DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Services
Food and Drug Administration

Memorandum

Date: 2/15

From: DMC (HFZ-401)

Subject: Premarket Notification Number(s): K122560/A001

To: Division Director: NE/DNPMD

The attached information has been received by the 510(k) DMC on the above referenced 510(k) submission(s). Since a final decision has been rendered, this record is officially closed.

Please review the attached document and return it to the DMC, with one of the statements checked below.

_____ Information does not change the status of the 510(k); no other action required by the DMC; please add to image file. (Prepare K-25) THIS DOES NOT APPLY TO TRANSFER OF OWNERSHIP. PLEASE BRING ANY TRANSFER OF OWNERSHIP TO POS.

_____ Additional information requires a new 510(k); however, the information submitted is incomplete; (Notify company to submit a new 510(k); [Prepare the K30 Letter on the LAN]

__X__ No response necessary (e.g., hard copy of fax for the truthful and accuracy statement, 510(k) statement, change of address, phone number, or fax number).

CLIA CATEGORIZATION refers to laboratory test system devices reviewed by the Division of Clinical Laboratory Devices (HFZ-440

_____ Information requires a **CLIA CATEGORIZATION**; the complexity may remain the same as the original 510(k) or may change as a result of the additional information (Prepare a CAT letter)

_____ Additional information requires a **CLIA CATEGORIZATION**; however, the information submitted is incomplete; (call or fax firm)

_____ No response necessary

This information should be returned to the DMC within 10 working days from the date of this Memorandum.

Reviewed by: _____

Date: 3/22/13

DNPMD Rec'd
FEB 20 2013
MYL

DNPMD Exited
MAR 24 2013
MYL

DLL 3/25

510(K) Summary, 510(k) K122550

Submitter: GN Otometrics A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
Registration number: 9612197
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173
(US) Phone: 847-534-2150
(US) Fax: 847-534-2153
Contact: Dan Sansonetti, Manager of Research and Development
Date Prepared: January 13, 2012

FEB 0 1 2013

1. **Identification of the Device:**
   Proprietary-Trade Name: **ICS Impulse**
   Classification Name: Class II, Product Codes: GWN and LXV, Device: Nystagmograph
   Common/Usual Name: Vestibular testing device

2. **Equivalent legally marketed devices**: Micromedical Technologies Inc. Vorteq, K891008 and Micromedical Technologies Inc. VisualEyes K964325.

3. **Description of the Device**: The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

4. **Indications for Use (intended use):** The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

5. **Safety and Effectiveness, comparison to predicate device**. This device has the same indications for use as the predicate device but employs different technology to accomplish the same tasks.

6. **Description of Testing:** The device passed UL Electrical Safety testing and EMC testing. Software validation and risk analysis was performed. Clinical testing compared test results to Scleral Search Coils test results. ICS Impulse adequately meets the design requirements and acceptance criteria.

7. Substantial Equivalence Chart

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interfaces	Firewire for Camera Data: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per UL2601 - IEC-60601.	Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

8. **Conclusion:** After analyzing bench testing, safety, EMC, software, and clinical validation testing we conclude that the ICS Impulse is as safe and effective as the predicate device, and has essentially the same indications for use, thus rendering it substantially equivalent to the predicate device.

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
10903 New Hampshire Avenue
Document Control Center – WO66-G609
Silver Spring, MD 20993-002

February 1, 2013

GN Otometrics A/S
% Mr. Daniel Kamm, P.E.
Principal Engineer
Kamm & Associates
8870 Ravello Court
Naples, FL 34114

Re: K122550
Trade/Device Name: ICS Impulse
Regulation Number: 21 CFR 882.1460
Regulation Name: Nystagmograph
Regulatory Class: II
Product Code: GWN, LXV
Dated: January 21, 2013
Received: January 23, 2013

Dear Mr. Daniel Kamm:

We have reviewed your Section 510(k) premarket notification of intent to market the device referenced above and have determined the device is substantially equivalent (for the indications for use stated in the enclosure) to legally marketed predicate devices marketed in interstate commerce prior to May 28, 1976, the enactment date of the Medical Device Amendments, or to devices that have been reclassified in accordance with the provisions of the Federal Food, Drug, and Cosmetic Act (Act) that do not require approval of a premarket approval application (PMA). You may, therefore, market the device, subject to the general controls provisions of the Act. The general controls provisions of the Act include requirements for annual registration, listing of devices, good manufacturing practice, labeling, and prohibitions against misbranding and adulteration. Please note: CDRH does not evaluate information related to contract liability warranties. We remind you, however, that device labeling must be truthful and not misleading.

If your device is classified (see above) into either class II (Special Controls) or class III (PMA), it may be subject to additional controls. Existing major regulations affecting your device can be found in the Code of Federal Regulations, Title 21, Parts 800 to 898. In addition, FDA may publish further announcements concerning your device in the Federal Register.

Please be advised that FDA's issuance of a substantial equivalence determination does not mean that FDA has made a determination that your device complies with other requirements of the Act or any Federal statutes and regulations administered by other Federal agencies. You must comply with all the Act's requirements, including, but not limited to: registration and listing (21 CFR Part 807); labeling (21 CFR Part 801); medical device reporting (reporting of medical device-related adverse events) (21 CFR 803); good manufacturing practice requirements as set

forth in the quality systems (QS) regulation (21 CFR Part 820); and if applicable, the electronic product radiation control provisions (Sections 531-542 of the Act); 21 CFR 1000-1050.

If you desire specific advice for your device on our labeling regulation (21 CFR Part 801), please go to http://www.fda.gov/AboutFDA/CentersOffices/CDRH/CDRHOffices/ucm115809.htm for the Center for Devices and Radiological Health's (CDRH's) Office of Compliance. Also, please note the regulation entitled, "Misbranding by reference to premarket notification" (21CFR Part 807.97). For questions regarding the reporting of adverse events under the MDR regulation (21 CFR Part 803), please go to http://www.fda.gov/MedicalDevices/Safety/ReportaProblem/default.htm for the CDRH's Office of Surveillance and Biometrics/Division of Postmarket Surveillance.

You may obtain other general information on your responsibilities under the Act from the Division of Small Manufacturers, International and Consumer Assistance at its toll-free number (800) 638-2041 or (301) 796-7100 or at its Internet address http://www.fda.gov/MedicalDevices/ResourcesforYou/Industry/default.htm.

Sincerely yours,

Joyce M. Whang

for Victor Krauthamer, Ph.D.
Acting Director
Division of Neurological
and Physical Medicine Devices
Office of Device Evaluation
Center for Devices and Radiological Health

Enclosure

Indications for Use

510(k) Number (if known): **K122550**

Device Name: **ICS Impulse**

Indications For Use:

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Prescription Use **X** AND/OR Over-The-Counter Use _____
(Part 21 CFR 801 Subpart D) (21 CFR 801 Subpart C)

(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Joyce M. Whang

(Division Sign Off)
Division of Neurological and Physical Medicine Devices (DNPMD)

510(k) Number **K122550**

K122550DIA1 K-57

GN OTOMETRICS A/S
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DENMARK DK-2630
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Schaumburg, IL 60173

This submission was prepared by:
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8870 Ravello Ct
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fda.help.now@gmail.com

February 6, 2013

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse K122550 (s001) Requested Additional Information: Replacement CDROM.

Dear Sir or Madam,

During a routine file inspection, I noticed that the WRONG FILE CONTENTS had been supplied to you as an e-Copy. Please accept my apologies. Enclosed is a replacement CDROM with the correct file contents.

The submission has already received marketing clearance, but I wanted to make sure your records are correct.

Respectfully submitted,

Daniel Kamm

Daniel Kamm, P.E.
(Regulatory Engineer, Submission Correspondent)

GN OTOMETRICS A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173

This submission was prepared by:
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8870 Ravello Ct
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Tel 239-234-1735
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fda.help.now@gmail.com

January 21, 2013

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse K122550 Requested Additional Information

CDROM Copy: A CDROM e-copy of this submission is hereby provided to serve as the second copy of the submission. It is in Acrobat format.

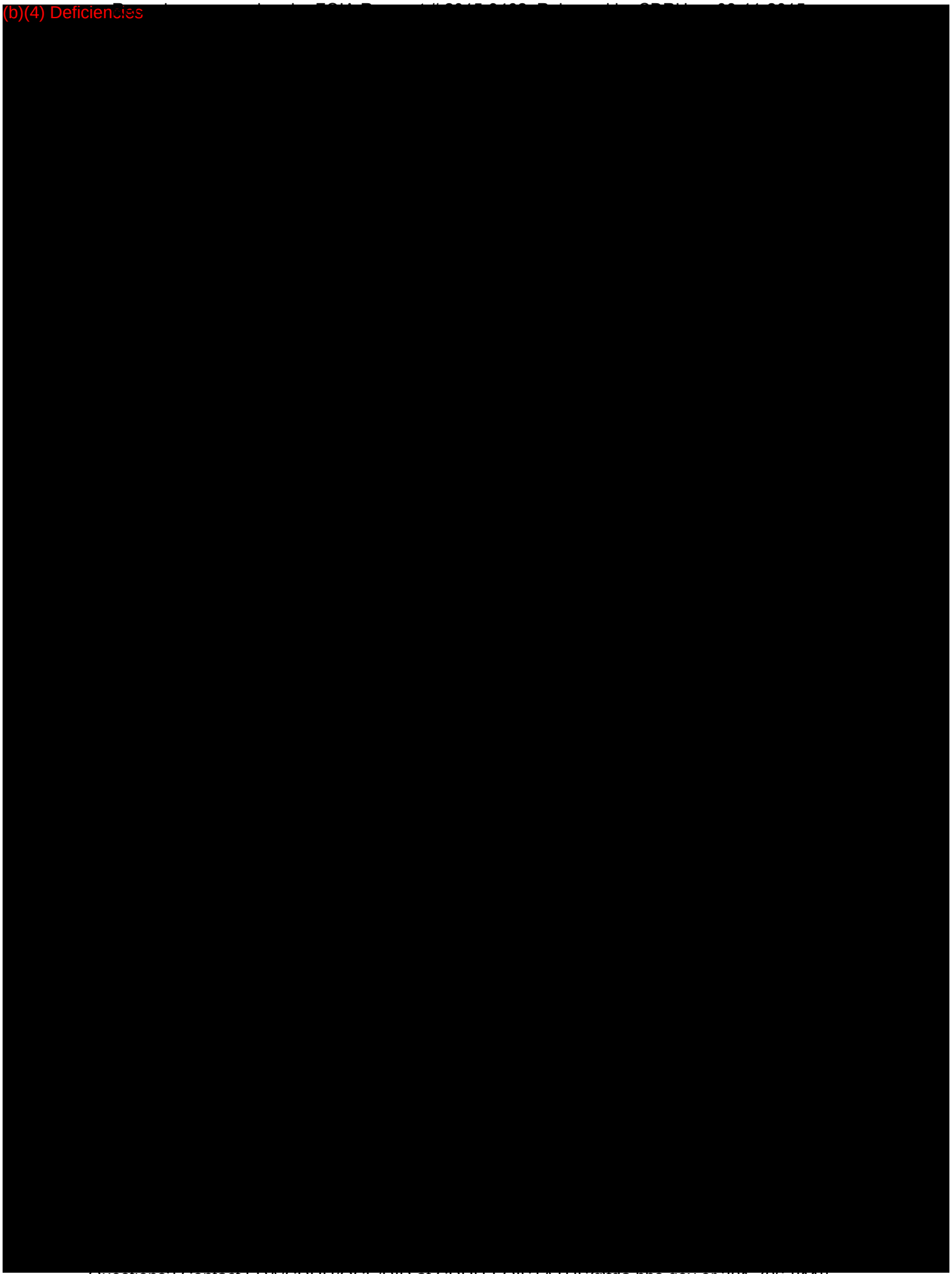
(b)(4) Deficiencies

[redacted]

(b)(4) Deficiencies

[redacted]

(b)(4) Deficiencies



(b)(4) Deficiencies

[redacted]

Respectfully submitted,

Respectfully submitted,

Daniel Kamm, P.E.
(Regulatory Engineer, Submission Correspondent)
Enclosures

Attachment Index

Attachment 1. Revised Indications for Use Statement

Indications for Use

510(k) Number (if known): K122550

Device Name: ICS Impulse

Indications For Use:

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Prescription Use X **AND/OR** **Over-The-Counter Use ___.**
(Part 21 CFR 801 Subpart D) **(21 CFR 807 Subpart C)**

(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Attachment 2. Revised 510(k) Summary

510(K) Summary, 510(k) K122550

Submitter: GN Otometrics A/S
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Taastrup,
DENMARK DK-2630
Registration number: 9612197
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173
(US) Phone: 847-534-2150
(US) Fax: 847-534-2153
Contact: Dan Sansonetti, Manager of Research and Development
Date Prepared: January 13, 2012

1. **Identification of the Device:**
   Proprietary-Trade Name: **ICS Impulse**
   Classification Name: Unclassified, Product Code: LXV, Device: Apparatus, vestibular analysis
   Common/Usual Name: Vestibular testing device

2. **Equivalent legally marketed devices**: Micromedical Technologies Inc. Vorteq, K891008 and Micromedical Technologies Inc. VisualEyes K964325.

3. **Description of the Device**: The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

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6. **Description of Testing:** The device passed UL Electrical Safety testing and EMC testing. Software validation and risk analysis was performed. Clinical testing compared test results to Scleral Search Coils test results. ICS Impulse adequately meets the design requirements and acceptance criteria.

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Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interfaces	Firewire for Camera Data: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per UL2601 - IEC-60601.	Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

8. Conclusion: After analyzing bench testing, safety, EMC, software, and clinical validation testing we conclude that the ICS Impulse is as safe and effective as the predicate device, and has essentially the same indications for use, thus rendering it substantially equivalent to the predicate device.

Attachment 3. Revised Users Manual Pages

1 Introduction

Congratulations! You are now the owner of a sophisticated new ICS Impulse system developed in collaboration with Drs. Ian Curthoys, Michael Halmagyi and others at University of Sydney.

To assist you in getting the most out of the ICS Impulse system, we have included this user manual and a training video. We hope you find it easy to use and that your use of the incorporated tips and information results in improved data collection accuracy as it relates to your assessment of vestibular-related disorders, test results reporting, and patient information retrieval.

1.1 Intended Use

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Note · *The ICS Impulse System is intended to be used only by qualified medical personnel.*

1.2 Intended User

This manual describes the use of the device in combination with the software. Readers are assumed to have prior knowledge of the medical and scientific facts underlying the procedure. For this reason, the examination methods are mentioned only to the degree that is necessary for a correct, safe application of the ICS Impulse System.

You can find more information in the ICS Impulse training video or at **www.headimpulse.com**.

3.1 Patient preparation

Warning · *A head impulse should not be performed on patients with a neck injury, or on patients who have been told by their physicians to limit or avoid neck movement activity.*

Prior to testing, provide the patient with these general recommendations:

- No alcohol for 48 hours before testing.
- Do not wear make-up around the eyes.
- Wear comfortable clothing.

3.2 Goggle preparation

3.2.1 Cleaning and maintenance

The ICS Impulse System equipment does not require preventive maintenance. Observe the following recommended guidelines regarding cleaning and maintenance.

- Keep the instrument clean and as free of dust as possible. Remove dust using a soft cloth or brush.
- If required, clean the goggle housing and interface box using a damp cloth moistened with a mild detergent and water solution. Do not allow any moisture to get inside the goggles.

Caution · *Never spray or immerse the goggle components with the cleaning solutions. This could contaminate the electronics and/or optics.*

- If required, clean the mirror using the supplied cleaning cloth. The presence of fingerprints on the mirror surfaces could cause inaccurate pupil detection.

Attachment 4 Gyroscope Data Sheets

(b)(4) Third Party Manufacturing Process

(b)(4) Third Party Manufacturing Process

(b)(4) Third Party Manufacturing Process

(b)(4) Third Party Manufacturing Process

(b)(4) Third Party Manufacturing Process

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Attachment 5 Camera Data Sheets

(b)(4) Camera Data Sheets

(b)(4) Camera Data Sheets

Attachment 6. Additional Risk/Hazard analysis

(b)(4) Risk Analysis

(b)(4) Risk Analysis

(b)(4) Risk Analysis

(b)(4) Risk Analysis

(b)(4) Risk Analysis

(b)(4) Risk Analysis

Attachmnet 7 Laser Data Sheet

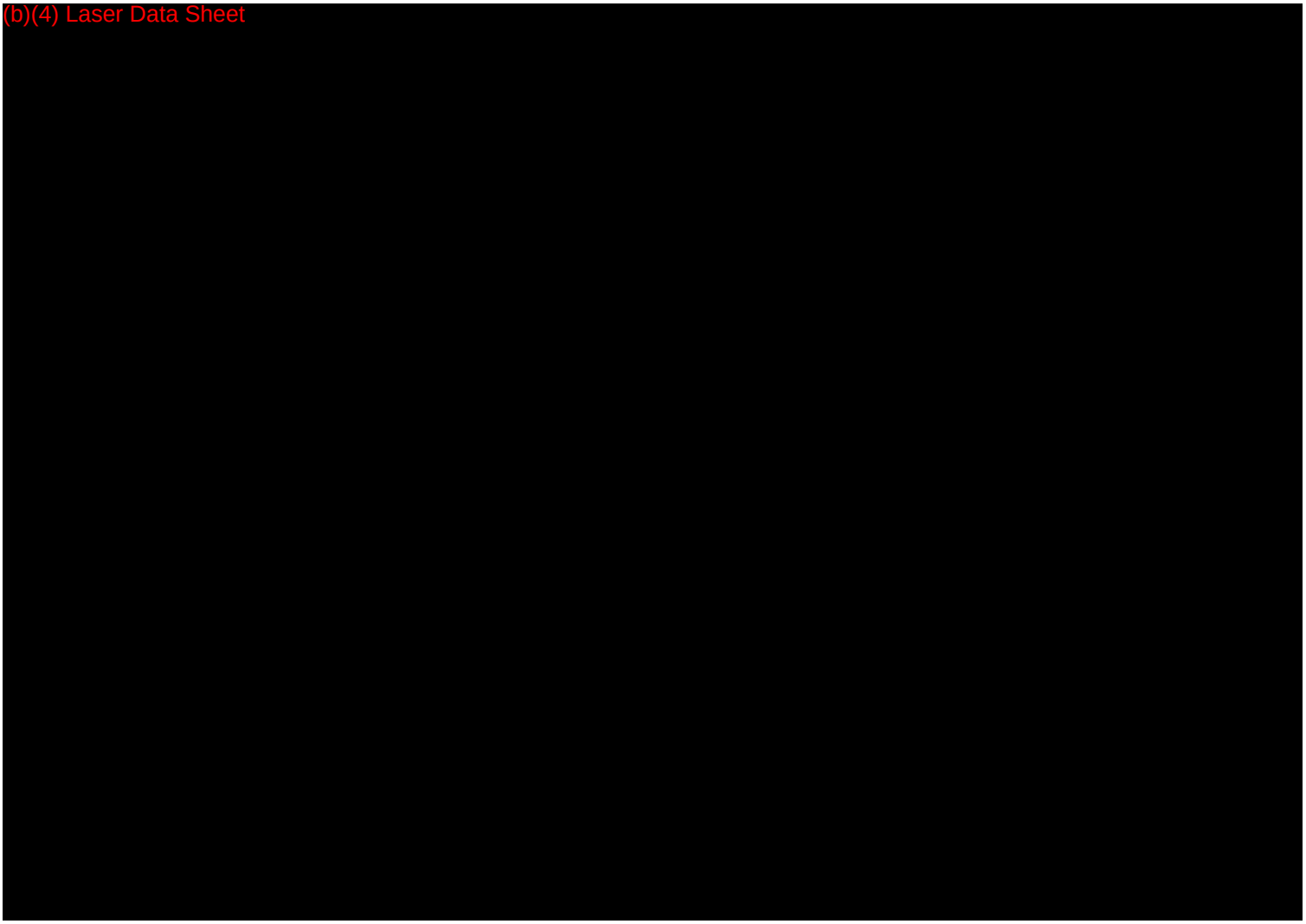
(b)(4) Laser Data Sheet

(b)(4) Laser Data Sheet

Photos of Test Jigs

Static

(b)(4) Laser Data Sheet

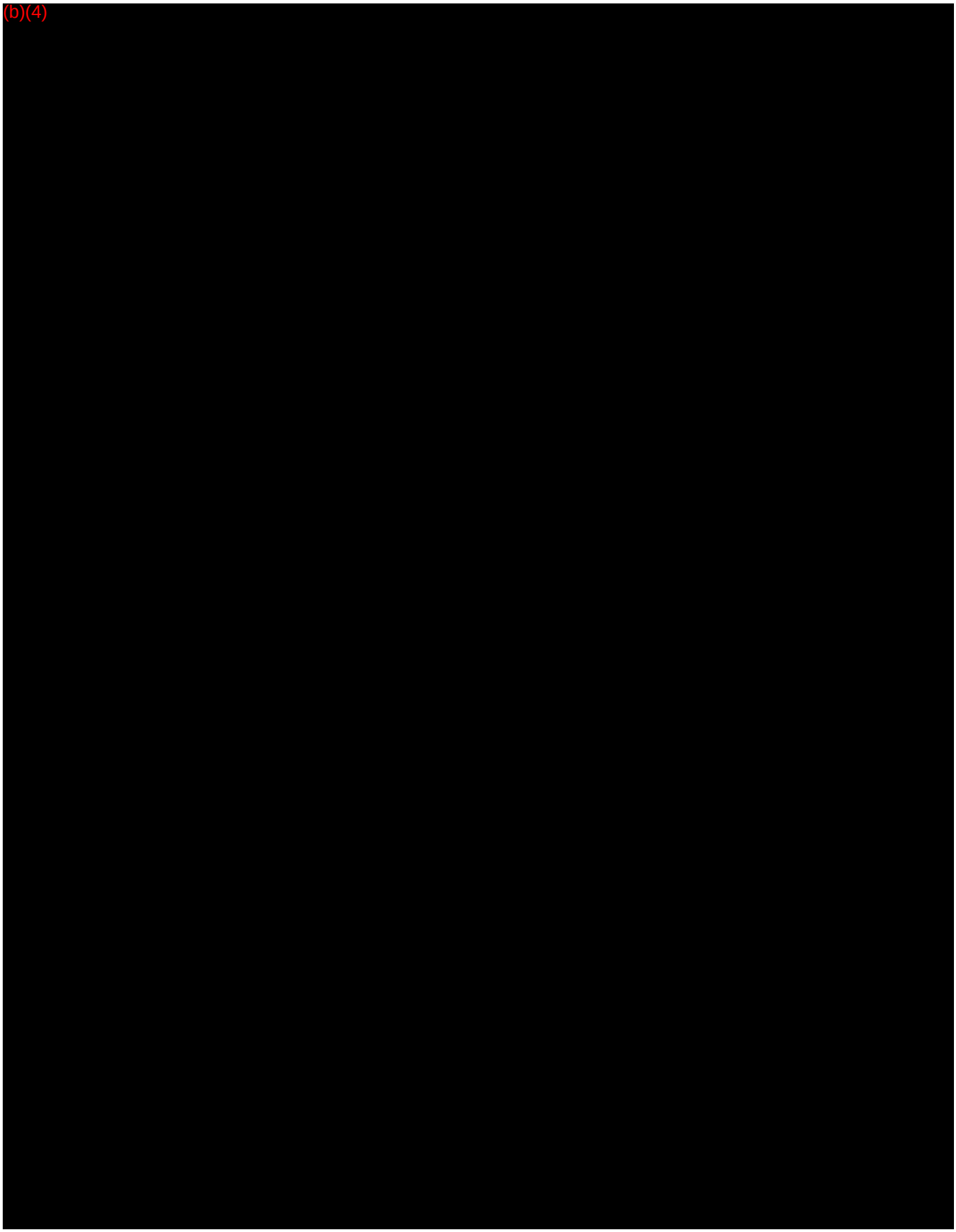


Dynamic

(b)(4) Laser Data Sheet

A3.8 VTM/VIDEYEO CALIBRATION AND VALIDATION

(b)(4)



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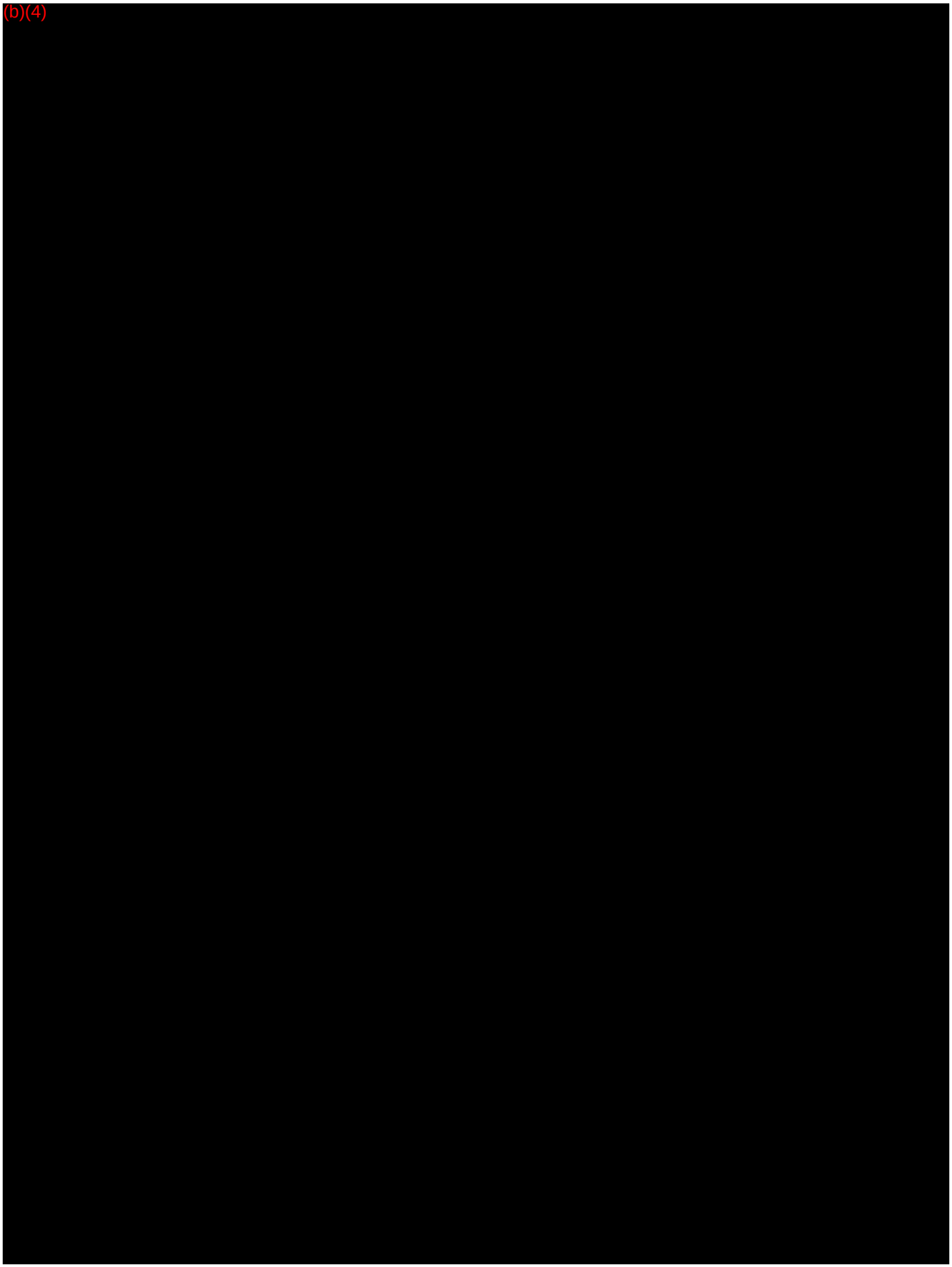

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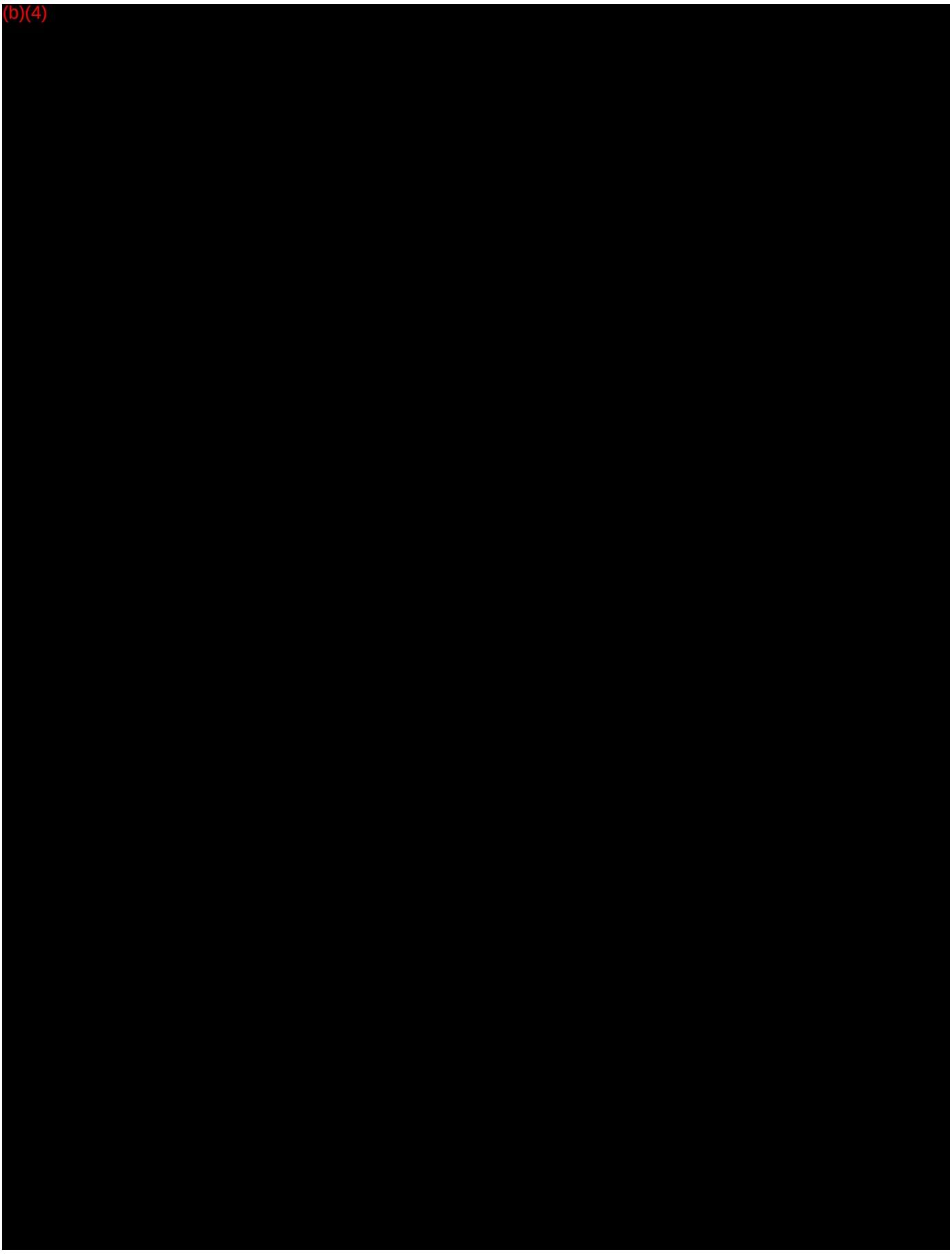

DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service

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10903 New Hampshire Avenue
Document Control Center – WO66-G609
Silver Spring, MD 20993-002

February 1, 2013

GN Otometrics A/S
% Mr. Daniel Kamm, P.E.
Principal Engineer
Kamm & Associates
8870 Ravello Court
Naples, FL 34114

Re: K122550
Trade/Device Name: ICS Impulse
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forth in the quality systems (QS) regulation (21 CFR Part 820); and if applicable, the electronic product radiation control provisions (Sections 531-542 of the Act); 21 CFR 1000-1050.

If you desire specific advice for your device on our labeling regulation (21 CFR Part 801), please go to http://www.fda.gov/AboutFDA/CentersOffices/CDRH/CDRHOffices/ucm115809.htm for the Center for Devices and Radiological Health's (CDRH's) Office of Compliance. Also, please note the regulation entitled, "Misbranding by reference to premarket notification" (21CFR Part 807.97). For questions regarding the reporting of adverse events under the MDR regulation (21 CFR Part 803), please go to http://www.fda.gov/MedicalDevices/Safety/ReportaProblem/default.htm for the CDRH's Office of Surveillance and Biometrics/Division of Postmarket Surveillance.

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Sincerely yours,

Joyce M. Whang

for Victor Krauthamer, Ph.D.
Acting Director
Division of Neurological
and Physical Medicine Devices
Office of Device Evaluation
Center for Devices and Radiological Health

Enclosure

Concurrence & Template History Page
[THIS PAGE IS INCLUDED IN IMAGE COPY ONLY]

Full Submission Number: K122550 / S1

For Office of Compliance Contact Information:

http://insideportlets.fda.gov:9010/portal/page?_pageid=197,415881&_dad=portal&_schema=PORTAL&org=318

For Office of Surveillance and Biometrics Contact Information:

http://insideportlets.fda.gov:9010/portal/page?_pageid=197,415881&_dad=portal&_schema=PORTAL&org=423

Digital Signature Concurrence Table	
Reviewer Sign-Off	
Branch Chief Sign-Off	
Division Sign-Off	Joyce M. Whang 2013.02.01 16:35:46 -05'00'

Template Name: K1(A) – SE after 1996
Edited: Latrina Crumlin 02/01/13

Template History:

Date of Update	By	Description of Update
7/27/09	Brandi Stuart	Added Updates to Boiler Table
8/7/09	Brandi Stuart	Updated HFZ Table
1/11/10	Diane Garcia	Liability/Warranty sentence added at bottom of 1st page
10/4/11	M. McCabe Janicki	Removed IFU sheet and placed in Forms
9/25/12	Edwena Jones	Added digital signature format

Indications for Use

510(k) Number (if known): **K122550**

Device Name: **ICS Impulse**

Indications For Use:

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Prescription Use **X** AND/OR Over-The-Counter Use _____
(Part 21 CFR 801 Subpart D) (21 CFR 801 Subpart C)

(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Joyce M. Whang

(Division Sign Off)
Division of Neurological and Physical Medicine Devices (DNPMD)

510(k) Number **K122550**

DEPARTMENT OF HEALTH & HUMAN SERVICES **Public Health Service**

Food and Drug Administration
Center for Devices and Radiological Health
Document Control Center WO66-G609
10903 New Hampshire Avenue
Silver Spring, MD 20993-0002

January 24, 2013

GN OTOMETRICS
C/O KAMM & ASSOCIATES
8870 RAVELLO CT
NAPLES, FLORIDA 34114
ATTN: DANIEL KAMM

510k Number: K122550

Product: ICS IMPULSE

The additional information you have submitted has been received.

We will notify you when the processing of this submission has been completed or if any additional information is required. Pleaseremember that all correspondence concerning your submission MUST be sent to the Document Mail Center at the above letterhead address. Correspondence sent to any address other than the one above will not be considered as part of your official premarket notification submission. Also, please note the new Blue Book Memorandum regarding Fax and E-mail Policy entitled, "Fax and E-Mail Communication with Industry about Premarket Files Under Review. Please refer to this guidance for information on current fax and e-mail practices at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089402.htm. On August 12, 2005 CDRH issued the Guidance for Industry and FDA Staff: Format for Traditional and Abbreviated 510(k)s. This guidance can be found at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm084365.htm. Please refer to this guidance for assistance on how to format an original submission for a Traditional or Abbreviated 510(k).

The Safe Medical Devices Act of 1990, signed on November 28, states that you may not place this device into commercial distribution until you receive a letter from FDA allowing you to do so. As in the past, we intend to complete our review as quickly as possible. Generally we do so in 90 days. However, the complexity of a submission or a requirement for additional information may occasionally cause the review to extend beyond 90 days. Thus, if you have not received a written decision or been contacted within 90 days of our receipt date you may want to check with FDA to determine the status of your submission.

Please ensure that whether you submit a 510(k) Summary as per 21 CFR 807.92, or a 510(k) Statement as per 21 CFR 807.93, it meets the content and format regulatory requirements.

If you have procedural questions, please contact the Division of Small Manufacturers International and Consumer Assistance (DSMICA) at (301)796-7100 or at their toll-free number (800)638-2041, or contact the 510k staff at (301)796-5640.

Sincerely,

510(k) Staff

Nichols, Karl *

From: (b)(6)
Sent: Thursday, January 24, 2013 2:53 PM
To: (b)(6)
Subject: FW: k122550/ S1 AI LETTER
Attachments: 2013_01_24_14_52_08.pdf

From (b)(6)
Sent: Thursday, January 24, 2013 2:53 PM
To: 'fda.help.now@gmail.com'
Cc: DCCLetters
Subject: k122550/ S1 AI LETTER

29

DEPARTMENT OF HEALTH & HUMAN SERVICES **Public Health Service**

Food and Drug Administration
Center for Devices and Radiological Health
Document Control Center WO66-G609
10903 New Hampshire Avenue
Silver Spring, MD 20993-0002

January 10, 2013

GN OTOMETRICS
C/O KAMM & ASSOCIATES
8870 RAVELLO CT
NAPLES, FLORIDA 34114
ATTN: DANIEL KAMM

510k Number: K122550
Product: ICS IMPULSE
On Hold As of 1/9/2013

We are holding your above-referenced Premarket Notification (510(k)) for 30 days pending receipt of the additional information that was requested by the Office of Device Evaluation. Please remember that all correspondence concerning your submission MUST cite your 510(k) number and be sent in duplicate to the Document Mail Center at the above letterhead address. Correspondence sent to any address other than the one above will not be considered as part of your official premarket notification submission. Also, please note the new Blue Book Memorandum regarding Fax and E-mail Policy entitled, "Fax and E-Mail Communication with Industry about Premarket Files Under Review. Please refer to this guidance for information on current fax and e-mail practices at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089402.htm.

The deficiencies identified represent the issues that we believe need to be resolved before our review of your 510(k) submission can be successfully completed. In developing the deficiencies, we carefully considered the statutory criteria as defined in Section 513(i) of the Federal Food, Drug, and Cosmetic Act for determining substantial equivalence of your device. We also considered the burden that may be incurred in your attempt to respond to the deficiencies. We believe that we have considered the least burdensome approach to resolving these issues. If, however, you believe that information is being requested that is not relevant to the regulatory decision or that there is a less burdensome way to resolve the issues, you should follow the procedures outlined in the "A Suggested Approach to Resolving Least Burdensome Issues" document. It is available on our Center web page at: http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/Overview/MedicalDeviceProvisionsofFDAModernizationAct/ucm136685.htm.

In accordance with 21 CFR 807.87(l), FDA may consider a 510(k) to be withdrawn if the submitter fails to provide additional information within 30 days of an Additional Information (AI) request. FDA generally permits submitters additional time to respond to such requests. FDA intends to automatically grant a maximum of 180 calendar days from the date of the AI request, even if the submitter has not requested an extension. Therefore, submitters are no longer required to submit written requests for extension. However, submitters should be aware that FDA intends to issue a notice of withdrawal under 21 CFR 807.87(l) if FDA does not receive, in a submission to the appropriate Document Control Center, a complete response to all of the deficiencies in the AI request within 180 calendar days of the date that FDA issued that AI request. In this instance, pursuant to 21 CFR 20.29, a copy of your 510(k) submission will remain in the Office of Device Evaluation. If you then wish to resubmit this 510(k) notification, a new number will be assigned and your submission will be considered a new premarket notification submission.

For further information regarding how various LDRs and industry actions that may be taken on 510(k)s should affect the review clock for purposes of meeting the Medical Device User Fee Amendments of 2012 (MDUFA III), to the Federal Food, Drug, and Cosmetic Act, you may refer to our guidance document entitled "Guidance for Industry and Food and Drug Administration Staff - FDA and Industry Actions on Premarket Notification (510(k)) Submissions: Effect on FDA Review Clock and Goals". You may review this document at <http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089735.htm>.

Please remember that the Safe Medical Devices Act of 1990 states that you may not place this device into commercial distribution until you receive a decision letter from FDA allowing you to do so.

If you have procedural questions, please contact the Division of Small Manufacturers International and Consumer Assistance (DSMICA) at (301)796-7100 or at their toll-free number (800)638-2041, or contact the 510k staff at (301)796-5640.

Sincerely yours,

Marjorie Shulman
Director, 510(k) Program
Premarket Notification Section
Office of Device Evaluation
Center for Devices and Radiological Health

Jones, Ashlee *

From: Microsoft Outlook
To: 'fda.help.now@gmail.com'
Sent: Thursday, January 10, 2013 9:13 AM
Subject: Relayed: K122550 Hold Letter

Delivery to these recipients or groups is complete, but no delivery notification was sent by the destination server:

'fda.help.now@gmail.com' (fda.help.now@gmail.com) <mailto:fda.help.now@gmail.com>

Subject: K122550 Hold Letter

82

DEPARTMENT OF HEALTH & HUMAN SERVICES **Public Health Service**

U.S. Food and Drug Administration
Center for Devices and Radiological Health
Document Control Center WO66-G609
10903 New Hampshire Avenue
Silver Spring, MD 20993-0002

August 21, 2012

GN OTOMETRICS
C/O KAMM & ASSOCIATES
8870 RAVELLO CT
NAPLES, FLORIDA 34114
ATTN: DANIEL KAMM

510k Number: K122550

Received: 8/21/2012

Product: ICS IMPULSE

The Food and Drug Administration (FDA), Center for Devices and Radiological Health (CDRH), has received the Premarket Notification, (510(k)), you submitted in accordance with Section 510(k) of the Federal Food, Drug, and Cosmetic Act(Act) for the above referenced product and for the above referenced 510(k) submitter. Please note, if the 510(k) submitter is incorrect, please notify the 510(k) Staff immediately. We have assigned your submission a unique 510(k) number that is cited above. Please refer prominently to this 510(k) number in all future correspondence that relates to this submission. We will notify you when the processing of your 510(k) has been completed or if any additional information is required. YOU MAY NOT PLACE THIS DEVICE INTO COMMERCIAL DISTRIBUTION UNTIL YOU RECEIVE A LETTER FROM FDA ALLOWING YOU TO DO SO.

Please remember that all correspondence concerning your submission MUST be sent to the Document Mail Center (DMC) at the above letterhead address. Correspondence sent to any address other than the one above will not be considered as part of your official 510(k) submission.

On September 27, 2007, the President signed an act reauthorizing medical device user fees for fiscal years 2008 - 2012. The legislation - the Medical Device User Fee Amendments of 2007 is part of a larger bill, the Food and Drug Amendments Act of 2007. Please visit our website at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/Overview/MedicalDeviceUserFeeandModernizationActMDUFMA/default.htm for more information regarding fees and FDA review goals. In addition, effective January 2, 2008, any firm that chooses to use a standard in the review of ANY new 510(k) needs to fill out the new standards form (Form 3654) and submit it with their 510(k). The form may be found at http://www.fda.gov/AboutFDA/ReportsManualsForms/Forms/default.htm.

We remind you that Title VIII of the Food and Drug Administration Amendments Act of 2007 (FDAAA) amended the PHS Act by adding new section 402(j) (42 U.S.C. § 282(j)), which expanded the current database known as ClinicalTrials.gov to include mandatory registration and reporting of results for applicable clinical trials of human drugs (including biological products) and devices. Section 402(j) requires that a certification form http://www.fda.gov/AboutFDA/ReportsManualsForms/Forms/default.htm accompany 510(k)/HDE/PMA submissions. The agency has issued a draft guidance titled: "Certifications To Accompany Drug, Biological

Product, and Device Applications/Submissions: Compliance with Section 402(j) of The Public Health Service Act, Added By Title VIII of The Food and Drug Administration Amendments Act of 2007" http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/HowtoMarketYourDevice/PremarketSubmissions/PremarketNotification510k/ucm134034.htm. According to the draft guidance, 510(k) submissions that do not contain clinical data do not need the certification form.

Please note the following documents as they relate to 510(k) review: 1) Guidance for Industry and FDA Staff entitled, "Interactive Review for Medical Device Submissions: 510(k)s, Original PMAs, PMA Supplements, Original BLAs and BLA Supplements". This guidance can be found at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089402.htm. Please refer to this guidance for information on a formalized interactive review process. 2) Guidance for Industry and FDA Staff entitled, "Format for Traditional and Abbreviated 510(k)s". This guidance can be found at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm084365.htm. Please refer to this guidance for assistance on how to format an original submission for a Traditional or Abbreviated 510(k).

In all future premarket submissions, we encourage you to provide an electronic copy of your submission. By doing so, you will save FDA resources and may help reviewers navigate through longer documents more easily. Under CDRH's e-Copy Program, you may replace one paper copy of any premarket submission (e.g., 510(k), IDE, PMA, HDE) with an electronic copy. For more information about the program, including the formatting requirements, please visit our web site at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/HowtoMarketYourDevice/PremarketSubmissions/ucm134508.html. In addition, the 510(k) Program Video is now available for viewing on line at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/HowtoMarketYourDevice/PremarketSubmissions/PremarketNotification510k/ucm070201.htm .

Please ensure that whether you submit a 510(k) Summary as per 21 CFR 807.92, or a 510(k) Statement as per 21 CFR 807.93, it meets the content and format regulatory requirements.

Lastly, you should be familiar with the regulatory requirements for medical devices available at Device Advice http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/default.htm. If you have questions on the status of your submission, please contact DSMICA at (301)796-7100 or the toll-free number (800)638-2041 , or at their internet address http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/default.htm. If you have procedural questions, please contact the 510(k) Staff at (301)796-5640.

Sincerely,

510(k) Staff

Grayson, Giovanna *

From: Microsoft Outlook
To: FDA.HELP.NOW@GMAIL.COM
Sent: Tuesday, August 21, 2012 2:05 PM
Subject: Relayed: ack letter

Delivery to these recipients or groups is complete, but no delivery notification was sent by the destination server:

FDA.HELP.NOW@GMAIL.COM (FDA.HELP.NOW@GMAIL.COM)

Subject: ack letter

K122330

Exhibit 3. 510(k) Cover Letter

GN Otometrics

GN OTOMETRICS A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173

This submission was prepared by:
Daniel Kamm, P.E.
Kamm & Associates
8870 Ravello Ct
Naples FL 34114
Tel 239-234-1735
Fax 206-260-4162
fda.help.now@gmail.com

August 13, 2012

FDA CDRH DMC
AUG 2 1 2012
Received

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse
Product Code: LXV, Device: Apparatus, vestibular analysis

Purpose of submission: This is to notify you of the intention by GN Otometrics to market a new but substantially equivalent, to a legally marketed, device: ICS Impulse vestibular analysis device. There have been no changes to the indications for use and many other essential characteristics as compared to the predicate devices.

Confidentiality: GN Otometrics considers the information contained in this submission to be confidential in nature (except for Exhibit 5 as required by the SMDA)

510(k) Summary: In response to the requirements addressed by the SMDA of 1990, a summary of the safety and effectiveness information upon which the substantial equivalence determination is based is enclosed. (Exhibit 5)

Email communications specifically authorized: Requests for additional information are hereby authorized and may be emailed to fda.help.now@gmail.com

CDRQM Copy: A CDROM of this submission is hereby provided to serve as the second copy of the submission. It is in Acrobat format. Training video images are also located on this disk.

Proprietary-Trade Name: ICS Impulse

Classification Name/Product Code:, Device: Apparatus, vestibular analysis. Product Code: LXV Review Panel: Ear Nose & Throat

Common/Usual Name: Vestibular analysis device

Equivalent legally marketed devices: Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.

Indications for Use (intended use): The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

Establishment address and registration number:

Manufacturer:
GN Otometrics A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
Registration number: 9612197

C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173
(US) Phone: 847-534-2150
(US) Fax: 847-534-2153

Design and Use of the Device

QUESTION	YES	NO
Is the device intended for prescription use (21 CFR 801 Subpart D)?	X	
Is the device intended for over-the-counter use (21 CFR 807 Subpart C)?		X
Does the device contain components derived from a tissue or other biologic source?		X
Is the device provided sterile?		X
Is the device intended for single use?		X
Is the device a reprocessed single use device?		X
If yes, does this device type require reprocessed validation data?		X
Does the device contain a drug?		X
Does the device contain a biologic?		X
Does the device use software?	X	
Does the submission include clinical information?	X	
Is the device implanted?		X

Traditional 510(k) Premarket Notification
Company: GN Otometrics (GN HEARING CARE CORPORATION)
Device: ICS Impulse (Model 1085)
Product Code: LXV, Device: Apparatus, vestibular analysis
Review Panel: Ear Nose & Throat

A Certified Copy of this submission has been attached in a CDROM to serve as copy #2. Acrobat format.

Exhibit 1. Medical Device User Fee Cover Sheet (Form FDA 3601)

Form Approved: OMB No. 0910-511. See Instructions for OMB Statement.

DEPARTMENT OF HEALTH AND HUMAN SERVICES
FOOD AND DRUG ADMINISTRATION
MEDICAL DEVICE USER FEE COVER SHEET

PAYMENT IDENTIFICATION NUMBER: (b)(4)
Write the Payment Identification number on your check.

A completed cover sheet must accompany each original application or supplement subject to fees. If payment is sent by U.S. mail or courier, please include a copy of this completed form with payment. Payment and mailing instructions can be found at: http://www.fda.gov/oc/mdufma/coversheet.html

1. COMPANY NAME AND ADDRESS (include name, street address, city state, country, and post office code)	2. CONTACT NAME
GN OTOMETRICS NORTH AMERICA 50 COMMERCE DRIVE Suite 180 SCHAUMBURG IL 60173 US	Daniel Kamm
1.1 EMPLOYER IDENTIFICATION NUMBER (EIN)	2.1 E-MAIL ADDRESS
*****9588	fda.help.now@gmail.com
	2.2 TELEPHONE NUMBER (include Area code)
	239-2341735
	2.3 FACSIMILE (FAX) NUMBER (Include Area code)
	206-2604162

3. TYPE OF PREMARKET APPLICATION (Select one of the following in each column; if you are unsure, please refer to the application descriptions at the following web site: http://www.fda.gov/oc/mdufma

Select an application type:	3.1 Select a center
[X] Premarket notification(510(k)); except for third party	[X] CDRH
[ ] 513(g) Request for Information	[ ] CBER
[ ] Biologics License Application (BLA)	3.2 Select one of the types below
[ ] Premarket Approval Application (PMA)	[X] Original Application
[ ] Modular PMA	Supplement Types:
[ ] Product Development Protocol (PDP)	[ ] Efficacy (BLA)
[ ] Premarket Report (PMR)	[ ] Panel Track (PMA, PMR, PDP)
[ ] Annual Fee for Periodic Reporting (APR)	[ ] Real-Time (PMA, PMR, PDP)
[ ] 30-Day Notice	[ ] 180-day (PMA, PMR, PDP)

4. ARE YOU A SMALL BUSINESS? (See the instructions for more information on determining this status)

[ ] YES, I meet the small business criteria and have submitted the required qualifying documents to FDA

[X] NO, I am not a small business

4.1 If Yes, please enter your Small Business Decision Number:

5. FDA WILL NOT ACCEPT YOUR SUBMISSION IF YOUR COMPANY HAS NOT PAID AN ESTABLISHMENT REGISTRATION FEE THAT IS DUE TO FDA. HAS YOUR COMPANY PAID ALL ESTABLISHMENT REGISTRATION FEES THAT ARE DUE TO FDA?

[X] YES (All of our establishments have registered and paid the fee, or this is our first device, and we will register and pay the fee within 30 days of FDA's approval/clearance of this device.)

[ ] NO (If "NO," FDA will not accept your submission until you have paid all fees due to FDA. This submission will not be processed; see http://www.fda.gov/cdrh/mdufma for additional information)

6. IS THIS PREMARKET APPLICATION COVERED BY ANY OF THE FOLLOWING USER FEE EXCEPTIONS? IF SO, CHECK THE APPLICABLE EXCEPTION.

[ ] This application is the first PMA submitted by a qualified small business, including any affiliates	[ ] The sole purpose of the application is to support conditions of use for a pediatric population
[ ] This biologics application is submitted under section 351 of the Public Health Service Act for a product licensed for further manufacturing use only	[ ] The application is submitted by a state or federal government entity for a device that is not to be distributed commercially

7. IS THIS A SUPPLEMENT TO A PREMARKET APPLICATION FOR WHICH FEES WERE WAIVED DUE TO SOLE USE IN A PEDIATRIC POPULATION THAT NOW PROPOSES CONDITION OF USE FOR ANY ADULT POPULATION? (If so, the application is subject to the fee that applies for an original premarket approval application (PMA).

[ ] YES [X] NO

PAPERWORK REDUCTION ACT STATEMENT

Public reporting burden for this collection of information is estimated to average 18 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to the address below.

Department of Health and Human Services, Food and Drug Administration, Office of Chief Information Officer, 1350 Piccard Drive, 4th Floor Rockville, MD 20850
[Please do NOT return this form to the above address, except as it pertains to comments on the burden estimate.]

8. USER FEE PAYMENT AMOUNT SUBMITTED FOR THIS PREMARKET APPLICATION

(b)(4) 27-Dec-2011

Form FDA 3601 (01/2007)

Exhibit 2. CDRH Premarket Review Submission Cover Sheet

DEPARTMENT OF HEALTH AND HUMAN SERVICES
FOOD AND DRUG ADMINISTRATION

CDRH PREMARKET REVIEW SUBMISSION COVER SHEET

Form Approval
OMB No. 9010-0120
Expiration Date: August 31, 2010.
See OMB Statement on page 5..

Date of Submission	User Fee Payment ID Number	FDA Submission Document Number (if known)
8/13/2012	(b)(4)	

SECTION A TYPE OF SUBMISSION

PMA	PMA & HDE Supplement	PDP	510(k)	Meeting
☐ Original Submission ☐ Premarket Report ☐ Modular Submission ☐ Amendment ☐ Report ☐ Report Amendment ☐ Licensing Agreement	☐ Regular (180 day) ☐ Special ☐ Panel Track (PMA Only) ☐ 30-day Supplement ☐ 30-day Notice ☐ 135-day Supplement ☐ Real-time Review ☐ Amendment to PMA &HDE Supplement ☐ Other	☐ Original PDP ☐ Notice of Completion ☐ Amendment to PDP	☒ Original Submission: ☒ Traditional ☐ Special ☐ Abbreviated (Complete section I, Page 5) ☐ Additional Information ☐ Third Party	☐ Pre-510(K) Meeting ☐ Pre-IDE Meeting ☐ Pre-PMA Meeting ☐ Pre-PDP Meeting ☐ Day 100 Meeting ☐ Agreement Meeting ☐ Determination Meeting ☐ Other *(specify):*

IDE	Humanitarian Device Exemption (HDE)	Class II Exemption Petition	Evaluation of Automatic Class III Designation (De Novo)	Other Submission
☐ Original Submission ☐ Amendment ☐ Supplement	☐ Original Submission ☐ Amendment ☐ Supplement ☐ Report ☐ Report Amendment	☐ Original Submission ☐ Additional Information	☐ Original Submission ☐ Additional Information	☐ 513(g) ☐ Other *(describe submission):*

Have you used or cited Standards in your submission? ☒ Yes ☐ No *(If Yes, please complete Section I, Page 5)*

SECTION B SUBMITTER, APPLICANT OR SPONSOR

Company / Institution Name	Establishment Registration Number *(if known)*		
GN OTOMETRICS	9612197		
Division Name *(if applicable)*	Phone Number *(including area code)*		
	(USA) 847 534-2150		
Street Address	FAX Number *(including area code)*		
Hoerskaetten 9	(USA) 847 534-2153		
City	State / Province	ZIP/Postal Code	Country
Taatrup		DK-2630	Denmark
Contact Name			
Dan Sansonetti			
Contact Title	Contact E-mail Address		
Manager of Research and Development	ddansonetti@gnotometrics.com		

SECTION C APPLICATION CORRESPONDENT (e.g., consultant, if different from above)

Company / Institution Name			
Kamm & Associates			
Division Name *(if applicable)*	Phone Number *(including area code)*		
	239-234-1735		
Street Address	FAX Number *(including area code)*		
8870 Ravello Ct	206-260-4162		
City	State / Province	ZIP/Postal Code	Country
Naples	FL	34114	USA
Contact Name			
Daniel Kamm, P.E.			
Contact Title	Contact E-mail Address		
Principal Engineer	fda.help.now@gmail.com		

SECTION D1 — REASON FOR APPLICATION - PMA, PDP, OR HDE

- ☐ Withdrawal
- ☐ Additional or Expanded Indications
- ☐ Request for Extension
- ☐ Post-approval Study Protocol
- ☐ Request for Applicant Hold
- ☐ Request for Removal of Applicant Hold
- ☐ Request to Remove or Add Manufacturing Site
- ☐ Change in design, component, or specification:
  - ☐ Software / Hardware
  - ☐ Color Additive
  - ☐ Material
  - ☐ Specifications
  - ☐ Other *(specify below)*
- ☐ Location change:
  - ☐ Manufacturer
  - ☐ Sterilizer
  - ☐ Packager
- ☐ Process change:
  - ☐ Manufacturing
  - ☐ Sterilization
  - ☐ Packaging
  - ☐ Other *(specify below)*
- ☐ Labeling change:
  - ☐ Indications
  - ☐ Instructions
  - ☐ Performance
  - ☐ Shelf Life
  - ☐ Trade Name
  - ☐ Other *(specify below)*
- ☐ Report Submission:
  - ☐ Annual or Periodic
  - ☐ Post-approval Study
  - ☐ Adverse Reaction
  - ☐ Device Defect
  - ☐ Amendment
- ☐ Response to FDA correspondence:
- ☐ Change in Ownership
- ☐ Change in Correspondent
- ☐ Change of Applicant Address
- ☐ Other Reason *(specify):*

SECTION D2 — REASON FOR APPLICATION - IDE

- ☐ New Device
- ☐ New Indication
- ☐ Addition of Institution
- ☐ Expansion / Extension of Study
- ☐ IRB Certification
- ☐ Termination of Study
- ☐ Withdrawal of Application
- ☐ Unanticipated Adverse Effect
- ☐ Notification of Emergency Use
- ☐ Compassionate Use Request
- ☐ Treatment IDE
- ☐ Continued Access
- ☐ Change in:
  - ☐ Correspondent / Applicant
  - ☐ Design / Device
  - ☐ Informed Consent
  - ☐ Manufacturer
  - ☐ Manufacturing Process
  - ☐ Protocol - Feasibility
  - ☐ Protocol - Other
  - ☐ Sponsor
- ☐ Report submission:
  - ☐ Current Investigator
  - ☐ Annual Progress Report
  - ☐ Site Waiver Report
  - ☐ Final
- ☐ Repose to FDA Letter Concerning:
  - ☐ Conditional Approval
  - ☐ Deemed Approved
  - ☐ Deficient Final Report
  - ☐ Deficient Progress Report
  - ☐ Deficient Investigator Report
  - ☐ Disapproval
  - ☐ Request Extension of Time to Respond to FDA
  - ☐ Request Meeting
  - ☐ Request Hearing
- ☐ Other Reason *(specify):*

SECTION D3 — REASON FOR SUBMISSION - 510(k)

- ☒ New Device
- ☐ Additional or Expanded Indications
- ☒ Change in Technology
- ☐ Other Reason *(specify):*

SECTION E ADDITIONAL INFORMATION ON 510(K) SUBMISSIONS

Product codes of devices to which substantial equivalence is claimed

1	LXV	2	GWN	3		4	
5		6		7		8	

Summary of, or statement concerning, safety and effectiveness information

☒ 510 (k) summary attached

☐ 510 (k) statement

Information on devices to which substantial equivalence is claimed *(if known)*

	510(k) Number		*Trade or Proprietary or Model Name*		*Manufacturer*
1	K891008	1	Vorteq	1	Micromedical Technologies
2	K964325	2	VisualEyes	2	Micromedical Technologies
3		3		3	
4		4		4	
5		5		5	

SECTION F PRODUCT INFORMATION - APPLICATION TO ALL APPLICATIONS

Common or usual name or classification

Device: Apparatus, vestibular analysis, Unclassified.

	Trade or Proprietary or Model Name for This Device		Model Number
1	ICS Impulse	1	Type 1085
2		2	
3		3	

FDA document numbers of all prior related submissions *(regardless of outcome)*

1	2	3	4	5	6
7	8	9	10	11	12

Data Included in Submission

☒ Laboratory Testing ☐ Animal Trials ☐ Human Trials

SECTION G PRODUCT CLASSIFICATION - APPLICATION TO ALL APPLICATIONS

Product Code: LXV

C.F.R. Section *(if applicable)*: Unclassified pre-amendment

Classification Panel: Ear, Nose, and Throat

Device Class

☐ Class I ☐ Class II ☐ Class III ☒ Unclassified

Indications *(from labeling)*

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

Note: Submission of this information does not affect the need to submit a 2891 or 2891a Device Establishment Registration form.	FDA Document Number *(if known)*

SECTION H MANUFACTURING / PACKAGING / STERILIZATION SITES RELATING TO A SUBMISSION

☒ Original ☐ Add ☐ Delete	FDA Establishment Registration Number: 9612197	☒ Manufacturer ☐ Contract Manufacturer ☐ Contract Sterilizer ☐ Repackager / Relabeler
Company / Institution Name: GN OTOMETRICS		Establishment Registration Number: 9612197
Division Name *(if applicable)*		Phone Number *(including area code)*: (US Number) 847 534-2150
Street Address: Hoerskaetten 9		FAX Number *(including area code)*: (US Number) 847 534-2153
City: Taatrup	State / Province	ZIP/Postal Code: DK-2630 / Country: Denmark
Contact Name: Dan Sansonetti	Contact Title: Manager of Research and Development	Contact E-mail Address: dsansonetti@gnotometrics.com

☒ Original ☐ Add ☐ Delete	FDA Establishment Registration Number: N/A	☐ Manufacturer ☒ Contract Manufacturer ☐ Contract Sterilizer ☐ Repackager / Relabeler
Company / Institution Name		Establishment Registration Number
Division Name *(if applicable)*		Phone Number *(including area code)*
Street Address		FAX Number *(including area code)*
City	State / Province	ZIP/Postal Code / Country
Contact Name	Contact Title	Contact E-mail Address

☒ Original ☐ Add ☐ Delete	FDA Establishment Registration Number	☐ Manufacturer ☒ Contract Manufacturer ☐ Contract Sterilizer ☐ Repackager / Relabeler
Company / Institution Name		Establishment Registration Number
Division Name *(if applicable)*		Phone Number *(including area code)*
Street Address		FAX Number *(including area code)*
City	State / Province	ZIP/Postal Code / Country
Contact Name	Contact Title	Contact E-mail Address

SECTION I			UTILIZATION OF STANDARDS		
Note: Complete this section if your application or submission cites standards or includes a *"Declaration of Conformity to a Recognized Standard"* statement.					
	Standards No.	Standards Organization	Standards Title	Version	Date
1	60601-1	IEC and UL	Medical Electrical Equipment - Part 1: General Requirements For Safety Medical Electrical Equipment - Collateral Standard: Safety Requirements For Medical Electrical Systems (5-4)	UL: 2003	2003
	Standards No.	Standards Organization	Standards Title	Version	Date
2	60601-1-2	IEC	Medical Electrical Equipment - Part 1: General Requirements For Safety 2. Collateral Standard: Electromagnetic Compatibility - Requirements And Tests (5-53)	2007	2007
	Standards No.	Standards Organization	Standards Title	Version	Date
3	60601-1-4	IEC	Collateral Standard: Programmable electrical medical systems (5-41)	1996 (First Ed.) + Am.1: 1999 (Consolidated 1.1 Ed.)	1999
	Standards No.	Standards Organization	Standards Title	Version	Date
4	60601-1-6	IEC	General requirements for safety - Collateral Standard: Usability (NR)	2010	2010
	Standards No.	Standards Organization	Standards Title	Version	Date
5	60825-1	IEC	Safety of laser products - Part 1: Equipment classification and requirements (12-220)	2007 2ed.	2007
6	Standards No.	Standards Organization	Standards Title	Version	Date
	62304	IEC	Medical device software – Software life cycle processes (13-8)	2006	2006
7	Standards No.	Standards Organization	Standards Title	Version	Date
	62366	IEC	Medical devices Application of usability engineering to medical devices (5-67)	2007 ed 1	2007
Please include any additional standards to be cited on a separate page. (SEE BELOW)					

Public reporting burden for this collection of information is estimated to average 0.5 hour per response, including the time for reviewing instructions, searching
existing data sources, gathering and maintaining the data needed, and completing reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including sugges ions for reducing this burden to:

Food and Drug Administration
CDRH (HFZ-342)
9200 Corporate Blvd.
Rockville, MD 20850

An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control

8	Standards No. 62471	Standards Organization IEC	Standards Title Photobiological safety of lamps and lamp systems (NR)	Version 2006 1ed	Date 2006
9	Standards No. ISO 10993-1	Standards Organization ISO	Standards Title Biological Evaluation of Medical Devices – Part 1: Evaluation and Testing (NR)	Version 2003	Date 2003
10	Standards No. ISO 10993-5	Standards Organization ISO	Standards Title Biological Evaluation of Medical Devices – Part 5: Tests for In Vitro Cytotoxicity (2-153)	Version 2009	Date 2009
11	Standards No. ISO 10993-10	Standards Organization ISO	Standards Title Biological Evaluation of Medical Devices – Part 10: Tests for Irritation and Delayed-type Hypersensitivity (2-87)	Version 2002/A1:2006	Date 2006
12	Standards No. ISO 10993-12	Standards Organization ISO	Standards Title Biological Evaluation of medical Devices – Part 12: Sample Preparation and Reference Materials (2-135)	Version 2007	Date 2007

NR = Not recognized by FDA.

Exhibit 3. 510(k) Cover Letter

GN OTOMETRICS A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173

This submission was prepared by:
Daniel Kamm, P.E.
Kamm & Associates
8870 Ravello Ct
Naples FL 34114
Tel 239-234-1735
Fax 206-260-4162
fda.help.now@gmail.com

August 13, 2012

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse
Product Code: LXV, Device: Apparatus, vestibular analysis

Purpose of submission: This is to notify you of the intention by GN Otometrics to market a new but substantially equivalent, to a legally marketed, device: ICS Impulse vestibular analysis device. There have been no changes to the indications for use and many other essential characteristics as compared to the predicate devices.

Confidentiality: GN Otometrics considers the information contained in this submission to be confidential in nature (except for Exhibit 5 as required by the SMDA)

510(k) Summary: In response to the requirements addressed by the SMDA of 1990, a summary of the safety and effectiveness information upon which the substantial equivalence determination is based is enclosed. (Exhibit 5)

Email communications specifically authorized: Requests for additional information are hereby authorized and may be emailed to fda.help.now@gmail.com

CDROM Copy: A CDROM of this submission is hereby provided to serve as the second copy of the submission. It is in Acrobat format. Training video images are also located on this disk.

Proprietary-Trade Name: ICS Impulse

Classification Name/Product Code:, Device: Apparatus, vestibular analysis. Product Code: LXV Review Panel: Ear Nose & Throat

Common/Usual Name: Vestibular analysis device

Equivalent legally marketed devices: Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.

Indications for Use (intended use): The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

Establishment address and registration number:

Manufacturer:
GN Otometrics A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
Registration number: 9612197

C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173
(US) Phone: 847-534-2150
(US) Fax: 847-534-2153

Design and Use of the Device

QUESTION	YES	NO
Is the device intended for prescription use (21 CFR 801 Subpart D)?	X	
Is the device intended for over-the-counter use (21 CFR 807 Subpart C)?		X
Does the device contain components derived from a tissue or other biologic source?		X
Is the device provided sterile?		X
Is the device intended for single use?		X
Is the device a reprocessed single use device?		X
If yes, does this device type require reprocessed validation data?		X
Does the device contain a drug?		X
Does the device contain a biologic?		X
Does the device use software?	X	
Does the submission include clinical information?	X	
Is the device implanted?		X

510(k) Screening Checklist

Title	Location	Present	Inadequate	N/A
MDUFMA Cover Sheet	Exhibit 1	X		
CDRH Premarket Review Submission Cover Sheet	Exhibit 2	X		
510(k) Cover Letter	Exhibit 3	X		
Indications for Use Statement	Exhibit 4	X		
510(k) Summary or 510(k) Statement	Exhibit 5	X		
Truthful and Accuracy Statement	Exhibit 6	X		
Class III Summary and Certification	Exhibit 7			X
Financial Certification or Disclosure Statement	Exhibit 8	X		
Declarations of Conformity and Summary Reports (Abbreviated 510(k)s)	Exhibit 9	X		
Executive Summary	Exhibit 10	X		
Device Description	Exhibit 11	X		
Substantial Equivalence Discussion	Exhibit 12	X		
Proposed Labeling	Exhibit 13	X		
Sterilization/Shelf Life	Exhibit 14			X
Biocompatibility	Exhibit 15	X		
Software	Exhibit 16	X		
Electromagnetic Compatibility/Electrical Safety	Exhibit 17	X		
Performance Testing – Bench	Exhibit 18	X		
Performance Testing – Animal	Exhibit 19			X
Performance Testing – Clinical	Exhibit 20 FORM FDA 3454, Certification: Financial Interests and Arrangements of Clinical Investigators N/A FORM FDA 3455, Disclosure: Financial Interests and Arrangements of Clinical Investigators: N/A	X		
FORM FDA 3654, Standards Data Report for 510(k)s	Exhibit 9	X		
Kit Certification	Not applicable			X

Respectfully submitted,

Daniel Kamm, P.E.
(Regulatory Engineer, Submission Correspondent)
Enclosures

Exhibit 4. Indications for Use Statement

Indications for Use

510(k) Number (if known): K11

Device Name: ICS Impulse

Indications For Use:

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.

Prescription Use X **AND/OR** **Over-The-Counter Use ___.**
(Part 21 CFR 801 Subpart D) **(21 CFR 807 Subpart C)**

(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Exhibit 5. 510(k) Summary or 510(k) Statement

See 510(k) Summary, below.

510(K) Summary, 510(k) K12

Submitter: GN Otometrics A/S
Hoerskaetten 9
Taastrup,
DENMARK DK-2630
Registration number: 9612197
C/O GN Otometrics North America
50 Commerce Dr Ste 180
Schaumburg, IL 60173
(US) Phone: 847-534-2150
(US) Fax: 847-534-2153
Contact: Dan Sansonetti, Manager of Research and Development
Date Prepared: August 13, 2012

1. **Identification of the Device:**
   Proprietary-Trade Name: **ICS Impulse**
   Classification Name: Unclassified, Product Code: LXV, Device: Apparatus, vestibular analysis
   Common/Usual Name: Vestibular testing device

2. **Equivalent legally marketed devices**: Micromedical Technologies Inc. Vorteq, K891008 and Micromedical Technologies Inc. VisualEyes K964325.

3. **Description of the Device**: The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

4. **Indications for Use** (intended use): The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use)..

5. **Safety and Effectiveness, comparison to predicate device**. This device has the same indications for use as the predicate device but employs different technology to accomplish the same tasks.

6. **Description of Testing:** The device passed UL Electrical Safety testing and EMC testing. Software validation and risk analysis was performed. Clinical testing compared test results to Scleral Search Coils test results. ICS Impulse adequately meets the design requirements and acceptance criteria.

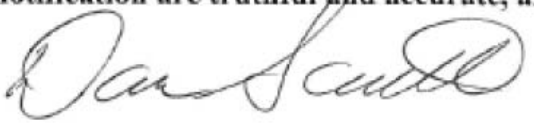
7. Substantial Equivalence Chart

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interfaces	Firewire for Camera Data: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per UL2601 - IEC-60601.	Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

8. Conclusion: After analyzing bench testing, safety, EMC, software, and clinical validation testing we conclude that the ICS Impulse is as safe and effective as the predicate device, and has essentially the same indications for use, thus rendering it substantially equivalent to the predicate device.

Exhibit 6. Truthful and Accuracy Statement as required per 21CFR807.87(k).

I certify that, in my capacity as Manager of Research and Development of GN Otometrics, I believe, to the best of my knowledge, that all data and information submitted in this premarket notification are truthful and accurate, and that no material fact has been omitted.



Dan Sansonetti
Date: August 13, 2012

Exhibit 7. Class III Summary and Certification

Not applicable. This is a Class II device.

Exhibit 8. Financial Certification or Disclosure Statement

See OMB Statement on Reverse. Form Approved: OMB No. 0910-0616, Expiration Date: 10-31-2011

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Food and Drug Administration

Certification of Compliance, under 42 U.S.C. § 282(j)(5)(B), with Requirements of ClinicalTrials.gov Data Bank (42 U.S.C. § 282(j))

(For submission with an application/submission, including amendments, supplements, and resubmissions, under §§ 505, 515, 520(m), or 510(k) of the Federal Food, Drug, and Cosmetic Act or § 351 of the Public Health Service Act.)

SPONSOR / APPLICANT / SUBMITTER INFORMATION

1. NAME OF SPONSOR/APPLICANT/SUBMITTER
GN Otometrics

2. DATE OF THE APPLICATION/SUBMISSION WHICH THIS CERTIFICATION ACCOMPANIES
Dec 7, 2011

3. ADDRESS *(Number, Street, State, and ZIP Code)*
Dybendalsvaenget 2
P.O. Box 119, Taastrup,
DENMARK DK-2630

4. TELEPHONE AND FAX NUMBERS *(Include Area Code)*
(Tel.) 847 534-2150
(Fax) 847 534-2153

PRODUCT INFORMATION

5. **FOR DRUGS/BIOLOGICS:** Include Any/All Available Established, Proprietary and/or Chemical/Biochemical/Blood/Cellular/Gene Therapy Product Name(s)
FOR DEVICES: Include Any/All Common or Usual Name(s), Classification, Trade or Proprietary or Model Name(s) and/or Model Number(s)
(Attach extra pages as necessary)

ICS Impulse Product Code: LXV, Device: Apparatus, vestibular analysis

APPLICATION / SUBMISSION INFORMATION

6. TYPE OF APPLICATION/SUBMISSION WHICH THIS CERTIFICATION ACCOMPANIES

☐ IND ☐ NDA ☐ ANDA ☐ BLA ☐ PMA ☐ HDE ☒ 510(k) ☐ PDP ☐ Other

7. INCLUDE IND/NDA/ANDA/BLA/PMA/HDE/510(k)/PDP/OTHER NUMBER *(If number previously assigned)*

8. SERIAL NUMBER ASSIGNED TO APPLICATION/SUBMISSION WHICH THIS CERTIFICATION ACCOMPANIES

CERTIFICATION STATEMENT / INFORMATION

9. CHECK ONLY ONE OF THE FOLLOWING BOXES *(See instructions for additional information and explanation)*

☐ A. I certify that the requirements of 42 U.S.C. § 282(j), Section 402(j) of the Public Health Service Act, enacted by 121 Stat. 823, Public Law 110-85, do not apply because the application/submission which this certification accompanies does not reference any clinical trial.

☒ B. I certify that the requirements of 42 U.S.C. § 282(j), Section 402(j) of the Public Health Service Act, enacted by 121 Stat. 823, Public Law 110-85, do not apply to any clinical trial referenced in the application/submission which this certification accompanies.

☐ C. I certify that the requirements of 42 U.S.C. § 282(j), Section 402(j) of the Public Health Service Act, enacted by 121 Stat. 823, Public Law 110-85, apply to one or more of the clinical trials referenced in the application/submission which this certification accompanies and that those requirements have been met.

10. IF YOU CHECKED BOX C, IN NUMBER 9, PROVIDE THE NATIONAL CLINICAL TRIAL (NCT) NUMBER(S) FOR ANY "APPLICABLE CLINICAL TRIAL(S)," UNDER 42 U.S.C. § 282(j)(1)(A)(i), SECTION 402(j)(1)(A)(i) OF THE PUBLIC HEALTH SERVICE ACT, REFERENCED IN THE APPLICATION/SUBMISSION WHICH THIS CERTIFICATION ACCOMPANIES *(Attach extra pages as necessary)*

NCT Number(s):

The undersigned declares, to the best of her/his knowledge, that this is an accurate, true, and complete submission of information. I understand that the failure to submit the certification required by 42 U.S.C. § 282(j)(5)(B), section 402(j)(5)(B) of the Public Health Service Act, and the knowing submission of a false certification under such section are prohibited acts under 21 U.S.C. § 331, section 301 of the Federal Food, Drug, and Cosmetic Act.
Warning: A willfully and knowingly false statement is a criminal offense, U.S. Code, title 18, section 1001.

11. SIGNATURE OF SPONSOR/APPLICANT/SUBMITTER OR AN AUTHORIZED REPRESENTATIVE (Sign)
Daniel Kamm

12. NAME AND TITLE OF THE PERSON WHO SIGNED IN NO. 11
(Name) Daniel Kamm, P.E.
(Title) Principal Engineer

13. ADDRESS *(Number, Street, State, and ZIP Code) (of person identified in Nos. 11 and 12)*
8870 Ravello Ct
Naples FL 34114

14. TELEPHONE AND FAX NUMBERS *(Include Area Code)*
(Tel.) 239-234-1735
(Fax) 206-260-4162

15. DATE OF CERTIFICATION
Dec 7, 2011

Form FDA 3674 (11/08) (FRONT)

Exhibit 9. Declarations of Conformity, FDA-3654 Forms

Declaration of Conformity:
Complies with voluntary standards:

1. Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA- 22.2 No. 601.1-M90, IEC60601-1, 2.ed.: 1988 + A1 + A2, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
2. IEC 60601-1-2: 2007 Collateral standard: Electromagnetic compatibility –Requirements and tests
3. IEC 60601-1-4 Collateral Standard: Programmable electrical medical systems (First Ed.) + Am.1: 1999 (Consolidated 1.1 Ed.)
4. IEC 60601-1-6 General requirements for safety - Collateral Standard: Usability
5. IEC 60825-1 Safety of laser products - Part 1: Equipment classification and requirements
6. IEC 62304 Medical device software – Software life cycle processes
7. IEC 62366 Medical devices Application of usability engineering to medical devices
8. IEC 62471 Photobiological safety of lamps and lamp systems
9. ISO 10993-1 Biological Evaluation of Medical Devices – Part 1: Evaluation and Testing
10. ISO 10993-5 Biological Evaluation of Medical Devices – Part 5: Tests for In Vitro Cytotoxicity
11. ISO 10993-10 Biological Evaluation of Medical Devices – Part 10: Tests for Irritation and Delayed-type Hypersensitivity
12. ISO 10993-12Biological Evaluation of medical Devices – Part 12: Sample Preparation and Reference Materials

Signature:

Dan Sansonetti, Manager of Research and Development
December 7, 2011

FDA-3654 Forms

Department of Health and Human Services
Food and Drug Administration

STANDARDS DATA REPORT FOR 510(k)s

(To be filled in by applicant)

This report and the Summary Report Table are to be completed by the applicant when submitting a 510(k) that references a national or international standard. A separate report is required for each standard referenced in the 510(k).

TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]

IEC 60601 1 MEDICAL ELECTRICAL EQUIPMENT PART 1: GENERAL REQUIREMENTS FOR SAFETY MEDICAL ELECTRICAL EQUIPMENT COLLATERAL STANDARD: SAFETY REQUIREMENTS FOR MEDICAL ELECTRICAL SYSTEMS

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 5-4	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

Title of guidance: _____

[1] The formatting convention for the title is: [SDO] [numeric identifier] [title of standard] [date of publication]

[2] Authority [21 U.S.C. 360d], www.fda.gov/cdrh/stdsprog.html

[3] http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfStandards/search.cfm

[4] The summary report should include: any adaptations used to adapt to the device under review (for example, alternative test methods); choices made when options or a selection of methods are described; deviations from the standard; requirements not applicable to the device; and the name and address of the test laboratory or certification body involved in conformance assessment to this standard. The summary report includes information on all standards utilized during the development of the device.

[5] The supplemental information sheet (SIS) is additional information which is necessary before FDA recognizes the standard. Found at http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfStandards/search.cfm

[6] The online search for CDRH Guidance Documents can be found at www.fda.gov/cdrh/guidance.html

Department of Health and Human Services
Food and Drug Administration

STANDARDS DATA REPORT FOR 510(k)s

(To be filled in by applicant)

This report and the Summary Report Table are to be completed by the applicant when submitting a 510(k) that references a national or international standard. A separate report is required for each standard referenced in the 510(k).

TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]

IEC 60601 1 2 MEDICAL ELECTRICAL EQUIPMENT PART 1: GENERAL REQUIREMENTS FOR SAFETY 2. COLLATERAL STANDARD: ELECTROMAGNETIC COMPATIBILITY REQUIREMENTS AND TESTS

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 5-53	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

Title of guidance: _____

[1] The formatting convention for the title is: [SDO] [numeric identifier] [title of standard] [date of publication]

[2] Authority [21 U.S.C. 360d], www.fda.gov/cdrh/stdsprog.html

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Department of Health and Human Services
Food and Drug Administration

STANDARDS DATA REPORT FOR 510(k)s

(To be filled in by applicant)

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TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
IEC 60601 1 4 Collateral Standard: Programmable electrical medical systems

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 5-41	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☐	☑
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance [6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

Title of guidance: _____

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TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
IEC 60601 1 6 General requirements for safety Collateral Standard: Usability

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☐	☑
FDA Recognition number [3]	# _____	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☐	☑
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance [6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

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TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
IEC 60825 1 Safety of laser products Part 1: Equipment classification and requirements

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 12-220	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance [6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

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STANDARD TITLE [1]
IEC 62304 Medical device software Software life cycle processes

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 13-8	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☐	☑
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

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STANDARD TITLE [1]

IEC 62366 Medical devices Application of usability engineering to medical devices

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 5-67	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☐	☑
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

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☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
IEC 62471 Photobiological safety of lamps and lamp systems

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☐	☑
FDA Recognition number [3] # _____		
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
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Is there an FDA guidance[6] that is associated with this standard?	☐	☑
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☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
ISO 10993 1 Biological Evaluation of Medical Devices Part 1: Evaluation and Testing

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☐	☑
FDA Recognition number [3] # _____		
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

Title of guidance: _____

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TYPE OF 510(K) SUBMISSION

☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
ISO 10993 5 Biological Evaluation of Medical Devices Part 5: Tests for In Vitro Cytotoxicity

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 2-153	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
Does this standard include more than one option or selection of tests? If yes, report options selected in the summary report table.	☐	☑
Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

Title of guidance: _____

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☑ Traditional ☐ Special ☐ Abbreviated

STANDARD TITLE [1]
ISO 10993 10 Biological Evaluation of Medical Devices Part 10: Tests for Irritation and Delayed type Hypersensitivity

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 2-87	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
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STANDARD TITLE [1]
ISO 10993 12 Biological Evaluation of medical Devices Part 12: Sample Preparation and Reference Materials

Please answer the following questions	*Yes*	*No*
Is this standard recognized by FDA [2]?	☑	☐
FDA Recognition number [3]	# 2-135	
Was a third party laboratory responsible for testing conformity of the device to this standard identified in the 510(k)?	☑	☐
Is a summary report [4] describing the extent of conformance of the standard used included in the 510(k)? If no, complete a summary report table.	☑	☐
Does the test data for this device demonstrate conformity to the requirements of this standard as it pertains to this device?	☑	☐
Does this standard include acceptance criteria? If no, include the results of testing in the 510(k).	☑	☐
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Were there any deviations or adaptations made in the use of the standard?	☐	☑
If yes, were deviations in accordance with the FDA supplemental information sheet (SIS) [5]?	☐	☐
Were deviations or adaptations made beyond what is specified in the FDA SIS? If yes, report these deviations or adaptations in the summary report table.	☐	☐
Were there any exclusions from the standard? If yes, report these exclusions in the summary report table.	☐	☑
Is there an FDA guidance[6] that is associated with this standard?	☐	☑
If yes, was the guidance document followed in preparation of this 510k?	☐	☑

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[4] The summary report should include: any adaptations used to adapt to the device under review (for example, alternative test methods); choices made when options or a selection of methods are described; deviations from the standard; requirements not applicable to the device; and the name and address of the test laboratory or certification body involved in conformance assessment to this standard. The summary report includes information on all standards utilized during the development of the device.

[5] The supplemental information sheet (SIS) is additional information which is necessary before FDA recognizes the standard. Found at http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfStandards/search.cfm

[6] The online search for CDRH Guidance Documents can be found at www.fda.gov/cdrh/guidance.html

Exhibit 10. Executive Summary

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. The key components are shown below:

- **High-speed camera (250 Hz)** The superior camera provides the best available technology for measuring fast eye movements. This provides the ability to record the eye while providing highfrequency head movements. Both covert and overt saccades can be identified.
- **Built-in gyroscopes** Dual-axis gyroscopes measure the head movement accurately allowing for direct comparison in head and eye velocities. They also provide instant feedback on the quality of the head impulse maneuver.
- **No slippage** Weighing 60 grams the goggle ensures no slippage and therefore providing accurate data collection without missing any important eye movements.
- **Built-in calibration laser** With a built-in calibration laser, the test can be performed anywhere there's a wall for calibration. There's no need for additional hardware.
- **USB/Firewire data transmission** The USB/Firewire interface box allows fast, accurate data transfer to the computer.

The testing is done via "head impulse testing." This is an ear-specific test that detects disorders of the vestibulo-ocular reflex and identifies which ear is affected in cases of peripheral vestibular loss. Patients with a vestibular loss will exhibit a corrective saccadic eye movement (a "catch-up" saccade) either during or after the head impulse and the gain of the head in comparison to the eye will not be equivalent. This is an assessment tool that provides quick, precise information about the vestibulo-ocular reflex to stimuli in the high-frequency range. It was first identified and described by Halmagyi and Curthoys in the 1988 article*, "A Clinical Sign of Canal Paresis." Said Halmagyi: "The eyes are the speedometers of the semicircular canals."

*See Bibliography in Exhibit 20.
Comparison table follows.

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interface	Firewire for Camera Other: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per IEC-60601.	Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA- 22.2 No. 601.1-M90, IEC60601-1, 2.ed.: 1988 + A1 + A2, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

Summary of Performance Testing

The following testing has been performed:

- Biocompatibility Testing: See Exhibit 15.
- Software Validation: See Exhibit 16
- Electrical Safety and EMC Testing: See Exhibit 17 for UL listing information and other safety test reports.
- Bench Testing: See Exhibit 18
- Clinical Testing: See Exhibit 20

Exhibit 11. Device Description (Detailed)

The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. The key components are shown below:

- **High-speed camera (250 Hz)** The superior camera provides the best available technology for measuring fast eye movements. This provides the ability to record the eye while providing highfrequency head movements. Both covert and overt saccades can be identified.
- **Built-in gyroscopes** Dual-axis gyroscopes measure the head movement accurately allowing for direct comparison in head and eye velocities. They also provide instant feedback on the quality of the head impulse manoeuvre.
- **No slippage** Weighing 60 grams the goggle ensures no slippage and therefore providing accurate data collection without missing any important eye movements.

- **Built-in calibration laser** With a built-in calibration laser, the test can be performed anywhere there's a wall for calibration. There's no need for additional hardware.
- **USB/Firewire data transmission** The USB/Firewire interface box allows fast, accurate data transfer to the computer.

The testing is done via "head impulse testing." This is an ear-specific test that detects disorders of the vestibulo-ocular reflex and identifies which ear is affected in cases of peripheral vestibular loss. Patients with a vestibular loss will exhibit a corrective saccadic eye movement (a "catch-up" saccade) either during or after the head impulse and the gain of the head in comparison to the eye will not be equivalent. This is an assessment tool that provides quick, precise information about the vestibulo-ocular reflex to stimuli in the high-frequency range. It was first identified and described by Halmagyi and Curthoys in the 1988 article*, "A Clinical Sign of Canal Paresis." Said Halmagyi: "The eyes are the speedometers of the semicircular canals."

*See Bibliography in Exhibit 20.

Minimum computer requirements

Note · Our recommended computer brands are Dell™, Hewlett Packard™, and Sony™. Installation problems were experienced with Acer™.

Operating System:	Windows XP 32-bit Professional SP3 or Windows 7 32-bit Professional or Windows 7 64-bit Professional
CPU	Intel i5 processor
Memory	32-bit (Windows XP or Windows 7): 4 GB, 64-bit (Windows 7): 6 GB
Disk Space	300 GB (3 GB of free disk space on C:\ drive for installed software)
Connectors	USB 2.0
IEEE	1394a (Firewire)
Note · It is preferable that the USB and Firewire ports are located on the same side of the computer.	
DVD Drive	DVD R/W
Monitor	1600 x 900 Screen resolution
Components	Mouse, keyboard
Internet access	An Internet connection on the ICS Impulse computer is strongly recommended during installation. It will reduce installation time by approximately 20 minutes

Technical Specifications

ICS Impulse System

Interface	USB 2.0 to PC 1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable
Type Identification	ICS Impulse System is Type 1085 from GN Otometrics A/S

Power Supply
Device is powered through USB - 5 V DC, 500 mA

Performance Characteristics	
Inputs	Monocular (Right eye only)
Sampling Rate	250 Hz for Head Impulse Test Option of 30, 60 or 120 Hz for Video Recording
Eye Tracking	100 pixels x 100 pixels.
Software	Windows Graphical User Interface; High Performance Analysis Software; Database Storage of Test Data; Sophisticated Patient and Test Data Management

Laser specifications	
Wavelength	Maximum 660 nm
Output power	Maximum 0.9 mW

Operating Mode	
Warm-up time	<1 min
Mode of operation	Continuous operation with intermittent loading Laser intermittence: 16%, Max. 5 min ON/ 25 min OFF Do not use the equipment in the presence of flammable anaesthetics (gases).

Operating Environment	
Temperature	+15° C to +29° C (59° F to +84.2° F)
Rel. Humidity	30 to 90%, non-condensing
Air Pressure	600 hPa to 1060 hPa
Operations at temperatures below -20° C (-4° F) or above +60° C (140° F) may cause permanent damage to the device.	

Storing and Handling	
Temperature	-20° C to +60° C (-4° F to +140° F)
Rel. Humidity	<90%, non-condensing
Air Pressure	500 hPa to 1060 hPa

Dimensions		
Goggles	Length	7.25 in (18.4 cm)
	Width	0.5 in (1.3 cm) to 1.75 in (4.4 cm)
	Height	1.75 in (4.4 cm)
Interface box	Length	5 in (12.7 cm)
	Width	2.75 in (7 cm)
	Height	1 in (2.5 cm)

Weight	
Goggles	2.1 oz (60 g)
Interface box	4.6 oz (130 g)

Calibration
Calibration of the system is not required

Classification
Class II Type B

Standards	
Safety	Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA-22.2 No. 601.1-M90, IEC60601-1, 2.ed.: 1988 + A1 + A2, IEC 62471, 1st.ed., IEC 60825-1, 2.ed.
EMC	IEC 6061-1-2: 2007

Accessories

Accessories		
Manuals/Videos	ICS Impulse Quick Guide	7-50-11300-EN 7-50-11300-DE 7-50-11300-ES 7-50-11300-FR 7-50-11300-IT
	ICS Impulse Training video	8-49-82700-US 8-49-82700-DE
Software	OTOsuite Vestibular	8-49-90300
Goggles	Face cushion 20/pkg	8-35-34400
	Strap assembly	8-35-34200
	Optical cleaning cloth ***Qty min 3***	7590527
	Fixation dot (2 sheets/pkg)	1-26-44000
Cables	USB 2.0 cable	8-71-79200
	1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable	8-71-89600
	Cable clip	8-35-36900
Case/Mount	Carrying case	8-35-36700
	Wall mount	8-62-45600

Type 1085, ICS Impulse System Specification

otometrics

Type 1085, ICS Impulse System Specification

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(b)(4)

3 Specifications

3.1 Software Specifications

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
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Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
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Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

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Document Title:	Type 1085 ICS Impulse System Specification	
Document No.:	(b)(4)	Revision: (b
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(b)(4)				

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
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Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

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Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
(b)(4)				

3.2 Hardware Specifications

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
HGD-1	HW-1	Angular Velocity Measurement	The design shall measure angular velocity with a sensitivity of 2mV/deg/s±10%about the X, Y, and Z axis'.	Type 1085, Hardware Verification Protocol 0-80-06520

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
HGD-2	HW-2	Laser Calibration Projection	The design must provide the ability to project two laser dots at an angle of 7.5° either side from the center of the head in the horizontal plane on to a flat surface (e.g., a wall). The resulting angle between the laser dots must be 15° +/- 1°.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-3	Monocular Design	The design shall include a right eye mounted eye / head movement detection system.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-4	Weight	The goggle weight should be less than 70 grams.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-4	HW-5	Power	The goggles will be powered by USB and will function over the full range of voltage supplied 4.75V and 5.25V with a maximum current of 0.5Amp. Note: The camera will be interfaced to the PC via 1394 (Firewire), but the power to the camera will be supplied via the PC's USB port.	Type 1085, Hardware Verification Protocol 0-80-06520
SDC-9	HW-6	Camera Field of View	The field of view shall be large enough to accommodate the entire eyeball to provide the ability for SW to center the eye movement on the PC screen. No mechanical controls shall be needed for image centering.	Type 1085, Hardware Verification Protocol 0-80-06520
(b)(4)				
HGD-4	HW-9	USB Interface	USB 2.0 is required to support the existing interface for acceleration and angular velocity data to the host computer.	Type 1085, Hardware Verification Protocol 0-80-06520

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
HGD-4	HW-10	1394 Video Interface	1394A (Firewire) is required to support the video interface to the host computer.	Type 1085, Hardware Verification Protocol 0-80-06520
HGD-4	HW-11	Goggle Interface	The interface shall have 1394 Tx, Rx, USB power and 6 connections for acceleration and Gyroscope data.	Type 1085, Hardware Verification Protocol 0-80-06520
SDC-9	HW-12	(b)(4)	[redacted]	Type 1085, Mechanical Verification Protocol 0-80-06521
SDC-9	HW-13	(b)(4)	[redacted]	Type 1085, Hardware Verification Protocol 0-80-06520
SDC-9	HW-14	(b)(4)	[redacted]	Type 1085, Hardware Verification Protocol 0-80-06520
SDC-9	HW-15	(b)(4)	[redacted]	Type 1085, Mechanical Verification Protocol 0-80-06521
SDC-9	HW-16	(b)(4)	[redacted]	Type 1085, Mechanical Verification Protocol 0-80-06521

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
SDC-9	HW-17	(b)(4)	(b)(4)	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-18	Goggle Strap	The goggle strap shall be 19mm wide, plus or minus 2mm.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-19	Goggle Strap	The overall minimum adjustable length shall be 230 mm, plus or minus 10mm.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-20	Goggle Strap	The overall maximum adjustable length shall be 405 mm, plus or minus 10mm.	Type 1085, Mechanical Verification Protocol 0-80-06521
HGD-3	HW-21	Goggle Strap	Strap will be constructed of an elastic type stretchable material.	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-1	HW-22	Materials Goggle	ABS Plastic shall be used in the Goggle, Camera Housing, Buckle, and Strap Clip design.	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-2	HW-23	Interface Box Material	An Interface Box shall be provided to house the Interface Electronics between the goggle and the PC.	Type 1085, Mechanical Verification Protocol 0-80-06521
CO-1	HW-24	Interface Box Weight	The Interface Box weight shall be no greater than 150 grams.	Type 1085, Mechanical Verification Protocol 0-80-06521

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
MA-1 Hazard Risk Analysis	HW-25	Face Foam Biocompatibility	Goggle face foam material that comes in contact with the patient must be biocompatible (and latex-free).	Type 1085, Face Cushion Biocompatibility Test Protocol, 0-80-06532
MA-1 Hazard Risk Analysis	HW-26	Head Strap / Plastic Component Biocompatibility	Head Strap material must be biocompatible (and latex-free).	Type 1085, Head Strap Biocompatibility Test Protocol, 0-80-06534
MA-1	HW-27	Goggle Color	Goggle color will be the anthracite grey used in the Chartr 200 with white lettering. (Anthracite Grey, RAL 7016)	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-1	HW-28	Camera Housing Color, exterior	The Camera Housing Color on the exterior will be the anthracite grey used in the Chartr 200. (Anthracite Grey, RAL 7016)	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-1	HW-29	Camera Housing Color, interior	The Camera Housing Color on the interior shall be (Anthracite Grey, RAL 7016). A matte finish is required to reduce glare.	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-1	HW-30	Buckle and Strap Clip Color	The Buckle and Strap Clip color shall be the Anthracite Grey used in the Chartr 200. (Anthracite Grey, RAL 7016)	Type 1085, Mechanical Verification Protocol 0-80-06521
MA-2	HW-31	Interface Box Color	The Interface Box color will be the Chartr 200 blue with white lettering (Genrian Blue, RAL 5010)	Type 1085, Mechanical Verification Protocol 0-80-06521
CL-1 Hazard Risk Analysis	HW-32	Cleaning New Goggles	Face cushion needs to be constructed of a material that is disposable.	Type 1085, Mechanical Verification Protocol 0-80-06521

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
MA-1	HW-33	Goggle Size	(b)(4)	Type 1085, Mechanical Verification Protocol 0-80-06521
SA-1 Hazard Risk Analysis	HW-34	Laser Hazard	The calibration laser shall not emit more than 1mW of optical power for class II. Design must be compliant with IEC 60825-1 Safety of Laser Products.	Type 1085, IR LED/Laser Verification Test Report 0-80-06522
SA-1 Hazard Risk Analysis	HW-35	IRLED Safety	The IR illumination LEDs shall not emit more than 30mW of radiant optical power each. Design must be compliant with IEC 60825-1 Safety of Laser Products.	Type 1085, IR LED/Laser Verification Test Report, 0-80-06522
SA-1 Hazard Risk Analysis	HW-36	Goggle Strap,	The goggle strap material must be compliant with ISO 10993-10 Biological evaluation of medical devices - - Part 10: Tests for irritation and sensitization.	Type 1085 Head Strap Biocompatibility Test Protocol, 0-80-06534
SA-1 Hazard Risk Analysis	HW-37	Face Cushion	The face cushion material must be compliant with ISO 10993-10 Biological evaluation of medical devices - - Part 10: Tests for irritation and sensitization.	Type 1085 Face Cushion Biocompatibility Test Protocol, 0-80-06532
SA-1 Hazard Risk Analysis	HW-38	Safety and Regulatory Requirements	The device must be compliant with EN 60601-1. Medical Electrical Equipment. Part 1: General requirements for safety.	Type 1085 Electrical and Mechanical Safety Test Report, 0-80-06524
SA-1 Hazard Risk Analysis	HW-39	Safety and Regulatory Requirements	The device must be compliant with CAN/CSA-C22.2 NO 601.1-90 Medical Electrical Equipment. Part 1: General requirements for safety.	Type 1085 Electrical and Mechanical Safety Test Report, 0-80-06524
SA-1 Hazard Risk Analysis	HW-40	Safety and Regulatory Requirements	The device must be compliant with EN 60601-1-1. Medical Electrical Equipment. Part 1: General requirements for safety. 1. Collateral standard: Safety requirements for medical electrical systems.	Type 1085 EMI/EMC Test Report, 0-80-06523

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
SA-1 Hazard Risk Analysis	HW-41	Safety and Regulatory Requirements	The device must be compliant with EN 60601-1-2 Medical Electrical Equipment. Part 1-2: General requirements for safety – Collateral standard: Electromagnetic compatibility – Requirements and test.	Type 1085 EMI/EMC Test Report, 0-80-06523
SA-1 Hazard Risk Analysis	HW-42	Safety and Regulatory Requirements	The device must be compliant with EN 60601-1-4. Medical Electrical Equipment. Part 1: General requirements for safety. 4. Collateral standard: Programmable electrical medical systems (Will soon be replaced by EN 62304).	Type 1085 60601-1-4 Test Report 0-80-06526
SA-1 Hazard Risk Analysis	HW-43	Safety and Regulatory Requirements	The device must be compliant with EN 60601-1-6 Medical electrical equipment. General requirements for basic safety and essential Performance. Collateral standard. Usability.	Type 1085 60601-1-4 Test Report 0-80-06525
SA-1 Hazard Risk Analysis	HW-44	Safety and Regulatory Requirements	The device must be compliant with ISO 10993-10 Biological evaluation of medical devices -- Part 10: Tests for irritation and sensitization.	Type 1085 Face Cushion Biocompatibility Test Protocol, 0-80-06532 , Type 1085 Head Strap Biocompatibility Test Protocol, 0-80-06534
SA-1 Hazard Risk Analysis	HW-45	Safety and Regulatory Requirements	The device must be compliant with IEC 60068-2 Environmental testing - Part 2: Tests WEEE Directive 2002/96/EC EN 980 Symbols for use in the labeling of medical devices.	Type 1085 Electrical and Mechanical Safety Test Report 0-80-06524
SA-1 Hazard Risk Analysis	HW-46	Safety and Regulatory Requirements	The device must be compliant with ISO 15223 Medical devices - Symbols to be used with medical device labels, labeling, and information to be supplied.	Type 1085 Electrical and Mechanical Safety Test Report 0-80-06524
SA-1 Hazard Risk Analysis	HW-47	Safety and Regulatory Requirements	The device must be compliant with EN 50419 Marking of electrical and electronic equipment in accordance with article 11(2) of Directive 2002/96/EC (WEEE).	Type 1085 Electrical and Mechanical Safety Test Report 0-80-06524

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
QR-1	HW-48	Data Collection Accuracy	(b)(4) [redacted]	Type 1085 Data Accuracy Validation Test Protocol 0-80-05613
QR-2	HW-49	Quality and Reliability *New Goggles*	The ICS Impulse system components selected are expected to perform for the 5-year lifetime of the product.	Type 1085, Hardware Verification Protocol 0-80-06520 Type 1085 Climatic Test Protocol 0-80-06527 Type 1085 Rough Handling Test Protocol, Device Freefall 0-80-06528

3.3 Other Specifications

3.3.1 Packaging Specifications

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
PA-1	PS-1	Hardware Packaging	Packaging designed to survive (3) consecutive drops on three different sides of the box from a height of one meter. The device should sustain no damage.	Type 1085, Rough Handling Test Protocol, Package Freefall 0-80-06540
PA-2	PS-2	Software Packaging	Delivered on CD-ROM in DVD case.	Type 1085, Mechanical Verification Protocol 0-80-06521

3.3.2 Labeling Specifications

Origin (e.g., DR No.)	Spec. No.	Spec. Name	Spec. Description	Verification Test Protocol
LA1-4 Hazard Risk Analysis	LS-1	Software Labeling	ICS Impulse software	Type 1085, Mechanical Verification Protocol 0-80-06521
LA1-4 Hazard Risk Analysis	LS-2	Otometrics Identification and Logo	Identification and Logo shall be provided on the goggle and the Interface Box.	Type 1085, Mechanical Verification Protocol 0-80-06521
LA1-4 Hazard Risk Analysis	LS-3	Model Number, Serial Number, CE Mark and other regulatory identifications	Model Number, Serial Number, CE Mark and other regulatory identifications shall be provided on the product.	Type 1085, Mechanical Verification Protocol 0-80-06521
LA1-4 Hazard Risk Analysis	LS-4	OP Manual	The ICS Impulse manual will be installed via the software CD. The manual will be accessible from the software by clicking on a button.	Type 1085, Mechanical Verification Protocol 0-80-06521

Exhibit 12. Substantial Equivalence Discussion
Detailed comparison chart

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	GN Otometrics ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor (gyroscope) mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing.	ICS Impulse is a monocular goggle. Built into the goggle is an angular velocity sensor (gyroscope) which measures head movement and a video camera which measures eye movement. The whole goggle system is lightweight (60g) to minimize goggle slippage during VOR testing.
Photo		
Interface	Firewire for Camera Other: Not specified	IEEE-1394a Firewire® for Camera USB 2.0 for Data Transfer
Electrical safety	Goggles - UL 60601-1, IEC 601-1, EN 60601-1 Vorteq Sensor - Electrical Safety per IEC-60601, UL 2601-1	Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA-22.2 No. 601.1-M90, IEC60601-1, 2.ed: 1988 + A1 + A2, IEC 62471, 1st.ed, IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.
Weight	1.6 oz – Vorteq Sensor (gyroscope) mounted on goggle 10.5 oz (298g) – goggle without cables	2.1 oz (60g) – goggles
Eye Measurement Cameras (frame rate)	Firewire Camera - 100 Hz	Firewire Camera – 250 Hz

Characteristic	**Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.**	**GN Otometrics ICS Impulse**
Head Measurement Gyroscope	Silicon Ring Angular Rate Sensor measures head velocity	3 Axis Angular Velocity sensor for X,Y, and Z planes
Mirrors	Visible spectrum – transparent to patient Infrared Spectrum – reflective for video capture	Identical
Face Cushion	Cleanable	Disposable
VOR Assessment	Horizontal and Vertical	Horizontal only
Target population	Children and Adults	Identical but Goggles must fit patient so the device is not intended for very young children.
Where Used	Hospitals, ENT/Neurology/Audiology Clinics	Identical
Operator	Trained Personnel	Identical
Computer Platform	Mid-tower PC or laptop	Laptop recommended for portability (any computer that meets the minimum specifications provided)
Power Source	Mains	Powered thru USB
Operating System Platform	Windows XP and Windows 7	Windows XP 32-bit Professional SP3 or Windows 7 32-bit Professional or Windows 7 64-bit Professional
User's Manual	Provided on CD and within software	Printed and provided within software

Predicate Similarities to Impulse

- Use Video Goggles and collect data monocularly
- Require calibration at the beginning of the test
- Patient sits 1 meter from stimulus. Stimulus for Vorteq is the center dot on the lightbar. Stimulus for Impulse is the fixation dot.
- Patients head is moved side to side and the goggles record the head and eye movement
- Head movement is similar in amplitude (how much head moves from side to side)
- Head movement is a quick motion
- Gain is measured (velocity of eye to head)
- Normative ranges are provided

Differences

- Head movement – Vorteq you have option of active (patient moves their head) or passive (tester moves patient's head) Impulse only uses passive.
- Head movement – Vorteq you are shaking your head side to side. Impulse the tester moves your head from point A to point B.

Substantial Equivalence Decision-Making Process

The conclusion that the ICS Impulse is substantially equivalent to the identified predicate devices is reached through substantial equivalence decision-making processes described in CDRH's August 12, 2005 Guidance: Format for Traditional and Abbreviated 510(k)s. The following questions are from the decision-making process flow chart illustrated in the Guidance on the Center for Devices and Radiological Health's Premarket Notification Review Program (K86-3).

Does new device have same indication statement? YES

Do the differences alter the intended therapeutic/diagnostic/etc. effect? NO

New device has the same intended use and may be 'substantially equivalent'? Yes.

Does the new device have the same technological characteristics, e.g. design, materials, etc.? YES, similar materials and configurations.

Are the descriptive characteristics precise enough to ensure equivalence? YES

Conclusion

Based upon the intended use, and upon the similarity of materials, product configuration and administration, it can be concluded the ICS Impulse is substantially equivalent to the identified predicate device in terms of intended use, safety and effectiveness.

Exhibit 13. Proposed Labeling

Predicate Brochure
Brochure
Device Labels
Operator Manual

Predicate Brochure (Micromedical Technologies Vorteq)

VORTEQ®

To Preserve and Improve Balance

Active Head Rotation & DVA-T Dynamic Visual Acuity Test

VORTEQ / DVA-T PROVIDES THE UNIQUE TESTING NECESSARY TO ACCURATELY MEASURE A PATIENT'S VOR AND DYNAMIC VISUAL ACUITY DURING NORMAL ACTIVE HEAD MOVEMENTS.

(AHR) ACTIVE HEAD ROTATION VOR

The Vestibular Ocular Reflex (VOR) serves to stabilize gaze during head movements by generating equal and opposite compensatory eye movements.

(DVAT) DYNAMIC VISUAL ACUITY TEST VVOR

A performance measure of the Visual Vestibular Ocular Reflex (VVOR), Dynamic Visual Acuity (DVA) is essential for retinal image stability when performing tasks where relative motion exists between the individual and the visual information which they must acquire and resolve in order to successfully perform a task (e.g. driving, flying, athletics).

PATIENT TESTING

AHR: During VORTEQ testing, patients simply shake their head "yes" (vertical VOR test) or "No" (horizontal VOR test) to the beat of an electronic metronome over a frequency range specified for that test.

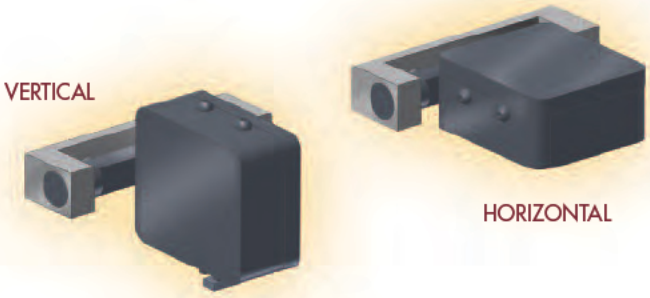
DVAT: The static acuity level is first determined. Then during headshake, the patient's head velocity must exceed a pre-set threshold for the character to appear on the screen. The characters are increased in size from the static acuity level until they can be accurately read during head shake. Eye movement recording is not required. Passive or active head shake and even head thrust testing can be used.

VISUALEYES VNG WITH VORTEQ OPTION

VORTEQ is available as an option on either the VisualEyes mid-tower or laptop systems with the VOR/DVAT tests integrated into the VNG protocol for a comprehensive evaluation of the balance disorder patient.

(VRT) VESTIBULAR REHABILITATION THERAPY

VORTEQ is particularly useful to help quantify visual acuity over a range of head velocities that occur in daily living. VOR and DVA improvements during Vestibular Rehabilitation Therapy can be serially documented to ascertain the amount of vestibular compensation

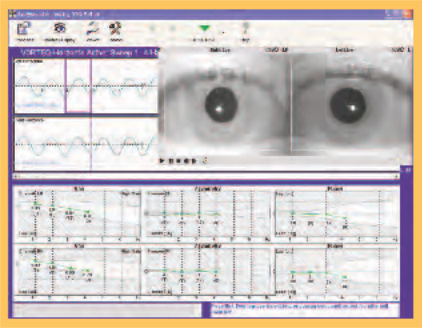


See images of high performance solid state sensor (above) that slides on to the back of the VisualEyes goggles or on the DVAT headband (right).

To Preserve and Improve Balance

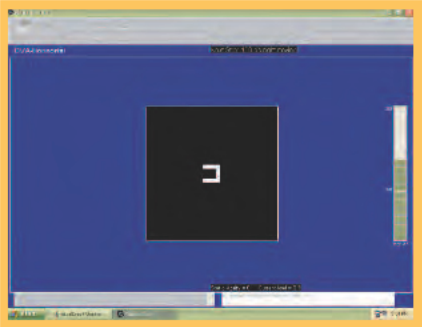
BALANCE CENTERS & FALL PREVENTION CLINICS SHOULD ALSO ACQUIRE:

- **VISUALEYES™ VNG** (Available in Midtower or Laptop)
- **BALANCE QUEST™** computerized dynamic posturography
- **SYSTEM 2000™** Rotational Vestibular Chair



AHR ANALYSIS SCREEN

Sample waveform collected from a normal subject showing eye position and head position. Gain, Phase and Asymmetry are calculated.



DVA TEST SCREEN

Characters presented are U's with possible up/down/left/right orientations that the patient must correctly identify during head motion.

VORTEQ SENSOR SPECIFICATIONS

Micromedical's solid state sensor measures head angular velocity using a state – of – the – art silicon ring gyroscope. This technology has unsurpassed accuracy and durability.

- Frequency Range: 1-5 Hz
- Velocity Range: +/- 500 degrees / second
- Size: 1.25" x 1.25" x 0.65"
- Weight: 1.6 oz

CUSTOMER CARE

Micromedical's knowledgeable staff is dedicated to assisting you and maximizing your investment by:

- Providing on-site installation and training using local representatives
- Providing technical, operational and interpretation assistance from Micromedical's experienced support staff
- Sponsoring continuing education courses
- Including a one year warranty on hardware
- Including one year of free software updates

SAFETY STANDARDS

VisualEyes systems are designed to meet applicable medical standards worldwide.

- UL2601-1
- IEC 60601-1 patient safety standard

HIPAA PRIVACY STANDARDS

Micromedical's Spectrum software is HIPAA ready, enabling healthcare providers or other covered entities to meet and maintain the confidentiality of patient medical information required by the Health Insurance Portability and Accountability Act of 1996.

Patient information can be de-identified for printed reports while the Windows® platform supplies the foundation for system security.

ISO 13485:2008
Revision

Micromedical™ TECHNOLOGIES INC.
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800.334.4154 *U.S. AND CANADA*
217.483.2122 *OUTSIDE U.S.*
FAX 217.483.1617
WWW.MICROMEDICAL.COM

VisualEyes™ VNG/ENG

Micromedical™ TECHNOLOGIES INC.

VisualEyes™ VNG/ENG

VISUALEYES IS THE ASSESSMENT TOOL OF CHOICE TO IDENTIFY ABNORMALITIES WITHIN THE VESTIBULAR SYSTEM. OBJECTIVELY DOCUMENT ABNORMAL EYE MOVEMENTS CAUSED BY TRAUMATIC BRAIN INJURY (TBI); BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV); LABRYNTHITIS AND MANY OTHER PERIPHERAL AND NEUROLOGICAL PATHOLOGIES.

VISUALEYES BINOCULAR GOGGLES

THE INNOVATIVE BINOCULAR DESIGN OF THE GOGGLES INCLUDES THE FOLLOWING KEY FEATURES:

- Full field of view with a minimum +/- 45 degrees horizontal and +20, -25 degrees vertical
- Integrated external focus knob and built-in software controlled fixation lights
- Comfortable, light-tight and easy to clean goggle
- Mirrors are mounted below eye level to reduce eyelid and eyelash interference.
- Goggle weight 10.5 oz. with cover 8 oz. with cover removed.
- Designed to utilize high speed Firewire® cameras that can record at up to 100 Hz

GOGGLES WITH COVER REMOVED

GOGGLES WITH COVER

To Preserve and Improve Balance

Providing Insight

into the Complexity of the Balance System.

HUMAN POSTURAL CONTROL RELIES ON THE MOTION AND GRAVITY SENSING ABILITY OF THE VESTIBULAR SYSTEM, AND THE SPINAL REFLEXES TO BALANCE THE BODY OVER THE RELATIVELY SMALL BASE OF SUPPORT PROVIDED BY THE FEET. WHEN THE INTEGRITY OF THE SENSORY INFORMATION TO THE CENTRAL NERVOUS SYSTEM IS COMPROMISED OR THE CNS ITSELF IS COMPROMISED, THE ABILITY TO STAY BALANCE IS REDUCED.

THE PREVALENCE AND DEBILITATING NATURE OF BALANCE DISORDERS CALLS FOR INGENUITY IN DESIGNING DIAGNOSTIC INSTRUMENTATION THAT CAN HELP THE CLINICIAN IDENTIFY ABNORMALITIES WITHIN THE CENTRAL OR PERIPHERAL BALANCE SYSTEM. THE VISUALEYES VNG IS THE PRIMARY DIAGNOSTIC TOOL AND THE IDEAL CHOICE FOR THE OBJECTIVE DOCUMENTATION OF VESTIBULAR AND BALANCE FUNCTION.

Micromedical™ TECHNOLOGIES INC

Spectrum™
balance software

SPECTRUM SOFTWARE PROVIDES YOUR CLINIC WITH STANDARD TESTING PROTOCOLS AS WELL AS PROTOCOLS THAT WILL GIVE YOU UNIQUE ASSESSMENT CAPABILITIES.

STANDARD PROTOCOLS

- Gaze
- Positional
- Dix-Hallpike
- Caloric
- Pursuit
- Saccade
- Optokinetic

UNIQUE ASSESSMENT CAPABILITIES

(some may require hardware or software enhancements)

- Active Head Rotation (VORTEQ)
- Dynamic Visual Acuity (DVA-Test)
- Pupillometry
- HVT 3D Eye Tracking – torsion measurement for ocular counter roll
- Head Impulse Test
- EOG amplifier to add ENG electrode recording capability
- Consensual Light Reflex (CLR)

ADVANCED CONCEPTS IMPLEMENTED IN THE CURRENT VERSION OF SPECTRUM (Specturm 8)

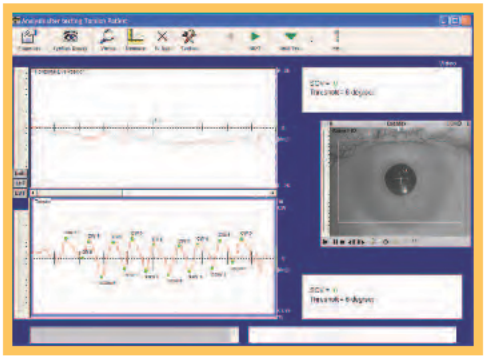
- Full software integration of Micromedical's **AirFx**™ air caloric irrigator and **AquaStim**™ water caloric irrigator
- Single mouse click eye centering removes the need to adjust cameras
- Freyss caloric diagram
- Consensual Light Reflex (CLR)

CONSENSUAL LIGHT REFLEX

Developed at the request of our interested neurology professionals to help find oculomotor nerve lesions, the CLR examines the integrity of the retina; ipsilateral optic nerve; ipsilateral oculomotor nerve and the contralateral oculomotor nerve.

HVT 3D EYE TRACKING

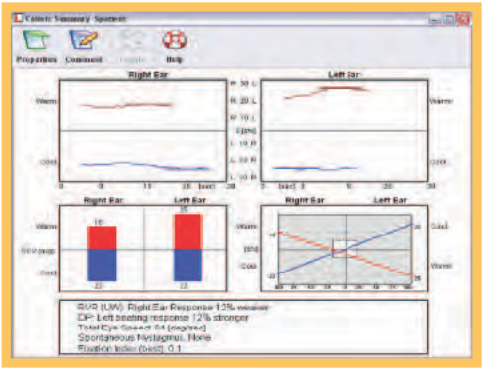
Torsional eye movement measurement provides a unique assessment of otolith function through an ocular counter roll test protocol.



- User selectable iris segment
- Torsion is recorded on one eye at 100 Hz with one degree accuracy
- Compensatory eye position is measured when the patient's head is tilted 45 degrees left or 45 degrees right

CALORIC SUMMARY

At a glance, Unilateral Weakness is identified graphically in both bar graph and Freyss diagram displays.



TURNKEY SYSTEM HARDWARE

- **COMPUTER:** Choose either Mid-Tower or Laptop configuration, with Windows® OS (computer hardware shown in photos may differ from what is supplied with your system due to our commitment to provide you with the latest technology)
- **TARGET STIMULUS:** Choose – Digital Light Bar; LCD Projector or Secondary Monitor

All systems include Micromedical's proprietary EyeMax video recording and management. EOG and VORTEQ options are available through DataLink connections

CUSTOMER CARE

MICROMEDICAL'S KNOWLEDGEABLE STAFF IS DEDICATED TO ASSISTING YOU AND MAXIMIZING YOUR INVESTMENT BY:

- Providing on-site installation and training using local representatives
- Providing technical, operational and interpretation assistance from Micromedical's experienced support staff
- Sponsoring continuing education courses
- Including a one year hardware warranty
- Including one year of free software updates

QUALITY AND REGULATORY STANDARDS

All equipment is designed and manufactured under our ISO 13485 certified quality management system to meet U.S. FDA; Canadian; European and International Standards.

- UL 60601-1
- IEC 601-1, EN 60601-1
- CMDCAS
- ANSI S3.45-2009
- Medical Device Directive (MDD) to comply with EC Directive 93/42

Micromedical™ TECHNOLOGIES INC.

To Preserve and Improve Balance

CE 500651

BSI ISO 13485:2003
FM 90233

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217.483.2122 *OUTSIDE U.S.*
217.483.4533 *FAX*
WWW.MICROMEDICAL.COM

Brochure

ICS Impulse

Great precision...

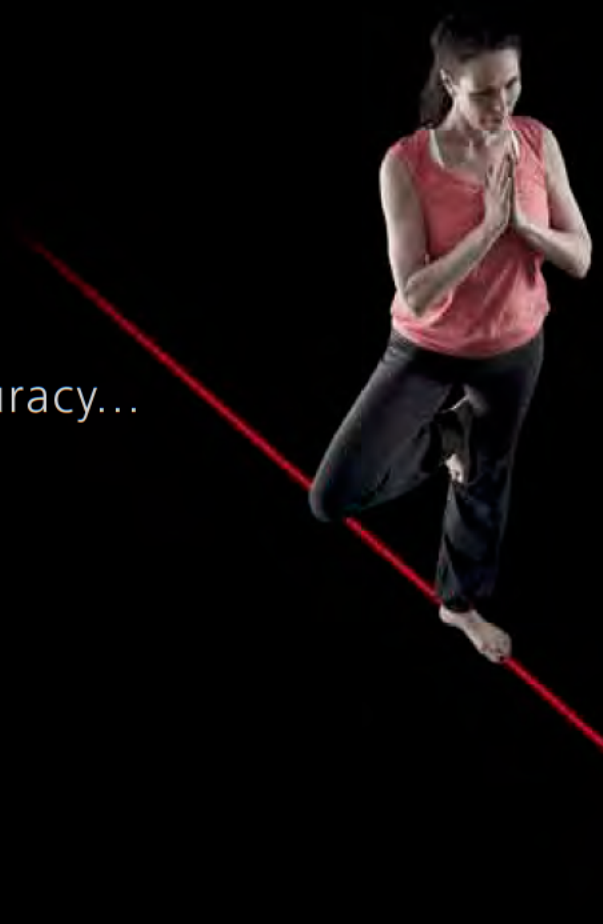
...fast diagnosis

otometrics
MADSEN · AURICAL · ICS

The head impulse test (HIT) provides quick, clear-cut ear specific assessment of the vestibulo-ocular reflex response to stimuli in the high-frequency range, the natural range of head movements. ICS Impulse from Otometrics is the world's first HIT device to combine gold-standard accuracy with unrivaled patient comfort, enabling you to perform head impulse testing with inarguable results. Fast, simple and precise, ICS Impulse is recommended as the first step in analysis, helping to improve your workflow and spend more time on patient care.

ICS Impulse: Bringing accuracy and efficiency into balance testing

Balancing accuracy...



...and great efficacy

BALANCE

ICS Impulse: A Powerful New Gold Standard in Vestibulo-ocular Reflex Assessment

More than two decades ago industry pioneers Drs. Michael Halmagyi and Ian Curthoys first described the head impulse test, and the industry has been trying to implement their findings ever since. Now, after almost 20 years of research, Otometrics has emerged with the new gold standard, ICS Impulse: an objective measurement of both head and eye movement using a fast and precise system to assess the gain of the vestibulo-ocular reflex. With unrivaled accuracy and efficacy, the ICS Impulse will forever change the way you work.

Stimuli replicating the patient's everyday situations

ICS Impulse provides precise, accurate data based on real-life stimuli. The high-frequency stimuli used in HIT is similar to that used in daily activity that occurs when crossing the street, sitting in a restaurant or quickly turning to a sound.

Test the vestibulo-ocular reflex in less than 10 minutes

In addition to providing an accurate, objective measure of the vestibulo-ocular reflex, the ICS Impulse allows clinicians to test patients with spontaneous nystagmus. Both overt and covert saccades can be detected allowing for proper diagnosis and rehabilitation recommendations. The vestibular function of both ears can be assessed and documented in less than 10 minutes from patient entry to reporting.

Improved Patient Care

Patient comfort is greatly enhanced by the lightest goggles in the industry. Due to the sophisticated cameras smaller velocity head impulses of only 15 to 20 degrees are used, making the test more pleasant for the patient. Direct comparison to the gold standard "scleral coils" has authenticated the ICS Impulse. ICS Impulse detects more abnormalities than visual observation and reduces false negatives. And because results are known immediately, treatment can begin much sooner.

Built on the work of Drs. Halmagyi and Curthoys

HIT thought leaders Drs. Michael Halmagyi and Ian Curthoys have collaborated with Otometrics to bring to market an assessment tool that optimizes their groundbreaking work. It is the first HIT testing device to meet their standards.

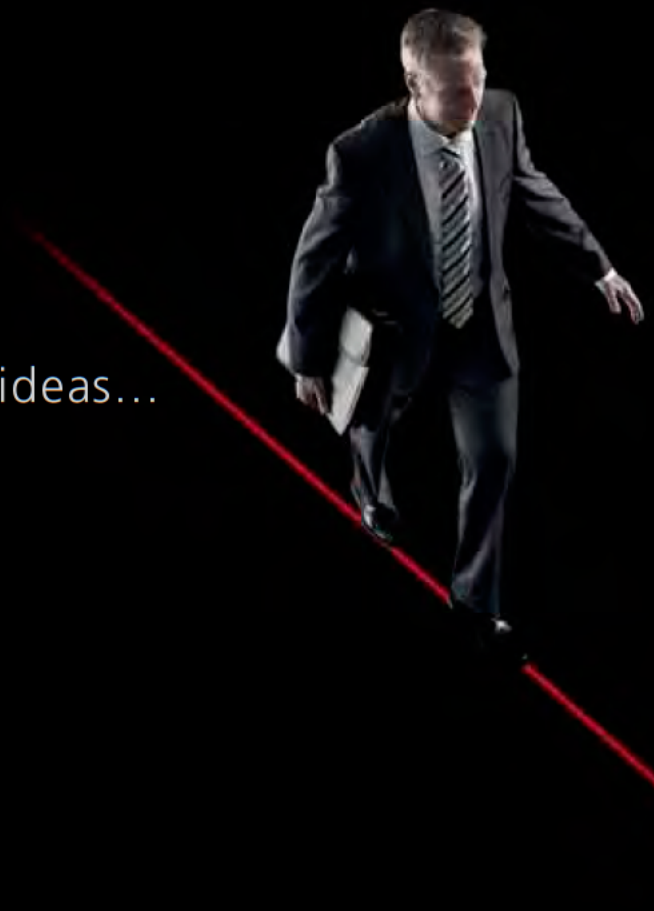
The ICS Impulse makes it possible to test children, bed-ridden patients, or anyone for which caloric testing is not an option.

What is Head Impulse Testing?

- An ear-specific test that detects disorders of the vestibulo-ocular reflex and identifies which ear is affected in cases of peripheral vestibular loss. Patients with a vestibular loss will exhibit a corrective saccadic eye movement (a "catch-up" saccade) either during or after the head impulse and the gain of the head in comparison to the eye will not be equivalent.
- An assessment tool that provides quick, precise information about the vestibulo-ocular reflex to stimuli in the high-frequency range.
- First identified and described by Halmagyi and Curthoys in the 1988 article, "A Clinical Sign of Canal Paresis." Said Halmagyi: "The eyes are the speedometers of the semicircular canals."

Watch videos of clinical applications of HIT and of Dr. Halmagyi's classroom lectures on **www.headimpulse.com/knowledge-center**

Connecting new ideas...



...to your workflow

BALANCE

The unique attributes of ICS Impulse bring a new flexibility to how and where you work

Simple, worry-free operation
ICS Impulse increases test quality by displaying head velocities which assists in the performance of unpredictable head impulses. Training curves provide a guide to assist you in performing quality head impulses of varying velocities. The only hardware needed is the ICS Impulse goggle and the USB/firewire data transmission box, which allows for a portable system that can be used in any location.

Superior Pupil Detection and Fast, Simple Calibration
Superior Pupil detection provides error free data. Calibration can be performed anywhere using Impulse goggles with built-in lasers. All you need is a small surface for which to project the laser dots. In seconds, you are ready to test.

Easier analysis and normative data
View analysis in 2D or 3D. Both display a gain graph with built-in published normative data. A clear 3-D picture facilitates easy identification of saccades. A powerful, dedicated database stores the patient's current status and charts progress by comparing results from multiple test sessions. Comparison of test sessions allows for validation of vestibular rehabilitation success.

Extensive reporting and Data Sharing
With documentation taking away an increasing amount of time from patient care, Otometrics designed the ICS Impulse with a customized report function to meet documentation requirements. Third-party data-sharing interfaces directly with third-party EMR systems. ASCII export is also available.

How does ICS Impulse fit into your workflow?

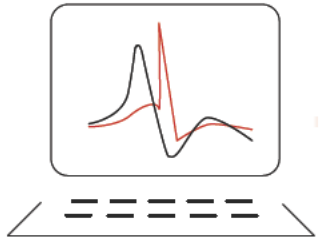
- It's the first step towards diagnosis and subsequently early treatment. Since the head impulse test is quick and won't produce an adverse patient reaction it's recommended that the test be performed at the beginning of the assessment workflow. This enables a quick assessment of the vestibulo-ocular reflex.

For a comprehensive, informative whitepaper on the clinical application of video head impulse testing, go to **www.headimpulse.com/knowledge-center**

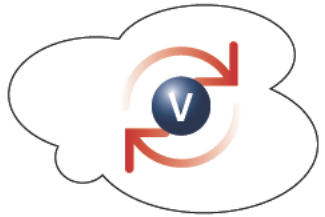
The goggle has been designed for quick and easy placement. At a mere 60 grams there is virtually no slippage. Calibration is quick and easy, using two lasers that are incorporated into the goggle itself, eliminating the need for any additional hardware.

A 15° angle is all that is required. Move the head quickly to the right or left and stop. After a short break return the head to the center, and repeat using varying velocites and unpredictable head impulses.

Watch the ICS Impulse training videos at www.icsimpulse.com



Visuals in the software provide immediate feedback on the quality of the head impulse.



Our proprietary OTOsuite™ Vestibular software captures, consolidates, and saves patient measurement data, allowing comparisons of multiple sessions through the use of progress graphs and data. The software also offers multiple reporting facilities and integration with third-party systems.

Cutting-edge technology...

...tested and documented

BALANCE

Years of research bring validity to ICS Impulse

More than two decades of testing

ICS Impulse is the first and only HIT system to be approved by Drs. Michael Halmagyi and Ian Curthoys, whose groundbreaking work first brought HIT to the attention of the world in 1988. Having developed both the prototype and the algorithms, Drs. Halmagyi and Curthoys were highly qualified to test and evaluate Otometrics' ICS Impulse. Their consensus was that it did indeed optimize their findings, providing thorough, precise results and excellent documentation.

Lightweight goggle brings significant benefits

ICS has developed the lightest video goggle in the industry, a fact that belies their strength. At 60 grams, the goggle virtually eliminates any possibility of slippage. A high-speed camera (250 Hz) facilitates the identification of covert and overt saccades. The goggle is designed to guarantee accurate data collection while ensuring the patient's comfort, which in turn makes the process easier for the tester.

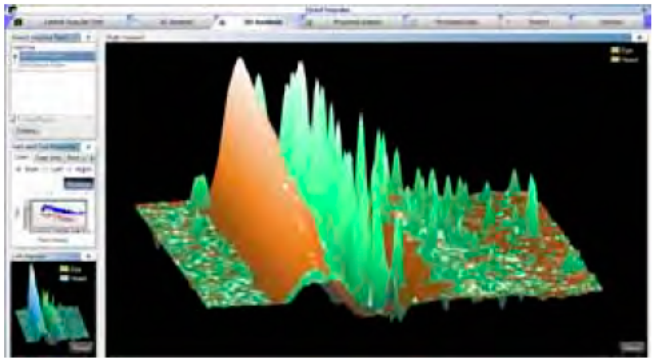
OTOsuite™ Vestibular software

- The OTOsuite™ Vestibular software optimizes workflow by capturing, automatically saving and displaying analysis, and sharing patient measurement data. It provides analyses (2-D and 3-D) with published normative data.
- Multiple test sessions can be compared using progress graph and progress data.
- Real time trace allows the clinician to monitor the patient's eye movement during head impulse testing.
- Record the eye movement at any time using the Video Record mode. Recorded eye movement can be played back in slow motion.

More about OTOsuite™ Vestibular
www.icsimpulse.com/downloads

1. **High-speed camera (250 Hz)** The superior camera provides the best available technology for measuring fast eye movements. This provides the ability to record the eye while providing high-frequency head movements. Both covert and overt saccades can be identified.
2. **Built-in gyroscopes** Dual-axis gyroscopes measure the head movement accurately allowing for direct comparison in head and eye velocities. They also provide instant feedback on the quality of the head impulse manoeuvre.
3. **No slippage** Weighing 60 grams the goggle ensures no slippage and therefore providing accurate data collection without missing any important eye movements.
4. **Built-in calibration laser** With a built-in calibration laser, the test can be performed anywhere there's a wall for calibration. There's no need for additional hardware.
5. **USB/Firewire data transmission** The USB/Firewire interface box allows fast, accurate data transfer to the computer.

Otosuite Vestibular provides 3D analysis allowing you to rotate the data 360 degrees makes viewing of saccadic eye movement simple. Published normative data in the gain graph makes determining if results are abnormal quick and easy.



Correct diagnosis...

...increased patient satisfaction

BALANCE

Meet us online to learn more about the features and benefits of Otometrics' ICS Impulse and head impulse testing.

Questions, comments, conversation
Join the conversation about ICS Impulse and head impulse testing. Ask questions, get answers from colleagues and balance experts or share your experiences and opinions.

More about ICS Impulse
www.icsimpulse.com opens up a wealth of information and resources to enhance your knowledge about ICS Impulse and HIT. A library of educational material offers white papers, articles by experts and e-learning options that include instructions on how to perform head impulse testing. Visit our blog and dialogue with the people behind the ICS Impulse and other balance experts.

More about head impulse testing
At www.headimpulse.com you can explore both the science and practical application of head impulse testing. The library houses research material, including videos of Dr. Halmagyi's classroom lectures and examples of HIT test results. Access a list of upcoming events to see the experts of HIT live. And you can learn more about how head impulse testing works and why it has made such a difference in the industry.

www.icsimpulse.com

www.headimpulse.com

Features and Benefits

High speed camera (250 Hz)
- Ultra-sensitive, requiring head turns of just 15°
- Records fast eye movements that allow for identification of overt and covert saccades
- Test takes less than 10 minutes from patient entry to reporting

Built-in gyroscopes
- Ensure accurate head velocity measurement
- Provide instant feedback on proper head impulse manoeuvre
- Compare head and eye movement to assess VOR gain

Built-in calibration lasers
- Calibration is quick and easy, using two lasers incorporated into the goggle itself
- No need for additional hardware
- Test can be performed anywhere

Built-in head impulse algorithms
- Developed by HIT pioneers Drs. Michael Halmagyi and Ian Curthoys
- Inaccurate head impulse data is automatically discarded
- Only accurate data is analyzed

Plug-and-go solution
- Small and compact for ultimate portability
- Easy and efficient for bedside testing
- Enables accurate testing of immobile patients

Video recording
Read the flyer about enhanced documentation of abnormalities
www.icsimpulse.com/downloads

See all the benefits of ICS Impulse online at
www.icsimpulse.com

ICS Impulse

Bringing accuracy and efficiency into balance testing

ICS - the leader in vestibular testing

ICS is a leading global provider of diagnostic devices for balance disorders. Founded in 1981, the company has a history of developing ground-breaking products that provide pinpoint accuracy for balance testing. ICS is an expert brand of GN Otometrics.

Meet us online to learn more about our thinking, ideas, solutions and the way in which we support you in your endeavours. We're always ready for and welcome a dialogue.

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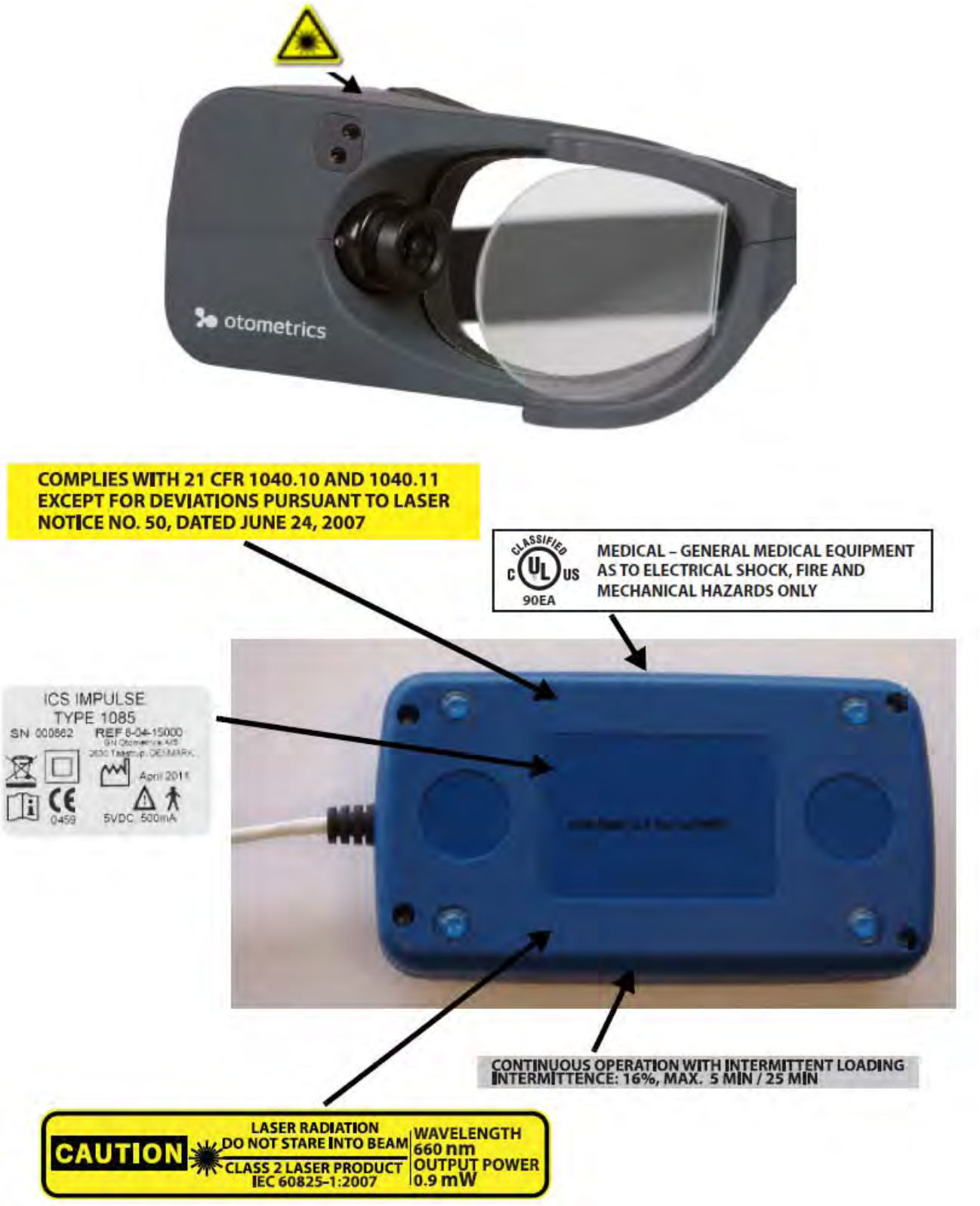
www.otometrics.com

otometrics
MADSEN · AURICAL · ICS

Device Labels



Operator Manual

ICS Impulse

Video Head Impulse Test

User Manual

Doc No. 7-50-1110-US_FDA
Part no. 7-50-11100-US

CE 0459

otometrics
MADSEN · AURICAL · ICS

Printed in Denmark by GN Otometrics A/S, Denmark

Version release date
13. January 2012

Technical support
Please contact your supplier.

Table of Contents

Table of Contents

1 Introduction

Congratulations! You are now the owner of a sophisticated new ICS Impulse system developed in collaboration with Drs. Ian Curthoys, Michael Halmagyi and others at University of Sydney.

To assist you in getting the most out of the ICS Impulse system, we have included this user manual and a training video. We hope you find it easy to use and that your use of the incorporated tips and information results in improved data collection accuracy as it relates to your assessment of vestibular-related disorders, test results reporting, and patient information retrieval.

1.1 Intended Use

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.

Caution · *US Federal Law restricts this device to sale by or on the order of a licensed physician.*

1.2 Intended User

Note · *The ICS Impulse System is intended to be used only by qualified medical personnel.*

This manual describes the use of the device in combination with the software. Readers are assumed to have prior knowledge of the medical and scientific facts underlying the procedure. For this reason, the examination methods are mentioned only to the degree that is necessary for a correct, safe application of the ICS Impulse System.

You can find more information in the ICS Impulse training video or at **www.headimpulse.com**.

1.3 About this manual

This is your guide to using the basic functions required for navigating in OTOsuite Vestibular and the various OTOsuite Vestibular modules. This includes key features such as printing test results, handling patient and user administration, and data and test device management.

Training
It is recommended that you make yourself familiar with the features provided by OTOsuite Vestibular and the test device before testing a patient.

1.3.1 Safety

This manual contains information and cautions which must be followed to ensure the safe performance of the ICS Impulse System.

Caution · *Local government rules and regulations, if applicable, should be followed at all times.*

Safety information is stated where it is relevant, and general safety aspects are described in Chapter 9 ICS Impulse System Safety ▶ 99.

1.4 Typographical conventions

The use of WARNING, CAUTION and NOTE
For safety reasons and appropriate use of the ICS Impulse System, the manual contains **WARNINGS**, **CAUTIONS** and **NOTES** which you should read carefully. The use of these headings is denoted as follows:

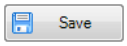
Warning · *Indicates that there is a risk of danger to persons, test device and data.*

Caution · *Indicates that there is a risk of damage to the device and/or data.*

Note · *Indicates that you should take special notice.*

1.4.1 Navigating this manual

Window tabs, icons and functions to select are shown in bold type, as for instance in:



- Click **Save**

Typographical conventions

2 Getting Started

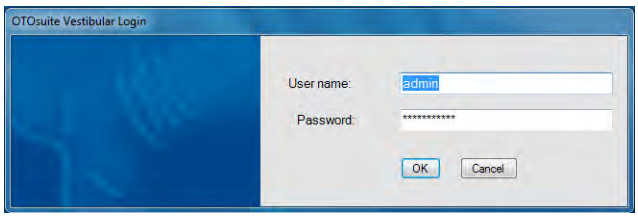
2.1 System startup

1. Switch on the computer.
2. Double-click the OTOsuite Vestibular icon.

2.2 Logging in

Note · *An Administrator user name and password are provided with a new installation of OTOsuite Vestibular. DO NOT delete the user name or password from the login screen until at least one new user name with password has been added. Ensure that at least one user has administrator privileges. Refer to* *System Settings* ▶ *63* *for information on how to add new users.*

At the login screen

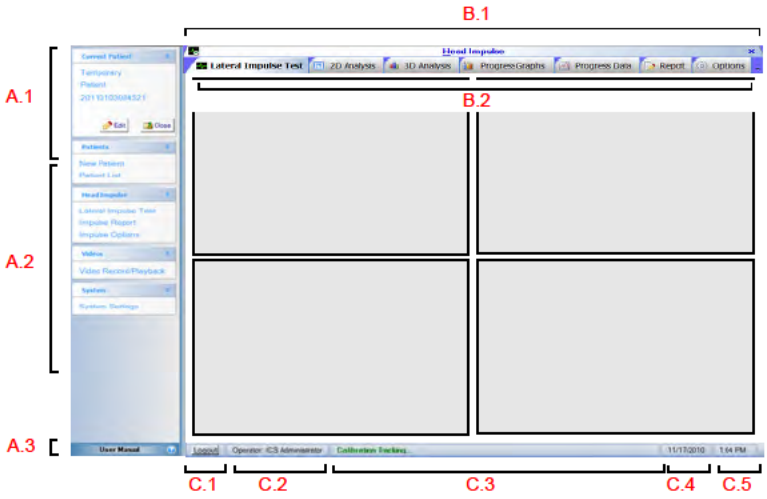


Note · *Both user name and password are case sensitive.*

1. Enter your user name.
2. Enter your password and click **OK.**

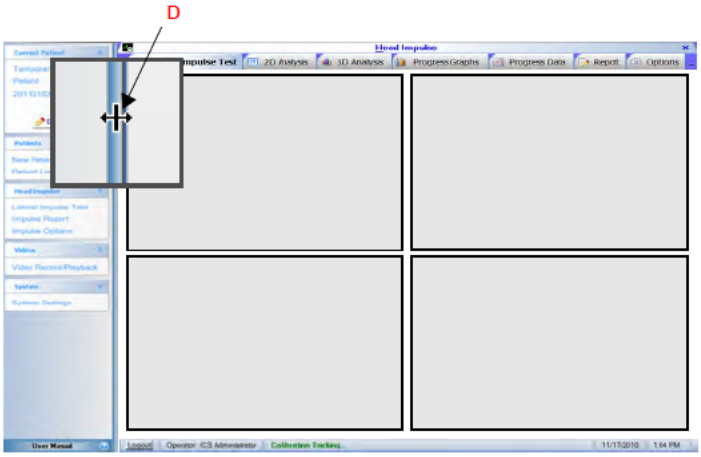
2.3 Understanding the OTOsuite Vestibular screen

The main areas of the OTOsuite Vestibular screen are the navigation panel (A), the tabbed windows for testing and other related functions (B), and the status bar (C).



Navigation panel	A.1 - Information about the current patient. A.2 - Items grouped by function A.3 - Button for access to the User Manual.
Tabbed windows	The tabbed windows correlate to the groups in the navigation panel. You can choose to display the tabbed windows for one or more groups. (To display a group of tabbed windows, click an item of that group in the navigation panel.) For example, the Head Impulse window group (B.1) is subdivided into tabbed windows (B.2) arranged from left to right from data collection, to reviewing and comparison of data results, to reporting, and test options.
Status bar	Logout button (C.1), current operator (C.2), system status (C.3), current date (C.4), and current time (C.5)

Note · *If a thick blue bar separates an area of the screen, the size of the areas can be changed. For example, to increase the width of the navigation panel, position the cursor over the blue bar until a double-headed arrow (D) appears. Click the left mouse button and drag the bar to a new location. These bars separate windows vertically as shown here but can also separate windows horizontally.*

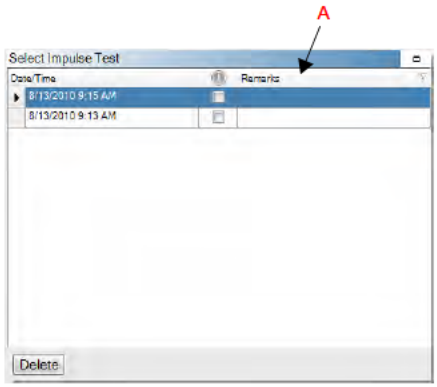


2.4 Entering test remarks/report findings

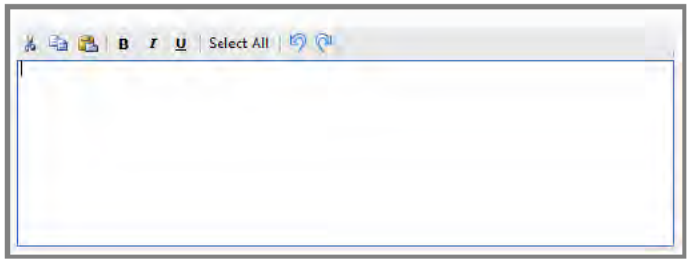
Test remarks can be entered in a test selection window or in a text field that includes editing tools. The remarks are saved for the head impulse or video test session in which they were entered. They can be viewed and edited in the following windows:

- Lateral Impulse Test
- 2D Analysis
- 3D Analysis
- Progress Graphs
- Progress Data
- Video Record/Playback

Remarks column (A) in a test selection window



Text field with editing toolbar



Toolbar buttons	
	Delete selected text
	Copy selected text
	Paste text that was cut or copied

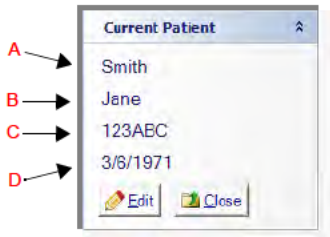
Toolbar buttons	
B	Apply bold formatting
I	Apply italic formatting
U	Apply underline formatting
	Undo actions (text entry, text formatting, etc.) starting with the most recent action and proceeding in reverse order of actions done.
	Reverse an undo action starting with the most recent undo and proceeding in reverse order of undo actions.

2.5 Patient data entry

2.5.1 Current Patient group

The **Current Patient** group provides information about the patient currently open.

- Last name (A)
- First name (B)
- Patient ID (C)
- Date of Birth (D)



- Click **Close** to close the current patient
- Click **Edit** to modify patient information

2.5.1.1 Temporary patient

If no patient is open, a temporary patient is available. The temporary patient allows testing to start without entering patient data. The patient is given a unique number based on the date and time the patient record was created (yyyy_mm_dd_hh_mm_ss where yyyy=year, mm=month, hh=hour, dd-day, mm=minute, and ss=second).

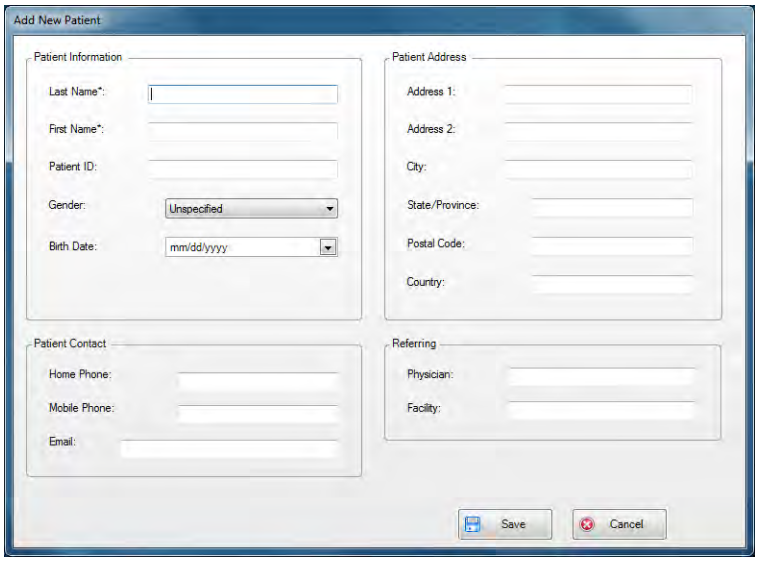
Once data is saved, you will be prompted to edit the temporary patient information. Refer to 2.5.2.2 Existing Patient ▶ 15 for information.

2.5.2 Patient group

This group allows you to create a new patient or view a list of existing patients.

2.5.2.1 New Patient

To enter information for a new patient, click **New Patient** and add patient information in the form:



Entries that cannot be left blank are marked with an asterisk (*). Patient ID can be numeric, alphabetical or a combination of numbers and letters. To set the gender, click the down arrow and select the appropriate entry. The date of birth format is determined by the computer setup and will display as mm/dd/yyyy or dd/mm/yyyy where mm=month, dd=day, and yyyy=year. Only numeric characters are allowed (alpha characters can not be used).

Click **Cancel** to close the form without saving the data. Click **Save** to save the data.

Note · *The system alerts you with a message with the first occurrence of a required entry that is blank.*

2.5.2.2 Existing Patient

From the **Patients** group, click **Patient List**. The window that displays provides access to existing patients.

Note · *Refer to* *Patient List operations ▶ 16* *for more information about how to work with the list.*

Each row has patient information separated into columns according to the type of information: Patient name, ID#, etc. The **HI** (Head Impulse) and **Video** columns are blank or have check marks to indicate the following:

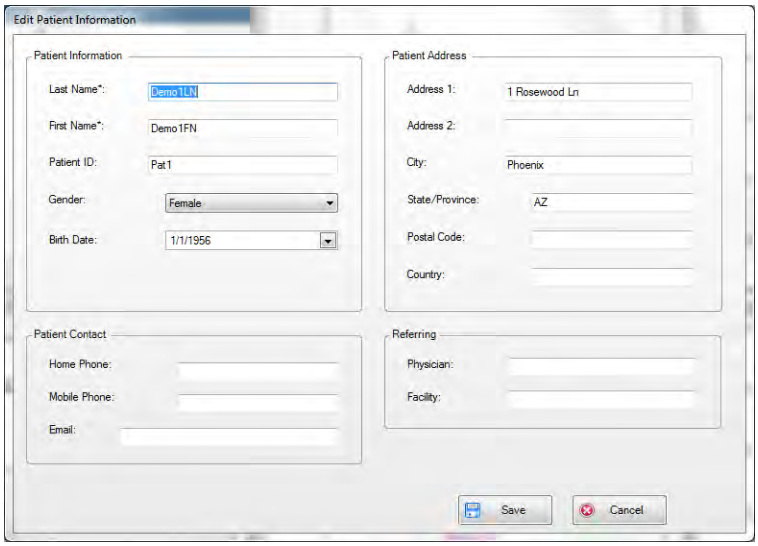
- **HI** - head impulse data is saved
- **Video** - video data is saved

Note · *The symbol*  *indicates the user has chosen to mark one or more tests or videos for a specific purpose (for example, it may refer to abnormal results, results to be used for a study, etc.)*

To select a patient, click the patient name.

- Click **Open** (or double-click) to open the patient to view test results
- Click **Edit** to make changes to the existing patient information

Modify patient information in this form:



Entries that cannot be left blank are marked with an asterisk (*).

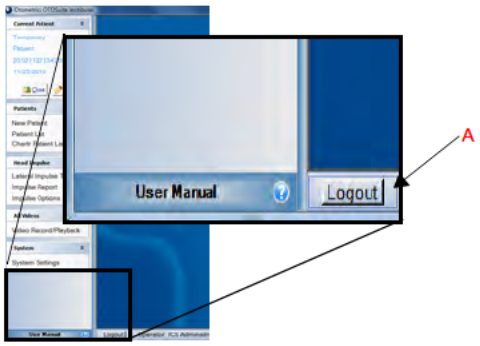
Patient List operations

Selecting more than one patient	Select all patients: Click **Select All**. To deselect all selected patients, click, **Deselect All**. Select a group of patients: Click the first name of the set, hold down the Shift key and click the last name in the set. Select individual patients: Keep the Ctrl key pressed as you click on each patient.
Adding patients	Click **New**. Refer to 2.5.2.1 New Patient ▶ 14 for instructions.
Deleting patients	Click **Delete** to delete the patient(s). **Note** · *You will be prompted to confirm deletions of the patient(s). If you choose Yes the data is permanently deleted. There is no possibility to retrieve the patient(s).*

Reordering lists	Click the column header, to reverse the order. For example • Click **Patient Name** to change the order from A-Z to Z-A. • Click **DOB** to change the order from oldest-youngest to youngest-oldest.
Changing column width	In the column heading of two adjacent columns, click the cursor on the line dividing two columns. The cursor changes to a double-headed arrow. Drag the cursor to increase or decrease the column width.

2.6 Logging out

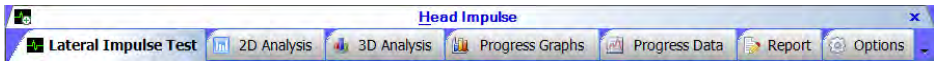
To log out, click on the Logout button (A).



Logging out

3 Head Impulse

These functions are available in the Head Impulse group:



- Lateral Impulse Test
- 2D Analysis (refer to 3.9 Lateral head impulse single test data analysis ▶ 34)
- 3D Analysis (refer to 3.9 Lateral head impulse single test data analysis ▶ 34)
- Progress Graphs (refer to 3.10 Lateral head impulse multiple-test data analysis ▶ 44)
- Progress Data (refer to 3.10 Lateral head impulse multiple-test data analysis ▶ 44)
- Report
- Options

3.1 Patient preparation

Warning · *A head impulse should not be performed on patients with a neck injury, or on patients who have been told by their physicians to limit or avoid neck movement activity.*

Prior to testing, provide the patient with these general recommendations:

- If in doubt, consult with a physician about the possible side effects of stopping a particular medication.
- Stop tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test.
- Continue medications that are vital, such as insulin, heart medications, seizure medications, and possibly antidepressants.
- No alcohol for 48 hours before testing.
- Do not wear make-up around the eyes.
- Wear comfortable clothing.

3.2 Goggle preparation

3.2.1 Cleaning and maintenance

The ICS Impulse System equipment does not require preventive maintenance. Observe the following recommended guidelines regarding cleaning and maintenance.

- Keep the instrument clean and as free of dust as possible. Remove dust using a soft cloth or brush.
- If required, clean the goggle housing and interface box using a damp cloth moistened with a mild detergent and water solution. Do not allow any moisture to get inside the goggles.

Caution · *Never spray or immerse the goggle components with the cleaning solutions. This could contaminate the electronics and/or optics.*

- If required, clean the mirror using the supplied cleaning cloth. The presence of fingerprints on the mirror surfaces could cause inaccurate pupil detection.

Caution · *Improper cleaning may scratch the mirror surfaces.*

- Replace the strap as required. Refer to 3.2.2 Replacing the strap ▶ 21.

3.2.2 Replacing the strap

1. Remove the face cushion.
2. Use a pen to push the plastic clip down and pull out the strap clip attached to the goggle.

3. Repeat on the other side.
4. Remove the cables from both clips on the strap.

5. Obtain a new strap assembly
6. Clip the strap clips into each side of the goggle.
7. Attach the cables to both clips on the strap inserting the small one first.

3.2.3 Replacing the face cushion

Note · *The single-use, disposable face cushion should be replaced for each new patient.*

1. To remove the face cushion, slightly flex the goggles out at the side opposite of the camera side and snap out the face cushion. Release the face cushion from the other side.
2. Properly dispose of the used face cushion.
3. Obtain a new face cushion.
4. Align the tab of the face cushion with the hole on the camera side of the goggles.
5. Ensure the face cushion is inside the nose piece.
6. Slightly flex the goggles at the opposite side, align the tab of the face cushion with the hole on this side of the goggles.
7. Double check both sides are fully inserted by pressing in at each side.

3.3 Test setup

The environment where the patient is tested can vary but must allow you to position the patient at least one meter from the wall (or other solid surface that can be used as a projection surface.).

1. Choose a wall that allows you to position the patient at least one meter in front of the fixation dot.
2. Apply one of the fixation dots supplied with the system to the wall in a location that allows you to position the patient directly in front of the fixation dot.

3.4 Goggle placement

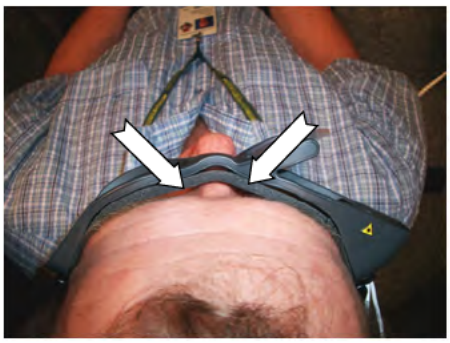
When goggles are placed properly they sit comfortably on the bridge of the nose and will not slip during the test. Poor goggle fit resulting in slippage can result in gain values being inaccurate.

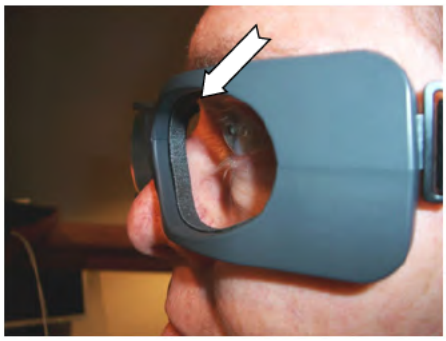
Note · *You can find more information in the ICS Impulse training video or at* ***www.headimpulse.com****.*

Caution · *Goggle Fit is Important. Improper goggle fit can result in inaccurate data collection. Goggle slippage often results in inaccurate gain values (too high).*

After placing the goggle on the patient and tightening the strap, look at how the goggle fits on the person's face. If there are gaps between the goggle and the patient's face the goggle may slip.

Improper goggle fit





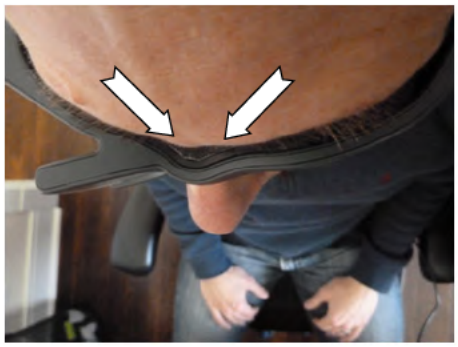
1. Before putting the goggles on the patient ensure
   - the goggles have a new unused face cushion. Refer to 3.2.3 Replacing the face cushion ▶ 22.
   - the mirror is clean. Refer to 3.2.1 Cleaning and maintenance ▶ 20.

Note · *The single-use, disposable face cushion must be replaced for each new patient.*

Caution · *Improper cleaning may scratch the mirror surfaces.*

2. If required, replace the strap. Refer to 3.2.2 Replacing the strap ▶ 21.
3. Position the goggles on the patient's face over the bridge of the nose.
4. Bring the strap above the patient's ears and around to the back of head.
5. Tighten strap tight enough to ensure that goggles will not shift horizontally during test.
6. Allowing some flexibility in the cables for head movement during testing, clip the cable clip to the patient's clothing at the top of the shoulder.
7. Ensure the eyes are wide open with eyelids positioned to not interfere with pupil detection. If required, adjust the skin around the eye:
   Tilt the bottom of the goggles out and away from the face, pulling the skin below the eye down and repositioning the goggles to hold the skin in place. Tilt the top of the goggles out and away from the face, pulling the skin above the eye up and repositioning the goggles to hold the skin in place.
8. Visually inspect goggle fit.

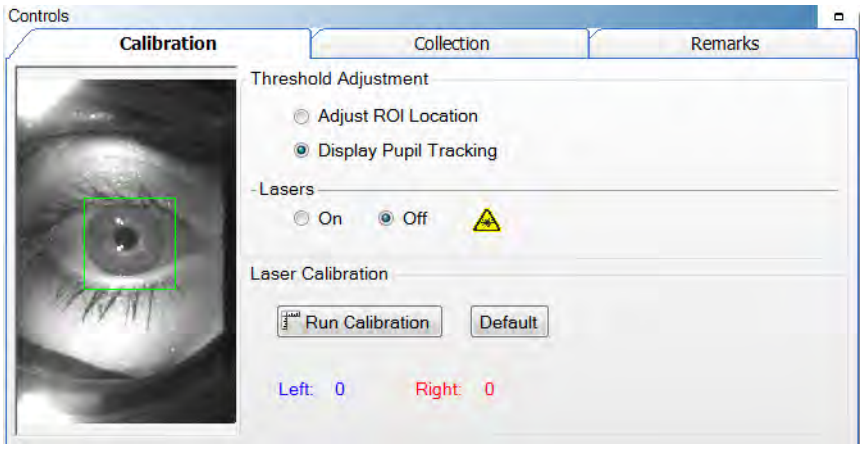
Proper goggle fit



3.5 Pupil detection

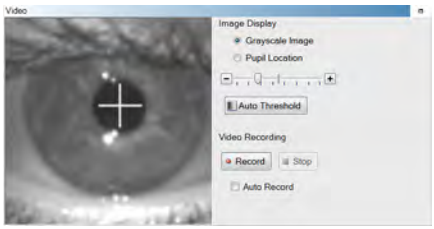
Pupil detection ensures that the system tracks the pupil properly during calibration and when collecting data.

1. Position the patient at least one meter in front the wall (refer to 3.3 Test setup ▶ 22 for information).
2. Put the goggles on the patient (refer to 3.4 Goggle placement ▶ 23 for information).
3. Position the ROI (Region of Interest) around the pupil:
   – use the mouse to center the ROI box on the pupil and click, or
   – click on the pupil to center the pupil inside the green box.
4. Select **Display Pupil Tracking**.

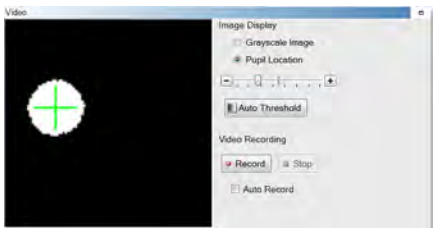


5. In the **Video** window, choose the type of image displayed: **Grayscale Image** (A) or **Pupil Location** (B).
6. Select **Auto Threshold**. The system centers the cross-hair on the pupil.
7. Ask the patient to stare at the fixation dot. Assess pupil tracking by observing the cross-hair: If the cross-hair fails to track the pupil (jumps around and does not stay centered on the pupil), move the threshold slider to adjust.

A



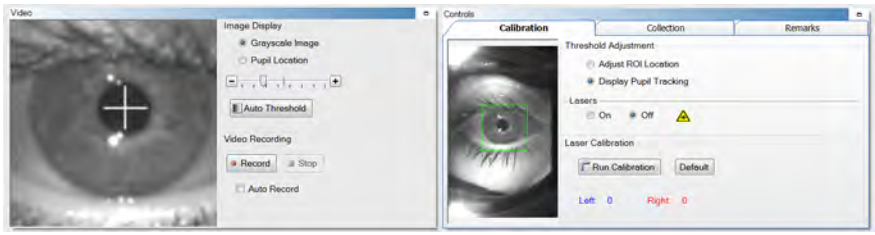
B



Note · *When* **Image Display** *is set to* **Pupil Location**, *make additional adjustments to remove any white dots outside the white circular image of the pupil.*

Caution · *During this procedure both lasers will turn on. Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*

8. Click **On** to turn on both lasers.



9. Ask the patient to position the left and right dots equidistant on each side of the fixation dot.

10. Ask the patient to look at the left dot, then at the right dot. In the **Video** window, check that the cross-hair continues to track the pupil.

Note · *Use the Real Time Trace window to monitor incoming data. By observing the head trace (red) and the eye trace (green), you can tell if the patient is moving their head or eyes (instead of staring at the fixation dot), blinking excessively, or not following instructions being given (not cooperating).*

11. If the cross-hair fails to track the pupil (jumps around and does not stay centered on the pupil), move the threshold slider to make further adjustments.
12. When pupil detection is set, start calibration (section 3.6).

3.6 Calibration

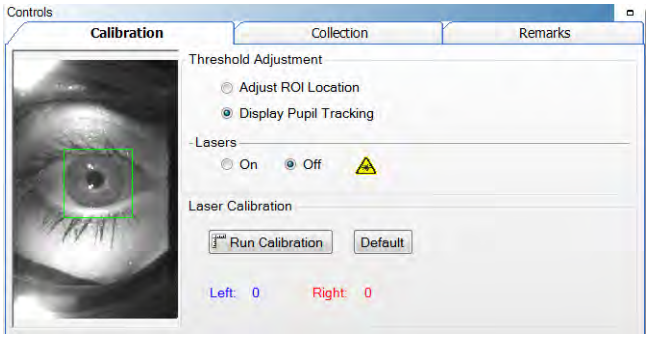
In the calibration procedure, the patient is asked to switch their gaze between the two dots that appear when the lasers are on. As the patient's gaze switches, the system tracks the movement of the pupil.

Note · *When a patient cannot be calibrated (for example the vision is so poor that the patient cannot see the fixation dot or laser beams dots), click* **Default** *to use the calibration default values (Left = 38, Right = 59, Δ = 21).*

The eye movement between the two laser beam dots is measured and calibrated against the known values between the two laser beam dots. The calibration values relate to the pixel location that equates to 7.5 degrees left and right of center. The difference between the left and right equates to the number of pixels for a 15 degree movement of the eye. These values are used to analyze eye movement during the lateral impulse testing.

Caution · *During this procedure one laser at a time will turn on. Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*

1. Click **Run Calibration**.



2. Ask the patient to face the fixation dot and hold the head still.
3. Ask the patient to look at the laser beam dot.

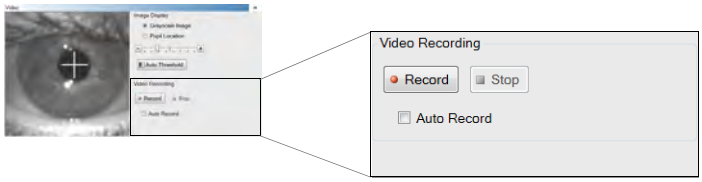
The calibration values are automatically saved. The **Collection** window opens and the software is ready to begin collecting head impulse data.

Caution · *Once calibration has been performed, it is recommended not to reposition the goggles on the patient's head.*

3.7 Video recording during head impulse testing

The **Video** window displays the image of the eye.

To start the video recording at the same time as data collection starts, select the check box **Auto Record**. To control the recording manually, click **Record** to start the recording and **Stop** to stop the recording.



3.8 Lateral head impulse data collection

The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity.

Stimulus

Displacement = 10° to 20°

Peak Head Velocity = 100°/s to 250°/s

Peak Head Acceleration = 1000°/s^2 to 2500°/s^2

Note · *You can find more information in the ICS Impulse training video or at* ***www.headimpulse.com****.*

The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response).

Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

3.8.1 Collecting head impulse data

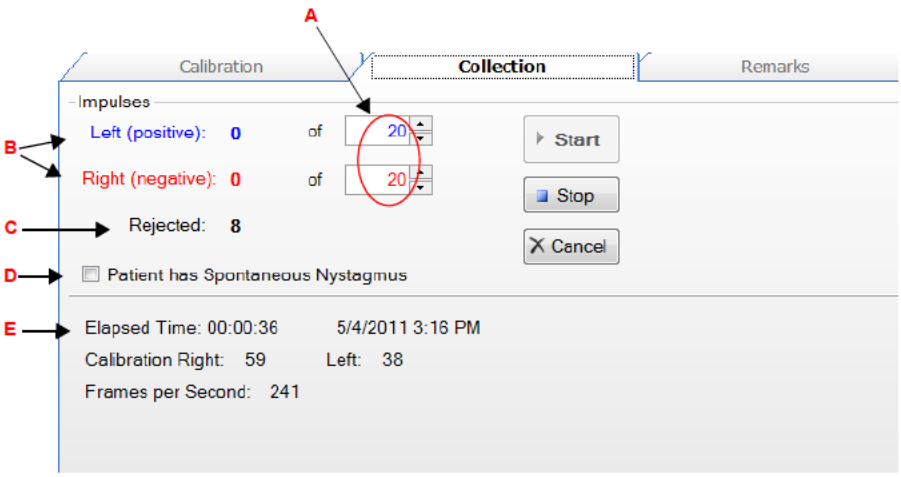
Note · *The temporary patient is available to allow testing to start without entering any patient data. Refer to* *2.5 Patient data entry ▶ 13* *to create a new patient or open an existing patient.*

1. If the Head Impulse window is not open, click **Lateral Impulse Test** in the **Head Impulse** group.
2. The **Impulses** settings (A) define how many valid left and right impulses are required before the test stops automatically. To change the number of suggested minimum head impulses for the current testing session, click the up or down arrows or type in the number directly. Twenty (20) is the recommended minimum number of head impulses for both leftward and rightward impulses.

Note · *Impulse settings can be any number up to 999. Refer to 3.12 Head impulse options ▶ 53 for information about setting the default numbers.*
During testing, valid impulses display for both left and right impulses (B). The left and right impulses that are not valid (those with velocities outside a pre-determined acceptable range) are combined into the rejected count (C).

3. For patients with spontaneous nystagmus, select the check box **Patient has Spontaneous Nystagmus** (D).

By selecting this check box for patient who have spontaneous nystagmus, it prevents good impulses from being rejected inappropriately.



4. A disabled (grayed out) **Start** button indicates the system is ready for head impulse data collection. Otherwise you must click **Start** to begin head impulse data collection.

Caution · *Touching the goggles or the goggle strap while moving the patient's head can result in moving the camera which produces artifacts in the collection data.*

5. Standing behind the patient, place your hands on the top of the patient's head well away from the goggles and the goggle strap.
6. Ask the patient to stare at the fixation dot and move the patients head as described in 3.8 Lateral head impulse data collection ▶ 30. It is important to have reviewed the ICS Impulse Training Video.
7. The system automatically stops when the minimum numbers for left and right impulses have been reached. To manually stop the test, click **Stop** (data collected is saved) or **Cancel** (data collected is NOT saved).

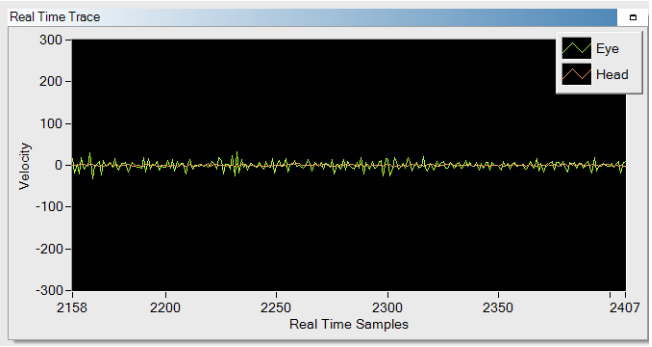
The data is automatically analyzed and displayed in the 2D Analysis window. The elapsed time (E) displays along with test date and test time.

3.8.2 Monitoring eye and head movement

While performing the head impulse test, both the **Real Time Trace** window and the **Lateral Impulses** window display head and eye traces to assist you in understanding the quality of data being collected.

3.8.2.1 Real Time Trace window

This window allows you to monitor both head and eye movement while performing the head impulse test. During the test, the maximum sample rate is 250 samples per second.

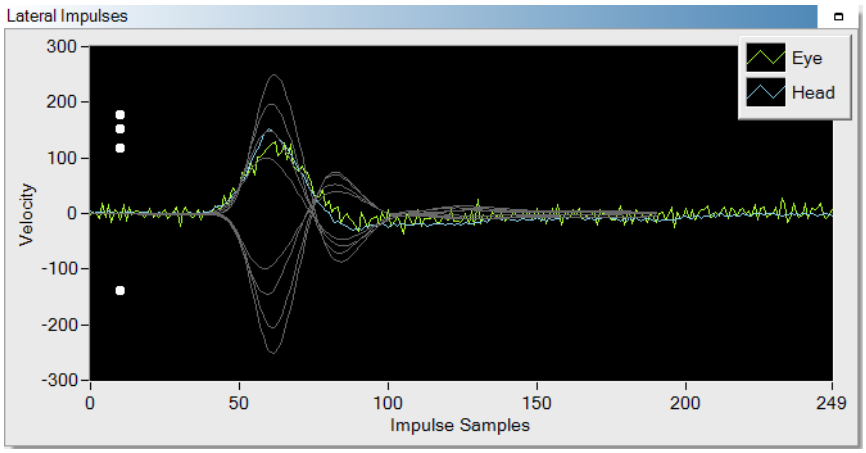


3.8.2.2 Lateral Impulses window

In this window, the gray training lines represent the shape of good head impulses at a variety of velocities: 50, 100, 150, and 200. Negative velocities represent rightward head impulses. Positive velocities represent leftward head impulses.

The actual head and eye traces are superimposed on top of the gray training lines. The head trace displayed in red and with negative velocities represents movement to the right. The head trace displayed in blue and with positive velocities represents movement to the left. The eye trace is always represented in green. White dots display along the Y axis to indicate the velocities of impulses collected.

The collection algorithm analyzes the data in real-time as it is being collected. If the head impulse meets the algorithm criteria, an accepted head impulse will be counted in the collection window. A head impulse that matches the training curve is typically counted as an accepted (valid) head impulse.



Comparing the actual head traces (in this example shown in blue) against the training lines helps ensure that the tester performs quality head impulses and that only good data is included in the analysis.

Note · *You can find more information in the ICS Impulse training video or at* ***www.headimpulse.com****.*

3.8.2.3 Adding remarks

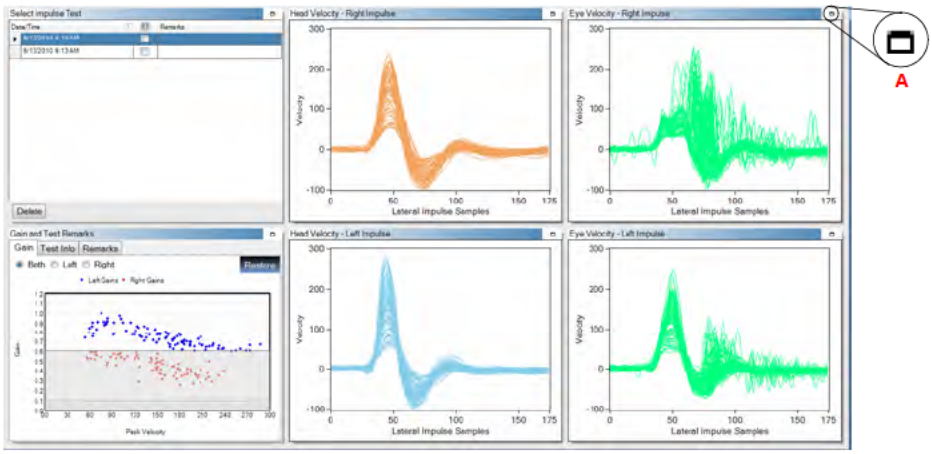
To enter remarks, click **Remarks** in the **Gain and Test Remarks** window. For information using the editing tools, refer to 2.4 Entering test remarks/report findings ▶ 12.

3.9 Lateral head impulse single test data analysis

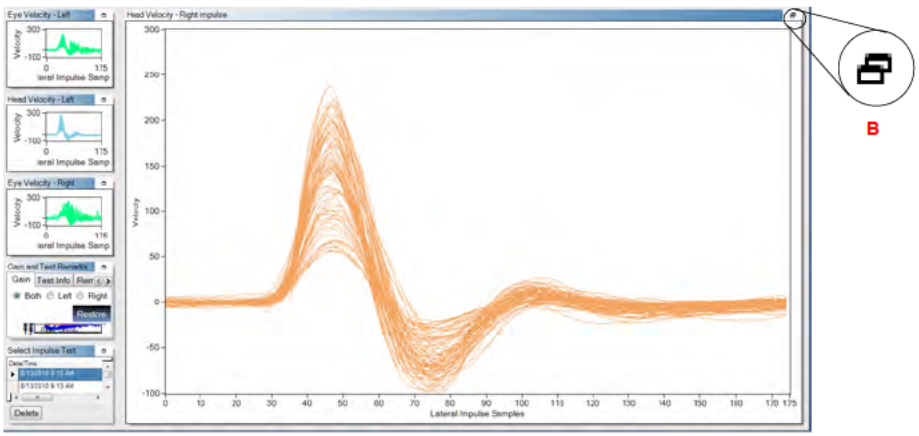
Analysis of the test results can be viewed in the **2D** or **3D Analysis** window group.

3.9.1 Switching between viewing modes

The normal viewing mode shows all windows at the same size as shown in this example of the **2D Analysis** set of windows. To enlarge a window, click the small box (A) in the top right corner.



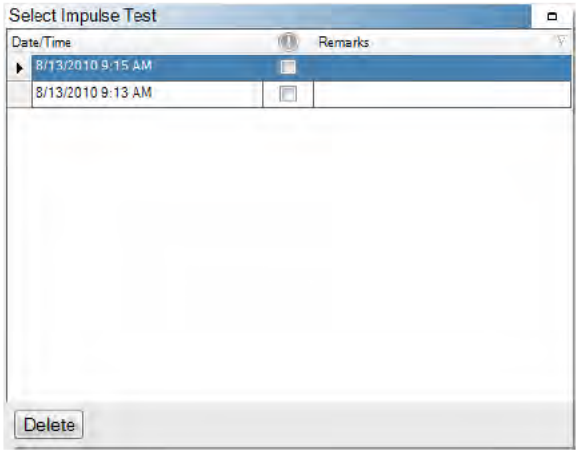
Repeat to enlarge a second window. (Only two windows can be enlarged at one time.) To return to the normal viewing mode, click the two overlapping boxes (B) in one of the enlarged windows.



Note · *Windows open with the normal viewing mode when OTOsuite Vestibular is restarted.*

3.9.2 Select Impulse Test

To view a specific test, click on the test row to highlight the test:



Reorder the list to make it easier to locate a specific test: Click the column header to change the order of how the tests display. For example

- Click **Date/Time** to change the order from ascending order (older to more recent) to descending order (more recent to older).
- Click **Remarks** to change the order from ascending order (A to Z) to descending order (Z to A).

To mark a test for a specific purpose (for example, to indicate abnormal results), click in the check box under the column heading marked with this symbol: .

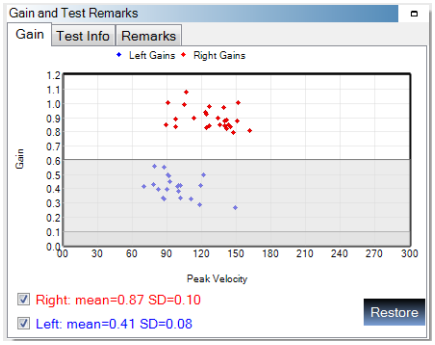
Add remarks in the Remarks column or in the **Remarks** window of the **Gain and Test Remarks** window.

Click **Delete** to delete the selected test.

Caution · *If you choose* **Yes** *at the prompt, the test data is permanently deleted. There is no possibility to retrieve the test.*

3.9.3 Gain and Test Remarks

Gain is the ratio of the eye movement velocity to the head movement velocity. The **Gain** window displays gain values along the Y axis and corresponding peak velocities along the X axis. (The peak velocity is the maximum velocity for each of the 175 samples representing that particular head impulse test).

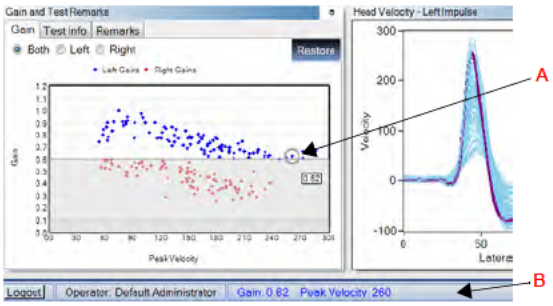


Select the **Left** or **Right** check box to view only left or right gains. To view both left and right gains, select both check boxes. At the top of the graph a legend identifies the colors used for left and right values.

Data in the white zone is within normal limits. Data in the light gray area indicates unilateral loss. Data in the dark gray area indicates bilateral loss. (Boundaries defined according to normative data research.[1]) All right and left gains are averaged. The mean and standard deviation (σ)are displayed below the gain graph.

Note · *Refer to 3.12 Head impulse options ▶ 53 to change the boundaries for normative data.*

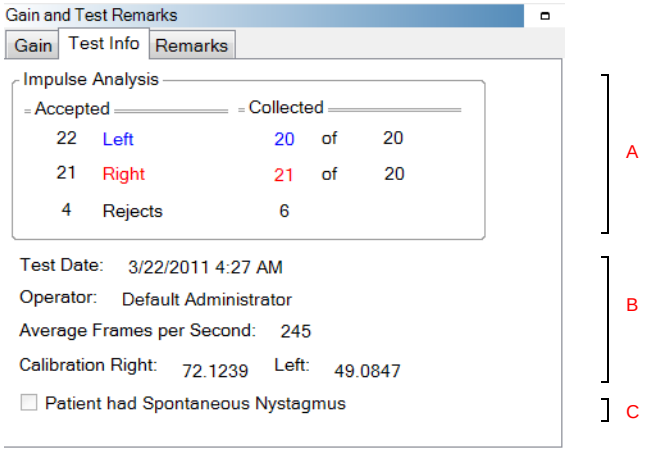
To see the gain and peak velocity values for a data point, move the cursor over the point. The gain value displays in the window next to the point (A). Both gain and peak velocity values display in the status bar (B).



With the value displayed, double-click the mouse to delete the point. Click **Restore** to restore all original data points.

[1] MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy. Neurology 73 (14): 1134-1141.

Click **Test Info** to view test information.



In the top part of the window (A) the **Impulse Analysis** lists both accepted impulses (the head impulse data that passed the collection and analysis algorithm) and collected impulses (the head impulse data that passed the collection algorithm as shown in the collection window). The analysis algorithm analyzes all the data as a whole, and more accurately identifies the impulses.

ICS Impulse uses different algorithms during collection and analysis, the number of collected and accepted impulses may vary. Typically the accepted number is less than or equal to the collected number. In some cases the analysis algorithm will find more valid head impulses than the collection algorithm. If the numbers vary greatly, this may be an indication that the impulse test is not being performed properly (e.g impulses generated too close together (within 0.5 sec), or impulses with multiple "peaks" being generated).

In the middle part of the window (B) the test parameters are listed: **Test Date**, **Operator**, **Average Frames per Second** (frequency of data acquisition by the camera in the goggles), and **Calibration** (**Right** and **Left**) values collected during calibration and used for the analysis.

In the bottom part of the window (C) the check box **Patient had Spontaneous Nystagmus** is checked if the tester selected the **Patient has Spontaneous Nystagmus** check box in the **Collection** window.

To add or modify notes regarding the test, click **Remarks**. For information about entering text or other text-related operations, refer to 2.4 Entering test remarks/report findings ▶ 12.

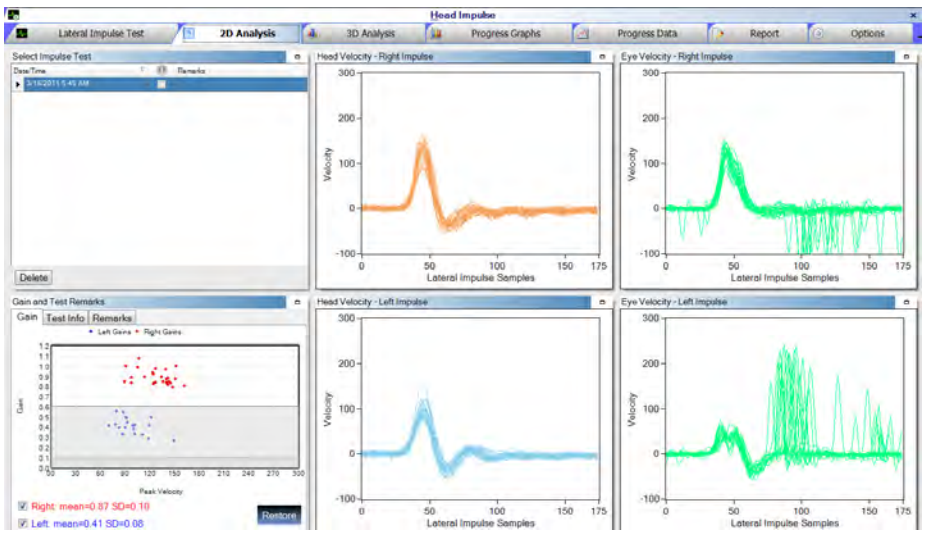
Note · *Text previously added in the* **Select Impulse Test** *window appear in the* **Remarks** *window.*

3.9.4 2D Analysis

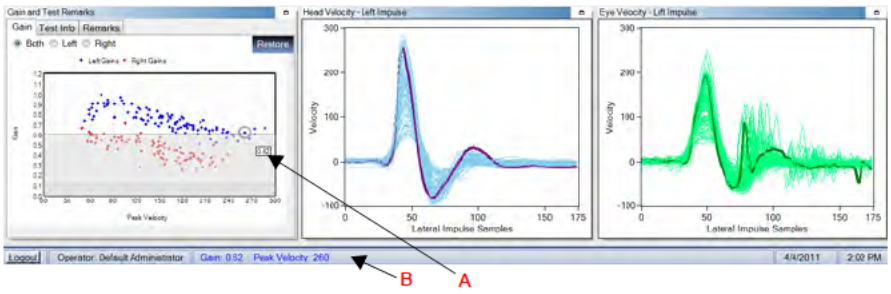
The graphs in the 2D Analysis window are organized to make it easy to compare left and right velocities

- right head and eye velocity data on the top
- left head and eye velocity data on the bottom

Head data is shown in purple. Eye data is shown in green. Data is shown with velocity plotted on the Y axis and impulse samples along the X axis.



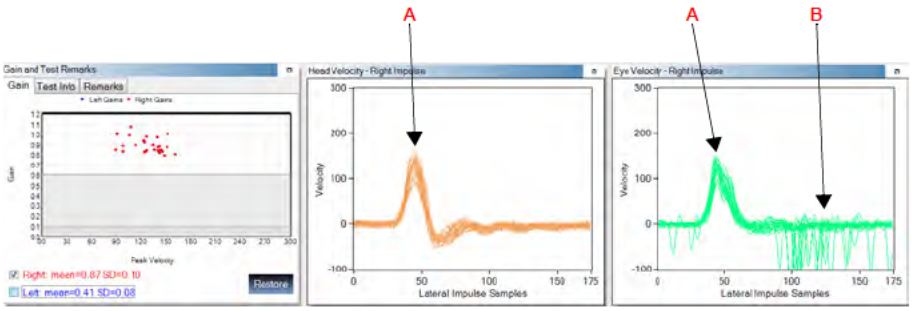
To see the gain and peak velocity values for a data point and the corresponding head and eye velocity traces, move the cursor over the point. The gain value displays in the window next to the point (A). Both gain and peak velocity values display in the status bar (B).



3.9.4.1 Understanding the 2D analysis

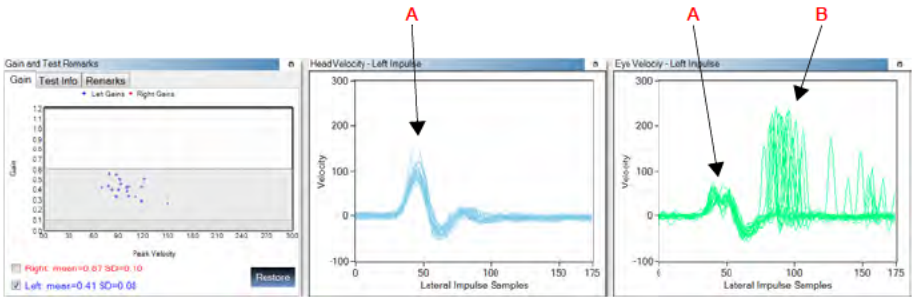
Within Normal Limits

This is an example of head impulse data that is within normal limits. Looking at the Gain graph the data points are all within the normal range (in the white area with a gain of 0.6 to 1). The head data shows very well performed head impulses and the eye data shows a vestibular ocular reflex (A) that mirrors the head velocities. There may be a few catch-up saccades. Downward spikes are a result of spontaneous nystagmus (B).



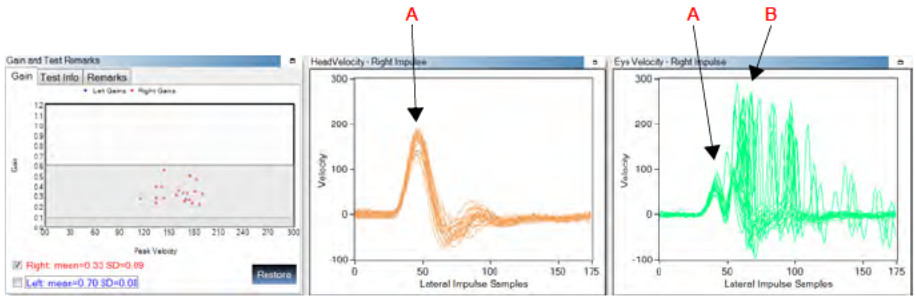
Overt saccades

This is an example of head impulse data that exhibits an abnormal vestibular ocular reflex indicated by the gain response and the catch-up saccades. Looking at the Gain graph the data points are all within the grey range indicating a unilateral loss. The head data shows very well performed head impulses and the eye data shows an inadequate vestibular ocular reflex (A) that does not mirror the head velocities. There are overt catch-up saccades present (B). Catch-up saccades are easier to visualize in the 3D analysis.



Covert Saccades

This is an example of head impulse data that exhibits an abnormal vestibular ocular reflex indicated by the gain response and the catch-up saccades. Looking at the Gain graph the data points are all within the grey range indicating unilateral loss. The head data shows well performed head impulses and the eye data shows an inadequate vestibular ocular reflex (A) that does not mirror the head velocities. There are covert catch-up saccades present (B). Catch-up saccades are easier to visualize in the 3D analysis.

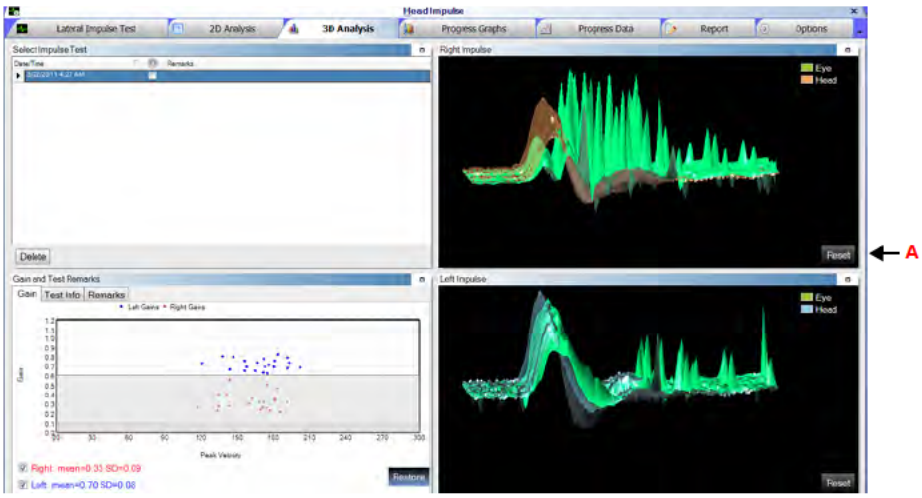


3.9.5 3D Analysis

The graphs in the 3D Analysis window are organized with the right impulse graphs on the top and the left impulse graphs on the bottom.

Eye data is shown in purple. Head data is shown in green.

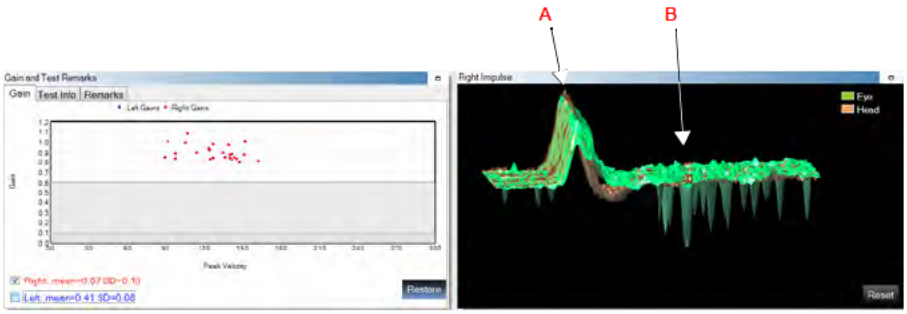
The data can be viewed from 360 degrees. To see the data from different views, hold down left-mouse button and rotate as desired. To return the data to the original view, click Reset (A).



3.9.5.1 Understanding the 3D analysis

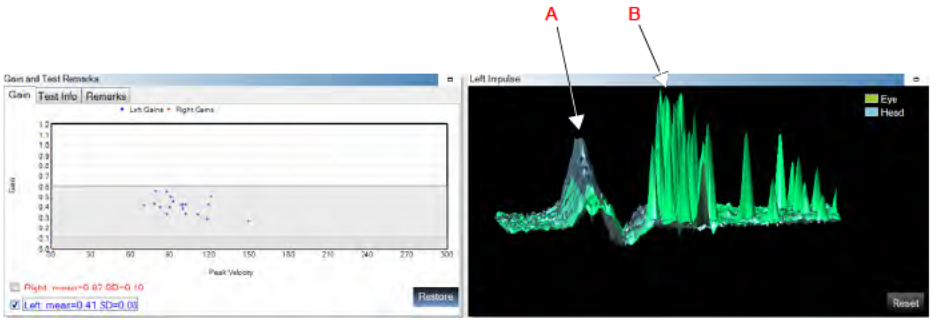
Normal

This is an example of head impulse data that is within normal limits. Looking at the Gain graph the data points are all within the normal range (in the white area with a gain of 0.6 to 1). The head data shows very well performed head impulses and the eye data shows a vestibular ocular reflex (A) that mirrors the head velocities. There may be a few catch-up saccades. (Downward spikes are a result of spontaneous nystagmus (B).



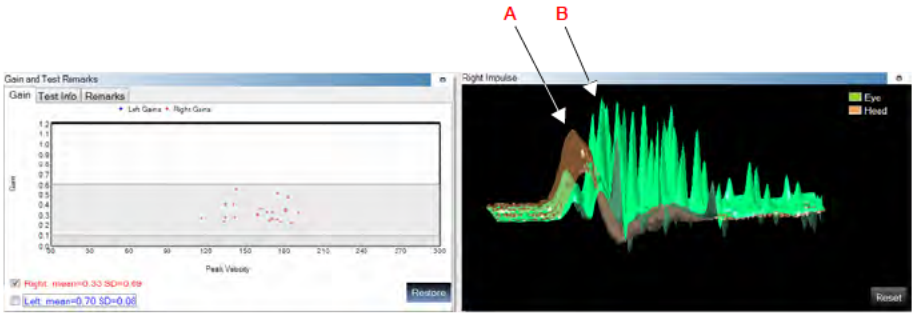
Overt saccades

This is an example of head impulse data that exhibits an abnormal vestibular ocular reflex indicated by the gain response and the catch-up saccades. Looking at the Gain graph the data points are all within the grey range indicating a unilateral loss. The head data shows very well performed head impulses and the eye data shows an inadequate vestibular ocular reflex (A) that does not mirror the head velocities. There are overt catch-up saccades present (B).



Covert saccades

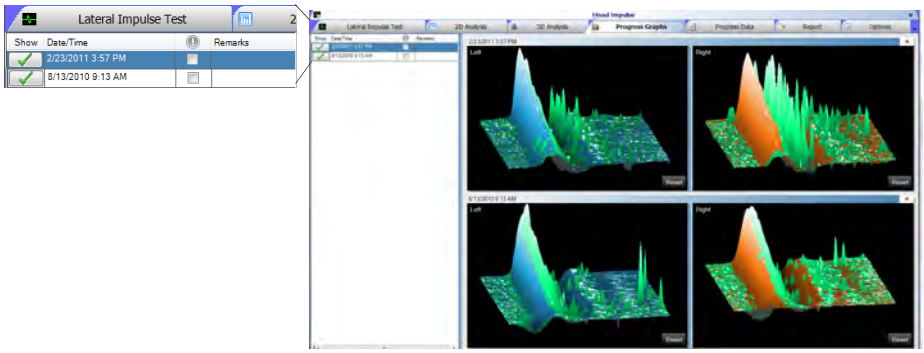
This is an example of head impulse data that exhibits an abnormal vestibular ocular reflex indicated by the gain response and the catch-up saccades. Looking at the Gain graph the data points are all within the grey range in indicating unilateral loss. The head data shows well performed head impulses and the eye data shows an inadequate vestibular ocular reflex (A) that does not mirror the head velocities. There are covert catch-up saccades present (B). Notice how the covert catch-up saccades are closer to the head data whereas the overt catch-up saccades were further to the right of the head data.



3.10 Lateral head impulse multiple-test data analysis

Analysis of multiple test sessions can be viewed in the **Progress Graphs** or **Progress Data** window group. This allows the user to compare a patient's results from different test dates. By comparing test sessions, the user can see if there is improvement or compensation has occurred.

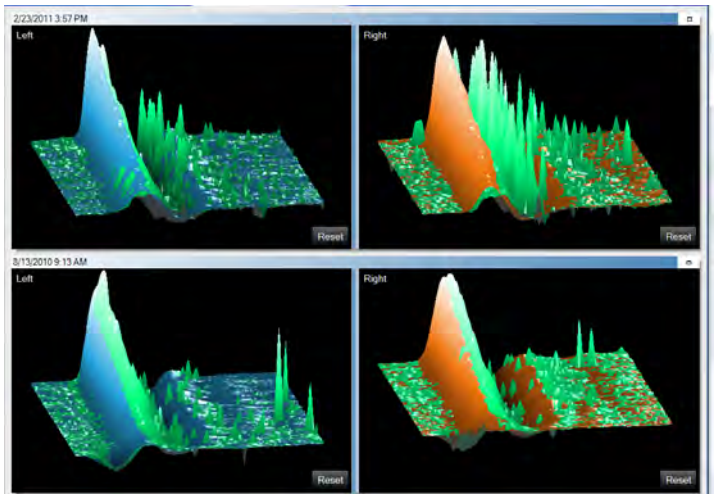
If there are three or more tests available, the three that display initially are the first test, the most recent test, and the test currently displayed in 2D and 3D window. To remove a test from the display, click the button next to the test name in the **Show** column. To add a test(s) to the display, click the button next to each test name in the **Show** column.



Note · *The viewing mode functions the same as the 2D and 3D Analysis. Refer to* *3.9.1 Switching between viewing modes ▶ 34*.

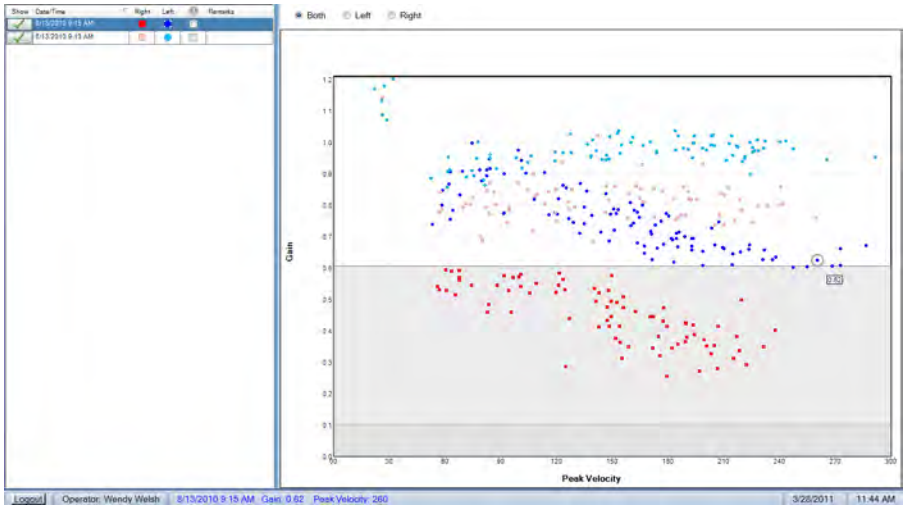
3.10.1 Progress Graphs

Three test sessions can be viewed together in this window. The 3D graphs display as described in 3.9.5 3D Analysis ▶ 42 except that this window displays multiple tests with the right impulse graphs on the right side and the left impulse graphs on the left side.



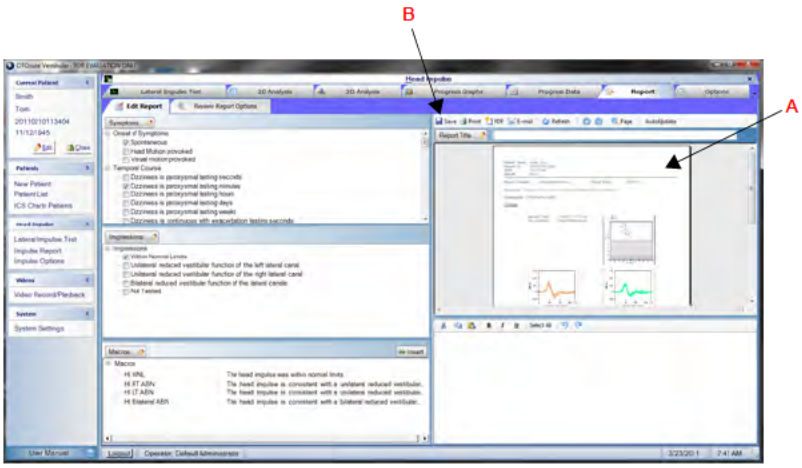
3.10.2 Progress Data

As many as ten test sessions can be viewed together in this window. The gain graph displays as described in 3.9.3 Gain and Test Remarks ▶ 36 except that the legend to identify the colors used for each test is shown in the test selection window.



3.11 Report

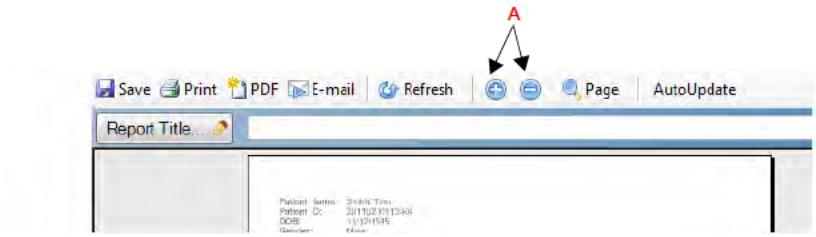
Click **Report**. The **Report** window opens with the default report displayed (A). If you want to save the report without making any changes, click **Save** (B).



Note · *Settings in the* **Report Settings** *window determine which patient data and facility data are included in all reports (6.4 Report Settings ▶ 69). Settings in the* **Review Report Options** *window determine which report options are included in the current report (3.11.4 Review Report Options ▶ 51).*

3.11.1 Report toolbar buttons

To view the report more easily, use the zoom buttons (A) to increase or decrease the magnification of the page. Click **Page** to see the entire page.



Click **Save** to save the default report with no changes made. Click **Print** to print the report, **PDF** to create a PDF of the report, or **E-mail** to email the report.

To change the default title (used for all future reports and for the current report), click **Report Title** and enter the new title. To change the title only for the current report, enter text in the field next to the button.

To make other changes, refer to 3.11.2 Edit Report ▶ 48, 3.11.3 Adding findings ▶ 51 and 3.11.4 Review Report Options ▶ 51.

Click **Refresh** to update the preview after a changes or a set of changes. Or, to update the preview after each additional change, click **AutoUpdate** to turn on automatic updating. Click **AutoUpdate** again to turn off automatic updating. (A rectangular outline around the button indicates automatic updating is turned on.)

3.11.2 Edit Report

Items can be added to the report by selecting from a list of symptoms and a list of impressions or by inserting a macro to enter pre-written text into the findings text field.

- Symptoms: List of symptoms the patient reports during the case history. When selected they display as a list in the report titled **Symptoms**.
- Impressions: The overall medical impression based on the test results. When selected they display as a list in the report titled **Impressions**.
- Findings: The overall finding for each test performed. You can type text directly in the findings field, or you can insert text in the findings field using macros that you create with your choice of commonly used text.

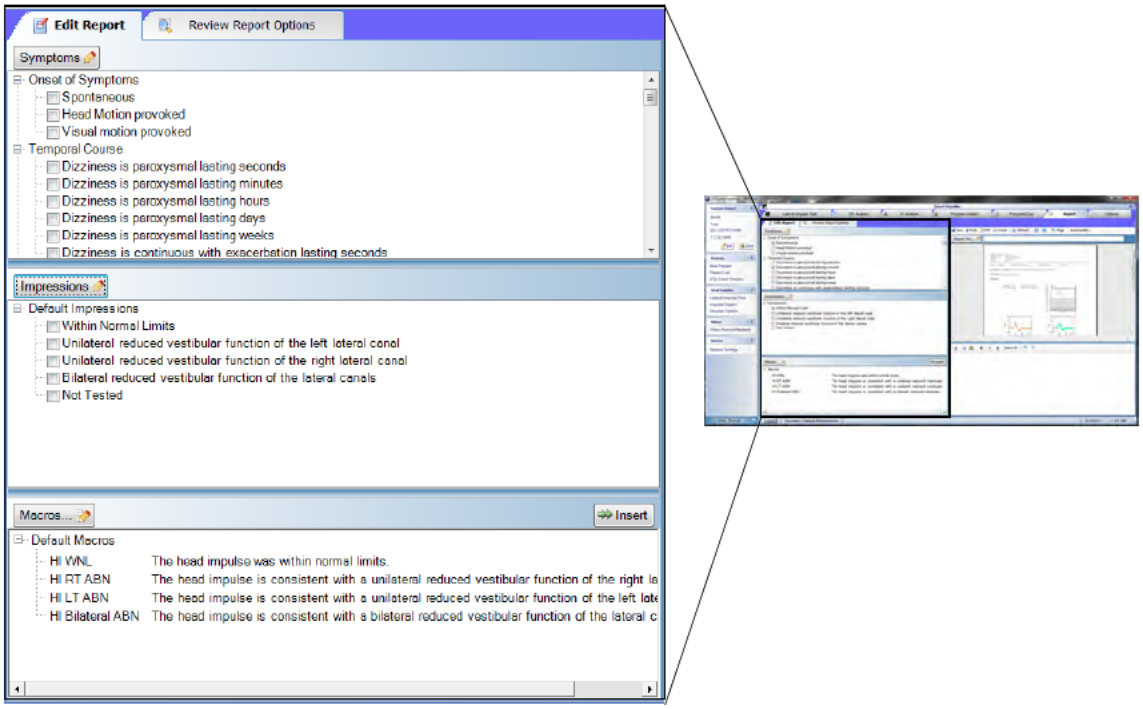
Note · *If the report lists do not display, they need to be imported. Refer to 7.1.7 Customizing for your facility ▶ 88.*

3.11.2.1 Selecting items from report lists

From the **Symptoms** list and **Impressions** list, click each check box that applies.

From the **Macros** list, click the name of the macro and click **Insert**.

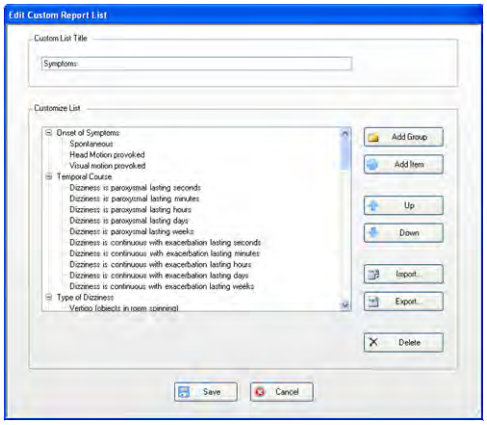
Note · *Some macros have text longer than what can be displayed in the window. To see the entire text, roll the cursor over the macro name.*



3.11.2.2 Customizing report lists

Note · *Report lists can be imported into one system, customized, and exported to other standalone workstation systems. This is useful when a facility has multiple standalone workstations.*

To customize report lists, click **Symptoms**, **Impressions**, or **Macros**.



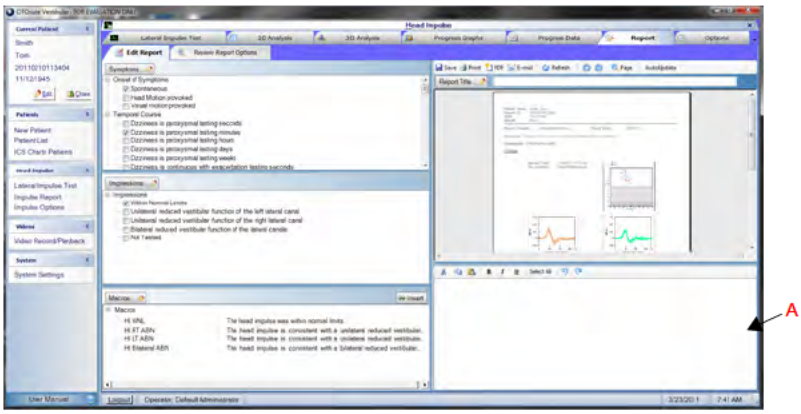
- To rename a group or item, click the item and enter the new name.
- To add a group, click **Add Group**.
- To add an item, click a group name, and click **Add Item**.
- To rearrange the order of items in a group or the order of groups in a list, click the item or group name. Click **Up** or **Down** to move the item or group.
- To import sample report lists, refer to 7.1.7 Customizing for your facility ▶ 88.
- To export the current report list, click **Export**. Change the file name and location as needed. Click **Save**.
- To delete an item or group, click the item or group, and click **Delete**. Click **OK** to permanently delete the item or group.

Note · *If a mistake is made, you can re-import the manufactured-supplied list.*

After making changes, click **Save** to save the changes. Click **Cancel** to discard the changes.

3.11.3 Adding findings

Text that is entered into the findings field (A) will appear in the Findings section of the report. For information about using the editing tools, refer to 2.4 Entering test remarks/report findings ▶ 12.



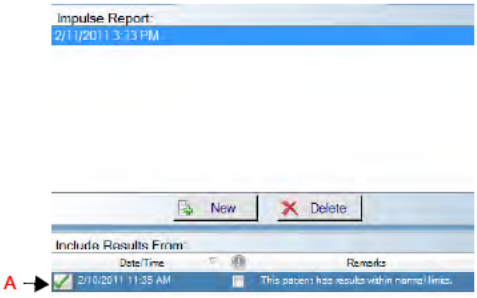
3.11.4 Review Report Options

In this window you can preview all ICS Impulse reports created for the patient including the default report automatically created for the current test.

You can make a new report that combines several ICS Impulse test sessions into one report. To create a new report, click **New**. To delete an existing report, click the report name and click **Delete**. After the report has been deleted it cannot be recovered.

Note · *If the current report is the default report without any changes made to the report, both* **New** *and* **Delete** *are disabled unless you press* **Save**.

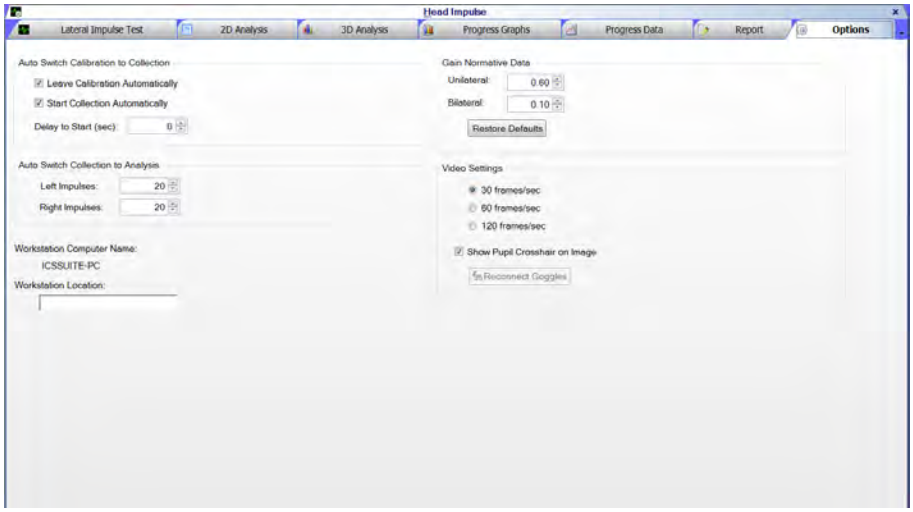
To add a test to the report, click the check box (A) at the left of the test name in the **Include Results From** list. Clicking the check box again removes the test from the report.



Click the check box for each report option to be included in the report. Click **Select All** to select all options. Click **Deselect All** to unsettle all options. The selections made are applied to all reports selected.

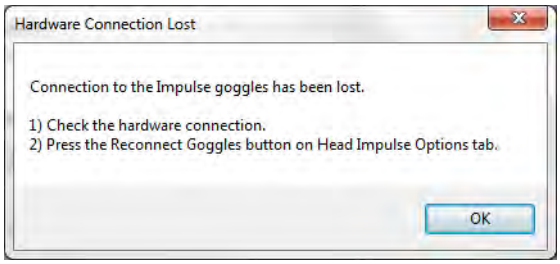
Note · *The gain graph along with the 2D impulse graphs from one test session should fit on one page. If multiple tests sessions are chosen, or a progress graph is included, then the report will include multiple pages. 3D graphs can not be included in the report.*

3.12 Head impulse options



Item	Choices
Auto Switch Calibration to Collection	**Leave Calibration Automatically** Collection window opens automatically as soon as calibration is finished.
	Start Collection Automatically This option is the default option and is recommended for optimum image quality. Only enabled if **Leave Calibration Automatically** is selected. Collection starts automatically x seconds after the collection window opens. Time is set in **Delay to Start (sec)** field.
Auto Switch Collection to Analysis	Number of impulses is set in the **Left Impulses** and **Right Impulses** fields.
Workstation Computer Name	Not possible to change. (The name is assigned to the computer in the Windows system control panel.)
Workstation Location	User-defined name to describe the location of this PC

Head impulse options

Item	Choices
Gain Normative Data	Set cutoff lines **Unilateral** - the cutoff line between normal and unilateral loss **Bilateral** - the cutoff line between unilateral loss and bilateral loss
	Restore Defaults - restores the normative data cutoff values as documented in published data and recommended by Otometrics. [a]
Video Settings	**30 frames/sec** **60 frames/sec** **120 frames/sec** Select one of the options to set the video record speed when collecting video during head impulse testing.
	Show Pupil Crosshair on Image Select to display the crosshair on the pupil in the Video window.
	Reconnect Goggles Click to reconnect the goggles (software-to-hardware connection) as instructed by this error message: 

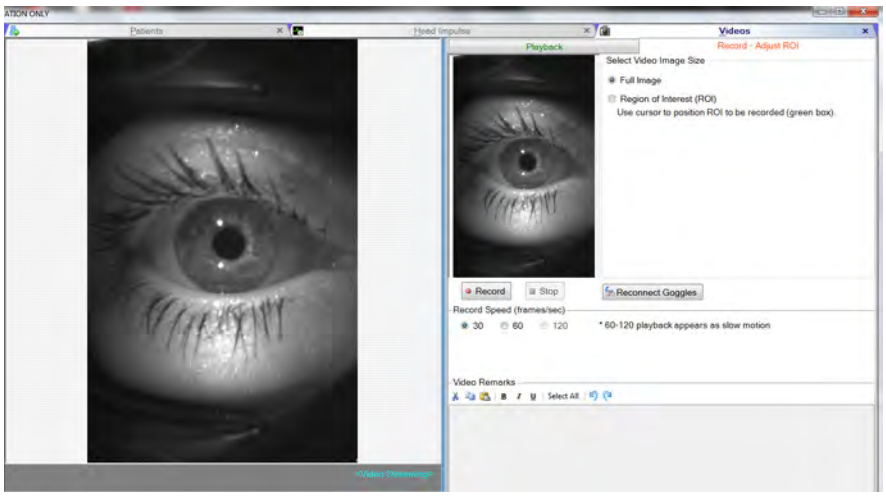
[a] MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy. Neurology 73 (14): 1134-1141.

4 Video Record/Playback

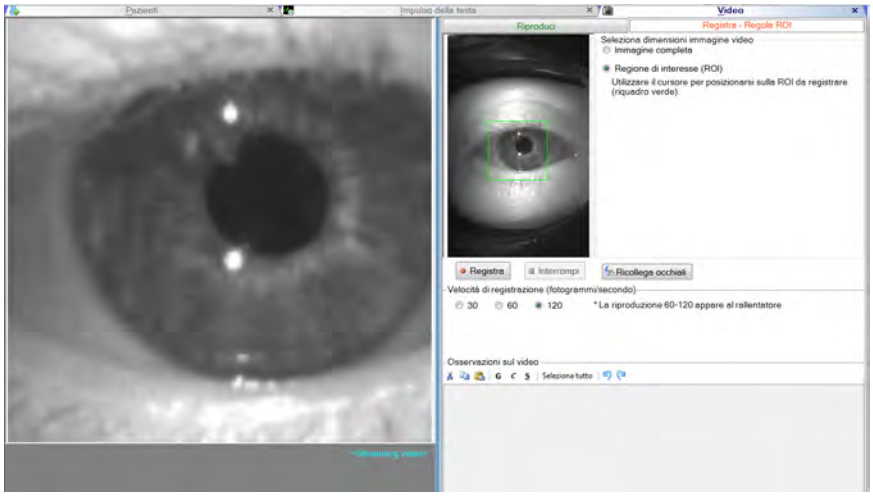
4.1 Record

When recording a video, there are 2 options: **Full Image** or **Region of Interest** (pupil).

Full image (30 and 60 fps only)



Region of Interest (ROI)

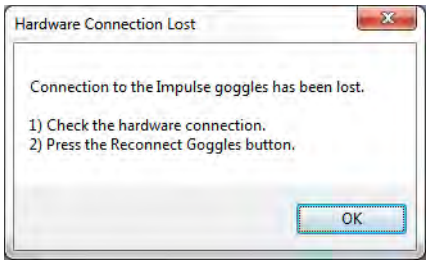


Record - start video recording

Stop - stop video recording

Note · *There is no limit to the length of the video recording; however, the longer the video recording is the larger the file will be on the hard drive.*

Reconnect Goggles - reconnect goggles (software-to-hardware connection) as instructed by this error message:

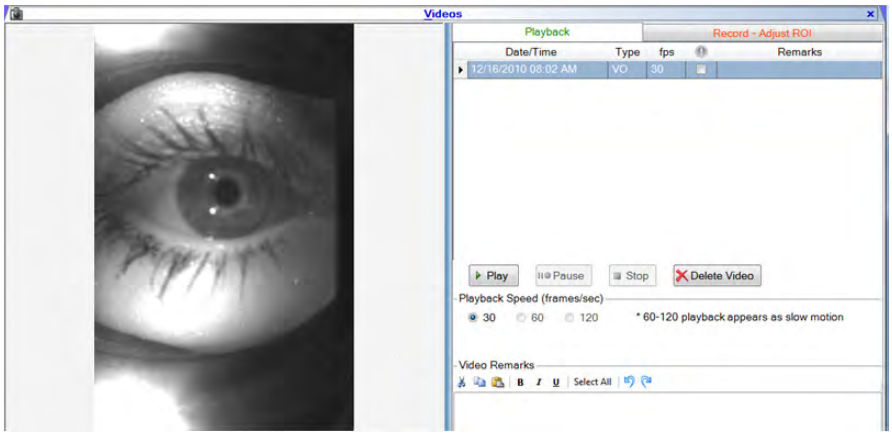


Record Speed - Video Recording can be performed at 30 or 60 frames per second. When using ROI the extra option of 120 frames per second is available. If the video is recorded at 60 or 120 frames per second it can be played back in slow motion or at normal speed. At normal speed some frames will be skipped.

Remarks can be entered directly in the text field (A). For information about entering using the editing tools, refer to 2.4 Entering test remarks/report findings ▶ 12.

4.2 Playback

The list of videos that have been collected for the selected patient is displayed. This includes videos collected during head impulse testing.



Date/Time - date and time the video was collected

Type - indicates if the video was collected during head impulse testing (HI) or separately (VO = video only)

Fps - the frame rate in which the video was recorded (frames per second)

Unique identifier - To mark a file for a specific purpose (for example, to indicate abnormal results), click in the check box under the column heading marked with this symbol: .

Play - plays the video

Pause - pauses the video

Stop - stops the video

Delete Video - deletes the video. After a video has been deleted it cannot be recovered. It is recommended to not save videos that are not needed in order to save disk drive space.

Playback Speed - The playback speed can only be selected if the video was collected at a speed other than 30 frames per second. If a video is played back at 30 frames per second it is normal speed. A video played back as 60 or 120 frames per second will play in slow motion because the refresh rate of the screen is 30 frames per second. By playing 60 or 120 frames within the same 30 seconds the video will appear to play in slow motion.

Video Remarks - Text can be entered directly in the text field (A). For information about entering using the editing tools, refer to 2.4 Entering test remarks/report findings ▶ 12.

5 Patient Lists

The **Patients** window lists the patient data entered into the **OTOsuite Vestibular Database**. The **Patients** window also includes the ability to view patient information from the **ICS Chartr Database** and the **Patient Export** and **Patient Import** windows from which you can export data out of and import data into the OTOsuite Vestibular database.

List operations

Each list has rows with patient information separated into columns according to the type of information: Patient name (last and first), ID, etc. For some of the lists the **HI** (Head Impulse) and **Video** columns are included. A check mark in these columns indicates the following:

- **HI** - head impulse data is saved
- **Video** - video data is saved

Note · *The symbol* ⓘ *indicates the user has chosen to mark one or more tests or videos for a specific purpose (for example, it may refer to abnormal results, results to be used for a study, etc.)*

Selecting more than one patient	Select all patients: Click **Select All**. To deselect all selected patients, click, **Deselect All**. Select a group of patients: Click the first name of the set, hold down the Shift key and click the last name in the set. Select individual patients: Keep the Ctrl key pressed as you click on each patients.
Reordering lists	Click the column header, to reverse the order. For example • Click **Patient Name** to change the order from A-Z to Z-A. • Click **DOB** to change the order from oldest-youngest to youngest-oldest.

Changing column width	In the column heading of two adjacent columns, click the cursor on the line dividing two columns. The cursor changes to a double-headed arrow. Drag the cursor to increase or decrease the column width.

5.1 OTOsuite Vestibular Database

From the **Patients** group, click **Patient List**. The window that opens provides access to all OTOsuite Vestibular patients. This list includes a column that shows the most recent date that the patient data was modified.

5.2 Chartr Database

From the **Patients** group, click **ICS Chartr Database**. The window that opens provides access only to the list of patients in the Chartr database. Test data collected in Chartr VNG/ENG or EP can not be viewed in the OTOsuite Vestibular application.

In order to reduce data entry time, patient's information in the Chartr database can be imported into the OTOsuite Vestibular database:

1. Select the patient(s) to be imported.
2. Click **Import**.
3. Click **OK**.

Imported displays in the **Status** column to indicate the process was completed. The record of imported patients clears when the application is closed.

The Chartr software must reside on the same computer as the OTOsuite Vestibular software for the database to be accessible. If the database is accessible, the ICS Chartr Patients item will appear in the navigation panel of the **Patients** group.

5.3 Patient Export

From the **Patients** group, click **Patient List**. Click **Patient Export**. The window that opens provides access to all OTOsuite Vestibular patients. This list includes a column that shows the most recent date that the patient data was modified.

To export one or more patients:

1. Select the patient(s).
2. Choose the export file type(s): XML, PDF, ASCII or some combination of these.

- **XML Files** (complete patient data as stored in the database to be used for electronic medical records)
- **PDF Reports** (all reports for this patient to be used for electronic medical records)
- **ASCII Test Results** (ICS Impulse test results formatted in a CSV - comma separated values - file to be used for research purposes with programs such as Excel or MatLab)

3. Click **Export**.

Exported displays in the **Status** column to indicate the process was completed. The record of exported patient(s) clears when the application is closed.

5.4 Patient Import

From the **Patients** group, click **Patient List**. Click **Patient Import**.

To import one or more OTOsuite Vestibular patient XML files into the OTOsuite Vestibular database:

1. Click **Open Folder** and navigate to the location of the files to be imported.
2. Click **OK**.
3. In the list of patient files, the patient name (last and first), ID, gender, date of birth are provided. Select the file(s).
4. Click **Import**.

Imported displays in the **Status** column to indicate the process was completed. The record of imported files clears when the application is closed.

Click 

to update the list of patient files in the **Import** folder.

6 System Settings

Some System Settings windows are only accessible to those with a user profile that includes one or both of these of these choices selected: **Add User as Administrator** or **Allow User to Change System Settings.** Refer to 6.1.1 Set up a new user ▶ 64.

6.1 Administrator

Only users with **Add User as Administrator** selected in their profile have access to this window.

Use this window to manage the list of users. Also, refer to this window to set or obtain the location of where video files are located. Refer to 7.1.9 Changing the shared network location for videos ▶ 90 for more information.

List operations

The user data is separated into columns according to the type of information: Last name, first name, user name, etc.

Reordering lists	Click the column header, to reverse the order. For example, Click **Last Name** to change the order from A-Z to Z-A.
Changing column width	In the column heading of two adjacent columns, click the cursor on the line dividing two columns. The cursor changes to a double-headed arrow. Drag the cursor to increase or decrease the column width.

6.1.1 Set up a new user

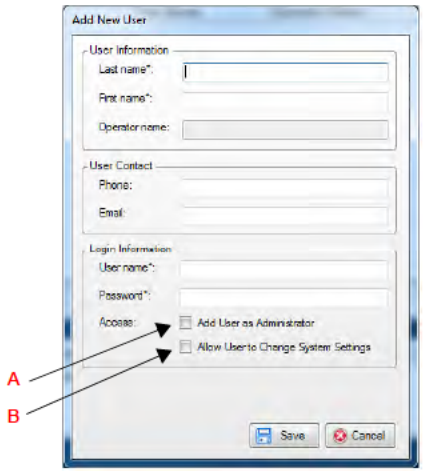
Note · *IMPORTANT! Set up at least one user with administrator privileges.*

1. Click **New**.
2. To give the user administrator privileges, click **Add User as Administrator** (A).
   This setting gives the user the ability to add, edit, or delete users as well as to specify the location for videos.
3. To give the user privileges to change the system settings, click **Allow User to Change System Settings** (B).
   This setting gives the user access to
   – Set cutoff lines for gain normative data (see 3.12 Head impulse options ▶ 53)
   – Enter e-mail settings used for mailing reports (see 6.2 Workstation Settings ▶ 66)
   – Choose patient, facility, and operator information to include in reports (6.4 Report Settings ▶ 69)
   – Perform database operations (6.5 Database ▶ 69)
   – Enter GDT interface settings (6.6 GDT Interface ▶ 71)

Note · *Both user name and password are case sensitive.*

4. Enter data. Entries that cannot be left blank are marked with an asterisk (*).

5. Click **Save** to save the changes. Click **Cancel** to discard the changes.



6.1.2 Edit user data

1. Select the user from the list.
2. Click **Edit**.
3. Edit the changes making sure all required fields marked with an asterisk (*) are completed.
4. Click **Save** to save the changes. Click **Cancel** to discard the changes.

6.1.3 Delete user

1. Select the user from the list.
2. Click **Delete**.
3. Click **OK**.

6.2 Workstation Settings

From the **System** group, click **System Settings**. Only users with **Allow User to Change System Settings** selected in their profile have access to certain fields in this window.

Complete the fields with information provided by the system administrator.

Item	Settings
Workstation Computer Name	Not possible to change. (The name is assigned to the computer in the Windows system control panel.)
Workstation Location	User-defined name to describe the location of this PC
Installation Profile	Type of installation that was chosen during the software install process - standalone, server, client or both (server/client)
Client/Server Database Port	Port number used by the client to access the database on the network
E-Mail Configuration	**E-mail Server** - the computer in the network responsible for receiving, routing and delivering email messages
	From E-mail - the email address from which emails are sent
	To E-mail - default email address to which the emails are sent (the user has ability to change or add to this email address)
SMTP Port (Default = 25)	This defines the simple mail transfer protocol port (25 is most typically used)
Outgoing Email Account	**Use default network credentials** - uses the default network credentials to log into the email server
	Log in with user name and password - uses the user name and password as specified in the fields below this choice to log into the email server

Item	Settings
Select Program Language	**Use Operating System Language** - when selected the OTOsuite Vestibular software will use the operating system language. If the operating system language is one that is not supported, the OTOsuite Vestibular software defaults to English. Instead of selecting **Use Operating System Language** choose from the list of languages.
Reset Navigation Bar	If changes had been made to the items in the navigation bar, click to reset the panel to the manufacturer default settings.

6.3 Facility Info

From the **System** group, click **System Settings**. Only users with **Allow User to Change System Settings** selected in their profile have access to certain fields in this window.

The information entered here is used in the reports as specified in the **Report Settings** window (Refer to section 6.4).

Enter text in the fields for the facility information (maximum number of characters is 32) and add a logo:

Note · *Logos are resized.*

1. Click **Browse...**.
2. Navigate to the location of the file to be used for the logo.
3. Select the file and click **Open**.

To remove the logo, click **Clear**.

6.4 Report Settings

From the **System** group, click **System Settings**. Only users with **Allow User to Change System Settings** selected in their profile have access to certain fields in this window.

Note · *Settings in this window determine which patient data and facility data are included in all reports. Settings in the* **Review Report Options** *window determine which report options are included in the current report (3.11.4 Review Report Options ▶ 51).*

Click on each item to be included in all ICS Impulse reports. To change the paper size, select either **Letter** or **A4**. Select **User who last updated the patient report**, or **User who collected test data** or both to include these names in the ICS Impulse reports.

6.5 Database

Note · *If the database is located on a server, all users must be logged off of client workstations prior to performing an archive or backup of the data.*

From the **System** group, click **System Settings**. Only users with **Allow User to Change System Settings** selected in their profile have access to database functions.

This window lists the OTOsuite Vestibular database version and a location where the database is stored.

Note · *Drag the horizontal thick blue bar to see more of the list displayed in the window. Refer to 2.3 Understanding the OTOsuite Vestibular screen ▶ 10 for more information about changing window sizes.*

6.5.1 Archiving

Note · *IMPORTANT! Archiving removes all patient data and videos from the OTOsuite Vestibular database.*

Clicking **Archive Now** first creates an SQL server backup copy of the OTOsuite Vestibular database. The process is identical to clicking **Backup Now** (6.5.2). After creating the backup copy, the patient information, test data and videos are archived to the location listed under **Archive All Patients**. Archiving exports the data to XML and AVI (video) files.

To cancel the archive process, click **Cancel**.

Note · *Archived patient data is removed from the OTOsuite Vestibular database but can be re-imported using the import operation.*

6.5.2 Backing up

It is recommended that a backup be performed monthly. The backup files should be stored in an alternate location.

Caution · *Backups are done for data safety only. Restoring backups can only be done under the direction of Otometrics personnel. Executing a restore will cause the permanent loss of any data collected subsequent to the backup. Data lost due to a restore cannot be recovered.*

Clicking **Backup Now** creates an SQL server backup copy of OTOsuite Vestibular database including all patient information and test data (video files are not backed up) in the location listed under **Database Backup**. Patient data and videos remain in the OTOsuite Vestibular database.

6.6 GDT Interface

From the **System** group, click **System Settings**. Only users with **Allow User to Change System Settings** selected in their profile have access to fields in this window.

The GDT interface is based on the documented GDT standard used only in Germany. When enabled the interface allows file transfers between a German practice management system (PMS) and the OTOsuite Vestibular system. The external PMS system can be used to launch the OTOsuite Vestibular system and open (or create) a specific patient. Once the impulse test is complete, a report is returned to the PMS system.

To enable the GDT interface to a Practice Management System, select the check box **Enable GDT Interface to Practice Management System**.

Item	Settings
OTOsuite Vestibular System Name	User-defined name. Limited to 4 characters. Used as part of the filename to identify the OTOsuite Vestibular system.
PMS System Name	User-defined name. Limited to 4 characters. Used as part of the filename to identify the PMS system.
Test Type	GDT-defined code used for file transfers to the Practice Management System. Limited to 6 characters.
File Transfer Timeout (secs)	Limit for the number of seconds OTOsuite Vestibular will attempt to obtain a file written by PMS.
Character Set	**ASCII** - standard character set **ANSI** - character set includes accented letters and other non-English language characters

Item	Settings
GDT Local File Transfer Directories	**Incoming Messages** - specifies the folder location for incoming GDT messages. The OTOsuite Vestibular system picks up messages from this folder. The specified folder must exist. Click **Browse...** to select the folder.
	Outgoing Messages - specifies the folder location for outgoing GDT messages. The OTOsuite Vestibular system places message here to be picked up by the PMS system. The specified folder must exist. Click **Browse...** to select the folder.

6.7 About

From the **System** group, click **System Settings**. Refer to this window to obtain specifications about the OTOsuite Vestibular and component software.

6.8 Error Logs

From the **System** group, click **System Settings**.

Item	Settings
Workstation Computer Name	Not possible to change. (The name is assigned to the computer in the Windows system control panel.)
Workstation Location	User-defined name to describe the location of this PC
System Error Logs	Location where systems error log files are stored
Debug Tracing	**Enable Debug Tracing** - turns on debugging level debug tracing
	Overwrite Existing Debug Files - if checked reuses existing debug files. If **not** checked creates new debug files.
	Trace Categories - fields filled in with information provided by Otometrics support.
	Location of debugging tracing log files

Caution · *Debugging and error tracing should not be used unless instructed by Otometrics representatives or support staff.*

Error Logs

7 Software Procedures

7.1 Installation and Setup

This section describes all steps required to complete the ICS Impulse System setup:

- Installing OTOsuite Vestibular software
- Activating the National Instruments software
- Connecting the goggles
- Importing sample report lists (not required for server-only installations)
- Importing a demo database
- Customizing for your facility
- Installing Abobe Reader
- Setting up a networked database and workstation (not required for standalone installations)

7.1.1 Minimum computer requirements

Operating System	Windows XP 32-bit Professional SP3 or Windows 7 32-bit Professional or Windows 7 64-bit Professional
CPU	Intel i5 processor
Memory	32-bit (Windows XP or Windows 7): 4 GB, 64-bit (Windows 7): 6 GB
Disk Space	300 GB (3 GB of free disk space on C:\ drive for installed software)
Connectors	USB 2.0 IEEE 1394a (Firewire) **Note** · *It is preferable that the USB and Firewire ports are located on the same side of the computer.*
DVD Drive	DVD R/W
Monitor	1600 x 900 Screen resolution
Components	Mouse, keyboard
Internet access	An Internet connection on the ICS Impulse computer is strongly recommended during installation. It will reduce installation time by approximately 20 minutes.

Note · *Our recommended computer brands are Dell™, Hewlett Packard™, and Sony™. Installation problems were experienced with Acer™.*

7.1.2 Software Installation

Note · *The OTOsuite Vestibular software requires 3 GB of free disk space available on the C:\ drive for software installation.*

7.1.2.1 Start installation

Note · *If firewall messages appear, please allow access. For Windows 7 systems, a message that refers to the User Account Control (UAC) may display. Accept this message as it does **not** indicate a problem.*

1. Save any files currently open and quit all programs.
2. Check if a previous version of the National Instruments software has been installed. If so, it is **not** recommended to install OTOsuite Vestibular. If it is required to install OTOsuite Vestibular on this computer, completely remove all previously installed National Instruments software prior to continuing.
3. Insert the OTOsuite Vestibular DVD.

Note · *If the computer is set up to AutoRun, the installation starts. If AutoRun is not initialized, browse the DVD to find this icon*  *.*

4. Click **setup**. (You may need to close the DVD content folder.)
5. If you have Windows 7, a security dialog will appear. Click **Yes**.
6. Installation of OTOsuite Vestibular requires certain applications (such as Windows Installer) to be installed on the computer. If an application(s) is missing, a dialog box lists the missing application(s).
   - Click **Install**. When the application(s) has been installed, the computer will restart.
   - Log in to computer. The installation continues automatically.
7. The Welcome screen appears. Click **Next**.
8. Select **I accept the terms in the license** and click **Next**.

Note · *During the installation of OTOsuite Vestibular, you will see references to various other associated programs that are being installed. These other programs include: SQL Server (OTOsuite Vestibular database), Access Database Engine (supports the Chartr database), National Instruments Vision Acquisition (video), National Instruments Run-Time Engine (pupil detection), National Instruments DAQmx (camera driver), and Infragistics (reporting).*

7.1.2.2 Choose installation type

OTOsuite Vestibular can be installed in a client/server or standalone configuration. In client/server, a single server hosts the database for one or more client workstations where the OTOsuite Vestibular application is installed. We recommend client/server installs to be set up using network domains. Use of workgroups is not recommended because it will require the same user name and password be used on each computer. In standalone a single computer hosts both the database and the OTOsuite Vestibular applications.

Choose how OTOsuite Vestibular will be installed:

- **Standalone installation**
  Hosts SQL database and OTOsuite Vestibular application (no additional clients)
  Select **Standalone** and click **Next.** Continue at 7.1.2.5 Activate the National Instruments software ▶ 80.
- **Network installation - Server**
  Hosts SQL database and a limited version of the OTOsuite Vestibular application (The limited version supports system settings changes and database management. It does not allow viewing tests or collecting test data.) Continue to 7.1.2.3 Server/Both installation ▶ 79.
- **Network installation - Client**
  Hosts OTOsuite Vestibular application
  Continue at 7.1.2.4 Client installation ▶ 79.
- **Network installation - Both (Client/Server)**
  Hosts SQL database and OTOsuite Vestibular application. Can support additional clients.
  Continue to 7.1.2.3 Server/Both installation ▶ 79.

7.1.2.3 Server/Both installation

Before starting the installation, determine the type of installation required:

- Server only - OTOsuite Vestibular database and a limited version of OTOsuite Vestibular software for archiving/backing up data will be installed.
- Both (Server and Client)- OTOsuite Vestibular software and database will be installed.

1. Select **Server** or **Both** and click **Next**.

Note · *Although it is possible to change the port number, we recommended you use the one supplied on the screen.*

2. Make note of the entries for **Port Number** and **Server Computer Name** as this information will be required for all client computers. (For computers connected to a printer, you can click **Print** to print out the information.)
3. If **Both** was selected, click **Next** and continue at 7.1.2.5 Activate the National Instruments software ▶ 80.
4. If **Server** was selected, click **Next** and continue at 7.1.2.6 Complete the installation ▶ 80.

7.1.2.4 Client installation

1. Select **Client** and click **Next**.
2. Enter the port number and the server computer name obtained from the server installation.
3. Click **Next** and continue to 7.1.2.5 Activate the National Instruments software ▶ 80.

7.1.2.5 Activate the National Instruments software

Note · *Before starting this procedure, locate the National Instruments product serial number found on the front of the envelope labeled* ***NI Vision Development Module Run-Time License****.*

1. If this computer is **not** connected to the Internet, click **Skip** and continue at 7.1.2.6 Complete the installation ▶ 80.
2. If this computer is connected to the Internet, complete every field in the form. Ensure that the product serial number is entered correctly.

Note · *IMPORTANT! This is the only opportunity to automatically activate the software. If the product serial number is not entered correctly, the following message will display at the end of the installation. It will be necessary to follow a multi-step procedure to manually activate the software.*

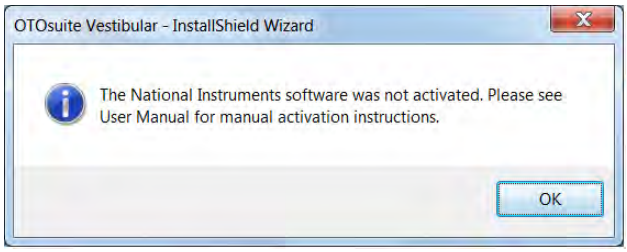
3. Click **Next** and continue to 7.1.2.6 Complete the installation ▶ 80.

7.1.2.6 Complete the installation

Note · *At the end of this procedure, after* **Finish** *is clicked, the computer restarts. A dialog stating* ***Repairing NI Device Driver Database*** *will appear during the restart process.*

1. Click **Install**. A status window appears reporting the progress of the installation.
2. For **server-only** installations, click **Finish** and continue at 7.1.6 Importing demo data ▶ 87
3. For all other installations, click **Finish** and continue at 7.1.4 Connecting the goggles ▶ 85.

   If this dialog appears, click **OK.**



   Click **Finish**. Continue to 7.1.3 Manually activating National Instruments software ▶ 81.)

7.1.3 Manually activating National Instruments software

Note · *Activation of National Instruments software is NOT needed for server-only installations. For all other installations, if automatic activation succeeded, continue at* *7.1.4 Connecting the goggles ▶ 85*.

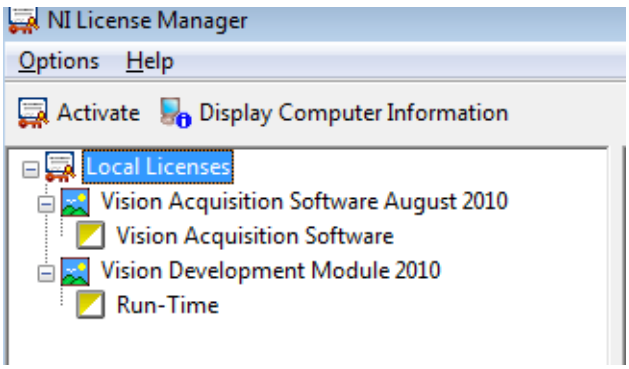
This software is required for data collection, processing, and display. It was installed when OTOsuite Vestibular was installed but requires activation before it can be used.

Note · *Before starting this procedure, locate the National Instruments product serial number found on the front of the envelope labeled* ***NI Vision Development Module Run-Time License***.

7.1.3.1 With Internet access on ICS Impulse Computer

1. Navigate to the required directory: From the desktop, click **Start** > **All Programs** > **National Instruments** > **NI License Manager**.
2. Click the plus sign (+) to display the contents for each license: **Vision Acquisition Software 2010** and **Vision Development Module 2010**.

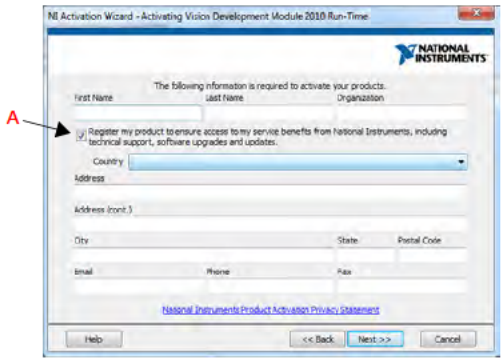
Note · *Solid green checkboxes indicate that the license has already been activated. Continue at* 7.1.4 Connecting the goggles ▶ *85*.



3. Right-click **Run-Time** and select **Activate**.
4. Select

and click **Next**.

5. Locate the product serial number on the front of the envelope labeled **NI Vision Development Module Run-Time License** (this is the bottom number, for example, M111xxxx), type in the number, and click **Next**.
6. Click **Next** and complete these fields: **First Name**, **Last Name**, and **Organization**.
7. Uncheck the check box (A) and click **Next**.

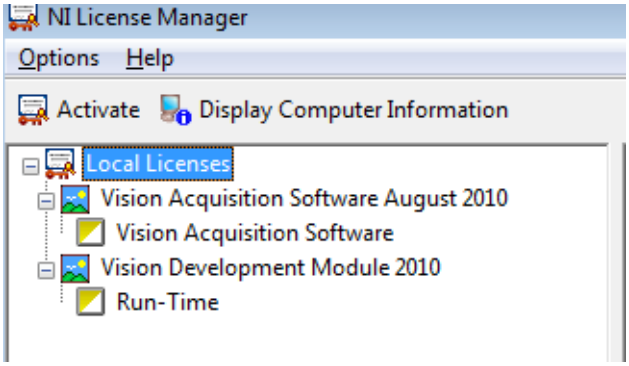


8. Click **Next**. (There's no need to send an email.)
9. Click **Finish**.
10. Right-click **Vision Acquisition Software** and select **Activate**.
11. Click **Finish**.
12. Close the **NI License Manager** window.
13. Continue at 7.1.4 Connecting the goggles ▶ 85.

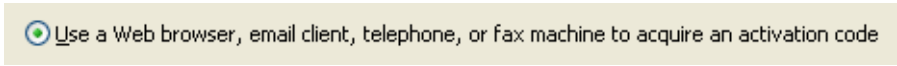
7.1.3.2 No Internet access on ICS computer

1. Navigate to the required directory: From the desktop, click **Start** > **All Programs** > **National Instruments** > **NI License Manager**.
2. Click the plus sign (+) to display the contents for each license: **Vision Acquisition Software 2010** and **Vision Development Module 2010**.

Note · *Solid green checkboxes indicate that the license has already been activated. Continue at* 7.1.4 Connecting the goggles ▶ *85*.



3. Right-click **Run Time** and select **Activate**.
4. Select

   and click **Next**.
5. Select

   and click **Next.**
6. Locate the product serial number on the front of the envelope labeled **NI Vision Development Module Run-Time License** (this is the bottom number, for example, M111xxxx), type in the number, and click **Next**.
7. Visit ni.com/activate from another computer or smart phone.
8. Two activation codes need to be obtained. On the website, under **Product to Activate**, choose **Vision**.

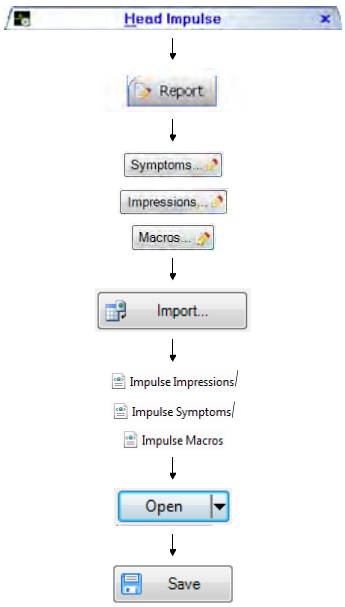
9. In the box below, choose **Vision RunTime**.
10. Under **Product Version**, choose **10.0**.
11. Click **Go**.
12. Enter the product serial number from the envelope.
13. Enter computer ID (located on the ICS Impulse computer).
14. Enter **Name**, **Organization**, and **Email address**.
15. Click **Continue**. Activation code will appear on the website.
16. On the ICS Impulse computer, click **Next**.
17. Enter the first activation code.
18. Visit ni.com/activate from another computer or smart phone.
19. On the website, **Product to Activate**, choose **Vision Acquisition**.
20. Under **Product Version**, choose **August2010**.
21. Click **Go**.
22. Enter the product serial number from the envelope.
23. Enter computer ID (located on the ICS Impulse computer).
24. Enter **Name**, **Organization**, and **Email address**.
25. Click **Continue**. The activation code will appear on the website.
26. On the ICS Impulse computer, under the first activation code, enter the second activation code.
27. Click **Next**.
28. Close the **NI License Manager** window.
29. Click **Finish**.
30. Continue to 7.1.4 Connecting the goggles ▶ 85.

7.1.4 Connecting the goggles

1. Remove the cap from the goggle lens.
2. Connect the goggle cables to the interface box.
3. Connect the interface box cables (USB power cable and Firewire data cable) to the appropriate connectors on the computer.
4. Click (OTOsuiteV icon found on the desktop) to open the application.

7.1.5 Importing sample report lists

These sample report lists can be imported: Symptoms, Impressions, and Macros.

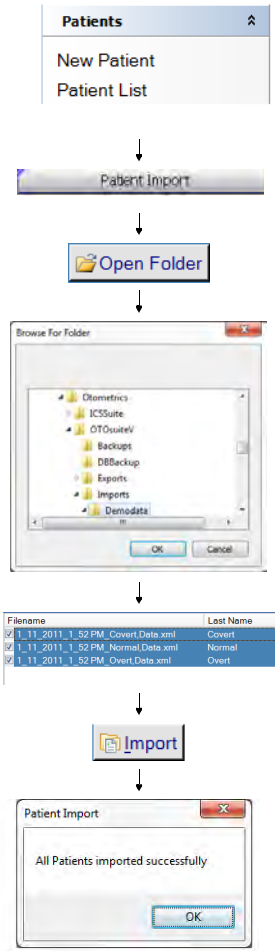


1. Click **Head Impulse** tab.
2. Click **Report**.
3. Click **Symptoms...**, **Impressions...**, or **Macros...**
4. Click **Import**.
5. Click the appropriate file in the correct language to import: **Impulse Symptoms...**, **Impulse Impressions**, or **Impulse Macros...**
6. Click **Open**.
7. Click **Save**.

7.1.6 Importing demo data

The demo data can be imported (patient files -Normal, Overt, Covert) giving you the ability to view patient data and analysis for these 3 findings.

1. If the **Patients** window is not open, click **Patient List**.
2. Click **Patient Import**.
3. Click **Open Folder**. The **Import** folder is highlighted.
4. Below the **Import** folder click the **Demodata** folder and click **OK**.
5. Select all 3 patient files that appear in the **Patient Import** window.
6. Click **Import**.
7. Click **OK**.

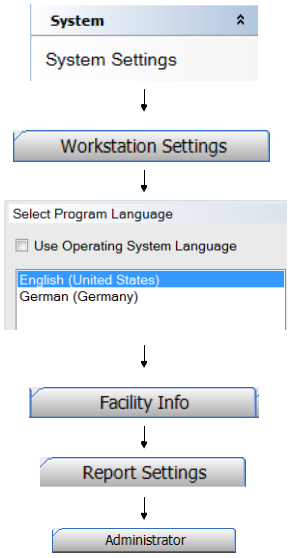


7.1.7 Customizing for your facility

Note · *For the initial login, the OTOsuite Vestibular provides a default user name and password. After one user with administrator privileges has been added, this default user name and password are no longer provided (the fields are blank). Use the new user name and password for subsequent logins.*

Customize the installation at each computer where the software is installed.

1. Click **System Settings**.
2. Click **Workstation Settings** and select the desired program language from the list.
3. Click **Facility Info**.
4. Add facility information. Refer to *6.6 GDT Interface* ▶ 71
5. Click **Report Settings** to modify the settings. Refer to *6.4 Report Settings* ▶ 69.



Note · *IMPORTANT! Set up at least one user in step 6 with administrator privileges.*

6. Click **Administrator** to set up a user. Refer to 6.1 Administrator ▶ 63.

7.1.8 Installing Adobe Reader

For access to the digital version of this manual, Adobe Reader must be installed on the computer. Install the copy provided on the OTOsuite Vestibular DVD if required.

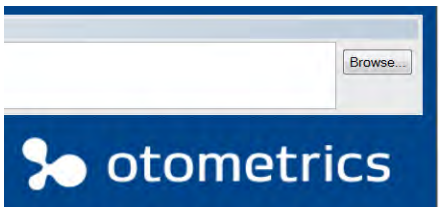
1. Browse the DVD to locate the Adobe Reader file.
2. Double-click Adobe Reader (choose language) to start the installation and follow the on-screen instructions.
3. The installation is complete for standalone installations.
   For all other installations, continue to 7.1.9 Changing the shared network location for videos ▶ 90.

7.1.9 Changing the shared network location for videos

Note · *This procedure is for Client or Both (Client/Server) installation types. Ensure that the permission level for the folder is set for sharing before continuing. (Refer to* *XP systems ▶ 91 or Windows 7 systems ▶ 91* *to set the permission level.)*

The default location for video storage is **\\<server-computer-name>\\OTOvideo**. Users that have **Add User as Administrator** selected in their profile can use this procedure to change where videos are stored.

1. From a workstation on the network, click  to open the application.
2. Click **System Settings** and click **Administrator**.
3. Click **Browse...** to browse to the shared folder.



4. Select the folder and click **OK**.

The network location for the folder displays in the **OTOsuite Vestibular Video File Storage Location** window. All video files recorded at any of the workstations on the network are now stored in this folder.

XP systems

1. Navigate to the required directory.
2. Right-click the Video folder.
3. Click **Properties**. Choose the **Sharing** tab.
4. Click **Share this folder**.
5. Click **Permissions**.
6. To add names to the list of users, click **Add** and type in the user name. Click **Check names**. Click **OK**.
7. To change the permission, click the name to highlight it.
8. Click the **Change** checkbox in the **Allow** column.
9. Repeat steps 6 and 8 until **Change** has been set for all users added to the list.
10. Click **OK**.
11. Click **OK**.

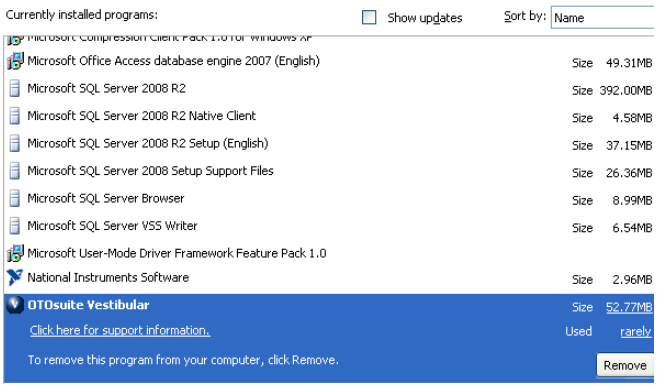
Windows 7 systems

1. Navigate to the required directory.
2. Right-click the video folder.
3. Click **Share with**. Choose **Specific people**.
4. Choose names.
5. Click in the field next to the Add button.
6. Select **Find people** and type in the name.
7. Click **Check Names**. Click **OK**.
8. To change the permission, click the user name to highlight it.
9. Click the drop-down arrow at the right and click **Read/Write**.
10. Repeat steps 8 and 9 until **Read/Write** has been set for all users added to the list.
11. Click **Share**.
12. Click **Done**.

7.2 Uninstalling

7.2.1 Windows XP

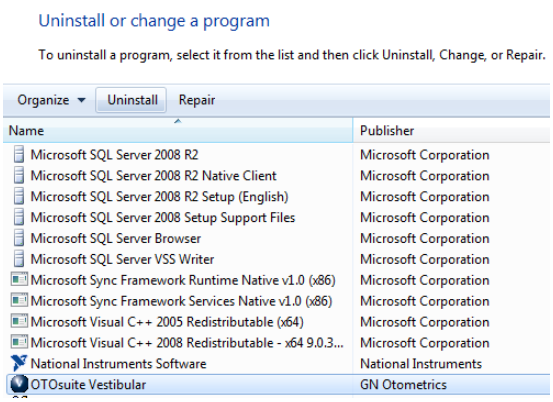
1. Open the **Control Panel**.
2. Click **Add or Remove Programs**.
3. Click **OTOsuite Vestibular**.



4. Click **Remove**.
5. Click **Yes** to the dialog: Are you sure you want to remove OTOsuite Vestibular from your computer?

7.2.2 Windows 7

1. Open the **Control Panel**.
2. Click **Programs**.
3. Click **Uninstall a program** under **Programs and Features**.
4. Click **OTOsuite Vestibular**.



5. Click **Uninstall**.
6. Click **Yes** to the dialog: Are you sure you want to uninstall OTOsuite Vestibular?

Note · *Uninstalling the software will remove OTOsuite Vestibular but not the SQL Server, National Instruments etc. that was part of the first time installation of OTOsuite Vestibular.*

7.3 Repairing

1. Save any files currently open and quit all programs.
2. Insert the DVD used when installing OTOsuite Vestibular.

Note · *If the computer is set up to AutoRun, the installation starts. If AutoRun is not initialized, browse the DVD to find this icon* 

.

3. Click **setup**. (You may need to close the DVD content folder.)
4. If you have Windows 7, a security dialog will appear. Click **Yes**.
5. The Welcome screen appears. Click **Next**.
6. Select **Repair** and click **Next**.
7. Click **Install**.
8. Click **Finish**.

7.4 Upgrading/Reinstalling

When upgrading or reinstalling the OTOsuite Vestibular application, it is not possible to change the type of installation previously chosen. Contact your local representative if you need to make changes to the type of installation.

Note · *If firewall messages appear, please allow access.*

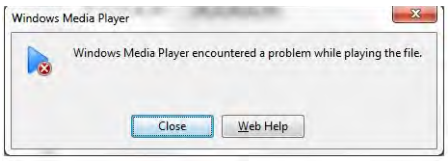
1. Save any files currently open and quit all programs.
2. If **upgrading** the OTOsuite Vestibular application, insert the DVD received with the upgrade of OTOsuite Vestibular application.
   If **reinstalling** the OTOsuite Vestibular application, insert the DVD used when installing OTOsuite Vestibular.

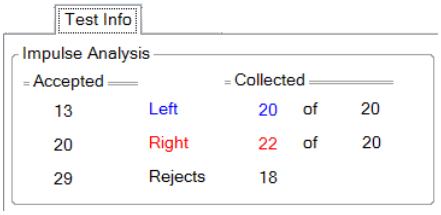
Note · *If the computer is set up to AutoRun, the installation starts. If AutoRun is not initialized, browse the DVD to find this icon* 

.

3. Click **setup**. (You may need to close the DVD content folder.)
4. If you have Windows 7, a security dialog will appear. Click **Yes**.
5. The Welcome screen appears. Click **Next**.
6. Select **I accept the terms in the license** and click **Next**.
7. Continue to click **Next** until the Ready to Install screen appears.
8. Click **Install**.
9. Click **Finish**.

8 Troubleshooting

Problem	Solution
Laser beam dots not seen on the wall or other projection surface	Make sure the light is not obstructed by hair or reflecting off another object in the line of projection. If obstruction is ruled out, Call Technical Support. **Caution** · *Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*
Eye image not displayed	Reconnect firewire and USB to the computer and click **Reconnect Goggles** in **Impulse Options** (part of the **Head Impulse** group).
When using Window 7 operating system, the following error dialog may be seen when trying to play a saved video file directly from Windows Media Player. This is done by either selecting the video file from Windows Media Player or by double-clicking on the video file from Windows Explorer. 	To correct this problem, locate the file **Win7codecs_v281.exe** inside the folder **Tools** on the OTOsuite installation DVD. Double-click the file to start the installation program and follow the prompts.

Problem	Solution
The following error dialog may be seen when trying to play the ICS Impulse Training DVD using Windows Media Player. 	Locate the file **k-lite_codec_pack_700_mega(3).exe** inside the folder **Tools** on the ICS Impulse Training DVD. Double-click the file to start the installation program and follow the prompts.
The **Left**, **Right** and **Rejected** counts are different in **Lateral Impulse Test** as compared to **Left**, **Right** and **Rejects** counts in **2D** and **3D Analysis**  	Counts result from two separate algorithms that in combination assure that only quality data are analyzed. **Collection** window of **Lateral Impulse Test**: The counts are the result of the **Collection algorithm** that assesses the head velocity data and rejects invalid head impulses. **Test Info** window of **2D** and **3D Analysis**: The **Accepted** counts are the result of the **Analysis algorithm** that assesses all the data a second time. This algorithm may reject data accepted by the **Collection algorithm** (displayed in the **Collected** column) if the corresponding eye velocity data is not valid. (For example, when the tester performed an acceptable head impulse but the patient looked away from the fixation dot.)

Problem	Solution
Frame rate too slow for accurate data collection (Error message appears during collection.)	The computer processor is too slow for acquiring the minimum frame rate needed for head impulse testing. • Verify that the computer meets minimum specifications • Close other software programs • Disable wireless Internet • Disable **Show Pupil Crosshair on Image** (located in the **Options** window of the **Head Impulse** group)

9 ICS Impulse System Safety

This User Manual contains information and cautions which must be followed to ensure the safe use of the ICS Impulse System. Local government rules and regulations, if applicable, should also be followed at all times.

When the ICS Impulse System is used in conjunction with a test device, make sure that all information, cautions, and warnings in the User Manual for the test device are followed.

For safety specifics concerning test modules and test devices, see the specific manuals.

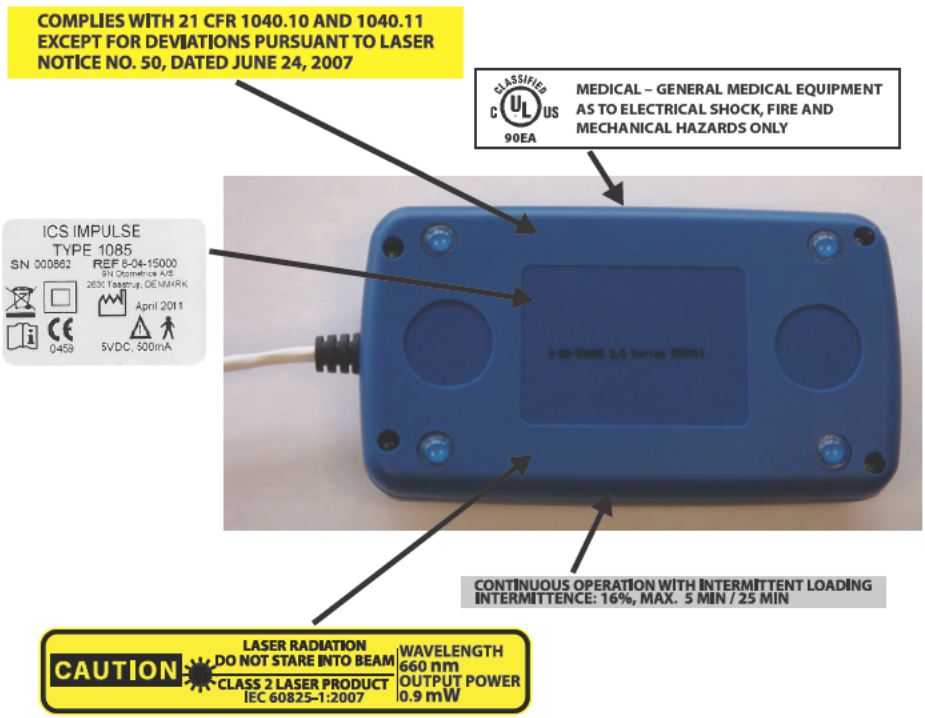
9.1 Symbols used

ICS Impulse System	Meaning
	CAUTION: Laser radiation. Do not stare into beam. Class 2 laser product.
	General warning sign.
	Consult instructions for use.
	Indicates compliance with Type B of the safety standard EN 60601-1.
	MEDICAL - General Medical Equipment as to electrical shock, fire and mechanical hazards only in accordance with UL 60601-1, first edition, 2003 CAN/CSA-22.2 No. 601.1-M90.

Symbols used

ICS Impulse System	Meaning
	The device is marked with this symbol to indicate that it is electronic equipment covered by the Directive 2002/96/EC on waste electrical and electronic equipment (WEEE). In European countries the crossed-out wheeled-bin WEEE symbol reminds you that all the electrical and electronic products, batteries, and accumulators must be taken to separate collection at the end of their working life. This requirement applies in the European Union. Do not dispose of these products as unsorted municipal waste. You can return your device and accessories to Otometrics, or to any Otometrics supplier. You can also contact your local authorities for advice on disposal.
CE xxxx	The system is CE-marked according to the Medical Devices Directive 93/42/EEC.
	The interface box is marked with this symbol to indicate to that it is suitable for direct current only.
	Indicates compliance with Class 2 of the safety standard EN 60601-1.

9.2 Label locations



9.3 Safety notes

GN Otometrics ICS products are not designed to be used in conjunction with any devices not approved by GN Otometrics. Summation of combined unapproved parts could result in increased electrical leakage. All parts of the ICS Impulse System are suitable for use within the patient environment.

Note 1:
There are no user-serviceable parts inside the ICS Impulse System goggles or interface box. For the sake of safety, and in order not to void the warranty, the goggles and interface box should only be serviced by authorized service personnel. In case of defects, please make a detailed description of the defect(s) and contact your supplier. Do not use a defective instrument.

Note 2:
Keep the equipment away from liquids. Do not allow moisture inside the equipment.

Note 3:
Do not use the equipment in the presence of flammable anaesthetics (gases).

Note 4:
A Class 2 Laser product is used for calibration. The laser beam projects from the front of the goggles onto a solid surface.

Caution · *Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*

Note 5:
No parts may be eaten, burnt, or in any way used for purposes other than head impulse testing or video recording of the eye.

Note 6:
The equipment can be disposed of as normal electronic waste according to local regulations.

Note 7:
For safety reasons, accessories connected to the equipment's outlet fittings must be identical to the type supplied with the system.

Note 8:
The device is disconnected from the mains by removing the USB cable.

Note 9:
Do not touch non-medical parts, such as the laptop/computer or printer and the patient at the same time.

Note 10:
Exposure to electromagnetic fields can result in interference with the process of recording correct measurements. ICS Impulse System cameras and gyroscopes are sensitive to electrical disturbances. Avoid static discharges and electromagnetic fields.

Note 11:
No defibrillators or high-frequency surgical equipment should be applied to the patient when connected to the ICS Impulse System at any time.

Note 12:
Immediately discontinue use of the equipment if skin irritation or discomfort occurs.

Note 13:
Installation of any third party software (applications, programs, or utilities) other than those specified by GN Otometrics can compromise the safety or effectiveness of this system.

Note 14: The ICS Impulse System needs to be installed and put into service according to the EMC information provided in this User Manual. Portable and mobile RF communications equipment can affect medical electrical equipment. The ICS Impulse System may be interfered with by other equipment with CISPR emission requirements.

Note 15: The use of accessories and cables other than those specified in the Accessories list of this User Manual may result in increased emissions or decreased immunity of the ICS Impulse System.

Note 16: The ICS Impulse System should not be used adjacent to or stacked with other equipment and that if adjacent or stacked use is necessary, the equipment or system must be observed to verify normal operation in the configuration in which it will be used.

Note 17: The lasers must not be left on longer than 5 minutes. Follow the testing instructions as provided in this User Manual to ensure that the lasers are on a minimum amount of time. In conditions where the ambient temperature approaches the maximum recommended operating temperature, 29° C (84.2° F), failure to follow the testing instructions, or to inadvertently leave the lasers on for an extended amount of time, may cause the goggles to overheat. Allow at least 25 minutes for the temperature of the goggles to return to normal if the lasers have been left on for 5 minutes.

9.4 Manufacturer

GN Otometrics A/S
9 Hoerskaetten
DK-2630 Taastrup Denmark
T: +45 45 75 55 55, **F**: +45 45 75 55 59
www.otometrics.com

9.4.1 Responsibility of the Manufacturer

The manufacturer is to be considered responsible for effects on safety, reliability, and performance of the equipment ONLY IF:

- All assembly operations, extensions, re-adjustments, modifications, or repairs are carried out by the equipment manufacturer or personnel authorized by the manufacturer.
- The electrical installation to which the equipment is connected complies with EN/IEC requirements.
- The equipment is used in accordance with the instructions for use.

The manufacturer reserves the right to disclaim all responsibility for the operating safety, reliability, and performance of equipment serviced or repaired by other parties.

10 Technical Specifications

10.1 ICS Impulse System

Interface	USB 2.0 to PC 1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable
Type Identification	ICS Impulse System is Type 1085 from GN Otometrics A/S

Power Supply
Device is powered through USB - 5 V DC, 500 mA

Performance Characteristics	
Inputs	Monocular (Right eye only)
Sampling Rate	250 Hz for Head Impulse Test Option of 30, 60 or 120 Hz for Video Recording
Eye Tracking	100 pixels x 100 pixels.
Software	Windows Graphical User Interface; High Performance Analysis Software; Database Storage of Test Data; Sophisticated Patient and Test Data Management

Laser specifications	
Wavelength	Maximum 660 nm
Output power	Maximum 0.9 mW

Operating Mode	
Warm-up time	<1 min
Mode of operation	Continuous operation with intermittent loading Laser intermittence: 16%, Max. 5 min ON/ 25 min OFF Do not use the equipment in the presence of flammable anaesthetics (gases).

Operating Environment	
Temperature	+15° C to +29° C (59° F to +84.2° F)
Rel. Humidity	30 to 90%, non-condensing
Air Pressure	600 hPa to 1060 hPa
Operations at temperatures below -20° C (-4° F) or above +60° C (140° F) may cause permanent damage to the device.	

Storing and Handling	
Temperature	-20° C to +60° C (-4° F to +140° F)
Rel. Humidity	<90%, non-condensing
Air Pressure	500 hPa to 1060 hPa

Dimensions		
Goggles	Length	7.25 in (18.4 cm)
	Width	0.5 in (1.3 cm) to 1.75 in (4.4 cm)
	Height	1.75 in (4.4 cm)
Interface box	Length	5 in (12.7 cm)
	Width	2.75 in (7 cm)
	Height	1 in (2.5 cm)

Weight	
Goggles	2.1 oz (60 g)
Interface box	4.6 oz (130 g)

Calibration
Calibration of the system is not required

Classification
Class II Type B

Standards	
Safety	Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA-22.2 No. 601.1-M90, IEC60601-1, 2.ed.: 1988 + A1 + A2, IEC 62471, 1st.ed., IEC 60825-1, 2.ed.
EMC	IEC 60601-1-2: 2007

10.2 Accessories

Accessories		
Manuals/Videos	ICS Impulse Quick Guide	7-50-11300-EN 7-50-11300-DE 7-50-11300-ES 7-50-11300-FR 7-50-11300-IT
	ICS Impulse Training video	8-49-82700-US 8-49-82700-DE
Software	OTOsuite Vestibular	8-49-90300
Goggles	Face cushion 20/pkg	8-35-34400
	Strap assembly	8-35-34200
	Optical cleaning cloth ***Qty min 3***	7590527
	Fixation dot (2 sheets/pkg)	1-26-44000
Cables	USB 2.0 cable	8-71-79200
	1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable	8-71-89600
	Cable clip	8-35-36900
Case/Mount	Carrying case	8-35-36700
	Wall mount	8-62-45600

10.3 Guidance and manufacturer's declaration tables

Electromagnetic Emissions

The ICS Impulse System is intended for use in the electromagnetic environment specified below. The customer or user of the ICS Impulse System should ensure that it is used in such an environment.

Emissions test	**Compliance**	**Electromagnetic environment - Guidance**
Radio Frequency (RF) Emissions CISPR 11	Group 1	The ICS Impulse System uses RF energy only for its internal function. Therefore, its RF emissions are very low and are not likely to cause any interference in nearby electronic equipment.
RF Emissions CISPR 11	Class A	The ICS Impulse System is suitable for use in all establishments other than domestic and those directly connected to the public low-voltage power supply network that supplies buildings used for domestic purposes
Harmonic emissions IEC 61000-3-2	Class A	
Voltage fluctuations/ flicker emissions IEC 61000-3-3	Complies	

Immunity - All Equipment and Systems

The ICS Impulse System was not tested for immunity to electromagnetic disturbances.

Guidance and manufacturer's declaration tables

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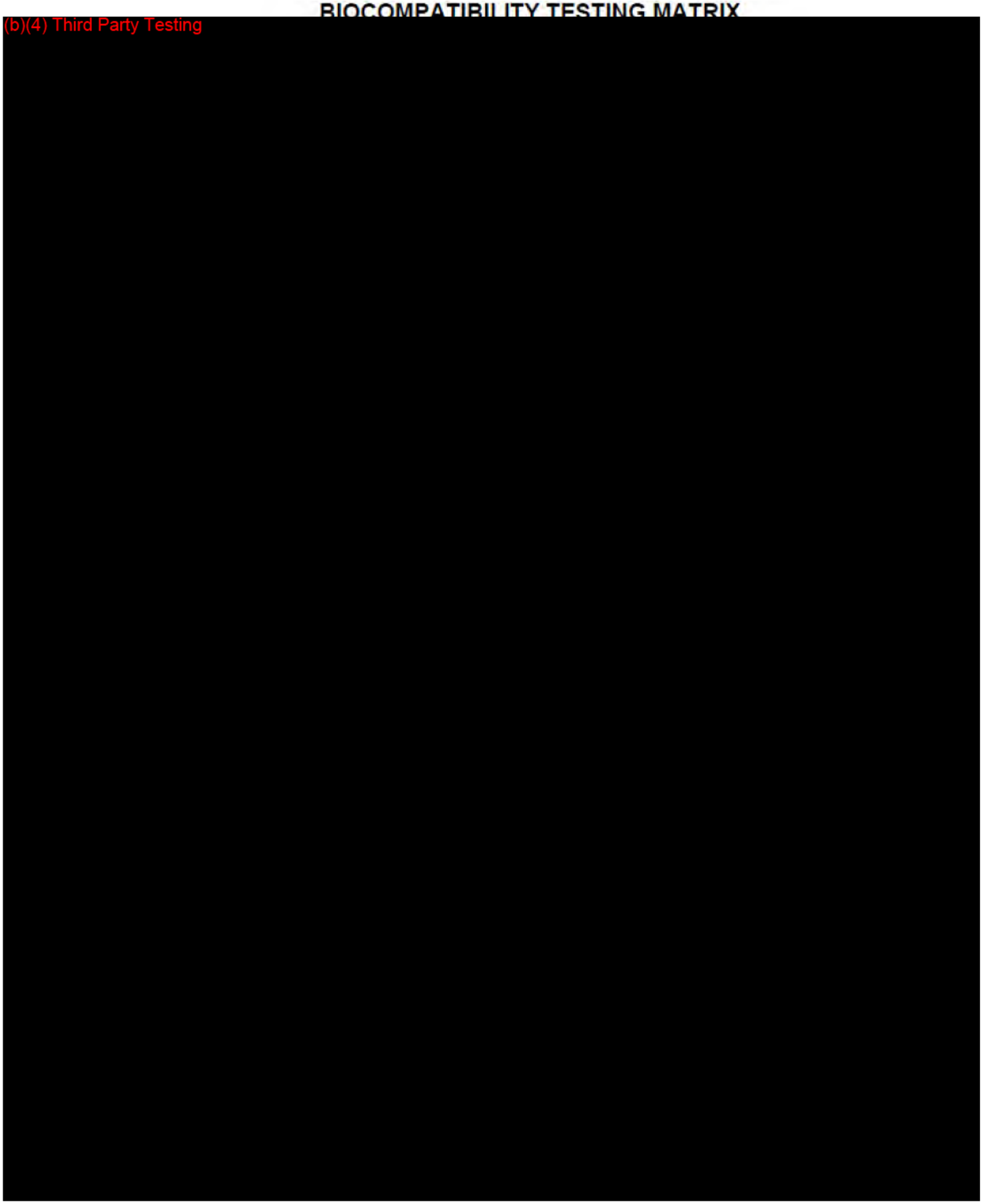
Exhibit 14. Sterilization and packaging
(Not Applicable)

Exhibit 15. Biocompatibility

BIOCOMPATIBILITY TESTING MATRIX

(b)(4) Third Party Testing



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Biocompatibility Test Protocol

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Exhibit 16. Software

Level of Concern: Minor.
Software Description
Device Hazard Analysis
Software Requirements Specification (SRS)
Architecture Design Chart (Not required for Minor Level of Concern)
Software Design Specification (SDS) (Not required for Minor Level of Concern)
Traceability Analysis
Software Development Environment Description (Not required for Minor Level of Concern)
Verification and Validation Documentation
Revision Level History
Unresolved Anomalies (Bugs or Defects) (Not required for Minor Level of Concern)

Level of Concern: Minor
We believe the level of concern is Minor if failures or latent design flaws are unlikely to cause any injury to the patient or operator.

FDA Checklists for Level of Concern:
Table 1, Major
1. Does the Software Device qualify as Blood Establishment Computer Software? NO
2. Is the Software Device intended to be used in combination with a drug or biologic? NO
3. Is the Software Device an accessory to a medical device that has a Major Level of Concern? NO
4. Prior to mitigation of hazards, could a failure of the Software Device result in death or serious injury, either to a patient or to a user of the device? Examples of this include the following:
a. Does the Software Device control a life supporting or life sustaining function? NO
b. Does the Software Device control the delivery of potentially harmful energy that could result in death or serious injury, such as radiation treatment systems, defibrillators, and ablation generators? NO
c. Does the Software Device control the delivery of treatment or therapy such that an error or malfunction could result in death or serious injury? NO
d. Does the Software Device provide diagnostic information that directly drives a decision regarding treatment or therapy, such that if misapplied it could result in serious injury or death? NO
e. Does the Software Device provide vital signs monitoring and alarms for potentially life threatening situations in which medical intervention is necessary? NO
Table 2, Moderate
1. Is the Software Device an accessory to a medical device that has a Moderate Level of Concern? NO
2. Prior to mitigation of hazards, could a failure of the Software Device result in Minor Injury, either to a patient or to a user of the device? NO
3. Could a malfunction of, or a latent design flaw in, the Software Device lead to an erroneous diagnosis or a delay in delivery of appropriate medical care that would likely lead to Minor Injury? NO

If the answers to all of the questions in Tables 1 and 2 above are No, the Level of Concern is Minor.

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Type 1085, ICS Impulse System Specification

Table of Contents

1 Introduction

1.1 Purpose

This is the System Specification for Release 1.0 the ICS Impulse product. The purpose of this document is to define the hardware, software, and system specifications for the initial release of the new 1085 ICS Impulse product.

1.2 Scope

The ICS Impulse product is part of the new Otometrics OTOsuite Vestibular. Future extensions to the OTOsuite Vestibular are expected to include the other ICS products (VNG, ENG, VEMP, EP, ASSR).

1.3 Definitions, Acronyms, Abbreviations

Term	Description
VOR	Vestibular Ocular Reflex
VNG	Videonystagmography
ENG	Electronystagmography
HIT/HHT	Head Impulse Test (aka Hamalgyi Head Thrust)
VAT	Vestibular Autorotation
DVA	Dynamic Visual Acuity
XML	Extensible Markup Language
EMR	Electronic Medical Record
GDT	Gerätedaten-Träger, which translates to “Device Data Carrier”. This an interface specification for data transfer between a PMS and another diagnostic system (in this case ICS Impulse).
PMS	Practice management system. Used in the GDT interface. This is the application which the practitioner uses to manage their patient data.
EDP	Electronic data processing. Used in the GDT interface. This term is often interchanged with PMS.
Data set type	Used in the GDT interface. This refers to a specific set of data fields to be transferred via file between the PMS and another diagnostic system (in this case ICS Impulse).

1.4 Design Input References

0-70-03000 Type 1085, Design Requirements
7-38-06501, Risk Management Plan
7-38-06502. Medical Device Questionnaire

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PART 1 - DATA AQUISITION

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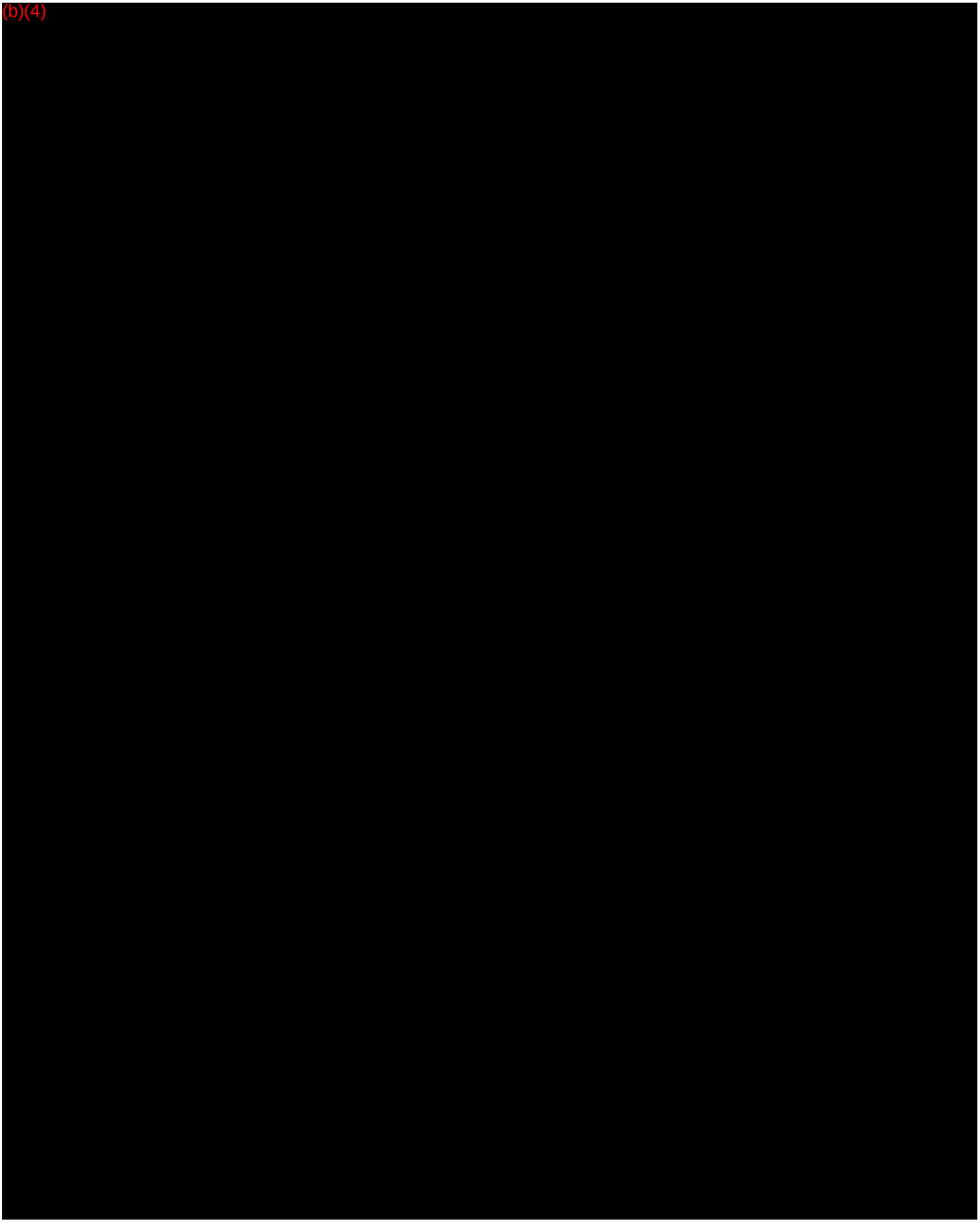

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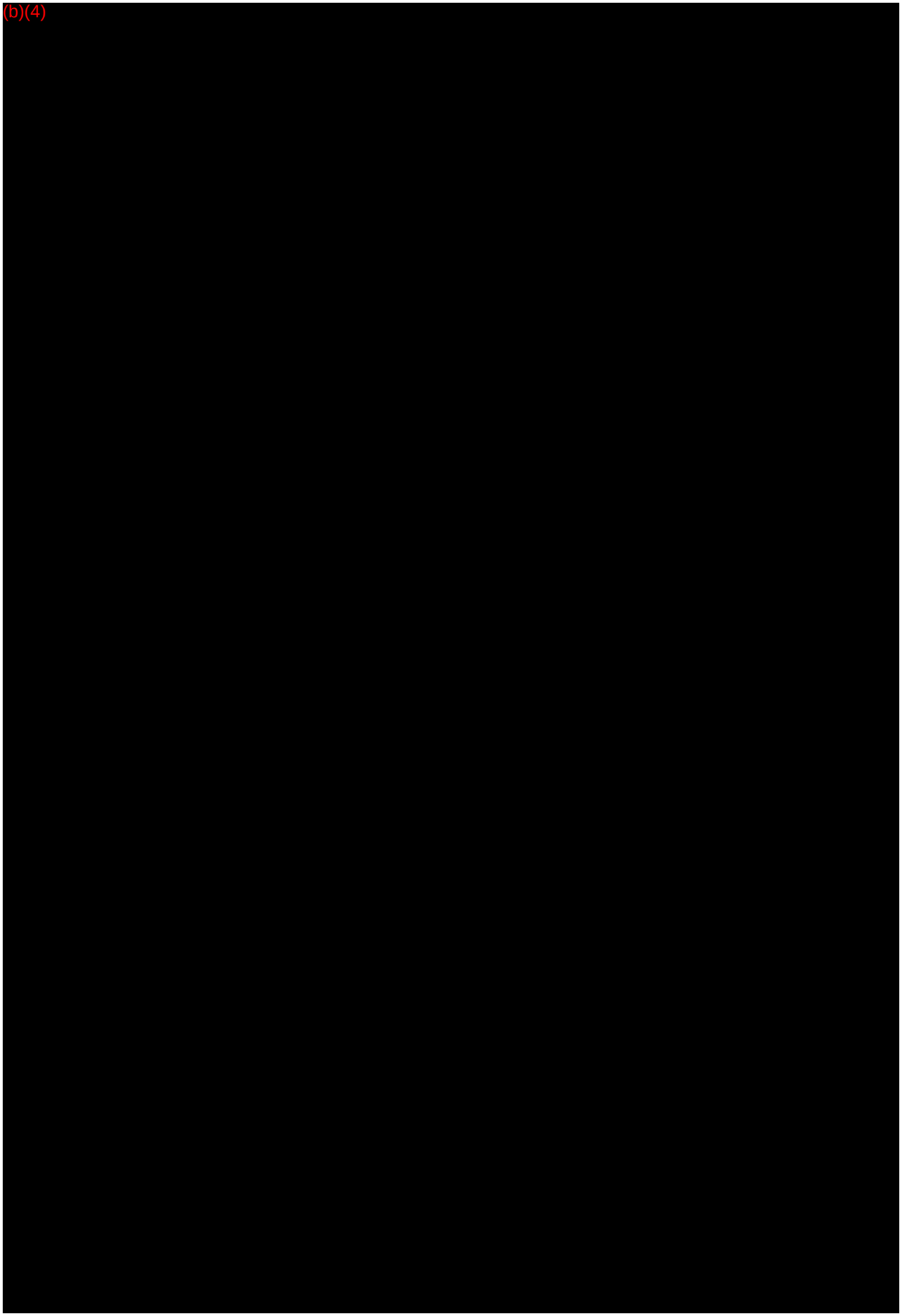

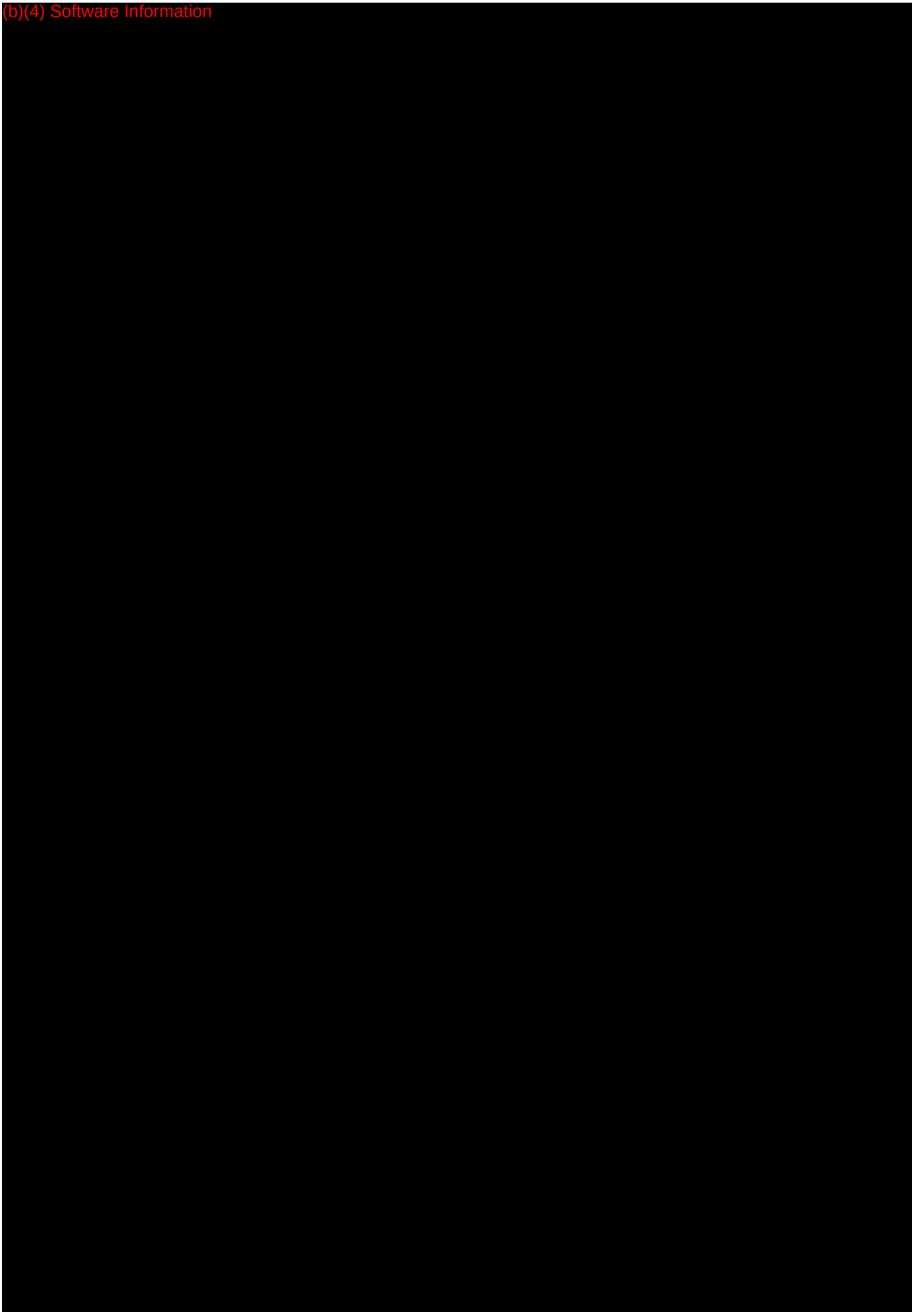
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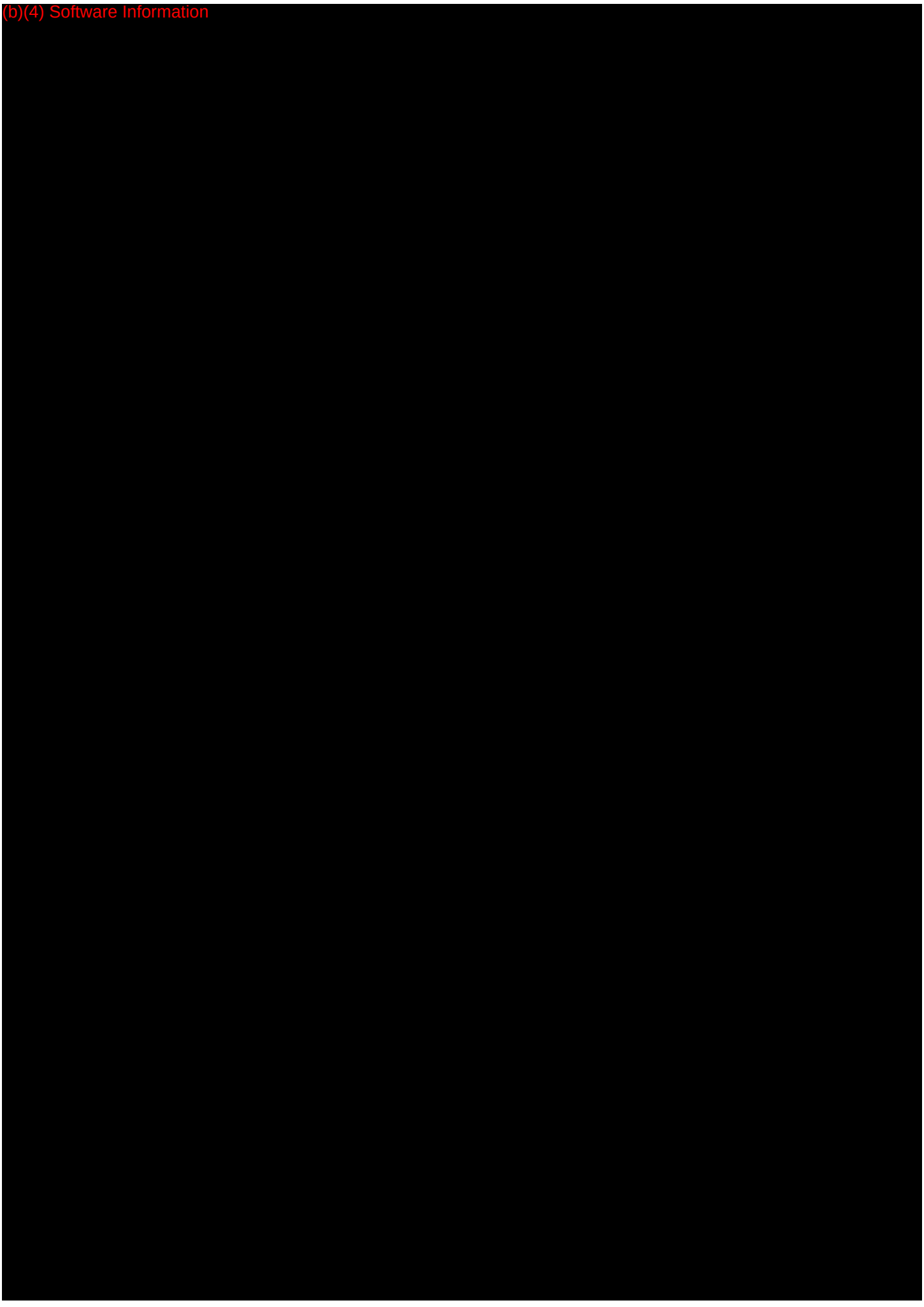

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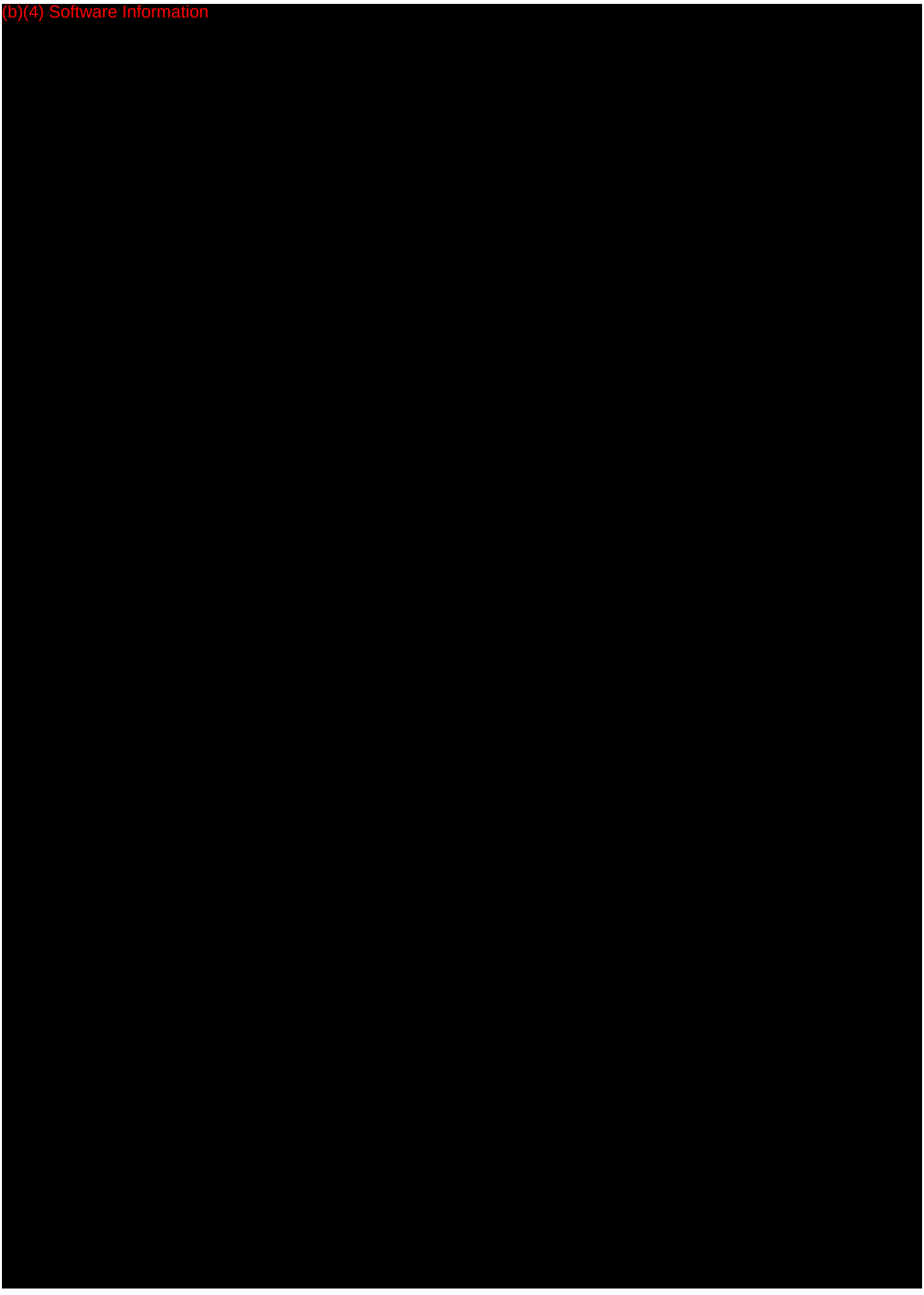

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Document Title:	(b)(4), Risk Management Plan	
Document No.:	(b)(4) 1	Revision: (b)
Owner:	Regulatory Affairs	Page 1 of 5

(b)(4) Risk Management Plan

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1.3 Reference Documents

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1.4 Acronyms and Abbreviations

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2 Risk Management

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Table 1 – Risk Management Activities

No.	Risk Management Activity	Owner	Minimum Approval	Phase Review Requirements
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2.3 Criteria for Risk Acceptability

Risk Analysis Approach

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Document Title:	(b)(4) Software Design for Reports	
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Owner:	R&D Software	Page 1 of 27

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Exhibit 17. Electromagnetic Compatibility and Electrical Safety
In order to receive UL listing, these requirements must be met:
The basic standard used to investigate products in this category is UL 60601-1, "Medical Electrical Equipment, Part 1: General Requirements for Safety," in addition to the applicable Particular and/or Collateral Standards. (Including the EMC standard)

UL Listing for ICS Impulse

ONLINE CERTIFICATIONS DIRECTORY

PIDF.E194318

Medical Equipment

GN OTOMETRICS A/S E194318
HORSKAETTEN 9
2630 TAASTRUP, DENMARK

Audio diagnostic testing systems, Model(s) Type Capella
Audiometers, Model(s) 1066, DA65, Type 1004, Type 1081
Hearing aid programmers, Model(s) NOAH link
ICS-Impulse, Model(s) type 1085
Middle ear analysers, Model(s) 1012
Otoacoustic Emissions and Automated ABR screener, "Serie Accuscreen TE, DP, TE/DP, ABR, ABR/TE, ABR/DP, ABR/TE/DP Lite TE, Lite DP", Model(s) type 1077
PC audiometers, Model(s) Siemens SD100
PC probe mics, Model(s) Siemens SD110
Real ear measurement equipment, Model(s) 1053
Video Otoscope, Model(s) Type 1076

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Enclosure

National Differences

Australia
Austria*
Belgium*
Canada
Czech Republic*
Denmark
Finland*
France*
Germany*
Greece*
Hungary*
Ireland*
Israel
Italy*
Korea
Netherlands*
Norway*
Poland*
Portugal*
Serbia and Montenegro*
Singapore
Slovakia*
Slovenia*
Sweden*
Switzerland*
Turkey*
USA
Ukraine*
United Kingdom*

* No National Differences Declared
** Only Group Differences

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Enclosures

Type	Supplement Id	Description
Marking Plate		
Collateral		
Particular	12-01	Laser Certificate
Particular	12-02	IR-diodes, CB-report
Photographs	3-01	Overall view
Photographs	3-02	Interface box, Internal view
Photographs	3-03	Goggle, rear side
Photographs	3-04	Goggle, Laser out
Photographs	3-05	Goggle, PWB's
Diagrams		
Schematics + PWB		
Manuals	6-01	User Manual - extract
Miscellaneous	7-01	Labels
Miscellaneous	7-02	Marking on packaging
Licenses		

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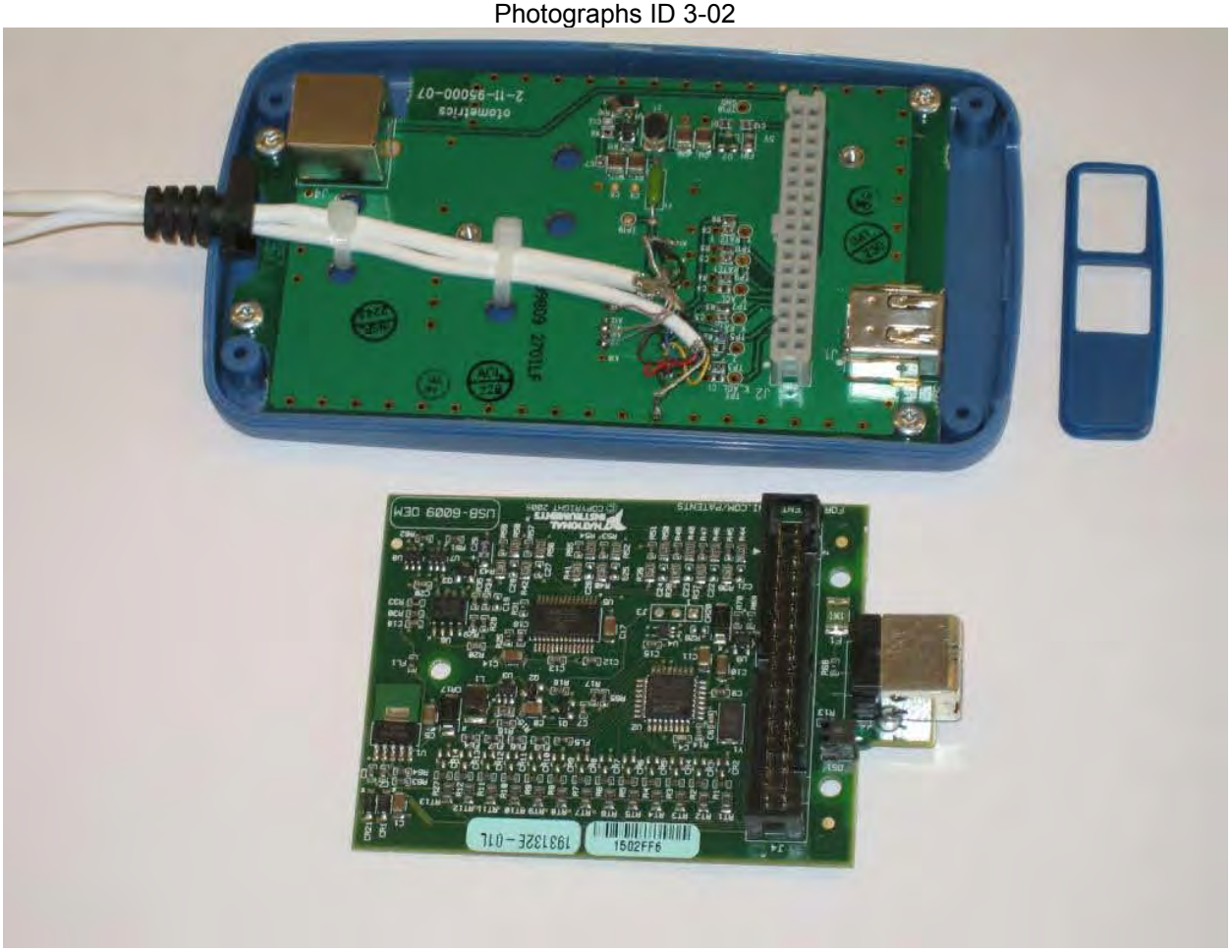
Photographs ID 3-01

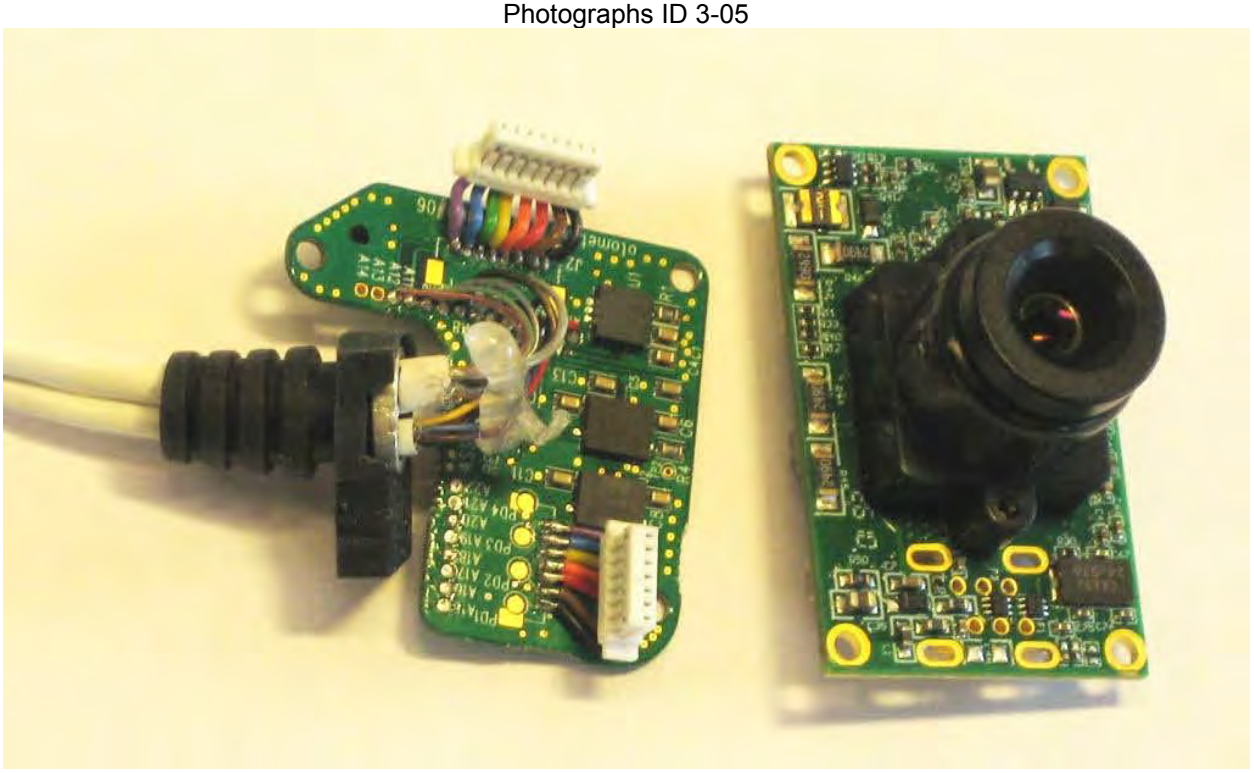
Photographs ID 3-02



Photographs ID 3-03

Photographs ID 3-04

Photographs ID 3-05

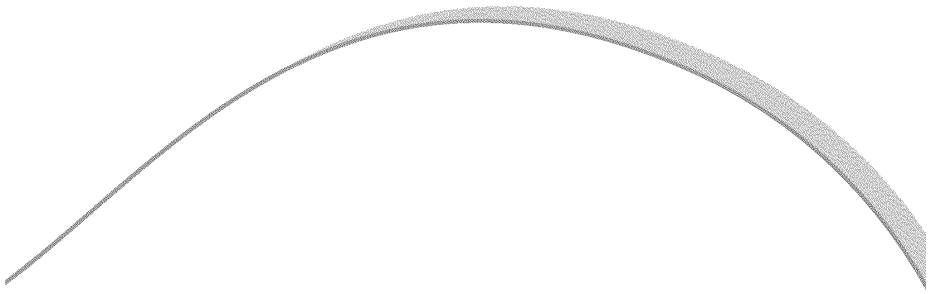


Manuals ID 6-01

ICS Impulse

ICS Impulse

User Manual



CE 0459

Doc No. 7-50-1110-EN/00

otometrics
MADSEN · AURICAL · ICS

Manuals ID 6-01

Version release date
21. May 2011

Technical support
Please contact your supplier.

Manuals ID 6-01

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Manuals ID 6-01

1 Introduction

Congratulations! You are now the owner of a sophisticated new ICS Impulse system developed in collaboration with Drs. Ian Curthoys, Michael Halmagyi and others at University of Sydney.

To assist you in getting the most out of the ICS Impulse system, we have included this user manual and a training video. We hope you find it easy to use and that your use of the incorporated tips and information results in improved data collection accuracy as it relates to your assessment of vestibular-related disorders, test results reporting, and patient information retrieval.

1.1 Intended Use

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.

Note · *The ICS Impulse System is intended to be used only by qualified medical personnel.*

1.2 Intended User

This manual describes the use of the device in combination with the software. Readers are assumed to have prior knowledge of the medical and scientific facts underlying the procedure. For this reason, the examination methods are mentioned only to the degree that is necessary for a correct, safe application of the ICS Impulse System.

You can find more information in the ICS Impulse training video or at **www.headimpulse.com**.

1.3 About this manual

This is your guide to using the basic functions required for navigating in OTOsuite Vestibular and the various OTOsuite Vestibular modules. This includes key features such as printing test results, handling patient and user administration, and data and test device management.

Training
It is recommended that you make yourself familiar with the features provided by OTOsuite Vestibular and the test device before testing a patient.

1.3.1 Safety

This manual contains information and cautions which must be followed to ensure the safe performance of the ICS Impulse System.

Caution · *Local government rules and regulations, if applicable, should be followed at all times.*

Safety information is stated where it is relevant, and general safety aspects are described in Chapter 9 ICS Impulse System Safety ▶ 99.

1.4 Typographical conventions

The use of WARNING, CAUTION and NOTE
For safety reasons and appropriate use of the ICS Impulse System, the manual contains **WARNINGS**, **CAUTIONS** and **NOTES** which you should read carefully. The use of these headings is denoted as follows:

Warning · *Indicates that there is a risk of danger to persons, test device and data.*

Caution · *Indicates that there is a risk of damage to the device and/or data.*

Note · *Indicates that you should take special notice.*

1.4.1 Navigating this manual

Window tabs, icons and functions to select are shown in bold type, as for instance in:

- Click **Save**

3.1 Patient preparation

Warning · *A head impulse should not be performed on patients with a neck injury, or on patients who have been told by their physicians to limit or avoid neck movement activity.*

Prior to testing, provide the patient with these general recommendations:

- If in doubt, consult with a physician about the possible side effects of stopping a particular medication.
- Stop tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test.
- Continue medications that are vital, such as insulin, heart medications, seizure medications, and possibly antidepressants.
- No alcohol for 48 hours before testing.
- Do not wear make-up around the eyes.
- Wear comfortable clothing.

3.2 Goggle preparation

3.2.1 Cleaning and maintenance

The ICS Impulse System equipment does not require preventive maintenance. Observe the following recommended guidelines regarding cleaning and maintenance.

- Keep the instrument clean and as free of dust as possible. Remove dust using a soft cloth or brush.
- If required, clean the goggle housing and interface box using a damp cloth moistened with a mild detergent and water solution. Do not allow any moisture to get inside the goggles.

Caution · *Never spray or immerse the goggle components with the cleaning solutions. This could contaminate the electronics and/or optics.*

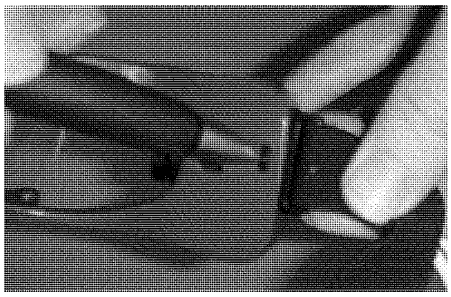
- If required, clean the mirror using the supplied cleaning cloth. The presence of fingerprints on the mirror surfaces could cause inaccurate pupil detection.

Caution · *Improper cleaning may scratch the mirror surfaces.*

- Replace the strap as required. Refer to 3.2.2 Replacing the strap ▶ 21.

3.2.2 Replacing the strap

1. Remove the face cushion.
2. Use a pen to push the plastic clip down and pull out the strap clip attached to the goggle.



3. Repeat on the other side.
4. Remove the cables from both clips on the strap.

5. Obtain a new strap assembly
6. Clip the strap clips into each side of the goggle.
7. Attach the cables to both clips on the strap inserting the small one first.

3.2.3 Replacing the face cushion

Note · *The single-use, disposable face cushion should be replaced for each new patient.*

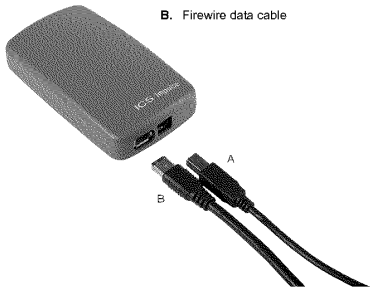
1. To remove the face cushion, slightly flex the goggles out at the side opposite of the camera side and snap out the face cushion. Release the face cushion from the other side.
2. Properly dispose of the used face cushion.
3. Obtain a new face cushion.
4. Align the tab of the face cushion with the hole on the camera side of the goggles.
5. Ensure the face cushion is inside the nose piece.
6. Slightly flex the goggles at the opposite side, align the tab of the face cushion with the hole on this side of the goggles.
7. Double check both sides are fully inserted by pressing in at each side.

3.3 Test setup

The environment where the patient is tested can vary but must allow you to position the patient at least one meter from the wall (or other solid surface that can be used as a projection surface.).

1. Choose a wall that allows you to position the patient at least one meter in front of the fixation dot.
2. Apply one of the fixation dots supplied with the system to the wall in a location that allows you to position the patient directly in front of the fixation dot.

7.1.4 Connecting the goggles

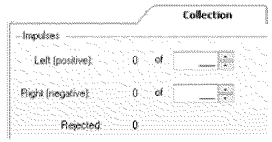
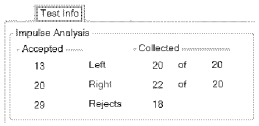


A. USB power cable
B. Firewire data cable

1. Remove the cap from the goggle lens.
2. Connect the goggle cables to the interface box.
3. Connect the interface box cables (USB power cable and Firewire data cable) to the appropriate connectors on the computer.
4. Click (OTOsuiteV icon found on the desktop) to open the application.

8 Troubleshooting

Problem	Solution
Laser beam dots not seen on the wall or other projection surface	Make sure the light is not obstructed by hair or reflecting off another object in the line of projection. If obstruction is ruled out, Call Technical Support. **Caution** · *Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*
Eye image not displayed	Reconnect firewire and USB to the computer and click **Reconnect Goggles** in **Impulse Options** (part of the **Head Impulse** group).
When using Window 7 operating system, the following error dialog may be seen when trying to play a saved video file directly from Windows Media Player. This is done by either selecting the video file from Windows Media Player or by double-clicking on the video file from Windows Explorer. 	To correct this problem, locate the file **Win7codecs_v281.exe** inside the folder **Tools** on the OTOsuite installation DVD. Double-click the file to start the installation program and follow the prompts.

Problem	Solution
The following error dialog may be seen when trying to play the ICS Impulse Training DVD using Windows Media Player. 	Locate the file **k-lite_codec_pack_700_mega(3).exe** inside the folder **Tools** on the ICS Impulse Training DVD. Double-click the file to start the installation program and follow the prompts.
The **Left**, **Right** and **Rejected** counts are different in **Lateral Impulse Test** as compared to **Left**, **Right** and **Rejects** counts in **2D** and **3D Analysis**  	Counts result from two separate algorithms that in combination assure that only quality data are analyzed. **Collection** window of **Lateral Impulse Test**: The counts are the result of the **Collection algorithm** that assesses the head velocity data and rejects invalid head impulses. **Test Info** window of **2D** and **3D Analysis**: The **Accepted** counts are the result of the **Analysis algorithm** that assesses all the data a second time. This algorithm may reject data accepted by the **Collection algorithm** (displayed in the **Collected** column) if the corresponding eye velocity data is not valid. (For example, when the tester performed an acceptable head impulse but the patient looked away from the fixation dot.)

Problem	Solution
Frame rate too slow for accurate data collection (Error message appears during collection.)	The computer processor is too slow for acquiring the minimum frame rate needed for head impulse testing. • Verify that the computer meets minimum specifications • Close other software programs • Disable wireless Internet • Disable **Show Pupil Crosshair on Image** (located in the **Options** window of the **Head Impulse** group)

9 ICS Impulse System Safety

This User Manual contains information and cautions which must be followed to ensure the safe use of the ICS Impulse System. Local government rules and regulations, if applicable, should also be followed at all times.

When the ICS Impulse System is used in conjunction with a test device, make sure that all information, cautions, and warnings in the User Manual for the test device are followed.

For safety specifics concerning test modules and test devices, see the specific manuals.

9.1 Symbols used

ICS Impulse System	Meaning
	CAUTION: Laser radiation. Do not stare into beam. Class 2 laser product.
	General warning sign.
	Consult instructions for use.
	Indicates compliance with Type B of the safety standard EN 60601-1.
	MEDICAL - General Medical Equipment as to electrical shock, fire and mechanical hazards only in accordance with UL 60601-1, first edition, 2003 CAN/CSA-22.2 No. 601.1-M90.

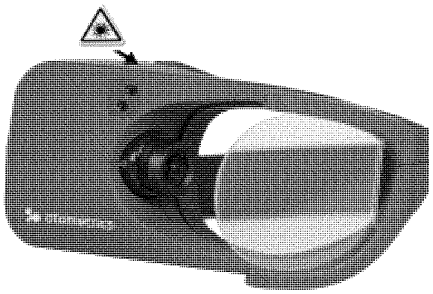
Manuals ID 6-01

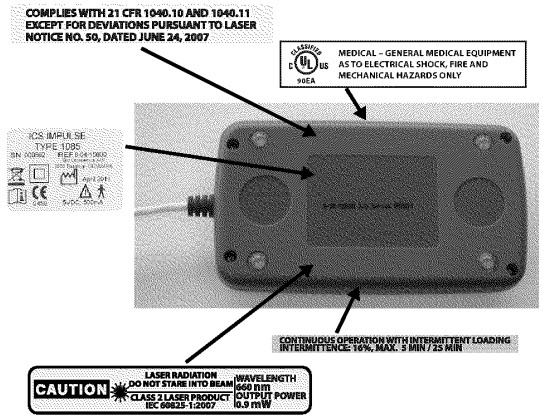
ICS Impulse System Safety

Symbols used

ICS Impulse System	Meaning
	The device is marked with this symbol to indicate that it is electronic equipment covered by the Directive 2002/96/EC on waste electrical and electronic equipment (WEEE). In European countries the crossed-out wheeled-bin WEEE symbol reminds you that all the electrical and electronic products, batteries, and accumulators must be taken to separate collection at the end of their working life. This requirement applies in the European Union. Do not dispose of these products as unsorted municipal waste. You can return your device and accessories to Otometrics, or to any Otometrics supplier. You can also contact your local authorities for advice on disposal.
CE XXXX	The system is CE-marked according to the Medical Devices Directive 93/42/EEC.
	The interface box is marked with this symbol to indicate to that it is suitable for direct current only.
	Indicates compliance with Class 2 of the safety standard EN 60601-1.

9.2 Label locations





9.3 Safety notes

GN Otometrics ICS products are not designed to be used in conjunction with any devices not approved by GN Otometrics. Summation of combined unapproved parts could result in increased electrical leakage. All parts of the ICS Impulse System are suitable for use within the patient environment.

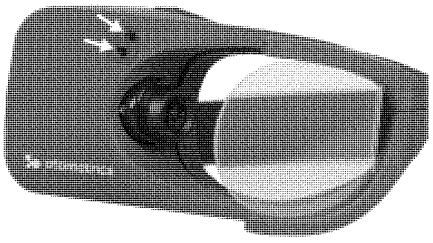
Note 1:
There are no user-serviceable parts inside the ICS Impulse System goggles or interface box. For the sake of safety, and in order not to void the warranty, the goggles and interface box should only be serviced by authorized service personnel. In case of defects, please make a detailed description of the defect(s) and contact your supplier. Do not use a defective instrument.

Note 2:
Keep the equipment away from liquids. Do not allow moisture inside the equipment.

Note 3:
Do not use the equipment in the presence of flammable anaesthetics (gases).

Note 4:
A Class 2 Laser product is used for calibration. The laser beam projects from the front of the goggles onto a solid surface.

Caution · *Do not look directly at the lasers. Use of controls or adjustments, or performance of procedures other than those specified herein, may result in hazardous radiation exposure.*



Note 5:
No parts may be eaten, burnt, or in any way used for purposes other than head impulse testing or video recording of the eye.

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Note 6:
The equipment can be disposed of as normal electronic waste according to local regulations.

Note 7:
For safety reasons, accessories connected to the equipment's outlet fittings must be identical to the type supplied with the system.

Note 8:
The device is disconnected from the mains by removing the USB cable.

Note 9:
Do not touch non-medical parts, such as the laptop/computer or printer and the patient at the same time.

Note 10:
Exposure to electromagnetic fields can result in interference with the process of recording correct measurements. ICS Impulse System cameras and gyroscopes are sensitive to electrical disturbances. Avoid static discharges and electromagnetic fields.

Note 11:
No defibrillators or high-frequency surgical equipment should be applied to the patient when connected to the ICS Impulse System at any time.

Note 12:
Immediately discontinue use of the equipment if skin irritation or discomfort occurs.

Note 13:
Installation of any third party software (applications, programs, or utilities) other than those specified by GN Otometrics can compromise the safety or effectiveness of this system.

Note 14: The ICS Impulse System needs to be installed and put into service according to the EMC information provided in this User Manual. Portable and mobile RF communications equipment can affect medical electrical equipment. The ICS Impulse System may be interfered with by other equipment with CISPR emission requirements.

Note 15: The use of accessories and cables other than those specified in the Accessories list of this User Manual may result in increased emissions or decreased immunity of the ICS Impulse System.

Note 16: The ICS Impulse System should not be used adjacent to or stacked with other equipment and that if adjacent or stacked use is necessary, the equipment or system must be observed to verify normal operation in the configuration in which it will be used.

Note 17: The lasers must not be left on longer than 5 minutes. Follow the testing instructions as provided in this User Manual to ensure that the lasers are on a minimum amount of time. In conditions where the ambient temperature approaches the maximum recommended operating temperature, 29° C (84.2° F), failure to follow the testing instructions, or to inadvertently leave the lasers on for an extended amount of time, may cause the goggles to overheat. Allow at least 25 minutes for the temperature of the goggles to return to normal if the lasers have been left on for 5 minutes.

9.4 Manufacturer

GN Otometrics A/S
2 Dybendalsvaenget
DK-2630 Taastrup Denmark
T: +45 45 75 55 55, **F:** +45 45 75 55 59
www.otometrics.com

9.4.1 Responsibility of the Manufacturer

The manufacturer is to be considered responsible for effects on safety, reliability, and performance of the equipment ONLY IF:

- All assembly operations, extensions, re-adjustments, modifications, or repairs are carried out by the equipment manufacturer or personnel authorized by the manufacturer.
- The electrical installation to which the equipment is connected complies with EN/IEC requirements.
- The equipment is used in accordance with the instructions for use.

The manufacturer reserves the right to disclaim all responsibility for the operating safety, reliability, and performance of equipment serviced or repaired by other parties.

10 Technical Specifications

10.1 ICS Impulse System

Interface	USB 2.0 to PC 1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable
Type Identification	ICS Impulse System is Type 1085 from GN Otometrics A/S

Power Supply
Device is powered through USB - 5 V DC, 500 mA

Performance Characteristics	
Inputs	Monocular (Right eye only)
Sampling Rate	250 Hz for Head Impulse Test Option of 30, 60 or 120 Hz for Video Recording
Eye Tracking	100 pixels x 100 pixels.
Software	Windows Graphical User Interface; High Performance Analysis Software; Database Storage of Test Data; Sophisticated Patient and Test Data Management

Laser specifications	
Wavelength	Maximum 660 nm
Output power	Maximum 0.9 mW

Operating Mode	
Warm-up time	<1 min
Mode of operation	Continuous operation with intermittent loading Laser intermittence: 16%, Max. 5 min ON/ 25 min OFF Do not use the equipment in the presence of flammable anaesthetics (gases).

Operating Environment	
Temperature	+15° C to +29° C (59° F to +84.2° F)
Rel. Humidity	30 to 90%, non-condensing
Air Pressure	600 hPa to 1060 hPa
Operations at temperatures below -20° C (-4° F) or above +60° C (140° F) may cause permanent damage to the device.	

Storing and Handling	
Temperature	-20° C to +60° C (-4° F to +140° F)
Rel. Humidity	<90%, non-condensing
Air Pressure	500 hPa to 1060 hPa

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Dimensions		
Goggles	Length	7.25 in (18.4 cm)
	Width	0.5 in (1.3 cm) to 1.75 in (4.4 cm)
	Height	1.75 in (4.4 cm)
Interface box	Length	5 in (12.7 cm)
	Width	2.75 in (7 cm)
	Height	1 in (2.5 cm)

Weight	
Goggles	2.1 oz (60 g)
Interface box	4.6 oz (130 g)

Calibration
Calibration of the system is not required

Classification
Class II Type B

Standards	
Safety	Complies with UL 60601-1, 1st ed.: 2003, CAN/CSA-22.2 No. 601.1-M90, IEC60601-1, 2.ed.: 1988 + A1 + A2, IEC 62471, 1st.ed., IEC 60825-1, 2.ed.
EMC	IEC 6061-1-2: 2007

10.2 Accessories

Accessories		
Manuals/Videos	ICS Impulse Quick Guide	7-50-11300-EN 7-50-11300-DE 7-50-11300-ES 7-50-11300-FR 7-50-11300-IT
	ICS Impulse Training video	8-49-82700-US 8-49-82700-DE
Software	OTOsuite Vestibular	8-49-90300
Goggles	Face cushion 20/pkg	8-35-34400
	Strap assembly	8-35-34200
	Optical cleaning cloth ***Qty min 3***	7590527
	Fixation dot (2 sheets/pkg)	1-26-44000
Cables	USB 2.0 cable	8-71-79200
	1m IEEE-1394a Firewire® 400 6-pin to 4-pin cable	8-71-89600
	Cable clip	8-35-36900
Case/Mount	Carrying case	8-35-36700
	Wall mount	8-62-45600

10.3 Guidance and manufacturer's declaration tables

Electromagnetic Emissions

The ICS Impulse System is intended for use in the electromagnetic environment specified below. The customer or user of the ICS Impulse System should ensure that it is used in such an environment.

Emissions test	Compliance	Electromagnetic environment - Guidance
Radio Frequency (RF) Emissions CISPR 11	Group 1	The ICS Impulse System uses RF energy only for its internal function. Therefore, its RF emissions are very low and are not likely to cause any interference in nearby electronic equipment.
RF Emissions CISPR 11	Class A	The ICS Impulse System is suitable for use in all establishments other than domestic and those directly connected to the public low-voltage power supply network that supplies buildings used for domestic purposes
Harmonic emissions IEC 61000-3-2	Class A	
Voltage fluctuations/ flicker emissions IEC 61000-3-3	Complies	

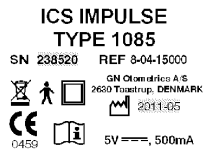
Immunity - All Equipment and Systems

The ICS Impulse System was not tested for immunity to electromagnetic disturbances.

Enclosures

Misc ID 7-01

Label 1
Size: 51 x 36 mm



Label 2
Size: 70 x 5 mm

Label 3
Size: 70 x 13 mm

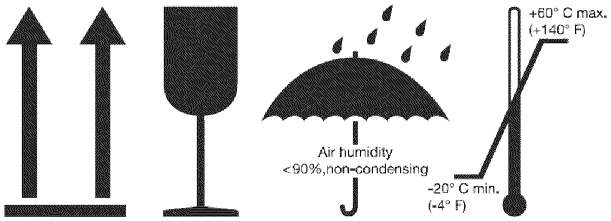
Label 4
Size: 78 x 10 mm

Label 5
Size: 60 x 9 mm

Label 6
Applied on goggle

Misc ID 7-02

Marking on packaging



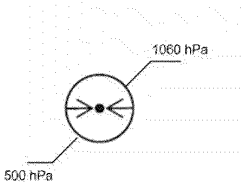


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Exhibit 18. Performance Testing – Bench

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Data Accuracy Validation Test Report

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(b)(4) Mechanical Verification Test Protocol

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Exhibit 19. Performance Testing – Animal

Not Applicable

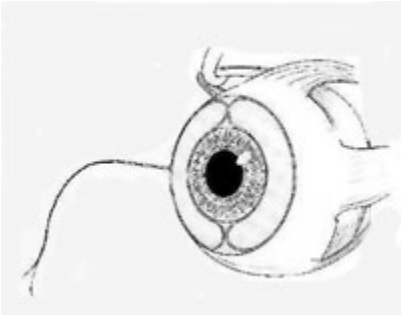
Exhibit 20. Performance Testing – Clinical

Fundamentals and applications of the Gold Standard: The Scleral Search Coil Technique
Type 1085 Comparison of Impulse to Scleral Searchex Coils Test Report
ICS Impulse and Micromedical Vorteq Comparison
1085 ICS Impulse Clinical Evaluation_7-38-06510 Rev 00

Fundamentals and applications of the Gold Standard: The Scleral Search Coil Technique
Accuracy Validation Test Report: ICS Impulse tested against Scleral Search Coil Technique

Fundamentals and applications of the Gold Standard: The Scleral Search Coil Technique

The principle of the scleral search coil technique is based upon the magnetic induction of a small coil (Robinson 1963). The induction coil is embedded in a flexible ring of silicone rubber which adheres to the limbus of the human eye concentric with the cornea (Collewijn et al. 1975). Around the head of the subject an alternating horizontal and vertical magnetic field (spatially and temporally in quadrature) is generated and consequently an alternating voltage will be induced in the coil. After amplification and phase-locked detection two analog voltages are obtained which are proportional to the sine of the horizontal and vertical eye position. In addition to this coil, which is wound in the frontal plane, a second coil is wound in the sagittal plane (Ferman et al. 1987). This combination coil simultaneously measures horizontal, vertical and torsional eye position.



This technique is used for physiological research of the oculomotor system in man and animals (Judge et al. 1980). Its high accuracy and bandwidth guarantees effortless recording of not only saccades, smooth pursuit, vergence, vestibular and optokinetic eye movements but also of miniature eye movements: tremor, drift and microsaccades. Applications include neuro-physiological, reading, psychological, psychiatric and visual studies.

Type 1085 Comparison of Impulse to Scleral Search Coils Test Report

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Test Report

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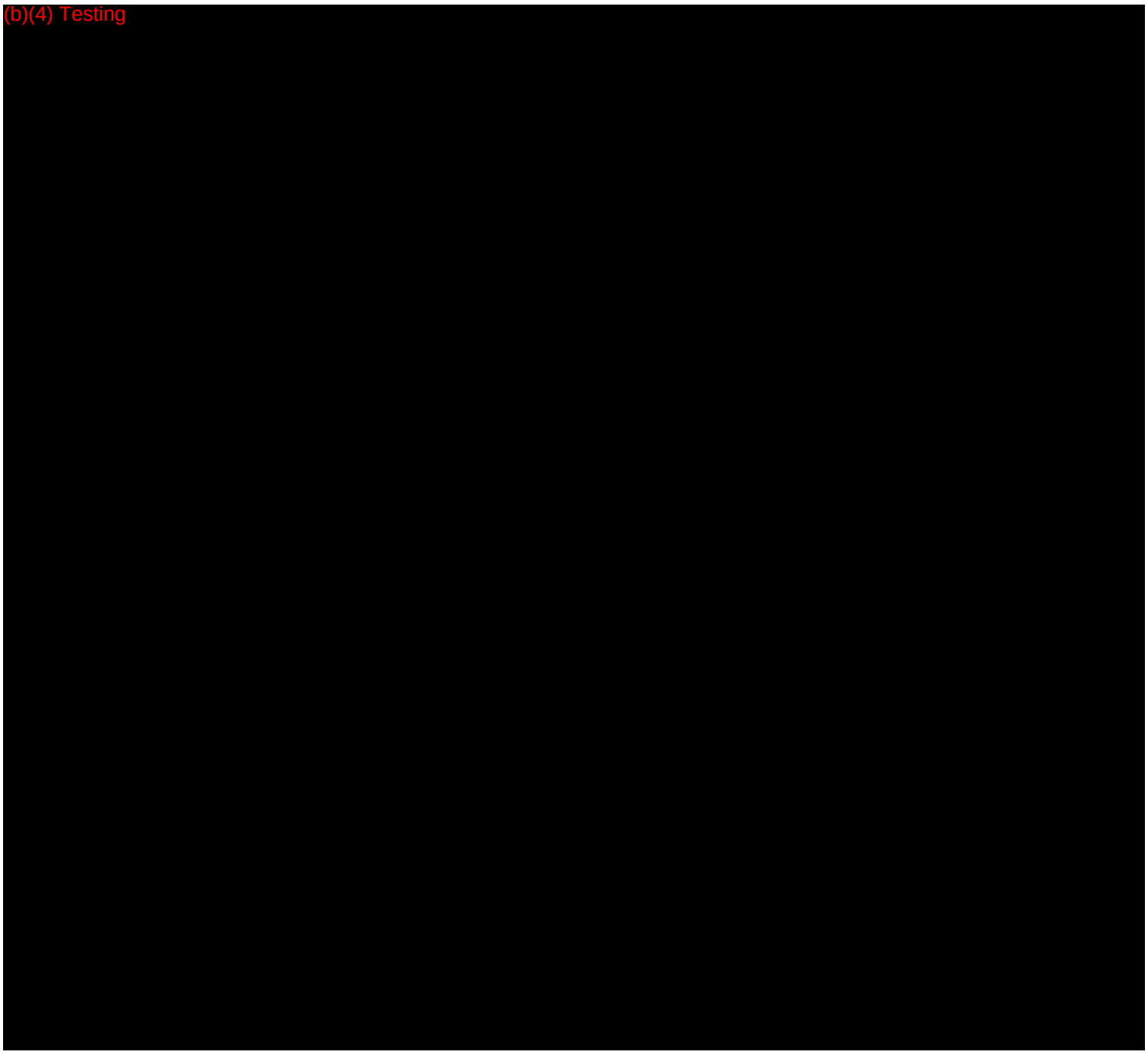
ICS Impulse and Micromedical Vorteq Comparison

ICS Impulse and Micromedical Vorteq Comparison



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ORIGINAL CONTRIBUTION

The Active Head-Impulse Test in Unilateral Peripheral Vestibulopathy

Ross A. Black, MBiomedE; G. Michael Halmagyi, MD; Matthew J. Thurtell, MB, BS; Michael J. Todd, MBiomedE; Ian S. Curthoys, PhD

Background: The head-impulse test, which is sensitive and specific for detecting severe unilateral peripheral vestibulopathy, is an accepted part of the neurological examination, especially in patients with vertigo and balance disorders.

Objective: To discover if the head-impulse test is just as useful diagnostically when patients are asked to rotate their own heads, the active head-impulse test, rather than when the clinician does so as in the standard passive head-impulse test.

Methods: Clinical observation of compensatory saccades and search coil measurement of compensatory eye rotations, during active and passive horizontal head-impulses in 6 patients with total unilateral vestibular deafferentation.

Results: Clinical observation showed the expected compensatory saccades with rotations toward the side with the lesion with passive head-impulses but not with active head-impulses. Search coil recordings revealed 2 reasons for this. With active head-impulses not only was vestibulo-ocular reflex gain higher, but compensatory saccade latency was shorter resulting in an occult saccade that occurred during, rather than after, head rotation.

Conclusions: Passive head-impulses are necessary to detect a severe unilateral peripheral vestibulopathy; active head-impulses will produce a false-negative result.

Arch Neurol. 2005;62:290-293

THE HEAD-IMPULSE TEST, since we first described it 15 years ago,[1] has become an accepted part of the neurological examination.[2] It detects severe unilateral loss of semicircular canal (SCC) function clinically and is important for the assessment of dizzy patients in the emergency department and physician's office.[3] In the emergency department the head-impulse test can distinguish between vestibular neuritis and cerebellar infarction in a patient seen with an initial attack of severe acute spontaneous vertigo: the result of the head-impulse test is invariably normal in a patient with a cerebellar infarction but abnormal in a patient with vestibular neuritis. In the physician's office the head-impulse test can identify a vestibular disorder in the patient with recurrent dizziness.

Could the head-impulse test be as useful with active head-impulses as with passive head-impulses, that is, with the patients themselves rotating their heads rather than the clinician doing so? To address this question we trained 6 patients with total unilateral vestibular deafferentation after surgery for vestibular schwannoma (acoustic neuroma) to make rapid, active head-impulses. We observed their eye movements clinically and then measured the horizontal vestibulo-ocular reflex (VOR) with active as well as with passive head-impulses. The velocity profiles of the active head-impulses matched those of the passive head-impulses. We found that not only is the horizontal VOR gain higher with active than with passive head-impulses[4] but that also with active head-impulses the compensatory saccade occurs during, rather than after, the head-impulse, thus giving the false impression of a normal horizontal VOR and, consequently, a spuriously negative finding for the head-impulse test in patients with total unilateral loss of SCC function.

METHODS

Three-dimensional eye and head positions were measured in a magnetic field (CNC Engineering, Seattle, Wash) with dual-axis search coils (Skalar, Delft, the Netherlands). Eye position was measured using a scleral search coil contact lens placed on the left eye. Head position

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was measured using a search coil mounted in a mouth guard composed of dental impression material (President Putty; Coltène/Whaledent Inc, Mahwah, NJ) in a disposable dental impression tray. Full details of the recording methods have been reported.[5,6]

We studied the VOR with active and passive head-impulses in 6 subjects with total unilateral vestibular deafferentation (5 men, 1 woman), aged 32 to 71 years, more than 2 years after vestibular schwannoma (acoustic neuroma) surgery. The head-impulse is a low-amplitude (15°-20°), high-acceleration (4000°/s²-6000°/s²) head rotation, in this case horizontal, with a peak velocity of 150°/s to 350°/s. Details of the head-impulse test have been previously published.[6,7] A passive head-impulse is delivered by an investigator who stands behind the patient and firmly holds the patient's head; at unpredictable times the investigator rapidly turns the patient's head to the left or right by 15° to 20° while the patient tries to keep staring at the target. The target is an earth-fixed laser dot located 94 cm straight ahead of the left eye. Active head-impulses were made by the patients themselves: each was asked to make rapid horizontal head rotations with velocities that fell within a template envelope representing the upper and lower 95% confidence intervals of the velocity of all passive head-impulses. The template was shown on a screen throughout the experiment. As the patient made an active head-impulse its velocity profile was instantaneously displayed on the head velocity template envelope (**Figure 1**). With this feedback all patients learned to make active head-impulses that fell within the passive head-impulse velocity envelope. Only those head velocities that fell within the envelope were analyzed. The peak velocities and durations of active and passive head-impulses were therefore matched. Written informed consent was obtained from subjects prior to testing, according to the Declaration of Helsinki. The experimental protocol was approved by the human ethics committee of the Central Sydney Area Health Service, Sydney, Australia.

The VOR with active and passive head-impulses was analyzed from the onset of head rotation to final gaze fixation—about 500 milliseconds. The gain of the VOR was calculated by finding the gradient of a line fitted to the mean eye velocity plotted as a function of head velocity in a time frame window from 30 to 75 milliseconds of head rotation onset. Full details of the analysis methods have been previously published.[6] Mean ± 2-tailed 95% confidence intervals were computed for head and eye velocity. A 2-tailed *t* test ($P<.05$) was performed to determine whether a statistically significant difference existed between head velocities and accelerations and VOR gains with active vs passive head-impulses.

RESULTS

All 6 patients learned to make active head-impulses that matched the passive head-impulses within about 20 attempts. While it was easy to see the compensatory saccade with passive ipsilesional head-impulses—the clinical sign of canal paresis[1]—we all had difficulty in doing so when the patient made active head-impulses because compensatory eye movements appeared smooth during most active head-impulses.

Using scleral search coil recordings, it was possible to make a valid comparison of the VOR in response to active vs passive head-impulses because the head velocity profiles in the first 75 milliseconds were the same (**Figure 2**). The results were that while ipsilesional VOR gain was abnormally low with both active and passive

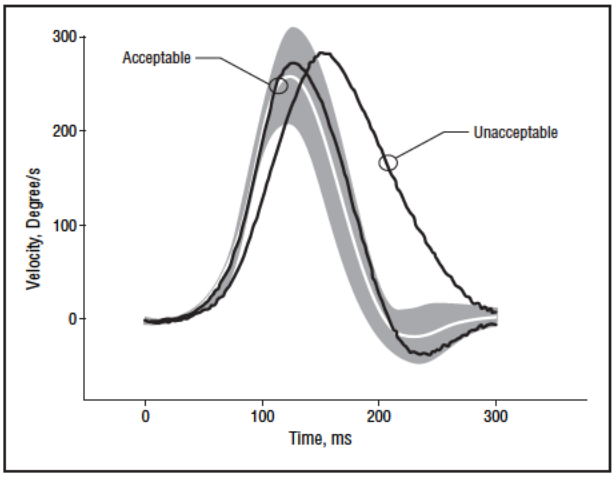

Figure 1. Acceptable and unacceptable head velocity template. Each patient's averaged passive head-impulse velocities were displayed on a screen as the template for active head-impulses. With active head-impulses, each patient would eventually make active head-impulses that had velocity profiles within the passive head-impulse velocity template. The figure shows an example of an acceptable and an unacceptable active head-impulse superimposed on the template.

head-impulses, with active head-impulses (mean ± SD, 0.38 ± 0.1), it was almost twice that seen with passive head-impulses (0.18 ± 0.02) (Figure 2). In contrast, contralesional VOR gain was only slightly higher with active head-impulses (0.78 ± 0.08) than with passive head-impulses (0.68 ± 0.06) (**Figure 3**).

Furthermore, the latency of the first saccade, which corrects the gaze position error accumulated during the ipsilesional head-impulse and forms the clinical sign of canal paresis,[1] was shorter with active than with passive ipsilesional head-impulses. With passive ipsilesional head-impulses, the mean latency of the compensatory saccade was 219 ± 102 milliseconds (mean ± SD) so that it invariably started only after the head had stopped rotating (Figure 2). In contrast with active ipsilesional head-impulses, the mean latency of the compensatory saccade was only 87 ± 11 milliseconds, so that typically it would begin while the head was still rotating. Furthermore, since VOR gain was higher with active than with passive ipsilesional head-impulses, the accrued gaze error and therefore the mean amplitude of the compensatory saccade was lower with active (approximately 4°) than with passive head-impulses (approximately 12°). Consequently, with active ipsilesional head-impulses, the eye position response appeared to be almost perfectly compensatory for head rotation (Figure 2)—creating the misleading impression of a near-normal VOR.

COMMENT

The technique and interpretation of the passive head-impulse test is generally straightforward as long as one recalls that SCC afferent neurons have a tonic firing rate at rest—about 40 spikes per second in the squirrel monkey.[8] Rotations away from an SCC (ie, in the off-direction) decrease the firing rate of the neuron whereas rotations toward an SCC (in the on-direction) increase its firing rate. With high-acceleration rotations, it is much

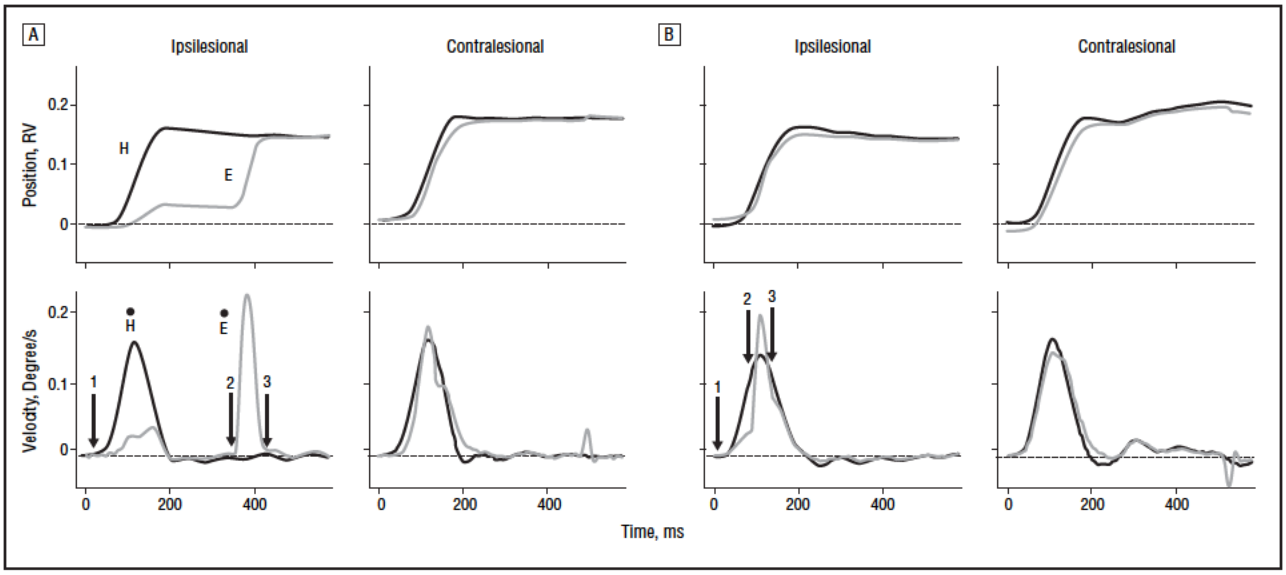

Figure 2. Passive (A) and active (B) head-impulse graphs. Typical active and passive, ipsilesional and contralesional head-impulses from a subject with unilateral vestibular deafferentation from just before the start of head rotation until just after the target is acquired. The start of head rotation is indicated by arrow 1, the start of the compensatory saccade by arrow 2, and the end of the compensatory saccade by arrow 3. With a passive ipsilesional head-impulse, a large gaze error is generated and it persists until after the end of head rotation, when it is corrected by a single saccade. With an active ipsilesional head-impulse, a compensatory saccade occurs during the head rotation so that the resulting gaze error is small. The higher initial vestibulo-ocular reflex gain with active head-impulses plus an early compensatory saccade result in an eye rotation response that is clinically indistinguishable from the head rotation stimulus. Contralesional head-impulses, both passive and active generate a vestibulo-ocular reflex within the normal range. All angles are shown in rotation vectors (RVs). Multiplying the RV value by 100 gives the approximate value in degrees. Hence, 0.1 RV is about 10°. H indicates the head position and velocity data; E, the eye position and velocity data.

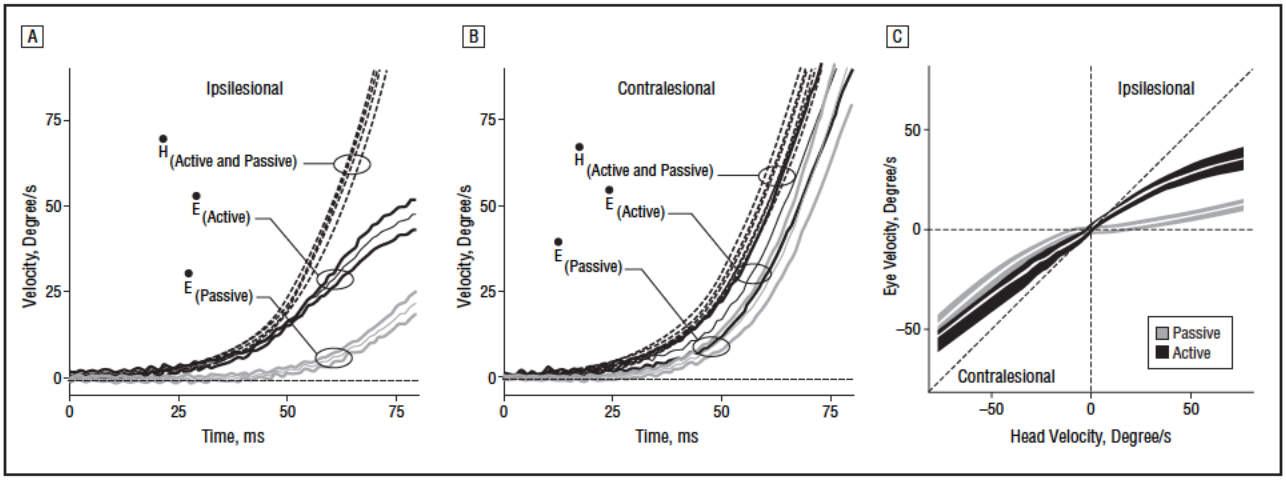

Figure 3. The mean±2-tailed 95% confidence intervals of head and eye velocities in the initial 75 milliseconds of active and passive ipsilesional (A) and contralesional (B) head-impulses from all subjects with unilateral vestibular deafferentation. Active and passive head-impulse velocity ($H_{[Active\ and\ Passive]}$) is shown as dotted black lines, eye velocity ($E_{[Active]}$) in response to active head-impulses as solid black lines, and eye velocity ($E_{[Passive]}$) in response to passive head-impulses as solid gray lines. Eye velocity has been inverted to aid comparison with head velocity. No statistically significant difference exists between the active and passive head-impulse velocities in the initial 75 milliseconds of ipsilesional or contralesional head rotations. A, Significant differences exist between the eye velocity responses to passive vs active ipsilesional head-impulses: the eye velocity response is higher (initial gain) with active than with passive head-impulses. Consequently, with active head-impulses, compensatory eye velocity at first closely follows the head velocity profile and then near peak head acceleration it diverges from head velocity. In contrast with passive ipsilesional head-impulses the initial compensatory eye response has lower magnitude and increases monotonically after onset. B, With contralesional head-impulses the magnitude of the eye velocity response to both active and passive impulses is higher than with ipsilesional head rotations and increases monotonically after onset. With active head-impulses the eye and head velocities are nearly matched. C, The mean±2-tailed 95% confidence intervals of horizontal eye velocity as a function of head velocity with active and passive ipsilesional and contralesional head-impulses. The statistically significant differences between active and passive impulses, in both directions of head rotation, are apparent even at low head velocities. Active head-impulses, in both directions, are associated with higher initial gains than passive head-impulses. The gains with contralesional head-impulses are higher than with ipsilesional rotations.

easier to saturate the firing rate of a vestibular neuron in the off-direction, that is, to silence it, than to saturate its firing rate in the on-direction.[9] In a patient with only one functioning labyrinth, rapid head rotations toward the side with the lesion will silence SCC afferents on the intact side and saturate the gain (eye velocity divided by head velocity) of the VOR.[6] Consider a patient with left vestibular neuritis: if the patient fixates a distant earth-fixed target while the clinician rapidly rotates the patient's head to the left, the passive head-impulse test, the patient will make 1 or 2 observable, compensatory, that is, rightward, saccades just after the head-impulse is over.

This is the head-impulse sign and it indicates that the gain of the horizontal VOR, which is generated from inhibition of the sole functioning, right, lateral SCC, rather than from excitation of the nonfunctioning left lateral SCC, is severely defective. In other words, the VOR gain is much less than 1.0. In contrast, when the clinician rotates the patient's head to the right, there will be no compensatory saccades indicating that the gain of the VOR, generated by excitation from the right lateral SCC plus any inhibition from the left lateral SCC, is close to 1.0. Not only can the compensatory saccades be observed clinically, the VOR deficit can be measured oculographically[4-7,10-12] not only from lateral but also from individual vertical SCCs.[6,13]

The present results show that active head-impulses, unlike passive head-impulses, are not an effective clinical way to show severe unilateral loss of vestibular function. They confirm that in unilateral vestibular loss ipsilesional VOR gain is abnormally low with both active[4,14] and passive head-impulses[5-7]; however, they also show that the VOR gain is significantly higher with active than with passive head-impulses. Although ipsilesional VOR gain is still low with active head-impulses resulting in a measurable, and potentially observable, gaze-error correcting compensatory saccade, the saccade latency is shorter with active head-impulses than with passive head-impulses so that the saccade is occult in that it occurs during, rather than after, the head-impulse. Because the velocity profile of the compensatory saccade resembles that of the head-impulse itself, it is easy clinically to mistake the compensatory saccade for a normal compensatory VOR and consider the findings of the head-impulse test to be normal. Consequently, for the diagnosis of a unilateral peripheral vestibulopathy, active impulses have 2 disadvantages compared with passive head-impulses: they not only produce a higher VOR gain, but they also generate an earlier compensatory saccade that can mimic a normal VOR.

Why is VOR gain higher and compensatory saccade latency shorter with active than with passive head-impulses? There is no theoretical reason why the VOR gain should be higher and there are no animal data because it has not proven possible to train an animal to make active head-impulses. Single neuron data from vestibular nucleus neurons in alert behaving monkeys show reduced activation with active vs passive head rotations.[15] However, in these experiments the animal's goal was refixation rather than fixation, a task that would benefit from a lower VOR gain. Active head rotations are normally made to refix rather than to fix gaze and that is presumably why for human subjects it feels unnatural to rotate the head rapidly while trying to look straight ahead. Saccadic latency might be shorter with active head-impulses than with passive head-impulses because of a learning effect. Our subjects were trained to match the velocity profile of their own passive head-impulses with an active head rotation. Therefore, our subjects may have learned to predict their own gaze error and to make compensatory express saccades[16] almost in time with head rotation. It is clear, therefore, that passive head-impulses rather than active head-impulses are needed to demonstrate a unilateral vestibular deficit.

Accepted for Publication: May 5, 2004.

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Funding/Support: This study was supported in part by the National Health and Medical Research Council, Canberra, Australia; the Garnett Passe and Rodney Williams Memorial Foundation, Melbourne, Australia; and the Royal Prince Albert Hospital Neurology Department Trustees, Sydney.

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ORIGINAL ARTICLE

Comparison of Head Thrust Test With Head Autorotation Test Reveals That the Vestibulo-ocular Reflex Is Enhanced During Voluntary Head Movements

Charles C. Della Santina, PhD, MD; Phillip D. Cremer, MBBS, PhD; John P. Carey, MD; Lloyd B. Minor, MD

Objectives: To compare 2 clinical tests of vestibular function, the head autorotation test (HART) and the head thrust test (HTT), and to determine why they give disparate results in patients with known unilateral vestibular deficiency (UVD) due to labyrinthectomy.

Methods: We used scleral coils to measure the horizontal (yaw) vestibulo-ocular reflex (VOR) in 5 healthy human subjects and in 11 patients who underwent labyrinthectomy. We used 2 paradigms. Using HART, subjects visually fixated a target during self-generated, swept-frequency, sinusoidal, horizontal head rotations. Using HTT, patients fixated the target during horizontal head thrusts delivered randomly in direction and time.

Results: In subjects without UVD, eye movements were almost perfectly compensatory for both paradigms. In subjects with UVD, VOR gain for ipsilesional head thrusts was low for both paradigms, but significantly ($P<.001$) higher (less abnormal) for HART (0.60±0.13) than for HTT (0.14±0.13). Contralesional gain was reduced for both, to 0.64±0.20 for HART and to 0.57±0.17 for HTT. Because ipsilesional and contralesional gains were not statistically different for HART ($P=.69$), comparison of VOR gains for half-cycle responses to the HART stimulus could not reliably identify the side of the known lesion. In contrast, HTT consistently identified the side of the lesion for all subjects with UVD. To investigate whether preprogramming contributes to the boost in VOR as measured by HART, we compared the gain and response delay of eye movements during actively self-generated and passively received head thrusts. For subjects without UVD, response delays were shorter for active (6±1 milliseconds) than for passive (12±1 milliseconds) HTT. For ipsilesional rotations of subjects with UVD, active HTT yielded a significantly higher gain (0.44±0.20) ($P<.001$) and a shorter delay (15±6 milliseconds) ($P<.001$) than did passive HTT (0.14±0.13 and 37±15 milliseconds, respectively). Contralesional test results revealed a similar performance boost for active head movements. Data are given as mean±SD.

Conclusion: When comparison of half-cycle gains is used to identify the lesion side, self-generated predictable head movement paradigms, such as HART and active HTT, are less accurate than passive HTT in the characterization of UVD, in part because preprogramming can augment the VOR during voluntary head movements.

Arch Otolaryngol Head Neck Surg. 2002;128:1044-1054

From the Department of Otolaryngology–Head & Neck Surgery, The Johns Hopkins Medical Institutions, Baltimore, Md (Drs Della Santina, Carey, and Minor); and the Eye and Ear Research Unit, Institute of Clinical Neurosciences, Royal Prince Alfred Hospital, Sydney, Australia (Dr Cremer).

DIZZINESS IS the ninth most common reason adults visit a primary care physician, and it affects approximately 90 million Americans.[1-4] Identification of the side, affected semicircular canals, and degree of unilateral vestibular deficiency (UVD) is an important goal in the examination of patients experiencing dizziness.

Two high-frequency rotational stimulus paradigms—the head thrust test (HTT) and the head autorotation test (HART)—are in wide use for assessing angular vestibulo-ocular reflex (VOR) function, yet have not been directly compared.

Researchers[3-8] have advocated HTT, in which high-acceleration impulsive head rotations are delivered in the plane of one pair of semicircular canals while a subject attempts to maintain visual fixation on a distant target. Implied by the second law of Ewald,[9] such head rotations elicit an asymmetric response in patients with UVD by maximally stimulating the neural pathway arising from one semicircular canal crista while maximally inhibiting or silencing the pathway from the coplanar canal. The approach has proved sensitive for the detection of vestibular dysfunction in subjects with known UVD.[6,10]

A second paradigm, HART, uses self-generated, swept-frequency, sinusoidal head rotations performed while the subject visually fixates a distant earth-fixed target. Fourier analysis of system gain and phase, along with formulaic measures of response asymmetry, are applied to the measured head and eye movements. Researchers[11-16] have reported extensively on this approach, and several commercially available vestibular testing systems are based on this paradigm.

We sought to compare the ability of HTT with that of HART to identify dysfunction in subjects with known UVD after surgical labyrinthectomy. There are 3 differences between these 2 stimulus paradigms that could contribute to a difference in measured VOR for a given patient. First, HTT stimuli are passive (ie, delivered by the examiner), while during HART, the head movement is active (ie, self-generated). Second, HTT stimuli are presented unpredictably in time and direction, while the sinusoidal frequency–sweep head movement during HART is predictable cycle by cycle. Third, HTT stimuli are often of higher acceleration and wider spectral bandwidth than are HART stimuli, because of some subjects' inability to make rapid, self-generated, sinusoidal head movements.

We hypothesized that during active predictable head movements, subjects with UVD can augment apparent VOR performance by using information about the intended/expected head movement to complement deficient vestibular function and guide compensatory eye movements. Such an effect would be expected to reduce the sensitivity of tests like HART that rely on self-generated head movement stimuli for detecting vestibular hypofunction. We tested these predictions by comparing VOR gains measured for subjects with and without UVD during HART and HTT, and by comparing subjects' responses to active and passive impulsive head rotations.

METHODS

SUBJECTS

We studied 5 human subjects without UVD and 11 with UVD due to labyrinthectomy. Subjects without UVD ranged in age from 32 to 55 years and were free of vestibular and ocular disease, except for wearing corrective lenses. Subjects with UVD ranged in age from 48 to 72 years, and the time from labyrinthectomy ranged from 3 to 120 months. All had undergone vestibular rehabilitation therapy postoperatively. The indication for labyrinthectomy was vestibular schwannoma or meningioma in 6 subjects and unilateral Meniere disease in 5. The side of labyrinthectomy was the left for 6 subjects with UVD and the right for 5. All subjects gave written informed consent, and the experimental procedures were approved by the Joint Committee on Clinical Investigation, The Johns Hopkins University School of Medicine, Baltimore, Md.

EXPERIMENTAL TECHNIQUE

Head and eye movements were recorded in 3 dimensions using magnetic search coils embedded in contact lenses and in a bite block. The instrumentation and technique have been described in detail elsewhere.[17] Eye and bite block angular positions were sampled at 500 Hz. The resulting signals were low-pass filtered with a single-pole analog filter with a 3-dB bandwidth of 100 Hz. Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line parallel to the horizon and the Frankfort line (from the top of the tragus to the infraorbital foramen) in the plane of head rotation. All rotational stimuli were in the horizontal (yaw) plane. During each trial, the room was completely dark except for a target light-emitting diode positioned 1.24 m anterior to the center of the head, at the same elevation as the pupils. All subjects were tested more than 20 minutes after removal of eyeglasses.

Each experiment began with the subject's head held rigidly at the starting position via connection of the bite block to a bar, while the subject performed calibration tasks. The bar was then removed, and all subsequent trials were performed with the head free to move during a stimulus, then returning to the starting position.

For HART, subjects attempted to visually fixate the target while sinusoidally shaking their heads horizontally (ie, rotating about an earth-vertical axis through the center of the cervical spine) at maximum tolerated velocity in time with a metronome swept logarithmically in frequency from 1 to 6 Hz for 20 seconds. Results from 3 trials were averaged.

For HTT, subjects fixated the same target during brief (100-200–millisecond) rotations in the earth-horizontal plane, at high acceleration (3000°-5000°/s^2) starting from a complete stop. Head velocity reached 25° to 75°/s at 40 milliseconds into the movement and 150° to 300°/s peak velocity by 100 to 120 milliseconds. The final position was 10° to 15° from center. For passive HTT (pHTT), stimuli were delivered manually at an unpredictable onset time and in a randomly varied direction, starting from center. The subject received no cues about the direction or time of an impending passive head thrust. For active HTT (aHTT), the subject generated the head thrust voluntarily. Only head movements reaching greater than 100°/s within the first 60 milliseconds were included in the analysis. A typical trial lasted about 3 minutes and included about 20 to 40 head thrusts to each side.

ANALYSIS

To facilitate comparison between eye and head angular velocities, both were analyzed in 3 dimensions in the head frame of reference. Horizontal rotational components were extracted and compared.[18] All results reported are for the horizontal components of rotation only. Angular velocity and acceleration were computed from angular position signals using a 30-Hz, band-limited, 10-element, linear phase differentiating digital filter designed using computer software (MATLAB; The Mathworks, Inc, Cambridge, Mass) and applied in zero-delay fashion.

We quantified the eye movement responses in terms of velocity gain and response delay. Gain was defined as the angular velocity of the eye divided by the angular velocity of the head at the instant head velocity crossed to above 120°/s, with the eye velocity inverted so that a gain of +1.0 denotes an ideal compensatory response. Using the method of Tabak et al,[7] we defined response delay as the time between the onsets of head and eye movements, where onset was defined as the time at which velocity crossed a threshold set at 2°/s plus 8 times the root-mean-square velocity in the 100 milliseconds preceding the stimulus.[8] We used 1- or 2-tailed *t* tests to compare gain and response delay distributions between experimental groups, with $P<.05$ considered significant. Data are given as mean±SD unless otherwise indicated.

RESULTS

HEAD AUTOROTATION TESTING

Figure 1 shows HART results for a healthy 33-year-old man without

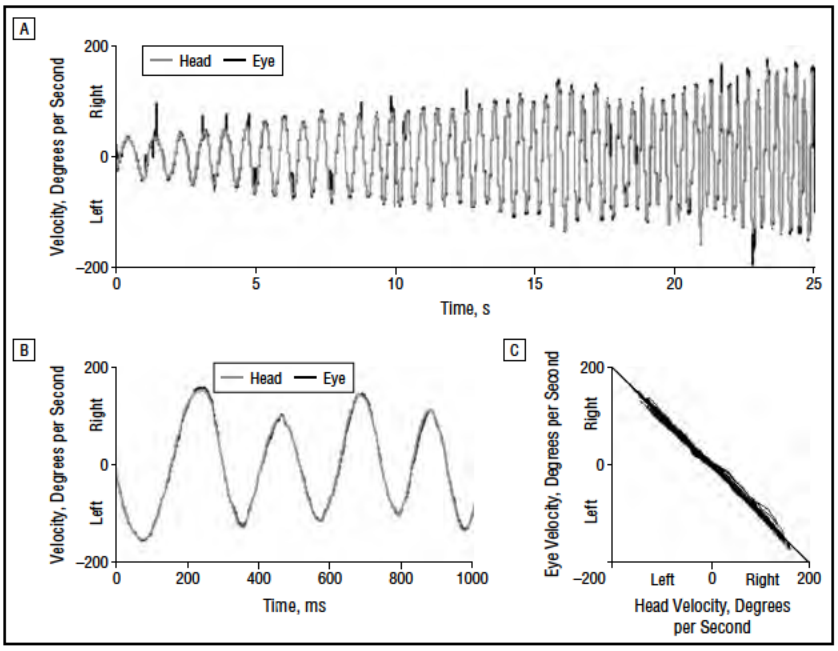

Figure 1. Horizontal head autorotation test results of a 33-year-old man without a unilateral vestibular deficiency. A, Head and eye velocity vs time, with the eye trace inverted for comparison with the head trace. (All similar figures use the same convention.) The eye velocity trace overlies the head trace almost exactly. In the trial shown, head velocity ranged from 40°/s at 1 Hz to 180°/s at 6 to 8 Hz. B, Expanded view of a portion of the trial. Eye velocity traces are nearly identical to head velocity traces. C, Eye velocity vs head velocity after removal of saccades. Data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect vestibulo-ocular reflex.

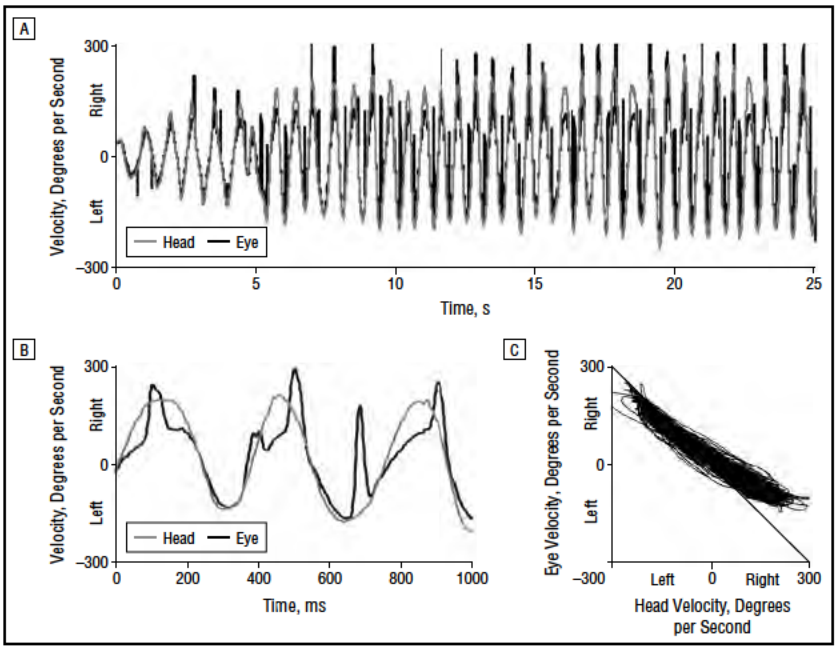

Figure 2. Head autorotation test results of a 63-year-old man 5 months after right translabyrinthine vestibular schwannoma resection. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity. Contralesional (leftward) head turns elicit a moderately degraded response, with a gain of 0.87±0.13 (mean±SD). Ipsilesional responses are more abnormal, with decreased velocity gain, increased retinal slip errors of up to 100°/s, and large-amplitude saccadelike catch-up eye movements.

UVD. In the trial shown, head velocity ranged from 40°/s at 1 Hz to approximately 180°/s at 6 to 8 Hz (Figure 1A). Eye velocity traces (inverted for comparison with head velocity) are nearly identical to head velocity traces, to within less than the 4°/s of retinal image slip for which visual acuity begins to degrade (Figure 1B).[19] When plotted as eye velocity vs head velocity, these data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect VOR (Figure 1C). This subject without UVD has rightward and leftward VOR velocity gains of 1.0±0.05 and 1.0±0.02, respectively.

Figure 2 shows HART results for a symptomatically well-compensated 63-year-old man who had undergone right translabyrinthine vestibular schwannoma resection and postoperative vestibular rehabilitation 5 months before testing. For this subject, the response during head turns toward the contralesional left side (Figure 2A and B) is somewhat degraded, but not significantly ($P=.07$) different from normal (gain, 0.87±0.13). During rightward head turns, there is a significantly ($P<.001$) abnormal response, with a velocity gain of 0.43±0.19 and retinal slip errors of up to 100°/s. Retinal slip not only degrades foveal visual acuity but also accrues to cause the subject to lose target fixation, eliciting large saccadelike corrective eye movements to reacquire the visual target. For this subject, HART results clearly reveal an asymmetry.

Such asymmetries between responses to ipsilesional and contralesional head rotations were not always apparent. **Figure 3** presents the HART responses from a 68-year-old man tested 38 months after undergoing surgery resulting in left UVD. At maximum effort, this patient achieved slower peak head velocity than the subject shown in Figure 2. The slow-phase components of the response to ipsilesional rotations were not markedly different from those observed in the contralesional half cycles. However, a gaze-correcting saccadic eye movement was often observed in the ipsilesional responses.

Figure 4A and B shows a summary of VOR gains measured using HART in 5 healthy subjects and in 11 subjects with UVD, respectively. The VOR gains for subjects without UVD are segregated into rightward and leftward head movements and illustrated separately as a check of test reliability. The VOR gains for subjects without UVD are 0.95±0.13 and 1.03±0.11 for rightward and leftward head rotations, respectively. There was no significant difference between right and left VOR gains

(P=.13), and the combined data set was insignificantly different from 1.0 (P=.64) (95% confidence interval, 0.93-1.05). The HART gains for subjects with UVD were segregated into ipsilesional and contralesional directions. Four findings were notable. First, the ipsilesional gain of 0.60±0.13 was significantly lower than normal (P<.001). Second, there was a wide distribution of ipsilesional gains for this sample population, ranging from 0.2 to 1.0. Third, the contralesional gain of 0.64±0.20 was also significantly decreased from normal (P<.001). Finally, HART gains were not significantly different for ipsilesional and contralesional rotations in these subjects with UVD and, therefore, could not reliably indicate the side of the known lesion (P=.69).

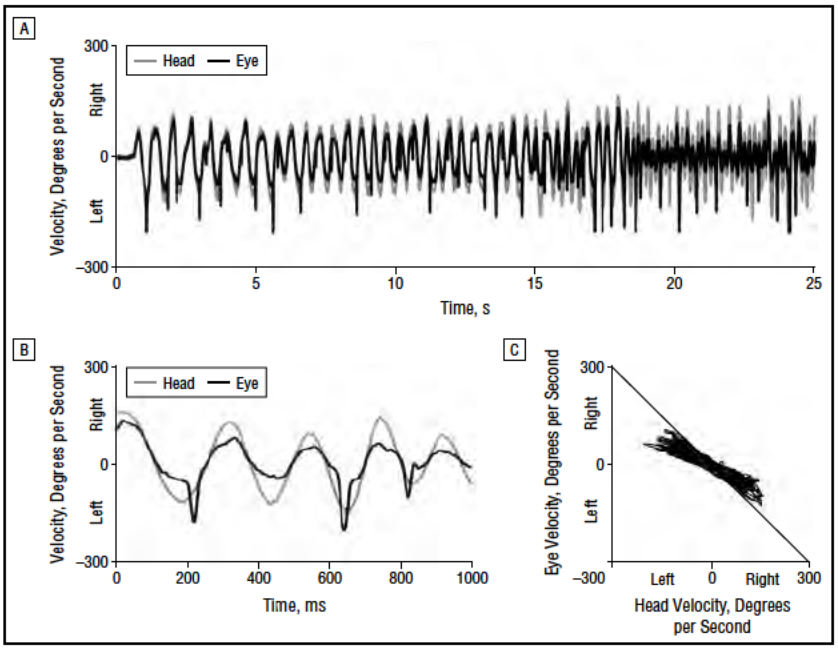

Figure 3. Head autorotation test results of a 68-year-old man 38 months after surgery for a left unilateral vestibular deficiency. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity after saccade removal. Saccadelike catch-up eye movements are directed toward the lesion side, but slow-phase eye movements show little asymmetry.

PASSIVE HTT

Passive HTT was performed on 4 of the subjects without UVD and on all 11 subjects with UVD who had been studied using HART. **Figure 5**A shows head velocity and concurrent eye velocity traces for 2 representative head thrusts from the same subject without UVD who was described in Figure 1. The high-acceleration head rotation transients reach a peak velocity of greater than 200°/s within 80 to

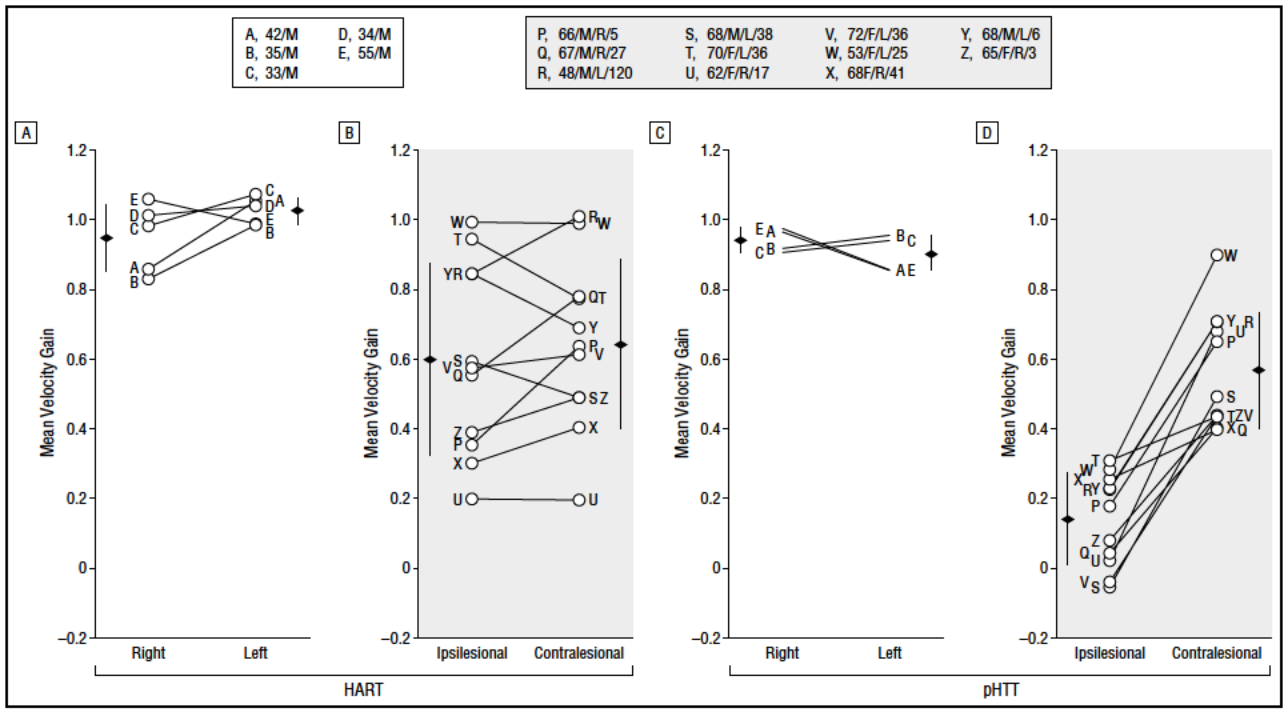

Figure 4. The head autorotation test (HART) gains measured in 5 healthy subjects (A) and in 11 subjects with unilateral vestibular deficiency (UVD) (B) are shown. A, The mean vestibulo-ocular reflex (VOR) gain for subjects without UVD is not significantly (P=.64) different from 1.0 for rightward and leftward head rotations. B, For this population of subjects with UVD, ipsilesional and contralesional HART gain distributions were not significantly different from each other (P=.69). The VOR gains for passive head thrust test (pHTT) in 4 subjects without UVD (C) and in 11 subjects with UVD (D) are also shown. C, Right and left pHTT gains of subjects without UVD are close to 1.0 and not significantly different from each other (P=.27). D, The gains of subjects with UVD were decreased for ipsilesional and contralesional head rotations, with a marked asymmetry between ipsilesional and contralesional gains for every patient tested. For A and C, the key provides subject age (in years)/sex (M indicates male; F, female); for B and D, subject age (in years)/sex/side (R indicates right; L, left)/time since labyrinthectomy (in months). Bars represent mean±SD.

100 milliseconds and are tracked accurately by compensatory eye movements, with a slight overshoot following the peak of head velocity. Figure 5B shows multiple head thrust stimuli and the corresponding eye movement responses for the first 80 milliseconds of each. Peak head accelerations ranged from approximately 3000° to 5000°/s^2. Although there is a slight early mismatch of eye and head traces because of VOR latency, retinal slip is kept below 5°/s throughout most of each trace. In Figure 5C, a plot of eye velocity vs head velocity reveals that all traces lie close to a $y=-x$ line, corresponding to nearly perfect performance. Rightward and leftward gains for this subject were 1.00±0.04 and 0.99±0.06, respectively. Subjects without UVD never complained of losing sight of the target during testing.

In contrast, **Figure 6** shows pHTT responses for the 68-year-old man with left UVD for whom HART data were described in Figure 3. In Figure 6A, a rightward head thrust is tracked fairly well by the eye, although the VOR gain is less than normal and a small catch-up saccade-like movement is required to correct the final eye position after the head movement ends. For a leftward head thrust, the VOR response decrease is more pronounced, as are the prominent catch-up eye movements. The second and third catch-up movements occur after the head stops moving; these are the corrective eye movements an observer can detect when using HTT as part of the physical examination. Figure 6B shows the first 80 milliseconds of multiple stimuli for this subject. For rightward head rotations, the eye tracks the head fairly well; however, the initial VOR-mediated eye movement response for leftward head thrusts is of low amplitude. Figure 6C reveals an asymmetry in the eye movement responses to contralesional and ipsilesional head thrusts, corresponding to the measured VOR gains of 0.50±0.15 and 0.06±0.09, respectively.

Figure 4C and D shows a summary of pHTT VOR gains for 4 subjects without UVD and 11 subjects with UVD, respectively. (The same subjects were used for pHTT and HART, except for 1 subject without UVD who was tested only with HART.) Right and left pHTT responses of subjects without UVD are shown separately in Figure 4C. Rightward (0.94±0.04) and leftward (0.90±0.05) pHTT gains were not significantly different ($P=.27$) for subjects without UVD. For subjects with UVD (Figure 4D), gains were decreased for ipsilesional and contralesional head rotations. In contrast to the HART VOR gains for the same population of subjects, there was a marked asymmetry between ipsilesional (0.14±0.13) and contralesional (0.58±0.17) gains. For every patient with UVD tested, ipsilateral gains were significantly lower than contralesional gains ($P<.001$). Ipsilesional and contralesional gains were each significantly different from normal ($P<.001$).

Comparison of HART and pHTT results reveals that although the ipsilateral VOR gains are reduced for the HART paradigm applied to subjects with UVD, a similar reduction in contralesional gains was such that HART could not reliably distinguish the side of the known lesion. In contrast, there was a marked and consistent asymmetry in the VOR gains of subjects with UVD measured using the pHTT paradigm, with the ipsilateral gains close to 0 and the contralesional gains close to gains measured using HART.

aHTT VS pHTT

Gain

To better identify why the VOR is apparently enhanced when measured by the HART paradigm, we compared VOR gains measured in subjects with and without UVD using head thrusts that were either passively and unpredictably received by the subjects as in the usual application of the test (pHTT) or actively generated by the subjects (aHTT). Analysis was limited to active and passive head movements that were similar in mean peak velocity, peak acceleration, and spectral content.

Figure 7 shows the first 80 milliseconds of head movements and corresponding eye movement responses to aHTT for the subject with left UVD shown in Figures 3 and 6. Whereas the ipsilesional pHTT response was essentially nonexistent for the first 80 milliseconds after stimulus onset (Figure 6B), the aHTT response VOR gain is enhanced, to 0.34±0.06, from 0.06±0.09 (Figure 7A). The contralesional VOR gain is also increased, to 0.74±0.05, from 0.50±0.15. Similarly, the time from stimulus onset to threshold response is shorter for aHTT than for pHTT.

Figure 8 shows pHTT and aHTT VOR gains for 3 subjects without UVD and 10 subjects with UVD (the same subjects used for HART except for those who did not generate aHTT head movements similar to those of pHTT). For subjects without UVD (Figure 8A), responses to aHTT and pHTT were not significantly different from each other or from 1.00 ($P>.05$ for all). For subjects with UVD, there was a significant ($P<.001$) increase in apparent ipsilesional VOR gain when measured using the aHTT paradigm, to 0.44±0.20 (from 0.14±0.13 for pHTT) (Figure 8B). The contralesional VOR gain also increased significantly ($P=.004$) under aHTT conditions, to 0.81±0.14 (from 0.58±0.17 for pHTT) (Figure 8C). This level was not significantly different from the VOR gains measured in subjects without UVD using pHTT ($P=.08$).

Response Delay

To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset and to compute a response delay. **Figure 9** shows pHTT and aHTT VOR response delays for 4 subjects without UVD and 11 subjects with UVD. For subjects without UVD (Figure 9A), the response delay was 11.0±3.3 milliseconds for pHTT and slightly, but significantly, lower at 5.0±1.8 milliseconds for aHTT ($P=.006$). For subjects with UVD, the response delay for ipsilesional head thrusts was significantly shorter for aHTT (19±16 milliseconds) than for pHTT (42±17 milliseconds) ($P=.006$) (Figure 9B). The response delay in subjects with UVD for contralesional head thrusts was also shorter for aHTT (11.0±5.1 milliseconds) than for pHTT

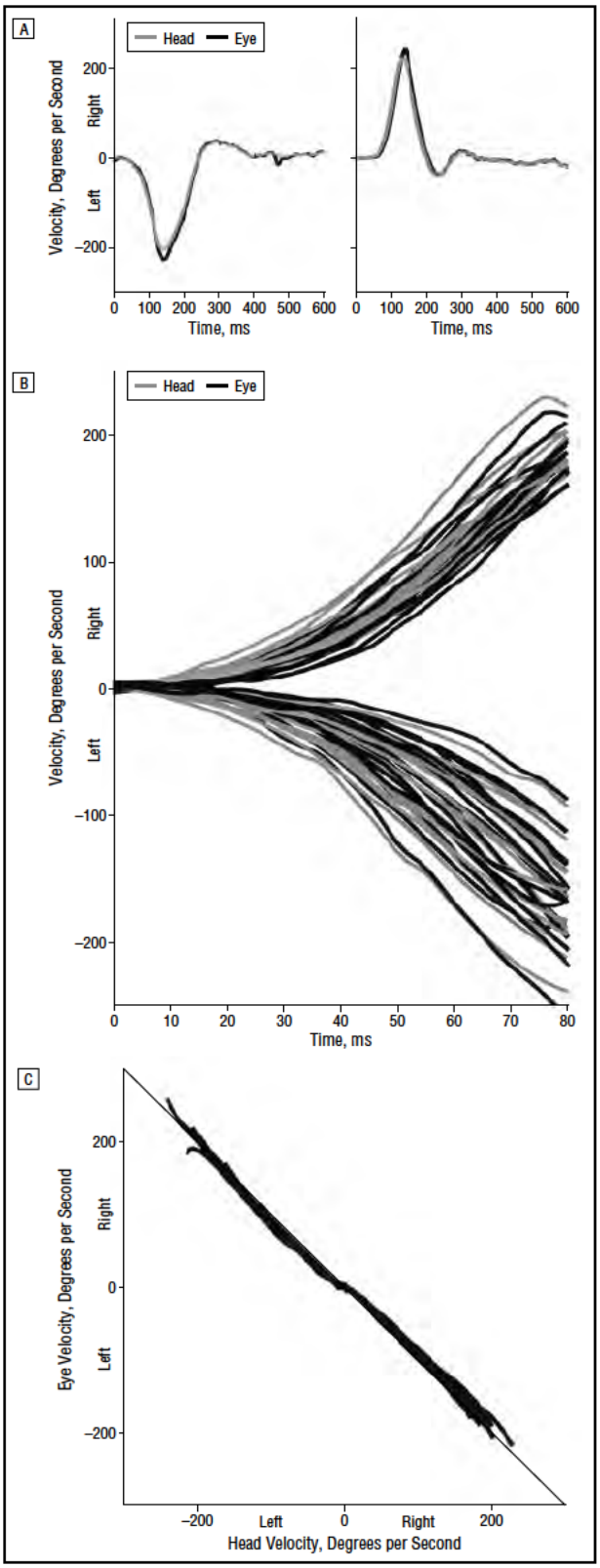

Figure 5. Passive head thrust test results for a healthy subject without a unilateral vestibular deficiency (UVD) (same subject as in Figure 1). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject has nearly perfect overlap of head and eye traces. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Traces nearly overlie each other for this subject without UVD. C, Eye velocity vs head velocity. Data lie closely along a $y=-x$ line, consistent with a near-perfect vestibulo-ocular reflex.

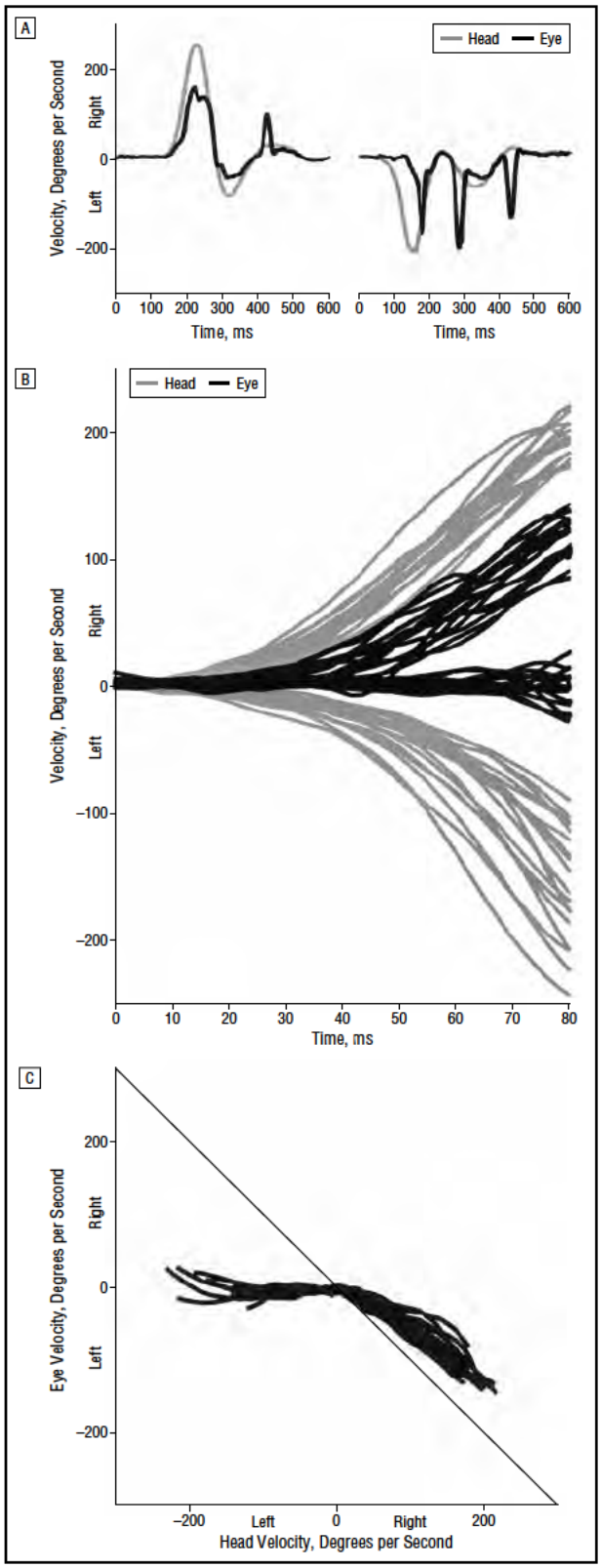

Figure 6. Passive head thrust test results in a subject with a unilateral vestibular deficiency (UVD) (same subject as in Figure 3). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject with a left UVD follows rightward head movement moderately well, but tracks leftward head thrusts poorly. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Response is delayed and of decreased magnitude, mildly for rightward head movements and dramatically for head movements toward the lesion side. C, Eye velocity vs head velocity for the first 80 milliseconds of responses.

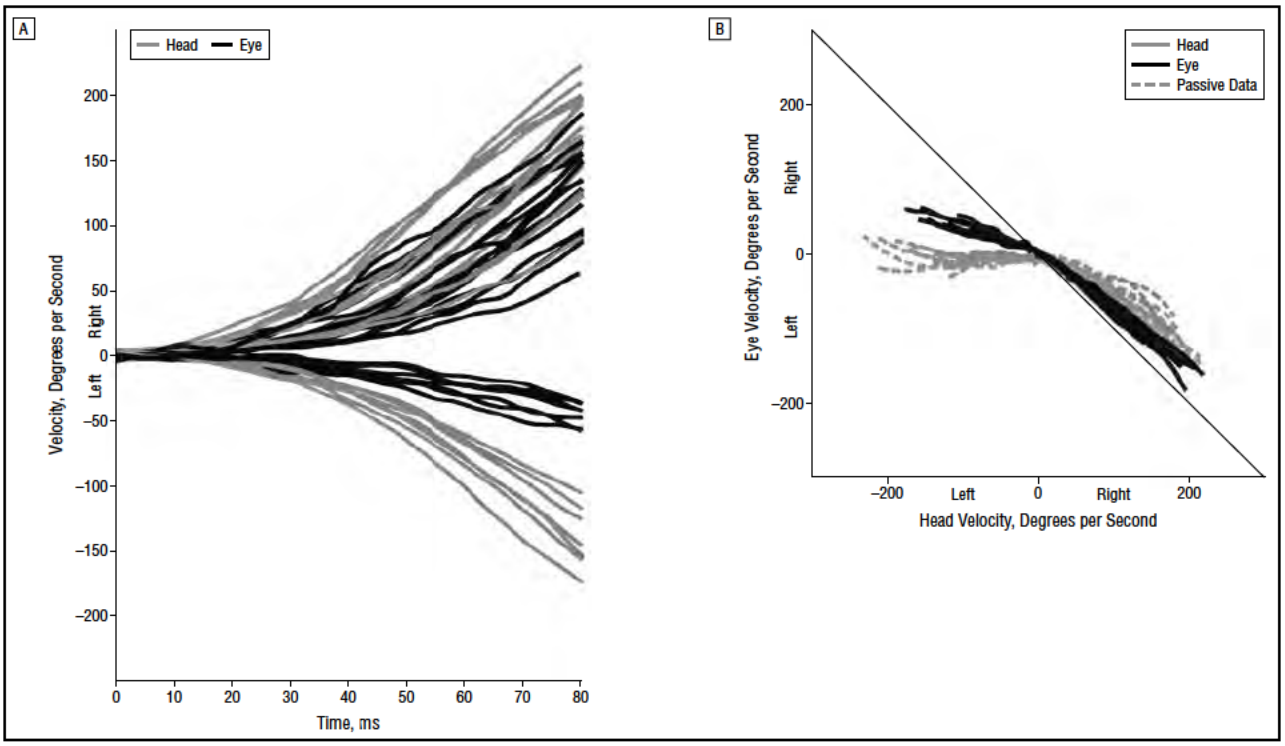

Figure 7. Active head thrust test results in a subject with a left unilateral vestibular deficiency (same subject for whom passive data are shown in Figure 6). A, Head and eye (inverted) velocities during the first 80 milliseconds of multiple active head thrust trials. B, Eye velocity vs head velocity. Although still not normal, the active eye movement response is significantly better than for passive trials, with shorter response delay and higher gains.

(15.0±4.5 milliseconds), but the difference was not significant ($P = .07$) (Figure 9C).

COMMENT

CLINICAL TESTS FOR IDENTIFICATION OF UVD

Traditional caloric and rotary chair tests for the identification of UVD fail to test the vestibular system using physiologically appropriate stimuli over the range of frequencies and accelerations for which the system apparently evolved. Caloric testing has the advantage of being strictly unilateral, but uses an unnatural stimulus (a thermal gradient) that, as measured by the eye movements it elicits, is the equivalent of a low frequency (0.025 Hz) and low amplitude (approximately 50°/s) head rotation. The caloric response can be altered by anatomic changes in the external ear canal and by individual variation in temporal bone anatomic features, and it only evaluates 1 of the 5 vestibular receptors (the lateral semicircular canal). Rotary chair vestibular testing is more physiologic; however, at the lower frequencies and velocities typically used in clinical vestibular laboratories, rotary chair tests are insensitive in the identification of chronic total unilateral vestibular hypofunction.[20,21] The sensitivity of rotary chair testing can be improved by comparison of responses to steps of acceleration with the head up and head down.[22]

In contrast, high-frequency and high-acceleration rotational stimuli unmask the inherent asymmetry in the vestibular system, as described by the second law of Ewald[9]—that the excitatory responses of vestibular pathway neurons encode motion over a larger dynamic range than do inhibitory responses. The eye movements elicited by such stimuli are principally due to excitation of the vestibular pathway arising from the canal ipsilateral to the direction of head acceleration and are, therefore, sensitive to a unilateral peripheral vestibular loss in that canal.

There are 2 high-frequency rotational stimuli in clinical use, pHTT and HART. The main objective of this study was to compare these 2 stimuli for the identification of unilateral vestibular hypofunction. The pHTT is a single, passive (operator delivered), unpredictable, high-acceleration (2000°-4000°/s^2), low amplitude (15°-30°) head rotation in the direction of a single semicircular canal. The HART is a continuous, active (self-generated), sinusoidal head rotation that begins slowly and becomes faster, covering a frequency range of about 1 to 6 Hz. During each test, subjects are required to fixate a visual target in front of them, and their ability to maintain visual fixation is accepted as a measure of VOR function. However, because HART is actively generated, non-VOR processes, such as predictive eye movements driven by an "efference copy" neural representation of the intended head movement, could contribute to maintaining gaze stability. As a result, VOR function could seem artificially enhanced, making HART a less sensitive and less accurate test of UVD.

We tested 11 patients who had undergone surgical labyrinthectomy and compared their responses with those of 5 subjects without UVD. The VOR gains for subjects without UVD using pHTT were insignificantly different from an ideal gain of 1.0, consistent with findings from previous studies.[23] For subjects with UVD, the VOR gains of 0.14±0.13 for ipsilesional head

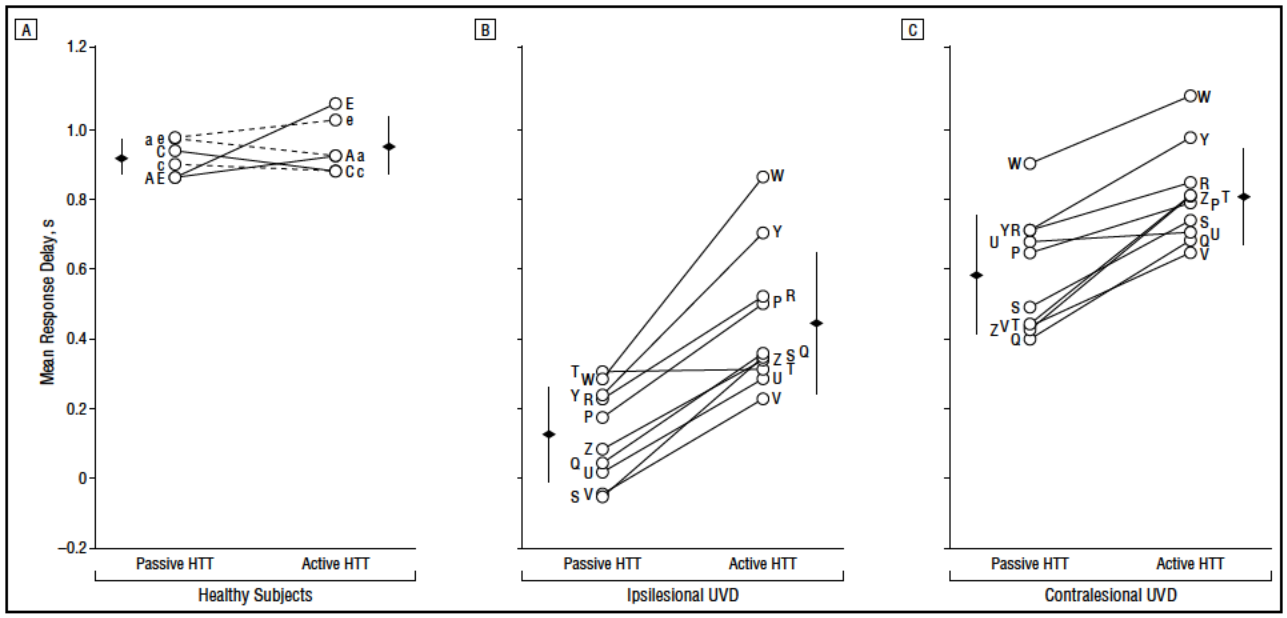

Figure 8. The vestibulo-ocular reflex (VOR) gains for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For healthy subjects without UVD, responses to active and passive HTT were not significantly different from each other or from 1.00 ($P>.05$ for all). In B, active HTT ipsilesional VOR gain is significantly ($P<.001$) higher than for passive HTT. In C, contralesional UVD VOR gain is also significantly ($P=.004$) closer to normal for active than for passive HTT. Bars represent mean±SD.

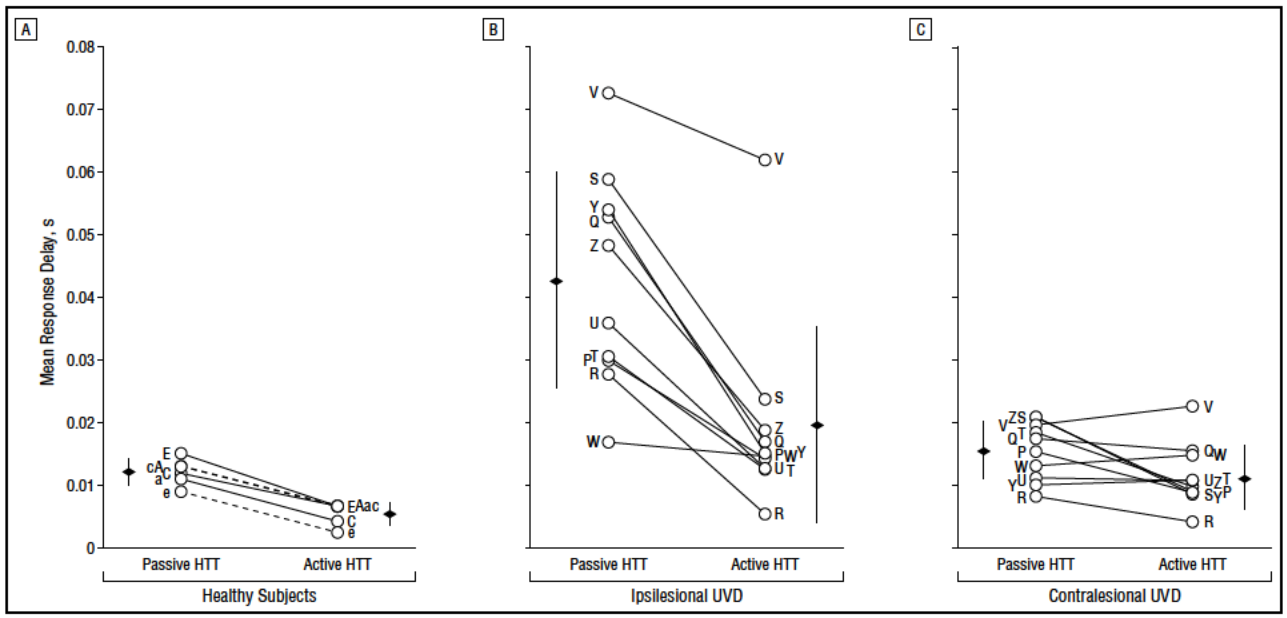

Figure 9. The response delays for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For subjects without UVD, the response delay was shorter for active than for passive HTT ($P=.006$. In B, for ipsilesional stimuli, response delay was significantly shorter for active HTT than for passive HTT ($P=.006$). In C, the response delay for contralesional stimuli was not significantly different ($P=.07$) between passive and active HTT. Bars represent mean±SD.

thrusts and 0.58±0.17 for contralesional head thrusts were significantly different, and the side with the lesion was clearly identified by pHTT in each patient, consistent with previous studies.[6,24] For HART, the VOR gain in subjects without UVD was essentially perfect, as one would expect. Surprisingly, however, although some subjects with UVD showed obvious asymmetry of VOR half-cycle gains on HART, there was no significant difference between the ipsilesional and contralesional VOR gains over the population with UVD ($P=.69$).

Other features of the response to the actively generated HART stimulus did provide an indication of the side of unilateral vestibular hypofunction. The occurrence of rapid, gaze-correcting, saccadic eye movements during head movements toward the side of the lesion, when they occur, is a reliable indication of the side of UVD. A bias velocity in the eye movement response during the HART stimulus (a direct current shift in the eye velocity trace toward the side of the lesion) pro-

vides another such indication. Head autorotation is, therefore, a useful paradigm, but comparison of VOR gains from half-cycle analysis of HART responses is not reliable for the identification of the side of UVD.

MECHANISMS FOR IMPROVED RESPONSES TO ACTIVE HEAD ROTATIONS

There are 2 main differences between HART and pHTT stimuli that might explain the improvement in VOR gain of patients with UVD for ipsilesional head movements during HART. First, the peak velocities and accelerations generated by the examiner in pHTT are greater than those that some patients are able to generate during HART. This constraint on stimulus intensity may limit HART's ability to discern unilateral weakness in such patients. In contrast, the stimulus used for pHTT is generated by the examiner and reaches a peak acceleration of 3000°/s^2 and a peak velocity of 250°/s. Although these head movements have low amplitude in displacement (10°-15°) and are well tolerated by patients, they are sufficient to elicit excitation-inhibition asymmetry and, thus, selectively probe the function of the excited canal.

A second difference is that the pHTT is passive, transient, and unpredictable, whereas the HART is a self-generated, sinusoidal, predictable stimulus. Self-generated, sinusoidal, steady-state rotations could allow for nonvestibular eye movement systems (eg, predictive eye movements, efference copy, and visual-following mechanisms) to contribute to the response. The existence of predictive mechanisms in augmenting vestibular responses is suggested by measures of visual acuity during head movement. Visual acuity is improved during active compared with passive head movements.[25,26] To further investigate these potential effects of self-generated stimuli, we asked patients to perform the HTT themselves, aiming to mimic the speed and amplitude of the operator-delivered head thrusts. Although patients had some difficulty reaching the top speed of operator-delivered stimuli (about 300°/s), they did reach speeds of approximately 200°/s, and we compared responses for active and passive head thrusts of similar peak velocity and acceleration. In subjects without UVD, the VOR gain was already insignificantly different from 1.00 for pHTT, so no significant change was observed for aHTT. For subjects with UVD, however, there was a marked improvement in VOR gain during active head movements, and the boost in VOR gain during active thrusts occurred from the onset of the head thrust, during the initial 20 to 40 milliseconds. The only 2 oculomotor systems with a latency of less than 40 milliseconds are the direct VOR and predictive eye movements. Saccades, the cervico-ocular reflex, and visual-following mechanisms, such as smooth pursuit, all have latencies that are 70 to 150 milliseconds.[27] From previous studies,[6,10] it has been shown that the 3-neuron VOR arc generates the response to pHTT, so the boost in gain during aHTT is, therefore, likely to be a result of predictive eye movements.

To study the effect of prediction, we measured the time between the onset of head rotation and the onset of eye rotation for active and passive head thrusts. We used manually applied passive head thrusts to approximate the usual clinical application of pHTT and the movements our subjects made during aHTT. Manual thrusts do not have an onset sharp enough or an acceleration constant enough to precisely measure VOR latency (estimated at 8.6 milliseconds in subjects without UVD by Collewijn and co-workers[28,29] using a torque-applying helmet in an HTT-type paradigm). To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset times (at which head and eye velocities cross set thresholds) and compute a response delay. This response delay probably does not precisely equal the true synaptic and axonal conduction delay of the VOR, because the finite rate of change of acceleration for manually applied stimuli and responses reduces the precision with which onset times for head and eye movements can be measured. However, this response delay provides a useful measure for comparison of responses to passive and active head thrusts.

For subjects without UVD, the response delay was shorter for active than for passive head thrusts. For subjects with UVD, the response delay during ipsilesional head thrusts was significantly longer for passive thrusts than for active thrusts. A similarly prolonged response delay for passive ipsilesional head rotations has been reported by Tabak et al.[8] The apparent reduction in the response delay to aHTT might be further evidence of preprogrammed eye rotation. Alternatively, some of the apparent difference in response delay may be due to a difference in VOR gain for the first 40 milliseconds of the response. The initial low velocity eye rotation makes it difficult to precisely define the onset of the eye rotation, and could give the appearance of a delayed onset.

TIMING OF CORRECTIVE EYE MOVEMENTS

Further evidence for preprogramming of eye rotations during active head thrusts was the reduced latency of rapid corrective eye movements during active compared with passive head thrusts. To compensate for a deficient VOR, patients with UVD used rapid eye movements to correct accrued gaze error and reacquire visual fixation of the target. During active head thrusts, these corrective movements occurred as early as 60 milliseconds after the onset of the head rotation, significantly earlier than during passive head thrusts (**Figure 10**). Under normal circumstances, the latency of voluntary true saccadic eye movements is 200 milliseconds.[25] This latency can be reduced to 100 milliseconds if the subject is trained in a specific task (so-called express saccades[30]). Tian et al[25] have shown that under pHTT conditions, these rapid eye movements have latencies shorter than those of express saccades. Our data indicate that these latencies can be reduced even further under conditions of active head movement.

CONCLUSIONS

Gain asymmetries on the pHTT reliably detected the presence of

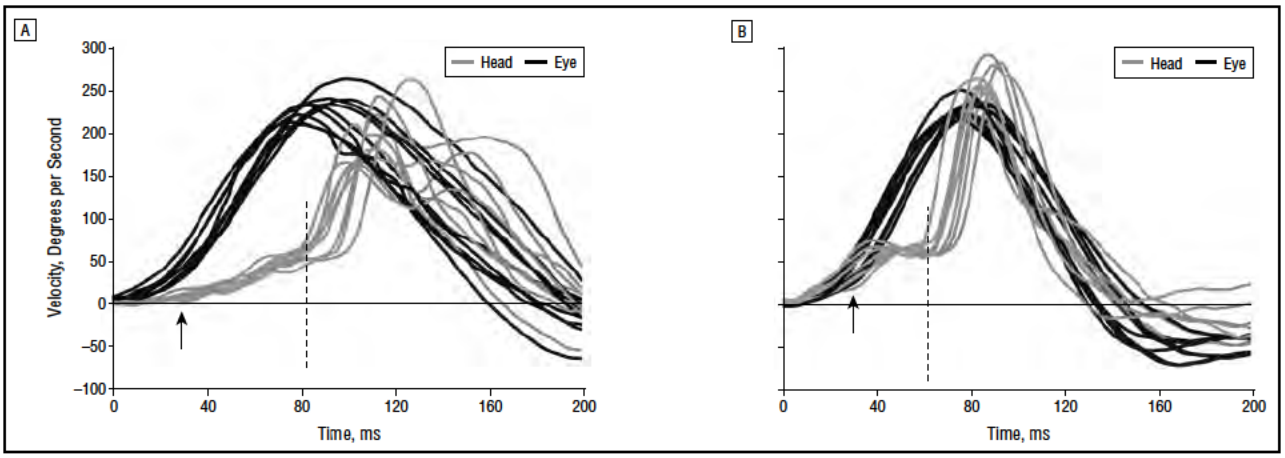

Figure 10. Corrective rapid eye movements during passive (A) and active (B) ipsilesional head thrust testing (HTT) in a 48-year-old subject with a unilateral vestibular deficiency 120 months after left labyrinthectomy (subject R shown in Figures 4, 8, and 9). Head and eye (inverted) velocities are shown. The response to active HTT has a higher initial vestibulo-ocular reflex gain (arrows) and earlier rapid compensatory eye movements (dashed lines).

known unilateral vestibular lesions in all patients with UVD tested, whereas the half-cycle gains measured from HART did not. Relative to pHTT, HART gains overestimated ipsilesional vestibular function and were, therefore, a less accurate indicator of unilateral hypofunction.

For subjects with UVD who are unable to generate high-acceleration sinusoidal head movements, HART may underestimate hypofunction simply because it relies on stimulus accelerations inadequate to silence inhibitory pathways arising in the intact ear. In contrast, the high-acceleration movements of pHTT reliably identify response asymmetry by more selectively probing the function of the excited canal. However, even for head movements of similar acceleration and time course, the measured VOR during self-generated head rotations (aHTT) has significantly higher gain and shorter response delay than does the VOR to passive unpredictable stimuli (pHTT).

Subjects with UVD may use a variety of strategies for improving visual fixation, including preprogrammed eye movements designed to augment the deficient VOR and to compensate for a planned or anticipated head movement. Tests using passive high-acceleration head thrusts delivered unpredictably in time and direction should, therefore, be more sensitive for discerning VOR hypofunction than tests using active and/or predictable stimuli.

Accepted for publication February 18, 2002.

This study was supported by the Garnett Passe and Rodney Williams Memorial Foundation, Parkville, Australia (Dr Cremer); the Royal Australasian College of Physicians, Sydney, New South Wales (Dr Cremer); grants K23 DC00196 (Dr Carey) and R01 DC02390 (Dr Minor) from the National Institutes of Health, Bethesda, Md; and a Johns Hopkins Clinician-Scientist Award (Dr Carey).

We thank Grace C. Y. Peng, PhD, David S. Zee, MD, Adrian G. Lasker, MS, and Dale C. Roberts, MS, for their assistance with data acquisition; and John K. Niparko, MD, and Howard W. Francis, MD, for their referral of subjects.

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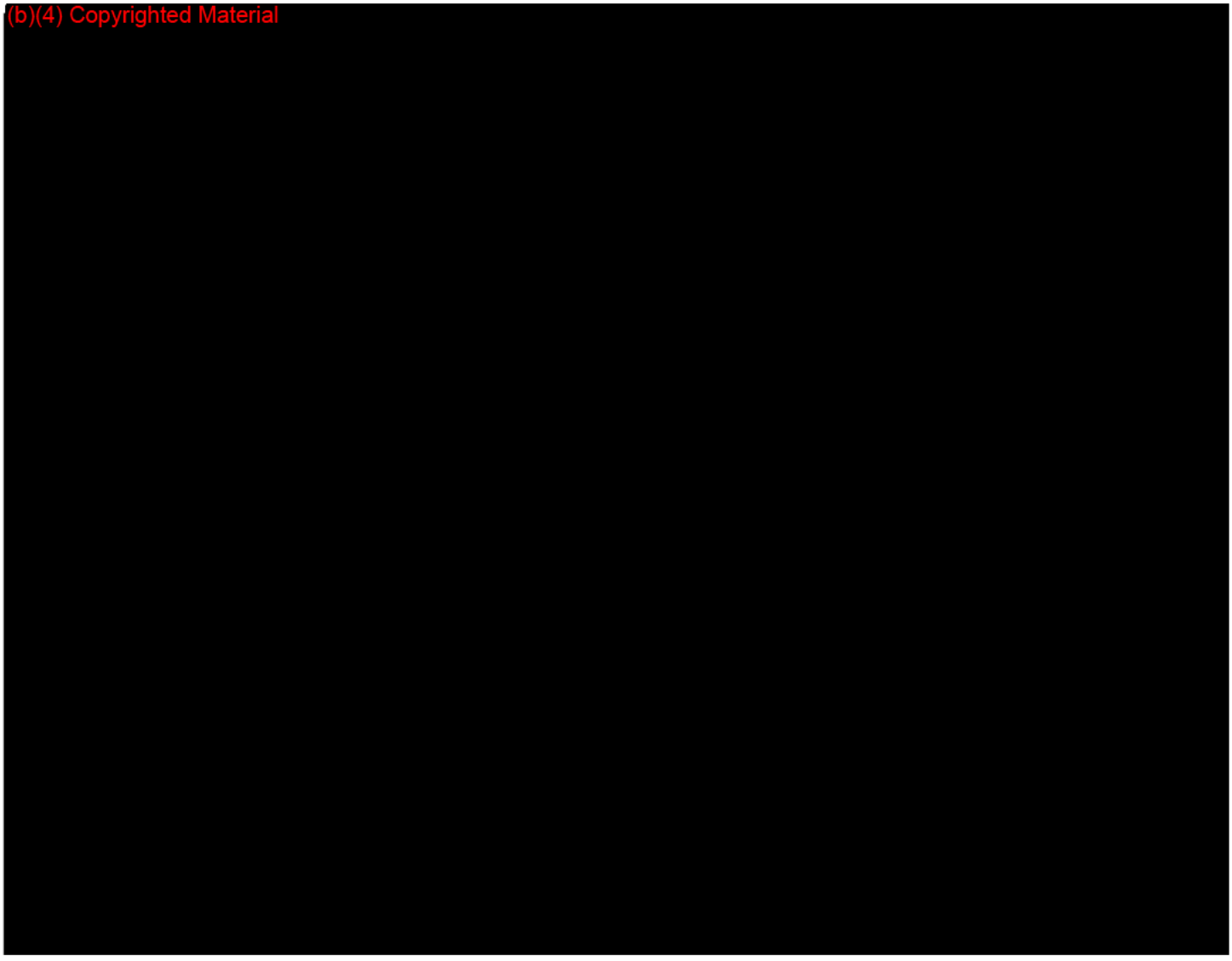
A Clinical Sign of Canal Paresis

G. Michael Halmagyi, MB, BS, Ian S. Curthoys, PhD



C. Kennard & R.J. Leigh (Eds.)
Progress in Brain Research, Vol. 171
ISSN 0079 6123

CHAPTER 3.6

Impulsive testing of semicircular canal function

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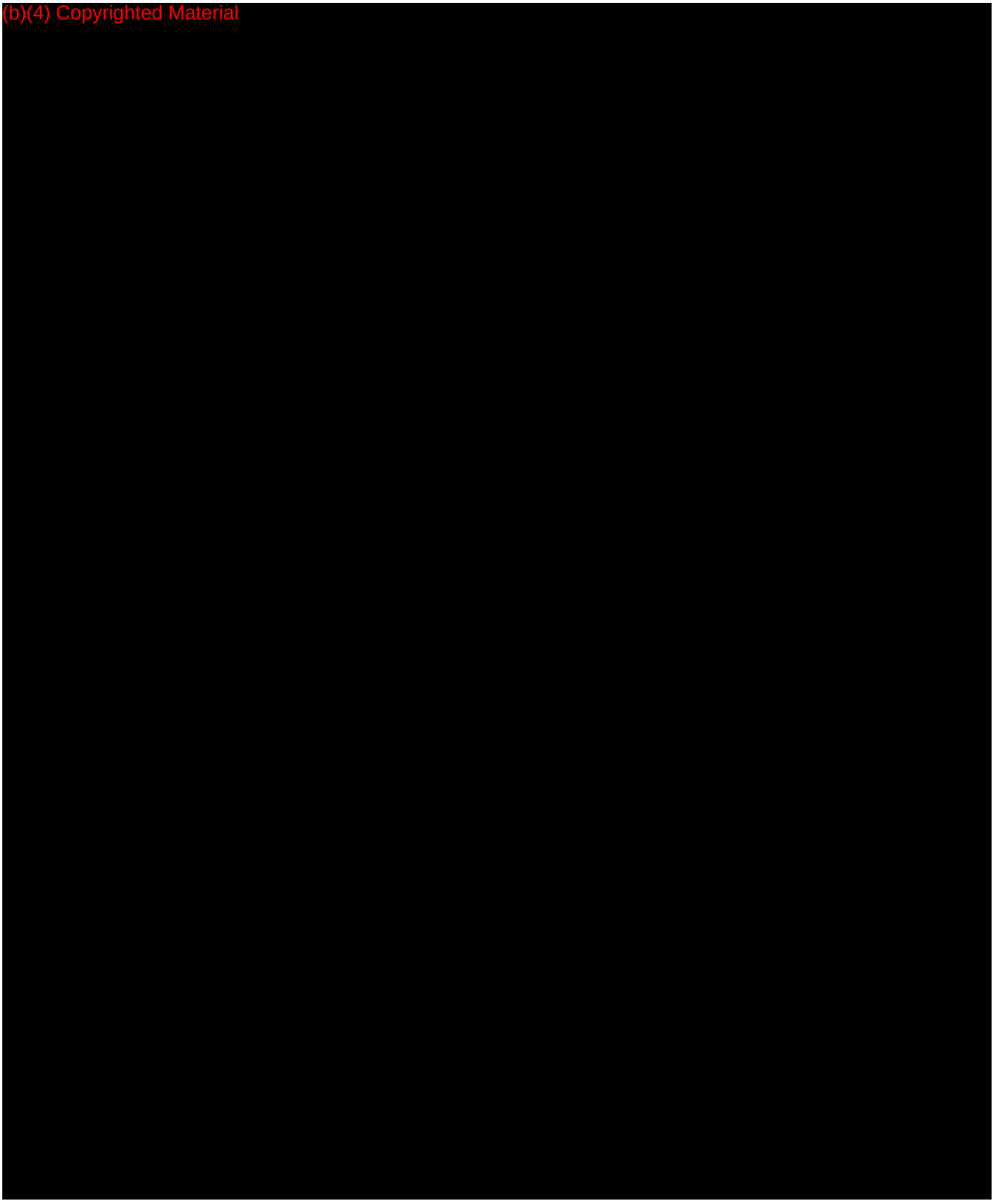
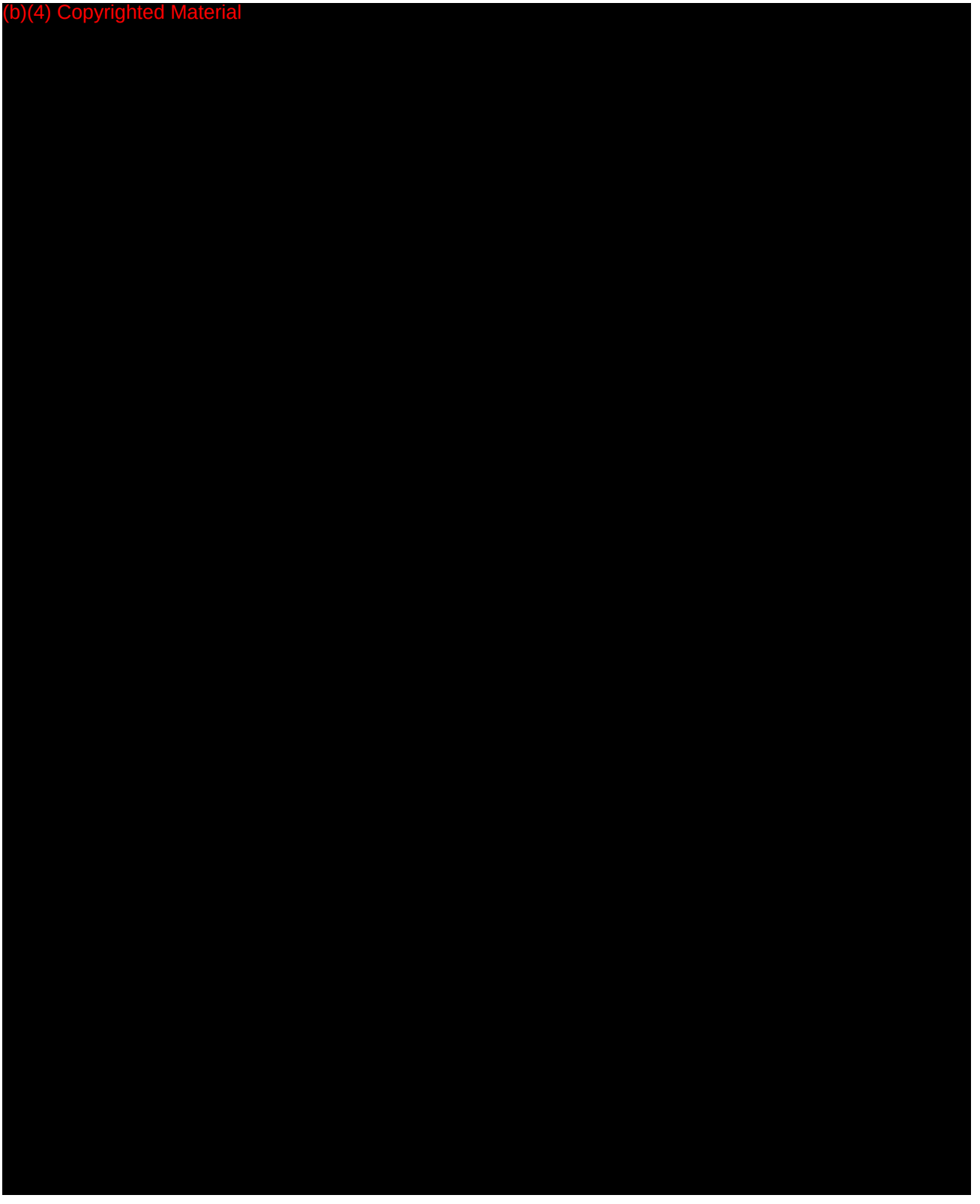
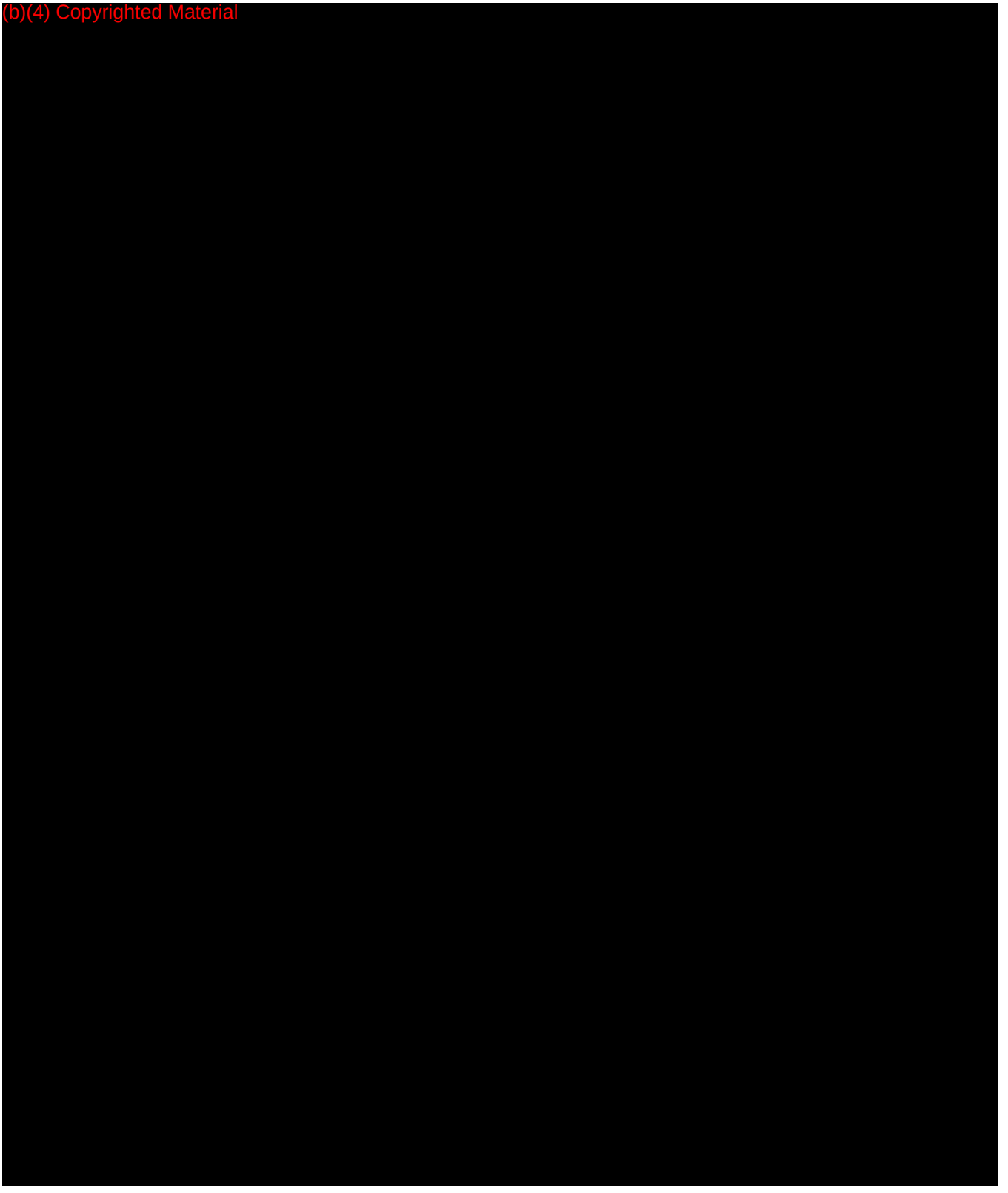
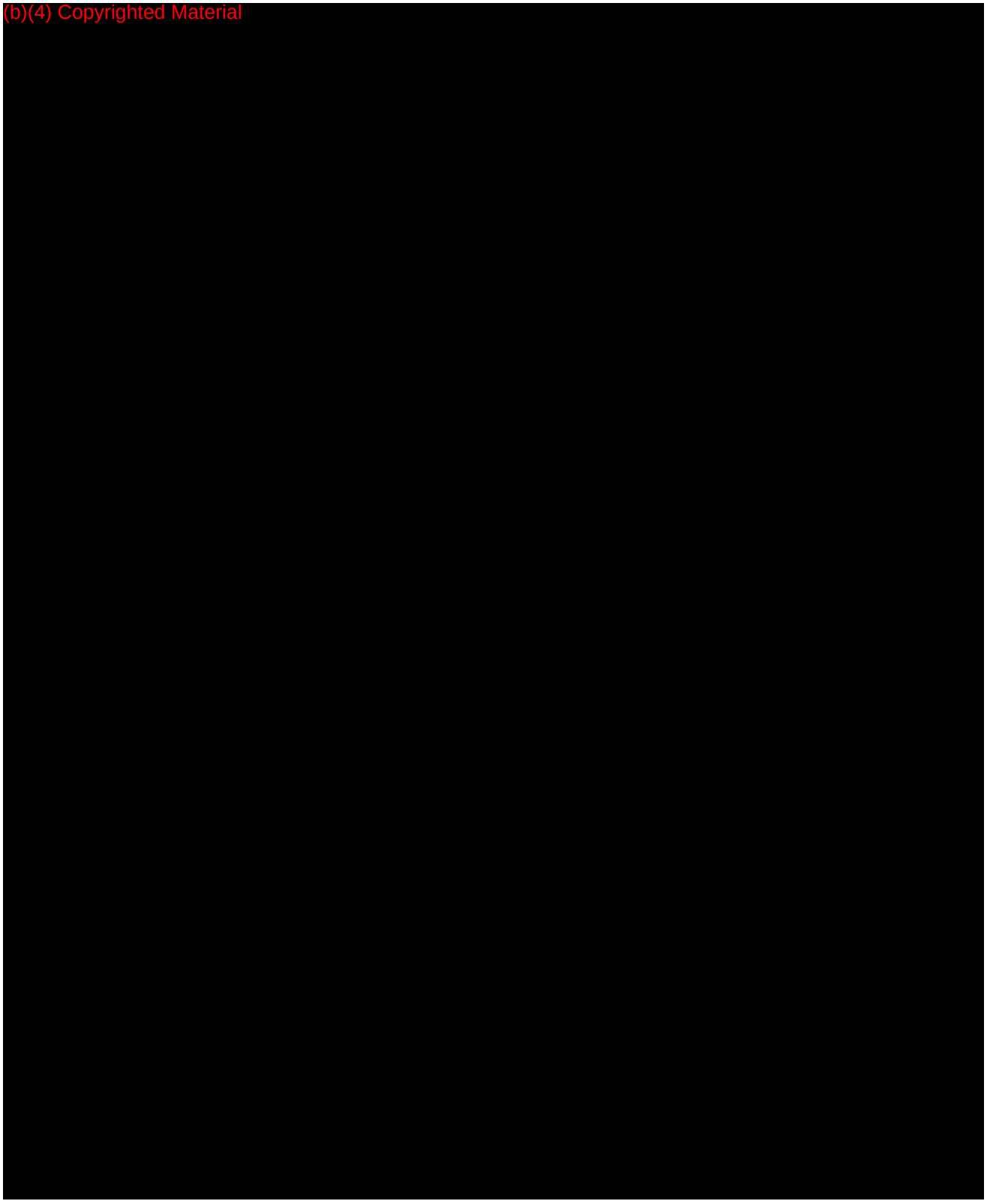
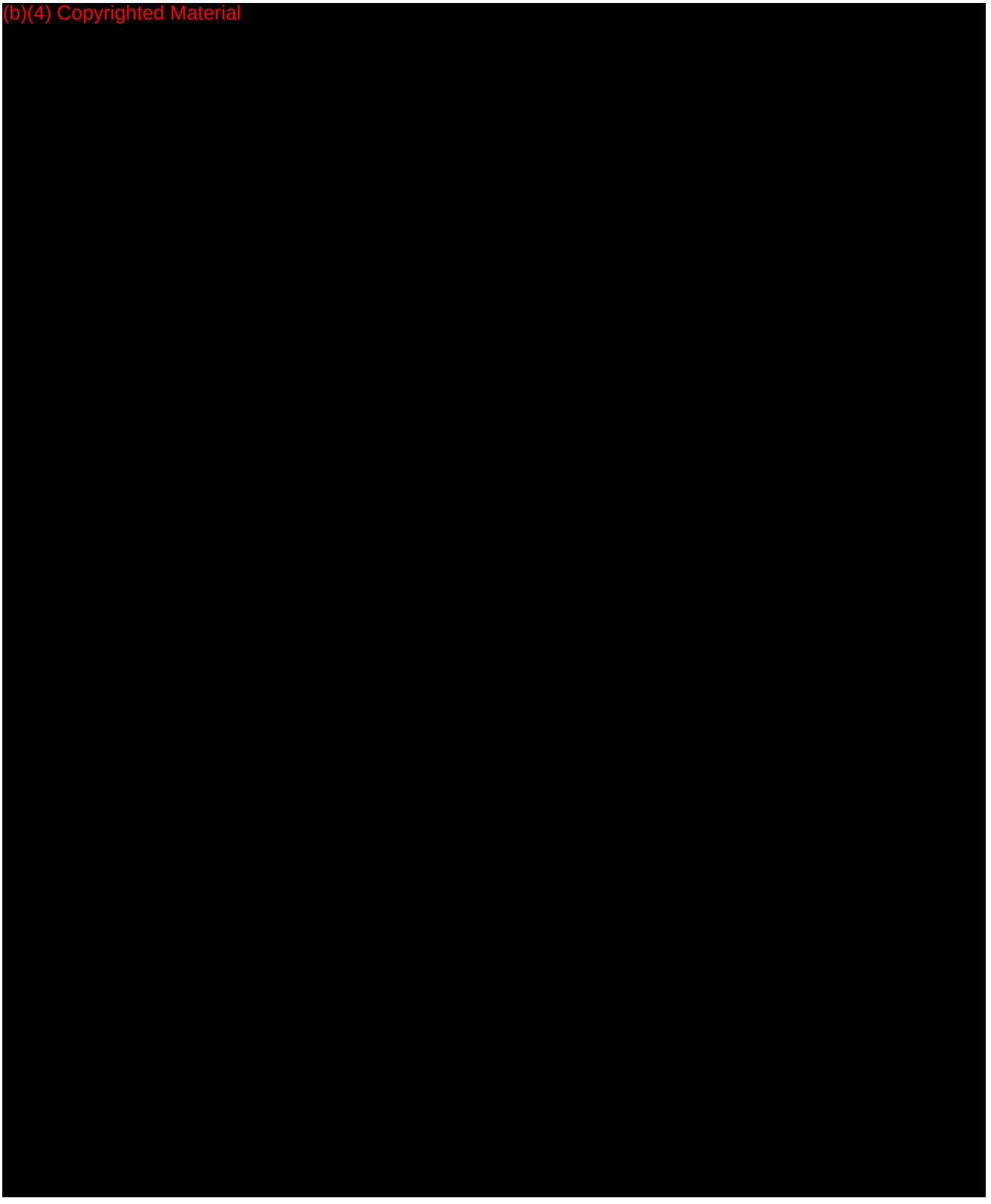
DOI: 10.1016/S0079 6123(08)00625 0

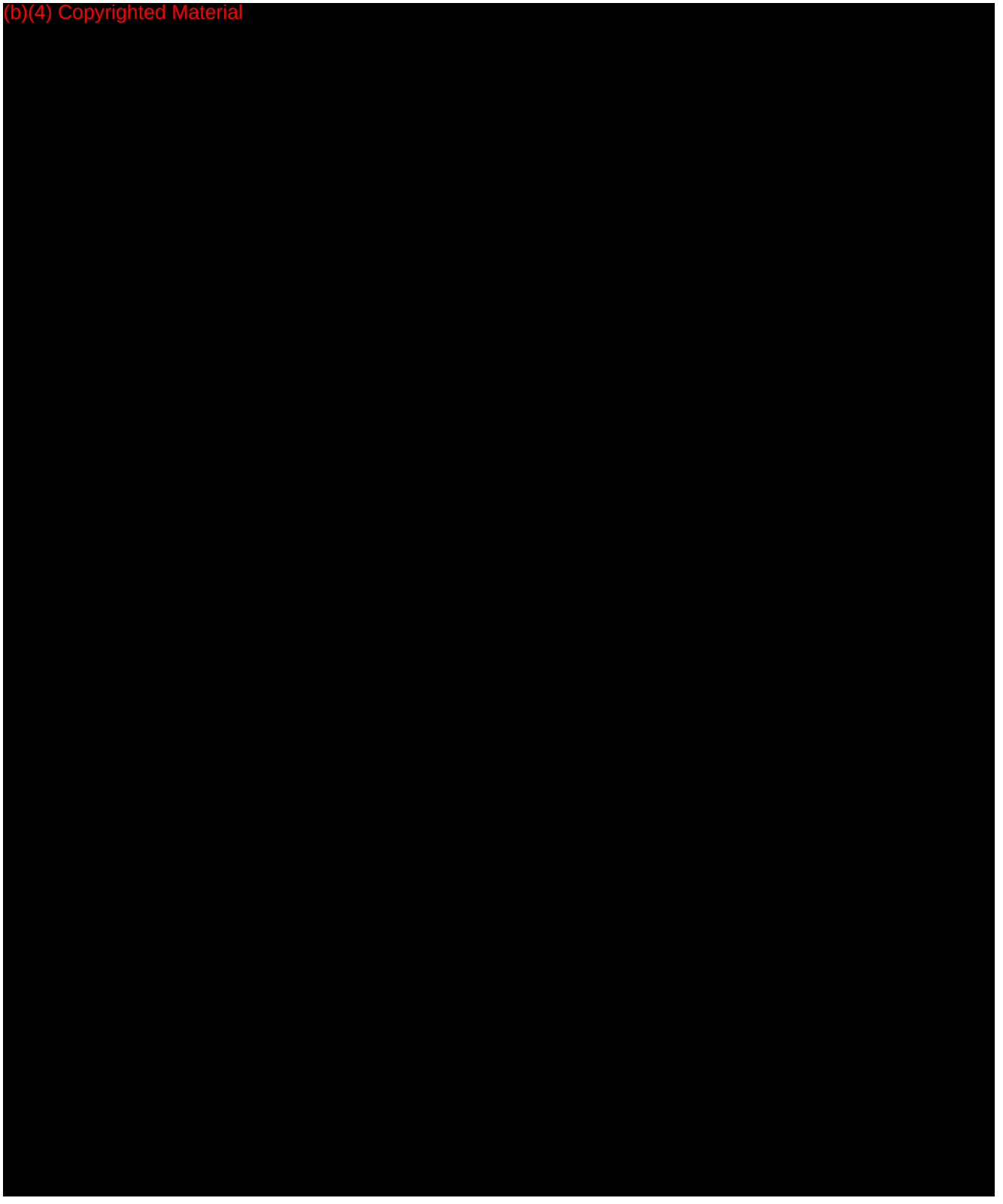


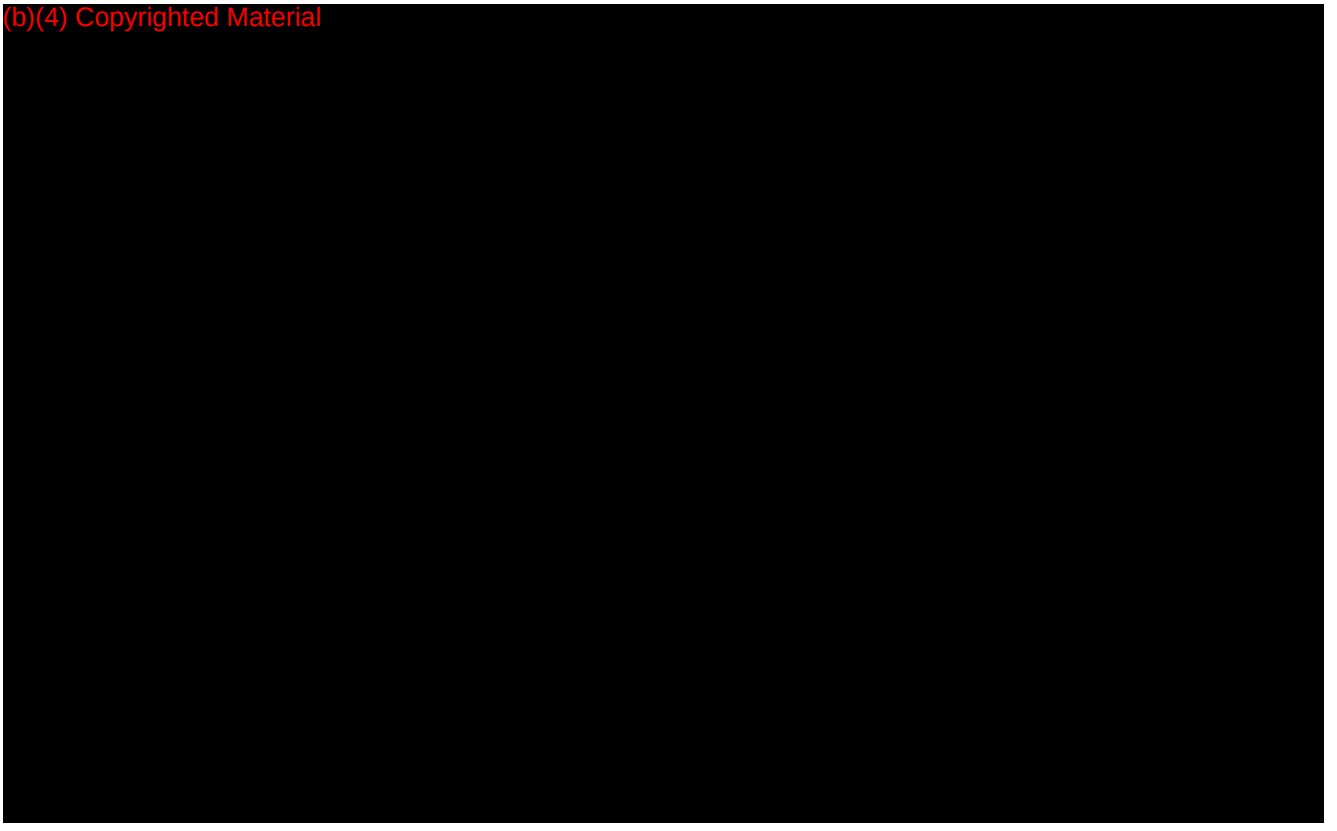












NEUROLOGY

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

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Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

The video head impulse test

Diagnostic accuracy in peripheral vestibulopathy

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ABSTRACT

Background: The head impulse test (HIT) is a useful bedside test to identify peripheral vestibular deficits. However, such a deficit of the vestibulo-ocular reflex (VOR) may not be diagnosed because corrective saccades cannot always be detected by simple observation. The scleral search coil technique is the gold standard for HIT measurements, but it is not practical for routine testing or for acute patients, because they are required to wear an uncomfortable contact lens.

Objective: To develop an easy-to-use video HIT system (vHIT) as a clinical tool for identifying peripheral vestibular deficits. To validate the diagnostic accuracy of vHIT by simultaneous measures with video and search coil recordings across healthy subjects and patients with a wide range of previously identified peripheral vestibular deficits.

Methods: Horizontal HIT was recorded simultaneously with vHIT (250 Hz) and search coils (1,000 Hz) in 8 normal subjects, 6 patients with vestibular neuritis, 1 patient after unilateral intratympanic gentamicin, and 1 patient with bilateral gentamicin vestibulotoxicity.

Results: Simultaneous video and search coil recordings of eye movements were closely comparable (average concordance correlation coefficient r_c = 0.930). Mean VOR gains measured with search coils and video were not significantly different in normal (p = 0.107) and patients (p = 0.073). With these groups, the sensitivity and specificity of both the reference and index test were 1.0 (95% confidence interval 0.69–1.0). vHIT measures detected both overt and covert saccades as accurately as coils.

Conclusions: The video head impulse test is equivalent to search coils in identifying peripheral vestibular deficits but easier to use in clinics, even in patients with acute vestibular neuritis.

Neurology® 2009;73:1134–1141

GLOSSARY

BVL = bilateral vestibular loss; **HIT** = head impulse test; **IMU** = inertial measurement unit; **ITG** = intratympanic gentamicin; **vHIT** = video head impulse test; **VN** = vestibular neuritis; **VOR** = vestibulo-ocular reflex.

The head impulse test (HIT) is a useful bedside examination to identify a peripheral vestibular deficit for example in patients with vestibular neuritis (VN).[1-4] The clinician briskly rotates the patient's head to detect "overt" catch-up saccades after head rotation as a sign of semicircular canal paresis. "Covert" saccades are saccades that occur during the head rotation that may be imperceptible to the naked eye and hence confound the diagnosis.[5,6] In patients with acute VN, spontaneous nystagmus also interferes with assessment of bedside HIT.

Up to now, the scleral search coil technique has been the gold standard for HIT measurements.[7-9] It quantifies the VOR deficit and shows the associated pattern of overt and covert catch-up saccades in vestibular deficient patients.[6,10] However, search coil measurements require the subject to wear an uncomfortable contact lens, are time intensive, are expensive, and are not practical for acute patients.

The goal of the study was to develop an easy-to-use high-speed video HIT system[11] (see video on the *Neurology*® Web site at www.neurology.org) as a clinical tool to identify a periph-

Supplemental data at www.neurology.org

*These co–first authors contributed equally.

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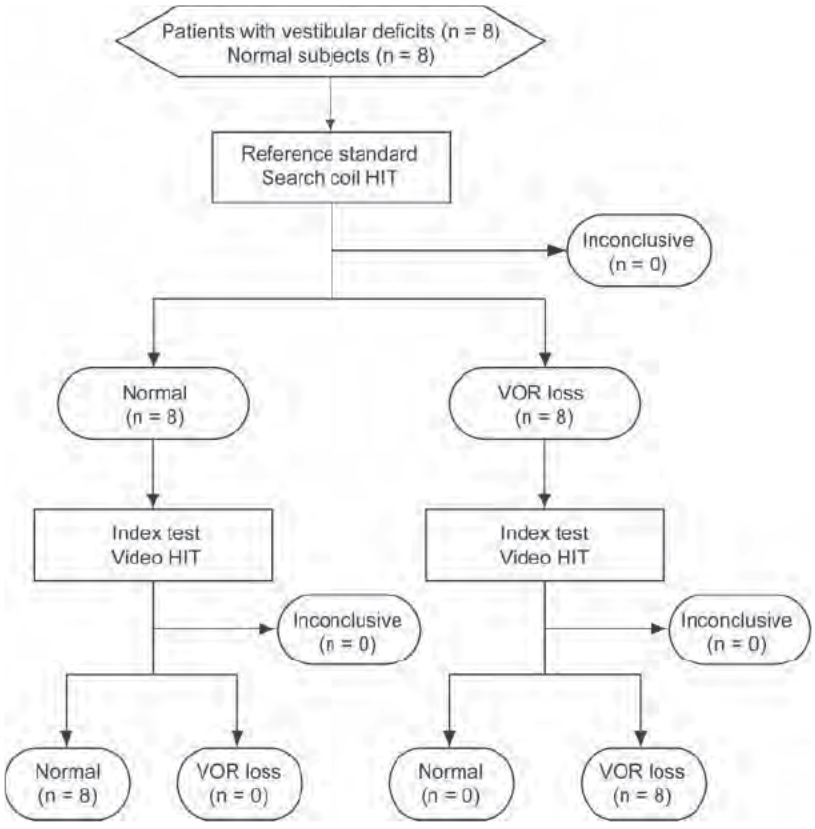
Disclosure: Author disclosures are provided at the end of the article.

eral vestibular deficit. To validate the diagnostic accuracy of our video HIT (vHIT) system, we compared the measures from video recordings with simultaneous measures from scleral search coil recordings of the same eye during head impulses in healthy subjects and patients with a wide range of previously independently identified vestibular deficits.

METHODS **Design.** The study was a prospective, cross-sectional comparison of the index test (vHIT) to the reference standard (HIT measured by scleral search coils) in patients with prior, independently identified vestibular deficits due to unilateral vestibular neuritis, intratympanic gentamicin, or systemic gentamicin and healthy asymptomatic control subjects (figure 1). Patients with a broad range of vestibular deficits were enrolled because we wished to establish how well each test identified vestibular deficits of varying severity.

Subjects. Sixteen subjects were recorded simultaneously with video-oculography and scleral search coils. Six patients with VN (mean 52 years, age range 38–59 years, 1 female) showed evidence of enduring unilateral loss of vestibular function after an illness with acute onset of prolonged rotational vertigo and postural imbalance associated with spontaneous nystagmus, nausea, or vomiting that fulfilled the clinical criteria of VN.[12] One patient with Ménière disease (53 years, female) with a unilateral vestibular deficit due to intratympanic gentamicin injection and a patient with bilateral vestibular loss due to systemic gentamicin vestibulotoxicity (72 years, male) were also tested. Eight healthy subjects without any history, symptoms, or clinical signs of vestibular disease (mean 35 years, age range 25–66 years, 2 females) served as controls. Diagnosis of a peripheral vestibular deficit was confirmed in all patients by bithermal caloric testing with water irrigation at 30°C and 44°C, resulting in a canal paresis factor greater than 25%.[13] Patients were tested between 5 months and 27 years after onset of symptoms. Two additional patients with acute VN (29 and 32 years, both female) were recorded within 36 hours after onset with video-oculography alone. Eligible patients were recruited at the Hearing and Balance Clinic, Royal Prince Alfred Hospital, Sydney, Australia. No potential subject was excluded. All subjects and patients were tested between August and October 2008.

Figure 1 **Flow chart for the comparison of video and search coil measures of head impulses**



HIT = head impulse test; VOR = vestibulo-ocular reflex.

Standard protocol approvals and patient consents. Written informed consent was obtained from all subjects. Written consent to disclose has been obtained from any recognizable persons in the published photograph and video. The protocol was approved by the Sydney South West Area Health Service Ethics Committee in accordance with the Declaration of Helsinki.

Experimental procedure. Subjects were instructed to fixate a laser dot on a screen at 91 cm distance in dim light. Approximately 50 horizontal head impulses to each side were manually applied with unpredictable timing and direction. Peak head velocity of the impulses was gradually increased from 50° to 250°/second (acceleration 750°–5,000°/second^2, amplitude 5°–20°) with the aid of visual feedback of head velocity for the experimenter.[5,6] The same eye was recorded simultaneously with video-oculography and scleral search coils (figure e-1). Two data sets were obtained for each recording session to show the reliability of the calculated gains and concordance of the video and search coil methods. All recordings were performed by the same team of 3 coauthors. Head impulses were always delivered by the same experimenter, unless acting as a subject. The experimenters were unmasked as to whether they were testing a patient or a healthy subject. All experimenters are graduates and have at least 5 years' experience in vestibular research. There were no adverse events from performing the tests.

Video-oculography. Right eye position was recorded at 250 Hz with a small, lightweight, high-speed digital (IEEE 1394a) video camera (Firefly MV, Point Grey Research Inc., Vancouver, British Columbia, Canada). The camera was mounted on a very lightweight motorcycle glasses frame with an elastic strap that locked comfortably onto the bridge of the nose and around the eye sockets to minimize slippage of the camera relative to the head. The image of the eye was reflected from a hot mirror to the camera. The eye was illuminated by 2 infrared light–emitting diodes (TSUS502, Vishay Intertechnology, Malvern, PA) run at 20 mA to keep infrared radiation far below exposure risk levels.[14] Head velocity was measured by a miniature 6-degrees-of-freedom inertial measurement unit (IMU) assembled from 2 dual-axis gyroscopes (IDG-300 InvenSense, Santa Clara, CA) and a 3-axis linear accelerometer (ADXL330, Analog Devices, Norwood, MA). The camera, hot mirror, and IMU were rigidly mounted onto the spectacle frame. The small mass of the system (approximately 60 g) minimized inertia during head rotation and so minimized slippage of the glasses. Eye position was calibrated in vivo with projected targets from a glasses-mounted laser. Video images were analyzed online to calculate eye position using a pupil detection method based on a center-of-gravity algorithm[15] written in LabVIEW (National Instruments, Austin,

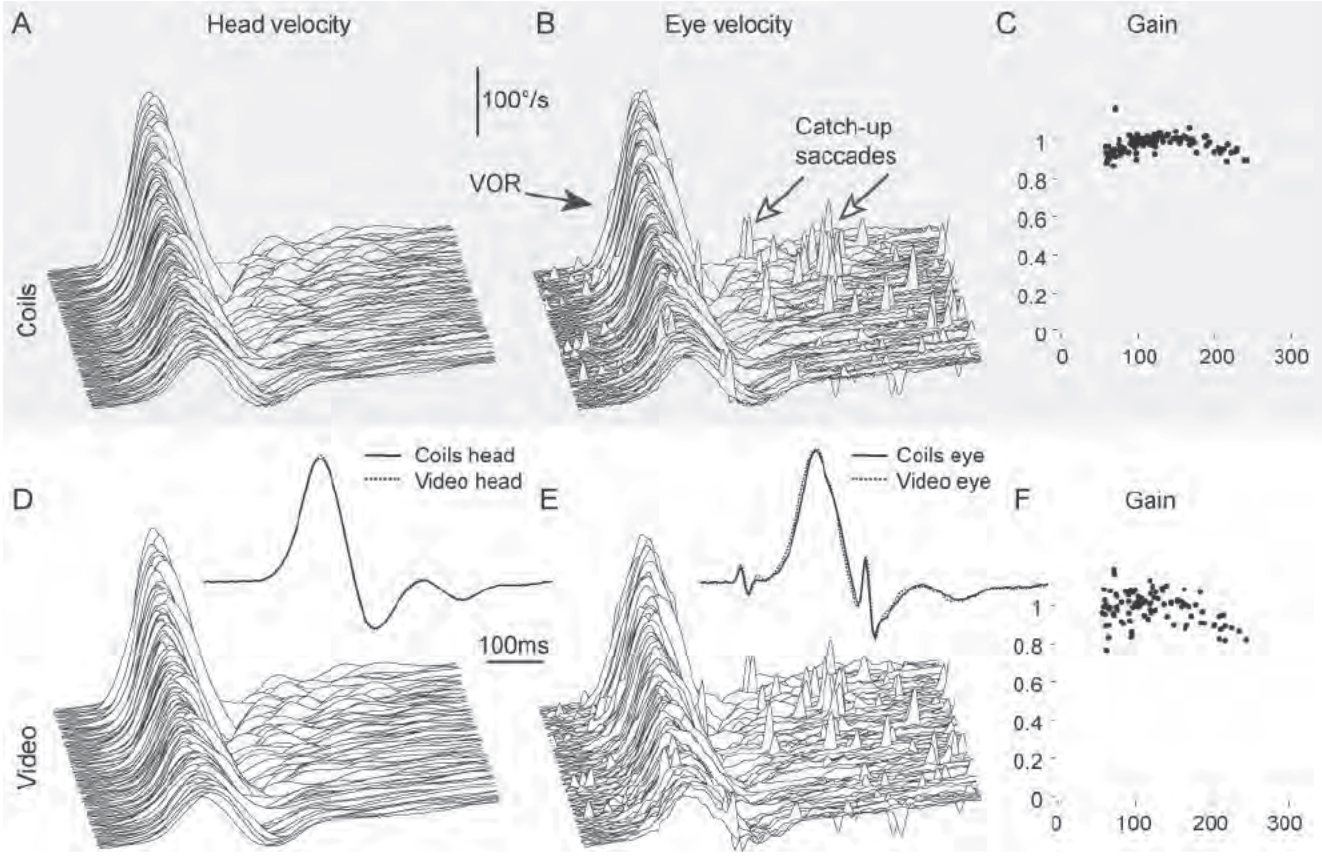
TX). Eye velocity was obtained from a 2-point differentiator and low-pass filtered (0- to 30-Hz bandwidth).

Scleral search coil recording. Right eye and head position were recorded with the scleral search coil technique in a 1.9 × 1.9 × 1.9-m magnetic coil frame (CNC Engineering, Seattle, WA).[2,9] Dual search coils (Skalar, Delft, The Netherlands) were precalibrated in vitro on a gimbal. The eye coil was inserted after topical anesthesia with Alcaine 0.5% eyedrops (Alcon Laboratories Australia Pty. Ltd., Frenchs Forest, Australia). The head coil was attached to a dental impression tray. Three-dimensional head and gaze position signals were sampled at 1,000 Hz, digitized with 16-bit precision, and low-pass filtered (0- to 100-Hz bandwidth). Three-dimensional rotation vectors and angular velocity vectors of head, gaze, and eye were derived from coil voltages.[16]

Data analysis. Offline analysis of the experimental data were automated with customized LabVIEW software. To synchronize the video and search coil measurements, a square wave signal produced by a signal generator was acquired by each system together with eye and head velocity measurements. Data from the 2 systems were then synchronized by aligning the square wave signals. Head impulses were automatically selected and aligned to peak head acceleration. Trials with blinks and outliers were automatically excluded, based on an envelope around the expected eye velocity response. Velocity gain of the horizontal VOR[7] was calculated for both recording methods as the ratio of mean eye velocity over mean head velocity during a 40-msec window centered at peak head acceleration. Data from both recording methods was processed simultaneously with the same automated algorithms to exclude any analysis bias. Invalid head impulses (e.g., with blinks) were excluded from both data sets, resulting in mirror-symmetric data. Both data sets were analyzed in all simultaneously recorded patients (no missing data sets). The criterion for a normal VOR velocity gain was that it should be 0.68 or greater, based on HIT data from 12 previously published healthy asymptomatic subjects[6,10] in which the mean HIT velocity gain measured by search coils with identical apparatus and procedures to those used here was 0.81 ± 0.068 SD, so that the mean ± 2 SD units incorporates 95% of the population and yields a lower cutoff of 0.68.

Statistical analysis. The results of the study are reported in accordance with the Standards for Reporting of Diagnostic Accuracy Studies (STARD).[17,18] The concordance correlation coefficient[19] was used to index the similarity between video and search coil recordings for each impulse. A coefficient was calcu-

Figure 2 Simultaneous video and search coil recordings of a horizontal head impulse test in a normal subject



Simultaneous angular head velocity recordings of a search coil mounted on a dental impression tray (A) and a gyroscope mounted on the video glasses (D) during graded horizontal head impulses. The close similarity between the 2 recordings demonstrates minimal slippage of the video glasses relative to the head. Simultaneous angular eye velocity recordings of a scleral search coil (B) and high-speed video-oculography (E) of the same eye. Both recording techniques accurately record the vestibulo-ocular reflex (VOR) and detect even the smallest catch-up saccades. Normal VOR gains of individual head impulses are comparable with search coil recording (C) and video-oculography (F). Scleral search coil recording is sampled at 1,000 Hz (A and B), video-oculography is sampled at 250 Hz (D and E), and both are plotted on the same time scale. Head and eye velocity traces from individual impulses are stacked according to increasing peak head velocity. (Insets D and E) Simultaneous video and search coil recordings are shown superimposed to facilitate comparison of single head and eye velocity traces.

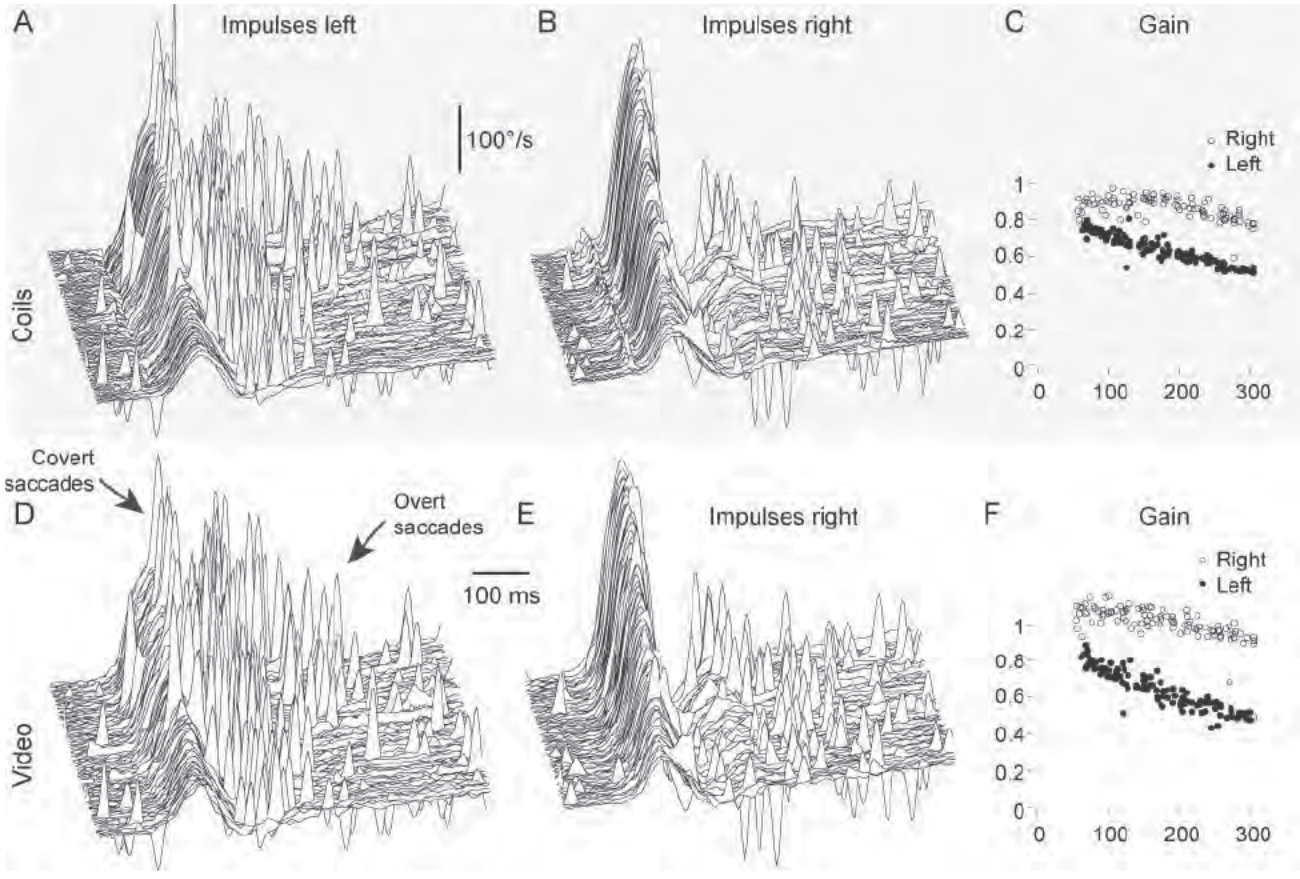
lated for each impulse and the values for an individual were averaged. Paired-sample t tests were used to test whether the VOR gains were different in video and search coil recordings. The Pearson product-moment correlation coefficient was used to determine test-retest reliability between the 2 separate test runs. Because of the small sample size, no subgroup analyses were performed by disease subtype or clinical examiner.

RESULTS **Head movement recording.** A close fit of the video glasses on the head is crucial for accurate eye movement recording during head impulses.[20] Simultaneous measurements of angular velocity from an inertial measurement unit (IMU) mounted on the glasses and from a search coil on a dental impression tray were virtually identical (figure 2, A and D), with an average concordance correlation coefficient[19] of $r_c = 0.999$.

Eye movement recording. Simultaneous measurements of video recording and scleral search coil of the same eye are shown in figure 2, B and E (normal subject), and figure 3, A, B, D, and E (patient with VN). Both recording techniques not only show VOR, but also record the smallest catch-up saccades (figures 2 and 3, arrows). Each method is highly repeatable, and the 2 methods are in very close agreement. The concordance correlations were calculated for every impulse in all subjects and patients: The mean r_c for each subject is shown in figure 4, and the average r_c of all subjects was 0.930.

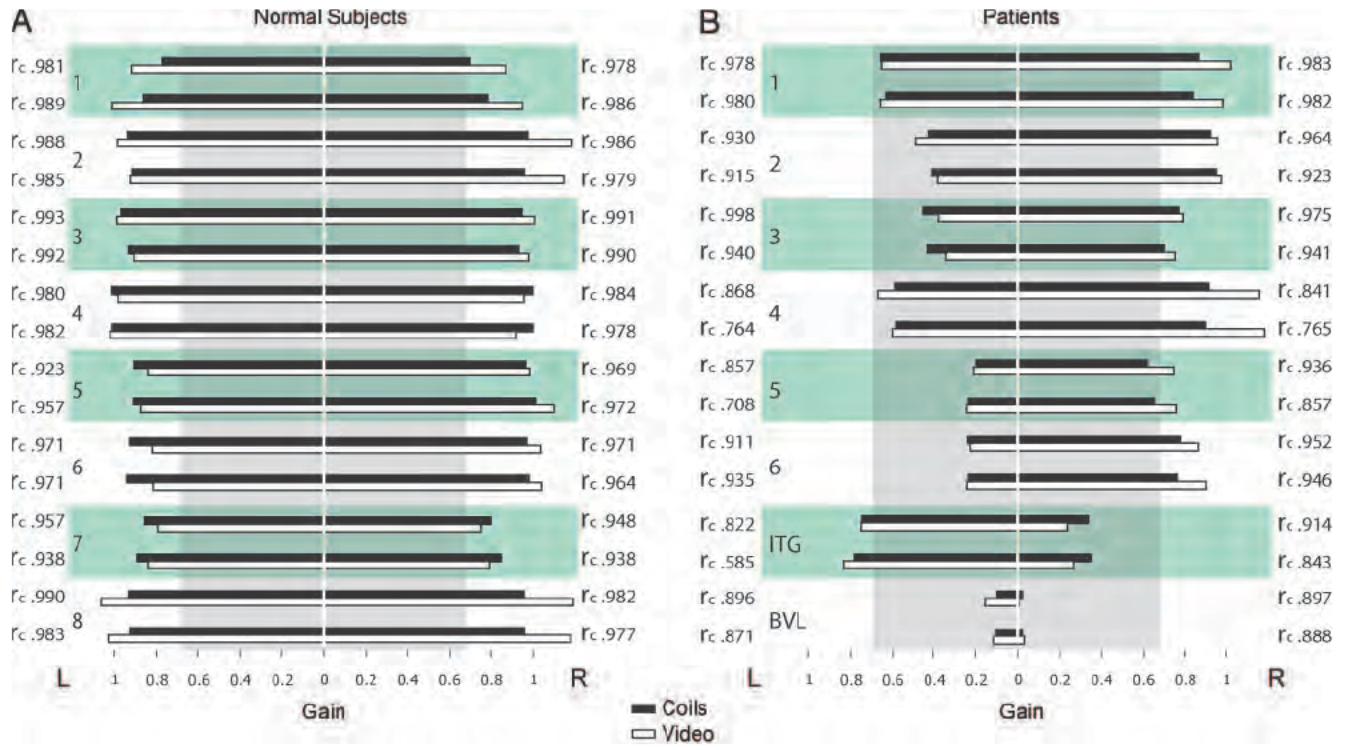
Detection of vestibular deficit. A VOR deficit was defined as being a HIT gain of less than 0.68. The reference standard, scleral search coils, correctly identified the VOR deficit in all patients, as did the video measures (figures 1 and 4). Thus, the sensitivity of the reference and index test were both 1.0 (95% confidence interval 0.69–1.0), and the specificity of both the reference and index test were 1.0 (0.69–1.0).[21] Using a paired t test, the difference between average VOR gain for search coils and video was not significantly different from zero for patients (mean difference = 0.040, n = 8, $t = 1.930$, $p = 0.073$) and for normal (mean difference = 0.043, n = 8, $t = 1.717$, $p = 0.107$). To test reproducibility, each

Figure 3 Simultaneous video and search coil recordings of a horizontal head impulse test in a patient after left vestibular neuritis



(A and D) Head impulses to the left (affected) side demonstrate the reduced vestibulo-ocular reflex (VOR) response. Both recording methods detect covert saccades during head rotation and overt saccades after head rotation (arrows). The pattern of catch-up saccades is identical for both recording methods. (B and E) Both recording methods demonstrate an almost normal VOR response to the healthy right side, with small overt saccades after head rotation. (C and F) Both recording methods clearly differentiate the reduced VOR gains of the left affected side (filled circles) from the right healthy side (empty circles). (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

Figure 4 VOR gain measures with search coils compared with video-oculography in normal subjects and patients with peripheral vestibular deficits



(A) The vestibulo-ocular reflex (VOR) gain for healthy subjects is almost identical for the 2 different methods of measurement. The 2 sets of data give highly reproducible values of VOR gain. (B) Video-oculography identifies the affected side in vestibular neuritis (1–6) and intratympanic gentamicin (ITG, 7) patients as reliably as search coil measurements. Patient 8 with bilateral vestibular loss (BVL) due to systemic gentamicin vestibulotoxicity demonstrates reproducibility of both methods at very low VOR gains. Bar graphs show the mean VOR gain measures with search coils (black bars) compared with video-oculography (white bars). For each individual, 2 data sets were recorded in the same session. Concordance correlation coefficients (r_c) index the similarity between search coil and video-oculography measurements. The vertical gray box indicates deficient VOR gain values (cutoff gain 0.68).

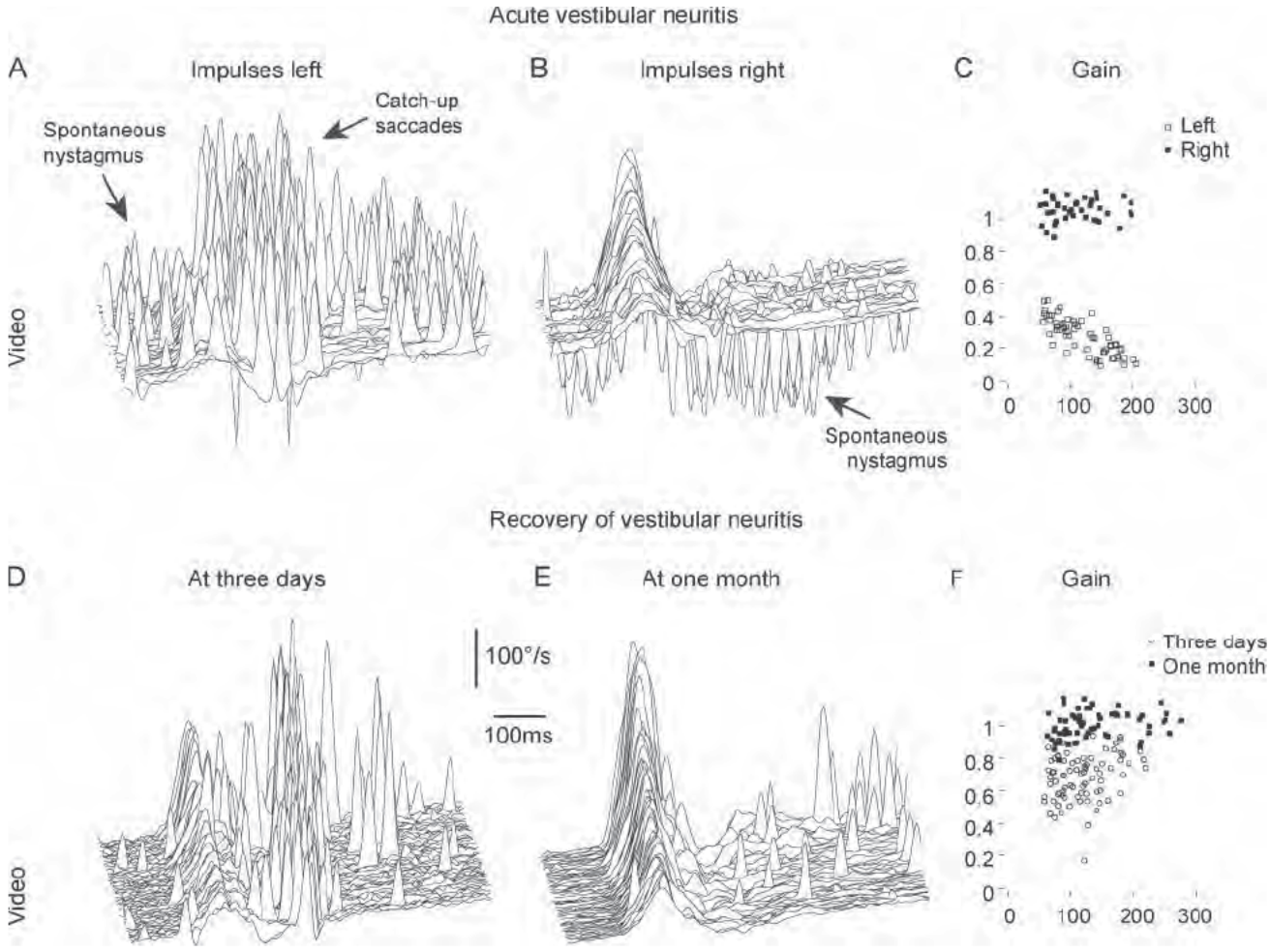
subject was given 2 separate test runs, and the test-retest reliability coefficient using a Pearson product-moment correlation was significant at 0.99 for search coils and 0.99 for the video measures. Both recording techniques readily detected covert saccades during head rotation as well as overt saccades after head rotation (figure 3, A and D).

Clinical application. This video method of recording eye movements during head impulses makes examination of acute vertigo patients possible where scleral search coil recordings are impractical. The unilateral VOR deficit of patients with acute VN can be detected even in the presence of spontaneous nystagmus (figure 5, A–C). The noninvasive and short (approximately 10 minutes) nature of vHIT also facilitates follow-up examinations to document recovery of vestibular function (figure 5, D–F).

DISCUSSION In 1988, 2 of the authors reported[1] a simple indicator that allowed clinicians at the bedside to identify peripheral deficits of horizontal semicircular canal function—the presence of a saccade after a small, rapid, passive, unpredictable, horizontal head rotation (a "head impulse") by the clinician while the patient attempted to maintain gaze on a target. If semicircular canal function is impaired, the slow phase eye velocity is inadequate, so the eyes move with the head (off the target), and at the end of the head rotation, the patient must make a saccade to return gaze to the target. That corrective saccade is easily detectable if it is made after the head rotation has stopped, and so these saccades are termed "overt" saccades.[6]

The HIT has found wide use as a qualitative clinical sign, but it has major limitations: 1) There is no objective measure of VOR gain or of the corrective saccade: the clinician's report is based on the subjective visual observation of the presence of an overt saccade. 2) Different clinicians carry out this impulse with very different trajectories, so the accelerations and velocities used differ considerably. 3) Usually only few head rotations are given, so there is not a range of stimuli for generating a stimulus-response function. 4) Some patients may hide their peripheral vestibular deficit with "covert" saccades during head rotation. As such, their peripheral pathology is missed, and it may be incorrectly concluded that they

Figure 5 Diagnosis of acute vestibular neuritis and documentation of recovery



(A–C) Video head impulse test of a patient 2 days after onset of acute vestibular neuritis. (A) In head impulses to the affected left side, catch-up saccades replace the deficient vestibulo-ocular reflex (VOR). The spontaneous nystagmus (scattered spikes) beats in the same direction as the catch-up saccades. (B) In head impulses to the healthy right side, the VOR is preserved and the spontaneous nystagmus beats to the opposite direction. (C) The VOR gain is deficient to the left (open squares) but preserved to the right (filled squares). (D–F) Video head impulse test of a patient 3 days (D) and 1 month (E) after onset of acute vestibular neuritis. Between the 2 recordings, the VOR gains returned toward normal (F), the majority of catch-up saccades disappeared, and the patient recovered from symptoms. (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

have a central vestibular disorder responsible for their symptoms. Such a "covert" saccade is almost impossible to detect by simple visual observation: it cannot be distinguished from the normal slow phase eye velocity needed for proper compensatory eye movement.

To overcome these limitations, we have developed a new lightweight, minimal-slip, high-speed video-oculography system[11] (vHIT, video) that measures eye velocity during head rotation. Importantly, the camera is mounted on a specially designed, very lightweight frame to minimize inertia and slippage (figure e-1). Instant feedback about every single head impulse allows the examiner to apply a set of standardized graded impulses. The system is easy to use in a clinical setting, provides an objective measure of the VOR, and detects both overt and covert catch-up saccades in patients with vestibular loss. Measurements are quick (approximately 10 minutes) and noninvasive, and the automated analysis software provides instant results.

The simultaneous video and search coil HIT recordings validate the diagnostic accuracy of high-speed video recording. Despite fundamentally different recording methods, we achieved head and eye velocity recordings that were closely comparable. Both methods correctly identified the peripheral vestibular deficit in patients with highly reproducible VOR gains and detected even the smallest catch-up saccades.

With the bedside HIT, clinicians have to deliver high head velocities to optimize the chance of detecting the corrective saccade.[6] Although high velocities also help to reveal VOR asymmetry in patients with VN,[6] lower velocities of approximately 100° to 150°/

second are sufficient to detect the deficit in acute patients (figure 5C). In practice, this is an advantage for vHIT because the effects of slippage of the glasses and inertia are smaller at lower head velocities, and patients with acute vertigo better tolerate these lower velocities.

The simple application of vHIT allows the clinician to diagnose patients with VN acutely while they are ill and assess them again after they have recovered, providing objective evidence of the VOR deficit and the extent of its recovery. As figure 5, A–C, shows, these measures are possible even in the presence of a very vigorous spontaneous nystagmus.

Bedside HIT remains a useful clinical sign to assess patients with acute spontaneous vertigo because it helps to distinguish between acute VN, where the test is positive, and a central vestibular lesion, where the test is usually negative. However, between 9% and 39% of positive clinical HIT results have been reported in patients with acute cerebellar or brainstem strokes.[22,23] vHIT will be a suitable tool to determine whether these cases are really due to reduced VOR gain or simply result from clinical misjudgment. This way, video HIT will help to improve diagnostic accuracy for patients with acute spontaneous vertigo in the emergency department.

ACKNOWLEDGMENT

The authors thank all the participants of the study, Anthony Anderson for recording of the video, and David Newman-Toker for advice.

AUTHOR CONTRIBUTIONS

The statistical analysis was conducted by the authors Hamish G. MacDougall and Ian S. Curthoys (Vestibular Research Laboratory, School of Psychology, University of Sydney, Australia).

DISCLOSURE

Dr. MacDougall receives research support from the National Aeronautics and Space Administration [NNJ04HF51G (Coinvestigator) and NNJ04HD66G (Coinvestigator)], the National Space Biomedical Research Institute [NNJ07ZSA002N (Co-PI) and SM00801 (Co-PI)], the European Space Agency [SPIN (Coinvestigator)], the Australian Research Council [DP0665402 (APD)], and the Garnett Passe and Rodney Williams Memorial Foundation [Project Grant 2009 (PI)]. Dr. Weber received a research fellowship from the Garnett Passe and Rodney Williams Memorial Foundation. Dr. McGarvie reports no disclosures. Dr. Halmagyi serves on the editorial boards of *Acta Otolaryngologica*, *Otology & Neurotology*, *Audiology & Neurotology*, and the *Italian Journal of Otolaryngology*; and receives research support from the National Health and Medical Research Council [570890 (Co-PI) and 457511 (Co-PI)]. Dr. Curthoys serves on the editorial advisory boards of *Experimental Brain Research* and *Brain Research Bulletin*; and has received research support from the Australian Research Council [DP-0665402 (Chief Investigator)], the National Health and Medical Research Council [457511 (Chief Investigator) and 509206 (Coinvestigator)], and the Garnett Passe and Rodney Williams Memorial Foundation [L2907 R6934 (Chief Investigator)].

Received March 14, 2009. Accepted in final form July 7, 2009.

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Editor's Note to Authors and Readers: Levels of Evidence coming to *Neurology*®

Effective January 15, 2009, authors submitting Articles or Clinical/Scientific Notes to *Neurology*® that report on clinical therapeutic studies must state the study type, the primary research question(s), and the classification of level of evidence assigned to each question based on the classification scheme requirements shown below (left). While the authors will initially assign a level of evidence, the final level will be adjudicated by an independent team prior to publication. Ultimately, these levels can be translated into classes of recommendations for clinical care, as shown below (right). For more information, please access the articles and the editorial on the use of classification of levels of evidence published in *Neurology*.[1-3]

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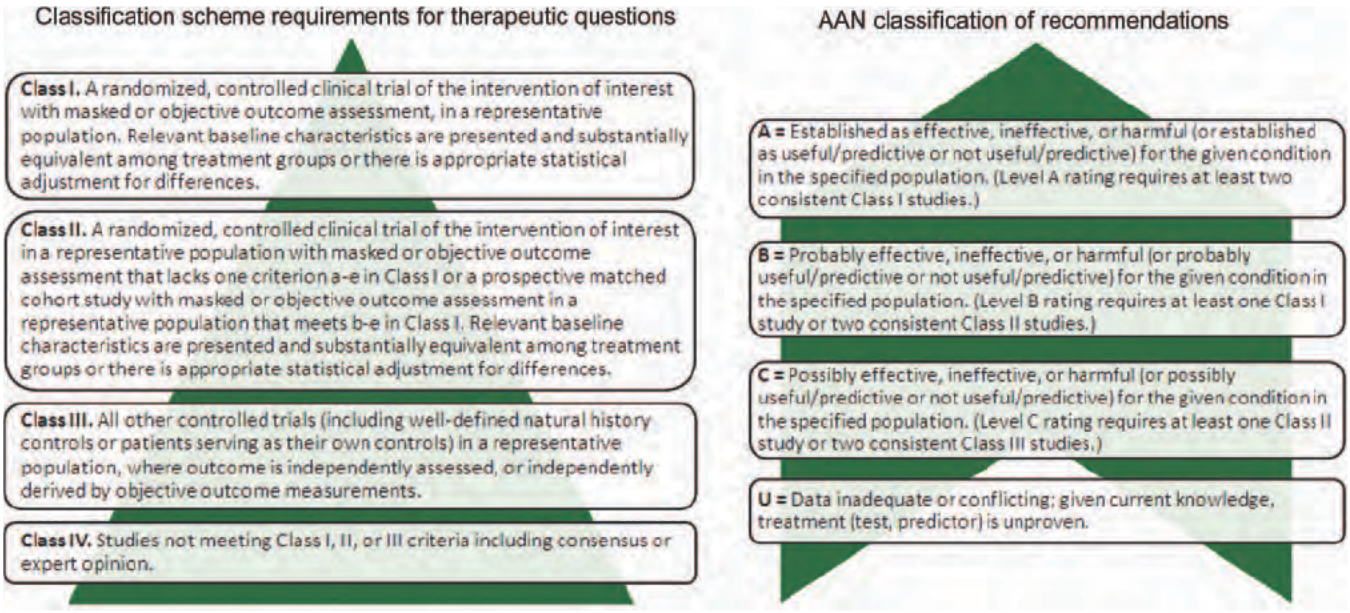

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

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Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

This information is current as of October 5, 2009

NEUROLOGY

Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis

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Neurology 2008;70;2378-2385
DOI: 10.1212/01.wnl.0000314685.01433.0d

Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis

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ABSTRACT

Objective: To test the diagnostic accuracy of the horizontal head impulse test (h-HIT) of vestibulo-ocular reflex (VOR) function in distinguishing acute peripheral vestibulopathy (APV) from stroke. Most patients with acute vertigo, nausea/vomiting, and unsteady gait have benign APV (vestibular neuritis or labyrinthitis) as a cause. However, some harbor life-threatening brainstem or cerebellar strokes that mimic APV. A positive h-HIT (abnormal VOR) is said to predict APV.

Methods: Cross-sectional study at an urban, academic hospital over 6 years. Consecutive acute vestibular syndrome patients at high risk for stroke underwent structured examination (including h-HIT), neuroimaging, and admission. Stroke was confirmed by neuroimaging (MRI or CT). APV was diagnosed by normal MRI and appropriate clinical evolution in follow-up.

Results: Forty-three subjects enrolled. One had an equivocal h-HIT. Patients with APV had a positive h-HIT (n = 8/8, 100%). Most patients with stroke had a negative h-HIT (n = 31/34, 91%). However, contrary to conventional wisdom, three patients with stroke (9%) demonstrated a positive h-HIT (1 vestibulocerebellar, 1 pontocerebellar, 1 pontocerebello-labyrinthine stroke).

Conclusions: Patients with lateral pontine and cerebellar strokes can have a positive horizontal head impulse test (h-HIT), so the sign's presence cannot be solely relied upon to identify a benign pathology. Additional clinical features (e.g., directionality of nystagmus, severity of truncal instability, nature of hearing loss) must be considered in patients with acute vestibular syndrome with a positive h-HIT before a central localization can be confidently excluded. Nonetheless, the h-HIT remains a useful bedside test—in acute vestibular syndrome patients, a negative h-HIT (i.e., normal VOR) strongly suggests a central lesion with a pseudo-labyrinthine presentation. *Neurology*® 2008;70:2378–2385

GLOSSARY

APV = acute peripheral vestibulopathy; **DWI** = diffusion-weighted imaging; **FLAIR** = fluid-attenuated inversion recovery; **h-HIT** = horizontal head impulse test; **VOR** = vestibulo-ocular reflex.

Acute vestibular syndrome is the rapid onset of vertigo, nausea, and vomiting (with nystagmus, unsteady gait, and head motion intolerance) over seconds–hours, lasting days–weeks. This presumed-viral, peripheral vestibular disorder is known as vestibular neuritis (without auditory symptoms),[1,2] labyrinthitis (with auditory symptoms),[3] or, generically, acute peripheral vestibulopathy (APV).[4]

Most acute vestibular patients have APV, but some have an acute central vestibular syndrome[5] resulting from posterior fossa stroke.[5-11] Roughly 16% of inferior cerebellar strokes present in pseudo-labyrinthine fashion.[11] The type of nystagmus evident on examination has been said to differentiate APV from central stroke mimics,[12] but assessment of nystagmus alone cannot distinguish all cases,[10] and perhaps half of patients with pseudo-labyrinthine stroke have unidirectional nystagmus mimicking APV.[11]

Supplemental data at www.neurology.org

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The preparation of this manuscript was supported partly by a National Institutes of Health grant (K23 RR17324-01, Building a New Model for Diagnosis of ED Dizzy Patients).

Disclosure: The authors report no disclosures.

In 1988, the horizontal head impulse test (h-HIT) of vestibulo-ocular reflex (VOR) function was described as a bedside test for peripheral vestibular disease.[13] The normal VOR response to a rapid, passive[14] head rotation as a subject fixates on a central target is an equal and opposite eye movement that keeps the eyes stationary in space (negative h-HIT). An abnormal response occurs when the head is rapidly rotated toward the side of a vestibular lesion. The loss of vestibular afferent input results in the inability to maintain fixation during the head rotation, requiring a corrective gaze shift once the head stops moving (positive h-HIT).

A positive h-HIT is sensitive for APV[15] and correlates with ipsilateral vestibular paresis.[13,16-18] Some have suggested a positive test confirms APV.[11,19] However, central lesions may simulate peripheral vestibular paresis when tested by other methods,[20] and prospective studies are lacking. Our objective was to assess the diagnostic accuracy of the h-HIT in distinguishing peripheral from central causes of the acute vestibular syndrome. We hypothesized that the h-HIT would be abnormal in APV, but normal in stroke mimics.

METHODS

We conducted a prospective, cross-sectional study of patients presenting for emergency evaluation of acute vestibular syndrome over a 6-year period, focusing on those at high risk for stroke. To ensure that findings were generalizable to others with posterior fossa stroke, we also studied those without the full-blown acute vestibular syndrome diagnosed as having cerebellar infarction during the same time period.

The study was conducted at a single urban, academic hospital serving as a regional stroke referral center for 25 community hospitals (~700 stroke admissions per year). The study was approved by the Institutional Review Board of The University of Illinois College of Medicine at Peoria. Two groups of patients consented to participate: group I—those with acute vestibular syndrome; group II—those without full-blown acute vestibular syndrome found to have ischemic cerebellar strokes by neuroimaging.

Group I (recruited based on symptoms). Consecutive patients (1999–2005) with acute vertigo, nausea, and retching-vomiting that 1) had at least one stroke risk factor and 2) were found to have nystagmus and truncal or gait instability on examination were eligible (figure 1A). Vertigo was defined as a sensation of spinning, either internal or external, self- or world-referenced. Excluded were those most likely to have APV or other peripheral vestibular disorder and those with obvious central oculomotor or neurologic signs (including limb ataxia) at initial assessment (figure 1B). The first (non-study) examiner (emergency physician or neurology house-staff) made a diagnosis of acute vestibular syndrome and contacted the study neuro-ophthalmologist (J.C.K.) if the patient had known stroke risk factors.

Group II (recruited based on diagnosis). During the same period (1999–2005), consecutive patients admitted with a radiographic diagnosis of ischemic cerebellar stroke were identified by neurology house-staff, who notified the study neuro-ophthalmologist of a potential subject without revealing clinical details. To be enrolled as a group II subject, patients were required to have truncal instability or gait imbalance (figure 1A), but not nystagmus, in the absence of significant limb weakness, limb ataxia, or other obvious brainstem signs (figure 1B).

All subjects. For patients consenting to screening, the neuro-ophthalmologist conducted a neurologic and vestibular examination (including h-HIT) according to a standard protocol (appendix e-1 on the *Neurology*® Web site at www.neurology.org). The h-HIT was performed from an eccentric head position to straight ahead, and several trials were assessed in each patient. An abnormal (positive) h-HIT response was determined based on the presence of a clear, reproducible, re-fixation saccade toward the affected side (video 1). As is standard practice, the intact side served as an intrasubject control/comparison response for each patient. All patients underwent neuroimaging, generally after standardized bedside evaluation. If neuroimaging was performed prior to the study evaluation, the study examiner was masked to these results at the time of clinical assessment. The examiner was not masked to other examination elements performed prior to the h-HIT.

All patients (including suspected APV patients), regardless of group assignment, were admitted for observation and underwent serial daily examinations for evolution of vestibular/neuro-ophthalmic signs or development of new neurologic findings. Patients were re-evaluated as outpatients for improvement within 2 weeks after discharge. At that time, most patients underwent electronystagmography or videonystagmography and several had their eye movements videotaped during repeat assessment of the h-HIT (videos 1–3).

The reference standard for a diagnosis of stroke was evidence of acute stroke by neuroimaging in the appropriate clinical context. MRI brain with diffusion-weighted imaging (DWI)[21] on the day of the index visit was generally the standard. Patients underwent stroke-protocol MRI (Siemens 1.5 Tesla Magnetom Vision/Plus) including 1) multiplanar T1, 2) axial T2 or fluid-attenuated inversion recovery (FLAIR), and 3) axial DWI sequences. Magnetic resonance angiogram was performed in roughly half.

Imaging studies were analyzed using the following method—sagittal T1, axial T1, and axial T2 or FLAIR images were used to define anatomic localization, and DWI was used to establish a diagnosis of acute ischemic stroke (except in three cases where MRI was not obtained, and the diagnosis was confirmed by CT). For anatomic and vascular localization, we used three axial sections through the cerebellum (appendix e-1).

The reference standard for a diagnosis of APV was absence of acute stroke in the brainstem or cerebellum by MRI, lack of neurologic signs on serial examination by a neuro-

Figure 1 Study inclusion and exclusion criteria

	Inclusion criteria	Exclusion criteria

History (Group I only)	ALL of the following:
	• Acute-onset vertigo
	• Nausea with retching or vomiting
	• Subjective gait instability
	• One or more stroke risk factors:
	  ○ Arterial hypertension
	  ○ Diabetes mellitus
	  ○ Hyperlipidemia
	  ○ Cigarette smoking
	  ○ Atrial fibrillation
	  ○ Hypercoagulable state
	  ○ Eclampsia
	  ○ Recent cervical trauma
	  ○ Prior myocardial infarction
	  ○ Prior stroke
		ANY of the following:
		• Preceding upper respiratory infection
		• Prior known diagnosis of Meniere disease
		• Previous attacks of vertigo with a history of fluctuating or long-standing hearing loss suggesting Meniere

Examination (Group I/II)	BOTH of the following:
	• Nystagmus* (Group I only)
	• Truncal or gait ataxia
		ANY of the following (unless chronic):
		• Hemiparesis
		• Hemisensory loss
		• Hemianopsia
		• Limb ataxia or dysmetria
		• Dysarthria, dysphonia, or dysphagia
		• Oculomotor signs other than horizontal nystagmus or impaired visual smooth pursuit tracking:
		  ○ Ophthalmoplegia (CN 3-4-6, INO, gaze palsy)
		  ○ Skew deviation
		  ○ Saccade dysmetria
		  ○ Nystagmus with dominant vertical or torsional vector
		• Other cranial neuropathy†

Inclusion and exclusion criteria are shown for group I (acute vestibular) and group II (cerebellar stroke) patients. Group I subjects were recruited based on symptoms, signs, and risk factors. Group II subjects were recruited based on radiographic evidence of a cerebellar stroke plus an examination finding of truncal or gait ataxia (nystagmus was not a prerequisite). *Nystagmus was sought during visual fixation (normal viewing) and again with fixation removed (behind Frenzel goggles). Frenzel goggles are occlusive goggles with high-plus diopter lenses that obscure the patient's vision (suppressing the ability to visually fixate) while providing the examiner a magnified, illuminated (or infrared) view of the eyes. †Lower-motor neuron facial palsy (7th) or deafness (8th) was not exclusionary. CN = cranial nerve; INO = internuclear ophthalmoplegia.

ophthalmology-trained neurologist (J.C.K.), and a characteristic clinical course in follow-up.

For predictive accuracy of the bedside h-HIT, we compared proportions of normal and abnormal h-HIT responses in APV and stroke patients. Fisher exact test was used to calculate p values for comparison of proportions using Stata v6.0 (College Station, TX). All p values were two-sided, with $p < 0.05$ considered significant.

RESULTS Of 71 patients screened, 27 were excluded—6 had previously-documented Ménière syndrome, and 21 had obvious signs pointing to a brainstem localization (appendix e-2); 15 of these had ischemic stroke as the cause. No patients were excluded for an antecedent history of upper respiratory symptoms. One eligible subject refused enrollment. Of 43 patients included, 33 were recruited in group I (acute vestibular syndrome, including nystagmus, without known localization), and 10 were recruited in group II (cerebellar stroke with truncal or gait instability but without nystagmus). All group I patients reported rapid onset (usually over minutes) of vertigo, nausea, and gait instability. Although not an inclusion criterion, the same was true of all but one group II patient. Retching and vomiting was reported or observed in all group I patients (per inclusion criteria), and 9/10 in group II. Thus, group II subjects differed from group I subjects only in the absence of nystagmus. In total, there were 8 APV patients and 35 brainstem or cerebellar stroke patients.

The study population was 65% men (n = 28/43). Mean age was 64 years (SD 15 years, range 26–92). The age range for patients with stroke was 26–92, with 7 patients under age 50, and 3 of these 40 or younger. In 30% (n = 13/43), only one stroke risk factor was present; the remaining 70% (n = 30/43) had two or more risk factors. Twenty-three presented initially to our emergency department, 4 were inpatients at symptom onset, 1 presented as an outpatient, and 15 were transferred to our neurology ward from other institutions. Thirty-five patients were examined within 24 hours of symptom onset (mean 10.4 hours in group I and 10.1 hours in group II, range 1–24). In 8 patients, the initial examination was delayed (range 26 hours to 8 days). In 4 patients, the precise time of examination relative to symptom onset was unclear,

because the precise time of symptom onset was unknown.

Most patients (91%, n = 39/43) underwent stroke-protocol MRI at the time of admission. One patient underwent CT followed by open MRI at another facility because of claustrophobia, and three underwent CT but no MRI (one was claustrophobic, one died, and one required ventriculo-peritoneal shunt placement and was too ill for MRI). All three who did not have MRI had unequivocal cerebellar stroke by CT. Initial imaging occurred within 6 hours of study examination in most (70%, n = 30/43). Three with initial negative MRI underwent repeat MRI when new neurologic signs developed during inpatient follow-up (appendix e-2). Other than one claustrophobic reaction, no patients suffered complications from diagnostic testing.

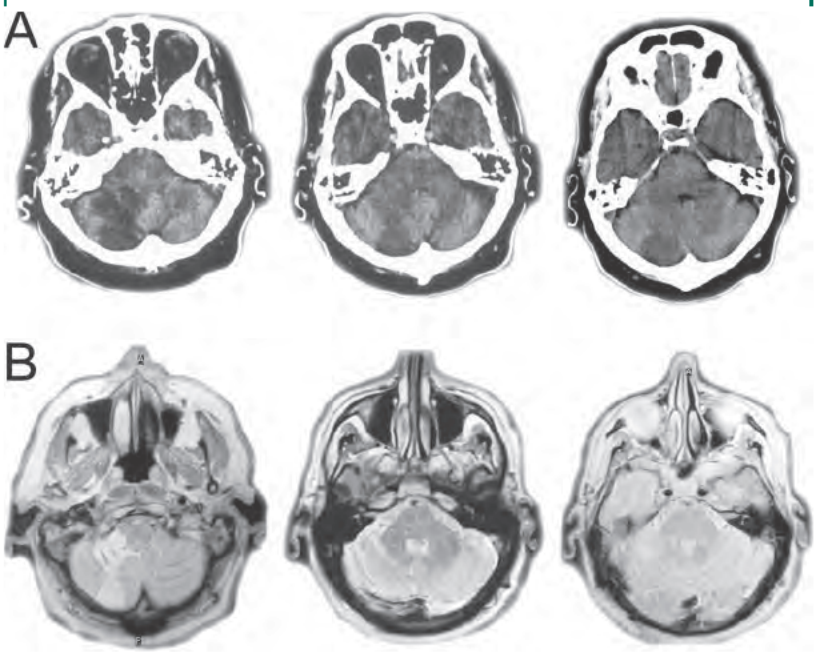
A detailed description of topographic stroke syndromes, associated symptoms, and individual patient findings can be found in appendix e-3. Aggregate results of h-HIT testing compared to neuroimaging are presented in the table. Three patients with stroke had a positive h-HIT. Each had subtle bedside clues suggesting a stroke syndrome. One with isolated inferior cerebellar infarction had primary-position left-beating nystagmus that reversed direction in right gaze. One with extensive pontocerebellar infarction was initially misdiagnosed as APV; in retrospect, the initial severity of the truncal instability (unable to stand without assistance) may have been an indicator of central disease. One with pontocerebellar and presumed cochleo-labyrinthine infarction presented with a pure pseudo-labyrinthine mimic; the sudden onset of total unilateral deafness in the context of known basilar stenosis and recent pontine infarction made a stroke syndrome fairly likely.

Table Head impulse test relative to neuroimaging in predicting the presence of stroke

	Negative imaging* (no stroke, n = 8)	Positive imaging† (stroke, n = 34‡)
Negative h-HIT (normal VOR)	0% (n = 0)	91% (n = 31§)
Positive h-HIT (abnormal VOR)	100% (n = 8)	9% (n = 3)

*All imaging that excluded a diagnosis of stroke was by MRI with DWI (n = 8/8, 100%).
†Most imaging that confirmed a diagnosis of stroke was by MRI with DWI (n = 32/34, 94%).
‡One of the 35 patients with stroke was excluded from this analysis, since no h-HIT result could be reliably obtained (the patient was too lethargic to visually fixate, which is a prerequisite for testing).
§All 10 subjects recruited in group II (recruited by diagnosis of cerebellar stroke, rather than symptoms) were patients with positive neuroimaging and negative head impulse test.
h-HIT = horizontal head impulse test; VOR = vestibulo-ocular reflex; DWI = diffusion-weighted imaging.

Figure 2 Imaging of acute peripheral vestibulopathy mimic, with pseudo-labyrinthine nystagmus, but normal horizontal head impulse test (h-HIT), suggesting stroke

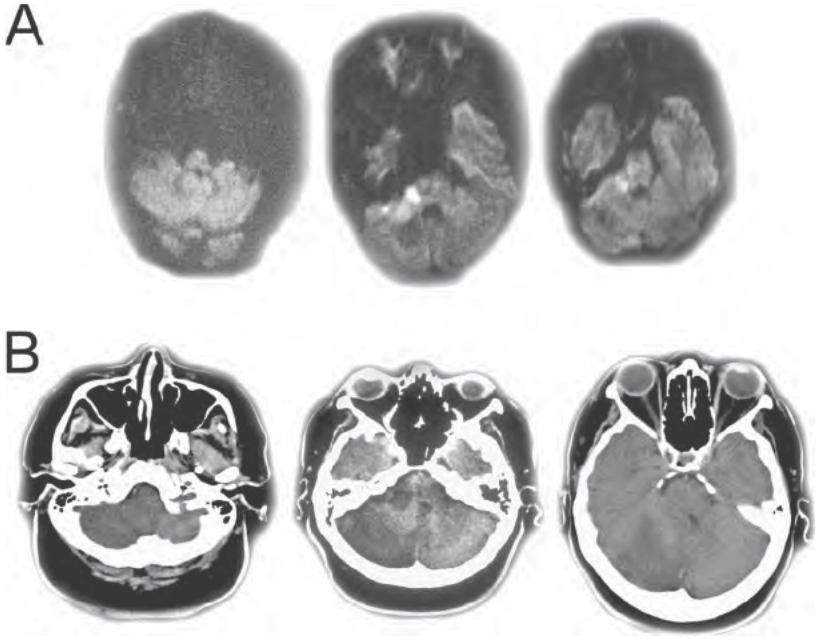


(A) Unenhanced axial CT of the head, obtained approximately 14 hours after symptom onset. There is a clear region of hypodensity involving the right inferior cerebellum, but the precise extent of the infarct or surrounding edema is difficult to delineate due to beam-hardening artifact on the CT image. The patient was claustrophobic, and could not tolerate MR imaging of the brain acutely. (B) Unenhanced axial T2 MRI of the brain, obtained in an open scanner 1 month after symptom onset. There is a well-circumscribed, wedge-shaped area of signal hyperintensity involving the right inferior cerebellum, consistent with subacute to chronic infarction.

The frequency of several associated symptoms and signs differed between stroke and APV patients, although none of these differences achieved significance in this relatively small sample: headache (43% stroke vs 13% APV, $p = 0.22$); severe truncal instability (34% stroke vs 0% APV, $p = 0.08$); direction-changing nystagmus (23% stroke vs 0% APV, $p = 0.32$); and impaired visual smooth pursuit in the absence of nystagmus (6% stroke vs 0% APV, $p = 1.0$). Together, one or more of these signs was present in only 60% of patients with stroke.

Neuroimaging by MRI revealed that all patients with APV had nonspecific areas of periventricular high signal intensity on T2 or FLAIR imaging but normal DWI, compatible with chronic gliosis, presumed secondary to ischemic leukoencephalopathy. Representative stroke imaging is shown in figures 2 and 3. The initial MRI, including DWI, was falsely negative in three patients with stroke (figure 4). These negative scans were obtained at 8, 12, and <30 hours after symp-

Figure 3 Imaging of acute peripheral vestibulopathy mimic, with pseudo-labyrinthine nystagmus and abnormal horizontal head impulse test (h-HIT), but evolution of additional oculomotor and neurologic signs, confirming stroke



(A) Unenhanced axial DWI MRI of the brain, obtained approximately 7 hours after initial vestibular symptom onset, after evolution of new neurologic signs including an infranuclear (lower motor neuron-type) facial palsy. Note, in particular, the areas of restricted diffusion in the right middle cerebellar peduncle and lateral pons (anterior inferior cerebellar artery territory), in the region of the right vestibular nerve root entry zone (center image). This root-entry-zone lesion likely accounts for the abnormal h-HIT, creating a nearly indistinguishable clinical mimic of a benign peripheral vestibular lesion at first presentation. (B) Unenhanced axial CT of the head, obtained 2 days after symptom onset. Follow-up imaging was obtained to evaluate the cause for increasing truncal and right limb ataxia. The scans suggest an extensive, new infarct in the posterior cerebellar hemisphere on the right (center image) with infarct or edema extending inferiorly and superiorly, and significant mass effect on the fourth ventricle (center and right images). Follow-up MR images obtained 1 year later (not shown) confirmed an extensive region of right cerebellar encephalomalacia, consistent with prior infarction.

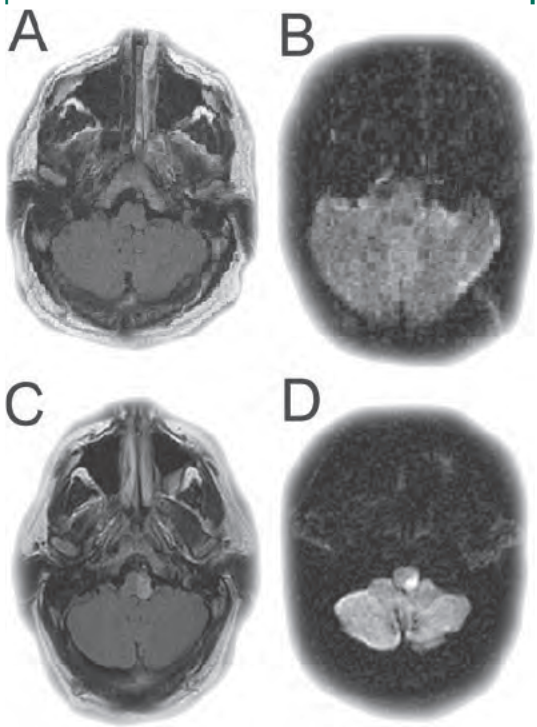
tom onset. Follow-up studies (3, 10, and 2 days later) revealed lateral medullary stroke in each case. Imaging evidence of mass effect was seen in the initial scan in seven patients, and in follow-up scan in one patient, all with at least some cerebellar involvement. Magnetic resonance angiogram was performed in 18 of 35 stroke cases; it revealed vertebral or PICA occlusion in 10 and was normal in 8. Three were diagnosed radiographically with vertebral artery dissection.

A negative h-HIT (i.e., normal VOR) was the most useful clinical sign for prospectively differentiating between central and peripheral causes of the acute vestibular syndrome (91% stroke vs 0% APV, $p < 0.001$). A negative h-HIT was a strong predictor of stroke (100%, n = 13/13), even in cases where the patient's nystagmus was frankly (n = 5) or potentially (n = 8) pseudo-labyrinthine. A negative h-HIT was particularly helpful for identifying patients with incipient lateral medullary stroke, whose initial MRI scans revealed no evidence of infarction, even on DWI sequences (n = 3).

In contrast to current wisdom, however, we found that a positive h-HIT (i.e., abnormal VOR) was insufficient grounds to conclude the acute vestibular syndrome was peripheral in localization. In our series, 30% (n = 3) of patients with acute vestibular syndrome with a positive h-HIT (n = 11) harbored an underlying stroke. Alarmingly, one of these patients had a pure pseudo-labyrinthine presentation that only declared itself clinically through evolution of neurologic signs and symptoms over the ensuing 24 hours after initial presentation.

DISCUSSION Our study confirms the utility of h-HIT in distinguishing peripheral from central causes of the acute vestibular syndrome, but departs from current neuro-otologic thinking by suggesting that the sign's absence may be more helpful than its presence. A negative h-HIT appears to be the single best predictor of stroke in

Figure 4 False negative MRI in a patient with lateral medullary stroke



Unenhanced axial FLAIR (A, C) and DWI (B, D) MR images of the brain at the level of the medulla. (A) and (B) show scans obtained approximately 8 hours after onset of acute vestibular symptoms, while (C) and (D) show matching scans obtained roughly 3 days later. Note the normal initial scans (A, B) and the clear evolution of FLAIR hyperintensity (C) and restricted diffusion (D) in the left lateral medulla, confirming the presence of an acute infarct, missed on the original scans.

patients with acute vestibular syndrome without frank neurologic symptoms or signs.

The simplest method for identifying those patients with acute vestibular syndrome at high risk for stroke is to select patients with known stroke risk factors. We and others[5] have used this strategy to identify a disease-enriched subpopulation for study. However, the utility of this approach should not be overestimated, since 12% of patients with cerebellar or brainstem stroke with identical symptoms have no known risk factors.[11] While some authors have suggested that frank neurologic symptoms or signs (e.g., hemi-body numbness or weakness, vertical nystagmus) are the keys to identifying pseudo-labyrinthine stroke mimics,[22,23] we found only 42% (n = 21/50) of strokes causing acute vestibular syndrome had such obvious findings (appendix e-2).

We speculate that the pathogenesis of the positive h-HIT was probably different in each of the three stroke cases. With the inferior cerebellar stroke, it was probably due to mass effect on the vestibular nucleus or 8th nerve root-entry zone in the pons, rather than labyrinthine infarction since 1) there was no associated hearing loss, 2) the primary-gaze-position nystagmus was in the wrong direction for a left-sided labyrinthine lesion, and 3) the cerebellar infarct was in the posterior inferior cerebellar artery rather than anterior inferior cerebellar artery distribution. In the pontocerebellar and presumed cochleo-labyrinthine infarction, it was probably the result of labyrinthine infarction, since the patient also developed sudden, unilateral, severe hearing loss. In the extensive ponto-cerebellar stroke, it was probably the result of vestibular nucleus or 8th nerve root-entry-zone infarction in the lateral pons (figure 3A). Although unlikely, it is conceivable that the positive head impulses in these cases were old and incidental; even if this were the case, they would still represent misleading, false-positive bedside results not to be relied upon.

Two points about neuroimaging in these patients are worth noting. First, three patients had false negative MRI with DWI—one perhaps as late as 30 hours after symptom onset. DWI has previously been demonstrated to be less than 100% sensitive,[24] particularly with strokes in the posterior circulation, where over 30% of MRIs obtained within a day of symptom onset can have negative DWI.[25] Therefore, we must be careful not to become over-reliant on imaging as the final arbiter of diagnosis in the acute-care setting for acute vestibular syndrome patients. Second, none of the eight peripheral (i.e., non-stroke) patients in our study had a normal MRI of the brain; all showed nonspecific gliosis. This is not terribly surprising given the inclusion criterion of "at least one stroke risk factor." However, it does raise an intriguing question about underlying etiology (i.e., viral vs vascular) in these well-localized peripheral vestibulopathy patients. Perhaps a much larger, population-based study could address the issue of MRI-negative, small labyrinthine infarcts vs viral causes, by assessing long-term stroke risk in vestibular syndrome patients and age-matched controls. More likely, we will have to await better imaging techniques to assess labyrinthine ischemia and infarction. For the time being, distinguishing those with large cerebellar infarcts will have to suffice. This more limited goal is still an important one, since large cerebellar infarcts place patients at significant risk of herniation,[26] and probably mark those at highest risk of subsequent brainstem stroke.[27]

Finally, it is notable that 20% (n = 7/35) of strokes in this series occurred in patients under age 50, three of whom were diagnosed as having vertebral artery dissection as a cause. Care should be taken not to overemphasize the importance of youth as "protective" factor for stroke in acute vestibular syndrome, lest we disproportionately misdiagnose young patients with cerebellar strokes.[28] Patients with vertebral artery dissections can present in pseudo-labyrinthine fashion[29] and dissection is the leading identifiable cause of stroke among young adults.[30,31]

The two major limitations to our study are one threat to internal validity (partially masked examiner) and one to external validity (sample from high-risk subpopulation). The study examiner (J.C.K.), though masked to the results of imaging, was not masked to the patient's clinical history, general neurologic examination, or eye movement findings at the time of testing the h-HIT. The h-HIT, like other bedside tests that require a degree of skill and interpretation on the part of an examiner (e.g., the plantar response[32]), is potentially subject to examiner bias, and we did not have multiple raters assess the h-HIT. An objective means of identifying patients with positive and negative head impulses (e.g., using magnetic search coils or video-oculography) could have been used to confirm the accuracy of our findings,[33] but this was not feasible. If such an observer bias was present in our study, it would have been based on the examiner's preconceived notion that patients with stroke should have a negative h-HIT. This bias would not have produced the novel and unexpected finding of a posi-

tive h-HIT in several patients with stroke, including one in whom the patient was initially misdiagnosed by the study examiner as APV based on the positive h-HIT response. If anything, this bias would have led us to underestimate (rather than overestimate) the frequency with which a positive h-HIT is found among pseudo-labyrinthine posterior circulation patients with stroke.

By design, the study population was not representative of the larger population of acutely dizzy patients seen in the acute-care setting. We included only patients with acute vestibular syndrome (not all acutely dizzy patients, some of whose transient dizziness resolved prior to presentation), and we further restricted this population to those with at least one stroke risk factor. Some additional patient selection (not by design) may also have occurred, since no study patients were found to have a cerebellar hemorrhage and none were excluded for antecedent viral illness. Therefore, care should be taken when drawing inferences about the population prevalence of stroke among patients with acute vestibular syndrome (81% [n = 34/42] in our study, compared to a prior report of 25% [n = 6/24]).[5] However, there is no reason to believe that use of a high-risk sample would substantially alter our conclusions regarding the h-HIT. Including more patients with vestibular neuritis in our study might have led to identifying some in whom the h-HIT was negative, particularly that minority with isolated inferior vestibular neuritis, whose h-HIT results are normal, despite having VOR deficits in the plane of the posterior semi-circular canal.[16] Including more such patients would have decreased the separation between pseudo-labyrinthine stroke and APV seen in our study (91% vs 0%). However, this would be unlikely to reduce the test's clinical value to any great extent. Studies suggest that roughly 8–18% of patients with APV have a negative h-HIT,[15,34] so the diagnostic utility of a normal VOR response in predicting stroke would be reduced only marginally. In considering any hypothetical loss of diagnostic utility, it is important to remember that the consequences of missing a large cerebellar infarct[28] likely far outweigh the costs of additional testing in that minority of patients with false negative h-HIT who turn out to have APV.

ACKNOWLEDGMENT

The authors thank Michael Zagardo, MD, who read all the imaging studies, and David Zee, MD, for his critical review and valuable advice regarding the manuscript and videos.

Received July 17, 2007. Accepted in final form December 28, 2007.

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Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis

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Neurology 2008;70;2378-2385
DOI: 10.1212/01.wnl.0000314685.01433.0d

Otology & Neurotology
24:913–917 © 2003, Otology & Neurotology, Inc.

Head-Impulse and Caloric Tests in Patients With Dizziness

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NEUROLOGY

Head impulse test in unilateral vestibular loss: Vestibulo-ocular reflex and catch-up saccades

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Neurology 2008;70;454-463
DOI: 10.1212/01.wnl.0000299117.48935.2e

Head impulse test in unilateral vestibular loss

Vestibulo-ocular reflex and catch-up saccades

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ABSTRACT

Background: Quantitative head impulse test (HIT) measures the gain of the angular vestibulo-ocular reflex (VOR) during head rotation as the ratio of eye to head acceleration. Bedside HIT identifies subsequent catch-up saccades after the head rotation as indirect signs of VOR deficit.

Objective: To determine the VOR deficit and catch-up saccade characteristics in unilateral vestibular disease in response to HIT of varying accelerations.

Methods: Eye and head rotations were measured with search coils during manually applied horizontal HITs of varying accelerations in patients after vestibular neuritis (VN, n = 13) and unilateral vestibular deafferentation (UVD, n = 15) compared to normal subjects (n = 12).

Results: Normal VOR gain was close to unity and symmetric over the entire head-acceleration range. Patients with VN and UVD showed VOR gain asymmetry, with larger ipsilesional than contralesional deficits. As accelerations increased from 750 to 6,000 °/sec^2, ipsilesional gains decreased from 0.59 to 0.29 in VN and from 0.47 to 0.13 in UVD producing increasing asymmetry. Initial catch-up saccades can occur during or after head rotation. Covert saccades during head rotation are most likely imperceptible, while overt saccades after head rotation are detectable by clinicians. With increasing acceleration, the amplitude of overt saccades in patients became larger; however, initial covert saccades also became increasingly common, occurring in up to about 70% of trials.

Conclusions: Head impulse test (HIT) with high acceleration reveals vestibulo-ocular reflex deficits better and elicits larger overt catch-up saccades in unilateral vestibular patients. Covert saccades during head rotation, however, occur more frequently with higher acceleration and may be missed by clinicians. To avoid false-negative results, bedside HIT should be repeated to improve chances of detection. *Neurology*® 2008;70:454–463

GLOSSARY

HIT = head impulse test; **UVD** = unilateral vestibular deafferentation; **VN** = vestibular neuritis; **VOR** = vestibulo-ocular reflex.

The head impulse test (HIT) assesses vestibular function with brisk, passive rotations of the head in the plane of parallel semicircular canal pairs.[1] In healthy subjects, the angular vestibulo-ocular reflex (VOR) stabilizes gaze in space by compensating the head rotations with equal eye rotations in the opposite direction.[2-5] When the VOR is deficient, the eyes move with the head, forcing the patient to make a catch-up saccade in order to re-fixate the target.[6-9] Hence, both residual VOR and catch-up saccades act synergistically to stabilize gaze.[10] Usually, HIT is applied in a non-standardized fashion at a single, examiner-dependent head acceleration.

Quantitative HIT records and analyzes the slow phase eye movement response during head rotation to determine the VOR gain.[2,11] This differs from bedside HIT, where the clinician identifies the catch-up saccades after head rotation as an indirect sign of VOR deficit.[1,12] These catch-up saccades are only visible after the head movement as they

Supplemental data at www.neurology.org

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Supported by the Garnett Passe and Rodney Williams Memorial Foundation.

Disclosure: The authors report no conflicts of interest.

cannot be distinguished from the VOR while the head is still moving. Therefore, bedside HIT depends on the timing and size of the consequent catch-up saccades rather than on the measured VOR gain.

In this study, we set out to relate the measurements from the quantitative HIT to the determinants of the bedside HIT. For this purpose, we measured the VOR gain as a function of head acceleration and analyzed the corresponding distribution pattern of catch-up saccades in patients after vestibular neuritis (VN) or unilateral vestibular deafferentation (UVD) compared to normal subjects.

METHODS

Patients. Thirteen patients (age range 36 to 68 years, mean ± SD 56.2 ± 8.5 years) with acute onset of prolonged rotational vertigo and postural imbalance associated with spontaneous nystagmus, nausea, or vomiting fulfilled the clinical criteria of VN.[6] All patients underwent bithermal caloric testing with irrigation at 30 °C and 44 °C. Canal paresis as determined by the Jongkees formula[13] showed more than 25% asymmetry between right- and left-sided responses in all patients. HIT was recorded between 2 weeks and 10 years after onset of symptoms.

Fifteen patients (age range 31 to 74 years, mean ± SD 52.7 ± 13.4 years) after UVD were studied as unilateral control subjects. Of these patients, 12 were operated as treatment for vestibular schwannoma and 3 underwent vestibular neurectomy for unilateral Ménière syndrome or intractable benign paroxysmal positional vertigo. HIT was recorded between 3 months and 20 years after the operation. Patients were compared with 12 healthy subjects (age range 27 to 65 years, mean ± SD 42.4 ± 13.5 years) without any history, symptoms, or clinical signs of vestibular disease.

Written informed consent was obtained from all subjects after the experimental procedure was explained. The protocol was approved by the Sydney South West Area Health Service Ethics Review Committee and was in accordance with the ethical standards laid down in the Declaration of Helsinki for research involving human subjects.

Experimental setup. Three-dimensional binocular eye and head position was recorded with scleral search coil techniques as previously described.[6,14] Tests were performed in a 1.9 × 1.9 × 1.9 m magnetic coil frame (CNC Engineering, Seattle, WA) with dual search coils manufactured by Skalar (Delft, The Netherlands) placed on both eyes after topical anesthesia with Alcaine 0.5% eyedrops (Alcon Laboratories Australia Pty Ltd). For reliable recording of head movements, the head coil was mounted on an individually molded dental impression bite bar. Dual search coils were precalibrated in vitro on a Fick gimbal about the three principal axes (±20° yaw, pitch, and roll) in 5° steps to determine gain and offset of each coil. Three-dimensional head and eye position signals were recovered by phase detection and low-pass filtered with anti-alias filters of 0 to 100 Hz bandwidth. The nine position signals were recorded at 1 kHz with 16-bit resolution. Minimum resolution of the system was 0.1 minute of arc with maximum cross coupling of 2% between orthogonal signals. An online feedback signal of the velocity profile of each head impulse was provided for the experimenter on an LCD monitor.[15]

Experimental procedure. Subjects were seated upright with the unrestrained head in the center of the magnetic coil frame. Under dimmed lighting, they were instructed to fixate a red laser dot projected at reference position straight ahead on a screen at a distance of 91 cm. Head impulses were manually delivered by the experimenter standing behind the subject. With the aid of a head velocity feedback display, passive head impulses in the horizontal plane with comparable time course (onset to peak velocity of 80 msec) were matched to six increasing peak velocities from 50 °/sec to 300 °/sec corresponding to accelerations of 750 to 6,000 °/sec^2 and amplitudes of 5 to 25°. At least 80 head impulses were recorded for each direction.

Data analysis. The experimental data were analyzed offline with a custom made analysis suite based on LabVIEW Version 7.1 (National Instruments, Austin, TX) and MatLab (The MathWorks Inc., Natick, MA). Individual head impulses were interactively selected and aligned to peak head acceleration. Three-dimensional head and gaze positions in space-fixed coordinates were derived from coil voltages and expressed as rotation vectors.[16] The signs of the rotation vectors were defined according to the right-hand rule with positive clockwise, downward, and leftward directions. The rotation vectors of the eye in the head-fixed coordinates were derived from gaze and head positions in space. From these rotation vectors, three-dimensional angular velocity and acceleration vectors of head, gaze, and eye were determined.

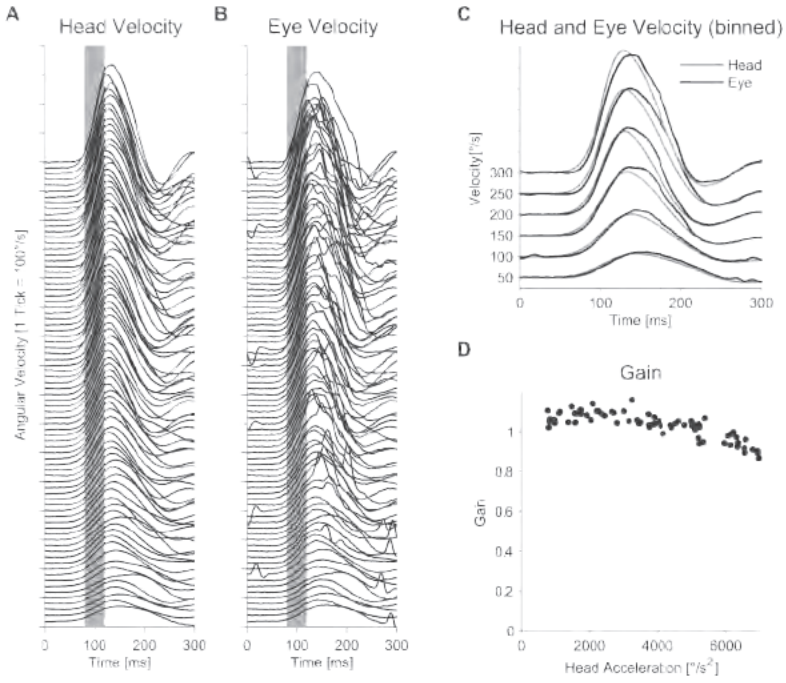
Gain of the VOR was calculated for individual trials as the ratio of the slope of a linear regression fitted to eye velocity over the slope of a linear regression fitted to head velocity during a 40-msec window centered at peak head acceleration.[11,17] Since the slopes of the two regressions over velocity represent acceleration, this method reflects an acceleration gain. The time window was chosen to end before the occurrence of early catch-up saccades in patients. Gains to each side were determined in the eye contralateral to the direction of the head movement. These are driven by the shortest disynaptic pathway from the ipsilateral horizontal semicircular canal to the contralateral lateral rectus muscle.[11,18]

Analogous to Jongkees formula for canal paresis in caloric testing, gain asymmetry g_s between ipsilesional (g_i) and contralesional (g_c) gains was determined as follows[5,13]:

$$g_s = \frac{g_c - g_i}{g_c + g_i} \times 100$$

Gains as a function of head acceleration for individual head impulses to each side (mean number: 78 ± 13 SD) were smoothed by a locally weighted regression using a least squares quadratic polynomial fitting algorithm[19] (robust LOESS, smoothing fraction f = 0.25) and interpolated for head accelerations between 750 and 6,000 °/sec^2. Means ± two-tailed 95% CI of LOESS functions were determined to describe gain as a function of head acceleration in patients and healthy subjects. Unpaired two-tailed Student t test was used to test for differences between the LOESS functions of patient groups and paired two-tailed t test was used to test for differences between the two sides of healthy subjects. Differences were considered significant if $p < 0.05$. Gains to

Figure 1 Scaled horizontal head impulse test of a healthy subject to the right



Head and eye velocity were always approximately equal and scaled almost linearly with an acceleration gain close to unity. Stacked inverted head velocity (A) and eye velocity (B) traces were aligned to peak head acceleration at 100 msec and sorted according to peak head velocity from about 50 °/sec to 300 °/sec (80 trials). Gray shaded areas indicate 40 msec time window centered at peak head acceleration where acceleration gain was determined. (C) Means of head and eye velocity from six bins between 50 °/sec and 300 °/sec (±25 °/sec) for visual comparison. (D) Acceleration gain of individual head impulses (filled circles) as a function of head acceleration.

both sides of healthy subjects were pooled to constitute the control group.

Onset of the head impulse was defined as the point where head velocity crossed 2% of peak head velocity. End of the head impulse was defined where head velocity crossed zero. Catch-up saccades during head rotation were occasionally difficult to distinguish from the VOR response and their onset was sometimes concealed. Therefore saccades were identified at peak eye velocity by detecting downward zero-crossings of the third derivative above a defined slope threshold.[20] Peak acceleration of saccades was used as a robust characteristic to define the saccade onset. Latency of the first catch-up saccade was measured between onset of the head impulse and peak acceleration of the saccade. Saccades were classified as covert if the onset occurred before the end of the head impulse and classified as overt afterwards. Gaze position error was determined as the difference between gaze position before and at the end of the head impulse.

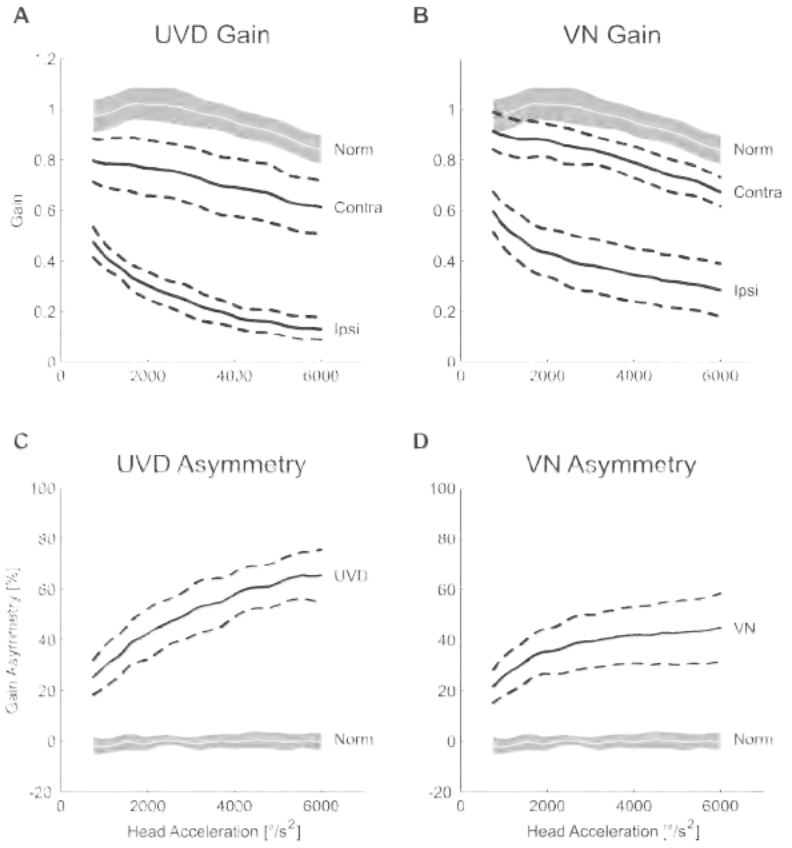
RESULTS We compared the HIT of patients after VN and UVD to normal subjects over a head acceleration range similar to natural head movements. Horizontal head impulses were scaled from peak head velocities of 50 °/sec to 300 °/sec corresponding to peak head accelerations of 750 °/sec² to 6,000 °/sec² (video 1 on the *Neurology*® Web site at www.neurology.org). The analysis included both the slow phase eye movement response to measure the VOR gain as well as the catch-up saccades which are the clinical sign of the bedside HIT.

Normal subjects. Twelve healthy control subjects were examined for comparison with patients with UVD and VN. Eye velocity of normal subjects was always approximately equal to inverted head velocity, so that gaze direction remained stable during horizontal head impulses (figure 1). The eye movement response correlated almost linearly with the head impulse stimulus over the whole acceleration range. Acceleration gains determined over a 40 msec time window centered at peak head acceleration were close to unity over the whole stimulus range. Gains of healthy subjects were not significantly different to either side and were pooled for further analysis. Mean gain across subjects was 0.98 ± 0.063 (mean ± 95% CI) at 750 °/sec², reached a maximum of 1.02 ± 0.062 at 1,717 °/sec², and slowly but significantly decreased to 0.84 ± 0.055 as head acceleration increased to 6,000 °/sec² (figure 2, A and B). Symmetry of gains to both sides was very tight in normal subjects over the whole acceleration range (−0.24% ± 2.3; mean ± 95% CI; figure 2, C and D).

Unilateral vestibular deafferentation. Patients after UVD showed markedly reduced eye velocity responses to head impulses mainly to the deafferented side resulting in unstable gaze direction and subsequent saccades to refixate the target (figure 3, A–C). The corresponding ipsilesional gains of 15 patients with UVD averaged 0.47 ± 0.06 (mean ± 95% CI) at 750 °/sec² relating to approximately half the gain of normal subjects (figure 2A). Ipsilesional gains decreased steeply with increasing head acceleration and asymptotically approached 0.13 ± 0.046 at 6,000 °/sec². Over the same acceleration range, mean contralesional gains linearly declined from 0.8 ± 0.085 to 0.61 ± 0.1. Mean ipsilesional gains showed approximately half the 95% CI compared to mean contralesional gains. The divergence of ipsilesional and contralesional gains led to a rising gain asymmetry that started with 25% ± 6.8 (mean ± 95% CI) at 750 °/sec² and increased asymptotically to 65% ± 10 at 6,000 °/sec² (figure 2C).

Vestibular neuritis. Patients after VN showed a gain pattern with a deficit predominantly to the affected side that was similar, but not as large as that of patients with UVD (figure 3, D–F). The corresponding ipsilesional mean gains of 13 patients with VN first decreased steeply from 0.59 ± 0.079 (mean ± 95% CI) at 750°/sec² to 0.43 ±

Figure 2 Summary of gain (A, B) and asymmetry (C, D) in patients after unilateral vestibular deafferentation (UVD, n = 15) and vestibular neuritis (VN, n = 13) compared to normal subjects (n = 2 × 12)



Both patient groups showed a steeply decreasing gain to the ipsilesional side and a linearly declining gain to the contralesional side resulting in a rising gain asymmetry with increasing head acceleration. Gain deficit and asymmetry was larger in UVD compared to VN. Means ± 95% CI of normal subjects (gray band). Ipsilesional and contralesional means (solid lines) ± 95% CI (dashed lines) of patients.

0.095 at 2,000 °/sec² head acceleration (figure 2B). Above about 2,000 °/sec² the mean gain declined more slowly to 0.29 ± 0.11 at 6,000 °/sec². Mean contralesional gains linearly declined from 0.91 ± 0.073 to 0.67 ± 0.058 over the entire acceleration range. The asymmetry between ipsilesional and contralesional gains of patients with VN first rose steeply from 22% ± 6.5 (mean ± 95% CI) at 750 °/sec² to 35% ± 8.8 at 2,000 °/sec² and then slowly approached 45% ± 14 at 6,000 °/sec² (figure 2D).

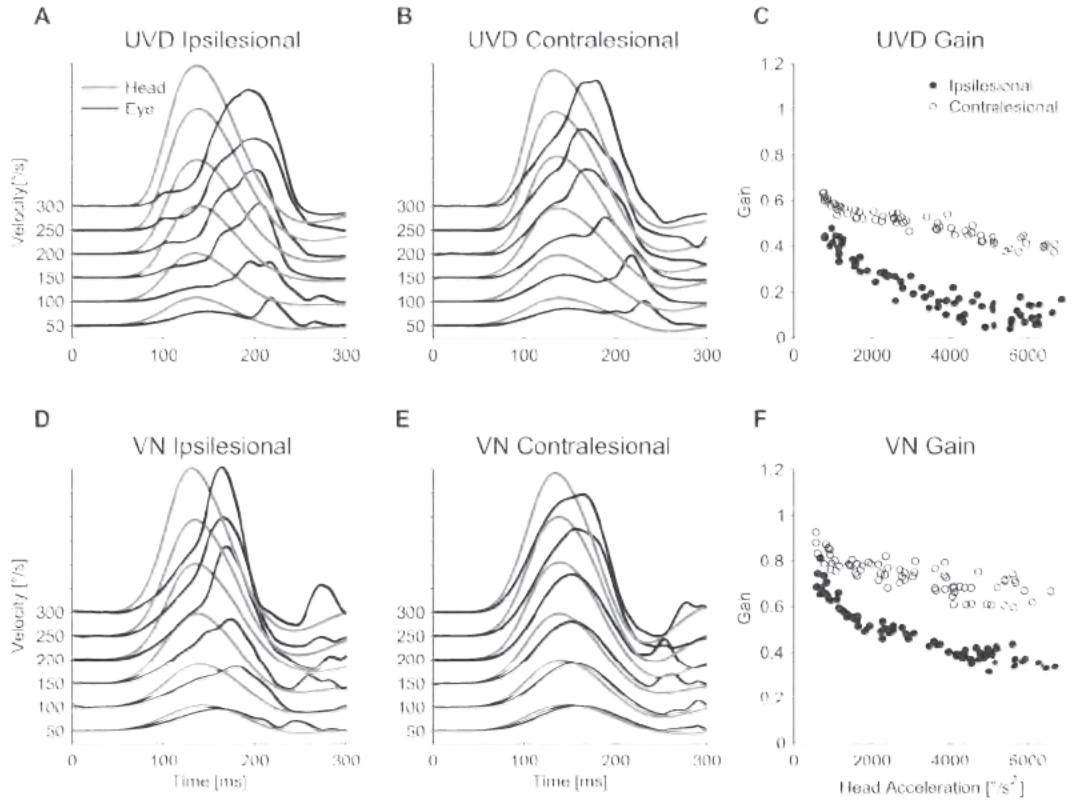
Comparison among normal subjects, UVD, and VN. While acceleration gains in normal subjects were close to unity, gains of patients after UVD showed a marked reduction mainly to the side of the lesion. Patients after VN showed reduced gains to the affected side as well, but the deficit was not always as large. With increasing head acceleration, the gain pattern of both patient groups was comparable with a decreasing gain to the ipsilesional side and a linearly declining gain to the contralesional side (figure 2, A and B). At higher acceleration, ipsilesional and contralesional gains of both patient groups decreased roughly parallel with but below the gains of healthy subjects. Compared to normal subjects, not only ipsilesional, but also contralesional gains of patients with UVD were reduced over the whole acceleration range ($p < 0.05$). In patients with VN, ipsilesional gains were significantly lower than those of normal subjects, but the difference in contralesional gains became significant only above a head acceleration of 1,158 °/sec². On average, contralesional gains were 26% below normal values in UVD and 16% below normal in VN. Mean gains of patients with VN were slightly higher compared to patients with UVD to either side. This difference was significant only for rotations to the ipsilesional side and for very low accelerations (<891 °/sec²) to the contralesional side.

While normal subjects showed symmetric gains over the whole acceleration range, gain asymmetry of both UVD and VN increased with head acceleration. Asymmetry between ipsilesional and contralesional gains of both patient groups was significantly different from normal subjects commencing from the lowest head acceleration tested. Asymmetry of gains for VN became significantly lower than for patients with UVD only above a head acceleration of 3,744 °/sec².

Catch-up saccades. In healthy subjects with a normal VOR, gaze remained on the target during HIT so that they rarely exhibited saccades during the head movement and only small catch-up saccades afterwards (figure 4, A and D, table e-1 on the *Neurology*® Web site at www.neurology.org). In patients with a deficient VOR, two types of catch-up saccades could be distinguished to keep the eye on target (video 2): 1) Early saccades that occurred on top of the residual VOR response (figure 4, B and E). With the head still moving, those covert saccades were most likely imperceptible to a clinical observer. 2) Late saccades that appeared during the fixation period after the head movement (figure 4, C and F). Depending on their amplitude, those overt saccades could be detected relatively easily by a clinical observer.[12] The three characteristic distribution patterns of catch-up saccades in time presented in figure 4 represented the boundaries of a continuous spectrum of individual saccadic responses among patients and normal subjects.

In normal subjects, the latency of the first catch-up saccade (252 msec ± 22; mean ± 95% CI; table) usually exceeded the duration of the head impulse (168 msec ± 6). Therefore, covert

Figure 3 Example of a patient after unilateral vestibular deafferentation (UVD, panels A–C) compared to a patient after vestibular neuritis (VN, panels D–F)



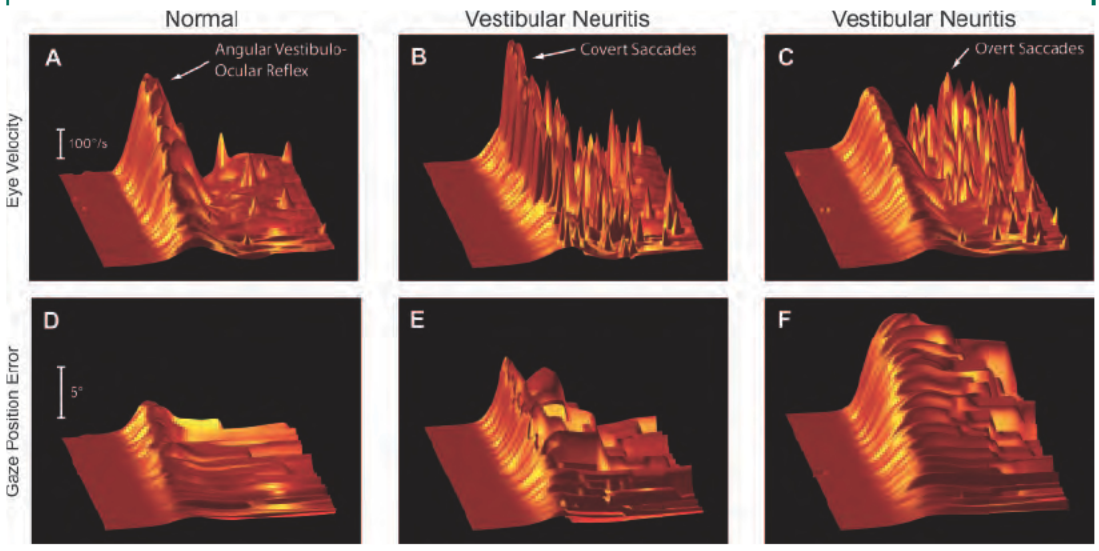
At low head velocity (50 °/sec), means of ipsilesional (A, D) and contralesional (B, E) eye velocity responses were almost symmetric in both patients. With increasing velocity, the ipsilesional deficit and asymmetry of the angular vestibulo-ocular reflex increased gradually. (C, F) Ipsilesional acceleration gains (filled circles) of both patients decreased steeply with increasing head acceleration whereas contralesional gains (open circles) declined only slightly. Both ipsilesional and contralesional gain levels were clearly below normal values (see figures 1 and 2), but higher in VN compared to UVD, suggesting residual ipsilesional canal function in the patient with VN.

saccades during head rotation were infrequent, but their mean occurrence increased slightly from 4% to 16% of trials as head velocity increased from 50 °/sec to 300 °/sec. The amplitude of overt saccades after head rotation remained very small and increased only slightly from 0.6° to 1.5° with increasing head velocity. With a normal VOR, gaze position error at the end of the head movement remained always very small (mean 0.1° ± 0.4).

In patients with UVD, ipsilesional head impulses showed a much shorter latency for the first catch-up saccade that decreased from 215 msec to 141 msec with increasing head velocity (table). Accordingly, the mean percentage of trials with covert saccades increased from 42% to 71%. The amplitude of overt saccades was much larger compared to normal subjects and linearly increased from 1.4° to 5.3° with stimulus magnitude. Over the same range, gaze position error increased from 1.5° up to 10° reaching about twice the amplitude of the consecutive overt saccades. In contralesional head impulses, the percentage of covert saccades and the amplitude of overt saccades were only slightly higher than in normal subjects: The percentage of trials with covert saccades increased from 14% to 20% and the amplitude of overt saccades increased from 0.6° to 2.5° (table e-2). Gaze position error of contralesional head impulses increased from 1° to 4.9° corresponding to about half the gaze position error of ipsilesional head impulses.

Patients with VN showed a qualitatively similar catch-up saccade pattern to patients with UVD. In ipsilesional head impulses, the latency of the first catch-up saccade decreased from 218 msec to 135 msec with increasing head velocity from 50 °/sec to 300 °/sec (table). Consequently, the percentage of trials with covert saccades increased from 32% to 73% over the same range. The amplitude of overt saccades increased from 1.1° to 4.2° and gaze position error rose from 1.3° to 8.2°. Contralesional head impulses in VN showed a saccadic distribution pattern similar to normal subjects. The

Figure 4 Characteristic distribution patterns of catch-up saccades in time observed in the head impulse test



Early covert saccades during head rotation are most likely imperceptible to the clinical observer and act to minimize gaze position error. Late overt saccades after head rotation are detectable by the clinical observer and act to nullify the gaze position error which accumulates during the head movement. (A, D) Normal subject (acceleration gain 0.87 at 6,000 °/sec²) without saccades during the head movement and only occasional small catch-up saccades in the consequent fixation period. Only a small transient gaze position error occurs during the head movement. (B, E) Patient 4 years after vestibular neuritis (VN, gain 0.40) with stereotyped covert catch-up saccades with a mean latency of 72 msec at 300 °/sec on top of the ipsilesional angular vestibulo-ocular reflex response. The covert saccades correct the gaze position error mainly during the head impulses. (C, F) Patient 2 weeks after VN (gain 0.51) with almost no covert saccades during the head movement. Overt catch-up saccades correct the large accumulated gaze position error afterwards. Three-dimensional time series (500 msec) of eye velocity (A–C) and gaze position error (D–F) in response to increasing head impulse stimuli. For detailed numerical data of examples, see table e-1.

percentage of trials with covert saccades increased from 10% to 17% while the amplitude of overt saccades increased from 0.6° to 2.0° (table e-2). In patients with VN, gaze position error of contralesional head impulses was considerably smaller than in patients with UVD and increased from 0.4° to 2.6°.

DISCUSSION Patients with unilateral vestibular disease showed a HIT with reduced ipsilesional VOR gains and compensated for this deficit with catch-up saccades to keep the eye on target. We found that HIT with high head acceleration revealed the VOR deficit better than with low acceleration. We identified two types of catch-up saccades that are important for the interpretation of the bedside HIT: early covert saccades, while the head is still moving, are most likely imperceptible to a clinical observer, whereas late overt saccades are visible as the indirect sign of vestibular deficit.

We found that during HIT in normal subjects the VOR stabilized gaze in space with gains close to unity over the entire tested acceleration range. This has been shown for single head acceleration in humans[2,5] and the linearity of the VOR could be demonstrated for head velocities up to 350 °/sec.[21]

In both of our patient groups, ipsilesional VOR gains decreased steeply with increasing head acceleration. This suggests that vestibular function of patients with unilateral vestibular disease was no longer linear, but deteriorated as the stimuli became more challenging. This relationship of VOR gain as a function of transient head acceleration has only been shown in animals after UVD with a similar decrease of ipsilesional gains from 0.8 to 0.4 with head accelerations between 3,000 and 12,000 °/sec².[22]

Contralesional VOR gains of both our patient groups were slightly but significantly reduced compared to normal subjects and gains declined linearly with increasing acceleration. In contrast to our results, contralesional gains of transient head perturbations over different accelerations were not attenuated after UVD in animals.[22] However, contralesional gain reductions comparable to our study are well established in humans at single head accelerations both after UVD and VN.[6,23-26]

In VN and UVD both ipsilesional and contralesional gain patterns were qualitatively similar. A previous comparison between the two patient groups showed no significant difference

Table Catch-up saccade characteristics of ipsilesional head impulses in patients after unilateral vestibular deafferentation (UVD) and vestibular neuritis (VN) compared to normal subjects (mean ± 95% CI)

Peak head velocity	Normal subjects (n = 2 × 12)	UVD ipsilesional (n = 15)	VN ipsilesional (n = 13)
Latency of first saccade (ms)			
50 °/s	269 ± 25	215 ± 22	218 ± 30
100 °/s	256 ± 27	189 ± 20	188 ± 29
150 °/s	239 ± 26	159 ± 21	160 ± 31
200 °/s	249 ± 28	144 ± 20	141 ± 29
250 °/s	248 ± 35	137 ± 22	137 ± 34
300 °/s	252 ± 35	141 ± 32	135 ± 37
Occurrence of covert saccades (% of trials)			
50 °/s	4 ± 3	42 ± 20	32 ± 17
100 °/s	9 ± 5	47 ± 19	45 ± 19
150 °/s	10 ± 5	55 ± 16	59 ± 20
200 °/s	10 ± 7	66 ± 15	70 ± 22
250 °/s	14 ± 9	73 ± 14	73 ± 23
300 °/s	16 ± 11	71 ± 18	73 ± 24
Overt saccade amplitude (°)			
50 °/s	0.6 ± 0.2	1.4 ± 0.3	1.1 ± 0.2
100 °/s	0.6 ± 0.2	1.9 ± 0.3	1.6 ± 0.3
150 °/s	0.8 ± 0.2	2.9 ± 0.7	2.5 ± 0.6
200 °/s	0.9 ± 0.2	3.5 ± 0.9	3.2 ± 0.7
250 °/s	1.1 ± 0.3	4.3 ± 0.8	3.7 ± 0.9
300 °/s	1.5 ± 0.4	5.3 ± 1.2	4.2 ± 0.9
Gaze position error (°)			
50 °/s (5.9°)*	0.3 ± 0.2	1.5 ± 0.5	1.3 ± 0.4
100 °/s (8.3°)*	0.2 ± 0.3	3.2 ± 0.5	2.3 ± 0.7
150 °/s (12.1°)*	0.0 ± 0.4	4.8 ± 0.9	4.1 ± 1.3
200 °/s (16.3°)*	−0.1 ± 0.5	6.4 ± 1.1	5.2 ± 1.6
250 °/s (21.3°)*	0.1 ± 0.6	7.9 ± 1.3	6.6 ± 1.8
300 °/s (23.8°)*	0.4 ± 0.7	10.0 ± 1.7	8.2 ± 2.4

Covert saccades occur during head rotation and overt saccades occur after head rotation; gaze position error is measured at the end of the head movement.
*Mean head impulse amplitude as reference.

for horizontal canal function at single head acceleration.[6] However, in our study both ipsilesional and contralesional gains were slightly higher in VN resulting in a lower asymmetry at higher accelerations compared to UVD. Presumably this difference reflects residual ipsilesional canal function in some of the patients with VN.

The characteristics of the gain curves might be explained by the push–pull cooperation between the horizontal semicircular canal pair: according to Ewald's second law, excitation of the ipsilateral canal drives the VOR better than inhibition of the contralateral canal.[27] At low head acceleration, the excitation of afferents from the ipsilateral canal and the disfacilitation of afferents from the contralateral canal probably both contribute to the VOR. With increasing acceleration, the contralateral afferents are gradually driven into inhibitory cutoff until predominantly the excitatory afferents of the ipsilateral canal contribute to the VOR.[28] If this excitatory contribution of the ipsilateral canal is missing, the VOR gain of patients is roughly halved at low accelerations and decreases steeply with increasing acceleration. If the disfacilitation of afferents of the contralateral canal is missing, the VOR gain decreases less with increasing acceleration due to its limited contribution at higher accelerations.

The gain curve in normal subjects was not exactly linear, but reached a maximum at about 1,700 °/sec^2. This maximum was clearly missing in the linear contralesional gain curves of patients with UVD and patients with VN. Its absence with the sole contribution of the ipsilateral canal in these patients suggests that the origin of the maximum may arise from an interaction with the contralateral semicircular canal. Possibly, it might reflect the inhibitory cutoff acceleration of the contralateral canal, above which only the ipsilateral canal contributes further to the VOR response.[3,29]

This study demonstrates symmetry of the binocularly measured VOR in healthy subjects over an acceleration range similar to natural head movements in accordance with previous studies.[4,5] Symmetric calibration of the VOR seemed to take priority over the absolute value of gain which varied slightly among individual subjects and depending on the size of the acceleration stimulus. The strong symmetry of HIT in normal subjects with a 95% CI of ±2.3% contrasts with the wide normal range of standard caloric and rotational chair testing: most vestibular laboratories accept a normal range of ±25% for the caloric canal paresis factor and ±30% asymmetry for constant acceleration stimuli on the rotational chair.[30]

Compared to normal subjects, VOR asymmetry of patients with VN and UVD was already significant at the lowest head acceleration and further increased with higher acceleration. A comparison of caloric and quantitative HIT after VN demonstrated that the HIT remained asymmetric even at a chronic stage whereas the caloric canal paresis factor returned to normal in 36% of the patients.[24] Given the tight symmetry range in normal subjects, quantitative HIT allows reliable diagnosis of the affected side, especially at higher accelerations.[7]

While the quantitative HIT directly measures the gain of the VOR, the bedside HIT relies on the observation of the catch-up saccades consequent to the head rotation. Clinical detection of this saccadic response, however, does not directly depend on the reduced VOR gain, but rather on the timing and size of the catch-up saccades relative to the head impulse. To account for this fact, we distinguished between early covert and late overt catch-up saccades in our analysis (video 2). Covert saccades, while the head is still moving, remain most likely imperceptible to the clinical observer, since they cannot be distinguished from the residual VOR response. On the other hand, overt saccades after the head movement could easily be detected by the clinical observer.[12] To assess the visibility of the catch-up saccades for the bedside HIT, we calculated the percentage of trials with covert saccades and measured the amplitude of overt saccades.

We found that the amplitude of overt saccades in patients with unilateral vestibular disease increased linearly with head acceleration (video 1). The average amplitude corresponded only to approximately 20% of the head impulse size. The mean percentage of initial catch-up saccades that were covert, however, increased up to about 70% with increasing head acceleration. Note that an initial covert catch-up saccade did not preclude the occurrence of subsequent overt saccades, but accounted for their reduced amplitude. Thus, covert catch-up saccades might lead to a false-negative bedside HIT and hence reduce the sensitivity of the test, estimated at about 70% according to a recent study.[12]

Tiny overt catch-up saccades occurred in healthy subjects as well and do not indicate a positive HIT. Likewise, some patients with unilateral vestibular loss showed not only large ipsilesional, but also smaller contralesional catch-up saccades. In this case the unilateral vestibular loss could be mistaken for a bilateral vestibular loss, but the asymmetry in amplitude of overt catch-up saccades should indicate the affected side.

Based on our catch-up saccade analysis, we recommend applying head impulses with high acceleration to elicit large overt catch-up saccades and repeating the test about 5 to 10 times to each side to circumvent possible covert catch-up saccades. For the future, this predicament calls for the development of readily available high-speed video systems to measure the VOR, if the bedside HIT is not conclusive.

The absence of a positive bedside HIT is the red flag in the assessment of a patient with a single, prolonged attack of acute, isolated spontaneous vertigo.[31] It should direct the clinician to suspect a cerebellar infarction and prompt further investigation with an MRI of the brain. In this context, a false negative HIT caused by covert catch-up saccades may lead to an unnecessary MRI examination, but no vital diagnosis would be missed. This is in contrast to a false positive HIT which could create a false sense of security of dealing with a peripheral vestibular disease in a patient with a cerebellar infarction.

Vestibular catch-up saccades with latencies as short as 70 msec, like we exemplified in our study, have been previously reported using transient mechanical whole-body rotation.[9] Note that these latencies are substantially shorter than those of the fastest visually guided saccades, called express saccades, with latencies of about 100 msec.[32] The significance of catch-up saccades with such short latencies for the interpretation of the bedside HIT has been recognized before, but only for active head impulses performed by the subject.[15] On average, these active head impulses elicited catch-up saccades about 130 msec earlier than passive head impulses.

The shorter the latency of the catch-up saccades in patients, the less gaze direction deviates from the target during the head impulse.[9] As exemplified in our two patients after VN, covert saccades during the head movement greatly reduce the gaze position error by the end of the head impulse. By correcting gaze position early, covert catch-up saccades most probably help patients to reduce oscillopsia.[33]

The question of what triggers those catch-up saccades has been previously addressed. In fact, it has been shown that the short latency could not be attributed to visual feedback or anticipation and concluded that those catch-up saccades are most likely triggered by vestibular input.[8,9] Clearly, the stimulus would have to come from the contralateral vestibular organ in patients after UVD. Given the very large number of stimuli delivered during our experiment, the subjects may have the potential to learn to trigger covert saccades more frequently, compared to the bedside HIT, where a limited number of stimuli are delivered.

It has been suspected that an adaptation period after the onset of a vestibular deficit might be necessary to develop early catch-up saccades.[9] In fact, one of our example patients showed no covert saccades 2 weeks after VN. But then another of our patients showed a very similar pattern 10 years after VN. One of our patients had been

measured twice after vestibular nerve section and showed an almost identical pattern with frequent covert saccades 2 weeks and 3 months postoperatively. Taken together, the saccadic distribution pattern in time seemed to be an individual fingerprint rather than a function of disease duration in our mostly chronic patient group. Further longitudinal studies of acute patients are necessary to demonstrate the adaptation process to a vestibular deficit with covert saccades.

ACKNOWLEDGMENT

The authors thank Dominik Straumann for suggestions, Anthony Anderson and Geoffrey Smith for help with recording the patient videos, Satendra Pratap for technical support, and all the participants of the study.

Received May 23, 2007. Accepted in final form August 6, 2007.

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Head impulse test in unilateral vestibular loss: Vestibulo-ocular reflex and catch-up saccades

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Neurology 2008;70;454-463
DOI: 10.1212/01.wnl.0000299117.48935.2e

Impulsive Testing of Semicircular-Canal Function Using Video-oculography

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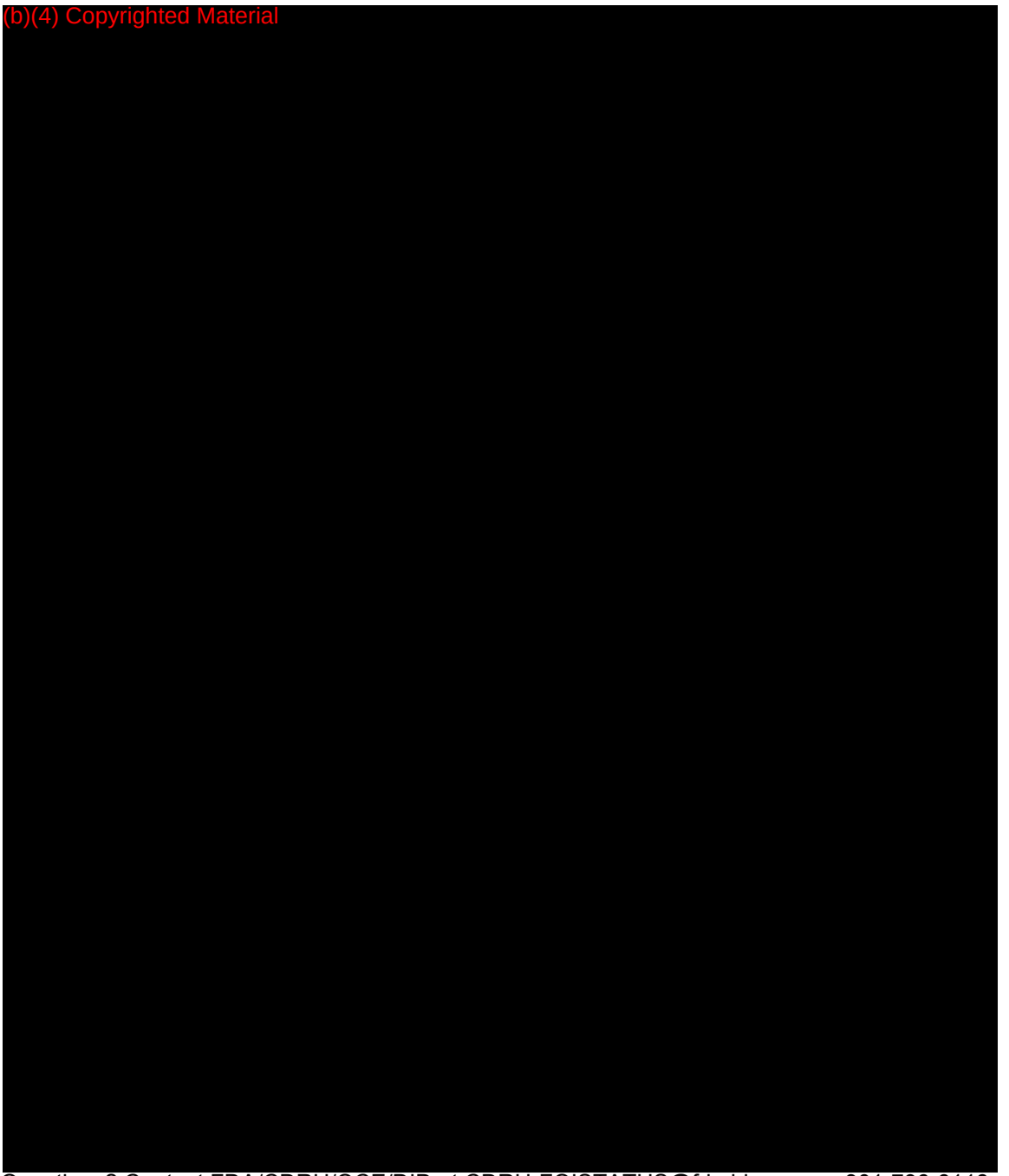

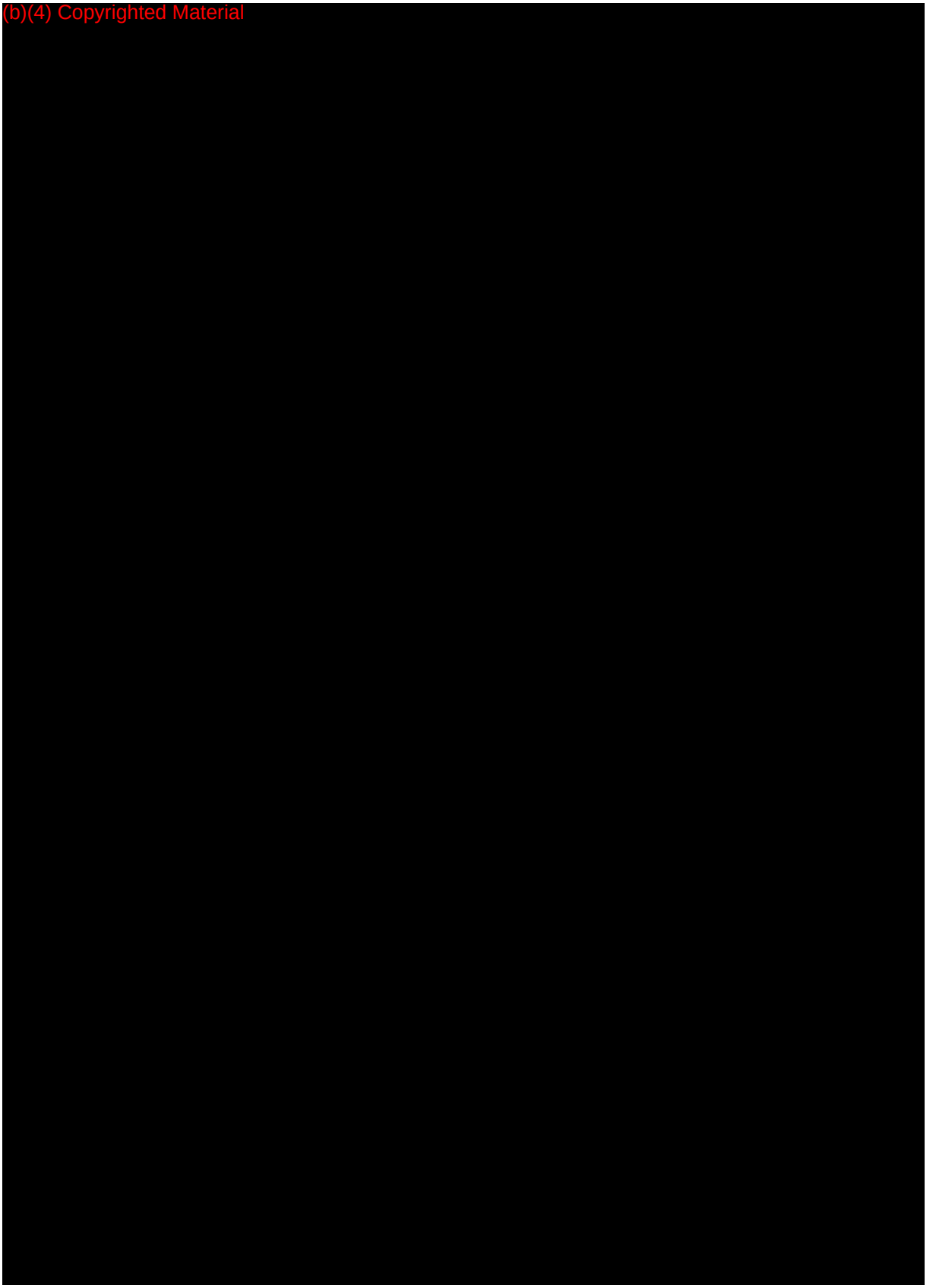

Research Report

Optimizing the Sensitivity of the Head Thrust Test for Identifying Vestibular Hypofunction

Background and Purpose. The head thrust test (HTT) is used to assess the vestibulo-ocular reflex. Sensitivity and specificity for diagnosing unilateral vestibular hypofunction (UVH) in patients following vestibular ablation is excellent (100%), although sensitivity is lower (35%–39%) for patients with nonsurgically induced UVH. The variability of the test results may be from moving the subject's head outside the plane of the lateral semicircular canals as well as using a head thrust of predictable timing and direction. The purpose of this study was to examine sensitivity and specificity of the horizontal HTT in identifying patients with UVH and bilateral vestibular hypofunction (BVH) when the head was flexed 30 degrees in attempt to induce acceleration primarily in the lateral semicircular canal and the head was moved unpredictably. **Subjects.** The medical records of 176 people with and without vestibular dysfunction (n=79 with UVH, n=32 with BVH, and n=65 with nonvestibular dizziness) were studied. **Methods.** Data were retrospectively tabulated from a de-identified database (ie, with health information stripped of all identifiers). **Results.** Sensitivity of the HTT for identifying vestibular hypofunction was 71% for UVH and 84% for BVH. Specificity was 82%. **Discussion and Conclusion.** Ensuring the head is pitched 30 degrees down and thrust with an unpredictable timing and direction appears to improve sensitivity of the HTT. [Schubert MC, Tusa RJ, Grine LE, Herdman SJ. Optimizing the sensitivity of the head thrust test for identifying vestibular hypofunction. *Phys Ther.* 2004;84:151–158.]

Key Words: *Head thrust test, Sensitivity and specificity, Vestibular hypofunction.*

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Individuals who sustain damage to their vestibular systems may experience vertigo, disequilibrium, gait ataxia, postural instability, and blurred vision during head motion (oscillopsia). One cause of oscillopsia is a deficient vestibulo-ocular reflex (VOR). The deficient VOR can reduce visual acuity during head rotation.[1] Because of the direct relationship between function of the vestibular receptors in the inner ear and eye movements produced by VORs, the bedside examination of eye movements can be of great importance in defining and localizing vestibular pathology.[2]

The head thrust test (HTT) has been widely accepted as a clinical test that is used to assess the angular vestibulo-ocular reflex.[3–7] During the HTT, the patient is asked to focus his or her eyes on a target. Next, the patient's head is gently grasped, and a small-amplitude (5°–10°) but high-acceleration (3,000–4,000°/s/s) thrust is applied by the examiner. Once the head stops moving, the eyes are observed for a corrective saccade. The corrective saccade is a rapid eye motion that returns the eyes toward the target and indicates a decreased gain (eye velocity/head velocity) of the VOR. Individuals with normal vestibular function do not use corrective saccades after the HTT (the eyes stay fixed on the target) (Figure, photographs A and B). People with vestibular hypofunction may use a corrective saccade after the head is thrust toward the side of the hypofunction (Figure, photographs C–E).

The specificity of the HTT for identifying lateral semicircular canal pathology for patients with unilateral vestibular hypofunction (UVH) is high (95%–100%)[3,8–11] yet the sensitivity is variable. For patients with complete UVH due to nerve section, the sensitivity and specificity are 100%.[3,8] For patients with nonsurgically induced unilateral hypofunction, a group that more accurately reflects a clinical population, the HTT has a sensitivity of 34% to 39% and a specificity of 95% to 100%.[9–11]

We believe the technique used to perform the horizontal HTT may be the cause of the low sensitivity in patients with UVH due to causes other than nerve section. We reasoned that position of the head and the unpredictability of the stimulus (random direction and random onset of head rotation) would be important components in identifying a peripheral vestibular lesion using the HTT. In none of the prior studies investigating the validity of data obtained with the HTT[3,8–11] did the authors state that the head was placed in a starting position of 30 degrees pitched down, a position that might optimize the acceleration signal being induced exclusively through the lateral semicircular canal. We hypothesized that if the horizontal HTT is done without initially pitching the head 30 degrees down, the head acceleration signal may not be isolated within the lateral semicircular canals. The vertical semicircular canals (anterior and posterior semicircular canals), therefore, may detect the head rotation signal and prevent cutoff of inhibitory input from the contralesional horizontal vestibular afferents.[12–14] Cutoff of the inhibitory input has been offered as an explanation for the positive HTT.[3,4]

Some researchers[9,10] administered the HTT with predictable timing. We have shown that when subjects with UVH made a predictable (volitional) head thrust, they generated a unique type of saccade more often than during an unpredictable (passive) head thrust.[15] This

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Each of the authors contributed to concept/research design and writing of the manuscript. Dr Schubert and Mr Grine performed data collection and analysis. Dr Tusa and Dr Herdman provided subjects, project management, and facilities/equipment. Dr Schubert, Dr Tusa, and Dr Herdman provided fund procurement. Dr Tusa, Mr Grine, and Dr Herdman provided consultation (including review of manuscript before submission). The authors thank John P Carey, MD, for assistance in generating the Figure.

Informed consent was obtained for a subset of the subject population from the Institutional Review Board of Emory University.

Preliminary findings of this work were presented at the Combined Sections Meeting of the American Physical Therapy Association, February 14–18, 2001, San Antonio, Tex, and at the American Academy of Neurology meeting, April 13–20, 2002, Denver, Colo.

This study was supported by the Foundation for Physical Therapy (MCS) and by National Institute on Deafness and Other Communication Disorders grant 03196 (SJH, RJT).

This article was received April 4, 2003, and was accepted September 4, 2003.

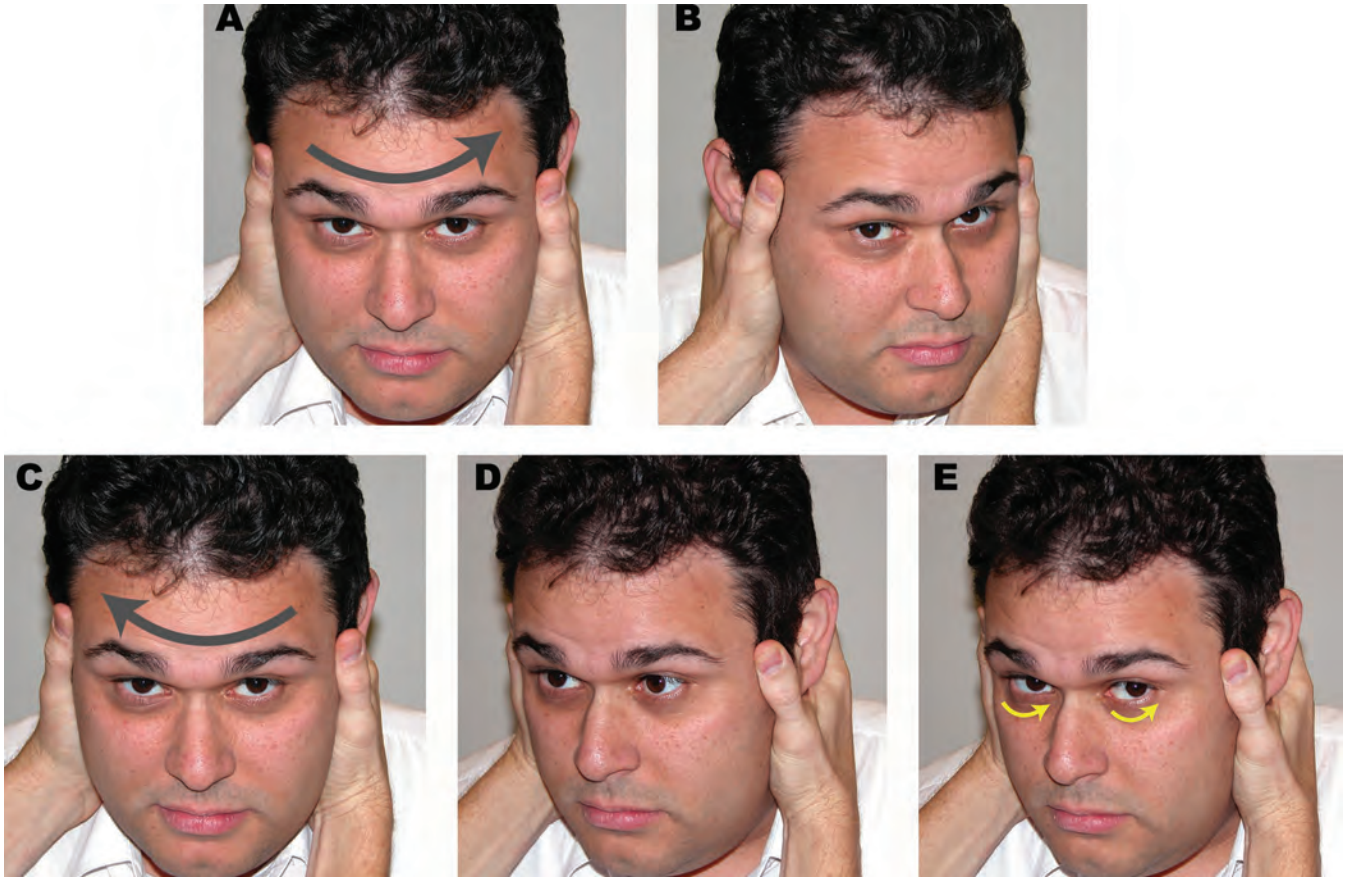

Figure.
Normal head thrust test to the left (A and B), abnormal to the right (C–E). Large arrow denotes direction the head will be thrust. (A) Initial starting position places subject's head into cervical flexion; eyes are focused on the target. (B) Upon stopping the head thrust, the eyes are still on target and no corrective saccade is observed. In photographs A and B, the subject's eyes stay fixed on the examiner's nose throughout the test. (C) Initial starting position places subject's head into cervical flexion; eyes are focused on the target. (D) As the head is thrust rapidly to the right, the eyes fall off the target and move with the head. (E) The subject must make a corrective saccade (small arrows) to bring the eyes back to the target of interest.

saccade occurred during the head thrust and was in the opposite direction of the moving head (preprogrammed saccade). The preprogrammed saccade returned the eye to the center of the orbit, often eliminating the need for a corrective saccade. This behavior, in part, also might explain the reduced sensitivity of the HTT.

The primary purpose of our study was to examine sensitivity and specificity of the horizontal HTT to identify patients with UVH when the test was done in a manner attempting to induce the acceleration signal through the lateral semicircular canals and to limit the effects of prediction (head pitched in 30° of cervical flexion and moved unpredictably). In addition, we report the sensitivity and specificity of the HTT to identify patients with bilateral vestibular hypofunction (BVH), which has not been previously reported.

Method

Subjects

Subjects were individuals complaining of dizziness or imbalance who were initially seen in a tertiary care facility from 1995 to 2001 by a neurologist specializing in dizziness and balance disorders. The neurologist was experienced in administering the HTT. Subjects were selected based on a retrospective chart review, and all data were obtained from their medical records. Inclusion criteria consisted of the subjects complaining of dizziness or imbalance and having undergone vestibular function testing. Exclusion criteria consisted of having a history of benign paroxysmal positional vertigo, anxiety disorders, cervical spine pathology, ocular malalignment, central nervous system pathology, or excessive alcohol use (greater than 59.13 mL [2 oz] of 100% alcohol per day).

Based on patient data obtained from the charts, which included the results of vestibular function testing, sub-

Table 1.
Group Characteristics[a]

	Subjects With Nonvestibular Dizziness	Subjects With Unilateral Vestibular Hypofunction			Subjects With Bilateral Vestibular Hypofunction		
		All	iUVH	cUVH	All	iBVH	cBVH
N	65	79	45	34	32	21	11
Age (y)							
$\bar{X}$	64.4	65.3	60.6	64.7	66.7	66.3	66.1
SD	16.8	16.2	14.1	17.6	13.3	18.7	6.5
Range	29–87	29–87	29–85	30–87	29–91	29–91	53–75
Sex ratio (male:female)	21:44	33:46	21:24	13:21	15:17	11:10	5:6

[a] i=incomplete (nystagmus reversal with a change in caloric temperature), c=complete (no response to ice water irrigation), UVH=unilateral vestibular hypofunction (ipsilesional), BVH=bilateral vestibular hypofunction.

jects were categorized as belonging to 1 of 3 groups: (1) subjects with UVH, (2) subjects with BVH, and (3) subjects with nonvestibular dizziness. This was done to determine the effectiveness of the clinical head thrust based on severity of hypofunction. Subjects with a diagnosis of UVH had a difference in slow component eye velocity of greater than 25% between the right and left sides on the caloric test (>25% unilateral weakness). Subjects with a diagnosis of BVH demonstrated a slow component eye velocity of less than 5°/s to cold, warm, and ice water irrigation in each ear and a gain of less than 0.1 on the rotary chair test (240°/s constant velocity). Typically, only subjects with BVH underwent both rotary chair and caloric testing. Subjects assigned to the nonvestibular dizziness group had complaints of dizziness but normal vestibular function test results.

Subjects with vestibular hypofunction were assigned to subcategories based on whether they exhibited incomplete or complete hypofunction as determined by caloric examination. Based on their medical records, patients with incomplete vestibular hypofunction demonstrated reversal of nystagmus between cold and warm water caloric irrigations. Patients with complete vestibular hypofunction showed neither reversal nor a response to ice water irrigation.

Data for a total of 176 subjects in all 3 groups were included in the overall analysis. The group characteristics are shown in Table 1. No difference in age was found across the groups (analysis of variance [ANOVA] $F=2.4$, $P=.64$).

Procedure

Data were obtained retrospectively from medical records from the Dizziness and Balance Centers of Emory University and the University of Miami. The rights of the patients were protected by 1 of 2 means: (1) Patient data were tabulated from a de-identified database (n=170), or (2) informed consent was obtained (n=6). The term "de-identified" means that the health information was stripped of all identifiers so that the health information does not identify an individual and does not present any reasonable basis by which the information can be used to identify the individual. Data from the de-identified database were originally collected before our intent to investigate sensitivity and specificity of the HTT.

All data were de-identified. Data collected included information regarding diagnoses, vestibular function test results, HTT results, and subject age. Individuals with the following peripheral vestibular diagnoses were included in the study: vestibular neuritis, vestibular ototoxicity, Ménière disease, vestibular nerve section due to vestibular schwannoma or Ménière disease, labyrinthitis, and idiopathic vestibular hypofunction.

For the purposes of clarity, all references to the HTT in this report refer to testing in the horizontal plane. Based on medical records, one investigator routinely did the HTT as part of the initial clinical examination before vestibular function testing was performed. This investigator was experienced in administering the HTT. The subjects were instructed to look at the investigator's nose (distance of 38 cm [15 in]). The investigator's nose was chosen because it provided a convenient near target. Individuals with vestibular hypofunction have more difficulty generating an adequate response for a near target because vergence of the eyes requires the gain of the VOR to be larger.[16–18]

A typical head thrust test was administered by first placing a subject's head into 30 degrees of cervical flexion. This was done using anatomical landmarks (imaginary line between the inferior rim of the ocular orbit through the external acoustic meatus) to match the lateral semicircular canal in situ. The subject's head would then be moved unpredictably to the right or left from center. The examiner attempted to keep the head thrust unpredictable by moving the head in a manner

that was random in direction and timing of onset. Total amplitude of head rotation was approximately 5 to 10 degrees. After each HTT, the head was slowly moved back to center. The test was repeated in each direction 3 times. Individuals with a positive head thrust were identified as having a corrective saccade in at least 2 of the 3 thrusts (toward one ear if the subject had a diagnosis of UVH and toward both ears if the subject had a diagnosis of BVH).

Sensitivity, specificity, positive and negative predictive values, likelihood ratios, and accuracy were calculated using the following formulas[19]:

$$(1)\quad Sensitivity = (True\ positives/[True\ positives + False\ negatives]) \times 100 = \%$$

$$(2)\quad Specificity = (True\ negatives/[True\ negatives + False\ positives]) \times 100 = \%$$

$$(3)\quad Positive\ predictive\ value = (True\ positives/[True\ positives + False\ positives]) \times 100 = \%$$

$$(4)\quad Negative\ predictive\ value = (True\ negatives/[True\ negatives + False\ negatives]) \times 100 = \%$$

$$(5)\quad Accuracy = (True\ negatives + True\ positives)/Total \times 100 = \%$$

$$(6)\quad Positive\ likelihood\ ratio = Sensitivity/(1 - Specificity)$$

$$(7)\quad Negative\ likelihood\ ratio = (1 - Sensitivity)/Specificity$$

"True positives" were those subjects with confirmed vestibular hypofunction (UVH or BVH) based on abnormal caloric or rotary chair test results and who had a positive HTT. "True negatives" were those subjects with normal vestibular function based on caloric or rotary chair test results and who had a negative HTT. "False negatives" were those subjects with confirmed vestibular hypofunction who had a negative HTT. "False positives" were those subjects with confirmed normal vestibular function who had a positive HTT. Sensitivity and specificity for subjects with vestibular hypofunction was assessed using subjects with nonvestibular dizziness as the criterion reference point. We chose subjects with complaints of dizziness and normal vestibular function as the criterion for comparison (instead of subjects with normal vestibular function and no complaints of dizziness) to provide what we believe is a more clinically appropriate assessment of test validity. Analysis of variance was used to assess differences in age ($P<.05$).

Table 2.
Contingency Table for All Subjects[a]

	Positive Dx	Negative Dx	Total
Positive head thrust	Nonvestibular dizziness=0 UVH=56 iUVH=26 cUVH=30 BVH=27 iBVH=16 cBVH=11	Nonvestibular dizziness=12 UVH=0 iUVH=0 cUVH=0 BVH=0 iBVH=0 cBVH=0	95
Negative head thrust	Nonvestibular dizziness=0 UVH=23[b] iUVH=19 cUVH=4 BVH=5 iBVH=5[c] cBVH=0	Nonvestibular dizziness=53 UVH=0 iUVH=0 cUVH=0 BVH=0 iBVH=0 cBVH=0	81
Total	111	65	176

[a] i=incomplete (nystagmus reversal with a change in caloric temperature), c=complete (no response to ice water irrigation), UVH=unilateral vestibular hypofunction (ipsilesional), BVH=bilateral vestibular hypofunction, positive Dx=positive diagnosis (positive caloric test [UVH and BVH] and rotary chair test [BVH]), negative Dx=negative diagnosis (negative caloric test [all 3 groups] and rotary chair test [BVH]), positive head thrust=corrective saccade observed after head movement stopped.
[b] No subject with UVH had a positive head thrust in both directions.
[c] Subjects with iBVH had a negative head thrust in one direction and a positive head thrust in opposite direction.

Results

Sensitivity and Specificity to Identify Peripheral Vestibular Hypofunction

Unilateral vestibular hypofunction. The contingency table for tabulating the sensitivity and specificity of the HTT to identify peripheral vestibular hypofunction according to the patient's HTT and diagnostic classification is presented in Table 2. Fifty-six of 79 subjects with UVH had a positive ipsilesional HTT, resulting in a combined sensitivity of 71% (incomplete and complete lesions). Of the 23 subjects with UVH who had a negative HTT, only 4 had a complete loss of vestibular function unilaterally. The difference in sensitivity for subjects with incomplete and complete UVH was 58% versus 88% (Tab. 3).

Bilateral vestibular hypofunction. Twenty-seven of 32 subjects with BVH had a bilaterally positive HTT, resulting in a combined sensitivity of 84% (incomplete and complete lesions). The other 5 subjects with BVH had a positive HTT in one direction only and were found to have incomplete loss of vestibular function. Similar to the sensitivity of the HTT for subjects with UVH, sensitivity improved depending on extent of the hypofunc-

Table 3.
Validity of the Data Obtained With the Head Thrust Test (HTT) for Identifying Subjects With Peripheral Vestibular Hypofunction Based on Extent of Lesion[a]

Validity Measures	All Subjects	All Subjects With UVH	All Subjects With BVH	Incomplete		Complete	
				UVH	BVH	UVH	BVH
Sensitivity (%)	75	71	84	58	76	88	100
Specificity (%)[b]	82						
PV+	87	82	69	68	57	71	48
PV−	65	70	91	74	91	93	100
LR+	4.16						
LR−	0.30						
Accuracy (%)	77	76	82	55	71	58	66

[a] Incomplete=nystagmus reversal with a change in caloric temperature, complete=no response to ice water irrigation, UVH=unilateral vestibular hypofunction (ipsilesional), BVH=bilateral vestibular hypofunction, PV+=positive predictive value, PV−=negative predictive value, LR+=positive likelihood ratio, LR−=negative likelihood ratio.
[b] Only subjects with nonvestibular dizziness (n=12) were found to have false positive HTT, resulting in overall specificity of 82%.

tion (76% for incomplete BVH and 100% for complete BVH) (Tab. 3).

All subjects. Eighty-three of 111 subjects with peripheral vestibular hypofunction (UVH and BVH) had a positive HTT in at least one direction. No subjects with nonvestibular dizziness were found to have a positive HTT and a positive vestibular function test. As a result, the overall sensitivity of the HTT in identifying subjects with vestibular hypofunction was 75% (Tab. 3). Twelve subjects with nonvestibular dizziness were found to have a false positive HTT. As a result, the overall specificity of the HTT to rule out vestibular hypofunction was 82%.

Discussion

Sensitivity and Specificity of the HTT

The method we used for administering the HTT to identify subjects with varying degrees of UVH had a sensitivity of 71%, as compared with sensitivity values of 34% to 39% previously reported.[9–11] We believe the improved sensitivity may be due to 2 factors. First, performing the HTT with the head pitched 30 degrees down places the lateral semicircular canals in the plane of rotation. If the HTT is done with the neck in a neutral position (no cervical flexion), the head acceleration may be distributed among the vertical semicircular canals.[14] We believe that, as a result, peripheral vestibular afferents and central vestibular neurons of the intact lateral semicircular canals are exposed to less acceleration and therefore are less likely to reach inhibitory cutoff. Second, ensuring that the head thrust is unpredictable in timing and direction may prevent preprogrammed saccades and the effects of prediction. When a head rotation is predictable, the gain of the VOR is greater compared with an unpredictable head rotation.[20–23] These phenomena have been suggested to be due, in part, to the central nervous system's ability to generate an appropriate VOR once the brain is able to predict the intended head rotation.[22–24] Some researchers[9,10] have done the HTT with predictable timing. Patients with vestibular hypofunction can generate preprogrammed eye movements during predictive head movements to stabilize gaze.[15] Preprogrammed eye movements may eliminate the need of corrective saccades, which can decrease the sensitivity of the HTT. Preprogrammed saccades have been shown to occur during predictable head thrusts as well as during pseudorandom whole-body rotations.[15,25] The effects of central preprogramming also have been suggested as the mechanism for improved visual acuity during predictable head rotation in patients with vestibular hypofunction.[26] We believe making the HTT unpredictable improves sensitivity of the HTT, in part, because the effects of central preprogramming are reduced.

Sensitivity of the HTT Related to Extent of Pathology

We found that the HTT was more sensitive in identifying subjects with complete versus incomplete loss of vestibular function. The HTT was more sensitive in categorizing subjects with complete BVH (100%) than in categorizing subjects with complete UVH (88%). Subjects with either UVH or BVH might be expected to have similar occurrences of corrective saccade use (and therefore similar sensitivities using the HTT), considering each group had a complete lesion as defined by caloric and rotary chair testing. One explanation may be the extent of the lesion. Because patients with BVH have a more extensive injury than those with UVH,[27] the response range of the VOR for head acceleration may be smaller for people with BVH. In contrast, people with UVH have intact contralateral peripheral vestibular afferents that we believe can respond to a broader range of head accelerations.

Limitations of Studies Comparing Results of the HTT and the Caloric Test

Twenty-two percent (12/53) of our subjects with dizziness not associated with vestibular dysfunction had a

positive HTT but negative caloric or rotary chair testing results. This finding reduced the overall specificity of the HTT to 82%. One explanation is that these 12 individuals may have a high acceleration defect in their VOR that could not be detected by the caloric testing. In our clinical experience, a small number of people with normal vestibular function based on caloric and rotary chair examinations have a positive head thrust test and complain of dizziness or imbalance only during rapid head motion. We have used the term "high acceleration defects of the VOR" to identify these individuals with normal vestibular function tests yet reduced performance of the VOR during rapid head accelerations. A more complete battery of vestibular tests (ie, one that includes accelerations at middle and high frequencies) may be needed to test this hypothesis.

An additional explanation for the reduced specificity of the HTT relates to differences with the caloric test. Although, the caloric test is recognized as the most useful test for identifying individuals with suspected peripheral UVH,[28,29] the information provided by caloric testing is dissimilar to the information provided by the HTT. The caloric test generates nystagmus as a result of an unnatural stimulation of the semicircular canals that is equivalent to a very-low-frequency head rotation. In contrast, the HTT represents a natural and high-acceleration head rotation. The caloric test, therefore, does not test the lateral semicircular canals with a stimulus equivalent to the HTT. As a result, factors other than technique used to perform the HTT likely contribute to the variability of the sensitivity of the HTT to identify individuals with UVH.

Importance of Training and Experience in Performing the HTT

In our study, a clinician with more than 20 years of experience administered the HTTs. We believe that proper training is necessary in order to perform the HTT correctly. Proper technique includes correct head position, inducing a rapid head thrust through a small amplitude, and making sure the head thrust is unpredictable both in direction and timing. We encourage all practitioners to study with someone who is skilled in performing the test.

Limitations of the Study

We did not examine the reliability of our examiner's findings or whether differences would have been found with multiple examiners. Similarly, we did not investigate whether proper training is necessary to perform the head thrust correctly. We believe, however, that performing the head thrust test is a learned skill.

Conclusion

The sensitivity of the HTT for identifying individuals with UVH is good when the head is thrust unpredictably in the plane of the lateral semicircular canals (keeping the head in 30° of cervical flexion). This degree of sensitivity is considerably improved compared with previous established values, although it is not sensitive enough to replace vestibular function testing. The HTT is more sensitive in patients with BVH. Correctly assessing the VOR with the HTT is an essential component of the clinical examination of the peripheral vestibular system. Proper position of the head, ensuring that the head thrust is unpredictable, and experience of the clinician are likely the most critical components for administering the HTT.

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Eur Arch Otorhinolaryngol
DOI 10.1007/s00405 010 1442 5

LETTER TO THE EDITOR

Rapid fluctuations in dynamic semicircular canal function in early Ménière's disease

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Received: 4 November 2010 / Accepted: 16 November 2010

Dear Sir,

Ménière's disease (MD) is characterized by fluctuations in labyrinthine function which are well known and objectively established for the auditory symptoms [1, 2]. It is also well known that it is disorders of balance, rather than hearing, which are the major symptoms during the early stages of the disease [3]. But to date there have been only a few measurements of the fluctuations in vestibular function around the time of the attack. This has been due to two factors. First, the difficulty of testing early MD patients around the time of their attack, which can have highly variable duration: each attack may last from 10 min to hours [2, 3]. Second, the limited range of vestibular tests available and the fact that the usual tests of vestibular function are so demanding that they are not feasible in patients around the time of the attack.

However, recently, we published results of a new non-demanding test of otolith function the n10 of the ocular vestibular-evoked myogenic potential which showed that there are fluctuations in vestibular function, with enhanced dynamic utricular function at the time of the attack compared to quiescence [4]. Here, we wish to address the complementary question as to whether dynamic semicircular canal function fluctuates as auditory and dynamic otolith function does and we present evidence of variations in dynamic semicircular canal function around the time of the MD attack.

The development of the video head impulse test (vHIT) has allowed non-demanding objective measures of semicircular canal function [5]. This is a very simple, fast way of measuring dynamic semicircular canal function accurately and has been validated by directly comparing it to simultaneous measures by the "gold standard" search coil test [6]. The gain measurements of the two tests are not significantly different and show very high concordance correlations [6]. With vHIT it is possible to test patients very quickly at short intervals and this kind of easily repeatable, high accuracy, minimally demanding test allows the measurement of the sequential changes in semicircular canal function at the time of the attack.

The vHIT test involves the clinician delivering brief, passive, high acceleration head impulses of yaw head rotation unpredictably to the right or left through an angle of about 10° 20° while the patient is instructed to keep looking at an earth-fixed target. The patient wears a set of minimal-slip goggles to which is attached a small lightweight high speed video camera to measure eye position and a 3-d sensor to measure head velocity. We used vHIT to measure the yaw VOR response of patients with evidence of early MD, both at quiescence and during an acute attack. Here, we report that the repeated tests at short intervals show that the VOR response changes substantially around the time of an attack (Fig. 1).

One important issue is that the patients for this study were a homogeneous group with early MD (6 subjects, 3

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Published online: 14 December 2010

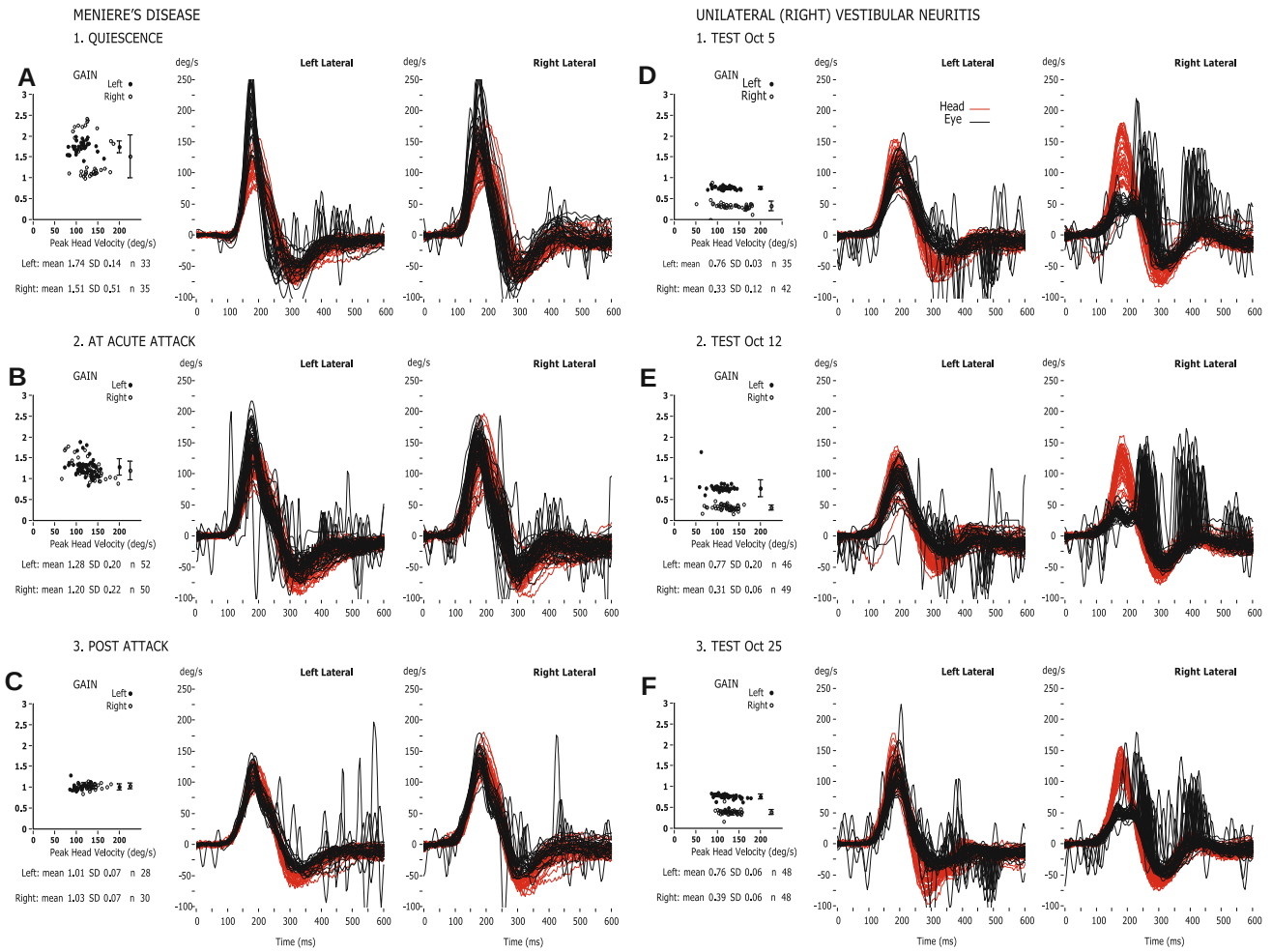

Fig. 1 Superimposed time series of eye velocity responses (*black*) to brief unpredictable horizontal angular head rotations (*red traces*). The *first column* shows successive measures in the same early MD patient from testing at quiescence (**a**), to test around the time of an attack (**b**), to test just after an attack (**c**). There are very large changes in the eye velocity response, even though the head velocity stimulus is comparable across the three occasions. These recordings are matched with three recordings (**d f**) from a patient with unilateral vestibular neuritis (VN) obtained on three different occasions, to show how stable the VOR is at repeated measures in vestibular neuritis in contrast to the rapid fluctuations in the VOR in the MD patient

males and 3 females, 26 64 age range) meeting the AAO-HNS criteria for stage 1 and 2 MD and who had at least two or more vertigo attacks [7]. Other studies in this area are difficult to interpret because of inhomogeneity of the sample of MD patients: for example, early MD patients frequently are not considered or their results are mixed with data obtained from stage 3 "burnt out" MD patients.

For every patient, a detailed medical history was taken and all cases were also assessed by means of a series of audio-vestibular tests: audiometric examination with accurate bone conduction threshold evaluation even if the air conduction threshold was within normal limits, tympanometry, speech audiometry, stapedial reflexes, caloric vestibular test (modified Fitzgerald-Hallpike) and Bone Conducted cervical and ocular VEMPs. The definitive diagnosis was established after the execution of high resolution CT scans and magnetic resonance imaging (MRI) of the posterior cranial fossa using paramagnetic contrast enhancement in all patients of our series to rule out other diseases. These patients then were tested with vHIT in various phases of their disease; quiescence; attack; just after attack, with at least one measure for each phase. Patients were free of any medication during the data acquisition period.

Dynamic VOR gain is very stable in healthy subjects [6] and to address the issue of repeatability of vHIT measures in patients with vestibular pathology, we contrast the results of the repeated testing of early MD patients (see Fig. 1a c, for an example of data from one MD patient) with repeated testing of a patient with vestibular neuritis (VN) in which disease the patient's loss of vestibular function persisted, relatively unchanged for days or weeks (Fig. 1d f).

Figure 1 shows the typical result; there are large changes in dynamic semicircular canal function from quiescence to attack. At quiescence MD patients are free of any sign of vertigo but vHIT measures show their VOR gain is typically enhanced (Fig. 1a). At attack, vertigo is intense and there is a characteristic rotatory spinning or a rocking sensation which may be associated with nausea and vomiting, which persists from 20 min to 24 h duration. There is a decrease in VOR gain around the time of the acute MD attack (Fig. 1b) and a gain value close to normal shortly after (see Fig. 1c). We stress that these rapid fluctuations in VOR performance are not found in healthy subjects or patients with other vestibular pathologies (e.g. repeatable data from the vestibular neuritis patient, see Fig. 1d f).

The cause of these rapid fluctuations in vestibular dynamic function is not known, but we think it is valuable to appreciate that rapid changes do occur around the time of the attack, as our objective measures show. These rapid changes in VOR function appear to correspond to some of the rapid changes in auditory function around the time of an attack [1].

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Semicircular canal plane head impulses detect absent function of individual semicircular canals

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Summary

We studied the human vestibulo ocular reflex (VOR) in response to head 'impulses' brief, unpredictable, passive, high acceleration (up to 4000°/s^2), low amplitude (20 30°) head rotations. We delivered the head impulses approximately in the plane of the semicircular canal (SCC) being tested. To test the anterior and posterior SCCs, the head impulses were delivered in a diagonal plane, midway between the frontal (roll) and sagittal (pitch) planes. We recorded head and eye position in three dimensions with scleral search coils in nine normal subjects, seven patients following unilateral surgical vestibular neurectomy and three patients following unilateral posterior SCC occlusion. In the post surgical patients we demonstrated a severe, permanent VOR gain deficit (0.2 0.3) for head impulses directed toward any single non functioning SCC. The sensitivity of the test depends on the physiological properties of primary vestibular afferents, and its specificity depends on the anatomical orientation of the SCCs. The diagonal head impulse is the first test of individual vertical SCC function in humans, and together with the horizontal head impulse, forms a comprehensive battery of SCC plane tests. These canal plane impulses could be useful in evaluating patients with vertigo or other vestibular disorders.

Keywords: vertical vestibulo ocular reflex; head impulse; semicircular canal

Abbreviations: LARP left anterior SCC/right posterior SCC (plane); RALP right anterior SCC/left posterior SCC (plane); SCC semicircular canal; uPCO unilateral posterior SCC occlusion; uVD unilateral vestibular deafferentation; VOR vestibulo ocular reflex

Introduction

Vestibular disorders are common and often difficult to characterize, because there are no clinical or laboratory tests for all of the various elements of the vestibular end organ. The vestibular end organ consists of five elements in each ear: the lateral, anterior and posterior semicircular canals (SCCs), which transduce angular acceleration, and the two otolith organs, the utricle and saccule, which transduce linear acceleration. We can test the function of the lateral SCC using thermal or rotational stimuli, but currently there is no test for individual anterior or posterior SCC function in humans.

The main bedside test of SCC function is the head 'impulse' test, which is limited to the lateral canals (Halmagyi and Curthoys, 1988). To perform this test, the examiner rapidly turns the patient's head to the right or the left through 20 30 , and instructs the patient to keep staring straight ahead. The patient's ability to maintain visual fixation is a measure of their horizontal vestibulo ocular reflex (VOR) gain. If the VOR gain is unity, the compensatory vestibular eye rotation has exactly the same speed as the head impulse, but in the opposite direction, so the patient will be looking straight ahead at the end of the impulse. If the VOR gain is low in one direction, the patient's gaze will be dragged in the direction of the head impulse. Because he had been told to look straight ahead, he will make a voluntary corrective eye movement back to the original fixation point, and it is this late saccadic eye movement that the clinician detects. We have confirmed these clinical findings with head and eye movement recordings, which demonstrate a severe VOR gain deficit (0.2 0.3) during horizontal head impulses directed

toward a lesioned lateral SCC (Cremer *et al*., 1988). The two lateral SCCs are approximately co planar with each other (Blanks *et al*., 1975*a*), and the horizontal head impulse is therefore a 'canal plane stimulus', because it is a head rotation in the plane of the lateral SCCs. This means that a horizontal head impulse will stimulate the lateral SCCs maximally. Despite the usefulness of the horizontal head impulse test, the corresponding vertical (pitch and roll) impulses have not been as helpful in diagnosing a lesion of individual vertical SCCs. Pitch head impulses are delivered in the sagittal plane about the inter aural axis, and roll impulses are delivered in the frontal plane about the naso occipital axis.

By recording head and eye rotation during pitch impulses (with scleral search coils), it is possible to demonstrate a modest reduction in VOR gain (~0.7) following ablation of an individual vertical SCC (Halmagyi *et al*., 1992; Aw *et al*., 1996*b*). Aw *et al*. (1996*b*) were also able to identify the side of the lesioned SCC, by analysing the direction of misalignment in the axis of eye rotation compared with the axis of head rotation. This was one of the first demonstrations of vertical SCC dysfunction in humans, but it required precise three dimensional recordings and complex off line mathematical analysis.

In this study we used a diagonal canal plane head impulse stimulus, oriented approximately in the plane of the vertical SCCs, and we were able to test each vertical SCC separately. The vertical SCCs are aligned diagonally in the head, and they form two approximately co planar pairs: the left anterior/right posterior SCC (LARP) pair, the right anterior/left posterior SCC (RALP) pair (Figs 1 and 2) (Blanks *et al*., 1975*a*; Curthoys *et al*., 1977). Because the LARP, RALP and lateral SCC pairs are roughly orthogonal to each other, a head impulse in the plane of one pair will stimulate mainly that pair, and not the other two SCC pairs. Furthermore, the asymmetric response of primary vestibular afferent nerve fibres dictates that the VOR during a canal plane impulse toward a particular SCC is driven largely by that SCC, and not by its co planar partner. Thus, for both anatomical and physiological reasons, diagonal canal plane head impulses test each vertical SCC separately.

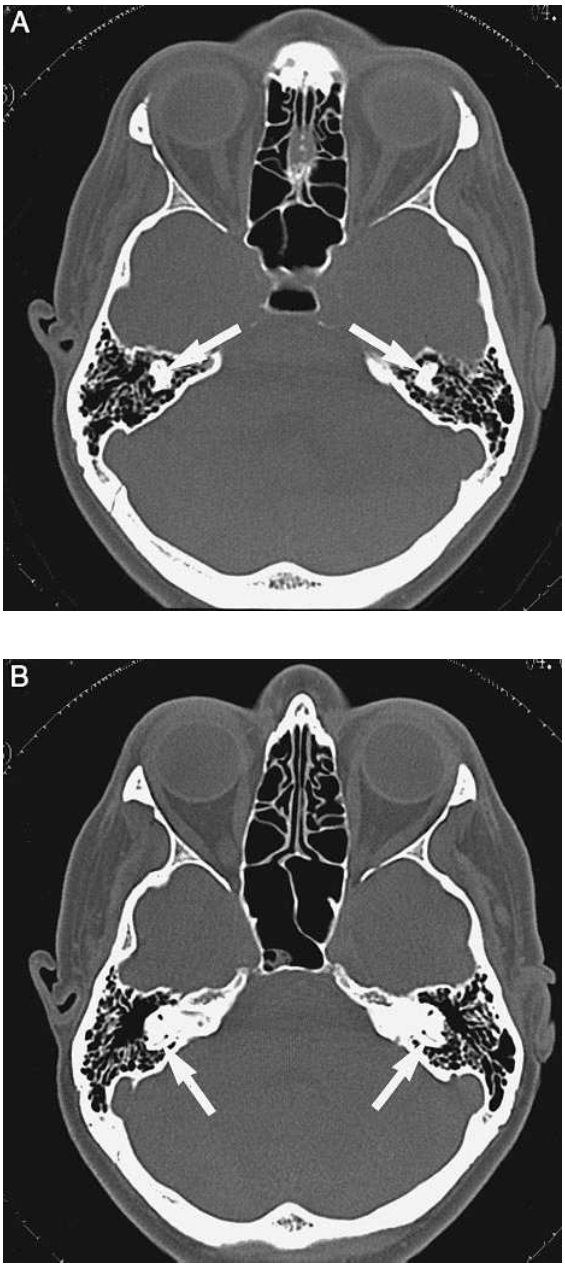

Fig. 1 Axial CT scans of the skull demonstrating that the anterior and posterior SCCs are oriented diagonally within the temporal bones (**A**) The left and right anterior SCCs are shown (arrows) (**B**) CT scan of the same subject, 4 mm caudal to **A** which shows the left and right posterior SCCs (arrows)

Methods

Subjects

Control subjects

We studied nine normal subjects (median age 28 years, range 25 48 years) without any history or clinical signs of vestibular disease.

Unilateral vestibular deafferented (uVD) subjects

We studied seven patients who had undergone surgical vestibular ablation for intractable vertigo, which was attributed to Ménière's disease (four), perilymph fistula (one), cholesteatoma (one) and intractable paroxysmal positioning vertigo (one, before the widespread use of repositioning manoeuvres and canal occlusion surgery). The median age of the uVD subjects was 65 years (range 43 83 years), and the lesions were right sided in four patients and left sided in three. The patients had all recovered from the acute affects of surgery (an average of 8 years before testing, range 36

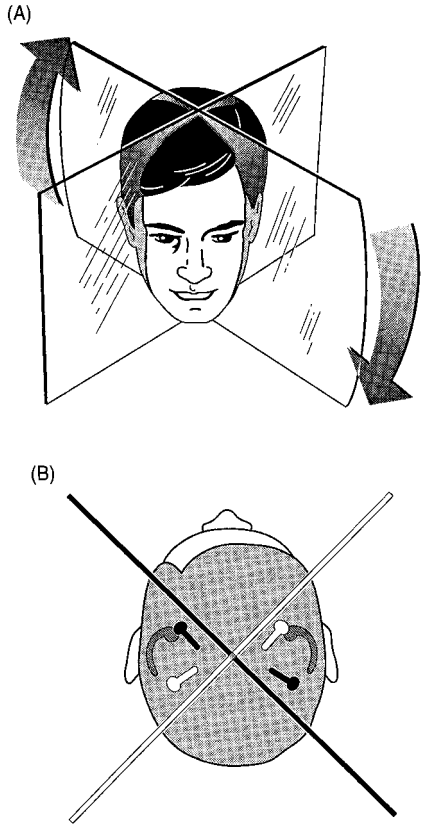

Fig. 2 (**A**) Diagram of the diagonal LARP and RALP planes in which the head impulses were delivered The arrows show the head about to move in the LARP plane, in the direction of the left anterior SCC (**B**) Axial view of the head from above, showing the LARP plane (black line) and the RALP plane (white line) The LARP plane is approximately co planar with the left anterior and the right posterior SCCs, shaded black The RALP plane is approximately co planar with the right anterior and left posterior SCCs, which are white

300 months), and none had significant spontaneous nystagmus (>2 /s) in the dark.

Unilateral posterior SCC occluded (uPCO) patients

We studied three patients following surgical posterior SCC occlusion for intractable benign paroxysmal positioning vertigo. The surgical technique (Pohl, 1996) was similar to that described by Parnes and McClure (1991). The median age of the patients was 66 years (range 58 67 years). The surgery was performed >3 years prior to testing (mean 49 months, range 44 56 months). Two patients underwent left posterior canal occlusion and one had surgery on the right. All had postoperative caloric testing and audiometry, which revealed intact function in the lateral SCC and cochlea, respectively.

Consent

Written, informed consent was obtained from all subjects prior to testing, according to the declaration of Helsinki. The project was supported by the Medical Ethics Committee of the Central Sydney Area Health Service.

Recording system

The recording system was the same as that used by Aw *et al*. (1996*a*, *b*). Three dimensional head and eye position was measured by the scleral search coil technique described by Robinson (1963) and Collewijn *et al*. (1985), using dual search coils (Skalar, Delft, The Netherlands). The eye coil, which had been calibrated in a Fick gimbal, was placed on the subject's left eye. The head coil was mounted on the nose piece of a pair of lightweight spectacle frames, which were fastened securely to the subject's head with a velcro strap. We then placed a latex swimming cap on the subject's head to ensure that the spectacle frames did not slip. The subject was seated with the head in the centre of a (1.9×1.9×1.9 m) wooden frame housing the magnetic field coils.

The head position and gaze position (eye in space) signals were recovered in three orthogonal planes (yaw, pitch and roll) by phase detection, and passed through anti alias filters with a bandwidth of 0 100 Hz. The six position signals were sampled at 1000 Hz by an IBM compatible personal computer running the LabVIEW program (National Instruments, Version 4.0) under Windows 3.1. The recording system has 16 bit resolution and is able to resolve an angular position of 0.1 minute of arc.

Triggering of data sampling was controlled by DAOS software running under TSX plus, on a PDP 11/73 processor (Digital Equipment Corporation). The PDP 11/73 also drove mirror galvanometers which controlled the position of a red laser fixation spot, which was back projected onto a screen 94 cm in front of the subject's left eye.

System calibration

In vitro *calibration*

The head and eye search coils were mounted in a perspex Fick gimbal at the centre of the magnetic field. The gimbal was rotated in successive 5 steps from 20 to 20 in each of the yaw, pitch and roll planes. The corresponding output voltages at each angular location were sampled from both search coils simultaneously. The horizontal, vertical and torsional coil signals were divided by the sine of the calibration angle, and a linear regression analysis was performed to determine the gains and offsets. The linear regression coefficient was >0.99 in each case. Cross talk between the orthogonal signals was <2%. These search coils are not responsive to translations which occur during head impulses (Aw *et al*., 1996*a*).

Following gimbal calibration, the eye coil was cleaned

with detergent and water to remove proteinaceous material, and then sterilized in bleach for 10 min. The bleach was rinsed off with sterile saline solution.

In vivo *calibration*

The eye coil was calibrated again after it was placed on the eye. The subject was asked to fixate the laser target as it was moved across the screen in 5 horizontal, vertical and oblique steps through a range of ±15 . Although the gimbal calibrations were used in the final data analysis, the *in vivo* calibration served as a check that the eye coil was working well and that there was no significant coil misalignment on the eye.

Experimental protocol

The stimulus

The head impulse is a passive, unpredictable, low amplitude (15 30), high velocity (200 400 /s), high acceleration (2000 4000 /s^2) head rotation (Halmagyi and Curthoys, 1988). For each subject we delivered 20 standard yaw (horizontal) head impulses, which are approximately co planar with the lateral SCCs, and 40 head impulses approximately co planar with the vertical SCCs, which we will refer to as 'diagonal' impulses. Diagonal head impulses have not been used previously, and they comprised 20 LARP impulses and 20 RALP impulses (Fig. 2). We define these diagonal planes to be earth vertical, and to lie midway between the pitch and roll planes.

To deliver the diagonal head impulses, the operator placed one hand on the subject's forehead and the other hand on the occiput, along one diagonal meridian (either LARP or RALP). She then delivered each impulse as a brief, abrupt one handed push or pull on the head along that meridian, using the other hand as a guide. This one handed technique was important in eliminating components of horizontal rotation from the head impulse. A good diagonal head impulse comprises equal components of pitch and roll, without any yaw head rotation. The operator was able to view these individual components of angular head velocity on a small LCD screen immediately after each head impulse, in order to maintain quality control of the stimulus. The diagonal impulses were not easy to deliver and the operator was trained extensively on normal subjects. The test was robust, because even if a head impulse did not contain equal amounts of pitch and roll, or if it contained some component of yaw rotation, we were still able to detect absent function in a single SCC. We later plotted the relevant vector component of head and eye velocity for the particular head impulse. For example, during a LARP plane impulse, only the LARP component of head velocity and the LARP component of eye velocity were plotted, thereby removing any 'out of plane' contribution to the VOR. This will be discussed in more detail later.

The diagonal head impulses were comfortable for all subjects, and none of the 19 subjects sustained any trauma from the test. Our protocol stipulates that the amplitude of the head impulses should not exceed 30 , in order to prevent damage to the cervical spine or vertebral arteries.

During each set of head impulses, the specific direction of the impulse within a canal plane (left/right, LA/RP or RA/LP) was randomly selected by the operator. The timing of the impulse was determined by a set of dim light emitting diodes, visible only to the operator, which were activated when the subject's head was repositioned inside a central head position 'window', set to ±1.5 in the yaw, pitch and roll planes. This ensured both that the head position was central at the onset of each impulse and that the timing was unpredictable to the subject.

In addition to these 60 scheduled head impulses, three normal subjects also received 10 roll and 10 pitch head impulses, about the naso occipital and inter aural axes, respectively.

At the beginning of the test, each subject was seated so that the left eye was positioned in the centre of the magnetic field. The room lights were dimmed and the subject was instructed to stare at a central red laser spot projected onto a screen 94 cm in front of the subject's left eye. The operator then delivered the head impulses and the subject's task was to maintain visual fixation on the red laser spot throughout the test.

Data analysis

For each head impulse, 1024 ms of data were acquired. Three dimensional head and 'gaze' (eye in space) position were calculated as Fick angles and then expressed in rotation vectors with roll, pitch and yaw coordinates (Haslwanter, 1995). The rotation vectors were then passively rotated 45 about the yaw (earth vertical) axis (Appendix, Fig. A1) by a matrix transformation (Appendix, Matrix 2) and expressed in canal plane coordinates (LARP, RALP and yaw). Orientation of the eye in the head (referred to as 'eye' position) was calculated from the gaze and the head position coordinates (Haslwanter, 1995).

Using head, gaze and eye position data we calculated the three dimensional angular velocity for head, gaze and eye. The method for calculating angular velocity takes into account the instantaneous head, gaze and eye positions as well as the mathematical differential of the position data (Hepp, 1990). Figure 3 shows some of our methods for data representation. Eye position and eye velocity have been inverted to allow comparison of the eye rotation response with the head rotation stimulus. If a subject had an ideal VOR with zero latency, the eye rotation traces would be superimposed on the head rotation traces. Head and eye position during RA and LP direction head impulses are plotted against time in row 1, and the corresponding angular velocity for each head impulse is plotted against time in row 2. We also plotted eye velocity versus head velocity for each head impulse (row 3), from

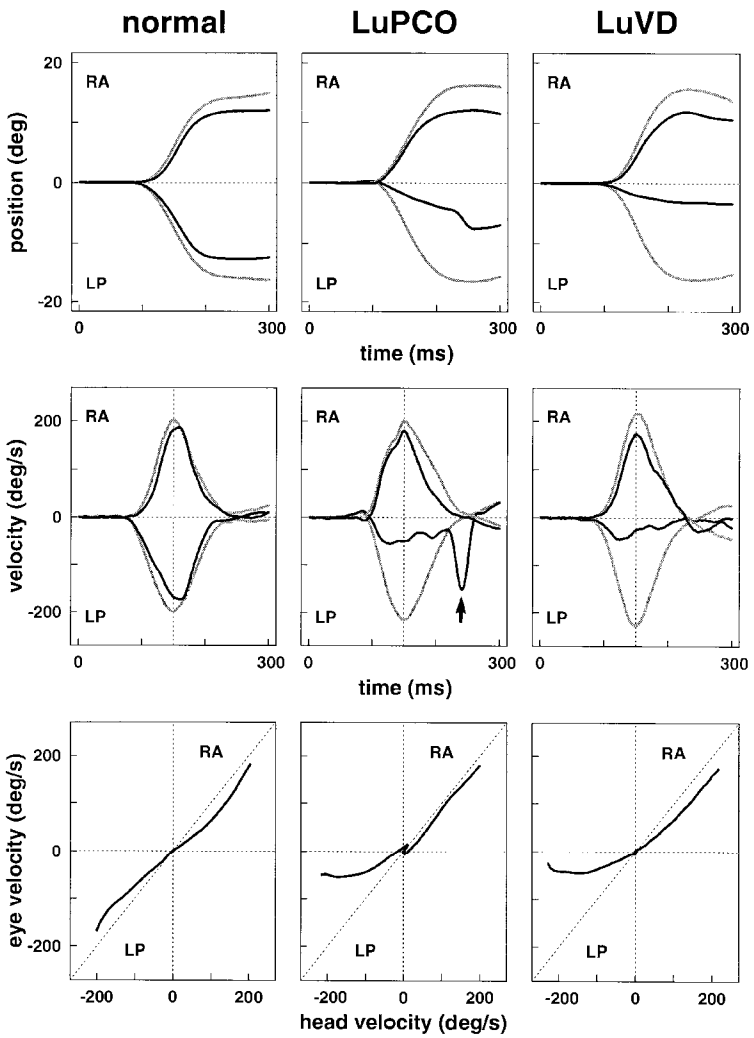

Fig. 3 Format of data representation in this study Head impulses from one normal subject are shown in the left column (column), one patient following left uPCO (column 2) and one patient following left uVD (column 3) Head position and head velocity are displayed in grey Eye position and eye velocity are displayed in black, and have been inverted for ease of comparison with the head rotation stimuli Row (top): head and eye position during one head impulse in the direction of the right anterior SCC (RA, top), and one head impulse in the direction of the left posterior SCC (LP, bottom), for each subject Row 2: head and eye velocity plotted against time, corresponding to the head impulses in row

The vertical broken lines show the point of maximum head velocity The sharp peak in eye velocity in row 2, column 2 (indicated by an arrow), is a 'catch up' saccadic eye movement Row 3: eye velocity plotted against head velocity for the same head impulses A perfect VOR would yield an eye velocity trace superimposed on the diagonal line The data are taken from the onset of the head impulse to maximum head velocity For the two patients (columns 2 and 3), the VOR is deficient for head impulses in the LP direction

the onset of the head impulse to the point of maximum head velocity (which is shown as the broken vertical line in row 2). In this representation, an ideal VOR would result in the eye velocity trace being superimposed on the diagonal broken line (in row 3). This early data plotted in row 3 corresponds to the first 50 100 ms of the head impulse, and was used in the analysis of VOR gain, because it is likely to be a true reflection of vestibular function. In this early phase, the short latency VOR is active, but other visual following systems such as pursuit, optokinetic, cervico ocular and predictive oculomotor systems are not operating, owing to their relatively long latencies (Halmagyi *et al*., 1990).

The VOR gain was then calculated for each subject by dividing the length of the total eye velocity vector (eye speed) by the length of the head velocity vector (head speed) (Haslwanter, 1995; Aw *et al*., 1996*a*).

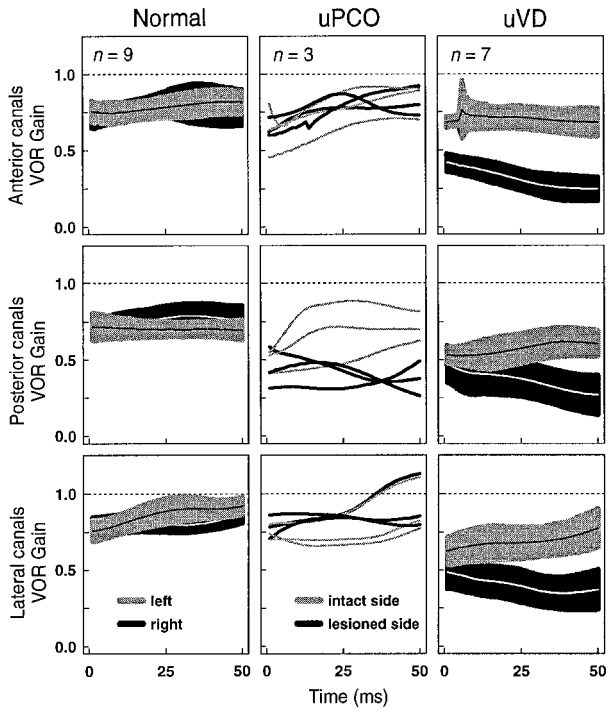

Fig. 4 Averaged VOR speed gain for nine normal subjects (column), three uPCO subjects (column 2) and seven uVD subjects (column 3) Instantaneous VOR gain is calculated for a 50 ms period prior to maximum head velocity VOR gain during the first 0 20 ms of the head impulse is not shown because of erratic results due to dividing by small numbers VOR gain is plotted for head impulses directed toward each anterior SCC (row), each posterior SCC (row 2) and each lateral SCC (row 3) For normal subjects and uVD subjects, the average VOR gain and 95% confidence intervals are plotted, and for uPCO subjects the mean gain for each of the three subjects is plotted separately For normal subjects the VOR gain during head impulses toward the right ear is plotted in black, and the VOR gain during head impulses toward the left ear is plotted in grey In uVD and uPCO subjects, the VOR gain during head impulses toward the lesioned side is plotted in black, and the VOR gain during head impulses toward the intact side is plotted in grey

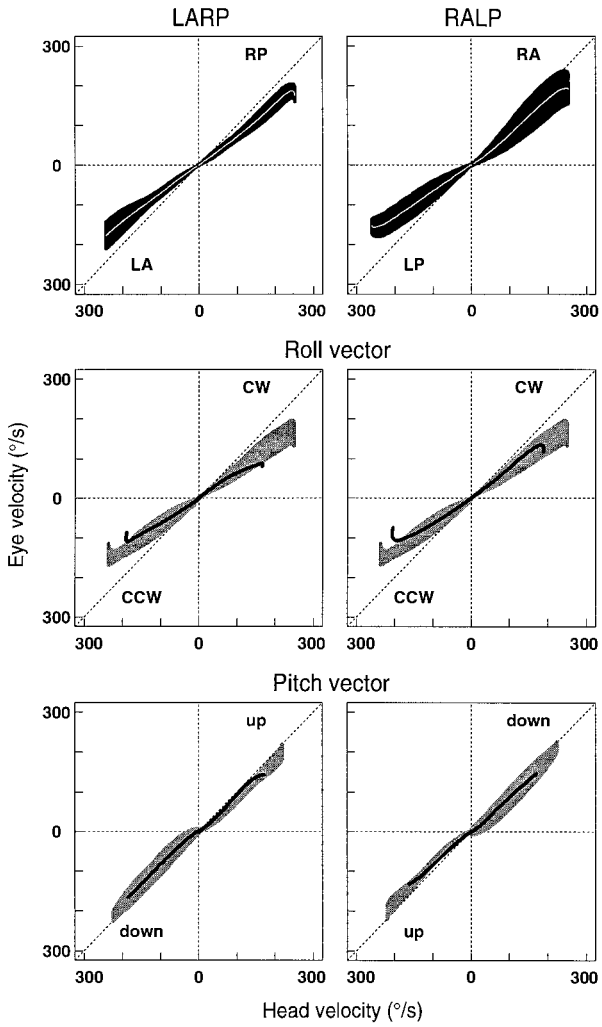

Fig. 5 Comparison of LARP and RALP plane with roll and pitch plane head impulses in three normal subjects In all graphs eye velocity is plotted against head velocity Row shows the average (± SD) for LARP and RALP plane impulses Rows 2 and 3 show the average roll and pitch component vectors of eye versus head velocity during LARP and RALP impulses (black lines) The grey bands in rows 2 and 3 represent the average (± SD) eye versus head velocity during roll and pitch plane impulses in the same three normal subjects The roll and pitch VOR appear constant, irrespective of whether the stimulus is a purely roll or pitch rotation, or whether the roll and pitch components are part of a more complex (LARP or RALP) stimulus

Results

VOR in normal subjects

Horizontal head impulses

The gain of the normal VOR during horizontal head impulses reaches a mean of 0.9 (±0.1, 95% confidence interval) at maximum head velocity (Fig. 4). In other words, during horizontal head impulses the eye rotates in the opposite direction to the head, with approximately the same speed.

Diagonal head impulses (LARP and RALP)

The average eye velocity responses (±95% confidence intervals) during diagonal head impulses in normal subjects are plotted against head velocity as the grey reference band in Fig. 7 (below). The VOR gain during diagonal head impulses is ~0.7 0.8 (Fig. 4). To investigate this deficient canal plane response in normal subjects, we resolved the LARP and RALP head and eye velocities into their separate pitch and roll vector components for three normal subjects (plotted as black lines in Fig. 5). The gain of the roll component was ~0.6, and the gain of the pitch component was ~0.9. This suggests that the deficiency in the diagonal canal plane responses was due to a deficit in the gain of the roll component. We tested the same three subjects with pure roll and pure pitch head impulses, and demonstrated similar

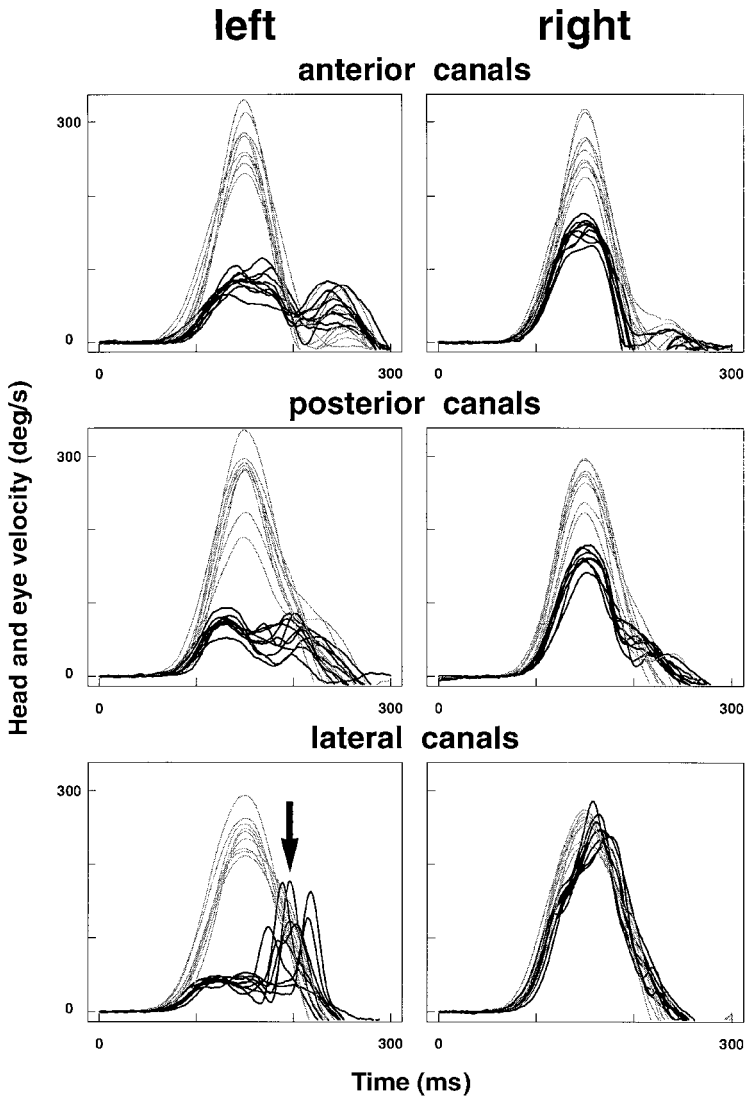

Fig. 6 Head velocity (grey) and eye velocity (black) are plotted against time; these responses are from a subject with a left vestibular neurectomy Eye velocity has been inverted for ease of comparison with the head velocity stimulus A total of 54 head impulses are shown, comprising nine head impulses toward each of the six SCCs For head impulses directed toward the left anterior, left posterior and left lateral SCCs, the VOR is markedly deficient The sharp peaks in the eye velocity traces in row 3, column (indicated by an arrow), are 'catch up' saccades

responses to the roll and pitch components of the diagonal VOR (grey bands in Fig. 5).

VOR in uVD patients

The VOR during head impulses directed toward each of the three deafferented SCCs was consistently deficient. Figure 6 shows nine head impulses toward each SCC in one representative patient following left vestibular neurectomy. The VOR during head impulses toward affected SCCs on the left side was poor, but the responses toward the intact right side were within normal limits. In addition, the eye velocity response reached a maximum of 50 100 /s and varied little for a wide range of stimulus magnitudes, from maximum head velocity of 180 320 /s. The VOR gain during head impulses toward the lesioned side was ~0.2 0.3 at maximum head velocity, regardless of which SCC was being tested (Fig. 4). Figure 7 (row 2) shows averaged eye velocity plotted against head velocity (in black), with 95% confidence intervals. There was a significant difference between the responses of the uVD subjects when tested toward their lesioned side and those in normal subjects (plotted in grey). For head impulses toward the intact side, the VOR gain was ~0.55 0.70, which overlaps the normal range at the low end.

Most subjects with a VOR deficit used saccades to refixate the target lost during the head movement. The subject shown

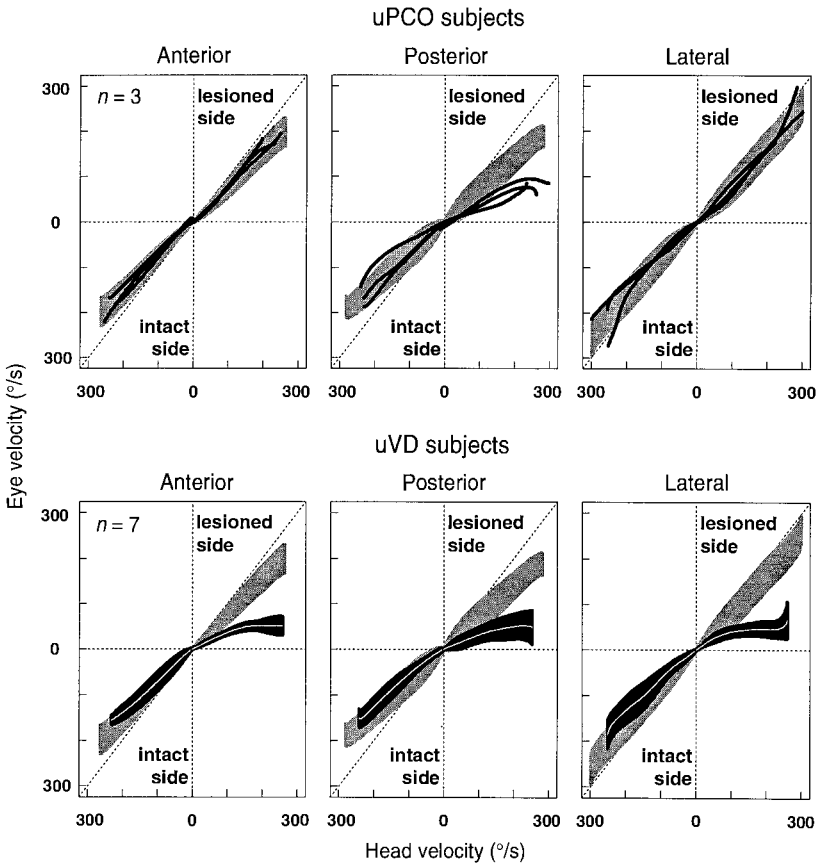

Fig. 7 Averaged eye velocity versus head velocity is plotted in black for the three uPCO subjects (row), and the averaged eye velocity (±95% confidence intervals) is plotted for uVD subjects (row 2) The VOR during head impulses directed toward the anterior SCCs is shown in column , toward the posterior SCCs (column 2), and toward the lateral SCCs (column 3) The normal range (average ±95% confidence intervals) is shown as a grey band in each plot The VOR during head impulses directed toward the lesioned side is shown on the right side of each graph, and for head impulses toward the intact side on the left side of each graph The eye velocity is limited to 50 00°/s during head impulses in the direction of any lesioned SCC

in Fig. 6 used early saccades (indicated by an arrow) during yaw impulses, with latencies of 110 150 ms. Other subjects used early saccades during diagonal head impulses. Most subjects also used late saccades (>500 ms after the head impulse) to refixate the target.

VOR in uPCO patients

There was a clear deficit in VOR gain (0.3) for head impulses directed toward the lesioned posterior SCC, and the VOR gain during head impulses toward all other SCCs was normal (Fig. 4). Figure 8 shows nine head impulses directed toward each SCC in one subject following left posterior SCC occlusion. During head impulses directed toward the five intact SCCs, the eye velocity response matches the head velocity stimulus. Yet for head impulses in the direction of the inactivated posterior SCC, the eye velocity response is markedly deficient. The eye velocity reaches a maximum of 50 100 /s during head impulses with a maximum velocity of 220 320 /s. In addition, there are early compensatory saccadic eye movements with latencies of 100 200 ms during head impulses toward the lesioned SCC.

Discussion

VOR in normal subjects

Horizontal head impulses

The gain of the normal VOR during horizontal head impulses reached 0.9 (±0.1, 95% confidence interval) at maximum head velocity. Therefore the eye rotation response compensates, in magnitude, for the head impulse stimulus. This confirms previous data for the horizontal VOR (Cremer *et al*., 1988; Halmagyi *et al*., 1990; Aw *et al*., 1996*a*).

Diagonal head impulses

The VOR in response to diagonal plane head impulses does not mirror the stimulus as faithfully as it does for yaw plane

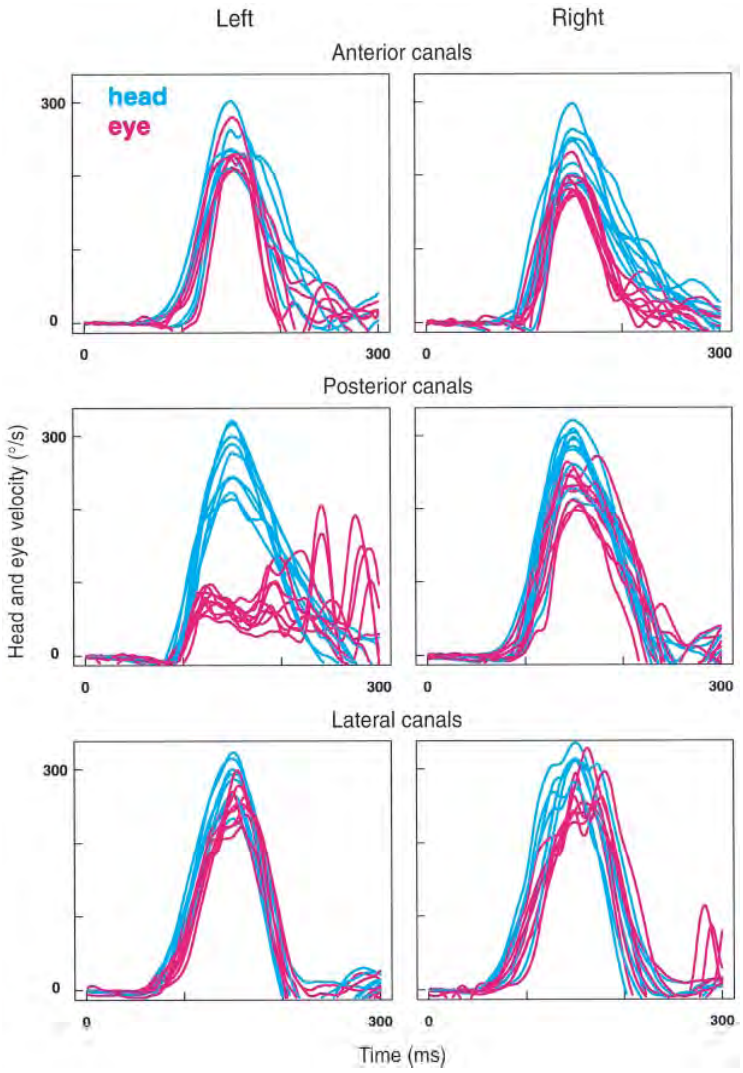

Fig. 8 Head velocity (blue) and eye velocity (red) are plotted against time in one subject following a left posterior SCC occlusion Eye velocity has been inverted for ease of comparison with the stimulus (head velocity) A total of 54 head impulses are shown, comprising nine head impulses toward each of the SCCs During head impulses directed toward the inactivated left posterior SCC the VOR is markedly deficient Yet head impulses directed toward any of the five intact SCCs elicit a normal response The sharp peaks in the eye velocity traces during head impulses directed toward the left posterior SCC are 'catch up' saccades, which partially compensate for the deficient vestibular response

stimuli. The gain of the response is only 0.7 0.8. This deficiency appears to be due to a physiological limitation of the roll VOR. The diagonal LARP and RALP planes lie midway between the pitch and roll planes, and the VOR during diagonal head impulses should be the vector sum of the pitch and roll VORs. The pitch VOR gain in normal subjects is 0.9 1.0, and the roll gain is 0.6 0.7 (Ferman *et al*., 1987; Leigh *et al*., 1989; Crawford and Vilis, 1991; Tweed *et al*., 1994; Aw *et al*., 1996*a*). We found the diagonal VOR gain to be between these values. To confirm this, we resolved the head and eye velocities of three normal subjects into their pitch and roll components, and the VOR gains were ~0.9 and 0.6, respectively (black traces in Fig. 5).

In addition, we delivered 10 pitch plane and 10 roll plane impulses in each of these three subjects (grey bands in Fig. 5), and the resulting VOR closely matched the pitch and roll components of the response during diagonal impulses. In other words, the VOR in response to a head impulse which deviates from the pitch and roll planes is the vector sum of the responses to its pitch and roll components. Crawford and Vilis (1991) reached a similar conclusion after rotating alert monkeys in the LARP and RALP planes. Fetter *et al*.

(1994) confirmed this in humans by rotating their subjects sinusoidally in the LARP and RALP planes and finding that the VOR was a vector sum of the pitch and roll components, with the roll gain being two thirds of the yaw or pitch gains. This has also been verified experimentally in canal plugged monkeys (Yakushin *et al*., 1995). This concept is not universally endorsed, since a study by Tweed *et al*. (1994) in humans showed that the roll VOR gain in response to a pure roll stimulus (0.37) was less than the gain when the torsional component was a small component of a predominantly yaw or pitch movement (0.52). This inconsistency might be due to the different stimuli used. Tweed *et al*. (1994) used sinusoidal oscillation with a maximum speed of 37.5 /s and torsional components of only 10 /s in their diagonal axis paradigm, whereas our head impulses reached velocities of 300 400 /s. It is possible that during the low velocity stimulation of 10 /s, which is generally considered to be at the low end of the operating range of the angular VOR, other non SCC systems, such as otolithic counter roll, could boost the response.

VOR in uVD patients

'Off' direction VOR

The VOR during head impulses directed toward any one of the three deafferented SCCs was uniform (Fig. 7), and the gain was ~0.2 0.3 at maximum head velocity (Fig. 4). A similar VOR gain deficit has been shown in the horizontal plane, reflecting a deficiency of lateral SCC function on the lesioned side (Cremer *et al*., 1988; Halmagyi *et al*., 1990, 1991; Tabak and Collewijn, 1995; Aw *et al*., 1996*b*; Haslwanter and Fetter, 1996). However, such a substantial gain deficit has not been shown for the anterior or posterior SCCs. Diagonal LARP and RALP plane impulses clearly detect a complete lesion of an individual anterior or posterior SCC.

Specificity of diagonal and yaw head impulses

Analysis of the stimulus plane

The specificity of diagonal head impulses (their ability to test individual SCC pairs) is predominantly due to the plane of head rotation being approximately co planar with the SCC pair being tested. The specificity is also due to analysis of the data in the LARP and RALP planes. The LARP, RALP and lateral SCCs operate in a push pull fashion, as three co planar pairs. Because the SCC pairs are approximately orthogonal, a head impulse in the plane of one pair will have minimal stimulatory or disfacilitatory effect on the other two SCC pairs, both of which are out of the plane of head rotation. For any given head rotation, the amount of acceleration transduced by a particular SCC is determined by the geometric 'projection' of that SCC into the plane of the head rotation (Estes *et al*., 1975). For example, a head rotation in the plane of a SCC causes maximum stimulation of that SCC, but a head rotation in the plane perpendicular to a SCC causes no activation. Intermediate projections cause intermediate SCC activation, as determined by the cosine of the angle between the plane of head rotation and the plane of the SCC (Blanks *et al*., 1975*b*; Bohmer *et al*., 1985). This principle has been validated in SCC plugged monkeys, whose VOR was shown to be a simple vector addition of the VOR generated by all six SCCs, weighted by their projection into the plane of head rotation (Yakushin *et al*., 1995).

We calculated the relative contribution of each SCC to the canal plane head impulses (Appendix). Using the canal plane data from Blanks *et al*. (1975*a*), and geometrically projecting the data into LARP and RALP planes, we calculate that during a head impulse in the LA direction, the left anterior SCC is stimulated by 0.99 of the total head acceleration, the right posterior SCC is disfacilitated by 0.93, the left lateral SCC is stimulated by 0.37, and the remaining SCCs each transduce <0.15 of the total head acceleration. Similarly, during a head impulse in the RA direction, the right anterior SCC is stimulated by 0.99 of the total head acceleration, the left posterior SCC is disfacilitated by 0.93, the right lateral SCC is stimulated by 0.37, and the remaining SCCs each transduce <0.15 of the total head acceleration. During a leftward yaw impulse with the head erect, the left lateral SCC is stimulated by 0.91 of the total head acceleration, the right lateral SCC is disfacilitated by 0.91, the right posterior SCC is stimulated by 0.32, the left posterior SCC is disfacilitated by 0.32, and the anterior SCCs each transduce <0.02 of the total head acceleration. This selectivity of yaw head impulses accounts for their usefulness as a clinical test of lateral SCC function (Halmagyi and Curthoys, 1988).

Analysis of the data in diagonal planes

Further specificity of canal plane impulses is derived from the plane of analysis of the head and eye rotations. We plotted the LARP component of head and eye velocity during LARP plane impulses, the RALP component during RALP impulses and the yaw component during yaw impulses (Figs 6, 7 and 8). By plotting only the in plane vector components of head and eye velocity, the data are not confounded by any out of plane components of the head velocity stimulus. Because the head impulses were delivered manually, they did not always lie exactly in a diagonal plane, and contained elements of yaw rotation or unequal components of pitch and roll. Yet if head and eye velocity are plotted in the LARP or RALP planes, these errors become unimportant, and a single defective SCC can be clearly identified (Fig. 8).

The physiological reason for this selectivity depends on the principle that each SCC generates eye rotations about an axis orthogonal to its own plane (Suzuki *et al*., 1964). In other words, the posterior SCCs generate predominantly downward and torsional slow phase eye rotation, and the lateral SCCs generate mainly horizontal eye rotation with a smaller torsional component. For example, during an ideal LA direction head impulse, even though the left lateral SCC

is stimulated by 0.37 of the total head acceleration, the resulting eye rotation generated by this SCC is largely horizontal. If we then plot the LARP component of eye velocity versus the LARP component of head velocity, the contribution of the left lateral SCC becomes very small. Using Robinson's methods (1982), and published VOR gain values (Aw *et al*., 1996*a*), we calculated that the left lateral SCC projects to the left medial rectus with a relative efficacy of 1.02, compared with 0.11 to the left superior rectus and 0.15 to the left superior oblique (Appendix). By projecting the plane of action of each extra ocular muscle into the LARP plane, it appears that only the left superior rectus has a significant action in this plane, with a relative efficacy of 0.94, compared with 0.01 for the left medial rectus and 0.13 for the left superior oblique. In other words, even though the left lateral SCC is stimulated by 0.37 of the total LARP plane head acceleration, the contribution of the left lateral SCC to LARP plane eye rotation is <0.04 (Appendix). This technique of excluding out of plane eye velocity, and therefore minimizing the contribution of out of plane SCCs, becomes even more important if a head impulse does not lie exactly in the LARP plane.

Even though plotting the component of head and eye velocity which corresponds to the intended plane of the head impulse increases the specificity of the test, the diagnostic ability of the diagonal impulses does not depend on analysing the data in canal planes. The calculation of VOR speed gain (Fig. 4) exposes the deficient SCCs, and is derived not from a particular component of head or eye velocity but from the total speed of eye rotation divided by the total speed of head rotation (Aw *et al*., 1996*b*).

Sensitivity of LARP, RALP and yaw head impulses

The sensitivity of canal plane head impulses, in detecting total loss of an individual SCC, depends not on the anatomy but on the physiology of the VOR during high acceleration stimuli. Rotating subjects in the LARP or RALP planes at low accelerations will expose only a small VOR gain asymmetry, despite complete functional loss of a single SCC (Kanayama *et al*., 1995).

Physiology of primary and secondary vestibular neurons

Ewald's Second Law (Ewald, 1892) implies that each SCC transduces angular acceleration in a non linear manner. Yet the normal VOR is linear because each SCC is oppositely polarized to its co planar partner, and the summed output is linear. Each SCC has a preferred 'on' direction response, and an opposite 'off' direction response which has a lower gain and which can be saturated. There is very little data on the behaviour of primary vestibular afferent neurons during high acceleration stimuli, but Goldberg and Fernandez (1971) have made neural recordings during moderate accelerations up to 150 /s^2. They demonstrated that during 'on' direction stimulation of any individual SCC in squirrel monkeys, the firing of primary vestibular afferent neurons can exceed 350 spikes per second from a tonic resting level of 90 spikes per second. The upper limit of firing was not reached at these accelerations. During acceleration in the 'off' direction, firing in primary afferent neurons decreased from 90 spikes per second to zero, with a sensitivity less than that in the 'on' direction. It is likely to be a combination of the reduced neural sensitivity and neural silencing during high acceleration, 'off' direction stimuli, that explains the VOR asymmetry described by Ewald's Second Law. This non linearity is propagated to, and probably enhanced in, vestibular nucleus neurons which receive input from one co planar pair (Shinoda and Yoshida, 1974). Reisine and Raphan (1992) found that LARP or RALP plane vestibular nucleus neurons were less sensitive to contralateral (off direction) acceleration than to ipsilateral (on direction) acceleration and, more importantly, the contralateral response saturated at high stimulus accelerations. These secondary vestibular neurons then form excitatory connections predominantly with neurons of a single extra ocular muscle in each eye whose action is roughly co planar with the SCC driving the response, but they also synapse with other agonist ocular motor neurons to a lesser extent (Ezure and Graf, 1984; Graf and Ezure, 1986; Baker and Peterson, 1991). The VOR is therefore largely driven by the peripheral and central vestibular system ipsilateral to the head acceleration. In other words, the vestibular response to head acceleration in the direction of the right anterior, right posterior or right lateral SCCs is primarily driven by the right sided SCCs and the right vestibular nuclei, with a weaker contribution from the SCCs on the left side. Under normal circumstances, this non linearity in the VOR is overcome by co planar vestibular nucleus neurons on opposite sides of the brainstem working synergistically to ensure that the VOR is symmetric regardless of the direction of head acceleration.

In uVD subjects the VOR in any plane is driven solely by the three SCCs on the intact side. It should therefore be possible to expose the inherent asymmetry of each remaining SCC by using a high acceleration, 'off' direction, saturating stimulus. This has been shown for the lateral SCCs only. Horizontal head impulses, with accelerations up to 4000 /s^2, have demonstrated a permanent, severe deficit of lateral SCC function in patients following uVD (Cremer *et al*., 1988; Halmagyi *et al*., 1990; Tabak and Collewijn, 1995; Aw *et al*., 1996*b*; Fletcher *et al*., 1996). In contrast, other studies in humans using lower acceleration stimuli on rotational chairs have failed to detect such a substantial VOR deficit (Honrubia *et al*., 1982; Olson and Wolfe, 1984; Black *et al*., 1989). Paige (1983) and Fetter and Zee (1988) demonstrated in primates that the horizontal VOR following inactivation of a single lateral SCC was symmetrical for low acceleration stimuli, and only became deficient for high acceleration stimuli in the direction of the inactivated SCC. This is

presumably because low velocity rotational stimuli, such as those used in the routine clinical testing of patients, lack sufficient acceleration to silence the primary vestibular neurons on the intact side, in contrast to the horizontal head impulses and high acceleration rotations. Although low velocity stimuli cannot reliably be used to assess the integrity of individual SCCs, they do provide useful clinical information about the mid to low frequency responses of the VOR, and about central velocity storage.

Kanayama *et al*. (1995) studied three patients following uPCO and one patient following posterior SCC nerve section. The subjects sat in a large gimbal which was oscillated sinusoidally in the diagonal LARP and RALP planes, at a frequency of 0.55 Hz. Six months after surgery there was a modest (16%) asymmetry in the pitch component of the diagonal VOR in the plane of the lesioned SCC, compared with a 5% asymmetry for normal subjects. The torsional component of the LARP and RALP responses was highly variable. This failure to demonstrate a substantial deficit in posterior SCC function after surgical inactivation was probably due to inadequate stimulus acceleration and failure to silence primary afferents from the contralateral anterior SCC. Another problem with the stimulus was its predictability, allowing non vestibular oculomotor systems, such as predictive vertical eye movements, to play a role. Fetter and Dichgans (1996) rotated 16 patients with vestibular neuritis in the LARP, RALP and yaw planes and found variable results. From the rotation axis of the spontaneous nystagmus, they concluded that both the lateral and anterior SCCs were deficient in many of their patients. Yet 14 out of 16 patients showed only modest asymmetry, or no asymmetry at all, in the VOR during LARP and RALP rotation. Again, the reason for this might be the low stimulus velocities used. Another possible explanation is that, because the anterior SCC lesions were pathological and not surgical, they might have been incomplete.

We used head impulses to examine the anterior, posterior and lateral SCCs, and we showed that in uVD subjects, the off direction VOR is remarkably similar for any SCC being tested. This is probably because the behavior of primary afferent neurons from each SCC is uniform (Goldberg and Fernandez, 1971). During all head impulses in the direction of a deafferented SCC, there appears to be an eye velocity response limit of ~50 75 /s, regardless of whether the maximum head velocity stimulus is 225 /s or 350 /s (Fig. 6). This eye velocity limit might reflect the neural saturation as primary vestibular neurons from the intact co planar SCC are silenced.

Comparison with pitch and roll head impulses

Previous efforts to study the anterior and posterior SCCs with the head impulse test have used pitch and roll impulses. Following uVD, the VOR gain during pitch up and pitch down impulses is symmetrical (0.5 0.9), regardless of whether the left or right vertical SCCs have been ablated (Halmagyi *et al*., 1992; Aw *et al*., 1994, 1996*b*; Fletcher *et al*., 1996; Haslwanter and Fetter, 1996). Analysing VOR gain during pitch head impulses is neither a sensitive nor a specific indicator of vertical SCC function in the uVD patient (Fig. 9). On the other hand, VOR gain during roll head impulses can indicate the side of the lesion, but does not provide any information about the function of individual SCCs (Fig. 9). Roll impulses elicit a severely attenuated VOR gain toward the lesioned side (0.2 0.3) versus 0.6 toward the intact side (Aw *et al*., 1996*b*; Haslwanter and Fetter, 1996). Using detailed off line three dimensional analysis of the axes of head and eye rotation, Aw *et al*. (1996*b*) were able to infer that an individual anterior or posterior SCC was defective, since the eye rotation axis deviated toward the lesioned side. However, this result is not apparent to the examiner performing the test, nor is it evident from looking at the VOR gain during each head impulse. The reason for this is that all four vertical SCCs participate in pitch head impulses and all six SCCs participate in roll impulses, so that the effect of any one or two defective SCCs is masked by the remaining intact SCCs. Compared with pitch and roll head impulses, diagonal head impulses accentuate the VOR gain deficit due to loss of a single vertical SCC.

We calculated the contribution of individual SCCs to pitch and roll head impulses using published canal plane coordinates in humans (Blanks *et al*., 1975*a*) projected onto the pitch and roll planes (Appendix). During a pitch down head impulse each anterior SCC is stimulated by 0.75 of the total head acceleration stimulus, each posterior SCC is disfacilitated by 0.56, and each lateral SCC is stimulated by 0.16. Similarly, during a roll clockwise head impulse, the right anterior SCC is stimulated by 0.65 of the total head acceleration, the right posterior SCC is stimulated by 0.76, the right lateral SCC is stimulated by 0.37, and the left sided SCCs are disfacilitated by corresponding amounts. In other words, pitch head impulses test all four vertical SCCs in normal subjects and the two remaining vertical SCCs in uVD subjects, and roll head impulses test all six SCCs in normal subjects and the three remaining SCCs in uVD subjects. For these anatomical reasons, pitch and roll head impulses have not been as useful in the clinic as horizontal head impulses in testing the function of individual SCCs.

Could the failure to silence primary vestibular afferent neurons from the intact vertical SCCs also have contributed to the failure of pitch impulses to detect a severe deficit in vertical VOR gain following uVD (Fig. 9)? The answer is probably yes. It is likely that the intact anterior SCC afferents are silenced during pitch up impulses, and that the intact posterior SCC afferents are silenced during pitch down impulses. From the canal orientations, we calculated that during a typical pitch down head impulse with a maximum acceleration of 2500 /s^2, the intact anterior SCC is being stimulated by a component equal to 1883 /s^2 and the intact posterior SCC is being disfacilitated by 1403 /s^2. Tabak and Collewijn (1995) demonstrated that head impulses with a maximum acceleration of 700 1300 /s^2 were sufficient to

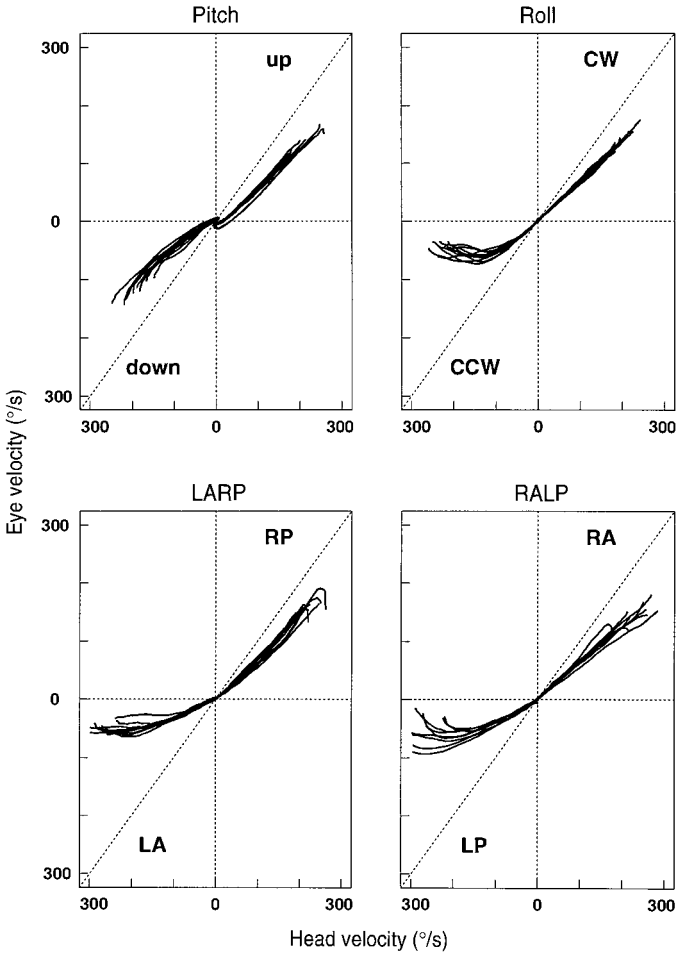

Fig. 9 Comparison of pitch , roll , LARP and RALP plane head impulses in two patients following left vestibular neurectomy Eye velocity is plotted against head velocity for 0 head impulses in each direction The VOR during pitch up and pitch down impulses is modestly reduced and symmetric The VOR during roll CCW (counter clockwise) is markedly reduced During LARP and RALP plane impulses toward the lesioned left anterior and left posterior SCCs, the VOR is markedly deficient The pitch and roll impulse data were kindly provided by Dr Aw (Aw *et al* , 996*b*)

expose severe asymmetry in the function of a single lateral SCC. This implies that these accelerations were sufficient to silence the intact primary vestibular afferent nerve fibres during head rotation toward the lesioned side. Therefore, during a pitch down head impulse the primary afferents from the intact posterior SCC are probably being silenced, and at the same time the intact anterior SCC is being stimulated in its 'on' direction by an acceleration of 1883 /s^2, and it is capable of generating a VOR gain of ~0.7 (Aw *et al*., 1996*b*). Similarly during a pitch up head impulse, primary afferents from the intact anterior SCC are probably being silenced, while the intact posterior SCC is being stimulated in its 'on' direction by an acceleration of 1403 /s^2. The simultaneous 'on' direction stimulation of one SCC masks the effects of the 'off' direction response from the other vertical SCC on the intact side, rendering pitch impulses ineffective in detecting a severe VOR gain deficit in the uVD patient.

Similar analysis reveals that a typical roll head impulse in a uVD patient toward the lesioned side disfacilitates the intact anterior SCC with an acceleration of 1630 /s^2, the intact posterior SCC with 1893 /s^2 and the intact lateral SCC with 913 /s^2. These stimuli are in the 'off' direction for all three SCCs, since all SCCs on one side contribute to the ipsilateral roll VOR. This explains why there is a severely limited roll VOR after uVD (Fig. 9). It also explains why the roll head impulse is a sensitive test of unilateral vestibular failure, but not a specific test of any individual SCC.

It is interesting that the roll VOR toward the lesioned side was similar to the LARP and RALP VOR toward the lesioned side in two patients following left sided uVD (Fig. 9). In other words, the result of silencing the primary afferents from three SCCs (during roll impulses), is not different from silencing the primary afferents from one SCC (during LARP and RALP impulses).

The VOR in uPCO patients

There was a clear deficit in VOR gain (0.3) for head impulses directed toward the lesioned posterior SCC, with the VOR gain during impulses toward all other SCCs falling within normal limits. The magnitude of the deficit in uPCO patients was similar to that found during head impulses toward the lesioned posterior SCC in uVD patients. This demonstrates the ability of LARP and RALP head impulses to detect a lesion in a single SCC, when the remaining five SCCs are normal. Previous studies using pitch and roll head impulses (Aw *et al*., 1996*b*) and using low velocity LARP and RALP rotation (Kanayama *et al*., 1995) have shown only a modest VOR gain deficit in uPCO subjects.

Use of LARP and RALP impulses as a clinical test

LARP and RALP canal plane head impulses expose a severe VOR gain deficit in the direction of a lesioned anterior or posterior SCC, much the same as the horizontal head impulses do for the lateral SCCs. Horizontal head impulses have gained acceptance as a useful bedside and laboratory investigation, and we believe that LARP and RALP head impulses complement the horizontal impulses, forming a comprehensive battery of canal plane tests. Potentially, this battery will be useful in assessing all patients with vestibular disorders, especially when pathology (e.g. vestibular neuritis) selectively affects certain elements of the vestibular end organ (Fetter and Dichgans, 1996).

The success of LARP and RALP impulses as a bedside test (i.e. without measuring head and eye rotation) depends on the examiner seeing a voluntary re fixation saccadic eye movement after completion of the head impulse. Clinically, we were able to detect an abnormal response during head impulses toward lesioned SCCs in the clinic, prior to measuring the VOR in this experiment. However, the predictive value of the these diagonal head impulses as a bedside test needs to be evaluated formally.

Conclusions

LARP and RALP plane head impulses have exposed a severe, permanent deficit in anterior and posterior SCC function following surgical lesions. These canal plane impulses are currently the only available method to evaluate each vertical SCC separately. Together with horizontal head impulses, they form a comprehensive battery of clinical SCC tests. The sensitivity of the head impulses in demonstrating a complete lesion of one SCC depends on exposing the inherent asymmetry in primary vestibular afferent neuron physiology, and the specificity of the test depends on the anatomical orientation of the SCCs.

Acknowledgements

We wish to thank Thomas Haslwanter and Ann Burgess for their help in reviewing the mathematics, and David Pohl for referring his patients following posterior SCC occlusion and vestibular neurectomy. Dr Cremer was supported by a grant from the National Health and Medical Research Council of Australia.

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Received May 23, 1997. Revised October 30, 1997.
Accepted November 19, 1997

Appendix

Analysis of the projections of the SCCs into the LARP, RALP, pitch, roll and yaw planes

Blanks *et al.* (975*a*) calculated the equations which describe the normals to the plane of the six SCCs in cardinal coordinates: roll, pitch and yaw (*x*, *y*, *z*) We have expressed these equations as matrices, representing the directional sensitivity of each SCC to head rotation in the roll, pitch and yaw planes; (the individual canal matrix [Ci]: Matrix) We have maintained the same Cartesian coordinate system: superior (+*z*), left (+*y*) and anterior (+*x*), but we have re defined the direction of the SCC sensitivity vectors to follow the 'right hand screw' convention In other words, a SCC is stimulated by angular acceleration in the direction of the advance of a right hand screw, where the axis of rotation is the directional sensitivity vector of that SCC For any head rotation, the change in firing of primary afferent neurons from a particular SCC is calculated by multiplying the roll, pitch and yaw components of head velocity by the SCC sensitivity matrix (Matrix) (Robinson, 982) It is important to note that this method calculates the geometric projection of the head acceleration vector onto the SCC sensitivity vector, but does not account for the physiological non linear firing in primary vestibular afferent neurons For example, during a leftward (+*z*) head rotation, the left lateral SCC is stimulated by 0 905 of the total head acceleration, the right lateral SCC is disfacilitated by 0 905, the right posterior SCC is stimulated by 0 320, and the left posterior SCC is disfacilitated by 0 320 of the total head acceleration During a pitch down head impulse with maximum head acceleration of 2500°/s^2, both anterior SCCs are stimulated by a component of 883°/s^2, both posterior SCCs are disfacilitated by 403°/s^2, and both lateral SCCs are stimulated by 395°/s^2 During a roll clockwise head impulse, the right anterior SCC is stimulated by 630°/s^2, the right posterior SCC is stimulated by 893°/s^2, and right lateral SCC is stimulated by 9 3°/s^2; and the left sided SCCs are disfacilitated by the corresponding amounts

Matrix 1 *The individual canal matrix [Ci]*

	la	lp	ll	ra	rp	rl
x	0 652	0 757	0 365	0 652	0 757	0 365
y	0 753	0 56	0 58	0 753	0 56	0 58
z	0 0 7	0 320	0 905	0 0 7	0 320	0 905

This is a directional sensitivity matrix for each SCC, expressed in roll, pitch and yaw coordinates (*x*, *y*, *z*) la = left anterior SCC; lp = left posterior SCC; ll = left lateral SCC; ra = right anterior SCC; rp = right posterior SCC; rl = right lateral SCC

In order to express the individual canal matrix in canal plane coordinates (RALP, LARP, *z*), we have passively rotated the (*x*, *y*, *z*) coordinate system by +45° about the earth vertical *z* axis (Fig A) This is equivalent to rotating all points 45° about the *z* axis, and is achieved by multiplying Matrix by Matrix 2 (Haslwanter, 995)

Matrix 2 *Rotation matrix which passively rotates the* x *and* y *axes by +45° about the* z *axis.*

cos (45°)	sin (45°)	0
sin (45°)	cos (45°)	0
0	0	

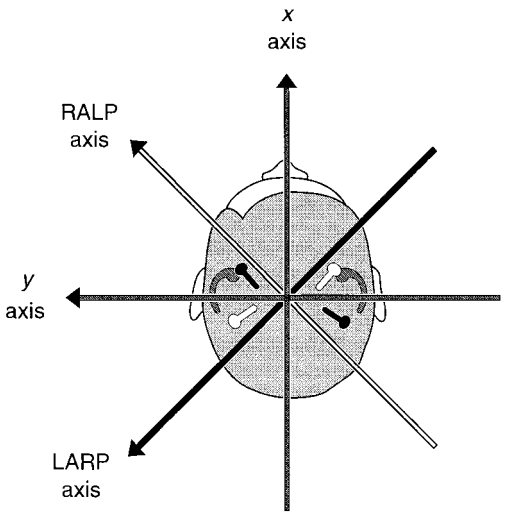

Fig. A1 Axial view of the head from above, showing the axes about which the head impulses were delivered The actual motion is perpendicular to these axes The roll (*x*) and pitch (*y*) axes are passively rotated +45° about the earth vertical *z* axis, to become the RALP and LARP axes, respectively Arrows indicate the positive direction for each axis, and they follow the 'right hand' convention For example a head impulse about the RALP axis in a positive direction, corresponds to a head impulse in the direction of the right anterior SCC (shaded white)

Matrix 3 *[Ci′] the modified individual canal matrix*

	la	lp	ll	ra	rp	rl
RALP	0 07	0 932	0 46	0 993	0 39	0 370
LARP	0 993	0 39	0 370	0 07	0 932	0 46
z	0 0 7	0 320	0 905	0 0 7	0 320	0 905

This is the directional sensitivity matrix for each SCC, expressed in canal plane coordinates (RALP, LARP, *z*) By convention, the LA and RA directions are positive

Using canal plane coordinates (Matrix 3) we calculate that during head acceleration in the direction of the right anterior SCC (+RALP), the right anterior SCC is stimulated by 0 99 of the total head acceleration, the left posterior SCC is disfacilitated by 0 93, the right lateral SCC is stimulated by 0 37 and the left lateral SCC is disfacilitated by 0 5 of the total head acceleration

Geometric alignment of the extra ocular muscles

The six extra ocular muscles in each eye act as three pairs of approximately co planar muscles, with each muscle pulling in the opposite direction to its partner The average plane of action for each pair (in the left eye) was calculated by Robinson (982), and we have represented that data in our coordinate system (Matrix 4)

Matrix 4 *The muscle matrix [M]*

	lmr	sir	sio
E*x*	0 0 5	0 424	0 788
E*y*	0 005	0 906	0 600
E*z*	0 999	0 0 6	0 40

Plane of action of the extra ocular muscles in the left eye (adapted from Robinson, 982) lmr = lateral and medial rectus; sir = superior and inferior rectus; sio = superior and inferior oblique Ex, E*y*, and E*z* refer to the vector components of eye velocity in the roll, pitch and yaw planes, respectively The first named muscle pulls in the direction indicated by the sign of the vector, and the second named muscle pulls in the opposite direction

We can also express the muscle matrix in canal plane coordinates (Matrix 5) by multiplying Matrix 4 by the rotation matrix (Matrix 2)

Matrix 5 *The modified muscle matrix [M′]*

	lmr	sir	sio
E*RALP*	0 007	0 34	0 98
E*LARP*	0 0 4	0 940	0 33
E*z*	0 999	0 0 6	0 40

Plane of action of the extra ocular muscles in the left eye lmr = lateral and medial rectus; sir = superior and inferior rectus; sio = superior and inferior oblique E*RALP*, E*LARP*, and E*z* refer to the vector components of eye velocity in the RALP, LARP and yaw planes, respectively The first named muscle pulls in the direction indicated by the sign of the vector, and the second named muscle pulls in the opposite direction

From this representation of the muscle matrix of the left eye, it is clear that the obliques pull predominantly in the RALP plane (0 98), the horizontal recti pull almost entirely in the horizontal plane (0 999), and the vertical recti pull predominantly in the LARP plane (0 94), but they also have some action in the RALP plane (0 34) By considering the direction of muscle action, the left superior oblique pulls in the RA direction (producing downward rotation and intorsion) The left superior rectus pulls mainly in the RP direction (producing upward rotation and intorsion) but also has a lesser action in the LP direction; the result is a largely upward eye rotation (0 906) with a smaller degree of intorsion (0 424) (Matrix 4)

The brainstem matrix

Robinson (982) derived the 'brainstem matrix' which determines the pattern and relative strength of the neural projection from each pair of SCCs onto each pair of ocular motor neurons The VOR is a product of three matrices: the canal pair sensitivity matrix [C], the brainstem matrix [B], and the muscle matrix [M] The canal pair sensitivity matrix [C] was calculated as the average plane for each SCC pair Thus if V is the eye velocity output and V_H is the head velocity stimulus, then

$$V = [M][B][C]V_H \qquad \text{(Equation)}$$

Robinson derived the brainstem matrix by assuming that the VOR is ideal, in other words, V = V_H If this is true, then

$$[M][B][C] = [\ I] \qquad \text{(Equation 2)}$$

where [I] is the negative of the identity matrix

Matrix 6 *[I] the negative of the identity matrix*

	0	0
0		0
0	0	

Robinson then calculated the brainstem matrix by rearranging Equation 2

$$[B] = [M^{-1}][\ I][C^{-1}] \qquad \text{(Equation 3),}$$

where $[M^{-1}]$ and $[C^{-1}]$ are the inverse of their respective matrices

We have modified the VOR gain element [I] to reflect a more realistic value for the roll VOR gain of 0 7 (Aw *et al* , 996*a*)

Matrix 7 *[VOR] the VOR gain matrix*

	0	0
0		0
0	0	0 7

[VOR] was used in our calculation of the brainstem matrix

$$[B] = [M^{-1}][\ VOR][C^{-1}] \qquad \text{(Equation 4)}$$

Using Equation 4, we calculated the brainstem matrix

Matrix 8 *The brainstem matrix [B]*

	Clrl	Clarp	Cralp
lmr	0 6	0 230	0 59
sir	0 06	0 877	0 33
sio	0 52	0 089	0 740

Clrl = canal pair sensitivity vector for the left and right lateral SCCs; Clarp = canal pair sensitivity vector for the left anterior/right posterior (LARP) SCC pair; Cralp = canal pair sensitivity vector for the right anterior/left posterior (RALP) SCC pair A positive value indicates that the first named SCC excites the first named extra ocular muscle, and the second named SCC excites the second named extra ocular muscle A negative value indicates that the first named SCC excites the second named extra ocular muscle, and vice versa

Specificity of canal plane head impulses

Because the vertical SCCs do not lie exactly in the diagonal LARP and RALP planes, and because the SCCs are not exactly orthogonal

to each other (Blanks *et al* , 975*a*), head impulses in these canal planes will stimulate out of plane SCCs to some extent For example, during a LA direction head impulse, the left lateral SCC is stimulated by 0 37 of the total head acceleration (Matrix 3) In this case, how much of the resulting eye velocity that we plot in the LARP plane is due to left lateral SCC stimulation?

From the brainstem matrix (Matrix 8), we can see that stimulation of the left lateral SCC causes contraction of the left medial rectus with a relative efficacy of 0 6, contraction of the left superior rectus (0 06) and left superior oblique (0 52) By multiplying the muscle matrix (Matrix 4) by column of the brainstem matrix (Matrix 8), we calculate that the resulting eye rotation is rightward (0 992) with some intorsion (0 50), without any vertical component (which is cancelled by the opposing actions of superior oblique and superior rectus) To calculate the LARP plane contribution, we multiply the modified muscle matrix (Matrix 5) by column of the brainstem matrix (Matrix 8) The relative LARP vector component of the eye velocity is 0 06 in the RP direction However, during a LA direction head impulse, the left lateral SCC is only stimulated by 0 37 of total head acceleration, so the relative LARP vector component of the VOR due to the left lateral SCC is only 0 039

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NEUROLOGY

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

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Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

The video head impulse test

Diagnostic accuracy in peripheral vestibulopathy

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ABSTRACT

Background: The head impulse test (HIT) is a useful bedside test to identify peripheral vestibular deficits. However, such a deficit of the vestibulo-ocular reflex (VOR) may not be diagnosed because corrective saccades cannot always be detected by simple observation. The scleral search coil technique is the gold standard for HIT measurements, but it is not practical for routine testing or for acute patients, because they are required to wear an uncomfortable contact lens.

Objective: To develop an easy-to-use video HIT system (vHIT) as a clinical tool for identifying peripheral vestibular deficits. To validate the diagnostic accuracy of vHIT by simultaneous measures with video and search coil recordings across healthy subjects and patients with a wide range of previously identified peripheral vestibular deficits.

Methods: Horizontal HIT was recorded simultaneously with vHIT (250 Hz) and search coils (1,000 Hz) in 8 normal subjects, 6 patients with vestibular neuritis, 1 patient after unilateral intratympanic gentamicin, and 1 patient with bilateral gentamicin vestibulotoxicity.

Results: Simultaneous video and search coil recordings of eye movements were closely comparable (average concordance correlation coefficient r_c = 0.930). Mean VOR gains measured with search coils and video were not significantly different in normal (p = 0.107) and patients (p = 0.073). With these groups, the sensitivity and specificity of both the reference and index test were 1.0 (95% confidence interval 0.69–1.0). vHIT measures detected both overt and covert saccades as accurately as coils.

Conclusions: The video head impulse test is equivalent to search coils in identifying peripheral vestibular deficits but easier to use in clinics, even in patients with acute vestibular neuritis.

Neurology® 2009;73:1134–1141

GLOSSARY

BVL = bilateral vestibular loss; **HIT** = head impulse test; **IMU** = inertial measurement unit; **ITG** = intratympanic gentamicin; **vHIT** = video head impulse test; **VN** = vestibular neuritis; **VOR** = vestibulo-ocular reflex.

The head impulse test (HIT) is a useful bedside examination to identify a peripheral vestibular deficit for example in patients with vestibular neuritis (VN).[1-4] The clinician briskly rotates the patient's head to detect "overt" catch-up saccades after head rotation as a sign of semicircular canal paresis. "Covert" saccades are saccades that occur during the head rotation that may be imperceptible to the naked eye and hence confound the diagnosis.[5,6] In patients with acute VN, spontaneous nystagmus also interferes with assessment of bedside HIT.

Up to now, the scleral search coil technique has been the gold standard for HIT measurements.[7-9] It quantifies the VOR deficit and shows the associated pattern of overt and covert catch-up saccades in vestibular deficient patients.[6,10] However, search coil measurements require the subject to wear an uncomfortable contact lens, are time intensive, are expensive, and are not practical for acute patients.

The goal of the study was to develop an easy-to-use high-speed video HIT system[11] (see video on the *Neurology*® Web site at www.neurology.org) as a clinical tool to identify a periph-

Supplemental data at www.neurology.org

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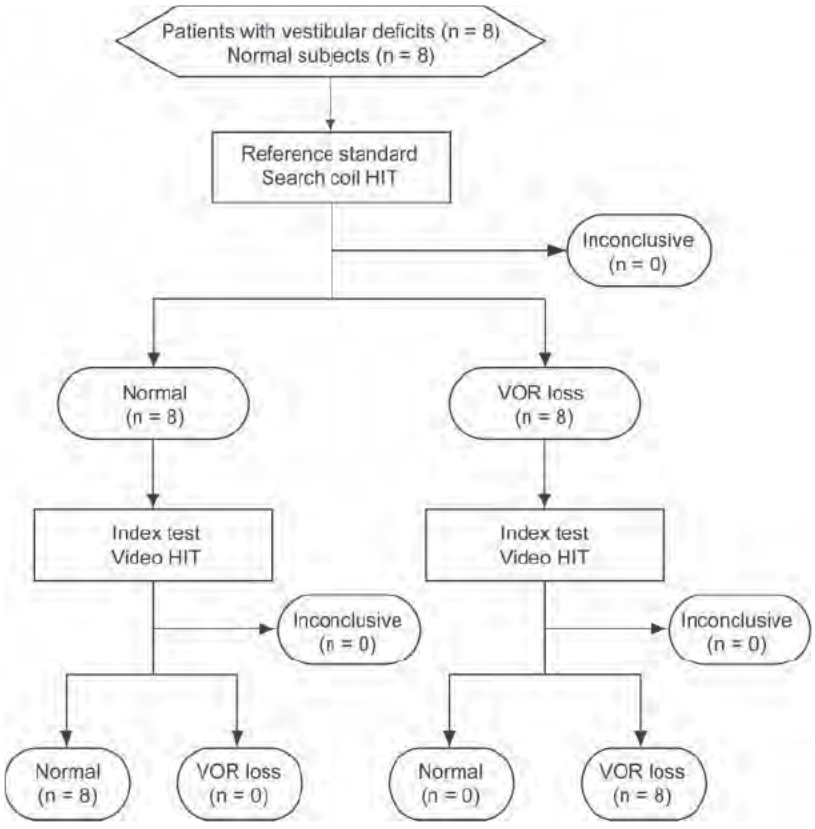
Disclosure: Author disclosures are provided at the end of the article.

eral vestibular deficit. To validate the diagnostic accuracy of our video HIT (vHIT) system, we compared the measures from video recordings with simultaneous measures from scleral search coil recordings of the same eye during head impulses in healthy subjects and patients with a wide range of previously independently identified vestibular deficits.

METHODS **Design.** The study was a prospective, cross-sectional comparison of the index test (vHIT) to the reference standard (HIT measured by scleral search coils) in patients with prior, independently identified vestibular deficits due to unilateral vestibular neuritis, intratympanic gentamicin, or systemic gentamicin and healthy asymptomatic control subjects (figure 1). Patients with a broad range of vestibular deficits were enrolled because we wished to establish how well each test identified vestibular deficits of varying severity.

Subjects. Sixteen subjects were recorded simultaneously with video-oculography and scleral search coils. Six patients with VN (mean 52 years, age range 38–59 years, 1 female) showed evidence of enduring unilateral loss of vestibular function after an illness with acute onset of prolonged rotational vertigo and postural imbalance associated with spontaneous nystagmus, nausea, or vomiting that fulfilled the clinical criteria of VN.[12] One patient with Ménière disease (53 years, female) with a unilateral vestibular deficit due to intratympanic gentamicin injection and a patient with bilateral vestibular loss due to systemic gentamicin vestibulotoxicity (72 years, male) were also tested. Eight healthy subjects without any history, symptoms, or clinical signs of vestibular disease (mean 35 years, age range 25–66 years, 2 females) served as controls. Diagnosis of a peripheral vestibular deficit was confirmed in all patients by bithermal caloric testing with water irrigation at 30°C and 44°C, resulting in a canal paresis factor greater than 25%.[13] Patients were tested between 5 months and 27 years after onset of symptoms. Two additional patients with acute VN (29 and 32 years, both female) were recorded within 36 hours after onset with video-oculography alone. Eligible patients were recruited at the Hearing and Balance Clinic, Royal Prince Alfred Hospital, Sydney, Australia. No potential subject was excluded. All subjects and patients were tested between August and October 2008.

Figure 1 **Flow chart for the comparison of video and search coil measures of head impulses**



HIT = head impulse test; VOR = vestibulo-ocular reflex.

Standard protocol approvals and patient consents. Written informed consent was obtained from all subjects. Written consent to disclose has been obtained from any recognizable persons in the published photograph and video. The protocol was approved by the Sydney South West Area Health Service Ethics Committee in accordance with the Declaration of Helsinki.

Experimental procedure. Subjects were instructed to fixate a laser dot on a screen at 91 cm distance in dim light. Approximately 50 horizontal head impulses to each side were manually applied with unpredictable timing and direction. Peak head velocity of the impulses was gradually increased from 50° to 250°/second (acceleration 750°–5,000°/second^2, amplitude 5°–20°) with the aid of visual feedback of head velocity for the experimenter.[5,6] The same eye was recorded simultaneously with video-oculography and scleral search coils (figure e-1). Two data sets were obtained for each recording session to show the reliability of the calculated gains and concordance of the video and search coil methods. All recordings were performed by the same team of 3 coauthors. Head impulses were always delivered by the same experimenter, unless acting as a subject. The experimenters were unmasked as to whether they were testing a patient or a healthy subject. All experimenters are graduates and have at least 5 years' experience in vestibular research. There were no adverse events from performing the tests.

Video-oculography. Right eye position was recorded at 250 Hz with a small, lightweight, high-speed digital (IEEE 1394a) video camera (Firefly MV, Point Grey Research Inc., Vancouver, British Columbia, Canada). The camera was mounted on a very lightweight motorcycle glasses frame with an elastic strap that locked comfortably onto the bridge of the nose and around the eye sockets to minimize slippage of the camera relative to the head. The image of the eye was reflected from a hot mirror to the camera. The eye was illuminated by 2 infrared light–emitting diodes (TSUS502, Vishay Intertechnology, Malvern, PA) run at 20 mA to keep infrared radiation far below exposure risk levels.[14] Head velocity was measured by a miniature 6-degrees-of-freedom inertial measurement unit (IMU) assembled from 2 dual-axis gyroscopes (IDG-300 InvenSense, Santa Clara, CA) and a 3-axis linear accelerometer (ADXL330, Analog Devices, Norwood, MA). The camera, hot mirror, and IMU were rigidly mounted onto the spectacle frame. The small mass of the system (approximately 60 g) minimized inertia during head rotation and so minimized slippage of the glasses. Eye position was calibrated in vivo with projected targets from a glasses-mounted laser. Video images were analyzed online to calculate eye position using a pupil detection method based on a center-of-gravity algorithm[15] written in LabVIEW (National Instruments, Austin,

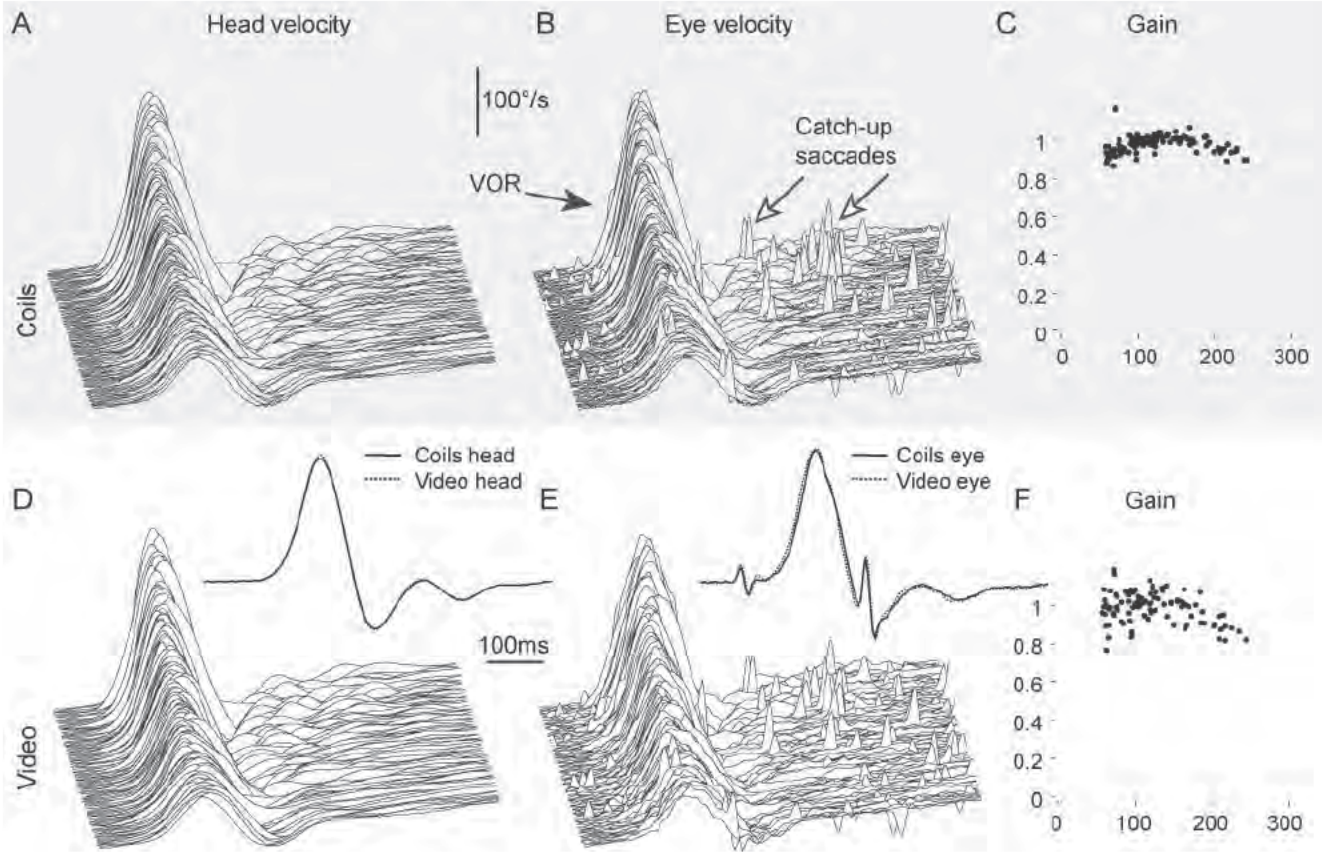
TX). Eye velocity was obtained from a 2-point differentiator and low-pass filtered (0- to 30-Hz bandwidth).

Scleral search coil recording. Right eye and head position were recorded with the scleral search coil technique in a 1.9 × 1.9 × 1.9-m magnetic coil frame (CNC Engineering, Seattle, WA).[2,9] Dual search coils (Skalar, Delft, The Netherlands) were precalibrated in vitro on a gimbal. The eye coil was inserted after topical anesthesia with Alcaine 0.5% eyedrops (Alcon Laboratories Australia Pty. Ltd., Frenchs Forest, Australia). The head coil was attached to a dental impression tray. Three-dimensional head and gaze position signals were sampled at 1,000 Hz, digitized with 16-bit precision, and low-pass filtered (0- to 100-Hz bandwidth). Three-dimensional rotation vectors and angular velocity vectors of head, gaze, and eye were derived from coil voltages.[16]

Data analysis. Offline analysis of the experimental data were automated with customized LabVIEW software. To synchronize the video and search coil measurements, a square wave signal produced by a signal generator was acquired by each system together with eye and head velocity measurements. Data from the 2 systems were then synchronized by aligning the square wave signals. Head impulses were automatically selected and aligned to peak head acceleration. Trials with blinks and outliers were automatically excluded, based on an envelope around the expected eye velocity response. Velocity gain of the horizontal VOR[7] was calculated for both recording methods as the ratio of mean eye velocity over mean head velocity during a 40-msec window centered at peak head acceleration. Data from both recording methods was processed simultaneously with the same automated algorithms to exclude any analysis bias. Invalid head impulses (e.g., with blinks) were excluded from both data sets, resulting in mirror-symmetric data. Both data sets were analyzed in all simultaneously recorded patients (no missing data sets). The criterion for a normal VOR velocity gain was that it should be 0.68 or greater, based on HIT data from 12 previously published healthy asymptomatic subjects[6,10] in which the mean HIT velocity gain measured by search coils with identical apparatus and procedures to those used here was 0.81 ± 0.068 SD, so that the mean ± 2 SD units incorporates 95% of the population and yields a lower cutoff of 0.68.

Statistical analysis. The results of the study are reported in accordance with the Standards for Reporting of Diagnostic Accuracy Studies (STARD).[17,18] The concordance correlation coefficient[19] was used to index the similarity between video and search coil recordings for each impulse. A coefficient was calcu-

Figure 2 Simultaneous video and search coil recordings of a horizontal head impulse test in a normal subject



Simultaneous angular head velocity recordings of a search coil mounted on a dental impression tray (A) and a gyroscope mounted on the video glasses (D) during graded horizontal head impulses. The close similarity between the 2 recordings demonstrates minimal slippage of the video glasses relative to the head. Simultaneous angular eye velocity recordings of a scleral search coil (B) and high-speed video-oculography (E) of the same eye. Both recording techniques accurately record the vestibulo-ocular reflex (VOR) and detect even the smallest catch-up saccades. Normal VOR gains of individual head impulses are comparable with search coil recording (C) and video-oculography (F). Scleral search coil recording is sampled at 1,000 Hz (A and B), video-oculography is sampled at 250 Hz (D and E), and both are plotted on the same time scale. Head and eye velocity traces from individual impulses are stacked according to increasing peak head velocity. (Insets D and E) Simultaneous video and search coil recordings are shown superimposed to facilitate comparison of single head and eye velocity traces.

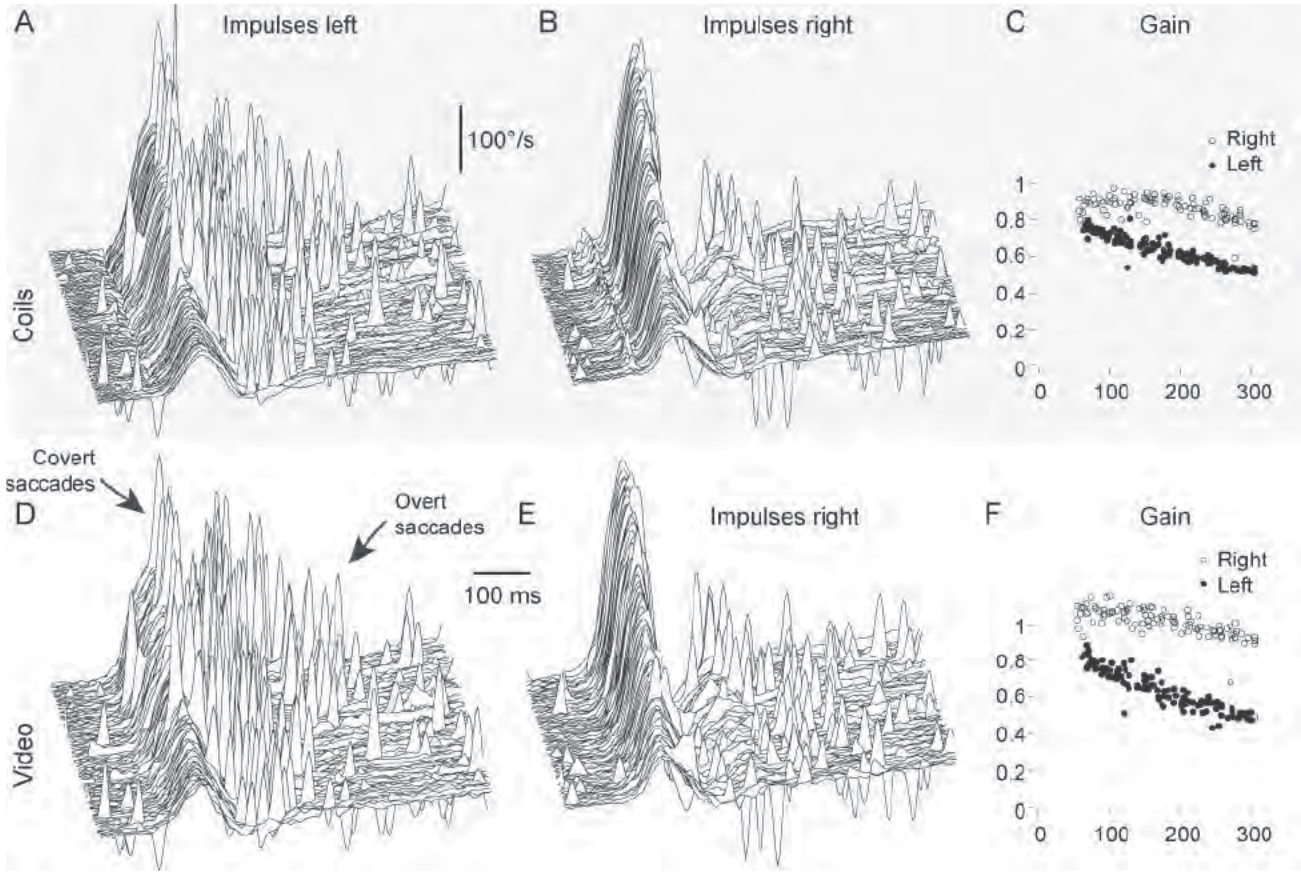
lated for each impulse and the values for an individual were averaged. Paired-sample t tests were used to test whether the VOR gains were different in video and search coil recordings. The Pearson product-moment correlation coefficient was used to determine test-retest reliability between the 2 separate test runs. Because of the small sample size, no subgroup analyses were performed by disease subtype or clinical examiner.

RESULTS **Head movement recording.** A close fit of the video glasses on the head is crucial for accurate eye movement recording during head impulses.[20] Simultaneous measurements of angular velocity from an inertial measurement unit (IMU) mounted on the glasses and from a search coil on a dental impression tray were virtually identical (figure 2, A and D), with an average concordance correlation coefficient[19] of $r_c = 0.999$.

Eye movement recording. Simultaneous measurements of video recording and scleral search coil of the same eye are shown in figure 2, B and E (normal subject), and figure 3, A, B, D, and E (patient with VN). Both recording techniques not only show VOR, but also record the smallest catch-up saccades (figures 2 and 3, arrows). Each method is highly repeatable, and the 2 methods are in very close agreement. The concordance correlations were calculated for every impulse in all subjects and patients: The mean r_c for each subject is shown in figure 4, and the average r_c of all subjects was 0.930.

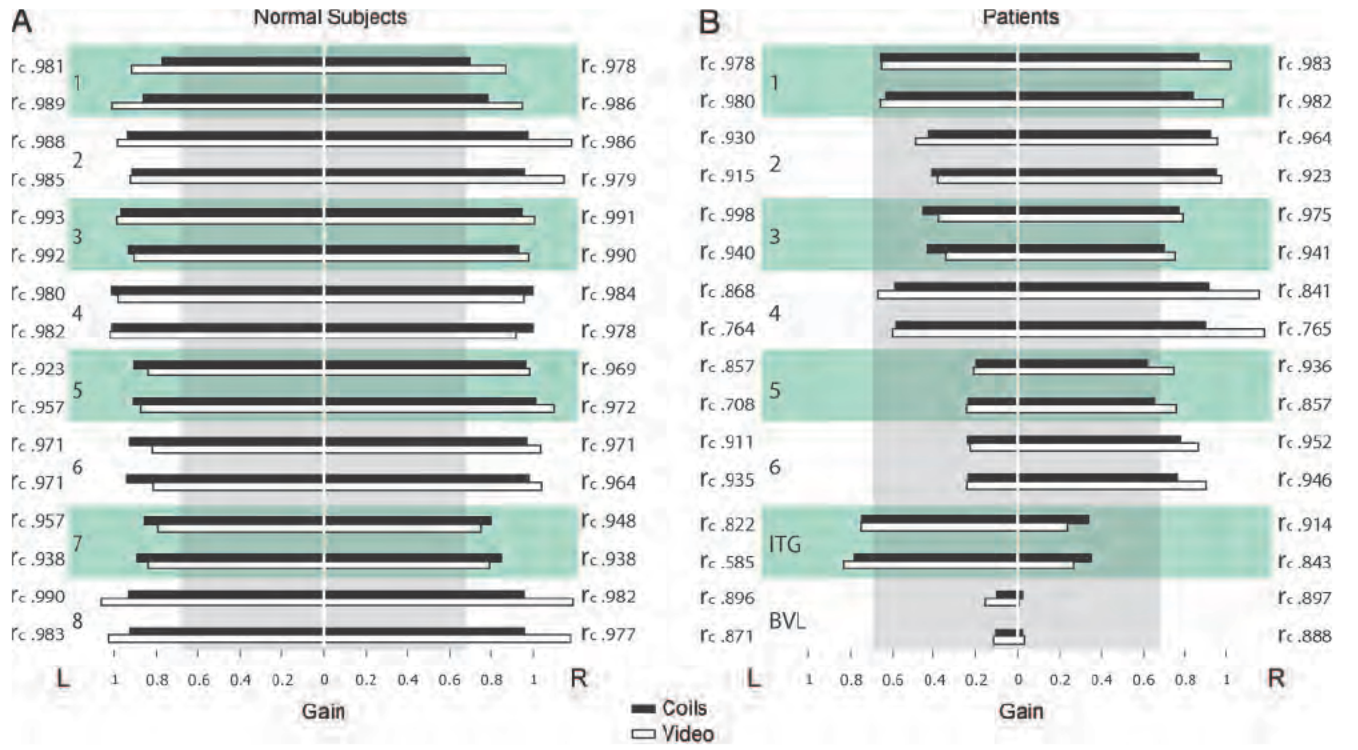
Detection of vestibular deficit. A VOR deficit was defined as being a HIT gain of less than 0.68. The reference standard, scleral search coils, correctly identified the VOR deficit in all patients, as did the video measures (figures 1 and 4). Thus, the sensitivity of the reference and index test were both 1.0 (95% confidence interval 0.69–1.0), and the specificity of both the reference and index test were 1.0 (0.69–1.0).[21] Using a paired t test, the difference between average VOR gain for search coils and video was not significantly different from zero for patients (mean difference = 0.040, n = 8, $t = 1.930$, $p = 0.073$) and for normal (mean difference = 0.043, n = 8, $t = 1.717$, $p = 0.107$). To test reproducibility, each

Figure 3 Simultaneous video and search coil recordings of a horizontal head impulse test in a patient after left vestibular neuritis



(A and D) Head impulses to the left (affected) side demonstrate the reduced vestibulo-ocular reflex (VOR) response. Both recording methods detect covert saccades during head rotation and overt saccades after head rotation (arrows). The pattern of catch-up saccades is identical for both recording methods. (B and E) Both recording methods demonstrate an almost normal VOR response to the healthy right side, with small overt saccades after head rotation. (C and F) Both recording methods clearly differentiate the reduced VOR gains of the left affected side (filled circles) from the right healthy side (empty circles). (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

Figure 4 VOR gain measures with search coils compared with video-oculography in normal subjects and patients with peripheral vestibular deficits



(A) The vestibulo-ocular reflex (VOR) gain for healthy subjects is almost identical for the 2 different methods of measurement. The 2 sets of data give highly reproducible values of VOR gain. (B) Video-oculography identifies the affected side in vestibular neuritis (1–6) and intratympanic gentamicin (ITG, 7) patients as reliably as search coil measurements. Patient 8 with bilateral vestibular loss (BVL) due to systemic gentamicin vestibulotoxicity demonstrates reproducibility of both methods at very low VOR gains. Bar graphs show the mean VOR gain measures with search coils (black bars) compared with video-oculography (white bars). For each individual, 2 data sets were recorded in the same session. Concordance correlation coefficients (r_c) index the similarity between search coil and video-oculography measurements. The vertical gray box indicates deficient VOR gain values (cutoff gain 0.68).

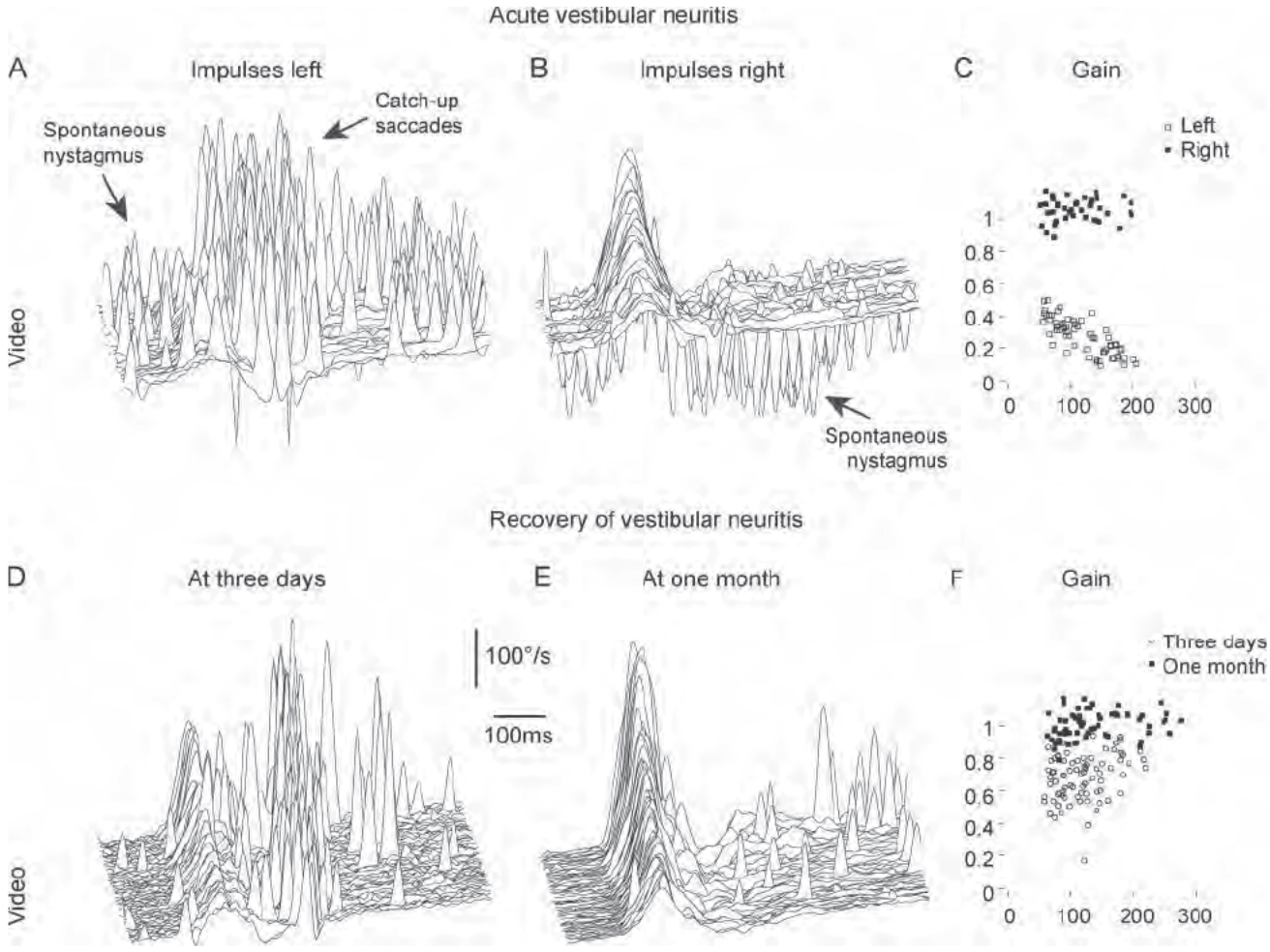
subject was given 2 separate test runs, and the test-retest reliability coefficient using a Pearson product-moment correlation was significant at 0.99 for search coils and 0.99 for the video measures. Both recording techniques readily detected covert saccades during head rotation as well as overt saccades after head rotation (figure 3, A and D).

Clinical application. This video method of recording eye movements during head impulses makes examination of acute vertigo patients possible where scleral search coil recordings are impractical. The unilateral VOR deficit of patients with acute VN can be detected even in the presence of spontaneous nystagmus (figure 5, A–C). The noninvasive and short (approximately 10 minutes) nature of vHIT also facilitates follow-up examinations to document recovery of vestibular function (figure 5, D–F).

DISCUSSION In 1988, 2 of the authors reported[1] a simple indicator that allowed clinicians at the bedside to identify peripheral deficits of horizontal semicircular canal function—the presence of a saccade after a small, rapid, passive, unpredictable, horizontal head rotation (a "head impulse") by the clinician while the patient attempted to maintain gaze on a target. If semicircular canal function is impaired, the slow phase eye velocity is inadequate, so the eyes move with the head (off the target), and at the end of the head rotation, the patient must make a saccade to return gaze to the target. That corrective saccade is easily detectable if it is made after the head rotation has stopped, and so these saccades are termed "overt" saccades.[6]

The HIT has found wide use as a qualitative clinical sign, but it has major limitations: 1) There is no objective measure of VOR gain or of the corrective saccade: the clinician's report is based on the subjective visual observation of the presence of an overt saccade. 2) Different clinicians carry out this impulse with very different trajectories, so the accelerations and velocities used differ considerably. 3) Usually only few head rotations are given, so there is not a range of stimuli for generating a stimulus-response function. 4) Some patients may hide their peripheral vestibular deficit with "covert" saccades during head rotation. As such, their peripheral pathology is missed, and it may be incorrectly concluded that they

Figure 5 Diagnosis of acute vestibular neuritis and documentation of recovery



(A–C) Video head impulse test of a patient 2 days after onset of acute vestibular neuritis. (A) In head impulses to the affected left side, catch-up saccades replace the deficient vestibulo-ocular reflex (VOR). The spontaneous nystagmus (scattered spikes) beats in the same direction as the catch-up saccades. (B) In head impulses to the healthy right side, the VOR is preserved and the spontaneous nystagmus beats to the opposite direction. (C) The VOR gain is deficient to the left (open squares) but preserved to the right (filled squares). (D–F) Video head impulse test of a patient 3 days (D) and 1 month (E) after onset of acute vestibular neuritis. Between the 2 recordings, the VOR gains returned toward normal (F), the majority of catch-up saccades disappeared, and the patient recovered from symptoms. (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

have a central vestibular disorder responsible for their symptoms. Such a "covert" saccade is almost impossible to detect by simple visual observation: it cannot be distinguished from the normal slow phase eye velocity needed for proper compensatory eye movement.

To overcome these limitations, we have developed a new lightweight, minimal-slip, high-speed video-oculography system[11] (vHIT, video) that measures eye velocity during head rotation. Importantly, the camera is mounted on a specially designed, very lightweight frame to minimize inertia and slippage (figure e-1). Instant feedback about every single head impulse allows the examiner to apply a set of standardized graded impulses. The system is easy to use in a clinical setting, provides an objective measure of the VOR, and detects both overt and covert catch-up saccades in patients with vestibular loss. Measurements are quick (approximately 10 minutes) and noninvasive, and the automated analysis software provides instant results.

The simultaneous video and search coil HIT recordings validate the diagnostic accuracy of high-speed video recording. Despite fundamentally different recording methods, we achieved head and eye velocity recordings that were closely comparable. Both methods correctly identified the peripheral vestibular deficit in patients with highly reproducible VOR gains and detected even the smallest catch-up saccades.

With the bedside HIT, clinicians have to deliver high head velocities to optimize the chance of detecting the corrective saccade.[6] Although high velocities also help to reveal VOR asymmetry in patients with VN,[6] lower velocities of approximately 100° to 150°/

second are sufficient to detect the deficit in acute patients (figure 5C). In practice, this is an advantage for vHIT because the effects of slippage of the glasses and inertia are smaller at lower head velocities, and patients with acute vertigo better tolerate these lower velocities.

The simple application of vHIT allows the clinician to diagnose patients with VN acutely while they are ill and assess them again after they have recovered, providing objective evidence of the VOR deficit and the extent of its recovery. As figure 5, A–C, shows, these measures are possible even in the presence of a very vigorous spontaneous nystagmus.

Bedside HIT remains a useful clinical sign to assess patients with acute spontaneous vertigo because it helps to distinguish between acute VN, where the test is positive, and a central vestibular lesion, where the test is usually negative. However, between 9% and 39% of positive clinical HIT results have been reported in patients with acute cerebellar or brainstem strokes.[22,23] vHIT will be a suitable tool to determine whether these cases are really due to reduced VOR gain or simply result from clinical misjudgment. This way, video HIT will help to improve diagnostic accuracy for patients with acute spontaneous vertigo in the emergency department.

ACKNOWLEDGMENT

The authors thank all the participants of the study, Anthony Anderson for recording of the video, and David Newman-Toker for advice.

AUTHOR CONTRIBUTIONS

The statistical analysis was conducted by the authors Hamish G. MacDougall and Ian S. Curthoys (Vestibular Research Laboratory, School of Psychology, University of Sydney, Australia).

DISCLOSURE

Dr. MacDougall receives research support from the National Aeronautics and Space Administration [NNJ04HF51G (Coinvestigator) and NNJ04HD66G (Coinvestigator)], the National Space Biomedical Research Institute [NNJ07ZSA002N (Co-PI) and SM00801 (Co-PI)], the European Space Agency [SPIN (Coinvestigator)], the Australian Research Council [DP0665402 (APD)], and the Garnett Passe and Rodney Williams Memorial Foundation [Project Grant 2009 (PI)]. Dr. Weber received a research fellowship from the Garnett Passe and Rodney Williams Memorial Foundation. Dr. McGarvie reports no disclosures. Dr. Halmagyi serves on the editorial boards of *Acta Otolaryngologica*, *Otology & Neurotology*, *Audiology & Neurotology*, and the *Italian Journal of Otolaryngology*; and receives research support from the National Health and Medical Research Council [570890 (Co-PI) and 457511 (Co-PI)]. Dr. Curthoys serves on the editorial advisory boards of *Experimental Brain Research* and *Brain Research Bulletin*; and has received research support from the Australian Research Council [DP-0665402 (Chief Investigator)], the National Health and Medical Research Council [457511 (Chief Investigator) and 509206 (Coinvestigator)], and the Garnett Passe and Rodney Williams Memorial Foundation [L2907 R6934 (Chief Investigator)].

Received March 14, 2009. Accepted in final form July 7, 2009.

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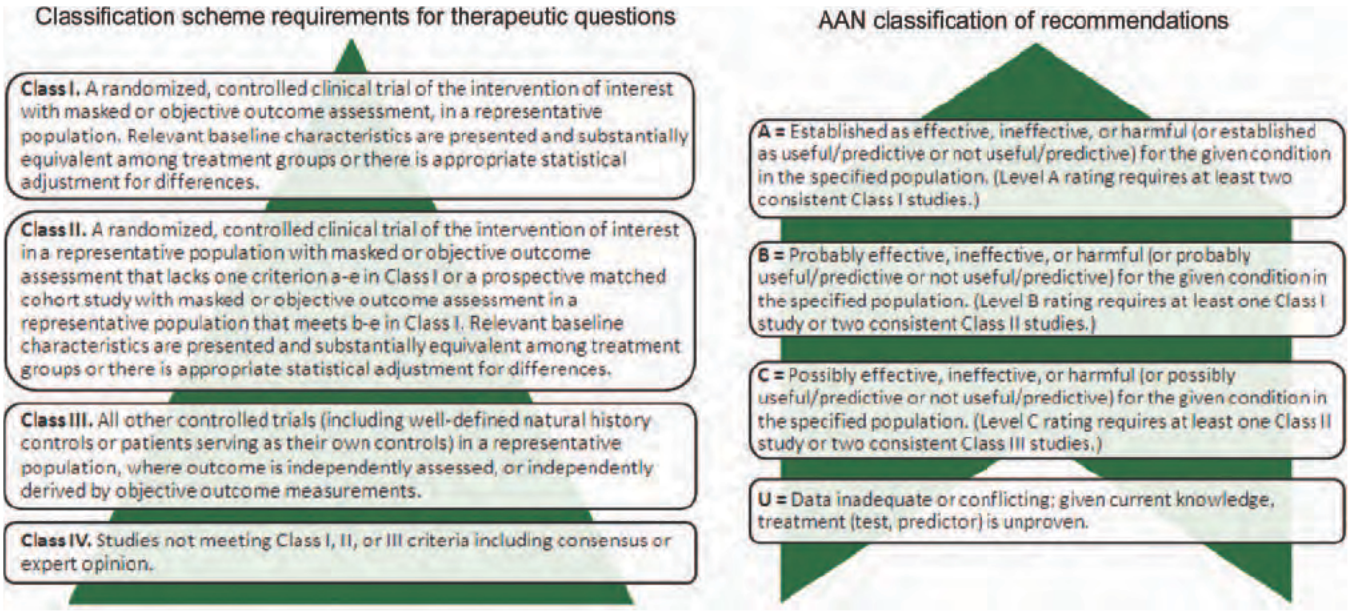

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

H. G. MacDougall, K. P. Weber, L. A. McGarvie, G. M. Halmagyi and I. S. Curthoys

Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

Clinical application of a new objective test of semicircular canal dynamic function – the video head impulse test (vHIT).

A safe, simple and fast clinical vestibular test.

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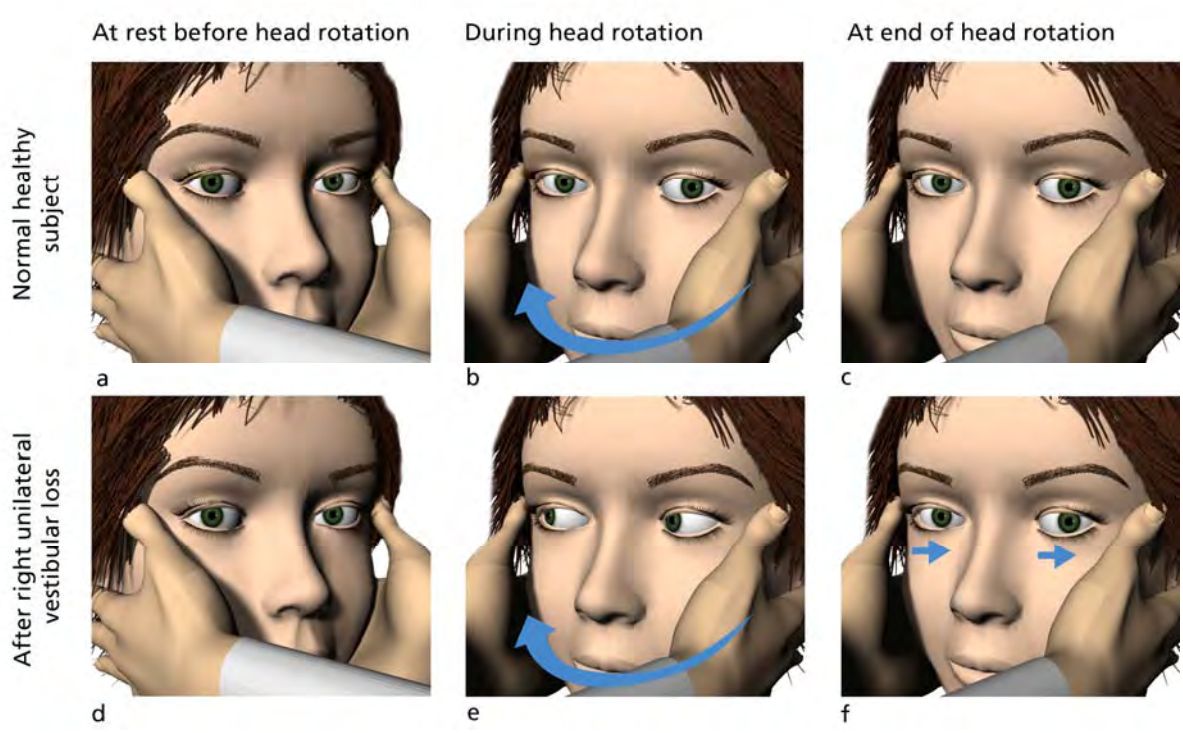
Preface

If you want to test hearing in the left ear, then you present a sound to the left ear to stimulate the auditory system and measure the response – for example whether a patient can hear the sound or whether there is a measurable physiological response to the sound. If you want to measure vision in the left eye, you present a visual stimulus to the left eye and ask the patient what they saw. Similarly if you want to test the semicircular canal function in the left ear ***you turn the person's head to the left*** to stimulate the semicircular canal in the left ear, and you measure the eye movement response while the patient tries to keep looking at a stationary target straight ahead. That is the response - just how well they can keep looking at the target. That head turn activates the receptors in the left horizontal semicircular canal and results in the eye movement response, which corrects for the head turn so that in healthy subjects the eyes stay looking at the target. So in the case of the semicircular canals, the response is that both eyes turn to correct for, or compensate, for the head turn - to keep looking at the target as the head is turned, so the person's gaze is stable during the head turn. The eye movement response is a tool to probe of the function of the semicircular canals of the inner ear. Of course to test the semicircular canals on the right you turn the patients head to the right.

What happens if the patient has ***no*** semicircular canal function in one ear? Now turn the head to that affected side but since the canal is not working, the eyes do not correct for the head movement. Instead of the eyes turning to correct for the head turn, ***the eyes move with the head***. So at the end of the head movement the patient must make a saccade back to the target. That saccade tells the clinician that the semicircular canal is not working properly – the response is just not adequate, so there is probably a deficit in the semicircular canal on that side. In most patients it is easy to see the corrective saccade at the end of the head turn and so we call it an ***overt saccade***. For example if, at the end of a head turn to the **left**, the clinician sees the patient has to make an overt saccade to get back to the target, then the semicircular canals on the **left** side are deficient.

This figure shows the difference between the responses of a healthy subject (top row) and a patient with a vestibular loss (bottom row), at comparable moments before, during and after the head rotation.



How can we objectively measure the adequacy of the semicircular canal response? One way is to measure the speed of the eye rotation and compare it to the speed of the head rotation. Eye velocity should be about equal and opposite head velocity and the ratio of the two velocities is called the vestibulo-ocular response (VOR) gain and in healthy subjects it is usually around 1.0. Or we can measure the saccades – whether they are there or not. The following explains how to do this test in real life.

The Head Impulse Test

The clinician stands before the patient, holding the patient's head in his hands, and the patient, who is looking straight at the clinician, is asked to keep staring at the earth-fixed target (the clinician's nose). If the clinician now turns the patient's head abruptly and unpredictably to the left or right, through a small angle (only 10-20 degrees - not a large angle), that head turn is what we call the **head impulse**. If the patient has a functioning vestibulo-ocular response they will be able to maintain gaze on the target because the vestibulo-ocular response drives the eyes to rotate to exactly compensate for head rotation and so maintain fixation. However if the patient's vestibulo-ocular response is inadequate then their eyes will be taken off target during the head rotation, because their eyes will not rotate at the correct speed to exactly compensate for head rotation. *So an inadequate VOR means that the eyes* ***go with the head during the passive unpredictable head turn*** *and will be taken off target by the head turn, so that at the end of the head turn the patient must make a corrective saccade back to the clinician's nose.* To the clinician watching the patient's eyes, this saccade is usually very clear, and we have termed it an ***overt saccade***. It is the tell-tale sign of inadequate semicircular canal function on the side to which the head was rotated. So an overt saccade after a leftwards head rotation means the left semicircular canal has a deficit. If there is any doubt, the clinician just repeats the head impulses until they are satisfied.

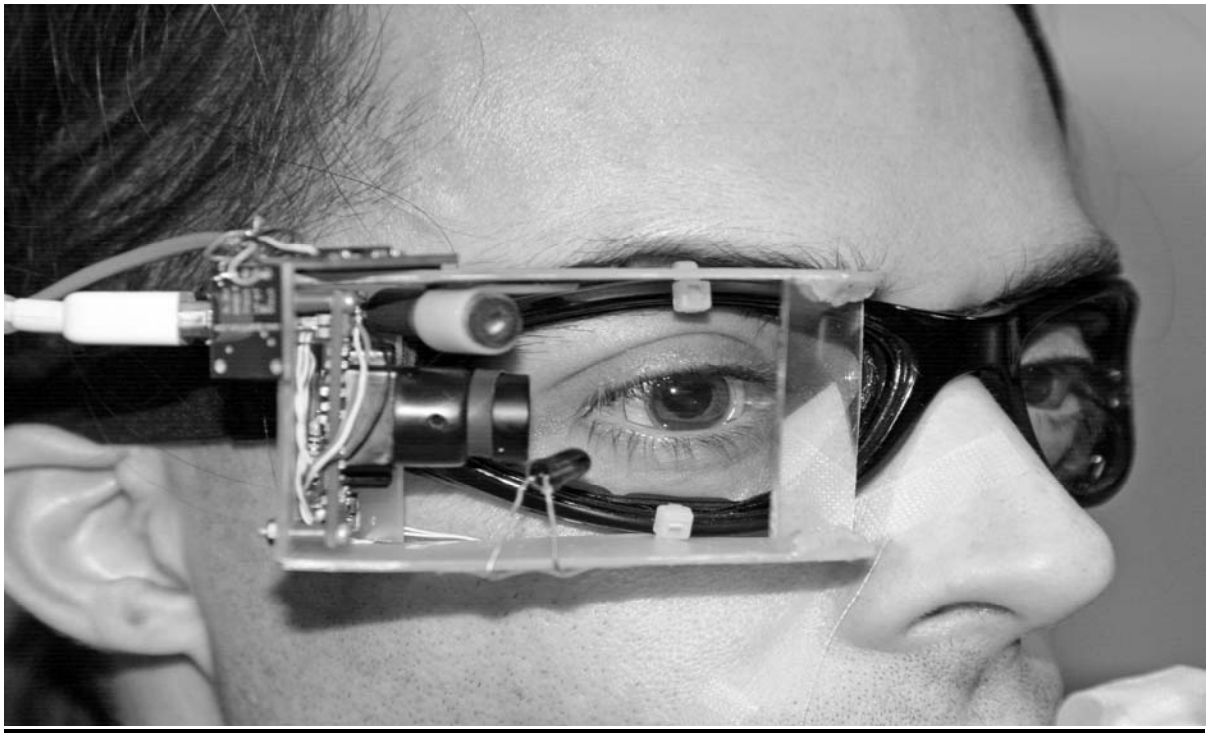
Covert saccades

Does the absence of an overt saccade mean that the canal is normal? No. Because our measures with scleral search coils (Weber et al 2008) showed that some patients with a semicircular canal deficit on one side could manage to generate small corrective saccades *actually during the head movement*, so that at the end of the head turn to their affected side hardly any overt saccades were necessary to bring the eyes back on target. These small hidden saccades during the head rotation had concealed their inadequate VOR. We have called these hidden saccades ***covert saccades***. It is important to realize that covert saccades can entirely obscure or conceal even a complete, total loss of canal function. These covert saccades are very fast and they occur *during* the head rotation and they are almost impossible to detect by the naked eye. It was only by using scleral search coils, the "gold standard" of eye movement measurement, during patient testing that we found them (Weber et al 2008), but clearly clinicians want to be able to detect them so they can accurately diagnose whether the patient has a vestibular loss or not and our new vHIT tests does just that. (Examples of recordings of overt and covert saccades are shown in the "Understanding vHIT Data" section)

The video Head Impulse Test (vHIT)

The simplest clinical indicator of a semicircular canal deficit is what I have just described - the head impulse test (also called the head thrust test, or the Halmagyi-Curthoys test, or the Halmagyi test). But detecting that saccade is ***subjective*** and relies on the clinician seeing the small corrective saccade after an abrupt head movement. The new indicator we describe below - the vHIT test uses a video camera to measure the eye movement and so it is ***objective*** and provides hard copy of the patient's performance. But first we will describe the test procedure and its logic.

This head impulse sign was described by Halmagyi and Curthoys in 1988, and from that time to the present, the clinical use of the head impulse test has been to indicate deficient canal function by virtue of the clinician (subjectively) observing whether there was an overt saccade or not at the end of the head turn. However some vestibular-deficient patients were missed by the head impulse test, even by expert clinicians, probably because of covert saccades. Clearly the ideal would be to have ***objective*** measure of both the head movement stimulus and the eye movement response using a system fast enough and accurate enough to detect covert saccades. The scleral search coil method of measuring eye movement achieves this aim, but it is clinically unrealistic, because of its huge expense, the high cost of each coil, the complexity of processing the data and the fact that patients do not like having a contact lens placed on their eye. However we have developed a new lightweight video system procedure – which we have called the video head impulse test (vHIT) – which does measure eye velocity and does detect covert saccades and is non-invasive and practical in clinics. Most importantly we have shown by direct comparisons that the accuracy of vHIT matches the accuracy of the "gold standard" search coil technique. vHIT has been validated by direct measures of VOR performance in healthy subjects and patients by two independent methods – search coils and vHIT. At ***exactly the same time: the same subject, the same eye movement responses were measured independently by these two methods and compared and found they both give essentially the same answer.***



How does the video head impulse test (vHIT) work?

The procedure is as described above, except that the patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage, because any slippage of the goggles will move the camera relative to the eye and so be registered as a movement of the eye and so generate artifactual data.

In testing, the clinician first conducts a quick calibration procedure, in which the patient is required to look between two laser spots projected from the goggles onto the wall. Then the clinician asks the patient to keep staring at an earth-fixed target, and gives the patient brief, abrupt, horizontal head rotations through a small angle (about 10-20 degrees), unpredictably turning to the left or right on each trial.

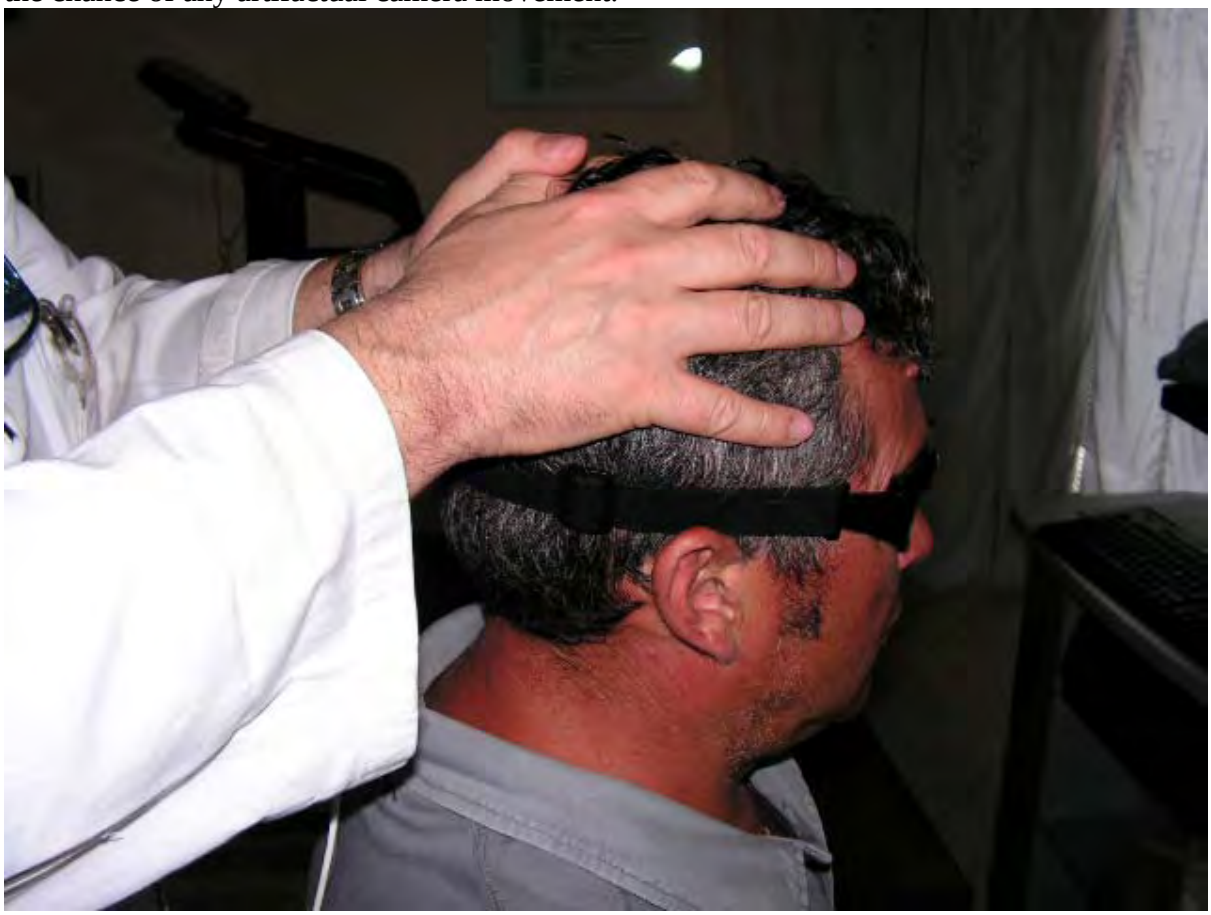
Stimulus

Displacement = 10° to 20°

Peak Head Velocity = 100°/s to 250°/s

Peak Head Acceleration = 1000°/s^2 to 2500°/s^2

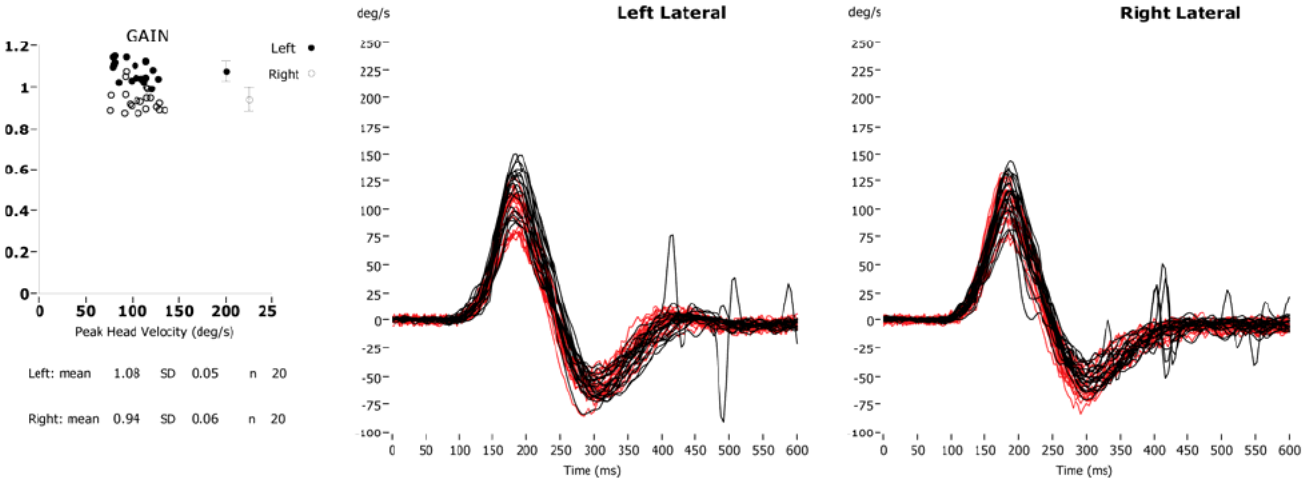
The clinician's hands must be well away from the goggles and the goggle-strap to minimize the chance of any artifactual camera movement.



The head movement speed is measured by the sensor in the goggles, and the image of the eye is captured by the high-speed firewire camera (250Hz) and processed by very fast software to yield eye velocity. At the end of each head turn the head velocity stimulus and eye velocity response are displayed simultaneously on the screen (figures below) so the clinician can see, just how good the stimulus and response were. In a full test usually around 20 impulses are delivered randomly in each direction and it may take 4 or 5 minutes to do that. At the end of the full test all the head velocity stimuli and eye velocity responses are superimposed and displayed on the computer screen, together with a graph of the calculated VOR gain for every head rotation as shown below in (Fig. 4). VOR gain is the ratio of eye velocity to head velocity, and so it should be ideally be about 1.0 for constant gaze during the head rotation. In practice normal healthy subjects typically have VOR gains less than 1.0 (around 0.8 - 0.9). But with vHIT any deficient response or VOR response asymmetry is easily seen. So in the space of about 5 minutes the clinician has an ***objective*** measure of the VOR response for both directions of rotation. The 250Hz video is fast enough that covert saccades can be detected and these are easily visible on the superimposed records (see below)

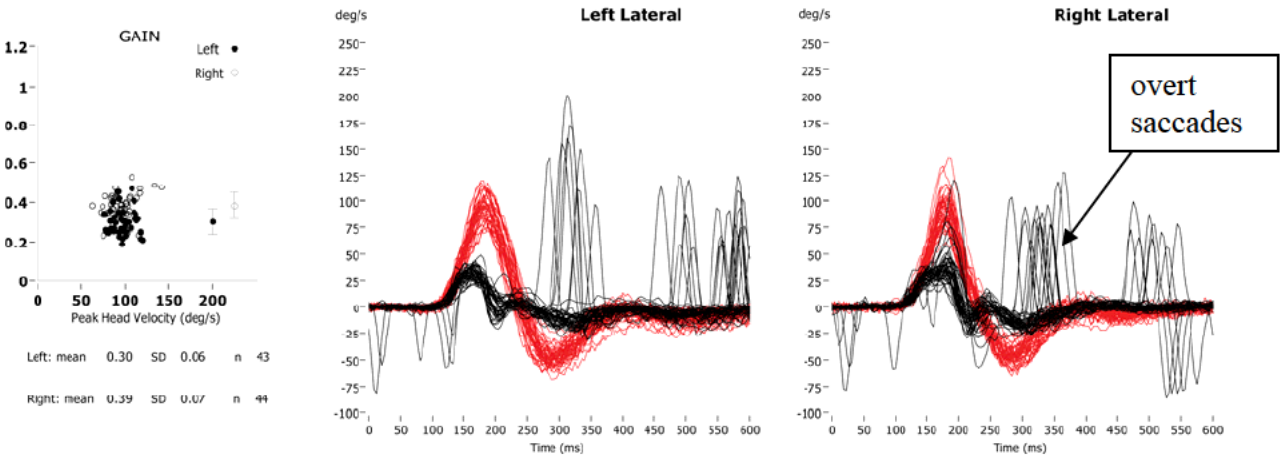
Examples of results from vHIT

1. Normal healthy subject



These and the following figures show superimposed records of eye velocity responses (black traces) to brief unpredictable horizontal head rotations (red traces) to the left and right. The first column shows the plot of the gain of the VOR for all of these (closed circles are for leftwards and open circles are for rightward impulses), as a function of peak head velocity. The average VOR gain is shown graphically and given numerically beneath the graph, together with the standard deviation and the number of impulses in each direction. Here the eye velocity matches head velocity so the head velocity traces and eye velocity traces are almost exactly superimposed showing that the eye velocity closely matches the head velocity. (We have inverted the eye velocity traces in these figures so you can more easily compare the eye velocity with head velocity.). Corresponding to that, the VOR gain values are good 1.08 for left and 0.94 for right. These VOR gain values are in the normal range and there is no asymmetry.

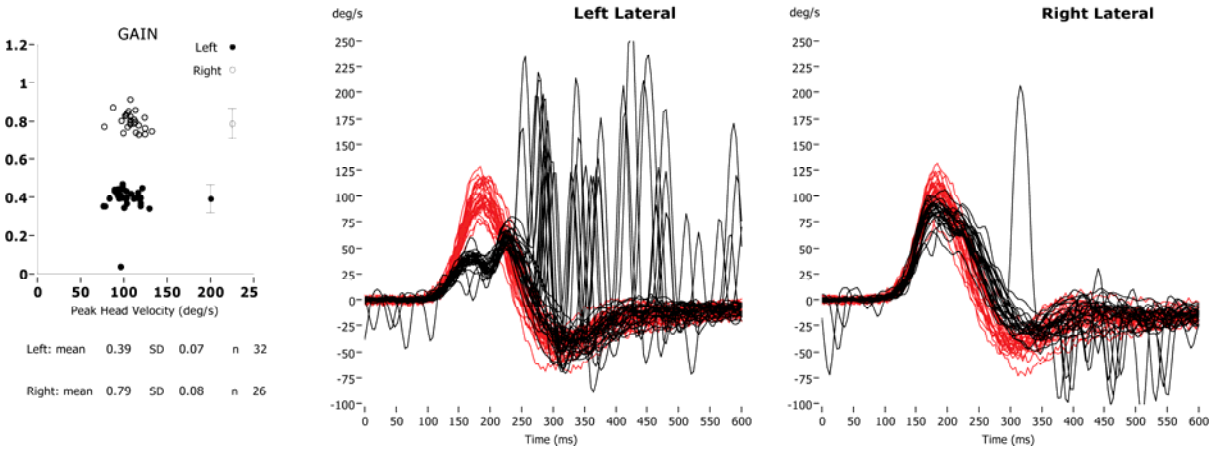
2. A patient with bilateral vestibular loss.



Comparable records from a patient with bilateral vestibular loss. Now the black traces (eye velocity) records do not follow the red (head velocity) records. The patient's VOR is clearly inadequate as the gain graph shows: the average VOR gain is 0.30 for left and 0.39 for right and both of these are significantly outside the normal range. In addition, there are large

saccades at the end of most head impulses and these are overt saccades and would be easily seen by the clinician at the end of the head turn.

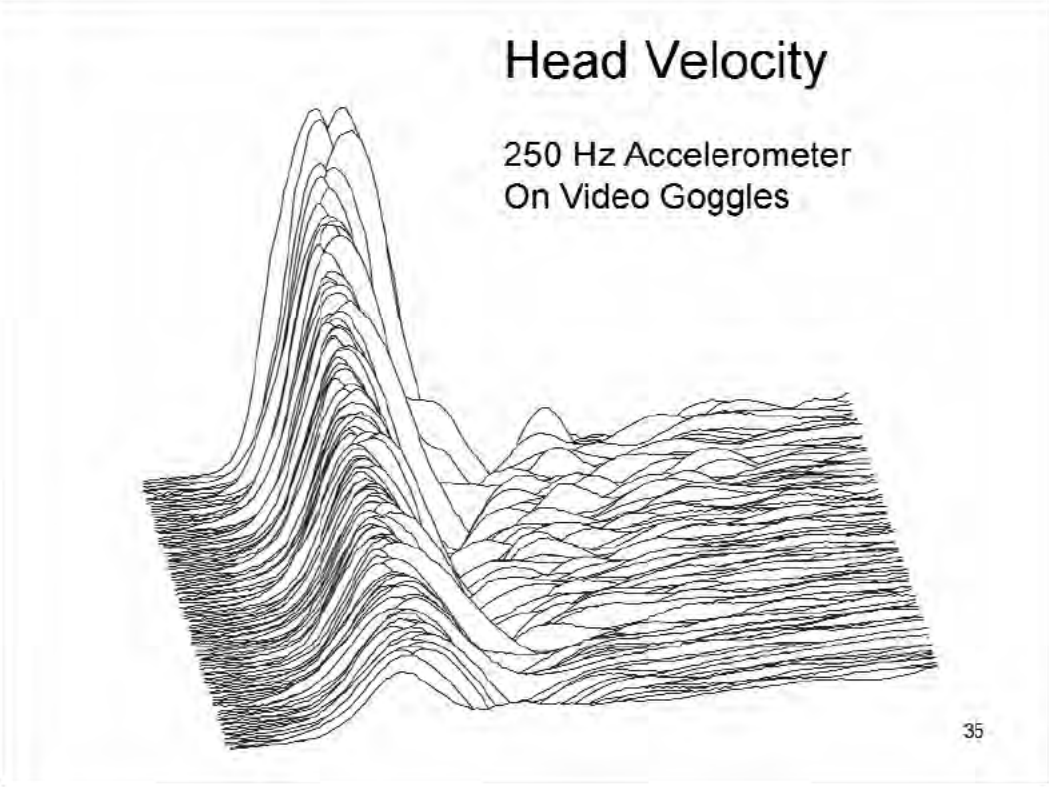
3. A patient with a unilateral vestibular loss

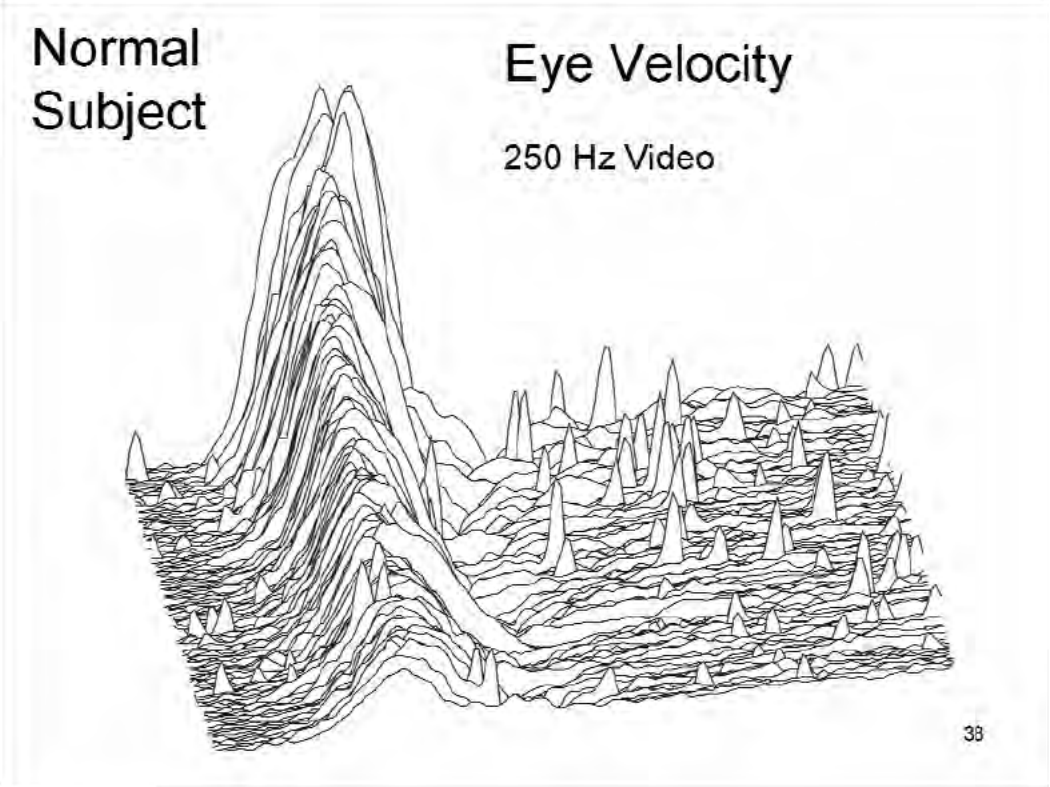


In this patient, eye velocity matches head velocity reasonably well for rightward head rotations, but not for leftwards head rotations (towards the patients affected side). Not only is the eye velocity insufficient, but there are many saccades which occur during the head impulse (covert saccades) and also some saccades which occur after the head impulse (overt saccades). The covert saccades would be very difficult for the clinician to detect by visual observation. The numerical values show the clear VOR gain difference for the two sides: to the right (healthy side) the average gain is 0.79 (in the normal range), whereas to the left (affected side) the average gain is only 0.39, significantly below the normal range. So this person has a severe left sided horizontal canal deficit.

The Graded series of head velocities

Instead of just giving one value of head velocity over and over again, as in the above examples, it is possible to give a graded series of increasing head velocities, which allows us to show the data in a 3-d format. Each line is a separate head impulse and they have been sorted so that the head velocity progressively increases from very small to large.

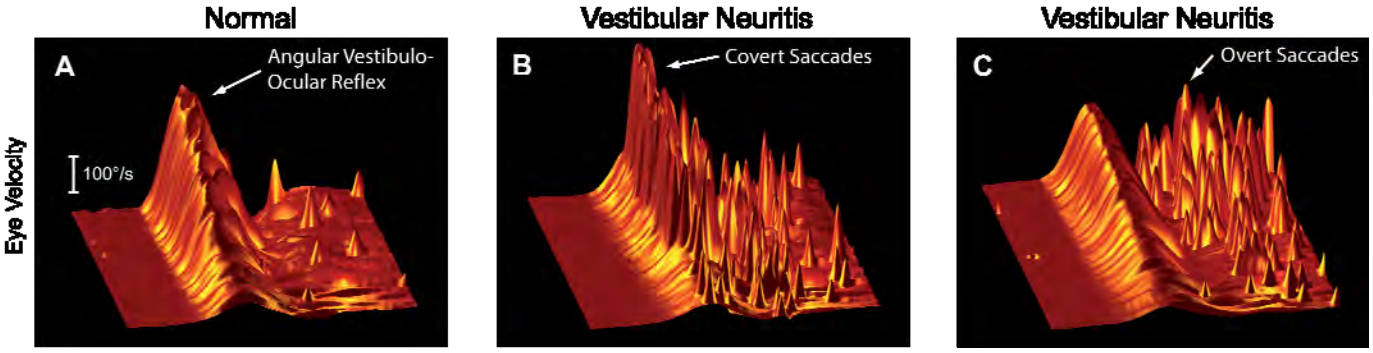




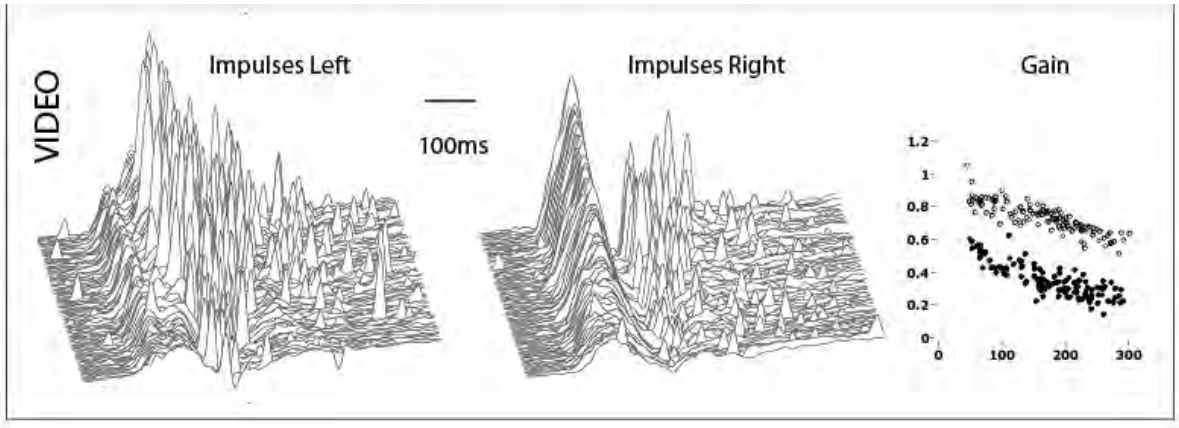
The figure above shows that as these head velocities increase, in a healthy subject there is a corresponding increasing series of matching eye velocities. (The small "stalagmites" are small saccades.) In fact it is usually sufficient just to show the graded series of eye velocity responses:

In the following examples a red surface has been fitted over the eye velocity responses (this example and the other "red surface figures" shown below have been recorded with search coils). Even in healthy subjects the VOR gain is not exactly 1.0, so even some healthy people occasionally make some very small overt or covert saccades. These are usually so small the clinician does not see them but the coils and video methods are so sensitive that they pick these up.

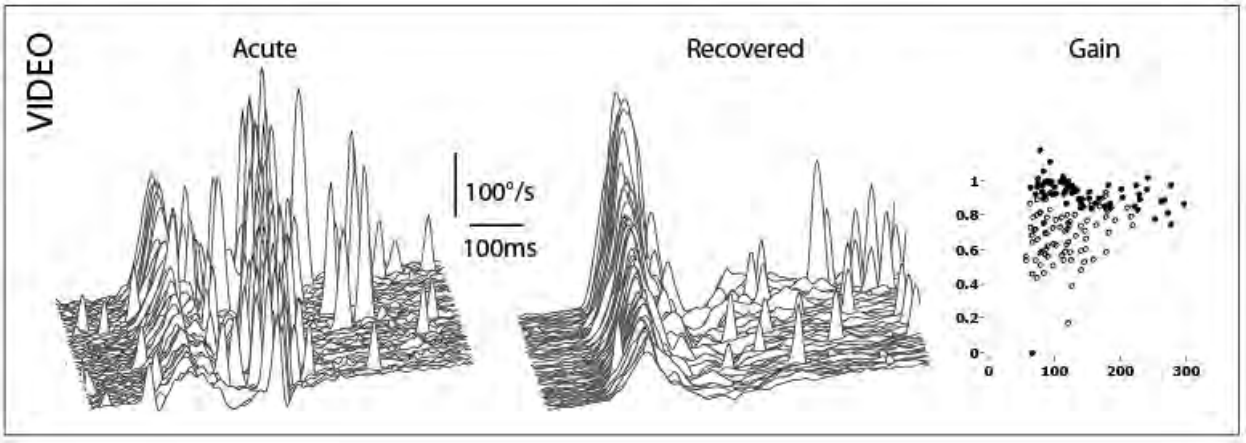
Now the saccades appear as small stalagmites, after the eye velocity response. This graded series method is especially good at showing the difference between overt saccades (panel C) and covert saccades (panel B).



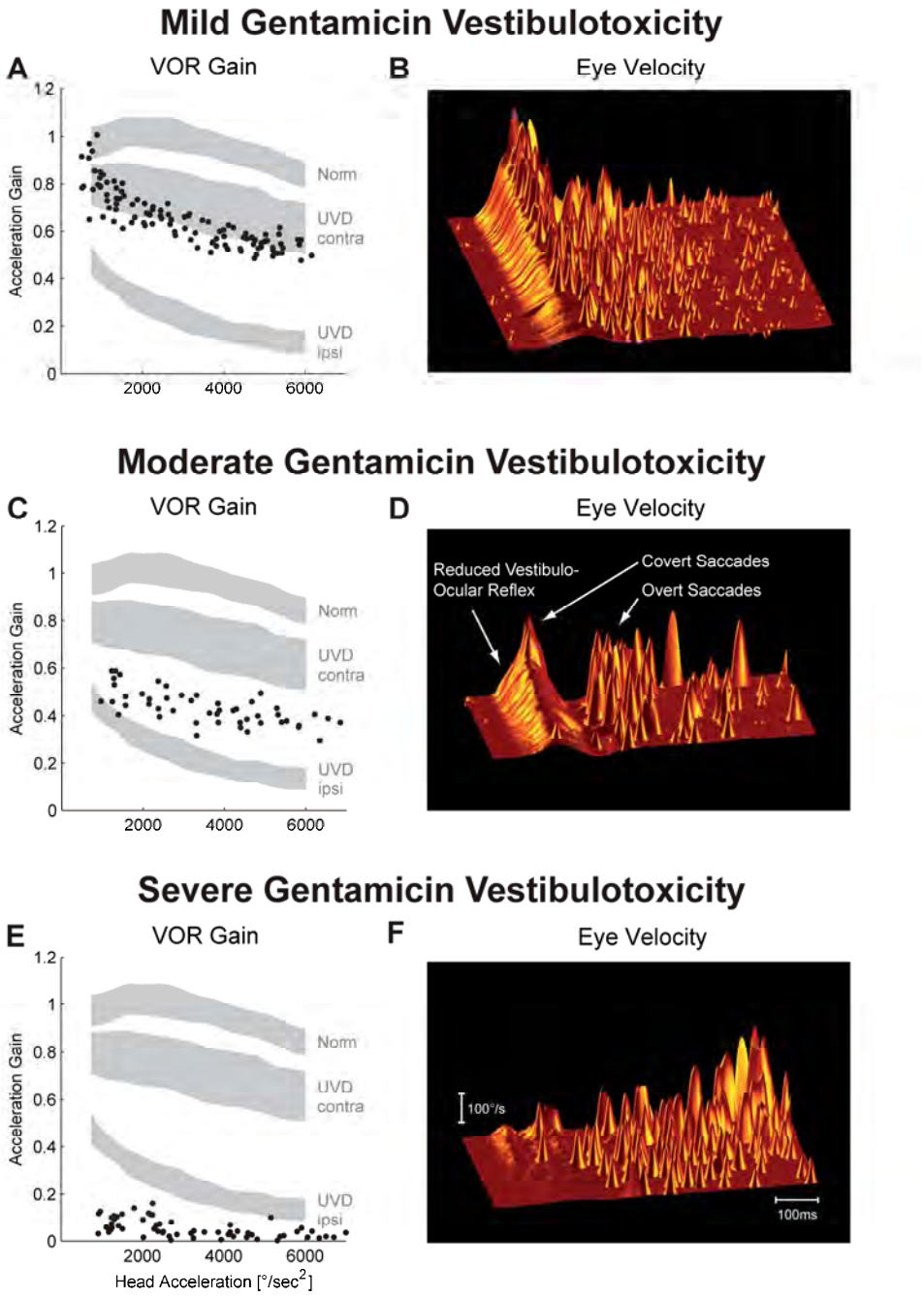
Below are the results of a patient who made many covert saccades during the head impulses to his affected ear, whereas his VOR gain for head rotations to his (right) healthy side are only slightly reduced compared to healthy subjects. The VOR gain shows the very clear, consistent difference in VOR gain for the two sides.



The objective measures of the eye movement response by vHIT allows the objective measurement of whether there is recovery of vestibular function after acute vestibular neuritis. Below is data from a patient who was measured at the acute stage of unilateral vestibular neuritis (with many overt saccades and a low VOR gain (open circles)). However when measured again at testing some weeks later (Recovered) there is clear objective evidence that the VOR gain has increased substantially (closed circles) and there are very few saccades.



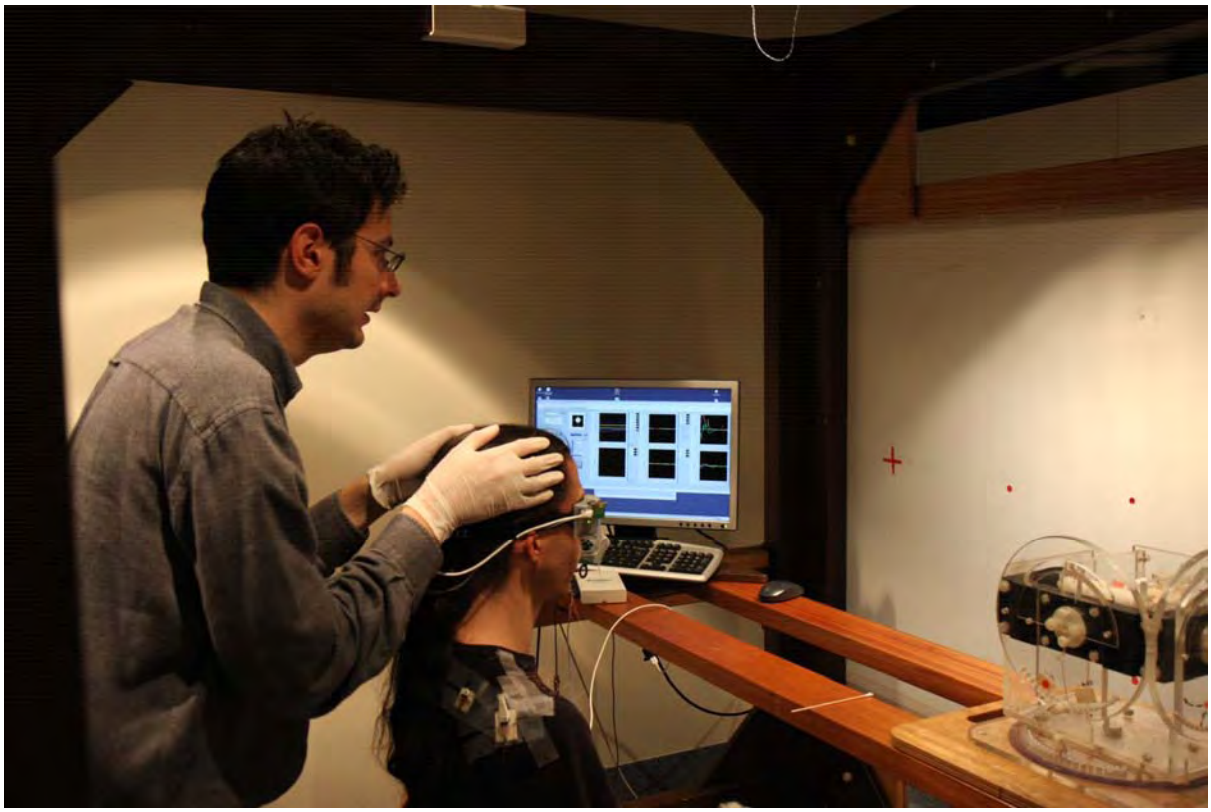
The following examples are search coil recordings of three different patients with varying degrees of progressive loss of vestibular function following systemic gentamicin toxicity, showing the decreased eye velocity response and the overt saccades after the head impulse.



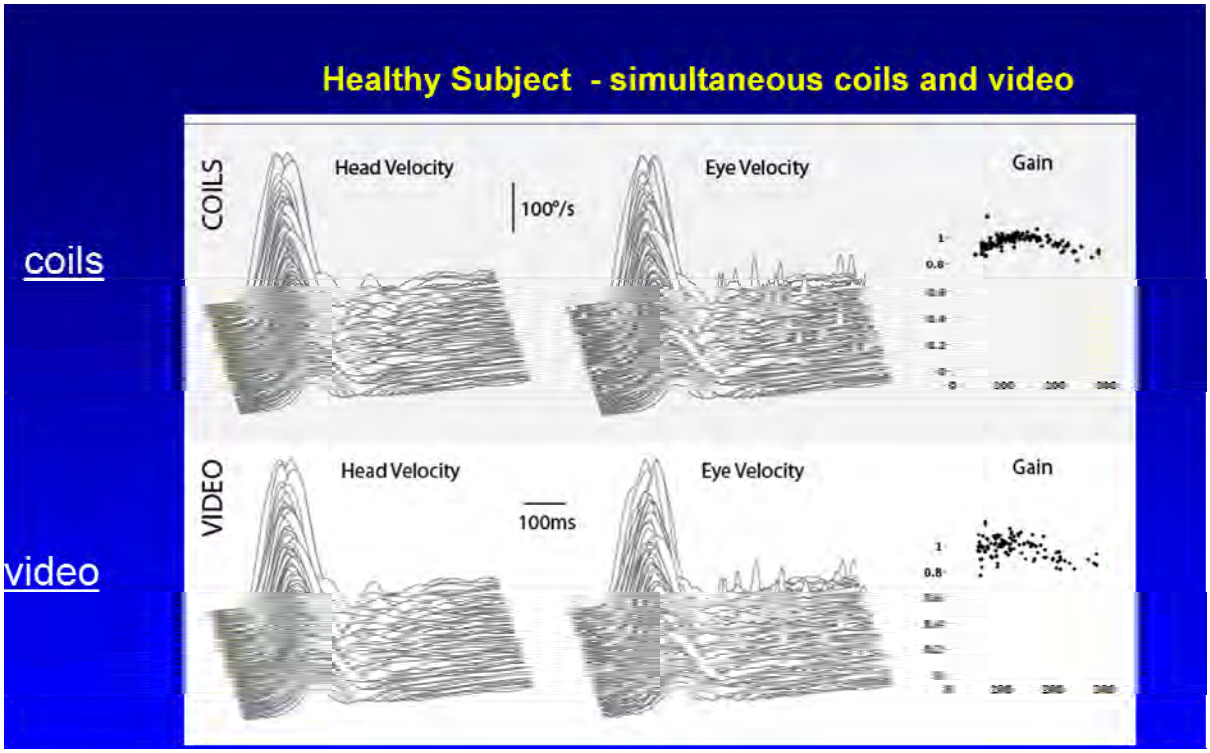
In the final case of severe gentamicin vestibulotoxicity there is almost no corrective eye movement during the head movement, so the VOR gain is close to zero, but there are many overt saccades after the end of the impulse.

Validation of vHIT

It is important to emphasize that the accuracy of the vHIT method has been carefully checked by simultaneous measures of eye movements during head rotations by two entirely independent systems (search coils and vHIT) measuring the stimuli and response of healthy subject and patients. This was done for 8 healthy subjects and 8 patients with various known vestibular losses. This is the way the simultaneous search coil and video measures were done (clinician Konrad Weber; subject Hamish MacDougall).



The evidence of this direct comparison was published in a peer-reviewed very high ranking journal in the field (*Neurology* -- MacDougall et al 2009) and it shows that the VHIT method and search coils gave very closely similar results – there were no significant differences in VOR gain, and the similarity of the eye velocity responses, as measured by the concordance correlation coefficient between the eye-velocity records for vHIT and for search coils was very high – meaning they both gave the same answer. The figure shows just how similar the measurements with search coils and video are.



Contraindications and Challenges vHIT cannot be used on everyone. Some people have very stiff necks or cannot relax their neck muscles sufficiently for the clinician to give them an unpredictable head rotation. In individuals who have previous neck trauma it is not advisable to carry out this rapid head turn. Blinks can be a problem – the patient must be asked to keep their eyes wide open during each head turn, and to try to keep looking at the fixation target. The patient must understand the instructions and attempt to maintain fixation. The goggles must be tightly fixed to the head, and the clinician's hands must be well away from the goggles and the goggle strap.

Why does vHIT detect semicircular canal loss? In a healthy subject a head rotation to the left activates receptors in the left horizontal semicircular canal, so the nerve fibres from the left canal generate nerve impulses which cause both eyes to rotate smoothly so that both eyes exactly compensate, or correct for, the head movement. So both eyes stay looking at the target during the passive unpredictable head movement. But if the person has a loss or deficiency in the ***left*** horizontal canal system, then the neural drive to the eyes will not be enough to drive the eyes to correct perfectly for a ***leftward*** head movement. So the eyes will move with the head and the result will be that at the end of the head movement the eyes will have been dragged off target and the patient will have to make a corrective saccade to get back onto the target. That is the *overt saccade* which the clinician sees. Obviously a right side canal deficit will cause a loss for rightward head movements. If just one side is affected and the other side is healthy then the corrective saccade will only occur for head rotations towards the affected side. That patient has a unilateral vestibular loss. This is the most frequent kind of deficit. When both sides are affected, the patient makes corrective saccades for both directions of head rotation – they have bilateral vestibular loss.

Summary of video HIT Advantages The vHIT method has now been in use for 18months by Dr Leonardo Manzari in the MSA Clinic at Cassino, Italy, as well as at two other locations (Sydney and Zurich), and the results show how extremely useful it is. The vHIT method provides ***objective*** measures of the eye-velocity response to the head-velocity stimulus, and shows the VOR gain for the two directions of rotation. It shows the presence of both overt

and covert saccades and has the very large advantage of being objective – these records of the eye movement responses and the VOR gain provide the hard objective evidence about the adequacy of semicircular canal function which clinicians require.

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Q & A

- ***Why is it necessary for the clinician to move the patients head? Why can't we just get the patient to turn their own head while they are looking at the target spot?***

Firstly that sounds a very easy thing to do but it isn't. Some people, even very intelligent people, just cannot do that task at all, try as they may! But more importantly, if the patient moves their own head they can voluntarily generate a corrective eye movement at the same time as they cause their head to move. Just as they can voluntarily control their own head movement, so they can voluntarily control their own eye movements. Active voluntary control of head movement and eye movement by the patient just does not provide the probe of the inner ear function which we get so well if the patient's head is turned in unpredictable directions by the clinician. We have found that patients doing the head impulse voluntarily (by actively turning their head) quickly learn to make an early saccade during the active head movement which is very very hard to detect – another version of the covert saccade. So the clinician cannot see any saccade even though the vestibular system may be non-functional.

- ***Why measure eye movements to test inner ear balance function? The patient's problem is in their ear so why not measure ear function rather than eye function?***

The answer is because the eye movement response to a head turn is a very sensitive indicator or tool or probe of just how well the balance mechanism of the inner ear is functioning. There are very strong fast neural projections from the inner ear to the eye muscles.

- ***Can we give the stimuli regularly (using a metronome)? and why is it necessary to randomize the direction of head turn?***

If each impulse is given in a very regular fashion (e.g. 3 seconds between each impulse and always alternating directions) the patient can quickly learn to either blink just at the start of the head turn, or generate a covert saccade, so the clinician misses seeing any deficit. The test should not be given at regular intervals – the timing when each head turn is delivered should be random. Unpredictable directions and unpredictable intervals minimize the chance of learning affecting the test results.

- ***How many impulses?***

We normally aim to get about 20 impulses for each direction. Although the very first impulses usually tell the whole story and the rest just confirm that story and give the clinician greater confidence in the results. In some patients it is difficult to deliver 20 impulses in each direction – the patient may have a stiff neck or not be able to totally relax their neck muscles. There is no absolute minimum, it really depends on the quality of the responses on each trial. If there is any doubt the stimulus and measurement is very easy and takes only an extra few minutes to give more. With a small number of impulses the calculation of VOR gain becomes less reliable because there are so few values.

- ***How big should the head movement be? (insert video into this section)***

Even very small head turns can be very valuable in showing a loss of function. The important thing is not how large the head turn is but ***how abruptly it starts.*** It should be an abrupt start, not through a big angle, but it should start abruptly.
If the head is turned through a small angle, slowly, then there is no need for the patient to make any corrective eye movement at all and so they don't and the clinician does not detect the deficit which may be there.

- ***Why do we have this vestibulo-ocular response?***

The corrective eye movement response is used to provide stable vision during the head movements of walking, running, driving and all the normal activities we get up to. This very simple eye movement response is an indicator of the function of one part of the balance system of the inner ear.

- ***Can we suppress this response?***

Healthy people can almost totally override the vestibulo-ocular response. For example if you are reading a book on a bus going around a corner - you want to keep your eyes on the text rather than having them being driven off the page by the vestibular input automatically correcting for the angular turn. It is abilities like that overriding (also called VOR suppression) which are an unnoticed but essential part of everyday living which make clinical testing of vestibular function more complex than testing hearing. In hearing there is such a simple output – do you hear that sound? and sounds cannot be suppressed or "shut out" in any way analogous to the way the vestibular information can be. But by restricting our measurements to just the start of these brief unpredictable head turns we can selectively probe the function of the semicircular canals since it takes a little time for that VOR suppression to work

- ***Is the patient's understanding of the instructions important?***

It is *VITAL* that the patient understand the instructions – that the patient has to keep looking at the fixed target – to try to keep their gaze stable and not to blink during this unpredictable movement. They ***must not*** try to "help" the clinician by looking ahead, or looking where their head is going - they must not turn their eyes with their head.

- ***Why has it taken so long to develop this system?***

Because we need very fast cameras which were very small and lightweight which can be comfortably be fitted to the head in goggles which have minimal slip.

- ***What is the worst kind of error with this system?***

Slip of the goggles. If the camera slips on the head then it appears as if the eye has moved relative to the camera A real eye movement and the movement of the camera relative to a fixed eye both generate the same effect at the camera - the image of the pupil of the eye moves across the camera sensor plane. This is the worst artifact. We want to measure real eye movements relative to a fixed camera, not artifactual eye movements. To avoid camera slippage, the camera should be tightly fitted to the head (the test only takes about 5 minutes so it will not cause discomfort for too long). But if it is not tight enough you might as well not do the test. The operator's hands must be well away from the goggles and the goggle strap.

- ***How do we maintain control over the stimulus?***

In clinical testing of hearing, precisely controlled stimuli are presented through calibrated headphones and the patient's responses are measured. In clinical testing of the vestibular system, this kind of presentation of controlled stimuli is just not feasible. Instead we present vestibular stimuli which are not well controlled – a head turn by a clinician can vary enormously from one trial to the next – but we rely on the fact that ***we measure the stimulus exactly each time and relate each response to that stimulus.*** In some respects this is an improvement over testing of hearing since we actually **measure** what the stimulus is on each and every trial, rather than assuming that a calibration check taken a few weeks before guarantees the stimulus value.

Conclusion

This has been a simple introduction. I have focused on the behaviour – the head turn stimulus and the eye movement response. But of course we would like to know about just how the head turn is detected by the receptors in the semicircular canals and how it is transformed into neural signals and how those neural signals result in the eye movement response, and how something like VOR suppression can occur. Those matters are taken up in the companion chapters.

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B-ENT, 2008, **4**, Suppl. 8, 23-25

Principle of the head impulse (thrust) test or Halmagyi head thrust test (HHTT)

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Key-words. Vestibular test; semicircular canal; head impulse test; head thrust test

The head impulse or head thrust test was first described by Halmagyi and Curthoys in 1988.[1] It has acquired an increasingly important place in the clinical examination of the vertigo patient. It detects severe unilateral loss of semicircular canal (SCC) function clinically; it is more sensitive and specific than the traditional Romberg and similar tests; and it is particularly important in the emergency unit, where it can distinguish between vestibular neuritis and cerebellar infarction, which can both generate similar symptoms suggesting an initial attack of severe acute vertigo. The result of the head thrust test is definitely normal in a patient with a cerebellar infarction but abnormal in a patient with vestibular neuritis.

General physiological background: the push-pull principle of the vestibulo-ocular reflex

The peripheral vestibular sensors transmit motion to the brain through frequency encoding. Like FM radios, our brains continuously receive 'frequency modulated' signals. A normal resting discharge rate of approximately 90 spikes per second is modulated such that any increase in this rate corresponds to excitation and a decrease to inhibition. The polarisation of the hair cells in the horizontal semi-circular canal is such that deflection of the stereocilia in the cupula towards the kinocilium (ampullo- or utriculopetal) results in hair cell depolarisation and the activity of the primary afferent neurons therefore increases. Deflection of the stereocilia away from the kinocilium (ampullo- or utriculofugal) results in hair cell hyperpolarisation and decreased primary afferent neuron activity.

The orientation of the left and right semi-circular canals in the head is such that any movement always induces an antagonistic response in both canals. Horizontal head movements in the yaw plane are an example. During rightward head rotation, the endolymph in the lateral semi-circular canals on both sides lags behind, bending the cupula of the right SCC towards the vestibulum (ampullo- or utriculopetal) and simultaneously deflecting the cupula of the left SCC away from the vestibulum (ampullo- or utriculofugal). A key difference is the polarisation of the hair cells. Indeed, since the the hair cells in the right and left canals are implanted in opposing directions (in a mirror image fashion), the deflection on the "leading" right side induces the movement of the stereocilia towards the kinocilium, whereas the movement of the stereocilia is away from the kinocilium in the opposing, "following" ear. As a result of this "push-pull principle", the activity of right lateral SCC primary afferent neurons increases, and, at the same time, the activity of left lateral SCC primary neurons decreases with respect to the normal resting discharge rate.

The activity of the lateral SCC primary afferent neurons is modulated by horizontal head rotation. The firing rate increases in the leading ear (the ear towards the movement is directed) and decreases in the following ear. This is the push-pull principle of the VOR.

The right medial vestibular nucleus in the brainstem receives an increased input from the right lateral SCC primary neurons (no crossing). This excites the activity of type I secondary vestibular neurons. These excitatory neurons drive the leftward compensatory eye movements of the VOR, to ensure gaze stabilisation. However, commissural disinhibition from the left lateral SCC primary neurons also contributes to the excitation of the type 1 neurons. Both excitation of the right SCC and disinhibition of the left SCC are therefore needed for an optimal VOR.

Figure 1
Left: the clinician holds the head of the subject firmly and turns it briskly to the left. Centre: After the rotation to the left, the subject maintains the gaze on the distant fixation point, *i.e.*, the eyes stay stable in space.
Right: After abrupt rotation to her right, the subject moves her eyes with her head and loses the target. A refixation is necessary to fixate the point again (not shown). The side towards the gaze fixation is lost is the deficit side, *i.e.*, the patient's right.

Head thrust test

The head thrust test is primarily based on the fact that inhibition of primary and secondary vestibular neurons cannot produce fewer than 0 spikes per second. Excitation can drive the discharge rate from 90 to 300 or more spikes per second. So when the healthy side is excited for a high acceleration head movement, the healthy side will generate the larger part of the VOR, since the disinhibition of the ipsilateral type-1 neurons by the contralateral SCC contributes relatively little to the VOR. Passive head impulses or thrusts should be typically rapid but with a small amplitude (± 20 degrees). Their velocity ranges up to 180 deg/s but high acceleration is particularly important (3000-4000 deg/s2). They have to be unpredictable since the patient very quickly learns to anticipate and this reduces the sensitivity of the test to a considerable extent. The examiner should therefore thrust the head of the patient firmly from left to right at random and from right to left a little later, *i.e.*, not immediately. The starting position should be such that the patient's head is turned slightly past the midline, and it should then be thrust just past the midline to the opposite side. Here, amplitude is low but acceleration can be considerable. This test demands some training, particularly with respect to the positioning of the hands on the side of the head and holding the head firmly. The instruction to the patient is to fix on a point in the distance behind the examiner.

When the subject's head is turned to the side of the lesion, the VOR is deficient and the eyes will move with the head so that they no longer fix on the point in the distance. The patient therefore needs a refixation saccade just after the thrust. When the head impulse is in the direction of the healthy side, the VOR will maintain the target on the fovea and no refixation saccade will be needed.

The head-thrust test is positive for the side that causes the refixation saccade upon thrust (Figure 1)

It is not only the lateral SCC that can be examined – this is, in a sense, a clinical approximation of the caloric test – but also the other SCC. Here, the patient's head must be thrust in the RALP or LARP planes (Right Anterior – Left Posterior or Left Anterior – Right Posterior SCC).[2]

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ORIGINAL ARTICLE

Comparison of Head Thrust Test With Head Autorotation Test Reveals That the Vestibulo-ocular Reflex Is Enhanced During Voluntary Head Movements

Charles C. Della Santina, PhD, MD; Phillip D. Cremer, MBBS, PhD; John P. Carey, MD; Lloyd B. Minor, MD

Objectives: To compare 2 clinical tests of vestibular function, the head autorotation test (HART) and the head thrust test (HTT), and to determine why they give disparate results in patients with known unilateral vestibular deficiency (UVD) due to labyrinthectomy.

Methods: We used scleral coils to measure the horizontal (yaw) vestibulo-ocular reflex (VOR) in 5 healthy human subjects and in 11 patients who underwent labyrinthectomy. We used 2 paradigms. Using HART, subjects visually fixated a target during self-generated, swept-frequency, sinusoidal, horizontal head rotations. Using HTT, patients fixated the target during horizontal head thrusts delivered randomly in direction and time.

Results: In subjects without UVD, eye movements were almost perfectly compensatory for both paradigms. In subjects with UVD, VOR gain for ipsilesional head thrusts was low for both paradigms, but significantly ($P<.001$) higher (less abnormal) for HART (0.60±0.13) than for HTT (0.14±0.13). Contralesional gain was reduced for both, to 0.64±0.20 for HART and to 0.57±0.17 for HTT. Because ipsilesional and contralesional gains were not statistically different for HART ($P=.69$), comparison of VOR gains for half-cycle responses to the HART stimulus could not reliably identify the side of the known lesion. In contrast, HTT consistently identified the side of the lesion for all subjects with UVD. To investigate whether preprogramming contributes to the boost in VOR as measured by HART, we compared the gain and response delay of eye movements during actively self-generated and passively received head thrusts. For subjects without UVD, response delays were shorter for active (6±1 milliseconds) than for passive (12±1 milliseconds) HTT. For ipsilesional rotations of subjects with UVD, active HTT yielded a significantly higher gain (0.44±0.20) ($P<.001$) and a shorter delay (15±6 milliseconds) ($P<.001$) than did passive HTT (0.14±0.13 and 37±15 milliseconds, respectively). Contralesional test results revealed a similar performance boost for active head movements. Data are given as mean±SD.

Conclusion: When comparison of half-cycle gains is used to identify the lesion side, self-generated predictable head movement paradigms, such as HART and active HTT, are less accurate than passive HTT in the characterization of UVD, in part because preprogramming can augment the VOR during voluntary head movements.

Arch Otolaryngol Head Neck Surg. 2002;128:1044-1054

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DIZZINESS IS the ninth most common reason adults visit a primary care physician, and it affects approximately 90 million Americans.[1-4] Identification of the side, affected semicircular canals, and degree of unilateral vestibular deficiency (UVD) is an important goal in the examination of patients experiencing dizziness.

Two high-frequency rotational stimulus paradigms—the head thrust test (HTT) and the head autorotation test (HART)—are in wide use for assessing angular vestibulo-ocular reflex (VOR) function, yet have not been directly compared.

Researchers[3-8] have advocated HTT, in which high-acceleration impulsive head rotations are delivered in the plane of one pair of semicircular canals while a subject attempts to maintain visual fixation on a distant target. Implied by the second law of Ewald,[9] such head rotations elicit an asymmetric response in patients with UVD by maximally stimulating the neural pathway arising from one semicircular canal crista while maximally inhibiting or silencing the pathway from the coplanar canal. The approach has proved sensitive for the detection of vestibular dysfunction in subjects with known UVD.[6,10]

A second paradigm, HART, uses self-generated, swept-frequency, sinusoidal head rotations performed while the subject visually fixates a distant earth-fixed target. Fourier analysis of system gain and phase, along with formulaic measures of response asymmetry, are applied to the measured head and eye movements. Researchers[11-16] have reported extensively on this approach, and several commercially available vestibular testing systems are based on this paradigm.

We sought to compare the ability of HTT with that of HART to identify dysfunction in subjects with known UVD after surgical labyrinthectomy. There are 3 differences between these 2 stimulus paradigms that could contribute to a difference in measured VOR for a given patient. First, HTT stimuli are passive (ie, delivered by the examiner), while during HART, the head movement is active (ie, self-generated). Second, HTT stimuli are presented unpredictably in time and direction, while the sinusoidal frequency–sweep head movement during HART is predictable cycle by cycle. Third, HTT stimuli are often of higher acceleration and wider spectral bandwidth than are HART stimuli, because of some subjects' inability to make rapid, self-generated, sinusoidal head movements.

We hypothesized that during active predictable head movements, subjects with UVD can augment apparent VOR performance by using information about the intended/expected head movement to complement deficient vestibular function and guide compensatory eye movements. Such an effect would be expected to reduce the sensitivity of tests like HART that rely on self-generated head movement stimuli for detecting vestibular hypofunction. We tested these predictions by comparing VOR gains measured for subjects with and without UVD during HART and HTT, and by comparing subjects' responses to active and passive impulsive head rotations.

METHODS

SUBJECTS

We studied 5 human subjects without UVD and 11 with UVD due to labyrinthectomy. Subjects without UVD ranged in age from 32 to 55 years and were free of vestibular and ocular disease, except for wearing corrective lenses. Subjects with UVD ranged in age from 48 to 72 years, and the time from labyrinthectomy ranged from 3 to 120 months. All had undergone vestibular rehabilitation therapy postoperatively. The indication for labyrinthectomy was vestibular schwannoma or meningioma in 6 subjects and unilateral Meniere disease in 5. The side of labyrinthectomy was the left for 6 subjects with UVD and the right for 5. All subjects gave written informed consent, and the experimental procedures were approved by the Joint Committee on Clinical Investigation, The Johns Hopkins University School of Medicine, Baltimore, Md.

EXPERIMENTAL TECHNIQUE

Head and eye movements were recorded in 3 dimensions using magnetic search coils embedded in contact lenses and in a bite block. The instrumentation and technique have been described in detail elsewhere.[17] Eye and bite block angular positions were sampled at 500 Hz. The resulting signals were low-pass filtered with a single-pole analog filter with a 3-dB bandwidth of 100 Hz. Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line parallel to the horizon and the Frankfort line (from the top of the tragus to the infraorbital foramen) in the plane of head rotation. All rotational stimuli were in the horizontal (yaw) plane. During each trial, the room was completely dark except for a target light-emitting diode positioned 1.24 m anterior to the center of the head, at the same elevation as the pupils. All subjects were tested more than 20 minutes after removal of eyeglasses.

Each experiment began with the subject's head held rigidly at the starting position via connection of the bite block to a bar, while the subject performed calibration tasks. The bar was then removed, and all subsequent trials were performed with the head free to move during a stimulus, then returning to the starting position.

For HART, subjects attempted to visually fixate the target while sinusoidally shaking their heads horizontally (ie, rotating about an earth-vertical axis through the center of the cervical spine) at maximum tolerated velocity in time with a metronome swept logarithmically in frequency from 1 to 6 Hz for 20 seconds. Results from 3 trials were averaged.

For HTT, subjects fixated the same target during brief (100-200–millisecond) rotations in the earth-horizontal plane, at high acceleration (3000°-5000°/s^2) starting from a complete stop. Head velocity reached 25° to 75°/s at 40 milliseconds into the movement and 150° to 300°/s peak velocity by 100 to 120 milliseconds. The final position was 10° to 15° from center. For passive HTT (pHTT), stimuli were delivered manually at an unpredictable onset time and in a randomly varied direction, starting from center. The subject received no cues about the direction or time of an impending passive head thrust. For active HTT (aHTT), the subject generated the head thrust voluntarily. Only head movements reaching greater than 100°/s within the first 60 milliseconds were included in the analysis. A typical trial lasted about 3 minutes and included about 20 to 40 head thrusts to each side.

ANALYSIS

To facilitate comparison between eye and head angular velocities, both were analyzed in 3 dimensions in the head frame of reference. Horizontal rotational components were extracted and compared.[18] All results reported are for the horizontal components of rotation only. Angular velocity and acceleration were computed from angular position signals using a 30-Hz, band-limited, 10-element, linear phase differentiating digital filter designed using computer software (MATLAB; The Mathworks, Inc, Cambridge, Mass) and applied in zero-delay fashion.

We quantified the eye movement responses in terms of velocity gain and response delay. Gain was defined as the angular velocity of the eye divided by the angular velocity of the head at the instant head velocity crossed to above 120°/s, with the eye velocity inverted so that a gain of +1.0 denotes an ideal compensatory response. Using the method of Tabak et al,[7] we defined response delay as the time between the onsets of head and eye movements, where onset was defined as the time at which velocity crossed a threshold set at 2°/s plus 8 times the root-mean-square velocity in the 100 milliseconds preceding the stimulus.[8] We used 1- or 2-tailed *t* tests to compare gain and response delay distributions between experimental groups, with $P<.05$ considered significant. Data are given as mean±SD unless otherwise indicated.

RESULTS

HEAD AUTOROTATION TESTING

Figure 1 shows HART results for a healthy 33-year-old man without

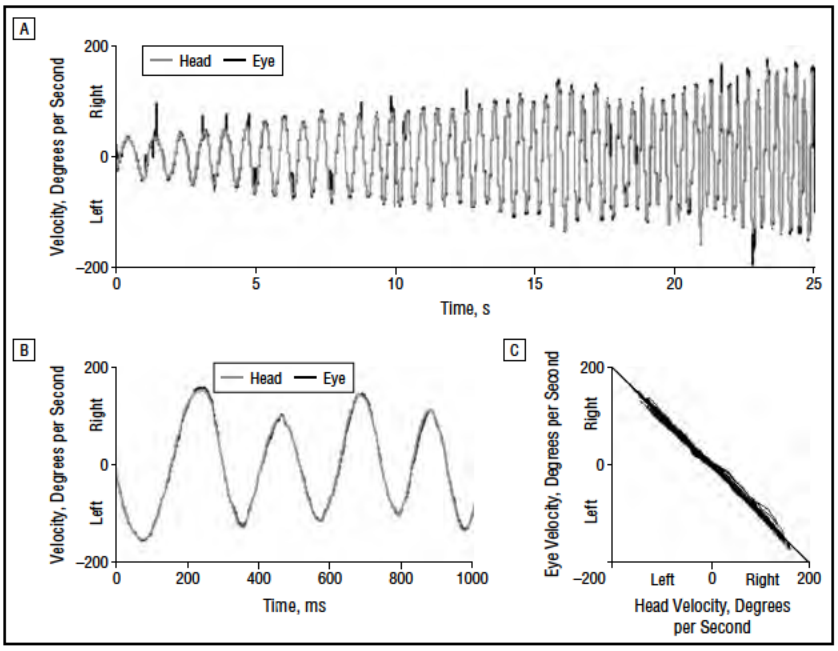

Figure 1. Horizontal head autorotation test results of a 33-year-old man without a unilateral vestibular deficiency. A, Head and eye velocity vs time, with the eye trace inverted for comparison with the head trace. (All similar figures use the same convention.) The eye velocity trace overlies the head trace almost exactly. In the trial shown, head velocity ranged from 40°/s at 1 Hz to 180°/s at 6 to 8 Hz. B, Expanded view of a portion of the trial. Eye velocity traces are nearly identical to head velocity traces. C, Eye velocity vs head velocity after removal of saccades. Data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect vestibulo-ocular reflex.

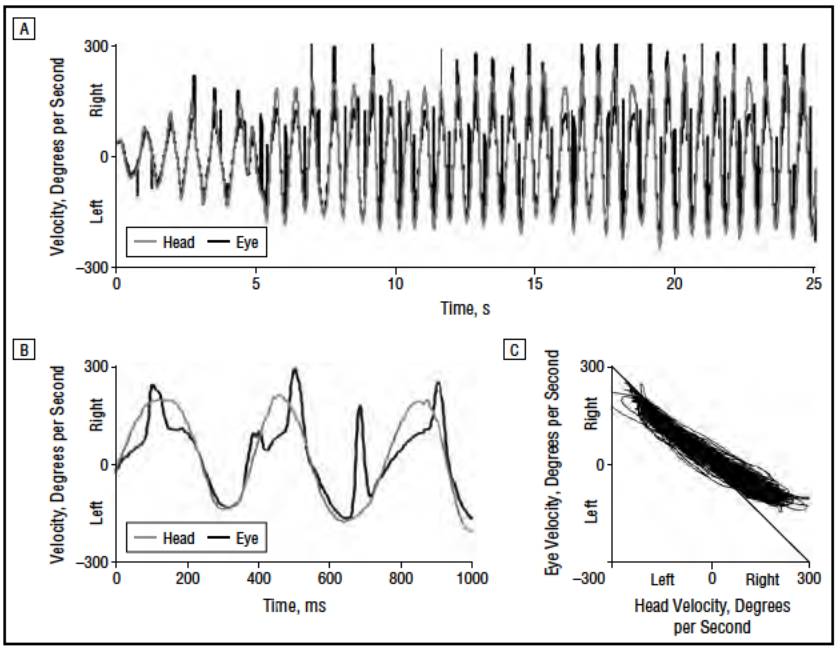

Figure 2. Head autorotation test results of a 63-year-old man 5 months after right translabyrinthine vestibular schwannoma resection. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity. Contralesional (leftward) head turns elicit a moderately degraded response, with a gain of 0.87±0.13 (mean±SD). Ipsilesional responses are more abnormal, with decreased velocity gain, increased retinal slip errors of up to 100°/s, and large-amplitude saccadelike catch-up eye movements.

UVD. In the trial shown, head velocity ranged from 40°/s at 1 Hz to approximately 180°/s at 6 to 8 Hz (Figure 1A). Eye velocity traces (inverted for comparison with head velocity) are nearly identical to head velocity traces, to within less than the 4°/s of retinal image slip for which visual acuity begins to degrade (Figure 1B).[19] When plotted as eye velocity vs head velocity, these data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect VOR (Figure 1C). This subject without UVD has rightward and leftward VOR velocity gains of 1.0±0.05 and 1.0±0.02, respectively.

Figure 2 shows HART results for a symptomatically well-compensated 63-year-old man who had undergone right translabyrinthine vestibular schwannoma resection and postoperative vestibular rehabilitation 5 months before testing. For this subject, the response during head turns toward the contralesional left side (Figure 2A and B) is somewhat degraded, but not significantly ($P=.07$) different from normal (gain, 0.87±0.13). During rightward head turns, there is a significantly ($P<.001$) abnormal response, with a velocity gain of 0.43±0.19 and retinal slip errors of up to 100°/s. Retinal slip not only degrades foveal visual acuity but also accrues to cause the subject to lose target fixation, eliciting large saccadelike corrective eye movements to reacquire the visual target. For this subject, HART results clearly reveal an asymmetry.

Such asymmetries between responses to ipsilesional and contralesional head rotations were not always apparent. **Figure 3** presents the HART responses from a 68-year-old man tested 38 months after undergoing surgery resulting in left UVD. At maximum effort, this patient achieved slower peak head velocity than the subject shown in Figure 2. The slow-phase components of the response to ipsilesional rotations were not markedly different from those observed in the contralesional half cycles. However, a gaze-correcting saccadic eye movement was often observed in the ipsilesional responses.

Figure 4A and B shows a summary of VOR gains measured using HART in 5 healthy subjects and in 11 subjects with UVD, respectively. The VOR gains for subjects without UVD are segregated into rightward and leftward head movements and illustrated separately as a check of test reliability. The VOR gains for subjects without UVD are 0.95±0.13 and 1.03±0.11 for rightward and leftward head rotations, respectively. There was no significant difference between right and left VOR gains

(P=.13), and the combined data set was insignificantly different from 1.0 (P=.64) (95% confidence interval, 0.93-1.05). The HART gains for subjects with UVD were segregated into ipsilesional and contralesional directions. Four findings were notable. First, the ipsilesional gain of 0.60±0.13 was significantly lower than normal (P<.001). Second, there was a wide distribution of ipsilesional gains for this sample population, ranging from 0.2 to 1.0. Third, the contralesional gain of 0.64±0.20 was also significantly decreased from normal (P<.001). Finally, HART gains were not significantly different for ipsilesional and contralesional rotations in these subjects with UVD and, therefore, could not reliably indicate the side of the known lesion (P=.69).

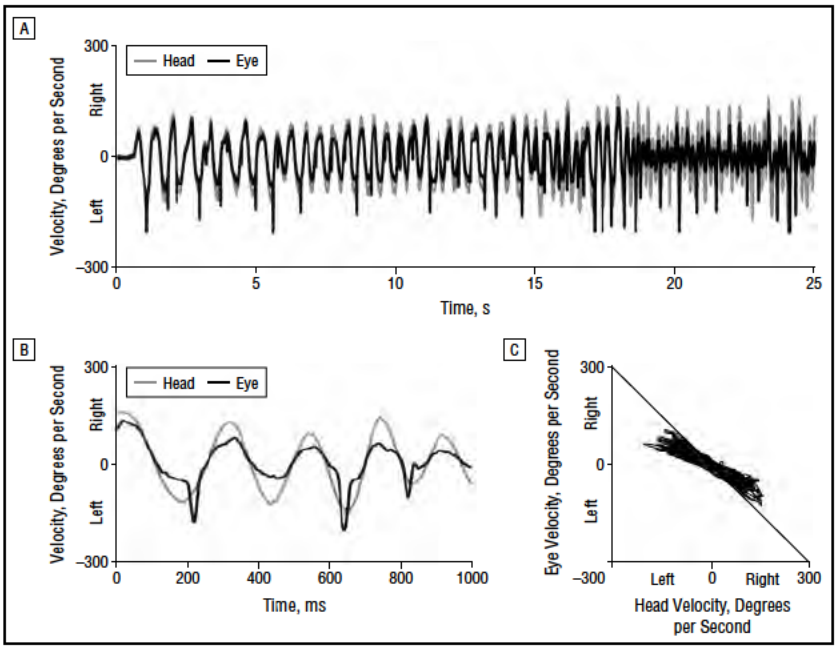

Figure 3. Head autorotation test results of a 68-year-old man 38 months after surgery for a left unilateral vestibular deficiency. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity after saccade removal. Saccadelike catch-up eye movements are directed toward the lesion side, but slow-phase eye movements show little asymmetry.

PASSIVE HTT

Passive HTT was performed on 4 of the subjects without UVD and on all 11 subjects with UVD who had been studied using HART. **Figure 5**A shows head velocity and concurrent eye velocity traces for 2 representative head thrusts from the same subject without UVD who was described in Figure 1. The high-acceleration head rotation transients reach a peak velocity of greater than 200°/s within 80 to

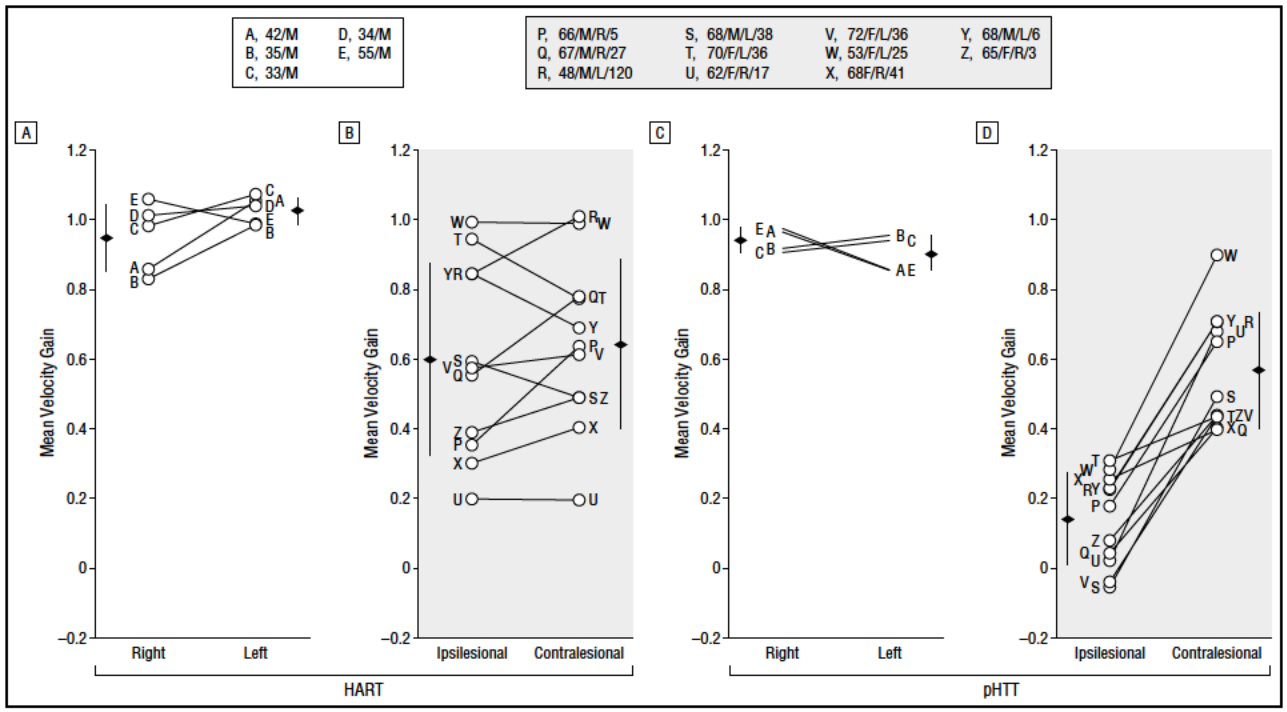

Figure 4. The head autorotation test (HART) gains measured in 5 healthy subjects (A) and in 11 subjects with unilateral vestibular deficiency (UVD) (B) are shown. A, The mean vestibulo-ocular reflex (VOR) gain for subjects without UVD is not significantly (P=.64) different from 1.0 for rightward and leftward head rotations. B, For this population of subjects with UVD, ipsilesional and contralesional HART gain distributions were not significantly different from each other (P=.69). The VOR gains for passive head thrust test (pHTT) in 4 subjects without UVD (C) and in 11 subjects with UVD (D) are also shown. C, Right and left pHTT gains of subjects without UVD are close to 1.0 and not significantly different from each other (P=.27). D, The gains of subjects with UVD were decreased for ipsilesional and contralesional head rotations, with a marked asymmetry between ipsilesional and contralesional gains for every patient tested. For A and C, the key provides subject age (in years)/sex (M indicates male; F, female); for B and D, subject age (in years)/sex/side (R indicates right; L, left)/time since labyrinthectomy (in months). Bars represent mean±SD.

100 milliseconds and are tracked accurately by compensatory eye movements, with a slight overshoot following the peak of head velocity. Figure 5B shows multiple head thrust stimuli and the corresponding eye movement responses for the first 80 milliseconds of each. Peak head accelerations ranged from approximately 3000° to 5000°/s^2. Although there is a slight early mismatch of eye and head traces because of VOR latency, retinal slip is kept below 5°/s throughout most of each trace. In Figure 5C, a plot of eye velocity vs head velocity reveals that all traces lie close to a $y=-x$ line, corresponding to nearly perfect performance. Rightward and leftward gains for this subject were 1.00±0.04 and 0.99±0.06, respectively. Subjects without UVD never complained of losing sight of the target during testing.

In contrast, **Figure 6** shows pHTT responses for the 68-year-old man with left UVD for whom HART data were described in Figure 3. In Figure 6A, a rightward head thrust is tracked fairly well by the eye, although the VOR gain is less than normal and a small catch-up saccade-like movement is required to correct the final eye position after the head movement ends. For a leftward head thrust, the VOR response decrease is more pronounced, as are the prominent catch-up eye movements. The second and third catch-up movements occur after the head stops moving; these are the corrective eye movements an observer can detect when using HTT as part of the physical examination. Figure 6B shows the first 80 milliseconds of multiple stimuli for this subject. For rightward head rotations, the eye tracks the head fairly well; however, the initial VOR-mediated eye movement response for leftward head thrusts is of low amplitude. Figure 6C reveals an asymmetry in the eye movement responses to contralesional and ipsilesional head thrusts, corresponding to the measured VOR gains of 0.50±0.15 and 0.06±0.09, respectively.

Figure 4C and D shows a summary of pHTT VOR gains for 4 subjects without UVD and 11 subjects with UVD, respectively. (The same subjects were used for pHTT and HART, except for 1 subject without UVD who was tested only with HART.) Right and left pHTT responses of subjects without UVD are shown separately in Figure 4C. Rightward (0.94±0.04) and leftward (0.90±0.05) pHTT gains were not significantly different ($P=.27$) for subjects without UVD. For subjects with UVD (Figure 4D), gains were decreased for ipsilesional and contralesional head rotations. In contrast to the HART VOR gains for the same population of subjects, there was a marked asymmetry between ipsilesional (0.14±0.13) and contralesional (0.58±0.17) gains. For every patient with UVD tested, ipsilateral gains were significantly lower than contralesional gains ($P<.001$). Ipsilesional and contralesional gains were each significantly different from normal ($P<.001$).

Comparison of HART and pHTT results reveals that although the ipsilateral VOR gains are reduced for the HART paradigm applied to subjects with UVD, a similar reduction in contralesional gains was such that HART could not reliably distinguish the side of the known lesion. In contrast, there was a marked and consistent asymmetry in the VOR gains of subjects with UVD measured using the pHTT paradigm, with the ipsilateral gains close to 0 and the contralesional gains close to gains measured using HART.

aHTT VS pHTT

Gain

To better identify why the VOR is apparently enhanced when measured by the HART paradigm, we compared VOR gains measured in subjects with and without UVD using head thrusts that were either passively and unpredictably received by the subjects as in the usual application of the test (pHTT) or actively generated by the subjects (aHTT). Analysis was limited to active and passive head movements that were similar in mean peak velocity, peak acceleration, and spectral content.

Figure 7 shows the first 80 milliseconds of head movements and corresponding eye movement responses to aHTT for the subject with left UVD shown in Figures 3 and 6. Whereas the ipsilesional pHTT response was essentially nonexistent for the first 80 milliseconds after stimulus onset (Figure 6B), the aHTT response VOR gain is enhanced, to 0.34±0.06, from 0.06±0.09 (Figure 7A). The contralesional VOR gain is also increased, to 0.74±0.05, from 0.50±0.15. Similarly, the time from stimulus onset to threshold response is shorter for aHTT than for pHTT.

Figure 8 shows pHTT and aHTT VOR gains for 3 subjects without UVD and 10 subjects with UVD (the same subjects used for HART except for those who did not generate aHTT head movements similar to those of pHTT). For subjects without UVD (Figure 8A), responses to aHTT and pHTT were not significantly different from each other or from 1.00 ($P>.05$ for all). For subjects with UVD, there was a significant ($P<.001$) increase in apparent ipsilesional VOR gain when measured using the aHTT paradigm, to 0.44±0.20 (from 0.14±0.13 for pHTT) (Figure 8B). The contralesional VOR gain also increased significantly ($P=.004$) under aHTT conditions, to 0.81±0.14 (from 0.58±0.17 for pHTT) (Figure 8C). This level was not significantly different from the VOR gains measured in subjects without UVD using pHTT ($P=.08$).

Response Delay

To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset and to compute a response delay. **Figure 9** shows pHTT and aHTT VOR response delays for 4 subjects without UVD and 11 subjects with UVD. For subjects without UVD (Figure 9A), the response delay was 11.0±3.3 milliseconds for pHTT and slightly, but significantly, lower at 5.0±1.8 milliseconds for aHTT ($P=.006$). For subjects with UVD, the response delay for ipsilesional head thrusts was significantly shorter for aHTT (19±16 milliseconds) than for pHTT (42±17 milliseconds) ($P=.006$) (Figure 9B). The response delay in subjects with UVD for contralesional head thrusts was also shorter for aHTT (11.0±5.1 milliseconds) than for pHTT

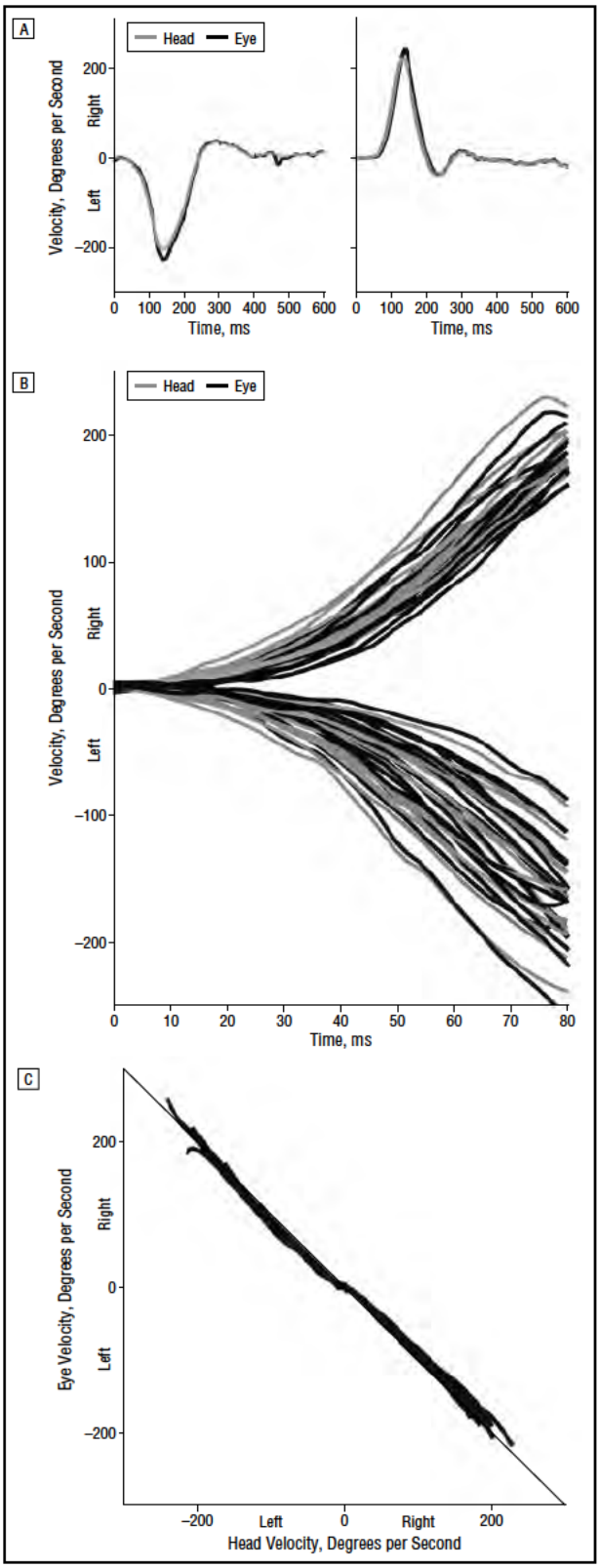

Figure 5. Passive head thrust test results for a healthy subject without a unilateral vestibular deficiency (UVD) (same subject as in Figure 1). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject has nearly perfect overlap of head and eye traces. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Traces nearly overlie each other for this subject without UVD. C, Eye velocity vs head velocity. Data lie closely along a $y=-x$ line, consistent with a near-perfect vestibulo-ocular reflex.

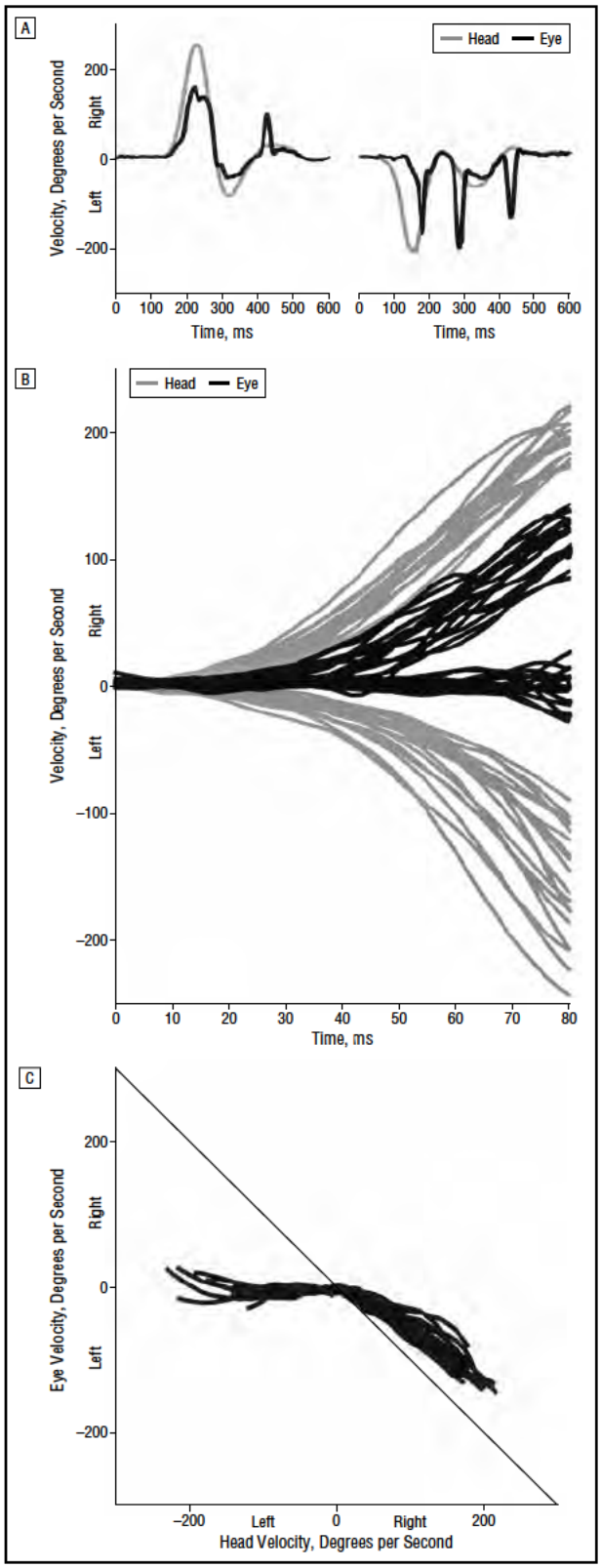

Figure 6. Passive head thrust test results in a subject with a unilateral vestibular deficiency (UVD) (same subject as in Figure 3). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject with a left UVD follows rightward head movement moderately well, but tracks leftward head thrusts poorly. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Response is delayed and of decreased magnitude, mildly for rightward head movements and dramatically for head movements toward the lesion side. C, Eye velocity vs head velocity for the first 80 milliseconds of responses.

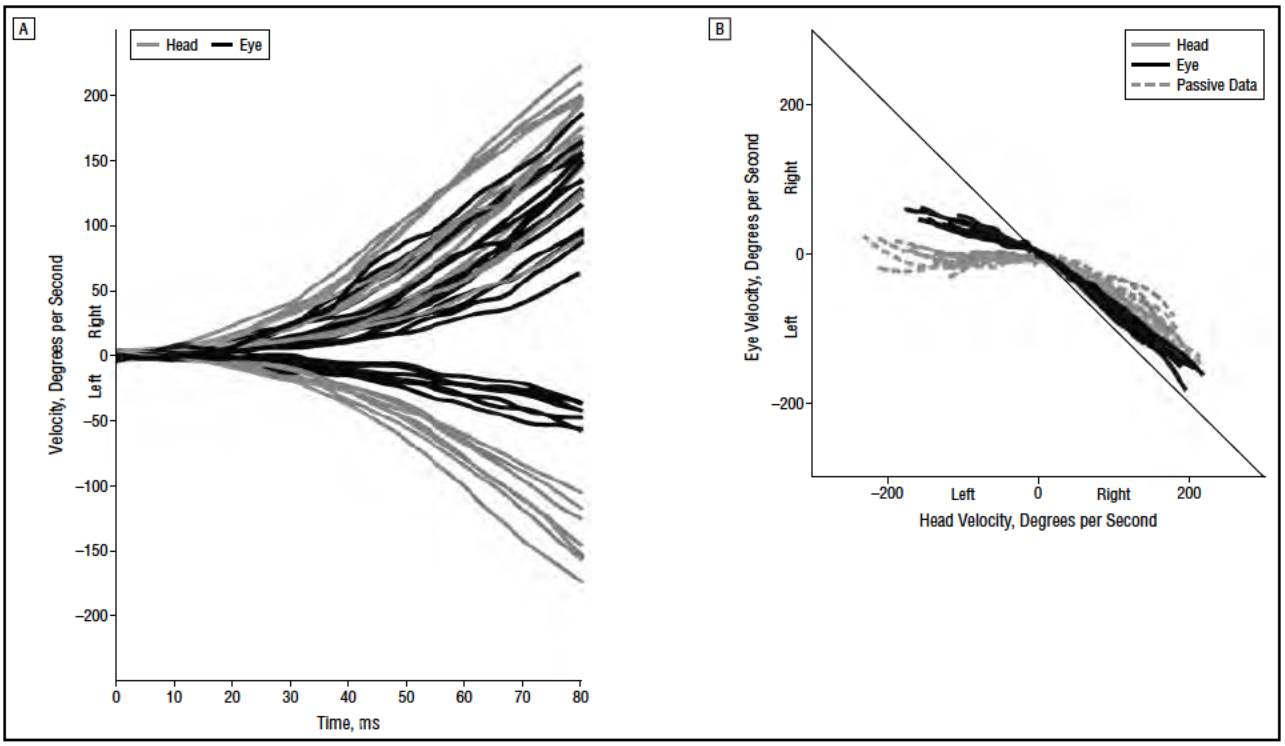

Figure 7. Active head thrust test results in a subject with a left unilateral vestibular deficiency (same subject for whom passive data are shown in Figure 6). A, Head and eye (inverted) velocities during the first 80 milliseconds of multiple active head thrust trials. B, Eye velocity vs head velocity. Although still not normal, the active eye movement response is significantly better than for passive trials, with shorter response delay and higher gains.

(15.0±4.5 milliseconds), but the difference was not significant ($P = .07$) (Figure 9C).

COMMENT

CLINICAL TESTS FOR IDENTIFICATION OF UVD

Traditional caloric and rotary chair tests for the identification of UVD fail to test the vestibular system using physiologically appropriate stimuli over the range of frequencies and accelerations for which the system apparently evolved. Caloric testing has the advantage of being strictly unilateral, but uses an unnatural stimulus (a thermal gradient) that, as measured by the eye movements it elicits, is the equivalent of a low frequency (0.025 Hz) and low amplitude (approximately 50°/s) head rotation. The caloric response can be altered by anatomic changes in the external ear canal and by individual variation in temporal bone anatomic features, and it only evaluates 1 of the 5 vestibular receptors (the lateral semicircular canal). Rotary chair vestibular testing is more physiologic; however, at the lower frequencies and velocities typically used in clinical vestibular laboratories, rotary chair tests are insensitive in the identification of chronic total unilateral vestibular hypofunction.[20,21] The sensitivity of rotary chair testing can be improved by comparison of responses to steps of acceleration with the head up and head down.[22]

In contrast, high-frequency and high-acceleration rotational stimuli unmask the inherent asymmetry in the vestibular system, as described by the second law of Ewald[9]—that the excitatory responses of vestibular pathway neurons encode motion over a larger dynamic range than do inhibitory responses. The eye movements elicited by such stimuli are principally due to excitation of the vestibular pathway arising from the canal ipsilateral to the direction of head acceleration and are, therefore, sensitive to a unilateral peripheral vestibular loss in that canal.

There are 2 high-frequency rotational stimuli in clinical use, pHTT and HART. The main objective of this study was to compare these 2 stimuli for the identification of unilateral vestibular hypofunction. The pHTT is a single, passive (operator delivered), unpredictable, high-acceleration (2000°-4000°/s^2), low amplitude (15°-30°) head rotation in the direction of a single semicircular canal. The HART is a continuous, active (self-generated), sinusoidal head rotation that begins slowly and becomes faster, covering a frequency range of about 1 to 6 Hz. During each test, subjects are required to fixate a visual target in front of them, and their ability to maintain visual fixation is accepted as a measure of VOR function. However, because HART is actively generated, non-VOR processes, such as predictive eye movements driven by an "efference copy" neural representation of the intended head movement, could contribute to maintaining gaze stability. As a result, VOR function could seem artificially enhanced, making HART a less sensitive and less accurate test of UVD.

We tested 11 patients who had undergone surgical labyrinthectomy and compared their responses with those of 5 subjects without UVD. The VOR gains for subjects without UVD using pHTT were insignificantly different from an ideal gain of 1.0, consistent with findings from previous studies.[23] For subjects with UVD, the VOR gains of 0.14±0.13 for ipsilesional head

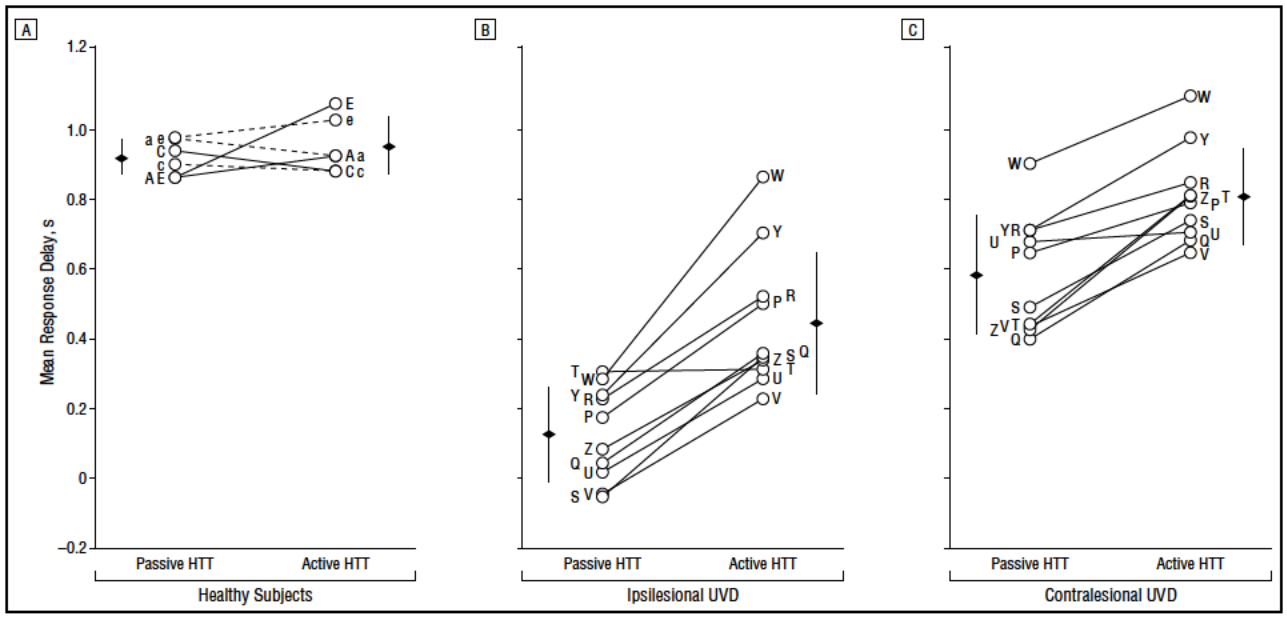

Figure 8. The vestibulo-ocular reflex (VOR) gains for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For healthy subjects without UVD, responses to active and passive HTT were not significantly different from each other or from 1.00 ($P>.05$ for all). In B, active HTT ipsilesional VOR gain is significantly ($P<.001$) higher than for passive HTT. In C, contralesional UVD VOR gain is also significantly ($P=.004$) closer to normal for active than for passive HTT. Bars represent mean±SD.

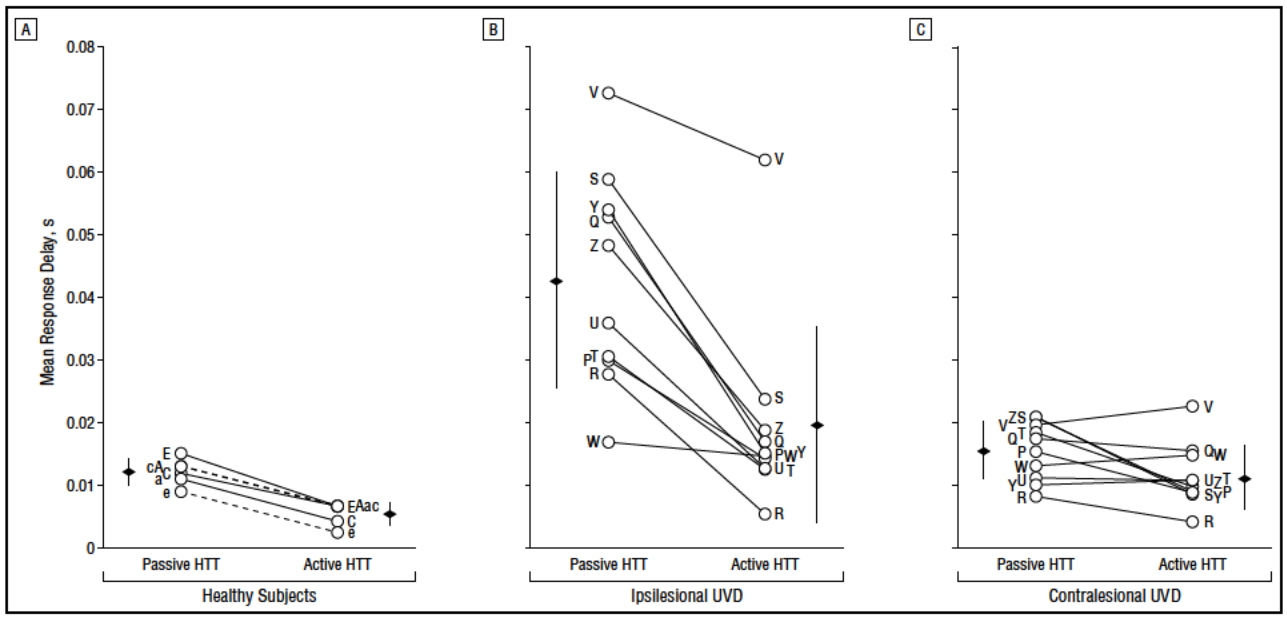

Figure 9. The response delays for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For subjects without UVD, the response delay was shorter for active than for passive HTT ($P=.006$. In B, for ipsilesional stimuli, response delay was significantly shorter for active HTT than for passive HTT ($P=.006$). In C, the response delay for contralesional stimuli was not significantly different ($P=.07$) between passive and active HTT. Bars represent mean±SD.

thrusts and 0.58±0.17 for contralesional head thrusts were significantly different, and the side with the lesion was clearly identified by pHTT in each patient, consistent with previous studies.[6,24] For HART, the VOR gain in subjects without UVD was essentially perfect, as one would expect. Surprisingly, however, although some subjects with UVD showed obvious asymmetry of VOR half-cycle gains on HART, there was no significant difference between the ipsilesional and contralesional VOR gains over the population with UVD ($P=.69$).

Other features of the response to the actively generated HART stimulus did provide an indication of the side of unilateral vestibular hypofunction. The occurrence of rapid, gaze-correcting, saccadic eye movements during head movements toward the side of the lesion, when they occur, is a reliable indication of the side of UVD. A bias velocity in the eye movement response during the HART stimulus (a direct current shift in the eye velocity trace toward the side of the lesion) pro-

vides another such indication. Head autorotation is, therefore, a useful paradigm, but comparison of VOR gains from half-cycle analysis of HART responses is not reliable for the identification of the side of UVD.

MECHANISMS FOR IMPROVED RESPONSES TO ACTIVE HEAD ROTATIONS

There are 2 main differences between HART and pHTT stimuli that might explain the improvement in VOR gain of patients with UVD for ipsilesional head movements during HART. First, the peak velocities and accelerations generated by the examiner in pHTT are greater than those that some patients are able to generate during HART. This constraint on stimulus intensity may limit HART's ability to discern unilateral weakness in such patients. In contrast, the stimulus used for pHTT is generated by the examiner and reaches a peak acceleration of 3000°/s^2 and a peak velocity of 250°/s. Although these head movements have low amplitude in displacement (10°-15°) and are well tolerated by patients, they are sufficient to elicit excitation-inhibition asymmetry and, thus, selectively probe the function of the excited canal.

A second difference is that the pHTT is passive, transient, and unpredictable, whereas the HART is a self-generated, sinusoidal, predictable stimulus. Self-generated, sinusoidal, steady-state rotations could allow for nonvestibular eye movement systems (eg, predictive eye movements, efference copy, and visual-following mechanisms) to contribute to the response. The existence of predictive mechanisms in augmenting vestibular responses is suggested by measures of visual acuity during head movement. Visual acuity is improved during active compared with passive head movements.[25,26] To further investigate these potential effects of self-generated stimuli, we asked patients to perform the HTT themselves, aiming to mimic the speed and amplitude of the operator-delivered head thrusts. Although patients had some difficulty reaching the top speed of operator-delivered stimuli (about 300°/s), they did reach speeds of approximately 200°/s, and we compared responses for active and passive head thrusts of similar peak velocity and acceleration. In subjects without UVD, the VOR gain was already insignificantly different from 1.00 for pHTT, so no significant change was observed for aHTT. For subjects with UVD, however, there was a marked improvement in VOR gain during active head movements, and the boost in VOR gain during active thrusts occurred from the onset of the head thrust, during the initial 20 to 40 milliseconds. The only 2 oculomotor systems with a latency of less than 40 milliseconds are the direct VOR and predictive eye movements. Saccades, the cervico-ocular reflex, and visual-following mechanisms, such as smooth pursuit, all have latencies that are 70 to 150 milliseconds.[27] From previous studies,[6,10] it has been shown that the 3-neuron VOR arc generates the response to pHTT, so the boost in gain during aHTT is, therefore, likely to be a result of predictive eye movements.

To study the effect of prediction, we measured the time between the onset of head rotation and the onset of eye rotation for active and passive head thrusts. We used manually applied passive head thrusts to approximate the usual clinical application of pHTT and the movements our subjects made during aHTT. Manual thrusts do not have an onset sharp enough or an acceleration constant enough to precisely measure VOR latency (estimated at 8.6 milliseconds in subjects without UVD by Collewijn and co-workers[28,29] using a torque-applying helmet in an HTT-type paradigm). To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset times (at which head and eye velocities cross set thresholds) and compute a response delay. This response delay probably does not precisely equal the true synaptic and axonal conduction delay of the VOR, because the finite rate of change of acceleration for manually applied stimuli and responses reduces the precision with which onset times for head and eye movements can be measured. However, this response delay provides a useful measure for comparison of responses to passive and active head thrusts.

For subjects without UVD, the response delay was shorter for active than for passive head thrusts. For subjects with UVD, the response delay during ipsilesional head thrusts was significantly longer for passive thrusts than for active thrusts. A similarly prolonged response delay for passive ipsilesional head rotations has been reported by Tabak et al.[8] The apparent reduction in the response delay to aHTT might be further evidence of preprogrammed eye rotation. Alternatively, some of the apparent difference in response delay may be due to a difference in VOR gain for the first 40 milliseconds of the response. The initial low velocity eye rotation makes it difficult to precisely define the onset of the eye rotation, and could give the appearance of a delayed onset.

TIMING OF CORRECTIVE EYE MOVEMENTS

Further evidence for preprogramming of eye rotations during active head thrusts was the reduced latency of rapid corrective eye movements during active compared with passive head thrusts. To compensate for a deficient VOR, patients with UVD used rapid eye movements to correct accrued gaze error and reacquire visual fixation of the target. During active head thrusts, these corrective movements occurred as early as 60 milliseconds after the onset of the head rotation, significantly earlier than during passive head thrusts (**Figure 10**). Under normal circumstances, the latency of voluntary true saccadic eye movements is 200 milliseconds.[25] This latency can be reduced to 100 milliseconds if the subject is trained in a specific task (so-called express saccades[30]). Tian et al[25] have shown that under pHTT conditions, these rapid eye movements have latencies shorter than those of express saccades. Our data indicate that these latencies can be reduced even further under conditions of active head movement.

CONCLUSIONS

Gain asymmetries on the pHTT reliably detected the presence of

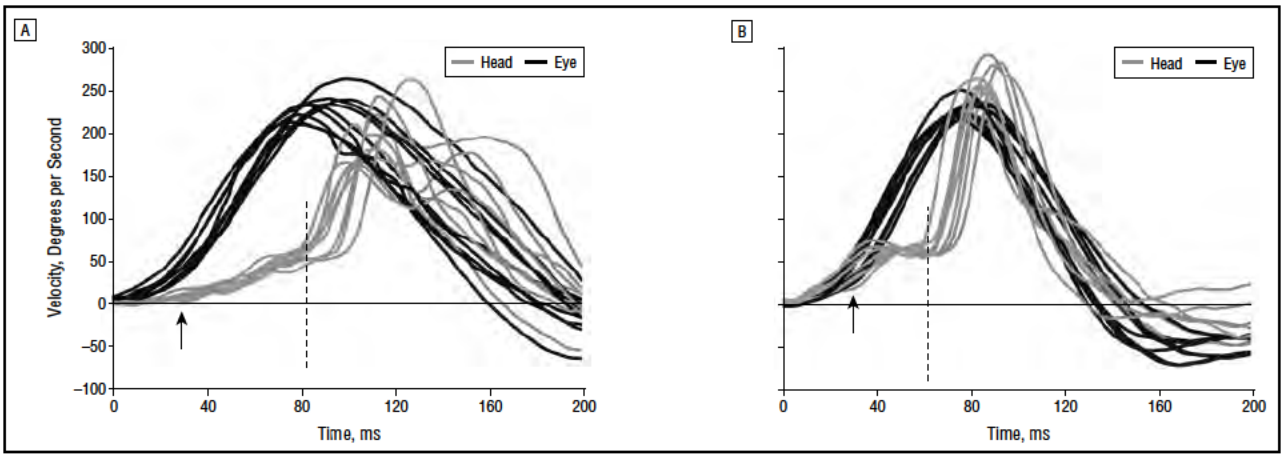

Figure 10. Corrective rapid eye movements during passive (A) and active (B) ipsilesional head thrust testing (HTT) in a 48-year-old subject with a unilateral vestibular deficiency 120 months after left labyrinthectomy (subject R shown in Figures 4, 8, and 9). Head and eye (inverted) velocities are shown. The response to active HTT has a higher initial vestibulo-ocular reflex gain (arrows) and earlier rapid compensatory eye movements (dashed lines).

known unilateral vestibular lesions in all patients with UVD tested, whereas the half-cycle gains measured from HART did not. Relative to pHTT, HART gains overestimated ipsilesional vestibular function and were, therefore, a less accurate indicator of unilateral hypofunction.

For subjects with UVD who are unable to generate high-acceleration sinusoidal head movements, HART may underestimate hypofunction simply because it relies on stimulus accelerations inadequate to silence inhibitory pathways arising in the intact ear. In contrast, the high-acceleration movements of pHTT reliably identify response asymmetry by more selectively probing the function of the excited canal. However, even for head movements of similar acceleration and time course, the measured VOR during self-generated head rotations (aHTT) has significantly higher gain and shorter response delay than does the VOR to passive unpredictable stimuli (pHTT).

Subjects with UVD may use a variety of strategies for improving visual fixation, including preprogrammed eye movements designed to augment the deficient VOR and to compensate for a planned or anticipated head movement. Tests using passive high-acceleration head thrusts delivered unpredictably in time and direction should, therefore, be more sensitive for discerning VOR hypofunction than tests using active and/or predictable stimuli.

Accepted for publication February 18, 2002.

This study was supported by the Garnett Passe and Rodney Williams Memorial Foundation, Parkville, Australia (Dr Cremer); the Royal Australasian College of Physicians, Sydney, New South Wales (Dr Cremer); grants K23 DC00196 (Dr Carey) and R01 DC02390 (Dr Minor) from the National Institutes of Health, Bethesda, Md; and a Johns Hopkins Clinician-Scientist Award (Dr Carey).

We thank Grace C. Y. Peng, PhD, David S. Zee, MD, Adrian G. Lasker, MS, and Dale C. Roberts, MS, for their assistance with data acquisition; and John K. Niparko, MD, and Howard W. Francis, MD, for their referral of subjects.

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A Clinical Sign of Canal Paresis

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NEUROLOGY

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

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Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

The video head impulse test

Diagnostic accuracy in peripheral vestibulopathy

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ABSTRACT

Background: The head impulse test (HIT) is a useful bedside test to identify peripheral vestibular deficits. However, such a deficit of the vestibulo-ocular reflex (VOR) may not be diagnosed because corrective saccades cannot always be detected by simple observation. The scleral search coil technique is the gold standard for HIT measurements, but it is not practical for routine testing or for acute patients, because they are required to wear an uncomfortable contact lens.

Objective: To develop an easy-to-use video HIT system (vHIT) as a clinical tool for identifying peripheral vestibular deficits. To validate the diagnostic accuracy of vHIT by simultaneous measures with video and search coil recordings across healthy subjects and patients with a wide range of previously identified peripheral vestibular deficits.

Methods: Horizontal HIT was recorded simultaneously with vHIT (250 Hz) and search coils (1,000 Hz) in 8 normal subjects, 6 patients with vestibular neuritis, 1 patient after unilateral intratympanic gentamicin, and 1 patient with bilateral gentamicin vestibulotoxicity.

Results: Simultaneous video and search coil recordings of eye movements were closely comparable (average concordance correlation coefficient r_c = 0.930). Mean VOR gains measured with search coils and video were not significantly different in normal (p = 0.107) and patients (p = 0.073). With these groups, the sensitivity and specificity of both the reference and index test were 1.0 (95% confidence interval 0.69–1.0). vHIT measures detected both overt and covert saccades as accurately as coils.

Conclusions: The video head impulse test is equivalent to search coils in identifying peripheral vestibular deficits but easier to use in clinics, even in patients with acute vestibular neuritis.

Neurology® 2009;73:1134–1141

GLOSSARY

BVL = bilateral vestibular loss; **HIT** = head impulse test; **IMU** = inertial measurement unit; **ITG** = intratympanic gentamicin; **vHIT** = video head impulse test; **VN** = vestibular neuritis; **VOR** = vestibulo-ocular reflex.

The head impulse test (HIT) is a useful bedside examination to identify a peripheral vestibular deficit for example in patients with vestibular neuritis (VN).[1-4] The clinician briskly rotates the patient's head to detect "overt" catch-up saccades after head rotation as a sign of semicircular canal paresis. "Covert" saccades are saccades that occur during the head rotation that may be imperceptible to the naked eye and hence confound the diagnosis.[5,6] In patients with acute VN, spontaneous nystagmus also interferes with assessment of bedside HIT.

Up to now, the scleral search coil technique has been the gold standard for HIT measurements.[7-9] It quantifies the VOR deficit and shows the associated pattern of overt and covert catch-up saccades in vestibular deficient patients.[6,10] However, search coil measurements require the subject to wear an uncomfortable contact lens, are time intensive, are expensive, and are not practical for acute patients.

The goal of the study was to develop an easy-to-use high-speed video HIT system[11] (see video on the *Neurology*® Web site at www.neurology.org) as a clinical tool to identify a periph-

Supplemental data at www.neurology.org

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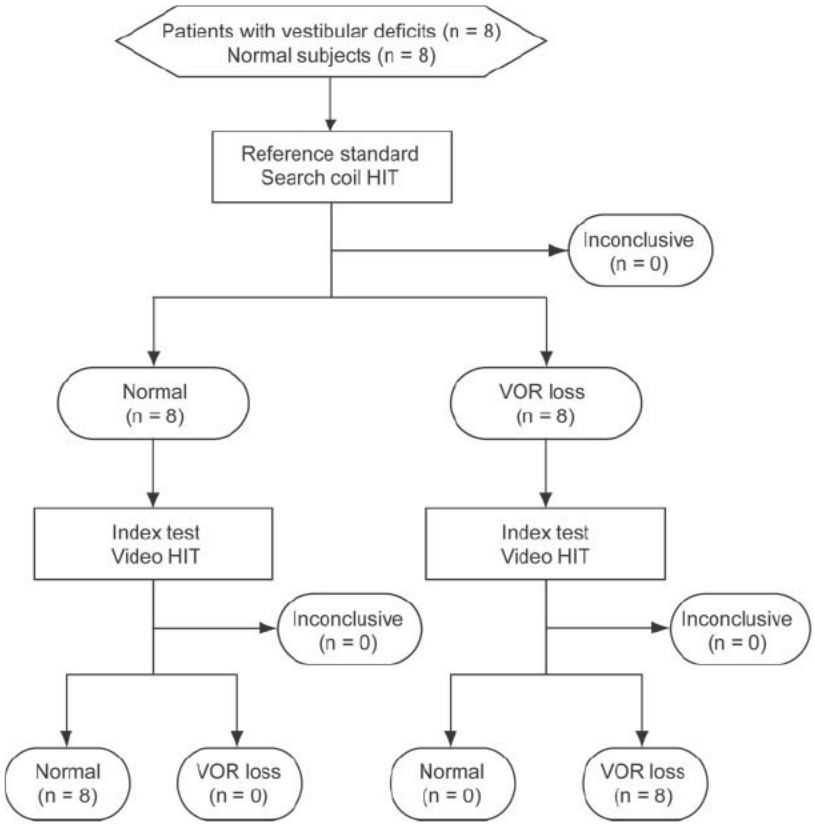
Disclosure: Author disclosures are provided at the end of the article.

eral vestibular deficit. To validate the diagnostic accuracy of our video HIT (vHIT) system, we compared the measures from video recordings with simultaneous measures from scleral search coil recordings of the same eye during head impulses in healthy subjects and patients with a wide range of previously independently identified vestibular deficits.

METHODS **Design.** The study was a prospective, cross-sectional comparison of the index test (vHIT) to the reference standard (HIT measured by scleral search coils) in patients with prior, independently identified vestibular deficits due to unilateral vestibular neuritis, intratympanic gentamicin, or systemic gentamicin and healthy asymptomatic control subjects (figure 1). Patients with a broad range of vestibular deficits were enrolled because we wished to establish how well each test identified vestibular deficits of varying severity.

Subjects. Sixteen subjects were recorded simultaneously with video-oculography and scleral search coils. Six patients with VN (mean 52 years, age range 38–59 years, 1 female) showed evidence of enduring unilateral loss of vestibular function after an illness with acute onset of prolonged rotational vertigo and postural imbalance associated with spontaneous nystagmus, nausea, or vomiting that fulfilled the clinical criteria of VN.[12] One patient with Ménière disease (53 years, female) with a unilateral vestibular deficit due to intratympanic gentamicin injection and a patient with bilateral vestibular loss due to systemic gentamicin vestibulotoxicity (72 years, male) were also tested. Eight healthy subjects without any history, symptoms, or clinical signs of vestibular disease (mean 35 years, age range 25–66 years, 2 females) served as controls. Diagnosis of a peripheral vestibular deficit was confirmed in all patients by bithermal caloric testing with water irrigation at 30°C and 44°C, resulting in a canal paresis factor greater than 25%.[13] Patients were tested between 5 months and 27 years after onset of symptoms. Two additional patients with acute VN (29 and 32 years, both female) were recorded within 36 hours after onset with video-oculography alone. Eligible patients were recruited at the Hearing and Balance Clinic, Royal Prince Alfred Hospital, Sydney, Australia. No potential subject was excluded. All subjects and patients were tested between August and October 2008.

Figure 1 **Flow chart for the comparison of video and search coil measures of head impulses**



HIT = head impulse test; VOR = vestibulo-ocular reflex.

Standard protocol approvals and patient consents. Written informed consent was obtained from all subjects. Written consent to disclose has been obtained from any recognizable persons in the published photograph and video. The protocol was approved by the Sydney South West Area Health Service Ethics Committee in accordance with the Declaration of Helsinki.

Experimental procedure. Subjects were instructed to fixate a laser dot on a screen at 91 cm distance in dim light. Approximately 50 horizontal head impulses to each side were manually applied with unpredictable timing and direction. Peak head velocity of the impulses was gradually increased from 50° to 250°/second (acceleration 750°–5,000°/second^2, amplitude 5°–20°) with the aid of visual feedback of head velocity for the experimenter.[5,6] The same eye was recorded simultaneously with video-oculography and scleral search coils (figure e-1). Two data sets were obtained for each recording session to show the reliability of the calculated gains and concordance of the video and search coil methods. All recordings were performed by the same team of 3 coauthors. Head impulses were always delivered by the same experimenter, unless acting as a subject. The experimenters were unmasked as to whether they were testing a patient or a healthy subject. All experimenters are graduates and have at least 5 years' experience in vestibular research. There were no adverse events from performing the tests.

Video-oculography. Right eye position was recorded at 250 Hz with a small, lightweight, high-speed digital (IEEE 1394a) video camera (Firefly MV, Point Grey Research Inc., Vancouver, British Columbia, Canada). The camera was mounted on a very lightweight motorcycle glasses frame with an elastic strap that locked comfortably onto the bridge of the nose and around the eye sockets to minimize slippage of the camera relative to the head. The image of the eye was reflected from a hot mirror to the camera. The eye was illuminated by 2 infrared light–emitting diodes (TSUS502, Vishay Intertechnology, Malvern, PA) run at 20 mA to keep infrared radiation far below exposure risk levels.[14] Head velocity was measured by a miniature 6-degrees-of-freedom inertial measurement unit (IMU) assembled from 2 dual-axis gyroscopes (IDG-300 InvenSense, Santa Clara, CA) and a 3-axis linear accelerometer (ADXL330, Analog Devices, Norwood, MA). The camera, hot mirror, and IMU were rigidly mounted onto the spectacle frame. The small mass of the system (approximately 60 g) minimized inertia during head rotation and so minimized slippage of the glasses. Eye position was calibrated in vivo with projected targets from a glasses-mounted laser. Video images were analyzed online to calculate eye position using a pupil detection method based on a center-of-gravity algorithm[15] written in LabVIEW (National Instruments, Austin,

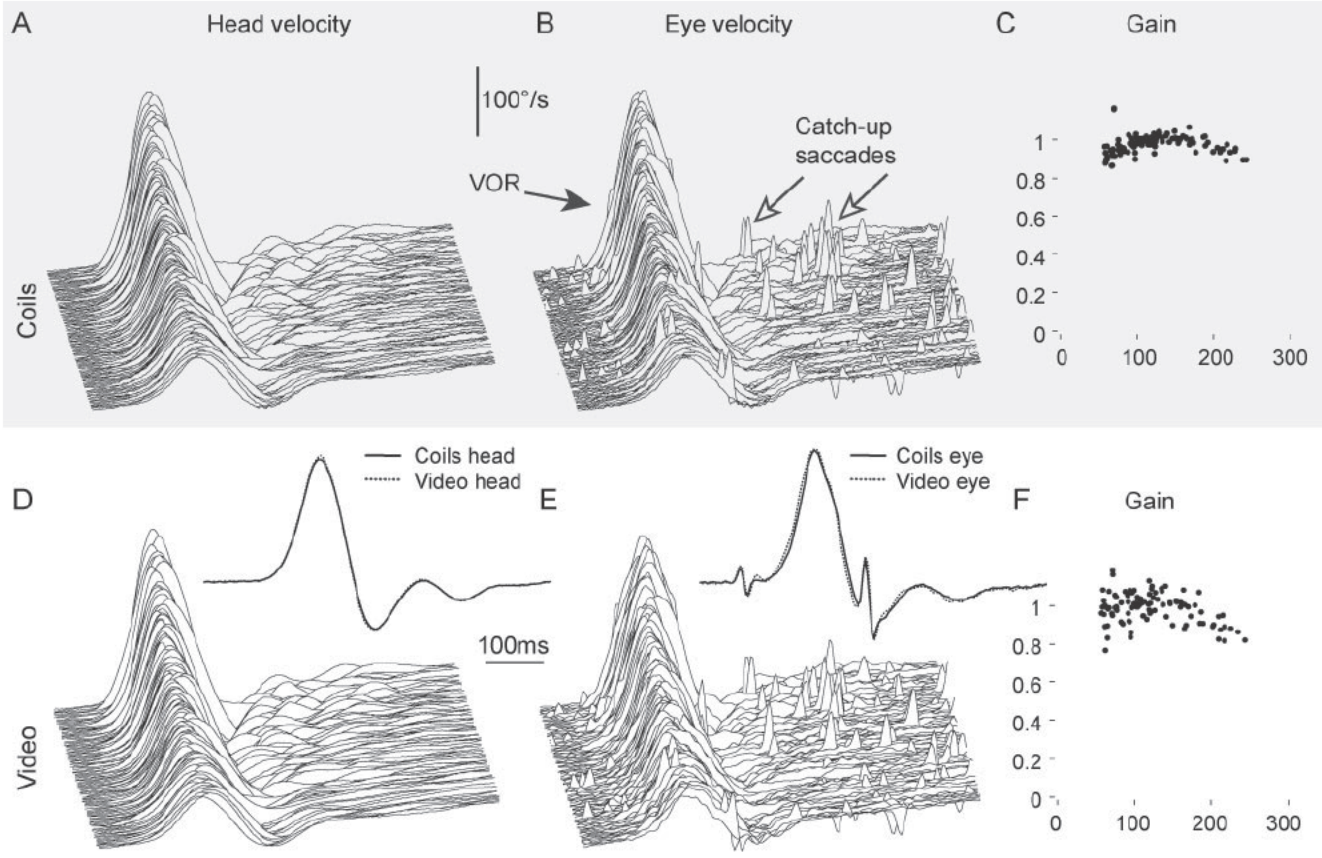
TX). Eye velocity was obtained from a 2-point differentiator and low-pass filtered (0- to 30-Hz bandwidth).

Scleral search coil recording. Right eye and head position were recorded with the scleral search coil technique in a 1.9 × 1.9 × 1.9-m magnetic coil frame (CNC Engineering, Seattle, WA).[2,9] Dual search coils (Skalar, Delft, The Netherlands) were precalibrated in vitro on a gimbal. The eye coil was inserted after topical anesthesia with Alcaine 0.5% eyedrops (Alcon Laboratories Australia Pty. Ltd., Frenchs Forest, Australia). The head coil was attached to a dental impression tray. Three-dimensional head and gaze position signals were sampled at 1,000 Hz, digitized with 16-bit precision, and low-pass filtered (0- to 100-Hz bandwidth). Three-dimensional rotation vectors and angular velocity vectors of head, gaze, and eye were derived from coil voltages.[16]

Data analysis. Offline analysis of the experimental data were automated with customized LabVIEW software. To synchronize the video and search coil measurements, a square wave signal produced by a signal generator was acquired by each system together with eye and head velocity measurements. Data from the 2 systems were then synchronized by aligning the square wave signals. Head impulses were automatically selected and aligned to peak head acceleration. Trials with blinks and outliers were automatically excluded, based on an envelope around the expected eye velocity response. Velocity gain of the horizontal VOR[7] was calculated for both recording methods as the ratio of mean eye velocity over mean head velocity during a 40-msec window centered at peak head acceleration. Data from both recording methods was processed simultaneously with the same automated algorithms to exclude any analysis bias. Invalid head impulses (e.g., with blinks) were excluded from both data sets, resulting in mirror-symmetric data. Both data sets were analyzed in all simultaneously recorded patients (no missing data sets). The criterion for a normal VOR velocity gain was that it should be 0.68 or greater, based on HIT data from 12 previously published healthy asymptomatic subjects[6,10] in which the mean HIT velocity gain measured by search coils with identical apparatus and procedures to those used here was 0.81 ± 0.068 SD, so that the mean ± 2 SD units incorporates 95% of the population and yields a lower cutoff of 0.68.

Statistical analysis. The results of the study are reported in accordance with the Standards for Reporting of Diagnostic Accuracy Studies (STARD).[17,18] The concordance correlation coefficient[19] was used to index the similarity between video and search coil recordings for each impulse. A coefficient was calcu-

Figure 2 Simultaneous video and search coil recordings of a horizontal head impulse test in a normal subject



Simultaneous angular head velocity recordings of a search coil mounted on a dental impression tray (A) and a gyroscope mounted on the video glasses (D) during graded horizontal head impulses. The close similarity between the 2 recordings demonstrates minimal slippage of the video glasses relative to the head. Simultaneous angular eye velocity recordings of a scleral search coil (B) and high-speed video-oculography (E) of the same eye. Both recording techniques accurately record the vestibulo-ocular reflex (VOR) and detect even the smallest catch-up saccades. Normal VOR gains of individual head impulses are comparable with search coil recording (C) and video-oculography (F). Scleral search coil recording is sampled at 1,000 Hz (A and B), video-oculography is sampled at 250 Hz (D and E), and both are plotted on the same time scale. Head and eye velocity traces from individual impulses are stacked according to increasing peak head velocity. (Insets D and E) Simultaneous video and search coil recordings are shown superimposed to facilitate comparison of single head and eye velocity traces.

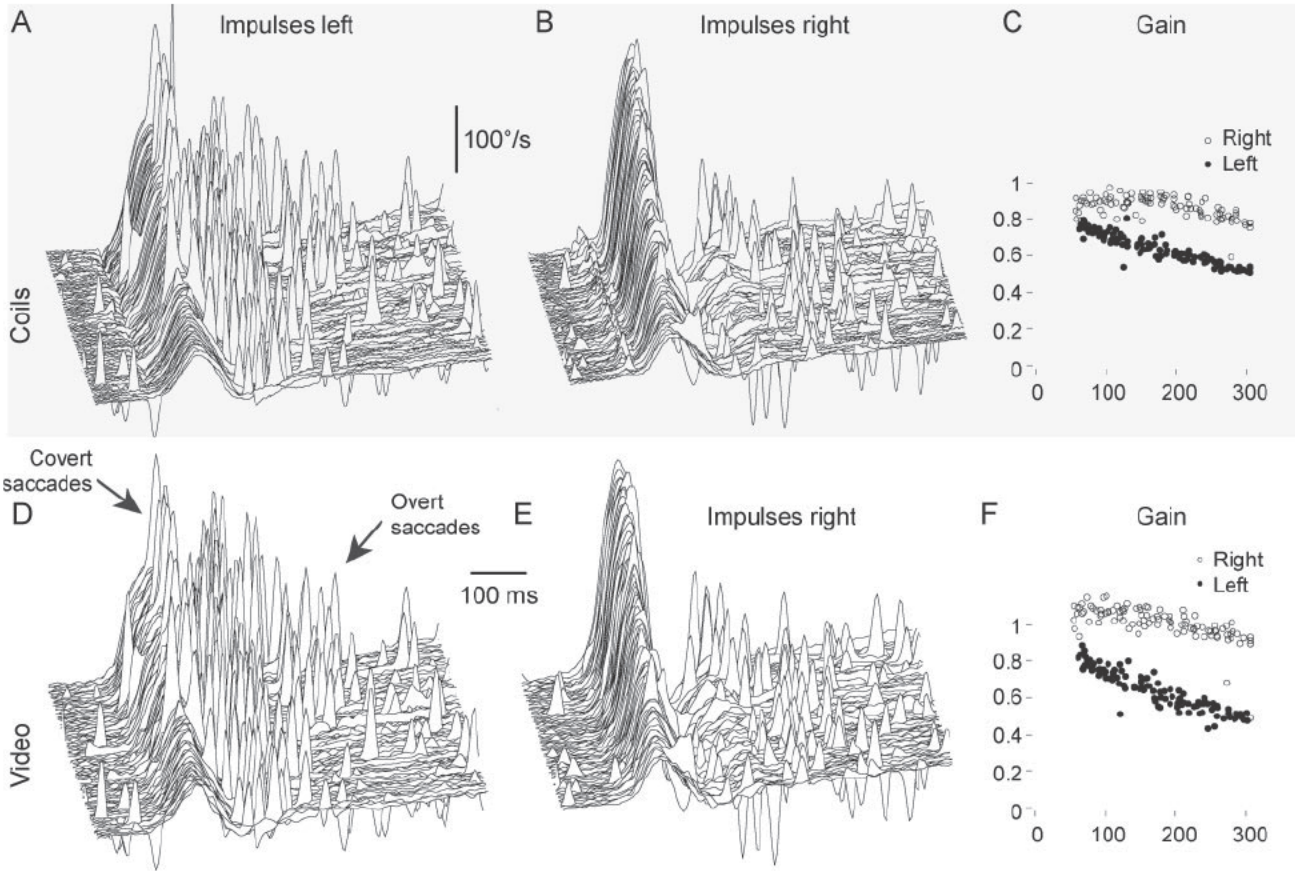
lated for each impulse and the values for an individual were averaged. Paired-sample t tests were used to test whether the VOR gains were different in video and search coil recordings. The Pearson product-moment correlation coefficient was used to determine test-retest reliability between the 2 separate test runs. Because of the small sample size, no subgroup analyses were performed by disease subtype or clinical examiner.

RESULTS **Head movement recording.** A close fit of the video glasses on the head is crucial for accurate eye movement recording during head impulses.[20] Simultaneous measurements of angular velocity from an inertial measurement unit (IMU) mounted on the glasses and from a search coil on a dental impression tray were virtually identical (figure 2, A and D), with an average concordance correlation coefficient[19] of $r_c = 0.999$.

Eye movement recording. Simultaneous measurements of video recording and scleral search coil of the same eye are shown in figure 2, B and E (normal subject), and figure 3, A, B, D, and E (patient with VN). Both recording techniques not only show VOR, but also record the smallest catch-up saccades (figures 2 and 3, arrows). Each method is highly repeatable, and the 2 methods are in very close agreement. The concordance correlations were calculated for every impulse in all subjects and patients: The mean r_c for each subject is shown in figure 4, and the average r_c of all subjects was 0.930.

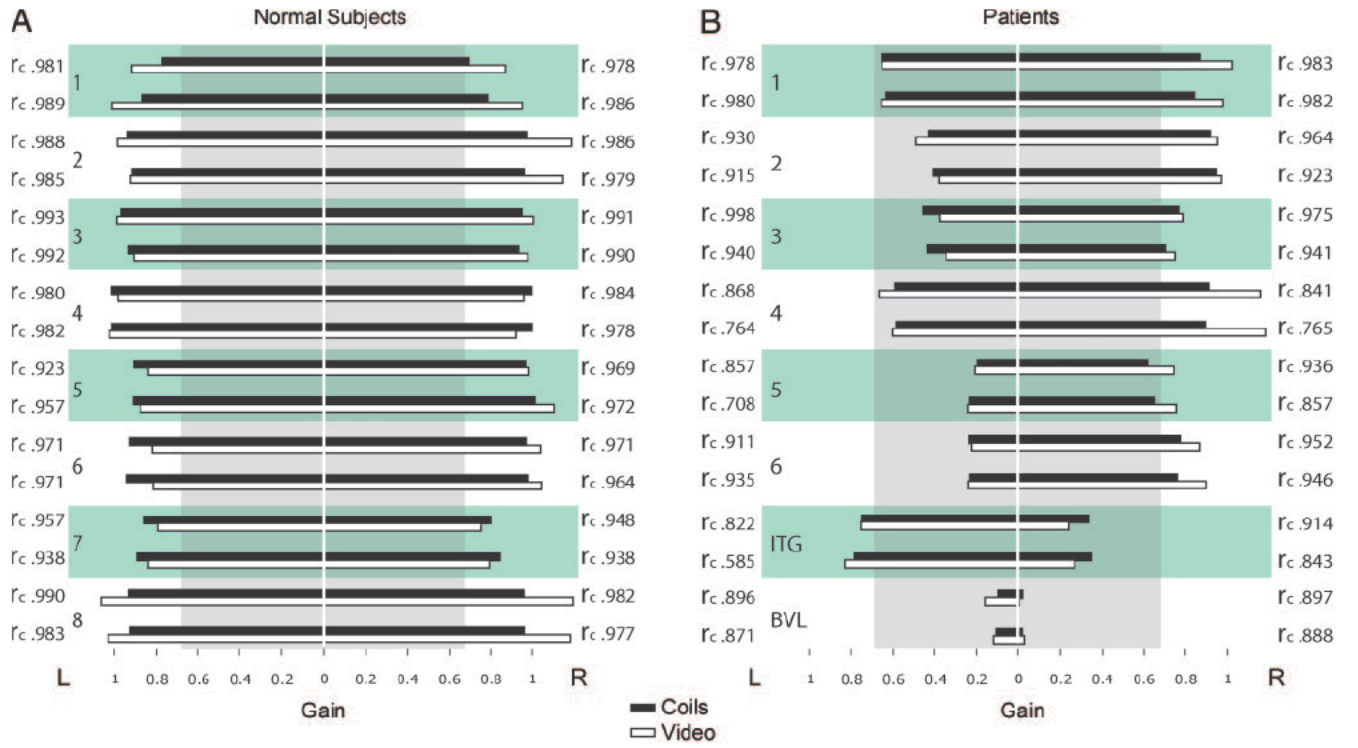
Detection of vestibular deficit. A VOR deficit was defined as being a HIT gain of less than 0.68. The reference standard, scleral search coils, correctly identified the VOR deficit in all patients, as did the video measures (figures 1 and 4). Thus, the sensitivity of the reference and index test were both 1.0 (95% confidence interval 0.69–1.0), and the specificity of both the reference and index test were 1.0 (0.69–1.0).[21] Using a paired t test, the difference between average VOR gain for search coils and video was not significantly different from zero for patients (mean difference = 0.040, n = 8, $t = 1.930$, $p = 0.073$) and for normal (mean difference = 0.043, n = 8, $t = 1.717$, $p = 0.107$). To test reproducibility, each

Figure 3 Simultaneous video and search coil recordings of a horizontal head impulse test in a patient after left vestibular neuritis



(A and D) Head impulses to the left (affected) side demonstrate the reduced vestibulo-ocular reflex (VOR) response. Both recording methods detect covert saccades during head rotation and overt saccades after head rotation (arrows). The pattern of catch-up saccades is identical for both recording methods. (B and E) Both recording methods demonstrate an almost normal VOR response to the healthy right side, with small overt saccades after head rotation. (C and F) Both recording methods clearly differentiate the reduced VOR gains of the left affected side (filled circles) from the right healthy side (empty circles). (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

Figure 4 VOR gain measures with search coils compared with video-oculography in normal subjects and patients with peripheral vestibular deficits



(A) The vestibulo-ocular reflex (VOR) gain for healthy subjects is almost identical for the 2 different methods of measurement. The 2 sets of data give highly reproducible values of VOR gain. (B) Video-oculography identifies the affected side in vestibular neuritis (1–6) and intratympanic gentamicin (ITG, 7) patients as reliably as search coil measurements. Patient 8 with bilateral vestibular loss (BVL) due to systemic gentamicin vestibulotoxicity demonstrates reproducibility of both methods at very low VOR gains. Bar graphs show the mean VOR gain measures with search coils (black bars) compared with video-oculography (white bars). For each individual, 2 data sets were recorded in the same session. Concordance correlation coefficients (r_c) index the similarity between search coil and video-oculography measurements. The vertical gray box indicates deficient VOR gain values (cutoff gain 0.68).

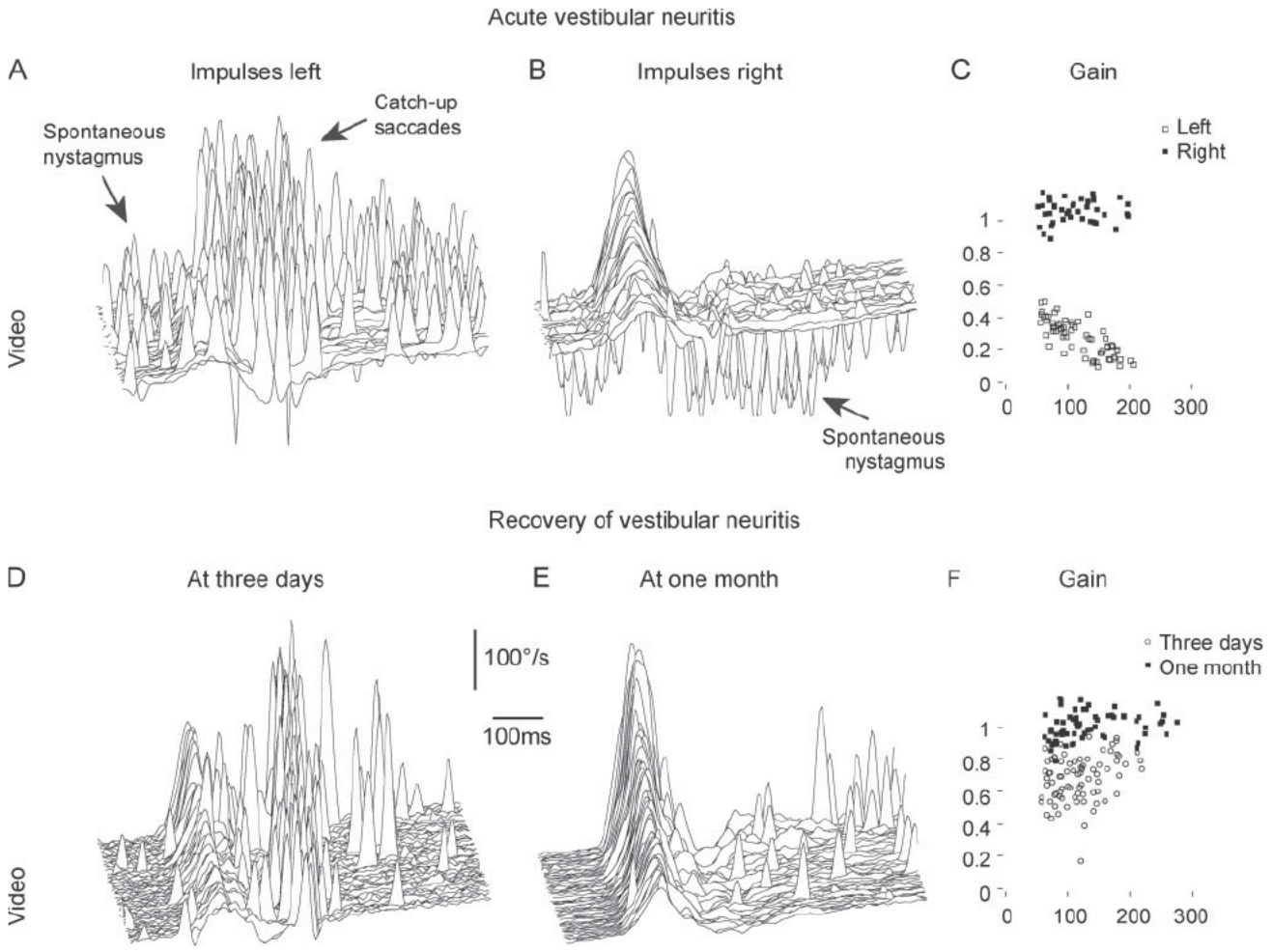
subject was given 2 separate test runs, and the test-retest reliability coefficient using a Pearson product-moment correlation was significant at 0.99 for search coils and 0.99 for the video measures. Both recording techniques readily detected covert saccades during head rotation as well as overt saccades after head rotation (figure 3, A and D).

Clinical application. This video method of recording eye movements during head impulses makes examination of acute vertigo patients possible where scleral search coil recordings are impractical. The unilateral VOR deficit of patients with acute VN can be detected even in the presence of spontaneous nystagmus (figure 5, A–C). The noninvasive and short (approximately 10 minutes) nature of vHIT also facilitates follow-up examinations to document recovery of vestibular function (figure 5, D–F).

DISCUSSION In 1988, 2 of the authors reported[1] a simple indicator that allowed clinicians at the bedside to identify peripheral deficits of horizontal semicircular canal function—the presence of a saccade after a small, rapid, passive, unpredictable, horizontal head rotation (a "head impulse") by the clinician while the patient attempted to maintain gaze on a target. If semicircular canal function is impaired, the slow phase eye velocity is inadequate, so the eyes move with the head (off the target), and at the end of the head rotation, the patient must make a saccade to return gaze to the target. That corrective saccade is easily detectable if it is made after the head rotation has stopped, and so these saccades are termed "overt" saccades.[6]

The HIT has found wide use as a qualitative clinical sign, but it has major limitations: 1) There is no objective measure of VOR gain or of the corrective saccade: the clinician's report is based on the subjective visual observation of the presence of an overt saccade. 2) Different clinicians carry out this impulse with very different trajectories, so the accelerations and velocities used differ considerably. 3) Usually only few head rotations are given, so there is not a range of stimuli for generating a stimulus-response function. 4) Some patients may hide their peripheral vestibular deficit with "covert" saccades during head rotation. As such, their peripheral pathology is missed, and it may be incorrectly concluded that they

Figure 5 Diagnosis of acute vestibular neuritis and documentation of recovery



(A–C) Video head impulse test of a patient 2 days after onset of acute vestibular neuritis. (A) In head impulses to the affected left side, catch-up saccades replace the deficient vestibulo-ocular reflex (VOR). The spontaneous nystagmus (scattered spikes) beats in the same direction as the catch-up saccades. (B) In head impulses to the healthy right side, the VOR is preserved and the spontaneous nystagmus beats to the opposite direction. (C) The VOR gain is deficient to the left (open squares) but preserved to the right (filled squares). (D–F) Video head impulse test of a patient 3 days (D) and 1 month (E) after onset of acute vestibular neuritis. Between the 2 recordings, the VOR gains returned toward normal (F), the majority of catch-up saccades disappeared, and the patient recovered from symptoms. (A, B, D, and E) Signs of eye velocity traces are inverted so that VOR responses and catch-up saccades always point upward.

have a central vestibular disorder responsible for their symptoms. Such a "covert" saccade is almost impossible to detect by simple visual observation: it cannot be distinguished from the normal slow phase eye velocity needed for proper compensatory eye movement.

To overcome these limitations, we have developed a new lightweight, minimal-slip, high-speed video-oculography system[11] (vHIT, video) that measures eye velocity during head rotation. Importantly, the camera is mounted on a specially designed, very lightweight frame to minimize inertia and slippage (figure e-1). Instant feedback about every single head impulse allows the examiner to apply a set of standardized graded impulses. The system is easy to use in a clinical setting, provides an objective measure of the VOR, and detects both overt and covert catch-up saccades in patients with vestibular loss. Measurements are quick (approximately 10 minutes) and noninvasive, and the automated analysis software provides instant results.

The simultaneous video and search coil HIT recordings validate the diagnostic accuracy of high-speed video recording. Despite fundamentally different recording methods, we achieved head and eye velocity recordings that were closely comparable. Both methods correctly identified the peripheral vestibular deficit in patients with highly reproducible VOR gains and detected even the smallest catch-up saccades.

With the bedside HIT, clinicians have to deliver high head velocities to optimize the chance of detecting the corrective saccade.[6] Although high velocities also help to reveal VOR asymmetry in patients with VN,[6] lower velocities of approximately 100° to 150°/

second are sufficient to detect the deficit in acute patients (figure 5C). In practice, this is an advantage for vHIT because the effects of slippage of the glasses and inertia are smaller at lower head velocities, and patients with acute vertigo better tolerate these lower velocities.

The simple application of vHIT allows the clinician to diagnose patients with VN acutely while they are ill and assess them again after they have recovered, providing objective evidence of the VOR deficit and the extent of its recovery. As figure 5, A–C, shows, these measures are possible even in the presence of a very vigorous spontaneous nystagmus.

Bedside HIT remains a useful clinical sign to assess patients with acute spontaneous vertigo because it helps to distinguish between acute VN, where the test is positive, and a central vestibular lesion, where the test is usually negative. However, between 9% and 39% of positive clinical HIT results have been reported in patients with acute cerebellar or brainstem strokes.[22,23] vHIT will be a suitable tool to determine whether these cases are really due to reduced VOR gain or simply result from clinical misjudgment. This way, video HIT will help to improve diagnostic accuracy for patients with acute spontaneous vertigo in the emergency department.

ACKNOWLEDGMENT

The authors thank all the participants of the study, Anthony Anderson for recording of the video, and David Newman-Toker for advice.

AUTHOR CONTRIBUTIONS

The statistical analysis was conducted by the authors Hamish G. MacDougall and Ian S. Curthoys (Vestibular Research Laboratory, School of Psychology, University of Sydney, Australia).

DISCLOSURE

Dr. MacDougall receives research support from the National Aeronautics and Space Administration [NNJ04HF51G (Coinvestigator) and NNJ04HD66G (Coinvestigator)], the National Space Biomedical Research Institute [NNJ07ZSA002N (Co-PI) and SM00801 (Co-PI)], the European Space Agency [SPIN (Coinvestigator)], the Australian Research Council [DP0665402 (APD)], and the Garnett Passe and Rodney Williams Memorial Foundation [Project Grant 2009 (PI)]. Dr. Weber received a research fellowship from the Garnett Passe and Rodney Williams Memorial Foundation. Dr. McGarvie reports no disclosures. Dr. Halmagyi serves on the editorial boards of *Acta Otolaryngologica*, *Otology & Neurotology*, *Audiology & Neurotology*, and the *Italian Journal of Otolaryngology*; and receives research support from the National Health and Medical Research Council [570890 (Co-PI) and 457511 (Co-PI)]. Dr. Curthoys serves on the editorial advisory boards of *Experimental Brain Research* and *Brain Research Bulletin*; and has received research support from the Australian Research Council [DP-0665402 (Chief Investigator)], the National Health and Medical Research Council [457511 (Chief Investigator) and 509206 (Coinvestigator)], and the Garnett Passe and Rodney Williams Memorial Foundation [L2907 R6934 (Chief Investigator)].

Received March 14, 2009. Accepted in final form July 7, 2009.

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Effective January 15, 2009, authors submitting Articles or Clinical/Scientific Notes to *Neurology*® that report on clinical therapeutic studies must state the study type, the primary research question(s), and the classification of level of evidence assigned to each question based on the classification scheme requirements shown below (left). While the authors will initially assign a level of evidence, the final level will be adjudicated by an independent team prior to publication. Ultimately, these levels can be translated into classes of recommendations for clinical care, as shown below (right). For more information, please access the articles and the editorial on the use of classification of levels of evidence published in *Neurology*.[1-3]

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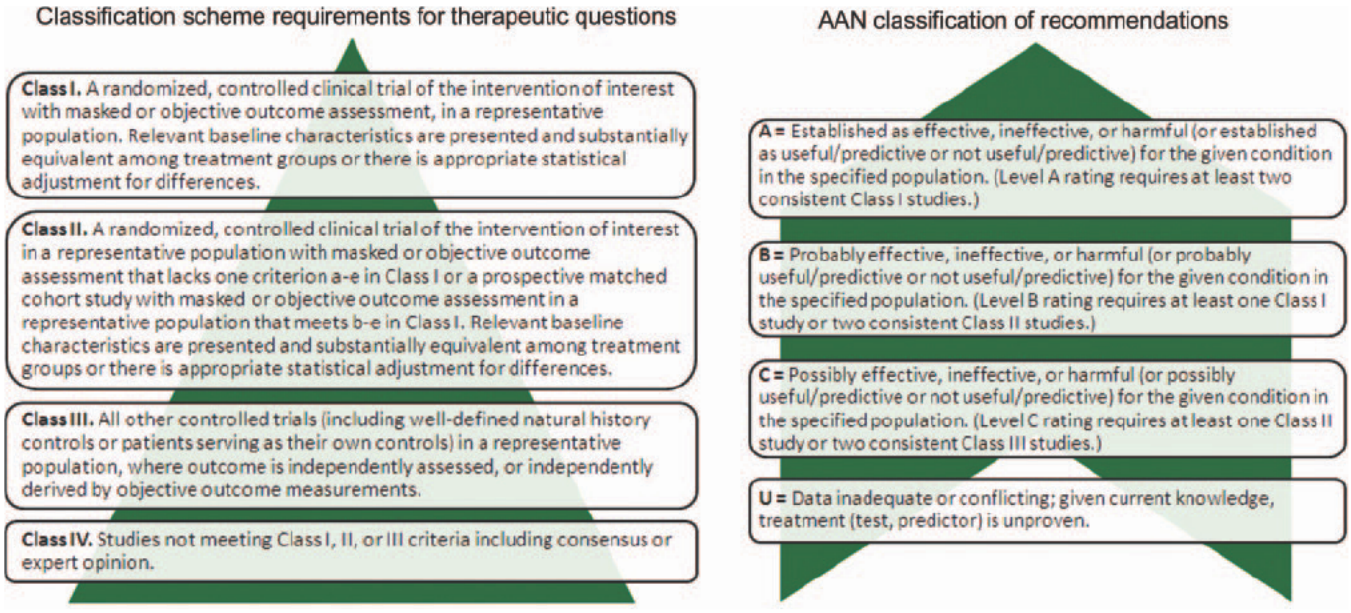

The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy

H. G. MacDougall, K. P. Weber, L. A. McGarvie, G. M. Halmagyi and I. S. Curthoys

Neurology 2009;73;1134-1141
DOI: 10.1212/WNL.0b013e3181bacf85

This information is current as of October 5, 2009

Clinical application of a new objective test of semicircular canal dynamic function – the video head impulse test (vHIT).

A safe, simple and fast clinical vestibular test.

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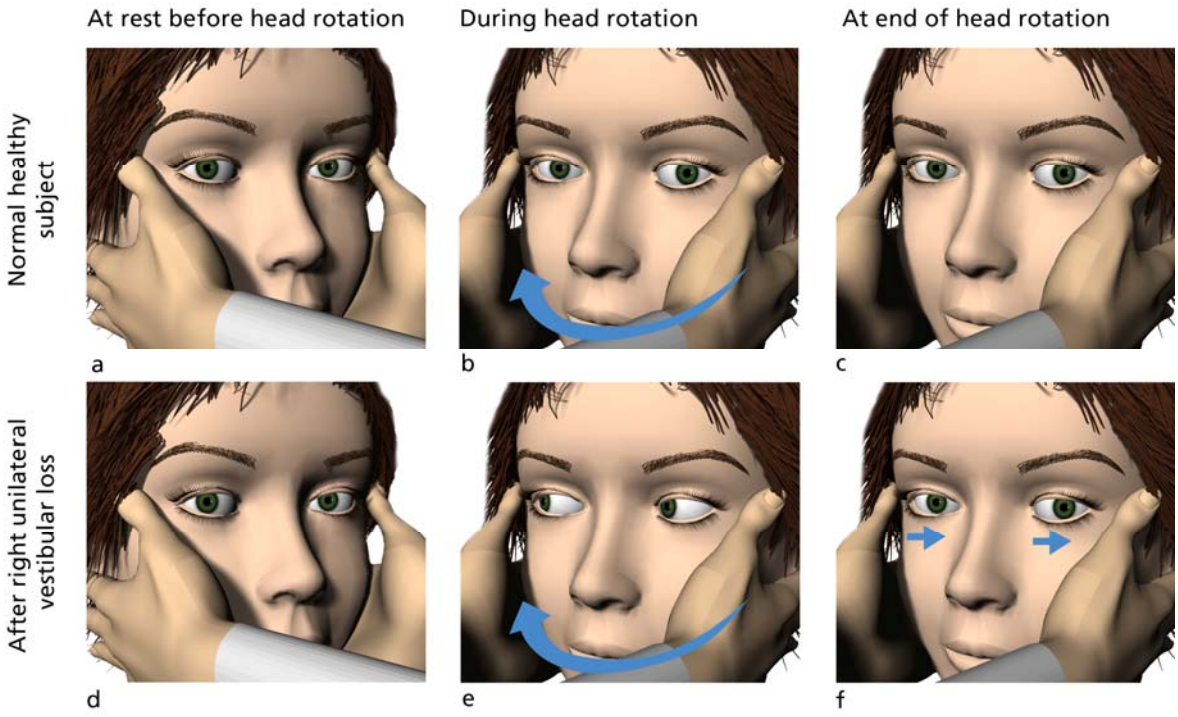
Preface

If you want to test hearing in the left ear, then you present a sound to the left ear to stimulate the auditory system and measure the response – for example whether a patient can hear the sound or whether there is a measurable physiological response to the sound. If you want to measure vision in the left eye, you present a visual stimulus to the left eye and ask the patient what they saw. Similarly if you want to test the semicircular canal function in the left ear ***you turn the person's head to the left*** to stimulate the semicircular canal in the left ear, and you measure the eye movement response while the patient tries to keep looking at a stationary target straight ahead. That is the response - just how well they can keep looking at the target. That head turn activates the receptors in the left horizontal semicircular canal and results in the eye movement response, which corrects for the head turn so that in healthy subjects the eyes stay looking at the target. So in the case of the semicircular canals, the response is that both eyes turn to correct for, or compensate, for the head turn - to keep looking at the target as the head is turned, so the person's gaze is stable during the head turn. The eye movement response is a tool to probe of the function of the semicircular canals of the inner ear. Of course to test the semicircular canals on the right you turn the patients head to the right.

What happens if the patient has ***no*** semicircular canal function in one ear? Now turn the head to that affected side but since the canal is not working, the eyes do not correct for the head movement. Instead of the eyes turning to correct for the head turn, ***the eyes move with the head***. So at the end of the head movement the patient must make a saccade back to the target. That saccade tells the clinician that the semicircular canal is not working properly – the response is just not adequate, so there is probably a deficit in the semicircular canal on that side. In most patients it is easy to see the corrective saccade at the end of the head turn and so we call it an ***overt saccade***. For example if, at the end of a head turn to the **left**, the clinician sees the patient has to make an overt saccade to get back to the target, then the semicircular canals on the **left** side are deficient.

This figure shows the difference between the responses of a healthy subject (top row) and a patient with a vestibular loss (bottom row), at comparable moments before, during and after the head rotation.



How can we objectively measure the adequacy of the semicircular canal response? One way is to measure the speed of the eye rotation and compare it to the speed of the head rotation. Eye velocity should be about equal and opposite head velocity and the ratio of the two velocities is called the vestibulo-ocular response (VOR) gain and in healthy subjects it is usually around 1.0. Or we can measure the saccades – whether they are there or not. The following explains how to do this test in real life.

The Head Impulse Test

The clinician stands before the patient, holding the patient's head in his hands, and the patient, who is looking straight at the clinician, is asked to keep staring at the earth-fixed target (the clinician's nose). If the clinician now turns the patient's head abruptly and unpredictably to the left or right, through a small angle (only 10-20 degrees - not a large angle), that head turn is what we call the **head impulse**. If the patient has a functioning vestibulo-ocular response they will be able to maintain gaze on the target because the vestibulo-ocular response drives the eyes to rotate to exactly compensate for head rotation and so maintain fixation. However if the patient's vestibulo-ocular response is inadequate then their eyes will be taken off target during the head rotation, because their eyes will not rotate at the correct speed to exactly compensate for head rotation. *So an inadequate VOR means that the eyes* ***go with the head during the passive unpredictable head turn*** *and will be taken off target by the head turn, so that at the end of the head turn the patient must make a corrective saccade back to the clinician's nose.* To the clinician watching the patient's eyes, this saccade is usually very clear, and we have termed it an ***overt saccade***. It is the tell-tale sign of inadequate semicircular canal function on the side to which the head was rotated. So an overt saccade after a leftwards head rotation means the left semicircular canal has a deficit. If there is any doubt, the clinician just repeats the head impulses until they are satisfied.

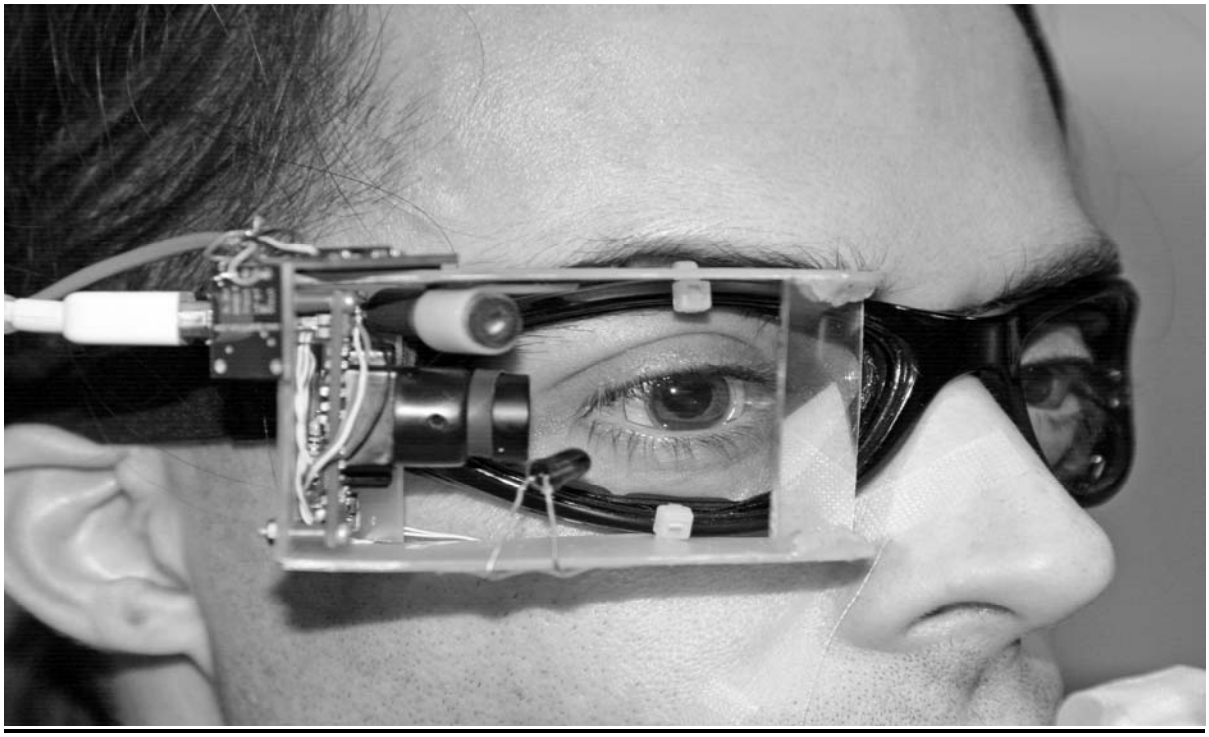
Covert saccades

Does the absence of an overt saccade mean that the canal is normal? No. Because our measures with scleral search coils (Weber et al 2008) showed that some patients with a semicircular canal deficit on one side could manage to generate small corrective saccades *actually during the head movement*, so that at the end of the head turn to their affected side hardly any overt saccades were necessary to bring the eyes back on target. These small hidden saccades during the head rotation had concealed their inadequate VOR. We have called these hidden saccades ***covert saccades***. It is important to realize that covert saccades can entirely obscure or conceal even a complete, total loss of canal function. These covert saccades are very fast and they occur *during* the head rotation and they are almost impossible to detect by the naked eye. It was only by using scleral search coils, the "gold standard" of eye movement measurement, during patient testing that we found them (Weber et al 2008), but clearly clinicians want to be able to detect them so they can accurately diagnose whether the patient has a vestibular loss or not and our new vHIT tests does just that. (Examples of recordings of overt and covert saccades are shown in the "Understanding vHIT Data" section)

The video Head Impulse Test (vHIT)

The simplest clinical indicator of a semicircular canal deficit is what I have just described - the head impulse test (also called the head thrust test, or the Halmagyi-Curthoys test, or the Halmagyi test). But detecting that saccade is ***subjective*** and relies on the clinician seeing the small corrective saccade after an abrupt head movement. The new indicator we describe below - the vHIT test uses a video camera to measure the eye movement and so it is ***objective*** and provides hard copy of the patient's performance. But first we will describe the test procedure and its logic.

This head impulse sign was described by Halmagyi and Curthoys in 1988, and from that time to the present, the clinical use of the head impulse test has been to indicate deficient canal function by virtue of the clinician (subjectively) observing whether there was an overt saccade or not at the end of the head turn. However some vestibular-deficient patients were missed by the head impulse test, even by expert clinicians, probably because of covert saccades. Clearly the ideal would be to have ***objective*** measure of both the head movement stimulus and the eye movement response using a system fast enough and accurate enough to detect covert saccades. The scleral search coil method of measuring eye movement achieves this aim, but it is clinically unrealistic, because of its huge expense, the high cost of each coil, the complexity of processing the data and the fact that patients do not like having a contact lens placed on their eye. However we have developed a new lightweight video system procedure – which we have called the video head impulse test (vHIT) – which does measure eye velocity and does detect covert saccades and is non-invasive and practical in clinics. Most importantly we have shown by direct comparisons that the accuracy of vHIT matches the accuracy of the "gold standard" search coil technique. vHIT has been validated by direct measures of VOR performance in healthy subjects and patients by two independent methods – search coils and vHIT. At ***exactly the same time: the same subject, the same eye movement responses were measured independently by these two methods and compared and found they both give essentially the same answer.***



How does the video head impulse test (vHIT) work?

The procedure is as described above, except that the patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage, because any slippage of the goggles will move the camera relative to the eye and so be registered as a movement of the eye and so generate artifactual data.

In testing, the clinician first conducts a quick calibration procedure, in which the patient is required to look between two laser spots projected from the goggles onto the wall. Then the clinician asks the patient to keep staring at an earth-fixed target, and gives the patient brief, abrupt, horizontal head rotations through a small angle (about 10-20 degrees), unpredictably turning to the left or right on each trial.

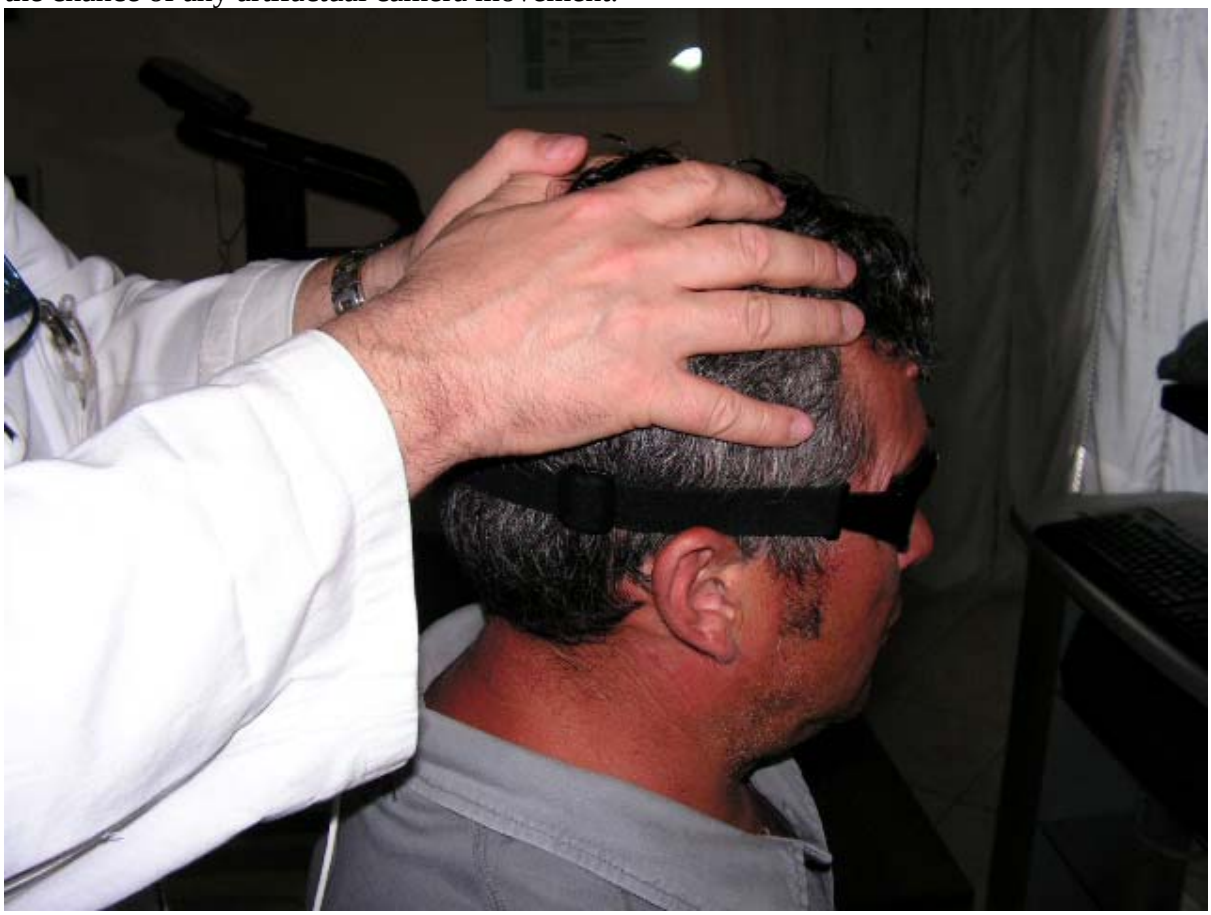
Stimulus

Displacement = 10° to 20°

Peak Head Velocity = 100°/s to 250°/s

Peak Head Acceleration = 1000°/s^2 to 2500°/s^2

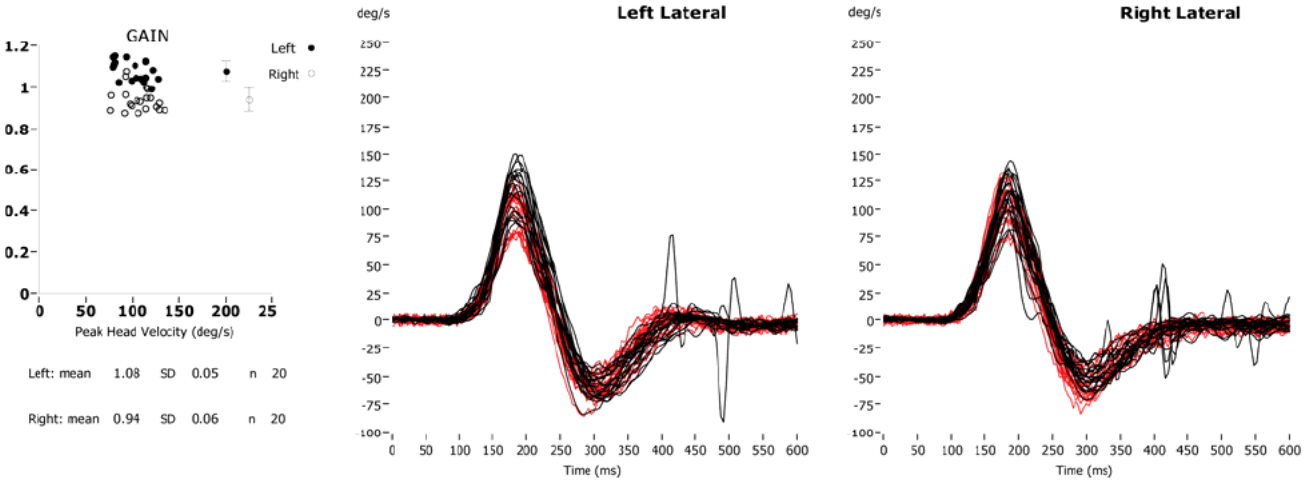
The clinician's hands must be well away from the goggles and the goggle-strap to minimize the chance of any artifactual camera movement.



The head movement speed is measured by the sensor in the goggles, and the image of the eye is captured by the high-speed firewire camera (250Hz) and processed by very fast software to yield eye velocity. At the end of each head turn the head velocity stimulus and eye velocity response are displayed simultaneously on the screen (figures below) so the clinician can see, just how good the stimulus and response were. In a full test usually around 20 impulses are delivered randomly in each direction and it may take 4 or 5 minutes to do that. At the end of the full test all the head velocity stimuli and eye velocity responses are superimposed and displayed on the computer screen, together with a graph of the calculated VOR gain for every head rotation as shown below in (Fig. 4). VOR gain is the ratio of eye velocity to head velocity, and so it should be ideally be about 1.0 for constant gaze during the head rotation. In practice normal healthy subjects typically have VOR gains less than 1.0 (around 0.8 - 0.9). But with vHIT any deficient response or VOR response asymmetry is easily seen. So in the space of about 5 minutes the clinician has an ***objective*** measure of the VOR response for both directions of rotation. The 250Hz video is fast enough that covert saccades can be detected and these are easily visible on the superimposed records (see below)

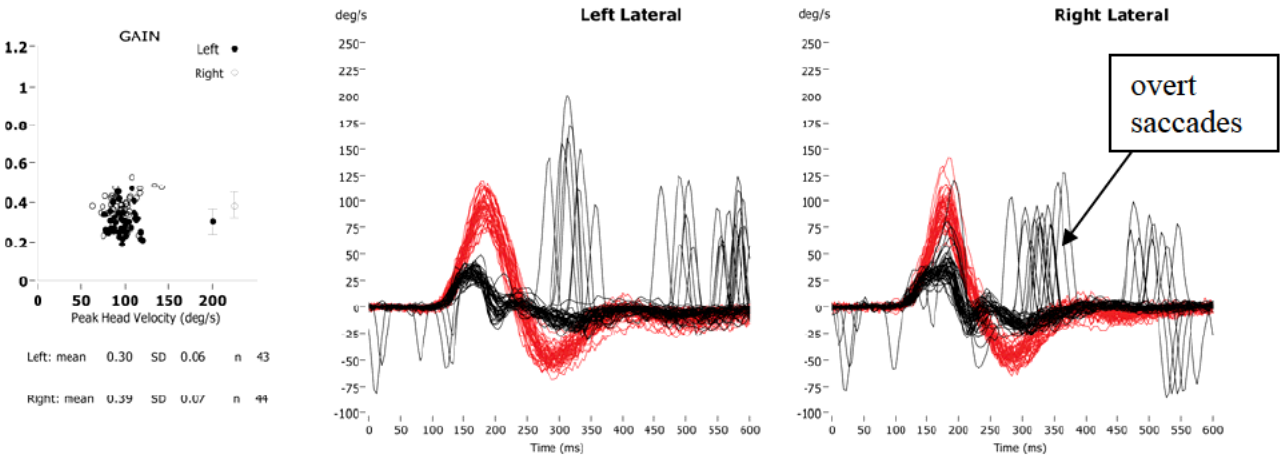
Examples of results from vHIT

1. Normal healthy subject



These and the following figures show superimposed records of eye velocity responses (black traces) to brief unpredictable horizontal head rotations (red traces) to the left and right. The first column shows the plot of the gain of the VOR for all of these (closed circles are for leftwards and open circles are for rightward impulses), as a function of peak head velocity. The average VOR gain is shown graphically and given numerically beneath the graph, together with the standard deviation and the number of impulses in each direction. Here the eye velocity matches head velocity so the head velocity traces and eye velocity traces are almost exactly superimposed showing that the eye velocity closely matches the head velocity. (We have inverted the eye velocity traces in these figures so you can more easily compare the eye velocity with head velocity.). Corresponding to that, the VOR gain values are good 1.08 for left and 0.94 for right. These VOR gain values are in the normal range and there is no asymmetry.

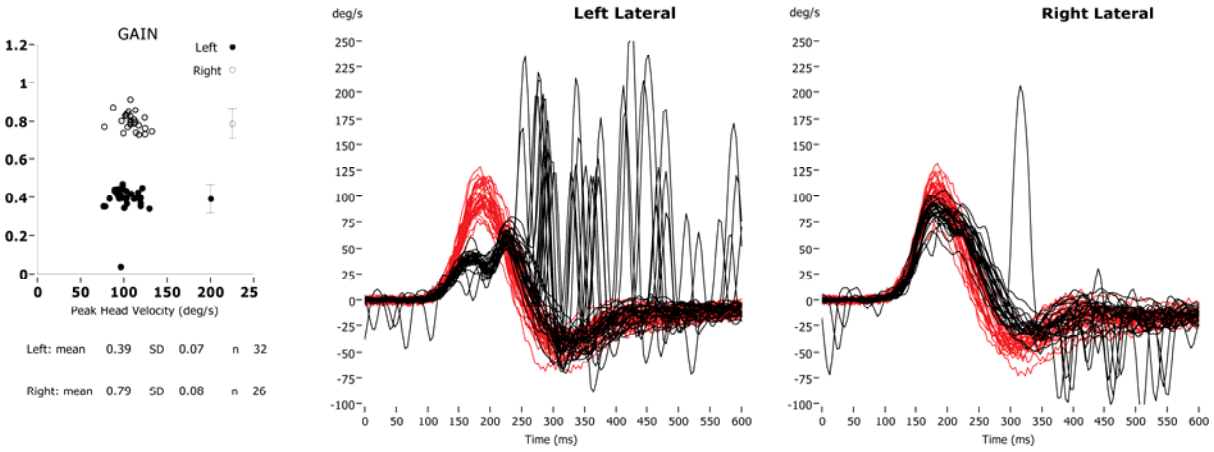
2. A patient with bilateral vestibular loss.



Comparable records from a patient with bilateral vestibular loss. Now the black traces (eye velocity) records do not follow the red (head velocity) records. The patient's VOR is clearly inadequate as the gain graph shows: the average VOR gain is 0.30 for left and 0.39 for right and both of these are significantly outside the normal range. In addition, there are large

saccades at the end of most head impulses and these are overt saccades and would be easily seen by the clinician at the end of the head turn.

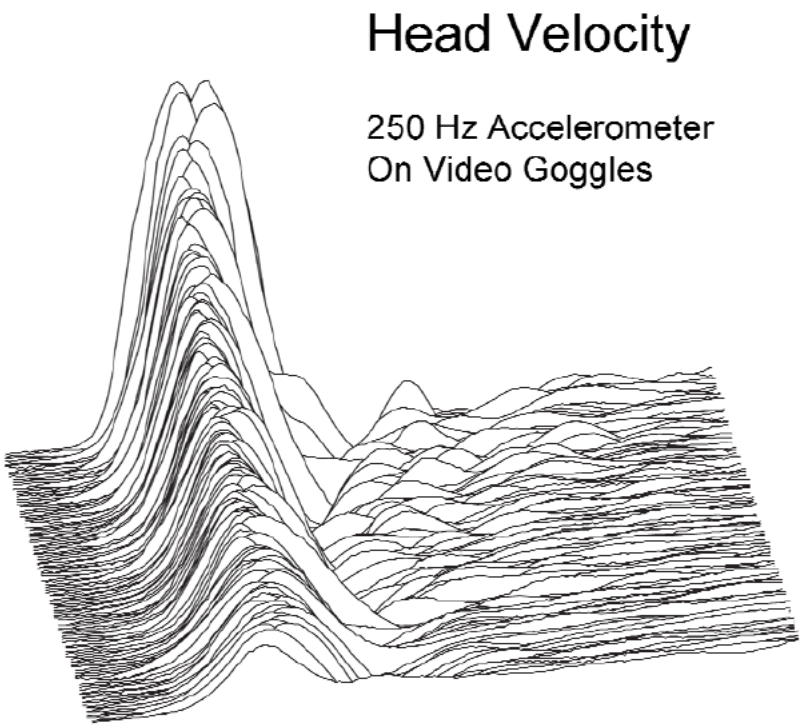
3. A patient with a unilateral vestibular loss



In this patient, eye velocity matches head velocity reasonably well for rightward head rotations, but not for leftwards head rotations (towards the patients affected side). Not only is the eye velocity insufficient, but there are many saccades which occur during the head impulse (covert saccades) and also some saccades which occur after the head impulse (overt saccades). The covert saccades would be very difficult for the clinician to detect by visual observation. The numerical values show the clear VOR gain difference for the two sides: to the right (healthy side) the average gain is 0.79 (in the normal range), whereas to the left (affected side) the average gain is only 0.39, significantly below the normal range. So this person has a severe left sided horizontal canal deficit.

The Graded series of head velocities

Instead of just giving one value of head velocity over and over again, as in the above examples, it is possible to give a graded series of increasing head velocities, which allows us to show the data in a 3-d format. Each line is a separate head impulse and they have been sorted so that the head velocity progressively increases from very small to large.



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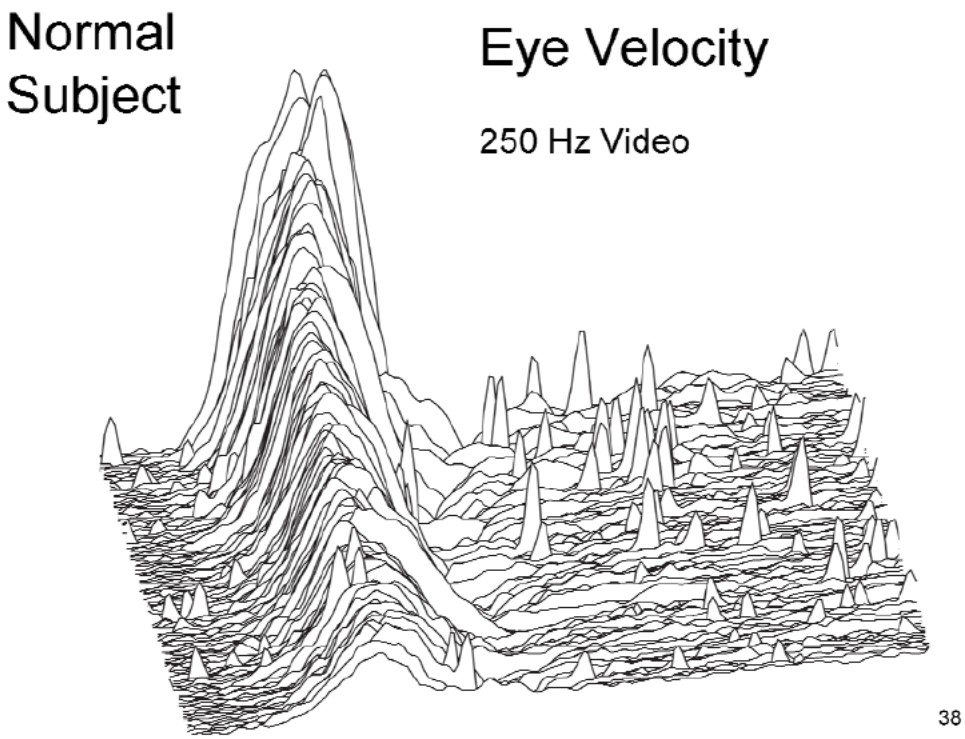

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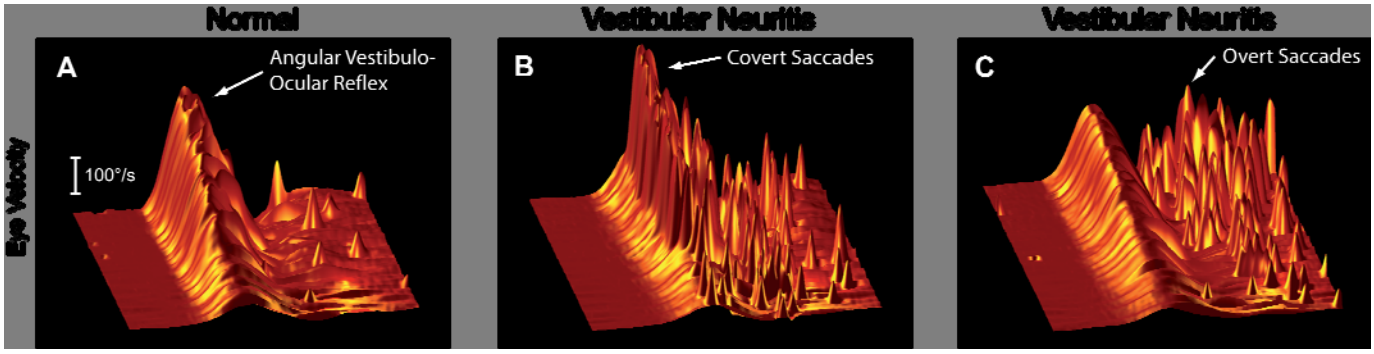
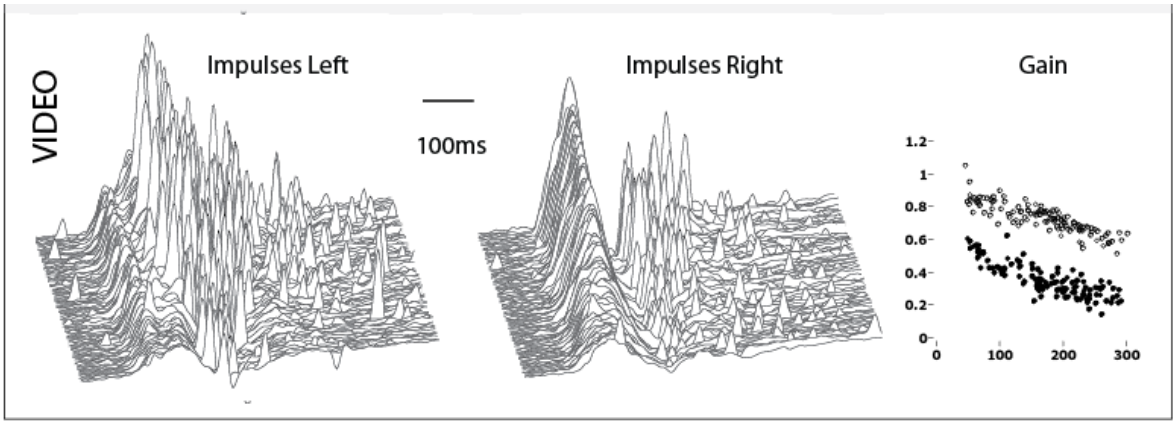
The figure above shows that as these head velocities increase, in a healthy subject there is a corresponding increasing series of matching eye velocities. (The small "stalagmites" are small saccades.) In fact it is usually sufficient just to show the graded series of eye velocity responses:

In the following examples a red surface has been fitted over the eye velocity responses (this example and the other "red surface figures" shown below have been recorded with search coils). Even in healthy subjects the VOR gain is not exactly 1.0, so even some healthy people occasionally make some very small overt or covert saccades. These are usually so small the clinician does not see them but the coils and video methods are so sensitive that they pick these up.

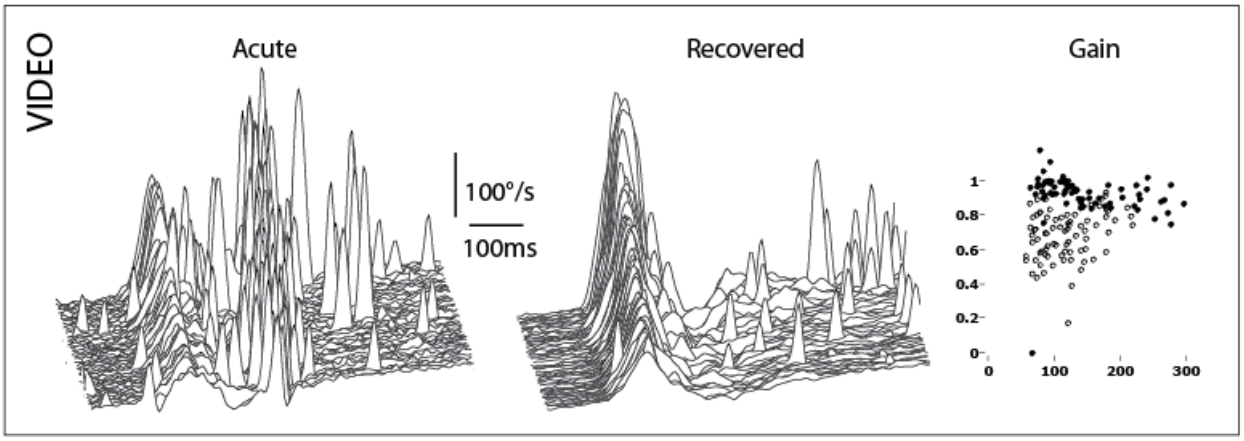
Now the saccades appear as small stalagmites, after the eye velocity response. This graded series method is especially good at showing the difference between overt saccades (panel C) and covert saccades (panel B).



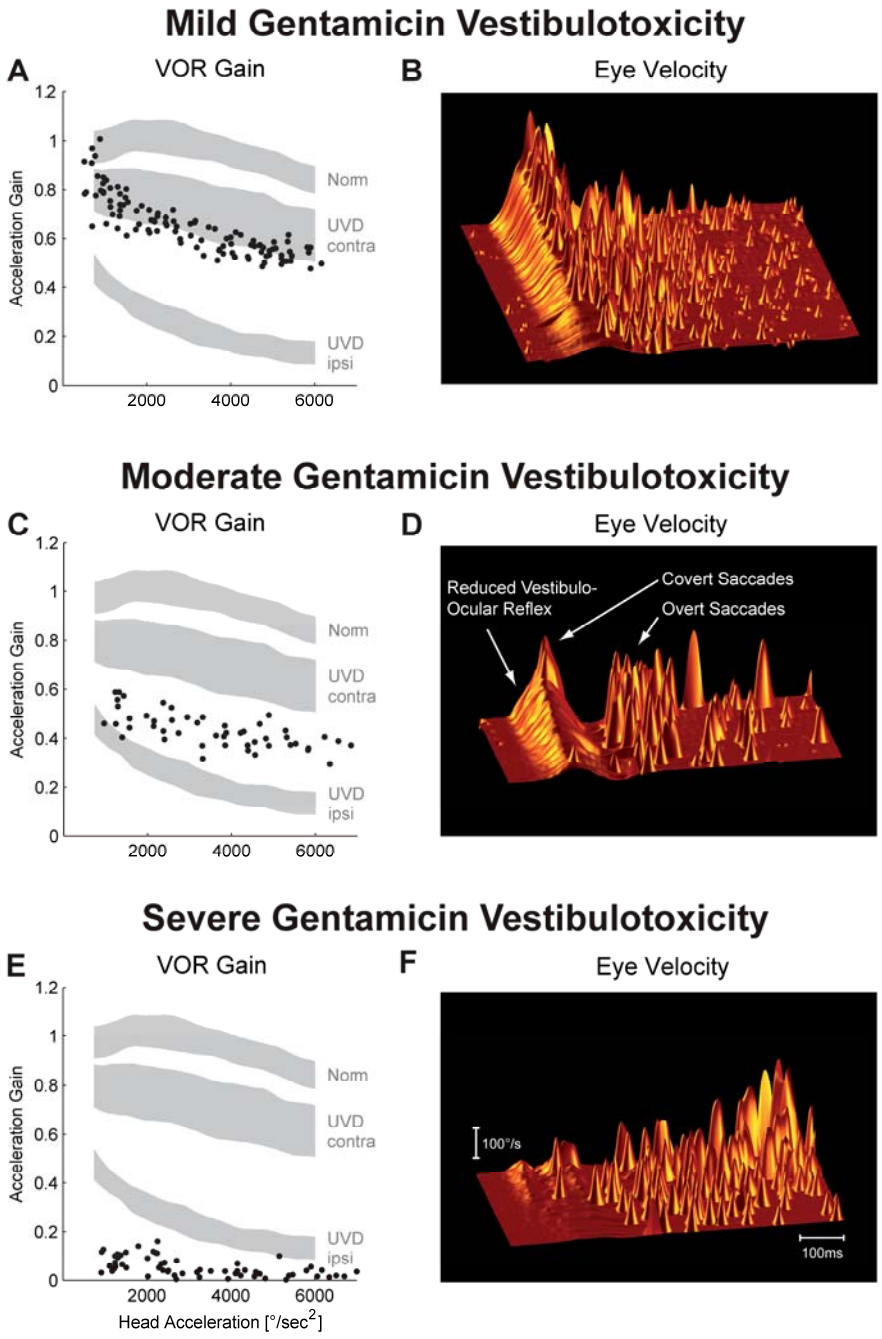
Below are the results of a patient who made many covert saccades during the head impulses to his affected ear, whereas his VOR gain for head rotations to his (right) healthy side are only slightly reduced compared to healthy subjects. The VOR gain shows the very clear, consistent difference in VOR gain for the two sides.



The objective measures of the eye movement response by vHIT allows the objective measurement of whether there is recovery of vestibular function after acute vestibular neuritis. Below is data from a patient who was measured at the acute stage of unilateral vestibular neuritis (with many overt saccades and a low VOR gain (open circles)). However when measured again at testing some weeks later (Recovered) there is clear objective evidence that the VOR gain has increased substantially (closed circles) and there are very few saccades.



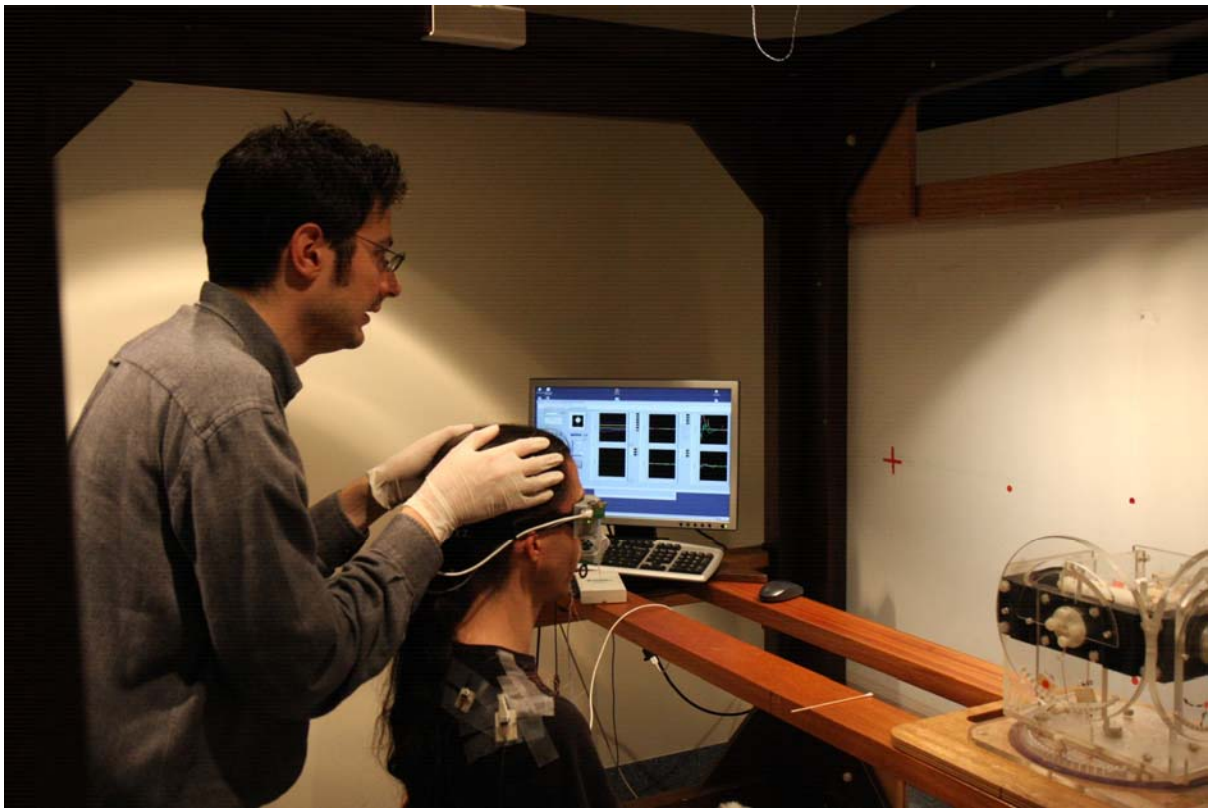
The following examples are search coil recordings of three different patients with varying degrees of progressive loss of vestibular function following systemic gentamicin toxicity, showing the decreased eye velocity response and the overt saccades after the head impulse.



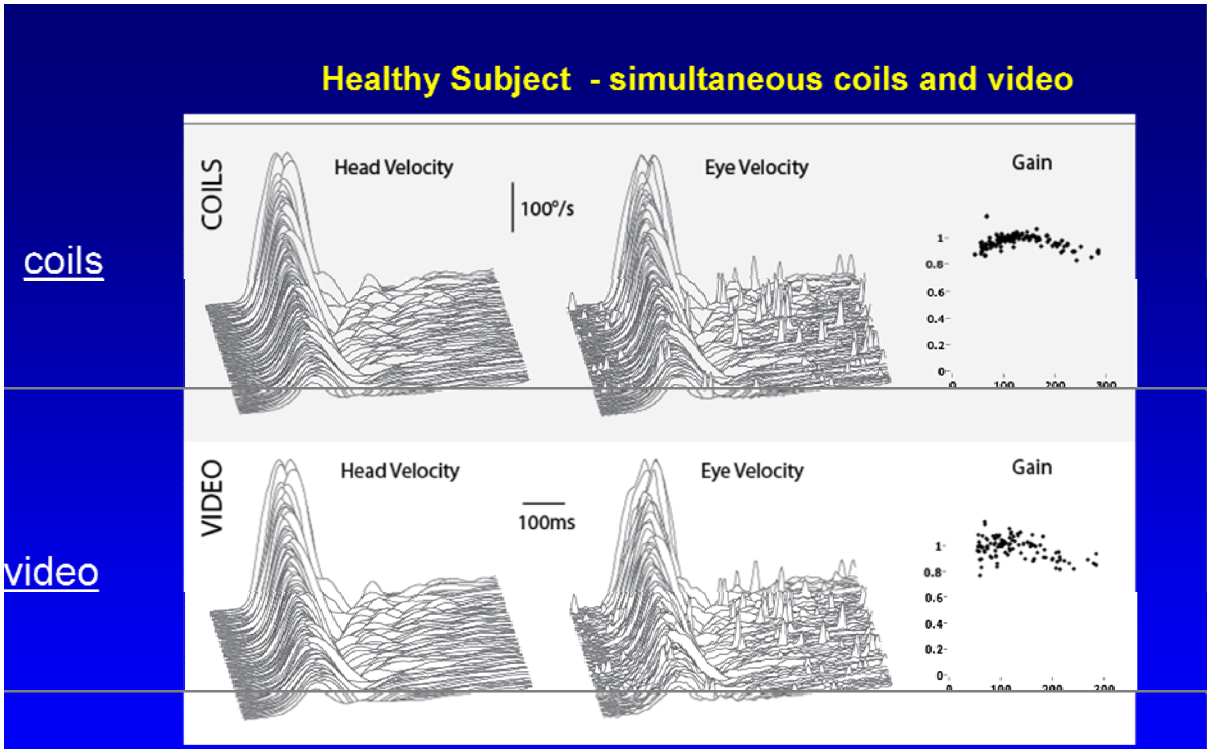
In the final case of severe gentamicin vestibulotoxicity there is almost no corrective eye movement during the head movement, so the VOR gain is close to zero, but there are many overt saccades after the end of the impulse.

Validation of vHIT

It is important to emphasize that the accuracy of the vHIT method has been carefully checked by simultaneous measures of eye movements during head rotations by two entirely independent systems (search coils and vHIT) measuring the stimuli and response of healthy subject and patients. This was done for 8 healthy subjects and 8 patients with various known vestibular losses. This is the way the simultaneous search coil and video measures were done (clinician Konrad Weber; subject Hamish MacDougall).



The evidence of this direct comparison was published in a peer-reviewed very high ranking journal in the field (*Neurology* -- MacDougall et al 2009) and it shows that the VHIT method and search coils gave very closely similar results – there were no significant differences in VOR gain, and the similarity of the eye velocity responses, as measured by the concordance correlation coefficient between the eye-velocity records for vHIT and for search coils was very high – meaning they both gave the same answer. The figure shows just how similar the measurements with search coils and video are.



Contraindications and Challenges vHIT cannot be used on everyone. Some people have very stiff necks or cannot relax their neck muscles sufficiently for the clinician to give them an unpredictable head rotation. In individuals who have previous neck trauma it is not advisable to carry out this rapid head turn. Blinks can be a problem – the patient must be asked to keep their eyes wide open during each head turn, and to try to keep looking at the fixation target. The patient must understand the instructions and attempt to maintain fixation. The goggles must be tightly fixed to the head, and the clinician's hands must be well away from the goggles and the goggle strap.

Why does vHIT detect semicircular canal loss? In a healthy subject a head rotation to the left activates receptors in the left horizontal semicircular canal, so the nerve fibres from the left canal generate nerve impulses which cause both eyes to rotate smoothly so that both eyes exactly compensate, or correct for, the head movement. So both eyes stay looking at the target during the passive unpredictable head movement. But if the person has a loss or deficiency in the ***left*** horizontal canal system, then the neural drive to the eyes will not be enough to drive the eyes to correct perfectly for a ***leftward*** head movement. So the eyes will move with the head and the result will be that at the end of the head movement the eyes will have been dragged off target and the patient will have to make a corrective saccade to get back onto the target. That is the *overt saccade* which the clinician sees. Obviously a right side canal deficit will cause a loss for rightward head movements. If just one side is affected and the other side is healthy then the corrective saccade will only occur for head rotations towards the affected side. That patient has a unilateral vestibular loss. This is the most frequent kind of deficit. When both sides are affected, the patient makes corrective saccades for both directions of head rotation – they have bilateral vestibular loss.

Summary of video HIT Advantages The vHIT method has now been in use for 18months by Dr Leonardo Manzari in the MSA Clinic at Cassino, Italy, as well as at two other locations (Sydney and Zurich), and the results show how extremely useful it is. The vHIT method provides ***objective*** measures of the eye-velocity response to the head-velocity stimulus, and shows the VOR gain for the two directions of rotation. It shows the presence of both overt

and covert saccades and has the very large advantage of being objective – these records of the eye movement responses and the VOR gain provide the hard objective evidence about the adequacy of semicircular canal function which clinicians require.

==========

Q & A

- ***Why is it necessary for the clinician to move the patients head? Why can't we just get the patient to turn their own head while they are looking at the target spot?***

Firstly that sounds a very easy thing to do but it isn't. Some people, even very intelligent people, just cannot do that task at all, try as they may! But more importantly, if the patient moves their own head they can voluntarily generate a corrective eye movement at the same time as they cause their head to move. Just as they can voluntarily control their own head movement, so they can voluntarily control their own eye movements. Active voluntary control of head movement and eye movement by the patient just does not provide the probe of the inner ear function which we get so well if the patient's head is turned in unpredictable directions by the clinician. We have found that patients doing the head impulse voluntarily (by actively turning their head) quickly learn to make an early saccade during the active head movement which is very very hard to detect – another version of the covert saccade. So the clinician cannot see any saccade even though the vestibular system may be non-functional.

- ***Why measure eye movements to test inner ear balance function? The patient's problem is in their ear so why not measure ear function rather than eye function?***

The answer is because the eye movement response to a head turn is a very sensitive indicator or tool or probe of just how well the balance mechanism of the inner ear is functioning. There are very strong fast neural projections from the inner ear to the eye muscles.

- ***Can we give the stimuli regularly (using a metronome)? and why is it necessary to randomize the direction of head turn?***

If each impulse is given in a very regular fashion (e.g. 3 seconds between each impulse and always alternating directions) the patient can quickly learn to either blink just at the start of the head turn, or generate a covert saccade, so the clinician misses seeing any deficit. The test should not be given at regular intervals – the timing when each head turn is delivered should be random. Unpredictable directions and unpredictable intervals minimize the chance of learning affecting the test results.

- ***How many impulses?***

We normally aim to get about 20 impulses for each direction. Although the very first impulses usually tell the whole story and the rest just confirm that story and give the clinician greater confidence in the results. In some patients it is difficult to deliver 20 impulses in each direction – the patient may have a stiff neck or not be able to totally relax their neck muscles. There is no absolute minimum, it really depends on the quality of the responses on each trial. If there is any doubt the stimulus and measurement is very easy and takes only an extra few minutes to give more. With a small number of impulses the calculation of VOR gain becomes less reliable because there are so few values.

- ***How big should the head movement be? (insert video into this section)***

Even very small head turns can be very valuable in showing a loss of function. The important thing is not how large the head turn is but ***how abruptly it starts.*** It should be an abrupt start, not through a big angle, but it should start abruptly.
If the head is turned through a small angle, slowly, then there is no need for the patient to make any corrective eye movement at all and so they don't and the clinician does not detect the deficit which may be there.

- ***Why do we have this vestibulo-ocular response?***

The corrective eye movement response is used to provide stable vision during the head movements of walking, running, driving and all the normal activities we get up to. This very simple eye movement response is an indicator of the function of one part of the balance system of the inner ear.

- ***Can we suppress this response?***

Healthy people can almost totally override the vestibulo-ocular response. For example if you are reading a book on a bus going around a corner - you want to keep your eyes on the text rather than having them being driven off the page by the vestibular input automatically correcting for the angular turn. It is abilities like that overriding (also called VOR suppression) which are an unnoticed but essential part of everyday living which make clinical testing of vestibular function more complex than testing hearing. In hearing there is such a simple output – do you hear that sound? and sounds cannot be suppressed or "shut out" in any way analogous to the way the vestibular information can be. But by restricting our measurements to just the start of these brief unpredictable head turns we can selectively probe the function of the semicircular canals since it takes a little time for that VOR suppression to work

- ***Is the patient's understanding of the instructions important?***

It is *VITAL* that the patient understand the instructions – that the patient has to keep looking at the fixed target – to try to keep their gaze stable and not to blink during this unpredictable movement. They ***must not*** try to "help" the clinician by looking ahead, or looking where their head is going - they must not turn their eyes with their head.

- ***Why has it taken so long to develop this system?***

Because we need very fast cameras which were very small and lightweight which can be comfortably be fitted to the head in goggles which have minimal slip.

- ***What is the worst kind of error with this system?***

Slip of the goggles. If the camera slips on the head then it appears as if the eye has moved relative to the camera A real eye movement and the movement of the camera relative to a fixed eye both generate the same effect at the camera - the image of the pupil of the eye moves across the camera sensor plane. This is the worst artifact. We want to measure real eye movements relative to a fixed camera, not artifactual eye movements. To avoid camera slippage, the camera should be tightly fitted to the head (the test only takes about 5 minutes so it will not cause discomfort for too long). But if it is not tight enough you might as well not do the test. The operator's hands must be well away from the goggles and the goggle strap.

- ***How do we maintain control over the stimulus?***

In clinical testing of hearing, precisely controlled stimuli are presented through calibrated headphones and the patient's responses are measured. In clinical testing of the vestibular system, this kind of presentation of controlled stimuli is just not feasible. Instead we present vestibular stimuli which are not well controlled – a head turn by a clinician can vary enormously from one trial to the next – but we rely on the fact that ***we measure the stimulus exactly each time and relate each response to that stimulus.*** In some respects this is an improvement over testing of hearing since we actually **measure** what the stimulus is on each and every trial, rather than assuming that a calibration check taken a few weeks before guarantees the stimulus value.

Conclusion

This has been a simple introduction. I have focused on the behaviour – the head turn stimulus and the eye movement response. But of course we would like to know about just how the head turn is detected by the receptors in the semicircular canals and how it is transformed into neural signals and how those neural signals result in the eye movement response, and how something like VOR suppression can occur. Those matters are taken up in the companion chapters.

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B-ENT, 2008, **4**, Suppl. 8, 23-25

Principle of the head impulse (thrust) test or Halmagyi head thrust test (HHTT)

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Key-words. Vestibular test; semicircular canal; head impulse test; head thrust test

The head impulse or head thrust test was first described by Halmagyi and Curthoys in 1988.[1] It has acquired an increasingly important place in the clinical examination of the vertigo patient. It detects severe unilateral loss of semicircular canal (SCC) function clinically; it is more sensitive and specific than the traditional Romberg and similar tests; and it is particularly important in the emergency unit, where it can distinguish between vestibular neuritis and cerebellar infarction, which can both generate similar symptoms suggesting an initial attack of severe acute vertigo. The result of the head thrust test is definitely normal in a patient with a cerebellar infarction but abnormal in a patient with vestibular neuritis.

General physiological background: the push-pull principle of the vestibulo-ocular reflex

The peripheral vestibular sensors transmit motion to the brain through frequency encoding. Like FM radios, our brains continuously receive 'frequency modulated' signals. A normal resting discharge rate of approximately 90 spikes per second is modulated such that any increase in this rate corresponds to excitation and a decrease to inhibition. The polarisation of the hair cells in the horizontal semi-circular canal is such that deflection of the stereocilia in the cupula towards the kinocilium (ampullo- or utriculopetal) results in hair cell depolarisation and the activity of the primary afferent neurons therefore increases. Deflection of the stereocilia away from the kinocilium (ampullo- or utriculofugal) results in hair cell hyperpolarisation and decreased primary afferent neuron activity.

The orientation of the left and right semi-circular canals in the head is such that any movement always induces an antagonistic response in both canals. Horizontal head movements in the yaw plane are an example. During rightward head rotation, the endolymph in the lateral semi-circular canals on both sides lags behind, bending the cupula of the right SCC towards the vestibulum (ampullo- or utriculopetal) and simultaneously deflecting the cupula of the left SCC away from the vestibulum (ampullo- or utriculofugal). A key difference is the polarisation of the hair cells. Indeed, since the the hair cells in the right and left canals are implanted in opposing directions (in a mirror image fashion), the deflection on the "leading" right side induces the movement of the stereocilia towards the kinocilium, whereas the movement of the stereocilia is away from the kinocilium in the opposing, "following" ear. As a result of this "push-pull principle", the activity of right lateral SCC primary afferent neurons increases, and, at the same time, the activity of left lateral SCC primary neurons decreases with respect to the normal resting discharge rate.

The activity of the lateral SCC primary afferent neurons is modulated by horizontal head rotation. The firing rate increases in the leading ear (the ear towards the movement is directed) and decreases in the following ear. This is the push-pull principle of the VOR.

The right medial vestibular nucleus in the brainstem receives an increased input from the right lateral SCC primary neurons (no crossing). This excites the activity of type I secondary vestibular neurons. These excitatory neurons drive the leftward compensatory eye movements of the VOR, to ensure gaze stabilisation. However, commissural disinhibition from the left lateral SCC primary neurons also contributes to the excitation of the type 1 neurons. Both excitation of the right SCC and disinhibition of the left SCC are therefore needed for an optimal VOR.

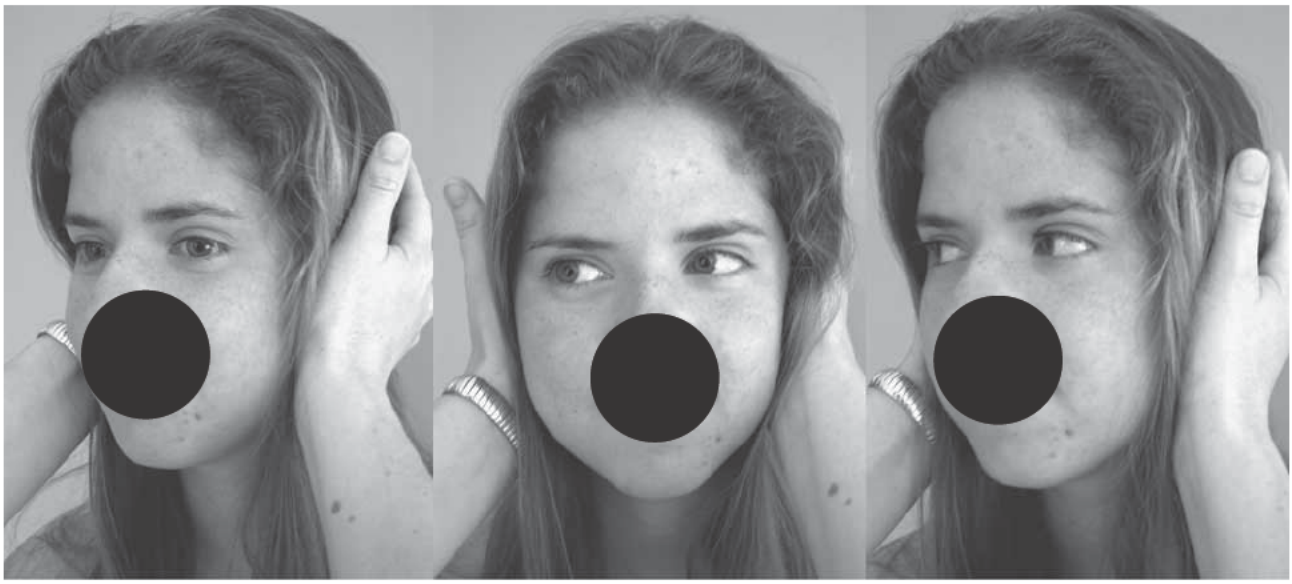

Figure 1
Left: the clinician holds the head of the subject firmly and turns it briskly to the left. Centre: After the rotation to the left, the subject maintains the gaze on the distant fixation point, *i.e.*, the eyes stay stable in space.
Right: After abrupt rotation to her right, the subject moves her eyes with her head and loses the target. A refixation is necessary to fixate the point again (not shown). The side towards the gaze fixation is lost is the deficit side, *i.e.*, the patient's right.

Head thrust test

The head thrust test is primarily based on the fact that inhibition of primary and secondary vestibular neurons cannot produce fewer than 0 spikes per second. Excitation can drive the discharge rate from 90 to 300 or more spikes per second. So when the healthy side is excited for a high acceleration head movement, the healthy side will generate the larger part of the VOR, since the disinhibition of the ipsilateral type-1 neurons by the contralateral SCC contributes relatively little to the VOR. Passive head impulses or thrusts should be typically rapid but with a small amplitude (± 20 degrees). Their velocity ranges up to 180 deg/s but high acceleration is particularly important (3000-4000 deg/s2). They have to be unpredictable since the patient very quickly learns to anticipate and this reduces the sensitivity of the test to a considerable extent. The examiner should therefore thrust the head of the patient firmly from left to right at random and from right to left a little later, *i.e.*, not immediately. The starting position should be such that the patient's head is turned slightly past the midline, and it should then be thrust just past the midline to the opposite side. Here, amplitude is low but acceleration can be considerable. This test demands some training, particularly with respect to the positioning of the hands on the side of the head and holding the head firmly. The instruction to the patient is to fix on a point in the distance behind the examiner.

When the subject's head is turned to the side of the lesion, the VOR is deficient and the eyes will move with the head so that they no longer fix on the point in the distance. The patient therefore needs a refixation saccade just after the thrust. When the head impulse is in the direction of the healthy side, the VOR will maintain the target on the fovea and no refixation saccade will be needed.

The head-thrust test is positive for the side that causes the refixation saccade upon thrust (Figure 1)

It is not only the lateral SCC that can be examined – this is, in a sense, a clinical approximation of the caloric test – but also the other SCC. Here, the patient's head must be thrust in the RALP or LARP planes (Right Anterior – Left Posterior or Left Anterior – Right Posterior SCC).[2]

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ORIGINAL ARTICLE

Comparison of Head Thrust Test With Head Autorotation Test Reveals That the Vestibulo-ocular Reflex Is Enhanced During Voluntary Head Movements

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Objectives: To compare 2 clinical tests of vestibular function, the head autorotation test (HART) and the head thrust test (HTT), and to determine why they give disparate results in patients with known unilateral vestibular deficiency (UVD) due to labyrinthectomy.

Methods: We used scleral coils to measure the horizontal (yaw) vestibulo-ocular reflex (VOR) in 5 healthy human subjects and in 11 patients who underwent labyrinthectomy. We used 2 paradigms. Using HART, subjects visually fixated a target during self-generated, swept-frequency, sinusoidal, horizontal head rotations. Using HTT, patients fixated the target during horizontal head thrusts delivered randomly in direction and time.

Results: In subjects without UVD, eye movements were almost perfectly compensatory for both paradigms. In subjects with UVD, VOR gain for ipsilesional head thrusts was low for both paradigms, but significantly ($P<.001$) higher (less abnormal) for HART (0.60±0.13) than for HTT (0.14±0.13). Contralesional gain was reduced for both, to 0.64±0.20 for HART and to 0.57±0.17 for HTT. Because ipsilesional and contralesional gains were not statistically different for HART ($P=.69$), comparison of VOR gains for half-cycle responses to the HART stimulus could not reliably identify the side of the known lesion. In contrast, HTT consistently identified the side of the lesion for all subjects with UVD. To investigate whether preprogramming contributes to the boost in VOR as measured by HART, we compared the gain and response delay of eye movements during actively self-generated and passively received head thrusts. For subjects without UVD, response delays were shorter for active (6±1 milliseconds) than for passive (12±1 milliseconds) HTT. For ipsilesional rotations of subjects with UVD, active HTT yielded a significantly higher gain (0.44±0.20) ($P<.001$) and a shorter delay (15±6 milliseconds) ($P<.001$) than did passive HTT (0.14±0.13 and 37±15 milliseconds, respectively). Contralesional test results revealed a similar performance boost for active head movements. Data are given as mean±SD.

Conclusion: When comparison of half-cycle gains is used to identify the lesion side, self-generated predictable head movement paradigms, such as HART and active HTT, are less accurate than passive HTT in the characterization of UVD, in part because preprogramming can augment the VOR during voluntary head movements.

Arch Otolaryngol Head Neck Surg. 2002;128:1044-1054

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DIZZINESS IS the ninth most common reason adults visit a primary care physician, and it affects approximately 90 million Americans.[1-4] Identification of the side, affected semicircular canals, and degree of unilateral vestibular deficiency (UVD) is an important goal in the examination of patients experiencing dizziness.

Two high-frequency rotational stimulus paradigms—the head thrust test (HTT) and the head autorotation test (HART)—are in wide use for assessing angular vestibulo-ocular reflex (VOR) function, yet have not been directly compared.

Researchers[3-8] have advocated HTT, in which high-acceleration impulsive head rotations are delivered in the plane of one pair of semicircular canals while a subject attempts to maintain visual fixation on a distant target. Implied by the second law of Ewald,[9] such head rotations elicit an asymmetric response in patients with UVD by maximally stimulating the neural pathway arising from one semicircular canal crista while maximally inhibiting or silencing the pathway from the coplanar canal. The approach has proved sensitive for the detection of vestibular dysfunction in subjects with known UVD.[6,10]

A second paradigm, HART, uses self-generated, swept-frequency, sinusoidal head rotations performed while the subject visually fixates a distant earth-fixed target. Fourier analysis of system gain and phase, along with formulaic measures of response asymmetry, are applied to the measured head and eye movements. Researchers[11-16] have reported extensively on this approach, and several commercially available vestibular testing systems are based on this paradigm.

We sought to compare the ability of HTT with that of HART to identify dysfunction in subjects with known UVD after surgical labyrinthectomy. There are 3 differences between these 2 stimulus paradigms that could contribute to a difference in measured VOR for a given patient. First, HTT stimuli are passive (ie, delivered by the examiner), while during HART, the head movement is active (ie, self-generated). Second, HTT stimuli are presented unpredictably in time and direction, while the sinusoidal frequency–sweep head movement during HART is predictable cycle by cycle. Third, HTT stimuli are often of higher acceleration and wider spectral bandwidth than are HART stimuli, because of some subjects' inability to make rapid, self-generated, sinusoidal head movements.

We hypothesized that during active predictable head movements, subjects with UVD can augment apparent VOR performance by using information about the intended/expected head movement to complement deficient vestibular function and guide compensatory eye movements. Such an effect would be expected to reduce the sensitivity of tests like HART that rely on self-generated head movement stimuli for detecting vestibular hypofunction. We tested these predictions by comparing VOR gains measured for subjects with and without UVD during HART and HTT, and by comparing subjects' responses to active and passive impulsive head rotations.

METHODS

SUBJECTS

We studied 5 human subjects without UVD and 11 with UVD due to labyrinthectomy. Subjects without UVD ranged in age from 32 to 55 years and were free of vestibular and ocular disease, except for wearing corrective lenses. Subjects with UVD ranged in age from 48 to 72 years, and the time from labyrinthectomy ranged from 3 to 120 months. All had undergone vestibular rehabilitation therapy postoperatively. The indication for labyrinthectomy was vestibular schwannoma or meningioma in 6 subjects and unilateral Meniere disease in 5. The side of labyrinthectomy was the left for 6 subjects with UVD and the right for 5. All subjects gave written informed consent, and the experimental procedures were approved by the Joint Committee on Clinical Investigation, The Johns Hopkins University School of Medicine, Baltimore, Md.

EXPERIMENTAL TECHNIQUE

Head and eye movements were recorded in 3 dimensions using magnetic search coils embedded in contact lenses and in a bite block. The instrumentation and technique have been described in detail elsewhere.[17] Eye and bite block angular positions were sampled at 500 Hz. The resulting signals were low-pass filtered with a single-pole analog filter with a 3-dB bandwidth of 100 Hz. Each subject was tested while seated upright and centered within a uniform magnetic field, with the interpupillary line parallel to the horizon and the Frankfort line (from the top of the tragus to the infraorbital foramen) in the plane of head rotation. All rotational stimuli were in the horizontal (yaw) plane. During each trial, the room was completely dark except for a target light-emitting diode positioned 1.24 m anterior to the center of the head, at the same elevation as the pupils. All subjects were tested more than 20 minutes after removal of eyeglasses.

Each experiment began with the subject's head held rigidly at the starting position via connection of the bite block to a bar, while the subject performed calibration tasks. The bar was then removed, and all subsequent trials were performed with the head free to move during a stimulus, then returning to the starting position.

For HART, subjects attempted to visually fixate the target while sinusoidally shaking their heads horizontally (ie, rotating about an earth-vertical axis through the center of the cervical spine) at maximum tolerated velocity in time with a metronome swept logarithmically in frequency from 1 to 6 Hz for 20 seconds. Results from 3 trials were averaged.

For HTT, subjects fixated the same target during brief (100-200–millisecond) rotations in the earth-horizontal plane, at high acceleration (3000°-5000°/s^2) starting from a complete stop. Head velocity reached 25° to 75°/s at 40 milliseconds into the movement and 150° to 300°/s peak velocity by 100 to 120 milliseconds. The final position was 10° to 15° from center. For passive HTT (pHTT), stimuli were delivered manually at an unpredictable onset time and in a randomly varied direction, starting from center. The subject received no cues about the direction or time of an impending passive head thrust. For active HTT (aHTT), the subject generated the head thrust voluntarily. Only head movements reaching greater than 100°/s within the first 60 milliseconds were included in the analysis. A typical trial lasted about 3 minutes and included about 20 to 40 head thrusts to each side.

ANALYSIS

To facilitate comparison between eye and head angular velocities, both were analyzed in 3 dimensions in the head frame of reference. Horizontal rotational components were extracted and compared.[18] All results reported are for the horizontal components of rotation only. Angular velocity and acceleration were computed from angular position signals using a 30-Hz, band-limited, 10-element, linear phase differentiating digital filter designed using computer software (MATLAB; The Mathworks, Inc, Cambridge, Mass) and applied in zero-delay fashion.

We quantified the eye movement responses in terms of velocity gain and response delay. Gain was defined as the angular velocity of the eye divided by the angular velocity of the head at the instant head velocity crossed to above 120°/s, with the eye velocity inverted so that a gain of +1.0 denotes an ideal compensatory response. Using the method of Tabak et al,[7] we defined response delay as the time between the onsets of head and eye movements, where onset was defined as the time at which velocity crossed a threshold set at 2°/s plus 8 times the root-mean-square velocity in the 100 milliseconds preceding the stimulus.[8] We used 1- or 2-tailed *t* tests to compare gain and response delay distributions between experimental groups, with $P<.05$ considered significant. Data are given as mean±SD unless otherwise indicated.

RESULTS

HEAD AUTOROTATION TESTING

Figure 1 shows HART results for a healthy 33-year-old man without

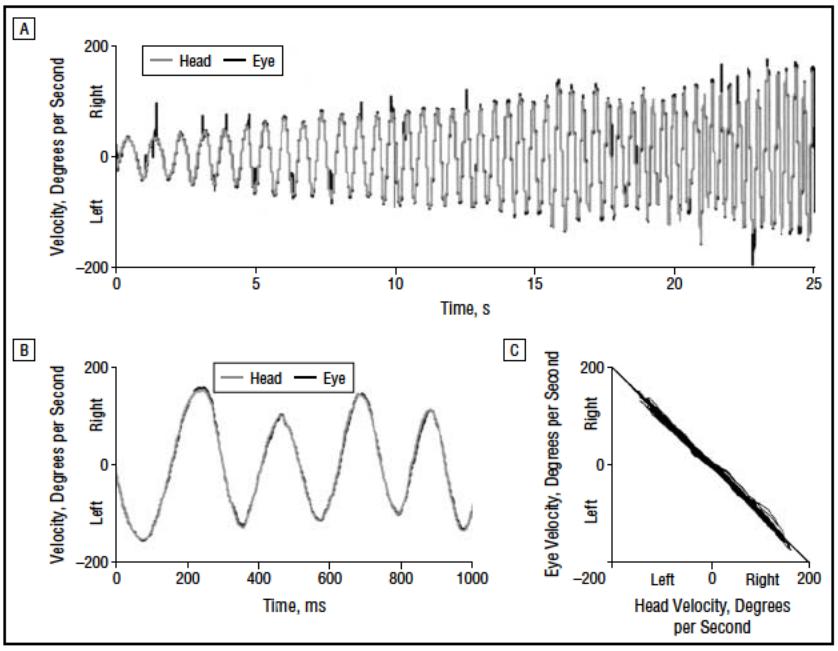

Figure 1. Horizontal head autorotation test results of a 33-year-old man without a unilateral vestibular deficiency. A, Head and eye velocity vs time, with the eye trace inverted for comparison with the head trace. (All similar figures use the same convention.) The eye velocity trace overlies the head trace almost exactly. In the trial shown, head velocity ranged from 40°/s at 1 Hz to 180°/s at 6 to 8 Hz. B, Expanded view of a portion of the trial. Eye velocity traces are nearly identical to head velocity traces. C, Eye velocity vs head velocity after removal of saccades. Data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect vestibulo-ocular reflex.

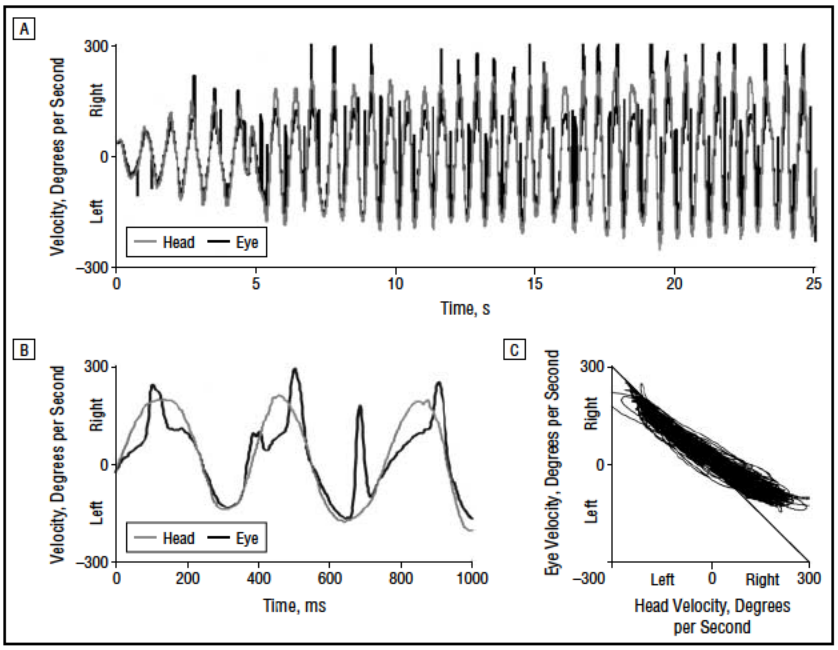

Figure 2. Head autorotation test results of a 63-year-old man 5 months after right translabyrinthine vestibular schwannoma resection. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity. Contralesional (leftward) head turns elicit a moderately degraded response, with a gain of 0.87±0.13 (mean±SD). Ipsilesional responses are more abnormal, with decreased velocity gain, increased retinal slip errors of up to 100°/s, and large-amplitude saccadelike catch-up eye movements.

UVD. In the trial shown, head velocity ranged from 40°/s at 1 Hz to approximately 180°/s at 6 to 8 Hz (Figure 1A). Eye velocity traces (inverted for comparison with head velocity) are nearly identical to head velocity traces, to within less than the 4°/s of retinal image slip for which visual acuity begins to degrade (Figure 1B).[19] When plotted as eye velocity vs head velocity, these data lie almost exactly along a $y=-x$ line, consistent with a nearly perfect VOR (Figure 1C). This subject without UVD has rightward and leftward VOR velocity gains of 1.0±0.05 and 1.0±0.02, respectively.

Figure 2 shows HART results for a symptomatically well-compensated 63-year-old man who had undergone right translabyrinthine vestibular schwannoma resection and postoperative vestibular rehabilitation 5 months before testing. For this subject, the response during head turns toward the contralesional left side (Figure 2A and B) is somewhat degraded, but not significantly ($P=.07$) different from normal (gain, 0.87±0.13). During rightward head turns, there is a significantly ($P<.001$) abnormal response, with a velocity gain of 0.43±0.19 and retinal slip errors of up to 100°/s. Retinal slip not only degrades foveal visual acuity but also accrues to cause the subject to lose target fixation, eliciting large saccadelike corrective eye movements to reacquire the visual target. For this subject, HART results clearly reveal an asymmetry.

Such asymmetries between responses to ipsilesional and contralesional head rotations were not always apparent. **Figure 3** presents the HART responses from a 68-year-old man tested 38 months after undergoing surgery resulting in left UVD. At maximum effort, this patient achieved slower peak head velocity than the subject shown in Figure 2. The slow-phase components of the response to ipsilesional rotations were not markedly different from those observed in the contralesional half cycles. However, a gaze-correcting saccadic eye movement was often observed in the ipsilesional responses.

Figure 4A and B shows a summary of VOR gains measured using HART in 5 healthy subjects and in 11 subjects with UVD, respectively. The VOR gains for subjects without UVD are segregated into rightward and leftward head movements and illustrated separately as a check of test reliability. The VOR gains for subjects without UVD are 0.95±0.13 and 1.03±0.11 for rightward and leftward head rotations, respectively. There was no significant difference between right and left VOR gains

(P=.13), and the combined data set was insignificantly different from 1.0 (P=.64) (95% confidence interval, 0.93-1.05). The HART gains for subjects with UVD were segregated into ipsilesional and contralesional directions. Four findings were notable. First, the ipsilesional gain of 0.60±0.13 was significantly lower than normal (P<.001). Second, there was a wide distribution of ipsilesional gains for this sample population, ranging from 0.2 to 1.0. Third, the contralesional gain of 0.64±0.20 was also significantly decreased from normal (P<.001). Finally, HART gains were not significantly different for ipsilesional and contralesional rotations in these subjects with UVD and, therefore, could not reliably indicate the side of the known lesion (P=.69).

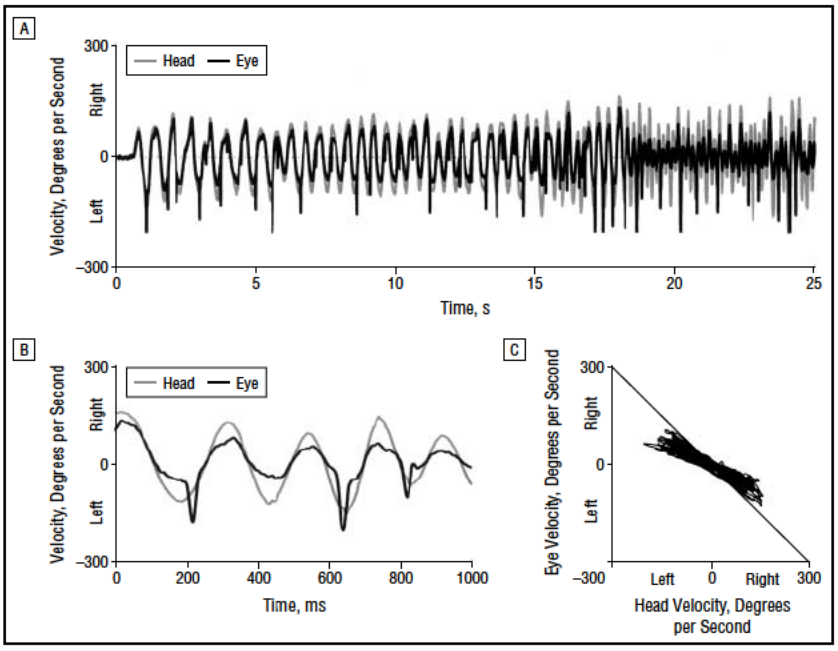

Figure 3. Head autorotation test results of a 68-year-old man 38 months after surgery for a left unilateral vestibular deficiency. A, Head and eye (inverted) velocity vs time. B, Expanded view of a portion of the trial. C, Eye velocity vs head velocity after saccade removal. Saccadelike catch-up eye movements are directed toward the lesion side, but slow-phase eye movements show little asymmetry.

PASSIVE HTT

Passive HTT was performed on 4 of the subjects without UVD and on all 11 subjects with UVD who had been studied using HART. **Figure 5**A shows head velocity and concurrent eye velocity traces for 2 representative head thrusts from the same subject without UVD who was described in Figure 1. The high-acceleration head rotation transients reach a peak velocity of greater than 200°/s within 80 to

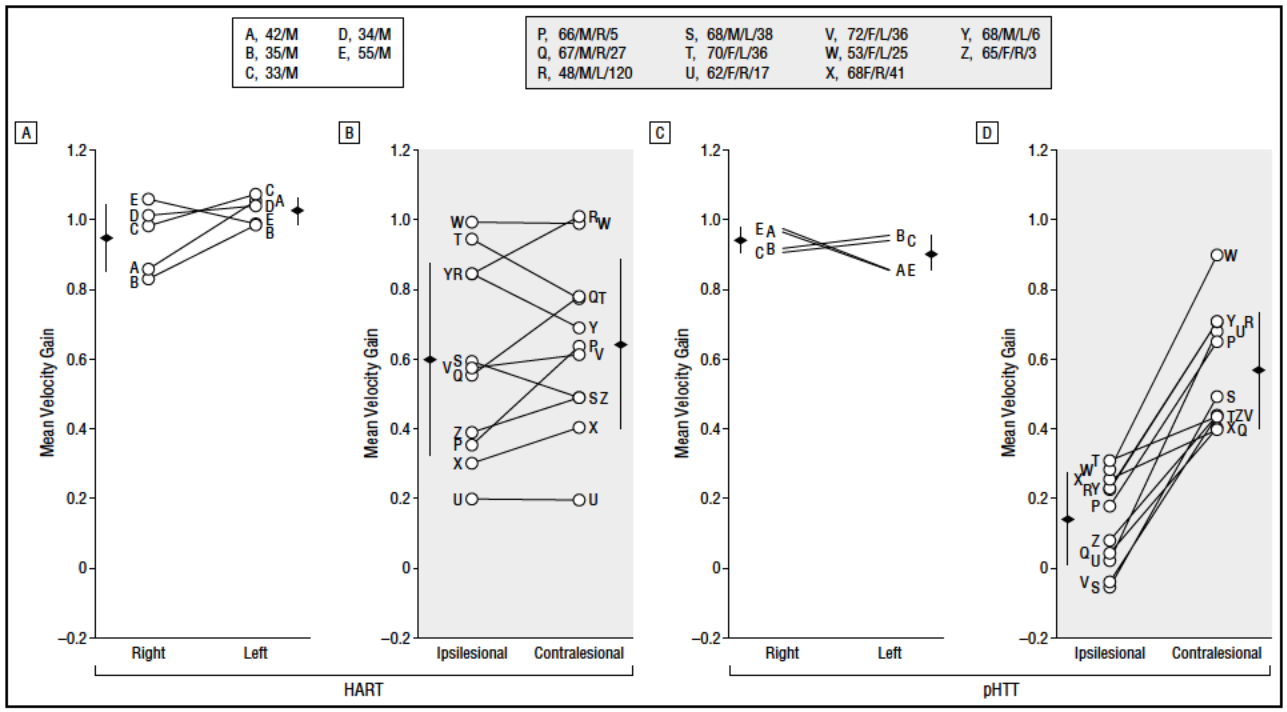

Figure 4. The head autorotation test (HART) gains measured in 5 healthy subjects (A) and in 11 subjects with unilateral vestibular deficiency (UVD) (B) are shown. A, The mean vestibulo-ocular reflex (VOR) gain for subjects without UVD is not significantly (P=.64) different from 1.0 for rightward and leftward head rotations. B, For this population of subjects with UVD, ipsilesional and contralesional HART gain distributions were not significantly different from each other (P=.69). The VOR gains for passive head thrust test (pHTT) in 4 subjects without UVD (C) and in 11 subjects with UVD (D) are also shown. C, Right and left pHTT gains of subjects without UVD are close to 1.0 and not significantly different from each other (P=.27). D, The gains of subjects with UVD were decreased for ipsilesional and contralesional head rotations, with a marked asymmetry between ipsilesional and contralesional gains for every patient tested. For A and C, the key provides subject age (in years)/sex (M indicates male; F, female); for B and D, subject age (in years)/sex/side (R indicates right; L, left)/time since labyrinthectomy (in months). Bars represent mean±SD.

100 milliseconds and are tracked accurately by compensatory eye movements, with a slight overshoot following the peak of head velocity. Figure 5B shows multiple head thrust stimuli and the corresponding eye movement responses for the first 80 milliseconds of each. Peak head accelerations ranged from approximately 3000° to 5000°/s^2. Although there is a slight early mismatch of eye and head traces because of VOR latency, retinal slip is kept below 5°/s throughout most of each trace. In Figure 5C, a plot of eye velocity vs head velocity reveals that all traces lie close to a $y=-x$ line, corresponding to nearly perfect performance. Rightward and leftward gains for this subject were 1.00±0.04 and 0.99±0.06, respectively. Subjects without UVD never complained of losing sight of the target during testing.

In contrast, **Figure 6** shows pHTT responses for the 68-year-old man with left UVD for whom HART data were described in Figure 3. In Figure 6A, a rightward head thrust is tracked fairly well by the eye, although the VOR gain is less than normal and a small catch-up saccade-like movement is required to correct the final eye position after the head movement ends. For a leftward head thrust, the VOR response decrease is more pronounced, as are the prominent catch-up eye movements. The second and third catch-up movements occur after the head stops moving; these are the corrective eye movements an observer can detect when using HTT as part of the physical examination. Figure 6B shows the first 80 milliseconds of multiple stimuli for this subject. For rightward head rotations, the eye tracks the head fairly well; however, the initial VOR-mediated eye movement response for leftward head thrusts is of low amplitude. Figure 6C reveals an asymmetry in the eye movement responses to contralesional and ipsilesional head thrusts, corresponding to the measured VOR gains of 0.50±0.15 and 0.06±0.09, respectively.

Figure 4C and D shows a summary of pHTT VOR gains for 4 subjects without UVD and 11 subjects with UVD, respectively. (The same subjects were used for pHTT and HART, except for 1 subject without UVD who was tested only with HART.) Right and left pHTT responses of subjects without UVD are shown separately in Figure 4C. Rightward (0.94±0.04) and leftward (0.90±0.05) pHTT gains were not significantly different ($P=.27$) for subjects without UVD. For subjects with UVD (Figure 4D), gains were decreased for ipsilesional and contralesional head rotations. In contrast to the HART VOR gains for the same population of subjects, there was a marked asymmetry between ipsilesional (0.14±0.13) and contralesional (0.58±0.17) gains. For every patient with UVD tested, ipsilateral gains were significantly lower than contralesional gains ($P<.001$). Ipsilesional and contralesional gains were each significantly different from normal ($P<.001$).

Comparison of HART and pHTT results reveals that although the ipsilateral VOR gains are reduced for the HART paradigm applied to subjects with UVD, a similar reduction in contralesional gains was such that HART could not reliably distinguish the side of the known lesion. In contrast, there was a marked and consistent asymmetry in the VOR gains of subjects with UVD measured using the pHTT paradigm, with the ipsilateral gains close to 0 and the contralesional gains close to gains measured using HART.

aHTT VS pHTT

Gain

To better identify why the VOR is apparently enhanced when measured by the HART paradigm, we compared VOR gains measured in subjects with and without UVD using head thrusts that were either passively and unpredictably received by the subjects as in the usual application of the test (pHTT) or actively generated by the subjects (aHTT). Analysis was limited to active and passive head movements that were similar in mean peak velocity, peak acceleration, and spectral content.

Figure 7 shows the first 80 milliseconds of head movements and corresponding eye movement responses to aHTT for the subject with left UVD shown in Figures 3 and 6. Whereas the ipsilesional pHTT response was essentially nonexistent for the first 80 milliseconds after stimulus onset (Figure 6B), the aHTT response VOR gain is enhanced, to 0.34±0.06, from 0.06±0.09 (Figure 7A). The contralesional VOR gain is also increased, to 0.74±0.05, from 0.50±0.15. Similarly, the time from stimulus onset to threshold response is shorter for aHTT than for pHTT.

Figure 8 shows pHTT and aHTT VOR gains for 3 subjects without UVD and 10 subjects with UVD (the same subjects used for HART except for those who did not generate aHTT head movements similar to those of pHTT). For subjects without UVD (Figure 8A), responses to aHTT and pHTT were not significantly different from each other or from 1.00 ($P>.05$ for all). For subjects with UVD, there was a significant ($P<.001$) increase in apparent ipsilesional VOR gain when measured using the aHTT paradigm, to 0.44±0.20 (from 0.14±0.13 for pHTT) (Figure 8B). The contralesional VOR gain also increased significantly ($P=.004$) under aHTT conditions, to 0.81±0.14 (from 0.58±0.17 for pHTT) (Figure 8C). This level was not significantly different from the VOR gains measured in subjects without UVD using pHTT ($P=.08$).

Response Delay

To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset and to compute a response delay. **Figure 9** shows pHTT and aHTT VOR response delays for 4 subjects without UVD and 11 subjects with UVD. For subjects without UVD (Figure 9A), the response delay was 11.0±3.3 milliseconds for pHTT and slightly, but significantly, lower at 5.0±1.8 milliseconds for aHTT ($P=.006$). For subjects with UVD, the response delay for ipsilesional head thrusts was significantly shorter for aHTT (19±16 milliseconds) than for pHTT (42±17 milliseconds) ($P=.006$) (Figure 9B). The response delay in subjects with UVD for contralesional head thrusts was also shorter for aHTT (11.0±5.1 milliseconds) than for pHTT

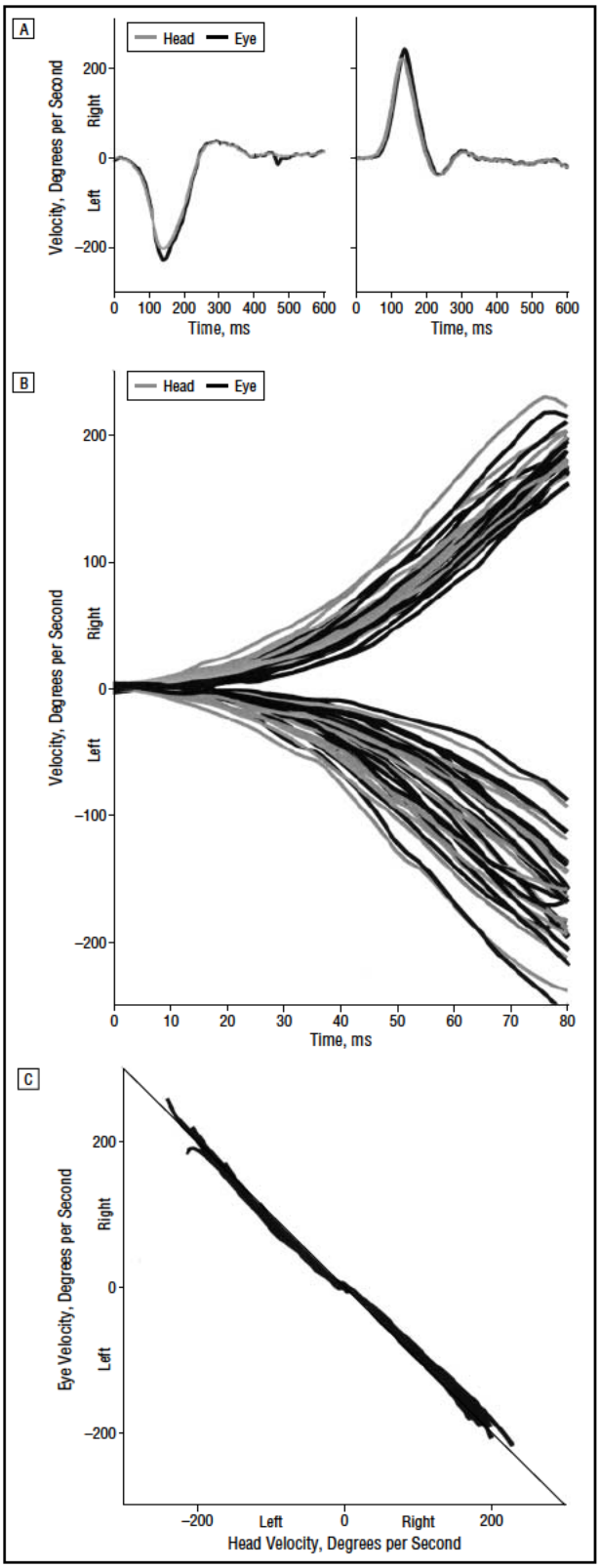

Figure 5. Passive head thrust test results for a healthy subject without a unilateral vestibular deficiency (UVD) (same subject as in Figure 1). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject has nearly perfect overlap of head and eye traces. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Traces nearly overlie each other for this subject without UVD. C, Eye velocity vs head velocity. Data lie closely along a $y=-x$ line, consistent with a near-perfect vestibulo-ocular reflex.

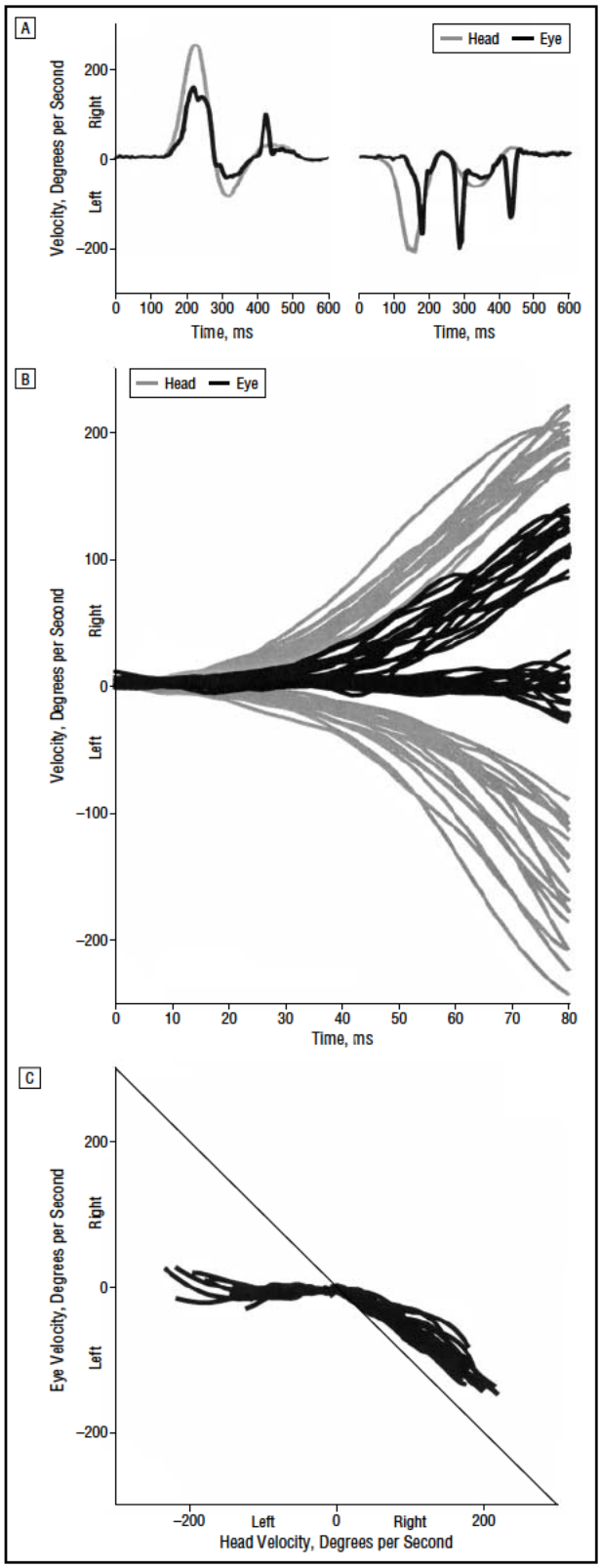

Figure 6. Passive head thrust test results in a subject with a unilateral vestibular deficiency (UVD) (same subject as in Figure 3). A, Head and eye (inverted) velocities during passive head thrusts to the right, then to the left (after returning to center [not shown]). This subject with a left UVD follows rightward head movement moderately well, but tracks leftward head thrusts poorly. B, Head and eye (inverted) velocities during the first 80 milliseconds of multiple passive head thrust trials. Response is delayed and of decreased magnitude, mildly for rightward head movements and dramatically for head movements toward the lesion side. C, Eye velocity vs head velocity for the first 80 milliseconds of responses.

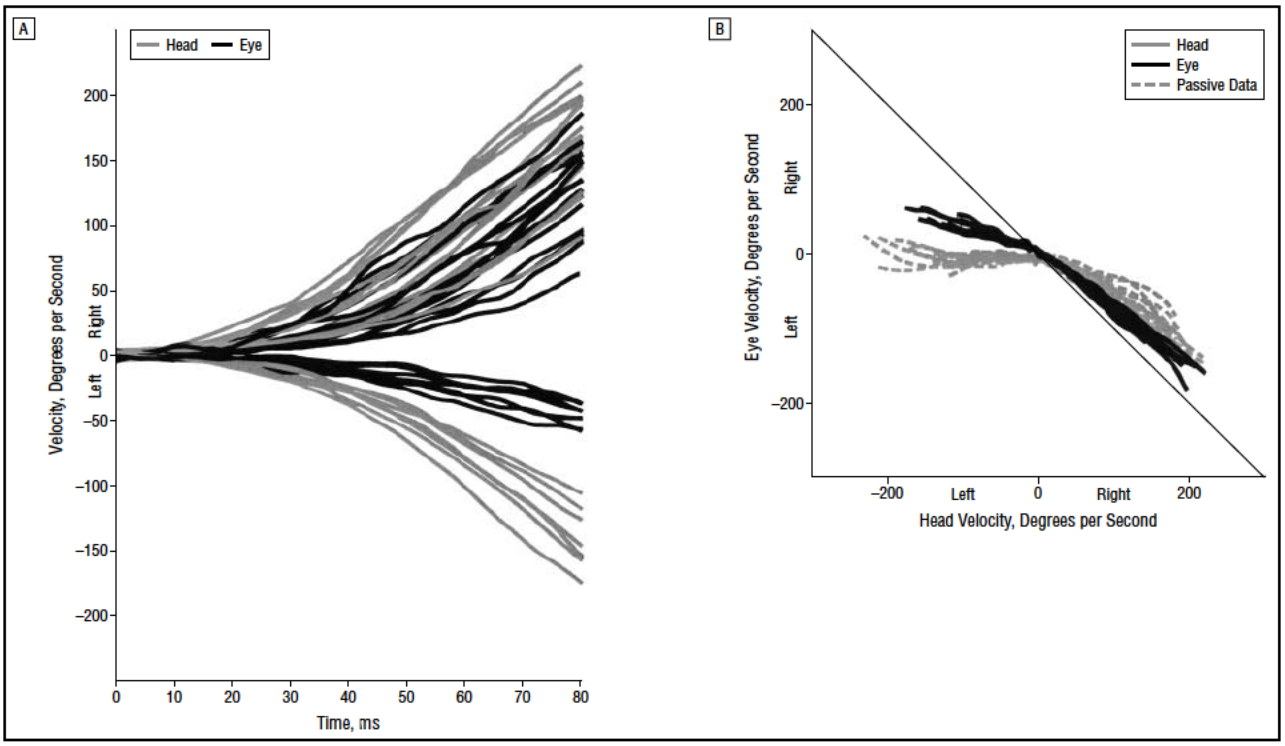

Figure 7. Active head thrust test results in a subject with a left unilateral vestibular deficiency (same subject for whom passive data are shown in Figure 6). A, Head and eye (inverted) velocities during the first 80 milliseconds of multiple active head thrust trials. B, Eye velocity vs head velocity. Although still not normal, the active eye movement response is significantly better than for passive trials, with shorter response delay and higher gains.

(15.0±4.5 milliseconds), but the difference was not significant ($P = .07$) (Figure 9C).

COMMENT

CLINICAL TESTS FOR IDENTIFICATION OF UVD

Traditional caloric and rotary chair tests for the identification of UVD fail to test the vestibular system using physiologically appropriate stimuli over the range of frequencies and accelerations for which the system apparently evolved. Caloric testing has the advantage of being strictly unilateral, but uses an unnatural stimulus (a thermal gradient) that, as measured by the eye movements it elicits, is the equivalent of a low frequency (0.025 Hz) and low amplitude (approximately 50°/s) head rotation. The caloric response can be altered by anatomic changes in the external ear canal and by individual variation in temporal bone anatomic features, and it only evaluates 1 of the 5 vestibular receptors (the lateral semicircular canal). Rotary chair vestibular testing is more physiologic; however, at the lower frequencies and velocities typically used in clinical vestibular laboratories, rotary chair tests are insensitive in the identification of chronic total unilateral vestibular hypofunction.[20,21] The sensitivity of rotary chair testing can be improved by comparison of responses to steps of acceleration with the head up and head down.[22]

In contrast, high-frequency and high-acceleration rotational stimuli unmask the inherent asymmetry in the vestibular system, as described by the second law of Ewald[9]—that the excitatory responses of vestibular pathway neurons encode motion over a larger dynamic range than do inhibitory responses. The eye movements elicited by such stimuli are principally due to excitation of the vestibular pathway arising from the canal ipsilateral to the direction of head acceleration and are, therefore, sensitive to a unilateral peripheral vestibular loss in that canal.

There are 2 high-frequency rotational stimuli in clinical use, pHTT and HART. The main objective of this study was to compare these 2 stimuli for the identification of unilateral vestibular hypofunction. The pHTT is a single, passive (operator delivered), unpredictable, high-acceleration (2000°-4000°/s^2), low amplitude (15°-30°) head rotation in the direction of a single semicircular canal. The HART is a continuous, active (self-generated), sinusoidal head rotation that begins slowly and becomes faster, covering a frequency range of about 1 to 6 Hz. During each test, subjects are required to fixate a visual target in front of them, and their ability to maintain visual fixation is accepted as a measure of VOR function. However, because HART is actively generated, non-VOR processes, such as predictive eye movements driven by an "efference copy" neural representation of the intended head movement, could contribute to maintaining gaze stability. As a result, VOR function could seem artificially enhanced, making HART a less sensitive and less accurate test of UVD.

We tested 11 patients who had undergone surgical labyrinthectomy and compared their responses with those of 5 subjects without UVD. The VOR gains for subjects without UVD using pHTT were insignificantly different from an ideal gain of 1.0, consistent with findings from previous studies.[23] For subjects with UVD, the VOR gains of 0.14±0.13 for ipsilesional head

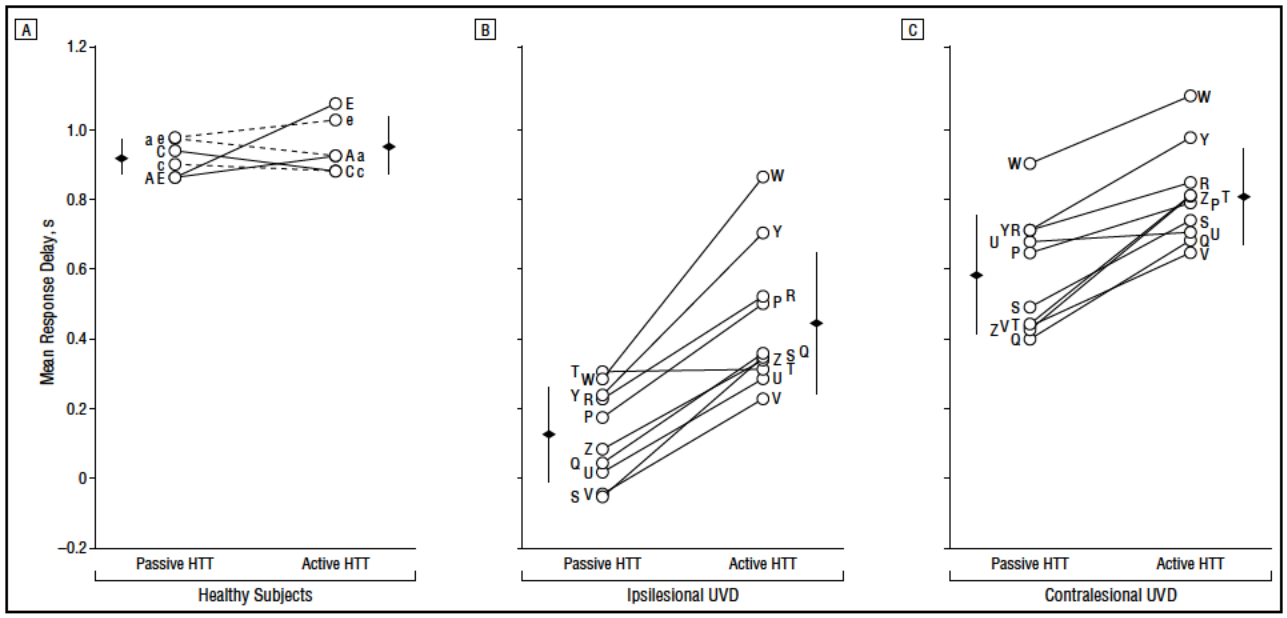

Figure 8. The vestibulo-ocular reflex (VOR) gains for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For healthy subjects without UVD, responses to active and passive HTT were not significantly different from each other or from 1.00 ($P>.05$ for all). In B, active HTT ipsilesional VOR gain is significantly ($P<.001$) higher than for passive HTT. In C, contralesional UVD VOR gain is also significantly ($P=.004$) closer to normal for active than for passive HTT. Bars represent mean±SD.

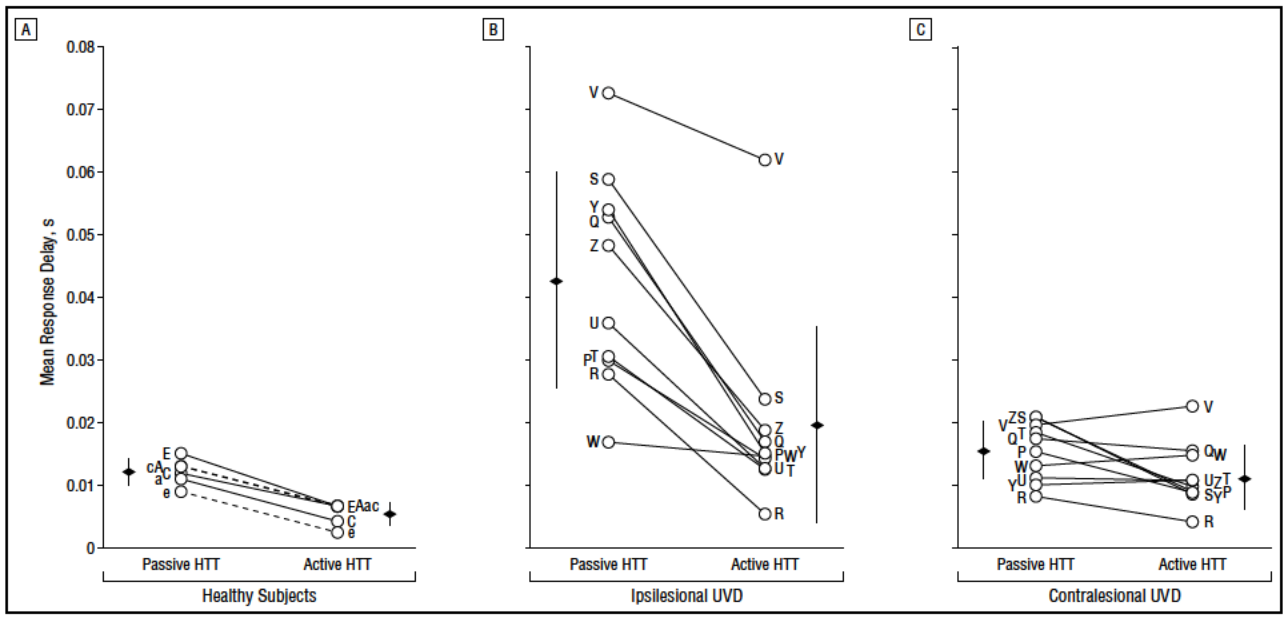

Figure 9. The response delays for passive and active head thrust test (HTT) of 3 subjects without unilateral vestibular deficiency (UVD) (A) and 10 subjects with UVD (B and C). The keys in Figure 4 provide characteristics of each subject. In A, lowercase letters indicate leftward thrusts; uppercase letters, rightward thrusts. For subjects without UVD, the response delay was shorter for active than for passive HTT ($P=.006$. In B, for ipsilesional stimuli, response delay was significantly shorter for active HTT than for passive HTT ($P=.006$). In C, the response delay for contralesional stimuli was not significantly different ($P=.07$) between passive and active HTT. Bars represent mean±SD.

thrusts and 0.58±0.17 for contralesional head thrusts were significantly different, and the side with the lesion was clearly identified by pHTT in each patient, consistent with previous studies.[6,24] For HART, the VOR gain in subjects without UVD was essentially perfect, as one would expect. Surprisingly, however, although some subjects with UVD showed obvious asymmetry of VOR half-cycle gains on HART, there was no significant difference between the ipsilesional and contralesional VOR gains over the population with UVD ($P=.69$).

Other features of the response to the actively generated HART stimulus did provide an indication of the side of unilateral vestibular hypofunction. The occurrence of rapid, gaze-correcting, saccadic eye movements during head movements toward the side of the lesion, when they occur, is a reliable indication of the side of UVD. A bias velocity in the eye movement response during the HART stimulus (a direct current shift in the eye velocity trace toward the side of the lesion) pro-

vides another such indication. Head autorotation is, therefore, a useful paradigm, but comparison of VOR gains from half-cycle analysis of HART responses is not reliable for the identification of the side of UVD.

MECHANISMS FOR IMPROVED RESPONSES TO ACTIVE HEAD ROTATIONS

There are 2 main differences between HART and pHTT stimuli that might explain the improvement in VOR gain of patients with UVD for ipsilesional head movements during HART. First, the peak velocities and accelerations generated by the examiner in pHTT are greater than those that some patients are able to generate during HART. This constraint on stimulus intensity may limit HART's ability to discern unilateral weakness in such patients. In contrast, the stimulus used for pHTT is generated by the examiner and reaches a peak acceleration of 3000°/s^2 and a peak velocity of 250°/s. Although these head movements have low amplitude in displacement (10°-15°) and are well tolerated by patients, they are sufficient to elicit excitation-inhibition asymmetry and, thus, selectively probe the function of the excited canal.

A second difference is that the pHTT is passive, transient, and unpredictable, whereas the HART is a self-generated, sinusoidal, predictable stimulus. Self-generated, sinusoidal, steady-state rotations could allow for nonvestibular eye movement systems (eg, predictive eye movements, efference copy, and visual-following mechanisms) to contribute to the response. The existence of predictive mechanisms in augmenting vestibular responses is suggested by measures of visual acuity during head movement. Visual acuity is improved during active compared with passive head movements.[25,26] To further investigate these potential effects of self-generated stimuli, we asked patients to perform the HTT themselves, aiming to mimic the speed and amplitude of the operator-delivered head thrusts. Although patients had some difficulty reaching the top speed of operator-delivered stimuli (about 300°/s), they did reach speeds of approximately 200°/s, and we compared responses for active and passive head thrusts of similar peak velocity and acceleration. In subjects without UVD, the VOR gain was already insignificantly different from 1.00 for pHTT, so no significant change was observed for aHTT. For subjects with UVD, however, there was a marked improvement in VOR gain during active head movements, and the boost in VOR gain during active thrusts occurred from the onset of the head thrust, during the initial 20 to 40 milliseconds. The only 2 oculomotor systems with a latency of less than 40 milliseconds are the direct VOR and predictive eye movements. Saccades, the cervico-ocular reflex, and visual-following mechanisms, such as smooth pursuit, all have latencies that are 70 to 150 milliseconds.[27] From previous studies,[6,10] it has been shown that the 3-neuron VOR arc generates the response to pHTT, so the boost in gain during aHTT is, therefore, likely to be a result of predictive eye movements.

To study the effect of prediction, we measured the time between the onset of head rotation and the onset of eye rotation for active and passive head thrusts. We used manually applied passive head thrusts to approximate the usual clinical application of pHTT and the movements our subjects made during aHTT. Manual thrusts do not have an onset sharp enough or an acceleration constant enough to precisely measure VOR latency (estimated at 8.6 milliseconds in subjects without UVD by Collewijn and co-workers[28,29] using a torque-applying helmet in an HTT-type paradigm). To obtain a measure of response timing, we used the method of Tabak et al[8] to define stimulus and response onset times (at which head and eye velocities cross set thresholds) and compute a response delay. This response delay probably does not precisely equal the true synaptic and axonal conduction delay of the VOR, because the finite rate of change of acceleration for manually applied stimuli and responses reduces the precision with which onset times for head and eye movements can be measured. However, this response delay provides a useful measure for comparison of responses to passive and active head thrusts.

For subjects without UVD, the response delay was shorter for active than for passive head thrusts. For subjects with UVD, the response delay during ipsilesional head thrusts was significantly longer for passive thrusts than for active thrusts. A similarly prolonged response delay for passive ipsilesional head rotations has been reported by Tabak et al.[8] The apparent reduction in the response delay to aHTT might be further evidence of preprogrammed eye rotation. Alternatively, some of the apparent difference in response delay may be due to a difference in VOR gain for the first 40 milliseconds of the response. The initial low velocity eye rotation makes it difficult to precisely define the onset of the eye rotation, and could give the appearance of a delayed onset.

TIMING OF CORRECTIVE EYE MOVEMENTS

Further evidence for preprogramming of eye rotations during active head thrusts was the reduced latency of rapid corrective eye movements during active compared with passive head thrusts. To compensate for a deficient VOR, patients with UVD used rapid eye movements to correct accrued gaze error and reacquire visual fixation of the target. During active head thrusts, these corrective movements occurred as early as 60 milliseconds after the onset of the head rotation, significantly earlier than during passive head thrusts (**Figure 10**). Under normal circumstances, the latency of voluntary true saccadic eye movements is 200 milliseconds.[25] This latency can be reduced to 100 milliseconds if the subject is trained in a specific task (so-called express saccades[30]). Tian et al[25] have shown that under pHTT conditions, these rapid eye movements have latencies shorter than those of express saccades. Our data indicate that these latencies can be reduced even further under conditions of active head movement.

CONCLUSIONS

Gain asymmetries on the pHTT reliably detected the presence of

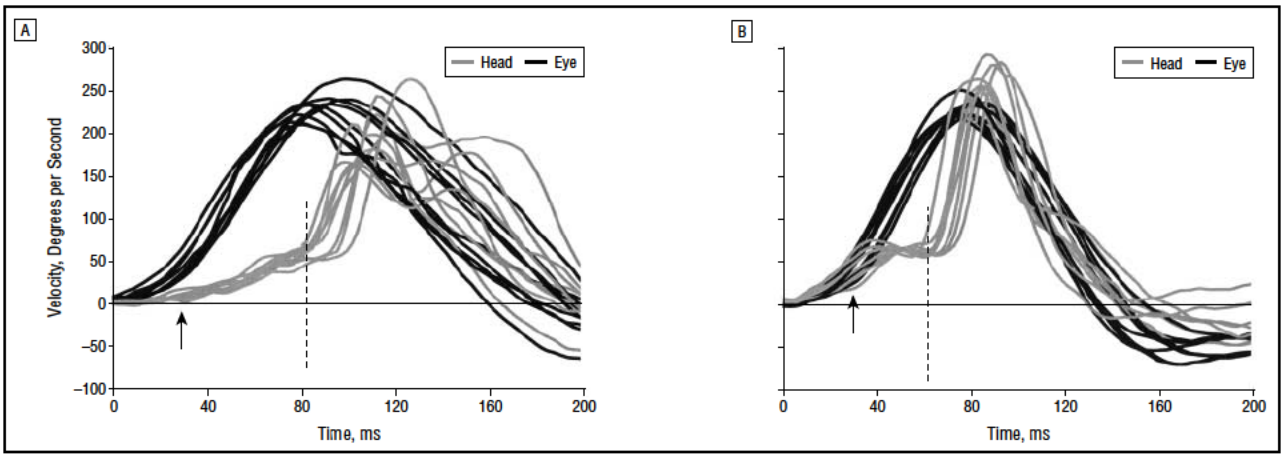

Figure 10. Corrective rapid eye movements during passive (A) and active (B) ipsilesional head thrust testing (HTT) in a 48-year-old subject with a unilateral vestibular deficiency 120 months after left labyrinthectomy (subject R shown in Figures 4, 8, and 9). Head and eye (inverted) velocities are shown. The response to active HTT has a higher initial vestibulo-ocular reflex gain (arrows) and earlier rapid compensatory eye movements (dashed lines).

known unilateral vestibular lesions in all patients with UVD tested, whereas the half-cycle gains measured from HART did not. Relative to pHTT, HART gains overestimated ipsilesional vestibular function and were, therefore, a less accurate indicator of unilateral hypofunction.

For subjects with UVD who are unable to generate high-acceleration sinusoidal head movements, HART may underestimate hypofunction simply because it relies on stimulus accelerations inadequate to silence inhibitory pathways arising in the intact ear. In contrast, the high-acceleration movements of pHTT reliably identify response asymmetry by more selectively probing the function of the excited canal. However, even for head movements of similar acceleration and time course, the measured VOR during self-generated head rotations (aHTT) has significantly higher gain and shorter response delay than does the VOR to passive unpredictable stimuli (pHTT).

Subjects with UVD may use a variety of strategies for improving visual fixation, including preprogrammed eye movements designed to augment the deficient VOR and to compensate for a planned or anticipated head movement. Tests using passive high-acceleration head thrusts delivered unpredictably in time and direction should, therefore, be more sensitive for discerning VOR hypofunction than tests using active and/or predictable stimuli.

Accepted for publication February 18, 2002.

This study was supported by the Garnett Passe and Rodney Williams Memorial Foundation, Parkville, Australia (Dr Cremer); the Royal Australasian College of Physicians, Sydney, New South Wales (Dr Cremer); grants K23 DC00196 (Dr Carey) and R01 DC02390 (Dr Minor) from the National Institutes of Health, Bethesda, Md; and a Johns Hopkins Clinician-Scientist Award (Dr Carey).

We thank Grace C. Y. Peng, PhD, David S. Zee, MD, Adrian G. Lasker, MS, and Dale C. Roberts, MS, for their assistance with data acquisition; and John K. Niparko, MD, and Howard W. Francis, MD, for their referral of subjects.

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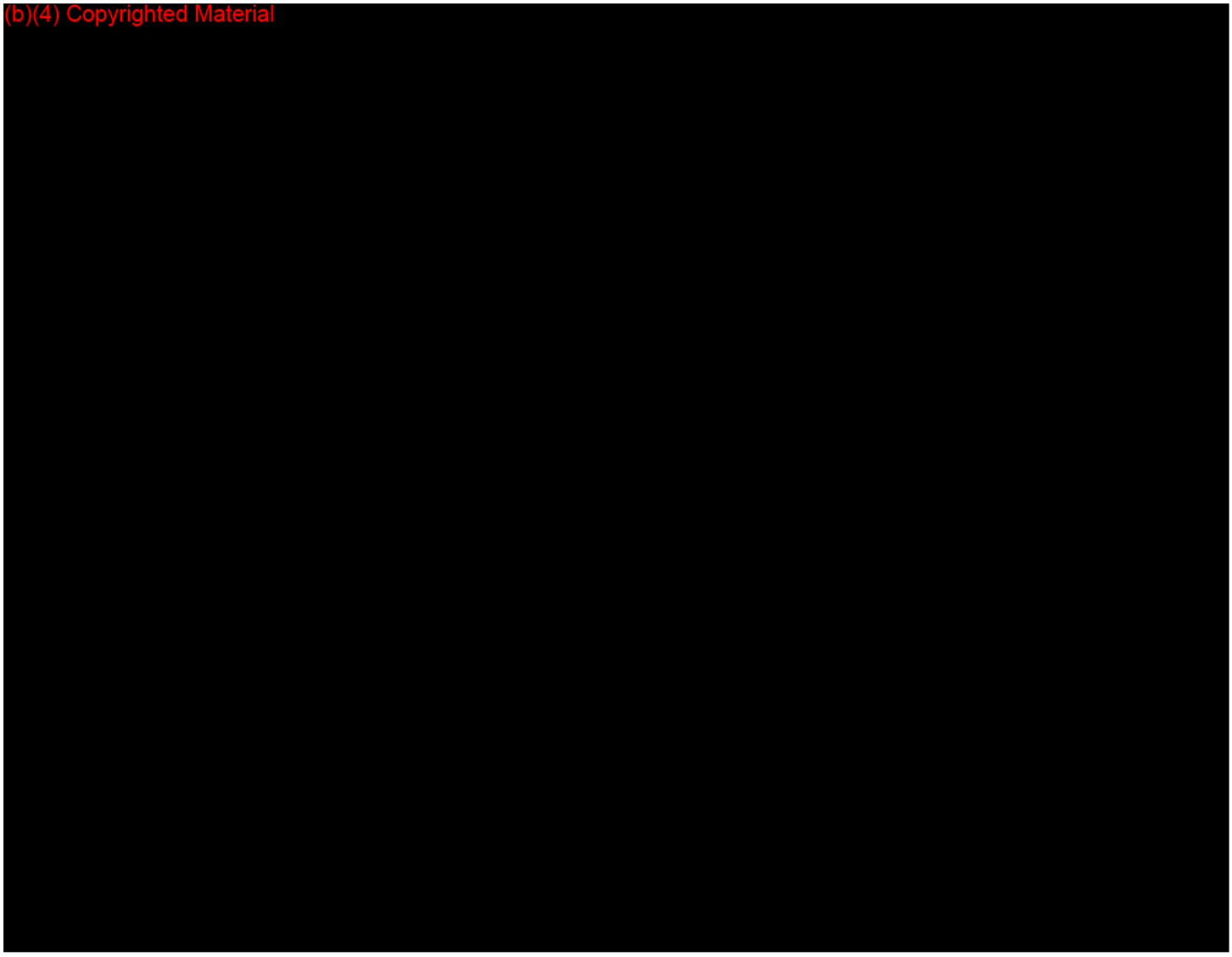
A Clinical Sign of Canal Paresis

G. Michael Halmagyi, MB, BS, Ian S. Curthoys, PhD



* * * COMMUNICATION RESULT REPORT (FEB. 5. 2013 12:46PM) * * *

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E-2) BUSY
E-3) NO ANSWER
E-4) NO FACSIMILE CONNECTION

DEPARTMENT OF HEALTH & HUMAN SERVICES Public Health Service

Food and Drug Administration
10903 New Hampshire Avenue
Document Control Center – WO66-G609
Silver Spring, MD 20993-002

February 1, 2013

GN Otometrics A/S
% Mr. Daniel Kamm, P.E.
Principal Engineer
Kamm & Associates
8870 Ravello Court
Naples, FL 34114

Re: K122550
Trade/Device Name: ICS Impulse
Regulation Number: 21 CFR 882.1460
Regulation Name: Nystagmograph
Regulatory Class: II
Product Code: GWN, LXV
Dated: January 21, 2013
Received: January 23, 2013

Dear Mr. Daniel Kamm:

We have reviewed your Section 510(k) premarket notification of intent to market the device referenced above and have determined the device is substantially equivalent (for the indications for use stated in the enclosure) to legally marketed predicate devices marketed in interstate commerce prior to May 28, 1976, the enactment date of the Medical Device Amendments, or to devices that have been reclassified in accordance with the provisions of the Federal Food, Drug, and Cosmetic Act (Act) that do not require approval of a premarket approval application (PMA). You may, therefore, market the device, subject to the general controls provisions of the Act. The general controls provisions of the Act include requirements for annual registration, listing of devices, good manufacturing practice, labeling, and prohibitions against misbranding and adulteration. Please note: CDRH does not evaluate information related to contract liability warranties. We remind you, however, that device labeling must be truthful and not misleading.

If your device is classified (see above) into either class II (Special Controls) or class III (PMA), it may be subject to additional controls. Existing major regulations affecting your device can be found in the Code of Federal Regulations, Title 21, Parts 800 to 898. In addition, FDA may publish further announcements concerning your device in the Federal Register.

Please be advised that FDA's issuance of a substantial equivalence determination does not mean that FDA has made a determination that your device complies with other requirements of the Act or any Federal statutes and regulations administered by other Federal agencies. You must comply with all the Act's requirements, including, but not limited to: registration and listing (21 CFR Part 807); labeling (21 CFR Part 801); medical device reporting (reporting of medical device-related adverse events) (21 CFR 803); good manufacturing practice requirements as set

Food and Drug Administration
Office of Device Evaluation &
Office of In Vitro Diagnostics

COVER SHEET MEMORANDUM

From: **Rahul Ram**

Subject: **510(k) Traditional - K122550**

To: **The Record**

Please list CTS decision code: **SE**

_ Refused to accept (Note: this is considered the first review cycle, See Screening Checklist http://eroom.fda.gov/eRoomReq/Files/CDRH3/CDRHPremarketNotification510kProgram/0_5631/Screening%20Checklist%207%202%2007.doc)
_ Hold (Additional Information or Telephone Hold).
X Final Decision (SE, SE with Limitations, NSE, Withdrawn, etc.)

Please complete the following for a final clearance decision (i.e., SE, SE with Limitations, etc.):		YES	NO
Indications for Use Pages	*Attach IFU*	Yes	
510(k) Summary /510(k) Statement	*Attach Summary*	Yes	
Truthful and Accurate Statement.	*Must be present for a Final Decision*	Yes	
Is the device Class III? If yes, does firm include Class III Summary?	*Must be present for a Final Decision*		No
Does firm reference standards? **Yes** (If yes, please attach form from http://www.fda.gov/opacom/morechoices/fdaforms/FDA-3654.pdf)		Yes	
Is this a combination product? (Please specify category _____, see http://eroom.fda.gov/eRoomReq/Files/CDRH3/CDRHPremarketNotification510kProgram/0_413b/COMBINATION%20PRODUCT%20ALGORITHM%20(REVISED%203-12-03).DOC			No
Is this a reprocessed single use device? (Guidance for Industry and FDA Staff – MDUFMA - Validation Data in 510(k)s for Reprocessed Single-Use Medical Devices, http://www.fda.gov/cdrh/ode/guidance/1216.html)			No
Is this device intended for pediatric use only?			No
Is this a prescription device? (If both **prescription** & OTC, check both boxes.) **Rx**		Yes	
Is clinical data necessary to support the review of this 510(k)? **Yes** Did the application include a completed FORM FDA 3674, *Certification with Requirements of ClinicalTrials.gov Data Bank*? **Yes** (If not, then applicant must be contacted to obtain completed form.)			No

Does this device include an Animal Tissue Source?	No
All Pediatric Patients age<=21	No Specific Claims
Neonate/Newborn (Birth to 28 days)	No Specific Claims
Infant (29 days -< 2 years old)	No Specific Claims
Child (2 years -< 12 years old)	No Specific Claims
Adolescent (12 years -< 18 years old)	No Specific Claims
Transitional Adolescent A (18 - <21 years old) Special considerations are being given to this group, different from adults age ≥ 21. (different device design or testing, different protocol procedures, etc.)	No Specific Claims
Transitional Adolescent B (18 -<= 21; No special considerations compared to adults => 21 years old)	No Specific Claims
Nanotechnology	No

Is this device subject to Section 522 Postmarket Surveillance? (Postmarket Surveillance Guidance, http://www.fda.gov/cdrh/osb/guidance/316.html)	*Contact OSB.*		No
Is this device subject to the Tracking Regulation? (Medical Device Tracking Guidance, http://www.fda.gov/cdrh/comp/guidance/169.html)	*Contact OC.*		No

Regulation Number	**Class***	**Product Code**
21 CFR 882.1460	II	GWN

(*If unclassified, see 510(k) Staff)

Additional Product Codes: LXV

Digital Signature Concurrence Table	
Reviewer Sign-Off	Rahul K. Ram -S (Affiliate) 2013.02.01 15:43:14 -05'00'
Branch Chief Sign-Off	Quynh T. Hoang 2013.02.01 16:15:00 -05'00'
Division Sign-Off	Joyce M. Whang 2013.02.01 16:36:41 -05'00'

DEPARTMENT OF HEALTH & HUMAN SERVICES
Food and Drug Administration
Center for Devices & Radiological Health

Division of Ophthalmic, Ear, Nose, and Throat Devices
Diagnostic & Surgical Devices Branch
10903 New Hampshire Ave
Silver Spring, MD 20993-0002
(301) 796-6620

SUPPLEMENTAL (S1) PREMARKET NOTIFICATION [510(K)] REVIEW

DATE: **February 1st, 2013**
TO: **RECORD**
FROM: **Rahul Ram**
SUBJECT: **Traditional, K122550/S1**

510(K) HOLDER: GN OTOMETRICS Hoerskaetten 9 Taatrup, DK-2630 Denmark	**OFFICIAL CORRESPONDENT:** Mr. Daniel Kamm Principal Engineer Kamm & Associates 8870 Ravello Court Naples, FL 34114 **Email:** fda.help.now@gmail.com **Phone:** (239) 234-1735 **Fax:** (206) 260-4162
DEVICE TRADE NAME: ICS Impulse (Model 1085)	
DESCRIPTION: Video Nystagmography System	
510(K) DATED DATE: January 21st, 2013	
510(K) RECEIVED DATE: January 23rd, 2013	

APPLICANT-IDENTIFIED PREDICATE DEVICES:

510(K) NUMBER	PRODUCT CODE	DEVICE NAME	510(K) HOLDER
K891008	LXV	Vestibular Ocular Reflex Test Equipment (Vorteq)	Micromedical Technologies
K964325	GWN	Visualeyes: Video Eye Monitor	Micromedical Technologies

RECOMMENDATION:

The submission concerning the **ICS Impulse** requires no additional information in order to proceed with the review and all safety / effectiveness concerns have been resolved. Therefore, I recommend that the device be found **substantially equivalent (SE).**

Regulation Number: **21 CFR 882.1460**
Regulation Name: **Nystagmograph**

Regulatory Class: **Class II**
Product Code: **GWN**
Produce Code Description: **Nystagmograph**
Additional Product Codes: **LXV**

INDICATIONS FOR USE (IFU) STATEMENT: ☒ **Prescription** ☐ **Over-the-Counter**

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

I SUBMISSION REVIEWERS

- Rahul Ram (ODE/DOED/DSDB) – Lead Reviewer
- Lawrence Rodichok (ODE/DNPMD/NSDB) – Consulting Neurological Clinical Reviewer
- Quynh Hoang (ODE/DNPMD/NNDB) – Branch-Level Concurrence

II PURPOSE OF SUBMISSION

As per the original (S0) submission cover letter, the applicant has submitted this 510(k) premarket notification in order to solicit marketing clearance for the **ICS Impulse** (Model 1085). As stated in Exhibit 10 ("Executive Summary") of the original (S0) submission, the device consists primarily of head-mounted video recording goggles and associated software to assess the vestibular-ocular reflex (VOR).

III FORMAT OF REVIEW

To ensure that this review is wholly inclusive of all pertinent information, it contains almost all the information from the review of the original submission (S0)). Please note the following conventions in this review:

1. Updated information as per the current submission (S1) is included as **bold** text (except *italicized* text, which is not bolded), and indicated by the following symbol: "▰".
2. New, S1 Review Comments are **bolded and thick-boxed**.
3. Review Comments from S0 are un-bolded and thin-boxed.
4. Minor edits to S0 text are unannounced.

IV SUBMISSION HISTORY

(August 13th, 2012) K122550/S0, received in FDA
(January 9th, 2013) K122550/S0, hold memorandum conveyed to applicant
(January 23rd, 2013) K122550/S1, received in FDA

V BACKGROUND

A VESTIBULOOCULAR REFLEX (VOR), BRIEF DESCRIPTION

The vestibular-ocular / vestibuloocular reflex (VOR) is one of three vestibular reflexes (three involuntary responses to strong stimulation to the vestibular system (the balance and spatial orientation component of the human nervous system)). The VOR contributes to image stability on the fovea of the retina by effecting involuntary eye movements compensate for head movements (rotational, translational, head tilt with respect to gravity) in directions opposite of those eye movements.

B TESTING

VOR can be evaluated by measuring eye motion and concurrent head movements; **gain** is the ratio of eye velocity to head velocity, and a normal value should be -1 (indicating an equally compensatory eye movement for any head movement). **Video-oculography (VOG)** is a method of concurrently measuring head movements and eye movements using digital video cameras and accelerometers.

C CURRENT DEVICE

The current device, the ICS Impulse, claims to perform video-oculography using infrared digital cameras to record eye movements and goggle-mounted gyroscopes to observe head movements.

VI OVERALL ADMINISTRATIVE REQUIREMENTS

REQUIREMENT	ORIGINAL SUBMISSION (S0) LOCATION
User Fee Cover Sheet (FDA Form 3601)	PAGE 6
CDRH Premarket Review Submission Cover Sheet (FDA Form 3514)	PAGE 7
Cover Letter	PAGE 14
Indications for Use Statement	PAGE 18
510(k) SUMMARY	PAGE 20
Truthful and Accuracy Statement	PAGE 23
Class III Summary or Certification	PAGE 24
Financial Certification or Disclosure Statement	PAGE 25
Clinical Certification Statement (FDA Form 3674)	PAGE 25
Standards Data Report(s) (FDA Form 3654)	PAGE 27 & FULL TEST REPORTS PROVIDED
Executive Summary	PAGE 40

10

S0 REVIEW COMMENT: Administrative requirements appear to have been met.
SECTION RECOMMENDATION: ADEQUATE

VII INDICATIONS FOR USE

The Indications for Use (IFU) statement, as reported in Exhibit 4 (Page 19) of the original (S0) submission, was the following:

> *"The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements."* (Prescription (Rx) Use)

Regarding this IFU, the following comments were noted in the original (S0) review:

S0 REVIEW COMMENT: Based on the above IFU, the following safety / effectiveness concerns are noted:

1. The applicant should validate the ability of the device to measure, record, display and analyze eye/head movements
2. The applicant should provide evidence that this analysis can be used to assess the vestibular-ocular reflex (VOR)

Regarding (1) and (2), please see sections of this review, below, for resolution of these concerns.

3. It is not clear why this device should be limited to patients "with complaints of dizziness, disequilibrium, and vertigo"; this suggests that the device has some clinical utility specific to this subpopulation; the applicant should either provide a justification of this utility or remove this restriction from the IFU

Regarding (3), please see the comments related to Deficiency 1, below.

S1 REVIEW COMMENT: **Regarding (1) & (2), please see sections of this review, below, for resolution of these concerns. Regarding (3), please refer to the discussion immediately below.**

Larry also noted concern (3) as a concern in his original (S0) review:

> **Reviewer's comments: Unless the sponsor can provide data to support accuracy for the stated population "patients with complaints of dizziness, disequilibrium, and vertigo, the indication should be limited to "assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements".**

To that end, Deficiency 1 was conveyed to the applicant in a hold memorandum dated January 9th, 2013 (reproduced below):

Your proposed Indications for Use (IFU) statement reads: "The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo." We do not consider this IFU acceptable since you have not provided adequate clinical data to support the accuracy of the device in evaluating this specific population. Please provide data to support the diagnostic accuracy of your device in this specific population. Alternatively, please consider the following modified IFU statement: "The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements."

In the current (S1) submission, the applicant responds to Deficiency 1 provides a revised IFU statement (Attachment 1) and 510(k) Summary (Attachment 2) in which the IFU statement is the following:

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

S1 REVIEW COMMENT: **The applicant has heeded the recommendation in full of the deficiency and provided an IFU which matches the recommended IFU verbatim. The applicant's response to Deficiency 1 is ADEQUATE.**

A CONSISTENCY THROUGHOUT SUBMISSION

Aside from in Exhibit 4, the applicant provided the original, unmodified, IFU statement in the following locations in the current (S0) submission:

- (Page 10) FDA Form 3514 (CDRH Premarket Review Cover Sheet) → ***Identical***
- (Page 21) 510(k) Summary → ***Identical***
- (Page 22) 510(k) Summary, albeit under heading "Intended Use" → ***Identical***
- (Page 72) Substantial Equivalence Discussion, albeit under heading "Intended Use" → ***Identical***
- (Page 106) Page 5 of User Manual, albeit under heading "Intended Use" → ***Identical***

As is stated above, in response to Deficiency 1, the applicant provided instances of the revised IFU in the IFU statement and the 510(k) Summary. These two instances are entirely consistent with each other.

S1 REVIEW COMMENT: **The instances of the IFU in the 510(k) Summary and IFU statement consistent with one another. This is ADEQUATE.**

B COMPARISON TO PREDICATE DEVICES

There exists no explicit IFU statement for one of the applicant's chosen predicate devices, the VORTEQ (cleared under K891008); the following is the intended use of that device, as stated by the reviewer of that submission (Melpomeni K. Jeffries):

The intended use of VORTEQ is to evaluate the vestibular ocular reflex.

The following is the IFU statement of the applicant's other chosen predicate device, the VisualEyes (cleared under K964325, obtained from IMAGE):

The Eye Monitor is used to observe eye movements from various stimuli used in vestibular diagnostic testing. It allows observation of horizontal, vertical and torsional eye movements. The Eye Monitor is mainly used to visually detect nystagmus positional maneuvers (e.g. Hallpike (e.g. Hallpike positional tests) or caloric tests. The device is especially useful in situations where the operator needs to observe the patient's eyes to distinguish nystagmus from artifacts. These observations are currently done using Fresnel lenses. The eye movements could also be recorded on suitable media (e.g. video tape) to provide documentation for any diagnosis based on observation of the Eye Monitor.

S1 REVIEW COMMENT: **The intended use of the ICS Impulse is the same as those of the predicate devices. The identified predicate devices are ADEQUATE.**

C DECISION-MAKING

- **SAME INDICATIONS FOR USE?**

  NO. The IFU of the current device includes only a subset of the claims of the IFU of the device cleared under K964325.

  ~~• The IFU of the ICS Impulse includes a intended use sub-population that is not included in the IFU statements of the predicate devices~~

- **DO THE DIFFERENCES ALTER THE EFFECT OR RAISE NEW QUESTIONS OF SAFETY AND/OR EFFECTIVENESS?**

  ~~**YES, HOWEVER…** the applicant can validate use of the device in this sub-population using performance testing. The applicant will be instructed to justify this claim.~~

  No. The claims contained in the IFU of the current device match existing claims in the IFU of the device cleared under K964325.

S1 REVIEW COMMENT: **All concerns regarding the IFU have been resolved.**

SECTION RECOMMENDATION: ADEQUATE

VIII TECHNOLOGICAL CHARACTERISTICS

The applicant provided a description of the device in Exhibit 11 ("Device Description") of the original (S0) submission. The following is a graphical depiction of the ICS Impulse, reproduced from Exhibit 11:

Although the applicant did not provide labels to accompany the numbered items in the graphic above, as described in Exhibit 11, the ICS Impulse is a goggles-based device that consists of the following gross physical components:

- Goggle Frame – houses other components
- Infrared LED – illuminates the right eye
- Digital Camera – mounted near the right eye, receives reflected light from the right eye for processing eye movements
- Lens – zero optical power, half-silvered lens in the frame location in front of the right eye that allows the device user to observe visual stimuli, but also reflects light from the eye to the camera
- Gyroscopes – records head movement
- USB/Firewire Interface Box (*labeled as "5"*) – connected, via cable, to the Firewire output of the camera to allow data transmission to the computer
- Calibration Laser

Not depicted is a personal computer on which the OTOSuite Vestibular software is intended to run, which stores and processes the head/eye movement data.

S0 REVIEW COMMENT: Please refer to the "Software" section of this review for more information regarding OTOSuite.

A MATERIALS, ALL DEVICE COMPONENTS

In review of the original (S0) submission, it appeared as though the applicant had not provided a list of the patient contacting materials and assurances that biocompatibility concerns have been mitigated. Accordingly, Deficiency 6 was conveyed to the applicant in the January 9th hold memorandum:

You do not provide a list of the patient-contacting parts of the device and the corresponding materials of which each part is composed. This is important to mitigate concerns regarding biocompatibility. Please provide this list, and evidence that biocompatibility concerns for each patient-contacting part of your device have been mitigated.

In the current submission (S1), the applicant responds to Deficiency 1 with the following:

A complete list of patient contacting materials was supplied in the original submission in Exhibit 15, including the only two materials which contact the patient: The headstrap and the face cushion. See pages 217 through 290. Full biocompatibility test reports were also provided. Also refer to pg 343 #2 of the Medical Device Hazard Questionaire. It is further supported by an additional Risk/Hazard analysis supplied herein as Attachment 6.

S1 REVIEW COMMENT: **The applicant has clarified the materials specified in Exhibit 15 of the original submission are the only patient-contacting materials of the device. Furthermore, given that these materials are short duration-contacting, and intact skin-contacting, the applicant has already provided sufficient evidence that biocompatibility concerns have been met (biocompatibility testing in the original submission). The applicant's response to Deficiency 6 is ADEQUATE.**

B INFRARED LEDS & DIGITAL CAMERA

On Page 64/1854 of the original (S0) submission, in a document titled "Type 1085 ICS Impulse System Specification," the applicant stated in specification number HW-7 that "*two infrared LEDs of 875 nanometers shall be provided to illuminate the patient eye.*" The applicant refers to "Type 1085, Hardware Verification Protocol 0-80-06520" for evidence of meeting this specification.

S0 REVIEW COMMENT: Please refer to the "Performance Testing" section of this review for an evaluation of optical radiation safety hazards associated with this light source.

The applicant stated, on Page 45/1854 of the original submission, that the sampling rate of the camera is 250 Hz for the "Head Impulse Test," and 30, 60, or 120 Hz for "video recording."

Regarding this information, the following comment was noted in the S0 review:

S0 REVIEW COMMENT: The applicant provides no description of the principle of operation of how the LEDs and camera collect information that ultimately allows for eye-tracking. This is important to substantiate performance provided to validate eye-tracking functionality. DEFICIENCY 3 SHOULD BE CONVEYED TO THE APPLICANT.

S1 REVIEW COMMENT: **Please see the "Performance Testing" section of this review, below, for an evaluation of resolution of this concern.**

C GYROSCOPES

On Page 306/1854 of the original submission, the applicant provided a specification for the device, stating that *"the design shall measure angular velocity with a sensitivity of 2mV/deg/s±10% about the X, Y, and Z axis."*

Regarding this information, the following comment was noted in the S0 review:

S0 REVIEW COMMENT: This is NOT ADEQUATE. The applicant should provide the following information:

- Gyroscope measurement range
- Measurement recording frequency
- Angular random walk (to assess variability)

This information is important to substantiate their performance in recording head movements. DEFICIENCY 2 SHALL BE CONVEYED TO THE APPLICANT.

For an evaluation of any performance provided in this regard, please see the "Performance Testing" section of this review.

Accordingly, Deficiency 2 was conveyed to the applicant in the January 9th hold memorandum:

On Page 306/1854, you provide a specification for the device, stating that "the design shall measure angular velocity with a sensitivity of 2mV/deg/s±10% about the X, Y, and Z axis." However, you do not provide any more information regarding the gyroscopes. Because measurements of head movements is a significant factor in the safety and effectiveness of your device, please provide testing protocol and results that demonstrate the ability of the gyroscopes to measure head movements, including the following:

a. Gyroscopes measurement range
b. Measurement recording frequency
c. Angular random walk (to assess variability).

In the current submission (S1), the applicant provides a narrative description of gyroscope specifications, as well as data sheets for the gyroscopes (Attachment 4). The applicant does not provide an angular random walk specification, but rather refers to an algorithm which attempts to minimize inherent noise. Finally, the applicant refers to testing in the Hardware Verification Test Protocol (HW-1), provided in the original submission for testing of the gyroscopes.

S1 REVIEW COMMENT: **This information is clear and raises no new concerns. The response to Deficiency 2 is ADEQUATE.**

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D USB/FIREWIRE INTERFACE BOX

The applicant stated, in the original submission, that the USB/Firewire interface box is intended to connect from the Googles to the computer to display, store, and analyze recorded data.

E CALIBRATION LASER

In the original submission, it appeared as though the applicant had provided little to no information regarding the physical characteristics of the calibration laser:

S0 REVIEW COMMENT: This is NOT ADEQUATE. The applicant should provide a diagrammatic and textual description of the calibration laser, including a principle of operation and rationale for how calibration improves data quality. DEFICIENCY 7 SHALL BE CONVEYED TO THE APPLICANT.

Accordingly, Deficiency 7 was conveyed to the applicant in the January 9th hold memorandum:

You provide no information regarding the characteristics of the calibration laser. Because the calibration laser may affect measurements made by your device, please provide a diagrammatic and textual description of the calibration laser, including a principle of operation and rationale for how calibration improves data quality.

In the current (S1) submission, the applicant references places in the original (S0) submission which describe the calibration laser and provide accompanying testing. Furthermore, the applicant states the following regarding the rationale of calibration:

According to ANSI/ASA S3.45-2009, "Calibration thus consists of an "end-to-end" assessment of the deflection of the pen on a strip-chart recorder, or other graphic output device, in response to the subject's shifts of gaze between small visual targets that are displaced by a specific angular amount." This is the rationale behind why calibration is performed for all measurements of eye movement using a video goggle.

S1 REVIEW COMMENT: **The information referred to from the original (S0) submission and the rationale provided in the narrative response raises no new concerns. The applicant's response to Deficiency 7 is ADEQUATE.**

F DESCRIPTION OF THE DEVICE - SOFTWARE

Level of Concern

On Page 53/1854 of the original submission, the applicant stated the following regarding software Level of Concern:

"The Software provides diagnostic information for evaluation without control or delivery of harmful energy and is considered a Moderate concern device."

However, on Page 291/1854 of the original submission, the applicant stated the following:

"We believe the level of concern is Minor if failures or latent design flaws are unlikely to cause any injury to the patient or operator."

Regarding this inconsistency, the following comment was noted in the S0 review:

S0 REVIEW COMMENT: The applicant should clarify this inconsistency and state the software Level of Concern. Furthermore, the applicant should provide software documentation as per FDA's premarket software guidance (http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089543.htm). When providing this documentation, the applicant should specify the page numbers (i.e., Page X to Page X) for each software section. DEFICIENCY 4 SHOULD BE CONVEYED TO THE APPLICANT.

Accordingly, Deficiency 4 was conveyed to the applicant in the January 9th hold memorandum:

On Page 53/1854 of the submission, you state the following regarding software Level of Concern: "The Software provides diagnostic information for evaluation without control or delivery of harmful energy and is considered a Moderate concern device." However, on Page 291/1854 of the submission, you state "we believe the level of concern is Minor if failures or latent design flaws are unlikely to cause any injury to the patient or operator." Please clarify this inconsistency and state the software Level of Concern. Furthermore, please provide software documentation as per FDA's premarket software guidance: http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089543.htm). When providing this documentation, please specify the page numbers (i.e., Page X to Page X) for each software section.

In the current (S1) submission, the applicant confirms that they intend to designate the software as of Minor Level of Concern.

S1 REVIEW COMMENT: **Based on the applicant's own risk analysis and Level of Concern designations for other, similar devices, the Minor designation is acceptable. Because the applicant has already provided all documentation required for Minor Level of Concern software, the response to Deficiency 4 is ADEQUATE.**

Software Description

In the original submission, it appeared as though the applicant had not provided a standalone description of the software:

S0 REVIEW COMMENT: This is NOT ADEQUATE. Although the applicant provides software specifications, these specifications do not clearly define all functions of the software, or more simply what software programs / modules are running on the personal computer. Therefore, the applicant should provide a list of all software programs / modules part of the device, including an indication of whether each is optional or required for operation and a statement of purpose for each. Furthermore, the applicant should provide a description of the following:

- Gyroscope and camera output formats
- Transformations of those outputs prior to display to the device user

DEFICIENCY 5 SHOULD BE CONVEYED TO THE APPLICANT.

In the current submission, the applicant refers to Pages 315 – 322 of the original (S0) submission, which includes a thorough description of data acquisition and data analysis.

S1 REVIEW COMMENT: **The information referenced is clear and thorough, and raises no new concerns. The applicant's response to Deficiency 5 is ADEQUATE.**

G DECISION-MAKING

- **SAME TECHNOLOGICAL CHARACTERISTICS?**

  NO. This is the first instance of marketing clearance for this device being sought, and the construction, specifications, and software architecture are different than the equivalent components of predicate devices.

- **COULD THE NEW CHARACTERISTICS AFFECT SAFETY AND/OR EFFECTIVENESS?**

  YES ~~/ NOT CLEAR.~~ **Please see the list of identified significant concerns, *immediately below*:**

  1. ~~Mitigation of biocompatibility concerns (cannot be evaluated until materials information regarding patient-contacting parts of the device has been provided) → *See Deficiency 6*~~ **Resolved**
  2. **Mitigation of optical radiation safety concerns of the LED light sources → *See Testing Below***
  3. **Ability of the gyroscopes to measure head movements, as compared to a suitable comparator → *See Testing Below***
  4. ~~Substantiation of the gyroscope testing by descriptive technological information → *See Deficiency 2*~~ **Resolved**

5. ~~Verification of software functionality (cannot be determined until more descriptive information has been provided)~~ → ~~*See Deficiencies 4 & 5*~~ **Resolved**
6. **Ability of device to accurately and precisely perform eye-tracking → *See Testing Below***
7. ~~Substantiation of the eye tracking testing by descriptive technological information~~ → ~~*See Deficiency 3*~~ **Resolved**
8. **Testing to mitigate electrical safety concerns → *See Testing Below***
9. **Testing to mitigate electromagnetic compatibility concerns → *See Testing Below***

- **ARE THERE NEW TYPES OF SAFETY OR EFFECTIVENESS QUESTIONS?**

  NO. These concerns are common to all video nystagmograph devices.

- **DO ACCEPTED SCIENTIFIC METHODS EXIST FOR ASSESSING THE EFFECTS OF THE NEW CHARACTERISTICS? YES.**

IX PERFORMANCE TESTING

- **ARE PERFORMANCE DATA AVAILABLE TO ALLOW FOR ASSESSMENT OF EQUIVALENCE? YES. See Below.**

A TESTING TO MITIGATE BIOCOMPATIBILITY CONCERNS

S1 REVIEW COMMENT: **The applicant has provided testing which sufficiently mitigates biocompatibility concerns associated with the head-strap and face cushion.**

B TESTING TO MITIGATE OPTICAL RADIATION SAFETY CONCERNS

On Page 1484/1854 of the original (S0) submission, the applicant provided a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60825-1, 2nd edition.

S0 REVIEW COMMENT: This testing appears to demonstrate that optical radiation safety concerns have been ADEQUATELY mitigated.

C TESTING TO VALIDATE ABILITY OF GYROSCOPES TO MEASURE HEAD MOVEMENTS

The applicant provides evidence of testing that validates the ability of the gyroscopes to measure head movements, as compared to an appropriate set of criteria or comparator in response to Deficiency 2 (see above).

S1 REVIEW COMMENT: **This is ADEQUATE.**

D SOFTWARE VERIFICATION & VALIDATION TESTING, VERIFICATION OF OVERALL FUNCTIONALITY TESTING

In Appendix 1 of a document titled "ICS Head Impulse Software Design," located in the original (S0) submission, the applicant provides testing which attempts to simulate device use scenarios to verify software functionality.

S1 REVIEW COMMENT: **In review of this information, it appears that the functionality of the software has been ADEQUATELY verified.**

E TESTING TO VALIDATE THE ABILITY OF THE DEVICE TO ACCURATELY AND PRECISELY TRACK EYE MOVEMENTS

To validate the accuracy of the device to perform eye-tracking, the applicant provided clinical performance testing, generated from their device and other similar devices in the original submission. Regarding this testing, Larry noted the following in his consulting review of the original submission:

> The sponsor provides data to support that the ICS device is accurate when compared to the gold standard for eye position, namely scleral search coils, using both simultaneous (3 subjects) and sequential (4 subjects) testing of normal volunteers and subjects with vestibular disorders. Neither predicate device provided this type of data. The sponsor then provides data to support that the ICS device is as accurate as the Vorteq device. However the Vorteq device used for the comparison is not the same as that in K891008. The device does correspond to a device found at the Micromedical technologies site which claims that the device is used to assess the VOR and also "DVA" – "dynamic visual acuity". However I am unable to find a clearance for this device which also claims to assess "Visual Vestibular Ocular Reflex (VVOR) in well subjects (pilots, athletes) in addition to patients with vestibular deficits." The apparent changes made to the device since K891008 do raise significant effectiveness issues and it would appear that this device should have been the subject of a new 510(k) application. Since I cannot substantiate that this device is cleared for any indication I do not believe that the comparison provided by the sponsor can be used to support that these new methods of assessing the VOR are accurate. The original device in K891008 was not validated against a standard and therefore at best is a tool to display head and eye movement. Despite the comparison to a version of the predicate that does not correspond to the cleared device, the sponsor does provide data that are adequate to support that the technologic changes in comparison to the cleared version of the predicate do not raise new effectiveness issues.

S0 REVIEW COMMENT: Based on Larry's recommendation, testing to validate the eye-tracking accuracy of the device is ADEQUATE; however, information to validate eye-tracking precision is lacking. The applicant shall be asked to provide this information. DEFICIENCY 3 SHOULD BE CONVEYED TO THE APPLICANT.

To obtain data to validate eye-tracking precision, Deficiency 3 was conveyed to the applicant in the January 9th hold memorandum:

You provide testing which compares the eye-tracking ability of your device and similar devices to the Vorteq (cleared under K891008) and scleral search coils. However, this testing does not ensure that your device is able to provide measurements with an appropriate level of precision. Therefore, please provide testing (e.g., via a motorized model eye) that demonstrates the precision of your device. Your test setup should test the entire range of eye movements you expect to see under actual use conditions, and should account for random motion of the goggles in relation to the eye. Furthermore, to corroborate eye-tracking performance testing, please provide a description of the principle of operation of how the LEDs and camera collect information that ultimately allows for eye-tracking.

In the current submission (S1), the applicant responds to Deficiency 3 by providing testing (Attachment 9) of the device using an artificial eye and a mechanical jig to simulate movements. This testing concluded that the device measured eye movements to within +/- 0.1 degree.

S1 REVIEW COMMENT: **In review of this information, it appears that the eye-tracking feature of the device has been ADEQUATELY verified and characterized for pre-clinical precision. The response to Deficiency 3 is ADEQUATE.**

F TESTING TO MITIGATE ELECTRICAL SAFETY CONCERNS

On Page 1263/1854 of the original (S0) submission, the applicant provided a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60601-1.

S0 REVIEW COMMENT: This testing appears to demonstrate that electrical safety concerns have been ADEQUATELY mitigated.

G TESTING TO MITIGATE ELECTROMAGNETIC COMPATIBILITY CONCERNS

On Page 1363/1854 of the original (S0) submission, the applicant provided a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60601-1-2.

S0 REVIEW COMMENT: This testing appears to demonstrate that electromagnetic compatibility concerns have been ADEQUATELY mitigated.

X LABELING

Regarding the labeling, Larry noted the following deficiency in his consulting S0 review, intended to be conveyed to the applicant:

In section 3.1/page 20 of the User manual, page 121/1854, the instructions suggest that a non-physician user should have the patient stop "tranquilizers, sedatives, or

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vestibular suppressants for at least 48 hours before the test" and "If in doubt, consult with a physician about the possible side effects of stopping a particular medication." This is not adequate since we do not consider it safe to withdraw prescribed medications without a specific order of a licensed physician. Please revise this instruction accordingly.

Accordingly, Deficiency 8 was conveyed to the applicant in the January 9th hold memorandum:

In Section 3.1 (Page 20) of the User Manual (Page 121/1854 of the submission), the instructions suggest that a non-physician user should have the patient stop "tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test" and "if in doubt, consult with a physician about the possible side effects of stopping a particular medication." This is not adequate since we do not consider it safe to withdraw prescribed medications without a specific order of a licensed physician. Please revise this instruction accordingly.

In the current (S1) submission, the applicant states that this instruction has been removed, and provides a revised copy of the labeling to that effect (Attachment 3).

S1 REVIEW COMMENT: **The applicant's response to Deficiency 8 is ADEQUATE.**

XI RECOMMENDATION

The submission concerning the ICS Impulse requires no additional information in order to proceed with the review and all safety / effectiveness concerns have been resolved. Therefore, I recommend that the device be found substantially equivalent (SE).

Reviewer Sign-Off:	Rahul K. Ram -S (Affiliate) 2013.02.01 15:55:04 -05'00'
Branch Chief Sign-Off:	Quynh T. Hoang 2013.02.01 16:05:47 -05'00'

Ram, Rahul

From: Daniel Kamm <fda.help.now@gmail.com>
Sent: Friday, February 01, 2013 4:26 PM
To: Ram, Rahul
Subject: RE: K122550 - 510(k) Summary
Attachments: Impulse revised summary K122550.pdf

Daniel Kamm, P.E.
www.fda-consultant.com
Tel +1-239-234-1735
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From: Ram, Rahul [mailto:Rahul.Ram@fda.hhs.gov]
Sent: Friday, February 1, 2013 4:24 PM
To: fda.help.now@gmail.com
Subject: K122550 - 510(k) Summary

Rahul K. Ram
LT, USPHS-CC
CDRH/ODE/DOED/DSDB

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510(K) Summary, 510(k) K122550

Submitter: GN Otometrics A/S
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Contact: Dan Sansonetti, Manager of Research and Development
Date Prepared: January 13, 2012

1. **Identification of the Device:**
   Proprietary-Trade Name: **ICS Impulse**
   Classification Name: Class II, Product Codes: GWN and LXV, Device: Nystagmograph
   Common/Usual Name: Vestibular testing device

2. **Equivalent legally marketed devices**: Micromedical Technologies Inc. Vorteq, K891008 and Micromedical Technologies Inc. VisualEyes K964325.

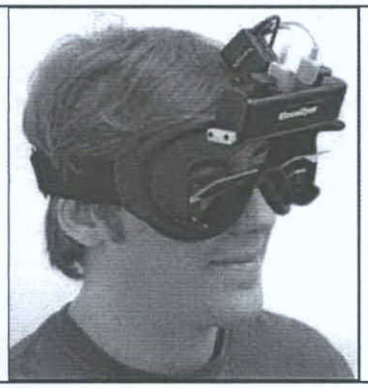
3. **Description of the Device**: The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

4. **Indications for Use (intended use):** The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

5. **Safety and Effectiveness, comparison to predicate device**. This device has the same indications for use as the predicate device but employs different technology to accomplish the same tasks.

6. **Description of Testing:** The device passed UL Electrical Safety testing and EMC testing. Software validation and risk analysis was performed. Clinical testing compared test results to Scleral Search Coils test results. ICS Impulse adequately meets the design requirements and acceptance criteria.

7. Substantial Equivalence Chart

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interfaces	Firewire for Camera Data: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per UL2601 - IEC-60601.	Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

8. Conclusion: After analyzing bench testing, safety, EMC, software, and clinical validation testing we conclude that the ICS Impulse is as safe and effective as the predicate device, and has essentially the same indications for use, thus rendering it substantially equivalent to the predicate device.

Ram, Rahul

From: Rodichok, Lawrence
Sent: Wednesday, January 30, 2013 12:31 PM
To: Ram, Rahul; Hoang, Quynh T.
Cc: To, Nam
Subject: RE: Welcome back, Rahul...pls pick up K122550/S1 fr NNDB box. Thanks!

Absolutely excellent

Lawrence Rodichok MD
Neurostimulation Devices Branch
Division of Neurological and Physical Medicine Devices
Center for Devices and Radiological Health
Food and Drug Administration

WO66 Rm 2457
10903 New Hampshire Ave
Silver Spring, MD 20993

tel: 301-796-6610

lawrence.rodichok@fda.hhs.gov

From: Ram, Rahul
Sent: Wednesday, January 30, 2013 12:30 PM
To: Rodichok, Lawrence; Hoang, Quynh T.
Cc: To, Nam
Subject: RE: Welcome back, Rahul...pls pick up K122550/S1 fr NNDB box. Thanks!

Larry,

You had two deficiencies – one regarding language in the IFU, and the other regarding language in a warning in the labeling. In response to the IFU deficiency, they have revised their IFU to reflect the suggested language which we sent to them. Regarding the labeling deficiency - in their original labeling, a non-physician user was instructed to remove medications up to 48 hours before testing. Accordingly, we sent them a deficiency regarding that instruction and they have since removed it.

Since you seemed so busy, to save you time I was just going to write my review and have you initial places in my review that talk about the responses to these two deficiencies.

Is that okay with you? Sorry for not being more communicative!

- Rahul

From: Rodichok, Lawrence
Sent: Wednesday, January 30, 2013 12:21 PM
To: Ram, Rahul; Hoang, Quynh T.

Food and Drug Administration
Office of Device Evaluation &
Office of In Vitro Diagnostics

COVER SHEET MEMORANDUM

From: Reviewer Name Rahul Ram

Subject: 510(k) Number K122550

To: The Record

Please list CTS decision code TH

☐ Refused to accept (Note: this is considered the first review cycle, See Screening Checklist http://eroom.fda.gov/eRoomReq/Files/CDRH3/CDRHPremarketNotification510kProgram/0_5631/Screening%20Checklist%207%202%2007.doc)

☑ Hold (Additional Information or Telephone Hold).

☐ Final Decision (SE, SE with Limitations, NSE (select code below), Withdrawn, etc.).

Not Substantially Equivalent (NSE) Codes

☐	NO	NSE for lack of predicate
☐	NI	NSE for new intended use
☐	NQ	NSE for new technology that raises new questions of safety and effectiveness
☐	NU	NSE for new intended use AND new technology raising new questions of safety and effectiveness
☐	NP	NSE for lack of performance data
☐	NS	NSE no response
☐	NL	NSE for lack of performance data AND no response
☐	NM	NSE pre-amendment device call for PMAs (515i)
☐	NC	NSE post-amendment device requires PMAs
☐	NH	NSE for new molecular entity requires PMA
☐	TR	NSE for transitional device

Please complete the following for a final clearance decision (i.e., SE, SE with Limitations, etc.):		YES	NO
Indications for Use Page	*Attach IFU*	✓	
510(k) Summary /510(k) Statement	*Attach Summary*	✓	
Truthful and Accurate Statement.	*Must be present for a Final Decision*	✓	
Is the device Class III?			✓
If yes, does firm include Class III Summary?	*Must be present for a Final Decision*		
Does firm reference standards? (If yes, please attach form from http://www.fda.gov/opacom/morechoices/fdaforms/FDA-3654.pdf)			✓
Is this a combination product? (Please specify category _____, see http://eroom.fda.gov/eRoomReq/Files/CDRH3/CDRHPremarketNotification510kProgram/0_413b/COMBINATION%20PRODUCT%20ALGORITHM%20(REVISED%203-12-03).DOC			✓
Is this a reprocessed single use device? (Guidance for Industry and FDA Staff – MDUFMA - Validation Data in 510(k)s for Reprocessed Single-Use Medical Devices, http://www.fda.gov/cdrh/ode/guidance/1216.html)			✓
Is this device intended for pediatric use only?			✓
Is this a prescription device? (If both prescription & OTC, check both boxes.)		✓	
Did the application include a completed FORM FDA 3674, Certification with Requirements of ClinicalTrials.gov Data Bank?		✓	
Is clinical data necessary to support the review of this 510(k)?			✓
For United States-based clinical studies **only**: Did the application include a completed FORM FDA 3674, *Certification with Requirements of ClinicalTrials.gov Data Bank*? (If study was			

conducted in the United States, and FORM FDA 3674 was not included or incomplete, then applicant must be contacted to obtain completed form.)

Does this device include an Animal Tissue Source? ✓

All Pediatric Patients age<=21 ✓

Neonate/Newborn (Birth to 28 days) ✓

Infant (29 days -< 2 years old) ✓

Child (2 years -< 12 years old) ✓

Adolescent (12 years -< 18 years old) ✓

Transitional Adolescent A (18 - <21 years old) Special considerations are being given to this group, different from adults age ≥ 21 (different device design or testing, different protocol procedures, etc.) ✓

Transitional Adolescent B (18 -<= 21; No special considerations compared to adults => 21 years old) ✓

Nanotechnology ✓

Is this device subject to the Tracking Regulation? (Medical Device Tracking Guidance, http://www.fda.gov/cdrh/comp/guidance/169.html) *Contact OC.* ✓

Regulation Number	Class*	Product Code
882.1460	II	GWN

(*If unclassified, see 510(k) Staff)

Additional Product Codes:

Review: J Hoang (Branch Chief) NNDB (Branch Code) 1/9/13 (Date)

Final Review: _____ (Division Director) _____ (Date)

To, Nam

From: To, Nam
Sent: Tuesday, January 08, 2013 1:19 PM
To: 'fda.help.now@gmail.com'
Cc: Hoang, Quynh T.
Subject: K122550
Attachments: Telephone Hold Memorandum.pdf

Dear Mr. Daniel Kamm,

Enclosed is our review of your submission for the ICS Impulse (Model 1085). Your submission has been placed on hold. Please contact me if you have any questions.

Regards,

Rahul K. Ram

LT, USPHS-CC

CDRH/ODE/DOED/DSDB

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Food and Drug Administration
10903 New Hampshire Avenue
Document Control Center – WO66-G609
Silver Spring, MD 20993-0002

Date: January 8, 2013

From: Rahul Ram
Lead Reviewer, K122550
Diagnostic and Surgical Devices Branch
Division of Ophthalmic and Ear, Nose, and Throat Devices
Center for Devices and Radiological Health

To: Mr. Daniel Kamm
Principal Engineer
Kamm & Associates
8870 Ravello Court
Naples, FL 34114
Phone: (239) 234-1735
Fax: (206) 260-4162
Email: fda.help.now@gmail.com

Subject: Traditional 510(k): K122550
Trade Name: ICS Impulse (Model 1085)
Dated: August 10, 2012
Received: August 13, 2012

Dear Mr. Kamm:

We have reviewed your Section 510(k) premarket notification of intent to market the device referenced above. We cannot determine if the device is substantially equivalent to a legally marketed predicate device based solely on the information you provided. We have stopped reviewing your 510(k) submission and placed it on telephone hold, while awaiting your submission of the following additional information:

(b)(4) Deficiencies

[redacted]

(b)(4) Deficiencies

[redacted]

(b)(4) Deficiencies

[redacted]

The deficiencies identified above represent the issues that we believe need to be resolved before our review of your 510(k) submission can be successfully completed. In developing the deficiencies, we carefully considered the statutory criteria as defined in Section 513(i) of the Federal Food, Drug, and Cosmetic Act (Act) for determining substantial equivalence of your device.

We also considered the burden that may be incurred in your attempt to respond to the deficiencies. We believe that we have considered the least burdensome approach to resolving these issues. If, however, you believe that information is being requested that is not relevant to the regulatory decision or that there is a less burdensome way to resolve the issues, you should follow the procedures outlined in the document titled "A Suggested Approach to Resolving Least Burdensome Issues" located at http://www.fda.gov/cdrh/modact/leastburdensome.html.

You may not market this device until you have provided adequate information described above and required by 21 CFR 807.87(l), and you have received a letter from FDA allowing you to do so. If you market the device without conforming to these requirements, you will be in violation of the Act. You may, however, distribute this device for investigational purposes to obtain clinical data if needed to establish substantial equivalence. Clinical investigations of this device must be conducted in accordance with the investigational device exemption (IDE) regulations (21 CFR 812).

In accordance with 21 CFR 807.87(l), FDA may consider a 510(k) to be withdrawn if the submitter fails to provide additional information within 30 days of an Additional Information (AI) request. FDA generally permits submitters additional time to respond to such requests. FDA intends to automatically grant a maximum of 180 calendar days from the date of the AI request, even if the submitter has not requested an extension. Therefore, submitters are no longer required to submit written requests for extension. However, you should be aware that FDA intends to issue a notice of withdrawal under 21 CFR 807.87(l) if FDA does not receive,

in a submission to the appropriate Document Control Center, a complete response to all of the deficiencies in this AI request within 180 calendar days of the date of this request. If you then wish to resubmit this 510(k) notification, a new number will be assigned and your submission will be considered a new premarket notification submission.

For further information regarding how various FDA and industry actions that may be taken on 510(k)s should affect the review clock for purposes of meeting the Medical Device User Fee Amendments of 2012 (MDUFA III), to the Federal Food, Drug, and Cosmetic Act, you may refer to our guidance document entitled "Guidance for Industry and Food and Drug Administration Staff FDA and Industry Actions on Premarket Notification (510(k)) Submissions: Effect on FDA Review Clock and Goals". You may review this document at http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089735.htm.
The requested information should reference your above 510(k) number and should be submitted in duplicate to:

U.S. Food and Drug Administration
Center for Devices and Radiological Health
Document Control Center – WO66-G609
10903 New Hampshire Avenue
Silver Spring, MD 20993-0002

If you have any questions concerning the contents of the letter and would like to set up a teleconference, please contact Mr. Rahul Ram at (301) 796-6620.

DEPARTMENT OF HEALTH & HUMAN SERVICES
Food and Drug Administration
Center for Devices & Radiological Health

Division of Ophthalmic, Neurologic, Ear, Nose, and Throat Devices
Ophthalmic Lasers, Neurostimulators, and Diagnostic Devices Branch
10903 New Hampshire Ave
Silver Spring, MD 20993-0002
(301) 796-6620

ORIGINAL (S0) PREMARKET NOTIFICATION [510(K)] REVIEW

DATE: January 8th, 2013
TO: RECORD
FROM: Rahul Ram
SUBJECT: Traditional, K122550/S0

510(K) HOLDER: GN OTOMETRICS Hoerskaetten 9 Taatrup, DK-2630 Denmark	**OFFICIAL CORRESPONDENT:** Mr. Daniel Kamm Principal Engineer Kamm & Associates 8870 Ravello Court Naples, FL 34114 **Email:** fda.help.now@gmail.com **Phone:** (239) 234-1735 **Fax:** (206) 260-4162
DEVICE TRADE NAME: ICS Impulse (Model 1085)	
DESCRIPTION: Video Nystagmography System	
510(K) DATED DATE: August 10th, 2012	
510(K) RECEIVED DATE: August 13th, 2012	

APPLICANT-IDENTIFIED PREDICATE DEVICES:

510(K) NUMBER	PRODUCT CODE	DEVICE NAME	510(K) HOLDER
K891008	LXV	Vestibular Ocular Reflex Test Equipment (Vorteq)	Micromedical Technologies
K964325	GWN	Visualeyes: Video Eye Monitor	Micromedical Technologies

RECOMMENDATION:

The submission concerning the **ICS Impulse** requires additional information in order to proceed with the review. Therefore, I recommend that the submission be placed on **telephone hold (TH).**

Regulation Number: **21 CFR 882.1460**
Regulation Name: **Nystagmograph**

Regulatory Class: **Class II**
Product Code: **GWN**
Produce Code Description: **Nystagmograph**
Additional Product Codes: **n/a**

INDICATIONS FOR USE (IFU) STATEMENT: ☒ **Prescription** ☐ **Over-the-Counter**

"The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movement."

I SUBMISSION REVIEWERS

- Rahul Ram (ODE/DOED/DSDB) – Lead Reviewer, S0
- Lawrence Rodichok (ODE/DNPMD/NSDB) – Consulting Neurological Clinical Reviewer
- Quynh Hoang (ODE/DNPMD/NNDB) – Branch-Level Concurrence

II PURPOSE OF SUBMISSION

As per the submission cover letter, the applicant has submitted this 510(k) premarket notification in order to solicit marketing clearance for the **ICS Impulse** (Model 1085). As stated in Exhibit 10 ("Executive Summary") of the current (S0) submission, the device consists primarily of head-mounted video recording goggles and associated software to assess the vestibular-ocular reflex (VOR).

III SUBMISSION HISTORY

(August 13th, 2012) K122550/S0, received in FDA

IV BACKGROUND

A VESTIBULOOCULAR REFLEX (VOR), BRIEF DESCRIPTION

The vestibular-ocular / vestibuloocular reflex (VOR) is one of three vestibular reflexes (three involuntary responses to strong stimulation to the vestibular system (the balance and spatial orientation component of the human nervous system)). The VOR contributes to image stability on the fovea of the retina by effecting involuntary eye movements compensate for head movements (rotational, translational, head tilt with respect to gravity) in directions opposite of those eye movements.

B TESTING

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VOR can be evaluated by measuring eye motion and concurrent head movements; **gain** is the ratio of eye velocity to head velocity, and a normal value should be -1 (indicating an equally compensatory eye movement for any head movement). **Video-oculography (VOG)** is a method of concurrently measuring head movements and eye movements using digital video cameras and accelerometers.

C CURRENT DEVICE

The current device, the ICS Impulse, claims to perform video-oculography using infrared digital cameras to record eye movements and goggle-mounted gyroscopes to observe head movements.

V OVERALL ADMINISTRATIVE REQUIREMENTS

REQUIREMENT	CURRENT SUBMISSION (S0) LOCATION
User Fee Cover Sheet (FDA Form 3601)	PAGE 6
CDRH Premarket Review Submission Cover Sheet (FDA Form 3514)	PAGE 7
Cover Letter	PAGE 14
Indications for Use Statement	PAGE 18
510(k) SUMMARY	PAGE 20
Truthful and Accuracy Statement	PAGE 23
Class III Summary or Certification	PAGE 24
Financial Certification or Disclosure Statement	PAGE 25
Clinical Certification Statement (FDA Form 3674)	PAGE 25
Standards Data Report(s) (FDA Form 3654)	PAGE 27 & FULL TEST REPORTS PROVIDED
Executive Summary	PAGE 40

S0 REVIEW COMMENT: **Administrative requirements appear to have been met.**

SECTION RECOMMENDATION: ADEQUATE

VI INDICATIONS FOR USE

The Indications for Use (IFU) statement, as reported in Exhibit 4 (Page 19) of the current (S0) submission, is the following:

"The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements." (Prescription (Rx) Use)

S0 REVIEW COMMENT: **Based on the above IFU, the following safety / effectiveness concerns are noted:**

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1. **The applicant should validate the ability of the device to measure, record, display and analyze eye/head movements**
2. **The applicant should provide evidence that this analysis can be used to assess the vestibular-ocular reflex (VOR)**

Regarding (1) and (2), please see sections of this review, below, for resolution of these concerns.

3. **It is not clear why this device should be limited to patients "with complaints of dizziness, disequilibrium, and vertigo"; this suggests that the device has some clinical utility specific to this subpopulation; the applicant should either provide a justification of this utility or remove this restriction from the IFU**

Regarding (3), please see the comments related to Deficiency 1, below.

A CONSISTENCY THROUGHOUT SUBMISSION

Aside from in Exhibit 4, the applicant provides the IFU statement in the following locations in the current (S0) submission:

- (Page 10) FDA Form 3514 (CDRH Premarket Review Cover Sheet) → ***Identical***
- (Page 21) 510(k) Summary → ***Identical***
- (Page 22) 510(k) Summary, albeit under heading "Intended Use" → ***Identical***
- (Page 72) Substantial Equivalence Discussion, albeit under heading "Intended Use" → ***Identical***
- (Page 106) Page 5 of User Manual, albeit under heading "Intended Use" → ***Identical***

S0 REVIEW COMMENT: **The instances of the IFU throughout the submission are consistent with one another. This is ADEQUATE.**

B COMPARISON TO PREDICATE DEVICES

There exists no explicit IFU statement for one of the applicant's chosen predicate devices, the VORTEQ (cleared under K891008); the following is the intended use of that device, as stated by the reviewer of that submission (Melpomeni K. Jeffries):

> *The intended use of VORTEQ is to evaluate the vestibular ocular reflex.*

The following is the IFU statement of the applicant's other chosen predicate device, the VisualEyes (cleared under K964325, obtained from IMAGE):

> *The Eye Monitor is used to observe eye movements from various stimuli used in vestibular diagnostic testing. It allows observation of horizontal, vertical and torsional eye movements. The Eye Monitor is mainly used to visually detect nystagmus positional maneuvers (e.g. Hallpike (e.g. Hallpike*

positional tests) or caloric tests. The device is especially useful in situations where the operator needs to observe the patient's eyes to distinguish nystagmus from artifacts. These observations are currently done using Fresnel lenses. The eye movements could also be recorded on suitable media (e.g. video tape) to provide documentation for any diagnosis based on observation of the Eye Monitor.

S0 REVIEW COMMENT: **The intended use of the ICS Impulse is the same as those of the predicate devices. The specific claims (*see review comment above*), however, should be validated. The identified predicate devices are ADEQUATE.**

C DECISION-MAKING

- **SAME INDICATIONS FOR USE?**

  NO. The following difference is noted:

  - The IFU of the ICS Impulse includes a intended use sub-population that is not included in the IFU statements of the predicate devices

- **DO THE DIFFERENCES ALTER THE EFFECT OR RAISE NEW QUESTIONS OF SAFETY AND/OR EFFECTIVENESS?**

  YES, HOWEVER... the applicant can validate use of the device in this sub-population using performance testing. The applicant will be instructed to justify this claim.

  Larry also notes this as a concern in his review:

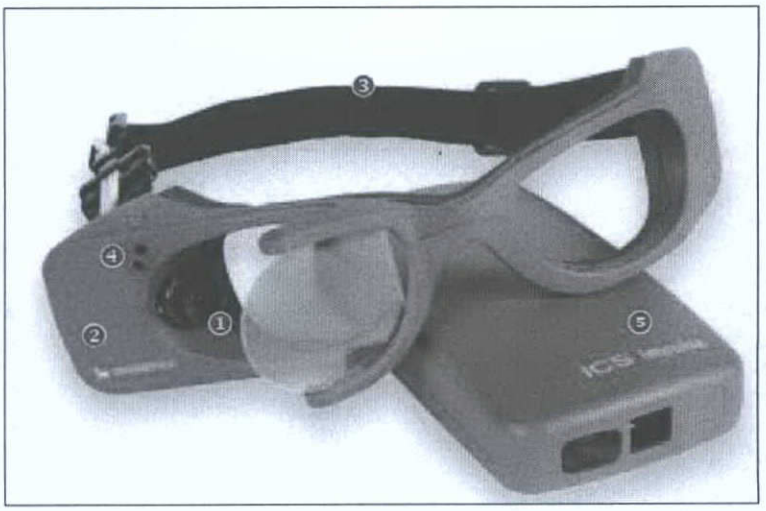
  Reviewer's comments: Unless the sponsor can provide data to support accuracy for the stated population "patients with complaints of dizziness, disequilibrium, and vertigo, the indication should be limited to "assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements".

S0 REVIEW COMMENT: **Regarding the concern above, DEFICIENCY 1 SHALL BE CONVEYED TO THE APPLICANT.**

SECTION RECOMMENDATION: NOT ADEQUATE

VII TECHNOLOGICAL CHARACTERISTICS

The applicant provides a description of the device in Exhibit 11 ("Device Description") of the current (S0) submission. The following is a graphical depiction of the ICS Impulse, reproduced from Exhibit 11:



Although the applicant does not provide labels to accompany the numbered items in the graphic above, as described in Exhibit 11, the ICS Impulse is a goggles-based device that consists of the following gross physical components:

- Goggle Frame – houses other components
- Infrared LED – illuminates the right eye
- Digital Camera – mounted near the right eye, receives reflected light from the right eye for processing eye movements
- Lens – zero optical power, half-silvered lens in the frame location in front of the right eye that allows the device user to observe visual stimuli, but also reflects light from the eye to the camera
- Gyroscopes – records head movement
- USB/Firewire Interface Box (*labeled as "5"*) – connected, via cable, to the Firewire output of the camera to allow data transmission to the computer
- Calibration Laser

Not depicted is a personal computer on which the OTOSuite Vestibular software is intended to run, which stores and processes the head/eye movement data.

S0 REVIEW COMMENT: **Please refer to the "Software" section of this review for more information regarding OTOSuite.**

A MATERIALS, ALL DEVICE COMPONENTS

The applicant has not provided a list of the patient-contacting parts of the device and the corresponding materials of which each part is composed.

S0 REVIEW COMMENT: **This is NOT ADEQUATE. The applicant should provide this information, as well as mitigation of biocompatibility concerns. DEFICIENCY 6 SHOULD BE CONVEYED TO THE APPLICANT.**

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B INFRARED LEDS & DIGITAL CAMERA

On Page 64/1854 of the current (S0) submission, in a document titled "Type 1085 ICS Impulse System Specification," the applicant states in specification number HW-7 that "*two infrared LEDs of 875 nanometers shall be provided to illuminate the patient eye.*" The applicant refers to "Type 1085, Hardware Verification Protocol 0-80-06520" for evidence of meeting this specification.

S0 REVIEW COMMENT: **Please refer to the "Performance Testing" section of this review for an evaluation of optical radiation safety hazards associated with this light source.**

The applicant states, on Page 45/1854, that the sampling rate of the camera is 250 Hz for the "Head Impulse Test," and 30, 60, or 120 Hz for "video recording."

S0 REVIEW COMMENT: **The applicant provides no description of the principle of operation of how the LEDs and camera collect information that ultimately allows for eye-tracking. This is important to substantiate performance provided to validate eye-tracking functionality. DEFICIENCY 3 SHOULD BE CONVEYED TO THE APPLICANT.**

C GYROSCOPES

On Page 306/1854, the applicant provides a specification for the device, stating that *"the design shall measure angular velocity with a sensitivity of 2mV/deg/s±10% about the X, Y, and Z axis."*

S0 REVIEW COMMENT: **This is NOT ADEQUATE. The applicant should provide the following information:**

- **Gyroscope measurement range**
- **Measurement recording frequency**
- **Angular random walk (to assess variability)**

This information is important to substantiate their performance in recording head movements. DEFICIENCY 2 SHALL BE CONVEYED TO THE APPLICANT.

For an evaluation of any performance provided in this regard, please see the "Performance Testing" section of this review.

D USB/FIREWIRE INTERFACE BOX

The applicant states that the USB/Firewire interface box is intended to connect from the Googles to the computer to display, store, and analyze recorded data.

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E CALIBRATION LASER

The applicant provides little to no information regarding the physical characteristics of the calibration laser.

S0 REVIEW COMMENT: **This is NOT ADEQUATE. The applicant should provide a diagrammatic and textual description of the calibration laser, including a principle of operation and rationale for how calibration improves data quality. DEFICIENCY 7 SHALL BE CONVEYED TO THE APPLICANT.**

F DESCRIPTION OF THE DEVICE - SOFTWARE

Level of Concern

On Page 53/1854 of the submission, the applicant states the following regarding software Level of Concern:

> *"The Software provides diagnostic information for evaluation without control or delivery ofharmful energy and is considered a Moderate concern device."*

However, on Page 291/1854 of the submission, the applicant states the following:

> *"We believe the level of concern is Minor if failures or latent design flaws are unlikely to cause any injury to the patient or operator."*

S0 REVIEW COMMENT: **The applicant should clarify this inconsistency and state the software Level of Concern. Furthermore, the applicant should provide software documentation as per FDA's premarket software guidance (http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089543.htm). When providing this documentation, the applicant should specify the page numbers (i.e., Page X to Page X) for each software section. DEFICIENCY 4 SHOULD BE CONVEYED TO THE APPLICANT.**

Software Description

The applicant provides no standalone description of the software.

S0 REVIEW COMMENT: **This is NOT ADEQUATE. Although the applicant provides software specifications, these specifications do not clearly define all functions of the software, or more simply what software programs / modules are running on the personal computer. Therefore, the applicant should provide a list of all software programs / modules part of the device, including an indication of whether each is optional or required for operation and a statement of purpose for each. Furthermore, the applicant should provide a description of the following:**

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- **Gyroscope and camera output formats**
- **Transformations of those outputs prior to display to the device user**

DEFICIENCY 5 SHOULD BE CONVEYED TO THE APPLICANT.

G DECISION-MAKING

- **SAME TECHNOLOGICAL CHARACTERISTICS?**

  NO. This is the first instance of marketing clearance for this device being sought, and the construction, specifications, and software architecture are different than the equivalent components of predicate devices.

- **COULD THE NEW CHARACTERISTICS AFFECT SAFETY AND/OR EFFECTIVENESS?**

  YES / NOT CLEAR. Please see the list of identified significant concerns, *immediately below*:

  1. **Mitigation of biocompatibility concerns (cannot be evaluated until materials information regarding patient-contacting parts of the device has been provided) → *See Deficiency 6***
  2. **Mitigation of optical radiation safety concerns of the LED light sources → *See Testing Below***
  3. **Ability of the gyroscopes to measure head movements, as compared to a suitable comparator → *See Testing Below***
  4. **Substantiation of the gyroscope testing by descriptive technological information → *See Deficiency 2***
  5. **Verification of software functionality (cannot be determined until more descriptive information has been provided) → *See Deficiencies 4 & 5***
  6. **Ability of device to accurately and precisely perform eye-tracking → *See Testing Below***
  7. **Substantiation of the eye-tracking testing by descriptive technological information → *See Deficiency 3***
  8. **Testing to mitigate electrical safety concerns → *See Testing Below***
  9. **Testing to mitigate electromagnetic compatibility concerns → *See Testing Below***

  - **ARE THERE NEW TYPES OF SAFETY OR EFFECTIVENESS QUESTIONS?**

    NO. These concerns are common to all video nystagmograph devices.

  - **DO ACCEPTED SCIENTIFIC METHODS EXIST FOR ASSESSING THE EFFECTS OF THE NEW CHARACTERISTICS? YES.**

VIII PERFORMANCE TESTING

- **ARE PERFORMANCE DATA AVAILABLE TO ALLOW FOR ASSESSMENT OF EQUIVALENCE? YES & NO. See Below.**

A TESTING TO MITIGATE BIOCOMPATIBILITY CONCERNS

S0 REVIEW COMMENT: **Until Deficiency 6 is resolved, mitigation of biocompatibility concerns is NOT ADEQUATE.**

B TESTING TO MITIGATE OPTICAL RADIATION SAFETY CONCERNS

On Page 1484/1854 of the current (S0) submission, the applicant provides a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60825-1, 2nd edition.

S0 REVIEW COMMENT: **This testing appears to demonstrate that optical radiation safety concerns have been ADEQUATELY mitigated.**

C TESTING TO VALIDATE ABILITY OF GYROSCOPES TO MEASURE HEAD MOVEMENTS

The applicant does not provide evidence of testing that validates the ability of the gyroscopes to measure head movements, as compared to an appropriate set of criteria or comparator.

S0 REVIEW COMMENT: **This is NOT ADEQUATE, as head movement measurements are a significant contributor to overall device safety / effectiveness. DEFICIENCY 2 SHOULD BE CONVEYED TO THE APPLICANT. Furthermore, more descriptive information regarding the principle of operation of the gyroscopes is necessary to substantiate this performance. DEFICIENCY 2 SHOULD BE CONVEYED TO THE APPLICANT.**

D SOFTWARE VERIFICATION & VALIDATION TESTING, VERIFICATION OF OVERALL FUNCTIONALITY TESTING

S0 REVIEW COMMENT: **Until Deficiencies 4 & 5 are resolved, verification of software functionality is NOT ADEQUATE.**

E TESTING TO VALIDATE THE ABILITY OF THE DEVICE TO ACCURATELY AND PRECISELY TRACK EYE MOVEMENTS

To validate the accuracy of the device to perform eye-tracking, the applicant provides clinical performance testing, generated from their device and other similar devices. Regarding this testing, Larry notes the following in his consulting review:

The sponsor provides data to support that the ICS device is accurate when compared to the gold standard for eye position, namely scleral search coils, using both simultaneous (3 subjects) and sequential (4 subjects) testing of normal volunteers and subjects with vestibular disorders. Neither predicate device provided this type of data. The sponsor then provides data to support that the ICS device is as accurate as the Vorteq device. However the Vorteq device used for the comparison is not the same as that in K891008. The device does correspond to a device found at the Micromedical technologies site which claims that the device is used to assess the VOR and also "DVA" – "dynamic visual acuity". However I am unable to find a clearance for this device which also claims to assess "Visual Vestibular Ocular Reflex (VVOR) in well subjects (pilots, athletes) in addition to patients with vestibular deficits." The apparent changes made to the device since K891008 do raise significant effectiveness issues and it would appear that this device should have been the subject of a new 510(k) application. Since I cannot substantiate that this device is cleared for any indication I do not believe that the comparison provided by the sponsor can be used to support that these new methods of assessing the VOR are accurate. The original device in K891008 was not validated against a standard and therefore at best is a tool to display head and eye movement. Despite the comparison to a version of the predicate that does not correspond to the cleared device, the sponsor does provide data that are adequate to support that the technologic changes in comparison to the cleared version of the predicate do not raise new effectiveness issues.

S0 REVIEW COMMENT: **Based on Larry's recommendation, testing to validate the eye-tracking accuracy of the device is ADEQUATE; however, information to validate eye-tracking precision is lacking. The applicant shall be asked to provide this information. DEFICIENCY 3 SHOULD BE CONVEYED TO THE APPLICANT.**

F TESTING TO MITIGATE ELECTRICAL SAFETY CONCERNS

On Page 1263/1854 of the current (S0) submission, the applicant provides a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60601-1.

S0 REVIEW COMMENT: **This testing appears to demonstrate that electrical safety concerns have been ADEQUATELY mitigated.**

G TESTING TO MITIGATE ELECTROMAGNETIC COMPATIBILITY CONCERNS

On Page 1363/1854 of the current (S0) submission, the applicant provides a test report whereby Underwriters Laboratory, Inc. evaluated device conformance to IEC 60601-1-2.

S0 REVIEW COMMENT: **This testing appears to demonstrate that electromagnetic compatibility concerns have been ADEQUATELY mitigated.**

SD

IX LABELING

Regarding the labeling, Larry notes the following deficiency in his consulting review, intended to be conveyed to the applicant:

> In section 3.1/page 20 of the User manual, page 121/1854, the instructions suggest that a non-physician user should have the patient stop "tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test" and "If in doubt, consult with a physician about the possible side effects of stopping a particular medication." This is not adequate since we do not consider it safe to withdraw prescribed medications without a specific order of a licensed physician. Please revise this instruction accordingly.

S0 REVIEW COMMENT: **As per Larry's recommendation, DEFICIENCY 8 SHALL BE CONVEYED TO THE APPLICANT.**

X RECOMMENDATION

I RECOMMEND THAT K122550 / S0 BE PLACED ON HOLD, AND THAT THE FOLLOWING DEFICIENCIES BE CONVEYED TO THE APPLICANT.

XI DEFICIENCIES

1. Your proposed Indications for Use (IFU) statement reads: "The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo." We do not consider this IFU acceptable since you have not provided adequate clinical data to support the accuracy of the device in evaluating this specific population. Please provide data to support the diagnostic accuracy of your device in this specific population. Alternatively, please consider the following modified IFU statement: "The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements."

2. On Page 306/1854, you provide a specification for the device, stating that *"the design shall measure angular velocity with a sensitivity of 2mV/deg/s±10% about the X, Y, and Z axis."* However, you do not provide any more information regarding the gyroscopes. Because measurements of head movements is a significant factor in the safety and effectiveness of your device, please provide testing protocol and results that demonstrate the ability of the gyroscopes to measure head movements, including the following:

   a. Gyroscopes measurement range
   b. Measurement recording frequency
   c. Angular random walk (to assess variability).

3. You provide testing which compares the eye-tracking ability of your device and similar devices to the Vorteq (cleared under K891008) and scleral search coils. However, this

testing does not ensure that your device is able to provide measurements with an appropriate level of precision. Therefore, please provide testing (e.g., via a motorized model eye) that demonstrates the precision of your device. Your test setup should test the entire range of eye movements you expect to see under actual use conditions, and should account for random motion of the goggles in relation to the eye. Furthermore, to corroborate eye-tracking performance testing, please provide a description of the principle of operation of how the LEDs and camera collect information that ultimately allows for eye-tracking.

4. On Page 53/1854 of the submission, you state the following regarding software Level of Concern:

   "The Software provides diagnostic information for evaluation without control or delivery of harmful energy and is considered a Moderate concern device."

   However, on Page 291/1854 of the submission, the you state

   "We believe the level of concern is Minor if failures or latent design flaws are unlikely to cause any injury to the patient or operator."

   Please clarify this inconsistency and state the software Level of Concern. Furthermore, please provide software documentation as per FDA's premarket software guidance (http://www.fda.gov/MedicalDevices/DeviceRegulationandGuidance/GuidanceDocuments/ucm089543.htm). When providing this documentation, please specify the page numbers (i.e., Page X to Page X) for each software section.

5. You provide no standalone description of the software. Although you provide software specifications, these specifications do not clearly define all functions of the software, or more simply what software programs / modules are running on the personal computer. Therefore, please provide a list of all software programs / modules part of the device, including an indication of whether each is optional or required for operation and a statement of purpose for each. Furthermore, please provide a description of the following:

   a. Gyroscope and camera output formats
   b. Transformations of those outputs prior to display to the device user

6. You do not provide a list of the patient-contacting parts of the device and the corresponding materials of which each part is composed. This is important to mitigate concerns regarding biocompatibility. Please provide this list, and evidence that biocompatibility concerns for each patient-contacting part of your device have been mitigated.

7. You provide no information regarding the characteristics of the calibration laser. Because the calibration laser may affect measurements made by your device, please provide a diagrammatic and textual description of the calibration laser, including a principle of operation and rationale for how calibration improves data quality.

8. In Section 3.1 (Page 20) of the User Manual (Page 121/1854 of the submission), the instructions suggest that a non-physician user should have the patient stop "tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test" and "if in doubt, consult with a physician about the possible side effects of stopping a particular medication." This is not adequate since we do not consider it safe to withdraw prescribed medications without a specific order of a licensed physician. Please revise this instruction accordingly.

Lead Reviewer Signoff:

Ka N. To
2013.01.08 13:23:56 -05'00'

Rahul Ram (ODE/DOED/DSDB)

Management Signoff:

Quynh T. Hoang
2013.01.08 16:08:29 -05'00'

Quynh Hoang (ODE/DNPMD/NNDB)

DEPARTMENT OF HEALTH AND HUMAN SERVICES MEMORANDUM

Food and Drug Administration
Office of Device Evaluation
10903 New Hampshire Ave.
Silver Spring, MD 20993

510(k) MEMORANDUM
Medical Officer Review

K122505

Date: November 19, 2012
To: Rahul Ram, Lead Reviewer **Division:** DOED
From: Lawrence Rodichok, M.D., Medical Officer **Branch:** NSDB

Device: ICS Impulse (Model 1085)
Sponsor: Company: GN Otometrics (GN HEARING CARE CORPORATION)

RECOMMENDATION: Additional information needed.

SUMMARY: This is a pre-marketing notification for a device to record, display and provide a calculation of some aspects of the vestibular ocular reflex (VOR). The applicant device uses tight fitting goggles in which are embedded devices for the detection of head motion (gyroscopes) and a video system to record eye position/movement to generate data used to compute the VOR. The output is essentially the ratio of the velocity of eye movement to the velocity of head movement. Ideally this ratio should be 1.00 since the velocity of eye movement should match that of head movement in the opposite direction in order to maintain eye position on a target. The sponsor proposes two predicate devices. One of these, K964325, "VisualEyes" is a device that includes a small video camera embedded in goggles to record eye movements in order to observe for evidence of nystagmus. It is not cleared to assess the VOR. The other predicate device, K891008, Vorteq, is a device that is intended to assess the VOR (although no formal IFU was identified in any available documents). However this device uses electrooculography electrodes for eye movement and an electromechanical sensor to detect head position. The sponsor provides data to support that the ICS device is accurate when compared to the gold standard for eye position, namely scleral search coils, using both simultaneous (3 subjects) and sequential (4 subjects) testing of normal volunteers and subjects with vestibular disorders. Neither predicate device provided this type of data. The sponsor then provides data to support that the ICS device is as accurate as the Vorteq device. However the Vorteq device used for the comparison is not the same as that in K891008. The device does correspond to a device found at the Micromedical technologies site which claims that the device is used to assess the VOR and also "DVA" – "dynamic visual acuity". However I am unable to find a clearance for this device which also claims to assess "Visual Vestibular Ocular Reflex (VVOR) in well subjects (pilots, athletes) in addition to patients with vestibular deficits." The apparent changes made to the device since K891008 do raise significant effectiveness issues and it would appear that this device should

have been the subject of a new 510(k) application. Since I cannot substantiate that this device is cleared for any indication I do not believe that the comparison provided by the sponsor can be used to support that these new methods of assessing the VOR are accurate. The original device in K891008 was not validated against a standard and therefore at best is a tool to display head and eye movement.

Despite the comparison to a version of the predicate that does not correspond to the cleared device, the sponsor does provide data that are adequate to support that the technologic changes in comparison to the cleared version of the predicate do not raise new effectiveness issues. However the IFU should be modified as indicated in deficiency 1 and a revision of the User manual is needed as described in deficiency 2.

MEDICAL REVIEW

(Sponsor in italics, FDA in plain text, reviewer comments in bold)

510(k) Number: K122550

Device Name: **ICS Impulse (Model 1085)**
Product code: LXV
Prior submissions:

SPONSOR LETTER:

August 13, 2012

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse

Product Code: LXV, Device: Apparatus, vestibular analysis

Purpose of submission: *This is to notify you of the intention by GN Otometrics to market a new but substantially equivalent, to a legally marketed, device: ICS Impulse vestibular analysis device. There have been no changes to the indications for use and many other essential characteristics as compared to the predicate devices.*

Confidentiality: *GN Otometrics considers the information contained in this submission to be confidential in nature (except for Exhibit 5 as required by the SMDA)*

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510(k) Summary: *In response to the requirements addressed by the SMDA of 1990, a summary of the safety and effectiveness information upon which the substantial equivalence determination is based is enclosed. (Exhibit 5)*

MEDICAL REVIEW

(Sponsor text in italics, FDA in plain text, reviewer comments in bold)

Brief Device Description

(page 21 of pdf submission)

3. Description of the Device: *The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.*

Exhibit 10. Executive Summary

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. The key components are shown below:

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- ***High-speed camera (250 Hz.)*** *The superior camera provides the best available technology for measuring fast eye movements. This provides the ability to record the eye while providing High frequency head movements. Both covert and overt saccades can be identified.*
- ***Built-in gyroscopes.*** *Dual-axis gyroscopes measure the head movement accurately allowing for direct comparison in head and eye velocities. They also provide instant feedback on the quality of the head impulse maneuver.*
- ***No slippage*** *Weighing 60 grams the goggle ensures no slippage and therefore providing accurate data collection without missing any important eye movements.*
- ***Built-in calibration laser.*** *With a built-in calibration laser, the test can be performed anywhere there's a wall for calibration. There's no need for additional hardware.*
- ***USB/Firewire data transmission.*** *The USB/Firewire interface box allows fast, accurate data transfer to the computer.*

The testing is done via "head impulse testing." This is an ear-specific test that detects disorders of the vestibulo-ocular reflex and identifies which ear is affected in cases of peripheral vestibular loss.
Patients with a vestibular loss will exhibit a corrective saccadic eye movement (a "catch-up" saccade) either during or after the head impulse and the gain of the head in comparison to the eye will not be equivalent. This is an assessment tool that provides quick, precise information about the vestibuloocular reflex to stimuli in the high-frequency range. It was first identified and described by Halmagyi and Curthoys in the 1988 article, "A Clinical Sign of Canal Paresis." Said Halmagyi: "The eyes are the speedometers of the semicircular canals."*

**See Bibliography in Exhibit 20.*

Comparison table follows.

Indications for Use (proposed)

The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

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Reviewer's comments: Unless the sponsor can provide data to support accuracy for the stated population "patients with complaints of dizziness, disequilibrium, and vertigo, the indication should be limited to "assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements".

Predicate Device Information:
K891008 – Vorteq – Micromedical technologies
K964325 – VisualEyes – Micromedical technologies

Indications for Use (predicate)
K964325
Indications For Use:

The Eye Monitor is used to observe eye movements from various stimuli used in vestibular diagnostic testing. It allows observation of horizontal, vertical and torsional eye movements.

The Eye Monitor is mainly used to visually detect nystagmus positional maneuvers (e.g. Hallpike (e.g. Hallpike positional tests) or caloric tests. The device is especially useful in situations where the operator needs to observe the patient's eyes to distinguish nystagmus from artifacts. These observations are currently done using Fresnel lenses.

The eye movements could also be recorded on suitable media (e.g. video tape) to provide documentation for any diagnosis based on observation of the Eye Monitor.

Reviewer's comments: This device is used primarily to detect eye movements using a small CCD camera mounted on goggles. This is technologically comparable to the camera used in the applicant's device. However the method used to analyze the result appears to have been by simple visual observation whereas for the applicant's device the position is detected using a computerized detection and velocity computed from that result. Although this does not raise any new safety issues, clinical data would be needed to support that the methodology being used is accurate for the proposed indication. See below for the clinical data provided.

K891008 – from the decision letter

Summary:
Micromedical Technologies, Inc. is planning to market VORTEQ an acronym for Vestibular Ocular Reflex Test Equipment. The submission includes a description of the device, indications for use, specifications, a users manual, software validation, patient data and predicate device information. The predicate devices are the Vestibular Autorotation Test (VAT),K 871466 and the Rotary Vestibular Testing System(RVT-50) marketed by ICS Medical Corp, K872093.

Analysis:
The intended use of VORTEQ is to evaluate the vestibular ocular reflex. Evaluation of the vestibular ocular reflex enables the clinician to distinguish between central and peripheral

pathology. Since the test relies upon head movements, it cannot differentiate bilateral Iron unilateral vestibular pathology. Both the VAT and RVT-50 have the same indications for use, and the same limitation.

The VORTEQ consists of a small electro-mechanical sensor mounted on a headband worn by the patient to measure angular velocity of the head, electro-oculograpy electrodes (EOG) and amplifier (K863424,) an IBM-AT compatible office computer and a computer program which records signals and compares head velocity to eye velocity when the patient voluntarily shakes their head no or yes. The VAT system has the same components while the RVT-50 consists of a rotary chair, EOG, computer and computer program. All three devices stimulate the vertical as well as the horizontal semi-circular canals, and presents calculations and graphs of gain, phase and gain asymmetry. The Vat and VORTEQ both use Fourier analysis to compute the results.

The software has been validated by processing files of known frequency, amplitude, asymmetries and phase delays (consultation with Monica Ferrante).

The alogrithm used to compute gain etc. are standard equation used in vestibular testing. Some patient data is included. However the VORTEQ has virtually the same specifications as the VAT which has been proven effective by data from over 400 patients, most of which has been published.

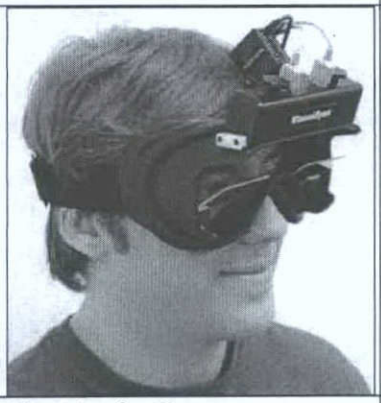
The user manual is very detailed and consistent with the intended uses. The VORTEQ is substantially equivalent in safety, intended uses, specifications, and effectiveness to the predicate devices.

The K891008 clearance describes a device which is A) "A small electro-mechanical sensor mounted on a headband and worn by a patient to measure angular velocity of the head, B) an "Electro-oculography(EOG) electrodes and amplifier to measure eye position (previously approved 5108 application # 8863424), C) A personal computer and interface which digitizes the signals from the angular velocity sensor and the EOG amplifier D) A computer program which records the above signals and compares head velocity to eye velocity when the patient voluntarily shakes their head no or yes."

Reviewer's comments: The predicate VORTEQ device is intended to record head and eye movement to assess the VOR but not using the same technology as the applicant device. It uses a somewhat different technology to detect head movement and EOG electrodes to detect eye movement. The method of computing velocity appears to differ as well. The IFU is not defined in the decision letter or in the CDRH database. The technology in this device differs from that in the applicant's device in that there is a different method used to detect head position and eye position. It is intended to record both head and eye movement but clearly using substantially different technology. Clinical data would be needed to support that the differences do not raise new issues of accuracy. See below for the data submitted.

Comparison to Predicate Device:

Characteristic	*Micromedical Technologies Inc. Vorteq, K891008 and*	*ICS Impulse*

	VisualEyes K964325.	
Intended Use:	*VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems*	*The ICS Impulse System is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. The assessment of the vestibular-ocular reflex (VOR) is performed by measuring, recording, displaying, and analyzing eye and head movements.*
Configuration	*VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing*	*The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement.* *The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage*
Photo		
Interfaces	*Firewire for Camera* *Data: Not specified*	*Firewire for Camera* *USB 2 for Data*
Electrical safety	*Electrical Safety per UL2601 - IEC-60601*	*Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed*
EMC	*Not specified*	IEC 60601-1-2: 2007
Calibration	*Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.*	*Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.*

Reviewer's comments: The picture above corresponds to that found at the Micromedical Technology website- http://www.micromedical.com/vorteqdva.html. I am unable to substantiate that this is a cleared device since it differs substantially from the device cleared under K891008.

Testing and Conclusions:

60

(b)(4) Testing

(b)(4) Testing

(b)(4) Testing

(b)(4) Testing

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(b)(4) Testing

1 Introduction

1.1 General

The purpose of this Clinical Evaluation is to provide a critical evaluation of the pre- and post market clinical data relevant to the intended use of the Type 1085, ICS Impulse manufactured by GN Otometrics. This may include data generated through scientific literature search, clinical experience, and/or clinical investigation. This is done in fulfillment of the requirements established within the guidelines for medical products 93/42/EEC, Annex X and MEDDEV 2.7.1. (Medical Devices).

Routinely, this clinical data evaluation report shall be reviewed and updated to reflect the implementation of any new relevant information as it is available. Regulatory Affairs and relevant clinically competent and experienced personnel shall determine what new information is relevant.

This clinical data evaluation report focuses on comparisons with equivalent predecessors and competitor products to determine the efficacy of those. Considerations of whether GN Otometrics' design and development processes are capable of developing medical device products that are safe and effective as the predecessors will be done.

1.2 Background

1.2.1 ICS Impulse is used in the assessment of patients with complaints of dizziness, disequilibrium, and vertigo. A head impulse maneuver is used to assess the vestibular-ocular reflex (VOR). The assessment of the VOR is performed by measuring, recording, displaying, and analyzing eye and head movements.

1.2.2 The head impulse was developed in 1988 by Drs. Ian Curthoys and Michael Halmagyi. Currently, the head impulse is performed and the physician/clinician would visually observe the eye movement. In a small number of hospital/research facilities in the world the "gold standard" scleral search coils are used. However, this device is very expensive, very large and therefore only utilized by a select few. The other option physicians were using was to perform the head impulse and visually observe the patient's eye movements. Visual observation is subjective and overt saccades are unidentifiable. Visual observation has proven not as accurate as the scleral search coils.

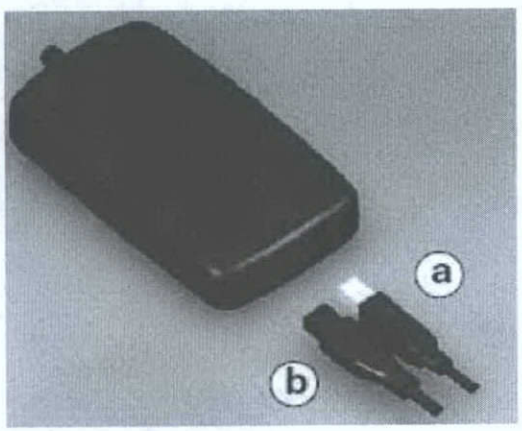
Drs Ian Curthoys and Michael Halmagyi have spent 20 years working on the development of video goggles for the use of recording eye movement during head impulse testing. It has not been until recently that the technology (video cameras, gyroscopes, accelerometers) has reached a small enough size that a goggle can be produced that works with head impulse testing. The goggle must be lightweight and fit close to the patient's head to avoid any slippage. Slippage of the goggle will result in the camera being unable to record the pupil during the head impulse test. The second technology improvement is in relation to the speed of these small cameras. A high speed camera is needed in order to capture the very fast eye movement that occurs during the head impulse test. In 2007/2008 Drs. Ian Curthoys and Michael Halmagyi and others at University of Sydney developed a prototype head impulse goggle. Drs. Ian Curthoys and Michael Halmagyi and others at University of Sydney have published several articles comparing their prototype goggle to the "gold

standard" scleral search coils and investigating various vestibular disorders. This prototype goggle was copied by GN Otometrics in order to deliver a system for head impulse testing which can be manufactured and produced in large quantities.
The formal clinical studies performed utilizing head impulse with visual observation and utilizing the prototype in clinical programs has proven the system to be extremely safe, efficient and effective. Currently, the prototype is in place in 4 major facilities Vestibular Research Laboratory/School of Psychology/University of Sydney, Department of Neurology/Royal Prince Alfred Hospital Sydney Australia, Universitäts Spital Zürich Switzerland, and Sapienza Università di Roma Cassino Italy.

1.3 Description of Device and Intended Application

1.3.1 This information can be found in Section 7 of the English User's manual. The interface box connects via USB and Firewire to the computer which has the OTOsuite Vestibular software loaded on it. The interface box is hardwired to the goggles (used to measure eye movement).
The computer may also be connected to a printer in order to print the test report or a network in order to network work stations together.

Connect the cables to the interface box:
a) USB power cable
b) Firewire data cable



Connect the interface box cables (USB power cable and Firewire data cable) to the computer.

1.3.2 The ICS Impulse is intended to measure, record and display involuntary movement of the eyeball.
It is a reusable device which is non-evasive and non-sterile. The goggle cushion is disposable and single-use with the intention that it will be replaced between patients. The goggles come into contact with the patient's face. For the head impulse procedure or video

recording, the patient wears the goggles for 5 to 10 minutes. Cleaning instructions of the mirror and goggle housing can be found in the English User's manual Section 3.

1.4 Indications and Claims

1.4.1 The typical user of this device is a neurologist, ENT, audiologist, physical therapist or technician supervised by one of the 3 mentioned. This device does not treat or diagnose the patient that is determined by the credentialed physician. The type of patient that this user would be evaluating is anyone who has a complaint of a vestibular nature. This age range could be young adult to elderly (dependent upon if the patient is old enough for the goggles to fit on their head). A list of common diagnoses (this list is not all inclusive) that the physician will conclude based on the test results using this device in combination with case history and other vestibular tests (e.g. dynamic posturography, rotary chair, gate assessment, VEMP testing, MRI, CT-scan etc) are Meniere's, Vestibular Neuritis, Vestibular Affrentation, Benign Paroxysmal Positional Vertigo, Neuroma (i.e., tumor), and Alcohol induced nystagmus (PAN I or PAN II). Without combining the test results with other tests the only conclusion can be made is that the patient is within normal limits or exhibits a reduced vestibular function of the right, left or both horizontal canal(s).

1.4.2 Please refer to Section 9 of the English User's Manual for safety claims.

1.5 Selection of Data Types

1.5.1 The equivalent devices are as follows:

VG-40 goggles (used with the Hortmann CNG Analyzer, Hortmann Vestlab, Hortmann Vestlab 100, Chartr VNG and Chartr 200 VNG) – this device was released in 2005 and utilizes similar video goggles as the ICS Impulse. The Micromedical Vorteq is a competitor system using video goggles for testing the vestibular-ocular reflex.

1.5.2 The literature selected was published data that was used for normative data within the system and articles which include data collected when performing head impulse and using the scleral search coils, visual observation and the video goggle prototype device (one copied to develop the ICS Impulse). All of these articles were found through an internet search (such as Medline, Pub Med) or by direct contact with the researchers. All published articles that references the video goggle prototype has been included as well as, the direct comparison to the scleral search coils. All anatomical research and animal research was excluded. The articles included justify the use of the head impulse test and the validation that the video goggle prototype functions as well as the gold standard (scleral search coils). There were no articles found that were unfavorable.

1.6 Summary of Clinical Data

The following sources of clinical data were used to conduct the evaluation:

a. Sources Generated from Clinical Experience

The attached report has been generated to identify complaint/service and repair events associated with GN Otometrics devices utilizing similar technology as that of the ICS Impulse product (e.g. a video goggle used to record eye movement). Specifically, the report evaluates the number of warranty complaints/repairs as a percentage of total units sold since 2005. The objective of the report is to demonstrate the safety and performance of the technology through analysis of the severity and number of complaint/service and repair events.

None of the GNO devices utilizing the technology in question have ever been associated with a reportable event.

b. Sources Generated from Scientific Literature Search

Applicable Clinical Reference Literature

These textbook chapters and peer-reviewed articles state the clinical efficacy of head impulse testing. The literature includes relevant references to: Head Impulse (aka Head Thrust, Halmagyi Manoeuvre, Halmagyi-Curthoys Test)

• Black RA, Halmagyi GM, Thurtell MJ, Todd MJ, Curthoys IS. The active head-impulse test in unilateral peripheral vestibulopathy. Arch Neurol 2005; 62(2):290-293. See Attachment 01.

Equipment Used: *Scleral search coils (this was the only diagnostic device for head impulse testing available at the time of publication)*

Article Type: *Peer-Reviewed Journal Article – Scientific Research Design*

Number of Subjects: *6*

Position: *Favorable*

Contains: Active and passive head impulses were performed on six adult subjects, using scleral search coils, who had been diagnosed with total unilateral deaffrentation more than 2 years after vestibular schwannoma (acoustic neuroma) surgery. The results were that while ipsilesional VOR was abnormally low with both active and passive head-impulses, with active head-impulses (mean +/- SD 0.38 +/- 0.1), it was almost twice that seen with passive head-impulses (0.18+/- 0.02). The results show that active head-impulses, unlike passive head-impulses, are not an effective clinical way to show severe unilateral loss of vestibular function.

Excerpts: "The Head-Impulse Test, since we first described it 15 years ago, has become an accepted part of the neurological examination. It detects severe unilateral loss of semicircular canal (SCC) function clinically and is important for the assessment of dizzy patients in the emergency department and physician's office."

Reviewer's comments: This publication used scleral search coils. The ICS device was not used in this study. Active (and unpredictable) head impulses are more sensitive than passive (and predictable) ones. It does not address the accuracy of the HIT for any specific disorder.

• *Della Santina, CC, Cremer PD, Carey JP, Minor LB Comparison of head thrust test with head autorotation test reveals that the vestibulo-ocular reflex is enhanced during voluntary head movements. Arch Neurol 2002: 128:1044-1054. See Attachment 02.*

Reviewer's comments: This study also supports the greater sensitivity of the active and unpredictable head impulses. The ICS device was not used in this study. It does not address the accuracy of the HIT for any specific disorder.

• *Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. Arch Neurol 1988; 45(7):737-739. See Attachment 03.*
• *Halmagyi GM, Weber KP, Aw ST, Todd MJ, Curthoys IS (2008) Impulsive testing of semicircular canal function. In: Kennard C, Leigh RJ (eds) Using Eye Movements as an Experimental Probe of Brain Function. Progress in Brain Research, volume 171, chapter 3.6, pp 187-194. (Elsevier, Amsterdam, in press). See Attachment 04.*

Reviewer's comments: The above 2 references lend general support to the use of the VOR but not to the ICS device specifically or even to a specific method of assessing the VOR.

• *MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy. Neurology 73 (14): 1134-1141. See Attachment 05.*

Reviewer's comments: This study validated the use of video instead of scleral search coils for the acquisition and analysis of the VOR. The two methods were nearly identical. The method did involve the use of active/unpredictable head impulses. Although the device used is somewhat similar to the ICS device, it did not use the ICS device. The study is based on 16 subjects ages 38-59, one female, with various types of vestibular dysfunction, and 8 healthy volunteers. There is no data on repeatability/reliability such as test-retest reliability, interrater reliability, etc. It is unclear if the data were analyzed using the same methods as in the study of the ICS device included in this application. The norm used for this study came from an earlier study of 12 healthy volunteers using the scleral search coil technique. The result was a mean of 0.81± 0.068 SD so that the lower cut-off was 0.68 – i.e. mean -2SD. In the publication by Weber et al (Neurology 2008;70:454-463), also using scleral coils, in 12 normal volunteers the norm was 0.98± 0.063 although this was affected by acceleration rate. It is similar to the norm for horizontal impulses reported by Cremer, Halmagyi et al in Brain (1998;121:699-716) in which the norm was 0.9± 0.1. It is therefore reasonable to use the proposed norm as the "default" norm as long as the source of the norm in provided. The device does allow for user modification of the norm.

• *Newman-Toker DE, Kattah JC, Alvernia JE, Wang DZ. Normal head impulse test differentiates acute cerebellar strokes from vestibular neuritis. Neurology 2008; 70(24 Pt 2):2378-2385. See Attachment 06.*

Reviewer's comments: The study did not use the ICS device. The remainder of the publications did not use the ICE device and only lend general support to the role of

assessing VOR by various methods in the evaluation of various peripheral vestibular and/or central nervous system disorders.

2.2 Safety

2.2.1 Since 2005, approximately 1550 units of the following products utilizing the similar technology as the ICS Impulse have been sold: Hortmann CNG Analyzer, Hortmann Vestlab, Hortmann Vestlab 100, Chartr VNG and Chartr 200 VNG. Approximately 235 warranty complaint/service and repair events (i.e., complaints, service, and/or repair activities) have occurred during the same time period.

2.2.2 Analysis of those events shows that none involved a high severity (i.e., actual or potential user or patient injury or death). Furthermore, there have been no reportable events associated with the aforementioned products.

2.2.3 In addition, the risk analyses for the VG-40 video goggles and ICS Impulse video goggles have been reviewed to confirm that there are no potential hazards that have an associated high risk priority number (RPN). All risks have been adequately mitigated and the complaint/service and repair data attached supports the accuracy of the current risk analyses for these devices.

Combined with the performance analysis above, we believe that the Company's risk management and post-market surveillance activities clearly indicate that the technology utilized by the ICS Impulse is both safe and effective.

Reviewer's comments: The data provided refer to the use of devices other than the ICS device. However the overall technologies and methods are similar enough that there is reasonable assurance that the ICS device safety will not be an issue.

Labeling

From user manual page 20 – page 121/1854 of pdf.

3.1 Patient preparation

Warning - A head impulse should not be performed on patients with a neck injury, or on patients who have been told by their physicians to limit or avoid neck movement activity.

Prior to testing, provide the patient with these general recommendations:

- *If in doubt, consult with a physician about the possible side effects of stopping a particular medication.*
- *Stop tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test.*
- *Continue medications that are vital, such as insulin, heart medications, seizure medications, and possibly antidepressants.*
- *No alcohol for 48 hours before testing.*
- *Do not wear make-up around the eyes.*

• *Wear comfortable clothing.*

Reviewer's comments: The above instructions suggest that a non-physician user should have the patient stop "tranquilizers, sedatives, or vestibular suppressants for at least 48 hours before the test" and "If in doubt, consult with a physician about the possible side effects of stopping a particular medication." No prescribed medications should be stopped without a specific order of a licensed physician.

3.9.3 Gain and Test Remarks

Gain is the ratio of the eye movement velocity to the head movement velocity. The Gain window displays gain values along the Y axis and corresponding peak velocities along the X axis. (The peak velocity is the maximum velocity for each of the 175 samples representing that particular head impulse test).

Data in the white zone is within normal limits. Data in the light gray area indicates unilateral loss. Data in the dark gray area indicates bilateral loss. (Boundaries defined according to normative data research[1]) All right and left gains are averaged. The mean and standard deviation (σ)are displayed below the gain graph.

Note · Refer to 3.12 Head impulse options 53 to change the boundaries for normative data.

3.9.4.1 Understanding the 2D analysis

Within Normal Limits

This is an example of head impulse data that is within normal limits. Looking at the Gain graph the data points are all within the normal range (in the white area with a gain of 0.6 to 1). The head data shows very well performed head impulses and the eye data shows a vestibular ocular reflex (A) that mirrors the head velocities. There may be a few catch-up saccades. Downward spikes are a result of spontaneous nystagmus (B).

3.9.5.1 Understanding the 3D analysis

Normal

This is an example of head impulse data that is within normal limits. Looking at the Gain graph the data points are all within the normal range (in the white area with a gain of 0.6 to 1). The head data shows very well performed head impulses and the eye data shows a vestibular ocular reflex (A) that mirrors the head velocities. There may be a few catch-up saccades. (Downward spikes are a result of spontaneous nystagmus (B).

Reviewer's comments: Gain of 0.6 to 1.0 is considered normal.

Gain Normative Data *Set cutoff lines*
Unilateral - the cutoff line between normal and unilateral loss
Bilateral - the cutoff line between unilateral loss and bilateral loss

Restore Defaults - restores the normative data cutoff values as documented in published data and recommended by Otometrics. [a]

[a] MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: Diagnostic accuracy in peripheral vestibulopathy. Neurology 73 (14): 1134-1141.

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(b)(4) Deficiencies

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Lawrence D. Rodichok
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K122550/S1

GN Otometrics

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January 21, 2013

U.S. Food and Drug Administration
Center for Devices and Radiological Heath
Document Mail Center - WO66 Room G609
10903 New Hampshire Avenue
Silver Spring , MD 20993-0002

Attention: Document Mail Clerk

Re: Traditional 510(k) Notification: ICS Impulse K122550 Requested Additional Information

CDROM Copy: A CDROM e-copy of this submission is hereby provided to serve as the second copy of the submission. It is in Acrobat format.

(b)(4) Deficiencies

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(b)(4) Deficiencies

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(b)(4) Deficiencies

[redacted]

Respectfully submitted,

Respectfully submitted,

Daniel Kamm

Daniel Kamm, P.E.
(Regulatory Engineer, Submission Correspondent)
Enclosures

Attachment Index

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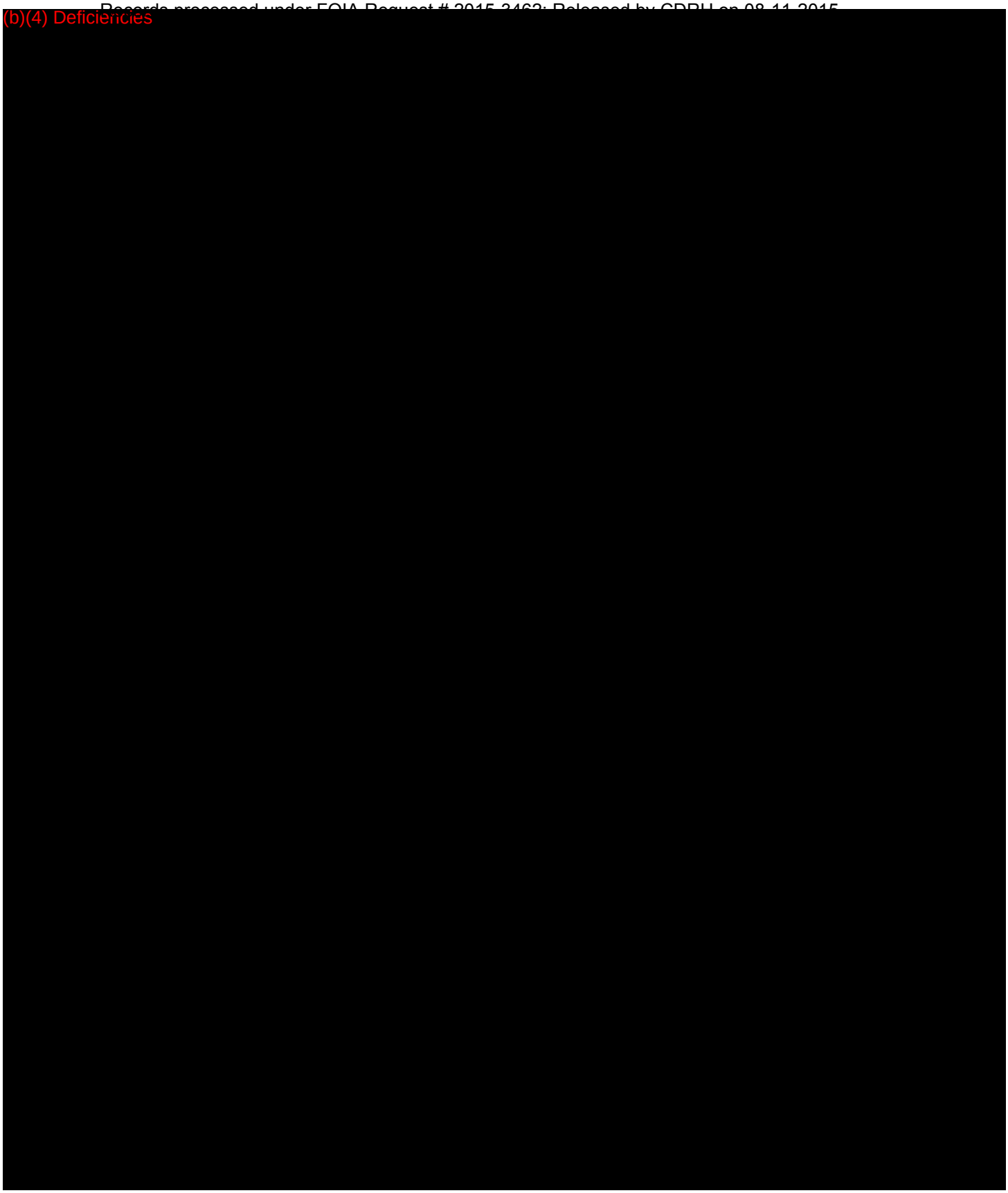

Respectfully submitted,

Respectfully submitted,

Daniel Kamm, P.E.
(Regulatory Engineer, Submission Correspondent)
Enclosures

Attachment Index

Attachment 1. Revised Indications for Use Statement

Indications for Use

510(k) Number (if known): K122550

Device Name: ICS Impulse

Indications For Use:

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Prescription Use X **AND/OR** **Over-The-Counter Use ___.**
(Part 21 CFR 801 Subpart D) **(21 CFR 807 Subpart C)**

(PLEASE DO NOT WRITE BELOW THIS LINE-CONTINUE ON ANOTHER PAGE IF NEEDED)

Concurrence of CDRH, Office of Device Evaluation (ODE)

Attachment 2. Revised 510(k) Summary

510(K) Summary, 510(k) K122550

Submitter: GN Otometrics A/S
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Contact: Dan Sansonetti, Manager of Research and Development
Date Prepared: January 13, 2012

1. **Identification of the Device:**
   Proprietary-Trade Name: **ICS Impulse**
   Classification Name: Unclassified, Product Code: LXV, Device: Apparatus, vestibular analysis
   Common/Usual Name: Vestibular testing device

2. **Equivalent legally marketed devices**: Micromedical Technologies Inc. Vorteq, K891008 and Micromedical Technologies Inc. VisualEyes K964325.

3. **Description of the Device**: The device is a combination of hardware and software. The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight but it must be secured tightly to the head to minimize goggle slippage. The software records and displays the information obtained during what is known as a "head impulse test" The basic head impulse test starts with the tester standing behind the patient who is wearing the goggles. While the patient is asked to stare at the fixation dot placed on a projection surface in front of them, the tester rotates the patient's head horizontally through a small angle (about 10-20 degrees) in a brief, abrupt and unpredictable manner, varying the direction and the velocity. The goggles collect both head and eye data. The gyroscope measures the velocity of the head movement (the stimulus). The high-speed camera captures the image of the eye. The OTOsuite Vestibular software processes the head velocity data and velocity data for eye movement (the response). Simultaneous displays of the data for head movement and for eye movement allow the clinician to determine if the response is within normal limits or not.

4. **Indications for Use (intended use):** The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements. (Prescription use).

5. **Safety and Effectiveness, comparison to predicate device**. This device has the same indications for use as the predicate device but employs different technology to accomplish the same tasks.

6. **Description of Testing:** The device passed UL Electrical Safety testing and EMC testing. Software validation and risk analysis was performed. Clinical testing compared test results to Scleral Search Coils test results. ICS Impulse adequately meets the design requirements and acceptance criteria.

7. Substantial Equivalence Chart

Characteristic	Micromedical Technologies Inc. Vorteq, K891008 and VisualEyes K964325.	ICS Impulse
Intended Use:	VORTEQ® is designed to provide information about the Vestibular Ocular Reflex (VOR) in patients with dizziness or balance problems.	The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.
Configuration	VORTEQ® utilizes an angular velocity sensor mounted directly to the VisualEyes™ FireWire Binocular Goggles. With the VisualEyes™ Monocular Goggles, the angular velocity sensor is attached to the back of the goggles headband for VORTEQ® testing	The patient wears a pair of lightweight, tightly-fitting goggles on which is mounted a very small, very light, very fast, fire-wire video camera and a half silvered mirror. This transparent mirror reflects the image of the patient's eye into the camera. The eye is illuminated by a low-level infra-red light emitting diode which is not visible to the patients. A small sensor on the goggles measures the head movement. The whole goggle system is lightweight (only about 60g) but it must be secured tightly to the head to minimize goggle slippage
Photo		
Interfaces	Firewire for Camera Data: Not specified	Firewire for Camera USB 2 for Data
Electrical safety	Electrical Safety per UL2601 - IEC-60601.	Complies with UL 60601-1, IEC 62471, 1st.ed., IEC 60825-1, 2.ed. UL Listed
EMC	Not specified	IEC 60601-1-2: 2007
Calibration	Performed using a Digital Lightbar, LCD projector or Secondary monitor. Stimulus +/- 15 degrees for horizontal and +/- 10 degrees for vertical.	Performed using 2 Built-In Laser (2) Class II @ +/-7.5 degrees.

8. Conclusion: After analyzing bench testing, safety, EMC, software, and clinical validation testing we conclude that the ICS Impulse is as safe and effective as the predicate device, and has essentially the same indications for use, thus rendering it substantially equivalent to the predicate device.

Attachment 3. Revised Users Manual Pages

1 Introduction

Congratulations! You are now the owner of a sophisticated new ICS Impulse system developed in collaboration with Drs. Ian Curthoys, Michael Halmagyi and others at University of Sydney.

To assist you in getting the most out of the ICS Impulse system, we have included this user manual and a training video. We hope you find it easy to use and that your use of the incorporated tips and information results in improved data collection accuracy as it relates to your assessment of vestibular-related disorders, test results reporting, and patient information retrieval.

1.1 Intended Use

The ICS Impulse System is used in the assessment of the vestibular-ocular reflex (VOR) by measuring, recording, displaying, and analyzing eye and head movements.

Note · *The ICS Impulse System is intended to be used only by qualified medical personnel.*

1.2 Intended User

This manual describes the use of the device in combination with the software. Readers are assumed to have prior knowledge of the medical and scientific facts underlying the procedure. For this reason, the examination methods are mentioned only to the degree that is necessary for a correct, safe application of the ICS Impulse System.

You can find more information in the ICS Impulse training video or at **www.headimpulse.com**.

3.1 Patient preparation

Warning · *A head impulse should not be performed on patients with a neck injury, or on patients who have been told by their physicians to limit or avoid neck movement activity.*

Prior to testing, provide the patient with these general recommendations:

- No alcohol for 48 hours before testing.
- Do not wear make-up around the eyes.
- Wear comfortable clothing.

3.2 Goggle preparation

3.2.1 Cleaning and maintenance

The ICS Impulse System equipment does not require preventive maintenance. Observe the following recommended guidelines regarding cleaning and maintenance.

- Keep the instrument clean and as free of dust as possible. Remove dust using a soft cloth or brush.
- If required, clean the goggle housing and interface box using a damp cloth moistened with a mild detergent and water solution. Do not allow any moisture to get inside the goggles.

Caution · *Never spray or immerse the goggle components with the cleaning solutions. This could contaminate the electronics and/or optics.*

- If required, clean the mirror using the supplied cleaning cloth. The presence of fingerprints on the mirror surfaces could cause inaccurate pupil detection.

Attachment 4 Gyroscope Data Sheets

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Attachment 5 Camera Data Sheets

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Attachment 6. Additional Risk/Hazard analysis

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Attachmnet 7 Laser Data Sheet

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