

DISABILITY MEDICINE

The Official Periodical of the American Board
of Independent Medical Examiners

Editorial Board

ABIME
American Board of Independent Medical Examiners

Contents

Editor-in-Chief

Mohammed I. Ranavaya, MD, MS, FFOM,
FRCPI, FAADEP, CIME

Assistant Editors

Thomas A. Beller, MD, FAADEP, CIME

Steven Mandel, MD

Rebecca McGraw-Thaxton MD, CIME

Editorial Advisory Board

Alan L. Colledge, MD, CIME

J. True Martin, MD, CIME

Stan Bigos, MD

Gordon Waddell, FRCS, *Glasgow, UK*

Charles N. Brooks, MD, CIME

Pete Bell, MD, CIME

Peter Donceel, MD, *Belgium*

Sigurdur Thorlacius, MD, PhD, *Iceland*

Clement Leech, MD, *Ireland*

Jack Richman, MD, *Canada*

Cristina Dal Pozzo, MD, *Italy*

Richard Sekel, MD, *Australia*

William H. Wolfe, MD, MPH, FACPM, CIME

Charles J. Lancelotta, Jr., MD, FACS

Kevin D. Hagerty, DC, CIME

Sridhar V. Vasudevan, MD

Frank Jones, MD, CIME

Alan K. Gruskin, DO

William Shaw, MD

Jan von Overbeck, MD, *Switzerland*

James Becker, MD

Altus J. Van der Merwe, MD, *Switzerland*

Jerry Scott, MD, CIME

Chet Nierenberg, MD, CIME

Charles Clements, MD, CIME

Kendal Wilson, DO

John Shimkus, MD

Brian T. Maddox, *Managing Director*

PAGE

Editorial:

The Discipline of Disability
Medicine: Independent Medical
Examiner vs. Attending Physician
.....

34

Original Research Article

Prevalence of Disability
in Iceland 2001 and Comparison
with Other Nordic Countries
.....

36

CME Question File
.....

41

Book Review

Spinal Cord Medicine
.....

42

Presurgical Psychological
Screening in Spinal Cord
Stimulator Implants – A Review
.....

43

Post-Traumatic Vertigo –
A Review
.....

49

Book Review

The Guides Casebook - 2nd Edition
ISBN #1-57947-264-8
.....

62

Book Review

Carpal Tunnel Syndrome and
Other Disorders of the Median
Nerve (2nd Edition)
.....

63



EDITORIAL:

The Discipline of Disability Medicine: Independent Medical Examiner vs. Attending Physician

BOARD OF DIRECTORS

Thomas A. Beller, MD, CIME
President
Kansas City, Missouri

Mohammed I. Ranavaya, MD, CIME
President Elect/Secretary
Chapmanville, West Virginia

Alex Ambroz, MD, MPH, CIME

Donald L. Hoops, PhD
Prospect Heights, Illinois

John D. Pro, MD, CIME
Kansas City, Missouri

Brian T. Maddox
Executive Director
Barrington, Illinois

BOARD OF ADVISORS

Robert N. Anfield, MD, JD
Chattanooga, TN

J. Frederic Green, MD
Moline, IL

Presley Reed, MD, CIME
Boulder, CO

David E. Brown, DC, CICE
Charlottesville, VA

Rebecca McGraw, MD, CIME
Morgantown, WV

Richard Sekel, MB,BS, CIME
Redfern, NSW Australia

Niall J. Buckley, BSc, MD, CIME
Halifax, NS, Canada

Gordon Waddell, DSc, MD, FRCS
Glasgow UK

Lester L. Sacks, M.D., Ph.D., FACOEM
Laguna Niguel, CA

Randall L. Short, DO, CIME
Chapmanville, WV

Robert L. Nierenberg, MD, CIME
Honolulu, HI

Alfred Taricco, MD, FACS
Manchester, CT

Pieter Coetzer, MB ChB., BSc., CIME
Capetown, South Africa

Clement Leech, MD, DMA
Dublin, Ireland

Stan Bigos, MD
Seattle, WA

William S. Shaw, MD, CIME
Denver, CO

David P. King, BSc, MD, CIME
Yellowknife, NT Canada

Jan Von Overbeck, MD
Zurich, Switzerland

Sigurdur Thorlacius, MD, PhD
Reykjavik, Iceland

Yat Cho Chu, MBBS, MSc(O.M.)
(London), CIME
Kowloon, Hong Kong

Datuk Dr. P. Krishnan, MBBS, DIH, FFOM
RCPI, CIME
Seremban, Malaysia

Anthony C.K. Thoo, MBBS, MSc Occ.
Med., FAFOM, CIME
Royal Park, SA, Australia

Mohammed Azman B. Aziz Mohammed,
MBBS, LFOMRCP, CIME
Kuala Lumpur, Malaysia

Rahman Gul, MBBS, MFOM RCP
(Ireland), CIME
Ipoh, Malaysia

Direct communication between attending/treating physician and Independent Medical Examiner sometimes become necessary, especially by phone, to clarify some issues when there is a question of medical necessity of a procedure or test as it relates to the claim. I have had to do this occasionally myself as an Independent Medical Examiner. I recall many instances when attending physicians became defensive and as the conversation progressed, I could hardly miss the derision in the attending physician's tone. Many IME doctors have experienced that they are regarded as company docs or even worse, (hired guns) responsible for the increasing physician hassle factor and participants in unconscionable cost containment strategies by workers comp carriers and insurance companies, which make obscene profits on the back of injured persons and their healthcare providers.

I have generally found though, that after explaining to the colleague on the other side in a non-threatening way as to what independent medical examiners do; and listening to them about what they are proposing to do, usually the outcome is satisfactory to both sides knowing more about each other and understanding different perspectives and hopefully each possessing a better understanding of each other and the role of independent medical examiner play in claim resolution process.

The discipline of Disability Medicine is like other medical disciplines. There is a core body of knowledge, which provides a scientific basis upon which the principles of impairment and disability evaluations are based. The practice of disability medicine demands knowledge, skill and ability in these core principles and can not be done without clinical experience and appropriate credentialing. The responsibilities of both the attending physician and the independent medical examiners are to the individuals whether a patient to the attending physician or an examinee of the independent medical examiner, and to the society at large. The domain of disability medicine is not only science but also include business and law.

However the most common communication between attending/treating physician and Independent Medical Examiner is not direct one but rather an indirect one via the medical record generated by the attending physician, which can be valuable in assessing an individual's claim of impairment or disability. It would be an Independent Medical Examiner's dream come true if the information in the attending physician's record were completely accurate, consistent and reliable. Unfortunately, this is often not the case.

No one will argue that in North America we are fortunate to have the finest medical care provided by most systematically trained and dedicated health care providers the world has ever known. Unfortunately for the society at



large, there is a tremendous pressure placed on these fine folks to exaggerate services performed to insure that the patients receive benefits and to some degree optimize reimbursement for the provider.

We are all aware of anecdotal evidence that suggests that the more diagnoses that are listed, the more likely the chance of payment being made and some providers may have taken this to creative heights. Obviously, where the payor is a liability company such as automobile insurance carrier or workers' compensation carrier, there is temptation to maximize disability. This has led some to conclude that there is an environment, which does nothing to correct the pressure to exaggerate medical illness and a system where

much money is available for those who are intentionally exaggerating the need for medical services or indicating the performance of a service in a workers' compensation claim which was not work-related, a phenomenon I regard as cost shifting.

In this regard, Independent Medical Examinations tends to offer a more neutral type of opinion; however, this system is also not without its abusers. We all know of some physicians who do nothing but plaintiff work and others that exclusively perform examination for defense. This ends up in some cases a shouting match between two equally qualified physicians described by some as dueling doctors phenomenon.

So then, what are we supposed to do? I believe that the quality and integrity of

the medical records coupled with the intellectually honest and objective analysis of all the information made available to the independent medical examiner is the appropriate course of action. It is imperative to evaluate the source of attending physician's information and weigh the veracity of these records by many factors. These should include sifting through data from variety of sources and appropriate second opinion consultation from colleagues with appropriate specialty. Hopefully the American Board of Independent Medical Examiners and the Journal Disability Medicine will continue to contribute to this interdisciplinary approach.

Mohammed I. Ranavaya, M.D., M.S., FRCPI, FFOM, FAADEP, CIME
Editor in Chief

General Information – Disability Medicine, Volume 3, Number 2

Disability Medicine is an educational publication of the American Board of Independent Medical Examiners (ABIME) intended to provide a forum for dissemination of a wide range of responsible scholarly opinions, research, and practice relevant to Independent Medical Examiners and disability medicine. The editors and all contributors to *Disability Medicine* enjoy a full latitude in expressing opinions on the subjects presented to better inform the readers. The views expressed by contributors are not necessarily those of the American Board of Independent Medical Examiners. *Disability Medicine* does not endorse or sponsor any articles, but rather presents them for the information and education of its readers.

Disability Medicine is published four times a year by the American Board of Independent Medical Examiners (ABIME, 111 Lions Drive, Suite 217 Barrington, IL 60010). American Board of Independent Medical Examiners claims copyright to the entire contents. All rights reserved. Reproduction in whole or in part without written permission is strictly prohibited.

Subscriptions: The subscription is free for one year to all currently certified diplomats of American Board of Independent Medical Examiners. The standard 1-year subscription rate in the USA and Canada is \$50 US per year (individual copies \$15 each); for outside

North America the one-year subscription rate is \$75 US per year (individual copies \$25). The two-year subscription rate is \$80 US in USA and Canada, and \$125 outside North America.

Inquiries Regarding Advertising and Subscriptions. Should be sent to Mr. Brian T. Maddox, Managing Director Disability Medicine, ABIME, 111 Lions Drive, Suite 217, Barrington Illinois 60010, phone (800) 234-3490.

Manuscripts should be addressed to Mohammed I. Ranavaya, MD, Editor-in-chief of *Disability Medicine*, RR 4, and Box 5C, Chapmanville, WV 25508. Fax (304)-855-9442, e-mail: mranavayamd@newwave.net.

Reprints, except special orders of 100 or more, are available from the authors.

Advertising Policy: *Disability Medicine* may carry advertisements either in *Disability Medicine*, as attachments to *Disability Medicine* or associated with the mailing of *Disability Medicine*. There may also be advertisements in association with *Disability Medicine* on ABIME web site dedicated to *Disability Medicine*. Services or products eligible for advertising will be relevant to the practice of disability medicine. These will general fall into the categories of: Independent Medical Examination services providers, Other relevant paramedical service organizations, publishers or professional textbooks or professional journals,

employment related advertisements, other services or products which are of particular interest to the practice of Independent Medical Examination services or disability medicine.

The appearance of advertising in *Disability Medicine* is neither an American Board of Independent Medical Examiners guarantee nor an endorsement of the product or service, or of the claims made for any product or service by the advertiser or manufacturer. The American Board of Independent Medical Examiners reserves the right to decline any submitted advertisement, in its sole discretion.

The Editor of *Disability Medicine* determines the eligibility of advertising and the placement of advertisements in *Disability Medicine* for products intended for diagnostic, preventive or therapeutic services. Scientific and technical data concerning a product or services safety and efficacy may be required before advertising is accepted for publication. In order to determine eligibility of advertising for books or journals a copy will usually be requested for review.

Instructions to Authors May be obtained by writing to *Disability Medicine* for a copy and outlines requirements for acceptance of manuscripts.



Original Research Article

Prevalence of Disability in Iceland 2001 and Comparison with Other Nordic Countries

Sigurdur Thorlacius^{1,2)}, Sigurjón Stefánsson¹⁾, Stefán Ólafsson³⁾

1)State Social Security Institute of Iceland, 2)Faculty of Medicine, University of Iceland, 3)Faculty of Social Sciences, University of Iceland

Correspondence:
Sigurdur Thorlacius
Tryggingastofnun ríkisins
Laugavegur 114, 150 Reykjavík, Iceland
Tlf: (+354) 5604400
Fax: (+354) 5604461
e-mail: sigurdur.thorlacius@tr.is

Keywords: disability, benefits, social security

Summary

Objective: To determine the prevalence of disability in Iceland according to gender and age and compare the results with data from the other Nordic countries.

Material and methods: The study includes all those receiving disability pension on the 1st of December 2001 as ascertained by the disability register at the State Social Security Institute of Iceland.

Results: On the prevalence day there were 11.235 persons living in Iceland who had applied for disability pension and satisfied the medical criteria; 10.263 satisfied the medical criteria for full disability pension, 6.085 women (59.3%) and 4.178 men (40.7%); 972 fulfilled the criteria for partial disability pension, 636 women (65.4%) and 336 men (34.6%). The prevalence of full disability pension was 5.5% and of partial disability pension 0.5%. Disability was

significantly more common among women than men ($p < 0.0001$) for full as well as partial disability pension and increased with age. Disability was most commonly associated with mental and behavioral disorders and diseases of the musculoskeletal system.

Conclusion: The percentage of the total population receiving full disability pension in Iceland is slightly higher than in Denmark but considerably lower than in Finland, Norway and Sweden. The following main explanations for this difference are suggested: the level of allowance and organization of the social security system is different in Iceland as compared with the other Nordic countries; the Icelandic unemployment level is lower and work participation higher, especially in the upper age groups in Iceland.

Introduction

The State Social Security Institute (SSSI) manages the national social insurance in Iceland, according to the National Social Security Act and the National Social Assistance Act. The national social insurance covers pensions, sickness insurance and accident insurance. The social insurance is mainly financed by taxes. The SSSI administers approximately 20% of the national budget of Iceland.

Disability pension is granted according to paragraphs 12 and 13 in the National Social Security Act.⁽¹⁾ A full disability pension (degree of disability at least 75%) is granted to those between the ages of 16 and 66 years suffering from considerable and prolonged disability. A partial disability pension (degree of disability 50-74%) is granted to those who have less pronounced disability or considerable expenses due to disability. This study is based on information on disability beneficiaries living in Iceland



on December 1st 2001. The main objective was to determine the prevalence of disability in Iceland and to examine whether there is a relative difference in disability according to gender and age.

Material and methods

Information was obtained from the disability register of the SSSI, which contains for each disability beneficiary his name, date of birth, gender, degree of disability and main diagnoses according to the International classification of diseases.⁽²⁾ From this register was extracted information on degree of disability, first (main) diagnosis, age and gender of all disability beneficiaries living in Iceland on December 1st 2001. Information was obtained on the number of inhabitants in Iceland aged 16-66 years on this same day and their age distribution.⁽³⁾ On the basis of this information the percentage of the population receiving disability pension in Iceland was calculated according to gender and age groups. Statistical significance was determined with the chi-square test.⁽⁴⁾

Results

On December 1st 2001 there were 10.263 individuals living in Iceland who had applied for and fulfilled the medical criteria for full disability pension (had disability assessed as being at least 75%), 6.085 women (59.3%) and 4.178

men (40.7%). In addition there were 972 who satisfied the criteria for partial disability pension (had disability assessed as being 50-74%), 636 women (65.4%) and 336 men (34.6%). Thus, there were totally 11.235 living in Iceland who had applied for and fulfilled the medical criteria for disability pension. The prevalence of full disability pension was 5.5% and of partial disability pension 0.5%

Disability was significantly more common among women than men ($p < 0.0001$) for full as well as partial disability pension.

Table 1 shows the age distribution of those who satisfied the medical criteria for full disability pension. The prevalence of full disability pension increased with age. Men and women showed a different age distribution ($p < 0.0001$). In people younger than 30 years the prevalence of disability was equal among men and women, but in people over 30 disability was more prevalent among women than men.

Table 2 shows the first (main) diagnosis among recipients of full disability pension according to diagnostic groups. This is the diagnosis the insurance physician at the SSSI regards as most important in his disability evaluation. Among women the most prevalent disease group was diseases of the musculoskeletal system, whereas in men mental and behavioral disorders were most prevalent. In both genders the two

most prevalent disease groups were mental and behavioral disorders and diseases of the musculoskeletal system; these two groups accounted for approximately 60% of cases.

Discussion

On December 1st 2001 the prevalence of full disability pension was 5.5%. In comparison with the other Nordic countries this ratio was in Iceland higher than in Denmark (4%) but lower than in Finland, Norway and Sweden (8-9%).⁽⁵⁾

In Iceland full disability pension was significantly more common among women than among men. As can be expected, the prevalence of disability increased with age. The most prevalent medical reasons for granting disability pension were mental and behavioral disorders and diseases of the musculoskeletal system and connective tissue. This is in line with the situation in Norway, Sweden and Great Britain.⁽⁶⁻⁸⁾

The age distribution of recipients of disability pension was different in Iceland compared with the other Nordic countries.⁽⁹⁾ Disability pension was relatively more prevalent under the age of 40 and relatively less prevalent over the age of 50 in Iceland as compared with Denmark, Finland, Norway and Sweden. Research in the Nordic countries has shown that receivers of disability pension have greatly



increased in numbers during the last decades, especially after 1970. The increase is greatest in Finland, Sweden and Norway, but less marked in Denmark.⁽¹⁰⁾ The increase is particularly marked among people in the higher age groups, notably above the age of 50. This development has also been observed in other Western-European countries and in fact in some of the countries on the continent of Europe it is greater than in the Nordic countries, for example in France, Holland, Germany and Britain. For males the increase is particularly significant in the age group 50-64, but among females it primarily shows up among those 60 years of age and older.⁽¹¹⁾ In Iceland, however, a comparison of the prevalence of disability pension in 1976 and 1996 did not show an increase, in fact it had slightly decreased.⁽¹²⁾

A number of explanations for differing development in the number of disability pensioners over time and across countries or regions have been put forth. One of the most common is the idea that pressure from the labor market leads to an increased number of disability pensioners. This explanation assumes that in periods of increasing unemployment rates and increasing competition among firms the more vulnerable part of the labor force, for example those with restricted abilities or physical inhibitions, are pushed out from the labor market. This is assumed to happen either directly and

consciously (for example by government induced policy aiming to lower measured unemployment rates) or indirectly and without any coordinated actions (for example by management which is pressed to perform well in tough competitive environments and therefore aims to hold on to the better able part of their labor force in times of downsizing and restructuring).^(13, 14)

Another explanation is that an inviting social security system with generous rights and benefits induces people who have some ailments or inhibitions to apply for disability benefits, even though they could undertake some paid work. In the Scandinavian countries and Finland the medicalized part of the disability evaluation process was reduced as against the social part of the evaluation, which allowed payment of benefits to people in various unfavourable social conditions. When this loosening of entitlement conditions went in hand with a raising of the actual benefit level the conditions for a great increase in the take-up rates of disability pensions were established and actualized when the opportunity conditions in the labor market became more restricted.⁽¹⁵⁻¹⁷⁾ In some continental countries in Europe it is believed that governments allowed or facilitated this development when unemployment increased significantly during the late 1970s and in the 1980s. In Norway and Sweden the above-mentioned changes in the entitlement conditions for

disability pensions (and “early retirement” pensions) had taken place before the advent of the unemployment crisis.⁽¹⁰⁾ Related to this explanation is the hypothesis that an improved management of the social security systems and increased knowledge among the public about rights and entitlements have had an independent impact on the number of disability pensioners.

The third kind of explanation of increasing rates of disability pensioners has to do with the internal organization of the social security systems themselves, i.e. the access of people with varying degrees of work inhibitions or ailments to the various benefit categories. Thus one can imagine that it may vary between countries how feasible it appears to members of the public to receive unemployment benefits, disability benefits, sick pay or direct financial assistance from the local commune. The financial amounts may also differ between these different types of support. This can of course vary greatly between countries and may be related to cultural factors, which affect perceptions of stigma and shame. On the whole it may be expected that those categories of support, which provide the higher benefit, may tempt more applicants. It is not always self-evident where in the support system each individual applicant should most correctly belong or be dealt with. Therefore it should be important to look



at other possibilities in the support system for people who might qualify as disability pensioners when varying rates of this group are explained between countries or regions and different periods. Part of the explanation may reside in other related support routes than disability pensions being more or less accessible and generous (for example the interaction with the unemployment benefit system may be particularly important in this respect).

The fourth kind of explanations is the eventual health or disability evaluation, i.e. the medicalized one. Many causes of disability are nearly completely conditioned by the prevalence of diseases and physical deviations, which should be similarly prevalent in countries at comparable levels of development. On the other hand it is of course possible that the prevalence of disability and physical wear and tear may be conditional upon the predominant industries in the said regions or countries. Some industries provide worse working conditions and are more prone to work related accidents and unhealthy pollution, both for the employees and neighboring populations. This may cause some difference in the long-term rates of disability between regions and/or countries. Fishing, farming and mining are for example cases in point which often are related to specific dangers of accidents and health risks.

Lastly we shall discuss how the above mentioned possible explanations may relate to the prevalence of disability pension receivers in Iceland as against the other Nordic countries.

If we firstly look at the theory of increasing pressure from the labor market it is quite clear that the Icelandic labor market has for most of the post-war period been characterized by excessive demand for labor.

Unemployment rates have been exceptionally low and employment participation rates have been high and the average work-week has also been longer than in the other Nordic countries, and in fact longer than in other Western countries. This indicates that the pressure from the labor market on the social security system has been very low in Iceland in this period.⁽¹⁸⁾

There has however been a change in this from 1992 to 1995, when unemployment increased to unprecedented levels (max. about 5%). Still unemployment in Iceland was low by international standards and in fact it lowered again gradually from 1995 to 2000.^(3, 9, 19) So ample employment opportunities in the Icelandic labor market have possibly given people with restricted employability more chances than elsewhere to pursue a working career instead of applying to the social security system for provision. This may have helped to keep the prevalence of disability pension relatively low.

This may thus explain the relatively low prevalence of disability pension in Iceland. On the other hand it is not particularly compatible with the higher rate among people under the age of 30 compared to the other Nordic countries. That might however be explained by the characteristics of the Icelandic social security system. The provision in the system for sick pay is one part of the overall system which might relieve pressure from the disability pension division, as regards those who lose ability to work temporarily due to sickness or accidents. This would of course only work if the access to sick pay was easy and the benefit more tempting than the disability pension. That however is not at all the case in Iceland since the Icelandic system for sick pay provides a much lower percentage of former pay than prevails in the other Nordic countries. Similarly the unemployment benefit has also been low in Iceland compared to these other countries.^(9, 20) On the whole the social security system in Iceland has not in general been likely to tempt people particularly to draw their subsistence from it instead of working. But the disability pension has perhaps been more tempting than the sick pay provision or the unemployment benefit and that may explain a relatively high rate among younger people.

A similar explanation may apply to the fact that the rate of disability pension is relatively low in Denmark, i.e. that other



parts of the social security and support system take a larger share of the potential applicants. For example the unemployment rate has been high in Denmark from the early 1970s compared to the other countries, except Finland.

In sum, as regards the significantly higher rate of disability pensioners for people over the age of 50 in Scandinavia and Finland, as against Iceland, a considerable part of the explanation has to do with the greater possibility to use the social security system, and especially the disability pension, to relieve tension due to unemployment in the labor market. That policy has not been applied in Iceland, and at any rate

the employment situation has been better in Iceland for most of the post-war period. In fact the employment rate for people over the age of 50 has been higher in Iceland than in any other Western country.⁽²⁰⁾

References

1. The National Social Security Act of Iceland (law no. 117/1993)
2. International Statistical Classification of Diseases and Related Health Problems. Tenth revision. World Health Organization, Geneva, 1994.
3. Information from Statistics Iceland.
4. Bland M. An Introduction to Medical Statistics. Oxford University Press, 1995.
5. Social Protection in the Nordic Countries 1999. Scope, expenditure and financing. Nordic Social-Statistical Committee, 8, Copenhagen, 2001.
6. Folketrygden – Nökkeltal 2001. Oslo, Rikstrygdeverket, www.trygdeetaten.no.
7. Social Insurance Facts. The National Social Insurance Board, Stockholm, www.rfv.se.
8. National statistics. Department for Work and Pensions, London, www.dwp.gov.uk
9. Social Protection in the Nordic Countries 2000. Scope, expenditure and financing. Nordic Social-Statistical Committee, 8, Copenhagen, 2002.
10. Kolberg JE, Hagen K. The Rise of Disemployment. In Kolberg JE (editor): Between Work and Social Citizenship. New York, M. E. Sharpe, 1992:114, 122.
11. Jacobs K, Kohli M., Rein M. The Evolution of Early Exit. I Kohli M, Rein M, Guillemard AM, Gunsteren H (editors): Time for Retirement. Comparative Studies of Early Exit from the Labor Force. Cambridge, CUP, 1991:44-49.
12. Thorlacius S, Stefánsson SB, Ólafsson S, Rafnsson V. Changes in the prevalence of disability pension in Iceland 1976-1996. Scandinavian Journal of Public Health 2002; 30: 244-248.
13. Halvorsen K. Arbeid eller trygd. Oslo, Pax Forlag, 1977.
14. Berglind H., Olson-Frick H. Förtidspensionering. Stockholm, Statens Offentliga Utredningar, no. 88, 1977.
15. Kjønstad A. Arsaker til økninger i antall uførepensjonister. Social trygd, nr. 10, 1976:272-279.
16. Hedström P. Disability Pension: Welfare of Misfortune? In Erikson R, Hansen EJ, Ringen S, Uusitalo H (editors): The Scandinavian Model. Welfare States and Welfare Research. New York, M. E. Sharpe, 1987.
17. Wadensjö E. Disability Pensioning of Older Workers in Sweden. A Comparison of Studies Based on Time-Series and Cross Section Data. Stockholm, SOFI, Meddelande, 1985.
18. Ólafsson S. The Rise and Decline of Work in the Welfare State. In Kolberg JE (editor): Between Work and Social Citizenship. New York, M. E. Sharpe, 1992:36-76.
19. OECD, Economic Surveys: Iceland. OECD, Paris, 1990-1997.
20. Ólafsson S. Íslenska leidin. The State Social Security Institute of Iceland, Reykjavik 1999

Table 1.
Percentage of age group of persons fullfilling the medical criteria for full disability pension in Iceland

December 1st 2001

Age in Years	Women	Men	Both genders
16-29	1.7	1.7	1.7
30-39	4.4	3.2	3.8
40-49	7.1	4.9	6.0
50-59	11.2	7.1	9.1
60-66	22.7	12.7	17.8
16-66	6.6	.4	5.5

Table 2.
First (main) diagnosis according to the International classification of diseases (ICD) among recipients of full disability pension in Iceland

December 1st 2001

	Women	Men
Diseases of the musculoskeletal system and connective tissue	2024 (33.3%)	668 (16.0%)
Mental and behavioural disorders	1936 (31.8%)	1762 (42.2%)
Diseases of the nervous system and sense organs	591 (9.7%)	486 (11.6%)
Diseases of the circulatory system	332 (5.5%)	416 (10.0%)
Injuries	276 (4.5%)	313 (7.5%)
Diseases of the respiratory system	182 (3.0%)	84 (2.0%)
Congenital mal/ deformations and chromosomal abnormalities	149 (2.4%)	161 (3.8%)
Endocrine, nutritional and metabolic diseases	146 (2.4%)	76 (1.8%)
Malignant neoplasms	122 (2.0%)	69 (1.7%)
Diseases of the skin and subcutaneous tissue	72 (1.2%)	21 (0.5%)
Diseases of the digestive system	69 (1.1%)	18 (0.4%)
Infections	52 (0.9%)	43 (1.0%)
Disease of the genitourinary system	47 (0.8%)	24 (0.6%)
Other diagnoses	87 (1.4%)	37 (0.9%)
Total	6085 (100%)	4178 (100%)



CME Questions File

Question #1

On December 1st 2001 the State Social Security Institute of Iceland administered approximately:

- a. 5% of the national budget
- b. 10% of the national budget
- c. 20% of the national budget
- d. 40% of the national budget

Question #2

On December 1st 2001 in Iceland:

- a. full disability pension was significantly more common among men than women but partial disability pension more common among women than men
- b. full disability pension was significantly more common among women than men but partial disability pension more common among men than women
- c. both full and partial disability pension were more common among men than women
- d. both full and partial disability pension were more common among women than men

Question #3

The prevalence of full disability pension On December 1st 2001 in Iceland:

- a. was equal among men and women in people younger than 30 years, but in people over 30 it was more prevalent among women than men
- b. was equal among men and women in people younger than 30 years, but in people over 30 it was more prevalent among men than women

- c. was higher among men than women in people younger than 30 years, but in people over 30 it was equal among men and women
- d. was higher among women than men in people younger than 30 years, but in people over 30 it was equal among men and women

Question #4

On December 1st 2001 in Iceland full disability pension was most commonly associated with:

- a. mental and behavioral disorders in women and diseases of the musculoskeletal system in men
- b. mental and behavioral disorders in men and diseases of the musculoskeletal system in women
- c. diseases of the circulatory system in men and diseases of the musculoskeletal system in women
- d. diseases of the musculoskeletal system in both men and women

Question #5

On December 1st 2001 the percentage of the total population receiving full disability pension in Iceland was:

- a. considerably higher than in the other Nordic countries (Denmark, Finland, Norway and Sweden)
- b. considerably lower than in the other Nordic countries
- c. slightly higher than in Denmark but considerably lower than in Finland, Norway and Sweden
- d. approximately equal to that in Finland, but considerably lower than in Denmark, Norway and Sweden

Question #6

On December 1st 2001 disability pension was:

- a. relatively more common under the age of 40 and relatively less common over the age of 50 in Iceland as compared with the other Nordic countries (Denmark, Finland, Norway and Sweden)
- b. relatively more common over the age of 50 and relatively less common under the age of 40 in Iceland as compared with the other Nordic countries
- c. more common in all age groups in Iceland than in the other Nordic countries
- d. less common in all age groups in Iceland than in the other Nordic countries

Question #7

At the close of 2001, in Iceland, the full disability pension prevalence was:

- a. 4.5%
- b. 5.5%
- c. 6.5%
- d. 9.5%

Answer Key for previous CME questions, Disability Medicine Vol 3, #1

The Questions can be found on page 26, Vol 3, #1 and were based on articles in previous issues of Disability Medicine.

Answers: 1.C, 2.B, 3.A, 4.C, 5.A, 6.A, 7.D, 8.D, 9.B, 10.B



Book Review

The Guides Casebook - Second Edition, ISBN #1-57947-264-8

Authors: Brigham DR, Ensalada LH, Talmage, JB

Publisher: AMA Press, 2002

Reviewer: John Walden, MD

Marshall University Joan C. Edwards School of Medicine

Division of Disability Medicine

1600 Medical Center Drive, Suite 1500

Huntington, WV 25701

Jefferson Medical College

The Guides Casebook, now in its second edition, owes its existence and usefulness to two realities in the world of permanent impairment evaluation: first, at the completion of medical school and residency, most physicians are woefully ill-prepared to perform an impairment evaluation because they have received little if any training in this area; second, the application of the basic information contained in the AMA Guides is not necessarily intuitive. The Guides Casebook serves as a complement to the AMA Guides to the Evaluation of Permanent Impairment by helping to bring physicians up to speed in the skills needed to perform proper exams and by addressing the vagaries and internal inconsistencies in the wording in the AMA Guides.

The editors chose 68 cases exemplifying clinical problems encountered in evaluating impairment. Each case is handily cross-referenced by sections, tables, figures and page numbers to the AMA Guides, Fifth Edition and Fourth Edition. Not surprisingly, the bulk of cases deal with commonly encountered issues related to the spine, upper

extremity and lower extremity.

Additional cases mirror the chapters in the AMA Guides and fall under the broad headings of Cardiovascular System, Respiratory System, Digestive System, Urinary and Reproductive System, Skin, Hematopoietic System, Endocrine System, Ear-Nose-Throat (and related structures), Visual System, Central and Peripheral Nervous System, Mental and Behavioral Disorders and, lastly, Pain.

Physicians new to the process of impairment evaluation will definitely gain from reviewing the cases, each of which reinforces how the Guides are applied. More experienced evaluators will benefit from a heightened appreciation of subtleties in the process as, for example, in instances where the Guides allows a considerable range of percentages and the evaluator is expected to exercise clinical judgment to select an appropriate specific percentage of the whole person.

The contributors and reviewers for this Second Edition are widely recognized for their impairment evaluation skills.

Fortunately, each case is presented in a straightforward, plain-English manner, so that confusion and uncertainty seem to melt away through the imposition of logic that flows from the discourse of these highly experienced evaluators.

More and more, the process of impairment evaluation strikes this reviewer as something akin to learning a foreign language: anything and everything seems to add to one's ability to master the nuances. The Guides Casebook, Second Edition, is a great aid in the passage from being essentially tongue-tied to fluency.



Presurgical Psychological Screening in Spinal Cord Stimulator Implants - A Review

Kenneth J. Devlin, M.A., Mohammed I. Ranavaya, M.D., MS., Charles Clements M.D., Jerry Scott M.D., Rabah Boukhemis M.D.
Division of Disability Medicine
Joan C. Edwards School of Medicine, Marshall Univ., Huntington WV

Editor's Note:

Implantable technologies are an alternative treatment option to chronic pain patients with specific conditions. The long-term success of these treatments is dependent on a number of variables including the surgeon, the procedure, and the selection process. Presurgical psychological screenings (PPS) as described in this article offers a way to improve outcome success rates.

Overview

This article provides an overview of the presurgical psychological screening (PPS) process as it applies to spinal stimulator implants and implanted drug delivery systems. The process has applicability to most spine surgeries with implications for bariatric surgeries. Readers interested in developing applied skills in the evaluation and treatment of these patients are referred to "The Psychology of Spine Surgery" (Block, A.R.; Gatchel, R.J.; Deardorff, W.W.; & Guyer, R.D., 2003) and Presurgical Psychological Screening in Chronic Pain Patients (Block, A.R., 1996) for a comprehensive discussion of the topic area.

Surgical Outcome

Many chronic pain patients believe surgical intervention leads to dramatic

improvement from their suffering when they contrast their hopes for surgical rescue to past disappointments from non-surgical interventions. In reality, spine surgery leads to about 50% reduction in pain levels and commensurate increases in function at the end of a protracted rehabilitation process. Approximately 25% of patients experience no pain relief (Block, A.R.; Gatchel, R.J.; Deardorff, W.W.; & Guyer, R.D., pg. 3,2003). These are general statistics and may vary on the basis of the surgeon and specific problem-procedure combination. Surgical outcome is also correlated with the surgeon's selectivity of patients on the basis of many factors to include those addressed in this article.

In addition to arriving at a surgical prognosis, our program also prevails upon the PPS process to assist in improving the success rate for surgical outcomes with the specific procedures noted. This additional feature is typically through specific psychological treatment recommendations and services. The basis of this information is based both on Block's (Block, A.R.; Gatchel, R.J.; Deardorff, W.W.; & Guyer, R.D., 2003), (Block, A.R., 1996) work and

the evolving implementation of this process for the past twelve years at our interdisciplinary pain center.

Overview of the Implantable technology

Spinal Cord Stimulators

Spinal cord stimulators (also know as dorsal column stimulators) are neurostimulation systems sometimes used for problems including: radiating pain; continued pain following back surgery; and Complex Regional Pain Syndrome (CRPS) previously termed Reflex Sympathetic Dystrophy. This type of system applies precisely controlled low-voltage electrical stimulation to the spinal cord through one or more precisely placed implanted "leads." The stimulation distorts the pain signal so that the patient feels a tingling sensation replacing part of the pain sensation. The system consists of one or more leads, a titanium implantable pulse generator which houses the battery and a programmer. The lead(s) is tunneled to the implanted pulse generator. An alternative system connects the lead(s) to an external transmitter which houses a 9-V battery. The latter system is usually reserved for



patients whose use pattern would rapidly drain implanted batteries, requiring frequent transmitter replacements. The patient has a handheld device that turns the unit off and on and can increase or decrease the signal. Broader stimulation parameters are adjusted by the clinician's office programmer.

Drug Delivery Systems

Implanted opiate catheters and intrathecal infusion pumps typically require far less quantities of drugs than is required by other parenteral routes to achieve equal pain reduction. Warfield and Fausett (Warfield, C.A., & Fausett, H.J., pg. 352, 2002) note that common side-effects such as nausea, sedation, and respiratory depression are reduced. This treatment approach is used in nonmalignant pain syndromes and, increasingly, for cancer pain. An intrathecal infusion pump system consists of a catheter placed in the intrathecal space tunneled to the implanted programmable pump. Refills are accomplished through a central diaphragm in the pump and require a needle stick to the patient. Dosing is controlled by external telemetry.

Health and Rehabilitation Psychology

The theoretical basis of successful PPS for chronic pain patients is based on the biopsychosocial model. Concisely, this model states that an individual's

experiences and responses are the product of the complicated interaction of basic biological mechanisms, individual differences, and the greater social context within which these forces operate in each individual. This perspective requires consideration of the longitudinal context of each patient in order to appreciate the clinical meaning of that patient's functioning at any point in time. An informative treatment of this model is found in Turk (Turk, D.C., & Gatchel, R.J., pp 6 - 23, 2002) and is a necessary area of knowledge to competently negotiate the many paths of prognoses and treatments required to effectively manage surgical candidate-patients.

Typically, psychologists who specialize in the PPS process and related treatment services are theoretically oriented to health and/or rehabilitation psychology. There is considerable overlap in these two areas of focus. Common to both is a strong expectation that the patient plays a strong, active role in their own treatment and health management as a member of the treatment team. Unless a patient accepts ownership of the pain problem they are unlikely to believe they can gain a sense of control over the pervasive influence of the problem in their life (Caudill, M.A., pg. 8, 2002).

Health psychology addresses both preventative services as well as pre-existing problems and is based on the biopsychosocial model. Clinical focus is

on health and short-term interventions. Common treatment approaches include brief cognitive-behavioral interventions in conjunction with active communications with other providers involved in the patient's care. This theoretical orientation is the emerging model in primary care settings, especially in rural areas. Health Psychology, Division 38 of the American Psychological Association, offers a wealth of information and support for practitioners interested in this area (American Psychological Association).

Rehabilitation psychology focuses on assessment and treatment of people who experience injuries and health-related disabilities incorporating much of what is described above under health psychology. It is represented by Division 22 of the American Psychological Association (American Psychological Association). This area of specialization requires expertise in areas to include: biobehavioral aspects of injury; disability; rehabilitation processes; specialized assessment and treatment strategies; and related ethical-legal issues (Boll, T.G. Ed., pg. 570, 2002).

An additional emerging specialty area is occupational health psychology which is targeted at developing, maintaining and promoting the health of employees and the health of their families (Quick, J.C., Tetrick, L.E., pg. 4, 2003).

Health and rehabilitation psychological interventions may occur in the absence



of a psychological/psychiatric diagnosis or focus exclusively on a medical problem even when a psychological diagnosis is present. In this latter case, new CPT health codes are applicable. These codes should not be used when treatment addresses psychological diagnoses. Alternately, psychology services can address a combination of medical and psychological diagnoses.

Patient Selection

In our pain center both the physician performing implants and the screening-treating psychologist work within the same treatment team. Implants are considered after alternative, less invasive treatment options have been exhausted. Due to the mature clinical relationship within the team, the potential pre- and post-surgical treatment options are broader and better controlled, allowing for greater variations and flexibility in psychological recommendations. For similar reasons, the physician performing the implants has refined the clinical judgment factors determining who may be an appropriate candidate before the PPS process begins. This combination of these factors is judged contributory to improved treatment outcomes.

Screening Format

Block (Block, A.R., 1996) has established a comprehensive protocol for PPS's. His protocol includes: 1. review of medical

records.; 2. semi-structured interview to identify surgical risk factors.; 3. observation of pain-related and other behaviors.; 4. psychological testing to identify additional risk factors.; and 5. determination of surgical prognosis. He defines the ultimate goal of PPS to arrive at a surgical prognosis, rating screened patients to have a "good", "fair", or "poor" surgical outcome.

The final prognosis for surgical outcome from Block's model is based on risk factors from three categories: medical risks (9 factors); psychological risks (8 factors); and judgment factors (5+ factors). Low psychological and medical risks suggest a good surgical outcome. A high level of risk in one domain and low on the other suggests a fair prognosis. High risk levels in both domains are associated with a poor prognosis. Standard recommendations in this model include: Clear for surgery, no psych necessary.; Clear for surgery, post-op psych.; Hold, pending psych intervention.; Do not operate, conservative care only.; Do not operate, recommend discharge.

Effective implementation of this requires a basic knowledge of the proposed surgical procedure, its level of destructiveness, and typical iatrogenic risks associated with the procedure. Knowledge of the general efficacy of the procedure is also necessary.

Psychological Factors

The psychological, physical and judgment factors put forth by Block (Block, A.R., 1996) serve as a foundation for PPS recommendations. In our experience factors including severe depression or anxiety, concurrent substance abuse and characteristics similar to those found in borderline personality disorders represent caution flags. These problem areas should show treatment response prior to the implant if they are not considered a contraindication.

Our center aggressively treats sleep disturbances to the extent possible prior to the implant. Patients with chronic pain are at higher risk for sleep problems than the general population. Factors that contribute to this risk include the injury itself, nocturnal pain, medication, decreased activity, lack of daily schedule, weight gain, uncertainty about the future, and problems with mood and anxiety. Other sources (Dement, W.C., pg. 144, 1999) validate our experience that increased sleep deprivation is associated with increased pain, especially myofascial pain.

Specific sleep disorders considered in the evaluation process include sleep apnea; restless legs syndrome (RLS), and periodic limb movement disorder (PLMD). These problems have been detected in many cases and are typically treated in house with the exception of sleep apnea which is referred to a



pulmonary sleep specialist. In addition to impacting implant outcome, the presence of these disorders increases the degree to which an individual is chronically sleep deprived. Sleep deprivation is associated with significant risk in occupational settings as well as general activities of daily living such as driving and family relationships.

A patient's treatment outcome expectations are critical in implant success. Poor outcome usually occurs when the patient's outcome expectations significantly exceed treatment results. The treatment team must assure that the patient does not believe s/he will be the same as they were pre-injury and pain free. One of our cases involved an implanted stimulator in a patient who was high functioning pre-injury and repeatedly professed to understand the treatment outcome limitations. After the implant he admitted that he "secretly" believed he would be "cured." In this case the actual pain relief far exceeded clinical projections. However, the patient was bitterly disappointed and developed severe depression resistant to treatment and required inpatient psychiatric treatment.

If the recommendation is for pre-surgical psychological intervention, target criteria for clearance should be established and agreed to by all involved parties. For a patient with severe pain it is more reasonable to

target improvement in depression and sleep than to require remission of these problems. The continuing severe pain is partially responsible for the maintenance of these other problems and is the target of the implant. At some point the clinician has to make a realistic judgment as to when maximum treatment response has been reached given the ongoing pain and limitations and plan to continue progress in all areas post-implant. This position has been successfully argued with third party payors.

Psychological Testing

Psychological testing serves the PPS process similarly to how it serves other assessment processes. There are limits to the time that can be allocated to a clinical interview as well as the ability of a clinician to objectively compare an individual's profile to a group of similar patients or the population at large. Additionally, patients often respond differently on test questions compared to similar questions posed in the interview. The resolution of these discrepancies leads to necessary refinement of the clinical picture.

Psychometrics should assist the clinician to refine clinical investigation areas to include: mood and anxiety states; anger; the individual's attitude towards his/her own pain; substance abuse; and a past history of abuse. Test results should be normed to relevant

populations and include validity measures.

The Minnesota Multiphasic Personality Inventory (MMPI) and its revision, the MMPI-2 continues to be widely used throughout clinical settings to include the PPS process. There is a substantial amount of retrospective research relating MMPI profiles to spinal surgery outcome (Block, A.R.; Gatchel, R.J.; Deardorff, W.W.; & Guyer, R.D., pgs. 79-99, 2003). The psychologist's familiarity with both the instrument and related research is critical since the MMPI was not originally designed with these purposes in mind.

The Battery for Health Improvement (Bruns, D., Disorbio, J.M., 1996) and the revised BHI-2 differ from many other test instruments by having been developed to address the psychomedical connection central to the issues relevant in the PPS process. It is designed to address areas of demonstrated importance in the diagnosis and treatment of this patient population. The test structure addresses three general factor categories: psychological; environmental; and somatic. The instrument is double normed for profile comparison to patients physical; rehabilitation and community populations. The design helps to reduce the problem of test results assigning excessive psychopathology to patients.

The Behavioral Assessment of Pain (BAP) (Tearnan, B.H. & Lewandowski,



M.J., 1992) is also an assessment instrument developed to measure the disability associated with chronic pain. The BAP measures over 35 factors relevant to the management of chronic pain patients. It includes validity scores and a disability index. The normative sample used in the development of the instrument was composed of sub-acute and chronic pain patients.

Patient Education

Successful treatment outcome is heavily dependent on adequate education of both the patient and members of the patient's family who are functionally involved in the patient's care. It has previously been stated that patients are expected to actively participate in their own care in this treatment model. This cannot occur without the patient being reasonably knowledgeable about all aspects of the process to include the role of each discipline, medications, procedures, progress curves and treatment goals.

Staff who work in medical settings often have a diminished ability to appreciate a patient's level to understand common medical jargon. This is often complicated by a patient's reluctance to request clarification. Consequently, it is more effective to offer excessive detail in the simplest of terms than to presume a patient understands because they are nodding their head in agreement.

Our center employs "teaching sheets" with all procedures. These are given to each patient and define each procedure with related outcome expectations. However, it is still necessary for a staff member to review each procedure in detail and, often, repeat this process a number of times. Teaching is usually followed by a non-threatening "quiz" about what the patient or family member understands.

Family members can significantly contribute to the success or failure of treatment. With the patient's permission, involved family members should be brought into the treatment early in the process and taught how they can positively contribute to progress and what may be counterproductive. Family members are also an excellent source of cross validation for problem areas or progress. In the area of sleep disorders, they are a necessity since the patient cannot self-observe during sleep.

It is equally important that family members as well as patients appreciate potential benefits and limitations of implants. Problems such as sexual dysfunction are likely to change little on the basis of the implant alone. Patients and family members should be informed that a broad range of clinical interventions will continue post-surgically to further minimize related symptoms and limitations.

Summary

Implantable technologies are an alternative treatment option to chronic pain patients with specific conditions. The long term success of these treatments is dependent on a number of variables including the surgeon, the procedure, and the selection process. PPS as described offers a way to improve outcome success rates.

Psychologists involved in this area of specialization typically work from a biopsychosocial model and are likely to have health and/or rehabilitation psychology orientations. These orientations bridge the gap between traditional medical and psychological perspectives and embody the integration required for the PPS process.

A research based PPS process is available to better guide psychologists screening candidates for implantable technologies as well as other spine surgeries. Psychological testing should be incorporated. Appropriate psychometric options include both historical and contemporary alternatives.

When there is an established relationship between the screening-treating psychologist and the physician performing the implant, the accuracy of surgical prognoses can increase and interventional options can be expanded for patient candidates. These refinements can be the basis of a



favorable prognosis in candidates who would otherwise be considered to have a poor surgical prognosis.

Patient education including involved family members is a salient factor in the design of a comprehensive program. Patients and family members should have a realistic perspective of both treatment outcomes and treatment limitations. Post-surgical treatment strategies should be part of a comprehensive plan.

A successful balance of the factors known to influence outcome in these surgical cases can usually be achieved. Psychologists involved in the evaluation and treatment of these patients can expect to contribute to an improved

patient selection process and increase the success rate for patients that might have had a marginal prognosis without appropriate psychological services. The general goals in all chronic pain management are to assist the patient to both better manage pain and to maximize function in all areas of life. These goals define the essential reason for the partnership between the psychologist, the medical team, and the patient. Including social, occupational, and family factors in this process closes the biopsychosocial circle.

REFERENCES

American Psychological Association; 750 First Street, NE, Washington, DC
 Block, A.R., (1996); Presurgical Psychological Screening in Chronic Pain Syndromes, Lawrence Erlbaum Associates, Mahwah, NJ

Block, A.R.; Gatchel, R.J.; Deardorff, W.W.; & Guyer, R.D., (2003); The Psychology of Spine Surgery; American Psychological Association, Washington, D.C.
 Boll, T.G. Editor-In-Chief (2002); Handbook of Clinical Health Psychology; American Psychological Association, Washington, DC
 Bruns, D., Disorbio, J.M., (1996); Battery for Health Improvement; National Computer Systems, Minneapolis, MN
 Caudill, M.A. (2002); Managing Pain Before It Manages You, Revised Ed.; The Guilford Press, NY, NY
 Dement, W.C., (1999); The Promise of Sleep; Delacorte Press, Random House Inc., NY, NY
 Minnesota Multiphasic Personality Inventory University of Minnesota Press, Minneapolis, MN
 Teaman, B.H. & Lewandowski, M.J. (1992); The Behavioral Assessment of Pain Questionnaire: The development and validation of a comprehensive self-report instrument. American Journal of Pain Management, 2, 181-191.
 Turk, D.C., & Gatchel, R.J. Editors, (2002); Psychological Approaches to Pain Management: Second Edition; The Guilford Press, NY, NY.
 Warfield, C.A., & Fausett, H.J. Editors, (2002); Manual of Pain Management Lippincott Williams & Wilkins, Philadelphia, PA
 Quick, J.C., Tetrick, L.E., Editors (2003); Handbook of Occupational Health Psychology; American Psychological Association, Washington, DC

We do our job so you can do yours!



A SOURCE ATTORNEYS USE IS NOW AVAILABLE TO YOU.

We compliment your IME practice by:

- **Preparing “critical fact” medical chronologies containing information applicable to YOU. This facilitates your exam, expedites your narrative report and helps you prepare for testimony.**
- **Outlining patient injuries, treatment response, complications, missing records and patient non-compliance.**

**MIDWEST
 MEDICAL LEGAL RESOURCES, INC.
 219-365-3113**



Post-Traumatic Vertigo - A REview

Charles G. Maitland, M.D.

Introduction

Balance may be defined as the ability to stand and move safely and securely despite destabilizing forces in the environment. Balance and equilibrium (spatial orientation) are paramount for activities of daily living such as transitions from sitting to standing, walking, reaching for objects, bending, etc. To maintain normal equilibrium, an individual must accurately identify his or her position in space with respect to the earth's gravitational forces and the immediate surroundings. An individual must also be able to identify and react to external forces with controlled muscle activity and to move in a world fraught with destabilizing circumstances. There exists in each of us a set of neural systems that act in a coordinated fashion to provide sensory information about ourselves and our immediate environment.

Three major sensory systems act in concert to provide information that assures equilibrium. The visual system provides information about the position and motion of objects in our outside world. The proprioceptive system recognizes changes in joint position in the limbs. Finally, the vestibular system conveys information about head and eye movement and orientation of head

and body position in space relative to gravity.

Although each sensory system provides a unique pattern of sensory input, the information provided is essentially redundant, providing a safety net in the event one of the systems fails. Sensory information provided through these primary sensory systems transcends multiple brain structures ultimately arriving at the parietal cortex from where commands responsible for maintenance of balance, guidance of eye and head movements, reaching behavior, and limb coordination are generated. While each system contributes to control of balance and equilibrium, the vestibular system exclusively evolved for this purpose. Of the three primary sensory systems, the vestibular system alone functions automatically and generally without an individual's conscious awareness. It is this fact that probably accounts for the difficulties individuals who suffer vestibular injury have in describing the nature of their symptoms.

The vestibular apparatus responsible for measurement of acceleration and gravitational information is particularly susceptible to head injury by virtue of its location. The labyrinthine cavities providing information of head movement and orientation of head in

space are housed in compact temporal bone at the base of the skull. The vestibular labyrinth is divided into sets of mechanical receptors. The otolithic receptors are responsible for detection of linear head movements (linear acceleration) and orientation of the head relative to gravity, while the semicircular canals, of which there are three in each inner ear, are responsible for detection of angular head movements and for corrections of eye positions in space following head movement. In each of these end organs, the sensory epithelium contains hair cells that convert a mechanical stimulus produced by displacement of those cells to neural discharges that travel through the vestibular nerve into the lower brain stem where they enervate a number of structures, most notably the vestibular nuclei, which serve as relay stations providing information throughout the central nervous system. Both sensory apparatus are stimulated by most natural behaviors, i.e. bending, walking, shifts of position, etc. The information they provide ultimately result in spinal reflexes, which correct for displacement of the body in space, and for ocular reflexes, which correct for eye position in response to head movement.¹

As there are two labyrinthine systems, one housed in either side of the temporal bone of the skull, their



function is coupled in specific ways. The process of reciprocal inhibition, i.e. when head movement excites one set of semicircular canals, it automatically produces inhibition of the partner canals in the opposite labyrinth, is operational. It is this balance between related inputs from two sides that drive vestibular reflexes and allows for normal balance and coordination of head and eye movement. Any disease process which selectively injures or produces asymmetry in balance between signals arising from these two sides will result in clinically reflexive imbalance in the form of aberrant eye movements producing subjective symptoms of vertigo and/or oscillopsia or vestibular spinal dysfunction resulting in loss of balance and equilibrium.²

Labyrinthine Concussion

Symptoms of vertigo, dizziness, and disequilibrium with or without hearing loss or tinnitus often follow blows to the head or may be the sequelae of whiplash injury. In the absence of clinical and radiologic evidence of fractures in the base of the skull, vestibular and auditory symptoms have been ascribed to labyrinthine concussion.³ Paradoxically, impact to the delicate labyrinthine membranes housed in the otic capsule at the skull base may be more severe when there is no fracture line to dissipate the force of the blow. Physiologic disruption of labyrinthine function associated with microscopic

hemorrhages in both the labyrinthine and cochlear systems may be present. The symptoms that arise following such injury may give rise to greatest disability in terms of return to normal activity and work.⁴ Oosterveld and colleagues report that symptoms of disequilibrium were secondary only to headache and neck pain in cases of whiplash injury and were present in a high percentage of patients, most commonly following so-called "rear end" collisions.⁵ Higher levels of trauma may result in damage to the sensory organ of the inner ear, the 8th nerve sub-serving the end organ, or its central vestibular connections, including both brain stem and cerebellum.

In any event, the symptoms that arise from such injury are frequently difficult for individuals to clearly describe and/or understand. Generally, nonspecific terms, i.e. dizziness, lightheadedness, swimmy-headedness, and unsteadiness are used to describe the non-physiologic sensations of motion and instability individuals experience. Vertigo, however, is a very definite sensation and is defined as an hallucination of motion of the environment and/or a sense of motion or turning of an individual within the environment; when present it is indicative of vestibular imbalance.

Dizziness, however, is a more generic non-specific term that may reflect a sense of disorientation due to injury to

any number of body parts. Head trauma, with or without temporal bone fracture, acceleration/deceleration non-contact head and neck injuries, and barotrauma are all significant mechanisms of injury and may coexist. Injury to the inner ear may be divided into cases with or without temporal bone fracture. Fractures of the temporal bone are usually classified as longitudinal or transverse with respect to the long axis of the temporal bone. Seventy to eighty percent of temporal bone fractures are longitudinal, and the remaining twenty to thirty percent are either transverse or oblique.⁶

Classification of temporal bone fractures on the basis of radiographic findings using high-resolution computerized tomographic CT scans yields a higher percentage of mixed fractures.⁷ Longitudinal fractures generally arise as a result of trauma to the temporoparietal area of the skull. A typical longitudinal fracture runs parallel to the anterior axis of the petrous pyramid housing the inner ear and ends in the middle cranial fossa near the foramen spinosum. Most commonly, posterior longitudinal fractures begin in the parietal bone and extend to mastoid air cells where the posterior external auditory canal lies. Such fractures usually produce a tear in the bony external canal skin and adjacent laceration to the tympanic membrane. If hearing loss is present, it tends to be of the conductive type, and



there is otorrhea caused by a dural tear at the tegmen tympani, and ecchymosis is present over the mastoid bones. This discoloration, which frequently appears several days after a fracture, is known as "Battle's sign."

Transverse fractures follow trauma to the orbit or occipital regions, generally the latter, and usually begin at the foramen magnum and extend perpendicular to the long axis of the temporal bone, fracturing the otic capsule and ending in the middle cranial fossa. The fracture may extend medially to traverse the internal auditory canal and petrous apex. With transverse fracture, the skin of the bony external canal and tympanic membrane is intact, but hemotympanum (blood behind the eardrum) is commonly observed. Patients with transverse fractures generally suffer severe sensorineural hearing loss and symptoms and signs of vestibular injury immediately. Dizziness and vertigo are reported symptoms in a high percentage of patients with temporal bone fracture.⁸ Patients with transverse fractures frequently suffer severe sensorineural hearing loss and symptoms and signs of vestibular injury immediately after trauma. Longitudinal fractures also produce similar symptoms but may be delayed and not uncommonly result in injury to the 8th nerve that is incomplete. The symptoms that follow are more generally likely to be classified

as characteristic of so-called "labyrinthine concussion syndrome."

Peripheral vestibular disturbances following blunt head injury produce almost immediately symptoms of vertigo, nausea, vomiting, and disequilibrium. Hearing loss may or may not be present. Vertigo is virtually always, in its acute phase, associated with clinical signs of unidirectional nystagmus (to and fro eye movements) with the fast phase beating in only one direction regardless of the position of the eyes and head in space. Central compensation results in dissipation of nystagmus over a relatively short period of time, whereas correction for vestibular spinal dysfunction reflected in symptoms and signs of disequilibrium and ataxia usually have a more protracted recovery time. Sensorineural hearing loss, if associated with vestibular symptoms, usually affects high frequency ranges, and the defect remains permanent.

Blunt head trauma may also cause brain injury by axonal shearing and microcavitation. The consequence of such injury is both multifocal petechial hemorrhaging and axonal and neuronal disruption. In the context of such diffuse injury, vertigo may arise as a component part but invariably is associated with multiple neurologic signs and frequently alterations of level of consciousness. However, as a general rule, isolated episodes of vertigo

following traumatic brain injury should not be attributed to brain stem injury but rather to injury of the peripheral sensory apparatus and/or the eighth nerve.⁹ Nevertheless, both central and peripheral vestibular abnormalities are frequently identified after minor head injury.¹⁰

Dizziness and vertigo as symptoms are certainly integral parts of the so-called posttraumatic syndrome associated with cerebral concussion. Subjectively, complaints of dizziness following traumatic brain injury are present in roughly fifty percent of patients at least one week after such minor head trauma.¹¹ In addition to symptoms of disequilibrium, lightheadedness, unsteadiness with positional shifts, and vertigo, psychophysiologic sequelae, including motion intolerance, sensory mismatching, chronic nausea, and anxiety, are frequent somatic (e.g. blurred vision) and psychologic (e.g. depression) features of traumatic brain injury.

Benign Paroxysmal Positional Vertigo (BPPV)

Traumatic brain injury, with or without impact, commonly results in the entity known as benign paroxysmal positional vertigo. BPPV is caused by dislocated calcium carbonate crystals (otoconia) that break free from the utricle, where normally they are attached to hair cells that provide gravitational information.



Free-floating calcium crystals migrate from utricle into the semicircular canals, where they can remain either free in any one of three different semicircular canals or attach to hair cells on the sensory receptor organs within a given canal.¹²

First described by Baranay¹³ and later by Dix and Hallpike,¹⁴ the condition has a number of stereotyped features: brief (generally less than one minute) episodes of vertigo, commonly severe, are precipitated by changes in head position, very commonly turning in bed or rising in the morning. Shifts of position during daytime hours in the pitch plan, i.e. looking up overhead, flexion and extension at the waist, etc. are also common precipitants. Affected individuals soon identify the precipitating position and seek to avoid it. They frequently discover that if an episode is intentionally precipitated by assuming a given position and can be endured for a matter of seconds, it will dissipate within seconds to minutes, and further attacks may not recur. It is not uncommon to find individuals who are self-treating based on their own observations. This disorder is often self-limiting and subsides, at times, within a few weeks or months following trauma. Patients may experience remissions and occurrences over a period of years.

In 1962, Schuknect and Ruby¹⁵ proposed that BPPV might be caused by detached utricular debris (otoconia) acting on the sensory receptor (cupula) of the

posterior semicircular canal. Years later, pathologic findings in the temporal bones of patients so affected revealed in fact calcium deposits on the cupula of the posterior canal of an ear that was most undermost during provocative head positions, confirming their hypothesis.

Ordinarily, a gelatinous material attaches calcium carbonate crystals to hair cells arising from the membranous macula of the utricle of the inner ear. Trauma causes fracture of otoconia and dislocation.¹⁶ The term cupulolithiasis implies that dislocated otoconia are attached directly to canal sensory receptors, whereas the term canalisiasis is used to describe the more common occurrence of loose otoconia which occasionally aggregate within one of the canals, generally the posterior semicircular canal, a canal which is dependent when an individual is upright. In greater than 90% of cases, otoconial debris is found to cause symptoms by interference with normal canal function in the posterior semicircular canal. The location of this canal, inferior to the utricle, permits easy entrance of free-floating otoconia.

A shift in position of aggregated crystals generated by head movement creates a plunger effect, which in turn forces fluid movement which acts on the sensory organ of the canal. The consequence is signal generation to a specific pair of eye muscles. Since each canal is yoked

to a single eye muscle in each orbit, the semicircular canal involved in benign positional vertigo can be determined by the direction of the nystagmus elicited during positional testing (see below). Each semicircular canal projects to a specific pair of eye muscles, one in each eye, called yoke muscles, which serve to drive the eyes in a given direction.

Nystagmus from BPPV, known as jerk nystagmus, consists of both a slow displacement phase and a quick recovery phase. A loose bit of otoconia within the canal generates the slow phase, and the quick phases are reflexive and corrective. Depending on canal involvement, nystagmus therefore may either be vertical or torsional, with the slow phase generally directed downwards and a recovery phase in the opposite direction in the case of the posterior semicircular canal, or horizontal with nystagmus being directed toward the underside ear (geotropic) in cases of canalisiasis and away from the ear (ageotropic) in cases of cupulolithiasis.¹⁷ Patients so affected may complain not only of a sense of vertigo and lightheadedness with shifts of position in either pitch or horizontal planes but also complain of difficulties with balance, may exhibit inappropriate motor strategies in response to forces that produce imbalance, and, in our experience, commonly become sensitized to motion in their visual environment resulting in a syndrome known as motion intolerance.



The diagnosis of benign paroxysmal positional vertigo is confirmed by the positional maneuver designed by Dix and Hallpike.¹⁴ In this test the individual patient sits on an examining table with his head turned 45 degrees to the direction of the canal being tested. He is then brought back rapidly so his head assumes a position hanging over the edge of the examining table. The test must be performed not only for position but also for speed, i.e., benign positional vertigo is not only positional but positioning. If canal debris is present, the usual response elicited is the subjective sense of vertigo and dizziness and, objectively, nystagmus that begins after a short latency, fatigues after a brief period of time, and is less obvious or no longer present when the patient is re-challenged.

It is worth noting that nystagmus, seen typically in patients with benign paroxysmal positional vertigo, has been seen by Brandt and colleagues¹⁸ in a rare case of perilymph fistula.

Associated changes in hearing or the development of tinnitus or other auditory complaint during provocative maneuvers, therefore, should alert the examiner to the possibility of a rare alternative diagnosis.

Many individuals in the posttraumatic period may experience spontaneous resolution of benign paroxysmal positional vertigo. Zuccag, et al.¹⁹ have reported that otoconia may dissolve

within inner ear fluid as a possible explanation for this experience.

Alternatively, as patients become more active they may distribute the otoconia throughout the canal in a fashion that does not permit aggregation of enough crystals to cause mass effect. Intuitively, therefore, increased activity including vestibular exercises should help promote such dispersion and reduce symptoms.

Three basic treatment modalities exist for this condition. In extreme cases, surgery is an option. Vestibular neurectomy, i.e. sectioning of a selective vestibular nerve, is no longer preferred because of the complete loss of vestibular function that follows. Alternatively, a number of procedures exist that are designed to plug the offending canal. These include insertion of bone chips and the use of both argon laser, CO₂ laser, and CO₂ laser followed by bone chip plugging.¹²

Patients treated operatively will invariably suffer a sense of sustained vestibular imbalance in the immediate postoperative period which generally resolves over a period of weeks.

However, the risk of sensory neural hearing loss is quite real. Despite this, there is a high rate of success with the major disadvantage being permanent defect in the selected canal function. To date, the most popular treatment consists of maneuvers that move otoconia within the semicircular canals

back into the utricle from which they migrated. The most popular of these maneuvers was introduced by John Epley.²⁰ Based on the theory that BPPV is the result of otoconia floating within a canal, Epley's procedure maneuvers the patient from position to position in such fashion that the utricle becomes dependent to the affected canal, and otoconia relocate back into the utricular system, where, presumably, they are reabsorbed into the macula. The success rate of this maneuver varies from study to study but is approximately 85%-95%. The rate of recurrence is variable, but it seems improbable that the entire population of otoconial debris is removed from the affected canal, and therefore recurrence is not uncommon.¹²

As an alternative treatment, habituation exercises introduced by Brandt and Daroff²¹ theoretically move debris in the semicircular canal in such a fashion as to dissipate plugging or clumping of otoconia, therefore reducing the risk of mass effect and secondary fluid movement within a given semicircular canal. During Brandt-Daroff maneuvers, a patient performs a series of sitting-to-lying maneuvers within the orientation of a specifically diseased canal. These maneuvers can be performed several times per day until vertigo dissipates. A number of variations on the technique exist, but the overall theory is to repeat exercises and maneuvers to a point where vertigo no longer develops. The procedure is frequently successful but



has a major disadvantage in that otolithic otoconia are not displaced from the canal and any resolution must depend on lack of aggregation or dissolution of individual otoconia within the canal. In our clinic, an individual patient who is successfully treated in this manner is instructed to re-institute treatment on his own if he is capable to do so should symptoms of vertigo reoccur.

One of the limiting factors in treatment of positional vertigo in the posttraumatic state is the present of comorbid cervical neck pathology and/or muscle stiffness of the neck or back that frequently prohibits the assumptions of the positions necessary to affect removal of offending debris. Not uncommonly, patients will respond to modified versions of this maneuver, although frequently such maneuvers need to be postponed and the patient cautioned to refrain from movements in planes likely to produce symptoms until such time as the neck pathology is brought under control.

Once canalithic debris has been cleared, conventional vestibular adaptation programs can be introduced to correct for the imbalance that frequently remains as the result of the original utricular injury. Early on before repositioning correction is accomplished, patient education, particularly as it relates to specific positions likely to provoke symptoms

(statistically, most likely in the pitch plane), is pragmatic. Patients should be taught to be extremely cautious when looking above the horizontal plane, avoid looking overhead, and avoid bending at the waist, rather bending at the knees, for example. Canaliasis is probably a lifelong condition.

It is worth noting that although the positioning maneuvers for this condition are highly successful statistically, one of the untoward complications of this treatment is an occasional translocation of otoconial debris from one canal into another, usually from the posterior to the horizontal canals. Therefore, symptoms of recurrent vertigo should not automatically be assumed to be the result of re-accumulation of otoconia in a previously affected canal but will require careful physical examination for accurate diagnosis and treatment. Nevertheless, once the defect is corrected, most individuals are able to perform daily activities without limitation and as a rule are not occupationally limited.

Perilymph Fistula

A perilymph fistula (perilymphatic fistula, labyrinthine fistula) is an abnormal communication between the fluid-filled perilymphatic space of the inner ear and the air-filled middle ear cavity. This abnormal connection may result in leakage of perilymph fluid

through either the round or oval windows separating inner from middle ears or both with clinical consequences of both labyrinthine and cochlear dysfunction.²²

In the normal condition, the vestibular end organ responds to angular or linear acceleration of the head by relative displacement of the cupula in the semicircular canals, and the macula in the case of otolithic organs. This displacement, in turn, causes deflection of stereocilia and kinocilia, and effects a change in the resting membrane potential of these hair cells, producing a change in the firing rate of vestibular afferents via the 8th cranial nerve.²³

Under normal circumstances, pressure in the inner ear tends to follow applied middle ear pressure. As a consequence, equilibration is present at all points and there is no stimulation of vestibular end organ despite changes of pressure within the middle ear.

If, however, there exists another place for venting of pressure in this normally closed system, change in middle ear pressure at the oval window may cause displacement of fluid out the other vent. According to Schuknecht,²⁴ the direction of fluid displacement crosses the ampulla of the semicircular canal, and in this manner fluid flow directly changes the resting membrane potential within the cupula via displacement of stereocilia. Shearing forces produced by velocity-rated flow of perilymph may



also play a role in changing resting membrane potential of hair cells. In these systems, a resultant afferent volleying response might reflect pressure, magnitude, and rate of change.

In any case, perilymphatic fistula represents a volume shift of perilymphatic fluid from vestibule into the middle ear space. This volume shift may be the consequence of an elevated intracranial pressure driving perilymph out of the middle ear or an increased middle ear pressure driving air into the inner ear or raising inner ear pressure and displacing perilymph outward. Penetrating head trauma is a recognized cause of perilymph fistula.²⁵ Goodhill²² proposed two main mechanisms for fistula formation following barotrauma, explosive and implosive. Explosive trauma implies that increased pressure within the cerebrospinal fluid compartment is translated to the perilymphatic space, possibly through the cochlea.

Implosive injury is attributable to excess of external pressure applied to the oval or round window membranes, either via the external auditory canal or the eustachian tube. Examples of explosive barotrauma include various forms of Valsalva's maneuver (e.g. lifting heavy weights, vigorous coughing). Implosive injury might follow tympanic membrane perforations from blows to the head, open hand slaps applied to the

external auditory meatus, or rapid airplane descent. Usually, perilymph fistulas arise spontaneously but are relatively rare occurrences. Spontaneous perilymph fistulas may in fact represent traumatic lesions in patients for whom the traumatic event was sufficiently mild as to remain unnoticed or forgotten.²⁶ However, more commonly they arise in direct temporal relationship to trauma.

Unfortunately, vertigo arising consequent to fistula formation is indistinguishable in form from that which follows the so-called "posttraumatic brain injury complex", the heterogeneous group of labyrinthine and central nervous system disorders discussed previously, among which indistinguishably exists perilymph fistulas. Post-traumatic vertigo may arise from damage to the sensory organ in the inner ear, the eighth nerve subserving the end organs, or the nerve's central vestibular connections.

Lack of specificity of clinical signs and symptoms implicating one etiology compared to the other produces a diagnostic dilemma. For example, phenomenon of canaliasis, secondary to otolithic injury, produces benign paroxysmal positional vertigo (see below). At times, patients with fistula formations also complain of brief episodes of posturally provoked vertigo, sometimes indistinguishable from the more benign condition of canaliasis.

Review of virtually every large series of clinical cases confirmed by exploratory tympanotomy to have perilymph fistula reveals prodromal symptoms of virtually every possible combination of tinnitus, hearing loss, and vertigo.^{27 28 29 30} Typical patients who present following trauma with episodic vertigo frequently suffer progressive sensorineural hearing loss that accompanies each attack. Valsalva's maneuver may precipitate such events, as may shifts in head position. Secondary symptoms of visual mismatching and chronic upright instability frequently aggravated by environmental motion may accompany the primary symptoms.

To further complicate matters, even when a perilymph fistula is suspected, diagnosis is difficult. Rizer and House found on surgical exploration, perilymph fistulas in approximately one-half of their suspected cases.²⁷ Vertigo and dizziness was present in approximately 50% of their cases as primary complaints; and in 50% of individuals with associated hearing loss, complaints of vertigo and dizziness were present two-thirds of the time as a secondary symptom. Symptoms of intermittent vertigo of variable duration, fluctuating or sudden drops in hearing which may or may not recover, and intermittent tinnitus are generally present in 75% of patients harboring fistulae. In short, however, the clinical manifestations of perilymph fistulae in most series are virtually



indistinguishable from those experienced by patients with medical conditions such as Meniere syndrome and transient benign vertigo of migraine.³¹

Lamentably, to date no test exists that reliably predicts the presence of a perilymph fistula. It should, however, be suspected in every individual with head trauma and/or barotrauma who complains of vertigo or fluctuating hearing changes and in cases of suspected canalisia (benign paroxysmal positional vertigo) that remain recidivistic to conventional canalithic repositioning maneuvers. Conventional testing, including audiometry may or may not be helpful. It may show normal patterns; typically it is said to show a flat or downward sloping pattern of hearing loss in cases with surgically demonstrative fistulae, but there are many exceptions to the rule, and variability is the hallmark. Pure-tone loss may be seen in low frequency, high frequency, or be flat. Furthermore, mixed hearing loss may be encountered as traumatic fistulae can also be associated with conductive hearing loss from middle ear pathology. Notably, hearing may be entirely normal in a substantial percentage of patients.³² Electronystagmography is not terribly helpful. It may or may not show positional or spontaneous nystagmus or caloric asymmetry in perhaps 50% of patients. However, the findings would not be specific for perilymph fistula.

Traditionally, the classical fistula test consists of application of both positive and negative pressures administered through a pneumatic otoscope applied to the external auditory canal. During such administration, eye movements are monitored for the development of nystagmus.³³ In at least one series of 51 patients operated on suspected perilymph fistula, such a connection was identified in 26 ears, one-third of those so identified had had a preoperative positive fistula test. This was, however, statistically significant compared with only an 8% false positive rate, suggesting that there is a fairly high specificity but low sensitivity with this test.³⁴

Black and colleagues studied the effect of postural responses in suspect individuals subjected to similar changes of external auditory canal pressure while being monitored on dynamic platform posturography. A test was considered positive if the patient's sway path became phase-locked or if sway amplitude increased more than 50% compared with control patients after positive and negative pressure was applied. In their series, almost all ears so tested showed positive moving platform posturography test results preoperatively, and perilymph fistulae were subsequently identified in 73 of the 75 ears so tested.³⁵ This would suggest extremely high sensitivity to detection of fistulas using this method. The authors, however, point out that

there are potentially unresolved theoretical and practical limitations to such fistula testing. Theoretically, any pathologic condition softening the otic capsule might result in a positive/negative pressure fistula test, so-called "Hennebert's sign." Any condition altering normal middle ear compliance might also alter the dynamics of external auditory canal pressure changes transmitted via tympanic membrane and consequently to inner ear fluids.

Assays of B2 transferrin leveled in spinal fluid, aqueous humor, and perilymph have been developed and reported to be positive in small series in a fairly high percentage of cases. Findings so far have been controversial.³⁶ Apolipoprotein-D as a marker for perilymph has also been reported to have the specificity of 75% but lacks confirmatory study.³⁷

Visualization techniques utilizing CT and magnetic resonance imaging scans can be a value in ruling out other conditions. For example, CT scan with bone windows may demonstrate temporal bone fractures and ossicular dislocation which suggests increase for perilymph fistula. The inner ear, however, is poorly visualized by standard CT technique. The value of MRI imaging at this point again largely excludes alternative diagnoses or explanations for recurrent vertigo and fluctuating hearing loss. Technical



problems still persist as, although the volumes of utricle and sacculi are within the resolving power of the MRI, the utricle and sacculi cannot be recognized because endolymph and perilymph signals are identical.³⁸ MRI images acquired following the injection of intrathecal gadolinium or the infusion intrathecal gadolinium do show evidence of passage of contrast from cerebrospinal fluid to perilymph and subsequent middle ear pooling. The technique, however, is both invasive and expensive. It is likely that in the future enhanced resolution MRI scanning would be utilized to localize fistulae. To date, however, in the absence of clearly reliable testing both sensitive and specific to fistula formation, the diagnosis is generally achieved by direct inspection of the oval and round windows at the time of surgical exploration.

Resolutions of symptoms in individuals with fistulae treated non-surgically do occur. Initially, conservative management seems pragmatic and generally consists of bedrest, during which time the head of the bed is elevated to 30 degrees above the level of the heart. The patient is instructed to avoid straining, lifting, and bending (avoidance of Valsalva's maneuver). Stool softeners may be administered. Medicine is of limited value, although symptomatic treatment with centrally acting GABA-minergic agents such as benzapines and peripherally acting

compounds such as scopolamine and/or promethazine may be administered to patients suffering from bouts of vertigo with nausea and emesis.

A protocol utilizing 24 hours to 48 hours of total bedrest followed by two weeks of restrained activity is a reasonable approach to patients suspected of harboring fistula. This protocol is in keeping with observations of patients recovering from stapedectomy surgery, which not uncommonly creates obvious perilymph fistulae. A longer period of bedrest from 5 days to two weeks may be advocated for patients who have chronic unremitting symptoms, as these fistulae do heal, at times, spontaneously. Patients who fail to improve after such a conservative therapy and/or who display progressive symptoms will require surgical exploration. The principle approach to repair consists of a graft of temporalis fascia or tragal perichondrium applied as patching to both round and oval window niches; results as high as 87% have been seen.³⁰ Vestibular complaints tend to resolve more favorably than does hearing recovery. Along with surgical intervention, postoperative care is about the same as that described following stapedectomy.

Cervical Vertigo

Vertigo, defined as an hallucination of environmental rotation or of self

rotation within the environment, probably does not arise from disruption of the integrity of the musculoskeletal system in the neck, although it is certainly feasible that injury to facet joints and/or musculoskeletal tissues play a role in the symptoms of dizziness and disequilibrium. One of the difficulties in establishing neck injury as the primary cause for the sense of vertigo is the likelihood of contiguous or comorbid injury to contiguous neurologic, vestibular, or neurovascular systems. For example, whiplash commonly affects the central nervous system and may produce dizziness, disequilibrium, and vertigo on a central basis due to axonal shearing. Likewise, injury to the vestibular system per se produces vertigo through a number of mechanisms. Lastly, neurovascular injury, particularly injury to the vertebral arteries as they ascend through bony columns in the neck, may also produce a true sense of vertigo on an ischemic basis.

The case for the existence of so-called cervical vertigo is based on the anatomic fact that the deep intervertebral neck muscles contain muscle spindles, which provide proprioceptive information. Selective injury to one set of proprioceptive signals theoretically might produce imbalance, a sense of lightheadedness, ataxia, and a sense of displacement in space. Experimentally, Karnath and colleagues³⁹ have demonstrated that vibration of neck



muscles, which produces a kinesthetic sense of muscle elongation, results in a sense of perception of trunk rotation and a shift in the subjective sense of an individual's location in space.

Strupp and colleagues⁴⁰ feel the perceived effects are secondary to change in eye positions rather than changes in cortical representation of body orientation. Patients so treated, however, do not develop clinically significant nystagmus, and therefore, no true sense of environmental rotation, i.e., no true vertigo.⁴⁰ Local anesthesia of deep muscle tissue in humans⁴¹ usually elicits a transient change in muscle tone, which results in a tendency for gait ataxia, pass-pointing, and falling toward the treated side, similarly to that reported in animals de-afferented in the high cervical area either surgically or by occipital anesthesia.⁴²

Nevertheless, the fact that somatosensory cervical proprioceptive input arising from muscle spindles converges with vestibular input to participate in the coordination of eye, head, and body position and location, as well as spatial orientation, makes it difficult to argue for a selective cervical syndrome. For example, there is little data to suggest that simple whiplash injury and/or cervical pain syndromes can produce such imbalance. Further, post-traumatic syndromes with minor traumatic brain injury or whiplash-type injuries affect structures beyond simple

neck proprioceptive muscle spindles.⁴³ As previously mentioned, the otolithic vestibular system, as well as brain stem structures, are particularly sensitive to identical trauma.

Although it is reasonable to assume that injury to the deep structures of the neck may produce non-specific symptoms of unsteadiness and alterations in postural control, involvement of neck reflexes as part of a multi-sensory intersegmental postural control mechanism makes it very difficult to establish isolated participation in any given syndrome. It is probably a mistake to assume that trauma to any of the musculoskeletal structures in humans can produce a significant nystagmus that may be subjectively experienced as vertigo. The clinical observation that post-traumatic lightheadedness and ataxia improve with either physical therapy, as it reduces neck pain, and/or the use of a neck collar⁴⁴ are intriguing but again fail to recognize that immobilization and physical therapy techniques undoubtedly have significant influence on the vestibular and ocular systems at the same time.

In this regards it should be noted that Brandt and Bronstein have pointed out that all clinical studies to date investigating cervical vertigo have had three weak points: the inability to confirm the diagnosis; the lack of a specific laboratory test; and the unexplained discrepancy between

patients with severe neck pain who do not experience vertigo and individuals experiencing only mild-to-moderate pain who bitterly complain of disabling vertigo. They further argue that even should cervical vertigo actually exist, since appropriate management seems the same as that for the cervical pain syndrome, the relevance as to any mechanism of cervical vertigo has more theoretical than practical value.⁴⁵

Visual Vestibular Mismatch (Sensory Mismatch)

It is a common experience to hear patients complain of a sense of dizziness, lightheadedness, or instability and unsteadiness that seems triggered by events in their environment. This is particularly true when a patient finds himself in surroundings that produce substantial visual motion, i.e. shopping malls, crowds, riding in cars as passengers on busy streets, etc. or in circumstances that produce repetitive visual patterns, i.e. crossing bridges, passing by a line of trees. This phenomenon, entitled physiologic vertigo, may be experienced by individuals free of disease. Other examples of this phenomenon include: the sense of dizziness that one experiences at heights, driving a car, or at sea. A surprising proportion of normal individuals with such symptoms are found to have a background history of migraine.



Despite the appearance of these symptoms in an otherwise normal population, identical feelings arise after vestibular insult, and the term “visual vestibular mismatch” has been coined to explain these symptoms. Guerrez and his colleagues⁴⁶ have offered an explanation for the development of such phenomenon following vestibular injury. The authors point out that some patients are lifelong “visually dependents.” In essence, a visually dependent person is someone who relies more on visual clues for spatial orientation than they do from information provided by proprioceptive information from the joint and/or information about position in space and acceleration from the inner ear. “Visually independent individuals,” on the other hand, are not particularly reliant on vision and proportionately use information equally from all three senses while making decisions about spatial orientation. The ability to weigh sensory information proportionately is of critical importance to the authors as they point out as soon as there is injury to a vestibular system, a neural process of recovery called vestibular compensation or adaptation begins. They speculate that if the process of compensation from a vestibular lesion is dependent on alternative sources of sensory information, i.e. visual or proprioceptive, then individual differences in the functional status of these systems may well have a critical

influence on clinical outcome following vestibular trauma.

In other words, a visually dependent individual who is unable to disregard visual information and accept contradictory information from either vestibular or proprioceptive systems or both are particularly handicapped if the vestibular system is injured. The injury to the vestibular system in such an individual with visual dependency would shift even more emphasis to the visual system, and as a consequence such individuals would be more prone to dizziness and associated symptoms and circumstances with excessive or disorienting visual stimuli, i.e. situations in which visual signals are referentially incorrect (flying in planes, walking in malls with a great deal of visual motion, walking down the aisles of grocery stores, etc.). In order to understand this concept, one must remember that in any given circumstance, sensory information may be inherently correct but referentially inappropriate. Seasickness, for example, arises when an individual does not visually recognize there is motion of the environment while at the same time the vestibular system provides information on constant acceleration in pitch or yaw planes. The inability to subconsciously decide which piece of information is correct produces the phenomenon known as visual vestibular mismatch and accompanying symptoms of dizziness, a sense of

disequilibrium, unsteadiness, and nausea.

Bronstein⁴⁷, in reviewing the works of Guerrez, concludes that the findings of the author and his colleagues support the view that individuals whose symptoms of dizziness are precipitated by disorienting visual surroundings are likely to have suffered underlying vestibular injury and be visually dependent, and that this combination can be highly debilitating and disabling to patients when exposed to disorienting visual environments in which both the visual signal and a faulty vestibular signal are unreliable. The consequence of such mismatching frequently results in psychophysiologic phenomenon to include anxiety, phobic symptoms, and secondary avoidant behavior. Individuals so affected are unable to identify the source of their emotional conflict, and treating physicians need to be most aware of the likelihood that these do not arise predominantly from a psychologic cause but result from automatic responses to conflicting sensory information. Thus, sensory mismatching can be identified objectively on quantitative tests of balance such as dynamic platform posturography.

Treatment of sensory mismatching is frequently difficult, but a number of therapeutic procedures exist,⁴⁸ and attention needs to be given to secondary psychologic manifestations. In our



experience, individuals so affected frequently respond positively when they are informed that their symptoms are not primarily psychogenic in origin.

Neurology of Otolithic Function

Special note should be given to those signs and symptoms which result from trauma to that part of the inner ear which singles information concerning the direction of gravity and the pattern of accelerated forces acting upon the body, the otolith system. The sensory receptors for the otolith organs, the utricle and saccule, known as maculae are membranous structures which project hair cells into the otolith membrane that overlies them. On the hair cells lies a dense layer of calcium carbonate crystals known as otoconia. The maculae of the utricle and saccule are oriented at approximately 90 degrees, and within each system sensory hair cells are organized in such a fashion that they may orient to respond to forces from all directions. Because of their capacity to respond to both gravity and to acceleration, they have essential functions in translating motion of the head, changes of the orientation of head and body with respect to gravity, and signaling static correction for posture shift with respect to gravity. They are also responsible for ocular motor responses to shifts of head position in pitch, yaw, and roll planes.⁴⁹ Unfortunately, because of their

interactions with the other sensory motor systems providing information about orientation, it has been virtually impossible to tease out specific techniques for identifying both normal and abnormal function within the system.

Furthermore, imaging techniques of the inner ear as yet lack sensitivity to identify what must be very small lesions as identifiable causes of the symptoms that arise from disruption of the system. Dysfunction within the otolith system is suspected when patients complain of sudden senses of veering, feelings of suddenly losing the sense of where they are in space, false feelings that the ground under them is moving, or that the environment appears to be tilted. Visual symptoms include double vision (diplopia) frequently provoked by shifts of head position and accelerating head movements in both vertical and oblique directions; oscillopsia, a sense that the environment is in motion during simple daily activities such as walking, running, etc.; and inability to establish true verticality, i.e. line up a picture on a wall, draw a line on a blackboard. Probably the most commonly identified sign suggestive of dysfunction in the system is the presence of nystagmus.⁵⁰ This nystagmus may be spontaneous, generally reflecting dysfunction of central signals within the brain stem, or positional which may represent either a central or peripheral mechanism for distortion of utricular signal.

The ocular tilt phenomenon, which consists of vertical skewing of the eyes in the horizontal plane, ocular torsional counter-rolling, and tilting of the head, represents one form of injury within the system which may be clinically identified. In trying to separate a peripheral versus central location for dysfunction within the system, one rule of thumb seems to hold fairly secure: patients with central dysfunction of the utricular system rarely complain of vertigo, i.e. hallucination of motion. This is not the case with patients with peripheral utricular injury.

The vestibulospinal system is equally involved, sometimes preferentially, but the findings are very nonspecific. Patients so affected frequently have abnormal postures, a tendency to tilt, a wide-based unsteady gait worsened by rapid position shifts, and a susceptibility to falls. The symptoms are not uncommonly an exaggeration of the normal phenomenon individuals without inner ear dysfunction experience with sudden position shifts, i.e. going from sitting to standing or retroflexing their head looking up at the sky, to a degree that the utricular system falls out of a position where it can effectively register gravitational information. The likelihood that the adjacent semicircular canal system may also be involved cannot be excluded. So the symptoms so generated cannot be categorically ascribed to the otolith system. The consequence of injury to the



system results in symptoms that seem quite bizarre and strange to given individuals. Those so afflicted frequently have a great deal of difficulty describing what it is they are experiencing, and as a consequence, the symptoms and signs are misunderstood and remain either without firm diagnosis or ascribed to an underlying psychopathology.

Of the limited tests available, dynamic platform posturography, which records body sway and displacement of sheer force downward, probably most closely identifies utricular dysfunction, but even then abnormalities and conditions presumed to have otolithic input also subserve vertical semicircular canal function. The best one can say, in circumstances where scoring is substandard in those conditions, is that the individual fails to maintain adequate balance using compensatory motor mechanisms in those conditions in which the vestibular system is the primary source of sensory information. On a positive note, traumatic injury to the peripheral vestibular apparatus, whether it involves the semicircular canal, the otolithic system, or both, is usually followed by a central compensation. Correction of ocular motor abnormalities usually occurs first with disappearance of nystagmus but is eventually followed by recovery of vestibular spinal function, often over periods of days to weeks.

In such cases, exercise, particularly those exercises generated to challenge the vestibular system, probably offer the best hope for expedient recovery. The time course for recovery from a labyrinthine injury may be substantially impeded by either the presence of comorbid injury, i.e. axonal shearing and/or microvasc lesions within the CNS, and/or the presence of vestibular suppressant medications such as antihistamines and medications designated to treat motion sickness, which by suppressing the vestibular system both centrally and peripherally prolong and sometimes prohibit rather than accelerate compensation. In our estimation, with the exception of rare cases of Meniere-like syndromes produced by trauma, and perilymph fistula, etc., vestibular suppressant medications should be withheld once the acute vegetative symptoms of nausea and vomiting have subsided.

BIBLIOGRAPHY

- 1 Baloh RW, Honrubia V. Vestibular function: an overview: part I. Clinical neurophysiology of the vestibular system edition II, F.A. Davis Co., 1990.
- 2 Baloh RW, Halmagyi GM. Overview of common syndromes of vestibular disease. Chapter 23. Baloh and Halmagyi, eds., Disorders of the Vestibular System. Oxford, NY: Oxford University Press, 1996:291.
- 3 Schuknecht HF. Mechanisms of inner ear injury from blows to the head. *Ann Otol* 78, 1969B:253-262.
- 4 Eide PK, Tysnes O-B. Early and late outcome in head injury patients with radiological evidence of brain damage. *Acta Neurol Scand* 86, 1992:194-198.
- 5 Oosterveld J, Kortschot HW, Kingma GG, et al. Electronystagmographic findings following cervical whiplash injuries. *Acta Otolaryngol (Stock 8)* 1991; 111:201-205.
- 6 Maitland, CG. Posttraumatic cranial neuropathies V, VII, and VIII. In Proceedings of the North American Neuro-ophthalmology Society meeting. Big Sky, Montana: 1993.
- 7 Aguilar E, Yeakley J, Ghorayeb A, et al. High resolution CT scan of temporal bone fractures. *Head Neck Surg* 1987; 9:163.
- 8 Wennmo C, Spandow AD. Fractures of the temporal bone-chain incongruencies. *Am J Otolaryngol* 1993; 141:38-42.
- 9 Mitchell DE, Adams JH. Primary focal impact damage to the brain stem in blunt head injuries: does it exist? *Lancet*, 1973; 2:215.
- 10 Davies RA, Luxon LM. Dizziness following head injury – A neuro-otologic study. *J Neurol* 1995; 242:222-230.
- 11 Levin HS, Mattis S, Ruff RM, et al. Neuro-behavioral outcome following minor head injury: a three-center study. *J Neurosurg* 1987; 66:234-243.
- 12 Tusa RJ. Benign paroxysmal positional vertigo. *Current neurology and neuroscience reports*, 2001; 1:478-485.
- 13 Barany R. Diagnose von krankheitserscheinungen im bereiche des otolithenapparates. *Acta Otolaryngol (Stock 8)* 1921; 2:434-437.
- 14 Dix MR, Hallpike CS. Pathology, symptomatology, and diagnosis of certain disorders of the vestibular system. *Proc Roy Soc Med* 1952; 45:341-354.
- 15 Schuknecht HF, Ruby RRF. Cupulolithiasis. *Adv Oto-laryng (Basel)* 1973; 20:443-445.
- 16 Hall SF, Ruby RR, McClure JA. The mechanics of benign paroxysmal vertigo. *J Otolaryngol* 1979; 8:151-168.
- 17 Maitland CG. Labyrinthine dysfunction. In Proceedings of the Rocky Mountain Neurophthalmology Society. 1984; 136-144.
- 18 Brandt T, Daroff RB. The multi-sensory physiological and pathologic vertigo syndromes. *Ann Neurol* 1980; 7:195-203.
- 19 Zucca G, Valli S, Valli P et al. Why do benign paroxysmal positional vertigo episodes recover spontaneously? *J Vest Res* 1998; 8:325-329.
- 20 Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1992; 107:399-404.
- 21 Brandt T, Daroff RB. Physical therapy for benign paroxysmal positional vertigo. *Arch Otolaryngol* 1980; 106:484-485.
- 22 Goodhill V. Sudden deafness and round window rupture. *Laryngoscope* 1971; 81:1462-1474.
- 23 Wall C 3d, Rauch SD. Perilymphatic fistula pathophysiology. *Otolaryngol Head Neck Surg* 1995; 112:145-153.
- 24 Schuknecht HF. Pathology of the ear. Edn 2. Philadelphia: Lea and Febiger; 1993.
- 25 Emmet R, Shea J. Traumatic perilymph fistula. *Laryngoscope* 1980; 90:1513-1520.
- 26 Friedland DR, Wackym PA. A critical appraisal of spontaneous perilymphatic fistulas of the inner ear. *Am J Otol* 1999; 20:261-279.
- 27 Rizer FM, House JW. Perilymph fistulas. The House Ear Clinic experience. *Otolaryngol Head Neck Surg* 1991; 104:239-243.
- 28 Davis RE. Diagnosis and management of perilymph fistula: the University of North Carolina approach. *Am J Otol* 1992; 13:85-89.
- 29 Seltzer S, McCabe BF. Perilymph fistula: the Iowa experience. *Laryngoscope* 1986; 94:37-49.
- 30 Fitzgerald DC, Getson P, Brousseau CO. Perilymphatic fistula: a Washington D.C. experience. *Ann Otol Rhinol Laryngol* 1997; 106:830-837.
- 31 Maitland CG. Perilymphatic fistula. *Current neurology and neuroscience reports* 2001; 1:486-491.
- 32 Singleton GT. Diagnosis and treatment of perilymph fistulas without hearing loss. *Otolaryngol Head Neck Surg* 1986; 94:426-429.
- 33 Daspt CP, Churchill D, Linthicum FH. Diagnosis of perilymph fistula using ENG and impedance. *Laryngoscope* 1980; 90:217-223.
- 34 Vartiainen E, Nuutinen J, Karjalainen S, et al. Perilymph fistula – A diagnostic dilemma. *J Laryngol Otol* 1991; 105:270-273.



- 35 Black FO, Lilly DJ, Peterkar J, et al. The dynamic posturographic pressure test for the presumptive diagnosis of perilymph fistulas. *Neurol Clin Diagnostic Neurotology* 1990; 8: No. 2:399-410.
- 36 Bassiouny M, Harsch BE, Kelly RH, et al. Beta two transferrin application in otology. *Am J Otol* 1992; 13:552-555.
- 37 Teilan SA, Disher MJ, Son Q, et al. Biochemical markers for identification of human perilymph. *Abstr Am Otol Soc* 1998; 131:39.
- 38 Buckingham RA, Valvassori GE. Inner ear fluid volumes and the resolving power of magnetic resonance imaging: can it differentiate endolymphatic structures? *Annal Otol Rhino Laryngol* 2001; 110:113-117.
- 39 Karnath O, Fetter M, Dichgans J. Ocular exploration of space as a function of neck proprioceptive and vestibular input – observations in normal subjects in patients with spatial neglect after parietal lesions. *Exp Brain Res* 1996; 109:333-342.
- 40 Strupp M, Arbusow V, Dietrich M, et al. Perceptual and ocular motor effects of neck muscle vibration in vestibular neuritis. *Brain* 1998; 121:677-685.
- 41 DeJong PTVM, DeJong JM, Cohen B, et al. Ataxia and nystagmus induced by injection of local anesthetics in the neck. *Ann Neurol* 1977; 1:240-246.
- 42 Manzoni D, Pompei O, Stampacchia G. Cervical control of posture and movements. *Brain Res* 1979; 169:615-619.
- 43 Ommaya AK, Faas F, Yarnall P. Whiplash injury and brain damage. *JAMA* 1968; 204:285-289.
- 44 Karlberg M, Magnusson M, Malmstrom EM, et al. Postural and symptomatic improvement after physiotherapy in patients with dizziness of suspected cervical origin. *Arch Phys Med Rehabil* 1996; 77:874-882.
- 45 Brandt T, Bronstein AM. Cervical vertigo. *J Neurol Neurosurg Psychiatry* 2001; 71: 8-12.
- 46 Guerrez M, Yardley L, Bertholon P, et al. Visual vertigo: symptom assessment, spatial orientation, and postural control. *Brain* 2001; 124:1646-1656.
- 47 Bronstein AM. Under-rated neuro-otological symptoms: Hoffman and Brookler 1978 Revisited. *Brit Med Bull* 2002; 63:213-221.
- 48 Pavlou M, Lingeswaran A, Davies RA, et al. Machine-based vs. customized rehabilitation for the treatment of chronic vestibular patients. *First World Congress of the International Society of Physical and Rehabilitation Medicine*, 2001, ed. Monduzzi: 139.
- 49 Highstein SM. How does the vestibular part of the inner ear work? In *Disorders of the vestibular system*. Baloh and Halmalgyi Oxford Press University. New York Oxford, 1996.
- 50 Gresty MA, Bronstein AM, Brandt T, et al. Neurology of otolithic function. *Brain* 1992; 115:647-673.

Book Review

Spinal Cord Medicine

By Steven Kirshblum, M.D., Denise I. Campgnolo, M.D., Joel A. DeLisa, M.D. (Eds)
Lippincott Williams & Wilkins
655 pages

Reviewed by Steven Mandel, M.D.
Clinical Professor of Neurology
Jefferson Medical College

In this multi-edited book, the authors successfully bridged the gap between basic anatomy and physiology and diseases that affect the spinal cord. They provide concise, easy-to-read chapters on primary disorders of the spinal cord, diseases that may have spinal cord involvement either degenerative, genetic, or acquired. An introduction to spinal cord rehabilitation, including assistive devices, provide a basic foundation for residents and practicing physicians for other healthcare workers involved in patients with diseases affecting the spinal cord. This book provides a solid foundation to understand the varying nature of disease entities, as well as treatment options.

Chapter 2 provides basic anatomy, including tracts, sympathetic nervous

system, and spinal cord vascular supply. It is written so that it can be understood by all healthcare providers, but provides the foundation to understand trauma and disease.

Chapter 3 provides an excellent review of anatomy and imaging of the spine, provides excellent drawings correlating with radiological pictures.

Chapters 4 and 5 start to build a foundation for spinal cord injury including assessment scales and anatomical localization.

Chapters 6 and 7 leads one to estimate outcome and sets the framework for further rehabilitation measures.

Chapters 8 through 25 describe the multiple organ complications that can accompany spinal cord injury and therapeutic modalities for symptom relief, as well as managing what could be life-threatening complications. Pressure ulcers, bladder care, and sexual function are addressed, as well as management programs, both short- and long-term.

Chapter 26 addresses pain, frequently overlooked. Pain may have a variety of causes which may emanate from central to peripheral. Patients may experience headaches, joint pains, reflex sympathetic dystrophy, with pain experienced both above and below the level of injury. Although there is a summary of anticonvulsants and antidepressants used in pain management, as well as muscle relaxants, and nonsteroidal drugs, it is sometimes difficult in an individual patient to predict which drug will be most effective..

Chapter 28 goes on to describe tendon transfers and behavioral assessments to improve quality of life and functioning in those with spinal cord injury, whether being a child as described in chapter 29 or an adult. Patients with spinal cord injury can lead productive lives, but require at times intensive management and surveillance for complications.

Chapters 30 through 37 describe multiple diseases that may affect the spinal cord, whether being motor neuron disease, vascular diseases, nutritional



deficiency, or post-polio syndrome. The same principles as outlined earlier in the book apply to understanding both anatomy, physiology, symptomatic treatment, treatment of underlying disease, and outcome.

For the physician evaluating patients with disability related to spinal cord

disorders must learn to not emphasize their restrictions, but their capabilities. Whether individuals have progressive neurological diseases that may fail to respond to medical treatment, those that might be reversible, and those that might be of a static nature, this book places all of those in a format well organized by the authors. They should

be applauded for this encyclopedic approach which is easy to read and reader friendly. It is an invaluable asset to any healthcare provider working in the area of spinal cord medicine. With a five-star rating system, I would rate this as being 5 stars.

Book Review

Carpal Tunnel Syndrome and Other Disorders of the Median Nerve (2nd Edition).

By Richard B. Rosenbaum, M.D. and José L. Ochoa, M.D., Ph.D., D.Sc.
Butterworth Heinemann
372 pages
Reviewed by Steven Mandel, M.D.
Clinical Professor of Neurology
Jefferson Medical College

Dr. Rosenbaum and Dr. Ochoa have written an excellent book on carpal tunnel syndrome and disorders affecting the median nerve. For physicians interested in median nerve abnormalities, the second edition provides an outstanding review of literature and differential diagnosis, aiding the physician in clinical decision-making.

The book starts out with a review of anatomy, identifying median nerve branches, sensory and motor distribution, and possible sites of entrapment. Included is a basic understanding of the anatomy, vascular supply, and the variations in branching.

In Chapter 3, in addition to Tinel's and Phalen's signs, other maneuvers such as median nerve compression test, closed fist test, "flicked" signs and others have been summarized. They point out that

provocative testing become positive in early in the diagnosis of carpal tunnel and in fact occur before electrophysiological testing becomes abnormal. In the flick sign, they point out "...patients often shook or "flicked" the wrist of the symptomatic hand when describing their attempts to alleviate carpal tunnel syndrome symptoms". There is a 93% true-positive rate in patients with nerve conduction evidence supporting the diagnosis with a false-positive rate being 5% in other neurogenic syndromes.

Authors have gone to great lengths to explain various clinical tests and their significance. For example, in the hand elevation test, patients are asked to raise their hands above their head and maintain that position until symptoms occur at most for 2 minutes. The authors describe among woman who had carpal tunnel syndrome and abnormal median

nerve conduction, three-fourths reported developing symptoms during this maneuver with the test being positive only 3 out of 200 control women.

Chapter 4 discusses the differential diagnosis to include radial neuropathy, brachial plexopathy, cervical radiculopathy, diseases of the central nervous system, and non-neurological diagnosis. The authors point out that sensory changes in the upper brachial plexus can overlap the median cutaneous sensory distribution.

Tendinitis which may accompany neurological syndromes such as de Quervain's tenosynovitis and lateral epicondylitis need to be differentiated from carpal tunnel syndrome and needs to be addressed along with other causes of wrist pain.



Chapter 5 discusses carpal tunnel syndrome with other medical conditions to include diabetes which is the most common systemic illness in association with carpal tunnel, both hyperthyroidism and hypothyroidism, and hormonal changes, especially during pregnancy. Chapter 6 addresses double crush syndrome with the author stating, "Observations in patients support this theoretical analysis", but later goes on to say, "These random observations of coincident pathology neither prove nor disprove the double crush hypothesis". The chapter goes on to describe space-occupying lesions, muscular anomalies, hereditary and acquired disorders, and a comprehensive list of unusual causes.

Chapter 7 addresses electrodiagnostic testing presenting a basic understanding of electrodiagnostic testing and validity of various electrodiagnostic criteria. Quantitative sensory testing was described as being valuable for small caliber fibers, nerve injuries, and neuropathic pain syndromes. Thermography, although frequently positive, was felt not to be useful for routine diagnosis of carpal tunnel.

Chapter 11 goes on to describe imaging techniques and the value of diagnostic imaging techniques, concluding that it is not part of the routine evaluation. Chapter 12 describes mechanical injuries with an excellent review of mechanisms of nerve injury, both clinically and experimentally.

Chapter 13 is of great interest to disability medicine physicians

attempting to address the worker and its relationship to occupation and development of carpal tunnel syndrome. Examples as given in the literature look at dose-response relationship concluding that, "...local median nerve pathology that is expressed as carpal tunnel syndrome is solidly supported by pathophysiologic observations". Later chapters go on to describe nonsurgical and surgical treatments with its rate of success.

Chapter 16 addresses complex regional pain syndrome cautioning physicians to be open minded and not labeling patients as having RSD when in fact other entities may be present. Chapter 17 describes multiple syndromes of the median nerve proximal to the carpal tunnel such as anterior interosseous and pronator syndrome as well as treatment options. Chapter 18 describes median nerve abnormalities distal to the carpal tunnel, emphasizing that the site of pathology needs to be definitively determined and all symptoms in a carpal tunnel distribution may not be secondary to an entrapment of the median nerve at the carpal tunnel, but may represent both proximal and distal abnormalities. Finally, tumors of the median nerve, both benign and malignant and those associated with neurofibromas, fatty tumors, and cysts and infections are described briefly.

The only deficiency within this book would be the paucity of information related to other nerve disorders in the upper extremity that may occur in association with findings in a median

distribution. Individuals with chronic inflammatory demyelinating polyneuropathy, multifocal motor neuropathy, neuropathies related to immunological causes such as monoclonal gammopathies, genetic disorders such as hereditary sensorimotor neuropathies and hereditary predisposition pressure palsies are not addressed in any detail. When individuals present with carpal tunnel syndrome and in the process of performing electrodiagnostic testing, other nerves may be found to be involved and problems that are first thought to be traumatically induced may in fact be of genetic or acquired conditions unrelated to their jobs. Toxic neuropathies that can either be demyelinating or axonal can present with neuropathies including being prone to entrapment syndromes.

The book provides excellent information on the median nerve. It is of benefit to all physicians who are interested in further studying the median nerve and its differential diagnosis. Physicians will need to consult other sources of information in regard to diseases of the upper extremity in addition to the information provided in this book to evaluate patients with upper extremity disorders. For those physicians performing disability evaluations, although the chapters on surgery are described, long-term outcome measures for those treated nonsurgically and those with surgery including absenteeism from work could have been better addressed.



*Get Some Action
With Your next Ad in
Disability Medicine*

Circulation 3000 IME physicians

*Next Deadlines:
October 22 & November 6*

Call 800-234-340
FAX 847-277-7912
www.abime.org

Dear List Renter:

The American Board of Independent Medical Examiners (ABIME) has a list rental service.

Please place all list orders by calling 800-234-3490, e-mailing infor@abime.org or faxing them to 847-277-7912.

ABIME is highly confident that we will expedite list orders to effectively meet your needs.

Thank you for your patronage. If you have any questions or concerns, please do not hesitate to contact me.

Sincerely,

Brian Maddox

Item	One-Time Use	Unlimited Annual Use
ABIME Physician List Rental	\$450 Flat	\$2,995 Flat
Phone Number Select	\$135 Flat	\$1,000 Flat
Fax Number Select	\$180/M	\$1,200/M
Email Broadcast Service	\$200 Flat	\$ 450 Flat
IME Schedulers	\$200 Flat	\$ 450 Flat

Reach 2,500 IME Physicians Per Issue

Disability Medicine Journal • Quarterly Publication

Advertising Price Sheet

Inside Back Cover Full Page.....\$500
Inside Back Cover Half Page.....\$300

Full Page Other\$400
Half Page Other\$250

The American Board of Independent Medical Examiners looks forward to your participation.

Name: _____ Title: _____

Company/Clinic: _____

Street Address: _____

City: _____ State: _____ Zip: _____

Phone: _____ Fax: _____ E-mail: _____

I am paying by credit care. Please circle appropriate card (Visa/Mastercard or American Express).

Credit Card Number: _____

Exp. Date: _____ Signature: _____

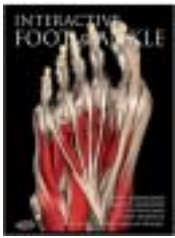
You may fax this form or send it with a check to:

The American Board of Independent Medical Examiners (ABIME) • 111 Lions Drive, Suite 217 Barrington, IL 60010 USA
Fax (847) 277-7912 • Phone (800) 234-3490 or (847) 277-7902 • www.abime.org



3D Interactive Series

The World's Finest Musculoskeletal Software



Foot & Ankle



Hand/Wrist



Hip



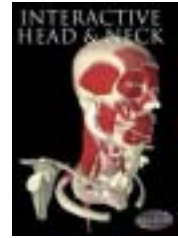
Knee



Shoulder/Elbow



Spine



Head & Neck

The exceptional 3D anatomical model on these disc are built from a full cadaveric MR data set. It can be viewed layer-by-layer, bone to skin, including such features as muscle attachment points, bony regions and dermatomes. Every visible structure can be clicked on to reveal detailed anatomical and clinical text, comprehensive enough for those in training and detailed enough for experts. Also included are hundreds of slides (MR, X-Rays, diagrams), fully labeled dissection pictures. Beautiful video clips of a moving dissection and biomechanics that demonstrate the dynamic actions for most major muscles.

Features Include:

- Windows or Macintosh Compatible
- Unsurpassed in Comprehensiveness, Resolution, and Clarity
- 3D Computer Graphic Models of Human Anatomy, Derived from CT and MR Scan Data
- Complete 3D Model, Can be Rotated and Every Visible Structure Labeled
- Layers of Anatomy Can be Added or Stripped Away
- Animations and Video Clips Show Functions, Biomechanics, and Some Surgical Procedures
- Print or Export Images and Text into Programs Such as PowerPoint
- Anatomical Text Links to Anatomical Dissections and Clinical Pathology Slides
- MRI Sections, Allowing Correlation of 3 Planar MRI to 3D Anatomy
- Wealth of Information on Therapy Related Topics and Clinical Pathologies
- Quizzes and Multiple Choice Questions
- Outstanding Educational & Lecture Tool

Range of Motion Testing

With the Acumar™ Digital Inclinometer Suite For Objective Evaluation

Features:

- Easy to read digital LCD
- Large liquid crystal display readout
- Covers 360° (displays +180° to -180°)
- Respect to gravity or zero at neutral position
- Multiple data storage
- Recall data for viewing
- Supports the AMA Guides



Digital Inclinometer



Dual Inclinometer

Compact, handheld unit, features large digital display for easy reading. Store measurements with the hold button, review maximum, minimum and average values. This full featured unit reduces examination time and enhances the objectivity of measurement and documentation. Supports range of motion evaluation as described in the AMA Guides* to the Evaluation of Permanent Impairment. Inclinometer comes with built-in wireless transmitter to optional computer interface.

Sports Medicine Technologies • 800-279-1479 • 602-971-4353 • Fax:(602) 404-2506
E-mail: tom@sportsmedtechnologies.com www.sportsmedtechnologies.com



SEAK, Inc. proudly presents three unique interactive CME programs:

2003 IME Summit

November 6-7, 2003

- Learn from a dynamic and diverse faculty which includes a distinguished IME physician, a workers' compensation hearing officer, experienced plaintiff and defense attorneys, an IME company executive, and an innovative IME office manager
- Write more persuasive, defensible, and valuable IME Reports
- Reduce your legal risks
- Improve the efficiency and profitability of your IME practice
- Obtain additional high-quality clients with repeatable business
- Effectively deal with the most difficult ethical problems associated with IMEs
- Defend your IME report from counsel's trick questions
- Build a premier IME practice

Medical Malpractice: Advanced Survival Training For Physicians

November 6-7, 2003

- How to be an effective witness in your own defense
- Effective risk management
- The hidden pitfalls of liability insurance policies
- What makes a malpractice jury tick
- Fundamental asset protection techniques
- How to be an effective member of the defense team
- Settlement: The good, bad and ugly
- How plaintiff and defense trial lawyers evaluate and try malpractice cases
- Law, procedure and tactics
- Deal with today's "Malpractice Crisis"

2003 Medical Witness Summit

November 8-9, 2003

- Learn from a highly knowledgeable faculty which includes a U.S. District Court Judge, experienced plaintiff and defense attorneys, an expert marketing consultant, a jury consultant, and an experienced medical witness
- Master deposition and trial skills
- Be far more effective in front of a jury
- Learn premium fee setting and collection techniques
- Solve ethical dilemmas
- Build a premier forensic practice
- Become a more valuable sought-after medical witness

All programs held at the DePaul Center, DePaul University®, Chicago, IL

To register or for further information call (508) 457-1111 or visit www.seak.com

A Distinction *that Sets You Apart*

Achieve certification through the American Board of Independent Medical Examiners (ABIME) and gain recognition from disability and compensation professionals. ABIME certification offers you added advantages:

- State-of-the-art training in AMA Guides
- Increased demand for your specialized services
- International promotion of your certification status to prospective clients
- Enhanced credibility and competency as a medical examiner
- Advanced knowledge and training in impairment and disability evaluation
- Added professionalism and career advancement

Be among the first in your area to earn this prestigious distinction. Fax us today to achieve the ABIME distinction that sets you apart.

2003-2004 Education and Certification Examination Schedule

Chicago, Illinois October 17-20
Orlando, Florida February 6-9, 2004
Puerto Rico March 26-28, 2004

Chicago, Illinois June 4-6, 2004
Chicago, Illinois July 30-August 2, 2004
Las Vegas, Nevada October 22-25, 2004

Yes, I'm interested in ABIME Certification.
Please send an information packet right away.

Name _____
Title _____
Company/Clinic _____
Address _____
City, State, Zip _____
Telephone _____ Fax _____
E-mail _____

For faster response, fax this form to 847-277-7912

ABIME
American Board of Independent Medical Examiners

111 Lions Drive, Suite 217
Barrington, IL 60010-3175
Telephone: 847-277-7902 or 800-234-3490
E-mail: info@abime.org **Website: www.abime.org**