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# DISABILITY MEDICINE

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## Editorial Board



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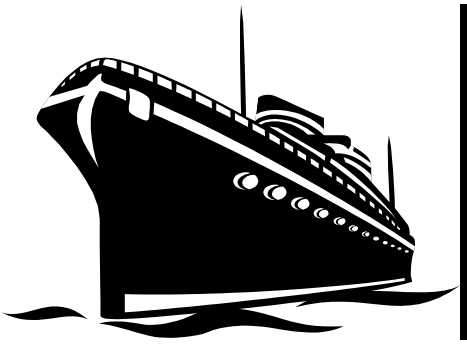

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# *EDITORIAL:* Re-certification by Alternate Pathway

The training program of the American Board of Independent Medical Examiners provides pre-eminent continuing medical education in impairment and disability evaluation. This education enhances physician knowledge, skills and ability, ensuring better impairment rating and disability evaluation. Physicians who pass the examination are certified and re-certified based on a standardized process. The re-certification assures addresses upgrading physicians' skills in disability medicine through continued education and training.

The American Board of Independent Medical Examiners (ABIME) requires re-certification by examination every five years after the initial certification. The objective is to ensure a high level of expertise and training for physicians certified as Independent Medical Examiner.

The ABIME Board of Directors has for some time now been petitioned by many currently certified individuals to authorize an alternate pathway for re-certification other than the examination. The Board, after giving it serious consideration, has unanimously voted in October 2001 Board of Directors Meeting to prescribe such an alternate pathway. Our President, Tom Beller, MD has formally asked me to organize and coordinate this process.

Effective June 1, 2002, the following rules shall govern the alternate re-certification pathway. *In this regard it should be noted that the initial certification would still require examination and is possible through examination only.* The following rules apply for *re-certification only* for an individual already initially certified by ABIME through examination and who is in good standing with the Board of Registry and whose certificate has not expired. The re-certification without examination pathway is designated the *alternate pathway.*

1. The re-certification without examination is an alternate pathway and the option to re-certification by examination remains and would continue to be available and an individual in the alternate pathway can switch to examination pathway by appropriately notifying ABIME.

2. The alternate pathway is available to individuals who have at least 2 years remaining before expiration of their certificate. *There will be an exception to the rule for the first 6 months, which will expire December 31, 2002.* During this initial phase anyone who has passed the ABIME exam previously and has been certified, would be able to enroll in the re-certification pathway. The following are components of the alternate pathway.

A. Enrollment-this will require filling out a declaration of intent to pursue the alternate pathway with a nonrefundable fee of $500.00. This fee will help defray the cost of establishing the Alternate Pathway track file, maintenance of various records by ABIME for documentation of continuous education in the field of Disability Medicine.

B. Earn 60 points of continuous education credit over the following two years as prescribed below. The Alternate Pathway candidate would be required to earn at least 60 points to become eligible for re-certification by Alternate Pathway.

*Continued Next Page*

1. Evidence of approved CME The Journal of Disability Medicine will have several CME articles with multiple-choice questions at the end. Re-certification candidates will read these articles and send the answers to the ABIME. Each correct answer is one point credit towards this category. The re-certification candidate needs a minimum of 25 points and may achieve up to 35 points from this activity towards the 60 points required for recertification.

2. The re-certification candidate will also be required to document a minimum of 25 hours of ABIME CME activities in Disability Medicine. The certification review course, sponsored by the ABIME currently offers 15 hours of CME and the Board intends to offer other advanced courses.

3. At the successful completion of all the required documentation, the re-certification candidate would be required to formally petition the ABIME board of directors for a re-certification through the alternate pathway.

## Continuous education credit Point System

| | Minimum Points | Maximum Points |
|---|---|---|
| **Required Educational Programs and Training**<br>ABIME approved Continuing Medical Education<br>(One Point for each CME hour) | 25 | 35 |
| **CME monitoring through CME questions**<br>in *Journal of Disability Medicine*<br>(One point for each correct answer)<br>60-point minimum required for Re-certification | 25 | 35 |

**Mohammed I. Ranavaya, M.D., M.S., FRCPI, FFOM, FAADEP, CIME, President Elect American Board of Independent Medical Examiners**

## CME QUESTIONS (Continued from page 102)

8. A 55-year-old male has 2 to 6 generalized tonic clonic seizures per week usually with a 1 to 2 hour post ictal state in spite of good compliance with medications. EEG was positive. According to the fifth edition of the AMA guides his whole person impairment is:

A. 39%
B. 49%
C. 59%
D. 69%

9. A 49 year old male with history of head trauma 2 years ago is unable to find his home or location, wanders out of the house, has poor short-term memory and is disoriented in time, place and person. According to the fifth edition of the AMA guides his whole person impairment is:

A. 29%
B. 39%
C: 49%
D: 59%

10. A 30-year-old female has been diagnosed with reflexes sympathetic dystrophy. She had right carpal tunnel release one year ago. Several stellate ganglion blocks temporary relieve the pain . Over time she noted colour changes and increase sweating. There is a deeper burning pain and increase sweating right arm weakness and inability to use the hand for any daily activities. On examination the right arm is held close to the body. The hand is dusky with the sweating palm. Three-phase bone scan was unremarkable. X-rays revealed diffuse demineralization.

According to the fifth edition of the AMA guides her whole person impairment is:

A. 30%
B. 40%
C. 50%
D. 60%

11. A 37-year-old female was in a motor vehicle accident and developed neck and right upper extremity pain. An MRI revealed a herniated disc at C 6. Currently she has some residual neck pain with physical activity. The upper limb symptoms have resolved. The diagnosis is herniated disc C-5 -- C6 with resolved right C6 radiculopathy.

What is the impairment category based on the DRE model of the fifth edition of the AMA guides?

A. DRE I
B. DRE II
C. DRE III
D. DRE IV

Answers for previous CME Questions from Disability Medicine Vol 2, #1
*Questions on Pg10: 1.b, 2.b, 3.b, 4.a, 5b*
*Questions on Pg19: 1.d, 2.b, 3c*

## *Original Research Article*

# Genetic factors associated with Complex Regional Pain Syndrome I: HLA DRB and TNFα promoter gene polymorphism

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## Editor's note

**Complex regional pain syndrome is classified as type I (also known as reflex sympathetic dystrophy, RSD) when it develops without overt nerve injury and as type II (also known as causalgia) when it follows a demonstrable nerve injury.[1]**

**Complex regional pain syndrome type I is a painful and disabling neuropathic pain syndrome that can develop after trauma or surgery or may occur spontanously. Even a trivial injury seems to be able to trigger a chronic, disabling condition. Clinical features include spontaneous pain, hyperalgesia, allodynia, impairment of motor function, swelling, changes in sweating, vascular abnormalities and trophic changes.**

**Complex regional pain syndrome type I is a diagnosis that is used to describe pain in any part of the body that is out of proportion to the degree of injury. This syndrome can involve high medical costs, long periods of lost time from work[1] and considerable permanent impairment[2].**

**Controversy exists about the classification of this syndrome[3] and there is limited evidence for the effectiveness of various therapeutic interventions4. Focused research is therefore a prerequisite for improved treatment and thus prevention of long standing disability due to this condition. One of the issues which need to be looked into is whether there is a genetic predisposition for Complex regional pain syndrome. The article by Michiel Vaneker et al in this issue of the journal is an important contribution in that direction.**

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## Abstract

*Background* Complex Regional Pain Syndrome I (CRPS I) is a complication occurring in an extremity, mostly following minor trauma or surgery. The main question of CRPS I is why one person develops this disease, while another person with a similar trauma does not develop this syndrome. For this reason, we analysed TNFα gene polymorphism and HLA class I and II antigens to clarify a possible genetic predisposition in CRPS I.

*Methods* The CRPS I patients (n=161) were included according to prospectively defined criteria. HLA typing was routinely carried out for A, B and DRB loci. TNFα typing, for the promotor gene polymorphism on position -308, was performed by PCR-SSP. Besides the whole population the following subgroups were separately analysed: male/female, primary skin temperature warm or cold and CRPS I involving one or more than one extremity.

*Results* HLA DR6 (50%, p<0.0003), as well as HLA DQ1 (99%, p<0.0002), were increased in the subgroup primarily cold CRPS I as compared to the control group, respectively (31%) and (83%). TNF2 allele (40%, p<0.002) was increased in the subgroup primarily warm CRPS I as compared to the control group (19%). Individuals homozygous for TNF2 allele (15%, p<0.008) were increased in the subgroup CRPS I involving more than one extremity, as compared to the control group (2%). In the total CRPS I population no significant results were seen.

*Conclusions* The association of HLA DR6 and HLA DQ1 suggests immune system involvement in primarily cold CRPS I, in which most complications occur. The presence of a TNF2 allele, associated with producing higher amounts of TNFα, suggests a role of the TNF2 allele in the pathogenesis, and may open new pathways in the treatment of CRPS I.

## Introduction

Complex Regional Pain Syndrome I CRPS I is a complication occurring in an extremity, mostly following minor trauma or surgery.[17] In some cases, it occurs spontaneously. The syndrome is characterised by pain, edema, vasomotor instability, limited range of motion and increase of these signs and symptoms after exercise.[26]

Various pathophysiological theories have been proposed to explain the appearance of CRPS I. These theories include an upregulation of the a-adrenoreceptors,[1;4] a psychological alteration,[7] and an exaggerated regional inflammatory response.[16;26]

Arguments supporting the exaggerated regional inflammatory response theory are typical histological findings,[21] the increased capillary permeability for large proteins in the acute phase,[16] and the therapeutic efficacy of corticosteroids,[2] and oxygen radical scavengers.[8;29]

Besides the discussion which pathophysiological mechanism is involved, it remains unknown why some patients develop CRPS I after injury, while others do not. Genetic differences in the response to injury may explain this difference. Mailis et al.[14] suggested HLA DR2 to predispose CRPS I, however their data did not reach the level of statistical significance. Kemler et al.[11] showed HLA DQ1 to be associated with CRPS I and found this result compatible with Mailis, because of the linkage between HLA DQ1 and HLA DR2. van Hilten et al.[24] could not confirm this linkage in a subgroup of CRPS I patients with progressive tonic dystonia, but found an association with HLA DR13.

TNFα is a potent cytokine with a wide range of proinflammatory activities, with a bi-allelic diversity in the promotor region of the TNFα gene, associated with high or low producer capacity.12 Because of its proinflammatory role as well as its immunomodulatory potential, we hypothesized the bi-allelic TNFα system, whether or not sustained by certain HLA-types, might play a role in a predisposition to CRPS I in response to injury.

Therefore, we analysed HLA class I and II antigens and TNFα gene polymorphism in CRPS I patients in an attempt to investigate their involvement as specific genetic factors that may predispose to CRPS I.

## Materials and methods

This study was approved by the institutional review board and performed according to the World Medical Association Declaration of Helsinki.

Inclusion criteria for the study were: Caucasian race and CRPS I diagnosed according to the following criteria.[17;26]

1) Four or five of the following signs and symptoms:

   Otherwise unexplained regional pain within the affected area

   Edema of the affected area

   Difference in skin temperature compared to the unaffected other extremity

Difference in skin colour compared to the unaffected other extremity

Limited active range of motion.

2) Occurrence or increase of above signs and symptoms after use of the extremity.

3) Above signs and symptoms present in an area larger than the area of primary injury or operation which must include the area distal to the primary injury.

All patients were seen at the outpatient clinic for CRPS I of the Department of Surgery by two investigators between September 1, 1998 to September 1, 2000. Patients were divided into the following subgroups: male/female, primary skin temperature warm or cold at onset of the complaints as mentioned by the patient, and CRPS I involving one or more than one extremity.

After informed consent and a written permission, venous blood was taken for serologic HLA typing 18 for A and B loci. HLA DRB typing was performed by PCR-SSO-ELISA-KIT after DNA isolation.[3] The TNFα typing for the promotor gene polymorphism on position -308 was carried out by PCR-SSP,19 using the sense-primer: AGG TTT GGG GCA TGG G* instead of the TNF1 sense-primer: AAT AAG TTT TGA GGG GCA TGG. The HLA DQ typing was derived from the HLA DR results by means of DR-DQ associations (table 1) to make a first analysis possible.

* modification made by Dr. B. Hepkema. University Medical Center, Gronigen, the Netherlands

### TABLE 1: HLA DR-DQ ASSOCIATIONS

| | |
|---|---|
| DQ1 | DR1, DR2, DR6, DR10 |
| DQ2 | DR3, DR7 |
| DQ3 | DR4, DR5, DR9 |

The detected HLA frequencies were compared with a database of HLA types in Caucasian individuals from the Bonemarrow donor registry of the Netherlands Europdonor the Nederlands (n > 10.000). TNFα frequencies were compared with a regional group of Caucasian bonemarrow donors (n=168).

Statistical analysis was carried out by the chi-square test for goodness of fit ($p < 0.05$) regarding the HLA typing, and Fisher exact test for a 2 x 2-table ($p < 0.05$) regarding the TNFα typing. Significant p values were corrected for the number of comparisons made by Bonferroni's principle of multiple comparison.

## Results

161 patients were included in the study. The gender was 45 male (28%) and 116 female (72%). The clinical characteristics are shown in table 2. The primary skin temperature of the affected extremity, as compared to the unaffected extremity, at the start of the CRPS I symptoms was cold in 82 patients (51%), similar or unknown in 16 (10%) and warm in 63 patients (39%). More than one extremity was affected by CRPS I in 27 patients (17%), 17 patients with 2 affected extremities and 10 patients with 3 or 4 affected extremities. In 107 patients (66%), CRPS I followed trauma and in 24 patients (15%) CRPS I followed surgery. In 30 patients (18%) no precipitating event could be identified.

In the total CRPS I population no significant results were seen. The subgroup primarily cold CRPS I showed an association with HLA DR6 ($p < 0.0003$) as well as HLA DQ1 ($p < 0.0002$). The presence of a TNF2 allele was associated with primarily warm CRPS I ($p < 0.002$). Looking only at homozygotes for the TNF2 allele in the subgroup CRPS I involving more than one extremity, we found a strong association ($p < 0.008$). The results are shown in table 3.

## Discussion

In this population, primarily cold CRPS I was strongly associated with HLA DR6 ($p < 0.0003$) as well as HLA DQ1 ($p < 0.0002$). Mailis et al.[14] suggested HLA DR2 to be associated with the development of CRPS I in Caucasian females. However this finding was not statistically significant, while the number of patients was small (n=15). Kemler et al.[11] reported a significant association of HLA DQ1 with CRPS I

## Table 2: Clinical characteristics

| | 1 (n=161) | | 2 (n=45) | | 3 (n=116) | | 4 (n=82) | | 5 (n=63) | | 6 (n=134) | | 7 (n=27) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N | % | n | % | n | % | n | % | n | % | n | % | n | % |
| Male | 45 | 28 | * | * | * | * | 20 | 24 | 22 | 35 | 41 | 31 | 4 | 15 |
| Female | 116 | 72 | * | * | * | * | 62 | 76 | 41 | 65 | 93 | 69 | 23 | 85 |
| Age at the onset of CRPS I(median) median | 13-75(45) | | 18-74(46) | | 13-75(45) | | 13-72(37) | | 14-75(54) | | 14-75(46) | | 13-60(38) | |
| Interval between onset of CRPS I and start of the therapy | | | | | | | | | | | | | | |
| 0-6 months | 113 | 70 | 36 | 80 | 77 | 66 | 48 | 59 | 55 | 87 | 97 | 72 | 16 | 59 |
| 6-12 months | 25 | 16 | 2 | 4 | 23 | 20 | 18 | 22 | 4 | 6 | 21 | 16 | 4 | 15 |
| > 12 months | 23 | 14 | 7 | 16 | 16 | 14 | 16 | 20 | 4 | 6 | 16 | 12 | 7 | 26 |
| Primary skin temperature | | | | | | | | | | | | | | |
| Cold | 82 | 51 | 20 | 44 | 62 | 53 | * | * | * | * | 63 | 47 | 19 | 70 |
| Warm | 63 | 39 | 22 | 49 | 41 | 35 | * | * | * | * | 57 | 43 | 6 | 22 |
| Normal | 16 | 10 | 3 | 7 | 13 | 11 | * | * | * | * | 14 | 10 | 2 | 7 |
| One extremity | 134 | 83 | 41 | 91 | 93 | 80 | 63 | 77 | 57 | 90 | * | * | * | * |
| More than one extremity | 27 | 17 | 4 | 9 | 23 | 20 | 19 | 23 | 6 | 10 | * | * | * | * |

1= total group (161)
2= male (45)
3= female (116)
4= primarily cold CRPS I (82)
5= primarily warm CRPS I (63)
6= CRPS I involving one extremity (134)
7= CRPS I involving more than one extremity (27)

## Table 3: Results

| | CRPS I % | Control-group% | p-value |
|---|---|---|---|
| Subgroup primarily cold CRPS I (n=82) | | | |
| DR6 | 50 | 31 | **< 0.0003** |
| DQ1 | 99 | 83 | **< 0.0002** |
| TNF2 positive | | | |
| 21 | 19 | 0,74 | |
| TNF2 negative | 79 | 80 | 1,00 |
| Subgroup primarily warm CRPS I (n=63) | | | |
| DR6 | 30 | 31 | 1,00 |
| DQ1 | 81 | 83 | 0,62 |
| TNF2 positive | 40 | 19 | **< 0.002** |
| TNF2 negative | 60 | 80 | **< 0.004** |
| Subgroup CRPS I involving more than one extremity (n=27) | | | |
| DR6 | 41 | 31 | 0,30 |
| DQ1 | 85 | 83 | 1,00 |
| TNF2 positive | 41 | 19 | **< 0.03** |
| TNF2 homozygotes | 15 | 2 | **< 0.008** |

(n=52) and found this result compatible with Mailis et al.[14] because of the linkage between HLA DR2 and HLA DQ1. However HLA DR6 is also linked to HLA DQ1. In a letter to the editor, van de Beek et al.[20] questioned the conclusions of Kemler et al.[11] and suggested an error in the data of the control population as not being representative for the Dutch population overall. The CRPS I patients included in our study were referred from all over the Netherlands in stead of from a particular region. Therefore a large control database resembling the Dutch population overall was considered best. van Hilten et al.[24] identified an association of HLA DR13 with a subgroup of CRPS patients suffering from progressive tonic dystonia. No association with HLA DQ1 was found. We also could not confirm the reported DQ1 association in our CRPS I population.

None of the three published studies mentioned above made a classification in different forms of CRPS I as was performed in the present study. This subdivision is clinically important, as primarily cold CRPS I has a worse functional outcome,[22] a higher incidence of recurrence, and a higher risk of developping CRPS I in another extremity.[25]

Hendriks et al.[9;10] showed HLA DR6 positiv atients to have a higher risk of rejection after a kidney transplantation and suggested HLA DR6 to be a marker for high immune responsiveness. Furthermore HLA DR6 has been associated with chronic autoimmune hepatitis 6 and vitiligo,[27] ailments also suggested to have an autoimmune component.15 One of the pathophysiological theories involved in CRPS I is an exaggerated inflammatory response.[2;8;16;21;26;29] It is yet unknown if the immune system is involved in the pathophysiology. The present finding that HLA DR6 and DQ1 are associated with primarily cold CRPS I, suggests an hyperresponsive immune system to predispose to these forms of CRPS I.

In our study, the TNF2 allele was associated with primarily warm CRPS I ($p < 0.002$). The association with CRPS I involving more than one extremity was only found in individuals homozygous for the TNF2 allele ($p < 0.008$), which may be a dosage effect due to the presence of two TNF2 alleles. TNF2 is strongly associated with HLA-A1 B8 DR3 haplotype.[28] The latter one did not show an association with CRPS I, indicating the TNF2 allele to be an independent contributing genetic factor. Individuals with the TNF2 allele produce higher amounts of TNFα.[12;13] The TNF2 allele may thus be involved in the pathogenesis of certain forms of CRPS I by, in situ, locally high TNFα production.

Thus, possibly an anti-TNFα therapy may reduce signs and symptoms in certain presentations of CRPS I, as was recently shown in Crohn's disease,23 and rheumatoid arthritis.[5] In this study no direct measurements of TNFα were performed. Systemic levels of TNFα may not be relevant due to the short half life of TNFα, while systemic concentrations of TNFα do not accurately reflect the actual concentration in situ. On the other hand in situ determination of TNFα is not easily done, especially in a CRPS I affected extremity.

In conclusion this study has demonstrated a statistically significant association of HLA DR6 and DQ1 in a subgroup of patients with CRPS I. For the first time it is shown that the presence of the TNF2 allele is associated with certain presentations of CRPS I. The finding of a significantly increased TNF2 allele, may open new pathways in the treatment of these presentations of CRPS I.

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# EVIDENCE-BASED DISABIITY DURATION GUIDELINES

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**Phil LeFevre MS, Philip L. Denniston, Jr. MBA**

The practitioners of disability medicine and other related disciplines have seen a massive paradigm shift in the past couple of decades in the use and demand for disability duration guidelines to achieve the ultimate and timely return to health and productivity of the injured workers all over the world. Insurers, employers, Third party administrators (TPAs), attorneys and various healthcare providers, all with their own scattered and sometimes misguided incentives, can easily make a circus of this conscious and reputable institution aimed at the restoration of health to humanity at work - the functional treatment of those men and women of the workforce.

Healthcare professionals have known for sometime that the longer the absence from work the less likely the return to work outcome. The cost of lost time from work is enormous for the economy and has been estimated to be $200 per worker per day. Further, the negative psychological impact of long-term absence from the work force can be devastating to an employee and his/her family and lifestyle. The need for scientifically based disability duration guidelines; indicators for modified duty, and treatment pathways for early return-to-work have become obvious. Healthcare professionals facing the challenge of making clinical decisions about the fitness for duty and timely return to work quite often discover that professional schooling traditionally lacked such training in their curriculum.

As not simply patients, but the great dukes of productivity, the injured men and women are at the center of the tug-of-war where it also happens the occupational physician hangs his hat. And now more than ever, to complement the polished armory of education, training, experience and good humor necessary to stay afloat in the sub specialty that is disability medicine, the occupational physician is turning to an outside source for disability duration information. Once thought to be reserved only for case managers, claims handlers and risk managers, disability duration guidelines, or "return-to-work guidelines", either electronic or traditionally bound, are showing an increased 21st century presence in the offices and clinics of doctors dealing with sick and injured workers. To earn the privilege to sit atop the shelf of the most experienced physicians and independent medical examiner the Return-to-work guidelines in the current market must meet stringent criteria for "evidence-based" medicine to be most effective, medically relevant and ward off litigation.

In conjunction with the quest for evidence-based medicine, the number of treatment guidelines has been mushrooming over the last few years. The National Guideline Clearinghouse, created by the US Government Agency for Healthcare Research and Quality (AHRQ), in partnership with the American Medical Association (AMA) and the American Association of Health Plans (AAHP), offers an Internet-based resource on clinical practice guidelines at www.guideline.gov. As of March 1, 2000, this site provided access to 700 clinical practice guidelines from 125 different organizations[1]. By December 2003, this guideline database is expected to contain a total of 3,500 clinical practice guidelines.[2] Publishers of these guidelines are primarily the professional societies of various healthcare specialty providers and the guidelines are oriented toward treatment by their own members, e.g., neurologists, orthopedic

surgeons, radiologists, physiatrists, chiropractors, etc. Consequently, the guidelines often have reputations for supporting the constituencies of their authors, and not always providing the best multi-disciplinary treatment pathways for each condition. Of the 125 different organizations, only five are commercial entities and the rest are nonprofit.

Unlike treatment guidelines, which recommend maps for treatment that include initial evaluations and specific treatment options, disability duration guidelines, which recommend lost time parameters for return-to-work, are available from only a few publishers. There are currently only four published versions of disability duration guidelines[3], Official Disability Guidelines from Work Loss Data Institute, Medical Disability Advisor from Reed Group, Health Management Guidelines from Milliman USA, and ACOEM's Occupational Medicine Practice Guidelines, the first three of which are published by commercial entities. Of these four guidelines, Official Disability Guidelines from Work Loss Data Institute best meets the criteria for "evidence-based" medicine in terms of lost time and disability duration. Developmentally, the duration recommendations of the remaining three sources are classified as consensus-based. Consensus-based recommendations are valuable as an additional frame of reference, but are not necessarily fair and/or defensible by today's standards.

The latest industry trend towards "evidence-based" disability medicine has largely patterned itself after changes to the admissibility of medical testimony in court. The legal trail is documented well by a California legal nurse consultant and regular testifier in workers' compensation litigation, Linda Stutzman, who explains, "Three U.S. Supreme Court cases, beginning with the 1993 Daubert Decision[4], which held that judges were obligated to evaluate the basis for expert testimony, and following with two additional expert testimony cases, GE v. Joiner[5] and Kumho Tire[6] have set the standards for the way federal courts approach expert testimony and are profoundly affecting State court practice as well. According to the Claims Support Professionals Association, experts with a long tradition of being readily admitted, such as clinical medical doctors, are now being excluded from court when they testify based on their opinions alone.[7]" The ripple effect here is that traditional guidelines, both disability duration and treatment, based on "expert" opinion and consensus judgment, as intellectual as they may be, are often also precluded.

As explained further by Stutzman, "A recent roundtable workshop was held by the federal guideline agency AHRQ and the Institute of Medicine, which concluded, 'Evidence-based medicine in practice defines the likelihood of something happening.' The workshop identified population-based evidence as the most important in court.[8] As a result, the Federal Rules of Evidence were amended in December 2000. The new rules state that statistical studies are admissible under the Federal Rules of Evidence, and that such methods generally satisfy important aspects of the "scientific knowledge" requirement articulated in the Daubert Decision.[9] Furthermore, it states that 'courts have described surveys as the most direct form of evidence that can be offered, and have drawn negative inferences from the absence of a survey.'[10] "

Hence "evidence-based" medicine, developmentally, uses statistical analysis of population-based material, preferably survey, to pattern outcomes, like disability durations, after experience. In her August 2001 article, "Evidence-Based Return-to-Work Guidelines", published in the *California Workers' Comp Enquirer*[11], Stutzman continues, "These developments have significant impact on the outcome of court cases involving workers' comp claims, and may even lessen the weight of Independent Medical Examination (IME) testimony that does not also reference evidence-based medicine. Furthermore, primary credence will be based on actual experience data, and not expert judgment in determining what should happen. When this experience

data is backed by a credible survey, such as the CDC National Health Interview Survey, it will have even more weight."

The above referenced National Health Interview Survey from the Centers for Disease Control and Prevention serves as the backbone and the very basis for *Official Disability Guidelines*, coupled with the Survey of Occupational Injuries and Illnesses from the Bureau of Labor Statistics, two of the most valuable lost time and restricted activity databases in North America today. Due primarily to this incredible stronghold in evidence-based methodology, *Official Disability Guidelines* from Work Loss Data Institute is playing a leading role in the industry migration, acting as a "home plate" to hunted base runners in the work comp and disability markets. This disability duration database is built around over 3 million total cases from CDC and OSHA BLS, so it holds true to the term "evidence-based". This is especially important now, with hotshot plaintiffs attorneys targeting healthcare providers and the workers' compensation systems in all 50 States, and patient & labor groups advocating to dismiss and discredit existing consensus-based disability guidelines.

For example, in a letter dated February 19, 2001 to the U.S. Department of Labor, the patient advocacy group for chronic fatigue and fibromyalgia, CFIDS, calls one of the leading consensus-based guidelines, the *Medical Disability Advisor* by Reed Group, "inconsistent with generally recognized medical standards"[12]. This letter requests that the Department provide additional detailed formal regulatory guidance as to the remedies available to plan participants and beneficiaries when an insurance company or other plan administrator fails to disclose the use of consensus guidelines from either *Medical Disability Advisor* by Reed Group or Milliman USA. In fact, guidelines from Milliman USA have taken the most heat in Texas and other States, as they have an aggressive reputation for representing a managed care constituency[13].

The scrutiny and credibility issues seem to be "just part of the territory" with consensus guidelines when used to judge or forecast outcomes in unique and individual cases, as all cases are. Expert opinions are easily and often just countered by each other. But why don't these issues play the role of obstacle in reference to evidence-based guidelines? The difference comes down to one thing: accountability. Using evidence-based guidelines to judge and forecast outcomes in unique and individual cases is similar to making inferences about a given population, based on statistically significant characteristics of a representative sample. The probabilities are high and have unquestionable value.

In this regards it should be noted that thelegitimate assumptions can be made about particular disability cases based on the duration behavior of large representative samples of either the working or civilian, non-institutionalized populations. In the case of *Official Disability Guidelines*, evidence-based medicine is not simply a catch phrase, but the accountability to the Survey of Occupational Injuries and Illnesses from BLS and the CDC's National Health Interview Survey, one of the oldest, most respected national health surveys in the United States, in continuous operation since July 1957.

Legal reasons aside, an experienced occupational doctor knows that evidence-based criteria in duration guidelines will promote "buy in" from all parties, including patients and employers, and allow for a focal point on which non-medical personnel can become comfortable, and a legitimate yardstick on which all parties can forecast downtime. An important aspect of promoting and coordinating recovery is to be fair and non-adversarial. While this behavior is usually the case, evidence-based guidelines serve as the proof. They allow for normal and effective recovery periods based on national statistics, so once injured, it is clear that each patient is not expected to outperform the rest of the free world. Physicians who make patients, case managers, insurers and employers aware of expectations can

show them to be consistent with national norms, ensuring to all parties they are realistic. With the availability of evidence-based guidelines from *Official Disability Guidelines*, the tug-of-war in the return to work process is beginning to vanish. With a little luck, everyone may someday be operating on the same team.

The industry migration speaks clearly, and the demand is for credible, experience, normative data as a fundamental ingredient to the construction of return to work guidelines. To an occupational physician measuring time away from work, while each patient is always more important than the norms, the norms are more important than anything else.

Above all, evidence-based guidelines in the arena of disability medicine make for a healthy and conducive recovery environment. Gone are the days when manipulators and puppeteers can graffiti the walls of this conscious and reputable institution with the filth of self-servitude. The industry is embracing Official Disability Guidelines from Work Loss Data Institute as it meets alone the stringent criteria for "evidence-based" medicine, and walks hand-in-hand with the silhouetted occupational doctor of tomorrow.

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# Vocational rehabilitation for prevention of disability in Iceland

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*Key words:* vocational rehabilitation, prevention of disability

## Abstract

*Objectives:* An evaluation of the effectiveness of vocational rehabilitation organized by the State Social Security Institute (SSSI) of Iceland.

*Material and methods:* The participants were 109 individuals who were unfit for work. They had been referred to a multidisciplinary team for assessment of rehabilitation potential and an advice on the appropriate type of rehabilitation. The outcome of the rehabilitation was evaluated 1-2 years after the appointment with the multidisciplinary team. The study group was compared to a similar group of people that began to receive rehabilitation benefits before the SSSI could offer vocational rehabilitation.

*Results:* Overall, the rehabilitation was successful, 72% of the study group stated that their fitness for work had increased after rehabilitation, 47% had returned to work and 23% were students. It is likely that a part of the students will return to work at a later date. Approximately half of the study group said that the rehabilitation programme had increased their self-confidence and self-reliance. Between one and two years after the rehabilitation evaluation 40.4% of the study group received no social security benefits and 40.4% received disability pension, whereas one and a half year after starting to receive rehabilitation benefits 17.7% of the comparison group received no social security benefits and 81.5% received disability pension

*Conclusion:* Vocational rehabilitation as organized by the SSSI is effective and can prevent disability and increase self-confidence and self-reliance amongst recipients.

## Introduction

Disability pension has been more prevalent among young people in Iceland than in the other Nordic countries (Denmark, Finland, Norway and Sweden).[1] One reason for this was a lack of suitable vocational rehabilitation in Iceland for those who are unfit for work due to health problems. Research has demonstrated that when people have been unfit for work for more than a few months, their self-esteem, self-reliance and footing on the labour market decrease rapidly.[2-4] The cost of disability for the society is high[5] and it can have a harmful effect on the quality of life of those that have to stop working. Therefore, it is important to be able to offer suitable rehabilitation at an early stage to prevent life-long disability. Money spent on vocational rehabilitation returns a multiple profit to society.[4, 6]

An amendment to the Icelandic law on social security came into force on 1 September 1999.7 In addition to establishing a new basis for disability evaluation (introduction of the British All Work Test) this law states that the physicians working for the State Social Security Institute (SSSI) can demand of those claiming disability pension that they try rehabilitation before their degree of disability is evaluated. The

SSSI put together a multidisiplinary team to evaluate the rehabilitation potential of those who have been unfit for work and are deemed not likely to return to work on their own accord and to advice on appropriate type of rehabilitation.[8] The team also guides people through the "jungle" of the welfare system. The team is led by a rehabilitation physician and also includes a social worker, a physical therapist and a psychologist. The physician decides which members of the team each individual needs to see. Referrals are from physicians working for the SSSI, often according to proposals from treating physicians. The SSSI also reached an agreement with two rehabilitation centres, Hringsja and Reykjalundur, about conducting the vocational rehabilitation.

The vocational rehabilitation centre Hringsja provides education and advice with the objective of increasing fitness for work or further studies. Each individual is evaluated and he is taught how to handle his own expectations, skills, capacities and limitations. A full vocational rehabilitation program takes one and a half to two years. The program is aimed at increasing general knowledge and skills needed for clerical or commercial work. In addition Hringsja offers shorter courses, such as personal computer training.

Reykjalundur is the largest rehabilitation clinic in Iceland, offering both medical and vocational rehabilitation. There the individual's capacity for work is evaluated. The emphasis is on education, improved body image, postures at work, increased working endurance and general strength. The individuals are assisted in setting realistic goals for themselves according to their skills and capacities and in finding a suitable job. The average length of stay at Reykjalundur is two months.

In order to evaluate the effect of rehabilitation offered by the SSSI, the progress of the 109 individuals who were evaluated by the multidisiplinary team in its first calender year of existence (the year 2000) was monitored.

## Material and methods

Participants were all those that were referred to the newly formed multidisciplinary team. In all there were 109 individuals. The participants were promised anonymity and full confidenciality. Answers were obtained from 83 (76.1%). Non-respondance (23.9%) was mainly due to residence abroad or lack of willingness to participate in the survey. No personal information was utilized in the survey.

The study material came from the medical reports of the multidisciplinary team, the SSSI disability register and from a telephone survey carried out by the Social Science Research Institute in October 2001. From the multidisciplinary team information was obtained on diagnoses, gender, age, marital status, number of children, level of education and place of rehabilitation. From the telephone survey information was obtained on the effect of rehabilitation on fitness for work, current employment status, self-confidence, self-reliance as well as an evaluation on the usefulness of the rehabilitation evaluation. An attempt was made to contact all 109 individuals who had been evaluated by the multidisciplinary team in the year 2000. Data was collected from the disability register at the SSSI on whether those evaluated by the multidisiplinary team received any social security benefits in November 2001. For comparison, a group that had started to receive rehabilitation benefits in November or December 1997, was used. In this period the SSSI could not offer vocational rehabilitation and the multidisiplinary team had not been formed.

Groups were compared with the chi-square test.[9] Otherwise, data was analyzed using descriptive statistics.

## Results

Of the 109 individuals evaluated by the rehabilitation team there were 74 women (68%) and 35 men (32%), aged between 18 and 57 years (mean age 35 years). In 85 cases (78%) the main diagnosis was a musculosceletal disorder, in 12 cases (11%) a mental

## Table 1. Marital status of the study group and the general population in Iceland*.

| | Study group | | The Icelandic population | |
|---|---|---|---|---|
| | Number | Percentage | Number | Percentage |
| Married or in co-habitation | 45 | 41.3 | 681 | 67.2 |
| Unmarried/not in co-habitation | 35 | 32.1 | 258 | 25.4 |
| Divorced | 25 | 22.9 | 49 | 4.8 |
| Widows/widowers | 4 | 3.7 | 26 | 2.6 |
| Total | 109 | 100 | 1014 | 100 |

* Information on the marital status of the Icelanders was obtained from a national survey carried out in the year 2000 by the Social Science Research Institute at the University of Iceland

## Table 2. Number of children supported by the study group and by the general population in Iceland*.

| Number of children | Study group | | The Icelandic population | |
|---|---|---|---|---|
| | Number | Percentage | Number | Percentage |
| 0 | 38 | 34.9 | 589 | 58.2 |
| 1 | 22 | 20.2 | 187 | 18.5 |
| 2 | 23 | 21.1 | 148 | 14.6 |
| 3 | 19 | 17.4 | 74 | 7.3 |
| 4 or more | 7 | 6.4 | 14 | 1.4 |
| Total | 109 | 100 | 1012 | 100 |

* Information on the number of children supported by the Icelanders was obtained from a national survey carried out in the year 2000 by the Social Science Research Institute at the University of Iceland

## Table 3. Educational level of the study group and of the general population in Iceland*.

| | Study group | The Icelandic population |
|---|---|---|
| Primary and lower secondary education | 80.7% | 44.0% |
| Grammar school or vocational training | 15.6% | 41.0% |
| University education | 3.7% | 15.0% |
| Total | 100% | 100% |

* Information on the educational level of the Icelanders in the year 2000 was obtained from Statistics Iceland

disorder was the main diagnosis and in the other 12 cases it was various other disorders. Many of those that suffered from musculosceletal disorders also suffered from mental disorders. Thus the main medical reasons for referral to the team were musculosceletal and mental disorders.

After evaluation 40 individuals were referred to a 2 months vocational rehabilitation at Reykjalundur rehabilitation clinic, 19 were referred to a 6 week computer training at Hringsja and further 15 to a longer (usually 18 months) rehabilitation programme in the same centre. In addition to these specific rehabilitation modalities offered by the SSSI, 46 individuals received other treatment (such as medical rehabilitation in a rehabilitation clinic, physical therapy or psychiatric treatment) or education.

Tables 1, 2 and 3 show the background of those evaluated by the rehabilitation team compared to the general population of Iceland. The evaluees were more likely to be unmarried or divorced than the general population ($X2 = 61.270$, $p<0.01$), had more children ($X2 = 37.923$, $p<0.01$) and a lower educational level ($X2 = 59.867$, $p<0.001$) than the general population.

Table 4 shows the social security benefits received by the study group in November 2001, almost two years after the evaluation by the multidisciplinary team. About 40% of the study group did

not receive any benefits in November 2001, about 18% received rehabilitation benefits, indicating that rehabilitation was not completed, and a further 40% received full or partial disability pension. The table also shows that in the comparison group about 18% did not receive any benefits from the SSSI approximately one and a half year after first receiving rehabilitation benefits. A comparison of the two groups shows that the comparison group is much more likely to receive some kind of benefits than the study group ($X2 = 14.409$, $p<0.01$).

The participants in the telephone survey were asked about the effect of vocational rehabilitation on their fitness for work. In all, 25% said it had great or considerable effect, for 47% the effect was moderate and for 28% it had very little or no effect. More than half (56%) of the participants considered themselves unfit for work, 36% partially fit for work and 8% fully fit for work. About 47% had been in paid employment at one time or another after finishing rehabilitation, 23% where students and 30% were out of work. One third of those who were able to work had done so as soon as they had finished rehabilitation, one third within 2 months and one third later. Of the 38 who had been employed after rehabilitation 51% had been in continuous employment and of those 74% were employed at the time of the survey (29 out of the 83 participants in

## Table 4. Social security benefits paid to the study group and the comparison group.

| | Study group | | Comparison group | |
|---|---|---|---|---|
| | Number | Percentage | Number | Percentage |
| No benefits | 44 | 40.4 | 21 | 17.7 |
| Rehabilitation benefits | 20 | 18.3 | 1 | 0.8 |
| Full disability pension | 32 | 29.4 | 86 | 72.3 |
| Partial disability pension | 12 | 11.0 | 11 | 9.2 |
| Maternity benefits | 1 | 0.9 | 0 | 0.0 |
| Total | 109 | 100 | 119 | 100 |

## Table 5. Effect of vocational rehabilitation on self-confidence and self-reliance in the study group.

| | Has increased | Has not changed | Has decreased | Mean-score | Standard deviation |
|---|---|---|---|---|---|
| Self-confidence* | 47.0% | 42.2% | 10.8% | 5.7 | 2.4 |
| Self-reliance* | 53.8% | 40.0% | 6.2% | 6.3 | 2.2 |

*The scale was from 0 to 10 and 0 meant that the situation had become much worse, 5 that it had not changed and 10 that the situation had become much better.

## Table 6. Usefulness of the rehabilitation evaluation according to the study group.

| | Great or considerable | Moderate | Slight or none |
|---|---|---|---|
| Usefulness of the rehabilitation evaluation | 47.0% | 22.9% | 30.1% |
| Usefulness of meeting the rehabilitation team | 54.2% | 24.1% | 21.7% |

## Table 7. The knowledge members of the study group had of available rehabilitation services.

| | Yes | No |
|---|---|---|
| Did you know what kind of rehabilitation services were available? | 16.9% | 83.1% |
| Where you referred to a service you did not know about? | 59.0% | 41.0% |

the survey). Out of those employed, 42% were in full employment, 46% in partial employment (50-80%) and 11% in less than 50% employment. Figure 1 shows that a considerably higher ratio of those who said that their fitness for work had increased after rehabilitation had been in paid employment compared to those who said their fitness for work had not improved.

## FIGURE 1: THE NUMBER OF THOSE IN PAID EMPLOYMENT ACCORDING TO THEIR OWN EVALUATION OF CHANGES IN FITNESS FOR WORK AFTER REHABILITATION

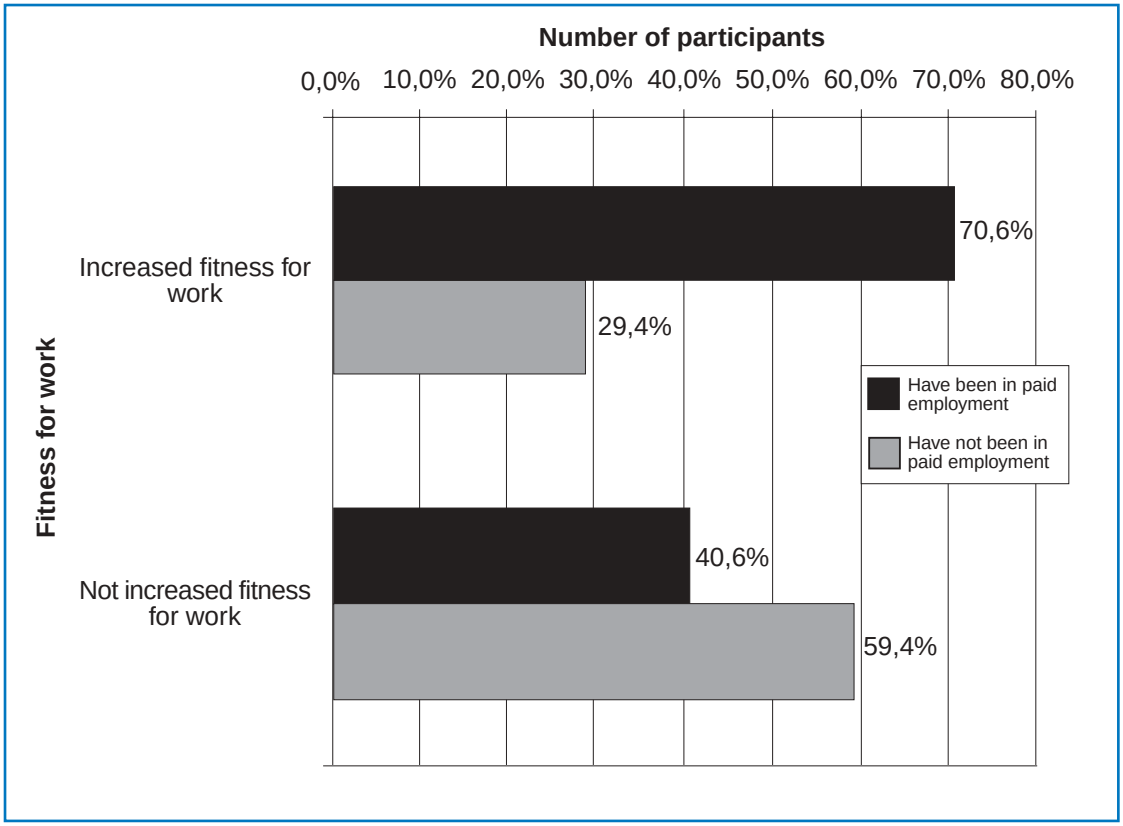


Table 5 shows the effect of vocational rehabilitation on self-confidence and self-reliance of the participants. Almost half (47%) said their self-confidence had increased and only a small minority (11%) said it had decreased. On the whole the self-confidence of the group had increased (M = 5.7). The effect on self-reliance was more pronounced as 54% said that it had increased and very few (6%) said it had decreased. On the whole the self-reliance of the group had increased (M = 6.3).

The usefulness of the rehabilitation evaluation was addressed in the survey. Almost 64% of the participants were positive towards the evaluation before it was carried out, 20% were neutral and 16% negative. Almost half (47%) felt that the evaluation had been useful and 54% felt that meeting the members of the evaluation team had been useful (table 6). The evaluation received from the team was in line with expectations for 42% of participants but did not meet expectations of 37% and 21% experienced it as more positive than expected.

The knowledge on available rehabilitation services was also addressed in the survey (table 7). Only 17% knew of the available rehabilitation services before the rehabilitation evaluation and 59% of the participants were referred to a rehabilitation service they did not know about.

## Discussion

The main finding of this study is that the new program for evaluation of rehabilitation potential and vocational rehabilitation introduced by the SSSI is successful. The characteristics of the study group are comparable to those that receive disability pension in Iceland.1, [10-13] The main medical reasons for referral for rehabilitation evaluation were musculosceletal and mental disorders. In the study group there were about twice as many women as men and the participants were more likely to

be unmarried or divorced, had more children and a lower educational level than the general Icelandic population. Lack of education beyond primary or lower secondary education makes it likely that further education will improve their position in the labour market and this is the backbone of the vocational rehabilitation offered by the SSSI.

The majority of the participants in the telephone survey stated that rehabilitation had increased their fitness for work. After the rehabilitation 47% had been in paid employment and 51% of them in continuous employment. Almost a quarter of the participants were students at the time when the survey was carried out. It is likely that a part of them will return to work when their studies are completed. In addition to the direct effect on working capacity, approximately half of the participants reported that their self-esteem and self-reliance had increased after participating in the rehabilitation program.

One to two years after the rehabilitation evaluation 44 (40%) out of the 109 people evaluated by the multidisciplinary team did not receive any social security benefits from the SSSI and only 18% were receiving rehabilitation benefits, as rehabilitation was not completed. Furthermore, only 29% received a full disability pension and 11% a partial disability pension. In the comparison group only 18% were without social security benefits from the SSSI after a comparable length of time from starting receiving rehabilitation benefits. It can be assumed that a much greater proportion of the study group would be receiving disability pension were it not for the vocational rehabilitation organized by the SSSI. This clearly demonstrates that this program of vocational rehabilitation is effective in preventing disability.

The male, female ratio, mean age, marital status and diagnoses of the study group are similar to those reported in a study on the effect of vocational rehabilitation organized by

the Social Insurance Office of Stockholm.[14] In that study approximately 45% were without social security benefits after one year and 36% two years after commencement of vocational rehabilitation. In another Swedish study 42% were without social security benefits one year after completion of vocational rehabilitation organized by the Social Insurance Office of Jämtland (in the northern part of Sweden)2 and in a study from the southern part of Sweden 48% were without social security benefits two years after completion of rehabilitation organized by Social Insurance Offices.15 These studies show comparable results as our study, where 40% of those referred were without social security benefits after one to two years. Thus, the preventive effect of vocational rehabilitation on disability in Iceland is comparable to that in Sweden.

In our study 54% of those evaluated by the multidisiplinary team stated that it was useful to meet the members of the team. This result is more positive than expected, as the primary role of the team is to evaluate rehabilitation potential and not to administer treatment. It can be expected that the majority of those referred to the team have gradually been loosing faith in their own ability to take care of themselves and would therefore not have been particularly positive towards any action by the SSSI other than granting them a disability pension. On the other hand, in the meetings with the members of the team they have presumably received encouragement, information and useful advice. The fact that 59% of those evaluated were referred to rehabilitation services they had not known about confirms this. These results are interesting and unexpected when compared to the results of a study on the opinion of people on vocational rehabilitation organized by Social Insurance Offices in three regions in the southern part of Sweden. The participants in this study had been evaluated by a team consisting of a physician, an occupational therapist, a physical therapist and a social worker. Only three out of 24 were satisfied with the rehabilitation, further three were ambivalent as to the benefits of the program and 18 (75%) stated that the rehabilitation had not been of any help at all. Some of them regarded the rehabilitation as some kind of test on behalf of the Social Insurance Office and many complained that the communication between the members of the team and also the communication between the team and the Social Insurance Office had been unsatisfactory.[15]

Conclusion: This study shows that vocational rehabilitation organized by the SSSI is effective in preventing disability and increasing self-confidence and self-reliance amongst those who receive their services.

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# Evaulation of Sexual Function Disability: A Systematic approach

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The role of sexuality in a person's life is subject to assessment that rarely can be independent from political, metaphysical and social conventions and tendencies. Yet, regardless of the disposition of any given medical provider, the medical profession must view sexuality as bona fide valid, indispensable and integral part of health. This approach is supported by the well-founded and often postulated assessment of sexuality as one of the foundations of unique psychosocial interactions that construe the human form of life.

Sexual dysfunction is a disabling condition in both sexes and in all genders and should be the focus of treatment, management and rehabilitation effort as much as any other condition that reduces the quality of life. Unfortunately, due to the innate communication disparity that pervades the provider-patient relationship, issues in sexuality are often left outside of the daily practice and are brought to the attention of the provider by the patient, an approach that may be deficient or ineffective.

The multidisciplinary approach to sexual dysfunction affords a number of benefits that include the finer understanding of specific pathophysiology of sexual function as well as the functional basis of neurological and psychosocial changes.

## COMMUNICATING WITH THE SEXUAL DYSFUNCTION PATIENT

Certain terminological and methodological issues need to be addressed to assure full and effective communication with the patient. Often even a brief explanatory statement by the clinician provides for a better framework and faster, more efficient information exchange. Examples of methodological issues that are often unknown to the patient include the fact that the term erectile dysfunction currently is used instead of the term impotence to indicate inability to achieve or sustain erection sufficient for satisfactory sexual function. From the factual standpoint it is often worth mentioning that statistical research indicates that more than 50% of men 40 years and older have experienced some degree of erectile dysfunction.

In both sexes sexual arousal is defined as the consequence of erotogenic psychological and physical response that leads to increased blood pressure, heart and respiratory rates, and the lubrication-swelling response of the genitalia. This clinical concept also needs to be elucidated to the patient, since a substantial majority of population lacks the knowledge of sexual physiology and the expected response. Both unnecessarily low and unsustainably high expectations have been observed in patients with sexual disability and the emphasis on realistic expectations is often the most important part of communication.

Sexual arousal is not to be confused with sexual response, since the latter involves sexual activity, most commonly in the form of sexual intercourse. There are numerous classifications of sexual response, of which the four stage classification (Excitation - Plateau - Orgasm - Resolution) due to Masters and Johnson is the most commonly used and is the easiest to explicate to the patient.

Communication problems due to speech and language deficits, such as nonfluent

speech, word finding deficits, and memory loss, may aggravate attempts to work out mutually agreeable solutions. Under such conditions a neutral intermediary, such as a counselor, may be helpful.

Patients need to be questioned about their perception ("self-assessment") of sensory changes. What previously was pleasurable may be irritating, and vice versa, while reduced sensation, reduced attention or increased distractibility as a presentation of a neurologic condition may often be the source of sexual dysfunction.

## PRELIMINARY INVESTIGATION OF THE VECTOR OF SEXUAL DYSFUNCTION

Since the sexual functioning sphere is highly liable to both psychological and organic challenges, the first step in determination of the best course of action should be the establishment of the primary vector of dysfunction. The following table (Modified from: BMJ 1999;318:518-521, ABC of sexual health: Sexual problems of disabled patients; **Clive Glass, Bakulesh Soni.** ) may be of use as a rough guide; however, the clinical judgment must only be formulated and implemented after a full and thorough evaluation of all aspects of the case.

| Mainly psychological cause of problem | Mainly organic cause of problem |
|---|---|
| ● Acute onset | ● Generally slower onset |
| ● General relationship with partner (excluding sexual problem) is poor | ● Good, reasonably harmonious relationship with partner |
| ● Symptoms not consistent in all situations or with all partners | ● S ymptoms consistent in all situations and with all partners |
| ● Major life events (births; deaths; potential or actual change in relationship, health, job) often precede or coincide with development of dysfunction | ● Major life events rarely present |
| ● Mental or physical comorbidity rarely present | ● Mental or physical comorbidity common |
| ● Can respond to self stimulation | ● No response to self stimulation |
| ● Commonly aged <50 years | ● Commonly aged >50 years |
| ● Genitalia (including prostate) and secondary sexual characteristics seem normal; men with erectile dysfunction have nocturnal or early morning erections | ● Genitalia and secondary sexual characteristics show abnormal structure or development; no nocturnal or early morning erections in men with erectile dysfunction |
| ● Normal results on examination and/or comprehensive laboratory evaluation | ● Abnormal results on examination and/or comprehensive laboratory evaluation |

*Modified from BMJ 1999;318:518-521; ABC of sexual health Sexual problems of disabled patients, Clive Glass, Bakulesh Soni.*

## EVALUATING THE PATIENT'S DISABILITY

If disability is observed, declared by the patient or suspected, the following key questions need to be answered and noted, especially in cases that involve extended history of being disabled (e.g., congenital defects) or substantial neurologic and/or cognitive changes in the patient.

## Key questions in cases of disability

Present condition

- Does the person have a congenital or an acquired disability?
- What is the patient-specific history of psychosocial changes caused by disability?
- Is the patient's condition static, stabilized or deteriorating?
- Is the patient's condition observable by others or co-

experienced by others (partners, caregivers etc.)?

- Effect of condition on sexuality
- Does the disability affect sexual function or sexuality?
- Does the disability impair cognitive, intellectual or communication ability?
- Are there associated iatrogenic factors?
- Is fertility and reproduction a concern?

*Modified from BMJ 1999;318:518-521; ABC of sexual health Sexual problems of disabled patients Clive Glass, Bakulesh Soni.*

Upon establishment of the underlying factors, the clinician should proceed with investigation of the presence and degree of the disability criteria. These criteria differ between the sexes and often require extensive examination, laboratory workup and clinical interpretation.

## DISABILITY CRITERIA IN MEN:

Deficits in the domains of erection, ejaculation, sensory, orgasmic function or libido are used to determine areas of sexual disability. The severity of each domain is assessed and may be quantitated by using the IIEF (International Index of Erectile Function).

Symptoms and subjective reports may be confirmed or contradicted by incorporation of the NPT, biothesiometry, intracavernous injection test and duplex Doppler ultrasound.

Criteria for impairment are based on whether dysfunctions require ongoing treatment, if sufficient erectile rigidity is maintained, and if adequate orgasmic, libido and sensory function is present.

## DISABILITY CRITERIA IN WOMEN:

Criteria for impairment determination of female sexual function are based on whether deficits require continue treatment and whether sexual intercourse and vaginal delivery are possible. Consideration should be given to integrity of sexual desire, vaginal lubrication and capacity. Grading of these symptoms may be quantitated at baseline and followed by using the Index of Female Sexual Function (IFSF).

### TABLE-1 Symptoms suggestive of organic Male Erectile Dysfunction:

- Inability to achieve or maintain erection
- Ejaculatory dysfunction (decreased force, volume, etc)
- Less pleasurable orgasm
- Diminished libido
- Genital numbness
- Angulated erection
- Genital pain

In the absence of standardized parameters, currently available laboratory testing is investigational and may be used in a confirmatory but not a diagnostic role.

### TABLE-2 Symptoms suggestive of organic Female Sexual Dysfunction:

- Loss of desire
- Dyspareunia/Genital pain
- Vaginismus
- Diminished sexual responsiveness
- Difficulty achieving orgasm
- Diminished genital sensation
- Vaginal dryness

## FEMALE MEDICAL/ PHYSIOLOGIC EVALUATION:

Comprehensive evaluation of FSD includes a full medical, surgical and sexual history (Table-2), physical and pelvic exam and hormonal profile (serum FSH, LH, prolactin, testosterone, SHBG, DHEA and estradiol). Medical conditions associated with hypothalamic-pituitary-ovarian dysfunction include menopause, chemotherapy, or surgical bilateral salpingo-oophorectomy. Classes of medications that adversely affect female sexual function include antihypertensives, diuretics, antidepressants, SSRI's, anxiolytics,

neuroleptics, anticonvulsants and anticancer agents. Complete laboratory evaluation of the components of the female sexual response is performed by hemodynamic, chemical, compliance and sensory studies (Table-3). Examination of these parameters is performed before and after visual and vibratory sexual stimulation. At present, efforts at standardization and definition of normal and abnormal parameters is underway. Until standardization of such normative laboratory parameters FSD impairment determination must rely on history and physical examination. Laboratory studies may be used in a confirmatory role. By contrast, laboratory testing in the evaluation of male erectile dysfunction is standardized and widely accepted. Thus such testing should be performed liberally in men, especially if there is any doubt as to whether the ED is organic or psychogenic or if there are prior comorbid risk factors such as smoking, vascular disease, offending medications or diabetes.

## MALE ERECTILE PHYSIOLOGY AND ANATOMY

Penile erection is a neurovascular event initiated by psychosomatic stimulation resulting in hemodynamic alterations required for erection.[45, 46] Cortical and hypothalamic activity influence erection through spinal reflexes and supraspinal influences. These neural pathways are modulated by serum testosterone, whose function is to maintain appropriate libido and ejaculatory function.

**TABLE-3**
**Laboratory assessment of the female sexual response:**

| FUNCTION | STUDY |
|---|---|
| Genital Blood Flow | Vaginal photoplethysmography<br>duplex Doppler ultrasound |
| Vaginal Lubrication | pH measurement |
| Vaginal Compliance | Vaginal pressure/volume balloon |
| Genital Sensation | Vibratory and temperature sensation thresholds |

Local arterial flow within the hypogastric-pudendal system, corporal smooth muscle relaxation and increased venous outflow resistance within the corpora cavernosa are the vascular events resulting from such neural stimulation. Corporal smooth muscle relaxation is synonymous with erection and is stimulated by local neurotransmitters. Penile flaccidity results from the release of norepinephrine from local sympathetic nerve endings, resulting in contraction of corporal smooth muscle. Thus, the tone of corporal smooth muscle is the determinant of penile erection whose primary neurotransmitter is nitric oxide (NO).

Normal erectile function requires intact neural stimulation of these hemodynamic events by the pelvic autonomic nerve fibers.

### Penile Arterial Supply

Primary arterial supply of the corpora cavernosa derives from the internal pudendal artery, which gives rise to the scrotal, bulbar and common penile arteries. The common penile artery further trifurcates into the bulbourethral artery, the cavernous artery and the dorsal penile artery. It is the cavernous artery that supplies the central inflow within each corpus cavernosum during erection.[47]

### Penile Venous Anatomy

Venous drainage of the penis consists of superficial, intermediate and deep venous systems. Beneath the capsule of the corpora cavernosa (tunica albuginea), course the emissary veins that run within the layer of this thick fibrous capsule and empty into the deep dorsal vein. This intermediate system drains into the internal pudendal vein. The glans penis is drained by the deep dorsal vein, which courses into the periprostatic plexus. The superficial dorsal vein drains the skin and subcutaneous tissue of the penis and

drains into the superficial external pudendal vein.[48] Appropriate veno-occlusive function, central to the maintenance of erection, is accomplished by compression of the emissary veins during increases in intracavernous pressure blocking venous outflow and establishing a durable erection.[49,50]

## Functional Neuroanatomy of Penile Erection

Parasympathetic input of the penis originates from the sacral spinal cord (S2-S4). Preganglionic fibers from the sacral roots form the pelvic nerves that are joined by fibers from the inferior hypogastric sympathetic system to form the pelvic plexus (inferior hypogastric plexus), which is situated between the rectum and bladder. Both the sympathetic and parasympathetic postganglionic fibers contribute to the formation of the cavernous nerve, which enters the corpus cavernosum.

Sympathetic neural outflow is responsible for penile flaccidity, seminal emission and ejaculation. Sympathetic preganglionic nerve fibers arise from the intermedial, lateral gray cell column of the thoracolumbar spinal cord (T10-L2). Preganglionic fibers leave the cord in the ventral roots and pass along the white rami communicantes of the paravertebral sympathetic chain. These fibers then leave the chain at the sacral level and course through the pelvic nerves to the pelvic plexus and contribute to the formation of the cavernous nerve. With the formation of the hypogastric nerves, sympathetic fibers supply the smooth muscle of the vas deferens, seminal vesicle, prostate and bladder neck.

Seminal emission is controlled by the hypogastric nerves originating from the T10-L2 spinal cord levels.

## Somatosenosry Innervation of the Penis

Penile sensory innervation, an important component of sexual function, derives from the dorsal penile nerve, which joins the pudendal nerve.51 Penile sensory function is modulated by the S2-S4 segments of the spinal cord. The motor neuron cells of the pudendal nerve form the ventrolateral group in the anterior gray column of the segments called Onuf's nucleus. These motor neurons supply the striated muscles, bulbocavernosus and ischial cavernosus and the perineum. Thus, a pudendo-pudendal reflex is completed by the sensory and motor components of the pudendal nerve. The pudendal somatic center (S2-S4) is involved with projectile ejaculatory function by involving a relaxation of the external urinary sphincter, rhythmic contractions of the ischiocavernosus and bulbocavernosus muscles and contraction of pelvic floor musculature.

## ANATOMY AND PHYSIOLOGY OF FEMALE SEXUAL FUNCTION

While female sexual dysfunction has historically been of a psychological focus, it has become increasingly evident that female sexual dysfunction (FSD) can have organic etiology. Ongoing research suggests that the risk factors of male and female sexual dysfunction share parallel risk factors and include aging, hypertension, smoking and dyslipidemia.[52, 53]

## Female Pelvic Anatomy

It is helpful to consider female pelvic anatomy in two categories: the external and internal genitalia. The organs of the external genitalia are collectively known as the vulva, consisting of the labia, interlabial space, clitoris and vestibular bulbs. The internal genitalia consists of the vagina, uterus, fallopian tubes and ovaries.

Innervation of the vagina is both autonomic and somatic. The autonomic innervation originates from the superior hypogastric plexus and the pelvic plexus. Sympathetic fibers originate in the lateral gray column of T11-L2 and form the hypogastric plexi. Parasympathetic fibers originate from the intermedial lateral cell column of S2-S4 and synapse in the pelvic plexus. Both sympathetic and parasympathetic fibers leave the pelvic plexus and course within the uterosacral and cardinal

ligaments along with respective blood vessels to supply the proximal 2/3 of the vagina and the corpora of the clitoris. Somatic motor fibers originate from the anterior horns of the sacral spinal cord (S2-S4) and travel within the pudendal nerve to innervate the bulbocavernosus and ischiocavernosus musculature. Sensory fibers innervating the introitus and perineum travel within the perineal and posterior labial nerves into the pudendal nerve.[54]

During sexual arousal, genital vasodilatation occurs resulting in lubrication and secretions by the uterine glands. Engorgement of the vaginal wall raises capillary blood pressure and creates a transudate of plasma through the vaginal epithelium.[55]

The clitoris , an erectile analog of the penis, shares a common embryologic origin with the corpora cavernosa in the male. It is composed of a head, body and innermost crura.

The clitoral response to sexual stimulation is increased blood flow to the clitoral cavernosal arteries, increasing clitoral intracavernous pressure and protrusion. Clitoral tumescence but not rigidity occurs during sexual arousal. Duplex Doppler ultrasound of the clitoris reveals increases in both length and diameter of these arteries and that peak systolic flow velocity doubles during sexual arousal.[53]

Uterine and cervical glands secrete mucous during sexual arousal for the purpose of vaginal lubrication. Understandably, uterine and pelvic surgical procedures can significantly impact on female sexual response and function. Uterine innervation, located adjacent to the bladder and vagina, can potentially be disrupted during any female pelvic surgical procedure, thus alterations in post-operative sexual function, arousal and orgasmic function can occur. Even hysterectomy alone without removal of the ovaries, can result in female sexual dysfunction.[56] Common post-operative sexual complaints can include loss of desire, decreased frequency of sexual activity, painful intercourse, diminished sexual responsiveness, orgasmic dysfunction and diminished genital sensation. The basis for sexual dysfunction symptoms following hysterectomy are likely combined neurovascular and hormonal in origin. Currently our understanding of precise neurovascular structures in the female pelvis is limited but nonetheless, vital to normal sexual arousal and function. We do know that hysterectomy and ligation of the arterial supply at the uterine pedicles can result in both ovarian atrophy and vaginal wall and clitoral smooth muscle fibrosis. Disruption of the uterosacral and cardinal ligaments and pelvic and cervical plexi are associated with genital arousal and orgasmic deficiency.

## Pelvic Floor Muscles

In addition to its function of abdominal and pelvic organ support, the pelvic floor musculature maintains continence and permits intercourse and parturition. The pelvic diaphragm is formed by the levator ani muscles, the urogenital diaphragm and the perineal membrane. The perineal membrane consists of the ischial cavernosus, bulbocavernosus and superficial transverse perineal muscles that are in close contact with the vestibular bulbs and clitoris and thus play an important role in the female sexual response. These muscles, when voluntarily contracted, can intensify both male and female orgasmic response. Involuntary pelvic floor spasm associated with vaginal penetration, or even speculum examination, is referred to as "vaginismus". This disorder is not an uncommon barrier to sexual intercourse and cause for dyspareunia and other sexual pain disorders. Alternatively, if laxity or hypotonia of pelvic floor musculature occurs due to aging, menopause or neurologic injury, symptoms of vaginal hypoanesthesia, coital anorgasmia, and even urinary incontinence during sexual intercourse may occur. Women with pelvic floor disorders often have coexisting urologic and sexual dysfunction complaints.

## NEUROGENIC MEDIATORS OF FEMALE SEXUAL RESPONSE

Preliminary studies suggest that vasoactive intestinal polypeptide (VIP) and nitric oxide (NO) are the non-adrenergic/non-cholinergic mediators involved in modulating vaginal relaxation and secretory processes. NO has been identified in human clitoris and the putative mediator of clitoral and labial engorgement.[57] These same investigators have identified PDE-5, the enzyme responsible for degradation of cGMP in human clitoral vaginal smooth muscle and vestibular bulb culture. The presence of sildenafil demonstrates enhanced intracellular cGMP synthesis

### TABLE - 4: Neurology-Specific Approach to History and Examination of Patients with Sexual Dysfunction

- Arousal
- Does the patient fail to become aroused under any conditions?
- Does arousal occur under conditions the patient may consider taboo?
- In men: Does erection fail during intercourse, but occur during nocturnal tumescence?
- Coexisting Depression/Anxiety
- What was the patient's premorbid self-image?
- Is there a history of sexual abuse?
- What role does personal or professional stressors play?
- Function- and Pathology-Specific Issues
- Does the patient experience pain associated with sexual performance?
- Are there signs of infection, swelling, or adenopathy of the genital and anal area?
- Is there pain in the lumbar, sacral, or pelvic regions to movement or palpation?
- Are there any signs of sensory pathology: dermatome-specific (can be answered after detailed evaluation of the sacral dermatomes);
  - generalized spinal sensory pathology (can be answered on the basis of evaluation of sensation in the torso for possible existence of a spinal sensory level);
  - "stocking and glove" sensory dysfunction (suggestive of a peripheral neuropathy)
  - “saddle anesthesia"?
- Reflexes and Other Objective Measures
- Is there hyperreflexia in the limbs as may be seen in a central neurologic lesion?
- Is there hyporeflexia as may be seen in a peripheral neuropathy?
- Is there an asymmetry of reflexes?
- Is the Babinski sign present?
- Are there abnormalities in skin moisture or pilomotor responses suggestive of autonomic nerve dysfunction?
  - In men the bulbocavernosus reflex is most indicative of the function of the sensory afferent fibers and motor efferent fibers both transmitted via the pudendal nerve.
  - (The reflex is evoked by a gentle stroke along the glans penis, which produces a contraction of the anal sphincter.)
  - Other reflexes of informative benefit include the anal reflex, a reflexive contraction of the anal sphincter (The reflex is evoked by stimulating the mucocutaneous junction of the anus with a pin); the scrotal reflex, which produces a slow contraction ("pull") of the scrotum (in response to cold stimulation in the region of the scrotum); and the cremasteric reflex which produces a contraction of the cremasteric muscle, elevating the testicle (in response to stroking of the inner thigh.) Since the cremasteric reflex travels via the ilioinguinal and genitofemoral nerves it is indicative of the condition of these nerves as well of the spinal segments L1 and L2.2
  - Perigential Areas and Systems
- Is there an abnormality of anal tone or the force of a voluntary contraction?
- Are there any concurrent abnormalities of bladder function (concurrent incontinence or retention)?
- Evaluation of bladder function may include urodynamic studies to assess for bladder contraction and sphincteric relaxation
- Does the patient have a normal sense of fullness of the bladder?
- Is there adequate emptying on voluntary voiding?

in these cultures and PGE-1 also produced marked increases in intracellular cGMP.[58] Dose-dependent relaxational smooth muscle response to sildenafil further suggests the role for NO as the mediator of clitoral cavernosal and vaginal wall smooth muscle relaxation.

Estrogen plays a significant role in the regulation of female sexual function. Estradiol levels affect peripheral and central nerve transmission. Declining serum estrogen has a resultant atrophy of vaginal wall smooth muscle and vaginal mucosal epithelium. These changes ultimately lead to vaginitis, UTI, incontinence and sexual dysfunction that are associated with aging.[59] Recently, estrogens have also been noted to regulate vaginal and clitoral NO synthase statement, an enzyme responsible for the production of NO. Oophorectomy and aging both result in decreased vaginal and clitoral NO synthase and statement. Importantly, estrogen replacement restores vaginal mucosal integrity and increases vaginal NO synthase statement.60 These findings suggest that drugs like sildenafil may have a potential role in the treatment of FSD that is associates with sexual arousal disorder.

Low testosterone levels in women are also associated with a decline in sexual arousal, genital sensation, libido and orgasm. Presenting symptoms include loss of pubic hair, skin wrinkling and a diminished sense of vitality. Treatment of diminished sexual desire in pre and post-menopausal women has been reported by testosterone replacement therapy,[61] as testosterone also improves libido in women following oophorectomy.[62] Currently there are no FDA-approved testosterone preparations for women, however, studies are under way assessing the potential benefits of testosterone in the treatment of hypoactive sexual desire and other sexual function complaints in women.

## NEUROLOGIC EXAMINATION

As it remains true for all neurologic evaluations, a proper assessment of sexual dysfunction must begin with the history and physical examination. Questions regarding sexual dysfunction must include a social history as to the patient's sexual preference, and the circumstances under which sexual dysfunction occur.

## NEUROLOGIC TESTS

Physiologic studies that may yield further insight into neurologic function may include the pudendal somatosensory evoked potential studies, in which a peripheral stimulation of the pudendal nerve, provokes an ascending response through the pudendal nerve,[4] entering the spinal cord and ascending the spinal cord to the subcortical level. Evaluation of evoked potential response latencies may yield clues as to localization of an area of dysfunction.

Peripheral neuromuscular dysfunction may be further assessed via pudendal nerve conduction studies, in which the pudendal nerve is directly stimulated via an electrical impulse with recording of the stimulated response along the length of the pudendal nerve. This may yield clues as to peripheral pudendal nerve dysfunction.

Further insight may be garnered via the sphincteric EMG, which may assess muscle motor response to sphincteric contraction. The presence of autonomic dysfunction may be suggested by the presence of orthostatic hypotension. Higher cortical causes of sexual dysfunction may be distinguished from autonomic or peripheral nerve dysfunction by assessment of nocturnal penile tumescence.[4]

## NEUROLOGIC LEVELS

### Peripheral Nervous System

Injuries to the peripheral nervous system may primarily affect the somatic nerves or the autonomic nervous system, or both. Lesions may occur in a focal, regional, or diffuse pattern.

Perhaps the most common cause of peripheral neuropathy in the population of the developed countries including USA is diabetes mellitus and it is important to recognize that diabetes

may also interfere with function of the central nervous system, an often overlooked neurologic fact.[3] In adult-onset diabetes mellitus, signs of neuropathy may predate diagnosis of the disorder, and the presenting feature may be sexual dysfunction, most commonly appearing as erectile dysfunction in men. This is most commonly a result of damage to autonomic fibers, but decreased sensation due to somatic nerve dysfunction may also play a role. In both males and females, sympathetic nerve dysfunction may interfere with orgasm or delay attaining it. Erectile dysfunction is seen in 50% of men with diabetes mellitus requiring treatment for over five years.[2]

Another condition that may present with erectile dysfunction as a result of its effect on the peripheral nervous system is amyloidosis, caused by amyloid deposition in the nerves of the autonomic nervous system.[5] Other causes of neuropathy associated with sexual dysfunction include uremia, alcoholism, B12 deficiency, syphilis, and certain inheritable diseases, most notably the Riley-Day syndrome (familial dysautonomia). In autonomic neuropathies, both reflex and psychogenic erections can be affected.[6]

Peripheral causes of erectile dysfunction may also be seen in lesions involving the cauda equina. This may be as a result of a partial cauda equina syndrome due to the mass effect of a herniated disc, tumor, or abscess.

It has been documented that pain arising from any source may interfere with sexual function and proper pain control and pain management is a necessity. While not necessarily associated with neurologic dysfunction, pain associated with lumbosacral root stretching or compression may impair the psychogenic contribution to sexual arousal or at least limit participation in coitus.[7]

Another possible cause of sexual dysfunction is the intrathecal opioid therapy for chronic pain. According to a recent ground-breaking study ((Roger Abs et al. J Clin Endocrinol Metab 85: 2215-2222, 2000)), patients receiving intrathecal opioids, are at high risk of development of hypogonadotropic hypogonadism. In addition, about 15 percent of these patients develop central hypocorticism, and another 15 precent develop GH deficiency. In view of these findings, patients on intrathecal opioids may require systematic endocrine work-up and potentially substitutive therapy with sex hormones.

## Spinal Cord

The improved life expectancy of patients with spinal bifida have allowed these patients to survive into sexual maturity. Sexual dysfunction has become a recognized association with this inherited disorder of neural tube formation and often requires extensive measures that lead to mediocre management and prognosis in regard of sexual function.

In erectile dysfunction secondary to spinal cord damage, the extent of dysfunction, as well as corresponding symptoms, depend upon the level of the injury in the cord, as well as the degree of the injury.[10] A complete cord transection may yield erectile dysfunction, bowel and bladder dysfunction, paraplegia, and a sensory level. A partial lesion may yield varying degrees of these findings, or may present as primarily erectile dysfunction. Spinal cord injury may be acute as in trauma or subacute due to external compression by a mass lesion. This may produce a complete or a partial functional transection of the cord. Lesions intrinsic to the cord may be due to ischemia, which may be produced by thrombotic or embolic vascular occlusion, hypotension, hemorrhage, or surgical disruption of vascular flow. Notably, while erectile dysfunction and bladder dysfunction are common in severe spinal trauma, they are infrequent in myelopathy due to cervical stenosis.

Other conditions that may disrupt ascending or descending spinal pathways include myelopathies, of which multiple sclerosis is a common example. Recent studies suggest that the percentage of multiple sclerosis patients

with some degree of sexual dysfunction may be as high as 70%.[11] Pudendal somatosensory evoked potentials are often abnormal in erectile dysfunction due to multiple sclerosis.[12] Spinal cord myelitis leading to sexual dysfunction may also be seen in a variety of viral infections including HIV myelopathy and HTLV I and II induced myelopathies.[13]

## Intracranial

### STROKE

The appearance of erectile dysfunction following a stroke is not uncommon, however, it is not clear whether the erectile dysfunction is primarily due to the ischemic insult or secondary to other factors, such as physical disability incurred in the stroke which interfere with participation in coitus; or depression due to altered self-image associated with loss of independence. Studies have also suggested that lesions of the left hemisphere may lead to increased severity of sexual dysfunction due to post-stroke depression. Another possible contributor is loss of socialization and/or communication and cognitive changes that follow language dysfunction. While motor dysfunction as a result of a hemiparesis syndrome may interfere with performance, there has also been noted decreased arousal, libido, and sexual satisfaction in hemisensory syndromes.[15, 16]

### Epilepsy

The development of sexual dysfunction is a well-recognized consequence of epilepsy, occurring in over 60% of patients.[17] The etiology appears to be multifactorial with contributors of neurologic, endocrine, social, psychological, and medication origin.[18, 19, 20] Decreased free testosterone has been observed in anticonvulsants that induce hepatic enzymes, with improved sexual functioning reported in patients taking lamotrigine, which does not.[21] The appearance of sexual dysfunction in female epileptic patients appears to be associated with hormonal dysfunction at the hypothalamic, pituitary, and gonadal levels. Arousal and vaginal lubrication are impaired.[19]

Independent of medications, there are observed differences in seizure types and foci regarding sexual activity. Temporal lobe foci are more commonly associated with decreased sexual activity. The decrease in seizure frequency associated with temporal lobectomy is also associated with an improved sexual activity. There are reports suggesting that patients with complex partial seizures are more prone to sexual dysfunction than are other seizure types.[2]

### Traumatic Brain Injury

The appearance of sexual dysfunction following a traumatic brain injury appears to be much greater than previously recognized. This has been associated with alterations in libido and personality. Depression associated with the postconcussive affects sexual interest and/or erectile function. Men with recurrent head trauma have exhibited higher incidences of erectile dysfunction.[22,23]

## Multilevel

Neurologic syndromes associated with erectile dysfunction often appear to affect the neural axis at multiple levels. Parkinson's disease and the other parkinsonian syndromes such as multiple system atrophy frequently exhibit erectile dysfunction. This may be due to impaired motoric function, a loss of sexual interest associated with depression, or autonomic dysfunction. Approximately 80% of Parkinson's patients show substantial reduction in sexual activity after several years of therapy.[24, 25] Similarly, disorders mentioned previously appear to have effects in erectile dysfunction in both central and peripheral locations. These include tabes dorsalis, B12 deficiency, HIV, and alcoholism.

As has been mentioned previously, depression associated with neurologic conditions may produce erectile dysfunction. Furthermore, treatment of this disorder may lead to functional improvement or iatrogenic erectile dysfunction.

Although a detailed discussion of iatrogenic sexual dysfunction is beyond the scope of this article, several suggestions appear to be the consensus of the practitioners. The patient with iatrogenic sexual dysfunction may be initially advised to wait four to six weeks for sexual side effects to resolve. In case of persistent dysfunction, a decrease of dosage, alteration of the timing of daily dose or introduction of a two-day drug "holiday" (especially recommended to patients on sertraline and paroxetine). In some cases, a sexual function antidote may be coadministered antidote. (See Table - 5); while in other cases substitution of another medication (e.g., nefazodone or bupropion instead of sertraline, paroxetine or fluoxetine).

## TABLE - 5 Published antidotes useful in management of iatrogenic sexual dysfunction induced by antidepressants

| Drug | Symptom | PRN Dosage | Daily dosage |
|---|---|---|---|
| Amantadine | Anorgasmia<br>Decreased libido<br>Erectile dysfunction | 100-400 mg<br>(for two days<br>prior to coitus) | 75-100 mg bid<br>or tid |
| Bupropion | Anorgasmia | 75-150 mg | 75 mg bid<br>or tid |
| Buspirone | Anorgasmia<br>Decreased libido<br>Erectile dysfunction | 15-60 mg | 5-15 mg bid |
| Cyproheptadine | Anorgasmia<br>Decreased libido<br>Erectile dysfunction | 4-12 mg | None published |
| Dextro-amphetamine | Anorgasmia | 5-20 mg | 2.5-5 mg bid<br>or tid |
| Pemoline | Anorgasmia | | 18.75 mg |
| Yohimbine | Anorgasmia<br>Decreased libido<br>Erectile dysfunction | 5.4-10.8 mg | 5.4 mg tid |

Adapted from Labbate et al, 1998; Shrivastava et al, 1995; Ashton, Rosen, 1998; Bartlik, Kaplan, Kaplan, 1995; Gitlin,1995

## CONCLUSION

Sexual dysfunction is commonly associated with a wide variety of disorders and is very commonly observed in neurologic patients. In assessment of sexual dysfunction, a systems approach must be employed to determine the etiology of sexual dysfunction, and the possible involvement of the nervous system. A thorough examination, an understanding of associated signs and syndromes, may help elucidate causation, and at which level in the nervous system a lesion may occur. These diagnostic findings play a crucial role in assessment of disability due to of sexual dysfunction and in management of the patient.

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## CME QUESTIONS FOR SEXUALITY AND DISABILITY

1. Acute onset sexual dysfunction in a 36-year-old recently divorced professional male suggests that upon further investigation and laboratory testing, the patient will exhibit:

   a. markedly reduced levels of testosterone and a successful intracavernous injection test
   b. normal levels of testosterone and a successful intracavernous injection test
   c. moderately reduced levels of gonadotropins and an unsuccessful intracavernous injection test
   d. normal levels of gonadotropins and unsuccessful intracavernous injection test

2. A 45-year-old female married for 20 years (2/2 pregnancies) complains of dyspareunia, loss of libido, and inability to achieve orgasm for several years which have prevented her from engaging in any kind of sexual activity for the last year. Upon examination, no signs of estrogen-deficient vaginitis or difficulty lubricating after vibratory sexual stimulation are observed. What course of action can be undertaken in this case?

   a. refer to psychological and/or sexual counseling
   b. order vaginal Doppler ultrasound and vaginal pH measurement
   c. consider low-dose estrogen/progestin replacement therapy
   d. all of the above

3. Which reflectory response in males is judged to be the most informative of the function of both the sensory afferent and the motor efferent fibers of the pudendal nerve?

   a. the anal reflex
   b. the cremasteric refle
   c. the Babinski sign
   d. the bulbocavernous reflex
   e. both b and c
   f. none of the above

4. Administration of intrathecal opioids for chronic back pain may lead to which endocrinological sequellae?

   a. hypergonadotropic syndrome
   b. alterations in ACTH/cortisol circadian rhythms
   c. central hypocorticism
   d. all of the above

5. Iatrogenic sexual dysfunction due to the adverse effects of antidepressants of the selective serotonin reuptake inhibitor variety (e.g. sertraline, paroxetine) may be addressed by which of the following management approaches?

   a. administration of dopamine agonists (e.g. levodopa-carbidopa)
   b. introduction of a two-day break in medication ("drug holiday")
   c. graduate increase of dosage of the antidepressant over four to six weeks
   d. all of the above
   e. none of the above


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# Book Review

## Writing and Defending Your Expert Report: The Step-by-Step Guide with Models

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**SEAK, Inc. Falmouth 2002**

**Reviewed by:**
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In the hills of our southern Appalachia it is a common saying " Opinions are like noses, every one has it and we all blow it often". However, an expert opinion on the on the other hand is based on special knowledge, skills and ability of expert which the adjudicators of a legal dispute and courts regularly rely on if found credible, usually after testing it in the crucible of cross examination. It is also known that an expert opinion is only as strong as One's expert report. Opposing attorney can and will use every tactic, fair and unfair, to turn expert's report against the expert. A well-written report is often the first and best line of defense from such attacks. Equally important is expert's ability to recognize Opposing attorney's tactics and neutralize them. We have, unfortunately, found far too many poorly written expert reports. These reports needlessly open up the expert to devastating cross-examination.

*Writing and Defending Your Expert Report: The Step-by-Step Guide with Models* is an excellent overview of the art of crafting the best possible expert report and expertly defending it in the crucible of cross examination. The book is ideally suited for the physician whose experience as an Independent Medical Examiner ranges from the beginner through the intermediate level and there are some pearls for so called experts as well.

The authors, Steven Babitsky and James J. Mangraviti, Jr., will be familiar to many readers of Disability Medicine through the texts, seminars, audiotape programs, videotapes and directories offered through their company, SEAK Inc. These products are geared toward non-attorney professionals, particularly physicians, who wish to be more effective expert witnesses and, in the case of this text, write high-quality, defensible expert reports.

*Writing and Defending Your Expert Report* is a to the point, easy-to-use reference text. Key elements in writing the expert report are illustrated by specific examples, which eliminate any doubt in the reader's mind as to what the authors are driving at. The introductory chapter sets the tone of the work by summarizing the salient points of the entire book. The additional 15 chapters include critically important topics such as: HOW TO BEST EXPRESS AND DOCUMENT DETAILED AND SPECIFIC FACTUAL ASSUMPTIONS; THE IMPORTANCE OF STAYING WITHIN ONE'S TRUE AREA OF EXPERTISE; STATING OPINIONS AND CONCLUSIONS IN A DEFENSIBLE MANNER; and MAKING YOUR REPORT POWERFUL, PERSUASIVE, AND UNDERSTANDABLE. There are chapters devoted to practical, but essential nuts-and-bolts matters such as FORMATTING; PROPERLY DISCLOSING PRECISE DOCUMENTS REVIEWED; HOW TO USE CITATIONS TO TEXTS, GUIDELINES, CODES, ARTICLES, AND OTHER AUTHORITY TO BOLSTER A REPORT'S CREDIBILITY; and PROOFREADING FOR MISTAKES.

On a personal level, I found the chapters on DAMAGING SUPERFLUOUS LANGUAGE AND INFORMATION THAT SHOULD NOT BE INCLUDED IN EXPERT REPORTS and RED-FLAG WORDS TO AVOID of particular interest because-Gulp! -As I read the chapters I realized that I routinely use some of the very language and red-flag words the authors properly recommend avoiding.

Chapter 16, DEFEATING COUNSEL'S TACTICS, alone makes this book worth the price. The authors give examples of the 40 most frequently used tactics experts can expect counsel to use when cross-examining them about their reports. The authors stress that the reader not try to memorize the tactics and replies but rather study the "manner in which the replies are formulated to develop a style to handle the tactics one is likely to face. Experts can answer even the most difficult questions truthfully and, at the same time, artfully." It is a beautiful thing . . .

A great strength of this book is Appendix B, MODEL REPORTS. In this section the authors have included 12 high-quality, well-written expert reports remarkable for their style, content and formatting; report #8, a model Independent Medical Evaluation, will be of particular interest.

Criticisms? Only one, actually, and it is minor. Appendix A: ADVICE FROM THE TRENCHES contains additional recommendations from experts and attorneys from across the country regarding the drafting of reports. While well intentioned, the inclusion of this "guest contributor" commentary does not add to or strengthen the work.

*Writing and Defending your Expert Report* is authoritative yet easy-in fact, pleasurable-to read and understand. If you want to improve your ability to write high-quality, defensible expert reports-this is the book to buy!

# CME Question / Answer file

## The following questions are based on the Articles from Disability medicine, vol 2, #2

***The correct answers will appear in the next issue of Disability Medicine***

*Original Research Article, Classifying Fibromyalgia: Taxonomic Lessons from the Icelandic Disability Registry*

1. In the ninth revision of the International Classification of Diseases (ICD 9) fibromyalgia
   A. is classifed under Diseases of the musculoskeletal system and connective tissue
   B is classifed under Mental disorders
   C. is classifed under Diseases of the nervous system and sense organs
   D. has no specific code

2. In this study a single diagnosis registered as basis for disability claim for women with the Fibromyalgia Syndrome
   A. was considerably more common than for women without the Fibromyalgia Syndrome
   B. was considerably more common than for women with schizophrenia
   C. was considerably less common than for women with multiple sclerosis
   D. was considerably less common than for women with anxiety or depression

3. A comparison of diagnoses by disease category registered as basis for disability claim for the study group (women with the Fibromyalgia syndrome) and the comparison group (women without the Fibromyalgia syndrome) revealed that

   A. diseases of the nervous system were statistically significantly more common in the study group than in the comparison group
   B. endocrine, nutritional and metabolic diseases were statistically significantly more common in the study group than in the comparison group
   C. mental disorders were statistically significantly more common in the study group than in the comparison group
   D. rheumatological disorders were statistically significantly more common in the study group than in the comparison group

4. The results of this study lend support to categorization of the Fibromyalgia Syndrome under

   A. internal medicine
   B. neurology
   C. psychiatry
   D. rheumatology

*Facial Pain, An Overview of Evaluation*

1. Which lab tests are recommended in a patient presenting with facial pain in order to screen for giant cell (temporal) arteritis:

   A. erythrocyte sedimentation rate (ESR)
   B. anti-nuclear antibodies (ANA)
   C. anticholinesterase (ACE) antibody
   D. C-reactive factor
   E. All of the above

2. The pathogenesis of secondary trigeminal neuralgia is best described as consisting of these factors, EXCEPT:

   A. demyalination of sensory fibers in the trigeminal nerve
   B. demyelination lesions in the brain stem
   C. impaction of the trigeminal nerve by the lateral cerebellar artery
   D. ectopic discharges in the nerve due to compression
   E. hyperexitation of the afferent neurons leading to synchronization of the after-discharge activity.

3. A 42-year-old woman presents with severe bilateral facial pain. History taking is difficult due to emotional response and the severity of pain prevents examination. Which of the diagnoses is least likely based on the evidence:

   A. sinusitis
   B. trigeminal neuralgia
   C. atypical facial pain
   D. temporomandibular joint disorder
   E. temporal arteritis

4. A 45-year-old male presents with facial pain felt in the medial orbital and glabellar areas that intensifies upon examination of the middle nasal turbinate. Which surgical procedure may be recommended upon confirmation of diagnosis of sphenopalatine neuralgia:

   A. TMJ arthroscopic surgery
   B. correction of the nasal septum
   C. frontal sinus drainage
   D. gamma knife ablation of the trigeminal ganglia
   E. endoscopic sinus surgery

5. Causes of impairment in patients with facial pain may include all of the following EXCEPT:

A. improper diet due to oral intake limitations
B. cosmetic deformities
C. mechanical allodynia
D. dysarthria
E. limited motor function in the trigeminal distribution

***NEUROPSYCHOLOGICAL ASSESSMENT***

1. The current gold standard for evaluation and confirmation of persistent cognitive impairment is:

A) An MRI of the brain with documented areas of encephalomalacia
B) An EEG with spectral analysis indicating slowing of the background
C) An abnormal PET scan or SPECT scan
D) Neuropsychological testing

2. Mechanical acceleration-deceleration forces generated at the time of whiplash injuries are maximal in the:

A) Occipital and frontal lobes
B) Frontal and temporal lobes
C) Parietal and occipital lobes
D) Evenly distributed throughout the brain

3. Patients commonly complain of memory disturbances with:

A) Depression
B) Anxiety disorders
C) Mild traumatic brain injuries
D) All of the above

4. Mild traumatic brain injury associated with loss of consciousness for less than 10 minutes has:

A) A poor prognosis for complete recovery
B) An excellent prognosis for complete recovery in 3-12 months
C) Been associated with progressive cognitive decline based on the formation of parenchymal scar tissue
D) Not been associated with any permanent injuries

***Following questions are based on current state of the art review of scientific literature related to Disability Medicine***

1. Approximatley what percent of randomly selected individuals will experience carpal tunnel syndrome as a part of daily living?

a. 5%
b. 10%
c. 15%
d. 20%

2. Among working age people what percent have low back pain each year?

a. 25%
b. 50%
c. 75%
d. 80%

3. Average lost days from work are highest for which of these conditions?

a. skeletal fractures
b. amputations
c. carpal tunnel syndrome
d. conditions a, b, and c result in approximately the same number of days

4. Approximate number of low back workers compensation claims annually?

a. 1 million
b. 2 million
c. 3 million
d. 5 million

5. The most common cause of disability is

A. mental problems
B. social problems
C. chronic pain
D. symptom magnification

6. Which of the following is a widely used health status measure that has been shown to reliably assess the impact of health problems on function and quality of life?

A. Pain Disability Index
B. SF-36
C. Sickness Impact Profile
D. Oswestry

7. The Luria Nebraska tests are used for

A. evaluation of schizophrenia
B. evaluation of intelligence
C. traumatic brain injuries
D. evaluation of chronic pain

*Continued on page 68*

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## 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 360º (displays +180º to -180º)
- 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.

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| | |
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| **Orlando, FL** | **January 31** |
| **Orlando, FL** | **February 1, 2** |
| **Orlando, FL** | **February 3 (*Advance Course*)** |
| **Sydney, Australia** | **February 15, 16** |
| **Sydney, Australia** | **February 17 (*Advance Course*)** |
| **Chicago, Illinois** | **May 3, 4** |
| **Chicago, Illinois** | **July 11** |
| **Chicago, Illinois** | **July 12, 13** |
| **Chicago, Illinois** | **July 14 (*Case Managers Workshop*)** |
| **Chicago, Illinois** | **October 17** |
| **Chicago, Illinois** | **October 19, 20** |
| **Chicago, Illinois** | **October 20 (*Advance Course*)** |

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