

GBS | CIDP

Foundation International

Summer 2012

The Communicator

Providing Strength Through Support

HIGHLIGHTS

- CIDP: An Update
- Immune-Mediated Small Fiber Neuropathy: A Treatable Condition That Can Mimic GBS or CIDP
- How Can Outcome and Disease Activity Be Measured in CIDP?
- What is MMN

Volunteer Opportunities

Do you have a passion to help individuals affected by GBS & CIDP?

If so, there are ways in which you can assist the Foundation. We are seeking out people interested in the following ways.

- Liaisons
- Points of Contact
- Board Committees:
 - Communications/ Public Relations/Web
 - Development
 - Finance
 - Advocacy
- Symposium Volunteers

If interested please contact us at info@gbs-cidp.org.



CONTACT US

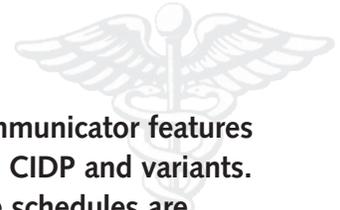
International Office
The Holly Building
104½ Forrest Avenue
Narberth, PA 19072

1.866.224.3301
1.610.667.0131

Fax: 1.610.667.7036

www.gbs-cidp.org
email: info@gbs-cidp.org

MEDICAL ISSUE



This annual Special Medical Issue of the GBS/CIDP Communicator features articles and comments from experts in the field of GBS, CIDP and variants.

We thank all the contributors whose schedules are demanding but nevertheless considered the needs of our readership in bringing us the latest information on these conditions.

We suggest that these newsletter issues be saved. Make them part of a reference library to serve as a ready resource for you or your physician. Additional copies are available upon request.



SAVE THE DATE
October 26 - 28, 2012
Fort Worth, Texas
 ★ ★ ★
 Plan to attend the
12TH INTERNATIONAL
GBS/CIDP SYMPOSIUM!



- ★ 23 Workshops
- ★ Meet world-famous neurologists
- ★ Welcome Reception
- ★ State Night Texas Barbeque
- ★ Learn About New Research
- ★ Symposium Walk-a-thon
- ★ Hospitality Room
- ★ Many opportunities to meet others and share experiences

Brochures will be mailed in the summer.
Watch for yours!

We take this opportunity to thank **CSL Behring** for their support in making this newsletter possible through an unrestricted educational grant.

Printed on recycled paper.

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What is MMN?

*Carol Lee Koski, MD**Medical Director, GBS/CIDP Foundation International*

MMN is an abbreviation for Multifocal Motor Neuropathy, a rare pure motor slowly progressive neuropathy reflecting focal damage to nerves that is primarily distal in the arms in two thirds of patients and in the distal legs in half the patients. Males from ages 22 to 66 years are 2.7 times more frequently effected than females. Only 0.6 individuals/100,000 population are involved at any one time. It is one of the least common of the inflammatory neuropathies. Since the process is focal, it involves some nerves more than others; it typically involves one arm or leg more than that on the other side of the body. Patients may note weakness or fatigue in muscles resulting in difficulty turning a key in a lock, dropping things out of their hand, not being able to retain a thong sandal while walking, or having a foot drop. Facial, swallowing and breathing muscles are not involved. Deep tendon reflexes are decreased or absent in the involved extremities while sensory functions (i.e. pain, light touch) are normal. Patients do not die from the condition but do experience significant disability over the chronic course of this condition. Other conditions such as primary motor neuron disease (Lou Gehrig's Disease) and inflammation of blood vessels or vasculitis can some times look like MMN and lead to a delayed diagnosis and treatment resulting in axonal damage and disability. In a recent study in the Netherlands diagnosis was on average delayed by five years in MMN patients.

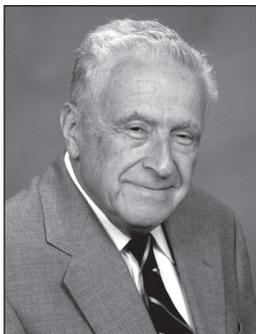
The cause of MMN is not fully understood. It is proposed that an immune system targets specialized areas of the motor axon or fiber. The axon extends from motor nerve cells located in the spinal cord out to muscle fibers. The specialized areas of the axon are rich in sodium channels that allow electrical impulses to travel rapidly down the motor axon and stimulate the muscles to contract or shorten leading to movement in the arms or legs. Damage to the axon causes focal muscle wasting and weakness. Antibodies

to the lipid ganglioside GM1 occur in 60-80% of patients and are higher in some patients with more severe weakness. Diagnosis of MMN requires recognition of the clinical signs discussed above and a well done series of nerve conduction studies that demonstrate focal block of the electrical impulses in motor but not sensory nerves at other than entrapment sites such as those at the wrist associated with Carpal Tunnel syndrome or at the elbow with ulnar nerve compression.

Treatment options for MMN are limited. In contrast to other inflammatory neuropathies, patients with MMN do not respond to corticosteroids and plasma exchange and may worsen with these treatments. Cytotoxic cancer therapy drugs such as cyclophosphamide can be effective but use over the long term is restricted by toxicity and potentially lethal side effects. High dose intravenous immune globulin or IVIG is generally safe and effective as demonstrated by a series of now five randomized, double blind and placebo controlled cross-over trials in MMN. The last of these trials involved 44 MMN patients from North America and Denmark and was completed in 2012. It demonstrated not only significant improvement in muscle strength but also in functional disability. The trial has been submitted to the FDA to support an indication for IVIG use in MMN patients which is currently off label. Delaying treatment with IGIV can result in irreversible physical impairment and supports the need for early diagnosis and treatment. IGIV maintenance treatment can be successfully used over years. However, over time and despite a regular IGIV treatment, a mild and slow decline in function can occur and be associated with signs of more widespread disease. This progression can be limited by increasing the IGIV dose or dose frequency. The median dose of IGIV gradually increases over years and may become as high as 1.6 grams/Kg per week. Early diagnosis and treatment will limit progression and disability in this chronic neuropathy.

Disclaimer Information Questions presented in the GBS/CIDP Newsletter are intended for general educational purposes only, and should not be construed as advising on diagnosis or treatment of the Guillain-Barré Syndrome or any other medical condition.

Privacy Policy In response to many queries: Intrusive practices are not used by the GBS/CIDP Foundation International. It does NOT sell its mailing list nor does it make available telephone numbers! The liaisons are listed in the chapter directory with their permission. Our CIDP and Miller-Fisher Groups share names only after a signed permission slip is received. We are proud that none of our members has ever been solicited or sent materials other than those concerning GBS. We respect your privacy.



It is with deep regret that the GBS/CIDP Foundation International announces that Robert (Bob) Benson, the inspiration for our Foundation, died on May 17th from cancer.

He was a graduate of Penn State University and received an MBA from University of Maryland.

In 1979 after having a bad cold and a touch of pneumonia, he came down with Guillain-Barré Syndrome. At that time there was no information, no one to turn to. While in Intensive Care for many weeks, he and his wife, Estelle, vowed that if and when he recovered they would start a support group to insure that no one would ever be alone with GBS again. A year later they kept their promise and with eight people in their home around a dining room table the organization began. The rest is history.

Over the years, it has grown to an international organization of over 30,000 people in 33 countries. Bob helped found the Board of Directors for the Foundation, and also served as Board President.



At his family's request, contributions in his memory may be made to:

THE ROBERT AND ESTELLE BENSON FELLOWSHIP IN NEUROMUSCULAR NEUROLOGY,
c/o GBS/CIDP FOUNDATION INTERNATIONAL, 104 1/2 FORREST AVE., NARBERTH, PA 19072
or please call 610-667-0131.

CIDP: An Update

David R. Cornblath, MD

Member, GBS/CIDP Foundation International Medical Advisory Board and Board of Directors

As many of you have hopefully heard, the GBS-CIDP FI has sponsored a series of talks around the country entitled, "CIDP, an Update." Supported by two anonymous donors, this program sponsors a member of the Medical Advisory Board to give a talk to people with CIDP and their supporters. So far, programs have been held in Baltimore, St. Louis, Chicago, Boston, and Philadelphia. Held on a Saturday, the purpose of the talks is several-fold.

First, members of the MAB believe that diagnosis of CIDP can be improved in the US. Based on experience from the Centers of Excellence program, there are a number of people who carry a diagnosis of CIDP but in fact have another diagnosis. Thus the talk starts with What is CIDP, How do doctors diagnose CIDP, and What looks like CIDP but is not. We ask those with CIDP to compare themselves to standard diagnostic criteria.

Second, people with CIDP should get the right treatment. There is a substantial body of medical evidence on "best" treatment for CIDP which is reviewed. Many people are being treated but possibly not optimally. People with CIDP should not be getting IVIg or other treatments if it does not help them.

Third, those with CIDP should always try to get off treatment. This initially sounds crazy, but the word "chronic" in the name CIDP refers to the onset of the disease not the fact that one has this forever. There is no reason to think you cannot be cured and eventually off all treatment. Strategies for this are discussed.

Fourth, those with CIDP plus another linked disease need additional thought especially if the other disease involves unusual blood proteins, which everyone with CIDP should be checked for. These tests are simple blood and urine tests and in some cases simple skeletal x-rays. Knowing whether or not you have a "paraprotein" can make a large difference in your treatment.

Finally, we ask those attending the meeting to consider Advocacy and Donations. The amount of research spending on neuropathy in general and CIDP in particular is very small in relation to the number of people affected. On both these fronts you will be hearing more from the Foundation.

We plan more of these talks and will eventually place the talk on the internet. In the meantime, if your local chapter would like such a talk, please contact info@gbs-cidp.org.

How Can Outcome and Disease Activity Be Measured in CIDP?

Kenneth C. Gorson, Professor of Neurology

St. Elizabeth's Medical Center, Tufts University School of Medicine, Boston MA

Member, Medical Advisory Board, GBS/CIDP Foundation International

What is the best way to define the prognosis of chronic inflammatory demyelinating polyneuropathy (CIDP)? How do treating clinicians determine when CIDP is active or in remission? These are some of the critical issues that neuromuscular physicians routinely encounter when evaluating patients with CIDP. Defining long-term treatment outcomes in patients CIDP is complicated by varying definitions of treatment “response” and differing outcome scales measuring impairment (neurological findings on examination), functional problems at the disability and social levels, or impact on quality of life. Some examples of disability assessments previously used in CIDP patients include the modified Rankin disability score, a gross measure of disability dependent mostly upon ambulation which has been frequently applied in older retrospective studies, perhaps due to its ease of application, but it may be relatively insensitive to small but clinically meaningful functional improvement. The Hughes scale (designed by our colleague Professor Richard Hughes) was used in one long-term retrospective study of CIDP, but this scale was originally intended to be applied to patients with Guillain-Barré syndrome and does not reflect upper extremity function. The Overall Disability Sum Score and the Overall Neuropathy Limitations Scale derived from it are validated disability scales and are probably the best measure of disability, but they have not been used to assess long-term outcome in CIDP. Furthermore, most clinical trials using these excellent scales were short-term, assessing the efficacy of various therapies with follow-up between 6 to 12 weeks. Recent studies have demonstrated efficacy from IVIg and high dose oral dexamethasone after 6-month and 1-year follow-up, but data on long-term outcome (e.g., 5 or more years) are limited. In such cases a uniform grading system assessing disease activity in relation to treatment status would complement disability and impairment scales and is especially desirable. Such a grading system in CIDP patients would promote better classification of outcome after many years, on or off therapy, and perhaps encourage better patient selection for those with active disease for new treatment trials. Accordingly, a new scale, the CIDP Disease Activity Status (CDAS) was designed specifically to examine the reproducibility of a grading system to assess disease activity status relative to treatment status which can be used in clinical practice and research studies, and to classify long-term outcome by applying the CDAS classification scheme to a well-defined cohort of CIDP patients.

The CDAS was created by an expert panel from the Guillain-Barré/CIDP Foundation International Medical Advisory Board using techniques that have been effective for classifying disease status for patients with myasthenia

gravis and other medical conditions. Points of discussion among the investigators included: 1) can a classification system be developed that allows for a variable disease course and treatment response, and a variable duration of follow-up, and yet be simple and easy to apply; 2) does the concept of “cure” apply to patients with CIDP; 3) how might remission be defined, allowing for different evaluators in a community or academic environment; 4) how should those patients with persistent but stable long-term deficits be classified; and, 5) how should stable patients requiring periodic treatment be classified. The following variables were considered relevant for the design of CDAS: 1) treatment status at the time of classification by the clinician (on or off therapy); 2) duration of treatment, 3) duration of follow-up (none to > 5 years); 4) neurological examination at last follow-up, defined as normal or abnormal; and 5) treatment responsive or not, as defined by the judgment of the treating physician.

The CDAS specifically was constructed for easy application by practicing clinicians and researchers using broad categories of patient disease status, such as “cured”, “remission”, “stable active disease”, “improving”, or “unstable active disease”. These disease status categories are to be determined by the treating clinician. Patients are classified as cured if they had a stable neurological examination (subcategories of either normal or abnormal) and were off all treatment for 5 or more years. If patients are stable off treatment and the duration of follow-up off treatment was less than 5 years, they are classified as in remission (with a stable normal or abnormal examination). Patients are considered to have stable active disease if they require ongoing immunotherapy to maintain clinical stability for a year or more; in contrast, those who recently initiated therapy (for 3 months or more, but less than 1 year) and are responding to treatment are classified as improving. All other patients (treatment naïve, treatment for less than 3 months, or those with a progressive or relapsing-progressive course despite immune therapy of any duration) are categorized as unstable active disease (Table).

Once the classification system was developed and consensus was reached among panel participants, the CDAS was applied to the case descriptions of a well defined cohort of 106 patients with idiopathic CIDP who satisfied a rigorous case definition of CIDP. These case descriptions arose from an earlier study to identify diagnostic criteria for CIDP, led by Carol Lee Koski, M.D. To assess the reproducibility of the CDAS, 60 of these cases were classified using the CDAS independently by 3 investigators who were blinded to the grading results of the others.

We found there was excellent agreement regarding

classifying these CIDP cases according to the CDAS among any 2 of the 3 raters, and ranged between 87 and 90 percent; this indicates that the scale is highly reproducible among different raters (i.e., different clinicians frequently classified the same cases into the same CDAS categories). Eleven percent of the patients were classified as cured, 20 percent were considered in remission, 44 percent had stable active disease on therapy, and 7 percent were improving. For those classified as cured, the average followup was 7.4 years (median, 8 years; range 5 - 12 years). Overall, 82 percent of treated patients had long-term stability off treatment, or they were stable or improving on treatment. In contrast, 18 percent were either untreated at the time of classification or refractory to previously administered immune therapy (Table).

Based upon the above data analysis, we have concluded the following:

1. The CIDP Disease Activity Status (CDAS) is a simple and reliable scoring system that potentially could be applied successfully both in clinical practice and research studies. The CDAS is intuitive as patients move easily from one category to another during follow-up and reassessment based upon: 1) treatment status; 2) duration on or off therapy; 3) stability of the neurological examination; and 4) response to treatment, as judged by the treating clinician. The CDAS also has high inter-rater reliability when applied by 3 independent and blinded investigators.

2. We applied a variation of the definition of cure that has been used as an outcome measure in cancer patients,

disease-free survival for 5 or more years off treatment. Using this paradigm, we defined cure in CIDP as stable disease off treatment for 5 or more years, and observed that this occurred in 11 percent of our cohort; all had received immune therapy, and many had chronic static neurological deficits. This observation suggests that in a minority of patients, CIDP is a curable condition. However, immune disorders theoretically could relapse several years after a period of stability, and the notion that the cancer definition of cure may not necessarily apply to patients with CIDP should be considered. Nonetheless, half of our CIDP cases who were considered cured were followed for at least 8 years and thus likely allows a sufficient time for observation for a relapse.

3. We found that an additional 20 percent of our patients were considered in remission and off therapy for less than 5 years (CDAS 2A and 2B). Fourteen percent of the cases had fixed neurological deficits (for example, foot drop, hand weakness with atrophy), yet were considered cured or in long-term remission by the CDAS classification (CDAS 1B and 2B). This is most likely due to stable and irreversible axon loss which persists in those no longer receiving treatment and does not change with long-term follow-up. If this finding extends to similar CIDP patients in general practice, it suggests that CDAS 1B and 2B patients actually may be over-treated; these long-term observations suggest that further treatment may not be indicated in the setting of chronic stable deficits, and the potential side effects, cost and patient inconvenience could be avoided.

4. Just over half of the patients in this study (51%)

continued on page 6

Table. CIDP Disease Activity Status (CDAS) of Patients with a Consensus Diagnosis of Idiopathic CIDP

The CDAS is based upon clinical assessment by the treating physician:

	Patients (n= 106)	
1. Cure: ≥ 5 years Off Treatment		11% Cure
A. Normal Examination	6 (6%)	
B. Abnormal Examination, Stable/Improving	5 (5%)	
2. Remission: < 5 years Off Treatment		20% Remission
A. Normal Examination	12 (11%)	
B. Abnormal Examination, Stable/Improving	10 (9%)	
3. Stable Active Disease: ≥ 1 year, On Treatment		44% Stable Disease
Normal Examination	18 (17%)	
4. Improvement: ≥ 3 months < 1 year, On Treatment		7% Improving
A. Normal Examination	0 (0%)	
B. Abnormal Examination, Stable/Improving	8 (7%)	
5. Unstable Active Disease: Abnormal examination with progressive or relapsing course*		18% Active Disease
A. Treatment Naïve or < 3 months	6 (6%)	
B. Off Treatment	7 (7%)	
C. On Treatment	5 (5%)	

*5B and 5C refer to patients who were treatment refractory from prior therapy or worsening despite ongoing therapy.

Three Research Grants Awarded!

In November 2011, 11 Letters of Intent were received for possible Research Grants by the GBS/CIDP Foundation International. Of that number, 3 were selected after careful review:

Tregitopes: A Novel Immunomodulatory Therapy for CIDP

Leslie Cousens, PhD
Director of Protein Therapeutics
EpiVax, Inc., Providence, RI



Comparing Sialylated IgG and Fc Fragments with IVIG in an Anti-ganglioside Antibody Induced Neuropathy Model of GBS

Cynthia A. Massaad, PhD
Research Scientist,
Department of Neurology
The University of Texas
Health Science Center, Houston, TX



CD4+CD25+ Regulatory T cells as Potential Biomarkers of Pathogenesis and Response to Therapy in CIDP Patients

Ericka P. Simpson, MD
Associate Professor,
Neurology Residence Program Director
Methodist Neurological Institute,
Houston, TX



How Can Outcome and Disease Activity Be Measured in CIDP?

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required continued treatment (CDAS 3 or 4), usually IVIg, plasma exchange, corticosteroids or various corticosteroid-sparing agents, and therefore were classified as “Stable Active Disease” or “Improving”, based upon the duration of therapy, because in the view of the treating physician treatment arrested further progression or improved impairment, functional disability, or quality of life. CDAS 3 and CDAS 4 categories included those with persistent neurological deficits or treatment related fluctuations. It is noteworthy that almost one-third of patients had a normal neurological examination at last follow-up (CDAS 1A, 2A, and 3A) regardless of treatment status at the time of case review.

5. We found that 18 percent of our cohort were classified as CDAS 5, “Unstable Active Disease”, yet 6 percent were treatment naïve at the time of case review (CDAS 5A) and thus probably would be reclassified based upon a high likelihood of a favorable initial treatment response. In contrast, 12 percent had severe disease with a progressive or relapsing course despite ongoing or prior therapy; 5 percent remained on treatment despite a lack of response (CDAS 5B), presumably to prevent further progression, but it was not clear from the case records that treatment was helpful. The remaining 7 percent had failed numerous treatments and were deemed treatment refractory by the treating clinician (CDAS 5C). With longer follow-up, those classified as CDAS 3 or 4 may be reclassified to CDAS 1 or 2 if they remain clinically stable and treatment can be discontinued, or CDAS 5 if there is clinical progression or a relapse and progression despite therapy.

6. Although the CDAS may be a potentially useful clinical outcome tool to assess the long-term efficacy of treatments for CIDP, it also may have practical utility for patient recruitment in future clinical trials. The CDAS can be used to identify patients with long-term inactive disease off therapy (CDAS 1 and 2) and those who are treatment refractory (CDAS 5B and C), thus avoiding inappropriate patient selection. It has been recognized from recent treatment studies that many CIDP patients are over-treated and have stable disease off therapy, and there have been placebo response rates as high as 40 percent in some clinical trials. In contrast, those with CDAS 3, CDAS 4, and CDAS 5A would be ideal candidates for trial recruitment.

Reference:

Gorson KC, Van Schaik IN, Merkies ISJ, Lewis RA, Barohn RJ, Koski CL, Cornblath DR, Hughes RAC, Hahn AF, Baumgarten M, Goldstein JM, Katz J, Graves M, Parry G, van Doorn P. Chronic inflammatory demyelinating polyneuropathy disease activity status (CDAS): recommendations for clinical research standards and use in clinical practice. *J Peripher Nerve Syst* 2010;15:326-333.

Important Information!

SAVE THE DATE:

Continuing Medical Education

Current Developments in the Diagnosis and Management of Inflammatory Neuropathies

Saturday, October 27, 2012 • Fort Worth, Texas, USA

- FACULTY:**
- Bart C. Jacobs, MD, PhD, Erasmus Medical College, Rotterdam, Netherlands
 - Richard J. Barohn, MD, University of Kansas Medical Center, Kansas, USA
 - Angelika Hahn, MD, London Health Sciences Centre, London, Ontario, Canada
 - Pieter A. van Doorn, MD, PhD, Erasmus Medical College, Rotterdam, Netherlands

Immune-Mediated Small Fiber Neuropathy: A Treatable Condition That Can Mimic GBS or CIDP

David S Saperstein, M.D. and Todd D Levine, M.D.

GBS/CIDP Center of Excellence at Phoenix Neurological Associates, Phoenix, AZ
Dr. Saperstein, Member, Medical Advisory Board, GBS/CIDP Foundation International

Over the years, much has been written in The Communicator about the typical symptoms and test results seen in GBS and CIDP. We wish to bring attention to a type of nerve disorder that is also immune-mediated and treatable but cannot be diagnosed by usual methods: small fiber sensory neuropathy (SFN).

Our nerves are made of nerve cells, or fibers, having different diameters. Small nerve fibers are involved in the sensation of pain and temperature. These fibers do not have myelin and, therefore, transmit electrical signals more slowly than larger nerve fibers. Consequently, small nerve fibers do not contribute to the signals measured in EMG/NCV tests. Therefore patients with SFN have normal EMG's and also have normal reflexes. In contrast, large nerve fibers are myelinated, transmit electrical signals quickly and are involved in balance. Abnormalities of larger nerve fibers can be detected on EMG and usually cause reflexes to be decreased or absent. In most patients with neuropathies both small and large nerve fibers are affected. In GBS and CIDP large nerve fibers are predominantly affected. This explains why abnormalities of reflexes and EMG are heavily relied upon for the diagnosis of these disorders. Small nerve fibers are abnormal in patients with GBS and CIDP, but the clinical picture is usually dominated by abnormalities of larger fibers.

One way to diagnose SFN is by means of a skin biopsy test. After injecting a small amount of a local anesthetic called lidocaine, a small circle of skin measuring 3 mm (about one-eighth of an inch) is removed from the surface of skin; usually from a spot on the leg. There is little to no pain. The biopsy site is covered with a band-aid and heals after a scab forms. This is an easy and quick procedure that can be done in a doctor's office. The skin specimen is sent to a specialized lab for processing that allows the appearance and number of nerves in the top layer of skin (epidermis) to be assessed. There are only a few labs that perform this testing because of the technical demands of this procedure. An abnormal skin biopsy can suggest that a SFN is present but cannot determine the cause of the neuropathy.

Another way to diagnose SFN is by means of tests of the autonomic nervous system, especially a test called quantitative sudomotor axon reflex testing (QSART).

However, autonomic testing, especially QSART, is generally only available at a limited number of specialized centers.

There are several reports describing patients with an abrupt onset of numbness and pain resembling GBS. However, neurological examination and EMG do not show the features we look for to diagnosis GBS (such as abnormal reflexes and EMG). Some patients may have elevated spinal fluid protein levels, as in typical GBS. However, most standard tests are usually normal, making definitive diagnosis difficult. QSART or skin biopsy testing to assess epidermal nerves can be very helpful to prove there is a SFN. Acute onset SFN can be immune-mediated and may respond to the same therapies used for GBS, such as intravenous immunoglobulin (IVIg). In contrast to GBS, however, patients with acute onset SFN may respond to corticosteroid medications such as prednisone.

Compared with GBS, CIDP can be more difficult to distinguish from other neuropathies because a lot of chronic neuropathies (those that have a gradual onset and are progressive) can have features similar to CIDP. There are many potential causes for chronic SFN, to include diabetes, vitamin deficiency, and exposure to certain medications or toxins. About half the time no cause can be determined. However, there are a number of patients with immune-mediated chronic SFN. As with acute onset SFN, some will have elevated spinal fluid protein. Others will have evidence for other immune-mediated diseases such as lupus or Sjögren Syndrome. Other patients may have something called a monoclonal protein in their blood (which can also be associated with some forms of CIDP). As discussed above, QSART or skin biopsy can be used to confirm a diagnosis of SFN.

As with CIDP, a number of immune-modulating therapies can be used to treat chronic immune-mediated SFN. Patients experience improvements in their numbness and pain. In our experience, repeat skin biopsies - performed next to the sites of initial biopsies - can be helpful by providing objective evidence of improvement (although more study needs to be done to determine the usefulness of repeat skin biopsies).

For the reasons outlined above we believe it is important that physicians and patients be aware of small fiber neuropathy and its diagnosis so that this entity can be considered in patients being evaluated for possible GBS or CIDP.

References:

1. Seneviratne U, Gunasekera S. Acute small fibre sensory neuropathy: another variant of Guillain-Barré syndrome? *J Neurol Neurosurg Psychiatry*. 2002; 72:540-2.
2. Dabby R, Gilad R, Sadeh M, Lampl Y, Watemberg N. Acute steroid responsive small-fiber sensory neuropathy: a new entity? *J Peripher Nerv Syst*. 2006; 11:47-52.
3. Levine T, Saperstein D. Improvement in Small Fiber Neuropathies Following Immunomodulatory Therapy. *Neurology* 2011; 76 (Suppl 4): A109.

Disclosure:

Drs. Saperstein and Levine have a financial interest in a lab that performs skin biopsy testing for the diagnosis of SFN.

CHANGE SERVICE REQUESTED

DIRECTORY

Check the enclosed chapter directory and contact the chapter nearest you. In addition, our "subgroups" are listed below.

- **"CIDP" Group**
For those with a diagnosis of chronic inflammatory demyelinating poly-neuropathy. Please identify yourself to the National Office in order to be put in contact with others around the country.
- **Children with GBS**
Call Lisa Butler, 215-628-2771
670 Penllyn Blue Bell Pike
Blue Bell, PA 19422
Son, Stuart had GBS at 5 1/2 years old
- **Children with "CIDP"**
For children diagnosed with chronic inflammatory demyelinating polyneuropathy. A separate registry has been created. Please contact the National Office for details.
- **Group for Having GBS Two Separate Times**
Please call the National Office for contact with others.
- **Miller Fisher Variant Group**
Please call the National Office for contact with others.
- **Wheelchair Limited Group**
Please call the National Office for contact with others.
- **AMSAN Group**
Please call the National Office for contact with others.
- **A Teenage Pen Pal Group**
Arielle Challander, 231-946-7256
413 Shawn Drive
Traverse City, MI 49684
E-mail: GBSTeenPenPal@hotmail.com
Arielle had GBS in 2006 at age 13. She is willing to share experiences that others might not understand. To have a teenage GBS'er pen pal, write, call or e-mail to Arielle.
- **Pregnant Women with GBS**
Robin Busch, 203-972-2744
264 Oenoke Ridge,
New Canaan, CT 06840
Robin has offered to share her experience with GBS which came about during her pregnancy. We have many such cases and reassurance from someone who has gone through this is needed support.
- **Bereavement Group**
A group for anyone who has lost a loved one due to GBS/complications. Please contact: Bereavement Group at the National Office.
- **The "Campy" Group**
Those whose GBS onset was identified as a result of the campylobacter bacteria. Numbers to be used for research purposes.