

THE VIRGINIAN

SERVING VIRGINIA & WEST VIRGINIA

VIRGINIA CHAPTER NEWSLETTER

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FROM THE PRESIDENT

Joe Powers

A Sad Ending, But Hopefully a New Beginning

The MGFA Advocacy Plan

First, our sincere thanks goes to Damon Wainscoat, our Chapter Vice President, for his outstanding leadership in developing our Congressional Advocacy Program that sought increased funding for MG/Autoimmune Research through the National Institutes of Health (NIH).

Regrettably, Damon's health has taken a turn for the worse. In spite of high doses of Mestinon, Prednisone and CellCept, he has not been able to effectively respond to the medications. Damon was not able to undergo a thymectomy – which might have helped – in view of lasting complications from his Viet Nam war wounds. He is now being treated at Walter Reed Army Hospital.

In view of this decline in health, Damon has submitted his resignation from MGFA's National Board of Trustees, as well as Chairman of MGFA's Advocacy Committee. This was also prompted in part by MGFA's inability to fully commit to the Advocacy Plan submitted to MGFA last May.

The Plan provided for voluntary participation of the Chapters and their members, emphasizing support for increased federal funding of MG/Autoimmune Research at NIH. For the Plan to have been effective, "grass roots" support at the state and chapter level would have been needed for members to contact their Congressional representatives and indicate their interest – and need – for additional – not less – funding for medical research. It is a process that every Patient Support Group (Heart-Cancer-AIDS) effectively

practices successfully, except MGFA, although the National By-Laws specifically require an Advocacy Program. The Plan as submitted would have corrected this deficiency if executed.

By definition, "Advocacy" implies that you are "for" or "against" a specific issue or program. For MGFA to elect a "neutral" position, being satisfied with the status quo and to remain a passive bystander rather than an active participant, doesn't get the job done – or bring more resources needed for improved therapies, medications – and a cure!

A Bit of MG History

A recent timeline published in MGFA's "In Focus" Newsletter (Spring 06), showed with some pride, a 56 year long process in slowly developing methods in dealing with MG:

Time: 50's

Development: Mestinon

Time: 60's – 70's

Development: Intensive Care and Ventilator Management

Time: 70's – 80's

Development: Corticosteroids and Thymectomy Surgery

Time: 80's – 90's

Development: Treatment with Cyclosporine, Azathioprine, Plasma Exchange, and IVIG

Time: 2000's

Development: Mycophenylate or CellCept and Thymectomy studies on their effectiveness.

Let's give credit when due! These interventions do keep your name out of the obituaries, and in many cases get you back to a "near normal" routine in life for some, but not for all. Moreover, each of these "interventions" have sometimes debilitating side effects – and if you don't have insurance, you can't afford them!

Pharmaceutical companies don't rush in with costly research programs, because there aren't enough MG patients to repay their investment. And even if we – MG patients – each gave \$1000 annually (or pick any number) that would barely approach the level of research funding needed. Ergo, the alternative of being a proactive advocate, reminding our Congressional representatives of the need for fewer "pork barrel" projects, and more down to earth support of healthcare. After all, it's your tax dollars!

A New Beginning

Hopefully, the last word isn't said – and that MGFA will find a way to assume a more positive advocacy role. The opportunity to do so is reflected in a second article in MGFA's Spring Newsletter by Dr. Robert Pascuzzi, M.D., Chair of the Myasthenia Scientific Advisory Board (MSAB) and Chairman of Neurology at Indiana University School of Medicine.

Dr. Pascuzzi correctly gives well deserved credit to MGFA and all those who have gone before us in the battle with MG – and the doctors and scientists who have made life itself possible for us as patients. And for their effort and dedication we are truly grateful.

Very succinctly, Dr. Pascuzzi summarizes what "we've" learned about MG over the last 50+ years, and what we don't know. Most of those questions revolve around possible causes of MG – and ways of prevention. To get the answers to these questions, Dr. Pascuzzi commits the MSAB to establishing a "Steering Committee for MG Research" that **“over the coming year will clearly articulate the research agenda and lay out a long-term strategic plan for research activities and desired accomplishments. This group will determine the key research goals that need to be achieved over the next decade. The goals will be prioritized and placed in a timeline and estimates provided regarding the relative cost**

in manpower and funding necessary...”

“MSAB will provide the research goals, targets and timeline as a five year and a ten year strategic research plan”

A Positive Step

This is a very positive – and long overdue step that needs our support It will not be the easiest thing to do considering that members of the MSAB already have demanding academic and clinical responsibilities. MGFA should consider offering “contract support” to MSAB to insure timely completion. That support could be scientific and technical, perhaps through an existing medical research center.

Complimentary Actions

The development of an MG Research Plan converges with NIH’s “Roadmap to the Future”, and the NIH reorganization, in which great stress is placed on an integrated, multi-disciplinary approach to research. This is typified by the recent NIH Autoimmune Research Plan.

In a May 8th conference in Washington, sponsored by the National Health Council, Dr. Raynard Kington, M.D., the NIH Deputy Director, described the new NIH Office of Portfolio Analysis and Strategic Initiatives (OPASI). I asked Dr. Kington if NIH would continue the “Autoimmune Disease Research Plan” mandated by Congress through the NIH Autoimmune Coordinating Committee. His reply was emphatically affirmative. Consequently, there is an immediate – and receptive audience for the MG Research Plan. To have it in place would further strengthen MGFA’s Advocacy Program as well – because there will be specific objectives with associated resource estimates needed to meet those goals.

Support Needed

In that sense we need to whole heartedly support the MSAB. Their efforts can be complimented by the Advocacy Plan proposed by Damon. Although MGFA hopes to initiate an Endowment Fund to support MG research, we should not give up on winning Congressional funding as well through an advocacy program in coordination with the National Health

Council and the Association of Autoimmune Patient Support Groups. We need both!

To show tangible support for both the research and advocacy programs, let’s begin by redoubling our financial contributions. Let’s be optimistic – and mark our contributions for “A New MG Beginning”.

Subsequent Cancers Noted in MG/Thymoma Patients

Scientists at the National Cancer Institute have reported on the incidence of malignant thymoma tumors and subsequent follow-on malignancies that can also occur. Although the exception, thymoma tumors can be associated with Myasthenia. Reviewing 849 cases extrapolated from SEER medical records covering a 25 year period, 66 subsequent malignancies were noted – or nearly 8%.

“The most notable excess risk for subsequent malignancy was for non-Hodgkin’s Lymphoma (B immunophenotype).” Also noted were digestive system cancers and soft tissue sarcomas. Incidence was higher in males and highest among Asians, occurring more likely in late adulthood. Variation and incidence by race suggested genetic factors may be relevant. With a diagnosis of Myasthenia, a CT scan or MRI of the thymus/thoracic area is usually made. The authors noted malignancies peaked in late adulthood, suggesting further study.

Source: “Malignant thymoma in the United States: Demographic patterns in incidence and association with subsequent malignancies” by E.A. Engels, and R.M. Pfeiffer. International Journal of Cancer, July 1, 2003; 105(4): 546-51. The authors are associated with the National Cancer Institute, Division of Cancer Epidemiology and Genetics, Viral Epidemiology Branch; Bethesda, MD.

Note: Our thanks to Dr. Mark Niehaus, M.D. for bringing this article to our attention! Dr. Niehaus is an Internal Specialist associated with Palmyra Medical Associates and Martha Jefferson Hospital in Charlottesville, VA.

Association Shown Between Autoimmune Diseases and non-Hodgkin’s Lymphoma

In a population-based case-control study in Denmark and Sweden, 3,055 non-Hodgkin’s lymphoma patients and 3,187 matched control subjects were asked about their history of autoimmune and chronic inflammatory disorders, markers of severity, and treatment.

The results of the study confirmed the associations between certain autoimmune disorders and the risk of non-Hodgkin’s lymphoma. They suggest that the associations may not be general but, rather, mediated through specific non-Hodgkin’s lymphoma subtypes. These subtypes develop during postantigen exposure states of lymphocyte differentiation, consistent with a role of antigenic drive in autoimmune-related lymphomagenesis.

Risks of all non-Hodgkin’s lymphoma (overall and subtypes) were increased in association with rheumatoid arthritis, primary Sjogren’s syndrome, systemic lupus erythematosus and celiac disease. All of these conditions were also associated with diffuse large B-cell lymphoma, and some were associated with marginal zone, lymphoplasmacytic, or T-cell lymphoma. Psoriasis, sarcoidosis, and inflammatory bowel disorders were not associated with increased risk of non-Hodgkin’s lymphoma.

Even use of nonsteroidal anti-inflammatory drugs, systemic corticosteroids, and selected immunosuppressants was associated with the risk of non-Hodgkin’s lymphoma in rheumatoid arthritis patients but not in subjects without rheumatoid arthritis. Also, multivariable adjustments for treatment had little impact on risk estimates.

Another study, this one conducted by researchers at the Mount Sinai Medical Center, in New York, concluded that autoimmune disease may account in part for the increase in non-Hodgkin’s lymphoma, especially in women.

To examine possible relationships, the researchers questioned 278 patients with non-Hodgkin’s lymphoma, who had been seen over a 10-year period, about prior autoimmune disease. They were compared with a control group of

317 patients who had other hematologic disorders. Overall, in comparing patients of the same sex and age, those with non-Hodgkin's lymphoma were 2.6 times more likely to have had a prior history of autoimmune disease.

Lead investigator, Dr. Janet Cuttner, said, "There has been an increasing incidence of non-Hodgkin's lymphoma over the past 20 years which is unexplained". She also stated, **"Autoimmune disease occurs predominantly in women, some of whom are receiving potent immunosuppressive therapy, which may possibly increase their risk of developing non-Hodgkin's lymphoma."** In the study, 56 percent of the non-Hodgkin's lymphoma patients with autoimmune disease received immuno-suppressive treatment compared to 38 percent of controls.

In commenting on the study, doctors Sharon Chambers and David Isenberg, of University College London, noted that although the researchers might have provided greater detail, "a dedicated attempt to study the link between cancer and autoimmune diseases is certainly a step in the right direction."

Sources: "Autoimmune and Chronic Inflammatory Disorders and Risk of Non-Hodgkin Lymphoma by Subtype," Karin Eskrom Smedby et al., *Journal of the National Cancer Institute*, January 4, 2006; "Autoimmune Disease Tied to Non-Hodgkin's Lymphoma," David Douglas, *Journal of Rheumatology*, 2005 Oct.

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Intravenous Immune Globulin in the Treatment of Myasthenia Gravis
By Steven Novella, M.D., Assistant Professor of Neurology, Yale University School of Medicine.

It has long been known that Myasthenia Gravis (MG) is an autoimmune disease; the immune system, which evolved elaborate mechanisms to identify self from non-self, is imperfect and may become activated against one's own tissue. In the case of MG, antibodies are formed which attach to the Acetylcholine receptor on the post-synaptic

neuromuscular junction, blocking the chemical signal connecting the nerve and muscle and resulting in weakness. (At least in most patients, there are subtypes of MG with similar but distinct mechanisms, such as the recently discovered MuSK Ab positive subtype.) Treatment of MG is therefore similar to all other autoimmune diseases – namely, immune function must be suppressed.

Over the years an armamentarium of immunosuppressive drugs and treatments has been developed to treat autoimmune diseases such as MG. One such treatment, intravenous immune globulin (IVIG) is the focus of recent interest for an expanding role in the management of MG.

IVIG is pooled human immunoglobulin type G (IgG) collected from blood donors. The IgG is purified from the plasma component of the blood, and it is estimated that tens of thousands of individual donors are represented in one batch of IVIG. Therefore, the product contains a large variety of IgG, which is likely important to its function. Immunoglobulins are the antibodies of the immune system. They are proteins that have one end, which is highly variable between different antibodies, so that they have varying affinity for different substances. This affinity determines what the antibodies can target. The other end interacts with other components of the immune system. Therefore, antibodies with an affinity for a particular protein on an invading organism, such as a virus, will bind to the protein with their variable end, while the other end activates the cellular components of the immune system, thereby targeting and destroying the invader. It also stimulates immune cells to produce more of the antibodies, thereby increasing the immune response against the invader.

IVIG has two primary medical uses, quite opposite in effect. Some individuals have an immunodeficiency syndrome which they cannot produce their own antibodies in sufficient amounts to maintain an immune defense against infection. **In these patients IVIG is given to supplement their immune system and increase their defense. In some autoimmune diseases such as MG, one particular type of**

antibody is produced in large numbers that is attacking the patient's own healthy tissue. In these cases IVIG can be given to suppress the immune system.

There are several known mechanisms of immune suppression with IVIG, although the relative importance of each in various diseases is still a matter of debate. The simplest mechanism is that the infused antibodies will, for a time, dilute the abnormal host antibodies that are causing the disease. The presence of large amounts of IgG will also suppress the production of host IgG (including, hopefully, the disease causing antibodies). The donated antibodies will also bind to the other components of the immune system thereby using up resources. There will therefore be fewer immune resources available to do damage. (This is like starting a fire to burn away the underbrush and thereby decrease the spread of a wild fire by depriving it of fuel.) The infused antibodies also may bind to host antibodies, including those causing disease, and take them out of action. IVIG also increases the breakdown of host immunoglobulin and decreases its production.

As an immunosuppressant treatment IVIG can be used in one of two basic strategies. The first is acute therapy: as a rapidly acting but relatively short lasting treatment for an autoimmune disease that is itself short lasting (days to weeks) or is currently flaring. A common example of this use is for Guillain Barre Syndrome, an acute autoimmune disease with about three weeks of immune activity. The second is for chronic autoimmune disease. In this case IVIG is given approximately once every 1-3 months over time for long term immunosuppression.

Side effects of IVIG are usually minimal, including headache, local skin reaction at the infusion site, and flu like symptoms. Less commonly patients may develop aseptic meningitis, although without long term consequence. Rare but severe side effects include thromboembolic events, such as pulmonary embolism, caused by the resulting increase in the blood's viscosity.

Of note, another treatment, plasmapheresis, is very similar to IVIG in effect and use. Plasmapheresis is the process of filtering the blood and removing all the protein. The process is not very selective, and most of the protein is removed. Albumen, a major blood protein, is then replaced so as to maintain the blood's normal amount of protein. Most of the antibodies, good and bad, are also removed. They are eventually replaced, but for a time there will be much less of the disease causing antibodies around. Like IVIG, plasmapheresis can be used as acute therapy or given intermittently as chronic therapy.

In many diseases, such as Guillain Barre Syndrome, IVIG and plasmapheresis are interchangeable and equally effective therapies.

Historically IVIG has been used in MG as an acute therapy for MG crisis. In this way it has primarily been an alternate therapy to plasmapheresis. IVIG has been shown to have similar efficacy to plasmapheresis in the treatment of acute MG exacerbations. It has also been shown to be effective in treating patients prior to thymectomy in order to improve their ability to handle the anesthesia and surgery. The evidence of effectiveness, however, is not yet definitive, and more studies would be helpful.

Another similar use of IVIG is for severe refractory MG patients who have not come under control with standard treatments. In these cases IVIG can be given over a short course in order to bring the patient under control, but traditional long-term medications are still used to maintain control.

Recently, there has been a great deal of interest in using IVIG to chronically treat MG. There are already small studies, which show that IVIG may be effective if used in this way. More study is required before IVIG is likely to be accepted broadly for this use.

The potential advantage is not that IVIG is likely to be more effective than current treatments. The published studies and consensus of clinical experience is that IVIG is likely to be

as, but not more, effective as plasmapheresis. Also, the standard combination therapy of prednisone for short term treatment (usually around one year of treatment tapering either to a very low dose or completely off) with long term immunosuppressive therapy with either azathioprine, cyclosporine, or Cellcept has proven to be very effective in controlling MG in most cases. It is unlikely that IVIG will prove to be more effective than these established therapies. **It is important to note, however, that these "established" therapies, although supported by some studies, are far from proven by large definitive clinical trials.**

What is driving the interest in IVIG for chronic therapy is not improved efficacy but decreased side effects. Plasmapheresis long term has the major disadvantage of requiring the placement of an intravenous catheter. Such catheters are prone to either clot off or become a site of infection. IVIG also requires venous access, but not as large a bore as for plasmapheresis. Therefore, routine peripheral venous access may be adequate in most cases.

Prednisone also has an unattractive side effect profile. Prednisone decreases resistance to infection more than IVIG. It also suppresses many of the symptoms of infection, like fever and swelling, and therefore an infection may go unrecognized longer. Prednisone counteracts the effects of insulin and may cause temporary diabetes. It also predisposes to gastric ulcers, weight gain, and osteoporosis. Less common, but very serious side effects include aseptic necrosis of the head of the femur (essentially, severe arthritis of the hips) and steroid induced myopathy (muscle damage). Long term steroid use can therefore, ironically, cause weakness.

Cost of IVIG treatment, however, is a concern, as prednisone is very cheap (cost estimates are at less than \$50 per year) and IVIG is very expensive (total cost would be in the tens of thousands of dollars per year). However, there are many hidden or secondary costs of prednisone therapy. Patients on prednisone must also be treated with

calcium supplements and perhaps also other agents to prevent osteoporosis, and they need to be monitored for diabetes. More importantly, even a single complication of prednisone, such as an opportunistic infection requiring a hospitalization, would have a cost similar or higher than that of IVIG. Therefore, future studies of the chronic use of IVIG in MG should include assessments of cost effectiveness.

Conclusion: So the current hope for IVIG in MG is that it will reduce or eliminate the need for prednisone. Plans are under way for a pilot study to look at exactly this. If successful this could lead to a significant shift in the standard management of MG, with more reliance upon intermittent treatments with IVIG and less reliance on prednisone. This will mean fewer side effects for MG patients and hopefully improve quality of life.

Source: AMPS: CT Nutmeg 1/04 (Note: We have requested a study update)

CDC Research Shows Differences Between Myasthenia And Chronic Fatigue Syndrome

Extensive research by the Centers for Disease Control (CDC) demonstrates the connection of Chronic Fatigue Syndrome (CFS) to the immune system, as well as relevant genetic and hormonal factors.

Many of our MG patients are all too familiar with chronic fatigue. Some of our patients have also been diagnosed with CFS – Chronic Fatigue Syndrome, and others suspect CFS as an additional explanation for their continuing fatigue.

The research distinctly confirms however that CFS is not to be confused with Myasthenia, although both involve fatigue, stress, and the immune system.

The Center for Disease Control and Prevention (CDC) in Atlanta has now reported on the causes of CFS and its correlation to the immune system. Several reports published in the April '06 issue of Pharmacogenomics, describe CFS as a "debilitating condition accompanied by unexplained extreme fatigue, memory and concentration problems, sleep disorders and chronic pain" – but not muscle weakness.

Results of several studies carried out concurrently, indicate that like cancer or heart disease, CFS is “very heterogeneous. It is not just one thing”, implying that there are different forms of CFS with different causes, primarily associated with genetic links as well as mutations or changes in genes that affect both the brain and the immune system, and the body’s response to stress.

According to Dr. William C. Reeves, the genetic links “should put to rest the idea that CFS is a made up diagnosis for a bunch of hysterical, upper-class white women” – pretty strong language for a scientist! The team of 20 different scientists – physicians, molecular biologists, epidemiologists, and mathematicians – studied 227 patients in a hospital setting, complete with blood and hormone tests, sleep studies, and psychological tests. Patients fell into one of four groups:

- One that fully met CFS diagnostic criteria
- A second that also met the criteria, but in addition had “melancholic depression which did not fit current CFS diagnosis standards
- A third group had all the essential characteristics of CFS but their fatigue was not sufficiently severe
- The last group – for comparison – was basically healthy.

The studies examined some 20,000 genes known to be active in response to emotional stress, infections, or injuries. Of these 20,000, several hundred were more – or less – active in CFS patients. Specifically, 26 genes were correlated with CFS and could be used to predict which of 6 different forms of CFS were present in the patient.

Out of another set of 50 genes, 5 were identified as having a “genetic glitch” and were associated with levels of serotonin and glutamate. Low levels of serotonin, a neurotransmitter, are associated with depression. Glutamate is associated with stress response.

Dr. Suzanne D. Vernon, a lead scientist on the project, observed that various levels of hormones related to

stress were also involved, as the body attempts to respond to various physical or emotional events, particularly in patients who are “genetically predisposed to handle stress poorly”. Stress is also noted as a probable “trigger” in MG. Dr. Julie L. Gerberding, Director of CDC, described the \$2 million dollar research as an important step in understanding CFS that will lead to better diagnostic tests and therapies for nearly 1 million Americans affected.

FDA Cautions on Use of Statins with Cyclosporine and Other Drugs

FDA has recently issued a warning about increased risk of severe muscle and kidney damage as a result of taking cholesterol lowering statin drugs along with various other drugs – one of which, Cyclosporine, is sometimes prescribed for MG patients. FDA singled out lovastatin, marketed by Merck and Co. as Mevacor – noting that the lovastatin dosage should be limited to 20 milligrams daily if taken with Cyclosporine. MG patients taking both medications should review the issue with their physicians.

The muscle injury which might result is known as rhabdomyolysis, characterized by tenderness, pain, and weakness in the affected muscles. The most serious possible effect however could involve the kidneys. As muscle cells are damaged as a result of the drug interaction, a toxin called myoglobin is released into the blood stream that the kidneys attempt to filter. These toxins however can block the kidneys and cause kidney failure thus requiring treatment, ranging from temporary dialysis to intravenous hydration, and/or diuretics to flush out the kidneys – or use of bicarbonate to prevent toxic compounds from forming. When muscle cells break down rapidly, potassium is released into the blood, and if high enough levels occur, heart rhythm disturbances can potentially result.

Other causes of rhabdomyolysis include drug overdoses of amphetamines, heat stroke or intolerance – or alcoholism. Symptoms in addition to overall weakness, fatigue, stiffness or aching, include urine that is dark, red or cola colored. Unintentional

weight gain may also occur. Specific diagnosis is made by blood and urine tests.

Other drugs noted with caution by FDA if used with statins (lovastatin) include Niacin – recommending that daily doses be limited to 1000 milligrams. Several antibiotics (Clarithromycin – with a “Do Not Use” note) – include a total of 15 different drugs. FDA also cautioned about the use of “large quantities of grapefruit juice”. The list is available on www.accessdata.fda.gov.

Source: Public Citizen Health Research Group (www.citizen.org/hrg)
Sidney M. Wolfe, M.D.

Internet Sites of Interest

The University of Maryland Thoracic Surgery Division has a page that discusses the thymus gland in myasthenia gravis and the various forms of thymectomies.

www.umm.edu/thoracic/myasthenia_gravis.htm?source=goto

From i-medicine.info, the Diseases Database provides a useful reference service for medical practitioners and researchers. The Database website offers a cross-referenced index and search portal that covers such topical areas as Symptoms and Signs, General Internal Medicine Disorders, Drugs and Medications, Congenital and Inherited Disorders, and more.

www.diseasedatabase.com/content.asp

Exercise programs...There’s everything from boating to horseback riding from an adaptive viewpoint, since this organization specializes in exercise for people with physical disabilities...from the National Center of Activity and Disability within the Department of Disability and Human Development (DHD) of the University of Illinois at Chicago.

www.ncpad.org

MED NOTES

Being a Myasthenic patient, one can easily become preoccupied and totally focused on MG problems, to the exclusion of other potential health issues. One of our program objectives was to concisely address other health

issues and research developments in our newsletter that may - or should be - of concern to our members. Accordingly in each issue, we have highlighted a few articles we hope you will find helpful and informative.

Standard Measurements for Diabetes Risk Lowered

The New England Journal of Medicine reports on the increased risk of diabetes depending on the amount of glucose or sugar in your blood.

Tracking 13,000 young Israeli soldiers over a 6 year period, those with initial levels around 90 milligrams of glucose/sugar per deciliter appeared to be twice the risk of diabetes than those with readings between 50 and 81. The risk tripled for men with readings between 95 and 99. Previously, readings below 100 were considered “normal” – that’s now changed.

Dr. Ronald Ayky, a Harvard Medical School Endocrinologist, who wrote the commentary, said: **“I now tell patients and members of my own family, several of whom have levels in their ‘90’s, that they need to keep their weight down and start exercising” – to get that reading substantially below 90.**

Reducing carbohydrate intake and following the “no white” rule is also helpful: no white bread, pasta or potatoes would be a step in the right direction, substituting whole grains instead while emphasizing fruits and vegetables.

Source: New England Journal of Medicine, October ‘05

Book Bytes

Attacking Myasthenia Gravis – A Key in the Battle Against Autoimmune Diseases by Dr. Ronald E. Henderson, M.D. Court Street Press, Montgomery, AL (2003), 294 pgs, \$25.95 + \$4.50 shipping/handling.

Dr. Henderson’s story has a unique twist. Not only does he suffer from Myasthenia Gravis (MG), but he is also a physician. He negotiated the healthcare system as a patient, and he became personally active in raising awareness with doctors to help diagnose his disease. He offers his 12 step plan to

help sufferers “take ownership” and manage their disease.

Early Symptoms

For almost 30 years, Dr. Henderson was a practicing physician. For over 25 years he specialized in obstetrics and gynecology. In 1994 he retired because he was too weak to continue. He was mysteriously losing strength in his muscles and losing overall stamina. Mysterious, because for his whole life he had enjoyed a high level of energy and stamina at his disposal. After experiencing difficulties with other symptoms such as weakness in his neck muscles and trouble chewing, he went through a battery of tests concluding that he did have MG.

Getting Familiar with MG

Once his diagnosis was confirmed, Dr. Henderson was anxious to learn as much as possible about MG. He wanted to be an expert on his disease. Some of the things he learned early is that MG is rare and very difficult to diagnose. It was not unusual for patients to have a delay of a year or two before getting a diagnosis. The complexity of the disease and the varied body parts that it attacks as well as the lack of in-depth education by doctors on the disease all contribute to this problem. The most important thing the doctor learned is that MG is a chronic, incurable, autoimmune disease which can possibly go into remission for an indefinite period of time.

Confronting Reality

After a conservative course of treatment with medication it became apparent that a stronger type of treatment was necessary as well as an adjustment to his lifestyle. And he had to accept the side effects of this stronger immunosuppressant therapy because he knew that in the long run it was going to help him get better and help him manage this disease. Eventually this course of treatment was successful, and Dr. Henderson was able to regain his strength and get back to many of the activities he was forced to give up.

Taking Care of His Health

Prior to getting MG, Dr. Henderson had a health-maintenance regimen that kept him healthy. He has added this health regimen to his MG regimen and the result is a style of healthy living that

helps prevent his MG from being worse than it is. His health regimen is:

1. Take medicines faithfully.
2. Be careful not to be exposed to infections.
3. Cooperate and interact with physicians to manage your healthcare.
4. Get your rest.

Staying Positive

Dr. Henderson has come to grips with the fact that he has a chronic, incurable disease. He has learned to ride with it and continue doing as much as he can in spite of it. It is his challenge to manage the disease. **And he does so with an incredibly positive attitude. He likes to say that “it’s not so much what happens in your life, but what you do with what happens in your life”.** And what he has done reaches far and wide – he has authored a book about his experience with MG (“Attacking Myasthenia Gravis”), started a foundation for autoimmune diseases and created a website for the foundation that increases awareness of these diseases and helps raise funds for research. He also speaks to MG and autoimmune groups across the country. It is through these extraordinary efforts that Dr. Henderson has touched the lives of so many and will touch many more in the future.

Source: HOVAK-Oklahoma Chapter of the Myasthenia Gravis Foundation Newsletter.

Ordering Information: Order from any bookseller or from one of the following:

By Mail: International Autoimmune Research Foundation, P.O. Box 131253, Birmingham, AL 35213.

By E-Mail: rehtvf@aol.com or info@newsouthbooks.com

Toll Free: 1-866-639-7688

Final Thoughts

Compassion is needed to heal the hurt and heart of others – Compassion is love in action.

You can give without loving – but you can’t love without giving.