Notes from the Bi-Annual Guillain-Barre/CIDP Symposium, November 2-5, 2006
Ted Asch
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Note: Spelling of drugs and medical terms may not be correct.

1.0 Location

The meeting was held at the Embassy Suites in Scottsdale, Arizona. The GBS foundation had blocked 250 rooms. There were 400 people attending and so there were not enough rooms at the hotel. The hotel was very wheelchair accessible although there were some comments that the carpets in some of the rooms were too thick for easy wheelchair mobility. The hotel had a very good complimentary breakfast and happy hour every afternoon. It was located about 0.25 miles from a large mall with many restaurants and shops.

2.0 Thursday Evening, November 2

Evening welcome reception. Supposed to be a desert reception but had many kinds of food, coffee, soda. Met many GBS and CIDP survivors. Each had similar but different stories. There were quite a few CIDP people who were going through tough times. One woman has had 9 attacks in 3 years. An attack is currently ongoing. Another had been on 6000 mg/day Neurontin, had been bedridden for 14 years, and had just finally recovered to some degree last April. Met one person (and others later) who had eaten not-cooked-enough chicken and had contracted a bad case of GBS. Another had contracted GBS from a flu vaccination.

3.0 Friday Morning, November 3

3.1 Plenary Session

400 attendees came from 36 states (including Alaska and Hawaii) and from the Netherlands, Germany, New Zealand, England, and Canada. Session was a review of GBS, nerves, myelin sheaths and their properties.

3.1.1 The Cochrane Library

The Cochrane Library is a collection of databases that contain high-quality, independent evidence to inform healthcare decision-making. Cochrane reviews represent the highest level of evidence on which to base clinical treatment decisions.

URLs:
http://www3.interscience.wiley.com/cgi-bin/mrwhome/106568753/ProductDescriptions.html
http://www.mrw.interscience.wiley.com/cochrane/cochrane_clsysrev_subjects_fs.html

Recent reviews of evidence in the Cochrane Library indicate that there is no difference in the results using either Plasma Exchange (plasmapheresis) or IVIG.

3.1.2 Issues Still Unresolved in GBS

A) New primary treatments for GBS and CIDP

B) New treatment for unresponsive GBS (GBS not responsive to primary treatments)
C) Enhance nerve regeneration
D) How to improve function in existing but partially repaired nerves.

3.1.3 CIDP review

Treatment must be given as early as possible once diagnosis is made. 80 % of people respond to steroids and plasma exchange, 70 % respond to IVIG.

Unresolved CIDP issues

A) How to accurately diagnose earlier.
B) Which is the best first line treatment? Are other treatments better?
C) What to do for CIDP over the long term?

3.1.4 Advocacy

Advocacy for research into GBS and CIDP must be made at the national level to US Congress. Doesn't help to advocate at NIH level.

3.1.4.1 Peripheral Nerve Society

The Peripheral Nerve Society is an international organization of physicians and scientists working together to develop and provide the best treatments for people who have peripheral nerve diseases. This goal is realized by cooperation - supporting research, training scientists and healthcare professionals, setting standards of care, creating new treatments, and facilitating clinical trials.

The Peripheral Nerve Society was founded in 1994, having evolved from two groups of academic investigators interested in understanding the basic biology and function of the peripheral nervous system - the nature of nerve injury and repair. These groups met periodically in closed meetings to discuss advances in this understanding and how this knowledge might be applied to care for patients with genetic, traumatic, toxic, or metabolic nerve disease. The interests of our members encompass all aspects of the peripheral nervous system, both clinical and scientific, and range from electrophysiologic tools for diagnosis to molecular mechanisms of disease and nerve fiber regeneration. The Society is incorporated, and open to general membership by those interested in the peripheral nervous system and its diseases.

URL: http://pns.ucsd.edu/

There is a Journal of Peripheral Nerve Society. Subscription is $207.

3.2 CIDP Diagnosis and Treatment (There was a concurrent session on Diagnosis and Treatment of GBS). Presented by Dr. Richard Lewis

There are many variants of CIDP: MMN, CMT, Lewis-Sumner, CNS, IGM, MGUS, IgM, POEM

CIDP may be monophasic (happens one time only).
CIDP can be Acute – whole episode occurs < 4 weeks (This is GBS-like)
Sub-acute – Episode occurs over 4-8 weeks

Chronic – Episode occurs > 8 weeks

In the acute mode, have increased central spinal fluid (CSF) proteins. Steroids are ineffective. In the subacute and chronic modes, also have increased CSF but steroids are effective. May have not antecedent event that can be related to cause episode. May have axonal nerve damage. Axons allow nerves to conduct electrical impulses to muscles. Demyelination causes the conduction to slow. The nerves have nodes along them called Node of Ramvier. This is where the current can be measured. Nerves can be shortened, thinned, or demyelinated. Nerves conduct electricity at around 50 meters/sec. Depending on the individual, 45-32 m/sec may also be ok. But much slower will not be ok.

Dr. Lewis showed a video by Michael Rasminsky, a neurologist in Toronto, who did research on nerve conduction in rats. Was able to correlate loss of conduction to damage of the actual nerve.

3.2.1 CIDP Diagnosis

A) Determine distal, proximal neuropathy
B) Look for features not length dependent (length meaning toes, fingers)
C) Use EMG to determine nerve electrical conduction slowing
D) Check if CSF protein increases => damage to the nerve roots
E) If EMG not conclusive, may need nerve biopsy

Can get double vision due to demyelination. Also fatigue due to the nerves not conducting the electricity in an efficient manner.

Diagnosticians are now using MRI's to support EMG's. Other disorders can also be determined with nerve biopsy.

3.2.2 CIDP Treatment Goals

A) Improve clinical status and functions (quality of life)
B) Remission
C) Control with least side effects
D) Cost is an issue

3.2.3 Therapy Strategies

A) IVIG for 4-8 weeks
B) If IVIG not effective, try Plasma Pheresis for 4-8 weeks
C) IF plasma exchange not effective, try immuno-suppression (steroids)

Can try Azathioprine (AZT) and mycophenolate. Also methotrexate has been shown to work on sensory CIDP. Must watch white cell count with AZT.

Alpha-interferon has been shown to both reduce CIDP but also cause CIDP. Etanercept can also cause CIDP.

Cellcept has shown promise. Does not effect the liver as does AZT. Several people in the audience were on Cellcept and were showing good results. Each pill is 500 mg. People usually take 2000 mg /day

3.2.4 Dr. Lewis strongly suggests a "contract" between patient and medical practitioner on what to expect from medicines and treatments. What will define a success or a failure and in what time frame?

3.3 Outcomes of Therapy in CIDP

Presented by Dr. Angelika Hahn, University of Western Ontario (medical research hospital), London, Ontario.

Dr. Hahn is studying the interaction between the trigger and immunological response? She strongly suggests not to delay the diagnosis and a precise diagnosis is very important. CIDP has not been shown to be inherited. Although individuals and families may have a predisposition for immunological disorders.

In CIDP have chronically-recurring destruction of myelin segments. Myelin are composed of Schwann cells that wrap the axonal nerves. There is a interdependence between myelin and the axons. Segmental demyelination causes failure of the electrical impulse conduction. However, the nerve conduction is quickly and fully reversed when nerve remyelinated. If can correct the conduction blockage, function comes right back. However, los of axons leads to atrophy which can be more permanent. The potential for re-growth is age dependent. If not the axons are not reconnected within 6-8 months, the Schwann cells go away and the deficit is irreparable.

A study was done on from 1972 to 2002. Reviewed patients with CIDP for at least 6 months. Patients sorted by first treatment prescribed. Patient was scored by degree of disability and response to treatment. 90 patients involved, ages 3-75. The median time for diagnosis was 5 months. Follow-up evaluations occurred over next 6 months to 28 years. 10 of 27 patients did not go into remission. 8 patients died. 9 patients moved or were lost to the follow-up evaluations. 25 % developed CIDP by a virus and 20 % were women who contracted CIDP after becoming pregnant. 65 % of patients had typical clinical presentation, 18 % were purely sensory CIDP, 10 % had asymmetric presentation, 7 % had more upper limb loss than lower limb loss.

100 % of patients responded to Prednisone. However, only 28% of patients went into remission. Plasma exchange had a 74% response rate but only 10% went into remission. 64% responded to IVIG but only 22% went into remission.

Overall, 66% of patients had full remission. The majority of patients that were not responding to the treatments were those who had the sensory-predominant CIDP.

Conclusion: CIDP highly treatable but needs early and aggressive therapy.

4.0 Friday Afternoon, November 3

4.1 Pain Management by Dr. Gareth Parry, University of Minnesota

Pain occurs in 15%-85% of GBS/CIDP patients. Pain occurs during all phases of GBS. Usually pain is the first symptom in 30%-50% of patients. The pain exacerbates the autonomic instability. The first pains are nociceptive (**nociceptive -** *caused by or in response to pain; "a nociceptive spinal reflex").* This is an inflammatory pain and not a neuropathic pain which can be pain deep within the body.

Severe pain during an attack means that severe paralysis will likely follow. i.e. The worse the pain, the worse the paralysis to follow.

To reduce this neuropathic pain, must use narcotic analgesics. However, you must be careful with a compromised pulmonary function due to the attack. As you fix the nerve problem,

the pain problem will also be resolved. Plasma pheresis and IVIG may reduce the pain as they reduce the GBS attack. Steroids will not improve GBS but they may improve the pain by reducing the inflammation.

The mechanism of pain is due to swelling and inflammation of nerve fibers and release of inflammatory mediators that activate the nociceptive receptors.\

During recovery can have pain. This is more common if had pain with the paralysis. So if have pain during the attack, will have pain during the recovery. This is more likely to occur if have axonal degeneration which is proportional to the severity of weakness experienced. The pain during an attack is usually distal (hands, feet), easily localized, and on the surface of the skin. The character of the pain is a burning, tingling, cold, and stabbing feeling. The pain can range from mild to severe. Will likely be worse after exercise and worse at night during inactivity.

Three types of pain:

Hyperalgeisa – Pain hurts worse than expected.
Allodynia – Something that would normally not cause pain but still hurts
Hyperpathia – Persisting pain.

Nociceptic pain – The prognosis for controlling is usually good. Must use narcotics including Tramadol, oxycontin, MSContin, methadone, Fentanyl patches.

Neuropathic pain – Must use something like Neurontin. Up to 1800 mg/day should help. At 1800 mg/day, body absorbs approximately 40%. At 3000 mg body only absorbs 25%. So if neurontin doesn't work at 1800 mg/day, it will likely not help any more at 3000 mg/day. Lyrica is absorbed by the body much more efficiently and may only need to take it twice day. With all these types of drugs, you must start at low doses and increase slowly. Should try multiple drugs to attack the pain and take the long view on treatment.

4.2 Men's Issues – Dr. Koppel Burke, Dr. Joel Steinburg

There was a concurrent session on women's issues with GBS.

The impact of GBS is on income, image and can leave some residual disability like sexual dysfunction. There are sympathetic nerves in the pelvis that are affected by GBS. These nerves are also demyelineated. A chemical in the body called Cyclic GMP allows blood to flow in the body to create an erection. Another chemical in the body, PDE5 – phosphdiasterase 5, blocks the flow of blood and stops the erection. Problem is that GBS allows for creation of too much PDE5.

The Kinsey Report indicated that for men 41-55 years of age, 7.7% would be impotent. However, 28% of men with GBS in the same age range are impotent. Of 396 men studied, 42% were impotent after GBS versus 8.8% for the same age ranges.

The more severe the GBS attack, the more severe the impotence. So there is significant impotence post main GBS recovery.

Pills like Viagra, Cialis, Levitra prevent the rapid breakdown of the CyclicGMP by PDE5. However, if taking nitroglycerin, you can't take any of these pills.

Other sources of impotence include circulation disorders, endochrine disorders, nerve dysfunction, and some medications that can cause impotence.

4.3 ASK THE EXPERTS

Dr.'s Asbury, Hahn, Gorson, Parry, Sladky, Koski, Lewis, Katzman, Brown, Cornblath, Lisek, McGovern, Saperstein, Burke

4.3.1 Does GBS cause high blood pressure?

Yes. The heartbeat, strength of beat, and tone of arteries are affected as the autonomic nervous system is affected by GBS. As GBS improves, so does stability of blood pressure.

4.3.2 Regarding Miller-Fisher Syndrome – Are headaches normal?

Yes. If you were going to have a headache without having Miller-Fisher, you will still have a headache with Miller-Fisher.

4.3.3 Is immediate treatment important for GBS/CIDP?

Yes. Very important. In GBS there is a 2 week window in which IVIG and Plasma exchange are most effective.

4.3.4 Is there a treatment for the numbness that comes with GBS?

No. Not treatable. Medications should be used to control the pain but not for numbness. Apparently some neuro's are using IVIG to control numbness which is a waste of good IVIG.

4.3.5 Vaccinations- Several questions came up:

Should people with GBS get flu vaccinations?
Travel vaccinations?
Children get chicken pox vaccine if GBS is in the family?
Should children going to college get meningitis shots?

Short answer: Yes, Yes, Yes, Yes

Flu vaccines are considered ok except for Swine flu vaccine. Vaccine should be of the killed or recombinant virus variety, not live variety. Nasal spray vaccine is of the live virus type and so should not be taken.

There is only very scant evidence that travel vaccines cause GBS or relapses in GBS.

Rabies, small pox, swine flu have been shown to cause GBS. The meningitis vaccine has not caused GBS. However, the rare patient could get GBS.

Dr. Koski said in their New England Journal of Medicine article that there is a 1:1,000,000 chance of getting GBS from a flu vaccine.

4.3.6 What treatments to use for GBS vs CIDP?

GBS is rapidly progressing while CIDP is slowly developing. So one must be more aggressive than the other but both must be aggressive. Also some evidence that some people who had GBS can later develop into CIDP.

4.3.7 Is it worth applying for disability from the government?

Yes. The government will help. However, the application must be filled out correctly. The Social Security Administration should help. Should call a SSA district office or go to ssa.gov. Caveat – Disability must be expected to last at least 12 months. This is a government entitlement program. Should call for an appointment at SSA. Should probably also get an attorney that specializes in disability claims to help fill out the form.

Medical records are required. This means that your doctor must write in your medical record that you are disabled. Must include examples. The doctors records are read by the judges who decide the claims. So the right information must be included in the doctors' notes. Bring this to the doctor's attention that you are considering filing a disability claim. This should clue them in to including the information that a judge will need to make the right decision.

4.3.8 Regarding CIDP- Will the residuals go away or you stable with residuals forever?

Maybe yes, maybe no. They think that you recover most of the way in the first 2 years after onset. However, may still continue to recover for several more years. Patients must realize that there may be long standing axonal nerve damage that can't be reversed. Damage usually due to prolonged lack of correct diagnosis and treatment.

4.3.9 Lack of correct diagnosis

If the paralysis worsens but the tests are not conclusive, the patient must insist on a vigorous exam by their doctor and then by a neurologist. This needs to be made clear the your primary care doctor.

4.3.10 Some people have problems with not enough muscle tissue covering their bones. When one bone lays across another, there is much bone pain.

In GBS and CIDP there is much muscle atrophy which means not much tissue to protect your bones. So you need more padding. However, you should note that there could be allodynia-type pain if there hasn't been much muscle and tissue atrophy.

4.3.11 Why can't hospitals get enough supplies of IVIG?

Companies are saying that the supply of IVIG, rationing of those supplies, and excessive use of IVIG where it isn't warranted have made IVIG unavailable in necessary quantities. However, the companies that are manufacturing IVIG have also closed several labs that produce IVIG in the hopes of further decreasing the supply and so raise the cost. Also because of Mad Cow disease in the UK, only blood donated in the US can be used to produce IVIG for use in the UK.

4.3.12 Is acupuncture effective?

There is no evidence that acupuncture helps GBS. It will help the pain but not the actual nerve problem.

4.3.13 If have GBS, can you get other immunological diseases/syndromes?

Yes. Some people are more susceptible to acquiring other immunological problems than others. There is a complex genetic relation between all the immune diseases. What is common is that they all attack the tissues of the body.

4.3.14 Is there research in other immunological diseases that will help GBS people?

Yes. There is a lot of research on MS (multiple schlerosis), which is similar to GBS in that the nerves are demyelinated but don't grow back, that will likely help GBS and vice versa.

4.3.15 Will stem cell transplants help GBS?

Probably. However, a connection has been shown between acquiring GBS and stem cell transplants. Some people have acquired CIDP from cell transplant grafts.

Bone marrow transplants have also caused CIDP and occasionally GBS as have some kidney transplants.

4.3.16 Will research on T-regulatory cells help GBS/CIDP?

Yes. In GBS the immune response is stimulated by something (many things). The T-cells are what turns off the immune response. There is some abnormality in CIDP that is regulated correctly. But, overall, this research is very hopeful in that it appears that the immune response can be modulated. May lead to more efficacious treatments.

4.3.17 Recovery rates – How/why are they different?

There is an apparent difference in the recovery rate depending on how GBS was acquired. i.e. viral vs bacterial. If the campha leflacta bacteria caused bad diarrhea, this can lead to bad cases of GBS. This is the prime cause of GBS in China. Apparently there is a 1 in 4000 chance of getting GBS in China due to this bacterium.

5.0 Saturday morning, November 4

5.1 What exactly are IVIG and Plasma Pheresis Treatments?

Presented by Dr. Mark Brown, Sarah White (RN for a PE company), Dave Drager (CEO for a IVIG distributor)

5.1.1 General treatments – Dr. Brown

The treatment for GBS involves protecting the patient from what is attacking them. So the correct diagnosis must be made and therapy started ASAP. Complications must be treated as they arise and plans must be made for a future after GBS.

The treatment for CIDP involves making the right diagnosis but deciding if therapy is necessary and to what degree to suppress the CIDP. Again, treat complications as they arise and also plan for a future with CIDP. Therapies for CIDP include steroids, plasmapheresis, and IVIG. Also azathioprine, cyclosporine, cytoxan, and cellcept – all meds from cancer treatments that are used to modulate the immune system.

5.1.2 Plasma Exchange – Sarah White

Basically this is a removal of a patient's plasma and, at the same time, replacing it with a similar fluid in which the disease mediator in the patient's plasma has been removed. This relieves or removes the symptoms in the patient. Note that this is done in conjunction with other therapies.

The procedure is relatively short, approximately 1.5 – 3 hours, depending on the size of the patient and how much plasma must be removed and replaced. The current procedure is much safer and healthier for the patient than in previous years. Previously, up to a quart of blood was removed at any one time with no replacement fluids input to the patient. This severely affected

the patient's blood pressure. Now, only a maximum of a cup of blood is removed at a time and it is immediately replaced as it comes out of the body. New sterile, disposable tubing is used for each patient. The patient should be able to watch the nurse set up the machine with new tubing.

The procedure involves setting up a vascular access and then separating the plasma into its parts. Vascular access could be through either the wrist by veripuncture, via a femoral artery catheter, a subclavian catheter (through the upper part of the chest), or through the jugular vein.

Once the blood is accessed, it is separated into its parts – Plasma, red blood cells, and platelets and lymphocytes. Anticoagulants are fed the patient to prevent clotting. These could be metabolized either quickly or slowly. Citrates are quick metabolizing anticoagulants. However they could cause hypocalcemia (not enough calcium). Slow acting anticoagulants include heparin – same as used after heart surgery. The replacement fluid is then input to the patient. This fluid contains some kind of colloid with proteins. Maybe 5% albumin and fresh frozen plasman.

The fluid balance between the patient and the plasma exchanger machine is always isovolemic – the fluid removed is always immediately replaced by an equal volume of new, good plasma.

The amount of blood plasma replaced is based on height, weight, sex. Usually 1 – 1.5 x plasma volume is replaced.

Treatment frequency for acute cases can be frequent – approximately 24-48 hours. For chronic cases, it is less frequent.

Side effects: hypocalcemia, hypertension, vasovagal (too low blood pressure), hyperventilation, and allergic reactions.

May take 4 treatments May take more. At some point, more treatments do not make a difference.

5.1.2 IVIG – Dave Drager

IVIG = IgG = IGIV = IVIg

Purified antibodies from the blood plasma are input to the patient. Blood consists of approximately 44% red blood cells, 55% plasma, and 1% white cells.

Antibodies in plasma are extracted and then separated. From each 1 liter of plasma, approximately 3 grams of IVIG are extracted. Viral safety methods continue to improve. No disease has been transmitted by IVIG since 1994. Cost ranges from ~$15,000 - $250,000 per person per year. By federal law, U.S. plasma must come from U.S. donated blood. As mentioned before, all British products come from the U.S.

IVIG is prepared in a pharmacy. The IVIG powder is put into a saline solution in a sterile environment.

Side effects: can be mild and/or temporary. Chills, aching, fever, and headaches are common. Side effects can't be eliminated but they can be modulated. Premedication, input of IVIG very slowly, good hydration, and use of different brands of IVIG help reduce side effects.

There have been some serious reactions to IVIG including anaphylaxis, thrombotic (blood clots), renal (kidney) failure, and aseptic meningitis (looks like an infection but isn't).

On the market are 7 different brands of IVIG. Similar but different properties. Access to all the brands may not be possible. The response time of treatments varies. Patients may feel better right away or may take several weeks. Multiple treatments may be necessary.

Medicare Part D will cover the IVIG but not the necessary accessories like the pump, supplies, or nurse to do the work.

Summary – IVIG is not for everyone. Best results will take time. IVIG is a therapy and not a cure.

6.0 Current Research on GBS and CIDP

6.1 Dr. Koski

Working on validation of the CIDP diagnosis. I.e. what is the best, most consistent, least damaging to the patient way to diagnose CIDP? They are trying to develop a consistent rule to make diagnoses. Currently using a panel of experts to decide CIDP in each case and to develop diagnostic criteria.

Current criteria include loss of motor function, sensory loss, muscle weakness, and symmetric presentation. CSF (spinal fluid) is used in some cases. Electrophysiology (EMG) and nerve biopsies also used. However, use of nerve biopsies rarely used and extraction of CSF also decreasing. Now mostly using clinical presentation, motor and sensory functions, loss of reflexes, and EMG to make the diagnosis. Developing something called INCAT criteria which confirms CIDP without having to extract CSF.

6.2 Dr. Cornblath

The Food and Drug Admin (FDA) has been approached to approve IVIG as a treatment for GBS/CIDP. However, the FDA did not approve the treatment because no controlled tests with placebos have been conducted. No one wants to knowingly (or unknowingly) not use IVIG to make an effective treatment for GBS and CIDP since treatment time lost can lead to permanent axonal nerve damage. This could affect insurance reimbursements in the near future.

However, a new study on GBS will start within a year, next April. It will look at drugs to improve functions of damaged nerves and to look at GBS residuals that appear stable after 1 year of recovery.

6.3 Dr. Robert Lisak

Doing Schwann cell research. Schwann cells are those cells which form the myelin sheaths around the axon nerves. They are doing a molecular approach to the study of GBS and CIDP. They are looking at growing and controlling myelinating genes which could lead to growing new Schwann cells. Dr. Hahn indicated that as soon as new Schwann cells have remyelinated the axonal nerve, recovery is apparent. Almost immediately.

6.4 John DeHart

The GBS foundation conducted a member/patient survey in 2004-2006.

83% of respondents had GBS, 14% had CIDP. Age ranges from 3 months to 98 years.

Correct diagnosis made: 57% in less than 1 week. 33% from 1 month to 6 months.

Treatments: 32% used steroids, 48% used plasma exchange, 46% used IVIG. Some used more than one treatment.

Patients had questions on cost of treatments, frequency of treatments, and what pain medications worked on what.

Looks like there are 3500 new cases of GBS each year.

6.5 Dr. Arthur Asbury

They are looking at new therapeutic approaches. Studying something called "complement" which is what actually attacks the nerves. The treatment concept is to find an agent that will interfere with the "complement" activity. Looking at complement receptors and how they stop complement activity. They have found some natural complement inhibitors that are currently used for treatment of hereditary anemia. Concept would be that if can supply complement inhibitors within the first 7 days of an attack, could severely impact amount of damage to the nerves. Idea would be to have a complement inhibitor IV in emergency rooms that could be applied right away.

There is also research being done on a new way to check spinal fluid protein levels in which the patient won't have to wait 2-3 weeks before something shows up. Those 2-3 weeks could be enough to cause permanent damage.

7.0 Saturday afternoon, November 4

Dr. Thomas McGovern, Psychologist at Arizona State and GBS survivor

Session was on emotional impacts of GBS/CIDP on patients. The doctor pushed the patient telling their story and the power that is released once the story is told.

Best comeback: Patient said they were told they hadn't prayed enough to get well. Another patient said they had prayed and that they had received enough understanding from God to not kill the person saying they hadn't prayed enough.

8.0 Sunday morning, November 5

Two sessions concurrently – One on patients in wheelchairs and the other on reimbursement. Attended the wheelchair session

Leader of session was a former British pilot who had contracted GBS in 1990. Still has major residuals. Legs and hand not functional. He wanted to make the point that wheelchairs are liberating versus limiting. If you didn't have a wheelchair you would be stuck in a bed. Especially the electrically powered wheelchairs. When you apply to the government for a wheelchair must emphasize upper body weakness.

Discussed so-called notion of wheelchair-accessibility or lack thereof. Mentioned that once accommodation is a swivel chair on the passenger side of a van. This would make transfers much easier. Liked gell wheelchair cushions because they mold to your body. When you go to a hotel you must specifically ask for a wheelchair accessible room and a roll-in shower.

This was generally a rap session on frustrations on being in a wheelchair.

9.0 Late Sunday morning, November 5

Rehabilitation – John Bargas, M.S. PT at some hospital in Phoenix

New orthotics are coming out that allow for more joint movement while still allowing support. PT's have found that joints remain too stiff in current braces, which will inhibit recovery later.

Strongly supported swimming pool exercises for both balance and strength training. Pool temperature should be around 94-98 degrees F

One patient who had had Miller-Fisher indicated that video games really helped him as an enjoyable eye exercise.