The 2012 Annual AGM and Conference of the Guillain-Barré Syndrome Support Group

The Conference did not officially start until Saturday, 21 April, at the Village Hotel in Bournemouth, but many of us took advantage of the good prices at the hotel and the opportunity of meeting old friends and new, by arriving on the Friday beforehand. After the local contacts met for training and discussion, the rest of the night was free. It was a time to see how others have progressed – both for the better and sometimes for the worse – to share a frustration and a laugh.

On Saturday morning members of the Support Group met to deal with the business of the group at the Annual General Meeting, then after tea and coffee all the delegates gathered for the Conference.

Caroline Morrice began with a report on the work of the group. The group's main function is support of patients and families, and in view of this in 2011 we had 363 referrals, most with GBS, some with Miller Fisher and CIDP, and some undiagnosed. Seventy local contacts dealt with patients through personal meetings, telephone calls and email. Our website offers much information, which many found helpful. The group is endeavouring to ensure that everything on our site is completely accessible to anyone who needs help. We had eighty calls a month on our Helpline, which is manned by twenty volunteers, and much is now automated to ensure that the time of the volunteers is used effectively. We now have a freephone for use by those in the Republic of Ireland. Our welfare fund received twenty-nine asking for help: seven were approved, two rejected and several were pulled by the requester. We have a new member of team – Gill – who is providing information for patients and is working with our local branches. The current branches are thriving, and we're hoping to have more – if you'd like more information about forming a branches, ask Gill.

With postage rates increasing, we're looking for our website to provide many people with information, particularly as the website will have our leaflets on the various diseases fully available. The website is also getting makeover, which we hope to have completed this year. We are focusing on technological advances in terms of getting information to patients and their families, and those in the medical fields. As always, awareness is of great importance. We are looking at mobile phone apps; our Facebook page has over 2,000 members; and we are looking at Twitter.

Leaflets will no longer be sent out in bulk but will be sent as needed to hold down costs. We now hold low stocks so that they can be easily updated as new information becomes available. Hospitals are no longer receiving our pens and post-it notes. We produced a calendar of people from group which was sent to many hospitals; according to a survey sent with the calendars, half of the hospitals had never heard of us. But they know us now!

Following the success of 2011 fund raising, our researchers were able to fund Claire White's control trial examining tailored home exercise programmes. Also funded is Hugh Willison's IGOS1000 programme – we are supporting the third year. The Craig Milne Fellowship has been formed for Craig, who died in Aberdeen; is has received £25,000. The family of a young woman who died have raised a lot of money for us. Fund raising in total for 2011 was £188,184.66, which included a legacy of £110,000

from someone who had never contacted the group. This has moved us to look more closely at legacies. Our rowers got almost to their destination before crashing on the rocks and having to be rescued, but they raised about £12,000. All sorts of things can be used to raise money. You don't have to climb Kilimanjaro: coffee mornings, bake sales, sponsored rambles – all of these work. An easy way of giving is through our JustTextGiving account – text GBS11 and insert the amount you wish to give. GiftAid is also a good way of giving– we can get an extra 20% claimed back on donation, and in 2011 gained some £8,000.

Since 2016 marks one hundredth anniversary of the discovery of GBS, we are endeavouring to raise more money in anticipation of that anniversary. Our patron Air Marshall Ian MacFayden helped significantly in this by hosting a gala dinner at Windsor Castle.

We are increasing our leaflet range, which the Medical Advisory Board are currently checking and updating. For our Christmas campaign this year, we are sending material to hospitals; and on the table is a form you can use to have material sent to your GP.

Dr Jane Pritchard said: I been on MAB for ten years, and my specialty is GBS and its variants. I find the peripheral nervous system fascinating. For a long time medical people didn't realise that the peripheral nerves could be damaged, but instead thought it was the brain and spine that were damaged. The first to note that peripheral nerves could be damaged was Robert Graves.

Landry was the first to describe five cases of GBS as a motor disorder causing flaccid muscles but in which there is no central nervous system damage. After that doctors began to note the pathology of the peripheral nerves. Landry examined soldiers that had nerve damage; he did lumbar punctures and noted raised protein in their spinal fluid.

In 1916 there was a polio outbreak, but Guillain and Barré noted a different pathology in some patients. People began to recognise a syndrome that included progressive weakness in multiple limbs, progression over more than four weeks – damage, plateau and recovery: GBS. GBS mimics other diseases, but needs to be treated differently. How can we be sure it's GBS? We listen to the patient's story, examine him, note what happens over time, run nerve conduction studies and blood tests, take lumbar puncture in which we look for normal white cell levels but raised protein.

There are variants in GBS – these have a similar process but not the same effect. Miller Fisher Syndrome was described in 1956; the symptoms were ataxia, an inability to move the eyes, and weakness. Other forms are pharyngeal.

Although there is an overlap between these variants and standard GBS, the classical syndrome can often be classed because of timing – GBS runs its course in four weeks, subacute four to eight weeks and CIDP more than eight weeks. Some clearly have one or the other, but there is also a recurrent GBS which can cause confusion. AIDP (what most people think of when they hear the term GBS) is an acute form; CIDP is chronic. There is also AMAN which isn't common here but is seen more often in China and

Japan. The differentiation between these helps in treating. Blood results help to classify the disorders. We see different antibodies in MFS and AIDP.

Nerves are like copper wires with insulation – demyelination damages the insulation while in the axonal type the nerve itself is damaged. Sometimes these overlap and those with demyelination also have axonal damage.

For treatment, most patients will have IVIg; plasma exchange also helps – this was the first type of treatment, but now everyone gets IVIg as first type of treatment. As we understand more about what causes and what drives GBS, we may be able to move to other more individually tailored treatments. Although we get good results, we still have unacceptably high death and damage rates.

Q: Is AMAN tied to race or location? A: we think there's an underlying genetic risk.

John Winer spoke on CIDP. I'm very grateful to be invited to speak; I've been to all but two of the twenty-eight conferences of the Support Group. Jane gave an overview of GBS; I'll be giving one on CIDP. CIDP isn't really any of the four words that make up its name – it's not always chronic, not necessarily really inflammatory, sometimes demyelinating rather than axonal, and not always a polyneuropathy.

In terms of chronic, the clinical distinction of CIDP is that it takes longer to come on – weeks or months. But sometimes it can come on fast or slow. And some are subacute forms which are more akin with GBS than CIDP.

Inflammatory: this is rare on biopsy in GBS where there are not as many inflammatory cells as in other inflammatory disorders. But I think T-cells are important in CIDP. Biopsy is less common in CIPD, and the information is not always useful; but it can rule out other things.

Demyelinating: this is not always seen in CIDP: sometimes it's axonal. It appears to be a macrophage mediated demyelination, with areas of demyelination perhaps guided by errant cells to myelin.

Polyneuropathy: some are very localised, and others start in one place and spread. There are multifocal variants such as MADSAM.

The diagnosis of CIDP is determined by a number of factors: clinical examination, nerve conduction studies, pathology, antibodies, lumbar puncture, cell functions. There are no single immunological tests that are useful.

We look for various clinical features: wasting, reflex changes, pattern of weakness (usually distal beginning although sometimes more global), sensory loss. The most useful texts are spinal fluid and nerve conduction study.

Nerve conduction studies are very useful in CIDP but not so much so in GBS. Sometimes there is conduction block. If you test higher in a part of the body, as in the arm or leg, sometimes you'll get a reduction in size of response; and the blockage is sometimes easily reversed using IVIg. NCS is very helpful in multifocal forms.

Scans in peripheral nerves disease are not terribly useful, but with more high resolution machines perhaps this will change.

Clues to pathogenesis are antibodies and gangliosides, paraproteins, and anti-TNF monoclonals. Clearly there's something in the blood – paraproteins may be a clue to what's going on.

Treatments: steroids are usually helpful in CIDP but not in GBS; IVIg is good in both as is plasma exchange; immunosuppression is also used.

In the rarer forms, MADSAM is asymmetrical and differs from MMN because of sensory involvement. MMN is purely motor, with conduction block, can be chronic, and mimics motor neurone. Weakness is due to conduction block, not necessarily much muscle wasting. Finger drop is seen because of slight weakness, very patchy, usually involving the fibres of only one nerve.

Anti-myelin Associated Glycoprotein Neuropathy: IgM has paraproteins, distal sensory loss, gait ataxia, and mild distal weakness. It is slowly progression, and doesn't respond to treatment. There are ongoing studies in how to treat these patients.

The diseases in classed under GBS overlap, with interaction between them; and sometimes they move from one to another.

With CIDP, the question is asked does it matter which type? It does only if treatment is going to be different. The purely motor forms respond better to IVIg over steroids.

Neurologists only see small number of CIDP patients, and they're not sure if one drug is better than another. We need to do a proper trial but they're very expensive and it's hard to find enough patients. We are trying to gather information from neurologists about how their patients have progressed to see how the drugs affect patients with different forms.

It's not all misery and depression: we're making little steps in understanding CIDP and treatments. The IGOS study, which Hugh Willison will discuss, is good but we need to do a similar study for CIDP.

Q: Is there a time scale when IVIg is ineffective? A: The earlier treatment of CIDP better. Doctors have to weigh whether it's better to treat or not. We don't always treat because side effects can be worse than current symptoms. The disease is not less responsive at onset, but sometimes we have to use very aggressive treatment.
Q: Do the standard treatments help pain? A: Sometimes it's better to give pain medication than to treat the disease itself.
Q: What about stem cell research? A: Hugh Willison has a good slide on this. I think stem cells will help in the future, but don't see them helping a great deal now.

Nicky Muir: I had GBS, and set out to raise money for the support group by abseilling down the Spinnaker Tower, despite suffering from heights.

I work in the tower doing photography and have always been very active. In 2009, when I was twenty years old, I took a summer break during my second year of

university. I was a hockey player, had never been ill, never been in hospital. I began feeling under the weather, and went to a walk-in where I was told to go home because I just had a cold with a little stiffness. Other doctors did nothing; they didn't know what was going on. I quickly became so ill that I was bedridden – exhausted all the time. Then one night I fell. My mother called our GP, but he was afraid of spreading swine flu so wouldn't come; Mum then had to carry me like a baby. On 9 July the GP finally came in; he was dressed in isolation clothes. He asked me to stand, and after a few moments told me I might have GBS. He called an ambulance which took me to hospital, where I was set up in an isolation room. I had two lumbar punctures; my arms and legs were unresponsive and I had facial problems. A five day course of IVIg, and I began to regain my legs after a few days.

I was soon able to get to the toilet by myself. Occupational Therapy sent me to rehab, where most patients were older than I. However, one patient was nineteen years old and had another similar disease. Daily physiotherapy soon enabled me to walk. There were still problems to overcome. A handheld fan got caught in my hair, and while I was in a wheelchair my brother rolled me into bush.

Despite it all, I made it back to university on time where I earned a first class honours degree despite being in a wheelchair and dealing with fatigue.

Now three years on I still have some fatigue and muscle weakness. But I decided I wanted to help raise money. Spinnaker Tower holding abseil day, so I decided to do it. My brother contacted the media and posted my plans on the internet. It is very rare to find people who have ever heard of GBS; we really need awareness so people will learn. Thus on 14 August 2011 I donned the equip and took the first step off the ledge around the tower. I learned to do tricks while abseilling but ended up with fat lip by hitting equipment. However, I raised £800 for support group.

Claire White is heading a project on exercise in rehabilitation which the support group is helping fund. She said: I'll be studying tailored home exercise versus simple advice for reducing disability, in GBS/CIDP/PDN – examining standard advice versus exercise tailored to patient in stable GBS and CIDP patients. This is the first controlled trial of exercise in UK. We need to employ a physiotherapist, obtain ethical approval, find participants – we'll be advertising soon, and may contact through the support group or doctors. GBS patients are a rare group of people; this conference is often the only time you meet with other patients.

An Australian study looked at multidisciplinary treatments. High intensity in which the patients had tailored exercise programmes involving three sessions a week for twelve weeks of physiotherapy and occupational therapy. Low intensity involved advice only, with thirty minutes of walking twice a week and some stretching. There was an overall impact on how they felt both physically and mentally. After twelve months the high intensity treatment showed improvement in 80% of patients; the improvements were small but noticeable in transfers, walking and constipation. In low intensity there was only an 8% improvement, with no overall real change in group. Improvement was based on the Functional Independence Measure, which examines eating, bathing, grooming, dressing, toilet, walking, stairs, transfers, with 1 being fully dependent to 7 being fully independent at normal speed with no aid. However, there are issues about the scale's consistency.

Some related findings were that there was an energy cost of in walking with GBS or CMT – it is harder to walk than normal even in patients with minimal impairment. One improvement was seen in the likelihood of falling: fall prevention exercises helped with confidence. Supervised exercise versus independent with advice from appropriate trainer showed more improvement.

One problem is that there is a lot of missing information in GBS/CIDP in terms of falls and physical activity. Other neuropathies note disability, falls more often, etc.; we need to determine in GBS/CIDP the frequently, consequences and circumstances of such falls. For physical activity we need to determine the frequency, type and intensity that would be best. We need to prepare questionnaires for these; and I might ask the members of the support group to provide information. Definition of falls includes dropping back into chair, etc.; we also need to determine how much the fear of falling limits activity. Department of Health has guidelines for type, amount, of physical activity needed. I would be glad to provide preliminary questionnaires to anyone would like to see them and provide information, views, etc.

Hugh Willison: It is wonderful to be here; these meetings remind me of what we're here to do. This, I'm afraid, is going to be a long and complicated presentation, because we need to get a feel for the research we're doing, its diversity, etc.

I'm so glad the group is dealing with CIDP: it as a much longer course than GBS although there are fewer patients. My favourite form is Multifocal Motor Neuropathy. We think of GBS as a disease of the developed world, but actually it's a global disease. There are probably 100,000 people with GBS. I received a box of nine hundred clinical samples from Dakar – nine hundred in India, and doubtless that's only a very small percentage. This is not just a Western disease but global. However, it's still incredibly rare: malaria affects five hundred million, so we're trying to help GBS patients in the context of much more major diseases. There is little research money and few people doing the research. Thus the GBSSG is very important for research. We want to move GBS from an important disease to an uncommon one – our goal to make it rarer, less debilitating, etc. Until recently the common cause of disability was polio, but it has moved from an important disease to an unimportant one.

We have a clear, broad (with gaps) grasp into what causes GBS. With campylobacter or infection, the body mounts an antibody, and that immune function starts to attack the nerves. When we understand more we can use it to help patients. What we know about campylobacter is that it's a food infection; the coating is a sugar, the shape of which is the same as that on the nerves, so the antibody attacks the myelin. Sugars determine which antibodies it makes. In MFS the antibody is to something in the eyes but not in other parts of the body; other antibodies in the arms and legs, etc., cause other forms. Antibodies in GBS bind on to things it thinks are infections.

AMAN in Chinese children is a seasonal epidemic, when it's warm and wet; Chinese children handle chickens and other poultry and the kids get infected. Often, because of lack of facilities, family members would have to vent the GBS-infected child with hand-held bag.

We need to undertake a number of trials, and if the trials work, what's left to do? Gene therapy, stem cell therapy – these may work at some point in the future, but at the moment, these are miles away in GBS. So don't go off to some other country for experimental therapy.

There are many things we don't know: why do some get GBS and some don't – supposing that the chicken at lunch was tainted, how many of the people in this room who had the chicken will get GBS? If we could determine who genetically are at risk, perhaps we would be able to vaccinate them. How can we get new diagnostic tests? Who will get a severe case and who mild – perhaps we wouldn't need to treat the latter? When does GBS stop – we know clinically when it does but we need to be able to tailor treatment? What about residual problems? Can we work out who might get it again?

This is a conundrum: if you lose a segment of myelin on each nerve you'd be completely paralysed, but it will regrow quickly. If you lose them all you'll also be paralysed but you have to do a lot more regrowth. And if it's close to the spine, you have a lot more distance to regrow. So how much and where the damage is, is critical; but the patients with either much or little demyelination look the same. So people working out various tests need to devise ways of determining these things.

My research subject is the antibodies and how they react with the sugars. We don't know if there is a sugar antibody although I believe there is and am trying to discover a blood test, etc., to diagnose GBS. How nice it would be to have a little stick – like with diabetes – to do a quick test. That's the dream.

One discovery made by a Japanese team is that the sugars wrap around each other, so you can have antibodies which when folded together produce complicated forms and can't be examined alone. The immune system looks to identify complicated shapes on the cell surface. A new machine has enabled us to see new antibodies that we didn't know existed, particularly in the combined sugars. The antibody often doesn't work against a single sugar but instead against the combined sugars, and the sugars can combine with various other sugars. Then when the material is found you have to study it, check it, try to understand it. Cluster analysis, in which computer can find correlations, etc., also need statisticians.

We have to think of GBS in a lot of different ways. Genetic risks, antibody markers in first presentation, treatment in acute phase: my work is in trying to find what causes GBBS and new treatments.

Internationalisation: IGOS funding is essential. We want to get together a big group of GBS patients and examine them down to the genetic level; we want a thousand people to study, especially as we approach the anniversary of GBS. Part of the funding for this comes from the GBS Support Group – you have supported researchers in our lab, for which we are grateful. A number of exciting things are happening.

Ask the Experts

Q: you used campylobacter; would the same apply to any other virus? A (Hugh Willison): yes, the principles the same. Viruses bud out of the cell and take on the coating of cell.

Q: when will subcutaneous be widely available? A (Michael Lunn): it's available but the problem is getting enough of it for people getting long-term IVIg for CIDP. Companies are now making it. We have to make sure it's safe, particularly for home use.
Q: how safe is IVIg? A (Michael Lunn): the risk of CJD can't be tested for in blood at the moment so theoretically it can get into IVIg; therefore our IVIg comes from USA and Spain. But never seen a case in which it is spread by IVIg.
Q: should be have vaccinations? A (Hugh Willison): you've all been vaccinated against polio, smallpox – thumbs up for vaccination. Of course there can be complications with vaccination, and a few have triggered GBS such as the swine flu, but the incidence is very small. If you think about GBS hitting within a month of vaccination, then one in twelve could be thought to cause the GBS, but if you're going somewhere where a disease is endemic, take the vaccination. But don't take vaccinations unnecessarily. (Richard Hughes): absolutely right; vaccination is safe for GBS, except old style rabies vaccination and the 1976 USA swine flu vaccination. These are now checked very carefully and are safe. For those who may have got GBS after vaccination, probably shouldn't have that vaccination again.
Q: thought my CIDP caused by campylobacter; does this mean I have MADSAM? A (John Winer): campylobacter doesn't determine what pattern of disease you have; antibodies probably would; campylobacter doesn't cause a particular type of CIDP.
Q: what is the role of antiviral? A (John Winer): the disease is caused by an immune response. In a small number of people perhaps the response could be stopped by antibiotics: if the patient is still secreting campy, perhaps, but otherwise no. (Richard Hughes): some in one group had been treated for campylobacter and still got GBS.
Q: is there a connection between arthritis and CIDP? A (John Winer): Lyme disease arthritis does appear at least very similar to CIDP. Lyme is an infection from a tick, causing facial weakness, neuropathy with arthritis – but usually no connection.
Q: what to do with fatigue with MFS? A (Claire White): very common with MFS. Two types of fatigue: weak muscles and more generalised fatigue not related to activity. No medical treatments have been found to be effective in fatigue, but graded exercise may help, spread activity out. Fatigue is very prevalent.
Q: any facial exercises that will help? A (Claire White): not much that I'm aware of but they do work with other diseases.
Q: why do my legs vibrate with CIDP? A (Claire White): CIDP gives odd sensations.
Q: are there racial differences in these diseases? A (Jane Pritchard): they are seen around the world in all types of people.
Q: does GBS progress to CIDP? A (Jane Pritchard): GBS is acute but CIDP chronic. GBS doesn't normally progress to CIDP, but sometimes the diagnosis changes. Doesn't happen often, so normal GBS sufferer shouldn't worry.
Q: how often will CIDP come back or worsen? A (Jane Pritchard): in some it's a recurrent disease – the majority – but it can burn out in the space of a few years – about a third do.
Q: what about SIDP? Doctors say the disease isn't active? A (Jane Pritchard): no blood test will tell so it would require clinical examination.
Q: my GP diagnosed me with CIDP after four month mystery. A (Richard Hughes): I think it would be very tough for a GP to diagnose.
Q: IVIg helped but I had severe pain for a couple of weeks until gabapentin. A (Richard Hughes): as the weakness improves the pain comes on – not a correlation between these, since the pain from different nerves than motor nerves. Sometimes legs go numb and as feeling comes back they hurt, like cold hands hurt as they warm

up. May be sign of recovery. Pain normally eventually improves or you learn to live with it.
Q: I've had MFS three times, fully recovered. A (Michael Lunn): we'd be happy to reassess you. Sometimes with recurrent needs a good reassessment.
Q: what of people having same symptoms as when they had GBS. A (Michael Lunn): there is a recurrent GBS which comes back later, and sometimes it's actually CIDP. Sometimes when nerves damaged, they're scarred, and sensory symptoms can come with other illnesses. But real recurrence is rare.
Q: I had MFS but didn't have treatment. A (Michael Lunn): why expose someone to treatment which can be problematic when you might get better anyway? IVIg only speeds up recovery but doesn't help with cure.
Q: still have MFS in my notes, and GP says I have inflammatory arthritis. A (Michael Lunn): most GPs have never seen GBS, CIDP or MFS, so don't really know about it.
Q: is there a treatment to help with my not being able to smile with MFS. A (Michael Lunn): nothing that will help with damage, but exercises.
Q: does GBS make blood sticky? A (John Winer): anticoagulants used for those confined to bed do, but there may also be slight stickiness; IVIg increases stickiness.
Q: DVLA and driving – with GBS listed as my disease do the DVLA need to be notified? Insurance? A (John Winer): if your GBS affects your driving, you need to contact the DVLA, and if you don't and have accident your insurance would be affected. I recommend some to go to a driving centre and have driving assessment. (Michael Lunn): required by law to tell DVLA and we're happy to send assessment. If GP sends you to mobility centre, there will be no cost. (Richard Hughes): have to tell DVLA, at the mobile centre you'll see an assessor and a mechanic.
Q: with slow onset CIDP followed by relapse and flu jab, where is most nerve damage done? A (John Winer): slow disease damage will accumulate. Rapid disease will reverse often quite quickly.

Michael Lunn: I'd like to speak today on Cochrane Evidence and IGOS1000. This is the 20th anniversary of the Cochrane Collaboration. Archie Cochrane is the father of evidence-based medicine, which he defined in 1972. He noted that we need trials to determine best treatment, etc.; high quality information can be used to help determine treatments. In 1992 the Cochrane Centre opened in Oxford, with its formal launch the next year; in 1998 the Neuromuscular Group was formed by Richard Hughes; in 2002 the Cochrane Library was opened to everyone; and in 2012 we passed 5,000 reviews in database. One key was to have international input.

The Cochrane Library includes systematic reviews: defined research questions, search strategy, type of study, peer review. It is updated every two years. We have some twenty-seven thousand people writing reviews. In this we use meta-analysis, combining trial reviews when information is similar. Richard Hughes is the mover in the Cochrane Collaboration.

The reviews attempt to bring together information, measuring when things work and don't. One problem that this attempts to overcome is that people want their preconceived notions to be borne out; pre-definition and strategy has to rule this out. Drug companies fund or do trials but these are sometimes biased. We need to run trials with enough patients, with everyone blinded.

The Library can be accessed at www.thecochranelibrary.org – it's not available in the USA for some unknown reason. There are one hundred and three reviews of neuromuscular disease. We aim to encourage people to do further work, since there's actually little work done in many areas. We're looking for clear evidence.

Richard Hughes did a random control test for steroids in GBS: after four weeks patients did worse than those who had no treatment. Plasma exchange helps and IVIg works as well as plasma exchange. A Chinese article shows there may be help with other medications. The Chinese also looked at acupuncture, but found it doesn't work. The research also showed that CIDP responded to treatment in the short term, but that steroids may make motor CIDP worse.

The GBSSG may provide funding for overviews of reviews, and we're trying to make them more user friendly for nonexperts.

IGOS 1000 study: John Winer, Hugh Willison and Michael Lunn are kicking off the IGOS study in next fortnight. This is a prospective study on the clinical and biological predicators of the disease course and outcome in GBS – it aims to determine, when you go to hospital the first time, how you're going to do in the long term. We know GBS is a spectrum of autoimmune diseases of the peripheral nerves, and the commonest cause of acute paralysis; it is probably post infection, and 10-20% remain disabled. We need biomarkers – things we can measure when you first go to hospital. Is it myelin, axonal, genetic; can it be measured in blood or spinal fluid, or thought age? We can assess axonal damage through emg, and other problems through lumbar puncture – we can measure neurofilament levels in spinal fluid, which is high in worst cases. Are there genetic factors or susceptibility? How does IVIg works in the body: some people react differently to IVIg based on levels in blood; we need to tailor it to individual. We need to design prognostic models, to identify those who will do poorly and optimise treatment. Reviews can be used to encourage some patients. We need to identify clinical and biological determinants for disease and recovery, to develop new prognosis models. We're looking to recruit one thousand patients worldwide, two hundred in the UK. We look to have a website for information on patients, blood samples, etc. We hope to have thirteen research centres in UK.

Q: why the differences between those who get IVIg? A (Michael Lunn): IVIg costs £5,600 per dose. Department of Health IVIg panel had to be persuaded government that we're not wasting IVIg, so the supply is safe for now with government funding. How fast individual metabolise determines something of dosage – probably those who metabolise slowly don't need it as often, others faster.
Q: how much of the research will be about CIDP? A (Michael Lunn): we want to set up similar to IGOS for CIDP; it has taken four years to set up current IGOS.
Q: will IGOS be of use for CIDP? A (Michael Lunn): possibly, but we want to set up other for CIDP.

Saturday night
The conference ended and we retired to our rooms to prepare for the banquet. It was a delicious meal, with lots of good conversation round the tables, and most of us went to bed late. Sunday morning dawned, and with it hugs good-bye and promises to see each other next year.