Myasthenia Gravis
Friends & Support Group WA (Inc)

A Handbook

For

MYASTHENICS

lotterywest supported

This booklet has been prepared and is

Presented by
The Myasthenia Gravis
Friends & Support Group WA (Inc)

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Whilst this handbook provides helpful information about Myasthenia Gravis it is not intended, nor does it constitute, medical or other professional advice. Diagnosis and advice on medical care or other assessment should be sought from a General Practitioner or a Neurologist or other suitably qualified practitioner.

Foreword

Considerable advances in the treatment of myasthenia gravis have been made in the recent years due to an improved understanding of the disease. Once you have been diagnosed, it is important to stay under regular medical supervision and to work closely with your general practitioner and specialist.

You and your family should request copies of consultation letters and reports, to keep your own records to show others if necessary, and get a Medic Alert bracelet in case of an emergency.

Modern treatment has a good chance of controlling or eliminating the effects of the disease over time.

Support from the Myasthenia Gravis Friends and studying this Handbook are also important elements in understanding and living successfully with myasthenia gravis.

Dr Rob Edis FRACP
Neurologist

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Why This Book Has Been Written

Myasthenia Gravis (MG) is a chronic auto immune disorder which results in muscle weakness and fatigue.

It is quite rare in our society and little known in the general community outside the small group of people who have been positively diagnosed. No current figures are available to tell exactly how many have MG in WA. Using recent estimated world wide figures of the prevalence of MG being 11-15 per 100,000 it could be assumed that there are approximately 250 sufferers in this state.

Of this number fifty have been in contact with a small support group allowing sufferers and family members to share their experiences and to learn much from each other.

Some of the group were diagnosed many years ago and some have even indicated that until they learned of this support group, felt they were the only person in WA with MG.

A common issue for all sufferers related to initially having no or limited information about Myasthenia Gravis followed by then having too much technical information.

The group, *Myasthenia Gravis Friends and Support Group of WA (Inc)* set out to review the literature available from within Australia as well as the UK and the USA. They soon became aware that Myasthenia Gravis, although technically the same in the way it manifests itself in the body, nevertheless produced quite different outcomes to individual people.

The group determined to produce this Handbook for Myasthenics - suitable to both newly diagnosed, their families and carers, and those who may have known for some years that they have MG but have had little opportunity to talk to others similarly affected.

Throughout this booklet the abbreviation MG is used for convenience but as will be shown in subsequent chapters, there are varying forms of the disease which was first described by Dr Willis in the U.K. in 1672.

This booklet is not presented as a medical textbook although the facts within have been subject to careful review by two Neurologists very familiar with the disease. Rather it is a sharing of common and uncommon experiences of some people in WA. In their own words they will describe how their symptoms vary, and some of the difficulties they have faced and overcome.

Definition

What is this little known disorder which can strike fear into the hearts of the newly diagnosed and their families? It is not until you discover people who have been diagnosed and have learned to cope and lead fulfilling lives that you gain reassurance.

MG is a disorder which presents as acute attacks or chronic weakness of the voluntary muscles which increases with use and decreases with rest.

The muscles which we use all the time such as those which keep our eyelids open are often, but not always the first to indicate that something is wrong. Troubles with our facial muscles, the ones we use to smile, speak, or swallow are also among the first to reveal that there is a problem. Other symptoms which may, or may not, be present include:

- Blurred or double vision,
- Slurred or nasal speech,
- Difficulty in holding the head up,
- Tiring easily just from the act of chewing and swallowing,
- Unstable gait,
- Weak or fatiguing arms and legs.

Although the term Myasthenia Gravis (MG) is used most commonly in referring to the disorder there are some variations of the condition

Ocular Myasthenia, where the condition is only seen in the eye muscles.

Lambert Eaton Myasthenic Syndrome (LEMS) This is a different problem at the neuromuscular junction producing weakness without fatigue-ability. Unlike general MG if anything some people with LEMS improve with exercise.

Congenital Myasthenia (CM) is a condition with which one is born. The onset of symptoms may appear shortly after birth or may not show for some years.

CM is caused by a faulty gene affecting the nerve to muscle signalling. Because this is not an autoimmune problem, none of the immune treatments are suitable. Non-immunological treatments such as Mestinon may still however be useful in alleviating weakness.

In other cases babies born to Myasthenic mothers may, for a brief period of about four weeks, exhibit transient myasthenic symptoms. They fully recover if properly supported during this period while the mother's antibodies clear from the baby's blood.

As we have referred to differences in the way the nerves activate the muscles it is useful to describe, briefly, how our muscles work. MG is generally only found in the voluntary muscles. That is the ones which we control. For a voluntary muscle to contract, the brain transmits a signal to a specific nerve. The nerve endings are very close to the muscles but do not actually touch, the space between them is referred to as the neuromuscular junction. The nerve releases a chemical known as Acetylcholine (Ach) which travels across the gap to sites on the muscles known as AC receptors (AchR) This in turn activates the receptors to generate an electrical impulse along the muscle causing the muscle to

contract. The malfunctioning of these receptors is at the root of the common form of autoimmune MG.

MG starts most commonly in young women, but also occurs in men and can start at any age. As patients often have the condition for many years and grow older with it, there are actually more older women than younger women with the condition at any time. In other words it is no respecter of race, age or gender.

We cannot describe MG without reference to the Thymus Gland (not to be confused with the Thyroid Gland). The Thymus is a small gland located within the chest cavity behind the breast bone. It is part of the body's immune system programming groups of cells which produce antibodies to attack foreign invaders in the body. In the case of MG, the rogue antibodies which are against the acetylcholine receptors (AchR)at the nerve-muscle junction are confused and start attacking the body they are meant to protect. (autoimmune disease)

A few general observations here will help to reinforce the fact that MG is no longer regarded as a fatal disease. Incurable yes, but with the present treatment available it is quite manageable. It is not contagious, nor is the acquired form with AChR antibodies considered to be hereditary. It is known however that other siblings in a family have an increased risk of being diagnosed with MG. Given the low incidence of the disease the chances of siblings experiencing MG is still uncommon.

The maximum level of progression of the disease is usually known in the first few years after diagnosis. If it remains

limited to ocular muscles (Ocular MG) for 2 years it is unlikely to spread to other muscles.

Your MG may be with you for your life, as remission occurs in only about 25% without treatment. Medical treatment to control the disease is often required to be taken life-long. (Just as in other conditions such as epilepsy or diabetes mellitus)

In the next chapter we will refer to the various treatments available depending upon the circumstances surrounding your diagnosis and your continuing symptoms.

Diagnosis

MG affects people initially in a variety of ways so that no two cases have exactly the same symptoms. The onset can be sudden, or more commonly, it starts gradually and develops slowly or intermittently. This fact makes it easily missed or only diagnosed after some time. Likewise, because the symptoms are similar to many other situations in which the limbs feel heavy and weak, it can be confused with other ailments.

Positive diagnosis however is now available, even though this is normally only achieved over a period of time and in some cases, requires confirmation by more than one test.

A preliminary test which suggests further investigation is the test for muscle strength and fatigue-ability. The limbs are tested for muscle strength after which the patient is asked to make multiple repetitive movements and then strength again tested to see if unusual muscle fatigue and weakness occurs.

We all know well how stupid one feels flapping our elbows up and down when this test is frequently carried out after diagnosis. However a treating neurologist suggests that this is a kinder method of assessing the degree of weakness present and hence the need for treatment than repetitive stimulation testing on every visit!.

If MG is suspected a blood test is carried out to try and establish the presence of AChR antibodies if they are present this is indicative of the common form of autoimmune MG.

On other occasions, where the problem is not clearly confirmed, your practitioner may determine that further tests are necessary. One such test is the Edrophonium (Tensilon) Test. This test is carried out under controlled conditions requiring the injection of a myasthenia treatment similar to Mestinon in the veins which has the effect of greatly stimulating the muscle for a brief period. Muscle strength, eyelid position, or eye movements are assessed before and after injection, and where a marked improvement is obvious, the result is considered positive. As Tensilon is only useful for testing for myasthenia it is no longer supported in the market by the manufacturer, so additional government paper work is required for this test.

Another test is called repetitive stimulation, where the nerve to the muscle is repetitively stimulated (shocked); this can help determine whether there is neuromuscular junction fatiguability present as the size of the muscle response gets smaller with successive stimulations.

In some cases an extra test called SFEMG (single fibre electro myography) is needed, where a tiny needle is placed within a number of individual muscle units (of which there are hundreds or thousands in each muscle) and the firing of each muscle unit is observed.

In about 20% of patients, who present with all the symptoms of generalised MG, tests show that they do not have AChR Antibodies. It has been recently discovered that about half of these patients have antibodies to another protein present at the neuromuscular junction, called Muscle Specific Tyrosine Kinase (MuSK). The role of the MuSK protein is still being clarified.

Treatment

We will outline in a later chapter how, just as the method of making a positive diagnosis of MG has developed, so also has the treatment. The important point being that in the last seventy years, MG has gone from being an almost unknown fatal disease to the current situation in which the symptoms can be controlled so that today we have ageing Myasthenics in our community.

There are different forms of treatment for MG, although often they are used together.

Non-immunological symptoms treatment accepts the immunological abnormality as a fact of life and compensates for its affects at the neuro-muscular junction by allowing the released ACh to work longer. Pyridostigmine (Mestinon), the anti-cholinesterase drug most commonly used, does nothing to cure MG or attack the rogue antibodies but assists the patient in coping by improving muscle strength temporarily with each dose. Other drugs in the same category are Ephedrine and Spironolactone.

Mestinon comes in 60 mg and 10 mg tablets, as well as a slow release 180 mg dose. Group members have come to realise that it is important to discuss the dosage with the Doctor, particularly if they are experiencing stomach cramps, diarrhoea, flickering eye lids and body cramps (indicating a relative overdosing). Mestinon assists the patient to a level where optimum strength is restored, but attempts to raise that level of strength by increasing the dose may have the reverse effect and actually increase the weakness. Where the dose is too high a Cholinergic Crisis may occur which is the result of

an overdose of the medication. The dose of Mestinon that works for a particular person may be very different to that required for someone else and a degree of trial and error may be required to get the best dose.

It is important to realise that, because Mestinon does not treat the underlying autoantibodies, immoderate or severe MG it is not enough and even at the optimum dose it may not relieve the symptoms or fully prevent weakness.

Immunological Disease Treatment concentrates on reducing the antibodies which cause such destruction to the muscle receptor's AChR. Certain drugs suppress the body's immune system (immunosuppressants) and are helpful in treating the cause of MG.

Corticosteroids (Prednisolone) (*not to be confused with anabolic steroids taken by some athletes*) are known to reliably improve MG after a delay of two to six weeks. It is generally known that such drugs have side effects in chronic use (see later section). By suppressing immunity generally, they raise the risk of infection but by taking reasonable steps to avoid being in certain environments this risk can be minimised. It is also of great importance that a patient who is on steroids should advise the surgeon and anaesthetist when facing surgery.

Dentists, optometrists, podiatrists and any health professionals with whom you have contact, should also be made aware that you suffer from MG and the medication you have been prescribed.

Patients being treated with steroids need to be aware that beneficial effects may take some time to be felt as the body needs to adjust.

Azathioprine (Imuran) suppresses anti–body production. The point to note about Imuran is that it can take up to a year to be effective. It may be used by itself or in conjunction with Mestinon and or steroids.

Mycophenylate another recently introduced immuno-suppressant drug with a similar action to Imuran is currently finding its place in the treatment of MG.

Plasmapheresis (Plasma Exchange) is a procedure where blood is separated into cells and plasma (which contains the antibodies). The plasma is then replaced with healthy plasma. This is not a long term treatment but is said to "buy time" while other treatments are becoming effective. It is very effective in patients with more severe MG. Strength usually improves after three days and lasts for about four weeks. This is a particularly useful procedure to strengthen the patient prior to surgery such as Thymectomy, or while patients are slowly responding to tablet immune suppression. It requires large intravenous lines which are unpleasant

IV Ig intravenous immunoglobulin. (Intragam) A procedure of injecting slowly into a vein the pooled antibody protein fraction from normal blood.

This probably works in MG by providing enough antibody in general that the body makes less of its own, including the undesirable AChR. Like Plasmapheresis it needs to be repeated at least every few weeks and is in short supply as it relies on thousands of blood donors. However, unlike

plasma exchange it only requires a little intravenous line to be given.

Thymectomy

The Thymus Gland is seen as the producer of these rogue antibodies. It has also been found that tumours (Thymomas) may develop in the Thymus Gland in about 10% of patients with MG. In the majority of cases these tumours are benign, however in a small number of cases they have been found to be malignant (Spread and invade local tissues).

Removal of the Thymus Gland (Thymectomy) for reasons other than MG was practised long before it was noticed that this surgery had a marked affect on about 15% of MG patients. On occasions MG patients (under 45 years of age), without Thymomas, but are antibody (AChR) positive may be recommended to undergo a Thymectomy. Some show great improvement, most have some benefit, but some do not improve post surgery.

Because we do not wish to enter the enormously complex world of medical research, we simply make some observations from the responses of MG sufferers within the group who have had the surgery. The surgery is major with the healing period being slow due to the need for the MG still to be treated using Steroids, and if there is to be a reduction of MG symptoms this takes place over a very long period (up to five years). So patients should not be disheartened if they do not see a dramatic change overnight.

In this booklet it is only possible to speak generally of these treatments and it will obviously be important to discuss your individual situation with your Doctor. Hopefully it does give you some idea of the range of treatments available and the

reasons they may be being used. Remember, this booklet is produced by a group of people many of whom can say *"Been there done that"* and are happy to share their experience.

Some neurologists consider it is an important benefit to join the MG Friends Support Group and attend their gatherings.

Side Effects of some Drugs Used in the Treatment of MG

Most medicines and drugs have some unwanted side effects. This includes those used to treat MG, where the side effects will vary from patient to patient and with the level and frequency of the dose.

Together with your doctor you must weigh up the benefits of the treatment against the risk of unwanted effects particularly when to cease the treatment could have serious consequences. **Generally the benefits of the treatment outweigh the risks.**

Let's look at the possible side effects of three of the main groups of drugs commonly used to treat MG.

Pyridostigmine (Mestinon) and **Neostigmine** (Prostigmin)
These drugs slow down the elimination of Ach helping to build its concentration on the muscle receptors. Ach plays an important role in many parts of the body in addition to the muscle receptors. The medication is not selective and thus can cause a number of unwanted effects. For instance it may affect the muscle in the wall of the bladder and bowel resulting in frequency of passing urine, even causing incontinence and stomach discomfort and diarrhoea. (may be controlled by reducing the dose or by adding medication to control the bowel) The pupils may be affected making it difficult to focus.

Some patients experience increase in saliva and bronchial mucus. Another side effect may be muscle cramps, muscle twitching (including around the eyes). **Generally the effects**

are minor in relation to the benefits. It should be noted, however, that if the dose is too high it results in **actual** muscle weakness. In other words **more** is not always better.

Azathioprine (Imuran)
This drug depresses the immune system causing the reduction of the antibodies responsible for MG. The drug is extremely useful as it allows smaller doses of other drugs, steroids in particular, to be prescribed. Unfortunately, in addition to reducing the antibodies, Imuran also reduces the formation of new blood cells. This effect must be monitored by regular blood tests.

Liver function may also be affected by Imuran but the damage is reversible on stopping the treatment, or reducing the dose.

Imuran is tolerated, by 90% of people, without serious adverse affects however the patient should contact their doctor immediately and stop the medication if any of the following warning signs occur:

- Nausea and vomiting
- Fever or chills (flu symptoms)
- Cough or shortness of breath
- Upset stomach including diarrhoea
- Skin rash
- Darkening of the skin
- Cold sores in the mouth
- Blood in the urine or stool
- Yellowing of the eyes and skin.

Steroids (Corticosteroids)

These are not to be confused with anabolic steroids taken by some athletes and roundly condemned.

Steroids occur naturally in the body and are part of the defence system. Those used in medicine are usually much stronger than those occurring naturally. The most commonly used drug in this group is Prednisilone, but others used are Prednisone, Hydrocortisone and Dexamethasone.

The most common undesirable side effect in the patients view is the increase in fat and fluid on the face and trunk. However this is not considered dangerous to health, and is dose related.

Steroids in chronic use can cause a weakening of the bones in the spine and the pelvis. This is worse for those already at risk, being women past child bearing age and older adults. Calcium supplements are usually suggested.

Steroids also make the body less capable of dealing with glucose and other sugars which may induce mild diabetes or aggravate already present diabetes.

Other possible side effects include:

- Salt and water retention causing puffiness or swelling
- Mood changes and insomnia
- Increase in appetite / weight gain
- Decreased resistance to infection
- High blood pressure
- Increased sweating / night sweats
- Cataracts

This list of possible side effects of steroids can be quite scary but the Doctor will be aware of this and alternate day dosing reduces the severity of unwanted effects.

For most of us, the benefits of steroid treatment far outweigh the risks.

Drugs not to have with Myasthenia Gravis

There are two classes of drugs that make Myasthenia Gravis dramatically worse while any amount of that drug remains in the body. While this does not mean that these medications must never be used, extreme care and precautions are necessary if so doing.

Note that that the effects are not persistent in the long term.

These are :

Aminoglycoside antibiotics. Fortunately these are for injection use only so it is unlikely you would receive them except in hospital. However, one of these gentamicin is commonly used in hospitals for urinary tract and abdominal (that is appendicitis) infections.

Neuromuscular blocking agents. These are for anaesthetic use and likewise injection only.
ALWAYS make sure your anaesthetist is aware you have Myasthenia Gravis. Note that this does not mean that you cannot have an anaesthetic, just that the technique may need modification.

Please ask the hospital to mark your file as allergic to these drugs.

There are a number of drugs where the product information suggests caution in taking these with Myasthenia Gravis. In many cases this is only a relative warning and the two may be able to be combined: as always, you should check with

your doctor before starting anything with such a warning. These include several types of blood pressure medications, most sleeping tablets, and various other drugs.

Little Bits of History That May Interest You

We think that MG has always been with us but one of the earliest descriptions was recorded in 1672 by Dr Thomas Willis, a well known London physician, who wrote: *"…in the mornings, they are able to walk firmly…. Or to take up any heavy thing. Before noon, the stock of the spirits being spent, which had flowed into the muscles, they are scarce able to move hand or foot….A prudent and honest woman has this spurious palsie since many years, not only in her members (limbs)…. But, after she has spoke long, hastily or eagerly, she becomes as mute as a fish; nor can she recover the use of her voice under an hour or two…"*
Does this sound familiar to you?

From that time until the 19th century, progress was slow, apart from the fact that in Germany around 1860 the distinction between Motor Neurone Disease (MND) and MG was made. Unlike MND, MG was not regarded as remorselessly progressive but that the disease was fatigable. That is the harder one tried the weaker they became.

In France, in 1850, research on the South American arrow poison Curare showed that it created conditions similar to MG in that it blocked the activation of the muscles. When an antidote for Curare was developed from the Calabar bean poison the way was paved for the production of Physostigmine. The modern Neuromuscular Blocking Agents (see above in Drugs Not to Have with Myasthenia) are derivatives of curare. An early test for myasthenia used an injection of a very small amount of curare that had no

effect on normal individuals but made myasthenics markedly worse, although hopefully they survived the test.

MG was first named Myasthenia Gravis or Grave Muscle Weakness about 1895. (prior to modern understanding of the disorder and treatments)

Then in 1934 a young hospital doctor in England, Mary Walker, thought that patients with myasthenia looked like patients who had been given too much curare. She tried the antidote for curare, pyridostigmine, on a patient with myasthenia who had a quick and remarkable improvement. She was subsequently involved in developing the oral equivalent: Mestinon.

Both Curare and Quinine, which is also produced from the bark of the rain forest trees, were used in diagnosis because they both exacerbated the MG symptoms. As you can imagine, this was very dangerous.

In America improved anaesthetics made the removal of the Thymus Gland possible when tumours had been identified. It was discovered that some patients with MG and Thymoma greatly improved following this surgery. This led to the practice of removing the Thymus Gland for MG patients even when no tumour was present resulting in significant improvement in the condition of some.

In 1973 scientists experimenting with snake venom to purify the ACh Receptors in electric fish were able to prove conclusively that MG could be caused by antibodies. This became a valuable diagnostic tool which is used throughout the world today.

The understanding of the role of the antibodies led to the use of plasma exchange (plasmapheresis), where about four litres of the patient's blood is drained a litre at a time and placed in a centrifuge and the plasma, where the antibodies are found, removed. The plasma is replaced by fresh plasma, leading to a lowering of the antibody level. This procedure is particularly useful where respiratory crisis situations exist, until other treatments become effective or where surgery is planned on a very weak patient. It is only effective for up to approximately four weeks.

A recent feature in the history of MG has been the increased incidence of older women being diagnosed. This has been observed in WA and in other parts of Australia, and studies in the Scandinavian Countries have confirmed this to be a world wide modern day phenomena. No definite reason has yet been advanced except that people are living longer.

Not all of the women, when tested, prove antibody positive and in most cases the Thymus gland is no longer active. It has been established that in a number of such patients a protein presence has been found at the neuromuscular junctions which has been given the name Muscle Specific Tyrosine Kinase (MuSK).

Sharing MG Experiences

This chapter is given over completely to the personal experiences of some members of the group.

Group members tell us of symptoms they experienced before diagnosis.

The contributors range from those who are still at school, wives who are at home with husbands working, men and women who are still able to work, be it sometimes part time, and those who have retired.

From these brief comments you will quickly appreciate just how difficult it is to diagnose MG as so many of the symptoms are non-specific.

Female 31-40 years
I was at a Conference Dinner and was laughing at something when I became aware that my face wasn't responding – later I discovered my arms were weak and I had double vision.

Female 14 years
Diagnosed at age three when in hospital with breathing difficulties. I was unable to swallow saliva. I also had droopy eyelids and muscle weakness in arms and legs.

Male 71+ years
At 75 years I developed difficulties in swallowing/ choking and muscle weakness in legs and arms. GP referred me to Neurologist who confirmed MG.

Female 51-60 years

I was diagnosed at 47 years after two operations when I had difficulty coming out of anaesthetic. I then experienced swallowing and speech difficulties, and muscle weakness.

Male 41-50 years
At 48 years of age I developed double and blurred vision and droopy eyelids. Underwent CT scans and eventually was referred to a Neurologist who diagnosed MG. Slurred speech and weakness in legs has occurred since I underwent a Thymectomy.

Female 21-30 years
I had been feeling very lethargic and heavy. My eyelids became droopy. The local GP on seeing me said immediately "This may be MG". I was referred to a Neurologist and have undergone a thymectomy.

Female 75 years
During a Busselton Health Survey (many years before) I was contacted by the Drs and told my blood test was abnormal. They told me to seek medical advice if I had any unusual sickness or symptoms. I later had blurred vision, droopy eyelids, difficulty swallowing and speaking and my gait was unstable. I had pain in neck muscles and at that time (I was 56 years old) doctors conducted all manner of tests to prove the Mysathenia because it was so rare a disease.

Male 71+ years
At 49 years of age when I developed double vision, droopy eyelids, difficulty swallowing and speaking and unstable gait with weakness in arm and leg muscles. A friend, a nurse, suggested I see a Doctor. The Doctor said I was depressed. Sought further advice and was diagnosed with MG.

Female 41-50 years
I was diagnosed at age 44. Whilst undergoing surgery for gall-bladder removal I had a reaction to anaesthetics. Tests after surgery confirmed M/Gravis. I had previously had difficulty with voice change which made me seek medical help (I was referred to a speech therapist) my symptoms were not associated with Myasthenia Gravis and I therefore was not diagnosed until I had gallbladder surgery.

Male 71+ years
At 67 years of age I woke one morning with droopy eyelid (L) and muscle weakness in (L) hand. Had been feeling tired over period of six months but not worried thought it was normal. My symptoms developed to include swallowing and choking difficulties, slurred speech and unstable gait

Female 51-60 years
I was diagnosed at age 36 when I was experiencing breathing difficulties, unable to lift head and repetitive action weakness etc. Looking back I recognised I had had symptoms prior to diagnosis, fatigue as a child and unable to pick up new born to be breastfed at 25.

Female 41-50 years
I was diagnosed at age 33 years. I already had SLE, then developed tumour of Thymus (Non Hodgkins Lymphoma) and Positive Ab discovered as part of work up. Retesting of blood tests taken 2 years earlier showed positive Ab. I had previously experienced general weakness, and an episode of failure to start breathing post op. Looking back on my symptoms, I am struck by how non specific they were for some years. It was only later that I had changes in my voice and dysphagia.

Female 71+ years
I was diagnosed at 75 years when left eye closed during sleep and remained closed. I also had double vision, slurred speech and unstable gait.

Female 51-60 years
I was diagnosed at age 30 in Malaysia and it was Ocular Myasthenia. I am one of the lucky ones. I am at present in remission for the last 12 years.

Male 61-70 years
Diagnosed at 54 years of age when one eye kept closing, double vision and trouble swallowing water which used to blow out my nose. Trouble saying words with the letter S.

Female 61-70 years
Just prior to leaving Britain in 1968 I was diagnosed as being stressed. On arriving in Australia, thyroid problems were wrongly diagnosed and then myasthenia was correctly diagnosed. I was experiencing double vision, droopy eyelids, and difficulty swallowing and muscle weakness. A Thymectomy was performed in 1987. Its success was not as good as I had hoped but nevertheless with the aid of drugs I manage most tasks fairly well (no long distance driving). Trying to lead a stress free lifestyle is an advantage.

Female 61-70 years
I was diagnosed at age 64. Admitted to hospital with fluid on lungs rapidly deteriorated to point where couldn't swallow, speak, keep eyes open or walk. Diagnosis took 3 weeks after numerous and extensive tests. Finally diagnosed with Tensilon Test. *This lady then underwent a Thymectomy and her symptoms are controlled by medication.*

Male 71+ years
When I was 60 years I was diagnosed with MG after a car accident. I had, and still have blurred vision, droopy eyelids, difficulty swallowing and an unstable gait. My leg muscles are weak.

Female 71+ years
Diagnosed at age 16 years when it was not known here in Australia. I was fortunate enough to have an uncle who was a physician who wrote to cousins in England who diagnosed MG. I think I had had symptoms from the age of 10 or 11 when I was always being admitted to hospital, and being told there was nothing wrong just nerves. I was sent to England operated on in 1947 and had a large tumour.

Female 71 + years
I was diagnosed at age 74 after experiencing double vision. The Neurologist confirmed his initial diagnosis of MG after Tensilon and electrical tests. I had been experiencing symptoms of droopy eyelids, difficulty swallowing and choking and muscle weakness in arms and legs, but these had not been recognised as symptoms of MG but rather symptoms of ageing. My condition is controlled by medication and I am able to lead, although restricted, a fairly normal life.

Male 71+ years
At 66 years of age I was driving a car when suddenly I saw two of everything! I also had droopy eyelids. My UK neurologist had my thymus removed immediately. Here in WA my consultant thought that rather 'over the top' and he would not have recommended it at my age.

Living with MG

At the support group's coffee mornings and in exchanges of e-mails the MG sufferers have shared their experiences since diagnosis and their coping methods.

Some of these are listed below in random order:

- Once my legs went on me and I leaned on the car. I was embarrassed as I was sure people thought I was drunk. A friend saw me and knew that if I rested for a while I'd be OK. I was.

- In the mornings I could move mountains so I usually do some preparation for the evening meal at that time. This means that late afternoon when the energy fails me a little, I can easily cook the dinner and enjoy my meal.

- I had a lot of trouble washing the car. I could drive a tractor but the repeated up and down movement of cleaning fatigued me quickly. I now tackle the car a little at a time.

- When I was first diagnosed I was shattered and didn't know who to turn to. I was grateful to find someone else who had been diagnosed and was happily getting on with their life.

- I find the weather affects me. That is the extremes of hot and cold and particularly humid stormy conditions. Solution. I just take it easy. The weather will improve

- My pharmacist once dispensed the wrong strength of steroids for me - 25mg instead of 5mg. I ended up in hospital. I now check the tablets not just the label.

- I buy all my prescribed medications from the same pharmacist. He understands my condition and the effects the medications may have on me.

- My chemist understands my MG and helps me when I need over the counter cough mixtures. I avoid anything with quinine.

- I have a list of drug which I know I need to avoid.

- I found that once I relaxed a little and stopped worrying about the MG and lived within my limits I was able to enjoy my life.

- If you have an ACROD sticker don't get fussed about those who think you look alright. You know why you have the sticker.

- I'm on medication which affects my immune system so I try to avoid crowded areas. My friends and family know not to visit if they have heavy colds or flu.

- If you are a carer remember you must have a life of your own.

- When I was recently admitted to hospital with kidney stones I intervened when they were about to administer a painkiller and let them know in no uncertain terms that I had MG. The doctor was called. Then the correct painkiller selected. All was well. It made me realise

that in any emergency situation if I was able I should let the Ambo or First Aider know that I have MG.

- When I get 'worked up' over something my MG seems worse. It's easy to say but hard to do, that is *avoid stress*. I understand stress does not cause MG but once you have it, stress will exacerbate the symptoms.

- I told my dentist I had MG. He checked with my Neurologist about the anaesthetics he was using. His nurse was instructed to be vigilant with the suction to avoid choking

Contacts you May Find Useful

MG sometimes causes situations where we need some help. Services are available subject to assessment and in some cases small fees are charged. Your GP can be a valuable resource in getting you connected with available services.

In the opening chapter we talked about people suffering with MG being able to lead fulfilling lives. Utilising any of the services offered may be one way you can achieve that goal and give your self time and energy to do some of the fun things in life.

Transport: Those wretched eyes stop you from driving. **HACC Transport (telephone 9309 8106)** provides a service for people attending medical, dental, optical appointments plus for some social events (This would include our Support Group gatherings). The service can be accessed by ringing the above number. An over the phone assessment as to your ability to drive is conducted.

Cleaning, Gardening and Transport: All these services are provided by a number of HACC groups throughout the state. To make contact ring HACC Services at the **Department of Health on 9222 4060** who will be able to give you details of the HACC Service nearest to you.
Website www.health.wa.gov.au/HACC

Parking: Sometimes you find it difficult to walk far. To enquire about an ACROD sticker and obtain an application form **ring 9242 5544.** Of interest is that holders of ACROD stickers may park anywhere in the city of Fremantle (not just disabled bays), free of charge, whilst City of Perth offers the first 2 hours free in any of their car parks.

Medic alert: 1800 882 222. Should you be in an accident or perhaps take suddenly ill it assists first aiders, ambulance officers and hospital staff, if you wear a Medic Alert bracelet.

Taxis: are an option some may use. It may be possible to be issued with Taxi Users' Subsidy Scheme vouchers. Ring Transport on 9216 8000 for an application form.

Carelink: 1800 052 222. Is a Commonwealth Government Department where you may be given assistance to locate the sort of help you may seeking.

Words You May Hear

Acetyl-Choline (Ach): is a chemical transmitter released from nerve endings on voluntary muscles. It is far too small to be seen on any microscope.

Acetyl Choline Receptor (AChR): is the spot on the muscle which when Ach binds to it, opens up the way into muscles to allow salt to enter and trigger an electrical impulse causing the muscle to contract.

Acetylcholinesterase (AChE): is a protein near the AChRs that destroys any spare Ach.

Anti-cholinesterases: are drugs that block AChE so that any Ach lasts longer giving it a better chance of triggering the muscle contraction. These drugs include Mestinon (Pyridostigmine) used for treatment and Tensilon (Edrophonium), used for diagnosis.

Antibodies: are proteins specially designed to destroy germs and block toxins.

Antibody negative MG: is a name that has been used but is not a good one because these patients do have typical MG which is caused by antibodies. The difference is that the antibodies do not work against the AChR but instead attack another muscle target called MuSK.

Apnoea/apnoeic attack: the sudden stopping of breathing.

Autoimmune diseases: are caused by cells or antibodies that can attack our own tissues or cell products.

Azathioprine (Imuran): is a drug that generally suppresses immune responses.

Chronic: a long lasting condition as opposed to a short term (acute) condition. The term chronic does not relate to the severity of the condition.

Congenital MG: usually means it can be seen at birth but may also first be noted later in life. Many of the faults are in the AChR, others are in other genes at the neuromuscular junction. Often both copies of the gene are affected, so the parents of an affected individual may be unaffected.

Diplopia: double vision.

Diuretic: causes an increased output of urine.

Dysarthria: is the difficulty of getting the words out. It's the physical movement of speech rather than finding the correct word in the brain (dysphasia) and is due to tongue or other mouth muscle weakness.

Dysphagia: difficulty in chewing and swallowing.

Dyspnoea: difficulty in breathing.

EMG (electromyography):. this is where the nerves are stimulated electrically with the resulting impulses measured in the muscles they supply. Repetitive stimulation of nerve to muscle may be used in diagnosis of MG.

Enzyme: a protein capable of producing a chemical reaction in the body.

Imuran (Azathroprine): is a drug that generally suppresses immune responses and the disease of MG.

IV Ig intravenous immunoglobulin: (**Intragam)**.
Procedure of injecting slowly into a vein the pooled antibody protein fraction from normal blood.

LEMS Lambert-Eaton Myasthenic Syndrome: Another form of autoimmune neuromuscular disease with weakness similar to, but different from MG **.**

Mestinon: is the commercial name for Pyridostigmine. This drug is not a cure for MG but assists in managing the symptoms.

Muscles: are long tubes of proteins woven together. In the case of voluntary muscles, when triggered, they shorten or contract causing movement.

Plasmapheresis or **Plasma exchange:** is a method of cleaning the blood of unwanted antibodies to temporarily improve strength.

Prednisone, Prednisolone: synthetic steroid drugs.

Tensilon (Edrophonium): is a short-acting anti-AChE drug. Used when diagnosing MG.

Thymus: a gland that produces 'T cells' especially before a person turns 45, and sends them out to the rest of the body. The gland sits between the breast-bone and the heart and is important in autoimmune MG.

A **Thymectomy** removal of the Thymus, seems to improve the MG in patients where the onset has been at a young age (below 45 years).

Thymoma: a tumour of the thymus found in about 10% of myasthenics and requiring Thymectomy.

We acknowledge

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Together with wise guidance by two treating Neurologists

Donations to the

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