Myasthenia Gravis

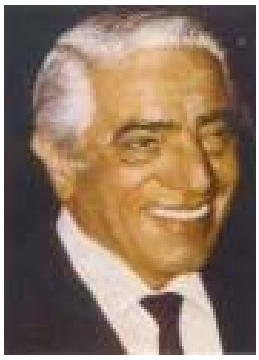
Information for Patients and their Families

MYASTHENIA GRAVIS FOR THE NON-SPECIALIST

HISTORY OF MYASTHENIA GRAVIS

The term "Myasthenia gravis" (MG) comes from the Greek (myasthenia = muscle illness) and Latin (gravis = grave) languages. The first description of myasthenia gravis dates back to the 17th century when Dr Thomas Willis wrote about 'a woman who spoke freely and readily enough for a while, but after a long period of speech was not able to speak a word for one or two hours.' It became clear in the 1930s that MG is due to a defect in nerve→muscle triggering. In the 1960s, it was predicted to be an autoimmune disease, suggesting an immune attack on the person's own muscles. This hypothesis was confirmed in the mid-1970's by Drs J. Patrick and J. Lindstrom by the discovery of specific immune **autoantibodies** in most patients that damage key targets at the junction between the nerve and the muscle. The normal job of antibodies is to destroy infectious bacteria or viruses. This makes MG one of the family of autoimmune diseases, which also include thyroid disease, diabetes in the young, multiple sclerosis, rheumatoid arthritis and lupus erythematosus.

WHO IS SUSCEPTIBLE TO MG?



Aristotle Onassis: one of the famous people with MG.

MG can affect anyone, from infants to the elderly, men and women. In terms of age of onset, there appear to be two incidence peaks; in women between 20-40 years old- termed early onset MG, and in men above 40 years old- termed late onset MG. It is not inherited and occurs in about 1 out of 10,000 people. However, about 2% of all myasthenics have inherited faults in nerve→muscle triggering. In this case, the disease is named 'congenital myasthenia' and does not involve the immune system, so immunosuppressive treatments (like steroids) are completely unsuitable unlike in autoimmune MG.

Newborn babies of MG mothers can also have short-term weakness caused by the transfer of **autoantibodies** from the mother to the baby via the placenta and/ or milk. That is not common: only about 10-15% of newborns from MG mothers show symptoms, which usually improve spontaneously after about 1-3 weeks and soon disappear. The MG in the mother normally helps to rule out the presence of inherited myasthenia.

WHAT ARE THE SYMPTOMS OF MG?

The main symptom is weakness of the voluntary muscles without sensory symptoms or pain. The weakness increases the more the muscles are used (as the day progresses), i.e. it is fatiguable, but gets better after resting. It often varies from day to day/ month to month. It can also get worse quite quickly, e.g. with infections. The first muscles affected in many MG patients are usually those that move the eyes, causing eyelid drooping or diplopia (double vision). Other patients experience weakness in the face, in chewing, swallowing or speaking and/or in the neck, trunk or limb muscles. In severe cases, breathing may be so weak that the patient needs a ventilator.

Three serial pictures to demonstrate fatigue of eyelid muscles and slight squint as the patient keeps looking up. After a few minutes of rest, the eyelids have returned to near-normal position (fourth picture).

HOW DOES MG PROGRESS?

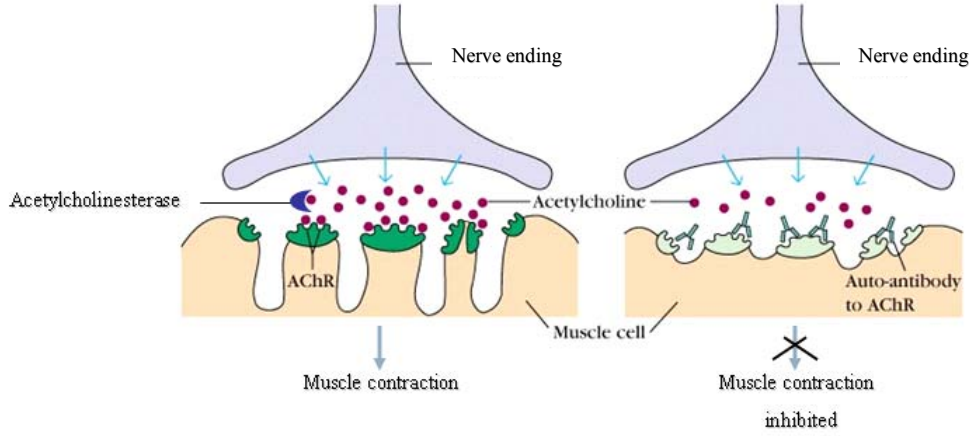
MG very often starts with eye muscle weakness. In 10-20% of patients, the weakness remains confined to eye muscles only for many years (ocular myasthenia). In the others, it starts to affect other muscle groups (listed above), usually within the first three years (generalised myasthenia). As in other autoimmune diseases, the symptoms may vary from time to time, or even go into remission in around 5% of patients per year. It was labelled 'gravis' because many patients used to die before better treatments were found in the 1930s - 1950s.

WHAT GOES WRONG?

Normal muscle function: When the brain sends an electrical signal along the motor nerves to the muscle to make a movement, a chemical transmitter – **acetylcholine (ACh)** – is released from the nerve endings. It instantly crosses to the muscle where it locks onto the **ACh receptors** (**AChR**), causing the muscle to contract. The spare ACh is broken down by ACh esterase, allowing the muscle to relax. Pyridostigmine (Mestinon®) blocks that breakdown, so that the ACh lasts longer and has a better chance of triggering.

In MG muscle, the presence of **autoantibodies** that bind to AChR causes loss of functional AChRs. Because we have very few AChRs in reserve, there are not enough of them for efficient nerve→muscle triggering. Some patients have autoantibodies to other molecules nearby the AChR in the NMJ which may indirectly act on the AChR (see below).

Normal situation **In case of Myasthenia Gravis**



Structure of the neuromuscular junction in normal and MG patients.

Problems with the immune system: It is not yet clear why the body produces these autoantibodies. There may be outside provoking factors, such as infections or drugs, but we still know very little about how and why the disease starts. Around 10% of MG patients, often between the ages of 40-60, have tumours in the thymus (**thymomas**). Thymomas are confined within the thymus gland (non-invasive) and tend to grow very slowly, but can rarely become malignant and may reappear locally many years after resection. It is therefore important that patients with thymomas have a regular follow up and worsening of myasthenic symptoms should warrant immediate examination. Furthermore, about 2/3 of the patients have milder abnormalities in the thymus, such as thymic hyperplasia in early onset MG and thymic atrophy in late onset MG. The link between thymic changes and MG is still not completely clear. Many researchers are striving to understand these processes more deeply so that they can devise better targeted treatments or even prevent susceptible individuals from getting MG in the first place.

HOW IS MG DIAGNOSED?

Clinical examination

MG can be diagnosed from the clinical history and the patient's muscle weakness, which is usually evident on examination. However, in mild cases, it may only be made obvious by testing muscle stamina, e.g. by lifting the arms time after time, or sustained up-gaze (for about 1 min) making the eyelids droop. Because MG is uncommon – and fluctuates – the diagnosis can easily be missed, especially in older people.

Presence of auto-antibodies

The diagnosis is confirmed by a blood test for anti-AChR antibodies in most patients. However, these antibodies are not found in about half of the patients with purely ocular MG and about 15% of those with typical generalised weakness; their MG nevertheless improves after plasma exchange that is used to wash antibodies away that are not detected in standard tests. We now know that, in about 1/3 of the patients who do not have anti-AChR antibodies, these other antibodies instead recognise the nearby target called **muscle-specific kinase** (**MuSK**), which is involved in clustering AChRs at the

nerve-muscle junctions. Their weakness affects the face and throat even more than in typical MG. The MG can be more severe and harder to treat in anti-MuSK than anti-AChR seropositive patients, but the thymus is often almost normal.

Electromyography and other tests
Electromyography (EMG) is a useful test of the muscle response to electrical stimulation of its nerve time after time. Typically in MG, the resulting electrical discharge in the muscle decreases progressively (~10%). Finally, the increase in muscle strength after giving ACh esterase inhibitors (see Fig. 3) can be measured before and after injecting the short-acting drug *edrophonium* (*Tensilon*® or *Camsilon*®) intravenously or by giving *pyridostigmine* (*Mestinon*®), a longer-acting version, by mouth. Once MG is diagnosed, scanning of the chest should be carried out to look for an associated thymoma. Special lung function testing that measures breathing strength can help to predict whether respiration may fail and lead to a myasthenic crisis.

HOW IS MG TREATED?

There are two kinds of treatments:

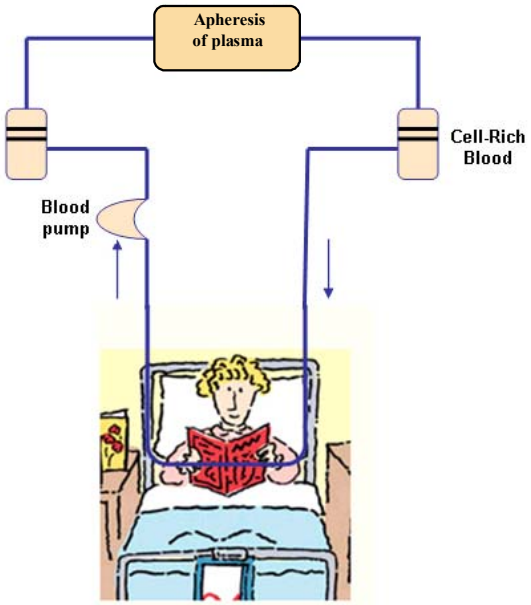
1. <u>Boosting nerve→muscle triggering,</u> mainly with *pyridostigmine* or *neostigmine*; these front-line drugs block ACh esterase, so that the ACh survives longer and has a better chance of triggering. These drugs only boost muscle triggering; many patients need something more to reduce the underlying immune reaction (see 2 below).

2. <u>Restoring AChR numbers by immune treatments by</u>:

I. Removing the damaging antibodies.

(a) **Plasmapheresis:** The simplest is plasma exchange (plasmapheresis), which is used to wash the patient's antibodies out of the bloodstream, while the blood cells are given back. It means being in hospital for about 5 days, after which the MG begins to improve. The benefits last only about 4-6 weeks because new antibodies are again produced in the patient. Plasmapheresis is especially useful when improvements are needed urgently, e.g. just before and after thymectomy, as well as while steroid treatment is being started (or sometimes while it is being continued in difficult cases). Plasmapheresis combined with steroids is recommended in severe forms of MG.

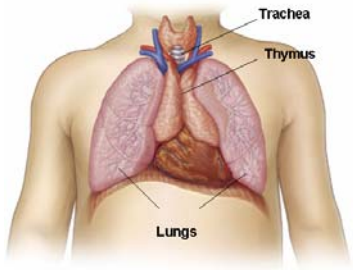
(b) **Intravenous Immunoglobulins (IvIg).** In the last few years, plasmapheresis has largely been overtaken by IvIg which means a transfusion of the antibody fraction pooled from thousands of healthy donors. That seems to work in MG by diluting or diverting the damaging antibodies. It may be used in combination with immunosuppressive drugs or when vascular access for plasma exchange is problematic. It takes longer to act than plasmapheresis, but its benefits can last several weeks. However, IvIg is very expensive and difficulties with immunoglobulin supply have been reported.



Plasmapheresis is used to remove the damaging MG antibodies.

II. Reducing antibody production.

(c) **Thymectomy.** The thymus gland plays an important role in the development of the immune system. Removing the thymus gland (thymectomy) has been used since 1940 for the long-term treatment of MG patients by re-balancing the immune system, and may lower antibody levels very slowly. Some neurologists feel that it helps patients with MG onset before the age of 45 years, especially if done early in the course of the MG, though that still awaits firm proof. Yet, when a thymoma is present, doctors agree that it should be removed to prevent spread, although its removal usually does not improve MG.



Thymus gland removal may play a role in MG treatment.

(d) **Immunosuppressive and anti-inflammatory drugs.** Such drugs, are the current standard for the treatment of moderate-to-severe MG. The most commonly used is prednisolone, especially in ocular MG. Corticosteroids are effective in decreasing the levels of anti-AChR autoantibodies, but can also cause complications, such as weight gain, high blood pressure, diabetes, anxiety/ depression/ insomnia, bone thinning, cataracts and gastrointestinal perforations. In the long-term, patients can often 'cruise' on lower doses by combining them with other immunosuppressive drugs such as *azathioprine* (*Imuran*®) or *cyclosporine A* (for *azathioprine*-intolerant patients). Alternatives that have proved successful in other immune-related diseases, such as rheumatoid arthritis and systemic lupus erythematosus, or in suppressing graft

rejection, are now undergoing clinical trials in MG, including Mycophenolate mofetil, Tacrolimus and Rituximab®.

HOW TO DEAL WITH MG?

There is no reason to find the next bits scary. **Starting on the bright side**:

- MG can nearly always be brought under good control, so most patients lead a pretty full life; very few people actually die of their myasthenia.
- The treatments for MG work better than for many other 'autoimmune' diseases; there is less pain and fewer serious long-term snags.
- These treatments are getting better all the time; with your help, we are trying to make sure that continues;
- Every MG patient should become their own 'special nurse' and work out their own ways of keeping their MG in its place. *Try not to let it take over your life*.

On the other hand, you should be warned that:

- Your MG may well be with you for years. It *can* fade away even without treatment, but only in about one patient in about 20-30 each year; so do not wait around – get treated;
- You will probably have to plan your day to make the most of the prime time when your strength is best;
- Other people may not always notice any weakness, especially when first meeting you; e.g. they may not realise that you are trying to smile.
- You are pretty sure to need some drugs, and they all have side-effects. Patients with MG usually manage on lower doses of steroids, taken every other day, than those with many other diseases.

It is also wise to avoid:

- Overexertion and unnecessary fatigue,
- Emotional stress,
- Catching infections (e.g. by staying out of crowds in the winter), and
- Certain drugs that directly affect nerve→muscle triggering, such as aminoglycoside (e.g. gentamicin) and especially ketolide antibiotics (e.g. telithromycin: Ketek®). Curiously, over-dosing with anti-cholinesterase drugs such as *pyridostigmine* or *neostigmine,* can increase the weakness or even cause cholinergic crises (overflow of saliva, tears, sweat and/ or vomiting) as well as increased weakness.

Eating a balanced diet, taking plenty of rest and some exercise (moderate, such as walking or ballroom dancing), and especially avoiding stress or infections, can help patients lead a fairly full life.

WHAT IS THE FUTURE?

The outlook for MG patients has improved dramatically in the last 30-40 years, with mortality rates currently near zero. As a result, the myasthenia is not "gravis" any more. Most present-day treatments evolved by trial-and-error, and some have serious side-

effects. As we learn more about autoimmune diseases, it should soon be possible to target treatments so that they selectively block only the damaging immune response in MG and not the whole immune system. With the knowledge accumulated on the immunopathology of MG and the role of the thymus and defects in immunoregulation, prospective antigen-specific therapies and novel technologies have been developed and are undergoing trials.
However, a lot still needs to be done. Since very few inherited or environmental risk factors are known for MG, we know almost nothing about how to prevent it. It is also interesting to know why some people are particularly susceptible to MG and others are not. Further research and optimisation of ongoing experimental approaches are therefore promising for the treatment of MG in the future.
Since MG is a rather rare disease, funding of relevant research is limited and therefore the research groups working towards understanding its mechanisms and developing more efficient treatments are very few in each country (often only one laboratory per country). This problem necessitates the coordination of efforts of the individual labs at a European level. The present European network has the aim of putting the efforts of various European research institutes together, in order to achieve faster progress in the understanding and treatment of the disease.

Frequently Asked Questions

MG: some facts

1. Is MG hereditary?
No, but there are some inherited risk factors for autoimmune diseases. The worst of these only increases the chances of getting MG from around 1 in 10,000 to around 1 in 2,000. So it is very rare to find two MG patients in the same family, though more have a relative with another autoimmune disease like young diabetes. There are also rare inherited ('congenital') myasthenias that are NOT caused by any immune attack, and should NOT be treated with immuno-suppressant drugs.

2. Is MG contagious?
We do not know the cause of MG (see 3), but there is absolutely **NO** evidence that MG can be caught from another MG patient.

3. How did I get MG?
For most patients, the cause of MG is not known. About 10% of MG patients have a thymoma, which seems to immunize them against muscle targets. In a very few cases, typical – but short-term – MG can be caused by the drug penicillamine, which was used to treat rheumatoid arthritis. It normally fades away a few weeks or months after the drug is stopped. In rare cases, MG may start during treatment with interferon alpha, or even after a bone marrow transplant.

4. Will my MG go away?
In some patients (around 5% per year), MG may go into remission for some time, or even permanently, and no longer needs any treatment. In the early stages, however, it

may be a wise choice to prevent it from getting worse – which happens more often than remissions, alas.

MG and quality of life

5. What is the quality of life for someone with MG?
Very few patients die of MG now, thanks to intensive care and modern treatments. Among these, it is nearly always possible to find a good combination that enables patients to lead a fairly active life. Tuning the treatments may take some time, and they will probably involve some side-effects.

6. Will I be able to continue working?
This partly depends on the nature of your job and your MG, but, with modern treatments, most MG patients continue working successfully.

7. Will I be able to drive safely? Should I inform my insurance company?
Double vision and muscle weakness can affect an MG driver's ability to respond to the road and others on it. You should discuss and evaluate your condition with your doctor first.

Women's and children's issues

8. Is it possible for MG women to have children?
Yes, many women with MG have successful pregnancies, especially if their MG is well controlled beforehand. Some find that their MG gets worse during pregnancy, and others afterwards, but that can be controlled. If they can't push well, they may need some help during the delivery. Most specialists prefer epidural to general anaesthesia (see 25).

9. Can a mother's MG affect her baby?
It is very rare for a mother's MG to affect the normal growth and development of the baby (a condition known as arthrogryposis). BUT the damaging antibodies are passed across from the mother (just like the good ones that protect babies against infections). They can cause short-term weakness in the newborn baby. That happens to about 1 mother in 8 or 10. If so:- (**a**) it usually fades away in 3 - 4 weeks; (**b**) it usually affects her next pregnancies similarly – i.e. each mother tends to be true to type; (**c**) breast-feeding is not advisable, despite its other benefits.

10. Are MG drugs safe to use during pregnancy?
Pyridostigmine (Mestinon®), prednisolone and azathioprine (Imuran®) are considered safe during pregnancy and breast-feeding. Other immunosuppressive drugs are either known to harm the baby's development or have not yet been shown to be safe, so they should be stopped several months in advance of any pregnancy. What is more, methotrexate and cyclophosphamide can affect sperm and egg generation. Men are therefore recommended to get some sperm banked before starting these drugs or stop taking these at least a year before giving sperm.

11. How does the menstrual cycle affect MG?
Some women may notice fluctuations in muscle strength during the time of their period, and may benefit from hormone therapy to prevent these fluctuations. However, others do not notice any changes. The menopause does not seem to have any effect on MG.

12. What should parents with MG children keep in mind?
In Europeans, autoimmune MG is rare in childhood. Its treatment is similar to that in adults, although high doses of steroids should be avoided because they interfere with growth.
Although a child's intelligence is not affected, droopy eyelids or double vision might make it difficult to see the blackboard, and MG may well limit other activities. It is therefore important that the child, parents, and school are fully informed about the condition; they should be warned about possible difficulties with eating, climbing stairs, or in games. Then they can provide the support that each child needs; for example, MG might make writing and reading harder, so a one-to-one helper, regular rests or more time (especially during exams) should be offered.

Treatment for MG

13. Do MG drugs have adverse side-effects?
As with all drugs, the proven benefits have to be balanced against the potential side effects. With careful instruction and close monitoring (e.g. regular blood tests), most patients find treatment options that help their MG without excessive side-effects. At standard doses, pyridostigmine (Mestinon®) and the related drugs neostigmine and distigmine can cause hyperactivity both of the 'automatic' muscles in the bladder and guts (etc.) and of their glands, leading to drooling or diarrhoea (see 14). At very high doses, these drugs can make MG weakness worse, or even cause 'cholinergic' crises.
Corticosteroids and immunosuppressive drugs are not clever enough to knock down only the damaging antibodies: unfortunately, they suppress the protective antibodies and immune cells too, and that increases the risk of infections. In addition, steroids have many side-effects, which include weight gain, mood and appearance changes, anxiety/depression/bad sleeping, failure to react to stress, greasy skin, high blood pressure, diabetes, bone-thinning, muscle damage, cataracts and peptic ulcer; if long-term use is expected, patients are routinely given a bisphosphonate plus calcium and vitamin D to prevent the bone-thinning that usually happens otherwise. Azathioprine (Imuran®) can cause liver and blood problems and allergic reactions; regular blood tests are essential to detect them early. About 1 in 200 people are over-sensitive to azathioprine, because of an inherited weakness in breaking it down. This can be checked in advance with a blood test.

14. Is it safe to use pyridostigmine (Mestinon®) in the long-term? How can its side effects be controlled?
There is no evidence that pyridostigmine (Mestinon®) is harmful in the long-term (at standard doses). Drooling and diarrhoea associated with Mestinon® can be overcome by propantheline, an antispasmodic drug which does not affect the voluntary muscles weakened by the MG.

15. Will I feel weak after I start taking medications?

Steroids can make muscle weakness worse in the first few days, especially if started at high doses. Patients may also continue to feel weak despite medications (see 14). Since pyridostigmine (Mestinon®) often does not return strength to normal, immuno-suppressive drugs may be needed to restore AChR numbers. Drugs like azathioprine (Imuran®), Mycophenolate (Cellcept®), methotrexate or cyclosporine may take 9 - 15 months to work, and may need fine-tuning. Your neurologist will advise whether your treatments need more time to act, or whether other medications need to be added.

16. Why might a patient not respond to treatment?

There are several possible reasons:

- Some patients' weakness may be worsened by another drug that is being used for another condition (like quinine for malaria).
- You may not be taking the correct dose. For example, the dose of azathioprine (Imuran®) is based on your whole body weight and larger people need larger doses.
- Other medical problems must be treated too. For example, for reasons not necessarily related to MG, a patient could develop depression, low thyroid or heart problems that produce fatigue not caused by their MG.
- Keep your expectations realistic. Are you trying to do too much? Are you expecting your body to behave like it did at an earlier age?
- Rare patients are misdiagnosed and do not have MG. Discuss with your doctor how definite the diagnosis of MG is.

17. What treatments are used when MG is severe?

The best short-term treatments for severe MG are plasma exchange and IVIg (intravenous immunoglobulin). Especially at high doses, steroids can increase strength within 2 - 3 months. They are often combined with another immuno-suppressant such as azathioprine (Imuran®), Mycophenolate (Cellcept®), methotrexate or cyclosporine, so that patients can eventually 'cruise' on lower doses of steroids, with fewer side-effects, though that may take many months.

Vaccinations in MG

18. Are there any vaccines that MG patients should have?

Medical decisions should always be discussed with your doctor. Patients on immunosuppressive treatment have a slightly higher risk of infection. It is therefore recommended that MG patients should receive additional vaccination to protect e.g. against pneumonia and flu. For example a flu vaccination in the autumn of each year can provide up to 70% protection for the coming winter. A jab for pneumoniae every 10 years is also recommended.

19. Are there any vaccines that MG patients should not have?

Patients should remember to mention their condition before vaccination. In general, immuno-suppressed patients should avoid live vaccines, but these are safe in MG patients under other kinds of treatment. Prednisolone, azathioprine (Imuran®) or other immunosuppressive drugs may depress patients' immune systems so much that they fail either to respond to the vaccine or to control the modified germ it contains. It may also be wise not to travel to high-risk areas.

20. Can vaccination trigger or worsen MG?
There is no convincing evidence linking vaccination to MG. Infections can make MG worse or even cause myasthenic crises, so it is sensible for MG patients to have appropriate vaccinations.

21. What is a myasthenic crisis?
A myasthenic crisis occurs when the muscles that control breathing are affected, as can happen during an infection (see 22). This can create a medical emergency requiring a respirator to help breathing.

Factors that can affect MG

22. What factors can aggravate MG?
Infections, fevers, strenuous exercise or emotional stress can make MG or congenital myasthenias worse. So can a variety of drugs (see 23-25). The risk is much greater in patients whose MG is not well controlled. It is therefore important that you discuss your condition with your doctor regularly.

23. Can any drugs make MG worse?
Obviously, drugs that interfere with nerve-muscle triggering can make MG worse and should be avoided; they include BoTox. Do remember: (**a**) such effects have been proven for some drugs: for others, they are only suspected; (**b**) any reaction to any drug can be very specific to an individual patient; (**c**) always check with your doctor before starting or stopping any drug; (**d**) although doctors will try to suggest safe alternatives, careful use of a suspect drug may be OK if it is really needed (perhaps a special antibiotic) and the MG is well controlled. Doctors and MG patients should be fully aware of all the potential problems and must be ready to deal with them.
Drugs that can make MG worse include some anti-malarials, beta-blockers used to treat heart conditions, and muscle relaxants (see 25); also, the antibiotic gentamicin; above all, **Telithromycin (Ketek®) should be avoided altogether by all MG patients; it can be dangerous in MG within hours.**

24. What kind of painkillers can a person with MG use?
Any of them can be used, but care is needed with the morphine (opiate) family of painkillers, which includes codeine. Because they can depress breathing and coughing, and even increase the chance of a respiratory arrest, they should be used with care.

25. Which anaesthetics may be used for MG patients?
In general, it is crucial that anaesthetists know in advance about the patient's MG or inherited myasthenia, and have a chance to make plans. If that is done, complications are very rare. The MG should first be brought under the best possible control, which might mean plasma exchange or IVIg a week or two beforehand. Local or regional anaesthetics should be used if possible, especially for operations below the waist. Shorter-acting local anaesthetics, such as lignocaine, combined with light sedation, avoid the depression of breathing that many general anaesthetics (or opiates) cause. If a general anaesthetic must be used, particular care is needed with muscle relaxants, which are used for easier access for deep operations; they paralyse the breathing muscles, and MG patients are 5 - 10 times more sensitive to them than healthy people.

26. Is there an association between asthma and MG? Is it safe for asthma patients to take steroids?
There is no evidence of an association between asthma and MG. Muscles that control breathing are affected in both conditions, so patients might experience difficulty in breathing and may require mechanical support.
Asthma patients on steroids should be aware of potential short-term worsening of their MG symptoms (see 15) and may benefit from inhaled steroids, which are less hazardous than tablets or injections because the dose is smaller.

27. Is it safe for an MG patient to be injected with X-ray contrast media?
It has been reported that injection with X-ray contrast media can aggravate MG, but there is no real clue as to the mechanism. You should discuss all medical decisions with your doctor.

28. Does dental hygiene affect MG?
Immunosuppressed patients are more likely to develop dental infections. Prevention of dental infections is vital, because they are stressful and can aggravate MG. Also, cyclosporine can cause overgrowth of the gums (gingival hyperplasia). Most dental procedures are safe for MG patients; if there are difficulties in closing the mouth, holding the head up or swallowing, the dentist should know how to prevent problems. It also helps to book appointments in the morning/when your strength is greatest, and to keep them short. Mercury in a dental amalgam (fillings) does not cause or worsen the disease. Oral anticholinesterase agents should be administered 1.5 hours before dental treatment to achieve maximal effect during the dental procedures.
Local anaesthetics, such as xylocaine and carbocaine, are preferred to general anaesthetics, and can be administered safely for dental surgery; carbocaine might be preferred because of the fewer side effects and the shorter duration of the anaesthesia effect. Nitrous oxide oxygen sedation may reduce stress and anxiety associated with dental treatment. When severe pain is expected, opioids can be prescribed for short term after consultation with your doctor.

29. What should I know about nutrition and MG?
There are no particular diets that help MG. It is important to avoid getting over-weight, which can strain the muscles and even cause diabetes, and limit treatment options. Preparing purées and smoothies may help to avoid difficulties with chewing and swallowing. Speech therapists may have valuable advice. Patients taking steroids need to make sure they take enough potassium; also vitamin D and calcium to prevent bone thinning. Additional vitamin B12, magnesium, and folate beyond the usual daily requirements are not needed. MG patients need to be aware that some tonic waters contain quinine (it is listed on the label) which can make MG muscles even weaker. Caffeine affects MG patients just like healthy people; it can make them feel more awake, shaky (with tremors) or anxious, and can cause palpitations. It is also recommended to limit consumption of alcohol, as it can cause muscular weakness, and salt, to minimise fluid retention caused by steroids.

30. Does the climate influence MG?
Some patients with MG or inherited myasthenias feel weaker in hot weather. As with fevers, the heat can affect nerve → muscle triggering.

31. How can I contact my nearest MG Support Group?
Each country to supply its own details.

USEFUL CONTACTS

- **The Myasthenia Gravis Association**
  Southgate Business Center, Normanton Rd,
  Derby DE23 6UQ, UK.
  Tel: (0044) 01332-290219, Fax: (0044) 01332-293641
  http://www.mgauk.org/

- **Myasthenia Gravis Foundation of America**
  1821 University Ave. W., Suite S256,
  St. Paul, MN 55104.
  Tel: (651) 917-6256 or (800)541-5454
  Fax: (651) 917-1835
  http://www.myasthenia.org/

- **Association Francaise Contre Les Myopathies**
  Rue de l'Internationale 91 000, Evry.
  Tel: (0033) 01 69 47 28 28
  http://www.afm-france.org/

- **The Australian Myasthenic Association**
  108 Bantry Bay Road, Frenchs Forest NSW 2086.
  Tel: (02) 4283 2815
  http://www.myasthenia.org.au/

- http://pages.prodigy.net/stanley.way/myasthenia/

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Information on myasthenia gravis for Patients and their Families

Euro Myasthenia