The author reviews the medical, surgical and anesthetic considerations necessary for patients with myasthenia gravis. The article points out that as many of the drugs used in anesthesia affect the activity of the neuromuscular conduction mechanism, the anesthetist should have an awareness of the nature of the disease and the impact these drugs have on it.

Myasthenia gravis is usually defined as a chronic disease of the neuromuscular junction that has a disputed etiology. It is characterized by weakness and variable fatigue of voluntary muscles, symptoms that gradually are alleviated with rest. There is a predilection for ocular and other cranial muscles including those that control facial expression, swallowing, phonation and respiratory function. There may be limb muscle involvement which may lead to atrophy.

The severity of the disease can range from mild (slight ptosis) to severe (complete respiratory failure), including death. There are no signs of neural lesion and partial reversibility can be accomplished by the administration of cholinergic drugs.

History

Willis first described the signs and symptoms of the disease in 1672. Erb in 1879, described it again as Erb-Goldflam's disease. In 1895, Jolly named it myasthenia gravis pseudoparalytica. Campbell and Bromwell modified the name to myasthenia gravis in 1900.

In 1934, Mary Walker demonstrated that the administration of physostigmine was followed by marked improvement of muscle function. This was a landmark discovery that led to the present concept of myasthenia gravis as a malfunction of neuromuscular transmission.

Incidence and prevalence

In the United States, the incidence of myasthenia gravis is 1 in 20,000 with a prevalence toward 30-year-old females. The disease can begin spontaneously at any age but occurs most frequently at about age 30. It affects women more than men at a ratio of 3 to 2, and the peak incidence of onset is the second to third decade in females and the fourth to seventh decade in males. In children and the elderly there is no sexual predilection.

Etiology

Although studied in-depth for many years, the etiology of myasthenia gravis...
had remained somewhat unclear. Recent findings, however, have left little doubt that this disease is caused by an autoimmune response.

For sometime there had been four generally accepted possibilities as to the cause of myasthenia gravis.

1. Diminished synthesis, storage or release of acetylcholine. In 1961, Desmedt made electromyographic recordings from myasthenic muscle. He observed that the myasthenic muscle responses to stimulation were similar to those of normal muscle treated with hemicholinium, a drug which inhibits either the synthesis or release of acetylcholine.\textsuperscript{10}

Dahlback observed through the use of electrical stimulation and measurement, that the miniature endplate potentials of myasthenic muscles were significantly reduced.\textsuperscript{11} In 1964, Elmqvist, et al concluded, from observing in vitro nerve-muscle preparations of myasthenic patients, that a reduction of the number of acetylcholine molecules in each quanta and not a total reduction in the number of quanta was the main cause of the neuromuscular transmission defect.\textsuperscript{12}

2. Altered response of the motor endplate to acetylcholine in myasthenic patients. There have been observations by some clinicians that there is a decreased sensitivity of the myasthenic endplate to acetylcholine, succinylcholine and other depolarizing compounds, such as decamethonium.\textsuperscript{13,14,15,16}

Normally, when acetylcholine com-
bines with the receptors at the neuromuscular junction, a transient phase of permeability to sodium and potassium takes place which results in electrical depolarization.

In 1961, Foldes\textsuperscript{17} presented a theory as to the cause of the decreased sensitivity of the endplate. He suggested that there could be a molecular structure alteration of the endplate receptor protein, an alteration of the permeability of the pores for sodium, potassium and other electrolytes and the presence of other neuromuscular blocking agents. Studies by Grob and associates corroborated these findings.\textsuperscript{18-20}

3. Virus theory. The presence of cytoplasmic inclusion bodies in the intramuscular nerve fibers and other histologic findings have suggested a possible viral origin to the disease.\textsuperscript{21}

4. Autoimmune aspects. In 1960, Nastuk noted that there were varying serum complement levels in the myasthenic patient during remission and exacerbation. He concluded that an autoimmune process was the possible cause of myasthenia gravis.\textsuperscript{22}

In this regard, an antigen-antibody response initiated by the thymus gland has been suggested. At an early age, the thymus is believed responsible for destroying mutations developed in the blood cells of normal people. As the person grows older, and these defective cells are destroyed, the thymus atrophies as it is no longer needed or used. The thymus is most active in the embryonic stage, but does function for a short while after birth.\textsuperscript{23}

It is felt that in myasthenia gravis the thymus loses its ability to distinguish between normal and abnormal cells and forms antibodies against normal skeletal muscle. These antibodies are then collected at the neuromuscular junction.

In 1973, Lindstrom, et al made a remarkable breakthrough in the autoimmune theory.\textsuperscript{24} He and his associates isolated a purified acetylcholine receptor protein from the electric eel and the electric ray. Immunization of rabbits with this receptor protein caused a myasthenic syndrome which was reversible with anticholinesterase drugs. Antibodies to this purified receptor protein were found in the rabbit sera which were capable of blocking the depolarizing acetylcholine receptor protein response.

This experiment has yielded the same results when conducted on rats, guinea pigs, goats and monkeys. The myasthenic syndrome which was elicited has been termed “experimental autoimmune myasthenia gravis” and has been accepted as a valid model for human myasthenia gravis.

Fambrrough and associates, in 1973, found that a snake poison called alpha bungarotoxin would bind specifically and irreversibly to the active site of acetylcholine receptors.\textsuperscript{25} They tagged acetylcholine receptors with \textsuperscript{125}I-labeled alpha bungarotoxin and found that the myasthenic muscles indicated a 70-89\% reduction in the number of acetylcholine receptors per neuromuscular junction as compared with control muscle.

This further supported the probability that an autoimmune response had destroyed acetylcholine receptors at the neuromuscular junction. Since then, other studies have found antibodies to the skeletal muscle in high titers in myasthenic patients.

Another important finding showed that when mice were injected with the globulin fraction of serum from myasthenic patients, a disease was caused in the animals that had myasthenic features.

Although previously mentioned etiological theories may remain valid, the findings by Lindstrom, Fambrrough and others have led to an almost total acceptance that myasthenia gravis is an autoimmune disease. This may also help to explain why steroids and thymectomy are so beneficial in its treatment.

Clinical Manifestations

A symptom of myasthenia gravis is that of muscle weakness involving
various parts of the body which resembles curare-like intoxication. It was this observation that led to the use of anticholinesterase drugs in the treatment of this disease. Steroids are also being used because of the possible autoimmune aspect, but the anticholinesterase drugs are the basic treatment.

The most commonly affected muscles are those of the eyes, mouth, pharynx and shoulder girdle. Swallowing is frequently affected and muscle weakness is most noticeable right after exercise.

The predominant symptom is ocular muscle weakness which is affected adversely by prolonged or repeated use, exposure to sunshine and emotional factors. The most frequent sign is ptosis, either unilateral or bilateral, and it is usually accompanied by blurred vision and dysopia. Nystagmus may be present and the ptosis may shift from one eye to the other.

Many cases exhibit symptoms of skeletal muscle weakness with the shoulder girdle most often affected. The hip, neck and back also may be involved.

In addition, there may be signs of respiratory distress with a decreased vital capacity and a greatly diminished maximal breathing capacity. The effort expended to maintain posture will usually cause pain in the muscles whether involved or non-involved in the myasthenic process. Ossermann noted other sensory changes such as ocular pain, paresthesias and headache in 14% of the patients he studied.

In advanced cases, patients have to support their chin with one hand to help them talk and may have difficulty holding their head erect. Ventilatory insufficiency occurs only in severely affected patients.

Neonatal myasthenia gravis occurs in infants born to myasthenic women, and these infants have a transient syndrome of muscle weakness. Small doses of cholinergic drugs may be administered therapeutically, but symptoms subside within 1 to 4 weeks. This occurs in 10 to 25% of the babies born to myasthenic women, and it is believed that a factor is passed through the placenta lending further credence to the autoimmune theory. In congenital myasthenia gravis, symptomatic infants are born to normal women. As opposed to the neonatal type myasthenia gravis, the symptoms exhibited in congenital myasthenia gravis persist.

About 5% of myasthenic patients have thyrotoxicosis at some time. Usually the two disorders occur simultaneously, but sometimes hyperthyroidism is evident for weeks or months before there are any myasthenic symptoms. Neuromuscular transmission in myasthenia gravis may be adversely affected by muscle relaxants, magnesium, choline, quinine, inhalation anesthetic agents, quinidine, barbiturates, tranquilizers, antibiotics, procainamide and ganglionic blocking agents.

Stress and emotional problems may have adverse effects on myasthenia gravis, and Meyer reports a higher mortality rate in these patients.

Diagnosis

In almost all cases, the diagnosis of myasthenia gravis is obvious and can be concluded from the patient's history and physical examination. The diagnosis is immediately confirmed by the response to cholinergic drugs. In order to provide further confirmation in some cases, the electromyographic response to ulnar nerve stimulation may be studied. A detailed account of some of the diagnostic tests possible follows.

**Edrophonium test.** Up to 10 mg of edrophonium (Tensilon®) is administered intravenously and muscle performance is observed. Some of these observations include swallowing, speech, ocular signs, chewing and measurement of vital capacity. Observations are made before and 30 to 90 seconds after administration of edrophonium. A definite improvement in muscle function is indicative of myasthenia gravis.
This test is also used to indicate the effectiveness of a known patient's anticholinesterase therapy. There may be fine, fascicular tremors in the eyelids of normal persons 30 to 40 seconds after administration of edrophonium. These tremors are absent in the myasthenic patient.

**Curare test.** Because this test entails some risk, adequate measures for artificial ventilation must be available. Foldes and McNall suggest that 0.5-2.0 mg of d-tubocurarine be administered over a 30-second period. In 5 minutes, muscle performance is assessed. An additional dose of 0.5-1.0 mg of d-tubocurarine may be administered if there is no significant deterioration. The total dose to be administered is 4.0 mg. A diagnosis is made if there is a significant decrease in muscle function.

The curare test, as described, is extremely dangerous and rarely used today. It has largely been replaced by a safer modification called the intravenous (IV) regional curare test.

**I.V. regional curare test.** The patient is prepared in the same manner as for an IV regional block, except that both the patient's arms are used. After the cuffs have been inflated to 40 torr above systolic pressure, 0.2 mg of d-tubocurarine diluted in 20 ml of 0.9 sodium chloride is administered in one arm and diluent alone in the other arm. The cuffs are released after 5 minutes; and 2 minutes following their release, muscle power is tested in both arms.

Other commonly used supplemental tests available in diagnostic confirmation are electromyography, muscle biopsy and immunological testing which includes measuring antibody titers and the mouse transfer assay for "myasthenogenic effect".

**Pathology**

Pathologic changes in myasthenia gravis affect skeletal muscle, the thymus, thyroid and heart. Skeletal muscles may appear normal, but there are often collections of lymphocytes around the blood vessels. In some cases there is evidence of degeneration of muscle fibers, and the inflammatory cellular response may be more extensive. Microscopic examination will show abnormal pre-and post-synaptic components of the neuromuscular junction.

There is an inflammatory reaction in the myocardium accompanied by a focal necrosis. This is a characteristic lesion found in myasthenic hearts. It is thought that this myocardial damage has been the cause of some reported sudden deaths in myasthenia gravis patients.

Thyroid disease is more common in myasthenic patients than in non-myasthenic patients. If the thyroid problem is corrected, there is often an improvement in the myasthenia.

The thymus gland is often abnormal and thymoma (an encapsulated tumor) occurs in approximately 10% of myasthenic patients. Thymomas will occasionally invade surrounding structures, but rarely metastasize to other organs.

**Treatment**

Management of myasthenia gravis may include drug therapy, radiation treatment or a thymectomy. The management is mainly symptomatic as the etiology is not completely certain.

**Drug therapy.** The treatment of myasthenia gravis with drugs is rather extensive and complex, therefore only the fundamentals will be discussed.

Of the few classes of drugs available with which to effectively treat the disease, the main one is the anticholinesterase group. These are quaternary bases which are structurally similar to acetylcholine. They combine with the cholinesterases and prevent the hydrolysis of acetylcholine.

Management of drug therapy of the myasthenic patient is not easy as underdosage or overdosage with these agents are likely possibilities. The ideal dose of anticholinesterase is one that is just large enough to increase the strength of the initial muscular effort.
thereby retarding the development of weakness or fatigue. An excess of anticholinesterase may augment muscle fatigue and cause fasciculations due to an accumulation of acetylcholine. Excess acetylcholine at the myoneural junction causes a depolarization or cholinergic block.

The common drugs for myasthenia gravis treatment in use today are: edrophonium (Tensilon®), neostigmine bromide (Prostigmin®), pyridostigmine (Mestinon®), and ambenonium chloride (Mytelase®). The latter two are preferred as they are slightly longer lasting and have fewer gastrointestinal disturbances. These drugs are administered on a trial and error basis to find the optimal dosage for each patient. Since these drugs have muscarinic side effects, the use of atropine may be indicated.

In some cases the endplate will not respond to any anticholinesterase drug; and apparently there is a desensitization to acetylcholine. These patients are given ACTH and/or corticosteroids along with their anticholinesterase drugs. If the endplates still do not respond, the anticholinesterase drugs are withdrawn for a “drying out” period and patients are maintained on the steroid regimen. Previously, endplate inactivity was achieved by the administration of d-Tubocurarine, with the patient being artificially ventilated and fed for a period of 7 to 10 days. This was generally felt to be sufficient time to restore endplate sensitivity.33

Other drugs in use are ephedrine, germine diacetate, potassium, ACTH, prednisone, methylprednisone, and guanadine. ACTH and adrenocorticosteroids are now being used with increasing frequency and a 90% success rate has been reported. The mechanism of action of these drugs is unknown; but it is felt that their relationship is to the immune response, as patients taking these agents have done very well, especially after having undergone thymectomy. As these drugs are used in relatively large amounts, their side effects must be carefully watched.

In some cases steroids, during the initial treatment of myasthenic patients, have had harmful effects such as muscle weakness and respiratory depression, leading to respiratory failure. Though these effects disappear as the therapy continues, the possibility of initial occurrence must be kept in mind.

Radiation. Radiation therapy is currently used only when thymectomy is contraindicated. It is administered to the anterior mediastinum in a dosage starting at 50 rads and going as high as 6,000 rads. As cholinergic crisis may develop during this therapy, the patient should be carefully observed and preferably hospitalized while undergoing treatment.

Thymectomy. Sauerbruch, in 1911, performed the first thymectomy on a myasthenic patient. Although the patient showed marked improvement, there were conflicting opinions for a long time afterward as to the value of this therapy. Perlo and associates34 outlined basic criteria for when a thymectomy is indicated. The principal indication for determining surgery is if the patient’s condition is deteriorating steadily and cannot be helped with pharmacological therapy. Another indication is if the patient is a young female; females respond best to this treatment and, in many cases, show complete remission of their symptoms.

Thymectomy is followed by significant improvement in approximately two-thirds of the myasthenic patients who have no tumor. Patients with thymoma tend to do worse whether or not the tumor is excised. Given today’s skillful and sophisticated surgical and anesthetic management techniques, as well as the advent of respiratory intensive care units, the risks of the operation itself are negligible. The transcervical approach to thymectomy has greatly reduced morbidity and allowed for a quicker postoperative recovery period.35
Myasthenic emergencies

Any of the aminoglycoside antibiotics (mycins and polymyxins) are capable of causing a myasthenic emergency. These drugs cause some degree of neuromuscular blockade in the normal person and interfere with the actions of muscle relaxants. Their use in myasthenic patients can have profound effects.

Basically, there are two types of myasthenic emergencies: (1) myasthenic crisis, which is related to undermedication, and (2) cholinergic crisis which is related to overmedication. These situations usually develop suddenly and can be life-threatening. The main symptom of both crises is muscle weakness; and in an emergency situation, it is often impossible to tell whether such weakness is due to too much or too little medication (anticholinesterase drugs).

In a myasthenic crisis, the principal entity is the increasing requirement of anticholinesterase drugs to the point where the patient is unresponsive to further therapy or, as stated earlier, the patient is undermedicated. For some reason certain patients can develop an endplate insensitivity to acetylcholine which can lead to this crisis.

Some other causes of the myasthenic crisis are the administration of non-depolarizing muscle relaxants during general anesthesia, the patient’s failure to take medications as prescribed, emotional stress, infection, and various other drugs known to cause adverse reactions in the myasthenic patient. In a true myasthenic crisis, the administration of edrophonium or neostigmine will usually result in a dramatic improvement.36

As previously stated, a cholinergic crisis is related to overmedication with anticholinesterase drugs. One of the major causes of this crisis is the patient who increases his or her dosage of the anticholinesterase drug hoping to improve muscle function. Another cause is a change in a patient’s response to anticholinesterase therapy. Usually the administration of 10 mg of edrophonium intravenously, with no improvement or a worsening of condition, can be used as an indication to differentiate this crisis from myasthenic crisis. Once the diagnosis has been made, atropine should be given intravenously to counteract the muscarinic effects of acetylcholine.

If diagnosis cannot be made between the two crises, all anticholinesterase medications must be stopped and respiratory support and tube feedings should be instituted.

If untreated, any myasthenic crisis will ultimately end in respiratory failure. The probability of artificial ventilation must be kept in mind, and the use of a cuffed endotracheal tube is mandatory to prevent aspiration and to facilitate suctioning since myasthenic patients usually have thick, copious secretions. If atropine is to be administered to counteract muscarinic effects, any hypoxia or respiratory acidosis that is present should be corrected first or serious cardiac arrhythmias may result.37

The mortality rate of crisis (both types) is now thought to be about 10% as compared to 50% before the advent of respiratory care units.

Prognosis

The course of myasthenia gravis is variable and the prognosis is uncertain. The disease may be restricted to ocular muscles for many years with no threat to life. Other patients are disabled to varying degrees with oropharyngeal or limb weakness and a few are crippled. Crisis occurs in about 25% of cases. The mortality rate for myasthenia gravis is about 15%.

Management during pregnancy

Pregnancy may cause exacerbations, remissions or have no effect at all on the myasthenic patient. Patients with myasthenia who have had no previous symptoms may exhibit major signs of
the disease upon becoming pregnant. Anticholinesterase therapy should be used with great caution, especially in the third trimester. At this time period the uterus has an increased sensitivity to anticholinesterase and premature labor may result.

For labor pain, short and medium-acting narcotics are preferable to long-acting ones and should be given in small doses, as these patients are susceptible to respiratory depression and do not have the respiratory reserves of the non-myasthenic patient. A narcotic antagonist, preferably naloxone (Narcan®), should be available.

Anesthesia for any method of delivery must be spinal or epidural except where contraindicated. In this case, nitrous oxide-oxygen and local infiltration may be used.

The postpartum clinical course usually is the same as before pregnancy, and patients should be treated according to the condition or stage of their myasthenia.

Relationship to anesthesia and surgery

As myasthenia gravis is a neuromuscular disorder, it can be greatly affected by the agents used in today's anesthesia practice.

A myasthenia gravis patient undergoing any kind of surgery should, if possible, be admitted to the hospital several days prior to the operation. Anticholinesterase therapy should be stabilized and reduced according to the patient's hospital activity. He should be on the minimum dosage that can be tolerated and still arrive in the operating room with no discomfort.

Regional or local anesthesia is preferred, if possible. However, if employing ester compounds (such as, procaine, or chloroprocaine), small doses should be used as their toxicity is increased in myasthenic patients. The cause of this is the fact these patients are usually on anticholinesterase therapy and cholinesterase is instrumental in metabolizing ester compounds.

If general anesthesia is indicated, most agents seem to work well, especially halothane with nitrous oxide and oxygen. Enflurane has been used with safety and is combined with a quick recovery time. Balanced techniques, for example, nitrous oxide-oxygen supplemented with thiopental sodium, droperidol or fentanyl, also work well.

For muscle relaxation, the non-depolarizing agents are the drugs of choice. For example, 0.5 to 2.0 mg of d-Tubocurarine will give profound muscle relaxation which can easily be reversed in the usual manner with atropine and neostigmine. A blockade monitor would be helpful, if not essential, for the proper use of muscle relaxants.

Depolarizing relaxants are much less predictable in the myasthenic patient, and the uninvolved muscles are usually resistant to depolarizing agents. This may necessitate large dosages of these drugs to give surgical relaxation to uninvolved muscles, which in turn will lead to a prolonged block in the involved muscles.

Today, it is generally accepted that depolarizing relaxants, especially succinylcholine, are contraindicated in patients with neuromuscular trauma or disorders. The reason for this is that depolarizing relaxants can cause a much higher release of potassium in the blood than would be found in normal persons.

Endotracheal intubation is mandatory for most surgical procedures involving the myasthenic patient in order to provide airway control and to remove secretions. Since respirations and/or cough mechanisms may be depressed for a few days postoperatively, either the endotracheal tube should be left in place or a tracheostomy performed before the actual removal of the tube. Though this is the general postoperative treatment, recently it has been found that if the patient is mentally alert, has good muscle tone, a tidal volume greater than 200 ml, a vital capacity of at least twice that of the tidal volume, and an inspira-
tory force of at least negative 11 cm/H$_2$O, there is no reason why the patient cannot be extubated and O$_2$ administered by face mask at an FIO$_2$ to maintain PaO$_2$ between 70-100 torr.$^{42}$

Caution must be taken when using Innovar® or frequent doses of fentanyl as the anesthetist may have to use a muscle relaxant to counteract chest wall spasm and the resulting apnea which is occasionally encountered with these agents.

Postoperatively, the readjustment of anticholinesterase therapy, pain relief, ventilation support and circulation support are the focal points. Myasthenia gravis patients should be placed in a respiratory care unit for a few days in order to receive necessary respiratory maintenance. Frequent blood gas monitoring is essential.

Circulation care should concentrate particular attention on cardiac function, blood volume, and fluids and electrolyte levels, with special attention to sodium and potassium.

Narcotics and small doses of tranquilizers can be employed for pain relief and to dispel restlessness. It must be kept in mind, however, that some narcotics are potentiated by anticholinesterase drugs.

Once a positive edrophonium test has been ascertained, anticholinesterase agents can be administered on an individual basis that will maintain adequate spontaneous respirations. Parasympatholytic drugs should be administered as needed.

**Myasthenic syndrome**

A condition has been reported that resembles an association between myasthenia gravis and bronchial carcinoma. Superficially, this syndrome resembles myasthenia gravis, but closer investigation shows marked differences.

The main symptom is peripheral muscle weakness which develops in patients with bronchial carcinoma. The major diagnostic tool for this syndrome is the electromyograph. The specific features$^{43}$ are:

1. Low voltage potential on twitch stimulation.
2. The “fade” of successive responses on twitch stimulation.
3. The “growth” of successive responses on tetanic stimulation.
4. The presence of post-tetanic facilitation.

Patients exhibiting this syndrome are sensitive to both depolarizing and nondepolarizing muscle relaxants; the use of all types of relaxants is contraindicated. Patients with myasthenic syndrome are almost entirely men, commonly 50 to 70 years of age. They generally have reduced or absent reflexes of tendons, and show post-tetanic facilitation. Small cell carcinoma of the bronchus is always present (but some cases have been reported with other tumors). There is a non-specific degeneration of nerve fibers and endplates, sensitivity to all types of relaxants, and an infrequent weakness of ocular and other cranial muscles. In addition, muscle pain is common, and there is a poor response to neostigmine.$^{44}$ The prognosis for this condition is poor and one can expect rapid deterioration and death.

**Conclusion**

Although a relatively uncommon disease, myasthenia gravis may be encountered by the anesthetist in the course of performing the diagnostic tests involved with such patients. This encounter may rise unexpectedly as in an emergency situation. Therefore, to ensure that these patients have optimal care, the anesthetist should be familiar with the disease and the profound effects most agents used in anesthesia practice can have upon it.

**REFERENCES**


(10) Wylie and Churchill-Davidson. Pg. 910-911.


(30) Wylie and Churchill-Davidson. pg. 914.


(36) Wylie and Churchill-Davidson. pg. 919.


(40) Wylie and Churchill-Davidson. pg. 808.


(43) Wylie and Churchill-Davidson. pg. 922.

(44) Wylie and Churchill-Davidson. pg. 923.
ADDITIONAL REFERENCES


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