Aquired Neuromuscular Junction Disorders

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NMJ Disorders

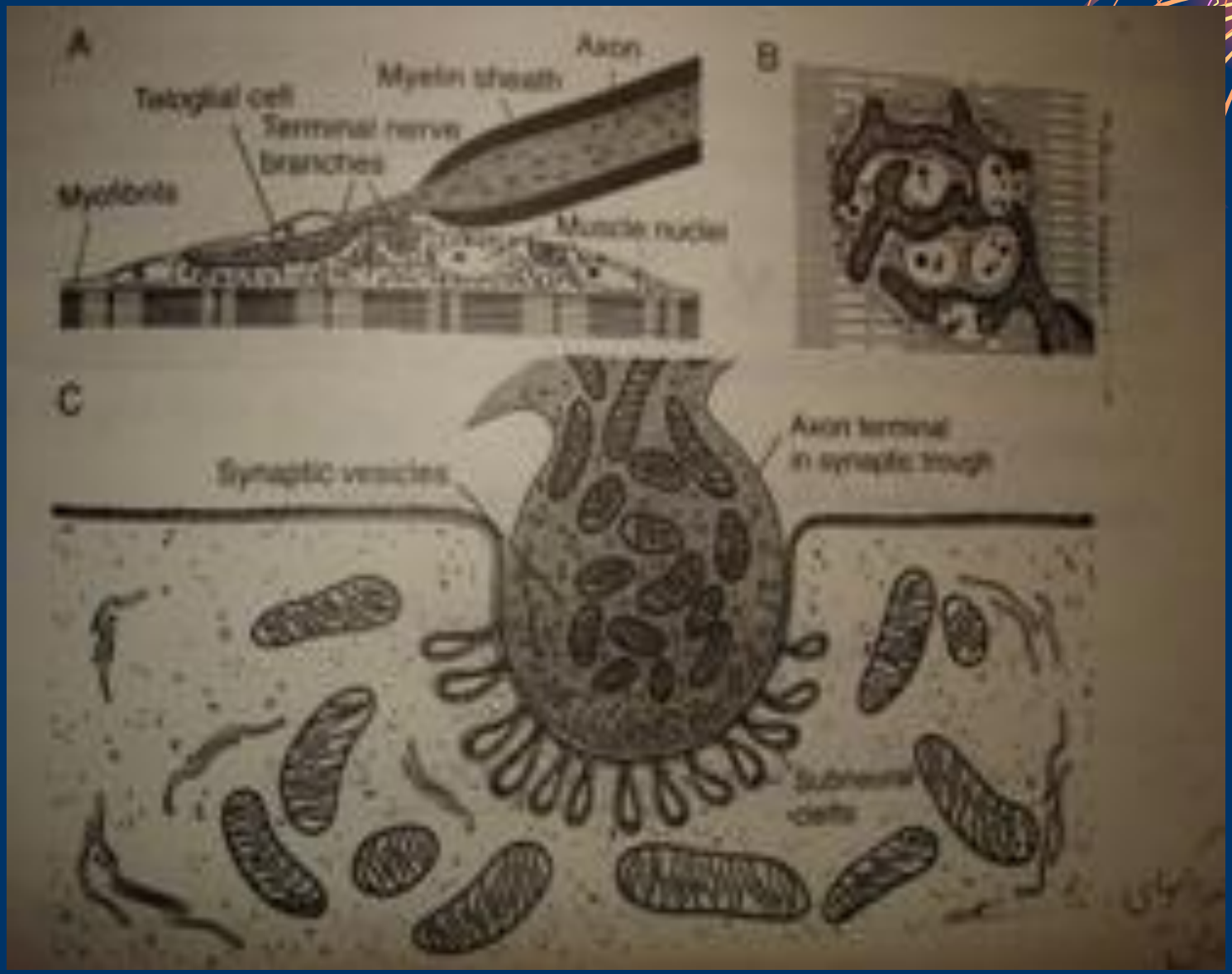
- **NMJ disorders can be classified into immune-mediated, toxic or metabolic, and congenital syndromes.**
- **Immune-Mediated Disorders**
- Myasthenia gravis
- Lambert-Eaton myasthenic syndrome
- **Toxic/Metabolic Disorders**
- Botulism
- Snake venom poisoning
- Organophosphates, insecticide poisoning (e.g., as from
- malathion, parathion), Hypermagnesemia
- **Congenital Myasthenic Syndromes**
- Presynaptic Choline acetyltransferase deficiency (ChAT).
- MuSK deficiency, Myasthenic syndrome associated centronuclear myopathies

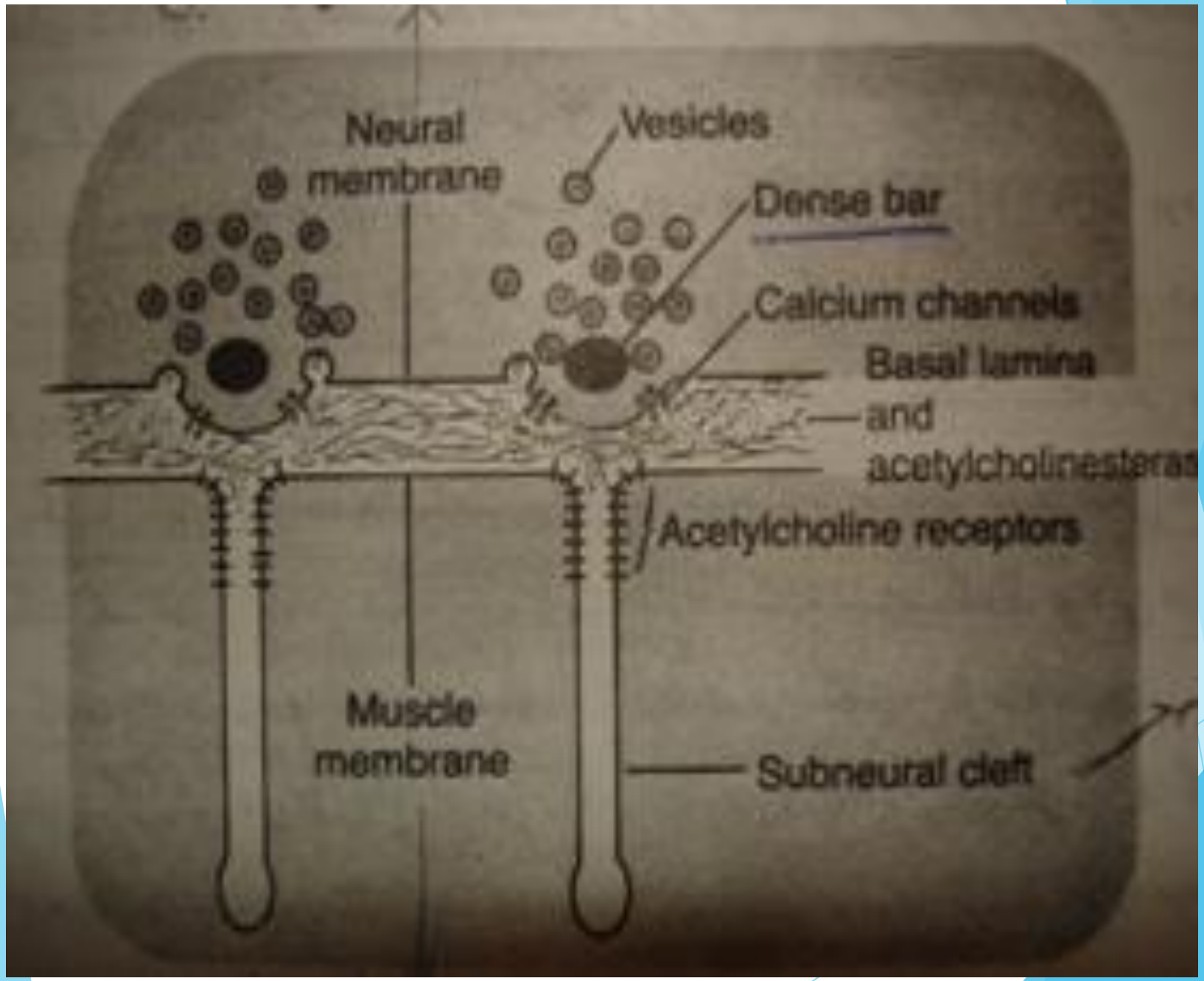
Postsynaptic Disorders

Myasthenia Gravis:

- MG, the best understood of all the autoimmune diseases, is caused by an immunoglobulin G (IgG)–directed attack on the NMJ, aimed specifically at the nicotinic acetylcholine receptor (ACHR) in the vast majority of cases.

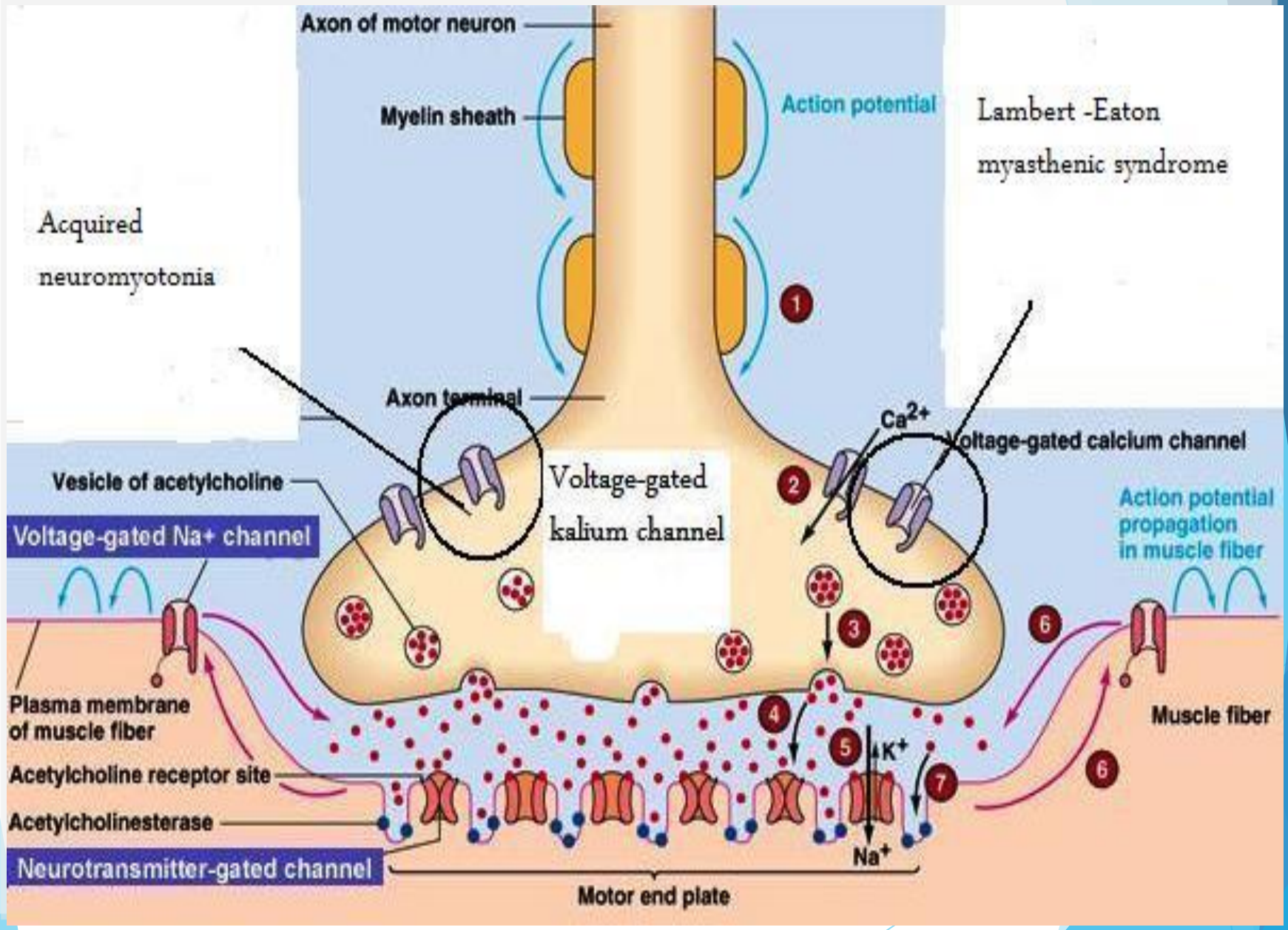
Physiology of NMJ




Neural membrane
Vesicles
Dense bar
Calcium channels
Basal lamina
and
acetylcholinesteras
Acetylcholine receptors
Muscle membrane
Subneural cleft

- The role of these anti-ACHR antibodies as the cause of MG has been proved through a variety of experimental steps: (1) antibodies are present in the serum of most patients with MG; (2) antibodies passively transferred to animals produce experimental myasthenia; (3) removal of antibodies allows recovery; and (4) immunization of animals with ACHRs produces antibodies and can provoke an autoimmune disease that closely resembles the naturally occurring disease.

- First, binding of the antibody to the receptor can directly block the binding of acetylcholine (ACH). Second, there is a complement-directed attack, with destruction of the ACHR and postjunctional folds. Last, antibody binding can result in an increase in the normal removal of ACHRs from the postsynaptic membrane (modulation).
- Thus, although the amount of ACH released is normal, there is reduced binding of ACH to the ACHR, resulting in a smaller endplate potential and a reduced safety factor of NMJ transmission.


Axon of motor neuron
Myelin sheath
Action potential
Lambert -Eaton
myasthenic syndrome
Acquired
neuromyotonia
1
Axon terminal
Ca2+
Voltage-gated calcium channel
Vesicle of acetylcholine
Voltage-gated
kalium channel
2
Voltage-gated Na+ channel
Action potential
propagation
in muscle fiber
3
6
Plasma membrane
of muscle fiber
4
Muscle fiber
5
K+
Acetylcholine receptor site
6
7
Acetylcholinesterase
Na+
Neurotransmitter-gated channel
Motor end plate

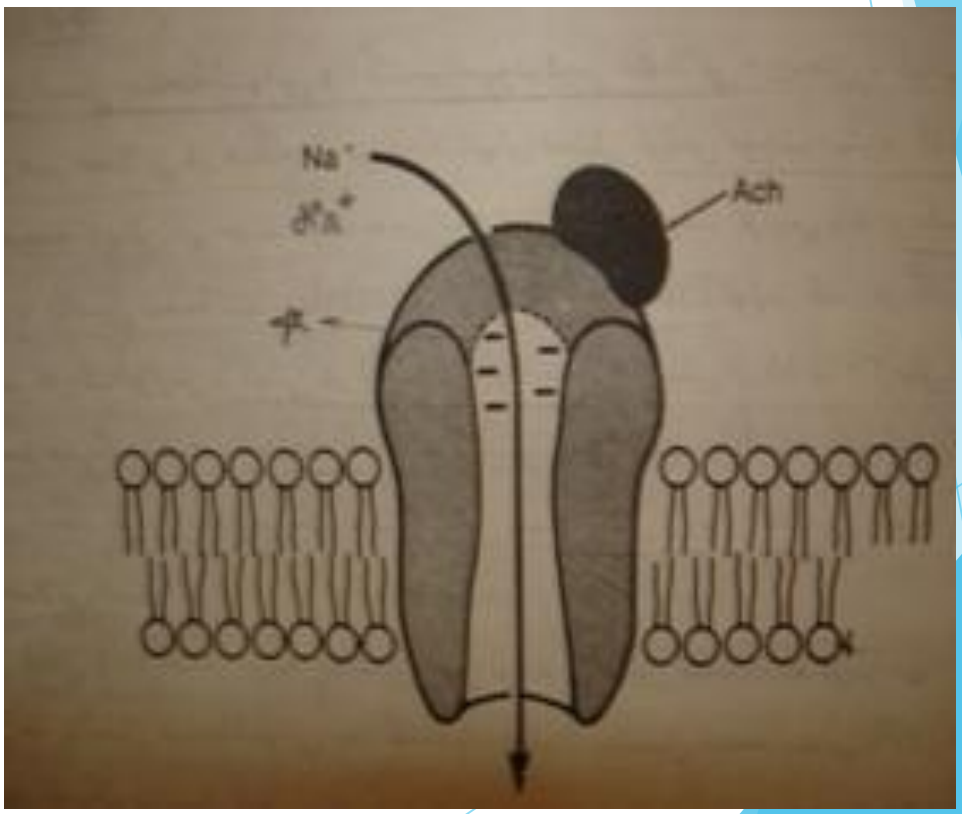
EPP is the potential generated at the postsynaptic membrane following a nerve action potential and neuromuscular transmission. Since each vesicle (quantum) released causes a 1 mV change in the postsynaptic membrane potential, this results in about 60 mV change in the amplitude of the membrane potential.

Safety factor. The safety factor is simply defined as the difference between the EPP and the threshold potential for initiating an action potential.

In normal conditions, the number of quanta (vesicles) released at the NMJ at the presynaptic terminal (about 60 vesicles) far exceeds the postsynaptic membrane potential change required to reach the threshold needed to generate a postsynaptic muscle action potential (7 to 20 mV). Hence, a nerve action potential results in an EPP that always reaches threshold and results in an all-or-none muscle fiber action potential (MFAP) . Also, the safety factor prevents NMJ failure despite repetitive action potentials.

Quantal release, AchR conduction properties, AchR density, and AchE activity contribute to the EPP.

- Postsynaptic folds form a high resistance pathway that focuses end plate current flow on voltage-gated sodium channels concentrated in the depths of the folds. Both these factors reduce the action potential threshold at the end plate and serve to increase the safety factor. Human junctions are smaller and with more extensive folding than other mammals, suggesting an evolutionary pressure towards postsynaptic modifications to enhance safety factor. All disorders of neuromuscular transmission are characterized by a compromise of the safety factor.



- A subset of patients with MG clinically (approximately 8%-15%) will not demonstrate antibodies to ACHR (so- called "seronegative" cases).
- In this subset, however, approximately 40%–50% will have an antibody to muscle-specific tyrosine kinase (MuSK). MuSK is a surface receptor involved in the clustering of ACHRs during development.
- Other rare seronegative patients have antibodies to the low-density lipoprotein receptor–related protein 4 (LRP4).
- LRP4 binds with agrin (another NMJ protein) to form a complex that assists with activation of MuSK.
- Patients without antibodies to ACHR, MuSK, and LRP4 are referred to as "triple seronegative."

Clinical findings

- Muscle fatigue and weakness
- In patients with antibodies to ACHR, myasthenic weakness characteristically affects the extraocular, bulbar, or proximal limb muscles.
- Eye findings are the most common, with ptosis and extraocular muscle weakness occurring In more than 50% of patients at the time of presentation and developing in more than 90% of patients sometime during their illness.

- Extraocular weakness frequently begins asymmetrically, with one eye involved and the other spared.
- A very small degree of extraocular weakness is experienced by the patient as visual blurring or double vision.
- Myasthenic weakness has been known to mimic third, fourth, and sixth nerve palsies and, rarely, an intranuclear ophthalmoplegia.
- **Unlike true third nerve palsies, however, MG never affects papillary function.**
- Fixed extraocular muscle weakness may occur late in the illness, especially if untreated.

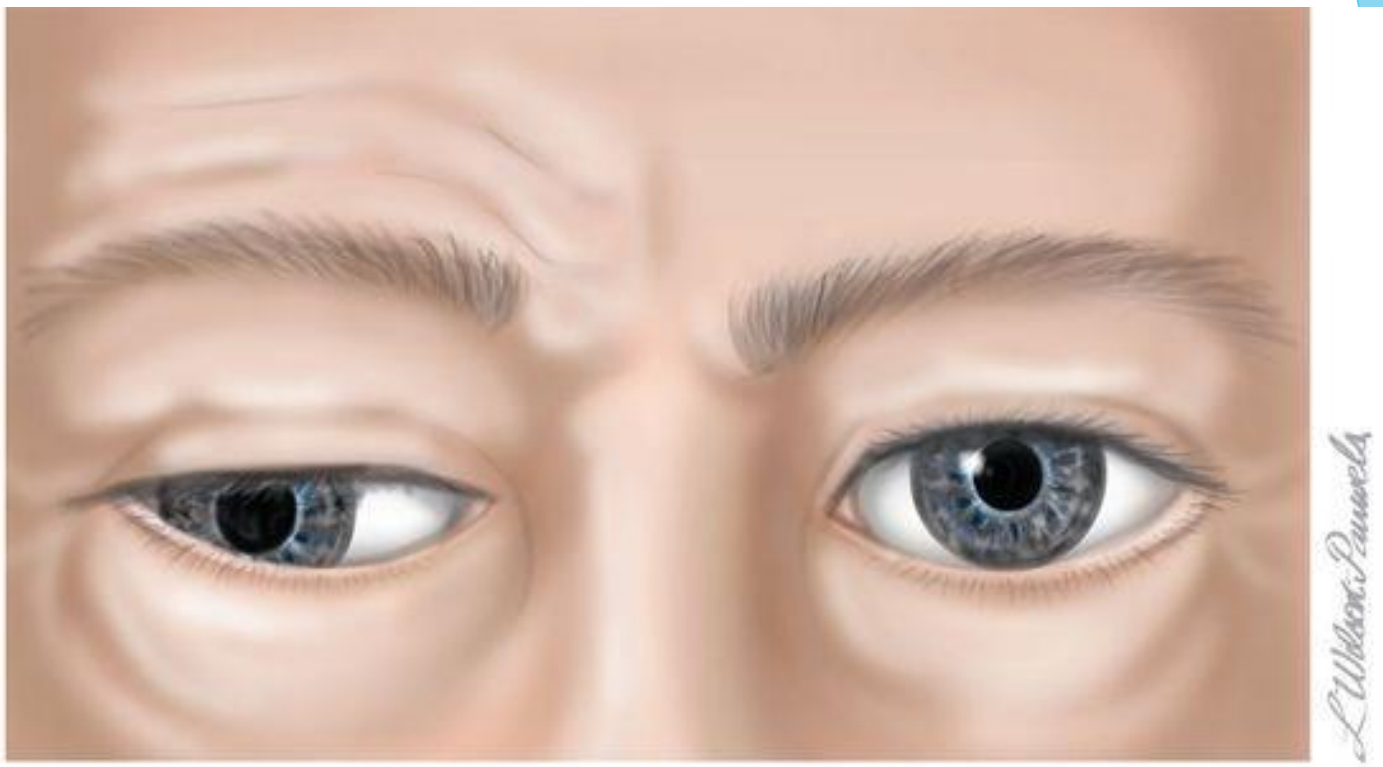


Figure III–13 Appearance of the eyes in right third nerve palsy.
The right side of Werner's face illustrates: a wrinkled brow due to the inability to raise the right eyelid; ptosis of his right eye lid due to the inactivation of the levator palpebrae superioris muscle; dilation of his right pupil due to the decreased tone of the constrictor pupillae muscle; and downward and outward movement of his right eye due to the unopposed action of the right superior oblique and lateral rectus muscles.

- Bulbar muscle weakness is next most common after extraocular weakness. This may result in difficulty swallowing, chewing, and speaking. Patients may develop fatigability and weakness of mastication, with the inability to keep the jaw closed after chewing.
- Myasthenic speech is nasal (from weakness of the soft palate) and slurred (from weakness of the tongue, lips, and face) but without any difficulty with fluency. Weakness of the soft palate may also resulting nasal regurgitation (i.e., liquid coming out the nose when drinking). When myasthenic patients develop limb weak it usually is symmetric and proximal.

- Patients note difficulty getting up from chairs, going up and down stairs, reaching with their arms, or holding up their head.
- Rare patients present with an isolated limb- girdle form of MG and never develop eye movement or bulbar muscle weakness.
- It is these patients who are most often misdiagnosed with myopathy.

- In contrast to the clinical syndrome seen in MG with anti- ACHR antibodies, the clinical characteristics of patients
- with anti- MuSK antibody–associated MG include female predominance, prominent bulbar, neck, shoulder and respiratory involvement, and a severe presentation that occurs at a younger age than MG associated with anti-ACHR antibodies.

- Three clinical patterns are present in anti-MuSK antibody–associated MG: (1) severe oculobulbar weakness along with tongue and facial atrophy, and tongue fasciculations;(2) marked neck, shoulder,and respiratory weakness with little or no ocular weakness; and (3) a pattern similar to anti-ACHR antibody–associated MG.
- In addition, patients With anti-MuSK antibody-associated MG are often unresponsive or intolerant to cholinesterase inhibitors, and some actually worsen.

- The distinguishing clinical feature of MG, whether seropositive (ACHR,MuSK, LRP4) or seronegative, is pathologic fatigability (i.e., muscle weakness that develops with continued
- use).
- Patients improve after rest or upon rising in
- the morning and worsen as the day proceeds. Although generalized fatigue is common in many neurologic and nonneurologic disorders, NMJ fatigue is limited to muscular fatigue
- alone, which progresses to frank muscle weakness with use.

- Patients with MG do not generally experience a sense of mental fatigue, tiredness, or sleepiness.
- To demonstrate subtle weakness, it is helpful to observe the patient performing functional tasks, such as rising from a chair or from the floor
- or walking, rather than relying on manual muscle strength testing alone.

- Pathologic fatigability may be demonstrated by having the patient look up for several minutes (to determine If ptosis or extraocular weakness Is present),
- count aloud to 100 (to determine if nasal or slurred speech is present),or by repetitively testing the neck or the proximal limb muscles (for example, with both shoulders abducted, the examiner repetitively pushes down on both arms several times, looking for fatigable weakness).

- Ice is applied over the forehead for several minutes to cool the underlying muscles. In MG, ptosis may improve markedly with cooling.
- The remainder of the neurologic examination should be normal. Deep tendon reflexes
- are generally preserved or,If reduced, are reduced in proportion to the degree of muscle
- weakness.

More recently, MG has been seen as a rare complication in cancer patients treated with immune checkpoint inhibitors (ICPIs). These are monoclonal antibodies that target cytoplasmic T-lymphocyte associated antigen-4 (CTLA-4), programmed cell death receptor-1 (PD-1), or programmed cell death ligand-1 (PD-L1). These agents inhibit normal nity. Although they are highly effective as immunotherapy in several types of refractory cancers, they can result in a large number of immune-related adverse effects (irAEs), including several neuromuscular conditions. Among these are MG, myositis, Guillain-Barré syndrome, and chronic inflammatory demyelinating polyneuropathy. In patients who develop MG, the ACHR antibodies are typically positive (although some are negative) and the electrophysiology is similar to that seen in sporadic autoimmune MG. In our experience, MG provoked by ICPIs is often associated with myositis, is very severe, and can be very difficult to treat. Plasma exchange and high-dose steroids are usually required, in addition to immediate cessation of the involved drug.

Autoimmune MG may be seen in two other groups of patients aside from those with idiopathic autoimmune myasthenia. First, transient neonatal MG may occur in babies born to mothers with MG. This occurs when maternal autoantibodies pass through the placenta, resulting in the same clinical syndrome in newborn infants. The illness usually is mild and self-limited and disappears after the first few months of life as the maternal antibodies are degraded. MG may also be seen in patients treated with penicillamine. The clinical syndrome is similar to idiopathic MG, including the presence of anti-ACHR antibodies, except that most patients slowly improve once the penicillamine has been discontinued.

Electrophysiologic Evaluation

- In any patient suspected of having MG, routine motor and sensory nerve conduction studies cannot be omitted. Normal CMAP amplitudes are an important and expected finding in MG, in direct contrast to LEMS, wherein baseline CMAPs usually are diffusely low.
- In only a small number of patients with MG (3%–15%), the baseline CMAPs at rest are below the normal range.

- A decrement on RNS can be seen in various denervating conditions (e.g., neuropathies, motor neuron disorders, inflammatory myopathies) and myotonic disorders, in addition to primary disorders of the NMJ.
- For instance, a decrement on RNS of the ulnar nerve may be seen in a severe ulnar neuropathy with denervation;such a finding in this context does not imply a primary NMJ disorder.

Repetitive Nerve Stimulation

After the routine nerve conduction studies are completed, RNS studies are performed. These studies are abnormal in more than 50%–70% of patients with

- Generalized MG but often are normal in patients with the restricted ocular
- Form of MG.

- A decremental response on RNS is the electrical correlate of clinical muscle fatigue and weakness.
- In normal subjects, slow RNS (3 Hz) results in little or no decrement of the CMAP, whereas in MG, a CMAP decrement of 10% or more is characteristically seen. Both distal and proximal nerves should
- be tested. Although distal nerves are technically easier to study, the diagnostic yield increases with stimulation of proximal nerves (e.g., spinal accessory or facial nerves).
- This is not unexpected, because the proximal muscles usually are much more involved clinically than the distal ones.

- Facial RNS is especially important to perform in suspected anti-MuSK
- antibody–associated MG, wherein the
- Yield of finding an abnormal decrement is much higher when examining a facial muscle than a limb muscle.
- This likely reflects the severe facial
- and bulbar involvement in some patients with anti-MuSK antibody–associated MG.

Exercise Testing

Exercise testing should be routinely used with all RNS studies (see Chapter 6). If there is no significant decrement on RNS studies at baseline (<10% decrement), the patient should perform 1 minute of exercise, followed by RNS at 1-minute intervals for the next 3–4 minutes, looking for a CMAP decrement secondary to postexercise exhaustion. If at any time, either at baseline or following exercise, a significant decrement develops, the patient should perform a brief 10-second maximum isometric contraction, immediately followed by slow RNS, looking for an increment in the CMAP and "repair" of the decrement secondary to postexercise facilitation (Fig. 37.1).

Box 37.2 Electrophysiologic Evaluation of Myasthenia Gravis

1. **Routine motor and sensory nerve conduction studies.** Perform routine motor and sensory nerve conduction studies, preferably a motor and sensory nerve in one upper and one lower extremity. CMAP amplitudes should be normal. If CMAP amplitudes are low or borderline, repeat distal stimulation immediately after 10 seconds of exercise to exclude a presynaptic NMJ transmission disorder (e.g., Lambert-Eaton myasthenic syndrome).
2. **Repetitive nerve stimulation (RNS) and exercise testing.** Perform slow RNS (3 Hz) on at least one proximal and one distal motor nerve. Always try to study weak muscles. If any significant decrement (>10%) is present, repeat to ensure that the decrement is reproducible. If there is no significant decrement at baseline, exercise the muscle for 1 minute, and repeat RNS at 1, 2, 3, and 4 minutes looking for a decrement, secondary to post-exercise exhaustion. If at any time a significant decrement is present (at baseline or following postexercise exhaustion), exercise the muscle for 10 seconds and immediately repeat RNS, looking for postexercise facilitation (repair of the decrement).

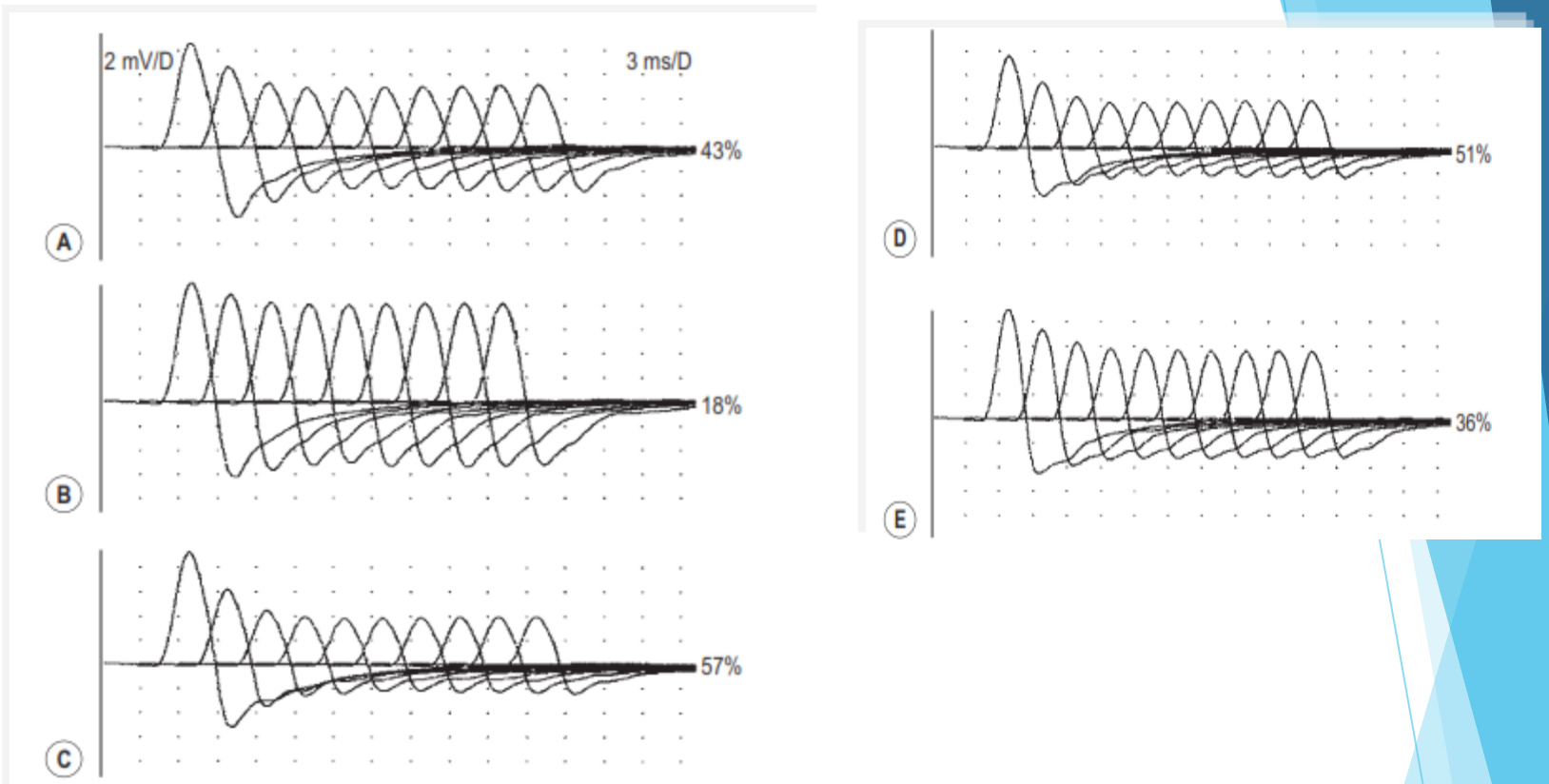


Fig. 37.1 Repetitive nerve stimulation (3 Hz) in myasthenia gravis. Stimulating the ulnar nerve at the wrist, recording the first dorsal interosseous. Maximal decrement noted to right of traces. (A) Baseline. (B) Immediately after 10 seconds of exercise (postexercise facilitation). (C, D) 2 and 3 minutes after 60 seconds of exercise (postexercise exhaustion). (E) Immediately after 10 seconds of exercise again (postexercise facilitation and partial repair of the decrement).

Needle electromyography (EMG). Perform routine needle EMG of distal and proximal muscles, especially weak muscles. Patients with moderate to severe myasthenia gravis may display unstable or short, small, polyphasic motor unit action potentials. Recruitment is normal or early. Needle EMG must exclude severe denervating disorders or myotonic disorders, which may display an abnormal decrement on RNS.

Single-fiber EMG (SF-EMG). If the previous items are normal or equivocal in a patient strongly suspected of having myasthenia gravis, perform SF-EMG in the extensor digitorum communis and, if necessary, one other muscle, looking for jitter and blocking. It is always best to study a weak muscle. Normal SF-EMG in a clinically weak muscle excludes an NMJ disorder.

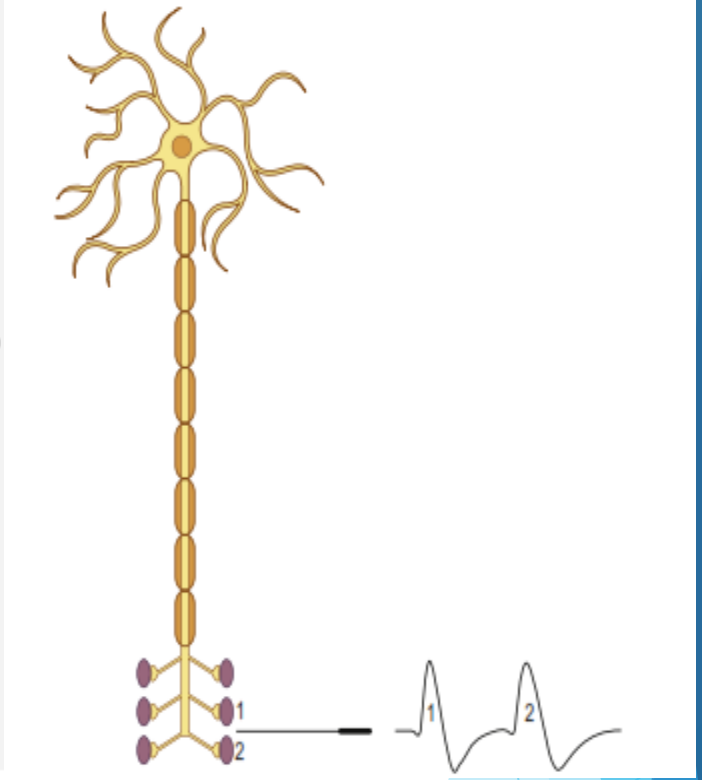
Table 37.1 Clinical Characteristics of Neuromuscular Junction Disorders.

Disorder	Temporal Onset	Ocular Sx	Bulbar Sx	Reflexes	Autonomic Sx	Sensory Sx	GI Sx
Myasthenia gravis	Subacute	+	+	Normal[a]	–	–	–
Lambert-Eaton myasthenic syndrome	Subacute	+/–	+/–	Reduced	+/–	+/–	–
Botulism	Acute	+	+	Normal[a]	+	–	+
Congenital myasthenia	Congenital or pediatric	+	+/–	Normal[a]	–	–	–

+, Commonly present; +/–, may be seen occasionally; –, usually not present; *GI*, gastrointestinal; *Sx*, symptoms/signs.
[a]May be reduced in proportion to the degree of muscle weakness.

Single fiber EMG

- When a motor axon is depolarized, the action potential normally travels distally and excites all the muscle fibers within that moto unit at more or less the same time.
- This variation in the time interval between the firing of adjacent Single muscle fibers from the same Motor unit is termed Jitter and primarily reflects variation in NMJ transmission time.



- If the NMJ is compromised, the time it takes for the endplate potential to reach threshold is prolonged, which results in greater-than- normal variation between firing of adjacent muscle fibers.
- If the prolongation is severe enough, the muscle fiber may never reach action potential, resulting in blocking of the muscle fiber.

- SF- EMG is used to measure the relative firing of adjacent single muscle fibers from the same motor unit and can detect both prolonged jitter as well as blocking of muscle fibers. It is important to note that, whereas the clinical correlate of blocking is muscle weakness, there is no clinical correlate to increased jitter. Thus the main advantage

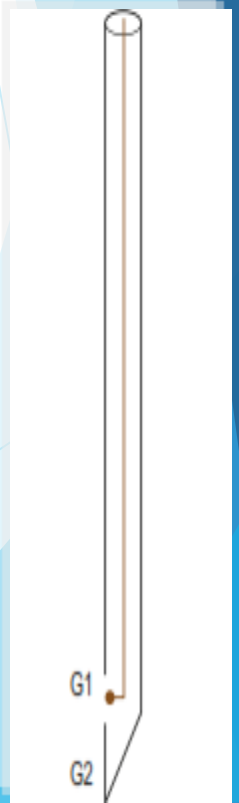
- Thus the main advantage of SF-EMG over RNS is that the single- fiber study may be abnormal, showing increased jitter, even in patients without overt clinical weakness.
- In contrast, for RNS studies to be abnormal, the NMJ disorder must be sufficiently severe that blocking (the electrophysiologic correlate of weakness) also occurs, leading to a decremental response.

- SF- EMG is best reserved for those electromyographers who are well trained in its use and who perform SF- EMG on a routine basis.
- It is a technically demanding procedure for both the patient and the electromyographer. In contrast to routine EMG, usually only one or two muscles are studied.
- Often, the extensor digitorum communis muscle in the forearm is selected for study. For most patients, this muscle can be steadily activated for a prolonged period and is relatively free of age- related changes.

- A normal single- fiber examination of a clinically weak muscle effectively rules out the diagnosis of MG.
- The goal of SF- EMG is to study two adjacent single muscle fibers, known as a pair, from the same motor unit.
- This is accomplished by first changing
- The filters on the EMG machine.
- The low-frequency filter (high-pass)
- is increased to either 500 or 1000 Hz (normally 10 Hz in routine EMG).

- By using a high- pass filter of 500 or 1000 Hz, the amplitudes of distant muscle fiber potentials are attenuated while those of the nearby fibers are preserved.
- The dedicated SF- EMG needle
- is a specially constructed needle ,The active electrode *(G1) is located in a port along the posterior shaft of the needle*,
- with a smaller leading surface area than a conventional concentric needle electrode. The reference electrode *(G2) is the needle shaft*.

- result of these two modifications is that single- fiber muscle action potentials are recorded only if they are within 200–300 μm of the needle. The needle is placed in the muscle, and the patient is asked to
- activate the muscle in an even and constant fashion.
- The needle is moved until a single muscle fiber potential is located.



- With this single muscle fiber potential triggered on a delay line, the needle is slightly and carefully moved or rotated to look for a second potential that is time locked to the first potential (signifying that it is from the same motor unit).
- SF- EMG is technically demanding.
- The optimal single-fiber potentials are those in which the amplitude is at least
- 200 μV in amplitude and the rise time is less than 300 μs.

- If a time- locked second potential is located, an interpotential
- interval between the two potentials (i.e., the pair) can be measured.
- By recording multiple consecutive firings of the muscle fiber action
- Potential pairs, the difference
- Between consecutive interpotential intervals can be calculated.
- This variation between consecutive interpotential intervals is the jitter.

- By recording 50–100 subsequent potentials, the mean consecutive difference (MCD), a measure of jitter, can be calculated between the triggered potential and the time- locked second single muscle fiber potential. Most modern EMG machines have programs that automatically perform the MCD calculation.
- This procedure is then repeated until 20 separate single- fiber pairs are collected, to calculate a mean MCD. This value is compared with the normal mean MCD for the muscle. There is also an upper limit of normal jitter for an individual pair, based on the muscle studied and the patient's age.

- ***Voluntary single fiber electromyography*:**
- **The most commonly used method: the patient activates and maintains the firing rate of the motor unit. This technique is not possible if the patient cannot cooperate (e.g., child, dementia, coma, severe weakness), and is difficult if the patient is unable to maintain a constant firing rate (e.g., tremor, dystonia, spasticity).**
- **With minimal voluntary activation, the needle is positioned until two muscle potentials (a pair) from a single motor unit are recognized. When a muscle fiber pair is identified, one fiber triggers the oscilloscope (triggering potential) and the second precedes or follows the first (slave potential). With voluntary activation, fifty consecutive discharges of a single pair is recorded. The interpotential interval (IPI) of the pair is then measured and a *mean consecutive difference (MCD or jitter)* of that pair is calculated as follows:**

$$\mathrm{MCD} = \frac{(\mathrm{IPI1} - \mathrm{IPI2}) + (\mathrm{IPI2} - \mathrm{IPI3}) + \ldots + (\mathrm{IPIN} - 1 - \mathrm{IPIN})}{\mathrm{N} - 1}$$

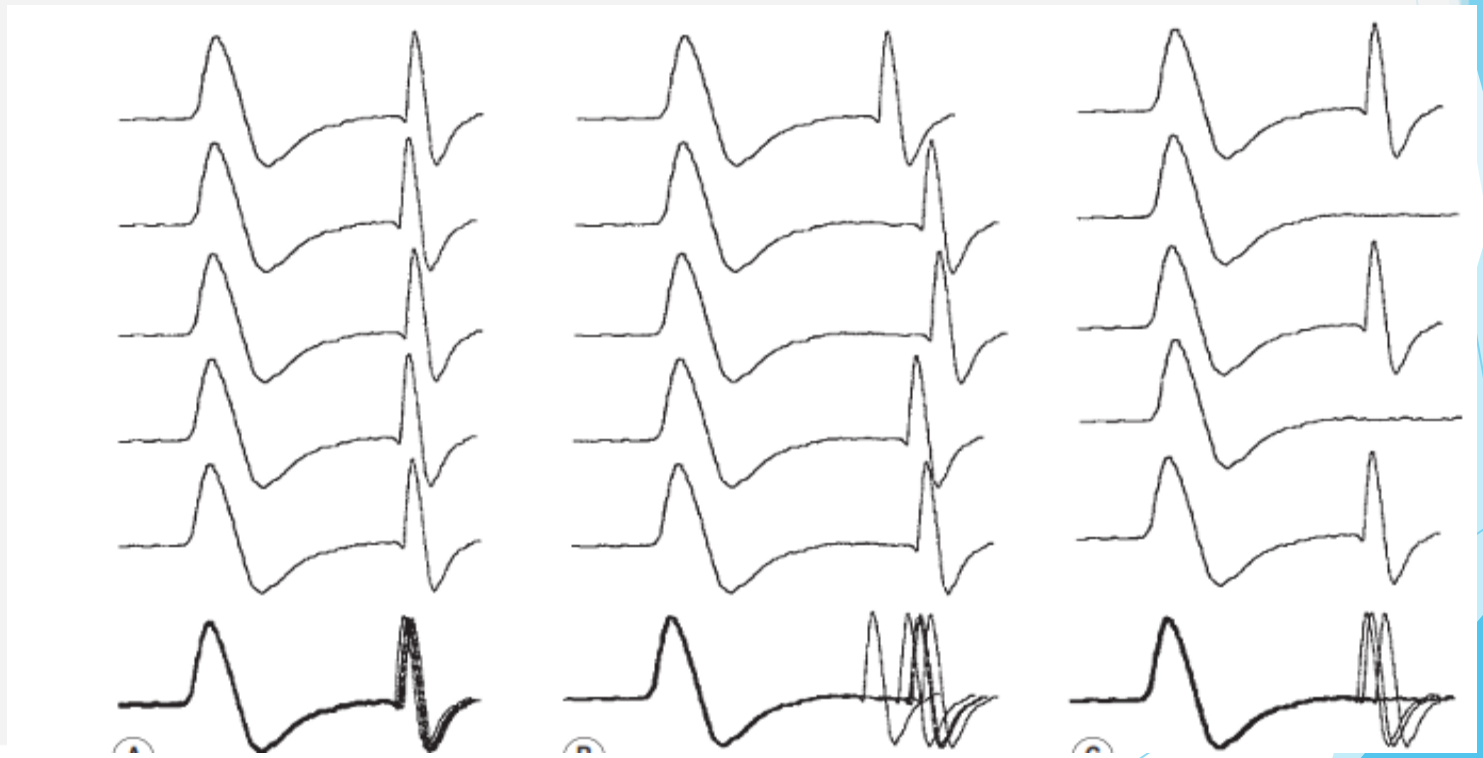
- the upper limit of jitter of a muscle to be deemed abnormal, more

  than 10% of the pairs must exceed the upper limit of normal (e.g., for 20 pairs, at least two must be abnormal).
- To make a diagnosis of an NMJ disorder:
- either the mean jitter must be abnormal
- or the upper limit of normal jitter

  must be abnormal in more than 10% of individual pairs.

- However, in most NMJ disorders, both measures will be abnormal. Increased jitter is consistent with an NMJ disorder (Fig.37.4).
- In addition to increased jitter, Blocking may be seen on SF-EMG. Two time-locked, single-fiber muscle potentials from the same motor unit normally fire together.
- If the triggered potential fires steadily while the second potential fires only intermittently, blocking is occurring.

- Blocking, which is another marker of NMJ disease, usually occurs only when the jitter is markedly prolonged
- (e.g., MCD > 80–100 μs).



this change. Despite the recording area from the smallest concentric needle being much larger than that of the dedicated SF-EMG needle, concentric needles have been found to be acceptable for SF-EMG studies. In one large multi-

- In one large multicenter study of normal subjects, the jitter of three muscles (orbicularis oculi, frontalis, extensor digitorum communis) was determined both for voluntary and stimulated
- Studies (Table 37.4).
- In general, the values for jitter are slightly
- smaller using a concentric EMG needle compared With a traditional SF-EMG needle.

Reference data for mean jitter in some muscles

Table 37.3 Reference Values for Jitter Measurements During Voluntary Muscle Activation.

Muscle	10 Years	20 Years	30 Years	40 Years	50 Years	60 Years	70 Years	80 Years	90 Years
Frontalis	33.6/49.7	33.9/50.1	34.4/51.3	35.5/53.5	37.3/57.5	40.0/63.9	43.8/74.1		
Orbicularis oculi	39.8/54.6	39.8/54.7	40.0/54.7	40.4/54.8	40.9/55.0	41.8/55.3	43.0/55.8		
Orbicularis oris	34.7/52.5	34.7/52.7	34.9/53.2	35.3/54.1	36.0/55.7	37.0/58.2	38.3/61.8	40.2/67.0	42.5/74.2
Tongue	32.8/48.6	33.0/49.0	33.6/50.2	34.8/52.5	36.8/56.3	39.8/62.0	44.0/70.0		
Sternocleidomastoid	29.1/45.4	29.3/45.8	29.8/46.8	30.8/48.8	32.5/52.4	34.9/58.2	38.4/62.3		
Deltoid	32.9/44.4	32.9/44.5	32.9/44.5	32.9/44.6	33.0/44.8	33.0/45.1	33.1/45.6	33.2/46.1	33.3/46.9
Biceps	29.5/45.2	29.6/45.2	29.6/45.4	29.8/45.7	30.1/46.2	30.5/46.9	31.0/48.0		
Extensor digitorum communis	34.9/50.0	34.9/50.1	35.1/50.5	35.4/51.3	35.9/52.5	36.6/54.4	37.7/57.2	39.1/61.1	40.9/66.5
Abductor digiti minimi	44.4/63.5	44.7/64.0	45.2/65.5	46.4/68.6	48.2/73.9	51.0/82.7	54.8/96.6		
Quadriceps	35.9/47.9	36.0/48.0	36.5/48.2	37.5/48.5	39.0/49.1	41.3/50.0	44.6/51.2		
Tibialis anterior	49.4/80.0	49.3/79.8	49.2/79.3	48.9/78.3	48.5/76.8	47.9/74.5	47.0/71.4	45.8/67.5	44.3/62.9

95% confidence limits for upper limit of mean jitter/95% confidence limits for jitter values of individual fiber pairs (us). This table was derived from data obtained

Reference data for voluntary and stimulated SFEMG

Table 37.4 Summary of Normal Jitter Values With a Concentric Needle.

	Muscle		
	Orbicularis Oculi	Frontalis	Extensor Digitorum
Voluntary SF-EMG			
Mean jitter (μs)	22.9	20.6	23.4
Mean jitter upper limit of normal (μs)	31	28	30
Upper limit of normal for any one pair (μs)	45	38	43
Stimulated SF-EMG			
Mean jitter (μs)	19.1	14.5	18.2
Mean jitter upper limit of normal (μs)	27	21	24
Upper limit of normal for any one fiber (μs)	36	28	35

From Stålberg E, Sanders DB, Ali S, et al. Reference values for jitter recorded with concentric needle electrodes in healthy controls: a multicenter study. Muscle Nerve. 2016;53(3):351–362.

SF EMG recording of a patient with MG dumitru data

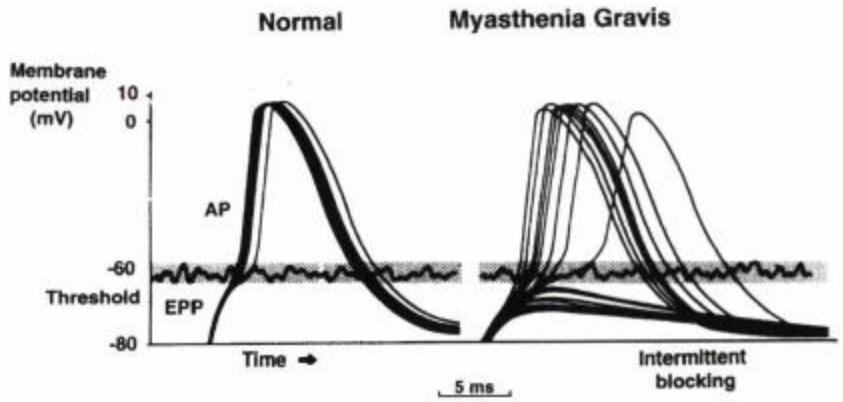


Table 25-5. Upper Limit of Normal Jitter Values (μs)[333,898]

Muscle	Largest Single Jitter Measurement	Mean
EDC	55	34
Biceps brachii	35	30
Deltoid	35	30
Frontalis	45	30
ADQ	60	49
TA	60	32.1
Rectus femoris	60	31

EDC, extensor digitorum communis; ADM, abductor digiti minimi; TA, tibialis anterior