# I need to rest every few minutes


## Case 2

*We Highly recommend you to have a look at the Phsiology and pharmacology lectures related to the case

**-Lecture (4) Indirect Cholinomimetics**

**-Lecture (6,7) Neuromuscular Transmission & Physiology of muscle contraction**

# Key points

- 30 years old; librarian-assistant.
- - She works in hospital library and she's responsible for placing books back to the bookshelves.
- - She feels tires, particularly after midday.
- - Recently, she noticed that she is unable to continue putting books, and she has to take rest every few minutes.
- - She noticed drooping of the end of her eyelid usually near the end of her work shift.
- - She said "my arms seem to become weak after placing few books".
- - Also, she said "in the morning I am much better, but I feel they are weak when I need to raise my arms".
- - She has difficult to climb the stairs.
- - These changes made her feel down and Frustrated.
- - She does not have shortness of breath or pain; also she does not have problems with swallowing and does not have tingling or numbness.

# New terms

- **Drooping**: to hang downward, pulled down from its normal place.
- **Tingling**: to have a sensation of slight prickles, stings, as from cold, a sharp blow or excitement.
- **Proximalmuscle**: Muscles closest to the center of the body (trunk) in anatomical position.
- **Numbness**: loss of physical sensation.
- **Frustrated**: disappointed.
- **Muscle wasting**: loss of muscle mass.
- **Muscle tone**: a continuous, passive, and partial contractionof skeletal muscles. in other words it is the muscle's resistance to passive stretch during resting state. It helps maintaining body posture.
- **Ptosis**: is a drooping or falling of the eyelid.
- **Serum anti-Acetylcholine Receptor (AChR) Antibodies**: Antibodies bind to AChR and prevent acetylcholine from doing its function which is stimulating the muscle contraction.
- **Cholinesterase inhibitors: (AntiCholinesterase )**: is a chemical that inhibits the acetylcholinesterase enzyme from breaking down acetylcholine and increasing the level and duration of action of the neurotransmitter acetylcholine

Clinical examination

Has no neurological and muscle problem

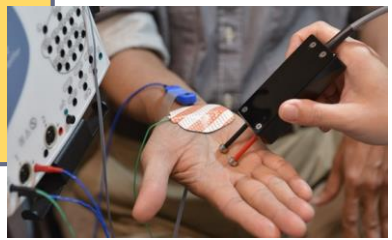
Eye eye ptosis and weakness of shoulder girdle and pelvic muscles

Blood test

Serum anti-acetylcholine receptor antibodies

EMG

Show decremental response in long thoracic nerve



Tensilon Test

Substantial improvement in weakness within a second

*For more Information about Tensilon Test

# Myasthenia Gravis

is an autoimmune disease which causes muscle paralysis because of inability of the neuromuscular junctions to transmit enough signals from the nerve fibres to muscle fibres. Pathologically antibodies that attack the acetylcholine receptors have been demonstrated in the blood of most patients with myasthenia gravis.

The antibodies destroy acetylcholine receptors at postsynaptic junction.

symptoms and signs :-

1- ptosis ( drooping of eyelid ) and diplopia (double vision )

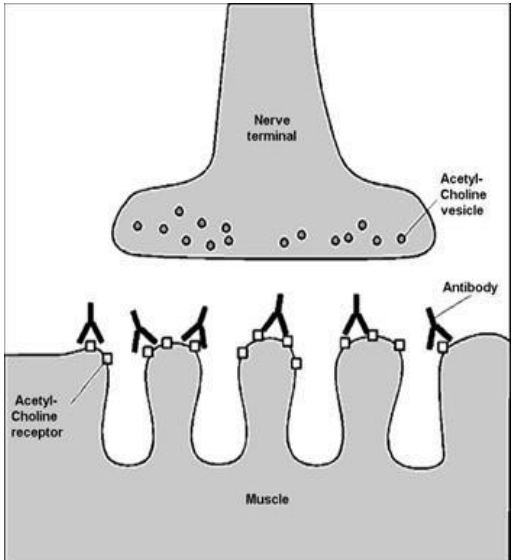
2-weakness of muscle in arm,hand,leg,and fingers

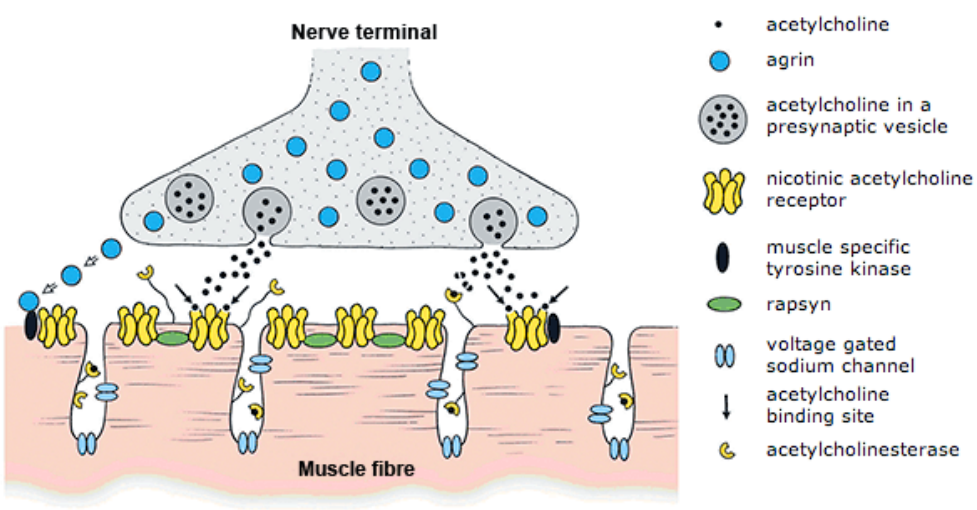
3-impaired speech

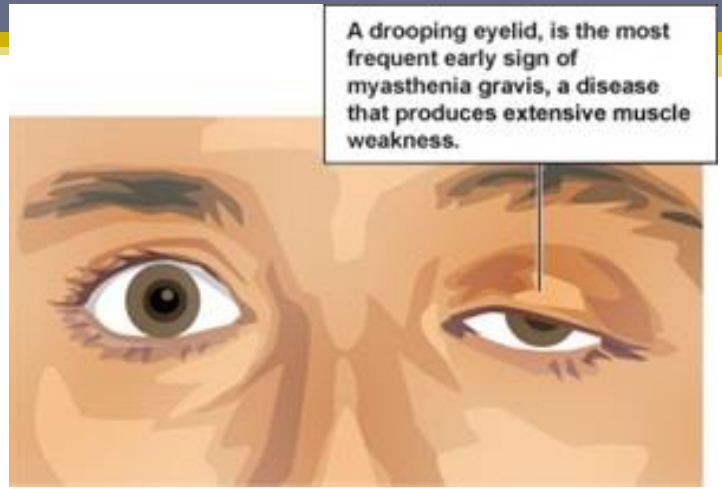
4- change facial expression

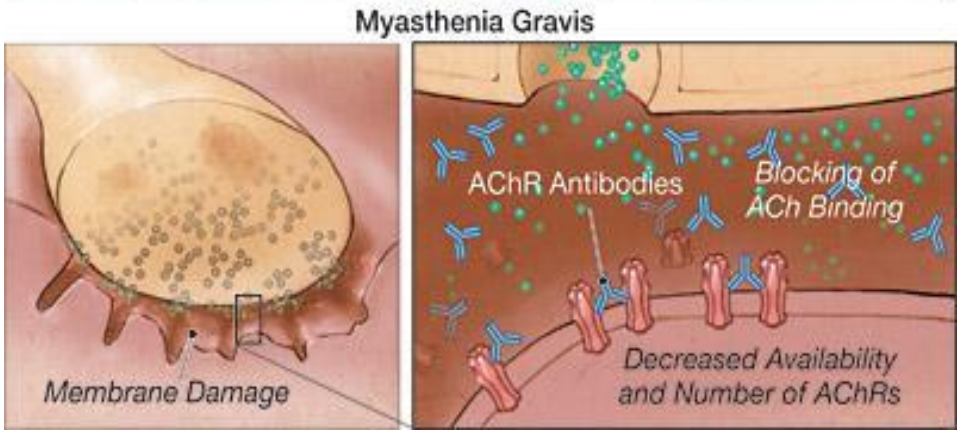
5- shortness of breath

http://www.youtube.com/watch?v=fTrWasKmHxY
more information about myasthenia gravis





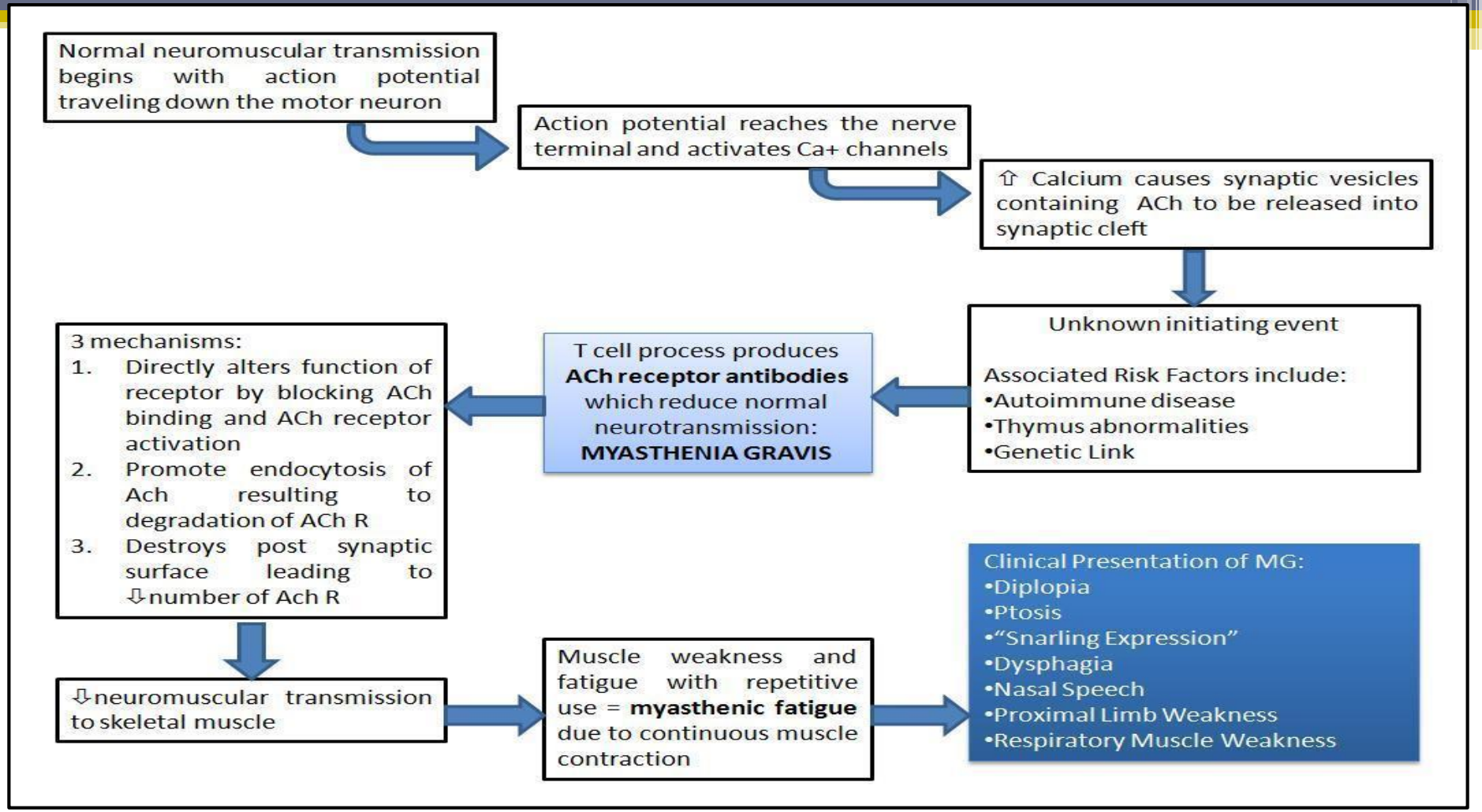




## Treatment by :

1- cholinesterase inhibitory drugs such as neostigmine or pyridostigmine

2- immunosuppression


Normal neuromuscular transmission begins with action potential traveling down the motor neuron
Action potential reaches the nerve terminal and activates Ca+ channels
⇧ Calcium causes synaptic vesicles containing ACh to be released into synaptic cleft
Unknown initiating event
Associated Risk Factors include:
•Autoimmune disease
•Thymus abnormalities
•Genetic Link
T cell process produces ACh receptor antibodies which reduce normal neurotransmission: MYASTHENIA GRAVIS
3 mechanisms:
1. Directly alters function of receptor by blocking ACh binding and ACh receptor activation
2. Promote endocytosis of Ach resulting to degradation of ACh R
3. Destroys post synaptic surface leading to ⇩number of Ach R
⇩neuromuscular transmission to skeletal muscle
Muscle weakness and fatigue with repetitive use = myasthenic fatigue due to continuous muscle contraction
Clinical Presentation of MG:
•Diplopia
•Ptosis
•"Snarling Expression"
•Dysphagia
•Nasal Speech
•Proximal Limb Weakness
•Respiratory Muscle Weakness

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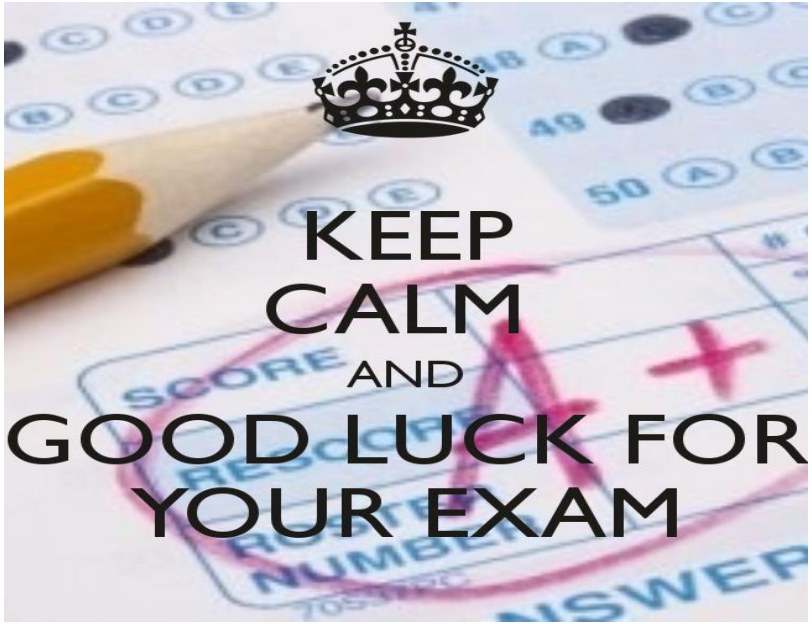


*Please if you have any complain please don't hesitate to tell us

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