PHYSIOLOGY OF SOMATIC MOTOR SYSTEM
MUSCLES - BODY MOVING SYSTEM

STRIATED

1. SKELETAL MUSCLES
Muscles enclosed in a connective tissues, that ends with a tendon
Contain hundreds of muscle cells – muscle fibers
Innervation by axons of spinal motoneurons
Enable voluntary control of movement and behavior

2. CARDIAC
Contracts in the absence of any innervation (PACEMAKERS)
Autonomic nervous system enhances (sympathetic) or
slows down (parasympathetic) the heart rate

SMOOTH

digestive tract – control of peristalsis
arteries – control of peripheral resistance and blood pressure
iris – control of light passing on to the retina
SPINAL CORD – SPINAL NERVES

SPINAL NERVES – 20 PAIRS

CAUDA EQUINA – 11 PAIRS

SPINAL NERVES INNERVATE THE PARTS OF THE BODY ACCORDING TO SPINAL SEGMENTS
SPINAL CORD – SPINAL NERVES

Cervical

C1-C3  Neck Muscles
C4    Diaphragm
C5    Deltoid (shoulder)
C6    Wrist
C7    Triceps
C7-C8  Fingers

Thoracic

T1    Hand
T2-T12  Intercostals (Trunk)
T7-L1    Abdominals
T11-L2  Ejaculation

Lumbar

L2    Hips
L3    Quadriceps
L4-L5  Hamstrings - Knee
L4-S1  Foot

Sacral

S2    Penile erection
S2-S3  Bowel and bladder

Coccygeal
Peripheral nerve

Is composed of number of axons of efferent and afferent neurons, myelin sheets and connective tissues

Types of fibres:

A alfa – thick, quick to 120 m/s, movement

A beta – thinner, to 70 m/s, touch, pressure

A gama – thinner, do 30 m/s, muscle tone

A delta – thinner, do 30 m/s, pain, warmth

B – thin and slow, 2 m/s, autonomic fibres

C – thin and slow, autonomic fibres, pain
ASCENDING TRACTS
(from periphery to brain)

Tractus spinothalamicus anterior
(coarse touch, pressure)
Tractus spinothalamicus lateralis
(pain, warmth)
Fasciculus gracilis
(cutaneous sensitivity, sensation from muscles, tendons, joints, fine touch, movement)

DESCENDING TRACTS
(from brain to the periphery)

Tractus corticospinalis (pyramidal tract)
Tractus vestibulospinalis (extrapyramidal tract)
Tractus reticulospinalis (extrapyramidal tract)
Three-neuronal afferent pathway from sensory receptors to the brain cortex

I. Order neuron
In the dorsal root ganglion

II. order neuron
In the spinal cord or in the medulla

III. Order neuron
In the thalamus

The exception from the three-neuronal rule is the pathway of the smell perception, which transmits the sensory signals directly from olfactory area in the nose to olfactory brain cortex.
MOTOR PATHWAYS

A  Pyramidal tract
Direct connection from motor cortex to skeletal muscles through motor end plate
Tractus corticospinalis

B  Extrapyramidal tracts
Indirect connections Through basal ganglia thalamus, cerebellum, brain stem
Tractus reticulospinalis
Tractus rubrospinalis
Protected in spinal column
Gray matter – neurons – butterfly shape
White matter – nerve fibers

**Dorsal horn** – sensory input
From muscle spindles
From spinal interneurons

**Ventral horn** – lower motor neurons
Motor output to spinal nerve
Motor input from upper motor neurons

Alpha motor neurons (motoneurons)
Gamma motor neurons (motoneurons)
Motor end plate = modified excitatory chemical synapse

**MUSCLE EXCITATION**

1. Action potential from alpha-motoneuron in the spinal cord reaches presynaptic membrane

2. Acetylcholine is released (excitatory mediator)

3. ACH on the receptors of postsynaptic membrane elicits (potential of motor end plate)

4. In case of threshold depolarization-depolarization of muscle membrane - **muscle potential** (AP of the muscle) in 2 ms muscle contracts
MOTOR END PLATE – NEUROMUSCULAR JUNCTION
MUSCLE FIBER – MUSCLE CELL
Its myofibrils behave according to the law „all or none“

SARCOLEMMMA

TRIAD:
TERMINAL CISTERNAE
TRANSVERSE TUBULE

SARCOPLASMIC RETICULUM

MITOCHONDRIA

MYOFIBRILLS

A - band
I - band
Z - line

from Z to Z = SARCOMERE
Muscle contraction occurs when myosin filaments within a sarcomere "walk" along adjacent actin filaments. This pulls the Z lines together shortening the sarcomere. Shortening of many sarcomeres along the myofibril causes the myofibril to contract.
EXCITATION – CONTRACTION COUPLING

- Action potential reaches motor end plate via axon of motoneurons in spinal cord
- Excitation (depolarization) of muscle membrane leads to the release of Ca ions from sarcoplasmic reticulum, Ca ions act on relaxation proteins unblocking actin receptors for myosin attachment
- Coupling of actin and myosin molecules and sliding of actin molecules upon myosin molecules (energy stored in myosin heads is consumed) from Z lines towards the centre of each sarcomere = muscle contraction
In skeletal muscle the action potential (AP) duration is about 2 ms and is over long before the peak of the twitch (twitch, being a very transient rise and fall of force). Repetitive stimulation can therefore lead to summation of twitches, more complete extension of the series elastic component and a rise in the force (tetanus).
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In the cardiac action potential, sodium channel opening is followed by inactivation. Sodium inactivation is accompanied by opening of slowly activating Ca\(^{2+}\) channels at the same time as a few fast K\(^+\) channels open. The balance between the outward flow of K\(^+\) and the inward flow of Ca\(^{2+}\) causes a plateau of variable length (150 ms). As the Ca\(^{2+}\) channels close, the plateau terminates and this leads to repolarisation. The muscle twitch is of almost the same duration as the action potential, and since the refractory period extends beyond the end of the AP, the muscle cannot readily be tetanised. This is important since tetany of the heart muscle would not allow effective pumping.
Monophasic Action Potential (Cardiac Muscle Cell)

-90 mV

350 ms

Action potential (Nerve Cell)

(c) 2007, Munther K. Horniou, MD
MOTOR UNIT IN SKELETAL MUSCLE

MOTOR UNIT = ONE ALPHA MOTONEURON PLUS ALL MUSCLE FIBERS IT INNERVATES ACCORDING TO LAW ALL OR NOTHING
DRUGS AFFECTING TRANSMISSION AT THE NEUROMUSCULAR JUNCTION

1) Drugs with ACH like action (nicotine) – have the same efect as ACH but are not destroyed by ACH esterase, so its action persists longer than ACH

2) Drugs that block neuromuscular transmission – curariform drugs prevent passage of impulses from the end plate to the muscle membrane by competing with ACH at the receptor sites

3) Drugs that inactivate acetylcholineesterase (physostigmine, neostigmine) Ach increases in quantity with successive nerve impulses, accumulation of ACH has potential military use as a powerful „nerve“ gas poison – cramps

MYASTENIA GRAVIS
– an autoimmune disease, patients have developed antibodies against their own Ach activated ion channels – end plate potentials are very weak to stimulate muscle fibers – paralyses – extremely dangerous in respiratory muscles – cessation of breathing (treated by neostogmin)
Muscle strength depends on:
- Number of stimulated muscle fibers
- Thickness of each m. fibre (thicker have more myofibrils)
- Resting length of muscle fibers

Ideal resting length is between 100% a 120% (2.0 – 2.25 micrometers), when maximum tension is produced (maintained by reflex contraction)
**REFLEX, REFLEX ARC**

**Main division:**
- **Somatic** (proprioceptive, exteroceptive)
- **Autonomic (Vegetative)**

According to the number of synapses:
- monosynaptic, polysynaptic

**Reflex arc –**
1) receptor,
2) afferent pathway,
3) CNS (spinal cord),
4) efferent pathway,
5) effector (skeletal muscle, heart, gland, smooth muscle..)

Reflex – stereotyped (always the same) response to stimulus – unconscious
The patellar tendon (knee jerk) reflex illustrates a monosynaptic stretch reflex and reciprocal inhibition of the antagonistic muscle.

**Stimulus:** Tap to tendon stretches muscle.

**Receptor:** Muscle spindle stretches and fires.

**Afferent path:** Action potential travels through sensory neuron.

**Integrating center:** Sensory neuron synapses in spinal cord.

**Efferent path 1:** Somatic motor neuron onto Effector 1: Quadriceps muscle

**Response:** Quadriceps contracts, swinging lower leg forward.

**Efferent path 2:** Interneuron inhibiting somatic motor neuron

**Effector 2:** Hamstring muscle

**Response:** Hamstring stays relaxed, allowing extension of leg (reciprocal inhibition).
CO-ACTIVATION OF ALPHA AND GAMMA MOTONEURONS

EXTRAFUSAL FIBRE
INTRAFUSAL FIBRE

SPINDLE SHEATH

PRIMARY AFF. FIBRE
SECONDARY AFF. FIBRE

GAMMA FIBRE
ALPHA FIBRE
CONTROL OF MUSCLE CONTRACTION

1) via pyramidal tract
   - alpha motoneurons
2) via reflex arch
   - muscle spindles,
   - Golgi tendon organs
3) via extrapyramidal tract
   - gamma motoneurons

Gama-activation
from higher motoneurons via extrapyramidal tract:
   - isometric contraction of muscle spindle
   - distension of the central region of the spindle
   - reflexive activation of alpha motoneuron
   - contraction of extrafusal fibres of the muscle

Function of gamma activation:
   - control of muscle tone
   - smoothness and preciseness of muscle contraction
Afferent impulses from muscle spindles stimulate directly alpha motoneuron to the agonist muscle (extensor) and via an inhibitory interneuron inhibit activity in the alpha motoneuron to the antagonist muscle (flexor) Ipsilateraly (on the same side of the body)
When a limb after painful stimulus is reflexively flexed, the antagonistic extensor muscles are passively stretched on the same limb - reciprocal innervation.
If one steps on a tack with the right foot, this foot is withdrawn by contraction of its flexor and relaxation of its extensor. Contralateral left foot extends to help support the body during withdrawal reflex – crossed extensor reflex = double reciprocal innervation.
An increase in muscle tension stimulates the activity of sensory nerve endings in the Golgi tendon organ. The sensory input stimulates an interneuron, which in turn, inhibits the activity of a motor neuron innervating that muscle. This is a disynaptic reflex and it helps prevent excessive muscle contraction or excessive passive muscle stretching.
CLASSIFICATION OF MOVEMENTS

LOCOMOTION = walking, running...transportation of the body in space
Muscles of lower limbs
Big motor units

MANIPULATION = writing, drawing, playing on music instruments, work
Muscles of upper limbs – hands
Small motor units, precise gradation of muscle contractions

VERBAL AND NONVERBAL EXPRESSION = speech, mimics...
Muscles of face and neck in coordination of breathing muscles

ANTIGRAVITATION = maintenance of orthostatic position
Muscles of trunk, neck and lower limbs – reflexive, muscle spindles involved
Big motor units

Hierarchic regulation:
1) Spinal reflexive centre
2) Supraspinal subcortical and cerebellar centres
3) Cortical centres
TYPES OF MUSCLE FIBERS

RED, SLOW

Have thin fibers, many mitochondria and high density of capillaries
= constant blood and oxygen supply (oxidative phosphorylation)
Long lasting muscle contraction
Muscle fatigue occurs later (POSTURAL MUSCLES)

WHITE, FAST

Have thick fibers, low capillary density, use anaerobic glycolysis,
Duration of contraction is short,
Muscle fatigue occurs quickly (BICEPS)
TYPES OF MUSCLE CONTRACTIONS

**ISOTONIC**
- Muscle remains constant muscle tone
- Muscle changes its length – shortens

**ISOMETRIC**
- Muscle remains constant length
- Muscle changes its tone – increases the tone - hardens

**AUXOTONIC**
- In normally functioning muscle under physiological conditions
- Muscle tone and muscle length are changed according to the needs of the body to stay or move.
Voluntary movements can be divided into three phases: planning, initiation, and execution. Sensory feedback allows the brain to correct for any deviation between the planned movement and the actual movement.
CONTROL OF VOLUNTARY MOVEMENTS

1. Sensory input
2. Planning and decision-making
3. Coordination and timing: cerebellar input
4. Execution: corticospinal tract to skeletal muscles
5. Execution: extrapyramidal influence on posture, balance, and gait
6. Continuous feedback

**KEY**
- Input
- Output
- Feedback

**Figure 13.10**
CONTROL OF VOLUNTARY MOVEMENTS

1. Sensory input
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KEY
- Input
- Output
- Feedback

- Prefrontal cortex
- Motor association areas
- Basal ganglia
- Thalamus
- Cerebellum
- Brain stem
- Motor cortex
- Sensory cortex
- Spinal cord
- Muscle contraction and movement
- Sensory receptors

Sensory receptors
Muscle contraction and movement
Spinal cord
Brain stem
Cerebellum
Motor cortex
Sensory cortex
Basal ganglia
Thalamus
Prefrontal cortex
Motor association areas
Continuous feedback
MUSCLE FATIGUE

FATIQUE ON THE SYNAPSE
Neuromuscular depression – on motor end plate – means that every consequent presynaptic stimulation causes weaker response on the postsynaptic (muscle) membrane.

CENTRAL FATIQUE
Inhibition on the level of motor neurons (always precedes cellular fatigue)
Lowered motivation to performance
Occurs mainly during linglasting stereotypic activities

CELLULAR FATIQUE
Fatique of muscle cell because of energy depletion
Accumulation of metabolites (lactic acid, lowered pH, carbon dioxide)
Physical and chemical changes in working muscle units (efflux of K ions, decrease of membrane potential disabling depolarization)
INCREASE OF NEUROMUSCULAR EXCITABILITY AS A FUNCTION OF HYPOCALCEMIA

NORMAL LEVELS OF PLASMA CALCIUM = 2.25 – 2.75 mmol/L

Calciemia depends on serum albumin, on acid-base balance (the higher the albumin concentration, the higher the plasma calcium, the lower the plasma pH, the higher the ionized plasma calcium.

**Hypocalciemia**

depends on absolute ionized calcium level
influences neuromuscular system – increase excitability of peripheral motoneuron
Painful tetanic spasms – **TETANY**
Occurs often following physical exhaustion and emotional stress
Hyperventilation leads to respiratory alcalosis and tetany

\[
\text{Excitability} = \frac{\text{Na}^+ + \text{K}^+}{\text{Ca}^+ + \text{Mg}^+ + \text{H}^+}
\]
SMOOTH MUSCLE
SMOOTH MUSCLE

Structure without striation

Muscle cells are thinner and shorter
(2 – 5 µm, 200-500 µm)

Contraction needs less energy
(10 - 300 times)

Muscle cells can stretch enormously
(uterus)

Muscle cells contract by means of actin and myosin
Myosin filaments are thicker, more actin filaments per cell (1:15)
Actin filaments insert into dense bodies and cell membrane
Less developed sarcoplasmic reticulum, Ca from ECF
Actin filaments lack troponin, but myosin filaments contain proteins binding Ca ions
Slow degradation of ATP in myosin heads – slow contraction – relaxation cycle (30 – times longer than in skeletal)
Maximum strength of contraction 4 – 6 kg/cm², skeletal 3 - 4 kg/cm²
Percentage of shortening to 30% of original length, skeletal to 65 – 75% of original length
SMOOTH MUSCLE

1. Non striated
2. Smaller cells one nucleus in each
3. Slow excitation and contraction
4. Excitation through hormones, local factors, passive stretch

Single – Unit

- GIT, uterus, ureter, arterioles
- Electrical synapses (gap junctions - syncitium)
- Myogenic stimulation (pacemakers – spont. activity)
- Respond to stretch

Multi - Unit

- m. ciliaris, m. sphincter et dilatator pupillae
- Separate innervation of muscle cells
- Neurogenic stimulation (via autonomic nerves)
- No response to stretch
Neuromuscular junctions
Autonomic fibers release neurotransmitter from varicosities to number of smooth muscle cells

Receptors for transmitter are on the entire surface of muscle Cells

Transmitters: NE, E, Ach, histamin angiotensin, serotonin, oxytocin,
• The top view shows a relaxed smooth muscle cell. Note the focal densities and the network of actin and myosin filaments.
• When contracted, the filaments slide together and pull the cell to a more rounded appearance.
• Sheets of smooth muscle cells work together because they are interconnected by gap junctions and connective tissue.
PHYSIOLOGY OF THE AUTONOMIC NERVOUS SYSTEM
AUTONOMIC NERVOUS SYSTEM

Motor system is divided to 1) SOMATIC NERVOUS SYSTEM 2) AUTONOMIC NERVOUS SYSTEM 3) ENTERIC NERVOUS SYSTEM

AUTONOMIC NERVOUS SYSTEM:

A) Sympathetic division
the mediator is adrenalin and noradrenalin

B) Parasympathetic division
the mediator is acetylcholin

Perform the action through two neurons
Preganglionic and postganglionic
Craniosacral division of ANS

Parasympathetic

Stimulates flow of saliva

Slows heartbeat

Constricts bronchi

Stimulates peristalsis and secretion

Stimulates release of bile

Contracts bladder

Sympathetic

Dilates pupil

Inhibits flow of saliva

Accelerates heartbeat

Dilates bronchi

Inhibits peristalsis and secretion

Conversion of glycogen to glucose

Secretion of adrenaline and noradrenaline

Inhibits bladder contraction

Thoracolumbar Division of ANS

Chain of sympathetic ganglia
The ganglion is situated paravertebrally in sympathetic division and near to the organs in parasympathetic division – spreading of the impulses for the release of the mediator – massive action throughout the body

Preganglionic fibers release ACETYLCHOLINE

Postganglionic fibers of sympathetic division release noradrenaline (norepinephrine)

Postganglionic fibers of parasympathetic division release acetylcholine
SYMPATHETIC NERVOUS SYSTEM

- decreases saliva production
- stimulates sweat glands
- causes mydriasis
- constricts peripheral vessels
- increase blood flow to skeletal muscles
- increase chronotropic and inotropic effects in the heart
- dilates bronchi
- reduces blood flow to abdomen
- decreases digestive activity
- relaxes smooth muscle in wall of bladder
- releases glucose stores from liver
ADRENAL MEDULLA

Stimulation of this „big sympathetic ganglion“ make it to release mostly adrenaline (epinephrine)
The adrenal medulla is also stimulated by the release of norepinephrine

The consequence of stimulation:
• noradrenalin & adrenalin release
• prolongation of the effects of sympathetic stimulation
• FIGHT OR FLIGHT
ADRENERGIC RECEPTORS

Alpha 1
- Peripheral vasoconstriction – increasing blood pressure
- Positive inotropic effect – increasing contractility
- Negative chronotropic effect

Alpha 2
- Peripheral vasodilation
- Limits release of norepinephrine
- Stimulated by excessive amounts of norepinephrine in synaptic cleft
ADRENERGIC RECEPTORS

Beta 1
- positive inotropic effect on heart - increased contractility and cardiac output
- positive chronotropic effect on heart - increased heart rate
- positive dromotropic effect on heart

Beta 2
- peripheral vasodilation
- Bronchodilation, relaxation of smooth muscles in bronchioli – increases lung capacity
- uterine smooth muscle relaxation
- GI smooth muscle relaxation
ALPHA AND BETA RECEPTORS

• The main actions of these receptors includes:
  – **Alpha-1**: vasoconstriction, gut smooth muscle relaxation, salivary secretion, glycogenolysis in the liver, contraction of gut sphincters and uterus.
  – **Alpha-2**: vasodilatation (central), vasoconstriction (peripheral), gut smooth muscle relaxation.
  – **Beta-1**: positive inotropy and chronotropy.
  – **Beta-2**: vasodilatation in muscle, gut and kidneys, bronchodilatation, pupillary dilatation, glycogenolysis.
SYMPATHOMIMETICS AND SYMPATHOLYPTICS

**Sympathomimetics** = medicaments that stimulate the sympathetic nervous system acting on adrenergic receptors (Alpha or Beta agonists)

- Direct effect: stimulation of alpha and/or beta-receptors,
- Indirect effect: stimulation of the release of noradrenaline (norepinephrine) by acting presynaptically. Drugs blocking the phosphodiesterase (phosphodiesterase inhibitors) have a postsynaptic action.

**Sympatholytics** = medicaments that inhibit the sympathetic system by blocking either alpha or beta adrenergic receptors. (Alpha or Beta antagonists)

- block effects of Alpha or Beta stimulation
SYMPATHOMIMETICS

The main drugs in this group are the naturally occurring catecholamines, adrenaline (epinephrine) and noradrenaline (norepinephrine). Adrenaline is an agonist at alpha and beta receptors, Noradrenaline has predominantly alpha agonist actions with minor beta-1 agonist activity.
SYMPATHOMIMETICS

Alpha receptor agonists

- Ephedrine: an indirectly acting sympathomimetic. It is taken up into presynaptic nerve terminals, thereby displacing noradrenaline resulting in alpha mediated vasoconstriction. Ephedrine also has a direct beta agonist effect increasing heart rate and cardiac output, the overall effect increasing blood pressure. These actions last for 10-15 minutes.
  Hypotension treatment
- Amphetamine: causes CNS stimulation by releasing and blocking uptake of neurotransmitters. Also has peripheral indirect sympathomimetic activity causing acute rises in blood pressure. Addiction
- Phenylephrine causes vasoconstriction and increasing blood pressure, coronary and cerebral perfusion pressure. Heart rate usually slows due to reflex bradycardia. Cerebral and coronary blood flow is minimally affected.
  hypotension treatment
- Alpha-2 receptors are found in the presynaptic membranes of adrenergic synapses and are widely distributed throughout the body including the CNS. They can be subdivided into three subtypes; alpha 2A (sedation, analgesia and sympatholysis), alpha 2B (vasoconstriction) and alpha 2C (CNS actions). Despite being agonists, their actions are generally more like sympatholytic drugs, but they are included here on the basis of their receptor activity.

Beta receptor agonist

- Isoprenaline: the first synthetic beta receptor agonist for clinical use, stimulating both beta-1 and -2 receptors. Usually given as an infusion because of its short duration of action. Used mainly to treat bradyarrhythmias and as a bronchodilator. Now largely replaced as a bronchodilator by beta-2 selective drugs because of the risk of cardiac arrhythmias.
SYMPATHOLYTICS

Alpha receptor antagonists (alpha blockers)
• **Phentolamine**: has similar CVS effects to phenoxybenzamine but the alpha blockade is shorter acting and reversible with alpha-agonists.

Beta receptor antagonists (beta blockers)
Since they were first synthesized over 50 years ago, “beta blockers” have evolved into a large family of drugs. The ones below are used to illustrate the key features of the differences between members of this family.

• **Propranolol**: relatively non-specific antagonist, blocking both beta-1 and beta-2 receptors. Decreases heart rate, blood pressure and cardiac output. Increases airway resistance in patients with asthma and COPD. Inhibits glucose metabolism and blocks sympathetic mediated “warning sings” of hypoglycaemia in diabetics. May adversly affect lipid profile. Its main use now is in the control of thyrotoxicosis, treatment of essential tremor, migraine and control of the somatic manifestations of stress.
PARASYMPATHETIC NERVOUS SYSTEM

- Makes pupillary constriction - miosis
- Increases secretion by digestive glands
- Increases smooth muscle activity along GI tract
- Makes bronchoconstriction
- reduces HR and have negative inotropic effects decreasing cardiac output
**ACETYLCHOLINE RECEPTORS**

**CNS receptors** (muscarinic and nicotinic): cholinergic neurotransmission at the CNS level is thought to regulate sleep, wakefulness, and memory.

**Autonomic receptors** (muscarinic or nicotinic)

**Neuromuscular junction:** acetylcholine receptors at the neuromuscular junction are exclusively nicotinic, they belong to the NN subtype.
MUSCARINIC RECEPTORS

Activation of muscarinic receptors occurs mainly at autonomic ganglia, organs innervated by the parasympathetic division of the autonomic nervous system and in the central nervous system.

M1, M4 and M5 receptors in CNS. These receptors are involved in complex CNS responses such as memory, arousal, attention and analgesia. M1 receptors are also found at gastric parietal cells and autonomic ganglia.

M2 receptors: heart. Activation of M2 receptors lowers conduction velocity at sinoatrial and atrioventricular nodes, thus lowering heart rate.

M3 receptors: smooth muscle. Activation of M3 receptors at the smooth muscle level produces responses on a variety of organs that include: bronchial tissue, bladder, exocrine glands, among others.
**N1 or NM receptors**: these receptors are located at the neuromuscular junction, acetylcholine receptors of the NM subtype are the only acetylcholine receptors that can be found at the neuromuscular junction.

**N2 or NN receptors**: these nicotinic receptors can be found both at cholinergic and adrenergic ganglia, but not at the target tissues.
PARASYMPATHOMIMETICS AND PARASYMPATHOLYTICS

Parasympathomimetics = drugs that stimulate parasympathetic nervous system
Physostigmine = reversible cholinesterase inhibitor, acts by interfering with metabolism of ACH, is used to treat myasthenia gravis, Alzheimer disease, delayed gastric emptying, mydriasis...

Parasympatholytics = drugs that block action of parasympathetic nervous system
Atropine = muscarinic receptor antagonist, parasympathetic blocker, oposes the action of vagus nerve, induces mydriasis, blocks eye accomodation by paralysing ciliary muscles
Adrenal medulla a modified sympathetic ganglion, receives sympathetic preganglionic fibers and releases epinephrine and norepinephrine into the blood.
### ACTIVATION OF THE SYMPATNICUS „FIGHT OR FLIGHT“

<table>
<thead>
<tr>
<th>RESPONSE</th>
<th>CONSEQUENCE FOR THE MUSCLE</th>
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<tbody>
<tr>
<td>Speeding up the heart rate</td>
<td>Increased blood flow (oxygen, glucose)</td>
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<tr>
<td>Strengthening the contractions</td>
<td></td>
</tr>
<tr>
<td>Dilatation of the vessels</td>
<td>Increased flow of oxygen and nutrients to skeletal muscles</td>
</tr>
<tr>
<td>Constriction of vessel in GIT</td>
<td>Blood redistribution to skeletal muscles</td>
</tr>
<tr>
<td>Emptying of the spleen</td>
<td>More blood in circulation</td>
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<tr>
<td></td>
<td>More oxygen and glucose to skeletal muscles</td>
</tr>
<tr>
<td>Dilatation of the airways</td>
<td>More oxygen to blood</td>
</tr>
<tr>
<td>Increase of ventilation</td>
<td>Increased oxygen in blood</td>
</tr>
<tr>
<td>Increase of perspiration</td>
<td>Transport of produced warmth to the environment</td>
</tr>
<tr>
<td>Increased metabolism of glycogen</td>
<td>More glucose to skeletal muscles</td>
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<td>to glucose</td>
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The complexity of the extrapyramidal tracts
INTEGRATION OF MUSCLE REFLEXES

Cerebrum

Sensory areas of cerebral cortex

Thalamus

Postural reflexes, hand and eye movements

Cerebellum

Brain stem

Spinal cord

Sensory receptors

Muscle contraction and movement

Feedback

1 Sensory input (→) from receptors goes to spinal cord, cerebral cortex, and cerebellum. Signals from the vestibular apparatus go directly to the cerebellum.

2 Postural and spinal reflexes do not require integration in the cortex. 
Output signals (→) initiate movement without higher input.
ENERGY SOURCES FOR MUSCLE CONTRACTION

Creatine\(\sim P\)

Creatine + \(~P~\)

Glycogen

Glycolysis

Lactic acid + \(~P~\)

Glucose or other fuel

Respiration

\(~P~\) + CO\(_2 + H_2O\)

ATP

Muscle Contraction

users.rcn.com
ENERGETICS OF MUSCLE CONTRACTION

- 100-meter; 9.86 s
- 1000-meter; 132.2 s
- Marathon; 7,600 s

*World Almanac, 1994*
CRANIAL NERVES
For head and neck innervation

**MOTOR CRANIAL NERVES**
leave motor nuclei in brain stem
Irritation leads to cramps, spasms
Lesion leads to muscle paralysis (palsy)

**SENSORY CRANIAL NERVES**
Transmit nerve impulses from sensory receptors (vision, hearing, olfaction, taste)
Lesion leads to disruption of sensory function

**SENSITIVE CRANIAL NERVES**
Transmit nerve impulses from skin
To cortex (gyrus postcentralis – sensory homunculus)
Lesion leads to hypesthesia
Irritation leads to hyperesthesia, paresthesia, pain
CRANIAL NERVES

I. n. olfactorius
II. n. opticus
III. n. oculomotorius
IV. n. trochlearis
V. n. trigeminus
VI. n. abducens
VII. n. facialis
VIII. n. vestibulocochlearis
IX. n. glossopharyngeus
X. n. vagus
XI. n. accessorius
XII. n. hypoglossus