LEC 19

SLIDE 2

the muscle structure is as follows:
1-myofibrils make the muscle fibers.
2- muscle fibers; which are the building units of the muscle (the muscle's cell).
3- fascicles; which are composed of a GROUP OF muscle fibers.
4- muscles; which are composed of a GROUP OF fascicles.
muscle fibers are composed of a muscle protein which are called protein filaments.

SLIDE 3

DO NOT FORGET (information from LECTURE 18), the T tubule is the extension that the action potential goes through. when the action potential goes through the T tubule, it stimulates Ca++ channels; which makes the muscles contract.
cisterna CAN NOT be seen in the picture.
the myofibrils have the units that are responsible of muscle contraction which are:
1- thick filaments = myosin.
2- thin filaments = actin.

SLIDE 4

in this slide we see a muscle contraction in a microscopic level.
the myosin (thick filaments) molecules structure looks like a golf club shape مضرب غولف:
1- tail
2- head
the tail is considered long and at the end of it the head is found, which is considered thick.
the head is composed of two special sites:
1- the actin-binding site, where the actin filaments (thin filament) bind with the myosin filaments (thick filament).
2- the ATPase site, which has the ATPase enzyme THAT PROVES that muscle contraction needs energy (ATP) to happen.

SLIDE 5

actin (thin filaments) structure are STRANDS of proteins.
the globular (G) actin is the monomer (the building block) of the actin filaments.
the globular (G) actin (thin filaments) has a myosin-binding site (the black dot in the picture) which binds with the myosin (thick filaments) head in the actin-binding site.
tropomyosin is the red line that can be seen in the last picture. its main function is to prevent interaction between actin and myosin. when this happens, the muscle relaxes.
you can see that the tropomyosin is ON TOP OF THE BLACK DOTS (the myosin-binding site, another name for it is "actin active site", that's how the tropomyosin prevents interaction between the actin and myosin.
what is the opposite of tropomyosin? troponin.
how? troponin works as a regulator for muscle contraction; as it binds with calcium which helps making the muscle contract AND works by moving the tropomyosin from the black dots (myosin-binding site).
NOT NEEDED but good to know! G actin is the monomeric form of actin and F actin is polymer form OF a group of G actin monomers.
basically, G actin makes F actin which is the ACTUAL filament.
the actin can be seen as double helical strands.

SLIDE 6

when the muscle is relaxed, the actin can be seen with tropomyosin COVERING the myosin-binding site AND the troponin is not linked with Ca++.

when the muscle is contracted, Ca++ is linked with troponin, the tropomyosin is MOVED AWAY by troponin which EXPOSES the myosin-binding site.

SLIDE 7

the myofibril can be seen like this when zoomed in.

the whole group is called SARCOMERE and it is found in the myofibrils, as they are the building unit of the myofibrils.

in this picture we can see the following:

1- the thick filaments (myosin) which can be NOTICABLE by the HEAD that is popping out (the ones in orange).

2- the thin filaments (actin) which can be NOTICABLE by its double helical structure (the ones in light blue).

we can see that the thick filaments bond with the thin filaments. this bonding is called "cross-bridge".

3- the Z line is the starting AND ending point of the sarcomere. so, we can say that the sarcomere starts FROM one Z line then the sarcomere ends AT the other Z line.

SLIDE 8

there are three places in the sarcomere:

1- H zone, which ONLY has MYOSIN filaments and the M line is found in this zone and it is the SMALLEST band.

the M line is a place THAT ONLY HAS MYOSIN FIBERS (thick filaments).

so, when there is a shortening/ relaxation in the H zone this means that ONLY the MYOSIN fibers have done the action (shortening/ relaxation).

2- A band, which has the myosin AND the actin filament and it is the LARGEST band.

3- I band, which ONLY has ACTIN filaments.

an easy way to remember the zones:

we have three zones, H, A, I

بنقدر نتذكرها بكلمة "هاي" بالعربي

1- H&M, so only Myosin is found in the H zone. H&M اتذكروا محل الملابس

2- MAA, so both Myosin AND Actin is found in the A band. اتذكروا صوت الخرفان

3- A.I, so only Actin is found in the I BAND. A.I هو اختصار للذكاء الاصطناعي

SLIDE 9

in this slide we see two pictures on the top.

the first picture is when the muscle is RELAXED and we see the following:

1- H zone is in its normal size and only myosin is found in it.

2- A band is in its normal size and both myosin and actin is found in it.

3- I band is in its normal size and only actin is found in it.

so, ALL THE ZONES ARE IN THEIR NORMAL SIZES WHEN THE MUSCLES IS RELAXED.

the second picture is when the muscle is CONTRACTED and we see the following:

1- H zone SHORTENS ,actin would get closer to the M line (which is the middle line) and would slide to be apparent in the H zone

2- A band stays in its normal size.

3- I band SHORTENS. so, myosin would get closer to the M line.

REMEMBER THAT the movement of actin is called SLIDING.

so, we can say that in contraction, the zones try to get CLOSER to the M line.

another easy way to remember what zones shorten is by saying:

contraction = HI

LEC 20

SLIDE 3

quick refreshment on lecture 18 that is IMPORTANT to remember.
ACh is the neurotransmitter that stimulates the muscles into having an action potential.
as we know, this process happens in the NEUROMUSCULAR JUNCTION. this junction is composed of the following:
1- presynaptic membrane which is the part in yellow in the picture (the axon terminal).
2- postsynaptic membrane which is the part that is in light brown in the picture (the motor end plate).
3- synaptic cleft which is the SPACE between the presynaptic and the postsynaptic membranes.

تبعوا مع الأرقام الي موجودة بالصورة

1- the ACh is stored in vesicles that ONLY reach the end of the axon's terminal wall by the help of several proteins. when it does, the ACh FUSES with the wall and gets released out of the axon terminal to the synaptic cleft; by the entrance of Ca++ in the axon terminal WHICH STIMULATES more vesicles to get out of the axon terminal.
the ACh then binds with receptors that are on the motor end plate's cytoplasm which are called "nicotinic receptors".
these receptors are responsible of opening Na+ ligand channels in the motor end plate to get an inflow inside the muscle cell to create an action potential, which has a SPECIAL NAME in the muscle called "end-plate potential".
2- now, the action potential propagates along the sarcolemma (the cell membrane's surface) and down the T tubule which is an extension coming from the sarcolemma.
* all of this was mentioned in lecture 18 so please read it.
3- in the picture, we can see that the T tubule is linked with the sarcoplasmic reticulum. the action potential that is going through the T tubule triggers the sarcoplasmic reticulum to release Ca++ from it. this is considered the starting point that is ESSENTIAL in making the muscles contract.
4- the Ca++ then moves on to bind with the troponin complex that is found in the actin filaments (thin filaments) that is dark blue in the double helical structure. when the binding happens, the troponin works on MOVING AWAY the tropomyosin strand from the myosin-binding site (the black dots in the actin) to EXPOSE IT.
5- when the myosin-binding site gets exposed, the myosin (thick filaments) that can be seen in orange would bind with it. how? the head of the myosin has an actin-binding site that CONNECTS with the myosin-binding site in the actin, this bond is called "cross bridge".
this is considered the start of the cross bridge cycle and the start of the muscle contraction.
when a cross bridge happens between the actin and myosin the contraction starts which in turn means that the myosin would SLIDE the actin to the middle (M line) and the muscle would shorten in two zones (HI):
1- the H zone.
2- the I band.
6- after the contraction is done, the Ca++ is then unbounded BY ACTIVE TRANSPORT from the troponin and the Ca++ gets back to the lumen sarcoplasmic reticulum; to be reused again for another contraction.
7- the most noticeable thing that we can see is that the tropomyosin GETS BACK to its original place (on top of the myosin-binding site) to BLOCK IT and the cross bridging breaks. this causes the muscle to get relaxed again.

SLIDE 4

the cross bridging cycle:

REMEMBER THAT the myosin head (the golf club shaped) has two sites:

1- the actin-binding site

2- the ATPase binding site, which has the ATPase enzyme.

the ATPase binding site shows that cross bridging and the muscle contraction needs ENERGY to happen.

KEYWORDS FROM BEFORE:

ATP = adenosine triphosphate, three groups of inorganic phosphate.

ADP = adenosine diphosphate, two groups of inorganic phosphate.

AMP = adenosine monophosphate, one group of inorganic phosphate.

hydrolyzed, hydrolyzation = having ATP broken down by water to (ADP + P) to release energy.

1- the binding of myosin (which has the actin-binding site) and actin (which has the myosin-binding site) happens. another name for this bond, cross bridge.

this happens by releasing ONE inorganic phosphate from the ATP (ADP + P) that is on the ATPase binding site so that the myosin would have ADP at the end of this step.

2- since the myosin has ADP in the ATPase site, the actin takes advantage of this to go towards the middle of the sarcomere (to the M line) to shorten the sarcomere. when this happens, it is called "power stroke" and is characterized by the release of the ADP.

3- because of the release of P in the first step and ADP in the second step, the myosin has lost the energy that is found in the ATPase binding site. when this happens, the myosin is considered a "rigor"/ in a low-energy form. BUT we still see that the myosin is STILL connected to the actin filaments and the muscle is considered in a stage of "stiffness"/ contraction.

stiffness = تشنج

in this stage, IF no ATP binds with the myosin head the muscle would stay contracted.

4- if ATP binds with the myosin head in the ATPase binding site, the myosin would UNBIND from the actin and the ATP would get hydrolyzed to have

ADP + P in the ATPase binding site.

5-the myosin becomes cocked (back to its normal place) and because the ATPase binding site has ADP + P then the myosin is in a high-energy form.

SLIDE 6

things to remember from lecture 18!!

when the ACh gets to the synaptic cleft (the space between the presynaptic and the post synaptic) IT HAS TO BREAK DOWN to acetyl + choline AND get back into the axon terminal (the presynaptic membrane). this is done by the acetylcholinesterase enzyme.

in spastic paralysis, we see a pathological change in which an inhibition of acetylcholinesterase enzyme happens and the ACh DOES NOT break down in the synaptic cleft.

the causes of spastic paralysis:

1- nerve gas poison (sarin gas) which inhibits the enzyme and prevented the ACh from breaking down. this causes the ACh to bond with the nicotinic receptors and to have an on-going inflow of Na+ inside the motor-end plate. so, the muscle would stay contracted.

2- ingestion of insecticides killer (يف باف)

it does the same as the nerve gas poison.

SLIDE 7

all neuromuscular blockers such as topical or general anesthesia or snake bites work on blocking the nicotinic receptors so that the ACh does not bind with it.

botulinum disease prevents the ACh from getting to the active zone of the axon terminal.

LEC 21

SLIDE 4

remember!! this graph shows something called "twitching" NOT action potential of a muscle.
twitching is the time needed for the muscle to contract and then relax.
in this graph we see three types of muscles:
1- extraocular muscle
2- gastrocnemius
3- soleus
in the X - axis, we see the time taken for the muscle to contract AND relax in milliseconds.
in the Y- axis, we see the tension of the muscle.
extraocular muscle, fast oxidative twitching.
gastrocnemius, fast glycolytic twitching.
soleus, slow oxidative twitching.

SLIDE 5

two things to distinguish for later on:
1- tension refers to the force IN the muscle fibers themselves.
2- force refers to an EXTERNAL force that is put on the muscle.
recruitment = تجنيد
the muscles work by RECRUITING motor units in an organized way.
MOTOR UNIT: is a bundle of muscle fibers that are innervated by a single neuron.
example! if one neuron innervates 5 muscle fibers and another neuron innervates 7 muscle fibers THEN the one that innervates MORE muscle fibers is called the BIGGER motor unit.
so, ↑ fiber units innervated by ONE nerve ↑ bigger motor unit.
how does recruitment work? there is a set of rules to recruit a motor unit. THE MAIN RULE IS motor units are recruited from SMALLEST to LARGEST.
this principle is called the henneman's size principle.
عشان نفهمها بشكل أكبر ناخذ هالمثال.
بالجيش، بيتم أخذ الشباب بنسبة أكبر من الكبار بالسن. ليش؟ لأنهم أكثر تحملًا وهيك بيمشي هادا المبدأ.
if we get more precise we see the following:
SMALL MOTOR UNITS ARE:
1- slow twitching.
2- low force.
3- fatigue-resistant.
fatigue-resistant = مقاوم للإجهاد
نرجع لمثالنا تبع الجيش، الشباب عندهم مزايا بتساعدهم
1- أقل غلبة، فيعني السيطرة عليهم بشكل أسهل
2- قوتهم بتكون ماشي حالها فبنقدر ندربهم بشكل أكثر
3- مقاومين للإجهاد بشكل أكبر
do not forget, SMALL motor units are found in SLOW OXIDATIVE muscles.
LARGE MOTOR UNITS ARE:
1- fast twitching.
2- high force.
3- less fatigue resistant.
do not forget, LARGE motor units are found in FAST GLYCOLYTIC muscles.
نرجع لمثالنا تبع الجيش، الكبار بالسن بيكون عندهم صعوبة
1- ما عندهم طولة بال فالسيطرة أصعب
2- قوتهم بتصعب على المدربين إنه يتم تحسينها
3- مقاومتهم للإجهاد قليلة

to understand the graph that is in the bottom, when a muscle wants to recruit we FIRST recruit the smaller motor unit THEN recruit the larger motor unit. so, we can say that the recruitment goes GRADUALLY.
this MINIMIZES the amount of fatigue for these motor units.
if the recruitment was the complete opposite the large motor units would not be able to handle for this long and it would be tired.

SLIDE 7
when does fatigue happen? when the muscle reaches the tetanus.

SLIDE 9
there are two types of smooth muscles:
1- multi-unit smooth muscle.
2- unitary smooth muscle.
multi-unit smooth muscles are NOT like what its name suggests. how?
multi-unit smooth muscles work independently in contractions AND are individually innervated. they are found in places that need precision or "fine control".
fine control = تحكم دقيق
so, even though their name is "MULTI" but they work alone NOT together.
examples of multi-unit smooth muscles:
1- ciliary muscle of the eye.
2- iris muscle of the eye.
3- piloerector muscle that causes erection of the hair when stimulated by the sympathetic nervous system.
بالعربي، هاد النوع من العضلات بالرغم من إنه إسمه بيوحي إنها عبارة عن مجموعة من الألياف العضلية الي بتشتغل مع بعض بس هم بيساووا العكس.
يعني، هاد النوع هو عبارة عن مجموعة من العضلات بس كل وحدة منهم الها عصب لحالها، وكل عضلة بتنقبض كمان لحالها.
unitary smooth muscle:
unitary = كوحدة
from the name, we can know that this type of muscle works as a group. this means that a hundred to thousands of smooth muscles CONTRACT TOGETHER.
unitary smooth muscles work differently than multi-unit smooth muscles.
how do they contract at the same time? they have gap junctions and these gap junctions allow them to COMMUNICATE BETWEEN EACH OTHER so that they can contract "coordinated contraction" and relax together. because of this, they are NOT considered precise like the multi-unit smooth muscles so they have "gross control".
gross control = تحكم إجمالي
unitary smooth muscles produce STRONG contractions.
multi-unit smooth muscles produce FINE contractions.

SLIDE 11

pacemaker activity basically organizes, regulates and generates action potential BUT it is not considered an action potential.

in the graph you can see the small waves that the pacemaker activity makes which are produced by the interstitial cells of cajal. they REGULATE THE ACTION POTENTIAL.

how? you can see that the spikes are ONLY on the top part of the slow waves and not on the lower part of the slow waves.

بالعربي، هاي الموجات بتنظم موضوع توليد الحركة وما بتصير إلا إذا الموجة كانت بالقمة. كيف يعني؟ الحركة الي بتعملها خلايا "كاجال" الي بتساويلنا رسمة الموجات البطيئة فأول ما تصير عملية الانقباض أو وصول الموجة للقمة بتحدث عملية توليد الطاقة الي هي الخطوط الي بالأحمر.

this makes the action potential organized. we can see this in the gastrointestinal smooth muscle to have a synchronized contraction and potential.

SLIDE 12

the difference between smooth muscle contraction and skeletal muscle contraction is the following:
the Ca++ binds in the smooth muscles with a protein called calmodulin (CaM) which activates the myosin light chain kinase (MLCK) that phosphorylates the light chains in myosin heads and increases the ATPase activity which in turn cross-bridges with actin to produce a contraction.

SLIDE 13

the difference between smooth muscle relaxation and skeletal muscle relaxation is the following:
there is an enzyme in the smooth muscles called "myosin phosphatase" which removes the phosphate group from the myosin head, which decreases the myosin ATPase activity and the muscle would get relaxed**.**

LEC 22

SLIDE 3

the sensory division is responsible of sensation.
the motor division is responsible of the final output (response) that is coming out from the CNS EFFERENT nerve.
reminder:
afferent nerve: a nerve that transmits impulses from PERIPHERAL ORGANS to CNS.
من الأطراف إلى المركز.
efferent nerve: a nerve that transmits impulses from CNS to PERIPHERAL ORGANS.
من المركز إلى الأطراف.
visceral: refers to smooth muscles, cardiac muscles and glands.
somatic: refers to skeletal muscles, tendons, joints and skin.
visceral motor division has two sub-categories:
1- sympathetic
2- parasympathetic

SLIDE 4

comparison, somatic VS. ANS.
somatic:
- its EFFERENT nerve comes out of the ventral root of the spinal cord as a LONG, THICK, HEAVILY MYELINATED nerve till it reaches its effector (from the previous note, the effector can be skeletal muscle, tendons, joints or skin).
so the pathway of the somatic system is:
spinal cord --> long, thick, heavily myelinated nerve --> effector.
somatic nervous system is responsible of voluntary actions.
target organs response to neurotransmitters in somatic:
- stimulatory ONLY. for example, contracting a muscle.
ANS:
- its EFFERENT nerve ALSO comes out of the ventral root of the spinal cord as a LONG, THIN, LIGHTLY MYEILNATED nerve.
the ANS differs from somatic; because its nerve gets separated to two:
1- pre-ganglionic nerve.
2- post-ganglionic nerve.
the pre-ganglionic nerve connects the spinal cord with something called the "ganglion".
ganglion: a connection of neuron cell bodies.
مكان تجمع كمية من الخلايا العصبية، اعتبروه نقطة تجمع.
now, a SHORT, UNMYELINATED nerve gets out of the ganglion and reaches the effector (from the previous note, the effector can be smooth muscles, cardiac muscle or glands). this nerve is called the post-ganglionic nerve.
so, the pathway of the ANS system is:
spinal cord --> long, thin, lightly myelinated nerve (pre-ganglionic nerve) --> ganglion --> short, unmyelinated nerve (post-ganglionic nerve) --> effector.
ANS nervous system is responsible of involuntary actions.
target organ response to neurotransmitters in ANS:
- stimulatory. for example, increases the pulse rate.
- inhibitory. for example, decreases the pulse rate.

SLIDE 7
we have two divisions in the ANS system:
1- sympathetic (+) زي دعاسة البنزين
2- parasympathetic (-) زي دعاسة البريك
in almost ALL VISCERAL ORGANS (smooth muscles, cardiac muscles, glands) we see both of these divisions BUT they cause opposite effects. this is called "dual innervation".
بالعربي، هدول الاثنين بيشتغلوا عكس بعض فكمثال إذا بدنا نرفع معدل نبضات القلب بنستعمل النوع الأول (+) وإذا بدنا ننزل المعدل بنستعمل النوع الثاني (-) وهيك بيصير عندنا تنظيم للحركة تاعت هاي الأعضاء اللإرادية.
الإشارات بتسهل عليكم تعرفوا كيف بيعمل هذا النوع (بمعظم الحالات) من ناحية الزيادة أو النقصان.

to understand the slide more,
there are three regions in the spinal cord that we need to know, which are called:
thoracic (T) region: it has 12 discs. so, from T1-T12.
lumbar (L) region: it has 5 discs. so,
from L1- L5.
sacral (S) region: it has 5 discs. so,
from S1-S5.
and a 4th region that is NOT a part of the spinal cord called "the cranial region" which has 12 CRANIAL nerves.

the sympathetic division:
- origin: THORACOLUMBAR region (from T1-L2) of the spinal cord.
so, the sympathetic nerves come out of the spine from the THORACIC (T) region AND the LUMBAR (L) region.
- the length of the nerve fiber:
first, SHORT PRE-ganglionic fiber then, LONG POST-ganglionic fiber
- location of ganglia (ganglion):
CLOSE TO THE SPINAL CORD
to imagine a sympathetic division consider it like this:
-- = shows the length of the sympathetic fiber.
spinal cord---ganglia-------effector

the parasympathetic division:
- origin: CRANIOSACRAL region (from S2-S4 and cranial nerve 3,7,9,10) of the spinal cord.
so, the parasympathetic nerves come out of the spine from CRANIAL region AND the SACRAL (S) region.
- the length of the nerve fiber:
first, LONG PRE-ganglionic fiber then, SHORT POST-ganglionic fiber
location of ganglia (ganglion): CLOSE TO THE EFFECTOR ORGANS.
to imagine a parasympathetic division consider it like this:
-- = shows the length of the parasympathetic fiber.
spinal cord-------ganglia---effector

SLIDE 8

notice how the sympathetic nerve fibers that are COMING OUT OF THE LATERAL HOLE OF THE SPINAL CORD are SHORT in the beginning (pre-ganglionic) and synapses with the sympathetic ganglion which is CLOSER to the spinal cord then the sympathetic nerve fibers become LONG at the end (post-ganglionic).
and notice how the parasympathetic nerve fibers are LONG in the beginning (pre-ganglionic) and synapses with the ganglion that is CLOSER to the effector organ then the parasympathetic nerve fiber becomes SHORT at the end (post-ganglionic).

SLIDE 9

remember that the SOMATIC NERVOUS SYSTEM always releases ACh and always has a stimulatory effect.
in the ANS NERVOUS SYSTEM:
1- pre-ganglionic ALWAYS releases ACh.
2- post ganglionic in SYMPATHETIC NERVE FIBERS release either NE (nor-epinephrine) or epinephrine.
but, in PARASYMPATHETIC NERVE FIBERS release ACh.
one exception in the ANS nervous system in the sympathetic nerve fiber is the adrenal medulla (a gland above the kidney) as the PRE-SYMPATHETIC nerve synapses with the medulla INSTEAD of a ganglion.

SLIDE 11

to understand the picture on the left more, we have AFFERENT and EFFERENTS that are numbered from 1-7
1- somatic efferent (not important)
2- somatic afferent (not important)
3,4,5- sympathetic efferent (important)
6,7- ANS afferent (not important)
3, 4, 5 go through a place called "white ramus communicant" where they communicate with each other and then synapse with a sympathetic ganglion EXCEPT the adrenal medulla.

SLIDE 12

in this picture we can see that there is a long chain of ganglions on top of each other which we either call it "ganglionic chain" OR "the sympathetic trunk"
OR "para-vertebral ganglions" which are close to the spinal cord.
there is ANOTHER type of ganglions that are CLOSE TO THE ORGAN which are called "co-lateral ganglions" OR "prevertebral ganglions" OR "greater splanchnic nerve.
in this picture we can see different pathways that pre-ganglionic nerves (the red lines) go through:
1- the pre-ganglionic nerve makes a synapse with the ganglion then the post-ganglionic (the black lines) synapses with the effector organ.
2- the pre-ganglionic nerve fiber does not synapse with the ganglion chain and they form together the "cardiac and pulmonary plexuses" (check in the picture) and then form the post-ganglionic nerve fiber which connects with the organs.
the nerve fibers can sometimes be ascending (like the innervation to the eye, the salivary gland, the heart, the lung) and descending which is linked with the sympathetic cervical ganglions.
3- from T5-T12 the pre-ganglionic nerve fibers do not synapse with the ganglionic chain BUT descend and synapse with the "co-lateral ganglions" and can be divided to:
a. greater splanchnic
b. mesenteric
c. inferior mesenteric
d. superior mesenteric
^^ not important ^^
4- sometimes, an innervation could come out of these divisions and create an outflow to certain organs such as the female organs or the male organs. this can be seen in the SACRAL (S) region and the coxae.

SLIDE 13

the ANS is controlled by the central nervous system, in specific the limbic system which consists of:

1- thalamus
2- hypothalamus
3- hippocampus
4- corpus callosum
5- brain stem (reticular formation)

SLIDE 14

the doctor only told us about the nerves

LEC 23

SLIDE 2

tract: a bundle of axons inside the central nervous system.

SLIDE 4

posterior column = dorsal column

SLIDE 5

the posterior column is responsible for certain actions such as:
1- fine-touch
2- vibration
3- pressure
4- proprioception
proprioceptive = the movement of joint or tendon or any skeletal muscles in space (knowing if it goes up, down, right, left).
how are these signals transmitted to the posterior column? a type of receptors need to be stimulated (check slide 6)
the stimulation would go UP through the DORSAL GANLGION ROOT which is found outside of the spinal cord where a synapse happens with the DORSAL ganglion (check slide 7), this is called a first-order neuron.
now, the pathway of the signal would go into the spinal cord.
we have two divisions for the posterior column:
1- fasciculus cuneatus
2- fasciculus gracilis
(check slide 8)
these two divisions would transmit the signals UP to the medulla oblongata where they synapse with the NUCLEUS GRACIILIS AND CUNATEOUS. this is called the second-order neuron.
the second-order neuron would CROSS OVER to the opposite side (in the picture it goes from the right side TO the left side).
* because of this, the right side of our body is controlled by the left side of our brain and vice versa.
the signal would then go up to the medial lemniscus in the mid-brain and go up to the fore-brain in the ventral nuclei of the thalamus.
ALL SENSORY SIGNALS HAVE TO TRANSMIT TO THE THALAMUS EXCEPT THE SMELLING SENSATION.
from the thalamus, all the sensory signals would transmit to cerebral cortex of the brain which has the following three regions.
the three regions are:
1- the projection fibers (responsible of ascending or descending information)
2-corpus callosum (connects the right side of the brain with the left side and transmits information between them)
3- parietal lobe/association area (transmits information between the anterior and posterior part of the brain)
there are three types of nerve fibers in our body:
1- type A (fastest)
2- type B
3- type C (slowest)
the posterior column is the fastest column in our body; because it uses type A nerve fibers which is the most thickly myelinated nerve fiber type. type A nerve fibers has three sub-categories:
1- alpha α (fastest)
2- beta β
3- delta δ (slowest)

SLIDE 6

in the picture on the right you'll find different types of receptors that are found in the epidermis.
each receptor is stimulated by a certain sensation.

1- merkel's disk: a type of receptor that is responsible of fine-touch/superficial touch sensation.
fine-touch can be described as an example when you sense a type of clothes texture.
مثال لهالنوعية من الإحساس: أول ما تروح تشتري بلوزة جديدة بتروح تمسك عشان تحس كيف نوعية القماش.

2- Meissner's corpuscle: a type of receptor that is responsible of distinctive touch sensation.
distinctive = التمييز
distinctive touch can be described as an example when someone puts their hand on your arm and know how many fingers are touching you,
Meissner's corpuscle is MORE sensitive than merkel's disk; as it can identify more localizations.
خلوا حدا يلمس ذراعكم بعدد معين من أصابعه بدون ما تشوفوا، عقلكم حيعرف كم إصبع لامس ذراعك بسبب هذا المستقبل.

3- ruffini ending:
a type of receptor that is found in the DERMIS layer of the skin and is responsible of sensing the skin's stretching.
زي لما حدا يشد خدودك.

4-pacinan's corpuscle:
a type of receptor that is found in the DERMIS layer of the skin and is responsible of sensing deep pressure and vibration.
vibration: the frequent stimulation (pressure) of the skin.
الضغط: زي لما حدا يمسك إيدك بقوة
الاهتزاز: زي لما تكون حاطط جوالك ع وضع الهزاز وتجيك مسج فتحس بالهزة تاعته.
as we can see, each receptor is involved in a certain type of stimulation/sensation to the posterior column tract.

how does a stimulation happen? for this example let's take the Pacinian corpuscle (which is responsible for the deep pressure stimulation).
when a pressure is applied, Na+ channels open in the NEURON ENDING in the corpuscle that would cause a DRAMATIC increase of Na+ inside in the neuron and an action potential would happen.
for this example,
↑ pressure ↑ Na+ voltage-gated channels open ↑ action potential

a general rule for ALL receptor:
↑ stimulation ↑ Na+voltage-gated channels open ↑ action potential

when more action potentials are created this causes something called "frequency"
frequency: the increasing of a stimulation per a unit of time.
frequency = تردد
عشان توضح بشكل أكبر، كل ما بيصير عندنا تحفيز للمستقبلات كل ما تكون عندنا سيلان عصبي، فكل ما زاد عنا كمية التحفيز صار عندنا زيادة لكمية السيلانات العصبية المتكونة.
التردد هو عبارة عن كمية التحفيز الي بتصير بفترة زمنية معينة، فكل ما زادت عدد مرات التحفيز كل ما صار عندنا تردد أكبر للسيلانات العصبية.
there is a certain level of frequency that is allowed. when it reaches the MAXMIUM FREQUENCY it gets stopped by the refractory period (absolute refractory period).

SLIDE 7

this picture shows a cross-section of the spinal cord.
the brown color in the picture is the white matter of the spinal cord.
the beige color in the picture is the grey matter of the spinal cord.
we have two roots coming out of the spinal cord:
1- the dorsal root
2- the ventral root
dorsal rootlets come out of the white matter to make the dorsal root and synapse with the dorsal root ganglion.
this pathway is ONLY for the sensory tract.
REMEMBER, that the sensory tract is ASCENDING so it would go from the place of the stimulation TO the spinal cord.
the ventral root does not synapse with a ganglion BUT both the VENTRAL AND DORSAL ROOTS join together to make the SPINAL NERVE.
the spinal nerve divides into four ways:
1- the dorsal ramus (thin, only responsible for the back muscles)
2- ventral ramus (thick, responsible for ALL the muscles besides the back muscles)
3- white ramus communicans (all sympathetic nerves pass through this division and synapse with the sympathetic chain ganglion)
4- grey ramus communicans (all signals would get carried out to a specific organ).

SLIDE 8

the two divisions of the posterior column can be seen in the picture. how to identify them?
in the middle of the spinal cord there is a place called the "fissure" which makes us easier to identify both divisions AND to know who is medial and who is lateral.
the part that is localized MEDIALLY (close to the fissure) is the fasciculus gracilis.
the part that is localized LATERALLY (farther from the fissure) is the fasciculus cuneatus.
the fasciculus gracilis is localized medially; because its sensory neurons come from a low level of the spinal cord (below C6).
the fasciculus cuneatus is localized laterally; because its sensory neurons come from a higher level of the spinal cord (above C6).

SLIDE 9

do not forget this order!
sensation in a certain part of the body --> stimulation by the posterior column up to the brain --> the association area of that part of the body would get stimulated in the SENSORY cortex --> the stimulation goes to the association area of that part of the body would get stimulated in the MOTOR cortex to send an appropriate response.

SLIDE 11

both anterior spinothalamic tract and lateral spinothalamic tract transmit in the same tract this is why it is called "anterior spinothalamic tract". but, to make it easier we will study each one alone.
lateral spinothalamic tract:
it is responsible of transmitting two types of sensations:
1- pain
2- temperature sensation
the anterior spinothalamic tract:
it is responsible of transmitting two types of sensations:
1- crude touch
2- pressure sensation

both crude touch and pain use a type of receptor called "nociceptors". sensation receptors use type C nerve fibers (which is the slowest) and type A delta nerve fibers. these nerve are what make the "pain pathway" which transmits the pain sensation to the brain.

there are two types of pain that can cause the stimulation of the receptors:
1- mechanical pain (that is caused by a crude touch "hard touch")
2- chemical pain (response to a mechanical pain by secreting a certain chemical such as potassium, protons, histamine, etc. which cause an inflammation)

type A delta fibers are responsible of transmitting a very fast pain sensation which is mainly caused by a mechanical pain.
مثال بيوضح هالنوع من الألم، أول ما تحس بوغز الإبرة هاد يعتبر ألم سريع ويتم نقله من خلال الديلتا فايبر.
type C fibers (which is unmyelinated) are responsible of transmitting very slow pain sensations which is mainly caused by a chemical pain.

SLIDE 12

85% of the C fibers reach the reticular formation region which is mainly responsible of sleeping/waking up.
because of this, you can't sleep when you are in a lot of pain.
only 15% of the C fibers reach the thalamus in a specific region called "intralaminar nuclei".
the C fibers would also go to other regions in the brain such as the hypothalamus (which is responsible of controlling the ANS system), by this we are controlling the autonomic response to pain.
for example, how does our body control pain? by increasing the heart rate, by increasing the blood pressure, etc.
another example, when giving birth the pain would be transmitted to the hypothalamus which would release oxytocin to increase uterine muscles contraction.

LEC 24

SLIDE 3

corticobulbar means:
it starts at the cortical part of the brain and ends at a part called the bulbar which is found in the medulla to give off its branches to the cranial nerve nuclei and then to cranial nerves that we will discuss.

the corticobulbar tract's origin is the cerebral cortex of the brain.
these tracts are going to descend throughout the whole brain to (in order):
1- thalamus
2- mid-brain
3-brain stem (specifically in the pons)
these are called the "upper motor neuron".
from the pons (in the cranial nerve nuclei) the nerves are going to branch into cranial nerves.
the cranial nerves that we need to know:
V (5) nerve: Trigeminal nerve (responsible of the voluntary action of the mastication muscles).
VII (7) nerve: Facial nerve (responsible of the voluntary action of the facial muscles).
IX (9) nerve: Glossopharyngeal nerve.
X (10) nerve: Vagus nerve
IX and X are responsible of the voluntary action of the pharynx, the soft palette and the uvula. can be called the "vocalizing muscles" or the "swallowing muscles.
XII (12) nerve: Hypoglossal nerve (responsible of the voluntary action in the tongue).
these are called "lower motor neuron".

SLIDE 4

the corticospinal tract starts from the cerebral cortical part of the brain and ends in the spine.
it goes down in the internal capsule and then to the medulla where it gives two sub-tracts:
1- lateral corticospinal tract
2- anterior corticospinal tract
till they reach the spinal cord.
THE UPPER MOTOR NEURON IS THE PART BEFORE THEY REACH THE SPINAL CORD.
ركزوا كثير على هالنقطة^^
the lateral tract then synapses with the anterior (ventral) grey horn of the spinal cord then their axons go to specific skeletal muscles.

SLIDE 5

there are two types of muscle fibers that we need to know about:
1- extrafusal fiber (outside of the skeletal muscle)
2- intrafusal fiber (inside the connective tissue of the muscle, can be called the muscle spindle)

SLIDE 6

in the muscle, we have two types of sensory neurons (AFFERENTS):
1- type Ia
2- type II
if we stretch a muscle, we would stimulate these sensory nerve fibers
which are also called "proprioceptive neurons" and the stimulation would go to the spinal cord.
the final response is received by the alpha nerve fiber which makes the muscle contract for an example.
this is called the stretch reflex or "myotatic reflex".

SLIDE 9

A lesion is any damage or abnormal change in the tissue of an organism.

lesion = جرح، تمزق

stroke = جلطة

SLIDE 14

if there is a decrease in ACh, the ACh nicotinic receptors activity would decrease.

when that happens, the muscle would get into a kind of "up regulation" to reach homeostasis.

the muscle would think that there are not enough nicotinic receptors on the surface of the skeletal muscle which would increase the amounts of ACh nicotinic receptors on the surface and would make the muscle more sensitive by increasing the sensitivity of ligand channels and increasing the sensitivity of the nicotinic receptors.

the increase in sensitivity would mean that even a small tap would stimulate the channels which would lead to the over-stimulation of a muscle.

بالعربي، أول ما يكون عندنا قلة بإنتاج الاستيل كولين حينتج منها قلة نشاط المستقبلات تاعتها الي بيكونوا موجودين ع سطح العضلة.

هاي الشغلة حتخلي العضلة تحس إنه مافي كمية كافية على السطح من المستقبلات فحتصير تكون كمية زائدة من المستقبلات الي حتخلي العضلة والمستقبلات تكون حساسة أكثر فيصير عندنا تحفيز مفرط للعضلة.

لدرجة إنه اي لمسة خفيفة ممكن تكون كافية لتحفيز العضلة .

fibrillation is a representation of fasciculation that could appear on the EMG.

EMG = electromyography

SLIDE 15

the UMN (from the cortical part TO the medulla) in the corticospinal tract is considered stimulatory.

the co-lateral tract in the corticospinal tract is considered inhibitory.

when there is a lesion in the UMN of the corticospinal tract (from the cerebral part to the medulla) there would be a decrease in the action potential.

this would increase the action potential of the LMN and the over-contraction (hypertonia) of the muscle which is caused by the alpha neuron and hyper-reflexia (gamma).

this is called "spastic paralysis".

if there is a lesion in the LMN there would be a decrease in the action potential which would lead a hypotonia (caused by the alpha nerve fiber) and hypo-reflexia (caused by the gamma nerve fiber).

SLIDE 17

if a patient has spasticity or spastic paralysis and we try to extend the arm fully, we would notice when trying to extend the arm that there is a high resistance in the patient's arm.

rigidity can be seen in two ways:

1- lead-pipe rigidity (has a very high resistance that is uniform, the patient cannot extend or flex the arm normally)

شكل الإيد بتكون زي إنبوب الكوع القائم

uniform = منظمة ودائمة

2- cog-wheel rigidity (has an on-off resistance and a zig-zag movement that is linked with tremors with patients that have Parkinson’s disease.

tremors = اهتزاز اليد بشكل غير طبيعي

Parkinson disease = الشلل الرعاشي

LEC 25

SLIDE 5

the heart has four chambers:
1- right atrium (RA)
2- left atrium (LA)
3- right ventricle (RV)
4- left ventricle (LV)
atriums are the small upper chambers
and ventricles are the big lower chambers.
left chambers are responsible of carrying oxygenated blood while the right chambers are responsible of carrying de-oxygenated blood.
the white gate-like structures in the picture are the valves.
valve = صمام
the valves are:
1- tricuspid valve (between RA and RV)
2- pulmonary valve (between RV and the pulmonary artery)
3- mitral valve/bicuspid valve (between LA and LV)
4- aortic valve (between LV and the aortic artery)

the de-oxygenated blood comes from two large veins called:
1- superior vena cava (found at the base of the heart that pumps the de-oxygenated blood from the upper part of the body to the heart)
2- inferior vena cava (found at the apex of the heart that pumps the de-oxygenated blood from the lower part of the body to the heart)

which both of them pump the blood to the right atrium. the blood would then go to the right ventricle by opening the tricuspid valve and then go to the lung (for the blood to be oxygenated) by the pulmonary artery which is done by opening the pulmonary valve to the pulmonary circulation.
the blood gets returned from the lung by the pulmonary vein (which is oxygenated) to the left atrium then to the left ventricle by the opening of the mitral valve and at the end the blood goes to the aorta by opening the aortic valve where the blood gets pumped to the systemic circulation.
REMEMBER!
arteries ALWAYS carry oxygenated blood
and veins ALWAYS carry de-oxygenated blood BUT the pulmonary artery is the ONLY artery that carries DE-OXYGENATED BLOOD and the pulmonary vein is the ONLY vein that carries OXYGENATED BLOOD.

there are no valves between the veins (vena cava and pulmonary vein) and the atriums (left and right) because the entrance of blood is easy as there is no pressure difference between them and no difference in size.
but valves are found between the atriums and the ventricles which are called "atrioventricular valves". such as the tricuspid valve and the mitral/bicuspid valve.
AND are also found between the ventricles and the arteries which are called "semilunar valves", such as the pulmonary valve and the aortic valve
the valves are between them because:
1- the difference in size between the atriums (smaller) and the ventricles (bigger), AND between the ventricles (smaller) and the arteries (bigger)
2- the pressure difference caused by the difference in size

NOTE that valves are only ONE-WAY. meaning that the blood can NEVER get back to the opposite direction (the atrium).
the valves would increase the pressure in the atrium by closing the pump to collect the blood. so when opened, ALL the blood would go easily to the ventricle in the case of the tricuspid and the bicuspid valve AND in the case of the pulmonary and the aortic valve where ALL the blood would get collected when the valves are closed and then when the ventricle is full, the pressure difference would make ALL the blood go to the arteries.
all of this happens in a PASSIVE motion, no need of energy.
a normal heart would pump THE WHOLE BLOOD from the atrium to the ventricle.

بالعربي، الصمامات بتعمل زي البوابة الي ما بتفتح لين ما تتعبى الغرفة بالكامل بحيث إنه يكون عندنا كمية كبيرة من الدم. أول ما تصير الغرفة مليانة بالدم حيطلع الدم للجهة الثانية بضغط عالي.

SLIDE 7
the right AV valve = tricuspid valve = looks like a leaflet = has three parts.

the left AV valve = bicuspid valve = matric valve = has two parts.
both AV valves has a structure called the papillary and chordae tendineae (the small strings that are seen on the right picture) that work on stabilizing and fixing the valves and not getting inverted
inverted = تقلب بالعكس
fixing = بسياق الجملة هون المقصود إنها تضل نفس ما هي
the aortic and the pulmonary valve = looks like a lunar-shaped moon
^^ شكلها زي الهلال

LEC 26

SLIDE 2

the heart can generate an action potential without the need of the CNS system.
the triggering of an action potential happens in an order.
the order is: SA node --> bachman's bundle --> internodal pathway --> AV node.
SA node = sinoatrial node
the SA node is considered the primary pacemaker; as it is the starting point of the action potential.
the internodal pathway has three parts:
1- anterior internodal tract
2- middle internodal tract
3- posterior internodal tract
the internodal pathways helps to make sure that all the atriums (left and right) get contracted.

SLIDE 3

after the action potential reaches the AV node it goes to the bundle of His.
the bundle of His is divided into two branches:
1- left bundle
2- right bundle
these two bundles can be seen in the picture on the bottom right passing with the inter-ventricular septum.
inter-ventricular septum: a wall between the left and right ventricle.
from the left and right bundles, it goes to the purkinje fibers; where it makes sure that the ventricular muscles are contracted simultaneously.
simultaneously = متزامنة
purkinje fibers are very fast fibers.
REMEMBER that the atriums get contracted first THEN the ventricles.

SLIDE 7

when we talk about the arterial blood pressure we are talking about the contraction AND the relaxation phase.
arterial blood pressure = systole/ diastole

SLIDE 8

the stroke volume (SV) is the difference between the end diastolic volume (EDV) and the end systolic volume (ESV).
SV= EDV - ESV
SV: is the amount of blood that is getting pumped PER A CONTRACTION to the systemic circulation.
SV = كمية الدم المضخة لكل انقباض واحد

EDV: is the amount of blood that remains in the ventricles BEFORE the contraction.
EDV = كمية الدم الموجودة بالبطين قبل الانقباض

ESV: the amount of blood that remain in the ventricles AFTER the contraction.
this means that the blood that has been accumulated.
accumulated = تتراكم
ESV = كمية الدم الموجودة بالبطين بعد الإنقباض
ventricle = بطين

NOTE: SV normal range is around 60 to 70 mmHg of blood per contraction.
بالعربي، كمية الدم المضخة لكل انقباض واحد عبارة عن فرق ما بين الدم الموجود قبل وبعض الإنقباض.

we need to understand two cases:
1- EDV is FAR GREATER THAN the ESV
2- EDV is LESS GREATER THAN the ESV

far greater than = أكبر بكثير
less greater than = أكبر بقليل

EDV is FAR GREATER THAN the ESV:
when the EDV is HIGHER than the ESV by a BIG difference this indicates that the heart is pumping the blood correctly and we would have a normal systemic circulation.

EDV is LESS GREATER THAN the ESV:
when the EDV is HIGHER than the ESV by a SMALL difference this indicates that the heart is NOT pumping the blood correctly and we would have problems in the systemic circulation.
why? because when the EDV is less greater than ESV this means that there is an amount of blood that is still accumulated in the ventricle.

to understand this in a better way:
EDV ↑ ↑ SV
ESV ↑ ↓ SV

INFORMATION FOR LATER:
there are two systolic volumes that can be measured:
1- early systolic volume (at the beginning of the ventricle contraction)
2- end systolic volume (at the end of the ventricle contraction)

this will also affect the pulmonary circulation and cause edema.
and also affect the systemic circulation.

to understand this in numbers, check these two examples and compare them on which is better.

if we have a patient that has an EDV of 115 ml and ESV of 50 ml then what would the stroke volume be and would there be any accumulation of blood in the chambers.
SV = EDV - ESV
SV = 115 ml - 50 ml
SV = 65 ml
this means that the blood is getting pumped from the right side to the left side normally and the blood is getting to the systemic circulation efficiently, so the patient is healthy.

another example,
if we have a patient that has an EDV of 80 ml and ESV of 50 ml then what would the stroke volume be and would there be any accumulation of blood in the chambers.
SV = EDV - ESV
SV = 80 ml - 50 ml
SV = 30 ml
this means that the blood IS NOT pumping all the blood efficiently from the right side to the left side and could face some health problems.

SLIDE 10
the vascular system order is the following:
1- arteries
2- arterioles
3- capillaries
4- venules
5- veins

after pumping the heart from the aortic artery, smaller arteries (arterioles)
deliver the blood to the rest of the body tissues. the capillaries is where the nutrient and gases exchange.
the blood would become de-oxygenated and then would go through the venules then to the veins then to the heart again by the inferior and superior vena cava.
the resistance or "total peripheral resistance" is mainly caused by the arteries as they have the MOST amount of smooth muscles compared to the other vessels.
if the arteries contract more than usual, the diameter would DECREASE and would have vasoconstriction.
vasoconstriction = تضيق الشرايين
if the arteries contract LESS than usual, the diameter would INCREASE and would have vasodilation.
vasodilation = توسع الشرايين
SLIDE 11
BP = CO * TPR
BP ↑ ↑ CO
BP ↑ ↑ TPR
CO = HR * SV
CO ↑ ↑ HR
CO ↑ ↑ SV
SLIDE 12
SNS = sympathetic nervous system
PSNS = parasympathetic nervous system
REMEMBER the CNS system DOES NOT control the heart rate BUT can modify it.
this is why the SNS and PSNS are called extrinsic conduction systems to the heart.
EPI = epinephrine
pre-load happens on the right side of the heart where more blood is getting pumped from the vena cavas to the right chambers.
after-load happens on the left side of the heart where more blood is getting from the left chambers out.
all negative effectors are called "negative inotropic agents".
and all POSITIVE effectors are called "positive inotropic agents".
SLIDE 15
agonists are drugs that are given to the patients.
antagonists are blocker drugs.
angina = ذبحة صدرية

LEC 27

SLIDE 3

CO = cardiac output
1 ml = cm^3
flow = cm^3/min
REMEMBER THAT, flow = cardiac output
another formula to find the cardiac output:
Velocity (cm^2/min) = Flow (cm^3/min)/cross sectional Area (cm^2)
V = F/A
so,
F ↑ ↑ V
A ↑ ↓ V

SLIDE 4

to understand the cross sectional area, check the arrows that are on the picture.
we see that the cross sectional area is the WHOLE LENGTH OF AN AREA of one type of vessels.
to understand more, we see that the arrow (1) in the aorta is the SHORTEST which means it has the SMALLEST cross-sectional area.
and when we reach the capillaries the arrow is the LONGEST which means it has the LARGEST cross-sectional area.
now, to remember the equation.
V = F/A
SO, when A INCREASES V DECREASES.
THIS MEANS THAT:
the velocity in the aorta is the HIGHEST
the velocity in the capillaries is the SLOWEST
a reason why the capillaries have the largest cross-sectional area and the slowest velocity; to have more time to exchange nutrients.
the velocity dramatically changes when the blood goes from:
from the aorta to the capillary (decreases)
from the capillary to the vena cava (increases)

SLIDE 7

when we are talking about the TPR we are talking about the smooth muscles in the arteries; as it has the highest percentage of smooth muscles.
*check the last lecture to remember vasodilation and vasoconstriction.
ohm's law:
F = delta P/R
F = Flow
delta P= delta of perfusion Pressure
R= total peripheral Resistance
F ↑ ↑ P
F ↑ ↓ R
REMEMBER THAT F IS THE SAME AS CO

poiseuille's law:
$R = 8*N*L/\pi r^4$
R = total peripheral Resistance
N = viscosity
L = length of the vessel
r = radius^4
R ↑ ↑ N
R ↑ ↑ L
R ↑ ↓ r
viscosity = اللزوجة
viscosity:
in patients with polycythemia, there would be an increase in the amount of red blood cells and osmolarity of the blood, which would increase the friction between the blood and the wall of the blood vessels and it would increase the viscosity of the blood.
friction = احتكاك
in patients with anemia, the would be a DECREASE in the amount of red blood cells, which would decrease the viscosity of the blood.
so,
polycythemia ↑ ↑ N
ANEMIA ↑ ↓ N
length:
when there is an increase in a person's:
1- weight
2- height
there would be an increase in the length of the vessels.
so,
weight ↑ ↑ L
height ↑ ↑ L
and
L ↑ ↑ R
radius:
the radius is the most important factor that affects the resistance. why? because it is r^4.
the relation between the radius and the resistance is the following:
r ↓ ↑ R
now, we know that the radius is affected by two things:
1- vasodilation (increases the radius)
2- vasoconstriction (decreases the radius)

SLIDE 8

in this slide we see the following.
a tank that has two tubes:
1- tube A
2- tube B
each tube has a different radius as we can see in the picture.
tube A's radius = 1
tube B's radius = 2
now, to calculate the resistance we use the following equation:
$R = 1/r^4$

so, we can know from this equation that:
tube A's resistance = 1/ (1) ^4
tube A's resistance = 1
and
tube B's resistance = 1/ (2) ^4
tube B's resistance = 1/16
so, tube B's resistance is LOWER than tube A'S resistance.
now, we can calculate the flow (cardiac output) by using this equation:
F = 1/R
so, we can know from this equation that:
tube A's flow = 1/1
tube A's flow = 1
and
tube B's flow = 1/ (1/16)
tube B's flow = 16
so, tube A's flow is LOWER than tube B's flow. why? because tube A has a HIGHER resistance.
so, we can finally say that:
R ↑ ↓ r
F ↑ ↓ R
VERY IMPORTANT TO KNOW that there is NO WAY that this difference in the radius to happen in the normal body as this difference would cause the blood vessel to blow, this is just an example.

SLIDE 10

in the graph we can see how the pressure GRADUALLY decreases going from the AORTA to the VENA CAVA.
the perfusion of the blood pressure is the difference of pressure between them.
IN A HUMAN BODY WE DO NOT USE ONE ABSOLUTE VALUE BUT WE USE THE PRESSURE DIFFERENCE TO DETERMINE THE FLOW.
the highest blood pressure is at the AORTA. why? it is the starting point of the systemic circulation.
تخيلوها زي كإنه حنفية وبربيش، الأورطة هو بداية البربيش والبطين الأيسر هو الحنفية والأوردة بيكونوا أخر البربيش.
فالضغط بيكون أقوى إشي ببداية البربيش وبيكون ضعيف بنهايته.
left ventricle = البطين الأيسر

SLIDE 11

the perfusion pressure is calculated as:
p = MAP - CVP
p = Perfusion pressure
MAP = Mean Arterial Pressure
CVP = Central Venous Pressure
the CVP is the pressure that is caused on the right side of the heart by the vena cavas.
in a NORMAL HEALTHY PERSON, the CVP is very small (around 3-8 mmHg) so we don't consider it in the equation.
this is why we say that:
P = MAP
now, the heart has two phases that are linked with the left ventricle and the aorta, we call them:
1- systolic pressure (the contraction phase)
2- diastolic pressure (the relaxation phase)
the blood pressure can be calculated through the following equation:
blood pressure = systolic pressure/diastolic pressure

the systolic pressure:
when the heart contracts the left ventricle and pushes the blood to the aorta. when the blood volume increases inside the aorta its diameter would increase and stretches its wall. but, because the aorta is made of elastic fibers it would recoil to get smaller.
WHEN THE AORTA STARTS TO STRETCH WE CAN MEASURE THE SYSTOLIC PRESSURE.
so, the systolic pressure is when the blood is getting pumped to the aorta and starts to stretch it out. the average systolic pressure is around 120 mmHg.

the diastolic pressure:
WHEN THE AORTA STARTS TO RECOIL AND PUSH THE BLOOD EITHER DOWNARD OR UPWARD TO GET BACK TO ITS NORMAL SIZE (relax) WE MEASURE THE DIASTOLIC PRESSURE.
so, the diastolic pressure is when the blood is getting pushed either upward or downward because of the recoil of the aorta to get back to its normal size. the average diastolic pressure is around 80 mmHg.

SLIDE 12

compliance = الطواعية للتمدد

we can measure the conductance by using the following equation:
C = delta V/ delta P
C = compliance
V = volume
P = pressure
so, we can say the following:
C ↑ ↑ V
C ↑ ↓ P
the conductance has a relation with the resistance as the following:
C ↑ ↓ R
and conductance can also be calculated through the following equation:
C = 1/ R
now, let's compare the arteries and the veins structure.
we see that the arteries have MORE smooth muscle layers in the tunica media than the veins.
this causes the arteries to have a SMALLER diameter compared to the veins.
so, we can say that the arteries have a LOWER COMPLIANCE than the veins.
compliance in numbers:
the veins have a 24 times more conduction than the arteries which is only 3 times as great.
this causes the veins to have about 8 times more compliance than the arteries.
this makes the veins have a function of containing the blood while the arteries have a function to push the blood.

LEC 28

SLIDE 3

laminar flow = normal blood flow

in the laminar flow, we can see that the blood is going in an ORGANIZED manner. we can also say that it goes as layers (not intercepting each other).

يعني الدم ماشي بطريقة منظمة وزي كأنهم طبقات متراصة مش متداخلة مع بعضها البعض.

an increase in viscosity in the laminar flow would mean as increasing the amount of layers (lines) and this would make the blood velocity DECREASE as there is MORE friction with the walls of the blood vessels.

a decrease in viscosity in the laminar flow would mean that the amount of layers (lines) and this would make the blood velocity INCREASE.

turbulent flow = pathological and physiological blood flow (abnormal)

in the turbulent flow, we can see the blood is going in a RANDOM manner AND NOT IN LAYERS AS THE LAMINAR FLOW.

a physiological condition that causes a turbulent flow:

the blood hitting the valves when going from the ventricles to the arteries. this can be heard as a murmur sound (look it up on YouTube to understand more).

بالعربي، هاي الحالة بتكون بسبب ارتطام الدم بالصمامات الموجودة بالقلب الي بتأثر إنه الدم ما يمشي بشكل منتظم.

a pathological condition that causes a turbulent flow:

in cases of atherosclerosis for an example, we have plaques inside the blood vessels which cause the blood flow to become turbulent. how? the plaques make up an occlusion so when the blood hits it acts in a randomized manner.

occlusion = blockage

SLIDE 4

the first graph on the top right shows the relationship between the flow and the perfusion pressure.

in the case of a laminar blood flow (normal blood flow):

perfusion pressure ↑ ↑ flow

linear relationship.

BUT THIS DOES NOT MEAN THAT IT WOULD ALWAYS INCREASE AS THE FLOW STOPS AT A POINT WHERE IT BECOMES CONSTANT

constant = ثابت

why? because regulation would happen to keep it in its normal range.

in the case of a turbulent blood flow (abnormal blood flow):

the flow INCREASES till a certain point. this is why we see in the graph that the line starts to become flat.

بالعربي، تدفق الدم حيزداد لحد معين، بعد هيك بتوقف تزداد ويتقدروا تشوفوا كيف الخط بيصير مسطح (بدون أي زيادة).

the second graph on the bottom right shows the relationship between the flow and the perfusion pressure AND how it correlates with resistance.

REMEMBER LECTURE 27: FLOW HAS AN INERVSE RELATIONSHIP WITH RESISTANCE.

Flow = 1/resistance

so, when Flow ↑ ↓ Resistance.

now we can say that:

1- laminar blood flow has a low resistance.

2- turbulent blood flow has a high resistance.

SLIDE 5
the equation that we have here is:
tension = pressure * radius
as we can see from the equation the relation between tension and the following are:
Tension ↑ ↑ Pressure
Tension ↑ ↑ Radius
now, when the perfusion pressure INCREASES the tension of the blood vessel walls INCREASES. BUT, the body regulates it by DECREASING THE RADIUS (vasoconstriction) so that it regulates the tension. how? by releasing certain agents to stimulate the DECREASE of the diameter.
DO NOT FORGET, when the radius DECREASES the blood flow would also DECREASE and the resistance INCREASES.
why does the body regulate the tension on the blood vessels? so it doesn't get torn.
torn = تمزق
the opposite happens if the pressure decreases.
SLIDE 8
REMEMBER, one cardiac cycle = one heart rate
diastole = relaxation phase
the first step of the cardiac cycle, mid to late ventricular diastole:
after pumping the de-oxygenated blood from the vena cavas to the atrium the atrial pressure INCREASES in both atriums (left or right, remember that the heart works simultaneously) and the pressure in the atriums would be > the pressure in the ventricles (left or right).
this triggers the AV valves to open.
AV valves:
1- tricuspid valve on the right side
2- mitral (bicuspid) valve on the left side
and the blood would flow to the ventricles.
now, the blood needs to go to the arteries (pulmonary artery on the right side and aortic artery on the left side) and because the arterial pressure is > than the ventricular pressure the SLV (semi-lunar valves) WOULD NOT OPEN UNTIL the ventricular pressure is > than the arteries.
SLIDE 9
because the AV valves have opened, the blood would flow to the ventricles and the pressure would in the VENTRICLES > than the atriums this would make the AV valves close, how? by the contraction of the chordae tendineae and the papillary muscles.
للي مش شايف الأرقام الموجودة على الصورة
the right ventricle has a pressure 7 mmHg and the pulmonary artery has a pressure 10 mmHg.
the left ventricle has a pressure of 60 mmHg and the aortic artery has a pressure of 80 mmHg.
this means that the ARTERIES pressure is still > than the ventricles pressure
this also means that the SLV valves would still be closed.
because of this step, we can put the stethoscope on the chest of the patient where the Av valves are found and we can hear a sound called "S1" which sounds like the word "LUB"
SLIDE 10
as the step before it, the AV valves would be closed because the ventricles pressure > atrial pressure.
BUT, the ventricular pressure would be > than the arterial pressure this makes the SLV valves open and the blood to flow from the ventricles to the arteries.
للي مش شايف الأرقام الي عالصورة:
the right ventricle has a pressure 25 mmHg and the pulmonary artery has a pressure 10 mmHg.
the left ventricle has a pressure of 120 mmHg and the aortic artery has a pressure of 80 mmHg.

SLIDE 11

the last step of the cardiac cycle is the isovolumetric relaxation.
the ventricles pressure would still be > than the atriums pressure and would still make the AV valves closed.
but the arterial pressure would be > than the ventricles pressure which would make the SLV valves close again.
when the SLV valves close again, they make the second sound which can be heard if we put the stethoscope on the SLV valves on the chest of the patient which is the "S2 sound" which sounds like the word "DUB".

LEC 29

SLIDE 2

just to remember,
cardiac output = cardiac rate * stroke volume
cardiac output ↑ ↑ cardiac rate
cardiac output ↑ ↑ stroke volume
DO NOT FORGET,
stroke volume = EDV-ESV
so,
Stroke volume ↑ ↑ EDV
how do we increase the EDV? by increasing the venous return which increases the quantity of the blood inside the chambers and increase the pressure and tension against the walls of the heart.
this is the principle of the frank-starling law where if you increase the quantity of the blood inside the chambers of the heart the tension would increase inside the heart and the stretching of the cardiac muscles, this would increase the EDV and in turn the stroke volume would be increased.
and as we said before, when the stroke volume increases the CO would increase.
as we studied before, the nervous system only MODIFIES the heart rate but DOES NOT CONTROL IT.
this can be either by:
1- increasing the heart rate
2- decreasing the heart rate
3- increasing the contractility
4- decreasing the contractility
PACEMAKERS SUCH AS THE NODAL CELLS ARE THE ONES THAT TRIGGER THE ACTION POTENTIAL.
cardiac rate can be modified with the SNS (Sympathetic Nervous System) and the PSNS (Parasympathetic Nervous System).
the SNS can:
1- increase the cardiac rate
2- increase the contraction strength (increase the stroke volume)
the PSNS can:
1- decrease the cardiac rate
both the SNS and the PSNS are somewhat controlled by the mean arterial pressure to keep it in its normal range.
we have two types of agents that can modify the contractility of the heart:
1- (+) inotropes (+ contractility)
2- (-) inotropes (- contractility)

SLIDE 7

in the graph we can see three cases of ventricular compliance:
1- decreased compliance
2- normal compliance
3- increased compliance

the graph shows how compliance is related to the pressure which is on the Y-axis and the volume in X-axis.

things to know about the graph:
EDP = end diastolic pressure
EDV = end diastolic volume
compliance = طواعية

as we studied in lecture 27, compliance is:
C= V/P
the graph shows that in the beginning there is somewhat of a linear relationship between the volume and the pressure BUT when it hits a certain point, the pressure and the volume DRAMATICALLY increase causing a non-linear equation.
linear = خطية
non-linear = غير خطية
this is because when the volume inside the ventricles increases the compliance of the wall decreases (STIFFNESS INCREASES) as the ventricular walls are stretched. this also means that the ventricles have reached the maximum volume and cannot be filled more this is why the pressure is increasing FASTER THAN THE VOLUME.

بالعربي، كل ما ازداد حجم الدم داخل البطين كل ما قلت الطواعية لإله فبيصير مشدود بشكل كبير بسبب التوتر وبتزداد قساوته.
تخيلوها زي البالونة، كل ما تنفخ كل ما بتصير قاسية بسبب امتلاءها بحجم كبير من الهواء.

check how the EDP is different in the three cases that we have.
first we set an EDV volume that we want to measure the pressure at which is around 120 ml (the highest volume that the ventricle can be filled) and now we measure the EDP pressure.
كل الأرقام الي حكيها ممكن ما تكون صحيحة بس المهم توصيل الفكرة
1- decreased compliance = 32 mmHg
2- normal compliance = 12 mmHg
3- increased compliance = 6 mmHg
now, we can understand that the pressure is completely different in the three types EVEN WHEN the volume is the same.