IN THE NAME OF ALLAH

page 19 :
one of the major features of skeletal muscles and cardiac muscles >> the presence of **T-tubules**
T-tubules: invagination of the cell membrane, deep into the muscle fiber
what for?? to conduct the **action potential**
action potential: wave of depolarization
at both sides of T-tubules, there are **terminal cisternea**
terminal cisternea: store for calcium
*So conduction of depolarization deep into the muscle fiber >> help in exit of Calcium from its stores
Ryanodine Ca+2- release channels called so >> because they affected by a drug called Ryanodine
BOTH (cardiac and skeletal muscles) need Ca+2 for contraction
the **increase in intracellular Ca+2 concentration** in muscle fiber >> it will combine with **troponin** (part of the thin filament) >> their binding moves **tropomyosin** away from the **myosin-binding sites on actin** >> give them chance to interact

page 20 :
what's **different** between cardiac and skeletal muscles?? in **skeletal** muscles, Ca+2 **only** originate from sarcoplazmic reticulum BUT in **cardiac** muscles, Ca+2 inter from outside to induce it's release from inside **((Ca+2 induced Ca+2 release))**
Inword Ca+2 current >> opens Ca+2 release channels in sarcoplazmic reticulum (induce Ca+2 release)
in **Both** (cardiac and skeletal muscles) >> the **increase** in intracellular **Ca+2** >> the **more forceful/powerful contraction**
the powerful of contractility of the muscle, proportionally depends on intracellular Ca+2 concentration
during **contraction**: **Ca+2 binds with troponin C**
to **relax** the muscle: **Ca+2 must return back to its stores (sarcoplazmic reticulum) and to the outside**
excitation-contraction coupling ازدواجية التنبيه والانقباض :
دائما **التنبيه الكهربائي** يتبعه **انقباض ميكانيكي**
action potential >> Ca**+2 induced Ca+2 release** >> **increase intracellular Ca+2 (electrical stimuli**) >> tension or contraction **(mechanical contraction)**
contraction of cardiac muscle (systole) must be followed by relaxation (diastole), and this is **NOT necessary in skeletal** muscles
in **mechanism**s by which **Ca+2 is lowered** to resting level to produce **relaxation** :
(A) and (B) Ca+2- **ATPase** pump because it move Ca+2 **uphill against consentration gradient** so it needs **energy**
(C) **Ca+2 - Na+ exchanger** :
Na+ enter **downhill** >> leads to exit of **Ca+2 uphill**
*the energy that result from Na+ entrance is used in Ca+2 exit
(D) action potential : depolarization followed by repolarization
during repolarization : **voltage gated Ca+2 channels close** >> so decrease intracellular Ca+2 concentration

page 22A :
BOTH (cardiac and skeletal muscles) contain **cross striations**
cross striations : actin and myosin arranged in **sarcomeres**

sarcomere is the distance between 2 Z-lines
major **differences between cardiac and skeletal muscles :
*point (2) : **sarcoplazmic reticulum less well developed in cardiac** muscles and it's smaller in size
(3) **T-tubules** :
in **skeletal** muscles: between 2 terminal cisternea
*terminal cisternea : is the collection of sarcoplazmic reticulum
(terminal cisternea _ T-tubule _ terminal cisternea) >> **triad**
in **cardiac** muscles: **diad**
here, **sarcoplazmic reticulum** does not form cisternea, it forms **small expansion** close to T-tubules (expansion _ T-tubule) >> diad
(4) always, **cardiac muscles more vascularized** than skeletal .. why??
because it's a **hard worker** muscle needs a **lot of nutrients and energy** >> so **more mitochondria** and **highly vascularized**
(5) ****skeletal** muscle **fiber** = skeletal muscle **cell**
inside the cell/fiber > there are fibrils > which consist of thick and thin filaments
BUT **cardiac muscle **fiber** = a group of **cells** (**myocytes**), joined together by **intercalated disc**
*the most important component of intercalated disc is ((**gap junctions**))
gap junction: an area of low electrical resistance, allowing direct spread of low electrical currents, make cardiac muscle function as **functional syncytium**
*when I stimulate one cardiac muscle cell, during seconds every cardiac muscle contract .. why ??
the action potential move from one cell to another Quickly through GAP JUNCTIONS, so every cell stimulated

page 22B :
types of contraction : ***isotonic** ***isometric**
both result from the **interaction of actin and myosin**, their interaction result in **transduction** of **chemical energy (ATP)** to **mechanical energy (طاقة حركة)**
***isotonic**: **muscle shortens**, **tension** remain **constant** ex: reflexion, extension
***isometric**: **length** does **not change**, **tension develops**
Both applied **for Both** cardiac and skeletal muscles
**systole in the heart (left ventricle)
*at the beginning **isometric contraction** : the muscle contracts with **no length shortening** >> the resulting **energy increase blood pressure inside the ventricles** >> when the blood **pressure in the left ventricle exceeds that of the aorta** >> aortic valve **opens**
*aortic valve opens>> now, **pumping action** is needed >> **isotonic** (muscle shortens) >> Pressing more and more on the blood >> pump it **(ejection)**
isotonic and isometric contractions occur **simultaneously

page 23 :
what happens if a **2nd action potential occurs in a muscle fiber .. **before complete relaxation** ??
the 2nd response will be **added to the first** (the 2nd contraction will be added to the first) >> **temporal summation**
does **tetanic contraction occurs in living bodies?? or does it exclusively occurs **after death (in rigor mortis)**?
of course, it **occurs during life**, when you **hold** sth and **keep it up** >> **continuous/sustained/tetanic contraction**
BUT this does**n't** occurs in the **heart**

in cardiac muscle, systole must be followed by diastole .. why??
during relaxation, heart filled with blood
if relaxing/diastole time shortens >> cardiac output will decrease
**tetanic contraction doesn't occur in cardiac muscles, and occurs in skeletal
*in **skeletal** muscle : the **action potential**= 0.1 millisecond (**very short time**)
the muscle twitch (contraction, relaxation) = 100 millisecond
if you **stimulate** the skeletal muscle **during**(contraction or relaxation) phase, you will get a **response** >> because **action potential duration** approximately is **over** (absolute)
*in **cardiac** muscle: the **action potential**=250-300 millisecond **(very long time), extend with muscle twitch** (contraction, relaxation)
during **plateau** : **Ca+2 enter** (Ca+2 induced Ca+2 release)
if you **stimulate** the **cardiac** muscle **during** (contraction or relaxation) phase, you wo**n't** get a **response (refractory period**)
it's a **boon of our god that **cardiac** muscle **physiologically** does **not tetanize**
according to its nature, contraction (systole) must be followed by relaxation (diastole)
**BUT if we give a patient Ca+2!
previously, in 40s of last century, patient went to the doctor with any disease, and becouse drugs were limited, cured by that magic drug ((IV Ca+2))
***if you need to give Ca+2 (IV), give it slowly**
ex: 10 ml during 10 min
if you give it very quickly, tetanization occurs ((continuous systole))
***so normally (physiologically) >> no tetanization**
artificially >> :/

page 24 :
we said that our body is divided into **motor units** BUT the **heart is not.**
motor unit: a **group of skeletal muscle fibers** and a **branch of nerve supplying them**
*the **motor unit of the small muscles of the hand**, that make you write, play piano ... , consists of **one or two fibers**
BUT in **gluteus maximus** the **motor unit** consists of **hundred fibers**
if you **stimulate motor unit, you will get **All or None response** (either it responds or not)
stimulus >> depolarization >> down to threshold >> opening of Na+ channels >> firing action potential >> contraction
Independent of the **stimulus intensity**, what's important is **down to threshold stimulus**
now, Cardiac muscle as a whole, let's say that it consist of **1000 motor unit, **each of them has it's threshold**
*the same thing is in **skeletal** muscle (**many motor unit**), if you **stimulate it**, you will get **graded response**
زيادة تدريجية في قوة الانقباض .. why is that ??
because muscle contains **many motor unit** >> **each** of them **has it's threshold .... increase intensity of stimulus** >> **stimulate more and more motor unit** >> **more and more powerful contraction**
this **phenomenon** is called : **motor unit recruitment ((spatial summation))**
at the level of **motor unit : all or none (intensity ×)
at the level of the **whole muscle : spatial, graded (increase the intensity of stimulus >> more powerful contraction)**
another way to **increase the force of contraction is to **increase** the **FREQUENCY** of action potential
*** increase intensity >> spatial summation**
*** increase frequency >> temporal summation**

so in **skeletal** muscle **graded** response may occur **BUT** of course this wo**n't** occur in **cardiac** muscle, because of **gap junctions** that conduct the action potential

page 26 :
length - tension relationship :
if we **stretch** the fibers of skeletal muscle, we will get **((passive)) tension**
*contraction is interaction between actin and myosin
increase the number of cross bridges >> increase the power of contraction
*there is another type of **tension** result from **stretching of CT (elastic tissue)**
if the skeletal muscle fiber become stimulated, and it's length become that certain length at which we get strong contraction : ((optimal length**)) : at which **sarcomere length = 2.2 micro meter**
*if the length of sarcomere is more or less >> tension differs
***resting position = optimal position = optimal length**
here, if you **stimulate** the muscle you will get the **strongest** contraction
and this indicates the **interaction between actin and myosin**
the **2nd case of sarcomere length:
actin overlapping over the myosin >> myosin pull it to the center of sarcomere (M line) >> make the two Z lines closer (lesser-in-length sarcomere)
BUT if the muscle was **over stretched (**130% of its normal length**) like in the **3rd case** >> there is **no interaction** between actin and myosin >> **lesser tension**
as in the **1st case if sarcomere length was **less than normal (70% of it's normal length**),((actin interact with actin)), **actin overlap with actin** instead of overlapping with myosin**, prevent actin-myosin interaction** >> **lesser tension**
****length - tension relationship** in case of **cardiac** muscle:
passive stretch occurs to the cardiac muscle .. how ?? by its **filling** with blood
if the filling was normal ((130 mille liter)) (end diastolic volume) and you make
***considerable stretch >> powerfull contraction**
*** excessive stretch >> weak contraction**
when does ventricle **over-filled**??
hypervolemia or **MI** >> **cardiac output decrease** >> **inadequate** amount of blood arrive to organs >> **ischemia**
ischemia of kidneys activate **renin-angiotensin aldoseron system**
renin released >> converts **angiotensiongen** into **angiotensin 1** >> which is converted in the **lungs** into **angiotensin 2**;
angiotensin 2 **functions**:
1. the most powerful vasoconstrictor >> ^ blood pressure
2. induce the release of aldosteron from supra-renal cortex >> induce Na+ release from the cells >> (water follow Na+)
so hypervolemia occurs in heart failure cases
>> overstretch >> weaker contraction and lesser cardiac output

****nomenclature:**
***end diastolic volume** : the volume of the blood that fill the ventricle during diastole (come from the atrium)
***venous return** : the blood that fill the atrium (return from all body organs except the lungs)
***cardiac filling pressure** : blood pressure in the filled atrium
increase venous return >> ^ cardiac filling pressure >> ^ end diastolic volume >> more and more stretch of the cardiac muscle (if it was within physiological limit > ^ powerfull of the contraction)

within physiological limits:
the more heart is filled with blood during diastole >>
the more it will pump during systole

ANATOMY
to examine X-ray chest, you have to assume it's volume, it's edges
****Anterior view**:
***Rt border : Rt atrium** (superior to it: SVC ,,, inferior to it: IVC)
*almost two thirds of the **anterior surface : Rt ventricle**
***ft border : ft ventricle**
most of the **ft ventricle** (2/3) appear in the **posterior view**
((the apex of the **left ventricle = **apex** beat))
posterior to it, **base which consists **mainly of left atrium**
****descending aorta is very close to esophagus**
only small part of **left atrium appears **anteriorly** called **auricle**
****pulmonary trunk** march out from the **Rt ventricle**, and it divides into **Rt & ft pulmonar arteries (**the **Rt is taller)**
**aorta march out from the ft ventricle; ascending > arch > descending
***from left to the right : SVC , ascending aorta , pulmonary**
****SVC** .. how it forms??
Rt & ft brachiocephalic veins (innominate veins)
****ft brachiocephalic vein is taller than the Rt**, and it **pass anterior to:**
1. ft common carotid artery
2. ft subclavian artery
3. Rt brachiocephalic artery

"الأطباء صاروا يعلموهم موظفين الدعاية!!"

(": احنا جيل التغيير)