Physiology CVs
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Lecture-9-

Ω Cardiac cycle:

- Is the set of cardiac events that occur from the beginning of one heart beat to the beginning of the next one.

I.e: from systole to the next systole or from Diastole to the next diastole and contain 1systole= contraction=ejection of blood,1Diastole = relaxation =filling of blood.

As we said we have 2syncitium**; Atria**l & **ventrical** so we have Atrial contraction "Atrial systole"

-Atrial relaxation "atrial diastole"

-Ventricular contraction "ventricular systole"

-Ventricular relaxation "ventricular diastole"

When we say diastole and systole without specification we mean "ventricular".

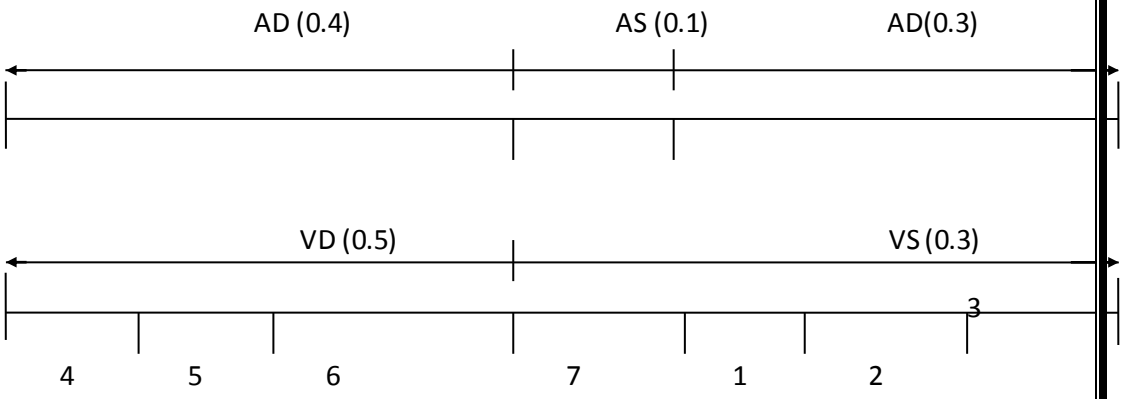
As we said before cardiac cycle normally=0,8sec for Atrium(systole=0.1 sec and diastole=0,7sec),for ventricle(systole=0,3sec and diastole=0,5sec).

Note:

Atrial systole occur while the ventricle is in diastole .

Ventricle systole occur while the atrium is in diastole.

So Atria l systole and ventricular systole will not occur simultaneously.



*Starting from **point 1**=the starting ventricular systole ,at this point the ventricle finish the diastole phase and is filled with blood .Here the ventricle pressure become**>** Atrial pressure so,

AV valve will close .here the aortic valve still closed because the ventricular pressure still **<** aortic pressure.

So, Atrial pressure **<** ventricular pressure **<** aortic pressure & all the valve are closed.

From1→2

The ventricular pressure start to rise by contraction to overcome aortic pressure ,but the 4 valve are closed so here **No** blood ejection occur →the volume does not change.

→here we have contraction without change in volume→"**Iso volumic contraction**".

Point 2

Ventricular pressure become **>** aortic pressure so the aortic valve will open and ejection of blood starting here.

From 2→3

Rapid ejection of blood occur here 70%of the blood is ejected in this phase although it is only 1/3 of the ejection phase.

Here the ventricle →still contracted →pressure still rising

aorta→ receive blood→↑pressure but ventricular pressure here > aortic

At point 3

Ventricle pressure reach a max value which called systolic pressure(normally120).

From 3→4

Here ventricle still contracted but the pressure start to decrease ,why?→ because ↓ in blood volume(ejected to aorta) . Aortic pressure start to decrease ,even though blood still flow to it from ventricle ,why ? because the blood is flow from aorta to distal arteries. So, ↓ in ventricular pressure,↓ in aortic pressure → but V>A.

But blood continue to flow ,but with slower rate→ slow ejection phase. Near the end of this phase aortic pressure become > ventricular pressure ,but blood still flow because aortic valve close because of the blood momentum.

At point 4

Aortic pressure > ventricular pressure so ,aortic valve will close → no more ejection of blood .So, from 1→4 is the ventricular systole which compose of→

- Iso volumic contraction
- rapid ejection phase
- Slow ejection phase

During the same period 1→4(AV valve is closed ,atrium is being filled with blood " atrial diastole").

Point 4 is the end of ventricular systole and the beginning at ventricular diastole .here aortic valve is closed →aortic pressure > ventricle pressure and AV valve still closed → atrial pressure < ventricle pressure.

→Aortic > ventricular > atrial

4→5:ventricle start to relax →pressure start to decrease→ to become < atrial pressure.

*All valves are closed →**no** change of volume

*↓ pressure ↔volume → Iso volumic relaxation.

At point 5

Ventricular pressure become < atrial pressure →AV valve open →flow of blood from atrium to ventricle start here.

5→6

Rapid flow of blood from atrium to ventricle →rapid filling phase.
Atrium still in the diastole phase → No contraction but blood flow because of the pressure gradient .

6→7

Ventricular filling continue but at slower rate.
The longest phase in cardiac cycle .
Called reduced ventricular filling **Diastasis**.

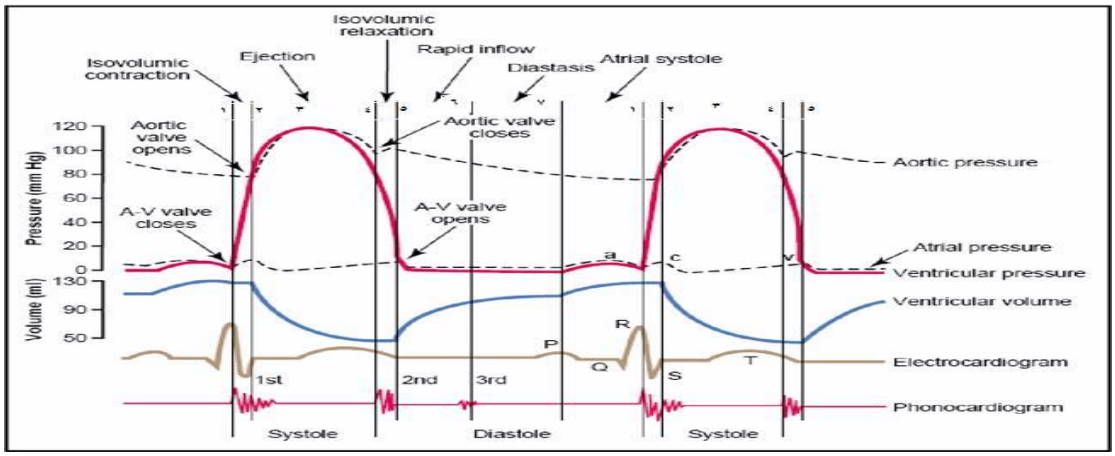
This period ↓by increasing heart rate. During the last 2 periods →rapid ventricular filling &reduced ventricular filling .The atrium still relaxed and the ventricle receive 75%of blood

7_1

The atrial systole .The atrium contract and push the remaining 25%of blood to ventricle .At **point 1** the ventricle is filled with blood . so, it's pressure become > atrial pressure → close of AV valve and the cycle again.

So 4→1 is the ventricular diastole which compose of:

- Isovolumic relaxation
- rapid ventricular filling
- reduced ventricular filling
- atrial systole



We will study some of the variable in the cardiac cycle..

Heart sounds;

When the valves close → it will cause sounds.

When the valves open →No sounds are generated

Normally there is 2 heart sound

"s1" **1st sound**

When the AV valve close ,at what point of the pervious figure?....

→ longer ,louder, low frequency

"s2" **2nd sound:**

When the semilunar valve close ,at what point?....

→shorter ,higher frequency

S1 is the end of diastole and the beginning of systole

S2 is the end of systole and the beginning of daistole

→**D** between s2-s1

→**S** between s1-s2

There are 2 other sounds Not related to valves which is→ **S3** due to rapid filling of ventricle.

Normal in young and pregnancy , pathological in adults.

S4 due to atrial contraction.

2-ventricular volume

At the end of diastole(point 1) the volume in the ventricle called End diastolic volume EDV.

During the iso volumic contraction(point 1→2) the volume remain the same.

During the rapid ejection phase(point 2→3) 70% of blood is ejected →sharp ↓ in ventricular valve.

During the slow ejection phase(point3→4) 30% of blood continue to flow→ the volume continue to decrease but at slower rate.

At the end of previous phase ,the aortic valve close.

→what has been ejected in the previous phase(systole) called stroke volume

→What remain in the ventricle called the End systolic volume(ESV).

Note: stroke volume = EDV-ESV

During the isovolumic relaxation (point4—5) the volume remain the same.

During the rapid filling phase(point 5—6) sharp ↑ in ventricular volume .

During the reduce filling phase (point6 –7) blood continue to flow from atrium to ventricle →the volume continue to increase

During the atrial contraction(point7—1) the volume continue to increase to reach the max at the End of diastole(EDV) at point 1.

3-ventricular pressure

At point 1 which resemble the End of diastole, AV valve close (why?) →vent p > atrial p but aortic valve still closed(why?) vent p< aortic p.

In the iso volumic contraction (from 1to 2)→sharp rise in ventricular pressure to overcome aortic pressure.

At the end of iso volumic contraction(point 2) ventricular pressure > aortic →aortic valve open.

During the rapid ejection phase (from 2—3) vent pressure continue rising to reach it max value at the end of this phase(point3).

From point 3—4 which resemble reduced ejection phase the vent pressure↓ because of loosing volume.

At the end of systole ,aortic valve close because vent pressure become < aortic

AV valve still closed,(why? →vent p > atrial p).

In isovolumic relaxation phase, **from 4 to 5**, the pressure decrease sharply ,so that atrial pressure > vent pressure and AV valve open.

During the rapid filling phase**(from 5—6)** and the reduced filling phase(**from6—7**)

Very slight rising In pressure because↑ volume but the vent pressure still< atrial pressure →flow continue in the atrial contraction(from 7—1) another slight ↑ in pressure because of more↑ in volume → the cycle continue…

4-Aortic pressure

During the ventricular diastole ,aortic pressure is decreasing ,because blood flow from the aorta to the small arteries.

At the End of isovolumic contraction(**point2**) the aortic pressure reach it's lowest value which called the diastolic pressure **normally80 mmHg** ,here the aortic valve open because vent p >aortic p and blood start to reach the aorta ,exerting force at the walls of aorta→ ↑pressure.

At the end of rapid ejection phase ,the aortic pressure reach it's max value →systolic pressure(normally 120) because of the huge amount of blood which exert more and more force on it's wall.

During the reduced ejection phase ,aortic pressure starts to ↓even though ,it still receiving blood from ventricle…why?→blood flow to smaller arteries.

At the end of the previous phase(point4) vent pressure<aortic pressure and aortic valve close.

During the next phases we expect to see continuous decreasing in the aortic pressure ,because blood flow to the smaller artery, but during the isovolumic relaxation there is slight ↑in aortic pressure called(**dicrotic notch**) or(**incisara**),because after closure of aortic valve some blood will flow back to the(aortic valve)from recoiling of the elastic arteries.

Toward the end of ventricular diastole ,the atrium will contract →this will cause ↑in atrial pressure which form**(a wave)**.In the isovolumic contraction →the blood in ventricle will push the AV valve →bulging of AV valve to the atrium→ another wave in the atrium pressure→(**C wave**).After the opening of aortic valve at the end of isovolumic contraction →blood flow to the aorta →the AV valve descened again →slight↓ in the pressure.

After that the pressure start to rise again..

Because filling with blood while AV valve is closed to react another peak which is (**V wave)** immediately before opening of AV valve..

***Clinical application**

In case of AV valve stenosis → ↑pressure in atrium when contract due to ↑ resistance

→ **A wave** will be more prominent and called Common wave.

Summery**:** **A wave** → atrial contraction ,associated w **S4**,preceded by P in ECG

C wav e→ during isovolumic contraction.

V wave →at end of isovolumic relaxation.

Note:

Ventricular pressure →**left** Diastole=0mmHg , systole=120 mmHg

Right → Diastole= o mmHg ,systole=25mmHg

Aortic P→ Diastole=80mmHg , systole=120 mmHg

Pulmonary P→ Diastole =8mmHg , systole=25mmHg

Clinical issues

The pressure gradient between ventricle and aorta during systole is very small 'about1-2mmHg' but in aortic stenosis →the vent pressure will ↑to overcome the resistance of valve →the pressure gradient will ↑ .

In case of aortic regurge the dicrotic notch will be less prominent because the blood will flow back to heart rather than exerting force on walls.

The most important in cardiac cycle is to know the correlation between different events ,and to know which come with which.

Examples.

1) QRS in ECG→ come before isovolumic contraction because electrical event come before mechanical occur

→associated with 1st heart sound →close of AV valve which occur before contraction of ventricle.

2)T wave →occur before isovolumic relaxation before elect Evert precedes mechanical.

→associated with 2nd heart sound →close of aortic which occur before relaxation of vent

p wave →comes before atrial systole

Cardiac output:.

During ventricular systole ,blood is ejected to the aorta.

During ventricular diastole ,blood flow from atrium to ventricles.

At the end of ventricular diastole ,the ventricles are filled with certain volume of blood ,we call it ,End diastolic volume(EDV):the volume in the ventricle at the End of diastole ,immediately before contraction.

During the systole ,not all the (EDV)will be ejected.

But: part will be ejected

Part will remain in ventricle .

The part of blood that remain in the ventricle after finishing the systole is called End systolic volume(ESV).

The part of blood that was ejected in systole is called **stroke volume**(S.V).

→So, EDV will be divided in 2 part:

●S.V→will be ejected

●ESV→what remain in the ventricles

EDV=SV+ESV

→ **SV=EDV-ESV**

SV is the blood volume that was ejected in 1 systole(in one cardiac cycle).

We know that cardiac cycle normally=0,8sec

So in 0,8 sec →the heart will eject SV

Q: in 1 minute what is the volume being ejected?

In 0,8sec→sv

60 sec→?

?=60/0,8 * sv

?=75*SV

75 is the heart rate→ munber of beat in 1 minute

So ?=SV*HR

?= the amount of blood being ejected in 1 minute and called cardiac output(CO).

→ C.O=SV*HR

Ejection fraction: used to determine the function of the heart =SV/EDV *100%

If heart work effectively →good contraction→ ↑ SV

→ ↑ ejection fraction

If heart is week→ week contraction→ ↓ SV, ↓ejection fraction.

Normally ejection fraction>55-75%

If we have 2 persons:

The 1st has SV=70 and ejection fraction 40%

The second has SV=70 and ejection fraction 65%

→although both have the same SV but the 1st one is abnormal because his ef is low

So: SV=EDV-ESV

C.O=SV* HR

e.f=SV/EDV*100%

cardiac output regulations:

C.O=HR*SV

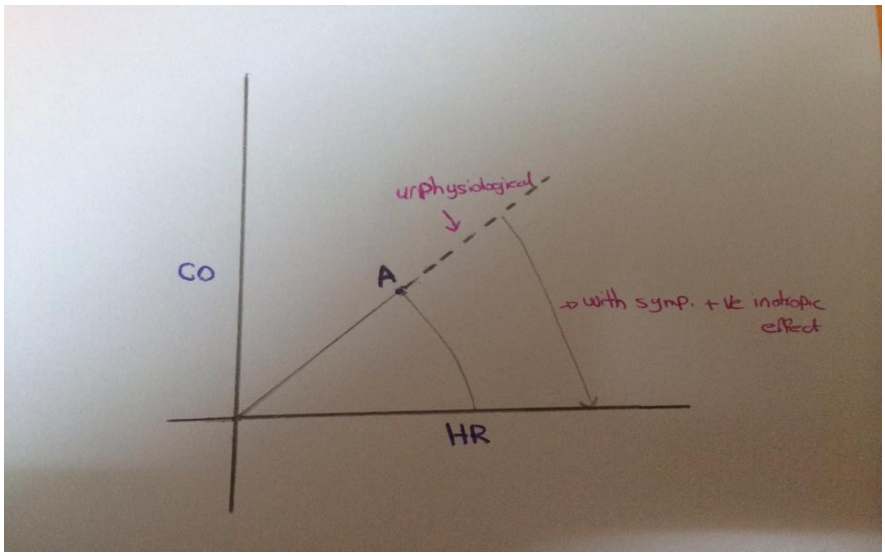
→Co is affected by changes in SV and changes in HR.

***Cardiac output and heart rate.**

We suppose that as we increase HR the cardiac output will increase too,(CO=HR*SV).

This is true up to certain point, were ↑ HR will decrease SV, why? As we study in the cardiac cycle, the ventricle are filled of blood in the diastole. If we increase the heart rate too much the time allowed for filling the ventricle will decrease → decrease filling→ decrease SV.

So the relationship between CO and HR will be as following curve



As HR ↑ the CO ↑ up to certain point A ,then as HR↑ the CO start to ↓ because SV↓ → because the time allowed for ventricle filling (diastole)↓.

In previous curve if there was sympathetic inotropic effect.(↑muscle contraction).

If the heart is facing afterload,it's contractility would increase (sympathatic activity)to comensate→ C.O is kept constant.

Conclusion: up to physiological level(normally170) cardiac output is net a fraction of after load.

C.O and SV: SV is affected by 3 major factors:

Preload , afterload]→intrinsic

Contractility →extrinsic

After load = the workload imposed on the heart after contraction and determined by the aortic pressure (diastolic one=80)

I,e the pressure that the ventricle must overcome to start ejection of blood.

If the heart is facing increasing afterload(BP), we suppose that the SV will decrease ,but in our body there is a compensatory mechanism which is sympathetic activity→ will increase contraction →SV will not decrease although afterload is↑ .

As long as systemic arterial blood pressure is less than 170mmHg,cardiac output remain constant.

At 250 mmHg ,cardiac output = zero.

So this is effect of afterload,what about preload??

Preload: the workload imposed on the heart before contraction which is the tension in the muscles before contration, it corresponds to (EDV ,Rt atrial pressure,venousreturn).

As we increase the length of muscle → the tension will increase up to certain point this is the length-tension relationship(frank-starling)

→ ↑length → ↑ tension up to certain point.

In cardiac muscle we used the EDV instead of length.

As we increase the EDV→ the tension in the ventricle will increase up to certain point

*summary:

preload→ is determined by EDv →↑ preload =↑ Edv= ↑ RTatrial p=↑ VR

this will ↑ the tension→ ↑ contraction force → ↑ SV

→ ↑ EDV → ↑SV up to certain point.

EDv→ ↑ co upto certain point

The third factor which affect SV is contractility which is extrinsic factor.

Contractility=force of heart muscle contraction

1-Which fixed EDV→ ↑ contractility will ↑ SV by ↓ ESV(not as frank starling in which ↑EDV cause ↑in ESV with fixed ESv.

2-↑By : sympathetic ,NE &epinephrine, Ca, glucagan.

↓by → acidosis ,↑$K^{+,}$ calcium channel blocker

finally

cardiac reserve the difference between max(CO) that can be reached by Frank starling low(which is 15L/min) and normal working CO (which is 5L/min).

it is different from max (CO) that can be reach by both: frank starling which may reach 35L/min in atria,and contractility.

The End

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