Cardiac Physiology

Valve Movements, Heart Sound, Cardiac Cycle

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Cardiac Valves

- No valves between the veins and the atria.
- Blood flows from the atrium to the ventricles→back into the veins
- Atrial contraction
  - not a very effective method of filling the ventricles.

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Cardiac Valves

- Right atrio-ventricular valve
  - tricuspid
- Left
  - mitral valve.
- Openings of these valves are
  - large so as to allow a rapid inflow of blood into the ventricles
Fig. 20.04b

Fig. 20.05

Superior view (the atria have been removed)
Fig. 20.06c,d

(c) Superior view with atria removed: pulmonary and aortic valves closed. Bicuspid and tricuspid valves open

(d) Superior view with atria removed: pulmonary and aortic valves open. Bicuspid and tricuspid valves closed

Fig. 20.06e

(e) Superior view of atroventricular and semilunar valves

Pectinate muscle of right atrium

TRICUSPID VALVE

PULMONARY TRUNK

Pulmonary trunk

RIGHT CORONARY ARTERY

AORTIC VALVE

BICUSPID (MITRAL) VALVE

Right ventricle

Left ventricle

Left coronary artery

Tricuspid valve (open)

Bicuspid valve (open)

Pulmonary valve (open)

Aortic valve (closed)

Right coronary artery

ANTERIOR

POSTERIOR
Cardiac Valves

- Leaflets
  - Thin
  - Freely movable
- When the ventricle contracts
  - valve leaflets are pushed together

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Cardiac Valves

- To prevent eversion of the leaflets
  - the edges are supported by the chordae tendinae which are connected to the papillary muscles.
- Contraction of the latter during ventricular systole keeps the chordae tendinae at a constant tension.

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Cardiac Valves

- Valves sometimes become seared or scarred so that they no longer meet in the middle, or the chordae tendinae become scarred and shortened. The valves cannot fully close, producing a **Valvular Insufficiency**.

- The valves, as a result of scarring, may tend to adhere together so that they are not able to swing apart freely when blood is flowing normally through them. **Valvular Stenosis**

Valvular Regurgitation

A condition in which blood leaks in the wrong direction because one or more heart valves closes improperly. Mitral valve prolapse (illustrated left) is a common case of regurgitation.
Cardiac Valves

- The pulmonary and aortic valves
  - Small
  - cup-like arrangements, which project into the stream
  - three cusps or cups
- Because of their shape they do not need any mechanical support such as provided by the chordae tendenae for the tricuspid and mitral valves.

Cardiac Cycle

- The cardiac cycle consists of a period of relaxation called diastole
- followed by a period of contraction called systole.
- During diastole, the heart chambers filled with blood
- During systole the blood is pumped forward into the arteries.
Records of the events in the human heart

- contraction of the left atrium begins before contraction of the right atrium,
- contraction of the left ventricle begins after but is completed before, that of the right ventricle.

Atrial Pressure
An impulse from the pacemaker reaches and spreads over the atria
- (P-wave in ECG).

Atrial systole follows and pressure develops in the atrium
- (“a” wave of atrial and central venous pressure curve).

During isovolumetric contraction of the ventricle
- the A-V valves bulges out into the atria causing a second pressure rise (the “av” wave or “c” wave)
- Filling of the atrium before the A-V valves open causes the atrial pressure to rise again (“v” wave).

The pressure continues to rise → exceeds the falling pressure in the corresponding ventricle → ativoventricular valves are pushed open → each atrium and its ventricle is a common chamber.

Opening of the ativoventricular valve → a rapid fall in atrial and in the intraventricular pressure due to
- the rapid relaxation of the ventricles

As the rate of relaxation of the ventricle becomes slowed, the rate of return of blood to the heart begins to exceed the rate of relaxation of the ventricles and the pressure then rises during the latter part of diastole.
Atrial Systole

Fig. 20.14

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The Ventricular Pressure Curve

Fig. 20.14
Excitation reaches the ventricles through the A-V node

- (QRS in ECG).

With the onset of ventricular contraction (ventricular systole – VS)
- the intra-ventricular pressure begins to rise slowly at first
- then very abruptly isovolumetric or isovolumic contraction – IC reaching a peak with a rounded summit rapid ejection
- then reduced ejection – RE.

During the latter part of RE
- the pressure starts to decline and the decline continues during the succeeding periods of isovolumetric relaxation (IR) and rapid inflow (RI).

Just before the next rapid rise, marking the onset of the next heart cycle
- there is usually a slow progressive rise (diastasis – D)
- reduced inflow followed
- shallow rounded rise (atrial systole – AS).

Prior to the onset of ventricular contraction
- the atrioventricular valves are open so that the atrium and ventricles are a common chamber
- the aortic and pulmonary valves are closed.

The point at which the intra-ventricular pressure starts its abrupt rise is associated with the onset of ventricular contraction.

Very shortly after the rise in ventricular pressure begins
- the atrioventricular valve closes producing the first heart sound.

From then on, for a brief interval of time, the ventricle is not in communication with any other chamber, pressure is building up the volume does not change, as shown by the fact that the ventricular volume curve during this interval of time writes a horizontal line.

A brief moment later the
- aortic valves open so that, from the point on the aorta and the ventricle are in communication with each other.

The interval of time between the closure of the atrioventricular valves and the opening of the aortic (and pulmonary) valves
- isovolumetric contraction phase of the heart cycle (IC)
During isovolumetric contraction
- the ventricle changes from flabby and elongated chamber to tense, globular form to provide a minimum of surface for the contained volume.

During the ensuing 0.2 to 0.3 seconds
- the ventricle is ejecting blood into the aorta
- ventricular pressure rises (rapid ejection) and then falls slightly (reduced ejection-RE).

Just past the peak of the ventricular curve
- the ventricular pressure starts falling more and more rapidly
- this point is assumed to mark the onset of ventricular relaxation (diastole–VD).

A moment after the ventricle starts to relax
- the aortic (and pulmonary) valves close
  - the second heart sound.

Protodiastole
End of protodiastole
- the ventricle is again an isolated chamber (isovolumetric relaxation –IR).

Pressure within the ventricle decrease below the atrium
- atrio-ventricular valve opens
- The interval of time from the closure of the aortic valve to the opening of the atrio-ventricular valve
  - isovolumetric relaxation period (IR).

Opening of the atrio-ventricular valve the ventricular pressure
- fall because the ventricle is relaxing more rapidly than it can fill
  - this is the phase of rapid inflow (RI). After 0.1 to 0.2 seconds, the pressure again starts to rise slowly, the phase of diastasis (D) or reduced inflow until the atrial systole (AS) occurs producing the rise in ventricular pressure just before the next ventricular contraction.

The period from the onset of rapid relaxation, that is, from the onset of protodiastole to the onset of the next rapid rise in intraventricular pressure
- ventricular diastole (VD).
AORTIC PRESSURE CURVE

Fig. 20.14
Ventricular diastole

- the aortic pressure decrease gradually.
- During isovolumetric contraction
  - brief jiggle in the aortic pressure curve → transmitted oscillation because of the sudden onset of the tension in the ventricle.
- Intraventricular pressure increase
  - the aortic valve is pushed open
  - the aorta and the ventricle become a common chamber.
- For 0.1 to 0.2 seconds (rising phase of the two pressures), ventricular pressure exceeds aortic pressure as the blood is being accelerated during its departure from the ventricle.
- At the same moment during the latter part of the ventricular systole, aortic pressure begins to exceed ventricular pressure.
- This coincides approximately with the declining phases of aortic and ventricular pressure and mark the phase of accelerating ventricular ejection.
Rising aortic and ventricular pressure during the first part of ventricular ejection are probably due
- blood entering the root of the aorta faster than it can move peripheralward.

The beginning decline during ventricular systole is due to
- blood is now leaving the ventricle to go into the aorta less rapidly than it is leaving the aorta to flow out to the periphery.

During the first part of systole ejection is more rapid than during the latter part.

Onset of protodiastole
- Aortic pressure starts to fall rapidly
- Blood starts to flow back into the ventricle from the aorta → the cusps of the aortic valve to be pushed close → brief rebound of pressure; the closure marks the bottom of the incisural notch.
- The sudden rise of pressure following the incisura is a water effect.
  - When any moving fluid is stopped suddenly, the energy of motion in the fluid is converted to energy of pressure.
- The aortic pressure then declines gradually throughout diastole as blood constantly flow out of the aorta through the arterioles and into the capillaries (peripheral run-off).
- The pressure drop is due to gradual diminution in the total quantity of blood in the aorta.
- During diastole there is a back and forth movement of blood in the aorta between the central and peripheral portions.
  - standing wave or the resonant wave.
The Ventricular Volume Curve

Ventricular volume is recorded
- upward movement → increase in the volume of fluid within the ventricle.
- During the atrial systole
  - the ventricular volume increases slightly
- Ventricular volume then remains relatively flat during isovolumetric contraction but, with the opening of the aortic valve and the onset of ejection, ventricular volume decreases quite rapidly.
- The rate of decrease in the ventricular volume becomes more gradual and then ceases
- Systole can thus be divided into three phases:
  - isovolumetric contraction
  - the phase of rapid ejection
  - phase of reduced ejection
    - there is no sharp point to divide rapid and reduced ejection.
The point at which shortening of the ventricle ceases and it starts to elongate
- the end of ventricular systole
- the beginning of protodiastole.

Protodiastole
- short

With the opening of atrio-ventricular valves → the ventricular volume rapidly increases
- This rapid initial increase is due to the sudden flow into the ventricles of blood which was stored up in the atrium during the preceding ventricular systole
- the atrium being elastic and distensible has simply accumulated a considerable quantity of blood during ventricular systole.

Phase of rapid increase in ventricular volume → large quantity of blood is transmitted into the ventricle from the atrium through the elastic recoil of the atrial wall.

When all the blood previously stored in the atrium has been emptied into the ventricle, the rate of filling of the ventricle becomes slower and now approximately coincides with the rate of return of the blood to the atrium and ventricles from the systemic veins.

Ventricular volume increases more gradually along with the progressive increase in the intra-ventricular pressure; this phase is called
- diastasis suggesting a period of “standstill”.

Following the end of diastasis or reduced inflow the next atrial systole occurs completing the heart cycle.
Magnitude of Pressures

- Left ventricular pressure oscillates
  - 5 mm (diastolic) and 90 to 140 mmHg (systolic)
- Right ventricular pressure
  - -0.5 and +6 mmHg (diastolic) and between 19 and 27 mmHg (systolic).
- Stroke volume output of both ventricles is essentially the same
  - the lower average in the right ventricular pressure reflects the lower resistance in the pulmonary as compared with the systemic circuit.
- The pressure in the atrium and ventricle just at the moment of onset of ventricular systole i.e., just before closure of the atrio-ventricular valve
  - “end diastolic pressure”.
- The pressure in the right atrium oscillate with respiration
  - -7 and +6 mm Hg measured with reference to atmospheric pressure at the level of the standard zero reference plane with the person supine.
- This average left atrial pressure
  - 5 mm Hg higher than the right.
  - This is due to a lesser distensibility of both the atrium and the ventricle on the left as compared with the corresponding right chambers.

Pulmonary Capillary Pressure

- The left atrial pressure has been thought to be closely approximated by measurement of
  - “pulmonary capillary pressure”.
- Recent studies suggest → left atrial pressure is high → mitral stenosis
  - the pulmonary capillary pressure may be only approximately half the true atrial pressure.
- The normal value for pulmonary capillary pressure
  - 5 to 12 mm Hg, the average value being 6 to 9 mm Hg.
Cardiac Movements

- The apical portion of the pericardium rigidly
  - attached to the relatively fixed diaphragm
- Basilar portion of the pericardium
  - attached to the superior mediastinal blood vessels and other structures which are distensible.
- Thus, since no space may be present between the epicardium and pericardium, the apex remains in contact with the diaphragm. Whereas the base descends with each ventricular systole.
- The myocardial fibers are arranged spirally.
  - heart rotates on its vertical axis with each systole
  - apical thrust on the palpation of the chest
  - apical thrust is also thought to be due to the recoil of the apex of the heart as the blood is ejected from the ventricles.

Fig.20.01

(a) Inferior view of transverse section of thoracic cavity showing the heart in the mediastinum.

(b) Anterior view of the heart in the mediastinum.
The aortic velocity curve
- distal to the aortic valve
- slight forward movement of blood just prior to the opening of the aortic valve
  - The movement of the aortic valve as tension on it is released by the rising intra-ventricular pressure.
- A more abrupt forward movement of the blood occurs with the opening of the aortic valve and this coincides with the anacrotic notch in the ventricular pressure curve.
- The maximum velocity \( \rightarrow \) the rapid ejection
  - coinciding with the most rapid drop in the ventricular volume curve.
- Greatest difference between ventricular and aortic pressures
  - during the rising phase of the velocity curve.
- The aortic and ventricular pressures
  - are equal at the summit of the velocity curve
  - whereas during the declining phase of the velocity curve, aortic exceeds ventricular pressure as the rate of ejection decelerates.
- Backflow is recorded for a brief interval before closure of the aortic valves.
  - The “resonant waves” in the aortic pressure curve are reflected in the diastolic portion of the velocity curve.
Heart Sounds

- Normal
- VSD
- Aortic Stenosis
- Mitral Stenosis

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First Heart Sound

- The first heart sound
  - produced primarily by the snapping shut of the two atrio-ventricular valves at the beginning of the isovolumetric contraction.
  - Contributory factors may be the sudden development of tension in the ventricular muscle and the chordae tendinae.
  - Rushmer suggests that contraction of the papillary muscles contributes to the first sound by pulling the atrio-ventricular leaves together.
  - The sound caused by closure of the mitral valve is heard best
    - at the point of apical impact, usually in the fifth interspace in the midclavicular line.
  - The sound from the closure of the tricuspid valve is heard best
    - in the sixth interspace just to the right of the sternum.

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First Heart Sound

- The interval from the beginning of the rise of ventricular pressure to the beginning of the first heart sound is minimal with a normal period between atrial systole and ventricular systole.
- In ectopic beats of the ventricle or in the absence of the atrial beat
  - the interval is significantly increased.
- It is thought that with normal ventricular beats, the orifices of the atrio-ventricular valves are nearly closed prior to ventricular systole by the preceding atrial beat, the open orifices of these valves must be closed by ventricular systole.

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Fig. 20.15b

(b) Anterior view of heart valve locations and auscultation sites

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Second Heart Sound

- The second heart sound
  - closure of the aortic valve
    - heard best in the second right interspace
  - that produced by the closure of the pulmonic valve
    - second left interspace.
- A split second sound may be produced when the two semilunar valves close asynchronously.

Third Heart Sound

- The third heart sound is associated
  - rapid inrush of blood into the ventricle during the descending limb of the V wave of the jugular and atrial pressure curves.
  - result from vibration of the ventricular walls because of their sudden distension, from sudden tensing of the atrio-ventricular valves by the wave reflected from the ventricular walls, and from tensing of the pericardium.
  - 26 to 85% of normal person, depending on the sensitivity of the recorder to vibrations of the frequency.
  - “opening snap” may be present 0.04 to 0.12 seconds after the onset of the second sound.
    - It is most common in the presence of mitral stenosis.
- In some instances
  - gallop rhythm results from intensification of the third heart sound.
Fourth Heart Sound

- The fourth heart sound
  - contraction of the atrium
  - 2% of normal person
  - audible in persons having left ventricular hypertrophy associated with hypertension
  - It may be produced by rapid flow of blood through the atrio-ventricular valve or sudden distension of the ventricle or by a rebound closure of the atrio-ventricular valve following rapid inflow accompanying atrial systole.

Intensity of Heart Sounds

- The Intensity of the First Heart Sound
  - augmented with increased cardiac output or elevated aortic pressure
  - diminished in myocardial damage from any cause
  - is related to the interval between the atrial and ventricular systole (in general, the longer the interval, the louder the first sound)
  - is louder after a short diastole than after a long diastole, i.e., it occurs during the phase of rapid ventricular filling
  - is frequently intensified with tachycardia, emotional tension, hyperthyroidism, anemia, infections and fever;
  - is loud and snapping in the presence of mitral stenosis
  - is louder the more atrio-ventricular valve leaflets are separated at the onset of ventricular systole, i.e., when the interval between atrial and ventricular systole is longer than normal.
Intensity of Heart Sounds

- The intensity of the second sound is related to the pressure in the appropriate vessel of the onset of isovolumetric relaxation.
- Normally, the aortic second sound in adults is louder than the pulmonic, but in left heart failure or mitral stenosis, the reverse may be found.
- In dilatation of the root of the aorta resulting from advancing age, the aortic second sound frequently has a tympanic tone.
- The second aortic sound is decreased in aortic stenosis.
- The first and second sounds may be split or even duplicated, probably because of asynchronous onset of cessation of contraction in the two ventricles.
  - This may be seen particularly in bundle branch block or ventricular premature beats.
- The intensity of both the first and second sound is louder in thin chested persons and soften in those with thick chests.

Gallop Rhythm

- Occasionally, a third heart sound is heard which gives rise to a triple beat that resembles the hoof beats of a galloping horse.
- The third sound may be produced in an early diastole during the phase of rapid inflow into the ventricle which follows opening of the atrio-ventricular valves (early ventricular or protodiastolic gallop).
- Ventricular gallop is most frequently associated with incipient or actual congestive failure; it may be caused by a momentary rise of ventricular pressure above atrial pressure, with temporary closure of the atrio-ventricular valve.
- Ventricular gallop may made to disappear by reducing output by standing, or by occlusion by tourniquet of the veins in the extremities.
- A presystolic or atrial gallop is produced by a third heart sound which occurs during atrial systole.
Gallop Rhythm

- This sound is heard frequently in patients with hypertension, myocardial infarction, or heart block.
- If the heart rate is rapid enough, the protodiastolic and presystolic phase may practically coincide, giving a summated effect which may be more readily audible as a third heart sound and producing a mid-diastolic gallop rhythm.
- It is generally loudest at the apex; usually with the patient recumbent.
- The exact cause of systolic gallop is not known; it is not believed to indicate a grave prognosis.
- When heard loudest at the aortic area, it may be the result of impact of the aorta against other structures.

THANK YOU!

For Not Listening!