4 CHAPTER Cardiac cycle and Cardiac output

CARDIAC CYCLE

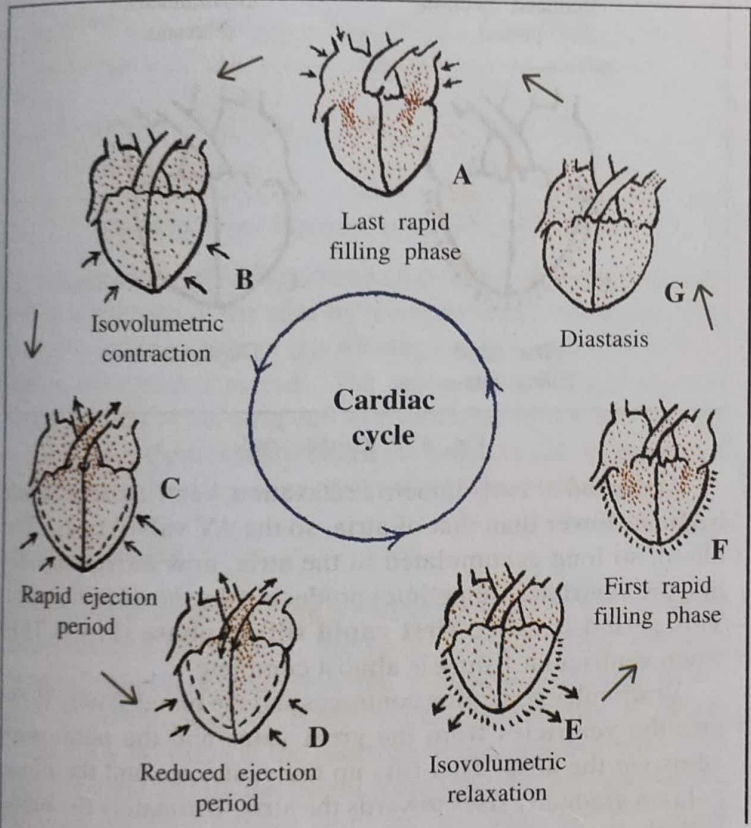
The sequence of events which occurs in the heart during a beat is repeated in each beat in the same order. This cyclical operation of the heart is called **cardiac cycle.** The cycle is completed once in each beat and the time required is called **cardiac cycle time**. The cycle time depends on the rate at which the heart is beating. When the heart beats faster, the cycle time is reduced and is prolonged in a slowly beating heart. When the rate is 75/min the cycle time is 0.8 sec, *i.e.*, one beat takes 0.8 sec to be completed.

Both the atria and the ventricles operate cyclically and for that reason the cardiac cycle is considered as **atrial cycle** and **ventricular cycle.** In each chamber, the cycle goes on as follows :

Systole ⟶ diastole ⟶ systole ⟶ diastole .....
(Electrical events also occur simultaneously as described in ECG)

In systole, the chambers contract to eject the blood from them, whereas, in diastole the chambers relax and blood collects in them. The diastole in the atria and the ventricles overlap, *i.e.*, both of them remain relaxed simultaneously (for some time, Fig. 6.74C). On the other hand, in a normal situation the systoles never coincide, *i.e.*, the atria and the ventricles never contract together (see nodal delay).

For a detailed study of cardiac cycle, the cycle of each chamber is subdivided as follows :



CARDIAC CYCLE

Atrial cycle (0.8 sec)		Ventricular cycle (0.8 sec)	
Atrial systole (0.1sec)	*Atrial diastole* (0.7sec)	*Ventricular systole* (0.3sec)	*Ventricular diastole* (0.5sec)
		(1) Isovolumetric contraction (0.05 sec).	(4) Protodiastole (0.04 sec)
		(2) Rapid ejection period (0.11 sec)	(5) Isovolumetric relaxation (0.08 sec).
		(3) Reduced ejection period (0.14 sec)	(6) First rapid filling phase (0.113sec).
			(7) Diastasis (0.167 sec)
			(8) Last rapid filling phase (0.1 sec).

A

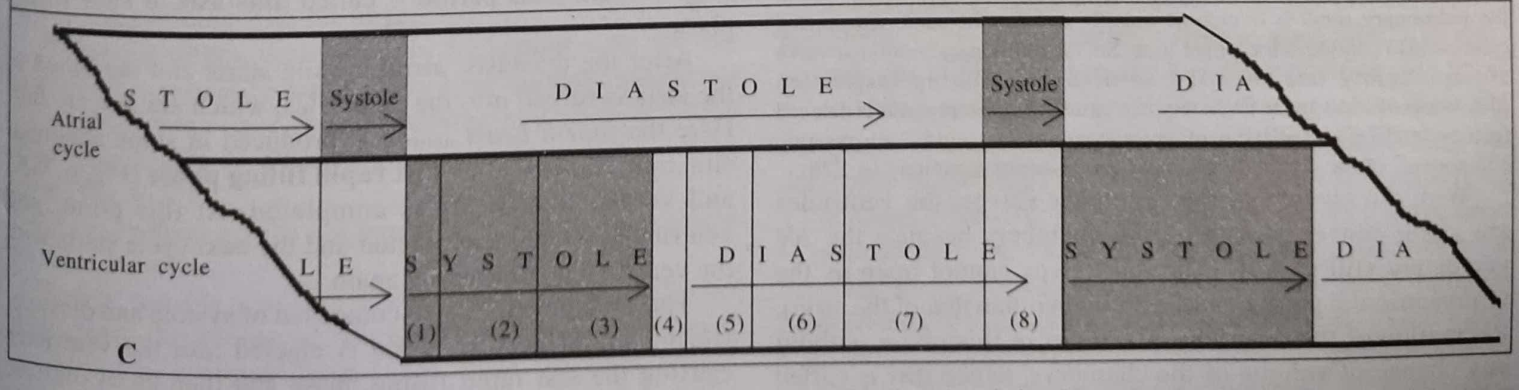


Fig. 6.74. *Events of the cardiac cycle, their timings and duration.*

Note : Numbers in the parenthesis in fig. 6.74C are same an in Fig. 6.74A and represent the same events

Description of the Cycle

(The mechanical events)

The cardiac impulse originates in the SA node, so the chambers to be activated first are the atria and these also contract first (Fig. 6.75A). After the **atrial systole** is over, ventricular contraction starts and intraventricular pressure rises. When the intra-ventricular pressure becomes more than in the atria, the AV valves close with the production of *first heart sound.* The papillary muscles also contract simultaneously to keep the valve cusps in position.

Now the ventricles are converted into closed chambers as the AV valves are closed and the semilunar valves yet to open. So, the blood from the ventricles cannot leave and the ventricles go on contracting at this stage without any change in the volume (as blood is not compressible). That is why it is named as the phase of **isovolumetric contraction** (Fig. 6.75B). (This is not a typical isometric contraction as some of the muscle fibres shorten). Intraventricular pressure rises rapidly in this phase (Fig. 6.76).

When the pressure inside the left ventricle becomes more than that of the aorta and of the right ventricle more than the pulmonary trunk, the respective semilunar valves open. Blood now goes out of the ventricles rapidly and the ventricular pressures continue to rise. This phase is named as **rapid ejection period** (Fig. 6.75C).

Later on, as some blood has moved out, the rate of ejection becomes slower, so it is named as **reduced ejection period** (Fig. 6.75D). In this phase, due to rapid reduction of the amount of blood in the ventricles, intraventricular pressure decreases but is always higher than in the great vessels.

Towards the end of the ventricular systole, inspite of continued contraction, intraventricular pressures fall further; the left ventricular pressure goes below that of aorta and that of right ventricle below the pulmonary trunk, but the blood flow continues for some time due to inertia of motion. This phase is called **protodiastole,** though it is a part of systole (Some authors believe that ventricles relax in this phase and include this in diastole, hence the name).

After the protodiastolic period, when the inertia of motion is overcome, blood wants to roll back towards the ventricles. This causes closure of the semilunar valves with production of the *second heart sound.* It marks the end of the clinical systole and ventricular diastole starts.

Note : Right ventricular ejection starts earlier because the low pressure in the pulmonary trunk is overcome quickly and for the same reason, the ejection also continues for longer time. So, the pulmonary semilunar valve closes slightly late than the aortic valve. During inspiration (due to more inflow to the RV, it requires more time to empty) this is delayed further leading to **splitting** of the second heart sound, *i.e.*, the two components of the second heart sound can be heard separately (p. 226).

With the closure of the semilunar valves, the ventricles are again converted into closed chambers because the AV valves are still closed. (The AV valves cannot open as the intraventricular pressures are still higher than that of the atria). Relaxation of the ventricles continues in this phase without any change of volume of the chambers, hence this is called **isovolumetric relaxation** (Fig. 6.75E). Intra-ventricular pressures fall rapidly in this phase.

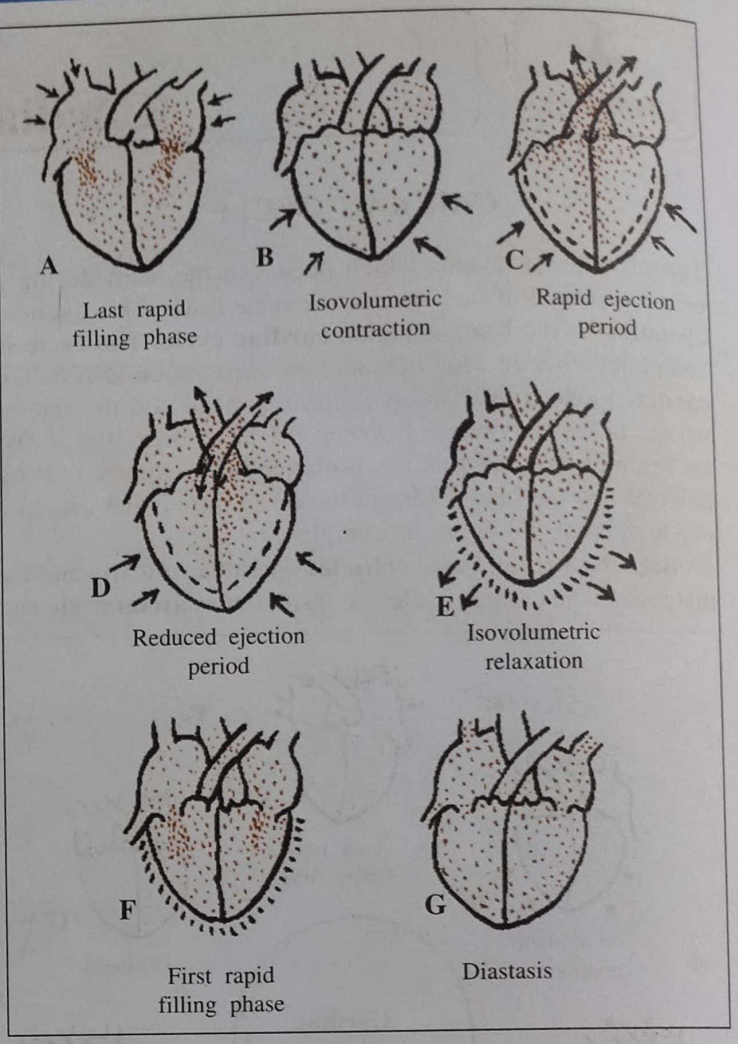


Fig. 6.75. *Cardiac cycle.*

At the end of isovolumetric relaxation, ventricular pressure becomes lower than that of atria, so the AV valves open. The blood, so long accumulated in the atria, now moves rapidly into the ventricles, sometimes producing the *third heart sound.* This period is called **first rapid filling phase** (Fig. 6.75F) when ventricular filling is almost complete.

Ventricular relaxation continues and the blood slowly flows into the ventricles from the great veins and the pulmonary veins *via* the atria. This fills up the ventricles and the blood column gradually rises towards the atria. Ultimately the blood in both the chambers becomes continuous as if a single cavity (Fig. 6.75G). This period is called **diastasis** or slow filling phase.

After the diastasis, atrial systole starts and the blood in the atria is forced into the ventricles, which are already full. Here the *fourth heart sound* is produced in some abnormal situation. This is called **last rapid filling phase** (Fig. 6.75A) and ventricular filling is completed. At this point, one ventricular cycle is completed and the next cycle starts with the ventricular contraction again.

The **atrial cycle** is also composed of systole and diastole. During **atrial systole** blood is ejected into the ventricles causing the last rapid filling phase and then atrial diastole starts. During **atrial diastole,** blood from the veins flows into the atria. In the later part of the atrial diastole, which coincides

situation. Therefore, this is not paradoxical but accentuation of the normal physiological findings.

Pulsus paradoxus is found distinctly in pericardial effusion, constrictive pericarditis, cardiac temponade, etc., when the heart cannot expand properly.

Pulsus alternans : It is the type which shows alternate strong and weak pulse (Fig. 6.83D). It is due to alteration of the stroke volume in successive beats in case of grave cardiac diseases like severe myocardial infarction, too much dilatation of the heart etc., *i.e.*, in conditions of cardiac failure.

HEART SOUNDS

These are the sounds normally produced within the heart during the cardiac cycle. It is not properly understood how the heart sounds are actually produced. The main cause seems to be either acceleration or deceleration of blood flow. Other causes are discussed with the individual types. Altogether there are four sounds produced in the heart, named as first, second, third and fourth heart sounds.

Of them, the 1st and the 2nd sound can be heard nicely by placing stethoscope on the chest but all four can be recorded by phonocardiograph (Fig. 6.76 and 6.84).

The third heart sound, sometimes may be heard in normal individual.

The fourth sound is never heard in normal heart. In some diseased states all the four sounds may be heard.

First heart sound

It is heard at the onset of the ventricular systole during closure of the AV valves. It almost coincides with the carotid pulse. Its duration is longer (0.15 sec) and its frequency is lower (25 to 45 Hz) than that of the second heart sound. It sounds like the word 'Lub'.

Causes of the first heart sound : The first heart sound is probably caused by three reasons : vascular, muscular and valvular.

(i) Vascular cause is due to turbulence created in the blood due to ventricular contraction on it.

(ii) Muscular cause is due to vibration of the walls of the contracting ventricles.

(iii) Valvular cause is due to vibration of the valve cusps during closure.

The first heart sound has two components : mitral (M_1) and tricuspid (T_1). The mitral component (M_1) is best heard in the mitral area, *i.e.*, in the left fifth intercostal space, 1 cm inside the midclavicular line *i.e.*, at the site of the apex beat. The tricuspid component (T_1) is best heard in the tricuspid area, *i.e.*, on the left side of the lower end of the sternum. Splitting of the first sound is rare.

Significance : A normal first heart sound signifies the start of systole, normal AV valve closure and normal ventricular contraction. The first heart sound is accentuated (intensified) during exercise due to associated tachycardia and increased contractility.

Abnormal accentuation is found in ventricular hypertrophy, hyperdynamic circulation and in mitral stenosis. First heart sound is soft in heart failure and is not heard if there is a concomitant murmur (see below).

Second heart sound

It is heard at the end of clinical systole during closure of the semilunar valves (the period between the first and second heart sound is the clinical systole). It is of shorter duration (0.12 sec) and is of higher frequency (50 Hz) than the first heart sound. It sounds like the word 'Dupp'.

Causes of the second heart sound : Like the first sound it has also three reasons : vascular, muscular and valvular.

(i) Vascular cause is due to oscillation/vibration of the blood column in the aorta and in the pulmonary trunk.

(ii) Muscular cause here means vibration of the walls of the pulmonary trunk and ascending aorta.

(iii) Valvular cause is due to closure of the semilunar valves.

The second heart sound has two components : aortic (A_2) and pulmonary (P_2). The aortic component is best heard in the aortic area, *i.e.*, in the right second intercostal space near the sternum and the pulmonary component is best heard in the pulmonary area, *i.e.*, the left second intercostal space near the sternum. The pulmonary and the aortic components of the second sound may be separated—a phenomenon called **splitting of heart sounds**.

This splitting can be distinctly heard during a deep inspiration which leads to a more delayed closure of the pulmonary semilunar valves. This is due to prolonged ejection by the right ventricle, as the pulmonary vascular resistance is low and increase of venous return to the right side of the heart during inspiration. Reverse splitting on expiration can also occur when the left ventricle takes more time to empty as in left ventricular failure and left bundle branch block. Normally the aortic component (A_2) is louder than the pulmonary component. In pulmonary hypertension P_2 becomes louder (accentuated) and A_2 is accentuated in systemic hypertension (high blood pressure). Second sound cannot be heard when there is a concomitant murmur.

Significance : A normal second heart sound signifies the end of clinical systole, normal semilunar valves which are closing normally.

Third heart sound

It is a low pitched soft sound of 0.1 sec duration, heard in the early diastole and is produced due to the first rapid filling phase, *i.e.*, after the second sound. It occurs probably due to vibration in the ventricular wall caused by the movement of blood. It can be heard sometimes in normal children and young individuals with thin chest and high venous return. Third sound in abnormal situation signifies heart failure, etc. It is heard best at the apical area.

Fourth heart sound

It is also called atrial sound and is produced during atrial contraction. It is heard just before the first sound, *i.e.*, late in the diastole. It is believed to be produced when the atria a.e

forcefully trying to pump blood in to noncompliant (stiff) ventricles (as in ventricular hypertrophy). It is not heard in normal individual but can be recorded by phonocardiograph. In abnormal situations like heart failure, after myocardial infarction, etc., the 4th sound is frequently heard.

Prosthetic sound

It is the sound produced due to normal closure and opening of a prosthetic (artificial) valve implanted in the heart.

'GALLOP' RHYTHM

Sometimes the sounds produced in the heart are heard like the hoop beats of a galloping horse. It is then called gallop rhythm. 'Gallop' is produced in presence of a third or fourth sound during tachycardia when the individual sounds cannot be identified separately. If it is due to 3rd heart sound, then the gallop is called diastolic gallop and due to fourth sound presystolic gallop is produced.

MURMUR AND BRUITS

Murmurs are the sounds produced within the heart, other than the heart sounds. These are produced due to turbulent blood flow and can be heard with stethoscope. This turbulence may be caused by abnormal flow with respect to speed and direction due to various reasons like narrowing of a valve or regurgitation through a valve, anaemia etc. Murmurs produced during diastole are named as diastolic murmur and those produced during systole are named as systolic murmurs. There are various sub types of each of them (Fig. 6.84). Bruits are sounds produced due to turbulence in a blood vessels but outside the heart. '**Thrills**' are palpable vibrations produced by murmurs.

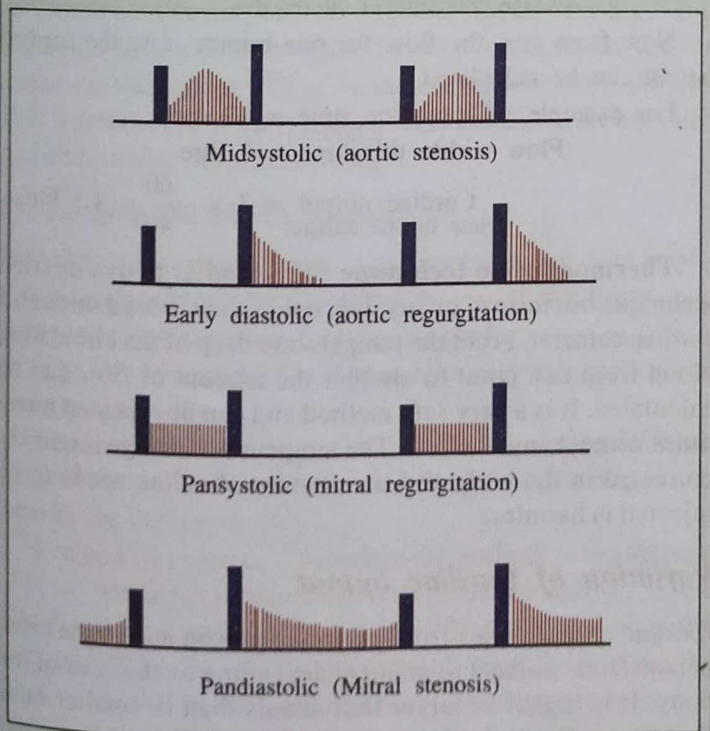


Fig. 6.84. *Cardiac murmurs—one example is given in each case but may be produced due to various reasons.*

CARDIAC OUTPUT

The main function of the heart is to pump blood to maintain circulation in the body. The right ventricle is responsible for circulation through the pulmonary circuit and the left ventricle supplies the rest of the body. This circulation of blood is essential for life. Therefore, the amount of blood ejected by the heart should be adequate, otherwise the tissues will suffer and here lies the importance of the cardiac output.

Definition : Amount of blood ejected by each ventricle per minute is called cardiac output (CO) It is expressed in litre per minute. Its value is almost same for both the ventricles and is about 5 litre/minute in a normal adult male.

[Amount of blood ejected per ventricle per beat/stroke is called **stroke volume.** It may be slightly different in the two ventricles (sometimes) and also in the same ventricle in different beats, hence it is not a good index of cardiac performance and one minute's value is taken.]

The amount of blood ejected in one minute, *i.e.*, CO will be equal to the number of beats per minute (heart rate) times the amount ejected per beat (stroke volume),

or, cardiac output = heart rate × stroke volume

or, CO = 72/min × 70 ml = 5 L/min (approx).

(assuming heart rate 72/min and stroke volume 70 ml)

Cardiac index (CI) : It is another way of expressing cardiac output. Cardiac index is the cardiac output in litres per minute per square metre of body surface area (SA). So, in an individual whose CO is 5 L/min and SA is 1.51 M^2,

$$\text{the cardiac index} = \frac{CO}{SA} = \frac{5 \text{ L/min}}{1.51 \text{ sqm}} = 3.32 \text{ L/min/M}^2.$$

Cardiac index is a better indicator of circulation than the cardiac output. It gives the idea about perfusion per unit area and is same in different individuals of different sizes, whereas the cardiac output will vary.

Distribution of cardiac oupput

The blood pumped by the heart is distributed throughout the body and the respective shares of each part at rest are approximately as follows :

Heart 5%; bronchial vessels 5%; gut, liver, pancreas and spleen 30%; kidneys 20%; skeletal muscles 15%; skin and bones 10%; brain 15%.

The share of blood to a part is not always constant and may change frequently according to the activity of the part concerned. A more active part receives more share of CO and flow through the other parts is then proportionally decreased.

Measurement of Cardiac output

Cardiac output is frequently measured to know whether the heart is working normally or not. It is estimated at rest as well as during a stress, *e.g.*, physical exercise.

Different methods are as follows :

(i) Ballistocardiography
(ii) Electromagnetic flowmeter
(iii) Dye dilution technique
(iv) Thermodilution technique
(v) Fick's principle

Estimation of Cardiac output by Fick s principle

Fick's Principle : It states that, *the amount of a substance taken up from blood is equal to the arterio-venous difference of the substance times the blood flow*. It is true for an organ or any part or whole of the body and can be employed to measure the blood flow through that part.