The Heart and its Function

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### Table

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![Diagram showing heart phases and pressure graph]

**FIGURE 12.16** Ventricular pressure during the cardiac cycle.
The Cardiac Cycle

• The human cardiac cycle lasts about 0.9 sec, for 67 beats/min

• Ventricular Filling
  • Duration 0.5 sec
  • Inlet valves (tricuspid, mitral): open
  • Outlet valves (pulmonary & aortic semilunar): closed

• Diastole lasts for nearly 2/3 of the cardiac cycle
Diastole

- Rapid-filling phase – fast over the initial 0.15 sec.
  - Filling pressure falls initially as contracted ventricle recoils from its systolic contraction
- At “natural relaxed volume” filling rate slows (diastasis).
- Final third of filling phase atrial contraction pumps extra blood into ventricle
  - Atrial systole boosts filling only 10-20% in young adults
  - Atrial systole boosts filling by 46% at 80 years old.
End-Diastolic Volume (EDV)

- At the end of the filling phase the EDV is \(~120\) ml in an adult human
  - End diastolic pressure (EDP) is 4 (R) - 9 (L) mm Hg.
**Diastole**

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**Pressure (mm Hg)**

- Start of atrial contraction
- Start of ventricular contraction
- **Diastole**

**Figure 12.16** Ventricular pressure during the cardiac cycle.
Isovolumetric contraction

- Duration: 0.05 sec
- **Inlet and outlet valves**: closed
- Atrioventricular flaps close as $P_V > P_A$
- Aortic & Pulmonary semilunar valves open when $P_V > P_{aorta}$ or $P_{pulmonary artery}$
Ejection

- Duration: 0.3 sec
- Inlet valves: closed
- Outlet valves: open
- Phase of rapid ejection: 0.15 sec
  - As ejection slows, outflow slows.
  - When outflow pressure > ventricular pressure, backflow begins and closes the semilunar valves – with < 5% of ejected volume leaking back.
- As semilunar valves close, a “notch” is noticed in the arterial pressure trace
**Figure 12.17** Aortic pressure during the cardiac cycle. An aortic pressure wave is shown along with periods of opening and closing of the aortic valve.
Isovolumetric Relaxation

- Duration: 0.8 sec.
- Inlet and outlet valves: closed
- Ventricular pressure falls rapidly due to elastic recoil of the deformed myocardium
- When $P_{\text{atrial}} > P_{\text{ventricular}}$, atrioventricular valves open and blood flows into the ventricles from the atria, which have been refilling during ventricular systole.
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**FIGURE 12.16** Ventricular pressure during the cardiac cycle.
Ventricular Pressure-Volume Loop

Figure 2.6 Pressure–volume cycle of human left ventricle.
Valve abnormalities - murmurs

- Aortic stenosis – narrowing of valve opening – high pressure gradient increases systolic pressure and increases ventricular work.
  - Ejection murmur – (peaks mid-systole)
- Mitral or tricuspid incompetence – during systole, blood leaks back into the atria
  - Pansystolic murmur (throughout systole)
- Aortic incompetence
  - Decrescendo murmur – (early diastolic)
The Frank-Starling law of the heart

• In 1895, Otto Frank, a German physiologist ligated a frog aorta and varied the diastolic fluid volume – measuring the systolic pressure.
  • When ventricle was stretched by increased diastolic fluid volume, systolic pressure generation increased.
  • Frog hearts have a myogenic pacemaker (like human) – however, being cold-blooded (poikilothermic), they have lower cardiac tissue O₂ requirements and hearts remain beating rhythmically at room temp.
Figure 6.7 Effect of diastolic volume on energy of contraction, as measured by systolic pressure in an isovolumetric frog ventricle (aorta ligated). (a) Active pressure generated between diastole (open circles) and systole (closed circles) increases as ventricular volume is raised from 1 to 4 (arbitrary units). (b) Effect of volume plotted out. Bottom curve shows passive pressure-volume relation; note the increasing stiffness (upswing) as the ventricle is distended. Top curve shows systolic pressure as a function of diastolic volume. Red curve shows pressure generated actively, i.e. systolic pressure minus diastolic pressure. (After Otto Frank’s seminal experiment of 1895.)
Starling’s Law of the Heart

- Using a similar experimental preparation in a dog in 1914, Ernest Starling established that the greater the stretch of the ventricle in diastole, the greater the stroke work achieved in systole.

- “The energy of contraction of a cardiac muscle fiber, like that of a skeletal muscle fiber, is proportional to the initial fiber length at rest” – Starling’s Law of the Heart
Stroke Work $(W) = \Delta P \times \Delta V$

- (change in Ventricular Pressure) $\times$ (change in Stroke Volume)
- Stroke work is the area inside the ventricular pressure – volume loop

![Figure 2.6 Pressure–volume cycle of human left ventricle.](image)
Control of stroke volume and cardiac output

Figure 6.11 (a) Pressure-volume cycle of the human left ventricle. A, opening of mitral valve; AB, filling phase; B, closure of mitral valve at onset of systole; BC, isovolumetric contraction; C, opening of aortic valve; CD, ejection phase; D, closure of aortic valve; DA, isovolumetric relaxation. Since systolic pressure reaches 140 mmHg here, this subject is probably middle-aged. The mechanical work performed equals the sum of all the $\Delta P \cdot dV$ strips within the loop, i.e. total loop area. The sketches indicate how the isovolumetric phase produces no external work despite large energy expenditure by the myocardial manikin. (b) Factors influencing the pressure-volume cycle. The lower border is set by the passive pressure-volume curve of the relaxed ventricle. The upper boundary is set by the systolic pressure that would be produced in a purely isovolumetric contraction from a given end-diastolic volume; this line represents the Frank-Starling mechanism. Loop 1 represents a control state. Loop 2 shows the effect of increasing the end-diastolic volume: stroke volume increases, provided arterial pressure is held steady. If arterial pressure is then raised (loop 3), stroke volume decreases, provided end-diastolic volume is held steady. Line 4 depicts a purely isovolumetric contraction.

1 = Normal left ventricular cycle
2 = The effect of increasing LEDV – possibly by lying down - Increased stroke volume.
3 = Negative effect of raising the arterial pressure – more energy is consumed raising the ventricular pressure so less remains for ejection. Stroke volume falls.
Determinants of CVP

- Central venous pressure (CVP) depends on total volume of blood in the circulation
- How the volume is distributed between the peripheral and central veins
- Venous volume distribution is affected by:
  - Gravity, peripheral venous tone, the skeletal muscle pump and breathing.
Low Blood Volume Reduces Filling Pressure

• About 2/3 of the entire blood volume is in the venous system.
• A fall in blood volume due to hemorrhage or dehydration will reduce CVP and result in a fall in stroke volume.
• Conversely, a blood transfusion raises CVP and increases stroke volume.
• Standing reduces CVP (venous pooling in legs).
• Sympathetic output constricts peripheral veins and shifts blood into the central veins increasing CVP.
• However, increasing CO with exercise puts more blood into the arteries and reduces CVP — ↓preload, ↑afterload.
• Venoconstriction during exercise increases CVP.
• Venodilation of the skin occurs in hot environments decreases CVP.
Transmural Pressure

- Ventricular filling is affected not only by the internal filling pressure but also by the external pressure around the heart.
- The true filling pressure is the difference between the internal and external pressures or Transmural Pressure.
- The external pressure is -5 to -10 cm H$_2$O
- This negative intrathoracic pressure increases venous return, but with each lung expansion, more blood is in the pulmonary vascular pool which temporarily decreases venous return.
Functions of the Frank-Starling mechanism

• Balance the outputs of the right and left ventricles
• Increase stroke volume in exercise
• Mediates postural hypotension
• Mediates hypovolemic hypotension
Figure 6.13 Guyton's analysis of the circulation. The cardiac output–CVP relation follows Starling's law of the heart, at constant heart rate. The venous return curve shows blood flow from the peripheral vasculature into the central veins (see text). MCP is mean circulatory pressure at zero flow. The normal operating point is where the two lines cross, i.e. output equals return (open circle). The output curve is shifted downwards in heart failure by a fall in contractility (short dashes). The venous return curve is shifted upwards if MCP is increased by vasoconstriction or increased plasma volume (long dashes); or downwards if MCP is reduced by hypovolaemia (not shown). (After Guyton, A. C., Jones, C. E. and Coleman, T. G. (1973) Circulatory Physiology: Cardiac Output and its Regulation, W. B. Saunders, Philadelphia.)
Heart Failure

- Guyton’s cross-plot provides insights into pathophysiological states
  - Cardiac failure: Cardiac Output curve is depressed by a reduction in contractility
  - Concomitant rise in mean circulatory pressure (MCP) due to venoconstriction and fluid retention increases venous return and cardiac output (see next figure).
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Laplace’s Law and Swollen Hearts

• For a hollow sphere (similar to the ventricle), the internal pressure, \( P \) is proportional to the wall tension, \( T \) and inversely proportional to the internal radius, \( r \).

\[ P = \frac{2T}{r} \]

• Increased wall tension will aid ejection early in systole
• As ejection proceeds, the radius decreases, also facilitating ejection
Law of Laplace: \[ P = \frac{2T}{r} \]

- \( T \) = wall tension
- \( P \) = internal pressure
**Figure 6.15** Pump function curves for a normal heart, failing heart and laboratory roller pump. W, normal working pressure. Raising the outflow pressure depresses stroke volume (point 1) if end-diastolic volume is held constant. Otherwise, ventricular distension restores stroke volume by shifting the pump function curve to a higher energy level (point 2) (Frank–Starling mechanism, see Figure 6.16). Impaired contractility (heart failure) shifts curve to a lower energy level (point 3), but the stroke volume can be improved by pressure-reducing drugs (point 4). (Adapted from work of Elzinga, G. and Westerhof, N. (1979) *Circulation Research*, 32, 178–186, and Nichols, W. W. and O’Rourke, M. F. (1998); see Further Reading, Chapter 8.)
Figure 6.20 Schematic pressure–volume loops for human left ventricle when myocardial contractility is increased. The upper dashed confine is the relation between systolic pressure and end-diastolic volume for a purely isovolumetric contraction (Frank–Starling mechanism). (a) Loop 1 represents a basal state. Loop 2 represents a state of increased contractility. Ejection fraction is increased, so end-diastolic volume falls unless actively regulated. Loop area (stroke work) is increased. (b) During exercise (loop 3), contractility is raised by sympathetic activity and end-diastolic volume is raised by peripheral circulatory adjustments (venoconstriction, muscle pump). The increase in stroke volume is now much greater.