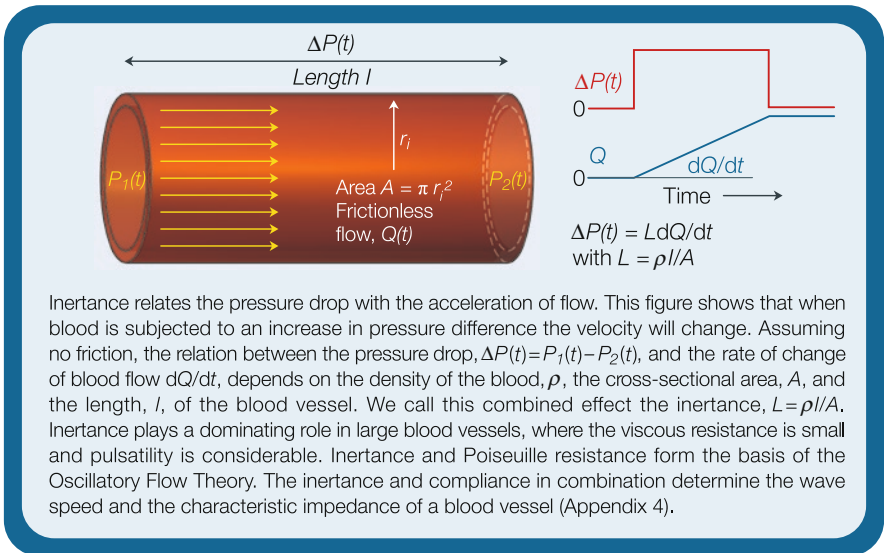


Chapter 7

Inertance



Description

Blood is accelerated and decelerated with every heartbeat, and the mass of the blood plays a role. The mass is density times volume, and the volume depends on the geometry of the blood vessel or heart. Blood density is a material property and is about 1.06 g/cm³. In hemodynamics, we calculate the effective mass and call it inertance. Inertance connects the oscillatory pressure drop with the rate of change of blood flow.

We can derive inertance by using Newton’s law relating force, F , mass, m , and the rate of change of velocity, dv/dt , which is the acceleration, a :

$$F = m \cdot a = m \cdot dv/dt$$

For a vessel, the net force $F(t) = (P_1(t) - P_2(t))A = \Delta P A$, A being the luminal cross-sectional area. The mass in the segment equals blood density, ρ , times the volume (length times area): $\rho(lA)$. The acceleration is the rate of change of velocity with time, i.e., dv/dt . In terms of volume flow this is $(1/A)dQ/dt$. With Newton's equation this gives:

$$\Delta P(t) \cdot A = \rho \cdot l \cdot A \cdot (1/A) \cdot dQ/dt = \rho \cdot l \cdot$$

and

$$\Delta P(t) = \rho \cdot l / A (dQ/dt) = L \cdot dQ/dt$$

where $L = \rho \cdot l / A$ is called inertance. We recall that resistance is inversely proportional to r_i^4 (Chap. 2) while inertance is inversely related to r_i^2 . Thus, in large vessels the inertance plays a larger role than resistance while in very small arteries and arterioles it is the resistance that plays the larger role.

The inertance in combination with the compliance of a vessel segment determines the characteristic impedance and the wave speed (see Chap. 20 and Appendix 3).

Addition of Series and Parallel Inertances

The principal rules for addition of inertance of vessels in parallel and in series are as for resistances (see Chap. 6).

Physiological and Clinical Relevance

The inertance is determined by the cross-sectional area and length of the blood vessel, and by blood density. Blood density varies little, even in pathologic conditions. Inertance is therefore primarily a geometrically determined parameter.

An example where the effect of the inertance can be seen is when left ventricular and aortic pressures are measured simultaneously (Fig. 7.1). During the ejection period aortic flow is first accelerating (early ejection) and then decelerating. When the blood is accelerated the left ventricular pressure is higher than aortic pressure. When the blood is decelerated the pressure difference reverses, as in the later phase of ejection. It may be seen that the pressure difference and the time derivative of flow are almost proportional in systole, suggesting inertance effects.

Inertance in combination with reflections (see Chap. 21) can result in flow reversal, i.e., negative flow during part of the cardiac cycle (Fig. 7.2). This negative flow is therefore physiologic. The mean flow is, of course, always in the direction of the periphery.

Another example (see Fig. 7.3) is the diastolic filling of the ventricle through the mitral valve. As a result of the inertance, flow persists when left ventricular pressure is higher than left atrial pressure.

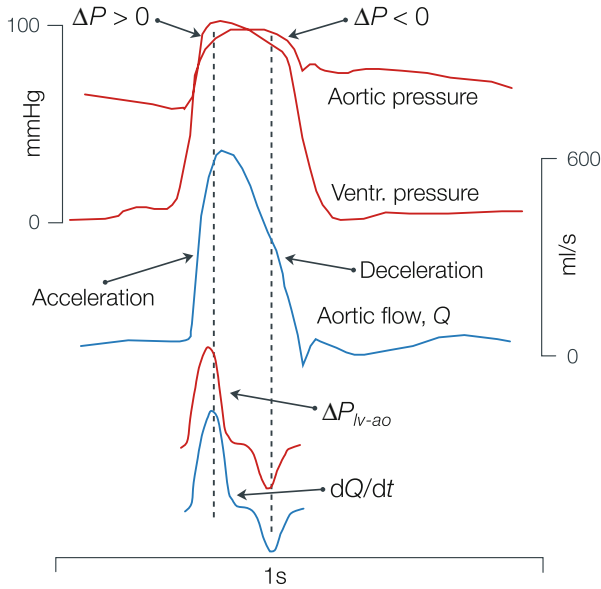


Fig. 7.1 Inertance plays a role in accelerating and decelerating the blood. In early systole, when left ventricular pressure is higher than aortic pressure the blood accelerates, i.e., flow increases. In late systole, aortic pressure is higher than ventricular pressure the blood still flows forward but the velocity decreases (deceleration). Adapted from ref. [1], used by permission

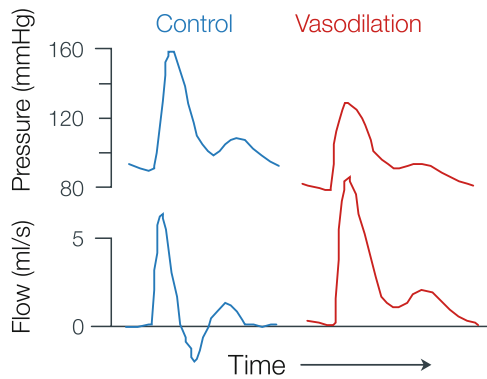


Fig. 7.2 Blood flow may be reversed, or negative, during part of the cardiac cycle. This results from inertia and reflections. With vasodilation the reflections decrease and flow reversal disappears (example from femoral artery). Adapted from ref. [2], used by permission

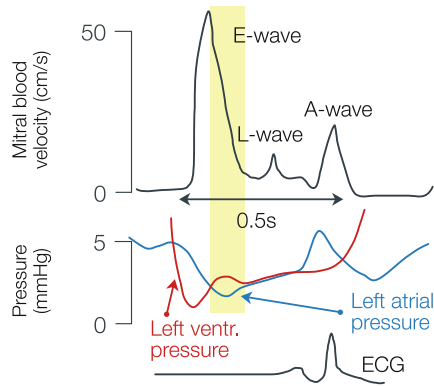


Fig. 7.3 In diastolic filling of the ventricle inertia plays a role. Flow is still forward, but decelerates, while the pressure difference between atrium and ventricle reverses. Adapted from ref. [3], used by permission

References

1. Noble MIM. The contribution of blood momentum to left ventricular ejection in the dog. *Circ Res* 1968;23:663–670.
2. O'Rourke MF, Taylor MG. Vascular impedance of the femoral bed. *Circ Res* 1966;18:126–139.
3. Solomon SB, Nikolic SD, Glantz SA, Yellin EL. Left ventricular diastolic function of remodeled myocardium in dogs with pacing induced heart failure. *Am J Physiol* 1998;274:H945–H954.