2 The Heart

2.1 Introduction

The heart is the prime mover of blood. By periodic stimulation of its muscles it contracts periodically and pumps blood throughout the body. How the pump works is the subject of this chapter.

In each cycle the left and right ventricles are first filled with blood from the left and right atria, respectively, in the diastolic phase of the cycle. Then by the deceleration of the blood stream a pressure field is generated, which closes the valves between the atria and the ventricles. The contraction of the heart muscle begins and the pressures in the ventricles rise. When the pressure in the left ventricle exceeds that in the aorta, and the pressure in the right ventricle exceeds that in the pulmonary artery, the aortic valve in the left ventricle and the pulmonary valve in the right ventricle are pushed open, and blood is ejected into the aorta and the lung. This is the systolic phase. The ejection continues until the deceleration of the jets of blood creates pressure fields to close the valves. Then the muscle relaxes, the pressures decrease, and the diastolic phase begins.

Thus, in the left ventricle, the blood pressure fluctuates from a low of nearly zero (i.e., atmospheric) to a high of 120 mm Hg or so. But in the aorta, the pressure fluctuation is much less. How does the aorta do it? How is the large fluctuation of blood pressure in the heart converted to the pressure wave in the aorta, with a high mean value and a smaller fluctuation? The answer was given by Stephen Hales (1733), who credited the feat to the elasticity of the aorta. An analogy was drawn between the heart-and-artery system and the old-fashioned hand-pumped fire engine. In the case of the fire engine, the fireman pumps water into a high-pressure air chamber by periodic injections at a higher pressure. Water is then drained from the air chamber, which has a high mean pressure that drives water out in a steady jet. This analogy was used by Otto Frank (1899) in his theory of the cardiovascular system, and is known as the Windkessel (German for air vessel) theory. In this theory, the aorta is represented by an elastic chamber and the peripheral blood vessels are replaced by a rigid tube of constant resist-
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Inflow Elastical chamber Peripheral vessel

**Figure 2.1:1.** The Windkessel model of the aorta and peripheral circulation.

Let $\dot{Q}$ be the inflow (cm$^3$/s) into this system from the left ventricle. Part of this inflow is sent to the peripheral vessels and part of it is used to distend the elastic chamber. If $p$ is the blood pressure in the elastic chamber (aorta), then the flow in the peripheral vessel is assumed to be equal to $p/R$, where $R$ is a constant called peripheral resistance. For the elastic chamber, its change of volume is assumed to be proportional to the pressure. The rate of change of volume of the elastic chamber with respect to time, $t$, is therefore proportional to $dp/dt$. Let the constant of proportionality be written as $K$. Then, on equating the inflow to the sum of the rate of change of volume of the elastic chamber and the outflow $p/R$, the differential equation governing the pressure $p$ is

$$\dot{Q} = K \frac{dp}{dt} + \frac{p}{R}. \quad (1)$$

The solution of this differential equation is

$$p(t) = \frac{1}{K} e^{-t/(RK)} \int_0^t \dot{Q}(\tau) e^{-\tau/(RK)} d\tau + p_0 e^{-t/(RK)}, \quad (2)$$

where $p_0$ is the value of $p$ at time $t = 0$. This gives the pressure in the aorta as a function of the left ventricle ejection history $Q(t)$. Equation (2) works remarkably well in correlating experimental data on the total blood flow $\dot{Q}$ with the blood pressure $p$, particularly during diastole (McDonald 1974, pp. 11, 310, 423; and Wetterer and Kenner, 1968). Hence, in spite of the severity of the underlying assumptions, it is quite useful.

Turning now to the question of stress in the heart itself, there is another very simple analysis that is quite good. Assume that the left ventricle can be approximated by a thick-walled hemispherical shell (Fig. 2.1:2), with