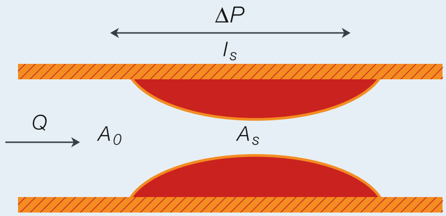
Chapter 5
Arterial Stenosis



$$\Delta P = a_1 \cdot Q + a_2 \cdot Q^2$$

Aortic coarctation and arterial stenosis is a localized narrowing in the arterial lumen, typically as a result of atherosclerosis. A stenosis is quantified by the ratio A_s/A_0, called the area ratio, often expressed as % area occlusion, given as $(1 - A_s/A_0) \cdot 100$. The relation between pressure drop across the stenosis, ΔP, and flow, Q, is quadratic, which means that stenotic resistance increases with flow. The linear term in the pressure drop-flow equation accounts for the viscous losses within the stenosis, whereas the quadratic term accounts for losses due to turbulence. In severe stenoses (area occlusion more than 85%), turbulent losses dominate. Severe stenoses add significant resistance to flow and can be potentially harmful by preventing adequate blood supply to distal beds.

Description

Stenosis, from the Greek word for 'narrowing', is a medical term used to describe a localized constriction in an artery. Stenoses are usually caused by the development of atheromatous plaques in the subintimal layer of the arterial wall, which subsequently protrude into the lumen of the artery, thus causing a narrowing to the free passage of blood.

A coarctation or arterial stenosis consists of a converging section, a narrow section, with the minimal luminal section defining the degree of stenosis, and a diverging section (Fig. 5.1). In the converging section, Bernoulli's equation holds (see Chap. 3). Within the narrow section Poiseuille's law is assumed to apply, provided that this narrow section is long enough with approximately constant diameter. In the diverging

N. Westerhof et al., *Snapshots of Hemodynamics: An Aid for Clinical Research and Graduate Education*, DOI 10.1007/978-1-4419-6363-5_5,

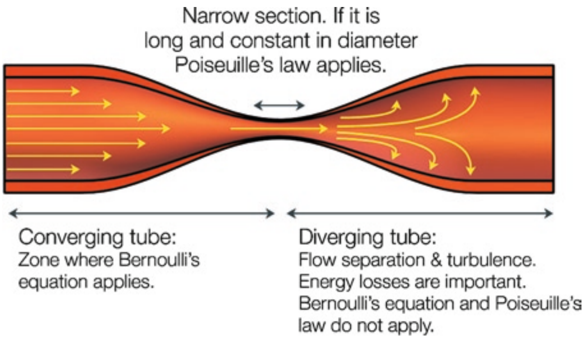


Fig. 5.1 A coarctation consists of a converging section, a narrow section and a diverging section, each with their particular pressure-flow relations

section flow separates and is often turbulent with significant viscous losses, which means that in this region neither Bernoulli's nor Poiseuille's law applies.

The severity of a stenosis can be expressed as % area or % diameter occlusion as $(1-A_s/A_o)\cdot 100$ or $(1-D_s/D_o)\cdot 100$, with subscripts s and o denoting stenotic and unstenosed vessel segments, respectively (Figure in the box). Pressure losses over a coarctation can be treated through semi-empirical relations. Such a relationship was developed by Young and Tsai [1] who performed a series of experiments of steady and pulsatile flows in models of concentric and eccentric stenosis. Young and Tsai found that the pressure drop, ΔP, across an arterial stenosis can be related to flow, Q, through the following relation:

$$\Delta P = \frac{8\pi\cdot\eta\cdot l_s}{A_s^{\,2}}\cdot Q + \frac{K_t\cdot\rho}{2A_0^{\,2}}\cdot[A_0 / A_s - 1]^2\cdot Q^2 = a_1 Q + a_2 Q^2$$

where A_0 is the unobstructed cross-sectional lumen area and A_s the minimal free cross-sectional lumen area within the coarctation (see Box Figure). The first term of the stenosis equation accounts for the viscous losses (Poiseuille's law) as blood flows through the narrow coarctation lumen. The second term accounts for the pressure losses distal to the stenosis and is derived from the mechanics of flow in a tube with an abrupt expansion. The K_t is an empirical coefficient approximately equal to 1.5, but strongly depending on the shape of the stenosis. The equation is derived for steady flow, but for oscillatory pressure-flow relations a similar equation holds [2].

Post-Stenotic Dilatation

The arterial diameter distal of a stenoses is often increased, a phenomenon called post-stenotic dilatation. The mechanism causing the dilatation is still not clear.

It may be due to abnormal shear stress and turbulent flow downstream of the stenosis, leading to extracellular matrix remodeling in the vessel wall. It has also been suggested that vessel wall vibrations distal to the stenosis cause the dilatation [3]

Physiological and Clinical Relevance

The best way to characterize a stenosis by measurement is by constructing the relation between flow and pressure across the stenosis (see Fig. 5.2).

The empirical formula for the pressure drop across a stenosis shows that both flow and area appear as quadratic terms. This is an important aspect of the hemodynamics of a coarctation. To illustrate the significance of the quadratic terms, let us assume that the stenosis length, l_s, is very small so that the first term in the equation above, $a_1 \cdot Q$, is negligible. The pressure drop is then proportional to the flow squared. Suppose that a patient with a mild coarctation in the femoral artery has, at rest, a pressure drop over the narrowed section of 10 mmHg. When the patient starts walking, and the peripheral bed dilates to allow for more perfusion flow, the drop in pressure increases. When flow needs to increase by a factor three the pressure gradient should increase to $10 \cdot 3^2 = 90$ mmHg. This is clearly impossible and the decrease in peripheral resistance of the leg does not help to increase flow sufficiently.

The pressure drop is inversely related to the square of the cross-sectional area in the stenosis. For a 80% area stenosis, the term $[A_0/A_s - 1]^2$ equals $[1/0.2 - 1]^2 = 16$, whereas for a 90% stenosis this term increases to 81. Thus a 90% stenosis is 81/16 or about five times more severe than an 80% stenosis in terms of pressure drop for

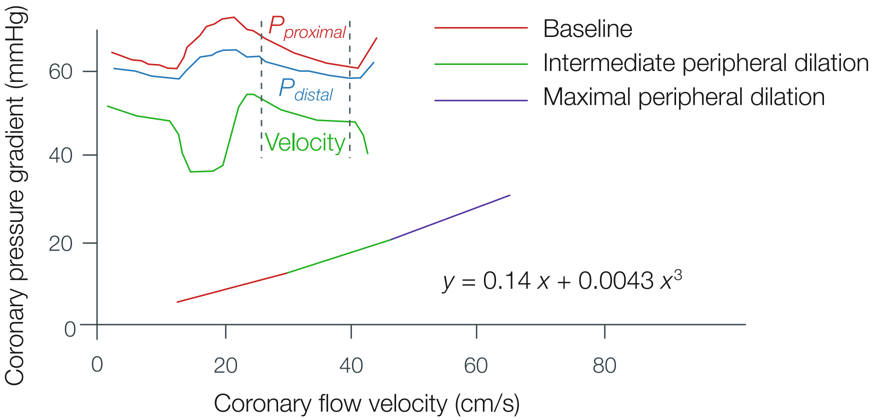


Fig. 5.2 Pressure drop over a coronary stenosis, as a function of blood flow velocity. The relations pertain to diastole and the range of velocities is obtained by vasodilation of the microcirculation. The quadratic expression can be applied. Adapted from ref. [4], used by permission

a similar flow, i.e., from 10 to 50 mmHg. This strongly nonlinear effect implies that complaints from ischemia will arise ‘suddenly’ when the narrowing becomes more severe, typically for a stenosis of >70%.

From Bernoulli’s equation it follows that at high velocity pressure is low (Chap. 3). This implies that when flow and thus velocity is high, as is the case during vasodilation, the pressure in the narrow section may decrease to low values. For stenoses with compliant walls the decrease in transmural pressure may lead to extra narrowing, thereby worsening the situation.

Flow Reserve

Angiographic data often do not give accurate information about the functional aspects of a stenosis or coarctation. This has led several investigators to propose methods to obtain a quantitative description in functional terms. One approach is the determination of flow reserve. The, absolute, flow reserve is defined as the ratio of flow during maximal dilatation and flow during control (Q_{max}/Q_c). In Fig. 5.3, pressure distal to a stenosis, P_d, is plotted as a function of flow, while proximal (aortic) pressure is assumed to be constant. It is apparent that when the periphery dilates, i.e., the peripheral resistance decreases, from R_c to R_d, the flow increases. However, in the presence of a severe stenosis (lower curve in Fig. 5.3), the flow increase is limited and distal pressure greatly decreases, and this pressure decrease is accentuated when flow is high. In control conditions, at rest, flow may be hardly affected by the presence of the stenosis, since peripheral dilation may compensate for the stenosis ‘resistance’, i.e., Q_c depends on stenosis severity and on microvascular resistance. At maximal vasodilation a severe stenosis limits maximal flow Q_{max} considerably, but the peripheral resistance remains playing a role. Thus, in presence of a stenosis, flows are not determined by the stenosis alone, but by both the stenosis and the microvasculature resistance. In other words, the flow reserve (Q_{max}/Q_c) is not determined by the severity of a stenosis alone.

Fractional Flow Reserve

Another estimate of stenosis severity is the Fractional Flow Reserve, FFR, which is the ratio of the maximal flow, $Q_{max,s}$ in the bed perfused by the stenosed artery and the maximal flow in a normal, unstenosed area, $Q_{max,n}$. The FFR is thus

$$\text{FFR} = [(P_d - P_v)/R_{st}]/[P_{prox} - P_v)/R_n] \cong P_d/P_{prox} \cong P_d/P_{aorta}$$

with P_d being the distal pressure during maximal dilation, and P_{prox} the proximal pressure. For coronary stenoses the proximal pressure equals aortic pressure. Under

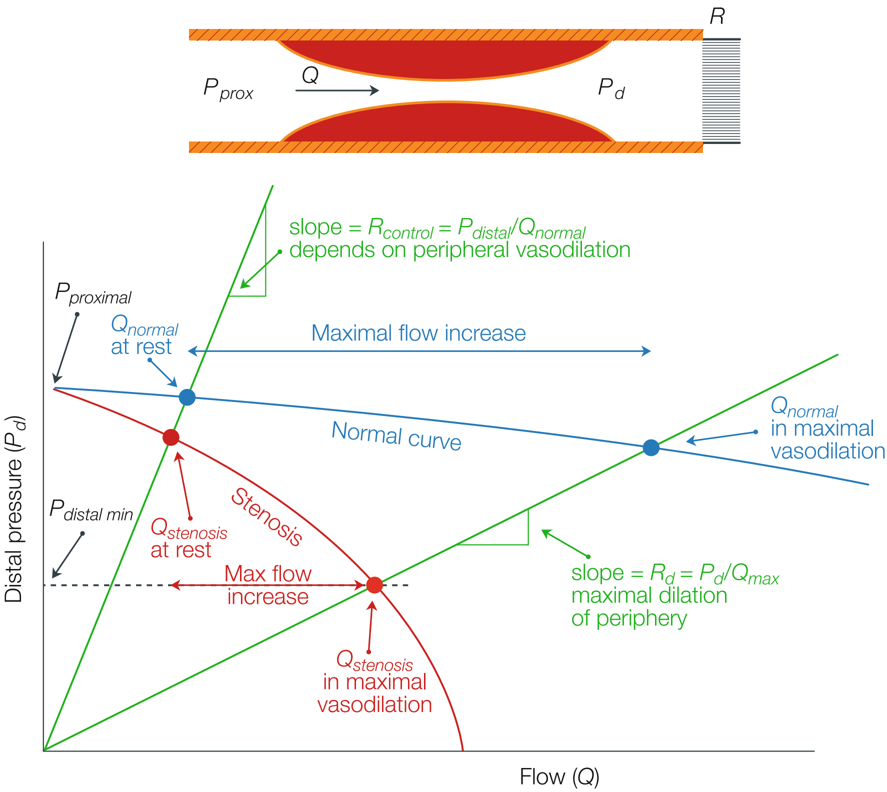


Fig. 5.3 Flow reserve is defined as the ratio of flow during maximal vasodilation and flow during control. In the unstenosed artery the ratio $Q_{max,n}/Q_n$ is much larger than when a stenosis is present, $Q_{max,s}/Q_s$. In this figure distal pressure is plotted as a function of flow. When the peripheral bed is maximally vasodilated, peripheral resistance decreases from R_c to R_d and flow increases, but distal pressure decreases. The decrease in distal pressure limits the maximal flow under vasodilation, thereby reducing the flow reserve. Thus, the flow reserve depends on the stenosis severity and microvascular resistance. The Fractional Flow Reserve, FFR, is the ratio of the maximal flow with the stenosis present and maximal flow in the unaffected bed, $Q_{max,s}/Q_{smax,n}$. The FFR also depends on the stenosis severity and how much the distal bed can dilate. The FFR is close to the ratio of the distal pressure during dilation and the proximal pressure, P_{dmin}/P_{prox}. The, nonlinear, relation between pressure drop over the stenosis and flow through it, $(P_{prox}-P_{dmin})/Q$, depends on the stenosis severity only

the assumption that the microvascular bed of the stenosed area has the same resistance as the bed of the normal area, $R_{st}=R_n$, and assuming that venous or intercept pressure, P_v, (Chap. 18) is small with respect to P_d it holds that the FFR is close to the ratio P_d/P_{aortax}. [5].

Although a normal periphery and the periphery distal to the stenosis may not have similar resistance, the FFR appears a workable parameter. The cut-off value of the FFR is 0.74, i.e., for values higher than 0.74 the stenosis is not considered functionally important.

For segmented stenoses, i.e., stenoses severity changes over its length, and for multiple stenoses the approach is more complicated.

Spaan et al. [6] have reviewed the principles and limitations of flow reserve.

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