

CHAPTER 7

VASCULAR OCCLUSION AND THROMBOSIS

VASCULAR OCCLUSION

To return to the present day, we shall first consider the problems of vascular occlusion.

Vascular occlusion may be arterial or venous and the effect of any occlusion will depend on:

- the type of tissue involved
- how quickly the occlusion develops
- availability of collateral circulation.

Collateral vessels provide an alternative route for the blood and they are sometimes able to compensate completely, especially if the occlusion develops slowly. The venous system has more collaterals than the arterial system. For example, there are anastomoses between the portal and systemic veins, around the lower end of the oesophagus and also linking veins between the deep and superficial venous plexuses in the leg. This means that occlusion of a deep vein in the calf does not produce haemorrhagic infarction of the foot but only a mild oedema of the tissues and congestion of the superficial veins because of their increased flow. Unfortunately, not all veins have a collateral system. If the central vein of the retina is occluded, as may happen in thrombosis of the cavernous sinus due to local infection, the tissue of the orbit becomes oedematous and congested so that the eye is pushed forward (proptosis) and there may be local haemorrhage as the small vessels rupture because of the increased pressure. In the worst cases the venous pressure rises until it exceeds the arterial pressure and prevents arterial flow. This produces infarction, i.e. death of the tissue, and the infarcted tissue is red or purple and swollen because of the haemorrhagic oedema. The word 'infarction' actually comes from the Latin '*farcire*' meaning to stuff and it is thought to have originally been used for the appearance of venous infarcts stuffed with blood.

Arterial collaterals exist in various areas such as the gut, circle of Willis and, to some extent, in the heart

(see Fig. 7.10). Arterial occlusion without the benefit of collaterals will produce ischaemic infarction where the tissue is pale without any swelling. Occasionally, arterial infarcts are haemorrhagic because there is reperfusion or some limited arterial flow leading to leakage of blood from necrotic small vessels. In incomplete arterial occlusion, the effects depend on the tissue's demand for metabolites. Brain and heart tissue are highly susceptible to ischaemic injury while bone and skeletal muscle are quite resistant. It is possible to reduce a tissue's demand by cooling the tissue as is done in some types of surgery.

Vascular occlusion can be due to:

- thrombosis
- embolism
- atherosclerosis
- external compression
- spasm.

We shall discuss the first three of these causes in some depth in this chapter, starting with thrombosis.

THROMBOSIS

Patients presenting with an **arterial thrombus** are generally middle-aged or elderly and may have circulatory problems due to atherosclerosis. Many will be smokers and some may suffer from diabetes. Their symptoms and signs will depend entirely on which vessel is affected. In contrast, a patient with **venous thrombosis** may be any age but generally will be rather immobile or forced to be immobile, such as after an operation. Such patients frequently complain of pain in a calf muscle and often swelling of the foot and ankle. But why should such people suddenly develop a thrombus? Much is known now about normal haemostatic mechanisms but the most important factors influencing thrombus formation were described more than a century ago by Rudolf Ludwig Karl Virchow (Fig. 7.1).

Virchow was born on 13 October 1821. As a child he excelled at school and his examination reports were