7

7.1 Introduction

Paraplegia has been a major concern of thoracic aortic surgeons ever since the first successful resection and replacement of a descending thoracic aneurysm in 1951 (which was, in fact, complicated by paraplegia [1]). Postoperative paraplegia remains the most devastating complication that faces patients undergoing surgery on the descending aorta because loss of lower-limb function imposes severe constraints on quality of life. Additionally, paraplegia is associated with higher postoperative mortality and morbidity.

Surgery on the thoracic aorta poses two distinct threats to the spinal cord. Firstly, to resect the aorta, the surgeon must temporarily interrupt lower-body aortic blood flow, rendering distal organs (including the spinal cord) ischemic intraoperatively. Secondly, replacement of the aorta may result in the permanent loss of spinal cord blood supply originating from the resected aortic segment. Being nervous tissue, the spinal cord tolerates ischemia poorly, and if infarction ensues, paraplegia results. In the early era of thoracic surgery, paraplegia rates in excess of 30% were reported, but with advances in surgical management, paraplegia rates below 10% became achievable in the 1980s [2].

In this chapter we review the contemporary anatomical and pathophysiological understanding of spinal cord blood supply, and present the scientific basis for clinical interventions used during descending aortic surgery to reduce the incidence of paraplegia.

7.2 Blood Supply of the Spinal Cord

7.2.1 Anatomy

The critical role of the descending aorta in the arterial blood supply of the spinal cord makes the spinal cord vulnerable to ischemia during disease processes or in-
terventions that involve the thoraco-abdominal aorta. The arterial supply to the spinal cord has been well studied [3]. The spinal cord is supplied via three longitudinal arteries: the anterior spinal artery, and the two posterior spinal arteries. The anterior artery is larger than the two posterior arteries, and provides 75% of spinal blood flow. There is little collateralization between the anterior and posterior arteries. Because the corticospinal tracts and motor neurons are largely supplied by the anterior spinal artery, it is occlusion or hypoperfusion of this vessel that is responsible for paraplegia. The anterior spinal artery is itself formed in the neck from the vertebral arteries, and continues caudally on the surface of the cord, receiving further blood supply via several segmental arteries (also called radicular arteries), which enter the spinal canal through the vertebral foramina [4]. These radicular arteries enter the cord in the three main regions of the spinal cord: cervical, thoracic and lumbar.

In the cervical region, the radicular arteries arise primarily from the vertebral, cerebellar, ascending cervical and other arteries, all of which arise from aortic arch vessels. As the input to the cervical cord is from the aortic arch, this part of the spinal cord is rarely compromised during descending aortic surgery. In contrast, in the thoracic and abdominal regions, the radicular arteries arise from the intercostal and lumbar arteries, which are branches of the descending aorta. The blood flow to the thoracic and lumbar cord therefore derives principally from the descending aorta, making this the region of the cord that is vulnerable during thoracic aortic surgery.

One segmental artery has assumed particular importance: the arteria radicularis magna (ARM), also known as the artery of Adamkiewicz, is an exceptionally large radicular artery that anastomoses into the mid-segment of the anterior spinal artery. Although large compared with other radicular arteries, the ARM is of variable diameter, ranging from 0.25 to 1.07 mm in cadaveric examinations [5]. Through the anterior spinal artery, the ARM supplies the majority of the flow to the lower thoracic and lumbar cord segments [3]. It can arise from any segmental artery between T7 and L4 on either side, or directly from the aorta, but frequently originates from one of the left segmental arteries between T8 and L1. In a study of 102 cadavers, Koshino et al. [6] found that approximately 70% of Adamkiewicz arteries originated from intercostal and/or lumbar arteries on the left side, frequently at the T8–L1 vertebral level.

Because the anterior spinal artery is generally continuous, loss of inflow from the Adamkiewicz artery alone does not generally result in paraplegia, since the anterior spinal artery will obtain sufficient inflow from the cervical and lumbar/hypogastric regions. On rare occasions, the anterior spinal artery may be poorly formed or discontinuous [4], so that loss of blood supply through the Adamkiewicz artery will render the lower anterior spinal territory ischemic. The incidence of discontinuous anterior spinal arteries is unknown, but two recent cadaveric studies did not find any instances of such discontinuity [5, 7]. Although the Adamkiewicz artery receives great prominence in anatomical texts, its importance is probably overstated. Some surgeons, notably Grieppe et al. [8], have questioned the clinical importance accorded to this artery, as they have routinely ligated the presumed origins of this vessel without clinical consequence.

There is additionally an extensive collateral network of vessels surrounding the length of the vertebral column, and communicating with the spinal arteries, which provides an alternate route of blood supply [4, 7]. Arteries feeding this collateral network form the so-called extrasegmental or extrinsic supply to the cord, and include branches of the subclavian artery (other than the vertebral arteries), the posterior vertebral and retrovertebral vessels, the intercostal and lumbar arteries (other than the ARM), the hypogastric arteries and the median sacral artery.

This collateral network becomes clinically relevant during aortic surgery. During aortic clamping, part of the spinal blood supply will route via collaterals from the subclavian arteries [9], making it important to maintain adequate proximal pressures during cross-clamping. Additionally, if segmental blood inflow is lost (such as by endovascular stenting or open repair with division of intercostals), the extrinsic collateral network becomes a major route of blood supply to the mid-cord. Sufficiently high blood pressure is probably necessary to drive blood through these collaterals. This anatomical feature may explain the observation that delayed onset paraplegia after aortic repair is often preceded by hypotension [10] (since a low blood pressure may be insufficient to drive blood through these collaterals to the spinal cord). If the collateral network has previously been disrupted, such as by earlier abdominal or pelvic surgery, then the segmental blood flow assumes greater importance [11]: such patients may be more prone to paraplegia, particularly if segmental vessels are sacrificed.

### 7.2.2 Determinants of Spinal Blood Flow

As there are no modalities for direct measurement of spinal blood flow in man, current understanding derives from animal experiments and clinical extrapolations. The principal determinants of spinal blood flow are anatomical, physiological and pathological factors.

#### 7.2.2.1 Integrity of Anatomical Pathways

Spinal cord blood flow depends upon anatomical integrity of the circulation described earlier. Anatomical in-