Malperfusion is a devastating complication afflicting 16–33% of acute type A aortic dissection (AAAD) [1]. It may result in myocardial, cerebral, spinal, extremity, renal and visceral ischaemia and is most common in extensive DeBakey type I dissection [2, 3].

In this issue of the Journal, Pacini et al. [4] report a cohort of 502 AAAD, of which 103 (20.5%) had a malperfusion syndrome (MPS). The presence of malperfusion increased hospital mortality three-fold and multivariable analysis identified mesenteric or cardiac malperfusion and shock (present in 39% of MPS cases) as independent predictors of early mortality.

The Emilia-Romagna region of Italy has a population of 4.4 million and is served by six surgical cardiac centres [5]. If all diagnosed AAADs undergo surgery, the annual incidence of (operated) AAAD is ~13 pmp and is comparable with the Swedish experience [6]; the real denominator is those reaching hospital alive and diagnosed AAAD but treated medically [16% as in International Registry of Acute Aortic Dissection (IRAD)].

The definition of malperfusion is important as was noted in the study. For MPS to occur, the patients in this Registry were required to have ‘signs and symptoms due to altered blood flow with clinical evidence of a lack of blood flow resulting in ischaemia with organ dysfunction’. While demonstration of radiological malperfusion may also be important, it is the ischaemic consequences of malperfusion and end organ dysfunction that compromise survival [7]. Thus, although a patient may have CT evidence of reduced true luminal innominate artery flow, the prognostic relevance of this is primarily determined by the presence of neurological ischaemia.

The low preoperative brain malperfusion rate (7.8%) in this series may explain the lower postoperative stroke risk (8.8%) in 44 patients (9 with preoperative malperfusion) than in either the IRAD or the German Registry for Acute Aortic Dissection Type A (GERAAD) registries [8]. Within GERAADA, preoperative neurological deficit occurred in 20.3% of all patients prior to surgery, 12.6% of which resolved postoperatively and 7.7% persisted. New neurological deficit after AAAD surgery occurred in 9.5% of patients. In agreement with GERAADA, stroke was more common in any anatomical MPS. Approximately 30% of cases were undertaken without deep hypothermic circulatory arrest (DHCA), 16% with DHCA and 53% with antegrade selective cerebral perfusion (15% with axillary artery cannulation). No differences in the outcome were noted between these perfusion strategies. Similarly, GERAADA reported no differences in the outcome between cerebral protection strategies [9].

The victims of acute aortic dissection have poor long-term survival, in the reported series, of 65% at 5 years. This was lower in the MPS patients, but remained at 70% for non-MPS patients. The causes of these deaths have not been reported but need to be examined carefully if the attrition rate is to be attenuated. However, it was probably not due to dissection-related aneurysmal disease as actuarial freedom from reoperation was 96.6% at 5 years in a 100% follow-up study. This provides food for thought for those advocating a more aggressive approach in primary type A dissection repair.

Thus, in keeping with other recent reports, the Emilia-Romagna Registry has given us more questions than answers. We can certainly say that malperfusion increases perioperative mortality and decreases long-term survival, but are we any closer to finding better ways of managing it?

REFERENCES


