Anaesthesia and cognitive function

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Postoperative cognitive decline (POCD) following cardiac and major non-cardiac surgery presumably reflects iatrogenic brain injury. The etiologic mechanisms, however, remain unclear, hindering our ability to develop strategies to reduce the incidence and severity of POCD and improve patient outcomes.

In both cardiac and non-cardiac surgical populations, increasing age is a strong risk factor for POCD (1-4). Apolipoprotein e4 genotype is not (5,6). In non-cardiac surgery, the extent of surgical trauma is associated with POCD, but surprisingly, anaesthetic technique and intraoperative episodes of hypotension and hypoxaemia are not (1,3,7-11).

In cardiac surgical patients, cardiopulmonary bypass has been assumed to be causal, given its potential for cerebral embolization of air and particulate matter. Early studies suggested that microemboli circulated during cardiopulmonary bypass were the etiologic basis of POCD. Several studies have shown a higher incidence of POCD in patients undergoing valve surgery vs. coronary artery bypass grafting (CABG) surgery, presumably due to greater frequency of microembolization (12,13). Comparisons of patients who underwent either on- or off-pump CABG, however, have not consistently found any difference in POCD incidence between the two groups (14,15).

With regard to temperature management, a prospective study demonstrated a clear causal relationship between rapid rewarming with hyperthermic perfusate during CPB and POCD (16). A retrospective study reported that postoperative hyperthermia was associated with POCD (17). Thus hyperthermia during the intraoperative phase should be avoided. It is less clear whether postoperative

hyperthermia prevention would improve outcomes, but it is prudent to limit the degree of hyperthermia.

There is some evidence that extreme haemodilution (haematocrit 15-18%) during cardiac surgery in the elderly is associated with worse neurocognitive outcome than moderate haemodilution (haematocrit >27%) (18).

The initial study examining the longitudinal course of POCD in cardiac surgical patients documented progressive decline over a 5year period from time of surgery (19). Progressive decline beyond the first year after surgery, however, evidently reflects progressive cerebrovascular disease. A study comparing patients with coronary artery disease (CAD) treated either medically or surgically (on- or off-pump CABG) with healthy heart control patients found that all CAD patient groups had lower pre-operative neurocognitive function and greater degrees of decline over a 6-year interval following surgery, with no differences between the medically treated vs. surgical subgroups (20). Another longterm study of neurocognitive function comparing patients undergoing CABG vs. percutaneous coronary intervention found no difference (21). Thus, a significant proportion of patients with advanced CAD have neurocognitive impairment preoperatively that is likely to progress, independent of surgery.

The study of POCD is especially important in patients undergoing aortic arch surgery who undergo various combinations of hypothermic circulatory arrest (HCA), retrograde cerebral perfusion (RCP) and selective cerebral perfusion (SCP). These surgical procedures are performed in referral centres drawing patients from large geographic regions, posing significant obstacles to neurocognitive out-

comes research, since many patients are physically inaccessible for follow-up testing. Thus, there are few neurocognitive outcome studies in this patient population and they are all limited by small sample size. In summary, this literature suggests that more complex surgeries requiring longer intervals of HCA, RCP and SCP are associated with worse neurocognitive outcomes than shorter intervals (22-27).

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