Cerebral perfusion and good neurological outcome appear most obviously at risk during operations on the aortic arch. However, it is also evident that from a numerical standpoint, otherwise uncomplicated coronary artery bypass grafting (CABG) accounts for the greatest number of perioperative strokes, as shown in a review of 7839 patients in which it was determined that the overall incidence of clinical stroke immediately apparent at extubation was 1.4% and that in addition to severe aortic calcification, cardiopulmonary bypass (CPB) time was also an independent risk factor [1]. Similarly, in a previous study of 13 897 CABG patients, it was also noted that the duration of CPB increased the risk of hypoperfusion strokes and that nearly 75% of all strokes occurred among the 90% of patients at low or medium preoperative risk [2].

These facts indicate that the duration of CPB and inadvertent cerebral hypoperfusion can directly influence the risk of perioperative stroke and adverse neurological outcomes. Clearly, for most patients undergoing cardiac surgery, survivalship is of first importance, but it is also readily apparent that for many patients, the risk of severe neurological injury, stroke and significant cognitive impairment are ever-present and particularly feared concerns [3].

While cerebral atheroemboli have been identified as a major factor in perioperative stroke, the influence of unrecognized cerebral hypoperfusion, whether as a consequence of underlying cerebrovascular disease, inadvertent cannula malposition, cerebral vasoconstriction attending unintentional hyperventilation, cerebral venous outflow obstruction, inadequate perfusion pressure, anemia or various combinations of these factors alone or in concert, contributes to, and can independently precipitate, perioperative neurological injury [4].

In a recent review of 190 cases of aortic arch surgery managed under deep hypothermic circulatory arrest (HCA) and antegrade selective cerebral perfusion (SCP), there was an acute stroke incidence of 5.6%, of which 2.7% were categorized as multiple embolic and 2.1% were felt to be due to hypoperfusion associated with prolonged brain ischemia [5]. A recent review has emphasized that even cerebral cortical border zone infarcts, which are now felt to be primarily embolic, are a reflection of lowered perfusion and thus impaired clearance of emboli from brain regions at the junction of anterior, middle and posterior cerebral arteries [6], and further adverse interactions between emboli and hypoperfusion in the genesis of acute stroke are also being increasingly recognized [7]. It is, thus, quite plausible that detection and avoidance of cerebral hypoperfusion during CPB—and particularly during aortic arch surgery—can substantially mitigate cerebral risk.

While HCA can be successfully employed for aortic arch surgery with <30 min circulatory arrest, for longer procedures diverse SCP strategies have been adopted in addition to employment of varying degrees of cerebral and systemic hypothermia [8]. What is also apparent is that monitoring the adequacy of cerebral perfusion using near-infrared spectroscopy (NIRS) to measure regional cortical oxygen saturation (rScO2)—to shine a light, can identify otherwise unrecognized cerebral hypoperfusion and has been shown to result in more optimal cerebral perfusion during aortic arch surgery [9-13].

Even during uncomplicated CABG surgery, venous cannula malposition causing cerebral oxygen desaturation has been identified by rScO2 and rapidly corrected, as has cerebral desaturation from a variety of other etiologies [4, 14]. Cerebral NIRS-guided pressure augmentation is also a developing technique that has been successfully employed to treat low cerebral saturation during prolonged retrograde SCP with good clinical outcome [9]. Additionally, there are now a number of case reports and clinical series during antegrade SCP in which cerebral NIRS has been essential in diagnosing otherwise clinically silent arterial cannula malposition, perfusion catheter balloon migration or inadequate contralateral cerebral perfusion [10-13].

Even in the absence of such technical problems, however, a variety of other physiological and pharmacological strategies have been developed to treat low rScO2 during CPB and have been associated with improved clinical outcomes [4, 15], and in at least one large retrospective series, cerebral oximetry monitoring has also been associated with a significant decrease in clinical stroke [16]. Conversely, in both retrospective and prospective clinical series, low rScO2 has been associated with adverse outcomes including prolonged ventilation, hospitalization, cognitive dysfunction, stroke and death [10, 14, 17-20].

So, why is there a degree of reluctance to employ cerebral oximetry monitoring on a more routine basis? One rationale appears to be based on the somewhat fatalistic attitude that

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nothing more can be done to mitigate cerebral risk—‘to curse the darkness’, or the related concern that if low rScO2 is detected and documented, it may give rise to medicolegal issues. Other concerns, however, may more realistically reflect the current limitations of commercially available devices.

The estimated area of brain cortex interrogated by cerebral NIRS devices is considered to be on the order of 1 cc [21]. Application over the frontal region provides an estimate of cortical tissue oxygen saturation in the watershed region at the confluence of anterior and middle cerebral arteries and can thus detect hypoperfusion in the anterior circulation. However, this does not provide information on other areas of brain, including brain stem and middle and posterior cerebral circulations. There is also an increasing concern about the degree to which signals from extracerebral tissue including skin and overlying structures may contaminate rScO2.

In a recent study, a decrease in rScO2 ranging from 6.8 to 16.6% was seen after inflation of a scalp skin tourniquet depending on which of three commercial devices was utilized [22]. The impact of extracerebral tissue and its influence on rScO2 are now being actively investigated and appear to be further influenced by the choice of vasoconstrictive pharmacological agents [23–26]. While various theories ranging from alterations in cardiac output [23, 24] to changes in carbon dioxide tension [25] have been proposed to account for the variable response of rScO2 to such pharmacologic agents, meticulous studies have indicated the most plausible mechanism to be a variable degree of skin vasoconstriction, producing an otherwise artefactual decrease in rScO2 associated with phenylephrine administration [26].

As such, it may be that some of the profound desaturation occasionally observed during HCA and in similar settings may be a consequence of hypothermic skin vasoconstriction and that depending on choice of vasopressor, specifically phenylephrine, increased extracerebral vasoconstriction and a further spurious decrease in rScO2 may ensue. However, it appears that in the absence of profound hypothermia and/or high-dose phenylephrine vasoconstriction, or with the development of unilateral cerebral desaturation, a decrease in rScO2 remains a robust sign of cerebral hypoperfusion and has been repeatedly associated with early identification and correction of adverse technical and physiological perturbations [4, 21].

Since the initial introduction of commercial cerebral oximetry in 1993 [21], there are now at least four commercially available devices. Additionally, a new generation of ultrasound based cerebral oximeter that measures cortical blood flow in addition to providing a more focused detection of cerebral oxygen saturation is in development and appears to offer the promise of the avoidance of extracerebral tissue contamination as well as providing real-time and continuous information on cerebral blood flow [27]. One implication of the high incidence of cerebral malperfusion of 11.4% seen in a series of 35 patients undergoing repair of aortic arch aneurysm using HCA with SCP was the observation that it was a drop in rScO2 <60% and/or low perfusion pressure that initiated transorbital ultrasound examination of retinal blood flow, and it was the associated identification of inadequate flow by ultrasound that confirmed hypoperfusion, prompting readjustment of cerebral perfusion catheters [13]. Integration of real-time cerebral flow and saturation measurements thus provides even further insight into the adequacy of cerebral perfusion and can provide corroborative or even synergistic information on the state of the cerebral circulation.

So, while there are a number of limitations associated with current cerebral oximetry devices, to date rScO2 monitoring has proven useful in diverse clinical settings and when used to guide therapy, has been associated with improved clinical outcomes during a variety of different surgical procedures [15, 21]. Understanding the limitations to existing devices as discussed here will only help improve their efficacy and facilitate more rational utilization and better guide intervention strategies, while the promise of greater cerebral specificity and concomitant flow-related information associated with next generation devices can only further enhance patient outcomes.

‘The future’s so bright I gotta wear shades’
Timbuk3

Conflict of interest John M. Murkin is a member of the Scientific Advisory Board for Ornim Medical, a manufacturer of cerebral oximetry devices.

REFERENCES


