however, had post-operative TND, which has been shown to be associated with long-term post-operative neuropsychological dysfunction [12]. We have no way of determining the proportion of patients who had neuropsychological dysfunction in the portion of the cohort that was lost to follow-up.

In light of the limitations of this retrospective analysis, we have not discontinued our use of jugular bulb oxyhemoglobin monitoring in thoracic aortic surgery, but are less concerned when prolonged cooling fails to raise the SjO2 above the 95% threshold. The technical ease and lack of complications associated with jugular bulb catheterization are such that no significant cost or time savings would result from discontinuation of SjO2 monitoring. Furthermore, analysis of a larger cohort of patients with SjO2 < 90% in the future may yield further information.

It is unlikely that cerebral hypothermia beyond the levels used in our institution confers additional neuroprotection in the thoracic aortic surgical population. Other methods must be explored to reduce the significant incidence of TND and neuropsychological dysfunction in thoracic aortic surgery requiring DHCA [6,13]. We are currently evaluating the use of right axillary artery cannulation, performing a separate graft to the brachiocephalic arteries (e.g. Tanaguchi procedure), and selective cerebral perfusion to reduce the period of cerebral ischemia in aortic arch reconstructions [14].

In conclusion, SjO2 monitoring and attempts to achieve SjO2 ≥ 95% prior to the onset of DHCA do not appear to improve neurological or neuropsychological outcome following thoracic aortic surgery requiring DHCA with the profound cooling regimen at the authors’ institution. Further investigation will be required to determine if lower SjO2 thresholds have prognostic significance in DHCA patients.

References


Appendix A. Editorial comment

Post-operative neuropsychological function unaffected by SjO2 monitoring in DHCA

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Three cerebral protection methods are currently being used in thoracic aortic repair: deep hypothermic circulatory arrest (DHCA) with or without retrograde cerebral perfusion (RCP) and antegrade selective cerebral perfusion (SCP).

Dr Griep, of Mt Sinai Hospital, having popularized this method of protecting the brain during aortic arch repair [1], is recognized as the father of DHCA, while Mt Sinai group as a whole is credited with numerous scientific papers regarding the neuroprotective effect of DHCA. However, appropriate criteria for initiating DHCA have not been established so far and tend to vary depending on the hospital. These have included electrical silence of the electroencephalogram, SjO2 monitoring or merely measurements of temperature at different sites (rectal, bladder,
esophageal, nasopharyngeal, and tympanic). At Mt Sinai, S\text{J}O_2 monitoring has been used as a marker of initiating DHCA because of the assumption that highly saturated jugular venous blood reflected global cerebral cooling as well as the concomitant cerebral metabolic suppression before the initiation of DHCA. In this issue of the journal, Reich and associates [2] from the Department of Anesthesiology at Mt Sinai Hospital, report on the implications of applying S\text{J}O_2 monitoring as the criterion for initiating DHCA during thoracic aortic surgery. Thoracic aortic surgical patients who had undergone both pre- and postoperative neuropsychological testing were divided into three groups: (1) those with S\text{J}O_2 \geq 95\% at DHCA onset, (2) those with S\text{J}O_2 < 95\% at DHCA onset, and (3) those without S\text{J}O_2 monitoring. No statistically significant differences were noted in the incidence of post-operative neuropsychological dysfunctions among the three groups of patients. Use of S\text{J}O_2 monitoring was associated with more profound hypothermia prior to DHCA due to more prolonged cooling in an attempt to bring the S\text{J}O_2 above the 95\% threshold. Although there are limitations for the current study including the high attrition rate, small sample size of the study, and the difference in mean cooling duration among the three groups, the study provides us with some valuable information. According to the study, S\text{J}O_2 monitoring does not increase neuroprotection in patients undergoing DHCA for thoracic aortic repair. The discrepancy between jugular bulb oxygenation saturation before the initiation of DHCA and the post-operative neuropsychological function, considered to be a sensitive measure of brain function, could be explained in several ways.

First, although the main mechanism of cerebral protection in DHCA has been suggested to be cerebral metabolic suppression, other hypothermia-induced mechanisms—reductions in ATP depletion, release of toxic neurotransmitters, generation of free radicals and reperfusion injury—may play a considerable role in protecting the brain from ischemic injuries. In addition to this, another factor, i.e. gaseous and particulate embolization during reperfusion, may also play a significant role in the incidence of neuropsychological dysfunction.

Secondly, even if the core temperature is brought down to 13°C, the cerebral oxygen consumption still remains at 20% of the baseline (37°C) [3]. Therefore, a limit to the circulatory arrest time must essentially exist. McCullough and colleagues [4] have reported that a safe DHCA duration at 15°C, based on the calculated cerebral metabolic rate, was 29 min. Moreover, Reich and coworkers [5] have previously reported that a DHCA of 25 min or more and an advanced age were associated with memory and fine motor deficits. In this series by Reich et al. although the mean cerebral ischemic time ranged from 0.30 to 0.37 h, which seemed to be within the ‘safe’ duration margin in terms of cerebral protection, their longest cerebral ischemic time ranged from 1.00 to 1.83 h in each group. It is widely recognized that the temperature during DHCA and the duration of circulatory arrest are important determinants of the adequacy of cerebral protection. It is therefore only to be expected that patients undergoing DHCA of 25 min or more have developed neuropsychological dysfunction regardless of S\text{J}O_2 monitoring. I am curious whether the difference would have been noted among the three groups if DHCA patients had been compared according to the duration of circulatory arrest in each group.

Thirdly, due to some theoretical limitations, S\text{J}O_2 monitoring could be applied as a global cerebral hypothermia, but not as cerebral metabolic suppression, as mentioned in the discussion.

Lastly, the authors refer to jugular venous desaturation during the rewarming phase of CPB. Since we have occasionally experienced the same phenomenon with a moderate hypothermic antegrade selective cerebral perfusion [6], it would be a good idea to investigate the effect of desaturation during the rewarming period on the postoperative neurocognitive dysfunction.

It is apparent from this study that patients undergoing DHCA within a ‘safe’ circulatory arrest duration had developed some neuropsychological dysfunction which seemed to be related to improper cerebral protection. Clearly, we must seek a safer alternative to DHCA. The question is whether another adjunctive cerebral protective technique will increase the neuroprotective effect evaluated by neuropsychological testing. Harrington and colleagues [7] reported that RCP with DHCA did not improve neuropsychometric outcome; similarly, Reich and associates [8] indicated that RCP had no beneficial effect (and most likely a negative one) upon cognitive outcome. We are looking forward to further investigation into whether SCP, which has been used routinely for complex and time-consuming aortic arch repair at our institute [9], improves post-operative neuropsychological outcome, as it supplies sufficiently oxygenated blood to the brain resulting in better cerebral metabolism.

Finally, I would like to congratulate Mt Sinai group on this excellent work which will definitely contribute to the field of aortic surgery.

References
[4] McCullough JN, Zhang N, Reich DL, Juvenon TS, Klein JJ, Spielvogel D, Arisan Ergin M, Griep RB. Cerebral metabolic suppression during...


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