

Category: **Technology assessment and health informatics:**
Monitoring technology

Title: MONITORING OF NON-INVASIVE ABSOLUTE BRAIN OXYGEN SATURATION TO DETECT CEREBRAL HYPERPERFUSION AFTER CAROTID ENDARTERECTOMY

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Text: Cerebral hyperperfusion syndrome, caused by inflow at normal blood pressure into maximally dilated fine vessels, is a recognized complication of carotid endarterectomy (CEA) Strict blood pressure control in the early postoperative period can minimize the risk of cerebral hyperperfusion. Until yet, diagnosis of cerebral hyperperfusion mainly relies on intermittent postoperative examinations (SPECT; CT angiography). Non-invasive absolute cerebral oxygen saturation (SctO₂ by Fore-Sight technology) was validated to jugular bulb saturation (SjO₂) monitoring with a constant difference of 10% higher for SctO₂ values. Previously, SjO₂ monitoring after severe head injury indicated cerebral hyperemia. In this study, we evaluated SctO₂ monitoring after carotid surgery as possible continuous on-line monitoring of cerebral hyperperfusion.

Fourteen pts scheduled for CEA were monitored for 12hrs postoperatively after CEA. Bilateral SctO₂ monitoring was started before induction of anesthesia and maintained until 12hrs postoperatively. Intra-operative EEG monitoring guided the decision to intraluminal shunt insertion. Strict blood pressure control was applied at maintaining normotensive levels throughout the clamping procedure. Early postoperative care focussed on strict maintenance of normotensive blood pressure.

In no pt, any change in EEG was observed after carotid clamping. In all pts, ipsilateral SctO₂ significantly decreased after carotid clamping, without any SctO₂ value below 55%. We observed no changes in contralateral SctO₂. Mean clamping time was 28min (19-37 min). In all pts, clamp release restored ipsilateral SctO₂ to baseline values. In all pts, emergence from anesthesia was uneventful, without any new neurological deficit. In 6 of 14 pts, significant increases (SctO₂ > 85%) in ipsilateral SctO₂ were observed in the postoperative period (mSctO₂ 87.5%), without any changes in contralateral SctO₂. This increase occurred at a mean of 3.4hrs after carotid declamping with a mean duration 5.3hrs. In these 14 pts, we could not make any significant correlation to arterial blood pressure, as none of these 6 pts needed more aggressive antihypertensive control. We noted that 4 of these 6 pts suffered from diabetes mellitus, while 5 of 6 pts revealed high (> 90%) contralateral stenosis. Further data will have to reveal the importance of these comorbide factors.

Non-invasive cerebral oximetry, enabling absolute cerebral oxygen saturation monitoring, could provide on-line estimation of cerebral perfusion state after CEA. This could allow bedside detection (and eventual therapeutic interventions) of cerebral hyperperfusion after CEA.