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Cerebral Air Embolism Recognized by Cerebral Oximetry

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Absolute cerebral oximetry is useful in clinical settings to identify "catastrophic events" that may occur during the course of surgeries that would otherwise have gone unrecognized. This study reports a case in which cerebral desaturation occurred after commencing cardiopulmonary bypass. Consequently, the source of air entrainment was discovered and therapeutic measures implemented.

Keywords: cerebral oximetry; cardiac surgery; air embolism; cerebral desaturation

Cerebral oximetry, based on near-infrared spectroscopy technology, provides information on the availability of oxygen in brain tissue at risk during numerous pathological conditions. We report the early detection of a cerebral air embolism during cardiopulmonary bypass (CPB) that could potentially have gone unrecognized if not for cerebral oximetry monitoring.

Case Report

A 62-year-old patient presented to our institution for aortic root replacement (Bentall procedure) and a stage I elephant trunk procedure. Past medical history was significant for ascending aortic aneurysm repair with a supracoronary tube graft 5 years previously, mild chronic obstructive pulmonary disease, and controlled arterial hypertension.

Monitoring for the case included standard ASA (American Society of Anesthesiologists) monitors, an arterial pressure monitoring catheter, a pulmonary artery catheter, a left-sided jugular bulb catheter, transesophageal echocardiography (TEE), and the ForeSight cerebral oximeter (CASMED, Branford, CT).

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Induction of general anesthesia was uneventful. After placement of invasive central monitors, bilateral cerebral oximetry sensors were placed on the patient's forehead. General anesthesia was maintained using a balanced technique consisting of isoflurane (0.6 to 1.2 vol%) and intermittent boluses of fetanyl, midazolam, and vecuronium.

Aneurysms of the aortic arch (diameter 5.2 cm) and descending aorta (diameter 5.8 cm), and moderate to severe aortic regurgitation (3+) were the only significant findings of the preprocedural TEE exam.

The right axillary artery was exposed but was unable to be cannulated directly, so a gortex graft was sutured (end-to-side) to the artery for arterial return from the CPB machine. Thereafter, a sternotomy was performed and the heart dissected free of adhesions. Venous drainage was achieved via a 2-stage cannula inserted into the right atrium. As moderate to severe aortic insufficiency would have limited the potential efficacy of antegrade cardioplegia delivered via the aortic root, cardioplegia delivery was to be via the individual coronary ostia once the aorta was cross-clamped. Consequently, the patient was systemically cooled awaiting spontaneous ventricular fibrillation. However, shortly after commencing CPB (and prior to cross clamping of the aorta) significant bilateral declines in cerebral tissue oxygen saturation ($SctO_2$) were detected. TEE examination revealed multiple air bubbles in the left atrium, left ventricle, and aorta (Figure 1). Further TEE examination revealed the air to be coming from the

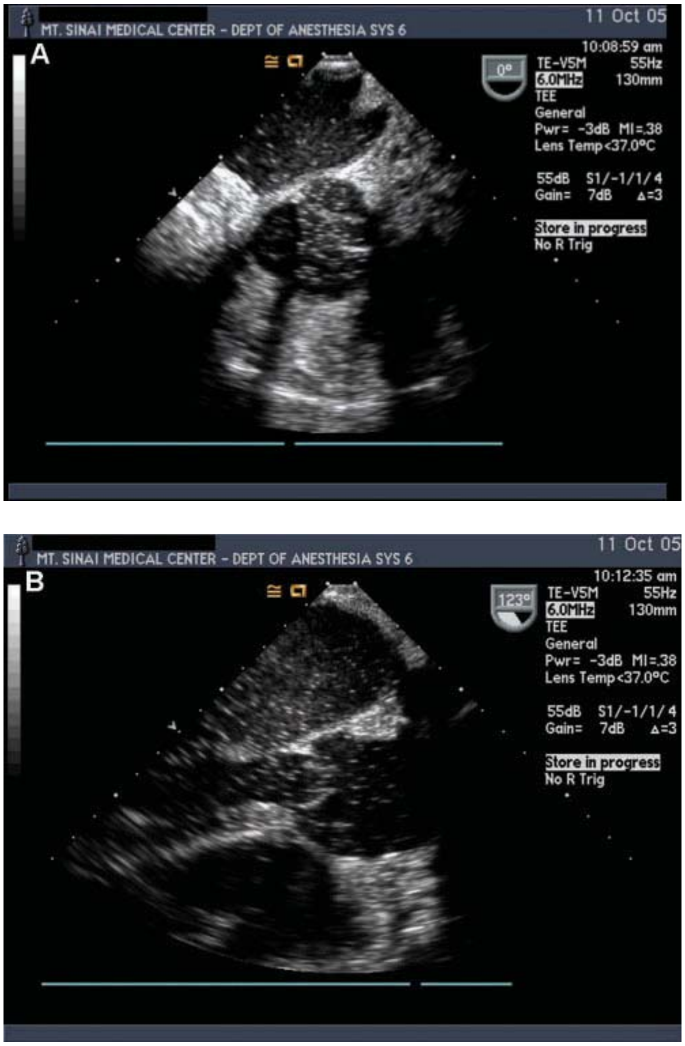

Figure 1. Transesophageal short axis (A) and long axis (B) views of the aortic valve. Air bubbles can be seen in ascending aorta, left ventricle, and left atrium.

innominate artery. The surgical team was immediately informed, the patient placed in steep Trendelenburg position and the axillary artery cannulation sight flooded with saline. This maneuver resulted in a dramatic and immediate decrease in the amount of air bubbles detected by TEE. Additional ligatures were placed around the arterial cannula in the axillary artery graft and further air entrainment was not detected by TEE for the remainder of the case.

Cerebral oximetry values remained much lower than expected despite adequate cooling (Figure 2). After 60 minutes of cooling, achieving a bladder temperature of 15°C and 2 consecutive jugular venous saturations >99%, deep hypothermic circulatory arrest (DHCA) was commenced and lasted for 33 minutes. After reinstitution of circulation the

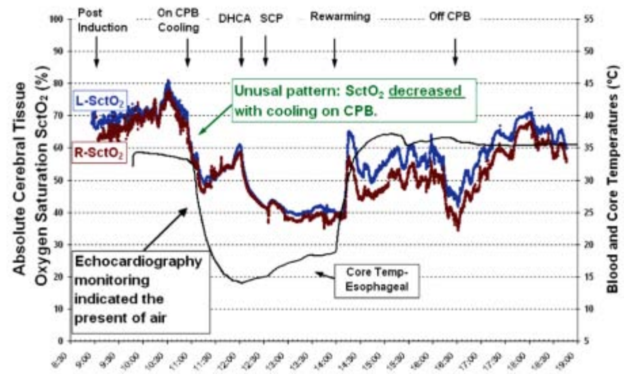

Figure 2. Cerebral oximetry values remained much lower than expected despite adequate cooling. Instead of mirroring core temperature, $SctO_2$ values continue to decline despite cooling of the patient.

patient was rewarmed gradually over the course of 65 minutes. The peripheral to core (esophageal to bladder) temperature gradient was not allowed to exceed more than 5°C. Additionally, the maximum acceptable arterial inflow temperature was limited to 37°C. At 35.5°C the patient was weaned off CPB on minimal inotropic/vasopressor support. The post-CPB course was uneventful. The $SctO_2$ readings remained in the 60s.

The postoperative course was complicated by severe postoperative delirium. Daily attempts were made at reducing the level of sedation, however, severe agitation made sedation necessary until the fifth postoperative day. Despite being extubated on the fifth postoperative day the patient remained disorientated, but gradually recovered to a fully lucid state over the subsequent 6 days. No focal neurological deficits were detectable at the time of hospital discharge on postoperative day 20.

Discussion

Cerebral oximetry, based on near-infrared spectroscopy technology, provides information on the availability of oxygen in brain tissue at risk during numerous pathological conditions.[1] Cerebral oximetry measures local concentrations of oxyhemoglobin and deoxyhemoglobin, and regional cerebral tissue oxygen saturation ($SctO_2$) at the microvasculature level (arterioles, venules, and capillaries only).[2-4] As a result, cerebral tissue oxygen saturation ($SctO_2$) is a mixed oxygen saturation parameter that has a value between arterial

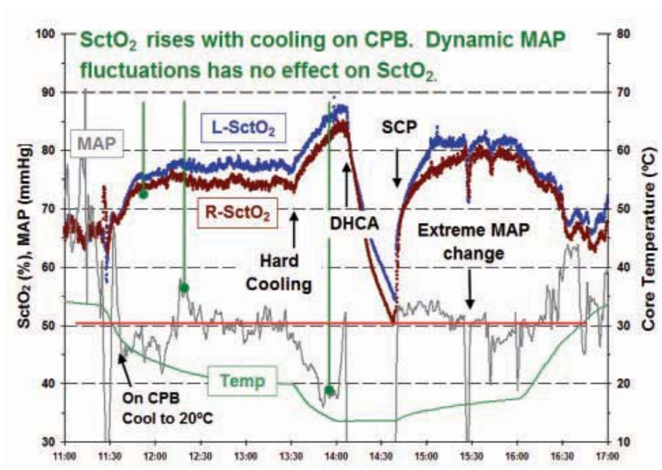

Figure 3. A typical pattern of recordings during cardiopulmonary bypass (CPB), cooling, deep hypothermic circulatory arrest (DHCA), selective cerebral perfusion (SCP), and rewarming.

(SaO_2) and jugular venous oxygen saturation ($SjvO_2$) under normal physiological conditions, therefore $SaO_2 > SctO_2 > SjvO_2$. Complementary to the arterial oxygen saturation (SaO_2) measured by pulse oximetry, $SctO_2$ reflects regional cerebral metabolism and the balance of local cerebral oxygen supply/demand. The advantages of cerebral oximetry are (*a*) it provides $SctO_2$ values continuously and noninvasively at the bedside[5] and (*b*) $SctO_2$ is a sensitive index of cerebral hypoxia and/or cerebral ischemia,[6,7] which is one of the main causes of brain injury in clinical settings.[8,9].

It has been our experience that a typical pattern for $SctO_2$ in patients undergoing aortic surgery with DHCA shows a post induction $SctO_2$ of approximately 70% (Figure 3). During cooling on CPB, a rise in $SctO_2$ mirroring the rate of core cooling should be expected. During DHCA, $SctO_2$ decreased as a consequence of ongoing metabolic demand without oxygen supply. Subsequently $SctO_2$ returns to pre-DHCA levels once cerebral circulation is returned using selective antegrade cerebral perfusion. Despite adequate cooling, this patient's $SctO_2$ continued to decline shortly after initiation of CPB leading the anesthesiologist to search for an explanation. In this case, TEE was able to identify the source of air entrainment into the aorta (and retrograde across the incompetent aortic valve into the left-sided cardiac chambers; Figures 1A and 1B).

Embolization of gaseous or particulate matter is common during cardiac surgery.[10] It is typically encountered during manipulation of the ascending aorta (cannulation, clamping, and unclamping) by the surgeon or interventions by the perfusionist while on CPB.[11] Due to the fact that embolic load is most likely linked to neurological outcome,[12] it is of paramount importance that the clinician monitor for excessive emboli and take action in order to prevent neurological sequelae.

This report highlights the ability of cerebral oximetry to recognize a potential neurological catastrophic event in the adult cardiac surgical population. A similar report can be found in the pediatric literature. Yeh et al[13] used a multimonitoring approach to detect catastrophic neurological events. During the fenestration phase of a Fontan procedure an acute deterioration in the readings of transcranial Doppler, cerebral oximetry, and electroencephalography monitoring was detected. Corrective interventions were swiftly implemented. No neurological sequelae resulted from the air embolism.

Cerebral oximetry offers the perioperative physician a noninvasive monitoring tool to directly assess the oxygenation of the patient's brain. This case clearly demonstrates the ability of cerebral oximetry to provide information that might have gone unrecognized with conventional monitoring techniques. As a consequence the cause of the acute cerebral desaturation was searched for and corrective action swiftly implemented.

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