Jugular venous desaturation following cardiac surgery†

M. J. SOUTER, P. J. D. ANDREWS AND R. P. ALSTON

Summary
Twenty-two patients undergoing coronary artery bypass surgery underwent postoperative measurement and recording of jugular venous oxygen-haemoglobin saturation (S\textsubscript{\text{o}}\textsubscript{2}) with desaturation defined as a value of less than 50% for 5 min or longer. Fifteen of the 22 patients monitored experienced desaturations. An average of four episodes were experienced by those 15 patients (range 1–14). The mean duration of a single episode was 46 (range 5–212) min whilst mean total duration of desaturation was 175 (range 5–570) min. The mean duration of desaturation in the 15 patients, expressed as a percentage of monitored time, was 21.1% (range 0.58%–61.96%). S\textsubscript{\text{o}}\textsubscript{2} desaturation, possibly indicating cerebral hypoperfusion, occurs in the early post-operative period following cardiac surgery. (Br. J. Anaesth. 1998; 81: 239–241)

Keywords: surgery, cardiac; complications; equipment, continuous in vivo oximetry; monitoring, jugular bulb oxygen saturation

Brain damage, ranging from stroke to cognitive dysfunction, is an important complication of cardiac surgery.\textsuperscript{1,2} The epoch of injury has commonly been assumed to be the intra-operative period. However, cerebral oedema and vasoconstriction occur in the early postoperative period which suggests that cerebral injury could also be occurring at this time.\textsuperscript{4,5}

During cardiac surgery, jugular venous oxyhaemoglobin (S\textsubscript{\text{o}}\textsubscript{2}) desaturation has been noted during several phases of normothermic and hypothermic cardiopulmonary bypass (CPB).\textsuperscript{6–9} Most importantly, S\textsubscript{\text{o}}\textsubscript{2} desaturation during rewarming has been associated with cognitive dysfunction.\textsuperscript{10} We hypothesized that cerebral hypoperfusion may be occurring in the early postoperative period and that it may be identifiable by S\textsubscript{\text{o}}\textsubscript{2} monitoring. The aim of this study was to determine whether S\textsubscript{\text{o}}\textsubscript{2} desaturation occurs postoperatively following cardiac surgery and, if so, to quantify the magnitude of such events.

Methods and results
After obtaining local ethical committee approval, informed consent was obtained from 22 patients scheduled for elective coronary artery bypass surgery.

Anaesthesia and ICU: Premedication was with lorazepam 1–4 mg orally at 10 pm on the night before surgery, then temazepam 20–30 mg orally 1 h induction. Anaesthesia was induced with midazolam up to 0.2 mg kg\textsuperscript{-1}, fentanyl 5 μg kg\textsuperscript{-1} and etomidate as required to abolish the eyelash reflex. Neuroumuscular block was obtained using pancuronium 0.15 mg kg\textsuperscript{-1}. Anaesthesia was maintained with fentanyl 0.1 μg kg\textsuperscript{-1} min\textsuperscript{-1} and midazolam 0.5 μg kg\textsuperscript{-1} min\textsuperscript{-1}, supplemented with isoflurane. Ventilation was controlled mechanically, before and after CPB, to obtain normocapnia (4.5–5.3 kPa). Following surgery, patients were transferred to the ICU and postoperative care was managed to avoid haemodynamic instability and hypoxia.

Extubation was contingent upon normothermia; stable haemodynamics and absence of significant arrhythmias; the patient awake and moving all four limbs to command without residual neuromuscular block; mediastinal blood loss less than 100 ml h\textsuperscript{-1}; P\textsubscript{\text{a}}\textsubscript{\text{O}}\textsubscript{2} greater than 8 kPa with an F\textsubscript{\text{I}}\textsubscript{\text{O}}\textsubscript{2} of 40% and P\textsubscript{\text{C}}\textsubscript{\text{O}}\textsubscript{2} less than 8 kPa with a spontaneous rate of greater than 8 breaths per minute.

Cardiopulmonary bypass: The CPB circuit consisted of a membrane oxygenator and a non-pulsatile roller pump and was primed with lactated Ringer’s solution 2 litres, and sodium bicarbonate 50 mmol. Acid base management was according to alphastat principles. Non-pulsatile flow was used at a rate of 2.4 litres min\textsuperscript{-1} m\textsuperscript{-2}. Moderate hypothermia (nasopharyngeal temperature 28 °C) was used. Mean arterial pressure (MAP) was controlled between 50–90 mm Hg using boluses of methoxamine 2 mg or phentolamine 1 mg as required.

Jugular bulb oximetry: Immediately after induction of anaesthesia, the right jugular vein was cannulated retrogradely and a spectrophotometric catheter (Opticath, Abbott Laboratories, Maidenhead, Berkshire, UK) inserted to lie in the jugular bulb. Catheter position was confirmed radiologically. The catheter was connected to a Oximetrix 3 System (Abbott Laboratories, Maidenhead, Berkshire, UK). The reading obtained was then calibrated in vivo against jugular venous samples analysed by a Corning 270 Co-oximeter. After surgery, the patient

M. J. SOUTER\textsuperscript{*}, FRCA, University Department of Anaesthetics, Royal Infirmary of Edinburgh, P. J. D. ANDREWS, FRCA, MD, Department of Anaesthetics, Western General Hospital, Edinburgh. R. P. ALSTON, FRCA, MD, Department of Anaesthetics, Royal Infirmary of Edinburgh. Accepted for publication: February 23, 1998.

*Present address for correspondence: Institute of Neurological Sciences, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF.

was transferred into the ICU, and the \( S_{\text{O}_2} \) catheter recalibrated. Monitoring was terminated on the day after surgery when patients were transferred out of the ICU.

Statistical analysis: Cerebral hypoperfusion was defined as a \( S_{\text{O}_2} \) less than 50% for five consecutive minutes.\(^7\) Data analysis was by analysis of variance (ANOVA) and the Kruskal-Wallis test, using SPSS-PC (SPSS Inc., Michigan, USA) and EPI-INF o (Center for Disease Control, Atlanta, Georgia, USA).

Sixteen males and six females were studied, and an average of three grafts per patient (range 2–4) performed. The mean age was 59 years (95% CI: 56, 62) with a mean height of 169 cm (95% CI: 166, 173) and weight of 77 kg (95% CI: 72, 81), giving a mean calculated BSA of 1.87 (95% CI: 1.81, 1.94). The duration of aortic cross-clamping was 46 min (95% CI: 41, 50), and the duration of CPB was 81 min (95% CI: 74, 87).

Postoperative monitoring was used for a mean time of 1025 min (95% CI: 95, 1093). Fifteen of the 22 patients monitored experienced \( S_{\text{O}_2} \) desaturations over the postoperative monitoring period. In those 15 patients, (i) the mean duration of a single episode was 46 min (95% CI: 41, 50), and the duration of CPB was 81 min (95% CI: 74, 87).

Postoperative monitoring was used for a mean time of 1025 min (95% CI: 95, 1093). Fifteen of the 22 patients monitored experienced \( S_{\text{O}_2} \) desaturations over the postoperative monitoring period. In those 15 patients, (i) the mean duration of a single episode was 46 min (95% CI: 41, 50), and the duration of CPB was 81 min (95% CI: 74, 87).

Postoperative monitoring was used for a mean time of 1025 min (95% CI: 95, 1093). Fifteen of the 22 patients monitored experienced \( S_{\text{O}_2} \) desaturations over the postoperative monitoring period. In those 15 patients, (i) the mean duration of a single episode was 46 min (95% CI: 41, 50), and the duration of CPB was 81 min (95% CI: 74, 87).

Postoperative monitoring was used for a mean time of 1025 min (95% CI: 95, 1093). Fifteen of the 22 patients monitored experienced \( S_{\text{O}_2} \) desaturations over the postoperative monitoring period. In those 15 patients, (i) the mean duration of a single episode was 46 min (95% CI: 41, 50), and the duration of CPB was 81 min (95% CI: 74, 87).

For all 22 patients monitored, the mean duration of cerebral desaturation expressed as a percentage of total time monitored postoperatively was 21.1% (95% CI: 10.8, 31.4); (iv) an average of four episodes were suffered by those patients experiencing cerebral hypoperfusion (range 1–14).

For all 22 patients monitored, the mean duration of cerebral desaturation expressed as a percentage of monitoring time was 14.4% (95% CI: 6.3, 22.5) with a range of 0.58% to 61.96%. Figure 1 expresses total duration of desaturation in relation to total time of monitoring in each patient.

The mean of the highest \( S_{\text{O}_2} \) value recorded was 81% (range 72–90), and the mean of the lowest was 37% (range 12–52).

The mean time from the last calibration of the monitoring system to onset of the first desaturation was 181 min (95% CI: 9, 354 min). Fifty-one of the 65 episodes commenced within 12 h of transfer to ICU, whilst all desaturations had terminated by 18 h after transfer. Fifty had terminated within 12 h. The mean time of the last desaturation terminating was 666 min from transfer (95% CI: 483,849 min).

Ten patients received inotropes postoperatively. Twenty-one of the 22 patients were extubated within 24 h with a mean time from transfer to extubation of 636 min (95% CI: 525–788 min) One patient was ventilated for three days, and was the only patient reopened for postoperative bleeding (with no desaturations). Eight patients experienced \( S_{\text{O}_2} \) desaturations before extubation, with three after, and four both before and after extubation. There were no recorded episodes of systemic hypoxia, seizure or pyrexia. There was no evident association between the jugular desaturations and (i) either the administration and timing of analgesia or inotropes; (ii) the time of extubation, or (ii) the presence of hypotension as recorded on the nursing records.

Comment
The Oximetrix \( S_{\text{O}_2} \) monitoring system has been criticized because it exhibits calibration drift.\(^1\) However, it has a 12-h period of accurate recording, with 5% limits of agreement between catheter and co-oximeter measurements. Although we did not recalibrate at 12 h, it is unlikely that the desaturations in the present study are a result of drift as the vast majority of desaturations started and finished within 12 h of calibration.

The mechanism of the desaturations in \( S_{\text{O}_2} \) found in this study is unclear. Desaturation is the consequence of relative cerebral hypoperfusion. Cerebral oedema and vasoconstriction have been found to
occur in the early postoperative period following cardiac surgery and both could produce an imbalance between cerebral oxygen supply and demand. Derangement of systemic haemodynamics and oxygenation could be important, despite a seeming independence of desaturations from the haemodynamic and ventilatory events noted. However, because of the periodic nature of the nursing observations used, the possibility remains that suboptimal levels of systemic arterial pressure or oxygenation may have provoked the desaturations, as these parameters were not prospectively recorded, and transient insults may have gone undetected. Pyrexia or seizure induce hypermetabolism and increase oxygen consumption, but there was no evidence of either. Further investigation is required to accurately determine whether there is any association between $S_iO_2$ desaturations and systemic haemodynamics and oxygenation.

The duration of $S_iO_2$ desaturations in the present study by far exceed those recorded by Andrews and Colquhoun, the only study so far to chronicle the duration as well as the degree of desaturation intraoperatively. They noted a mean duration of 27 min when $S_iO_2$ was less than 50%, with a range of 4 to 51 min and a maximum desaturation to 19%. In the present study, the mean total postoperative duration of desaturation was 197 min (95% CI 106, 289) which is over six times longer. If these postoperative $S_iO_2$ desaturations do represent cerebral hypoperfusion then it may be that cerebral damage is occurring in the early postoperative period. Additionally, analogous to the established deleterious effect of secondary physiological insult upon head injuries, intraoperative cerebral damage may be being exacerbated by secondary hypoperfusion insults postoperatively. Although Croughwell and colleagues have found that low $S_iO_2$ during surgery is associated with decrements in cognition,

References