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ORIGINAL RESEARCH

Changes in cerebral oxygen saturation during transcatheter aortic valve replacement

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Abstract Cerebral oxygen saturation (rSO_2) is a non-invasive monitor used to monitor cerebral oxygen balance and perfusion. Decreases in rSO_2 >20 % from baseline have been associated with cerebral ischemia and increased perioperative morbidity. During transcatheter aortic valve replacement (TAVR), hemodynamic manipulation with ventricular pacing up to 180 beats per minute is necessary for valve deployment. The magnitude and duration of rSO_2 change during this manipulation is unclear. In this small case series, changes in rSO_2 in patients undergoing TAVR are investigated. Ten ASA IV patients undergoing TAVR with general anesthesia at a university hospital were prospectively observed. Cerebral oximetry values were analyzed at four points: pre-procedure (baseline), after tracheal intubation, during valve deployment, and at procedure end. Baseline rSO_2 values were 54.5 ± 6.9 %. After induction of general anesthesia, rSO_2 increased to a mean of 66.0 ± 6.7 %. During valve deployment, the mean rSO_2 decreased <20 % below baseline to 48.5 ± 13.4 %. In two patients, rSO_2 decreased >20 % of baseline. Cerebral oxygenation returned to post-induction values in all patients 13 ± 10 min after valve deployment. At procedure end, the mean rSO_2 was 67.6 ± 8.1 %. As expected, rapid ventricular pacing resulting in the desired decrease in cardiac output during valve deployment was associated with a significant decrease in rSO_2 compared to post-induction values. However, despite increased post-induction values in all patients, whether related to increased inspired oxygen fraction or reduced cerebral oxygen consumption under anesthesia, two patients experienced a significant decrease in rSO_2 compared to baseline. Recovery to baseline was not immediate, and took up to 20 min in three patients. Furthermore, baseline rSO_2 in this population was at the lower limit of the published normal range. Significant cerebral desaturation during valve deployment may potentially be limited by maximizing rSO_2 after anesthetic induction. Future studies should attempt to correlate recovery in rSO_2 with recovery of hemodynamics and cardiac function, provide detailed neurological assessments pre and post procedure, determine the most effective method of maximizing rSO_2 prior to hemodynamic manipulation, and provide the most rapid method of recovery of rSO_2 following valve deployment.

Keywords Transcatheter aortic valve replacement · Cerebral oxygen saturation · Rapid ventricular pacing · Aortic stenosis

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1 Introduction

Cerebral oxygen saturation (rSO_2) is used in traditional cardiac surgery as a continuous monitor of cerebral oxygen balance and brain perfusion [1–3]. Declines in rSO_2 are associated with increased neurologic complications (stroke,

postoperative cognitive decline) and prolonged length of ICU and hospital stay [1, 4–7]. Published normal baseline values for rSO_2 in adults ranges between 55 and 78 % [8]. A decline from baseline of ≥20 % is considered significant and is a threshold for intervention to decrease the risk of cerebral ischemia [5, 9].

Symptomatic aortic stenosis is associated with a high mortality rate if left untreated [10]. Surgical correction dramatically improves survival, but traditional open techniques to replace the aortic valve involve significant perioperative risk, morbidity, and mortality. Transcatheter aortic valve replacement (TAVR) has been established as an alternative when patients are not candidates for surgery [11, 12]. During TAVR, patients undergo intense hemodynamic manipulation, including rapid ventricular pacing at up to 180 beats per minute, to decrease cardiac output and forward flow of blood across the valve during deployment. This minimizes heart translocation during valvuloplasty and valve deployment, facilitating accurate prosthesis placement, but creating an extremely low cardiac output state with inherent risk of cerebral and systemic hypoperfusion.

The risks and complications of TAVR are distinct from those associated with open surgery, including vascular access complications, valve embolization, conversion to open surgery, neurologic and myocardial ischemia, delayed return of spontaneous circulation, perivalvular aortic regurgitation, aortic dissection, obstruction of coronary ostia, and conduction abnormalities [11–14].

This observational case series was performed to determine the duration and severity of changes in rSO_2 during TAVR in patients under general anesthesia. Changes in rSO_2 were recorded during rapid ventricular pacing and valve deployment, and compared with values obtained at baseline and post-induction. An observation of perioperative complications was also made.

2 Materials and methods

After institutional review board approval and informed consent, all patients undergoing TAVR from March 2011 to July 2011 were enrolled. Ten patients were enrolled (6 male and 4 female, ranging from 77 to 94 years of age), with severe to critical aortic stenosis. Valve areas ranged from 0.4 to 0.8 cm^2. All patients had previously been denied open surgical replacement of the aortic valve and were to receive an Edwards Sapien transcatheter aortic valve (Edwards Life Sciences Corp, Irvine, CA). Patient demographic data and baseline characteristics are shown in Table 1. Although four patients had a history of cerebrovascular disease, none had baseline neurologic impairment prior to the procedure. This was a 10-patient pilot study and the protocol did not require standardization of anesthetic techniques. However, in every patient, general anesthesia was induced with etomidate and fentanyl after pre-oxygenation with 100 % oxygen for at least 5 min. Endotracheal intubation was facilitated with muscle relaxant of choice. Anesthesia was maintained with sevoflurane, 100 % inspired oxygen (as is usual practice at our institution), fentanyl, and muscle relaxant. Cerebral oxygen saturation was monitored continuously, and values at procedural milestones underwent statistical analysis. "Baseline" values were obtained prior to anesthesia induction and prior to administration of oxygen. "Post-induction" was the period immediately after tracheal intubation during ventilation with 100 % oxygen. The nadir value for rSO_2 during rapid ventricular pacing was used as the "pacing and valve deployment" analytic value. "End" was the period upon conclusion of the procedure, prior to tracheal extubation.

Table 1 Patient demographic data

Demographic	Mean (SD)
Age (years)	87 ± 6 (77–94)
Aortic valve area (cm^2)	0.6 ± 0.1 (0.4–0.8)
Left ventricular ejection fraction (%)	59 ± 10 (50–70)
Male/female	6/4
NYHA class III–IV	8
Coronary artery disease	9
Previous CABG	6
Previous PCI	2
Arterial hypertension	8
Previous aortic valvuloplasty	6
Diabetes mellitus	5
Chronic pulmonary disease	3
Chronic renal insufficiency	3
Cerebrovascular disease	4

NYHA New York Heart Association, *CABG* coronary artery bypass grafting, *PCI* percutaneous coronary intervention

Cerebral oxygen saturation (rSO_2) was measured continuously via INVOS 5100c surface pads (Covidien, Mansfield, MA) placed bilaterally on the forehead. The electrode pads were positioned according to the manufacturer's instructions. Left and right-sided rSO_2 values were similar in all patients and averaged for data analysis. Throughout each procedure, values for rSO_2 were blinded to the anesthesiologist. All other parameters, including electrocardiography, invasive arterial blood pressure, pulmonary arterial pressure, pulse oximetry were monitored by the anesthesia provider. Perioperative transesophageal echocardiography (TEE) was performed and monitored by the anesthesiologist.

2.1 Statistical analysis

Descriptive statistics were reported as mean and standard deviation for continuous variables. Further statistical analysis is not included given the small number of patients involved and the nature of the study.

3 Results

The mean age of our small patient cohort was 87 years. Baseline rSO_2 prior to induction was 54.5 ± 6.9 %. After induction of anesthesia, rSO_2 increased above baseline values in all patients, to 66.0 ± 6.7 %. All patients experienced a decline in rSO_2 during rapid ventricular pacing. Cerebral oxygen saturation during rapid ventricular pacing and valve deployment was 48.5 ± 13.4 %. Eight patients experienced a ≥20 % decline compared to post-induction values, and two patients within this group showed a ≥20 % decline compared to baseline values. One patient was found to have a left atrial appendage thrombus on intra-operative TEE, and underwent an aortic valvuloplasty without TAVR.

Return to baseline rSO_2 values was seen after 6 ± 7 min (range 0–20 min) following cessation of rapid ventricular pacing and successful valve deployment. Cerebral oxygenation returned to post-induction values after 13 ± 10 min (range 1–22 min). End of procedure rSO_2 was 67.6 ± 8.1 %. Figure 1 shows the percent changes in rSO_2 at the specific procedural milestones.

Perioperative complications are detailed in Table 2. Individual patient complication rates varied widely, including respiratory complications in six patients, acute renal failure in four patients, and cardiovascular complications in two. Multi-organ failure and death occurred in one patient prior to hospital discharge. No patients were noted to have symptomatic neurologic complications prior to hospital discharge. Blood transfusion was required in three patients due to decreased hematocrit and blood loss during procedure, permanent pacemaker insertion for complete heart block in one patient, and intra-aortic balloon pump insertion and chest compressions in one patient. All patients were extubated at the end of the procedure.

4 Discussion

This small case series shows that during rapid ventricular pacing in patients with severe to critical aortic stenosis undergoing TAVR, a potentially profound but apparently reversible decline in cerebral oxygen saturation is seen as monitored by rSO_2. Recovery to baseline rSO_2 values may not be immediate, and in our series took up to 20 min. The clinical impact of this cerebral desaturation is unclear from our current analysis.

Baseline rSO_2 values were in the low-normal range in this patient group (54.5 ± 6.9 %, compared to published normal range of 55–78 %) [8]. This indicates a decreased cardiac and neurological reserve, as may be expected in this population [6, 15]. Induction of general anesthesia resulted in an increase in rSO_2 above baseline in all patients. This is consistent with previously published literature, and can be explained by an improvement in cerebral oxygen supply and decreased oxygen demand under general anesthesia [2]. The increase in rSO_2 following induction of general anesthesia may be particularly valuable to patients undergoing TAVR. By favorably altering the cerebral oxygen balance, these patients with low-normal baseline rSO_2 may be protected from a decline in cerebral oxygenation below baseline during rapid ventricular pacing. We observed this pattern in our patients, where the rSO_2 declined ≥20 % below post-induction values in eight patients, but due to the post-induction increase in rSO_2, only two patients experienced a decline ≥20 % below baseline values.

These observations suggest that in patients undergoing TAVR, consideration of a treatment protocol based on

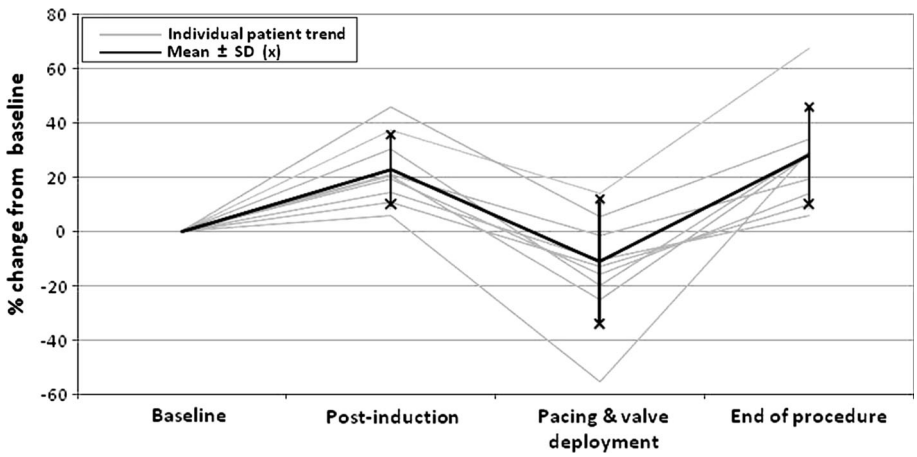

Fig. 1 Changes in cerebral oxygenation measured by cerebral oximetry during TAVI

Table 2 Perioperative complications

Complication	N (%)
Acute renal failure	4 (44)
Respiratory complications (pleural effusion, pulmonary edema, pneumonia, respiratory failure)	6 (66)
Procedure abandoned (atrial thrombus)	1 (11)
Paravalvular leak	1 (11)
Atrial fibrillation	1 (11)
Multi-organ failure	1 (11)
Death	1 (11)

maximizing initial rSO_2 and minimizing declines in rSO_2 during rapid ventricular pacing may be warranted. This protocol should focus on four specific goals known to improve rSO_2: increasing cerebral blood flow and perfusion pressure, decreasing cerebral metabolic rate, increasing the oxygen content of blood, and increasing cardiac output. Maneuvers that effectively influence these goals include deepening general anesthesia, increasing $PaCO_2$, inotropic and vasopressor infusions when indicated, intra-aortic balloon pump support, adjusting head position, and blood transfusion (where indicated) [2]. Choice of vasopressor has also been shown to have an impact on cerebral blood flow and therefore may have an impact on cerebral oxygen saturation (norepinephrine improves rSO_2 better than phenylephrine) [5].

4.1 Limitations

This study was limited primarily by a small sample size and its observational nature. While a post-induction increase in rSO_2 seems to be protective from excessive desaturation during pacing and valve deployment, this conclusion cannot be drawn from such a small series of patients. The duration of cerebral desaturation following rapid ventricular pacing and valve deployment appears variable, but it is not clear if longer durations are associated with worsened outcomes. Although the specific anesthetic technique was not standardized for the study, medication selection for general anesthesia was similar enough to allow for comparison of the oximetry results. All patients were induced with etomidate, and maintained on muscle relaxant, fentanyl, sevoflurane and 100 % oxygen. In addition, measurements of PaO_2, hemoglobin, cardiac output and $PaCO_2$ at the time of rSO_2 readings were not noted during this study. These variables cannot be ignored and would be useful information in future studies.

5 Conclusion

This small prospective case series demonstrates that in patients undergoing TAVR, there is a decline in cerebral oxygen saturation measured by rSO_2 during rapid ventricular pacing and valve deployment. Significant decline in rSO_2 appears to be limited by the increase seen after induction of general anesthesia. Following cessation of rapid ventricular pacing and successful valve deployment, rSO_2 increases with variable delays. Further large, randomized, prospective studies are warranted to investigate the ability of rSO_2-directed therapy to decrease morbidity and mortality of patients undergoing TAVR, specifically looking at correlation of recovery in rSO_2 with changes in hemodynamics, cardiac function by echocardiography and cardiac output calculation, as well as analysis of pre versus post-procedure complications including a detailed neurological function.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

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