CASE REPORT

Can entropy predict neurologic complications after cardiac surgery?

Mohamed R. El Tahan

Department of Anaesthesia and Surgical ICU, College of Medicine, University of Dammam, Al Khubar, Saudi Arabia

Address for correspondence:
Dr. Mohamed R. El Tahan, Department of Anaesthesia and Surgical ICU, College of Medicine, University of Dammam, P.O. 40081, Al Khubar, Saudi Arabia.
E-mail: mohamedrefaateltahan@yahoo.com

ABSTRACT

Electroencephalography can detect both cerebral ischemia/hypoxia and seizures and can measure hypnotic effects. The author reported two patients with left main coronary artery disease and myocardial infarction scheduled for urgent coronary artery bypass grafting surgery; they developed abrupt decreases in response entropy (RE) and state entropy (SE) values to isoelectric silence during target-controlled propofol-sufentanil anesthesia. After that, low RE and SE values persisted throughout the intraoperative period. Both patients showed delayed awakening after surgery and brain CT revealed nonhemorrhagic tempro-parietal cerebral infarctions. Intraoperative entropy-based monitoring could predict poor neurological outcome after cardiac surgery during target-controlled propofol and sufentanil anesthesia.

Key words: *Cardiac surgery, entropy, neurologic outcome, stroke*

INTRODUCTION

Cardiac-surgery-associated neurological complications are common, multifactorial, and often preventable if they are detected early. Entropy monitoring permits more controllable and predictable anesthesia to be achieved and reduces anesthetic dosages in patients undergoing cardiac surgery.[1,2]

Response entropy (RE) and state entropy (SE) may predict the patients with good neurologic outcomes during the first 24 h after out-of-hospital cardiac arrest (P=0.011 and P=0.008, respectively).[3] Hence, this advantage may be extended to predict the patients with worse neurologic sequels during cardiac surgery.

CASE REPORTS

Case 1

A 59-year-old, 74 kg, 165 cm, female was presented with a non-ST segment elevation myocardial infarction. Preoperative echocardiography findings included an ejection fraction (EF) of 50%, tight aortic valve stenosis, peak systolic gradient across the aortic valve of 45 mm Hg, trivial aortic regurgitation, no detected wall motions abnormalities, and normal right ventricular (RV) diameters. Cardiac catheterization showed two-vessel disease and fair left ventricular (LV) function. She was treated with clopidogrel, aspirin, captopril, bisoprolol, and intravenous infusions of heparin and nitroglycerin. She underwent an urgent bypass grafting for the left-anterior descending (LAD), diagonal and right coronary arteries (RCA), and prosthetic replacement of the aortic valve under the standardized target-controlled (TCI) propofol-sufentanil anesthesia and cisatracurium infusion.[4] After heparinization, aortic cannulation was performed. It resulted in an abrupt decreases of RE and SE values from 49 and 46, respectively, to isoelectric silence. Transoesophageal echocardiogram (TOE) failed to detect aortic intraluminal thrombi and plaques. Target effect site concentrations (Ce) of propofol and sufentanil were lowered to 1 and 0.1 μg/mL, respectively, and continued till the end of surgery. Mild hypothermic cardiopulmonary bypass (CPB) was used. The cross-clamp time was 148 min and the CPB time was 289 min.

Isoelectric silence persisted till the end of reperfusion time. Then, gradual increases in RE and SE toward the low 30 s levels were noted and they persisted till the transfer of the patient to the intensive care unit (ICU).

The patient was subsequently successfully separated from CPB with intravenous epinephrine (7 μg/min).

Access this article online	
Quick Response Code:	Website: www.saudija.org
	DOI: 10.4103/1658-354X.105899

The cisatracurium, sufentanil, and propofol infusions were discontinued after surgical homeostasis, sternal closure, and skin closure, respectively.[4]

Mean arterial and central venous blood pressures (MAP and CVP, respectively), arterial oxygen saturation (SaO_2), normocapnia, and euglycemia were well maintained throughout the perioperative period. Rectal temperature >36°C was maintained before and after the CPB.

Postoperative ventilation was performed with synchronized intermittent mandatory (SIMV). Mediastinal bleeding of 750 mL during the first three postoperative hours was treated with transfusion of two U-packed red blood cells, five U platelets, and three U fresh frozen plasma. Unfortunately, the lady did not wake up even 8 h after the discontinuation of anesthetics. After that, she showed agitation and left-sided paresis. Brain CT showed a nonhemorrhagic right tempro-parietal infarction. Dexmedetomidine infusion was used to alleviate her agitation. Her consciousness was recovered and her trachea was extubated on the second postoperative day.

Case 2

A 65-year-old, 85 kg, 152 cm, male was presented with a ST segment elevation myocardial infarction. Preoperative echocardiography showed an EF of 25%, and LV akinetic apex and anterolateral wall and dyskinetic of the inferior wall. Cardiac catheterization showed 95% occlusions of both RCA and left main coronary artery with severely impaired LV function. Circulatory support was achieved with intra-aortic balloon pump counter-pulsations (IABP), dobutamine, and nitroglycerin. Heparin infusion was going on.

He underwent an emergent bypass grafting for the LAD, diagonal and RCA under the standardized TCI propofol-sufentanil anesthesia, and cisatracurium infusion. After heparinization, mild hypothermic CPB was established. An abrupt decreases of RE and SE values from 52 and 48, respectively, to isoelectric silence was noted. Propofol Ce and sufentanil Ce were lowered to 1 and 0.1 μg/mL, respectively, till the end of surgery. This was followed with gradual increases in RE and SE toward the low 10 s that persisted till the end of surgery.

The cross-clamp time was 120 min and the CPB time was 240 min. The patient was subsequently successfully separated from CPB with IABP and intravenous infusions of epinephrine (18 μg/min), dobutamine (5 μg/kg/min), and nitroglycerin (1 μg/kg/min). The cisatracurium, sufentanil, and propofol infusions were discontinued.

Stable MAP, CVP, and SaO_2, normocapnia, and euglycemia were maintained throughout the perioperative period. Normothermia (rectal temperature >36°C) was maintained before and after bypass.

The patient was shifted to the ICU ventilated with SIMV. He did not wake up even seven days after surgery. His Glasgow Coma Scale (GCS) was 4/15. Brain CT showed an extensive nonhemorrhagic left tempro-parietal infarction. His postoperative course was complicated with vasodilatory shock, renal failure, and sepsis. The patient passed away on the seventh postoperative day.

DISCUSSION

Neurologic injury after CPB remains a devastating complication of cardiac surgery. Possible causative factors include hypoperfusion, lack of pulsatile flow, and cerebral embolization of gaseous or particulate.[5]

Bispectral index (BIS), a monitor for the depth of anesthesia, is a very early predictor to identify patients with a good chance of recovery during therapeutic hypothermia after cardiac arrest.[6]

Unfortunately, monitoring of brain electrical activity, blood flow velocity, and oxygenation during cardiac surgery is not standardized yet among the worldwide centers.[7]

RE and SE are comparable with the BIS but showed superior resistance against artifacts. Thus, SE is more suitable than the BIS during propofol-remifentanil anesthesia in cardiac surgery patients.[8]

We documented isoelectric entropy silences in two cases with left main coronary artery diseases during emergent CABG surgery. After bypass, RE and SE values increased gradually to the 10 to 30 s levels and persisted till the end of surgery. These changes were correlated to postoperative delayed awakening and documented cerebral ischemic infarctions.

Similarly, others reported remarkable reduction in BIS value from 45 to 5 after injection of air to confirm a resected tumor lesion during video-assisted thoracoscopic segmentectomy under TCI propofol anesthesia. Brain CT and MRI confirmed cerebral air embolism.[9]

In the present case series, the noted abrupt decreases in RE and SE values during CABG surgery may be attributed to the confounding effects of anesthetics, hypothermia, hypotension, changes in arterial carbon dioxide tension ($PaCO_2$) and roller-pump artifacts.[10] Fortunately, these problems are less likely to be the causative for the reductions in entropy variables as a fast-track cardiac anesthesia protocol was adopted,[4] mild hypothermic bypass

was used, and stable hemodynamics and normocapnia were maintained throughout the intraoperative period. To show the evidence of cerebral injury, we documented postoperative cerebral infarctions in the brain CT scanning.

It is unlikely that either of the patients had neurological injury secondary to hypoglycaemia, hyperglycaemia, or cerebral hypoperfusion, hyperthermia or deoxygenation, as these variables were well maintained throughout the intraoperative course. Unfortunately, cerebral oxygenation was not monitored during the perioperative management of the presented two patients.

Further studies are needed to test the sensitivity and specificity of entropy, especially if it is combined with near infrared cerebral oximetry to predict the neurological outcome after cardiac surgery.

In conclusion, intraoperative entropy-based monitoring could predict poor neurological outcome after cardiac surgery during target-controlled propofol and sufentanil anesthesia.

REFERENCES

1. Arutiunian OM, Iavorovskiĭ AG, Guleshov VA, Dutikova EF, Buniatian AA. Role of entropy-based neuromonitoring during cardiac surgery.Anesteziol Reanimatol 2010;5:78-82.
2. Jiahai M, Xueyan W, Yonggang X, Jianhong Y, Qunhui H, Zhi L, *et al*. Spectral entropy monitoring reduces anesthetic dosage for patients undergoing off-pump coronary artery bypass graft surgery. J Cardiothorac Vasc Anesth 2012;26:818-21.
3. Wennervirta JE, Ermes MJ, Tiainen SM, Salmi TK, Hynninen MS, Särkelä MO, *et al*. Hypothermia-treated cardiac arrest patients with good neurological outcome differ early in quantitative variables of EEG suppression and epileptiform activity. Crit Care Med 2009;37:2427-35.
4. El Tahan MR, Khidr AM. Low Target Sufentanil Effect-Site Concentrations Allow Early Extubation After Valve Surgery. J Cardiothorac Vasc Anesth 2012.
5. Kilo J, Dzerny M, Gorlitzer M, Zimpfer D, Baumer H, Wolner E, *et al*. Cardiopulmonary bypass affects cognitive brain function after coronary artery bypass grafting. Ann Thorac Surg 2001;72:1926-32.
6. Seder DB, Fraser GL, Robbins T, Libby L, Riker RR. The bispectral index and suppression ratio are very early predictors of neurological outcome during therapeutic hypothermia after cardiac arrest. Intensive Care Med 2010;36:281-8.
7. Erdös G, Tzanova I, Schirmer U, Ender J. Neuromonitoring and neuroprotection in cardiac anaesthesia. Nationwide survey conducted by the Cardiac Anaesthesia Working Group of the German Society of Anaesthesiology and Intensive Care Medicine. Anaesthesist 2009;58:247-58.
8. Baulig W, Seifert B, Schmid ER, Schwarz U. Comparison of spectral entropy and bispectral index electroencephalography in coronary artery bypass graft surgery. J Cardiothorac Vasc Anesth 2010;24:544-9.
9. Kiribayashi M, Nakasone M, Moriyama N, Mochida S, Yamasaki K, Minami Y, *et al*. Multiple cerebral infarction by air embolism associated with remarkable low BIS value during lung segmentectomy with video assisted thoracic surgery (VATS) technique: A case report. Masui 2010;59:480-3.
10. Levy WJ. Monitoring of the electroencephalogram during cardiopulmonary bypass. Know when to say when. Anesthesiology 1992;76:876-7.

How to cite this article: El Tahan MR. Can entropy predict neurologic complications after cardiac surgery?. Saudi J Anaesth 2012;6:426-8.

Source of Support: Nil, **Conflict of Interest:** None declared.