Patient management algorithm combining processed electroencephalographic monitoring with cerebral and somatic near-infrared spectroscopy: a case series

Algorithme de gestion du patient combinant le monitoring de l'électroencéphalogramme traité et la spectroscopie cérébrale et somatique dans le proche infra rouge : une série de cas

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Abstract

Purpose Cerebral oximetry is a monitoring tool used in the perioperative care of cardiac surgery patients to ensure adequate cerebral perfusion and oxygenation. When combined with somatic oximetry, the differential diagnosis of cerebral desaturation can be better identified and managed more specifically, as somatic oximetry serves as a global or localized perfusion monitor (depending on its regional position). The use of processed electroencephalography (pEEG) in cardiac surgery could further guide the management of desaturation episodes, as reductions in pEEG activity without a change in the anesthetic agent level indicate potential cerebral ischemia. Continuous integration of multiple monitoring modalities are thus desirable to assess organ perfusion and organ function.

Clinical features Four clinical cases are presented in which the combination of pEEG and cerebro-somatic

oximetry assisted with understanding the mechanism of cerebral desaturation encountered during cardiac surgery.

Conclusion Integrating combinations of different monitoring modalities such as cerebral and somatic oximetry with pEEG can help the diagnosis and treatment of organ malperfusion and related dysfunction.

Résumé

Objectif L'oxymétrie cérébrale est un outil de monitoring utilisé dans les soins périopératoires des patients de chirurgie cardiaque pour s'assurer que leur cerveau est adéquatement perfusé et oxygéné. Quand on la combine à l'oxymétrie somatique, le diagnostic différentiel de désaturation cérébrale peut être mieux identifié et géré de manière plus spécifique, car l'oxymétrie somatique permet un suivi de la perfusion globale ou localisée (selon l'emplacement du capteur). L'utilisation de l'électroencéphalographie traitée (pEEG) en chirurgie cardiaque pourrait entraîner une meilleure gestion des épisodes de désaturation dans la mesure ou une baisse de l'activité pEEG sans modification du niveau de l'agent anesthésique indique une ischémie cérébrale potentielle. L'intégration continue de multiples modalités de monitoring est donc souhaitable pour évaluer la perfusion et le fonctionnement des organes.

Caractéristiques cliniques Quatre cas cliniques sont présentés dans lesquels la combinaison de la pEEG et de l'oxymétrie cérébro-somatique a aidé à comprendre le mécanisme de désaturation cérébrale rencontrée au cours de la chirurgie cardiaque.

Conclusion Les combinaisons intégrant différentes modalités de monitoring, telles que l'oxymétrie cérébrale

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et somatique avec la pEEG, peuvent contribuer au diagnostic et au traitement des troubles de la perfusion des organes et des dysfonctionnements qui en découlent.

Cerebral oximetry is a non-invasive monitoring modality based on several physical principles that allow it to act as an indirect continuous indicator of oxygen supply-vs-consumption balance. As a monitor of cerebral perfusion adequacy, it has numerous clinical and research-related applications. Our group has previously described a clinical management algorithm using cerebral near-infrared spectroscopy (NIRS),¹⁻⁴ and a new algorithm combining both cerebral and somatic NIRS monitoring has also been proposed.⁵⁻⁷ Nevertheless, the use of processed electroencephalography (pEEG) has not been formally integrated into a NIRS cerebral desaturation management algorithm. The use of pEEG, particularly in the elderly population, was recently introduced as part of the clinical care at our institution, consistent with recent European guideline recommendations.⁸ In this report, we describe how the combination of cerebro-somatic NIRS and pEEG monitoring can be used to manage several patients undergoing cardiac surgery.

Case descriptions

Written consent was obtained from all the patients presented in this case series. The first patient was a 73-yr-old male with a EuroSCORE II⁹ of 2.8. The patient was scheduled for an elective aortic valve replacement. His preoperative medication included an angiotensin converting enzyme inhibitor and a calcium channel blocker. He had no other significant co-morbidities. His left and right systolic ventricular function were both normal and there was only mild left ventricular diastolic dysfunction presenting as a relaxation abnormality. In addition to standard monitors,¹⁰ we used a pulmonary artery catheter, radial and femoral arterial catheters,¹¹ O3™ Regional Oximetry with SedLine (Masimo Corporation, Irvine, CA, USA), and transcranial Doppler (TCD) (ST3, Spencer Technology, Seattle, WA, USA) using a previously reported localization technique.¹²

Figure 1 summarizes the pEEG and NIRS changes that occurred during the case. In Fig. 1A, the awake state was associated with an elevated patient state index (PSI) and the percentage of the pEEG signal representing muscular activity was also high due to initially elevated electromyography (EMG) values. An expected decrease in these two parameters was observed after induction of

anesthesia and the onset of neuromuscular blockade. A gradual increase was also noted in the bilateral cerebral NIRS signals due to the reduction in cerebral metabolic rate of oxygen (CMRO₂) induced by the anesthesia agents used, including propofol, fentanyl, and isoflurane.

At time point #1 in Fig. 1A, there was an increase in PSI, which coincided with an increase in the percentage of the pEEG signal resulting from EMG and the increased artifact signal. No significant changes were noted in the NIRS signal, which continued to rise and then plateaued. At this point, it was felt that the increase in PSI was mostly related to an EMG artifact related to surgical manipulation. No intervention was performed, and the value spontaneously returned to a target range (PSI between 25–50, or spectral edge frequency < 15 Hz). At time point #2 in Fig. 1A, another increase in PSI was observed. Nevertheless, it was not associated with any artifact or significant EMG signal. At the same time, a reduction in the cerebral NIRS signal was observed suggesting that the CMRO₂ increased because of possible awakening. The anesthesia agent was thus increased, resulting in a decreased PSI along with a gradual increase in the cerebral regional oxygen saturation. At time point #3 in Fig. 1A, which occurred during weaning from cardiopulmonary bypass (CPB), there was a significant reduction in PSI that was associated with a simultaneous reduction in cerebral NIRS values. These reductions were thought to be due to a reduction in cerebral blood flow (CBF) resulting from an episode of hypotension shown in Fig. 1B (i.e., on the hemodynamic monitor). The hypotension was mostly likely the result of transient right ventricular dysfunction secondary to air embolism (with transient myocardial ischemia) diagnosed with transesophageal echocardiography (TEE) and confirmed using TCD. These air emboli can be seen in Fig. 1C, where the numerous white arrows indicate high intensity transient signals (HITS) detected using TCD at the end of CPB. In our prior experience, when HITS are detected, there is a significant risk of air emboli in the right coronary artery and resultant right ventricular dysfunction. In patient #1, hemodynamic support consisted of maintaining adequate perfusion pressure using noradrenaline. The patient completely recovered and was transferred without vasopressors.

The combined reduction in PSI and cerebral saturation is also shown in patient #2, a 49-yr-old male with a EuroSCORE II of 3.4 undergoing a Ross procedure. As illustrated in Fig. 2A, hypotension (mean arterial pressure = 48 mmHg) during CPB was associated with a PSI reduction. A transient but significant decrease in the spectral edge frequency (SEF) combined with an increase in the blue color displayed on the density spectral array (DSA) indicated a reduction in the high pEEG frequencies

Fig. 1 A) Processed electroencephalography (pEEG) using SedLine and O3™ Regional Oximeter System (O3™ System) (Masimo Corporation, Irvine, CA, USA) in a patient undergoing aortic valve surgery (patient #1). At point #1, there is an increase in patient state index (PSI) associated with an increase in the electromyography artifact (ARTF) signal, and minimal change in regional oxygen saturation (rSO₂). At point #2, the increase in PSI is associated with a reciprocal reduction in rSO₂; additional anesthetic drugs were subsequently administered. At point #3, a reduction in PSI is associated with a reduction in rSO₂ resulting from hypotension, as shown in panel B. In panel C, hypotension is associated with a new onset of high intensity transient signals (HITS) (white arrows) seen by

transcranial Doppler (TCD) monitoring. These changes represent cerebral microemboli that likely also entered the right coronary artery resulting in right ventricular ischemia and dysfunction, which triggered hypotension and secondary reductions in the PSI and rSO₂. EDV = TCD end-diastolic velocity; ETco₂ = end-tidal carbon dioxide; MV = TCD mean velocity; PDV = TCD peak diastolic velocity; Pfa = femoral arterial pressure; PI = TCD pulsatility index; Ppa = pulmonary artery pressure; Prad = radial artery pressure; PSV = TCD peak systolic velocity; SaO₂ = oxygen saturation; SEFL = spectral edge frequency on the left; SEFR = spectral edge frequency on the right; SR = suppression ratio

followed by a brief period of bilateral burst-suppression (black horizontal line). In Fig. 2B, the PSI reduction was associated with a simultaneous reduction in bilateral cerebral NIRS values, which were secondary to transient reduction in blood pressure combined with hemodilution upon initiation of CPB. The postoperative course was uneventful.

Figure 3 is from patient #3, a 76-yr-old female with a EuroSCORE II of 9.7 who was scheduled for reoperation for coronary revascularization and aortic valve replacement. Reduction in cerebral saturation values were observed both upon initiation of and during CPB. A concomitant burst-suppression signal was seen on the pEEG and is shown in Fig. 3B (white arrow). It resolved by increasing the blood pressure and the CPB flow with

resulting improvement in CBF. Towards the end of the procedure, bilateral cerebral desaturation occurred that was a similar magnitude to that during CPB. Nevertheless, as shown in Fig. 3C, desaturation was not associated with a decrease in PSI but was most likely due to a reduction in the depth of anesthesia. It resolved following administration of a small bolus of propofol and fentanyl. The DSA and the SEF reduced following drug administration, which can be seen in Fig. 3C (white arrows). The patient did well postoperatively with no complications.

Patient #4 was a 33-yr-old woman with dilated cardiomyopathy undergoing cardiac transplantation. This case illustrates the integration of somatic oximetry in our proposed algorithm (Fig. 4). The patient was monitored

Fig. 2 A) Processed electroencephalography (pEEG) monitoring using two bilateral raw EEG channels, patient state index (PSI), density spectral array (DSA), and regional oxygen saturation (rSO₂) data in a patient with intraoperative hypotension (arrows) (patient #2).

B) Same patient with the graphical display of both PSI and rSO₂. Note the simultaneous significant reduction in the PSI associated with a reduction in DSA on Fig. 2A and in bilateral cerebral oximetry in Fig. 2B. AUC = area under the curve

with both cerebral and somatic oximetry in addition to TCD. After CPB, the TCD pulsatility index (PI) was 0.70 on the right side and 0.78 on the left side (Fig. 4A) and it increased to 1.63 on the right side and 2.05 on the left side (Fig. 4B) during an episode of hypotension without any significant change in the peak systolic velocities. This episode of hypotension was secondary to a superior vena cava (SVC) anastomotic stenosis characterized by a 22 mmHg pressure gradient between the SVC and right atrium that was diagnosed using a pulmonary artery catheter (Fig. 4C). During this episode, the PSI decreased with bilateral cerebral desaturation (Fig. 4D). Somatic oximetry monitoring demonstrated a desaturation limited only to the upper extremity (Fig. 4E white arrow) without any changes in the lower extremity, thus confirming that the venous drainage of the brain and upper body (and hence upper extremity) was compromised due to this SVC syndrome.^{1,5} In this particular situation, a reduction in diastolic arterial blood flow velocities secondary to the increase in cerebral venous pressure resulted in a reduction of the PI.^{13,14} These changes are similar to those seen in situations associated with intracranial hypertension.¹⁵ The anastomotic stenosis was thought to be secondary to edema of the surrounding tissues. The patient was observed and remained asymptomatic without any neurologic deficit postoperatively.

Discussion

In this case series outlining the combined use of cerebral and somatic oximetry with additional pEEG, we report a patient monitoring algorithm that integrates both modalities into clinical decision making. First, if both cerebral saturation and the pEEG signal decrease (with reduction of PSI, SEF, or reduction of high frequency pEEG in the DSA), then a reduction in CBF, which can be associated with either reduced oxygen transport (Fig. 1A point #3, or Fig. 2, or Fig. 3B) or in some cases venous congestion (Fig. 4) should be suspected. The next question is whether this reduction is from localized reduced brain perfusion or is secondary to a reduction in cardiac output, acute anemia, or hypoxemia.

If the changes in either pEEG monitoring or cerebral saturation are unilateral, and the peripheral or somatic NIRS saturation is normal, localized cerebral hypoperfusion should be suspected. Furthermore, cerebral investigation should be pursued, such as a carotid, jugular, and brain ultrasound, TCD,¹⁵ optic nerve sheath measurement,^{16,17} and/or computed tomography. On the other hand, if the cerebral saturation reduction is bilateral and symmetrical and the somatic NIRS saturation is also reduced, then a non-cerebral etiology such as cardiogenic shock, hemorrhagic shock, hypoxemia or obstruction to adequate central venous drainage (superior or inferior vena cava) is more likely, as previously reported.^{5,6} The specific location of the somatic oximetry probes allows a more

Fig. 3 A) Near-infrared spectroscopy (NIRS) signals in a 76-yr-old woman with two specific and symmetrical episodes of cerebral desaturation (patient #3). B) Processed electroencephalography (pEEG) monitoring using two bilateral raw EEG channels, patient state index (PSI), and density spectral array (DSA). Note the presence of burst-suppression associated with reduce NIRS signals (arrow). C) The same patient with the graphical display of both PSI and DSA during the second episode of brain desaturation. Note the increase in

the PSI followed by its decrease following deepening of anesthesia (arrows). ARTF = artifact; AUC = area under the curve; CÉRÉ G = cerebral left NIRS value; CÉRÉ D = cerebral right NIRS value; CPB = cardiopulmonary bypass; EMG = electromyography; SEFL = spectral edge frequency left; SEFR = spectral edge frequency right; SR = suppression ratio; RF = reference range; rSO₂ = regional oxygen saturation

regional assessment of local oxygen extraction and perfusion compared with blood gas oxygen saturation taken from central venous catheter or pulmonary artery catheter, which represents a more global value. Bedside surface ultrasound imaging or TEE can rapidly clarify several of these diagnoses.¹⁸

If the reduction of cerebral saturation is associated with an increase in DSA or pEEG activity (Fig. 1A point

#3, and Fig. 3C), then reasons for an increase in CMRO₂ should be suspected, including awakening, reduction in the anesthesia state (as in Fig. 3C), hyperthermia,¹⁹ or, more rarely, seizure activity.²⁰ Conversely, a simultaneous increase in cerebral saturation and pEEG values without any artifact could be associated with increased CBF. In the absence of any artifact, the etiology would most likely be awakening or an increase

Fig. 4 Transcranial Doppler before A) and after B) a transient hypotensive episode in a 33-yr-old woman undergoing cardiac transplantation. Note the significant reduction in the end-diastolic velocities (EDV) and the increase in the pulsatility index (PI) without any significant change in the peak systolic velocity (PSV). The increase in PI was secondary to cerebral venous congestion resulting from a superior vena cava (SVC) anastomotic stenosis with a 22 mmHg gradient between the pressure measured in the SVC (Psvc) and the right atrial pressure (Pra) (C). Note the reduction in both left (SEFL) and right spectral edge frequency (SEFR) and also bilateral cerebral regional saturation (rSO₂) during the episode (arrows) (D).

in brain perfusion (as shown in Fig. 2) following correction of the hypotensive episode (i.e., reactive hyperemia). Nevertheless, as showed in the first patient, artifacts can be associated with an elevated PSI. In those patients, cerebral saturation will typically be slightly elevated or unchanged. Finally, when cerebral saturation increases and the pEEG signals are reduced, this is typically due to increased sedation, which can be seen right after anesthesia induction or following propofol administration (Fig. 1C). Combining pEEG as a monitor of cerebral activity with cerebral oximetry as a monitor

The somatic desaturation was limited only to the upper extremity (Figure 4E white arrow) without any changes in the lower extremity NIRS signal, confirming that the venous drainage was compromised in both the brain and the upper extremity. The anastomotic stenosis was thought to be secondary to edema. The patient was observed and remained asymptomatic without any neurologic deficit postoperatively. ARTF = artifact; AUC = area under the curve; HITS = high intensity transient signal; HR = heart rate; MV = mean velocity; Pfa = femoral arterial pressure, Prad = radial arterial pressure; PSI = patient state index; RF = reference range; SaO₂ = oxygen saturation

of cerebral oxygenation and perfusion represents an interesting way to establish the cause of reduced cerebral activity. Figure 5 summarizes our approach in combining NIRS and pEEG.

In summary, combining both NIRS and pEEG allows for a much more nuanced understanding of the etiology of cerebral desaturation. Future studies are needed to investigate if the combination of both modalities is more prognostic than each alone. Every cerebral oxygen desaturation is not equal.

Fig. 5 Approach to combined cerebral oximetry monitoring and processed electroencephalography (pEEG). Reduction in cerebral oximetry can be associated with a reduction or increase in pEEG. In the presence of simultaneously reduced oximetry and pEEG, somatic oximetry is assessed. If the somatic oximetry is normal then the reduction in cerebral blood flow or venous congestion is of cerebral origin, which can be either unilateral or bilateral. If somatic oximetry is associated with simultaneous desaturation, the etiology of brain desaturation has an extracerebral origin. Typically, it results from cardiac dysfunction, blood loss, hypoxemia, or venous congestion from a superior vena cava (SVC) or inferior vena cava (IVC) syndrome. If cerebral desaturation is associated with an increase in

pEEG, then awakening, hyperthermia, or seizures should be suspected. Increases in cerebral oximetry can be associated with a reduction or increase in pEEG. If both cerebral oximetry and pEEG are increased, then this can cause increased brain perfusion after cerebral desaturation such as hyperemia. It can be associated with an artifact diagnosed using an electromyogram (EMG). When the oximetry increases but the pEEG decreases, this is typical of anesthesia induction or the administration of sedative agents. CBF = cerebral blood flow; CMRO₂ = cerebral metabolic rate of oxygen; ICP = intracranial pressure; NIRS = near-infrared spectroscopy; PaCO₂ = partial pressure of carbon dioxide in arterial blood; PaO₂ = partial pressure of oxygen in arterial blood

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