Near Infrared Spectroscopy: A Light into Patient Safety?

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Abstract

Near infrared spectroscopy (NIRS) is a noninvasive monitor that can be used with clinical skill to help determine cerebral oxygen supply and demand balance even during times when pulsatile flow is absent. Patients for cardiac surgery are at risk for significant post-operative cognitive dysfunction. High flow rates during cardiopulmonary bypass is a risk factor for embolization, while low flows may produce insufficient MAPs to maintain cerebral perfusion. Interventions based on NIRS data has proven beneficial. Other high risk patient populations stand to benefit from using the technology as well. NIRS may help to guide the clinician producing shorter hospital stays and a decrease in post-operative cognitive dysfunction.

What is NIRS?

Light of wavelengths near the infrared bandwidth are able to transcend the calvarium. Wavelengths of peak light absorption for oxyhemoglobin and deoxyhemoglobin are unique to each molecule, about 850 and 750 respectively. Both molecules absorb light around 800 nm. A pad placed on a patient’s forehead containing a light source and sensor can then be used to determine intensity of light absorption at each peak versus absorption bands that correlate to both molecules. Some NIRS devices attempt to hone their measurements to brain tissue by performing this calculation on superficial and deep tissue. Superficial measurements may correlate with skin, dura, and bone. Deep tissue measurement may account for skin, dura, bone, and brain matter.

Proprietary algorithms are used by some manufactures to subtract superficial from deep measurements [1]. Often the attempted focus of monitoring is a watershed region at the junction of the middle and cerebral artery [2]. NIRS differs from pulse oximetry in that it works without being triggered by pulsatile flow. This means it functions during cardiopulmonary bypass.

Why is cerebral ox important in cardiac surgery?

Cardiac surgery may provide substantial increases in a person’s functional capacity. However, at times, these gains are over shadowed by a decrement in neurologic status. For most the decrease is minor and short lived, but the incidence remains significant, as high as 30-60% [3]. In addition, there is a minority of cases, 1%, in which patients suffer a cerebral hypoxic event. Stroke following cardiac surgery may place a 10-fold increase in risk of mortality and carries the burden of long term rehabilitation costs.

Debate

The clinical utility of knowing cerebral oxygen saturation is a topic of debate. Possible mechanisms thought to cause cerebral ischemia during cardiac surgery include microembolic shower, embolism, decrease in oxygen carrying capacity, and insufficient cerebral perfusion. Prediction of poor neurologic outcomes in cardiac surgery by embolism is not a goal of NIRS. Brain tissue that becomes ischemic secondary to embolism may not use oxygen and therefore NIRS may not be sensitive to this mechanism. It has been argued that NIRS may produce normal data in cadavers, which is not surprising because the metabolic rate of dead tissue is very low. Those who are in favor of using NIRS as a routine monitor in cardiac surgery feel that NIRS can give reliable data during times of ischemia in which the predominant mechanism is not embolization. Other counter arguments include the expense at greater than $200 per patient. Also, absolute normal values have not been established and there may be great variation between devices.

Does it work?

Orishi et. al correlated post-operative stroke with prolonged intra-operative decrease in cerebral saturation [4]. They prospectively studied 59 patients undergoing aortic arch surgery by the same surgeon. All patients had Cardiac bypass initiated after body temp was reduced to 25°C and anterograde cerebral perfusion. A post anesthesia induction ratio of cerebral oxy to deoxyhemoglobin was set as a baseline of 1.0. Total time spent below 65% and 55%, of the initial value, were recorded. Post-operative neurologic changes were determined to have occurred in 16 of the 59 patients by anisocoria, mydriasis, motor deficit, or convulsion. Operative time and total time spent below 65% and 55% of the initial value was significantly more for the group

of 16 as compared to the other 49. Six of the 16 patients had a positive a CT or MRI for stroke, as read by a radiologist blinded to the NIRS data. Two of the infarcts were in watershed regions and another study was read as multiple infarcts in the cerebellum and posterior lobe probably due to basilar artery hypoperfusion. Each of these three infarcts had significantly more time spent below 60% than the other three infarcts that were segmental and thought to be due to embolus.

If it works then can we use the data to know when to intervene and can we intervene

Deschamps et al. [5] aimed to evaluate strategies to reverse instances of NIRS cerebral desaturation during high risk cardiac surgery. They prospectively observed 279 patients for the onset of cerebral desaturation defined by at least 15 seconds of a decline by 20% or more, from pre-anesthetic induction values. An algorithm for intervention was constructed based on previous NIRS literature to attempt systematic reversal of the desaturation. Variables assessed and attempted to optimize included laterality, head and cannula position, MAP, systemic saturation, end tidal CO2 or PaCO2, hemoglobin, cardiac function and venous saturation. There were 267 desaturation events, 235 events were reversed by 298 interventions. Therefore, the authors feel that their algorithm produced a successful outcome 78.9% of the time. They then wanted to determine the extent to which systematic intervention employed with NIRS monitoring may alleviate cerebral desaturation. 48 patients were randomized, who were not undergoing circulatory arrest, into an interventional and control group. Cerebral desaturations in the interventional group were monitored by NIRS and subsequent interventions were employed via the aforementioned algorithm. In the non-interventional group the anesthesiologist was blinded to the NIRS monitor, and the anesthetic was carried out as if this information was unavailable. They then calculated a desaturation load. Using the same criteria they calculated the desaturation load by adding the sum of the 15 second interval desaturations and multiplied by the desaturation depth. There was a statically significant increase in the desaturation load during the intra-operative and post-operative periods for the control group. It can be seen that an increase in load by adding the sum of the 15 second interval desaturations. A weakness of this study may be that defining desaturation in time increments of five minutes is significantly longer than many other studies in this field.

CONCLUSION

The addition of monitoring regional cerebral saturation by NIRS may help guide a clinician through certain mechanisms of ischemia. The best possible patient outcomes may occur when a systematic approach to reversing regional desaturation is employed. In addition to cardiac surgery cerebral saturation monitoring may be useful in other surgical fields with patients at high risk for post-operative cognitive delay or frank stroke. While NIRS monitoring may seem expensive at greater than $200 per patient the bigger picture is that interventions employed on the behalf of this monitor may reduce length of hospital stays which has the possibility of decreasing total cost and increasing patient satisfaction.

REFERENCES
