Chapter 10
Cerebral Oximetry

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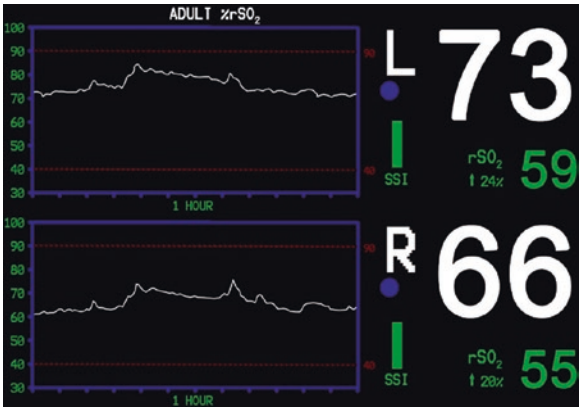

Fig. 10.1 An example of a cerebral oximeter monitor with normal values

Questions

1. What is cerebral oximetry?
2. How does it work? Is it similar to pulse oximetry?
3. In what clinical scenarios might the cerebral oximetry be used?
4. What are normal values? What are abnormal values?
5. What interventions can be performed to improve rSO_2 values?
6. What are some interference sources for NIRS?
7. What are the current FDA-approved cerebral oximetry devices in the United States?

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Answers

1. Cerebral oximetry (CO) has been available to clinicians for more than two decades [1, 2]. Currently this monitor can be used as a "first alert" of impending organ dysfunction [3]. The cerebral cortex is an area of the brain that is particularly susceptible to changes in the demand and supply of oxygen and has a limited oxygen reserve. CO estimates the oxygenation of regional tissue by transcutaneous measurement thru the cerebral cortex.

2. Cerebral oximeters consist of adhesive sensors applied over the frontal lobes which both emit and capture reflected light based on near-infrared spectroscopy (NIRS). CO depends on the ability of light to penetrate the skull to determine hemoglobin oxygenation from the underlying brain tissue according to the amount of light absorbed by hemoglobin [4]. NIRS uses two photodetectors with each light source. Selective sampling of tissue beyond a specified depth beneath the skin is measured by the technology. Near-field photodetection can be subtracted from far-field photodetection to provide selective measurements of tissue oxygenation. Tissue sampling is mainly from venous (70–75%) rather than arterial (25%) blood (Fig. 10.1). It is independent of pulsatile blood flow. As opposed to pulse oximetry, which monitors arterial blood hemoglobin saturation (SpO_2), cerebral oximetry monitors hemoglobin saturation in mixed arterial, venous, and capillary blood in cerebral tissue ($SctO_2$). As a result $SctO_2$ is determined by two physiologic considerations. The first is the proportional volumes of arterial, venous, and capillary blood in the brain region illuminated by cerebral oximetry. $SctO_2$ is higher if the sample has an increased ratio of saturated arterial blood to desaturated venous blood and conversely lower if the ratio is decreased. The volume percentage of each blood compartment is not fixed. It varies interindividually and possibly between different brain regions of the same individual. It may also change with hypoxia, hyper-/hypocapnia, neural excitation, and vasoconstrictor administration [5].

   The second consideration is the balance between cerebral oxygen supply and demand. Cerebral oxygen supply is determined by cerebral blood flow and arterial blood oxygen content. If arterial blood content is stable, an increase in CBF will expand arterial blood volume and shift the volume ratio toward more arterial blood. Cerebral oxygen demand is determined by cerebral metabolic rate of oxygen. If cerebral oxygen supply is stable, an increase in cerebral metabolic rate of oxygen will expand venous blood volume ratio toward more venous blood. These physiologic processes alter $SctO_2$ readings. When $CMRO_2$, arterial blood content, and the volume percentage of different blood compartments are all relatively stable, $SctO_2$ can be regarded as a surrogate of cerebral perfusion [5].

3. Clinical Scenarios:
   Cardiac Surgery

   Multiple clinical outcome studies [3, 6, 7] support the concept that CO may allow clinicians to use the brain as an index organ that points to the adequacy of tissue perfusion and oxygenation of other vital organs. Data from the Society of Thoracic Surgeons (STS) National Database strongly suggest that the intraoperative use of CO in cardiac surgery patients frequently (23%) served as a "first

alert" indicator of an intraoperative dynamic that could lead to potential adverse clinical outcomes in both adult and pediatric patients. The cerebral frontal cortex is a vulnerable watershed tissue that is sensitive to small decreases in oxygen saturation and therefore can provide an "early warning" about compromised oxygen delivery to the rest of the brain and other major organs [8].

Patients whose saturations fell below 75% of preoperative levels and who were treated spent less time in the ICU and had less morbidity/mortality than the untreated group [6, 9, 10].

Cerebral oximetry has been shown to predict the lower limits of autoregulation during cardiopulmonary bypass [11]. Real-time monitoring of rSO_2 provided more accurate information than routine blood pressure monitoring in identifying the lower limit of autoregulation.

Cerebral Vascular Surgery, Geriatric Surgery, and Thoracic Surgery

Cerebral oximetry preinduction value and/or an intraoperative decrease in rSO_2 value can guide in advance decisions regarding blood pressure manipulation or elective shunting for carotid endarterectomy. For cerebral vascular disease, a cutoff value of 25% or 20% below baseline for prolonged hypoperfusion is used to opt for shunting [12].

Aggressively treating values that fall below 75% of baseline rSO_2 in general surgery and geriatric patients improved or maintained scores on the Mini-Mental State Examination at postoperative day 7 and reduced the length of stay in the postanesthesia care unit [7].

Early cognitive dysfunction after thoracic surgery with single lung ventilation was found to be directly related to intraoperative decline of rSO_2 [13, 14].

Trauma

NIRS cerebral oximetry has been found to correlate with cerebral blood flow in trauma patients with brain injuries [14]. This monitor has found a use in trauma patients on the scene and en route to the hospital providing valuable information [15]. Cerebral oximetry may be a useful technique for predicting mortality and/or adequacy of CPR from cardiac arrest.

Heart Failure and ECMO

In heart failure patients, rSO_2 may be a potential important biomarker and useful monitor of target organ perfusion. When ECMO must be used for a prolonged period of time, brain perfusion in the setting of normal vital signs is undetermined. Sensors measuring rSO_2 can be placed on the forehead and lower extremities to monitor perfusion. When rSO_2 values drop to below 40 or greater than 25% of baseline, interventions such as fluid administration, increase in ECMO flow, vasopressors, or replacement of a functioning distal perfusion catheter can be initiated to reduce the incidence of stroke or limb ischemia [16].

Beach Chair Position

This is an emerging area of cerebral oxygen saturation monitoring. Cerebral malperfusion may be unappreciated in this setting. Blood pressure monitoring may not be optimal, head position may impede cerebral venous drainage thereby decreasing CBF, and positive pressure ventilation impedes an already compromised decreased venous return to the heart because of beach chair positioning [8, 17].

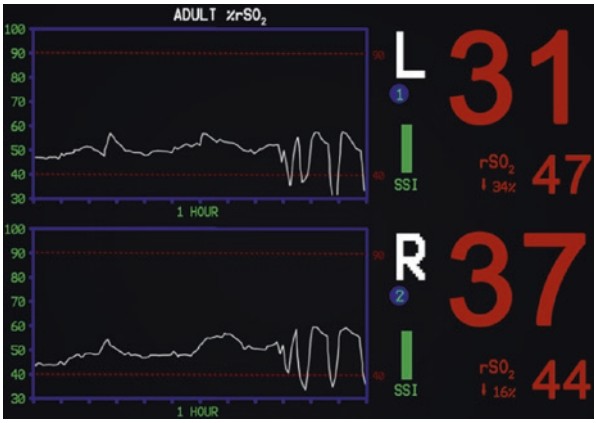

Fig. 10.2 An example of a cerebral oximeter monitor with abnormal values

4. In this technology, near-field photodetection is subtracted from far-field photodetection to provide selective tissue oxygenation measurement beyond a predefined depth [3]. Normal $Sr0_2$ baseline values would be 60–80%. Generally speaking, greater than 25% decrease or 20% decrease from baseline or a SrO_2 value less than 40% is considered a trigger for intervention.

5. The guiding principle in the treatment of cerebral desaturation (Fig. 10.2) is to increase oxygen delivery to the brain and/or decrease cerebral metabolic rate of oxygen utilization. Ways to augment CBF include:

   (a) Increasing cerebral perfusion pressure if it is below the lower limit of cerebral autoregulation and autoregulation is intact
   (b) Increasing cerebral perfusion pressure irrespective of the lower limit if autoregulation is impaired
   (c) Augmenting cardiac output
   (d) Avoiding hyperventilation and hypocapnia, maintaining $PaCO_2$ greater than or equal to 40 mmHg
   (e) Administering a cerebral vasodilator
   (f) Using inhalational anesthetic agents based on their intrinsic cerebral vasodilating properties at less than 1 MAC
   (g) Checking head position to assure optimal cerebral venous outflow
   (h) Augmenting cerebral venous drainage with 30 degree reverse Trendelenburg position.

   Additionally, interventions capable of improving arterial blood oxygen content such as increased inspired oxygen fraction and red blood cell transfusion should be considered to boost oxygen delivery to the brain.

   On the consumption side, deepening anesthesia causes a progressive decline in cerebral metabolic rate of oxygen until EEG becomes isoelectric. Too deep of an anesthetic though causes hypotension and abolishes autoregulation which would be counterproductive [5].

Interventions depending on the clinical scenario but would include:

Cardiac Surgery: correction of patient or cannula positioning, increasing blood pressure, increasing cardiac output or CPB flow to greater than 2.5 L/m2/min, increasing $FI0_2$, increasing $PaC0_2$ to >40 mmHg by decreasing minute ventilation or decreasing oxygenator fresh gas sweep flows during CPB, administering anesthesia and/or muscle relaxants as indicated, and administering a red blood cell transfusion if the hematocrit is <20%.

Carotid Endarterectomy: all of the above maneuvers aside from those related to CPB would be appropriate. Additionally, if rSO_2 values are particularly low on the one side or the other, elective shunting would be indicated for the procedure rather than just clamping the vessel.

6. Essentially any pharmacologic or anatomic abnormality which might involve blood flow, hemoglobin abnormalities affecting light absorption in the same spectra as NIRS, or distance between the near and far-field photodetection. Variations in oximeter design, use of systemic vasoconstrictors, and underlying skin pigmentation may affect the accuracy of cerebral oximetry readings [18]. Deeper anatomical structures such as the skull and frontal sinus may also play a role. Hyperostosis frontalis interna with the resultant shallow frontal sinus may cause unreliable rSO_2 readings. With skull thickness causing low readings, moving the oximeter probes to a more lateral or more cephalad positions where the skull is not as thick or the sinus as superficial might improve readings [19].

   Bilirubin dampens the spectrophotometry determined cerebral saturation at 733 and 809 nm. Normal absorption spectra for this technology are 700 to 1000 nm. A bilirubin level of 370 mmol/L, tissue pigment deposits, or both may render cerebral oxygen saturation impossible [18].

7. Current devices approved by the FDA for CO monitoring include:
   INVOS (Somanetics Corporation, Troy, MI, recently COVIDIEN, Boulder, CO)
   FORE-SIGHT (CAS Medical Systems, Inc., Branford, CT)
   EQUANOX (NONIN Medical, Inc., Minneapolis, Minn.)

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