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Role of INVOS in critical congenital heart disease during cardiac catheterization

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Abstract

Neurobehavioral impairments are common in infants undergoing surgery for CHD, and they occur across a wide spectrum; cognition, motor, social interaction and behavior, language, inattention, and executive function. Many studies have discussed the factors related to surgery or postoperative care on this issue. However, some studies suggested that brain damage may occur during the early postnatal life of infants with CHD, which may lead to impaired neurodevelopment, in this article, we aimed to reviewed the role of INVOS in critical congenital heart disease during cardiac catheterization. Many centers, and even entire countries, have adopted near-infrared spectroscopy as standard of care. The available data suggest that multimodality monitoring, including near-infrared spectroscopy, may be a useful adjunct. The current literature on the use of near-infrared spectroscopy alone, however, does not demonstrate improvement in neurologic outcome. The data correlating near-infrared spectroscopy findings with indirect measures of neurologic outcome or mortality are limited. Although near-infrared spectroscopy has promise for measuring regional tissue oxygen saturation, the lack of data demonstrating improved outcomes limits the support for widespread implementation.

Keywords: congenital heart disease, cardiac catheterization, cerebral oxygenation

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1. Introduction

NIRS and cerebral oximetry are identical technologies. They rely on the relative transparency of biological tissue to near infrared light (700 to 900 nm) where oxy-, deoxyhemoglobin and cytochrome aa3 have distinct absorption spectra. By monitoring light signals at several wavelengths, it is possible to determine ScO_2, concentrations of oxy- and deoxyhemoglobin, and cytochrome aa3 redox state [1]. At present, the devices are based on continuous-wave or frequency-domain technology. Continuous wave devices have been available for several years and monitor the intensity of the detected light relative to the emitted light; they describe oxygenation changes over time from an unknown baseline [2]. Cerebral oximetry and pulse-oximetry differ in several respects. Although both use near infrared light signals, pulse oximetry monitors the pulsatile signal component reflecting the arterial circulation, whereas cerebral oximetry monitors the non-pulsatile signal component reflecting the tissue circulation (arterioles, capillaries and venules) [1]. This noninvasive technique is accomplished by placing a sensor on the skin over the frontal and temporal regions of the brain. By passing a low intensity near infrared light into the brain, this technique allows the capability of measuring changes in tissue chromophores such as hemoglobin [3]. As light is returned at two distances from its source i.e., the spectral absorption of oxygenated and deoxygenated hemoglobin in the brain can be measured and the relative differences can be compared with time). This measurement is displayed as an oxygen saturation index ($rS0_2$) of the mixed arterial and venous blood in the cerebral cortex [4].

2. Critical congenital heart diseases

Critical congenital heart defects are usually defined as structural malformations of the heart that are present at birth and require intervention in the 1st year of life. Congenital heart disease has been reported to be responsible for 30–50% of infant mortality due to birth defects, During the period from 1999 to 2006 (before the advent of critical congenital heart disease [CCHD] newborn screening), more than 13,000 infant deaths caused by congenital heart defects were reported in the United States [5]. The American Heart Association (AHA) defines critical CHD as any CHD, which

requires intervention within the first year of life. A more practical definition would be CHD, which "required intervention or resulted in death within 28 d of life". Applying this definition, the categories of CHD, which could benefit from an early screening module would include [6].

2.1. Epidemiology

Congenital heart disease (CHD) is one of the most frequently diagnosed congenital disorders afflicting approximately 0.8% to 1.2% of live births worldwide. Generally, CHD is defined as a structural abnormality of the heart and (or) great vessels that is present at birth. Although numerous etiologic investigations have been conducted, only approximately 15% of cases of CHD can be attributable to a known cause [7]. A meta-analysis of 1.3 million live births, with regional and specific CHD differences, showed that the global average prevalence of CHD at birth was 8.2 per 1000 births during 1970–2017. Critical CHD (CCHD) is any CHD requiring urgent medical intervention during early infancy, representing about 25% of babies with a CHD [8]. In Egypt, Congenital heart disease (CHD) is one of the relatively common congenital disabilities whose prevalence ranges from 3.5-17.5 per 1000 live births. They are becoming an increasing cause of pediatric mortality, especially in the developing countries [9].

3. Classification of CHD

CHD can be classified into three main categories in clinical point of view [10].

3.1. Life-threatening CHD

Structural cardiac malformations in which cardiovascular collapse is likely and compromised if not treated early. They include TGA, COA/IAA, AS, and HLHS/mitral atresia, PA and obstructed TAPVR.

3.2. Clinically significant CHD

Structural cardiac malformations that have effects on heart function but where the collapse is unlikely to be need early intervention. Most common defects in this group are ventricular septal defect (VSD), complete atrioventricular septal defect (AVSD), atrial septal defect (ASD) and tetralogy of Fallot (TOF) with good pulmonary artery anatomy.

3.3. Clinically non-significant CHD

Anatomically defined cardiac malformations but no functional and clinical significance. They include small VSD, atrial septal defect (ASD), mild pulmonary stenosis (PS), only detectable with echocardiography and requiring no treatment.

3.4. Cyanotic congenital heart disease

Cyanotic CHDs usually have multiple defects of the heart that result in right-to-left shunt. Obstruction to pulmonary blood flow (for example tetralogy of Fallot), complete admixture of pulmonary and systemic venous returns (for example, total anomalous pulmonary venous return and double-inlet left ventricle) and parallel rather than in-series circulation (transposition of the great arteries) are the usual causes of right-to-left shunts and cyanosis. In cyanotic CHD, the primary physiological abnormality is arterial desaturation due to right to left shunt across the atrial/ventricular septal defects or patent ductus arteriosus, mixing of pulmonary and systemic venous returns, or secondary to parallel circulation in transposition of the great arteries [11].

4. Clinical Presentation of Critical CHDs in 2019 and Their Immediate Management

In the current era, there are 3 clinical presentations of neonates with a critical CHD: Prenatal diagnosis, Clinical recognition of CHD and Pulse Oximetry Screening (POS).

4.1. Clinical Recognition of CHD in Neonates

The recognition of a murmur has long remained the cornerstone of diagnosing CHD in a neonate. Unfortunately, most critical CHD in neonates present without a murmur. The clinical recognition is further complicated by the fact that there is considerable overlap in presentation between CHD and other more common neonatal conditions. Thus, most infants with HLHS or critical left ventricular outflow tract obstructions will be wrongly labelled as septic shock while pulmonary atresia is often misdiagnosed as Persistent Pulmonary Hypertension of the Newborn (PPHN). However a focused clinical examination can give valuable clues to suspecting a CHD.

4.1.1. Cyanosis

Cyanosis is bluish discoloration of the skin and mucous membranes due to an increased amount of deoxygenated hemoglobin. Most neonatologists are well versed in differentiating central cyanosis from the more common peripheral cyanosis which occurs due to hypothermia and poor peripheral perfusion. Central cyanosis may be due to respiratory or cardiac causes. It is not always easy to differentiate these 2 causes clinically. Cardiac cyanosis more commonly results in quiet tachypnea where the work of breathing is not particularly increased [13].

4.1.2. Systemic Hypo perfusion

Systemic Hypo perfusion the commonest causes of shock in neonates are sepsis and hypovolemia. It is virtually impossible to differentiate cardiogenic shock from other causes clinically. However, certain clues can point towards the diagnosis of cardiogenic shock. The presence of cardiomegaly as well as low post ductal SpO2 is the most important clinical clues for a cardiac cause. The CHD which present with systemic hypoperfusion are the left ventricular outflow obstructions which include aortic stenosis, coarctation of aorta/interrupted aortic arch and HLHS at the more severe end of the spectrum. These commonly present when the arterial duct closes, an event which occurs before 96 h of life in a vast majority [14].

4.1.3. Severe Respiratory Distress and Pulmonary Edema

A clinical picture of pulmonary edema with tachypnea, chest wall retractions and a ground glass appearance on chest X-ray may be due to disorders associated with pulmonary venous obstruction (Fig. 3). These include TAPVC and cortriatriatum. Clinical differentiation from respiratory causes is impossible. However if there is lack of improvement or worsening with conventional respiratory management, the possibility of a cardiac cause should be entertained and an early echocardiogram is mandatory. Neonates with these conditions are often the sickest neonates with CHD but improve dramatically with definitive surgery [15].

4.1.4. Cerebral oxygenation (INVOS)

For CHD patients undergoing cardiac surgery or cardiac catheterization, brain injury could occur before and after interventions, and neurodevelopmental impairment could affect patients' cognition, motor skills, social interaction and behavior, language, concentration, and executive function [16]. Preoperative and postoperative hypotension and hypoxemia are significant risk factors for brain injury in pediatric CHD patients. Thus, real-time neurological monitoring is necessary for children with CHD undergoing cardiac surgery or cardiac catheterization. Cerebral perfusion and oxygen saturation are important factors affecting neurological functions that must be monitored perioperatively to assure satisfactory patient outcomes [17].

4.2. Pulse Oximetry

Pulse oximetry is sometimes referred to as the fifth vital sign; it is a quick and non-invasive monitoring technique that measures the oxygen saturation in the blood by shining light at specific wavelengths through tissue, most commonly the fingernail bed [18]. Pulse oximetry is a noninvasive means to rapidly and accurately determine oxygen saturation. This modality is one of the standard monitor parameters used in the critical care setting and during anesthesia cases and is recommended by the American Society of Anesthesiologists [19].

4.2.1. Pulse oximetry use for CCHD screening

Addition of pulse oximetry screening raised the diagnosis rate to 82%. Several other studies mostly from Europe revealed similar findings. These were summarized in a systematic review and meta-analysis of pulse oximetry screening for CCHD in the newborn nursery, which included 13 studies with 229,421 infants [20].

4.3. Cerebral Oximetry

Cerebral oximetry with NIRS is a noninvasive, continuous assessment of brain oxygen delivery and use. NIRS-based cerebral oximeters quantitate a venous-weighted ratio of oxygenated and deoxygenated hemoglobin in the region of cerebral cortex underlying the sensors, usually placed on the forehead and sometimes overlying the kidney. By comparison with pulse oximetry, cerebral oximeters trend venous-weighted measurements because the entire returned signal is measured rather than just the pulsatile measurements that make pulse oximetry specific to arterial blood oxygen saturation [21]. Because cerebral oximetry interrogates all hemoglobin in the reflectance arc (including arterial, venous, and capillary hemoglobin), the resulting number is biased toward the larger venous hemoglobin mass, which is consistently higher than, but correlated with jugular venous oximetry. Whereas the pulse oximeter is a useful trend of pulmonary function and the a-A gradient, cerebral oximetry trends the ratio of regional oxygen delivery and use to detect cerebral ischemia [22].

5. Clinical interpretation of cerebral oximetry measurements

Adequate cerebral oxygenation is dependent upon adequate cerebral blood flow and oxygen content. Factors affecting either of these will result in a reduction in cerebral oxygenation and a reduction in cerebral oximetry values. Anatomical variations, for example, an incomplete Circle of Willis, or severe carotid artery stenosis can create errors in cerebral oximetry values; therefore, it is recommended that cerebral oximetry is performed bilaterally [23].

6. Cerebral Oxygenation in critical congenital heart diseases

6.1. Pediatric Cardiac Surgery

Many groups have advocated for monitoring $rScO_2$ in pediatric patients undergoing cardiac surgery for congenital heart defects. Another small study in 25 pediatric surgery patients showed a positive correlation between $rScO_2$ and mixed venous oxygen saturation [24]. Whereas Bhutta et al., [25] reported a correlation between cerebral oximetry and superior vena cava oxygen saturation in pediatric patients after heart transplant. Moreover, intraoperative $rScO_2$ has been shown to be a sensitive indicator for the adequacy of cerebral perfusion in pediatric surgical patients who weigh less than 10 kg, as mixed venous oxygen saturation in those patients is more representative of lower body oxygenation [26]. A recent systematic review of the role of rScO2 in pediatric patients undergoing surgery for congenital heart disease identified 54 manuscripts (47 case series, 4 randomized trials, and 3 retrospective studies) and 13 review articles. Taken together, the evidence failed to show that rScO2 monitoring leads to a clinical improvement in short-term neurologic outcome in this patient population [27].

6.2. Cerebral Oxygenation and the Patent Ductus Arteriosus

The hemodynamically significant patent ductus arteriosus (PDA) remains a controversial topic. Clinicians and researchers are still debating whether or not it should be treated, what the best treatment strategy is and when would be the best time to intervene [28]. Infants who need surgical PDA closure are often exposed to low $rScO_2$ values for a longer period of time, A, and are therefore at risk of cerebral injury. Additionally, a further reduction in cerebral oxygenation occurs during ductal surgery [29].

Table 1. Common cyanotic congenital heart defects [12]

5 Ts	Other Defects
Tetralogy of Fallot	Hypoplastic left heart syndrome
Transposition of the great arteries	Pulmonary atresia with intact ventricular septum
Tricuspid atresia	Double-outlet right ventricle
Total anomalous pulmonary venous connection	Double-inlet left ventricle and univentricular hearts
Truncus arteriosus	

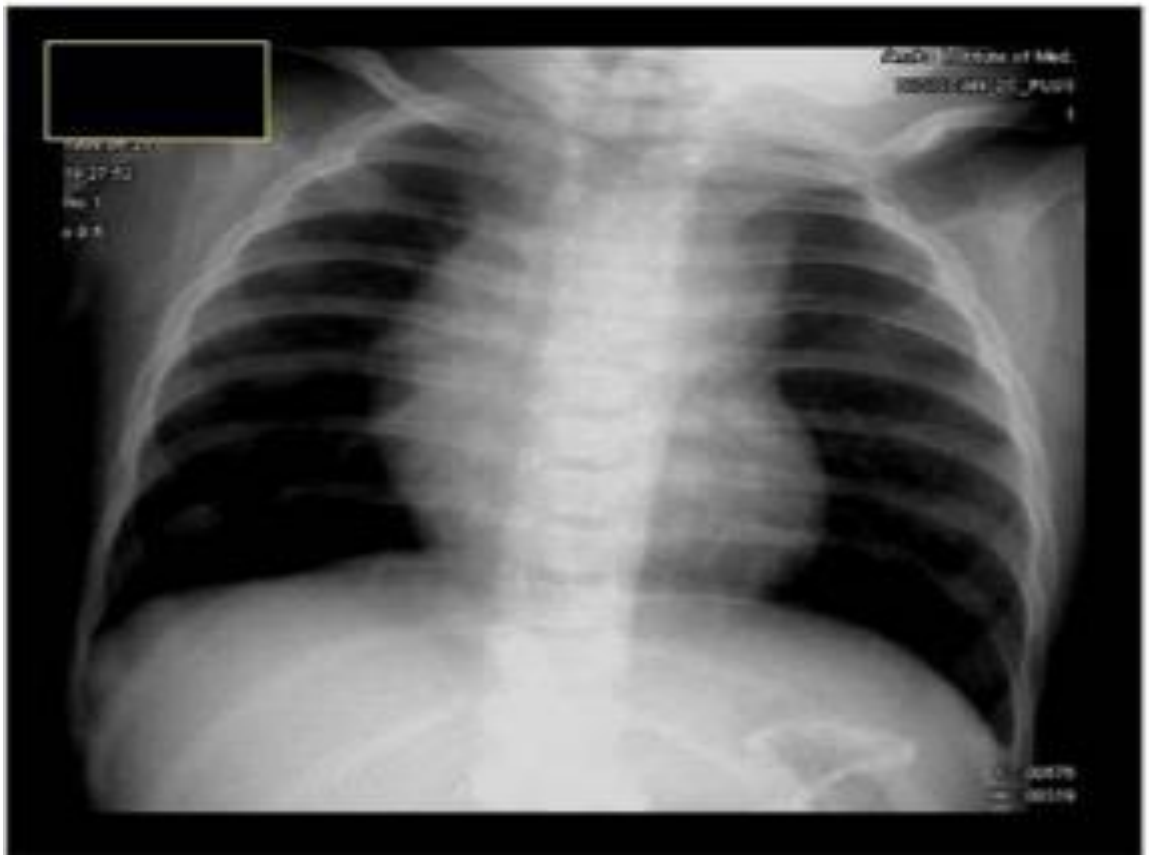

Figure 1. Chest X-ray of a cyanotic neonate demonstrating mesocardia, a normal cardiac size and decreased pulmonary vascularity consistent with the cardiac diagnosis of pulmonary atresia [6]

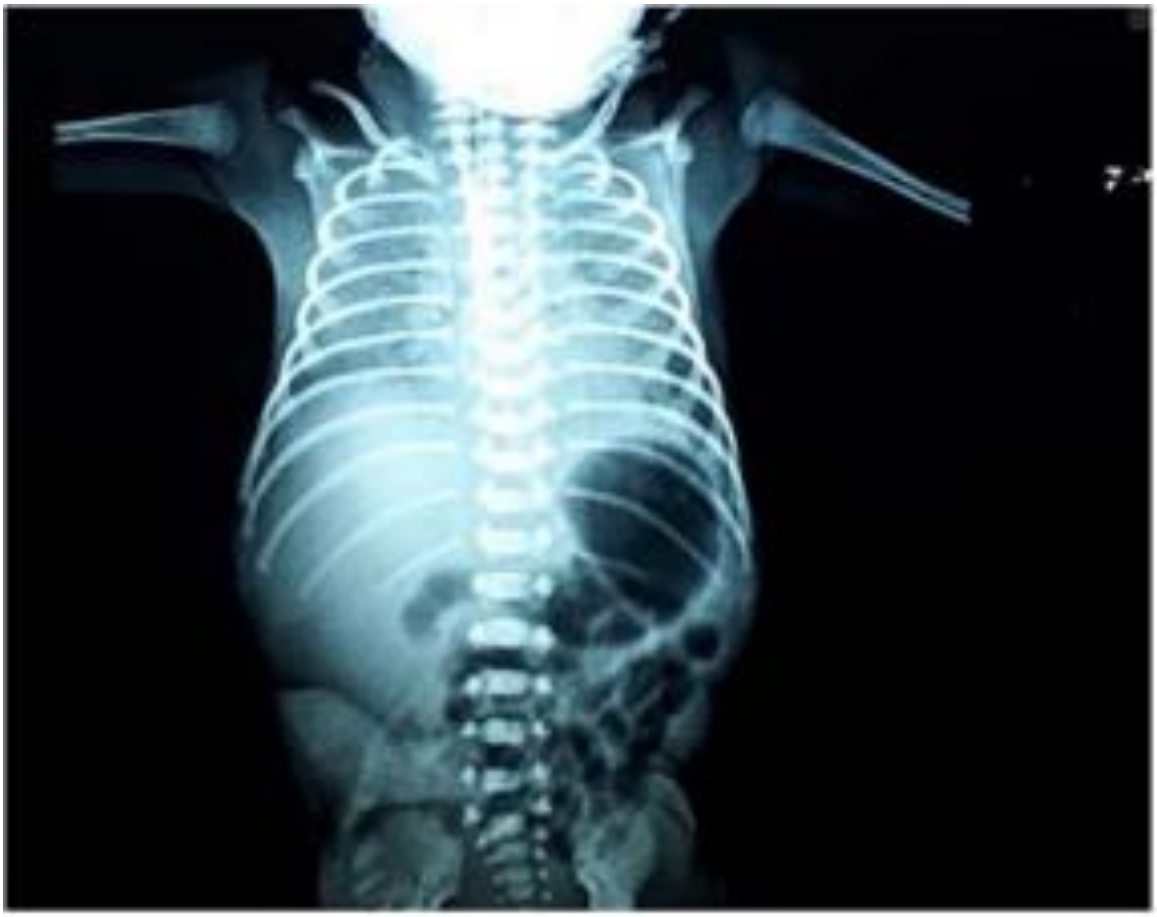

Figure 2. Chest X-ray of a neonate with cyanosis and markedly increased work of breathing demonstrating a ground-glass appearance consistent with the diagnosis of obstructed infra-cardiac total anomalous pulmonary venous connection [6]

Table 2. Summarizes some factors that may result in reduced cerebral oxygenation values caused by alterations in blood flow or oxygen content [23]

Cerebral blood flow	**Oxygen content**
Cardiac output	Haemoglobin concentration
Acid–base status	Haemoglobin saturation
Major haemorrhage	Pulmonary function
Arterial inflow/venous outflow obstruction	Inspired oxygen concentration

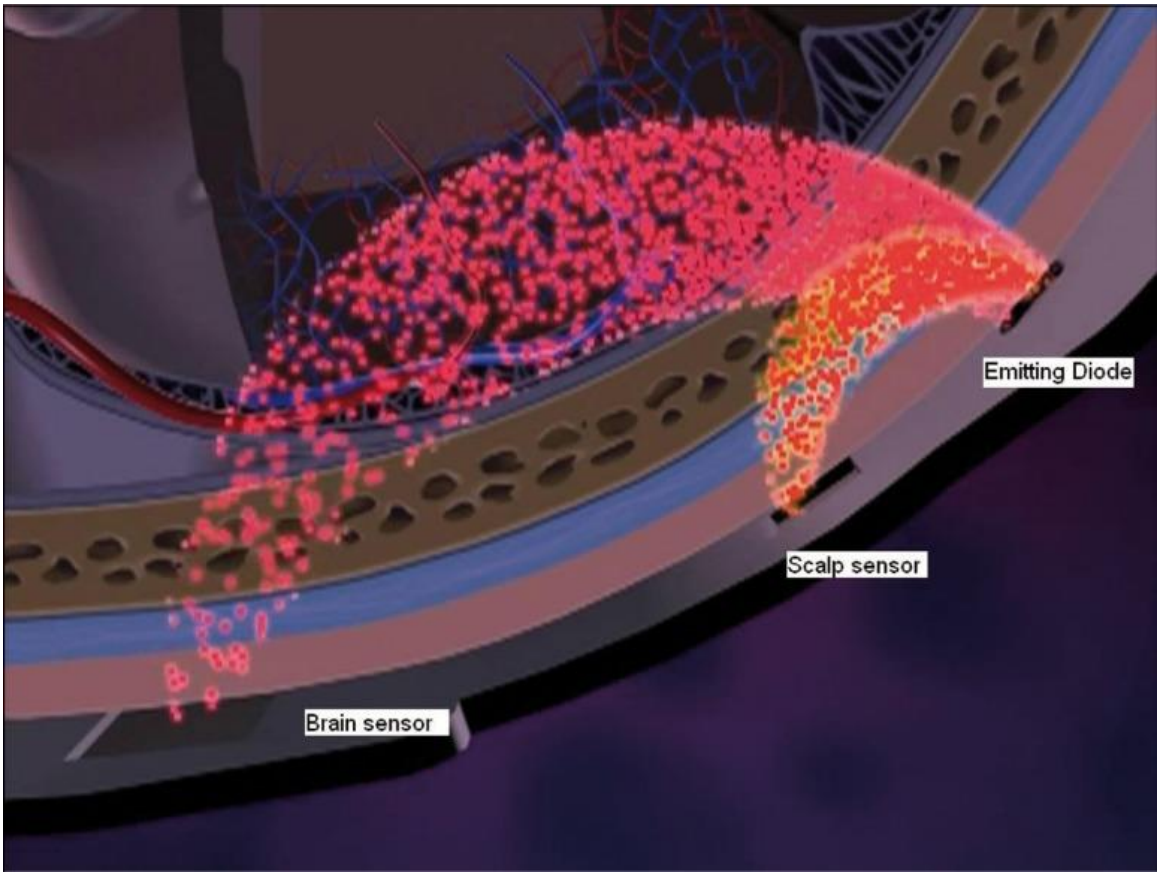

Figure 3. By subtracting the measurements obtained from the brain detector from the scalp detector, extracererbal contamination can be minimized [33]

6.3. Cerebral Oxygenation and Carotid Artery Surgery

Carotid endarterectomy (CEA) surgery reduces the risk of stroke for patients with symptomatic carotid stenosis and subgroups of patients with asymptomatic stenosis. These benefits are only realized if the rate of perioperative neurologic complications, including stroke, can be minimized. The risk for stroke after CEA depends on many factors, including the acuity and severity of symptoms and the individual center experience [30]. Cerebral oximetry index (COx) recording from a patient undergoing cardiopulmonary bypass. The top graph is the time series for mean arterial pressure (ABP) and the raw left and right regional cerebral oxygen saturation (rSO_2) [31].

7. The evolution of cerebral oximetry

A "biological spectroscopic window" exists at the wavelength range 660-940 nm because only a few chromophores like Hb and HbO_2 strongly absorb light in this spectra range, allowing light to penetrate tissue to a great distance. In this wavelength range, absorption of light due to other biological compounds and tissues such as water, lipids, skin, and bone is lower in magnitude, and these biological compounds generally have a flat absorption spectra when compared to Hb and HbO_2 [23]. In order to guarantee that only cerebral oxygen saturation is being measured most commercially available oximeters minimize extracerebral contamination by equipping the sensors with 2 light detectors located at fixed distances from the light source. The mean penetration depth of the photons is proportional to the distance between the emitting source and receiving detector. Consequently the detector (scalp or near detector) located closer to the light source measures saturations within the scalp, whereas the detector located further away measures both cerebral as well as scalp saturations (brain or far detector) [32] (Figure 3). The physical principles upon which NIRS is based have been described. In general terms, NIRS utilizes the absorption and reflectance spectra of near-infrared light to quantify oxygenation of tissues underlying the sensor. Both INVOS and Foresight use the modified Beer–Lambert law to measure tissue oxygen saturation, and eliminate the contribution of extracerebral tissue by using the principle of

spatial resolution (depth of photon penetration proportional to the source–detector separation). However, some important technical differences exist [34]. Near-infrared spectroscopy (NIRS), which is an attractive, noninvasive method for regional monitoring of cerebral oxygenation, has been used in cardiac surgery patients during intra- and postoperative periods. This method can result in earlier detection of cerebral complications, and its use has been associated with improved clinical outcomes in these patients. NIRS is easy to use and provides continuous real-time assessment of regional cerebral oxygen saturation (rSO_2) [35]. The NIR light is absorbed by HHb and O2Hb in both arterial and venous vessels, in a 25:75% ratio, and thus NRS reflects mainly cerebral venous oxygen saturation. The NIR light is absorbed by both superficial tissues and the cerebral cortex [36]. Other NIRS techniques, such as time-resolved spectroscopy and frequency-resolved spectroscopy, are now able to assess cerebral blood volume and quantify absolute concentrations of HHb and O2Hb, respectively. However, these techniques have not yet been proved practically useful in neonatal care [37]. CW spectroscopy emits light at a constant intensity and measures the intensity of the light emerging from the tissue. FD, time domain, and DCS are more advanced techniques used predominantly for research and are described in detail elsewhere [38]. Because of the limited depth of NIR penetration, the value of somatic NIRS in patients greater than 10 to 20 kg is questionable.14 Most of the published somatic studies have been in the neonatal and infant populations in whom the relatively superficial location of the renal, splanchnic, and hepatic vascular beds allows for NIRS based measurements of regional oxygen sufficiency [16]. There are several different NIRS devices and sensors commercially available. A number of comparative studies have shown that the overall correlation between NIRS devices is acceptable, although they differ in technique and algorithm. Smaller and more flexible sensors have been designed for neonatal use [39]. Regional cerebral oxygen saturation represents a mixed saturation largely determined by the venous component (75%), which is why NIRS validation studies have often focused on venous saturation. However, venous saturation does not reflect mixed arterial and venous saturation as NIRS does, and there is no “gold standard” to measure venous oxygen saturation. A good correlation has been reported between oxygen saturation in the jugular vein and NIRS-monitored cerebral oxygenation, with a mean difference of 5%, for different manufacturers [40]. Cerebral fractional tissue oxygen extraction (cFTOE) has been validated against central cerebral venous saturation in newborn piglets. Brain perfusion assessment with NIRS has been compared to perfusion assessment with MRI, which has shown strong correlations. Both $rScO_2$ and TOI have shown good reproducibility [41]. Several studies have analyzed changes in $rScO_2$ with advancing postnatal age. $rScO_2$ is between approximately 40 and 56% directly after birth (irrespective of delivery mode), increases up to 78% in the first 2 days after birth and then slowly stabilizes during 3–6 weeks after birth with values between 55 and 85%. Several studies have published reference ranges immediately after birth, which show a gradual increase during the first 15 min of life [42].

8. Conclusions

Neurobehavioral impairments are common in infants undergoing surgery for CHD especially cardiac catheterization, and they occur across a wide spectrum; cognition, motor, social interaction and behavior, language, inattention, and executive function. Real-time neurological monitoring is necessary for children with CHD undergoing cardiac surgery or cardiac catheterization. Although INVOS has promise for measuring regional tissue oxygen saturation, the lack of data demonstrating improved outcomes limits the support for widespread implementation.

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