

Using the EQUANOX™ 7600 Regional Oximetry System to Identify Profound Cerebral Hypoxemia in a Child with Sickle Cell Disease, Pneumonia and Severe Anemia



Case Overview

This case demonstrates how the Nonin Medical EQUANOX™ Model 7600 Regional Oximetry System identified profound cerebral hypoxemia in a child with Sickle Cell Disease (SCD), pneumonia and severe anemia. The rSO₂ trend provided objective evidence of rapid improvement in brain oxygenation following an emergency blood transfusion and administration of oxygen, allowing for IV antibiotic therapy and providing reassurance to the clinical team regarding the decision not to use mechanical ventilation and general anesthesia.

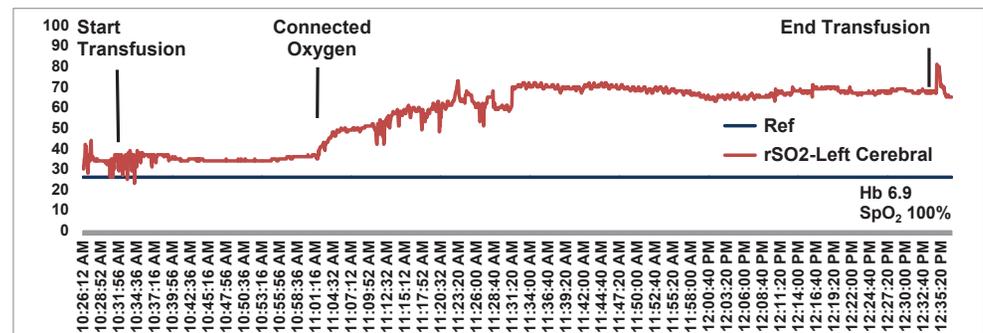
Background:

- **Patient:** 4 year old, 13 kg child presenting to a pediatric emergency room at Mulago Hospital's Acute Care Unit, Kampala, Uganda, with one week history of fever and cough and three days of breathing difficulty.
- **Diagnoses:** Homozygous sickle cell anemia, acute bronchopneumonia, severe anemia.
- **Physical Findings:** Vital signs were: (RR-37/minute; HR-122/minute; BP-101/54 mmHg; SpO₂ on room air- 94%). Physical exam showed patient was conscious and showed moderate tightening in the rib cage (retractions) and nasal flaring, crackling (crepitation) in the right chest (infra-scapular region) and enlarged liver and spleen (hepatosplenomegaly, 5-6cm).
- **Laboratory Findings:** Blood composition, gas and chemistry were well out of normal range, and malaria smear and other malaria tests were negative (Hemoglobin (% hematocrit) on arrival, 3.3 g/dL (9.4%); white blood cell count, 24,940/uL with 60% neutrophils and platelet count, 162,000/uL; initial blood lactate, 185.45 mg/dL; baseline blood chemistries showed K+, 5.0; Base Excess, -6; bicarbonate, 19.5 mEq/L; and anion gap, 18 mEq/L).

Operative Monitoring (During Transfusion)

The patient was given an emergency blood transfusion over two hours with frequent observation of vital signs and clinical status (O+, leukoreduced red blood cells (RBC) at 10 ml/kg). An EQUANOX™ Model 8004CB Pediatric Regional Oximetry sensor was placed on the left forehead for continuous monitoring of the response to transfusion (*Figure 1*). The child also received supplemental oxygen (nasal prongs), intravenous antibiotics (IV Ceftriaxone), and oral analgesic and fever-reduction therapy (acetaminophen-based paracetamol). At hour four (two hours after the completion of the transfusion), the hemoglobin and blood chemistry had improved (to 6.8 g/dL, 20%, lactate 30.91mg/dL). The child was no longer in respiratory distress (respiratory rate 34/min), and the pulse and blood pressure had improved (to 116/min and 102/57 mmHg) (*Figure 2*). The patient was discharged from the hospital in good condition four days after arrival.

Figure 1: The EQUANOX 7600 Regional Oximeter rSO₂ trend graph from an 8004CB sensor placed over the left forehead demonstrates rescue from profound cerebral hypoxemia by a red blood cell transfusion in a child with Sickle Cell Disease, pneumonia and severe anemia.



What the EQUANOX System Showed

The EQUANOX 7600 Regional Oximetry System immediately identified that this child had profound cerebral hypoxemia, triaging the child into a group at much higher risk for complications including seizures and acute stroke. The initial regional cerebral saturation (rSO₂) value of 31% represents the extreme of the limit of consciousness. The absence of stupor and coma in this child was probably due to adaptation to the chronic anemia of SCD, and the patient was judged to be at high risk for cardiopulmonary arrest. The rSO₂ trend provided

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Figure 2: Monitored Parameters and Laboratory Results

Time (Hr)	HR (BPM)	RR (BPM)	BP-Cuff (mmHg)	SpO ₂	L rSO ₂	TCO ₂ /HCO ₃ /Base	Hgb/HCT (g/dL / %)	Lactate (mg/dL)
0 (Baseline)	122	37	101/54	94% on RA	31	20 / 19.5 / -6	3.3 / 9.4%	185.45
2 Hrs	127	40	106/69	100% on O ₂	67			47.27
4 Hrs	116	34	102/57	99% on O ₂	N/A	22 / 21.6 / -1	6.8 / 20%	30.19

The rSO₂ trend, combined with the overall clinical status, provided substantial reassurance to the clinical team regarding the decision not to use mechanical ventilation and general anesthesia.

objective evidence of rapid improvement in brain oxygenation following transfusion and administration of oxygen, allowing for IV antibiotic therapy. Indeed, this finding, combined with the overall clinical status, provided substantial reassurance to the clinical team regarding the decision not to use mechanical ventilation and general anesthesia.

Discussion

According to the World Health Organization (WHO), more than 300,000 babies are born each year with severe haemoglobin disorders.¹ The cerebral complications of sickle cell anemia are profound and include acute stroke and seizures. Recently, silent cerebral ischemia has been recognized as an important cause of neurologic damage in children with sickle cell anemia, affecting up to 27% of children before their sixth birthday. Repeated small cerebral infarctions lead to progressive neurologic damage and encephalomalacia (a local softening of brain tissue due to hemorrhage or inflammation). This can result in decreased cognitive ability, poor school performance and neurologic

deficits in adulthood. In a recent prospective randomized controlled trial, chronic RBC transfusion programs were shown to decrease the rate of silent cerebral ischemia in children with SCD.²

The goal of RBC transfusion is improved tissue oxygenation. Despite a century of blood transfusion therapy, clinicians have not had, until recently, any non-invasive method to measure tissue oxygenation and have relied on changes in hemoglobin concentration as a surrogate of tissue oxygenation. Near infra-red spectroscopy (NIRS) provides an immediate readout of tissue oxygenation of critical organ beds and can supplement other clinical assessments in patients receiving transfusion for critical anemia.³ Studies show that cerebral oximetry offers promise as a tool to manage patients with SCD.^{4,5}

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References:

- ¹World Health Organization; Sickle cell disease and other haemoglobin disorders; Fact sheet N°308; January 2011.
- ²DeBaun MR, Armstrong FD, McKinstry RC, et al. Silent cerebral infarcts: a review on a prevalent and progressive cause of neurologic injury in sickle cell anemia.
- ³DeBaun MR, Gordon M, McKinstry RC, et al. Controlled trial of transfusions for silent cerebral infarcts in sickle cell anemia. N Eng J Med 2014; 371: 699-710.
- ⁴Dhabangi A, Ainomugisha B, Cserti-Gazdewich C, et al. Cerebral oxygenation during transfusion for profound anemia. Transfusion 2014; 54: 2802.
- ⁵Quinn CT et al; Ped Blood and Cancer 2012; 59:881-7.



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