Cerebral and Somatic NIRS during Anesthesia in Normal Children
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Introduction:
Hemodynamic changes during induction of the anesthetic state result from complex interactions of direct and indirect pharmacologic effects and alterations in autonomic tone. Standard physiologic measures do not detect organ-specific hemodynamics, but changes in the distribution of blood flow during anesthesia and surgery may be detected by monitoring regional oxyhemoglobin saturation (rSO2) by near infrared spectroscopy (NIRS). Our objective was to characterize the changes in regional hemodynamics monitored by NIRS during the course of uncomplicated anesthesia.

Methods:
This was an observational study of measured and derived indices of regional oxygenation and perfusion during anesthesia for catheter ablation of dysrhythmia substrates in otherwise healthy patients. Anesthesia techniques were not constrained by study enrollment. Physiologic measures were collected at 1-minute intervals for all patients from pre-induction to emergence. For each patient, the major case event sequence was temporally marked. Cerebral and renal-somatic regional saturation (rSO2C, rSO2R) was measured using frontal and L4-5 flank probes (Invos 5100, Covidien-Medtronics). Arterial saturation (SET algorithm, Masimo), inspired and end-tidal gas concentrations (Apollo, Drager NA), and non-invasive or invasive arterial pressure were measured using a standard physiologic monitor (GE B850). All measures were temporally aligned and electronically recorded. Regional a-v differences (davSO2) were calculated from synchronous SpO2 and rSO2 assuming a 25% arterial contribution to hemoglobin saturation. Changes in regional oxygenation and in the distribution of cardiac output can be monitored using two-site NIRS. In relatively healthy patients undergoing anesthesia induction for elective procedures, the average response early after sevoflurane induction is an increase in cerebral oxygenation and a decrease in somatic oxygenation, indicating a shift in blood flow away from somatic beds. Individual variation exists in these responses.

Results:
The study population included 32 patients, age 13.3±3.0 (5-17) years, weight 61±25 kg (20-114). Twenty-nine patients received sevoflurane as the major anesthetic agent, and three received intravenous anesthetics without airway instrumentation. Physiologic measures were collected over 6130 minutes, average 185 minutes/patient. At baseline, the SpO2 was 99±1% (96-100), rSO2C 82.0±5.8% (70-95), rSO2R 86.2±5.5% (70-95), davSO2C 26±10% (4-57), davSO2R 13±8% (5-40), and drSO2RC 0.87 (0.57-1.0). Anesthesia induction affected regional saturation and hemodynamics differentially, with a direct increase in cerebral rSO2 by 8.7±4.1%, and a decrease in somatic rSO2 by 3.6±0.5%, and a corresponding decrease in cerebral davSO2 by 11.3±1.1% and an increase in somatic davSO2 by 5.7±0.9%. The somatic-cerebral difference fell by 11.9±0.6%, indicating a shift in regional blood flow from somatic to cerebral beds during induction. These changes are illustrated graphically (figure 2).

Conclusions:
Changes in regional oxygenation and in the distribution of cardiac output can be monitored using two-site NIRS. In relatively healthy patients undergoing anesthesia induction for elective procedures, the average response early after sevoflurane induction is an increase in cerebral oxygenation and a decrease in somatic oxygenation, indicating a shift in blood flow away from somatic beds. Individual variation exists in these responses.