The goal of the current study was to assess changes in cerebral and tissue oxygenation using non-invasive near infrared spectroscopy (NIRS) following awake spinal anesthesia (SA).

Our study hypothesis was that cerebral oxygen saturation does not change after awake SA. Our secondary hypothesis was that lower extremity tissue oxygen saturation increases after awake SA.

Methods

- Following IRB approval, patients who were consented for awake SA were enrolled in the study.
- Prior to placement of SA, the NIRS monitors were placed on the forehead (cerebral) and the thigh (tissue) for baseline measurements.
- Spinal anesthesia was placed using 0.5% isobaric bupivacaine (1 mg/kg) with epinephrine 1:200,000 and clonidine 1 µg/kg.
- NIRS values were recorded every 1 minute after SA, for 30 minutes.
- The change from baseline was calculated by subtracting the baseline value from each measure obtained after block placement.
- Results were modeled as a function of time elapsed using mixed effects linear regression with cluster-robust standard errors.
- Secondary variables recorded included hemodynamic parameters of blood pressure (BP), heart rate (HR), and oxygen saturation (pulse oximetry).

Results

The study cohort included 7 patients, age 7 ± 2 months (Table 1). Seven patients had a successful spinal block placed and were included in further analysis.

The tissue saturation increased significantly over the baseline value (Figure 1), with each minute elapsed being associated with an additional 0.12 increase over baseline (95% CI: 0.03, 0.20; p=0.006). All patients breathed spontaneously on room air without changes in oxygen saturation.

Clinically insignificant changes in hemodynamic variables were noted after SA. In mixed effects regression models, there was no change over time in systolic BP (coefficient = -0.31; 95% CI: -0.70, 0.08; p=0.012), diastolic BP decreased by 0.53 mm/Hg per minute (95% CI: 0.17, 0.88; p=0.003), mean arterial pressure decreased by 0.52 mm/Hg per minute (95% CI: 0.24, 0.80; p<0.001), and HR decreased by 1.3 beat/minute with each minute elapsed (95% CI: 0.3, 2.2; p=0.010).

Discussion

There was no significant change in cerebral oxygen saturation following placement of SA. The change in cerebral oxygen saturation did not depend on minutes elapsed (coefficient = -0.08; 95% CI: -0.31, 0.16; p=0.521) (Figure 1). The tissue saturation increased significantly over the baseline value (Figure 1), with each minute elapsed being associated with an additional 0.12 increase over baseline (95% CI: 0.03, 0.20; p=0.006). All patients breathed spontaneously on room air without changes in oxygen saturation.

Clinically insignificant changes in hemodynamic variables were noted after SA. Data provide further evidence of the safety and neutral effects of SA on parameters of cerebral oxygenation.

Increase in tissue oxygenation likely resulted from the SA-induced sympathetochy and increased regional blood flow.

Conclusion

- Preliminary results support our hypothesis that cerebral oxygen saturation is maintained after awake SA.
- Data provide further evidence of the safety and neutral effects of SA on parameters of cerebral oxygenation.
- Increase in tissue oxygenation likely results from the SA-induced sympathetochy and increased regional blood flow.

References