Abstract 36

Cardiovascular Effects of Spinal Cord Stimulation in Hypertensive Patients

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Background: A number of animal studies have shown that thoracic spinal cord stimulation (SCS) may decrease mean arterial pressure (MAP). A recent study demonstrated a trend towards reduction in MAP at the T5-T6 spinal level in sedated normotensive subjects. Another study sponsored by Medtronic (Minneapolis, Minnesota) demonstrated that chronic SCS at subthreshold stimulation level (85% of the voltage that causes paresthesia) significantly improved angina attacks and 6-minute hall walk distance in drug-refractory angina patients. This study was an acute, single-center, randomized feasibility study and was designed to determine if SCS at two different stimulation strengths would decrease MAP and heart rate (HR) during baseline conditions and during activation of the sympathetic nervous system by the cold pressor test (CPT).

Methods: Six hypertensive patients and 11 normotensive patients were evaluated in this study. All subjects were clinically indicated for SCS therapy to manage their neuropathic pain. Arterial BP was continuously measured at the finger using beat-to-beat photoplethysmographic recordings (Finometer; Finapres Medical Systems, Amsterdam, The Netherlands) at rest and during CPT. SCS at threshold (100%: SCS-100) and subthreshold (80%: SCS-80) intensities were randomly performed in the T5-T6 region of the spinal cord during normal conditions as well as during CPT. Each subject underwent three CPTs with the placebo (control) CPT always performed first. The approximate recovery time between the serial CPTs was 10 to 12 minutes. CPT was performed by immersion of the right hand of each subject into slurry of ice water for 90 seconds. Thirty seconds of beat-to-beat data prior to starting each CPT (baseline) was analyzed. During the 90-second CPT, the median values of the last 30 seconds of data were used for analysis. Heart rate variability (HRV) during baseline and SCS (3–5 minutes of inter-beat-interval data derived by the Finometer) was computed using the Kubios HRV Analysis software (University of Kuopio, Finland). The HRV analysis included measurements in both the time and frequency domain. Repeated measures ANOVA and the Wilcoxon's signed rank test were used to compare groups.

Results: SCS did not significantly alter MAP or HR at baseline nor did it appear to blunt changes in MAP or HR in response to CPT. In the normotensive group, the MAP was significantly elevated by 19.3 mm Hg ($P < .001$) during the placebo phase, and by 16.1 and 15.3 mm Hg during the SCS-80 and SCS-100 phases ,respectively. However, in hypertensive subjects, an enhanced response to the CPT was observed. In the hypertensive group, the MAP was significantly elevated by 26.9 mm Hg ($P < .001$) during the placebo phase, and by 20.8 and 23.4 mm Hg during the SCS-80 and SCS-100 phases respectively. The HR in both the groups did not show any significant changes during the three CPTs. Compared with normotensive subjects, HRV tended to decrease in both the time and frequency domain in hypertensive subjects, although this decrease was not statistically significant.

Conclusion: Acute SCS at the T5-T6 region did not significantly alter MAP or HR compared with baseline (no SCS) in subjects without sedation, supporting our previous findings in sedated subjects. In contrast to acute SCS, chronic SCS may have a different effect on BP and HRV and should be explored in the future.