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Cardiac Sympathetic Blockade during Spinal Anesthesia Involves Both Efferent and Afferent Pathways

To the Editor:—We would like to thank Drs. Thrush and Downs for their interesting case report, "Vagotonia and Cardiac Arrest during Spinal Anesthesia."¹ These authors discuss the contribution of sympathetic and parasympathetic nervous system imbalance during spinal anesthesia as a mechanism of asystole. They state that in a patient with vasovagal syncope, the combination of cardiac sympathetic blockade and vagal stimulation disturbed this autonomic balance even further. It is certainly intuitive to think that because the spinal anesthetic blocks cardiac sympathetic efferent nerves and not vagal efferent nerves, a relative parasympathetic dominance or vagotonic state would result. However, if this was the case, why would severe bradycardia and asystole not occur more often with high-spinal and epidural anesthesia?

As discussed in previous work, another neural pathway is probably involved.²⁻³ Instead of a relative vagal dominance, there was evidence of a decrease in both sympathetic and vagal outflows (efferents) in patients with cardiac sympathectomy after spinal anesthesia. The vagal outflow, which was not directly blocked by the anesthetic, was somehow concomitantly reduced or inhibited in parallel with the anesthetic blockade of the sympathetic efferent outflow.³ Therefore, a state of reduced sympathetic and vagal outflow resulted, which fortunately maintained sympathetic and vagal balance and maintained the baseline heart rate.

It has been proposed that complete blockade of sympathetic afferent pathways, which have an anatomical distribution similar to the sympathetic efferent pathways, will interrupt necessary visceral communication lines to central neural centers and will result in a self-protective reduction of parasympathetic activity.⁵ The activity of sympathetic and parasympathetic (vagal) efferents are normally under the continuous influence of and modulation by sympathetic and parasympathetic afferent input to central control centers.^{4,5} Therefore, blockade of both sympathetic efferent and afferent activity has significance in the overall mechanism of cardiac arrest during spinal anesthesia. We think that sympathectomy of the heart after spinal anesthesia should be thought of as a condition with the potential to develop clinically

significant vagal dominance or vagotonia. Bradycardia, asystole, and cardiac arrest from autonomic imbalance during spinal anesthesia are more likely to result if precipitating events resulting in vagal stimulation occur while cardiac sympathetic blockade exists, as reported by Thrush and Downs.¹ We agree with these authors that during spinal anesthesia the clinician should have a high index of suspicion for at-risk scenarios that could result in cardiac arrest and a low threshold for the initiation of prophylactic or resuscitative treatment throughout the perioperative period.

Robert P. S. Introna, M.D.

Anesthesiologist
rintrona@gaanes.com

John R. Blair, M.D.

Anesthesiologist
Northside Anesthesiology Consultants
Atlanta, Georgia

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