Asystole during Spinal Anesthesia in a Patient with Sick Sinus Syndrome

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Sick sinus syndrome is a syndrome encompassing a number of sinus nodal abnormalities that include: 1) persistent spontaneous sinus bradycardia not caused by drugs and inappropriate for the physiological circumstance, 2) apparent sinus arrest or exit block, 3) combinations of sino-atrial and atrial-ventricular conduction disturbances, or 4) alternation of paroxysms of rapid regular or irregular atrial tachyarrhythmias and periods of slow atrial and ventricular rates (tachycardia-bradycardia syndrome). This diversity of sinus node dysfunction is partly a consequence of a dual innervation with both sympathetic and parasympathetic components. The following is a case report describing a patient with sick sinus syndrome in whom spinal anesthesia resulted in asystole.

REPORT OF A CASE

The patient was a 79-yr-old man, height 180 cm, weight 72 kg, with carcinoma of the prostate scheduled for a transurethral prostate resection. He was previously diagnosed as having sick sinus syndrome, as manifest by persistent sinus bradycardia with an attenuated heart rate response to exercise (as demonstrated by Holter monitor recording). Other than a single episode of atrial fibrillation associated with a pulmonary embolus occurring 7 yr prior to this admission, no reported symptoms or documentation of any tachycardic episode were present. The patient never required either drug or pacing therapy for his sinus node dysfunction. The remainder of his history was non-contributory, and his only medications were diethylstilbestrol, aspirin, and persantine. Preoperative EKG revealed a sinus bradycardia with a rate of 58 bpm and an incomplete right bundle branch block; blood chemistries were normal; serum potassium was 4.5 mEq/l. Just before induction of subarachnoid block, monitoring was established with EKG, automatic arterial blood pressure cuff, and pulse oximeter; arterial blood pressure was 130/70 mmHg and heart rate was 62 bpm.

Spinal anesthesia was induced with the patient in the sitting position. A 25-gauge needle was introduced at the L3-4 interspace via a midline approach, and 80 cc. 1.5 ml of 5% solution plus epinephrine 0.2 mg was injected into the subarachnoid space. The patient was immediately placed supine, oxygen at 3 l/min flow was given via nasal cannula, and his legs were lifted into the lithotomy position. The operating room table was then tilted through various degrees of head-down position to achieve a T8 level in response to pin prick at 10 min. With the table finally in the neutral position, the operation was begun. Over the next 10 min, the heart rate gradually decreased to 48 bpm, but the arterial blood pressure remained at least 110/70 mmHg, the pulse oximeter continued to indicate an oxygen saturation in the 97–100% range, and the patient remained lucid, alert, and oriented. Within the next minute, he became pale and unresponsive; the EKG revealed a 6-s sinus pause, followed by normal sinus beats, followed by asystole. Closed cardiac massage and controlled ventilation by mask with 100% oxygen was immediately begun, and atropine 1 mg iv was administered. This rapidly led to a return of heart rate and arterial blood pressure to preoperative levels; the patient again became responsive, alert, and oriented, and no further pharmacologic intervention was required. The level of spinal anesthesia in response to pin prick was determined to be approximately T6 bilaterally at this time.

The operation proceeded uneventfully, and the patient’s recovery from anesthesia and subsequent hospital course were unremarkable.

DISCUSSION

The preceding case provides an illustration of how the interplay between a dysfunctioning sinus node and the autonomically mediated hemodynamic effects of spinal anesthesia can produce an asystolic cardiac arrest. Alternate causes for bradycardia and asystole can be reasonably well excluded in this instance. Neither hypoxemia nor hypercapnia were present, since the patient was unsedated and alert, had spontaneous, unobstructed respiration, was always receiving oxygen via nasal cannula, and always had an oxygen saturation of between 97 and 100%. Electrolyte disturbances, particularly serum K abnormalities, were not present on the blood chemistry screen performed the previous day. Significant acid-base abnormality was not present in light of a normal serum HCO3 ion and a normal respiratory pattern. No cardioactive medications were being taken, and no cardiac or anesthetic agents, other than the local anesthetic used for subarachnoid block, were administered. Further, the dose of local anesthetic used was too small to produce any cardiac effects secondary to vascular absorption. Maneuvers or manipulations that enhance vagal tone (Valsalva, traction on the peritonum, carotid sinus massage, etc.), other than those related to the spinal anesthetic itself, were not employed. Finally, myocardial ischemia was unlikely to have occurred in the presence of a normal arterial blood pressure, adequate oxygenation, and the absence...
of symptoms or morphologic EKG changes. Indeed, the most likely EKG lead to detect ischemic changes that would predispose to bradycardia is one of the inferior leads, and lead II was being continuously monitored during the course of the anesthetic. No such changes occurred.

Electrophysiologic studies have revealed that any of several different pathophysiologic mechanisms can produce the clinical manifestations of sick sinus syndrome. By measuring intrinsic heart rate, sino-atrial conduction time, and sinus node recovery time before and after autonomic blockade with atropine, propranolol, and the combination, sinus node dysfunction may be classified as secondary to: 1) an intrinsic abnormality of sinus node automaticity independent of autonomic nervous system influences, 2) an extrinsic abnormality of sinus node automaticity due to enhanced or reduced sympathetic tone despite normal intrinsic conduction, or 3) an intrinsic abnormality of sinus node automaticity masked by exaggerated sympathetic activity. Patients with sick sinus syndrome, on the basis of either of the latter two mechanisms, may be particularly vulnerable to the autonomic perturbations associated with spinal anesthesia. In and of itself, this anesthetic technique can often predispose to bradycardia, the causes of which may be twofold. First, there may be a block of the effent sympathetic cardiac accelerator fibers that arise from spinal levels T1-T5. Second, a reflexly enhanced vagal action on the heart may result from the decreased afferent activity of right atrial stretch receptors responding to decreased venous return. The superimposition of sympatholysis ascending to upper thoracic levels upon an intrinsically diseased sinus node whose automaticity is being maintained by enhanced basal sympathetic tone, or the further enhancement of vagal tone upon a sinus node whose original dysfunction is due to increased parasympathetic tone, can clearly lead to deleterious decreases in heart rate. Further, certain patients with sick sinus syndrome exhibit findings suggestive of "catecholamine dependency:" an obligatory role of catecholamines on cardiac pacemakers. The fact that plasma catecholamine levels are markedly suppressed in patients who have a spinal anesthesia level of T6 or above (corresponding to the known innervation of the adrenal medulla by spinal nerves from cord levels T6-L2) can, thus, only contribute to the decrease in sinus node automaticity resulting from a block of the cardiac accelerator fibers.

Patients with primarily intrinsic nodal disease are less vulnerable to significant autonomic modulation of heart rate, and will probably not respond dramatically to high thoracic levels of spinal anesthesia. However, patients with an appreciable autonomic component to their sinus node dysfunction are quite susceptible to the resultant alterations in sympathetic and parasympathetic tone. Nonetheless, prophylaxis for bradycardia during spinal anesthesia in patients with sick sinus syndrome is not indicated, because 1) in the absence of electrophysiologic testing, the subcategory of sick sinus syndrome cannot be determined, 2) a spinal anesthetic level that does not result in sympatholysis of the upper thoracic dermatomes will usually not yield clinically important slowing of heart rate, 3) deleterious increases in heart rate in a primarily elderly patient population and the potential for subsequent myocardial ischemia and congestive heart failure may occur, and 4) close observation of EKG trends, coupled with an awareness of the potential for bradycardia in this setting, will allow for early intervention.

In conclusion, while sinus bradycardia with normal arterial blood pressure is generally not a cause for alarm or treatment (indeed, it is beneficial in terms of reducing myocardial oxygen demand), progressive sinus bradycardia in the setting of sick sinus syndrome and spinal anesthesia warrants heightened awareness and, possibly, earlier intervention. In the patient described above, atropine was effective in restoring pacemaker activity to the sinus node. The rapidity in which asystole may develop makes close attention to EKG trends especially essential.

References