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Psychogenic Cardiac Arrest during Extensive Sympathetic Blockade

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Extensive epidural or spinal blockade alters the balance between the sympathetic and parasympathetic nervous systems. When this occurs, situations that increase parasympathetic tone may be more likely to result in clinically significant vagal effects, such as bradycardia. A case is described in which a young athlete suffered emotionally induced asystole during an epidural blockade.

REPORT OF A CASE

A 75-kg, 30-yr-old male physician presented for arthroscopic examination of a knee that had been injured by chronic abuse during a wide variety of athletic endeavors. Past medical history was unremarkable. Physical examination revealed a well-developed young man, 180 cm tall, with an arterial blood pressure of 110/65 mmHg and a heart rate of 56 bpm that was felt to be due to a training effect.

After the patient's arrival in the operating room unpremedicated, an epidural block was administered at the third lumbar interspace, with injection through a Tuohy needle over an 8-min period using a careful test-dosing procedure. A combination of bupivacaine 0.5% (150 mg) and fentanyl 50 μg was employed, resulting in a sensory level at the third thoracic dermatome. No intraoperative sedation was employed, since the patient wished to view the procedure on a video monitor and discuss any pathologic condition with the surgeon.

The arthroscopy proceeded uneventfully with the patient and his wife (a resident in OB-GYN) observing the procedure. Approximately 35 min after skin incision, the surgeon began to discuss the severe pathology found, and the fact that reconstructive surgery could be attempted but would be unlikely to improve the poor prognosis. The patient was informed that absolute curtailment of athletic activity would be necessary to prevent further deterioration of the joint. At this point, in response to the surgeon's asking whether he should proceed with further surgery, the patient turned to his wife, expressed disappointment, asked her for her opinion, suddenly developed a bradycardia to a heart rate of 30 bpm from a prevailing rate between 70 and 80 bpm, became asystolic, and lost consciousness.

Following two precordial "thumps," intravenous administration of a total of 2 mg atropine in 1-mg aliquots over 1 min, controlled ventilation with a FI_{O_2} of 1.0 *via* a mask, and external cardiac compression, spontaneous cardiac rhythm (nodal tachycardia) and respiratory effort returned within 2 min. Consciousness was regained rapidly thereafter, and the surgical procedure was concluded without further event. Upon rechecking, the sensory level was found to be unchanged from that measured immediately before surgery began.

A 12-lead electrocardiogram performed in the recovery room showed no evidence of dysrhythmia or myocardial damage. The patient's memory of the event was of feeling very disheartened about the news given to him by the surgeon and then losing consciousness. After discharge from the hospital on the following day, cardiography was performed for a 24-h period by Holter® monitor. This revealed a

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resting heart rate of approximately 45 bpm, with occasional episodes of bradycardia between 35 and 40 bpm.

Discussion

A reduction in resting heart rate is one of the typical physiologic adaptations to recurrent, strenuous physical activity. Whether this results from an increase in parasympathetic influence or a decrease in sympathetic effect is controversial.[1,2]

The term "vagotonia" is commonly employed to describe the clinical situation present in approximately 7% of the population manifested by resting bradycardia, first- or second-degree AV block, or complete AV dissociation. Well-trained athletes account for a large number of the people with this condition. In these individuals, prolonged sinus arrest can occur when procedures known to increase vagal activity are performed.[3]

Psychologic stress is associated with autonomic nervous system activity, and can produce increases in either sympathetic tone or parasympathetic tone. The actual human cardiovascular response to stress is environmentally determined: when stress occurs in a passive-coping environment (*i.e.*, a situation in which the subject has no opportunity to modify his position), the resultant cardiovascular effects are predominantly vagally mediated.[4] This is in contrast to the "active-coping" environment, in which subjects can influence the situation through performance. Here, cardiovascular effects are mostly the result of sympathetic, specifically, beta-adrenergic, activity.

In general, increases in resting vagal tone produce cardiovascular benefit by reducing myocardial oxygen consumption and increasing diastolic filling time. Anesthetics can contribute to the balance between sympathetic and parasympathetic cardiovascular effects. Epidurally administered local anesthetics can produce a level of sympathetic blockade much higher than the level of sensory loss.[5] In a recent study of the cardiac effects of morphine administered into the thoracic epidural space, significant reductions in heart rate, atrioventricular-nodal conduction time and refractoriness, and dP/dt_{max} were measured, even though there was no concomitant use of local anesthetics.[6] These effects were completely reversed by bilateral vagotomy and, therefore, the electrophysiologic effects of epidural morphine were presumed to be the result of an increase in parasympathetic tone, perhaps due to activation of specific spinal opioid receptors.

When vagal tone is not effectively opposed by modulating effects of the sympathetic nervous system, as occurs during epidural blockade to a high thoracic level, the potentially beneficial effects of a reduced heart rate may be overshadowed by the risk of life-threatening bradycardia that can progress to sinus arrest. If a further increase in vagotonia occurs for any reason, the likelihood of significant bradycardia is heightened.

Our patient was not sedated, and was resuscitated with relative ease, despite the presence of a high level of sympathetic blockade. The relationship between high spinal anesthesia, excessive intraoperative sedation, and unexpected cardiac arrest is well known.[7] Often overlooked, however, is the potential for difficult or failed resuscitation resulting from peripheral vasodilation with a consequent reduction in cardiac filling. Here, alpha-adrenergic stimulation can improve venous return and enhance the effectiveness of external cardiac compression.[8]

In summary, we presented a case in which a patient with increased vagal tone received an epidural anesthetic and, in conjunction with emotional stress, developed bradycardia and subsequent asystole. We feel that the possible etiologic factors discussed in the report were probably additive, and would caution against discussing poor prognosis with conscious patients in whom a high level of sympathetic blockade has been produced.

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