Editorial

During Spinal Anesthesia, Bradycardia may occur.

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An elective knee arthroscopy was planned for a 20-year-old female athlete. Only the history of fainting was remarkable in the medical history. At the L4-5 interspace, a spinal anaesthetic containing 60 mg lidocaine 2 percent plain was administered. The patient chose to watch the video display instead of being sedated and stayed fully awake. The patient began to feel nauseous thirty minutes after the spinal was injected. Her heart rate had dropped to 28 beats per minute from 50 beats per minute at the start. The patient went into asystole all of a sudden. Despite positive pressure ventilation and 100% oxygen supplied via a mask, she soon became cyanotic. During spinal anaesthesia, this patient's heart rate dropped suddenly. Bradycardia can be harmful if it occurs during spinal anaesthesia. Both anesthesiologists should have a plan in place to recognise patients who are at risk for bradycardia and treat them right away if it happens. Unfortunately, despite the fact that all 14 patients in this study were resuscitated intraoperatively, six of them developed serious neurologic deficits and died in the hospital. The other eight had significant neurologic consequences. Despite the fact that all of the patients were stable, resuscitation proved difficult, with poor neurologic outcomes. As a result, despite its rarity, this complication can go unnoticed and have catastrophic consequences.

EDITORIAL

The most common form of bradycardia, which occurs during spinal or epidural anaesthesia, is non-threatening, takes a long time to develop, is not associated with significant hemodynamic changes, and is simple to treat. Most patients with this form of bradycardia will experience a rise in heart rate after taking atropine or ephedrine. If phenylephrine or some other isolated agonist is used to treat hypotension, Bradycardia is often exacerbated. Bradycardia that is more sudden or extreme should be treated as soon as possible. If given early, before cardiac output drops significantly, a direct-acting -agonist is required and usually effective. The role of early intravenous epinephrine administration in bradycardia cardiac arrest was discussed previously, and we've also spoken about how to use epinephrine to treat or avoid bradycardia. When epinephrine was compared to phenylephrine during epidural anaesthesia, it resulted in higher cardiac output, stroke volume, cardiac index, and a more efficiently regulated heart rate. It is difficult to overstate the importance of prompt availability and initiation of pharmacologic therapy.

Epinephrine is regularly diluted to 4 g/mL and drawn up in a syringe for immediate availability at the author's institution. Also available should be atropine, ephedrine, and isoproterenol. Finally, as with any major regional anaesthetic, airway control and positive pressure ventilation equipment should be available.

Routine spinal or epidural anaesthesia has been related to sudden and extreme bradycardia or asystole. To identify variables that place patients at an increased risk, prospective, controlled studies are needed. Medical, socioeconomic, and anaesthetic factors are among them. Treatment, which is initially aimed at the cardiovascular system, must be started as soon as symptoms appear or as soon as bradycardia or asystole is detected. A resuscitation with a poor outcome will result from even a brief delay in providing proper therapy.

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