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Ballistocardiography Complicating Tympanoplasty

To the Editor:—We recently anesthetized a healthy 9-yr-old, 30-kg boy with chronic otitis media and perforated tympanic membrane for tympanoplasty. The child underwent induction and maintenance of anesthesia with halothane, nitrous oxide, and oxygen. Intubation was performed under deep halothane anesthesia following placement of an intravenous catheter. Ventilation was controlled without a muscle relaxant. After 2.75 h, with inspired halothane at 1.2%, the nitrous oxide was discontinued in preparation for placement of the graft on the perforated tympanic membrane. Fifteen minutes after stopping the nitrous oxide, the heart rate increased from 105 to 125 beats per min without a change in blood pressure. Five minutes later the surgeon noted that there was a disruptive "bouncing" of the surgical field under the microscope. The patient had been placed with his head on what is normally the foot of the bed in order to allow the surgeons to be seated comfortably and operate with the microscope. The operating table and the microscope were checked for stability, and the table was weighted with sandbags in an attempt to damp the distracting movement under the microscope.

Upon further inspection by a senior anesthesiologist, the movement of the field was seen to coincide rhythmically with the heart rate. A ballistocardiogram, in effect, was being observed in the movement of the patient under the microscope. Since the patient had not been forcibly coupled to the operating table, his body was free to respond to cardiac ejection—*i.e.*, to produce the ballistocardiogram. The patient was given 0.5 mg/kg esmolol, which decreased the heart rate from 125 beats per min to 105 beats per min, and the distracting "bouncing" under the microscope stopped. No further β-adrenergic blockade was necessary because the operation finished in approximately 20 min.

Ballistocardiography was a technique employed until the early 1970s to measure cardiac output and force of contraction. In fact, it was used regularly to monitor the rejection of the cardiac homograft during early human cardiac transplantation.[1] The ultra-low-frequency (ULF) ballistocardiogram is based on measuring the reactive movement of the human body caused by ejection of blood from the heart with each heart beat. The force of contraction is directly proportional to the contractile state of the heart, and in young children with healthy and efficient ventricles, the ballistocardiogram may be evident when standing on a bathroom scale (the pointer moves slightly with each heart beat) and when lying in bed at night (children may mention that the room moves rhythmically). After the nitrous oxide was discontinued, our young patient demonstrated an increase in the contractile state of the heart, which decreased his anesthetic depth and increased his cardiac contractile state. The short-acting, effective β-blocker esmolol alleviated this phenomenon and allowed surgery to continue unencumbered.

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A Complication Following Prophylactic Blood Patch: Spinal or Subdural Anesthesia?

To the Editor:—Leivers reports a complication, apparent total spinal anesthesia, which he attributes to the performance of a prophylactic epidural blood patch.[1] Leivers hypothesizes that the pressure of the epidural blood forced cephalad a sufficiently large volume of lidocaine-containing cerebrospinal fluid to produce total spinal anesthesia.

I would like to suggest subdural anesthesia as an alternate mechanism. Suppose the tip of the epidural catheter migrated into the subdural space between the penultimate and final lidocaine doses. Subdural migration may have been more likely because of the previous dural puncture.[2,3] The patient would then have received 10 ml 1.5% lidocaine with 1:200,000 epinephrine 37 min before the apneic episode. Leivers's case report sounds suspiciously similar to Massey Dawkin's description of "massive extradural" (*i.e.*, subdural) anesthesia: "All goes well for about 20 min. Then respiration slowly fails; the pupils dilate, but the blood pressure does not fall. . . . Assuming that 2% lidocaine was used, the patient suddenly wakes up after 1¾ h, resumes breathing,

and appears perfectly normal. This sequence of events is quite dissimilar from that of a total spinal, where apnea occurs in 2 min with dilation of the pupil and an unrecordable blood pressure; and where on recovery it takes 30 min from the first faint chin tug to the time when the jaw can be left unsupported."[4] Leivers's patient regained consciousness 115 min following the last dose of lidocaine.

I agree with Leivers's suggestion that performance of a prophylactic epidural blood patch be postponed until the epidural anesthetic has resolved, for the patient is then able to report back pain if too much blood is injected too quickly. It is possible that the blood injection hastened or worsened the apparent subdural anesthetic. However, it is not clear that the blood injection caused the reported complication.

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In Reply:—I agree with Dr. Leighton that the possibility of subdural injection of local anesthetic in this case[1] had to be considered. The fact remains, however, that the sudden rapid increase in the level of a previously stable block occurred immediately following the blood injection. In addition, if the catheter had migrated into the subdural space, it is unlikely that the injection of 15 ml of blood would have been subsequently so benign. The respiration did not "slowly fail" but ceased abruptly. Massey Dawkins[2] gave only a general description of his "massive extradural," but it is possible that some of his 28 cases would now be considered to be examples of subdural injection. Sykes[3] earlier had reported details of a case which coincide more with the typical subdural injection. In all instances the progress was more protracted than in my case.

Recently, another patient experienced a sudden increase in the level of a previously stable epidural block, when 10 ml of saline with 5 mg morphine was injected. This patient had also had an accidental dural puncture prior to the establishment of satisfactory analgesia *via* an epidural catheter. In similar circumstances, I suggest that the mechanism postulated in my case report should be considered as a possible alternative explanation to subdural injection.

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Value of Spinal Block in Central Pain

To the Editor:—The recent report by Crisologo *et al.*[1] concluded that the value of spinal block in making the diagnosis of central pain is questionable. This conclusion is not warranted, although the authors are correct to the extent that the interpretation of the effect of spinal blocks may be difficult. The use of spinal block to aid in the diagnosis of central of psychogenic pain is still useful in the absence of a positive response (relief of pain). However, a positive response including "cure" may still be entirely consistent with a central pain diagnosis. Whenever lidocaine (or any local anesthetic) is used, any conclusion made from a positive response must be made cautiously, since systemic absorption can cause pain relief in a wide variety of painful disorders.* In order to definitely conclude that the effects of the injection are due to the local effects of the block, one would need to compare the results to the results obtained after systemic injection (with equivalent serum lidocaine concentrations) without noticeable nerve block. Such a test is impossible to perform in a patient who remains improved after spinal block; in this instance the test is really a moot point.

In addition, the cases cited that did have a positive response could very well have had a peripherally mediated, sympathetic maintained pain syndrome since these have been described after strokes.[2]

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