(all of which are consistent with a high anesthetic level with secondary respiratory impairment) were the initial clues to the eventual cardiac arrest in 93% of the cases. Instead of invoking some "poorly understood potential" of spinal anesthetics to cause the cardiac arrests in these patients, we suspect that these patients developed high levels of spinal anesthesia that lead to hypotension, bradycardia, and respiratory embarrassment. When these well-known complications were not promptly detected by the anesthetist, cardiac arrest rapidly ensued. Thus, one need not invoke mysterious physiological explanations in these cases, but, rather, the cases reported by Caplan *et al.* most certainly represent the consequences of a lack of vigilant monitoring on the part of the anesthetist.

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# Cardiac Arrest during Spinal Anesthesia. III.

*To the Editor:*—Caplan *et al.* are to be congratulated for attempting to improve the anesthetic care our patients receive through the use of a unique resource—the American Society of Anesthesiologists (ASA) closed claims data base. In their manuscript, they theorize that ". . . spinal anesthesia—conducted under routine conditions and in a standard manner—carries a poorly understood potential for sudden cardiac arrest and severe brain injury in healthy patients."[1] Keats suggested in the accompanying editorial that their review of a "collection of rare events" characterized a previously undescribed "phenomenon."[2] We believe two related questions need to be answered before accepting the conclusions of Caplan *et al.* First, is this a clinical reality, or a result of the retrospective methodology of closed claims analysis? Second, did Caplan *et al.* really uncover "sudden" cardiac arrests, or did the attending individuals *suddenly recognize* the cardiac arrests?

Caplan *et al.* state that anesthetic care was adequate in all 14 patients, and believed they were able to make "an accurate assessment of vigilance" from the documentation available. This speculation is the primary shortcoming in their methodology, and clouds their analysis. An example illustrating the difficulties of assessment of vigilance from retrospective documentation is the dichotomy in adequacy of care when one evaluates the pre- and post-arrest care the 14 patients received. If pre-arrest care was adequate and appropriately vigilant, why did it take 7.5 ± 6.2 min to first administer epinephrine after the patients' cardiac arrests were recognized? It is perplexing that these "vigilant anesthetists" took that amount of time to administer epinephrine. It emphasizes that Caplan *et al.* seem to have uncovered inadequate resuscitations, rather than a new phenomenon, since potential for profound bradycardia during spinal anesthesia has been recognized for years.[3]

We have been aware of the potential for cardiac arrest during seemingly uncomplicated spinal anesthesia for a number of years, and have been observing patients for similar patterns of cardiovascular change. Three patients at our institution in the last year have appeared to exhibit rapid decreases in heart rate during spinal anesthesia. Figure 1 details that, in each of our three patients, the profound decrease in heart rate appeared to be a sudden event when viewed over a short time interval (the expanded circles), when, in fact, each patient's heart rate had decreased for many minutes prior to the profound change. Each of the profound bradycardias was treated by intravenous atropine (0.4 mg) without adverse outcome. This pattern of heart rate change in our patients following spinal anesthesia hints to us that the suddenness of cardiac arrest in the patients in the review of Caplan *et al.* may have mimicked this pattern, and was really the *suddenness of recognition.*

During the years 1982–1987, 10,080 spinal anesthetics were performed at our institution, without an episode of cardiac arrest resulting in neurologic injury. We believe this is most likely related to our vigilance during the technique, and a willingness to utilize intravenous atropine (0.4–0.6 mg), ephedrine (25–50 mg),

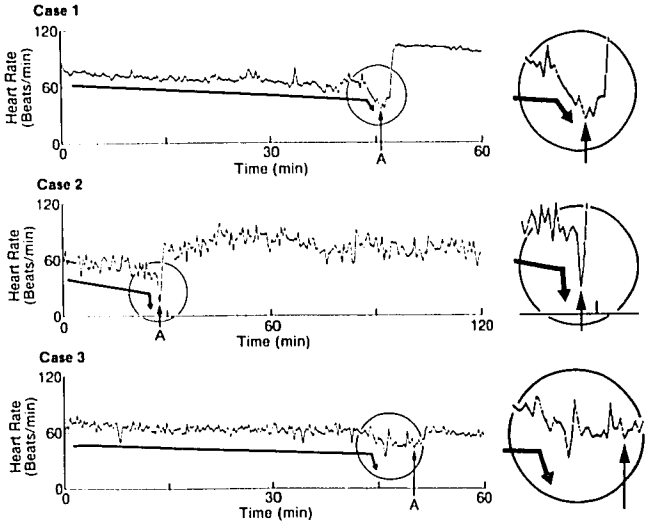


FIG. 1. Heart rate patterns in three patients (cases 1–3) following spinal anesthesia. The traces were obtained by using trending feature of electrocardiography monitors (Model #90603/90651, Spacelabs Inc, Redmond, WA). The enlarged circle seems to indicate that the profound bradycardia was a sudden event; however, when a longer view is taken, the decrease in heart rate was more gradual. Please note that the time scales for case 1 and 3 are similar, while case 2 was printed over a 2-h interval. A = administration of 0.4 mg of iv atropine.

and epinephrine (0.2–0.3 mg) in stepwise escalation of therapy when profound bradycardia develops following spinal anesthesia. Indeed, there are situations where epinephrine may be the most appropriate first choice.

In conclusion, thanks to Caplan *et al.*, the catastrophic possibility of bradycardia during spinal anesthesia has once again been emphasized, and the need for its prompt recognition and treatment stressed. We agree wholeheartedly with the two principal points they make in the paper's abstract, *i.e.*, "unappreciated respiratory insufficiency may have played an important role" in the cardiac arrests, and that there may be an "interaction between sympathetic blockade during high spinal anesthesia and the mechanisms of cardiopulmonary resuscitation." However, their implication that, in spite of vigilant anesthetists, fatal cardiac arrests can occur *unexpectedly* and rapidly during spinal anesthesia is misleading. This implication may discourage the appropriate use of a technique with a long history of safety. We would emphasize that the ASA motto of "vigilance" is as important during regional anesthesia as during general anesthesia.

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