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Cardiac Arrest during Spinal Anesthesia. I.

To the Editor:—With apologies to Drs. Keats and Caplan *et al.*, there is nothing new about hypoxia under spinal anesthesia. There is also nothing new about the respiratory effects of high spinal anesthesia or of oversedation.

The purpose of this letter is not to negate the rigorous work and time devoted by the authors. Regardless of how one interprets the data, the subject matter is so vitally important that it is always worthy of our efforts and discussion.

The case for applicability of the sentinel event theory rests upon the presence of vigilance and the validity of "worst case" time estimates.

Vigilance is one of those interesting "absolute" nouns. It is either present or absent. Vigilance cannot be "adequate." Particularly in view of the outcomes, vigilance can surely not have been present in these cases.

Worst case time estimates have a serious limitation in that they make the dangerous assumption that, at a reference time point, when all vital signs were normal, the patient was not yet hypoxic. If one accepts that this assumption may be false, these worst case estimates become valueless in defining a period of hypoxia. If these worst case estimates are accurate, we must conclude that some patients do not tolerate as long a period of hypoxia as was traditionally believed. I find it tempting to speculate that the patient receiving conscious sedation may be more at risk for a specific period of hypoxia than a comparable patient receiving inhalation anesthesia with an agent associated with some measure of brain protection.

Clinical anesthesia in the late 1980s is blessed with pulse oximetry and capnography. As important as these devices are in the setting of general endotracheal anesthesia, they probably have a more important place in settings where respiration is not controlled. The capnographic trace provides excellent qualitative respiratory monitoring when used with mask ventilation or with a nasal cannula (which is easily modified to house the capnographic port). This is a good adjunct to (not a replacement for) the precordial stethoscope.

It is our job to monitor the ingress of good air and the egress of bad. Failure to detect the inadequacy of this phenomenon means that vigilance is absent. This is a very simple concept!

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REFERENCES

1. Keats AS: Anesthesia mortality—A new mechanism. ANESTHESIOLOGY 68:2–4, 1988
2 Caplan RA, Ward RJ, Posner K, Cheney FW: Unexpected cardiac arrest during spinal anesthesia: A closed claims analysis of predisposing factors. ANESTHESIOLOGY 68:5–11, 1988

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Cardiac Arrest during Spinal Anesthesia. II.

To the Editor:—The recent paper by Caplan *et al.*[1] on cardiac arrest following spinal anesthesia attempts to elucidate the precipitating factors that lead to the poor outcome in these patients. We were disturbed, however, by the concluding sentence of the article which states ". . . we wish to emphasize 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." We feel that this statement is not supported by the data presented in the article and implies some mysterious factor that causes healthy patients' hearts to stop beating during spinal anesthetics.

A review of the data (most of which was obtained from persons directly involved in the care of these patients and likely to be biased) reveals that 12 of 14 patients had received intravenous sedation, and, in at least half of these, there was no spontaneous verbalization. Respiratory rate, which was recorded in only five cases, is not reported. Bradycardia, cyanosis, and hypotension

(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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REFERENCE

1. Caplan RA, Ward RJ, Posner K, Cheney FW: Unexpected cardiac arrest during spinal anesthesia: A closed claims analysis of predisposing factors. ANESTHESIOLOGY 68:5–11, 1988

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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),