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Cardiac Arrest during Spinal Anesthesia. IV.

To the Editor:—Caplan *et al.*[1] are to be commended for their thoroughness in reviewing the records and their superb analysis. Two of my observations in 36 yr while practicing and teaching anesthesia should be called to your attention.

First, it is assumed that T4 is the level when a patient first feels the needle prick at the nipple line. Over the years, I have personally performed or supervised the performance of cervical plexus blocks for many different operations. Curiosity led me to define the extent of all regional anesthetics. I learned a lot about nerve distribution, including the fact that cervical 4 (C4) overlaps the upper thoracic segments down to the nipple line. I have seen patients who had "T4 spinal levels" who actually had T1 levels, and one who had a C7 level. These were identified by checking the inner aspect of the arm and continuing down to and including the fingers on the one occasion. Whenever a patient first feels the needle at the nipple line, check the arm!

Second, we tend to allow the operation to begin as soon as the operative site is well anesthetized, approximately 5–10 min after injection for spinal anesthesia, and 15–20 min after that for epidural anesthesia. In the case of spinal anesthesia, the level at 10 min does not mean that the same level will be present 20 min later. The length of time to establish a "final level" varies from patient to patient, and depends on all the factors that influence the level of spinal anesthesia.

These thoughts occurred to me as I noted that the majority of the patients had bradycardia and hypotension as the initial "clues." Although my observations do not necessarily cover all the facets in this article, nor do they explain the results, future studies of this type can be enhanced if these types of observations are charted when appropriate.

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REFERENCE

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In Reply:—The purpose of our study[1] was to search for new mechanisms of anesthetic injury. Because of the limitations imposed by closed claims data and retrospective analysis, we do not expect the reader to be completely convinced by our findings and conclusions. Rather, our intent is to present data which point to new *hypotheses.* We hope these hypotheses will stimulate discussion and encourage further research in appropriate experimental models and clinical settings.

We sympathize with the common sentiment expressed in the letters by Drs. Abramowitz, Zornow and Scheller, and Brown *et al.*—it would *seem* most plausible to ascribe the poor neurologic outcomes in our study to inadequate vigilance. However, after extensive analysis of charts, anesthetic records, personal narratives, depositions, and multiple eyewitness accounts, we simply could not find data to support this view. We cannot dismiss the possibility that poor vigilance may, indeed, have played a role, but we do not wish to mislead the reader by suggesting that we found convincing evidence. In contrast, we were impressed by the recurring pattern of relatively late epinephrine administration during otherwise prompt and aggressive cardiopulmonary resuscitation. This led to our principal hypothesis that poor neurologic outcome may have been linked to an inadequate appreciation of the interaction between sympathetic blockade during high spinal anesthesia and the mechanisms of cardiopulmonary resuscitation.

We wish to assure Dr. Abramowitz that it is not our intent to denigrate the importance of vigilance in the practice of anesthesiology. In fact, we think our paper conveys just the opposite message. If cardiac arrest can occur so suddenly and unexpectedly during spinal anesthesia, then it behooves the anesthesiologist to be highly vigilant and thoroughly prepared for prompt and appropriate action. We are not as confident as Drs. Zor-

now and Scheller that the mechanisms underlying sudden cardiac arrest during spinal anesthesia are simple and well-understood. As examples, we cite two case reports. In the first,[2] which appears in this issue of ANESTHESIOLOGY, an athletic young physician underwent knee arthroscopy with epidural anesthesia. He had a sudden asystolic cardiac arrest when his surgeon informed him that the outlook for further sports activity was poor. In the second,[3] which appeared in the May, 1988, issue, a patient with clinically asymptomatic sick sinus syndrome received spinal anesthesia for transurethral prostate resection. During the 10-min interval preceding sudden asystole, he was alert, lucid, and a pulse oximeter gave no indication of hypoxemia. After a brief and successful resuscitation, pinprick testing revealed bilateral sensory blockade to T_6.

We direct the reader's attention to several valuable comments contained in these letters. Dr. Brown and colleagues at the Mason Clinic report an impressive record of safety in the conduct of over 10,000 spinal anesthetics. Of note, this group advocates the early use of epinephrine in the management of profound bradycardia during spinal anesthesia. This approach is consistent with the principal hypothesis that we advance. Dr. Abramowitz suggests that the risk of hypoxia may be greater during conscious sedation than during general anesthesia. This is an interesting question that is currently under investigation in the ASA Closed Claims Study. Finally, Dr. Jones offers practical clinical advice about the timing and detection of high spinal anesthesia.

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Hypoxia and Anesthetic–associated Liver Injury in Guinea Pigs

To the Editor:—In the recently published work by Hursh *et al.*[1] concerning hepatic oxygen supply in guinea pigs during anesthesia, it was shown that halothane decreases hepatic oxygen delivery to a much greater extent than does isoflurane. It was also demonstrated that, in the guinea pig, a very small fraction (2%) of the total hepatic blood flow was contributed by the hepatic artery as compared to other species where the fraction is 20–35%.

From these data, it was inferred that the guinea pig is highly susceptible to hypoxic insult to the liver, and that the hepatic lesion observed in guinea pigs following halothane anesthesia[2,3] may be the result of "hepatic oxygen deprivation *per se* without a direct involvement of halothane metabolism."[1] If a hypoxic mechanism is the culprit behind the observed halothane-associated hepatotoxicity in the guinea pig model, then it should be possible to magnify the severity of the lesion and to duplicate it with other anesthetics by utilizing severely hypoxic (8–10% O_2) exposure conditions. However, exposure of inbred strain 13 guinea pigs to 1% v/v halothane and 10% O_2 for 4 h produced no increases over animals breathing 40% O_2 in either the incidence of centrilobular necrosis or the severity of injury (table 1). The hypoxic stress to the livers of guinea pigs breathing 10% O_2 during halothane exposure was obviously much greater, as indicated by halothane metabolite levels in the plasma. With hypoxia, plasma concentrations of fluoride ion, indicative of reductive (lack of oxygen) biotransformation of halothane, increased four-fold, while levels of the oxidative metabolite, trifluroacetic acid (TFA), decreased three-fold. Total halothane metabolism, indicated by plasma levels of bromide ion, was identical between the groups. An additional indication of the severity of hypoxic stress to the livers of animals breathing halothane and 10% O_2 is that three of eight animals demonstrated extensive areas of panlobular coagulative necrosis within the