Correspondence

Anesthesiology
47:75, 1977

Malignant Hyperthermia during Regional Anesthesia

To the Editor:—I would like to correct a statement made by Dr. Wadhwa that the malignant hyperthermia syndrome has not been previously reported to occur in association with regional anesthesia.[1] We reported a case of a patient who survived one episode of malignant hyperthermia under general anesthesia and who had a somewhat attenuated but otherwise similar episode during subsequent spinal anesthesia.[2] At least one other case of malignant hyperthermia occurring during local anesthesia has been described, although not documented in detail.[3]

These case reports, along with that of Dr. Wadhwa, are in support of my general impression that the syndrome of malignant hyperthermia may be triggered by a host of nonspecific stresses in addition to the pharmacologic stimuli that have been identified in the laboratory. Indeed, it has been suggested that anxiety alone may induce an episode of malignant hyperthermia.[4] Dr. Wadhwa's point is well taken, "regional techniques . . . are not foolproof."

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REFERENCES

1. Wadhwa RK: Obstetric anesthesia for a patient with malignant hyperthermia susceptibility. ANESTHESIOLOGY 46: 63–64, 1977
2. Katz JD, Krich LB: Acute febrile reaction complicating spinal anaesthesia in a survivor of malignant hyperthermia. Can Anaesth Soc J 23:285–289, 1976
3. Britt BA: Recent advances in malignant hyperthermia. Anesth Analg (Cleve) 51:841–850, 1972
4. Wingard DW: Malignant hyperthermia: A human stress syndrome. Lancet 1450–1451, 1974

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More Than V_5 Needed

To the Editor:—Drs. Kaplan and King[1] have properly stressed the importance of lead V_5 of the ECG for detecting the occurrence of myocardial ischemia during anesthesia. While we have been using this for some time in open-heart procedures, a potential danger exists if one assumes no other lead need be monitored. Lead V_5 is expected to evaluate the anterior–lateral surface of the heart effectively, and often shows changes more clearly than leads I or AVL. In contrast, the inferior surface of the heart (supplied to a large extent most often by the right coronary artery) is readily evaluated with a tracing of leads II, III, or AVF,[2,3] but frequently not with lead V_5.

We want to emphasize this with the attached cardiogram (fig. 1) obtained as a continuous strip from a patient who had a period of myocardial ischemia following cardiopulmonary bypass for valve replacement. No evidence of ST change is evident from the V_5 lead, but all the inferior leads show marked ST elevation. These subsequently improved with continued close supportive care. While V_5 is indeed a valuable monitor, one must not neglect to check other leads frequently, especially leads II, III, and AVF.

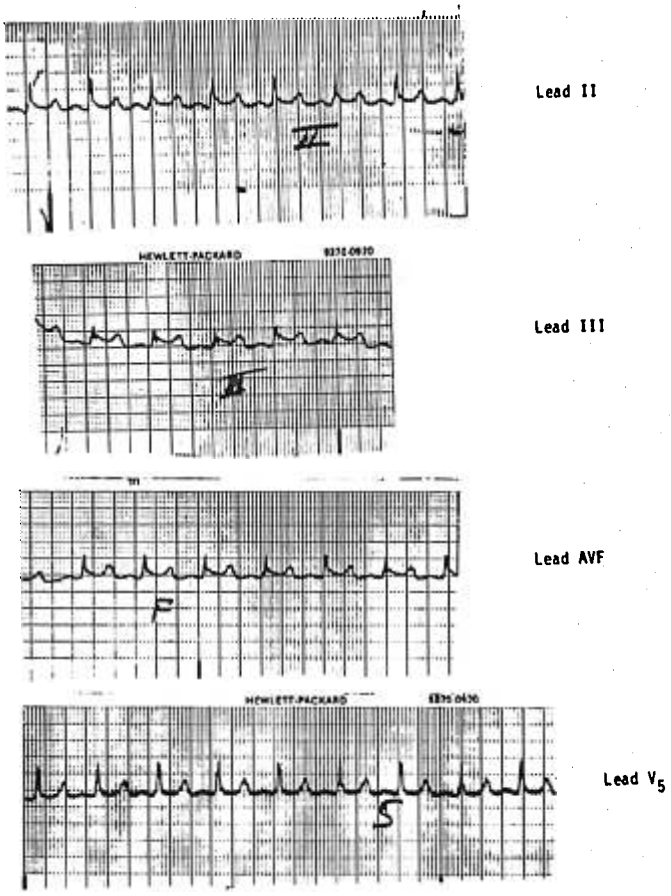

FIG. 1. Cardiogram from patient who had myocardial ischemia after cardiopulmonary bypass.

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REFERENCES

1. Kaplan JA, King SB: The precordial electrocardiographic lead (V_5) in patients who have coronary-artery disease. ANESTHESIOLOGY 45:570–574, 1976
2. Hurst JW (editor): The Heart: Arteries and Veins. New York, McGraw-Hill, 1974, p 312
3. Goldman MJ: Principles of Clinical Electrocardiography. Los Altos, Lange, 1970, pp 158–188

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Are Effects of Halothane on Hepatocytes "Pathologic"?

To the Editor:—As an author of an article that was cited in the recent special article by Chang and Katz,[1] I wish to raise some concerns regarding interpretation of our studies related to the liver. In our paper we reported hepatic morphologic changes after repeated exposures of rats to halothane, .25 per cent.[2] In hepatocytes of treated rats, smooth endoplasmic reticulum, lipid droplets and microbodies increased, in addition to other minor alterations. None of these alterations was regarded as a "lesion" or "hepatic damage," nor were they described as such in our paper. Therefore, I should like to record my objection to the reference by Chang and Katz that we reported halothane induced "lesions." In my opinion, the changes we recorded should be regarded as reversible and as an adaption to the administration of the xenobiotic.

Another area of concern is that of cell sampling. In their original report, Chang *et al.* showed electron micrographs of abnormal hepatocytes at moderately high magnification.[3] In the text of that paper they made several references to "many" relatively normal cells. In their special article, Chang and Katz published micrographs that are also at moderately high magnification, and all clearly show the existence of abnormal cells. The reader is unable to gain an impression of the frequency of abnormal cells. Since, in any tissue, some cells that are abnormal, dying or poorly fixed, are always found, the omission of any mention of sampling methods or number of cells sampled is disturbing. One way to deal with this problem is to use lower magnification or even light microscopy for orientation of the reader. A good example of the effective use of light microscopy appears in the same issue of ANESTHESIOLOGY in the report by Sipes and Brown.[4] The light micrographs presented there provide clear evidence of the extent of cellular damage.

In studies of the effects of anesthetics on organ function or organ change, the effects of the anesthetic state should be separated from the effects of the anesthetic drug *per se* to the extent that this is possible. Could the observed cellular effects be due to the effects of altered feeding, intestinal absorption, etc.? Were the terminal weights of the treated animals comparable to the terminal weights of the untreated controls? Data to answer these questions are not provided. Finally, most morphologic studies can be considerably strengthened and interpretation simplified when they are accompanied by simultaneous measurement of other indices (*e.g.*, correlation of biochemical and structural changes).

In their conclusion, Chang and Katz acknowledge that their findings are still subject to critical debate. I agree.

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REFERENCES

1. Chang LW, Katz J: Pathologic effects of chronic halothane inhalation: An overview. ANESTHESIOLOGY 45:640–653, 1976
2. Ross WT, Cardell RR: Effects of halothane on the ultrastructure of rat liver cells. Am J Anat 135:5–22, 1972
3. Chang LW, Dudley AW, Lee YK et al: Ultrastructural studies of the hepatocytes after chronic exposure to low levels of halothane. Exp Mol Pathol 23:35–42, 1975
4. Sipes GI, Brown BR: An animal model of hepatotoxicity associated with halothane anesthesia. ANESTHESIOLOGY 45:622–628, 1976

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