Platelets and Malignant Hyperthermia

To the Editor:—In their recent paper, Lee et al.1 raised concern that the platelet may not be affected by malignant hyperthermia. However, the previous work of Basrur et al.2 has established the presence of morphologic changes in platelets of pigs specifically related to malignant hyperthermia. These observations have recently been extended to humans by O'Toole et al.3 It should also be noted that the five-fold higher centrifugal force and the smaller amount of heparin used by Lee et al.1 deviate appreciably from the method of Solomons and Masson.4 These differences are reflected in the high-performance liquid chromatography (HPLC) chromatogram of Lee et al.,1 where the platelet extract gave a response ten-fold weaker than that of the preparations of Solomons and Masson.4 This suggests an unacceptably poor yield of platelets or a selective platelet subpopulation being studied. Because Lee et al.1 chose to eliminate the internal standard from their HPLC runs in order to save time, they could not alert themselves to the low adenosine triphosphate (ATP) levels relative to the recommended standard. This then led to the use of the HPLC detector at near maximum sensitivity with unnecessarily excessive baseline noise relative to adenosine monophosphate and hypoxanthine seen in the published chromatogram. In another variation of technique, Lee et al.1 have neutralized their perchloric acid (PCA) extracts with KOH. In our hands precipitation of potassium perchlorate can absorb variable amounts of purines and is not recommended. Storage of the extracts is also not advisable. When these and other conditions explicitly stated in detail by Solomons and Masson4 are adhered to, consistent results were found both at sea level and 3,000-mile altitude. Lee et al.'s group of patients are not homogeneous and have widely variable and incomplete clinical symptoms as the basis for the diagnosis of malignant hyperthermia.

In summary, differences in analytical technique preclude the comparison of the work of Lee et al.1 with that of Solomons and Masson,4 and independent evidence suggestive of the involvement of the platelet in malignant hyperthermia is available.

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REFERENCES


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In reply—Dr. Solomon's claim that both our group and Kaplan's group were unable to repeat his work is correct. His claim that this irreproducibility is due to differences in technique is incorrect.

Interpretation of platelet morphology is currently filled with controversy. An excellent paper on the subject is recommended.1

Currently, the platelet contractile mechanism is believed to be different from that of muscle. Thrombin is believed to be the physiologic stimulus to platelet aggregation and contraction, while electrical stimulus and acetylcholine are the stimulus for muscle contraction. Moreover, the skeletal muscle filaments of actin and myosin are highly organized, while those filaments in platelets seem to be reticular prior to contraction and possibly radial postcontraction. We are dealing then with contractile mechanisms of two different natures dependent on two different physiologic stimuli.

The platelet fraction used by Solomons and Masson is not just large-size platelets, but also contains erythrocytes,