Pulmonary artery (PA) pressure monitoring was indicated for proper IV fluid management during anesthesia and surgery. It was planned to insert a Swan-Ganz® catheter into the right internal jugular vein. The initial identifying venipuncture was made with a 2.5-cm 22-gauge needle attached to a 5-ml syringe. The needle was advanced lateral to the carotid artery, toward to the right ipsilateral nipple, and at about 30-degree angle to the skin. After the vein was located, a 16-gauge needle with syringe was inserted. Two attempts to cannulate the internal jugular vein failed. Finally, the needle was advanced parallel to the trachea. Aspiration of bloody fluid occurred momentarily, but subsequently nothing was aspirated. The inner needle was removed, and a soft guide wire was replaced and threaded. The dilator insertion was facilitated by the wire with no difficulty. A Swan-Ganz® catheter was threaded into the dilator and advanced about 20 cm from the skin. Aspiration was negative at this point. When the balloon was inflated with 0.8 ml air, suddenly the patient’s right leg was twitched once. Then an accidental insertion of the Swan-Ganz® catheter into intrathecal space was suspected. A chest roentgenogram was immediately performed, which showed the catheter in the spinal canal. Examination of the fluid aspirated through the catheter revealed the following: specific gravity 1.008, protein 15 mg/dl, cell 3/3, and glucose 57 mg/dl. The Swan-Ganz® catheter was extracted. After this episode the patient was examined thoroughly by neurologists. Although motor neuron function was intact, sensory neurons were slightly injured since bilateral hypalgesia at T1–T3 was recognized. The nerve injury improved progressively, and no neurologic deficit was observed at the third postoperative day. She was discharged without any neurologic deficit 20 days after the episode.

This complication occurred because proper judgment or correct practice was not used. A guide wire never should be advanced unless venous blood flows freely. The error is surely predictable when imprudent practices are followed. Another important problem is the indication of PA monitoring. The decision to catheterize obviously should be stringent. This case adds another complication associated with insertion of a Swan-Ganz® catheter.

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Low Molecular Weight Proteins in Human Malignant Hyperthermia Muscle

To the Editor.—We wish to comment on a laboratory report recently published in ANESTHESIOLOGY describing two low molecular weight proteins in skeletal muscle from malignant hyperthermia (MH) susceptible humans not found in controls. In response to an earlier abstract by the same group, we began similar studies in hopes of confirming their results. Using an approach very similar to that used by Blanck et al., we also observed the two “novel” protein bands; however, we observed the proteins in preparations from both control and MH-susceptible patients. Our study concluded that the two low molecular weight protein bands reported by Blanck et al. might be the result of serum contamination, as the molecular weights of these bands corresponded to those of the alpha and beta chains of hemoglobin.

Modifying our method of sample preparation to include a 1-h 15,000 X g centrifugation step (Eppendorf microcentrifuge), testing several different buffers (sucrose–EDTA, TRIS–EDTA, Krebs), and including four protease inhibitors during sample preparation has not altered our findings (unpublished observations). The two low molecular weight proteins were still present in vastus lateralis from all control (n = 4) and MH-susceptible (n = 4) patient samples; however, we have made no attempt to quantify the proteins. Due to the lower centrifugal force used in our studies, our preparations may have contained a greater contamination by microsomes than the preparations of Blanck et al. However, none of the studies to date, including ours, have determined the degree of microsomal contamination in the “soluble protein” fraction used. Based on our results, the low molecular weight proteins exist in normals and MH-susceptible patients. If these proteins normally are associated with the microsomal fraction, then it is possible that the proteins are freed by enzymatic activity from
these vesicles and therefore appear in the soluble protein fraction of MH susceptibles. This possibility could be addressed by Blanck et al., or others, by comparing preparations derived from the supernatant of a low-speed centrifugation (ca. 20,000 × g) with that of a much higher speed centrifugation (ca. 100,000 × g). In both cases the presence or absence of microsomes should be confirmed. There still remains the possibility of serum contamination, which is unavoidable in preparing the samples.

We do not recommend the method of diagnosis proposed by Blanck et al.1,2 until the nature of these proteins is understood more clearly.

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In reply.—We appreciate the efforts of Fletcher and Rosenberg to clarify the putative electrophoretic differences between MH and normal muscle. Although we never stated that electrophoresis should be used as a diagnostic criterion for MH, we do agree with them that the extra protein bands are probably hemoglobin. We have determined this in three ways. MH and control muscle proteins and a standard hemoglobin sample on the SDS gels were transferred to nitrocellulose paper. The transferred patterns were then stained for hemoglobin with benzidine. The benzidine reaction stains heme proteins in general, therefore, positive staining with benzidine could relate to any other heme protein as well as hemoglobin. The low molecular weight proteins from the MH sample stained positively for hemoglobin; no staining was evident in the electrophoretic sample from the control patient. The hemoglobin sample transferred to the nitrocellulose paper as expected stained positively for hemoglobin. We also took an aliquot of the supernatant from an MH and a normal control patient and directly added benzidine to them and found that the MH sample was markedly positive for hemoglobin while the control sample only showed a trace amount of staining. Gel Ioselectric® focusing of hemoglobin and the MH muscle samples demonstrated a yellowish band in unstained gels at exactly the same pI for both hemoglobin and MH samples. This further substantiates that the extra MH proteins are probably only hemoglobin contaminants. Finally, vastus lateralis samples from several more patients who have undergone orthopedic surgery have also demonstrated the extra protein found in their gel electrophoretic patterns that we had originally found only in the MH patients.

The question arises as to why the original control samples did not contain the hemoglobin contaminants that were found in the MH patients muscle? The most obvious explanation is that half of the six control patients underwent knee surgery and had a tourniquet applied to their thigh during the time when the muscle specimen was obtained. This resulted in less bleeding, and therefore less hemoglobin contamination was evident in our biopsy specimen. The other patients fortuitously had less traumatic surgery compared with our MH patients, resulting in less hemoglobin contamination.

In conclusion, electrophoresis of MH muscle samples cannot be used at this time to distinguish MH from normal muscle. Future investigations, however, may yet demonstrate differences in protein composition of MH compared to normal muscles.

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