In reply: Drs. Solomons and Masson question our methods. One of us (UG) has been working for several years with in vitro cell systems and is aware of the fragility of platelets and their metabolism. We paid great attention to their handling and performed many controls to avoid artifacts. Most of the quality controls of our technique have been described under “Experimental Procedures” and “Results” of our article but are mentioned for completeness again here.

1. Platelets of an MH-susceptible patient and one control subject always were tested simultaneously in order to depict even small differences in technique.
2. Dose and time dependency of halothane on platelets as well as its reversibility by dantrolene were used to detect minor changes between control and MH-susceptible patients.
3. Each platelet preparation and treatment group assay were performed in duplicate, showing very reproducible results.
4. EDTA was added at the end of the incubation according to Rao et al.¹ and others, although its deletion appeared not to influence our results.
5. In order to get complete recovery of nucleotides from platelets and not to have destruction (tested by the addition of nucleotide standards), the mixing and sonication time as well as strength were chosen carefully. It is understandable that the sonication step needs to be optimized in any laboratory according to the instrument used.
6. Immediate freezing of platelets before extraction of nucleotides was done as previously described by Rao et al.¹ The “cold stress” must have been minimal, since extraction and analysis of nucleotides from unfrozen platelets did not change the nucleotide levels significantly.
7. Complete recovery of adenosine nucleotides was achieved with the described extraction procedure (i.e., no nucleotides were found upon further extractions).
8. Our ATP, ADP, and AMP levels in platelets were comparable to values derived with different techniques in various other laboratories as discussed in our publication.
9. The use of external standards as we did (linearity between 0.2–20 nmol of nucleotides) is a well-accepted technique. The standard deoxyuridine or adenine added after incubation, as published in Solomons and Masson’s abstract,² does not assure complete recovery of endogenous nucleotides from platelets.
10. We and other laboratories have used this platelet model but were unable to reproduce the preliminary reports of Solomons et al.²–⁴ that have not yet been published as an original full article.

We, therefore, conclude that our methods are reliable. We await a noninvasive technique to diagnose MH susceptibility that can be reproduced in many centers.

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Catheter Doubling in Left Main Pulmonary Artery

To the Editor:—The balloon-tipped (Swan–Ganz®) pulmonary artery catheter usually is inserted percutaneously¹–³ via the jugular (IJV, EJV), subclavian (SCV), or antecubital vein. In adults, the distal end of the catheter is in the pulmonary artery (PA) at a distance of 40–45 cm from the percutaneous insertion site when inserted via IJV and SCV. The pulmonary artery usually can be occluded at a distance of 50–55 cm. We have observed
The reason for the increased incidence of doubling when the catheter enters the left PA is not known. We speculate that the more vertical and acute downward course of the left lower lobe PA may cause the catheter to double on itself. Doubling can be suspected when an occlusion pressure (PAOP) is not obtainable despite insertion of the catheter to at least 55 cm (with a functioning balloon). Advancing the catheter in the presence of doubling could result in formation of loops in the right ventricle, knotting in PA, or exaggeration of the doubling loop with pulmonary valve insufficiency. We recommend, that, if PAOP cannot be obtained after insertion of the catheter to 55 cm via IJV or SCV, the catheter should be withdrawn into the right ventricle after balloon deflation and refloated following balloon inflation.

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