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Hypertension associated with cardiopulmonary bypass

To the Editor:

In the recent article by Townsend *et al*[1] the authors conclude that the renin-angiotensin system is not the primary mediator of cardiopulmonary bypass associated hypertension, at least during fentanyl anaesthesia. In that study, as outlined in the method, all patients were anaesthetised with a single bolus of fentanyl and received halothane or enflurane if hypertension occured prior to cardiopulmonary bypass. After institutions of cardiopulmonary bypass no further anaesthestic was given. Recent studies[2,3] have indicated that fentanyl levels on cardiopulmonary bypass may fall to subtherapeutic levels very quickly. One study has demonstrated that this may be a result of binding of fentanyl to certain membrane oxygenators and siliconized tubing.[3] In the absence of any other anaesthetic agent, I question therefore whether CPB associated hypertension is hypertension associated with lack of anaesthesia.

I would suggest that any study which involved the use of fentanyl as a primary anaesthetic agent during cardiopulmonary bypass is subject to the possibility of patients having inadequate anaesthesia and massive sympathetic output may occur on that basis. Perhaps studies including fentanyl as a major component of anaesthesia during bypass should include drug levels as part of that study to demonstrate whether anaesthesia is adequate during the period of study.

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REPLY

We are grateful for the opportunity to reply to Dr. Goresky's letter. The abstracts he quotes provide convincing evidence of the sequestration of fentanyl in the lungs and in the membrane oxygenator during cardiopulmonary bypass (CPB) and explain the substantial decrease of plasma fentanyl concentration that has been observed during CPB. We use bubble rather than membrane oxygenators during CPB and, in a previous study,[1] did not see any dramatic decrease in plasma fentanyl concentration with institution of CPB.

We agree that the most likely cause of the hypertension in our patients was an adrenergic response which, perhaps, could have been diminished by administration of additional anaesthesia. However, the purpose of our study was to determine whether the renin-angiotensin system also had a causative role in CPB-associated hypertension. Our results suggest it does not.

We dispute whether it is possible to determine the adequacy of anaesthesia by measuring plasma fentanyl concentrations. When fentanyl is administered as a continuous infusion there is a tendency for higher plasma levels to be associated with a reduction in the incidence of hypertension during CPB.[1] However, the relationship is inconsistent and unpredictable and we suspect,[2] like others,[3] that it is not possible to block the response to noxious stimuli completely, at least at fentanyl doses used in clinical practice.

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Diagnosis of malignant hyperthermia

To the Editor:

Rosenberg and Gronert[1] have recently challenged the diagnosis of malignant hyperthermia (MH) as reported by Grinberg *et al.*[2] They believe that Grinberg *et al.* had insufficient data to support their contention that the episodes reported by them were MH. Rosenberg and Gronert feel that labeling a patient MH susceptible has far-reaching implications in the medical care of such patients and that the diagnosis should be made with care. It is difficult to disagree with them on this latter point.

However, to err the other way and fail to make the clinical diagnosis usually leads to a worse outcome than incorrectly labeling someone MH susceptible. There are still too many deaths occurring annually from MH. In many of these cases there has been sufficient data to make the presumptive diagnosis of MH, yet the anaesthetists fail to make the proper diagnosis for a variety of reasons. One of the most common reasons is an unwillingness to accept the diagnosis of MH. There are many anaesthetists who do not believe in the syndrome. All too frequently there is a willingness to place the blame on human error as a cause of the deaths. One of the reasons for this is that many authorities still insist that MH is a rare condition and should have a low priority in the differential diagnosis of complications of anaesthesia. In those areas of the United States where there is an emphasis on the diagnosis of MH, the mortality from it has fallen to almost zero. In other areas where there is hesitance to make an early diagnosis, the mortality rate is still quite high. These latter areas of the country seem to centre around spheres of influence which convince anaesthetists that MH has a low priority as a cause of death.

A second major reason for MH deaths is the unwillingness of anaesthetists to properly monitor their patients, particularly the body temperature. I don't understand the hesitance to use simple monitors such as those used for body temperature when many anaesthetists will go to great lengths to insert Swan-Ganz catheters, arterial catheters, etc. In the majority of the cases of MH in which there has been a major complication, there has been evidence that the patient's temperatures were rising. To my amazement, some anaesthetists are still observing body temperatures rising to extraordinarily high levels without considering the diagnosis of MH.

MH is a clinical syndrome. All of the diagnostic techniques have been developed from patients or animals who developed the clinical syndrome. To my knowledge no one has developed a test which can assure the diagnosis of MH. Until the supporters of the muscle biopsy prove that they can accurately make the diagnosis of MH in everyone, the diagnosis must be made on a clinical basis. Our primary goal must be to reduce the mortality and morbidity from MH. It is far better to overdiagnose MH than to underdiagnose it. To my knowledge there has never been a death from MH in a known susceptible patient when appropriate precautions have been used. The same cannot be said for the undiagnosed patient, or worse yet, the suspected susceptible patient who had a procedure performed without adequate precautionary measures.

Occasionally patients may be upset that they are labeled incorrectly. This is the exception rather than the rule. Usually they are more concerned that someone who is unfamiliar with MH will be taking care of them.

I am appalled at the number of anaesthetists who are unfamiliar with the MH clinical syndrome or do not even know that it exists. Likewise, it is generally not appreciated by those who are familiar with the syndrome that it is more likely to be seen and diagnosed in the postoperative period than in the operative period. In my opinion we need reports such as that by Grinberg *et al.* to help us further ellucidate the diagnosis of MH and to bring it to the attention of the medical profession. I agree with them in their reply to Drs. Rosenberg and Gronert.

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