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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, 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. However, the relationship is inconsistent and unpredictable and we suspect, like others, 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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