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REPLY

Drs. Lang and Berenbaum have argued convincingly that the incidence of cardiomyopathy that is "clinically significant" may indeed be lower than was suggested in our review article on anaesthesia for pheochromocytoma. Van Vliet's pathological study of human hearts in patients having died of the consequences of their pheochromocytomas was just that - a post mortem study.¹ We will not demur on this point and agree that the true incidence of cardiomyopathy that is of clinical significance is unknown and probably less than the incidence that is obtained at postmortem. It still behooves the clinician, however, when confronted with a pheochromocytoma patient, to look specifically for any evidence of congestive failure and also at the ejection fraction obtained echocardiographically. We still contend that a Swan-Ganz catheter should be inserted if the patient has abnormal left ventricular function, assuming that the patient's size and the anaesthetist's skill and facilities allow. Mihm's study of five pheochromocytoma patients showed that the relationship between right atrial pressure and pulmonary wedge pressure was poor and especially so in those with a history of congestive heart failure and "that misleading information may have been obtained in any of the five patients with PA catheters if only right-sided heart pressures were monitored."²

As to their second point about the incidence of hypertension persisting for a few days after removal of a solitary pheochromocytoma, that certainly is our and Dr. Roizen's personal experience. The reference to the statement that "fully 50 per cent of pheochromocytoma patients will remain hypertensive for days postoperatively" was listed incorrectly in our review and should be quoted from Dr. Roizen's review of the condition in the 1983 ASA review course lectures pages 124: 4-5. My recent personal communication with him confirms that about half of his patients also remain hypertensive for days following tumour removal.

This academic interchange re-emphasizes that the disease is certainly protean in nature and that at the very least, the clinician should be wary of the possible existence of abnormal ventricular function in a pheochromocytoma patient. Even when a solitary tumour is excised, the blood pressure may not return to normal values promptly when catecholamine levels abate.

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Caudal mepivacaine and ventilation

To the Editor:

Takasaki and Kosaka have concluded that resting ventilation is impaired but the ventilatory response to CO₂ is improved by caudal block with one or two per cent mepivacaine.¹ Their data do not support the conclusion that resting ventilation is impaired. Although V_E is decreased the PaCO₂ did not significantly increase. Since PaCO₂ = (VCO₂/V_A) × K and PaCO₂ did not change, the evidence seems to suggest that either CO₂ production decreased, due to a decrease in oxygen consumption, or V_{PDS} decreased leaving a constant V_A at the decreased V_E.

In any case, the level of resting ventilation was appropriate as the resting PaCO₂ did not change. These two facts, i.e., normal resting PaCO₂ with increasing slope of the CO₂ response curve would suggest that the respiratory stimulatory effect of the mepivacaine is acting at the central chemoreceptors.

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REPLY

Thank you for the opportunity of replying to the letter from Dr. Edelist. We demonstrated that resting ventilation was impaired by caudal block with one or two per cent mepivacaine. Expired minute volume (V_E) and respiratory frequency decreased significantly after the caudal blocks. However, end-tidal CO₂ partial pressure (PETCO₂) and PaCO₂ remained unchanged following caudal block with one per cent mepivacaine, although PETCO₂ increased significantly and PaCO₂ increased slightly following caudal block with two per cent mepivacaine. The same effects of caudal blocks on respiratory function were observed in our previous report.¹ Following caudal block with 1.5 per cent lidocaine, PETCO₂ and PaCO₂ increased significantly, but