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In reply:—The letters of Engel and of Turnbull *et al.* nicely reiterate the conflicting biases of Keats and Hamilton as to causes of anesthetic mortality. Drawing from a much wider experience as a malpractice defense attorney, Engel's perceptions parallel my own. Turnbull *et al.* presumably find many more "possibly preventable" deaths than "fortuitous" or "unassessable" deaths, thus supporting Hamilton's view. It is to be hoped that they will one day give us their numbers and the bases for their judgments.

I would be saddened if readers of these two essays reduced their essence to a drug-versus-user debate. The major thrust, occupying more than half of my paper, was the painful documentation of the error-bias that has pervaded out specialty *since its inception* and still pervades it. Error clearly plays a large but as yet undetermined role, and no whitewash was intended. But I did urge that we also look beyond error. Where? Look at the adverse drug reactions. Look for new mechanisms. Look into the obligatory death rate associated with hospitalization. Most importantly, I urged that we accept the existence of anesthetic deaths without human or machine error in patients not expected to die and our ignorance as to cause. This would be the first step toward investigation of such deaths. To do otherwise would be to conclude we already know all there is to know and to convert our error-bias to certain knowledge that every anesthetic injury and mortality was preventable. Even Dr. Hamilton did not go this far.

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Abduction of the Vocal Cords

To the Editor:—In considering congenital vocal-cord paralysis, Dr. Maze and Mr. Bloch[1] repeat the venerable assertion that abductor fibers of the recurrent laryngeal nerve are more easily damaged than adductor fibers. The basis for this time-honored opinion has been challenged by a recent study, which suggests that there is no truly abductor muscle for the human vocal folds.[2] There is none for the vestibular folds either. Abduction of the vocal folds is effected indirectly by the unfolding attendant on inspiratory caudad descent of the larynx as a whole. The inspiratory muscles responsible for this descent supply a much more powerful opening element than the reputed abductors, the relatively tiny cricoarytenoids, whose role in man is to keep the arytenoid cartilages in balance for the operation of folding and unfolding.[3]

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Rare Cause of Stridor

To the Editor:—The review by Maze and Block[1] of stridor in pediatric patients brings to mind a recent case of a 3-year-old boy who was seen in the hospital emergency department because of stridor. A diagnosis of croup was made and the boy was sent home with instructions for conservative therapy. The stridor worsened, and he returned to the hospital later the same day. At this time a lateral roentgenogram of

the neck showed a lesion in the larynx. An emergency tracheostomy and laryngeal biopsy were performed with halothane–oxygen anesthesia via a 3-mm (ID) endotracheal tube. Biopsy revealed a rapidly growing mixed sarcoma. Ten days later, a laryngectomy was performed with halothane–nitrous oxide–oxygen anesthesia given through the tracheostomy via a 4.5-mm (ID) wire-reinforced cuffed tube. The cuff was inflated with 2–3 ml air. Midway through the procedure, manual ventilatory assistance became progressively more difficult, and ultimately the lungs could not be ventilated. The tracheostomy tube was quickly removed and exchanged for a 5-mm (ID) uncuffed plastic tube, which restored manually assisted ventilation to normal. Examination of the removed tube revealed that despite precautions, blood from the surgical field had leaked around the cuffed tube and occluded the distal end.

Maze and Block emphasize the importance of establishing an adequate airway, and this case illustrates a problem in its maintenance. Cancer of the larynx is rare in children, but should be considered in any case of a child with stridor whose condition does not respond to conventional therapy.

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Use of Pulmonary-artery Catheter not Justified

To the Editor:—The article by Silverstein *et al.*[1] did not convince me that the use of a pulmonary-artery catheter is either necessary or desirable during elective resections of abdominal aneurysms. The sole manifestation of depressed myocardial function following aortic cross-clamping seems to have been the 23 per cent decrease in cardiac output. Significant changes in all other measured values, which would have indicated heart failure, were not seen. I believe that, from the data given, none of these patients was in heart failure, and hence none needed additional vasodilator therapy intraoperatively. They had already received large amounts of morphine, an effective drug for the treatment of left ventricular failure.

Heart failure is said to occur when cardiac output is unable to satisfy the metabolic demands of the body, even when blood volume is normal. Now it is manifestly impossible for the heart to satisfy the metabolic demands of the pelvic viscera and legs (where a fifth of the cardiac output usually goes) when the aorta is clamped below the renal arteries. However, the other four fifths of the cardiac output is all that is necessary to provide normal circulation to the remainder of the body which the heart is being allowed to perfuse. Thus, a state of heart failure—by definition—does not exist in this situation, although the cardiac output is only 80 per cent of its usual value. A similar, permanent, decrease in cardiac output would occur following a hemicorporectomy operation. Since the cardiac index for the perfused part of the body in these cases was normal, it follows that the real increase in total peripheral resistance was only 9 per cent.

I have worked in various hospitals during the past 19 years, and I am still not aware of any peculiar association between elective abdominal aortic aneurysm resection and intraoperative pulmonary edema. If the usual vital signs, the pulse, blood pressure and, perhaps, central venous pressure, are kept within reasonably normal limits, no notable change in cardiac function seems to occur. Under these conditions the increase in afterload, which presumably precipitates left ventricular failure, is minimal. The main argument in favor of the pulmonary-artery catheter is that, by detecting minute changes in left atrial pressure, it can give an early warning of left heart failure before pulmonary edema or right heart failure is detectable. I am not convinced that such accuracy is necessary in clinical medicine, especially if it cannot be achieved in an innocuous manner. Early pulmonary edema