

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 defini-

tion—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 hemicolectomy 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

should be detected by an esophageal stethoscope and left heart failure alone during anesthesia is still a rarity. However, if intraoperative pulmonary edema is becoming a problem (and this same institution has reported a disturbing 4 per cent incidence for it postoperatively²), perhaps a critical reappraisal of intraoperative fluid therapy would be more useful than resorting to pulmonary-artery catheters. The patients in this series received 1 liter of lactated Ringer's solution per hour, and so many probably had totals of 3 liters or more. Intraoperative sodium intake, therefore, may have been more than 400 mEq, which is equal to the usual intake over a period of four or five days. This presents no problem to the young, healthy patient who can excrete it in 12 hours, but for this population, the response might be very different. Seventy-three per cent of the patients were found to have hypertension or tachycardia, or both, immediately postoperatively. This finding may have

been due to excessive fluid therapy intraoperatively, combined with the diminishing effects of the morphine, a circumstance reported by Flacke *et al.*³

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In reply:—It is unfortunate that Dr. Morley remains unconvinced of the value of pulmonary-artery catheterization during elective abdominal aortic operations in selected patients with serious myocardial ischemia or dysfunction. Although he appreciated that significant hemodynamic changes did not occur after aortic clamping, he arrived at the wrong conclusion. As the title of our paper indicates, we intervened and preserved optimal hemodynamics despite aortic clamping and declamping in a group of patients at risk for the consequences cited in the references. Our concerns were not for intraoperative pulmonary edema or congestive heart failure, but the consequences of aortic cross-clamping (hypertension, myocardial ischemia, and arrhythmias) and declamping-induced hypotension. Of course, these patients were not in heart failure. Vasodilator therapy was used intraoperatively not to prevent heart failure, but to prevent myocardial ischemia and its manifestations by avoiding increases in myocardial oxygen demand. In addition, we endeavored to balance this goal with that of maintaining high enough filling pressures by careful volume administration to avoid the hazards of aortic declamping.

How would Dr. Morley propose to keep the "pulse, blood pressure and, perhaps, central venous pressure" within normal limits? Will he use nitroprusside, nitroglycerin, propranolol, more or less halothane, narcotics, vasopressor drugs, fluid restriction or fluid loading, etc.? How will he fine-tune any of these therapeutic maneuvers to avoid the above-mentioned com-

plications? Since Dr. Morley does not see the need for monitoring, how can he state that "no notable change in cardiac function seems to occur"? He also fails to mention the electrocardiogram as an essential monitor (precordial lead with or without a limb lead). The electrocardiogram is a good indicator of overt myocardial ischemia, but our goal was to avoid such ischemia rather than identify it.

Major discrepancies between central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) do occur in patients undergoing elective abdominal aortic operations.¹ Five of 55 patients (9 per cent) had significant decreases (4-5 torr) in CVP when cardiac index was stable and PCWP was increasing. "In those patients, there was no substitute for indirect measurement of PCWP." We never stated that the main argument in favor of pulmonary-artery catheterization is early warning before left heart failure, pulmonary edema or right heart failure develops. We did state that by use of appropriate intraoperative intervention with sodium nitroprusside, and as we've learned subsequently, with nitroglycerin, none of our patients experienced decompensation sufficient to cause overt left ventricular failure, irritability, or increased myocardial ischemia. Why does Dr. Morley raise the issue of pulmonary edema when it was not mentioned in the article?

Of equal importance is intraoperative prevention of declamping-induced hypotension with nitroprusside and volume loading before clamp removal. How would Dr. Morley guide volume therapy in order to prevent