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 postoperatively[2]), 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 complications? 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

declamping-induced hypotension? With just a peripheral intravenous line or, perhaps, central venous pressure? Bush *et al.* similarly reported their ability to volume-load the left ventricle, thereby increasing cardiac output such that after declamping, the vasodilated lower extremities were supplied without a decrease in blood pressue.[2] They too, believe that close monitoring using pulmonary-artery catheters is necessary to avoid excessive or inadequate volume infusion. We certainly do not advocate that all patients undergoing abdominal aortic operations have pulmonary-artery catheters placed as a prerequisite to anesthetic and surgical management, but most definitely urge their use for patients who have histories of myocardial ischemia or ventricular dysfunction. The devastating effects of acute myocardial ischemia, declamping-induced hypotension and all its sequelae, justify the very small but definite risk of complication from pulmonary-artery catheterization in these high-risk patients.

We sincerely hope that Dr. Morley rereads and understands our article for what it is, a means to prevent myocardial ischemia and declamping-induced hypotension, rather than for what he has interpreted it to mean, namely, heart failure and pulmonary edema. Perhaps he would then be convinced of the efficacy (in selected patients) of pulmonary-artery catheterization for monitoring, appropriate volume loading, and vasopressor and vasodilator therapy.

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Does "Self-taming" Decrease Postoperative Myalgia in Outpatients?

To the Editor:—We read with interest the recent article by Brodsky and Brock-Utne[1] regarding the possible prevention of postoperative myalgia by use of a "self-taming" dose of succinylcholine. At our hospital we give anesthesia to a relatively large number of outpatients and are interested in minimizing postoperative patient discomfort. Fifty healthy outpatients scheduled for oral surgical procedures were randomly divided into two groups. Group I received a "self-taming" 10-mg dose of succinylcholine 1 min prior to an intubating dose of 1 mg/kg. Group II received a placebo in place of the "self-taming" dose of succinylcholine. All patients were given fentanyl, 0.05–0.10 mg, and atropine, 0.4 mg, iv, for premedication. Anesthesia was induced with thiopental, 4–5 mg/kg, and maintained with enflurane, nitrous oxide, and oxygen.

Of the 25 patients in Group I, ten had some degree of postoperative myalgia, determined by a telephone interview the first postoperative day. Of the 25 patients in Group II, ten also had evidence of postoperative myalgia. Muscle fasciculations were decreased by the "self-taming" dose of succinylcholine. Only 4 per cent of the patients receiving the "self-taming" dose had +3 fasciculations (vigorous in trunk and extremities), while 44 per cent of those receiving a placebo had +3 fasciculations.

These findings correlate with those of Brodsky *et al.*, and fail to show any advantage in using a "self-taming" dose of succinylcholine to decrease postoperative myalgia in outpatients.

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