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Differentiating Interstitial Fluid from Cerebral Spinal Fluid

To the Editor:—Recently, an 89-year-old man with severe chronic obstructive pulmonary disease underwent emergency surgery for intestinal obstruction at our hospital. An epidural catheter was placed in the immediate postoperative period to provide pain relief with intermittent epidural morphine injections. It was hoped that the high degree of analgesia characteristic of this technique would help to prevent the occurrence of pulmonary complications. Catheter placement in the epidural space was confirmed when, after a negative aspiration test, injection of 5 ml xylocaine 1.5% produced a T-10 analgesic band unaccompanied by paresis of the lower extremities.

Twenty-four hours after insertion, copious amounts of crystal clear fluid began leaking through the puncture site and around the catheter. Although fluid could not be aspirated from the catheter itself, a cerebrospinal fluid leak was strongly suspected and the catheter was removed. Several hours later, deep pitting edema was noted in the lumbosacral area, and large amounts of fluid continued to leak from the former epidural puncture site. The question was raised as to whether this was cerebrospinal fluid or interstitial fluid.

Samples of the unknown fluid, cerebrospinal fluid (obtained by lumbar puncture), and blood were submitted to the laboratory. Although the values for BUN, creatinine, and sugar were similar in the three samples, the protein concentration of the unknown fluid was 326 mg/100 ml and that of the cerebrospinal fluid was 46 mg/100 ml.

Since the concentration of protein in the unknown sample was about 600% greater than that of the spinal fluid, and since it is well known that the concentration of protein in the interstitial space is normally much higher than that of the cerebrospinal fluid,¹ it seemed reasonable to assume that the interstitial space and not the subarachnoid space was the source of the fluid. Increased usage of epidural catheters for postoperative pain relief in the elderly and debilitated may well give rise to this problem more frequently in the future. A determination of protein concentration in the suspected fluid can provide valuable information with respect to such patients.

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An Alternate Method to Manage Patients with Protamine Hypersensitivity for Cardiac Surgery

To the Editor:—In a recent issue, Campbell *et al.*¹ reviewed their experience and knowledge of the management of cardiac surgery patients with known protamine hypersensitivity. We present an alternate technique that we deployed for a cardiac patient with a documented history of an anaphylactic reaction to protamine.

A 61-year-old, 56-kg woman had an uneventful carotid endarterectomy that included protamine reversal of bovine heparin.² Two months later, she had an aortofemoral bypass during which she had

a severe hypersensitivity reaction to protamine reversal of bovine heparin. The extreme hypotension necessitated a brief period of external cardiac massage as well as appropriate fluid and pharmacologic therapy, and she later had both adult respiratory distress and Dressler's syndromes develop. Six months later she was scheduled for aortic and mitral valve replacements. As with the second patient of Campbell *et al.*, she was pretreated with steroids and antihistamines, although our plan of management intended to avoid, if possible, the need for protamine.

Upon exposure of the heart, a canula was placed in the right atrium and connected to two 1,000-ml citrated blood donor bags via a Y-designed collecting system. During several minutes 2,000 ml of

autologous blood was removed while her blood pressure was supported with a phenylephrine infusion. She was then heparinized with 13,000 units of bovine heparin. The atrial canula was replaced with routine two-stage atrial canulae. The 1,300-ml priming volume of the bubble oxygenator (Bos 10, American Bentley, Irvine, California) included 3 units of packed erythrocytes to insure that the patient had adequate oxygen-carrying capacity during cardiopulmonary bypass. Adequate heparinization was confirmed by frequent measurements of the activated clotting time (ACT) and titrations of the blood heparin levels (Hepcon System A-10, Hemotec Inc., Englewood, Colorado). With the support of dopamine and phenylephrine she was weaned off a bypass lasting 100 min. Her blood was then slowly emptied via the venous canula into the oxygenator until her systolic blood pressure was decreased to 60 mmHg. The right atrial canulae were then reexchanged, and the 2,000 ml citrated autologous blood (with calcium coverage) was rapidly returned to the patient. The phenylephrine was replaced with a sodium nitroprusside infusion. The remaining 1,600 ml of the oxygenator blood was washed through a cell saver apparatus (Hemonetics, American Bentley, Irvine, California) and the erythrocyte concentrate reinfused to the patient. She also received empirically 3 units of fresh frozen plasma. Within 45 min of decanulation, the heparin titration system indicated absence of any heparin activity, the ACT had returned to control, and clinically adequate hemostasis with clot was noted, despite a minimally elevated partial thromboplastin time. The patient's postoperative chest drainage during the initial 24 h was 350 ml, and the remainder of her hospital course was uneventful.

In conclusion, our technique employs the withdrawal of a large percentage of the patient's blood volume prior to systemic heparinization, its anticoagulation with

nonheparin chemistry, and its return to the patient immediately following bypass. Proper pharmacologic manipulation of the cardiovascular state not only maximizes the volume of blood that may be so maneuvered, but also facilitates the largest volume of oxygenator erythrocytes that can be washed free of heparin.

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Intravenous Nitroglycerin Dosage to Prevent Intraoperative Myocardial Ischemia during Fentanyl-Pancuronium Anesthesia

To the Editor:—We read with great interest the article by Thomson *et al.* concerning the failure of intravenous nitroglycerin to prevent intraoperative myocardial ischemia during fentanyl-pancuronium anesthesia.¹ This appears to be the final product of work reported by these authors in abstract form.² In both articles the patients received an $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ intravenous nitroglycerin infusion 20 min prior to induction of anesthesia and during the entire operation, in order to assess the possible prevention of ischemic ECG changes. The authors reported that intravenous nitroglycerin was not superior to placebo in preventing ECG changes of myocardial ischemia. We should like to refer the authors to the article of Coriat *et al.* on the efficacy of intravenous nitroglycerin in the prevention of intraoperative myocardial ischemia during noncardiac surgery.³ They com-

pared the incidence of intraoperative myocardial ischemia in patients with angina pectoris undergoing noncardiac surgery during fentanyl N_2O -pancuronium anesthesia with prophylactic intravenous nitroglycerin infusion. They concluded that with a dose of $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ there is still a high incidence of intraoperative myocardial ischemia in these patients and they even compared their own results with those of Mutch *et al.*² They went further with this investigation and administered nitroglycerin at a dose of $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and found it to be highly effective in preventing intraoperative ischemic episodes. It seems to us that if Thomson *et al.* had used $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ instead of $0.5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ they would have achieved the same prevention of ischemic episodes. As the authors suggest, and our experience confirms, adequate hydration before