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In Reply—The conclusion drawn by Beriault that individuals having a history of more sinister herpesvirus infections are unacceptable candidates for intraspinal opiates cannot be drawn from the data presented in our study.¹ The reported association between epidural morphine and herpes simplex virus (HSV) labialis in a puerperal population does not equate to reactivation of HSV encephalitis or HSV keratitis. The far-reaching conclusion of “turning a cold cheek” to the use of epidural opioids in an anesthesia practice is again not supported by the data presented. We have reported an unusual side effect of the use of epidural morphine in a specific subset of the population (parturients). It is quite conceivable that epidural morphine is associated only with HSV labialis reactivation in pregnancy and with no other type of reactivation.

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Bronchospasm Following Revascularization of Cadaver Kidney Graft

To the Editor—Wheezing during anesthesia may result from many phenomena other than simple reactive bronchospasm. Anaphylactoid drug reactions also are characterized by bronchospasm, and almost every drug given during anesthesia and surgery can be implicated in this type of reaction. The following case describes an episode of bronchospasm following revascularization of a transplanted kidney, in all likelihood induced by the kidney preservation solution.

A 30-yr-old, 65-kg man with Alport's syndrome was scheduled for cadaveric kidney transplantation. His hypertension was controlled with clonidine 2 mg orally; the chronic renal failure was treated by hemodialysis, the most recent on the day of surgery. He had no history of allergy or hypersensitivity to any drugs, and smoked cigarettes occasionally. Preoperative blood pressure was 140/90 mmHg, and heart rate was 86 beats per min.

Preoperatively he had received 8 mg/kg methylprednisolone, azathioprine 2 mg/kg, and 10 mg OKT 3 intravenously as part of our transplantation protocol. Monitoring consisted of ECG, noninvasive arterial blood pressure, central venous catheter, pulse oximeter, capnometer, peripheral nerve stimulator, Foley catheter, and temperature probe.

Anesthesia was induced with thiopental 5 mg/kg, fentanyl 2 µg/kg, droperidol 2.5 mg, and atracurium 0.5 mg/kg intravenously. The patient's trachea was intubated without difficulty. Anesthesia was maintained with isoflurane 0–2% and N₂O/O₂ 50/50%. The lungs were mechanically ventilated to achieve an end-tidal carbon dioxide tension (PETCO₂) of 35–40 mmHg. Additional atracurium 5 mg was given as needed.

His vital signs remained stable throughout the procedure. After the release of the vascular clamps, increased inspiratory pressure and bilateral wheezing was noted, and the PETCO₂ increased to 46 mmHg.

After the most likely etiologies of bronchospasm (mechanical obstruction, endobronchial intubation, or light anesthesia) were ruled out, two to three puffs of fenoterol was applied endotracheally, followed by epinephrine 0.5 mg and promethazine 50 mg intravenously.

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During the next 15 min the wheezing gradually diminished and the PETCO₂ returned to the 32–35 mmHg range. At the end of the procedure, after appropriate antagonism of the residual neuromuscular blockade, the trachea was extubated.

Emergence and recovery were uneventful and the patient had satisfactory diuresis. No episodes of further wheezing were noted, and he was discharged home with good renal function.

Since all other possible causes of bronchospasm were considered and ruled out, and the onset of the wheezing was immediately after the release of the vascular clamps, we concluded that the bronchospasm was induced by the solution used to preserve the cadaver kidney. The Collins solution, which is widely used to perfuse the cadaver kidney (with penicillin G added to it) can cause rash and wheezing¹ at the onset of graft perfusion.

In our case, the kidney was perfused with HTK protective solution

TABLE 1. Composition of the Two-perfusion Solution

| | Concentration (mM) | |
|------------------------------|---|--|
| | Collins | HTK |
| Na ⁺ | 10 | 15 |
| K ⁺ | 115 | 10 |
| Ca ²⁺ | — | 0.015 |
| Mg ²⁺ | — | 4 |
| Cl ⁻ | 15 | 50 |
| SO ₄ ⁻ | — | — |
| Substrates | glucose 198 | tryptophan 2 α-ketoglutarate 1 mannitol 30 |
| Buffer | HPO ₄ ⁻ 43 H ₂ PO ₄ ⁻ 15 HCO ₃ 10 | histidine 180 his-HCl 18 |
| Osmolarity | 406 | 310 |

(Custodial, Kohler Chemie GMBH, FRG), which does not contain penicillin (table 1). Mannitol, one of the other components, has been described to induce anaphylactoid reactions.^{2,3}

To confirm the allergic origin of the wheezing, blood samples were drawn two and twelve h after the occurrence of bronchospasm, and showed increased plasma histamine, normal immunoglobulin E (IgE), and slightly depressed complement 3 and normal complement 4 level, supporting an anaphylactoid reaction was the cause of the adverse reaction.⁴

Because of the preoperatively administered corticosteroid and immunosuppressive treatment, fortunately this was not a life-threatening reaction. Our case emphasizes the fact that in the immunocompromized patient, an allergic reaction can manifest as bronchospasm possibly caused by the solution used to perfuse the harvested organs.

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Delayed Onset of Pneumothorax Following Internal Jugular Vein Cannulation

To the Editor:—Pneumothorax following placement of central venous catheters occurs in approximately 1-3% of attempts at access. Previous reports of this complication note intrapleural air immediately, or soon after, placement of the catheter.^{1,2} We report a case of 100% pneumothorax occurring immediately following removal of an internal jugular vein catheter that had been inserted 8 days earlier.

A 53-yr-old woman presented for posterior fossa craniotomy in the sitting position to excise both a right-sided cerebellar meningioma and a left-sided acoustic neuroma. After induction of general endotracheal anesthesia, an 8.5-French sheath (Arrow-Flex Radiopaque, Arrow International) was placed in the right internal jugular vein with ease. A multiorificed single-lumen catheter was inserted through the diaphragm of the sheath. The internal catheter was removed in the recovery room. Radiography of the chest in the recovery room and on postoperative days 1, 2, and 5 revealed no abnormalities. On the 8th postoperative day, a nurse removed the sheath, and within 1 min the patient developed profound dyspnea and tachypnea. Venous air embolism was suspected, but physical examination revealed absence of breath sounds over the right lung field. A radiograph of the chest showed a 100% right-sided pneumothorax. Tube thoracostomy was performed, and intercostal nerve block provided the patient with relief of pain from her chest tube.

To our knowledge this is the first report of a pneumothorax following removal of a central venous catheter. We theorize that the pleura, and possibly the lung parenchyma, was damaged during sheath insertion. This defect was temporarily sealed by the sheath. At the time of removal, the defect opened, resulting in pneumothorax. This represents

a case of uncommon timing of a fairly common complication, and highlights the necessity of observing the patient after removal of intravascular catheters used for monitoring.

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