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Spinal Headache and Air Travel

To the Editor:—Vacanti, in 1972,[1] reported a severe recurrence of a post-lumbar-puncture headache precipitated by change in atmospheric pressure associated with an airplane flight. We have recently treated a post-lumbar-puncture headache with an epidural blood patch, only to have a recurrence of headache occasioned by air travel.

A healthy, 33-year-old woman had laparoscopy with epidural anesthesia, during which a dural puncture with a 19-gauge needle was accidentally performed. She noticed a positional headache the next morning, and was treated immediately with an epidural blood patch, with relief. Her headache recurred 48 hours later and she was successfully treated with a second epidural blood patch four days after the initial puncture. Eight days thereafter, her headache again recurred while on a commercial airline flight to her home in Alaska. She noticed a positional headache that then persisted for four weeks, despite additional treatment with oral fluid intake, analgesic drugs, and bed rest. It became associated with an increasingly frequent, intermittent, non-positional headache. She consulted a neurologist, who could identify no neurologic disease. A third epidural blood patch, six weeks after the initial puncture, provided resolution of her positional headache. Her non-positional headaches were believed to be related to depression secondary to enforced inactivity, and proceeded to improve after she finally was able to return to work, eight weeks after her operation.

The aggravation of post-lumbar-puncture headaches by acute decreases in atmospheric pressure is possibly related to an increased pressure gradient between the subdural and epidural spaces. The mechanism is still unclear, but it does appear, from this case, that such exacerbations may occur despite a previously successful epidural blood patch. It may be judicious to advise patients who plan air travel shortly after lumbar puncture or epidural blood patch that there may be an associated risk of recurrence or exacerbation of headache.

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REFERENCE

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Unexplained Brachial-plexus Palsy

To the Editor:—Damage to the brachial plexus, while uncommon, is the most frequent type of postoperative neuropathy. We had a patient in whom a right partial brachial plexus palsy developed following a left hemithyroidectomy. We surveyed the literature and have not found a similar case report.

The patient, a 42-year-old, 90-kg man, was admitted for removal of an enlarging mass in the left thyroid lobe, which had been present for a year. His past history was unremarkable. He had no muscle weakness, pain, or paresthesia in the neck or upper extremities. There was a 5 × 4-cm mass occupying the entire left thyroid lobe. The remainder of the physical examination disclosed no abnormality. Roentgenograms of the chest were negative except for a displacement of the trachea to the right by the thyroid mass. A thyroid scan demonstrated a nonfunctioning left thyroid lobe. The patient received premedication intramuscularly in the right arm, presumably in the deltoid region. The injection was painless and without paresthesia. Throughout the entire operation the patient was positioned with both arms at his sides. His shoulders were elevated 4–5 cm from the table by a folded sheet. The head and neck were moderately hyperextended and maintained in the midline. The operation lasted three and a half hours. The left thyroid lobe and isthmus were resected. The right thyroid lobe was not explored.

On the evening of the operative day, the patient complained of inability to move his right shoulder.

Examination of the right upper extremity showed zero deltoid action and minimal triceps muscle action. The rotator cuff was good; elbow and wrist flexors and finger motion were normal. There was no sensory loss. A diagnosis of brachial plexus contusion involving C5 and C6 of the posterior cord was made. Therapy was conservative, the arm was put in a supporting sling. The patient was discharged on the seventh postoperative day with obvious improvement in shoulder function. Examination two years after operation showed good recovery, although the patient complained of some shoulder weakness. The impairment did not interfere with the patient's normal routine.

The pathogeneses of brachial-plexus injury during anesthesia has been considered by many. Stretch or compression of the brachial plexus associated with malposition of the body during anesthesia is responsible for many of the reported neuropathies. We can only speculate on the possible causes of brachial-plexus damage in this patient. It is possible that the intramuscular injection of the premedicant drugs in the right arm injured the axillary or radial neurons. This appears improbable, as the patient had no pain or paresthesia. A congenital anomaly of the cervical vertebrae could produce compression of C5 and C6 cords with moderate hyperextension of the head. There is no support for this in the patient's history. Finally, a member of the surgical team could have rested on the patient's right shoulder. Continuous downward shoulder pressure in a paralyzed patient could produce posterior displacement of the humeral head or clavicle, with prolonged stretching of the brachial plexus and consequent damage. This suggestion is, of course, pure speculation.

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Prevention of Anaphylaxis from Contrast Media

To the Editor:—Drs. Millbern and Bell suggest that pretreatment with steroids and diphenhydramine should be considered prior to giving radiopaque contrast agents to patients with previously documented sensitivity to these agents to avoid anaphylactic responses.[1] However, we have recently found that pretreatment with these agents failed to prevent an anaphylactic response to contrast medium. The patient was a 60-year-old white man who complained of increasing claudication in both legs, and for whom aortic angiography was planned. He had a well-documented history of anaphylactic responses, including cardiac arrests on two occasions when he had been given intravenous pyelogram dye. In preparation of the angiographic study, he was hospitalized and received a five-day course of prednisone, 20 mg, and diphenhydramine, 50 mg, orally, twice daily. On the morning of angiography, and with informed consent, he was premedicated with prednisone, 50 mg, and diphenhydramine, 50 mg, orally, and he was given methylprednisolone, 100 mg, and diphenhydramine, 25 mg, intravenously on arrival in the angiography suite. Monitors included an electrocardiogram, precordial stethoscope, blood pressure cuff, and transduced arterial waveform obtained from the femoral arterial catheter to be used for the angiography. The patient was sedated with diphenhydramine, 75 mg, morphine, 15 mg, and diazepam, 10 mg, intravenously, and was sleepy but easily rousable. Vital signs were pulse, 70 beats/min, blood pressure, 150/100 torr, respiration rate, 18/min, with spontaneous respirations. A test injection of Renografin-76® contrast material, 10 ml, resulted in no change in vital signs. Angiography of the abdominal aorta and both legs was then performed with a single mechanized injection of 75 ml of the same contrast agent. Immediately after the injection, the pulse decreased to 50 beats/min, blood pressure decreased to 60/20 torr, and the patient became very flushed. Marked bronchospasm, tachypnea, and dyspnea were present. The patient remained conscious and complained of severe generalized burning and pain. He was successfully resuscitated with intravenous fluids and epinephrine. Four hours after the incident he had completely recovered.

Pretreatment with methylprednisolone just prior to challenge failed to prevent the anaphylactic response in both our patient and Dr. Millbern's patient, even in conjunction with diphenhydramine therapy. For our patient the five-day course of orally administered prednisone also apparently had little or no effect. These experiences and other reports[2,3] suggest that pretreat-