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Regional anesthesia for a patient with hereditary neuropathy with liability to pressure palsies

To the Editor:

Hereditary neuropathy with liability to pressure palsies (HNPP) is a focal, recurrent, hereditary, demyelinating neuromuscular disorder characterized by weakness and paresthesia following apparently trivial compression injury.[1] The most commonly affected sites are the peroneal nerve (from compression against the fibular head), the ulnar nerve (from prolonged resting on the elbow) and the radial nerve (from compression at the spiral groove in humerus). The brachial plexus is also frequently affected.

Hereditary neuropathy with liability to pressure palsies was first described by De Long, who studied a family of three generations suffering from recurrent peroneal neuropathy. Sausage-shaped swellings of the myelin sheath ("tomacula") are found on biopsy.[1] In most cases, the genetic anomaly is a deletion of 1.5 million base pairs in chromosomal region 17p11.2, which contains the gene coding for peripheral myelin protein 22 (PMP-22).[1] Despite such knowledge, implications for anesthesia in these patients remain provisional, and few reports on anesthetic management have been published, two being obstetric cases[2,3] and one case diagnosed following breast surgery.[4]

We recently provided anesthesia to a 27-yr-old male with HNPP who underwent surgery for arthroscopic anterior cruciate ligament reconstruction. The diagnosis of HNPP was made eight years previously when the patient suffered peroneal nerve injury with weakness and numbness in the left leg after prolonged sitting with his legs crossed. Electrophysiological studies demonstrated blocked peroneal nerve conduction at the level of the fibular head and signs of peripheral diffuse neuropathy at other sites. A Southern blot study identified a characteristic deletion on chromosome 17. The patient was initially treated with betamethasone, cobalamin and electrostimulation, with substantial recovery. At the time of the patient's hospital admission, the anesthesiologist, neurologist and orthopedic surgeon met to plan perioperative management; little helpful literature was found.

We decided to employ regional anesthesia with the goal of avoiding the prolonged immobility with general anesthesia that presumably might increase the risk of a pressure palsy. We performed a L2–L3 spinal anesthetic using a Sprotte needle, and administered 12 mg of hyperbaric bupivacaine with the patient in the sitting position. The patient was then turned to the left lateral position to obtain a unilateral block. For surgery, he was positioned supine with his arms abducted to an angle under 90°. In addition, pads were positioned under both legs, and especially under the popliteal fossa of the operated leg, with the knees flexed slightly. We also encouraged the patient to move his arms and his right leg to maintain comfort. Another surgical precaution was to avoid using a tourniquet. The surgery was uneventful, lasting 90 min; after three hours the block completely regressed. There were no neurological complications and no complaints of pressure palsy. Neurological examinations performed six and 12 hr postoperatively did not reveal any abnormalities. The patient was discharged on the third postoperative day without event. Neurological examinations repeated after a week and after three and six months were negative.

Although there are no existing reports discussing the effect of surgical positioning in patients with HNPP, there are a number of potential causes of nerve palsy in these patients. Consequently, special effort was made to avoid any nerve stretching on the non-operated leg while the surgeon positioned the operated leg so as to avoid any pressure on the peroneal nerve at the fibular head. As noted earlier, we also avoided the use of a tourniquet, which is usually applied to reduce bleeding and provide better surgical conditions, to

avoid direct extrinsic pressure on any nerves. In conclusion, patients with HNNP present special surgical positioning challenges for which regional anesthesia may provide specific advantages.

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A case of paradoxical cerebral thromboembolism after removal of a central venous catheter

To the Editor:
Thrombotic complications of central venous catheters (CVCs) are reported to occur in 2–26% of patients, and all thrombi have the potential to embolize.[1] Paradoxical cerebral air embolism during placement or withdrawal of CVCs has been described in numerous case reports,[2] but cerebral thromboembolism after central venous catheterization procedures is a rare complication. We present a case of pulmonary and cerebral embolism immediately after removal of an internal jugular venous (IJV) catheter with evidences of right-side thrombi and right-to-left shunt.

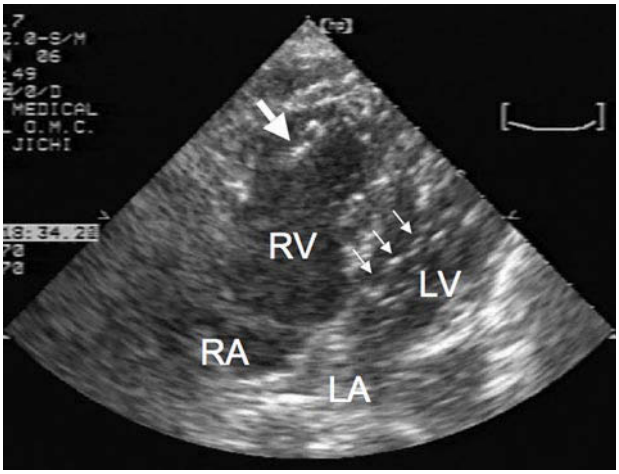

FIGURE Four chamber view of transthoracic echocardiography demonstrates a linear thrombus tethered to the right ventricular trabeculae (large arrow) and snowstorm-like bubbles in the left heart (small arrows). RV = right ventricle; RA = right atrium; LV = left ventricle; LA = left atrium.

A 78-yr-old male, who had a history of stroke with residual right hemiparesis, underwent a distal gastrectomy for cancer. The postoperative course was complicated with an anastomotic leak, which required a re-exploration of the abdomen and correction on postoperative day 11. In the operating room, a double-lumen, heparin-coated CVC (M2714HSI, Edwards Life Science, Irvine, CA, USA) was uneventfully placed into the right IJV for postoperative nutritional support.

One week after the re-exploratory surgery, the patient still spent most of the day in bed due to his preoperative morbidity and delayed wound healing, while *sc* heparin administration for deep venous thrombosis (DVT) prophylaxis was already discontinued. Two days later, when the patient became febrile, the IJV catheter was removed. The removal procedure was uneventful with the legs elevated, and the puncture hole covered with a tight bandage. Soon after this procedure, the patient developed respiratory distress, hypotension, loss of consciousness, and a seizure. While standard resuscitation was initiated, the diagnostic interventions began with transthoracic echocardiography, which revealed a clot trapped in the right ventricle (Figure) and moderate tricuspid insufficiency suggesting an elevated pulmonary artery pressure. Also, snowstorm-like bubbles in the right heart, accidentally created by a rapid resuscitation fluid bolus, were simultaneously seen in the left heart