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Failure of an iv fluid warming device

To the Editor:

We recently experienced a failure of a fluid warmer (Level 1- H1000, Fast Flow Fluid Warmer, Smiths Medical, Rockland, MA, USA) which could have resulted in significant harm to a patient. Although we could find no other reports of this particular failure, it has been documented in other types of counter-current fluid warmers.[1] The incident occurred during the elective repair of an abdominal aortic aneurysm, under general anesthesia, with the placement of a thoracic epidural for postoperative analgesia. There were no problems during the case from either a surgical or anesthetic point-of-view. The patient was transfused with blood from the cell saver during the case. This blood was transfused through the Level 1, under pressure.

At the end of the operation, a small pool of blood was observed near the base of the Level 1 fluid warmer. Further investigation revealed that fluid in the reservoir of the Level 1 was mixed with blood. We assumed that a communication must have existed between the infused fluid, and the warming fluid within the counter-current aluminum heat exchanger of the warmer. We could not establish if the exchange of fluid occurred unidirectionally (from the *iv* infusate into the warming fluid), or if the patient had been transfused with fluid from the warming reservoir.

We were concerned about the potential for infection because the fluid reservoir is not sterile. Electrolyte disturbances and hemolysis were also potential problems, because of the hypotonicity of the warmer fluid. The patient was continued on prophylactic antibiotics, and cultures of the patient's blood, and the reservoir fluid were obtained. The patient experienced a transient bacteremia, however, the isolates from her blood did not match the isolates from the reservoir fluid. Fortunately, the patient did not suffer any ill effects from this mishap.

We reported the problem to our quality assurance officer, and to the manager of the anesthesia technicians. They involved the Biomedical Engineering Department of the hospital, whose investigators discovered a small hole in the aluminum tube of the counter-current heat exchanger (Figure). It did not appear that this hole was the result of mishandling

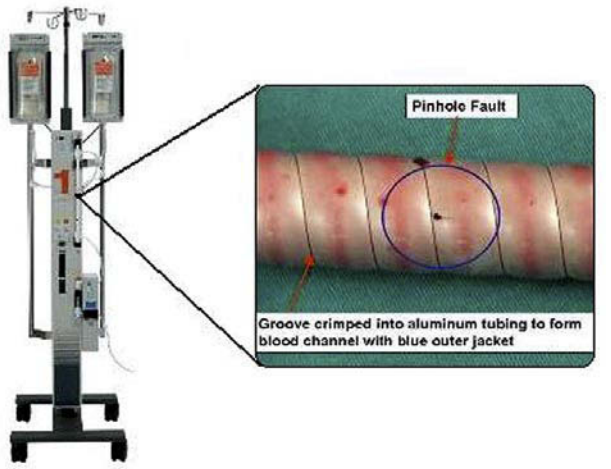

FIGURE The pinhole which allowed communication between the infused fluid and the warming fluid. The fault was found in the aluminum tube inside the heat exchanger disposable unit, as shown.

or faulty installation of the heat exchanger and tubing assembly prior to use. The source of the defect remains unresolved, and the Level 1 manufacturer has been advised of the issues.

This case highlights the importance of testing the integrity of the lines of fluid warming devices prior to their use. The testing is simple, as indicated by this excerpt from our departmental policy for this device:[A] *"attach the disposable set to the Level 1 Fast Flow Fluid Warmer at steps 1, 2 and 3. The unit may then be turned on before connecting the disposable set to any fluid intended for administration to the patient. The appearance of fluid in the disposable set, within a one-minute period, would indicate failure of the disposable and would require a replacement set with a re-test."* The anesthesia technician who set up the room could not recall with certainty that the set had been tested.

A larger issue that this incident raises relates to the safety of counter-current heat exchangers which use fluids as the heat transfer medium. While failures like the one reported are rare, the potential complications from an infection control perspective could be serious. Preoperative inspection and ongoing vigilance when using these devices are warranted. Further, as new technologies emerge which appear to be safer, and equally effective,[2–4] perhaps we could eliminate one more risk from the operating room environment by adopting alternative fluid-warming methods.

A Personal communication with Daniel Cashen, Manager, Department of Anesthesia, Queen Elizabeth II Health Sciences Center, Halifax, NS, Canada.

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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