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Introducer Sheath Malfunction Producing Insidious Air Embolism

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Venous air embolism from the use of catheter introducer sheaths which were not self-sealing¹ has led to the use of self-sealing sheaths. Despite use of a sheath with this safety feature, we encountered an unusual presentation of probable venous air embolism (VAE) which led to pulmonary edema. We describe herein the mechanism of self-sealing valve malfunction and resultant air entrainment.

CASE REPORT

A 64-yr-old man underwent retroperitoneal lymph node dissection. Induction and maintenance of general anesthesia were uneventful. An 8.5 F sidearm introducer sheath (Walrus, Medical Parameters, Inc., Woburn, MA) was placed in the right internal jugular vein for central venous access. As had occurred during a previous laparotomy, peritoneal traction led to sinus arrest, with 2-4-s pauses. Therefore, a consultant cardiologist placed a 6F bipolar pacing wire (USCI Division, C. R. Bard, Inc, Billerica, MA) through the sheath assembly. Although the pacing wire could not be passed into the right ventricle, it did allow effective atrial pacing, and was used intermittently throughout the procedure. At the conclusion of operation, neuromuscular blockade was reversed, and the trachea was extubated in the operating room.

In the recovery room, the patient was alert, with normal vital signs, and breathing comfortably in the semi-sitting position. Approximately 3 h later, a systolic arterial blood pressure of 90 mmHg was noted, and was treated by return to the supine position and iv administration of 300 ml of lactated Ringer's solution. Sinus rhythm continued at 75 bpm. Hypotension again occurred, and was accompanied by an increase in tidal volume and respiratory rate. The patient was placed in the head-down position and given more iv fluid. The pulse oximeter was placed, revealing an arterial oxygen saturation (Sa_{O_2}) of 85% while breathing oxygen *via* facemask. Pa_{O_2} was 33 mmHg, a pH 7.27, and $Paco_2$ 43 mmHg. Electrocardiogram (ECG) and chest radiograph were unchanged from their preoperative appearances. Arterial blood pressure rose to 110/70 mmHg, and cardiac rhythm remained sinus. Central venous pressure (CVP) was below 5 cm H_2O . An endotracheal tube was placed and conventional mechanical ventilation was begun, with Fi_{O_2} = 1.0, positive end expiratory pressure (PEEP) = 5 cm H_2O . Arterial blood gases were then Pa_{O_2} 85 mmHg, pHa 7.36, $Paco_2$ 43 mmHg.

Intermittent hypotension ensued, responding to iv fluids and a dopamine infusion. At this point, a screw cap on the introducer sheath was noted to be missing, with fluid leaking around the pacing wire. Sheath and pacing wire were removed and replaced with a new sheath. A pulmonary artery catheter was inserted several hours later. Initial measurements were pulmonary artery (PA) pressure of 30/12 mmHg, pulmonary artery wedge (PAW) pressure of 12 mmHg, cardiac output of 5.5 L/min, and systemic vascular resistance of 750 dynes \cdot sec \cdot cm^{-5} . A chest radiograph taken 3 h after the initial episode showed diffuse pulmonary edema without cardiomegaly.

Mechanical ventilation was maintained over the next 3 days, during which time the Fi_{O_2} was reduced from 1.0 to 0.5 and PEEP from 12 cm H_2O to 5 cm H_2O , while maintaining Pa_{O_2} greater than 70 mmHg. The patient remained hemodynamically stable, and the iv dopamine infusion was discontinued within 48 h. Serial ECGs and creatine phosphokinase values gave no suggestion of myocardial infarction, and PAW pressures remained below 15 mmHg. The trachea was extubated on the third postoperative day, by which time the chest radio-

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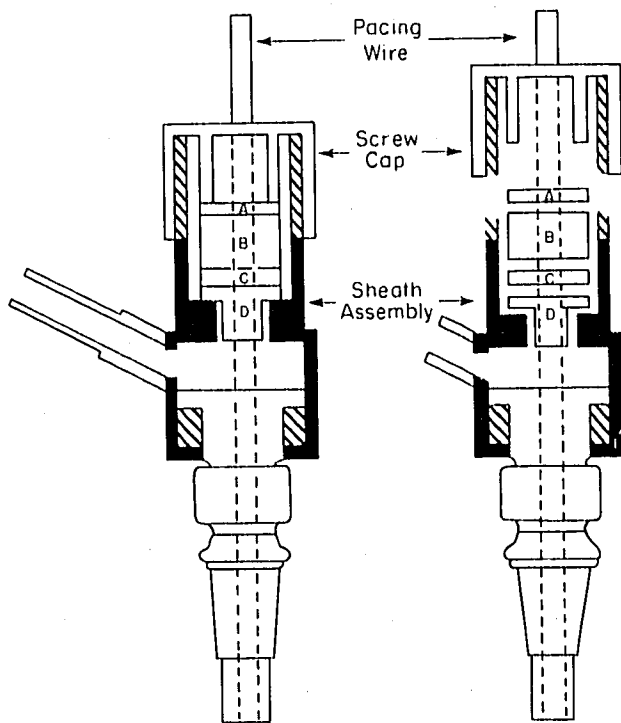


FIG. 1. Diagram of introducer sheath assembly. The left-hand figure shows the properly seated valve components. The right-hand figure illustrates how removal of the screw cap and passage of a wire allows valve components a, b, c, and d, to separate, creating a passage for air entrainment between the components and the sheath assembly. (a, c) spacing washers (b) Tuohy-Borst® type valve (d) anti-reflow self-sealing "duck-bill" valve.

graph had improved dramatically. However, pulmonary dysfunction was still evident on the fifth postoperative day, when P_{aO_2} was 44 mmHg while breathing room air, improving to 81 mmHg with the addition of 50% oxygen by facemask. The patient continued to improve, and he was discharged from the hospital in good condition on the 13th postoperative day.

The introducer sheath and pacing wire were saved intact and tested in the laboratory. A pressure transducer (Gould P231D) was attached to the distal lumen and a 50-ml syringe to the sidearm stopcock. On application of a 5-mmHg subatmospheric pressure, air was drawn through the valve assembly at a rate of 100 ml/min. When the screw cap was correctly replaced, syringe aspiration to pressures of 40 mmHg failed to entrain air. On careful examination of the valves with screw cap removed, it was evident that minor movements of the pacing wire caused separation of the valve components and failure of valve competence (fig. 1).

DISCUSSION

This was an unusual presentation of venous air embolism in the early postoperative period. In the operating room, with the patient in the supine position and receiving positive-pressure ventilation, there was no driving force to entrain air through the misassembled introducer sheath. Once in the recovery room, with the patient in the semi-sitting position, breathing spontane-

ously, and with a low CVP, a pressure gradient could easily occur, allowing insidious intravascular air entrainment. A slow continuous infusion of air, as might occur, would lead to hypotension and hypoxia.^{2,3} Central venous and pulmonary artery pressures would increase with slow infusion of air, probably as a result of pulmonary vasoconstriction, while rapid entrainment of a quantity of air large enough to produce an airlock in the right heart would cause an acute fall in arterial and PA pressures.² As the patient developed respiratory distress, increasing respiratory rate and tidal volume would enhance air entrainment, exacerbating the situation.

While others have described pulmonary edema owing to VAE,⁴⁻⁶ all noted clinically apparent VAE, then related development of pulmonary edema. Venous air embolism was predicted to be a common occurrence in those situations, so monitoring techniques were chosen to rapidly and specifically detect VAE. Venous air embolism occurred in our patient in the recovery room, in a situation in which monitoring to detect VAE is not ordinarily utilized. As a result, VAE was not considered in our patient until the appearance of pulmonary edema and the discovery of the source of air entrainment. There was no clinical or hemodynamic evidence to implicate other common causes of postoperative pulmonary edema, such as congestive heart failure, sepsis, aspiration, or forceful inspiration against a closed airway. Given a source of air entrainment, the appearance of pulmonary edema with no other likely etiology, and the satisfactory resolution with only supportive therapy, we felt confident in making a clinical diagnosis of VAE. A demonstration of increased physiologic dead space (V_D/V_T) or appropriate findings on ventilation-perfusion scanning would have provided more objective documentation of VAE, as well as its resolution.

An introducer sheath can be a source of VAE of significant magnitude, even with only a small pressure gradient favoring air entry.⁷ Our simple technical examination clearly demonstrated that substantial amounts of air could be entrained with minor negative pressures (5 cm Hg), if the valve assembly was mis-seated. This degree of air entrainment was comparable to that reported by Conahan *et al.*⁸ for a defective self-sealing valve (1.36 ml/sec at 30 cm H₂O negative pressure).

Venous air embolism is a well-known complication of central venous cannulation, especially when a large bore catheter is utilized.^{1,8} The 8.5 F introducer sheath inserted into this patient relies on a Tuohy-Borst® type valve, as well as a "duckbill" valve to prevent air leak when the sheath is *in situ* and a catheter or pacing wire has been guided through it, as well as when there is no catheter or pacing wire in place. In addition, the manu-

facturer designed a removable screw cap to seal the port and insure proper alignment of the valve components. Although the apparatus should provide a seal even without the screw cap, passage of the pacing wire, which required a number of attempts, both deformed the valves' seal around the wire and distorted their alignment within the sheath. Without the screw cap in place, a path for air entrainment existed, either through the valves or around them.

Although this report clearly demonstrates misuse of this device by unfamiliar operators, we believe that this episode exemplifies inherently unsafe equipment design. Other introducer sheaths are designed without the removable cap (e.g., Arrow International, Inc., AK-09803, Reading, PA), thus obviating the potential for error. Although not previously reported with this device, another patient at similar risk has been observed earlier by one of us. Postoperatively, this patient was breathing spontaneously, in the 45° head-up position, with the same introducer sheath in the internal jugular vein. Both pulmonary artery catheter and sheath sealing screw cap had been removed, but, fortunately, the valve components had retained their sealed configuration. Experienced anesthesiologists, cardiologists, surgeons, and nurses were involved in these incidents. Redesign of the valve system of this sheath so that it does

not depend on a removable component would improve the safety of the device.

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Is the EEG a Useful Monitor during Cardiac Surgery? A Case Report

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Open cardiac procedures carry the risk of neurologic damage.¹ The use of a neurologic monitor (such as the

electrocephalogram-EEG) during open cardiac procedures would therefore seem logical. However, the role of EEG monitoring during open cardiac surgery has not been defined. Because of the difficulties associated with the intraoperative use of the standard multi-channel EEG, a number of techniques for computerized EEG analysis have been developed²⁻⁵ and incorporated into EEG monitors that are available commercially.

We describe a patient's neurologic status which was being monitored during cardiac surgery by a 2-channel density spectral array EEG (Cerebro Trac™ 2500 EEG Monitor, SRD, Peekskill, NY). In this device, after Fast Fourier Transformation of the EEG signal to produce a power/frequency spectrum, the EEG is displayed as a density-spectral array.² The areas of maximum density of dots correspond to those frequencies making the largest contribution to the EEG spectrum (fig. 1). The spectral edge frequency (SEF; the frequency below

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