CORRESPONDENCE

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The IJ Guide-wire Is "Stuck"

To the Editor:—We encountered an instance of a guide-wire that could not be withdrawn after cannulation of the internal jugular vein. The eventual explanation involved sufficient patient hazard to prompt us to describe it for the benefit of others.

A 75-yr-old man with chronic renal failure (creatinine 13.1, BUN 96) was brought to the operating room for placement of a left forearm A-V fistula and a temporary internal jugular hemodialysis access catheter. An axillary block was performed. The surgical team, using local anesthesia, then undertook the placement of a Hemo-Cath (Medcomp, Harleysville, PA) double lumen catheter in the right internal jugular vein. The vein was entered quickly and uneventfully with an 18-gauge "thinwall" needle. The J-tipped, 70-cm guide-wire was introduced and the vascular access catheter was passed over it. However, the guide-wire could not be withdrawn. The catheter was removed. The guide-wire still could not be withdrawn. The suspicion was that there was a knot in the wire. The possibility that the knot involved other structures or objects was entertained, but was dismissed because no other intravascular foreign bodies (catheters, pacemakers) were present within the great vessels of the head or neck. The possibility of the chordae tendineae of the tricuspid valve being included in the knot was considered.

An image intensifier was obtained. The wire was followed from the neck through the heart and into the abdomen, where an inferior vena caval filter of the Greenfield type (Medi-Tech, Watertown, MA) was identified. The J-tip of the guide-wire appeared to be wedged between two of the struts of the filter (fig. 1). Consultation by the Interventional Radiology Service was sought. The radiologist passed, using the original internal jugular access site and guide-wire, a rigid catheter to and beyond the Greenfield filter. This had the effect of pulling the J-tip caudally out of its wedged location. The tip of the wire was withdrawn to the thorax. The rigid catheter was removed and the dialysis access catheter was passed. The guide-wire was withdrawn uneventfully. The A-V fistula was then completed in the left forearm.

Entrapment of guide-wires or dislodgement of caval filters have been reported six times, beginning with a report in 1993.[1-6] In addition, an *in vitro* evaluation of the phenomenon was reported very recently.[6] Kaufman *et al.* observed entrapment only with J-tipped and not straight guide-wires. That investigation revealed that certain types of filters, in particular Vena-Tech filters (B. Braun Vena-Tech, Evanston, IL), are more likely to entrap wires than others. Their report notes that all of the six reported clinical events involved a Vena-Tech filter (including the one whose title identifies the filter as a "Greenfield"[4]). However, our experience indicates that Greenfield-type caval filters are not exempt from this potential complication.

The particular concern in this instance was that over-vigorous attempts to withdraw the wire, with the unsuspected filter attached, could have resulted in a disruption of the inferior vena cava, with a potential for retroperitoneal exsanguination. When a caval filter is known to be present, care to avoid unnecessarily deep passage of a guide-wire appears appropriate. In addition, anesthesiologists should add unrecognized caval filters to the list of foreign bodies and anatomic structures that can cause entrapment of guide-wires.

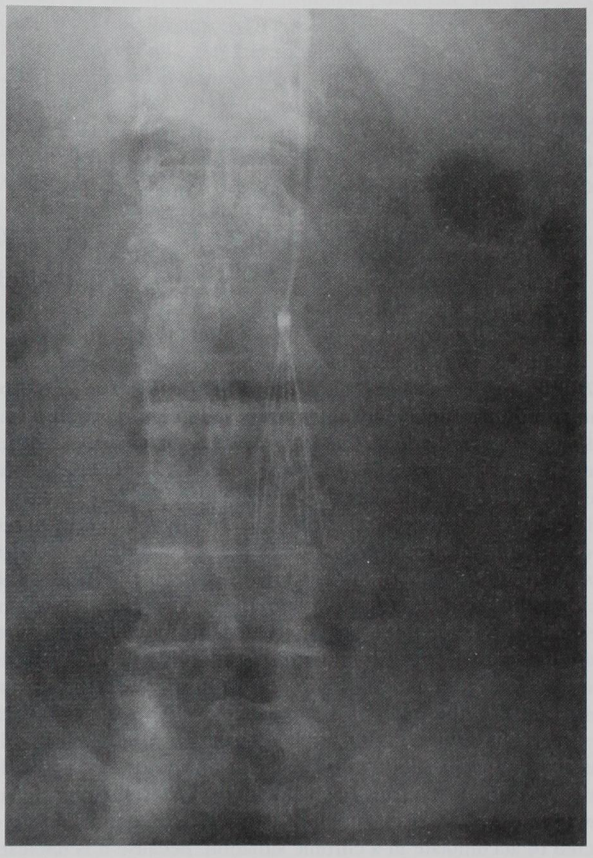

Fig. 1. Anterior-posterior radiograph of the abdomen demonstrating the J-wire engaged with the apex of a Greenfield-type filter located in the inferior vena cava.

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Pharmacokinetic Considerations for the Use of the Ovine Model in Cocaine Research

To the Editor:—In a recent study by Bernards *et al.*, the authors report that chronic cocaine administration reversibly increases isoflurane minimum alveolar concentration in sheep.[1] This study illustrates the dilemma often encountered by researchers when trying to answer a clinically relevant question with a less than ideal animal model.

The authors do acknowledge that no animal metabolizes cocaine in a manner identical to humans, and that the clearance of cocaine in sheep is significantly more rapid than in humans. However, there are other pharmacokinetic considerations that were not mentioned by the authors that are essential whenever using this species for cocaine and drug interaction research. Specifically, the metabolism of cocaine in sheep is quite different from its metabolism in humans and other commonly used laboratory animals.[2] The primary metabolite of cocaine in sheep is ecognine methyl ester, whereas in other species (*e.g.*, human, subhuman primate, rat, guinea pig), benzoylecgonine is the major metabolite. These metabolites are cleared more slowly than the parent compound, and, consequently, would be expected to accumulate in a chronic situation. Because it is unclear as to whether the accumulation of these metabolites alters minimum alveolar concentration, ideally, an animal model in which benzoylecgonine, rather than ecognine methyl ester, would be expected to accumulate would be preferable.

In addition, the investigators used a subcutaneous osmotic pump in this study to deliver cocaine continuously so as to maintain a steady, low background plasma cocaine concentration, ranging from 15 to 69 ng/ml. Although the authors state that this background cocaine infusion was intended to mimic heavy cocaine use, the pharmacodynamic rationale behind this "chronic exposure" with such pharmacologically ineffective low plasma concentrations is unclear. In addition, because of the drug's short elimination half-life in this species, a cocaine infusion rate of 0.4 mg/kg/min over 10 min without an initial loading dose would not produce a pattern of plasma drug concentrations or hemodynamic responses similar to that observed in human "binge" abusers. As previously demonstrated in our continuous intravenous administration study in sheep, the plasma cocaine concentration decreased rapidly when the cocaine infusion was terminated, whereas the benzoylecgonine concentration continued to increase (note that ecognine methyl ester was not determined in this early study). The elimination half-life of cocaine in this study was 5 min.* In addition, in this species, "binge" cocaine doses at 1-h intervals probably do not mimic one important aspect of human "binge" abuse, namely cocaine accumulation. It is unfortunate that the authors did not measure cocaine concentrations during the "binge," because this might have provided more information in regard to drug accumulation.

Based on this knowledge, the investigators similarly should have kept plasma cocaine concentrations in a range occurring in human "binge" abusers by administering a programmed constant intravenous infusion at a high initial rate followed by a gradual decrease in rate.

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* Morishima HO, Chao CR, Matsuo M, Abe Y, Cooper TB: Placental transfer and disposition of cocaine in the pregnant sheep (abstract). ANESTHESIOLOGY 1992; 77:A1038.