discussed in our original article.[3] However, the potential confounding factors of equipment familiarity and preparation for a posttest would be unlikely to be eliminated by a control group. To prevent an improvement in performance from residents preparing for the posttest, we did not warn them in advance. As for the instructional video, hands-on teaching was shown to be far superior to didactic teaching for procedural skill acquisition.[4]

The larynx of the Laerdal SimMan is anatomically correct in contrast to the low-fidelity model used and is commonly referred to in the literature as a high-fidelity model. We agree that the issues of cost, availability, and portability are central to the interpretation of our findings as highlighted in our article. Cadavers, although obviously an anatomically perfect model have their own drawbacks, including the stiffness of the tissues and the technical difficulty of aspirating air from the formalin-filled trachea. The main problem with cadavers is of course their scarcity, which was one of the most important reasons we undertook this study.

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Propofol: Pro- or Anticonvulsant Drug?

To the Editor:

As part of a review discussing the occurrence of seizures associated with general anesthetics, Voss et al.[1] stated that it is still unclear whether propofol is pro- or anticonvulsant. The typical electroencephalogram pattern after propofol sedation in healthy subjects is an initial increase in frequency from α to β waves, followed by a slowing to δ waves.[2,3] In our prospective study,[4] we demonstrated distinct electroencephalogram patterns after propofol-induced sedation in children with epilepsy and children with learning difficulties. Whereas children with learning difficulties did not develop spike-wave patterns, the most notable characteristic seen in children with epilepsy was suppression of spike-wave patterns. None of our study patients developed seizure-like phenomena.

The effects of propofol on epileptic discharge and seizure activity may be related to its uniform depressant action on the central nervous system, to a potentiation of γ-aminobutyric acid-mediated pre- and postsynaptic inhibition by enhancing inward γ-aminobutyric acid_A Cl^- currents, and by decreasing the release of excitatory transmitters glutamate and aspartate.[5,6] Conversely, a potential mechanism for the proconvulsant properties of propofol may be due to intrinsic subcortical glycine antagonism as suggested by animal data.[7]

A systematic review demonstrated a predominance of seizure-like phenomena during induction, emergence, or delayed after anesthesia or sedation with only a few events during maintenance (when the level of anesthesia is relatively constant) in nonepileptic patients, indicating that seizure-like phenomena tend to occur during changes of blood and brain tissue levels of propofol.[8] However, most of these events were classified as seizure-like phenomena of nonepileptic origin (events with an increased tone with twitching and rhythmic movements not perceived as generalized tonic-clonic seizures, opisthotonos, or involuntary movements). In patients with epilepsy, seizure-like phenomena of epileptic origin after propofol were very rare, and most often they occurred during recovery.[8] It is also important to note that pro- and anticonvulsant mechanisms are modulated by the given dose of propofol as inhibitory central nervous system structures are more sensitive to depression than excitatory ones.[9]

In summary, there is increasing evidence from clinical studies demonstrating that propofol possesses anticonvulsant properties.[1,6] Only in very rare circumstances is the administration of propofol associated with seizure-like phenomena, most commonly of nonepileptic origin.[8]

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In Response:

We agree with Meyer et al.[1] that true seizures with propofol are rare, but not zero; the most risky periods for both true seizures and abnormal nonseizure movements are during the recovery or induction phases of anesthesia, and that propofol has predominantly depressive effects on the cortex.

However, we also maintain that seizures with thiopental are probably even more rare than those after propofol (although in the absence of an accurate denominator this could be disputed) and that induction of cortical depression is often associated with paradoxical cortical hyperexcitability. Meyer et al. seem to agree with this, as their sentence: "It is also important to note that pro- and anticonvulsant mechanisms are modulated by the given dose of propofol as inhibitory central nervous system structures are more sensitive to depression than excitatory ones" would support a proepileptogenic mechanism of propofol. In our article, we termed this mechanism "inhibiting-the-inhibitors."[2]

We would therefore suggest that the correct title of this letter should be: "*Propofol: Pro- and Anticonvulsant Drug*."

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Intrathecal Morphine Pump Malfunction Due to Leakage at the Catheter Connection Site: A Rare Problem and Its Prevention

To the Editor:

Implanted intrathecal drug delivery systems provide excellent pain relief and reduced drug toxicity in patients with refractory cancer pain.[1,2] Common complications include postpuncture headache (15%), external leakage of cerebrospinal fluid (3.5%), CSF hygroma (1.5%), catheter tip dislodgement (1.5%) and catheter system leakage (1.5%). Infection of implanted systems are a rare occurrence, mostly in the first 3 mo after implantation.[3]

We report a pump malfunction resulting from pump-catheter connection failure and a strategy to avoid this complication. A 72-yr-old patient with bladder cancer metastatic to the sacrum and lumbar plexus was receiving the equivalent of 750 mg morphine per day postoperatively, but with significant sedation and still inadequate pain control. A percutaneous intrathecal catheter was placed and an infusion of morphine begun. The patient improved rapidly, showing much lower pain scores while being alert and mobile. Subsequently, an intrathecal drug delivery pump (Medtronic Synchromed II 8637) was implanted. An epidural fibrin glue patch was performed at the catheter entry site to the dura to minimize the risk of cerebrospinal fluid leakage along the catheter.[4] In the first postoperative days, the daily morphine dose had to be increased rapidly without improvement of the pain scores. At the same time, swelling and fluctuance over the pump pocket was noted. The pump pocket was punctured and blood-tinged fluid aspirated. The catheter side port of the pump was accessed under fluoroscopy and, upon injection of contrast medium, a leak was identified at the pump-catheter connection site (Fig. 1). Upon surgical revision, the catheter connector was in place but could be disconnected easily. The snap connector extension was replaced and the new piece reattached to the pump with considerable extra force. The connection was then manually tested for leak using a saline injection in the catheter access port while occluding the catheter distally, a procedure we now routinely use for all pump implantations. Laboratory examination of the aspirated fluid showed no bacterial growth and was positive for β transferrin, confirming a retrograde cerebrospinal fluid leakage to the pump pocket. According to an oral communication from the Medtronic

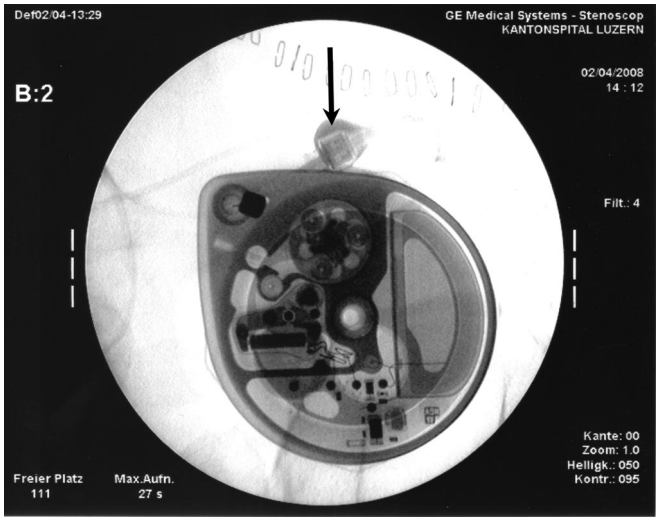

Figure 1. Leakage of contrast dye at connection site of intrathecal catheter and pump.