

Letters to the editor

Inadvertent subcutaneous infusion of propofol by a syringe pump

Rumiko Uda

Department of Anesthesiology, Osaka Medical College, 2-7 Daigaku-cho, Takatsuki, Osaka 569-8686, Japan

To the editor: A 19-year-old, 51.5-kg healthy woman underwent arthroscopy and open reduction for fractures of the tibial spine. Atropine and hydroxyzine were administered 30 min before induction. Anesthesia was induced with propofol and pentazocine, and vecuronium was given to facilitate endotracheal intubation. Continuous propofol infusion was initiated (10 mg·kg⁻¹·h⁻¹) with a syringe pump exclusive for this agent (Syringe Pump Model STC-525X, Terumo, Tokyo, Japan) via an extension tube and a three-way stopcock attached to a lactated Ringer's solution line connected to a 20-gauge cannula placed in the left cephalic vein. Ten minutes after initiation, the infusion rate was reduced to 5 mg·kg⁻¹·h⁻¹, maintained for 170 min, followed by 3 mg·kg⁻¹·h⁻¹ for the final 20 min of the operation. Before incision, lidocaine containing epinephrine was infiltrated. The anesthesia was maintained with 67% nitrous oxide and additional vecuronium without apparent sign of light anesthesia and with no operational difficulties with the intravenous line or the syringe pump. At the completion of the 3-h operation, the surgical drapes were removed, and edema was observed in the patient's entire left forearm and hand. The left radial artery was palpable, and pulse oximetry measurement on the second digit of the affected hand was stable at 98%–99%, with normal pulse waveform. Emergence was not delayed. The patient's trachea was extubated after she was able to follow oral commands. She never complained of pain in the arm and slept well overnight. During the first 15 postoperative hours, she could not recall any events following induction. The left arm edema completely subsided.

I did not note extravasation until the arm was uncovered because of the stable anesthesia and correctly functioning pump. The lower limit of the occlusion detection pressure of the pump, which was initially set at 300 mmHg, has been reset at 400 mmHg since 1996 by the manufacturer, because there

was a complaint about false detection during infusion [1]. On trial, when a stopcock connected to the intravenous line was completely closed or the line was clamped proximally to the patient, it took 8 and 15 min, respectively, for the detection alarm of the STC-525X to operate. This fact suggests that the alarm may be delayed if propofol infusion is partially interrupted, or may never be in operation due to less resistance if extravascular infusion occurs. The patient may be awake before the alarm functions unless blood levels of anesthetics are sufficient.

Even a large aliquot of intraarterial propofol appears benign and without need of treatment [2]. However, there have been rare reports of transient cyanosis [3], local pain, swelling, blisters, and/or necrosis following accidental extravasation [4]. Fortunately, this patient had no major symptoms or sequelae. If extravasation occurred during the positioning change 50 min after induction, the amount of 1% propofol would be 646 mg administered subcutaneously and it would be diluted 10 times for 130 min. Dilution and a slow infusion rate could reduce the incidence of severe damage.

Interestingly, this young patient had no intra- or postoperative recall in spite of failed propofol administration. To my knowledge, there are no reports of systemic effects such as postoperative sedation or respiratory or cardiovascular depression caused by possible reabsorption following extravasation of propofol [2–4]. Because the sedative effect itself was found to be highly variable among individuals [5], propofol administered extravascularly may have been slowly reabsorbed, resulting in postoperative sedation and memory impairment.

I suggest that attention should be paid to extravasation irrespective of alarm operation in the present setting of STC-525X, which might cause intraoperative awakening and possibly postoperative systemic side effects.

References

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