

CASE REPORTS

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Unintentional Arterial Catheterization and Bupivacaine Toxicity Associated with Continuous Interscalene Brachial Plexus Block

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Continuous interscalene brachial plexus block with bupivacaine has been used effectively in our hospital for patients undergoing shoulder surgery and for postoperative pain relief.¹⁻³ No serious complications have ensued despite doses of bupivacaine that generally exceed the recommended doses.

We report an unintentional intraarterial catheterization and toxic bupivacaine reaction associated with the catheter approach to the brachial plexus.

CASE REPORT

The patient was a 55-yr-old man in good general health (weight 88 kg and height 187 cm) admitted for manipulation of the right shoulder. Preanesthetic medication included oral diazepam 15 mg and intramuscular oxycodone 14 mg on the ward and intravenous fentanyl 0.1 mg in the operating room. He was given an interscalene brachial plexus block using the technique described by Winnie.⁴ A nerve stimulator (DualStim®, Life-Tech, Houston, TX) connected to the proximal end of the metal inner needle of a plastic plexus block cannula (Contiplex®, B. Braun Melsungen AG, Germany) was used to identify the nerve plexus. A volume of 20 ml 0.75% bupivacaine with epinephrine (5 µg/ml) was injected through the cannula into the interscalene space with frequent repeated aspirations during the injection. An interscalene catheter (OD 0.85 mm, Contiplex®, B. Braun-Melsungen AG, Germany) then was introduced easily through the plastic plexus cannula. The catheter was fixed to the skin with a tight suture. The remainder of the local anesthetic volume, 6 ml, to achieve the total dose of 26 ml, was injected through the catheter, again with careful aspiration during and after the injection. No blood or cerebrospinal fluid was detected in the catheter. It later was noted that the distance of the catheter tip from the skin was 12 cm.

A few seconds after the injection through the catheter the patient complained of an uncomfortable feeling. Shortly thereafter he lost consciousness and his breathing became shallow. Manual ventilation with 100% oxygen was started *via* bag and mask, and the hemoglobin oxygen saturation was monitored. The arterial blood pressure decreased to 70/50 mmHg and etilefrine (an α - and β -adrenergic agonist) 4 mg was given intravenously. Blood pressure returned to normal and remained between 90/60 and 130/80 mmHg. Except for transient

tachycardia after etilefrine (heart rate 130 beats per min), no dysrhythmias were detected, and ECG monitoring showed normal sinus rhythm, with a heart rate of 55-65 beats per min. Naloxone hydrochloride 0.2 mg was given three times to antagonize the effect of the opioid premedication. The patient's pupils were dilated and showed no reaction to light, and he was apneic.

After 35 min the trachea was intubated, and ventilation was mechanically controlled. Sixty minutes thereafter the patient opened his eyes, was conscious, and started to breathe spontaneously. The trachea was extubated 2.5 h after the beginning of the toxic symptoms. The patient had sensory and motor block in the area innervated by the nerves derived from the right brachial plexus and no signs of nerve block on the opposite side or on the lower extremities.

Arterial blood samples were taken during the toxic phase. The plasma concentrations of bupivacaine measured by gas chromatography⁵ were 2.37 µg/ml at 5 min, 1.49 µg/ml at 10 min, 1.30 µg/ml at 30 min, and 1.23 µg/ml at 60 min. The planned manipulation of the shoulder was performed during the period of unconsciousness. Postoperative analgesia was good. The catheter, from which aspiration of any fluid was impossible, was left in place until the next day, when it was injected with contrast medium in 1-ml increments (fig. 1). This revealed that the catheter was situated quite medially close to the cervical vertebrae, making a sharp angle of about 90° in the caudad direction. After each injection, the contrast medium disappeared immediately from the site of the catheter tip. Still at the time of removal of the catheter, after the radiographs, aspiration of fluid gave a negative result. The patient's recovery was uneventful.

DISCUSSION

Central nervous excitation with convulsions followed by coma are common manifestations of severe systemic local anesthetic toxicity.⁶ Generalized bupivacaine toxicity is associated also with cardiovascular depression and ventricular arrhythmias.⁷ Resuscitation after bupivacaine-induced cardiac dysrhythmias has been found to be difficult, and deaths have been reported.^{8,9}

In this patient unexpected instantaneous coma without convulsions was the first sign of toxicity a few seconds after injection of 45 mg bupivacaine with 30 µg epinephrine through the plexus block catheter. The reason for the lack of convulsive activity may be related in part to the diazepam premedication, which has been shown to decrease the incidence of local-anesthetic-induced seizures in animals.¹⁰ The arterial bupivacaine blood concentration of 2.37 µg/ml was at a level occasionally observed during successful continuous brachial plexus block without toxic symptoms.² However, the bupivacaine concentration of the blood reaching the brain probably was much greater, since the local anesthetic was injected directly into the

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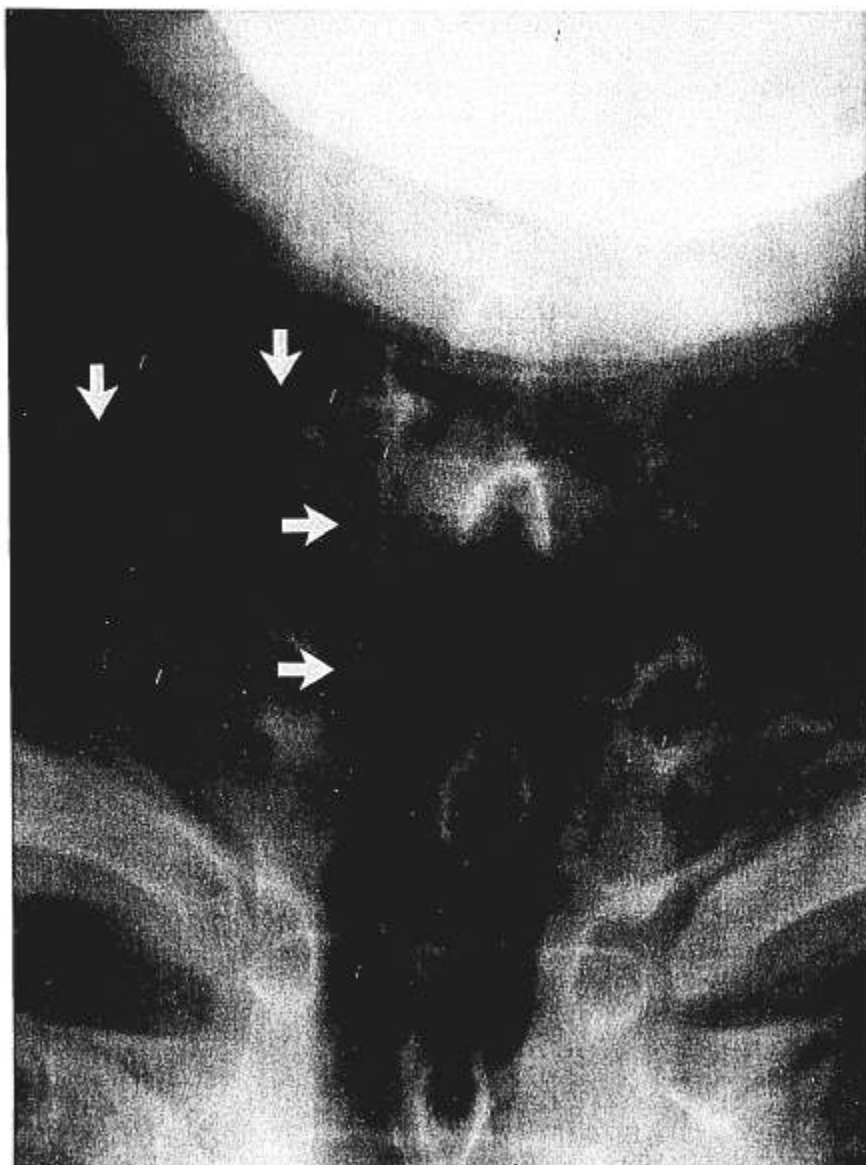
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FIG. 1. Radiograph of the intraarterially positioned plexus catheter (0.85 mm OD) visualized with contrast medium. The arrows outline the catheter and indicate the 90° bend in the catheter.



vertebral artery.¹¹ The rapid administration of a substantial dose of bupivacaine directly into the brain resulted in central nervous system (CNS) depression with apnea, but without a typical initial excitation phase. The long duration (95 min) of unconsciousness and apnea was due probably to the strong binding of bupivacaine to brain tissue. The CNS toxicity could not have been related to bupivacaine plasma concentration (1.23 $\mu\text{g}/\text{ml}$ at 60 min).

Although bupivacaine may excite the heart indirectly *via* the CNS,^{12,13} the general anestheticalike deep CNS depression may also be the reason for the lack of cardiovascular toxicity by this particular mechanism. Considering the binding of bupivacaine to the brain, uptake of a large amount of the drug in the lungs during the first passage,¹⁴ and dilution by mixing with venous blood, the arterial concentration of bupivacaine reaching the coro-

nary arteries had declined significantly and thus was not deleterious to the heart. Actually, the only sign of cardiac toxicity in our patient was a modest decrease in arterial blood pressure.

Absorption of bupivacaine into the circulation from the initially injected bupivacaine dose must have contributed to the measured plasma concentrations. In our recent study of continuous interscalene brachial plexus block, the bupivacaine plasma concentrations measured 5, 30, and 60 min after the initial dose did not show a peak (1.63 $\mu\text{g}/\text{ml}$) until 30 min; at 5 min the mean concentration was 0.82 $\mu\text{g}/\text{ml}$.¹⁵ In the current patient, the bupivacaine concentration reached its highest level at 5 min and decreased steadily thereafter.

In this patient the catheter introduced for continuous interscalene brachial plexus block was located apparently

in the vertebral artery. Ipsilateral anesthesia of the arm and shoulder and lack of anesthesia of the lower extremities speak against spinal or epidural location of the tip of the catheter. Kinking of the catheter, observed as a 90° angle in the radiographs, might explain why aspiration of blood before and during the injection was unsuccessful. This patient had had four interscalene brachial plexus blocks performed within 15 months. Therefore, tissue damage caused by the block needles or catheters as well as by the local anesthetics may have contributed to the ease of arterial perforation.¹⁶

Based on our clinical experience, including interscalene catheterization as a routine procedure, the catheter sometimes can be advanced more than the usual 2–3 cm past the tip of the introducer cannula, *i.e.*, 4–8 cm from the skin. Despite the relative softness of the plexus block catheter (polyamide), vascular puncture is possible, as our case shows. Repeated careful aspiration before and during the injection of the local anesthetic injection did not prevent a serious toxic complication.

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