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Minimizing the Risk of Intravascular Injection during Ultrasound-guided Peripheral Nerve Blockade

To the Editor:—We read with interest the recently published case reports by Loubert *et al.*¹ and Zetlaoui *et al.*² regarding suspected inadvertent intravascular injection of local anesthetic (LA) and LA toxicity during ultrasound-guided axillary brachial plexus blockade. While ultrasound likely reduces the risk of accidental vascular puncture compared with “blind” peripheral nerve stimulation,³ these two case reports demonstrate that the risk of intravascular injection persists despite ultrasound guidance.^{1,2} In 2006 and 2007 at the Toronto Western Hospital, we performed 1,797 ultrasound-guided brachial plexus blocks without any sign or symptom of LA toxicity. Good fortune notwithstanding, there are several important principles that merit thoughtful consideration to improve detection of accidental intravascular injection and possibly prevent LA toxicity during ultrasound-guided peripheral nerve blockade. We believe that the most reliable feature during real-time ultrasound imaging indicative of intravascular injection is the failure to visualize a hypoechoic fluid bolus on the ultrasound monitor during and/or after injection of as little as 1 ml of injectate. Indeed, we customarily initiate LA injection with a 1-ml bolus to exclude intravascular or intraneural^{4,5} needle tip placement before proceeding with 5-ml increments of injectate. Visualization of the needle tip does not preclude intravascular injection *per se*; it is the real-time observation of hypoechoic fluid causing tissue dispersion that most consistently excludes intravascular injection. The absence of a discernible extraneural hypoechoic fluid bolus on the ultrasound monitor means that either the needle tip is intravascular or the plane of imaging is inaccurate.

We are also hesitant to recommend the use of ultrasound for peripheral block techniques as described by Loubert *et al.*¹ Rather, we contend that perineural LA deposition is the safest application of ultrasound technology. There can be multiple veins traveling alongside landmark pulsatile arteries, especially in the axilla. Veins are exquisitely collapsible with even the slightest amount of pressure applied by the transducer to the skin, and can therefore vanish from sonographic view, fooling even the most experienced providers, as demonstrated by these two recent case reports.^{1,2} We therefore use systematic scanning of the intended block site before needle insertion. Our

systematic sonographic survey includes sliding the transducer distally and proximally to trace the target nerve along its expected course and examine the surrounding vasculature and tissues. Scanning is performed with varying degrees of pressure, with and without the use of color Doppler, to identify any hazards that may cross the planned trajectory of the needle. In addition, applying pulse wave Doppler over a nearby vessel during perineural injection may help to detect inadvertent intravascular injection by the characteristic high-pitch sound of turbulent flow associated with a sudden rush of fluid. Finally, however contentious,⁶ we strongly believe that patients undergoing ultrasound-guided peripheral nerve blockade should remain awake with judicious sedation so that signs and symptoms of LA toxicity can be recognized, communicated, and treated immediately upon onset.

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Ultrasound-guided Peripheral Nerve Blocks and Intravascular Injection

To the Editor:—In the April issue of *ANESTHESIOLOGY*, Loubert *et al.*¹ and Zetlaoui *et al.*² reported about possible intravascular injection after an ultrasound-guided axillary block. Their reports highlight the need for vigilance in the performance of ultrasound-guided blocks. This and similar reports of complications³⁻⁵ after ultrasound-guided regional blocks reinforce the need for proper training, and the understanding that ultrasound, after all, is only a tool. Any tool should be used with full cognizance of its limitations. The major limitations of ultrasound-

guided blocks are technical,⁶ including the angle of incidence, needle visualization, and possibly artifacts.^{7,8} Training in the proper holding of the probe while analyzing and while injecting help overcome some of the complications. Sometimes even with proper training, complications do occur.⁴

The reports^{1,2} have similarities and differences besides the ultrasound-guided axillary block and intravascular complication leading to seizure. One of them described the changes in vital signs,¹ and the

other reports some technical difficulties in getting the data.² Needle visualization was an issue in both reports. Using an out-of-plane approach may have prevented proper needle visualization because only a cross section of the needle anywhere in the length of the needle may be seen and mistaken for the tip, although tissue movement may have been seen. With the in-plane approach used in the other report, needle artifacts may have prevented proper visualization, which will only be discerned when the injectate spread is noticed. Both reports mention distortion of tissues, one due to probe pressure and the other due to local anesthetic already injected.

There is no documentation in either of the reports of having seen other vessels in the proximity before the actual needle placement. Assuming they used color flow Doppler, the default settings for the color Doppler cannot detect small vessels unless the color velocity range and the angle of steering are adjusted. It is possible that they did not visualize the needle during the performance of the block and hence did not adhere to one of the safety principles that they have mentioned. Any of these situations could have led to the complication. Most importantly, they were both performed by residents.

My practice is to perform a preliminary scout scan, including a color flow study, to visualize the target and its associated neighboring structures and demonstrate to the trainee. This permits proper guidance during the actual performance of the block. Could they have avoided the intravascular injection by using landmarks or nerve stimulation? Probably not.

To elevate ultrasound-guided to the next level and call it a "bullet-proof technique" by the more "vocal proponents"⁶ is a dream awaiting fruition with some more technological advancements and changes in needle design. In the meantime, adhering to some basic principles will avoid potential complications. To blame the ultrasound for complications due to technical and possibly inadequate training is, in my opinion, tarnishing a useful technique without understanding its ad-

vantages and mainly its limitations. There is an increasing need for a proper curriculum and training to fully understand the technique, the potential pitfalls, and the complications of ultrasound-guided blocks.⁹

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Accidental Intravascular Injection of Local Anesthetic?

To the Editor:—I read with great interest the recent case report detailed by Loubert *et al.*¹ I respectfully disagree with their conclusion of this being a case of local anesthetic toxicity. Presuming, based on their case description, that only 5 ml local anesthetic was injected into a blood vessel and minimal perivascular uptake occurred from the previous injections, a maximum of 75 mg lidocaine was inadvertently injected intravascularly.¹ This amount of local anesthetic is unlikely to produce the necessary blood levels to create central nervous system symptoms.²

An alternative explanation is that the associated intravascular administration of epinephrine, which expectedly caused a hypertensive response, disrupted the blood-brain barrier and the defective blood-brain barrier produced sufficient cerebral edema to generate the witnessed symptoms.³ The patient's symptoms of agitation and loss of consciousness were likely from hypertensive encephalopathy or reversible posterior leukoencephalopathy syndrome.^{1,4} Clinical manifestations of both of these hypertensive-related syndromes overlap with central nervous system local anesthetic toxicity and include restlessness, confusion, altered consciousness, seizures, and coma.^{3,4} These symptoms stem from altered cerebral autoregulation and endothelial dysfunction.³

The patient, assumed from her American Society of Anesthesiologist physical status of I to be normotensive, had a documented blood pressure of 280/130 mmHg during the described symptoms.¹ Hypertensive encephalopathy has been seen with diastolic readings of as low as 100 mmHg in patients without preexisting hyperten-

sion.⁴ As blood pressure exceeds the threshold of cerebral autoregulation, a hyperperfusion situation exists that may disturb the blood-brain barrier and cause cerebral edema.⁵ The resultant cerebral edema can lead to symptoms not dissimilar to those described by the patient in question.⁵ In cases of autoregulatory failure, the rate of blood pressure elevation is pivotal in the pathogenesis of both hypertensive encephalopathy and reversible posterior leukoencephalopathy syndrome.⁶ A rapid increase in blood pressure, from the alleged intravascular epinephrine, was no doubt present in the case report.¹ Neuroimaging, although not performed in this case, may have revealed cerebral edema.⁷ When cerebral edema is primarily localized into the posterior cerebral hemispheres and is coupled with the clinical picture of restlessness, confusion, altered consciousness, seizures, or coma, a diagnosis of reversible posterior leukoencephalopathy syndrome should be entertained.⁷ With reversible posterior leukoencephalopathy syndrome, a complete recovery is typically seen after blood pressure is controlled and stabilized.³

It seems that the rapid onset and offset of symptoms in this case would likely correlate with epinephrine, not lidocaine or bupivacaine, serum levels. Patient symptomatology paralleled the elevation and subsequent normalization of the recorded blood pressures. In summary, I propose the intravascular epinephrine provided a positive stress test to the patient's blood-brain barrier and that the concomitantly intravenously administered local anesthetic may have been an inert bystander.