other reports some technical difficulties in getting the data. 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 was 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” 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 advantages 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.

Hariharan Shankar, M.B.B.S., Clement Zablocki VA Medical Center and Medical College of Wisconsin, Milwaukee, Wisconsin. hshankar@mcw.edu

References


(Accepted for publication August 7, 2008.)

Accidental Intravascular Injection of Local Anesthetic?

To the Editor—I read with great interest the recent case report detailed by Loubert et al. 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 peripheral uptake occurred from the previous injections, a maximum of 75 mg lidocaine was inadvertently injected intravascularly. This amount of local anesthetic is unlikely to produce the necessary blood levels to create central nervous system symptoms.

An alternative explanation is that the associated intravascular administration of epinephrine, which usually caused a hypertensive response, disrupted the blood-brain barrier and the defective blood-brain barrier produced sufficient cerebral edema to generate the witnessed symptoms. The patient’s symptoms of agitation and loss of consciousness were likely from hypertensive encephalopathy or reversible posterior leukoencephalopathy syndrome. 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. These symptoms stem from altered cerebral autoregulation and endothelial dysfunction.

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. Hypertensive encephalopathy has been seen with diastolic readings of as low as 100 mmHg in patients without preexisting hypertension. As blood pressure exceeds the threshold of cerebral autoregulation, a hyperperfusion situation exists that may disturb the blood–brain barrier and cause cerebral edema. The resultant cerebral edema can lead to symptoms not dissimilar to those described by the patient in question. 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. A rapid increase in blood pressure, from the alleged intravascular epinephrine, was no doubt present in the case report. Neuroimaging, although not performed in this case, may have revealed cerebral edema. 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. With reversible posterior leukoencephalopathy syndrome, a complete recovery is typically seen after blood pressure is controlled and stabilized.

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.

Anesthesiology 2008; 109:1143–4
In Reply:—We thank Drs. Brull et al., Shankar, and Nelson for responding to our case report of accidental intravascular injection of local anesthetic and epinephrine during ultrasound-guided perivascular axillary block.1

The suggestions provided by Dr. Brull’s group for improved safety during ultrasound-guided axillary block seem reasonable. The large case series of axillary blocks recently published by Dr. Brull et al. bears witness to their experience of significantly reduced (but not completely eliminated) rates of accidental intravascular injection with the adoption of ultrasound guidance compared with the blind transarterial or neurostimulator-guided techniques used and taught until recently at their institution.2 Further large case series such as theirs, or the establishment of a complication registry will be needed to quantify the relative safety benefits of various preblock precautions and ultrasound-guided approaches to axillary blockade (including perivascular vs. perineural injection). However, there seems to be little doubt that future improvements in block safety lie in the optimal application of ultrasound training and imaging, and technical advances including echogenic atrumatic needles specifically designed for regional anesthesia.

To Dr. Shankar, the problems we wished to highlight in our case report include modification of anatomical relations by injection of local anesthetic leading to migration of the needle tip into a blood vessel, and the existence of small, compressible, low-flow veins that are difficult to detect with even the most sophisticated ultrasonic equipment, experienced operators, and careful scanning techniques. These problems may be mitigated by technical and educational improvements, but we wished to emphasize that continued adherence to traditional safety rules such as fractionated injection is necessary even in the ultrasound age of regional anesthesia. Blaming ultrasound guidance for the complication we present in our report would constitute in our opinion a misinterpretation of the events we related.

Dr. Nelson brings up the interesting point that 75–100 mg lidocaine would not be expected to result in the neurologic symptoms presented in our report, and proposes the alternative diagnosis of hypertensive encephalopathy or reversible posterior leukoencephalopathy secondary to the epinephrine in the block solution. Although we agree that the dose of lidocaine administered intravenously was relatively small (due to fractionated injection with ultrasonographic confirmation), we believe the time course of our patients’ symptoms (minutes, rather than days for the other evoked diagnostic possibilities) are more consistent with a high but transient peak concentration of lidocaine, possibly potentiated by the epinephrine in the solution.3

Christian Loubert, M.D., Stephan R. Williams, M.D., Ph.D.*
*Centre Hospitalier de l’Université de Montréal, Montreal, Quebec, Canada. stephan.williams@umontreal.ca

References


(Accepted for publication August 7, 2008.)