Local Anesthetic Cardiac Toxicity can Present as Late-onset Hypotension, Bradycardia, and Asystole

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To the Editor:

We applaud Dr. Polley and Dr. Santos for reminding us in their recent editorial of the potential risks of anesthetic toxicity in peripheral nerve block and of the need to employ standard safety methods to reduce the risk of cardiac toxicity in all forms of regional anesthesia. However, we believe their discussion missed another, equally important, lesson to be learned from these interesting case reports of ropivacaine-induced cardiac arrest.

Both cases presented with hypotension, bradycardia, and asystole. Drs. Polley and Santos state that as bupivacaine would be expected to produce ventricular tachycardia or fibrillation, the arrhythmias produced in humans by ropivacaine and bupivacaine are different. In fact, severe hypotension and bradycardia leading to asystole are commonly seen in animal models of bupivacaine cardiac toxicity, and we believe their occurrence during a regional anesthetic should lead to the inclusion, not exclusion, of local anesthetic toxicity from the differential diagnosis.

More importantly, Chalazon et al. report cardiac toxicity 90 min after a ropivacaine-based sciatic block and 30 min after smaller supplementary injections for inadequate sensory anesthesia. Traditionally, the natural history of local anesthetic toxicity is described as occurring immediately after inadvertent intravascular injection of anesthetic. However, as this case indicates, patients can present with delayed onset severe local anesthetic cardiac toxicity. This phenomenon is sufficiently different from the standard textbook presentation that it can be easily overlooked or misdiagnosed. For instance, we recently commented on a case report of cardiac arrest occurring 105 min after a combined bupivacaine-mepivacaine brachial plexus block. The possibility of anesthetic toxicity was not mentioned in the report and was later dismissed by the authors on the basis of the long interval between injection and the arrest and because the presenting arrhythmia was junctional bradycardia leading to asystole, not ventricular tachycardia or fibrillation.

We believe that the natural history of local anesthetic toxicity may encompass a much broader clinical phenotype than is indicated in standard textbook descriptions. It is likely that atypical cases of local anesthetic toxicity go unrecognized and are underreported. Given the recent evidence for a possible form of therapy specific for local anesthetic cardiac toxicity, it is particularly important to contemplate this diagnosis in patients having signs of cardiac dysfunction in the setting of regional anesthesia, even if the arrhythmias or time course vary from a classic presentation.

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