Correspondence

Endpoint for successful, ultrasound-guided infraclavicular brachial plexus block

To the Editor:

We congratulate Dr. Morimoto *et al.*[1] for their excellent work demonstrating the existence of a septum that restricts the diffusion of local anesthetic (LA) during ultrasound-guided infraclavicular block. This study confirms our clinical impression that a septum, or a fascia, must be pierced on the posterolateral aspect of the subclavian artery (SA) to ensure a reliable block of the brachial plexus.

At our institution, we perform ultrasound-guided infraclavicular block in a manner very similar to that described by Dr. Morimoto; however, we use a 20G Tuohy needle (BBraun, Bethlehem, PA, USA) and we aim to position the tip of the needle at the very posterior aspect of the SA before the injection. Using such a non-cutting needle allows us to consistently feel the passage of the posterolateral septum. In fact, this fascial click has become our primary endpoint to confirm a good needle position. We have observed that this technique strongly predicts a U-shaped distribution of the LA and an anterior displacement of the SA. In our practice, the combination of these three factors; fascial click, U-shaped distribution of LA, and anterior displacement of the SA, is highly predictive of a rapid and complete block of the entire arm. In contrast, if the fascial click is not perceived before the injection, even if the needle tip is posterior to the artery, a lateral distribution of the LA and a caudad or posterior displacement of the SA is often observed. This scenario often leads to an incomplete block, or to a delayed onset, before adequate anesthesia is achieved.

The existence of this septum can explain why studies of neurostimulation-guided, infraclavicular block show better success rates, when administering injections on a posterior cord motor response, rather than on a medial or lateral cord motor response.[2,3] It may also explain the findings of Dingemans *et al.*[4] who demonstrated an excellent success rate when obtaining a U-shaped distribution of the LA.

Sauter *et al.*[5] recently reported an MRI study which delineates the position of the three cords in relation with the SA. Based on their observations, it is suggested that an optimal target point exists closest to all three cords on the posterolateral aspect of the SA.[5] In our experience, using this injection point seldom pierces the septa and often leads to incomplete block. As anesthesiologists gain more knowledge regarding the anatomical relations of the brachial plexus with the use of ultrasound, it becomes clear that for regional anesthesia of the upper limb, we need to think in terms of the diffusion compartment, instead of focusing on pure needle-to-nerve distance.

Simon Lévesque MD
Nicolas Dion MD
Marie-Christine Desgagné MD
CHA Hôpital Enfant-Jésus, Québec City, Canada
E-mail: simlevesque@hotmail.com
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4 *Dingemans E, Williams SR, Arcand G, et al.* Neurostimulation in ultrasound-guided infraclavicular block: a prospective randomized trial. Anesth Analg 2007; 104: 1275–80.

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Reply:

We sincerely thank Dr. Lévesque and his colleagues for their excellent comments. In particular, we were quite intrigued to learn about the "three factors" to predict the

outcome of the infraclavicular brachial plexus blocks. At our institution, instead of making use of "three factors," we utilize one common endpoint to ensure the success of the blocks. It is the deposition of the local anesthetic (LA) around the brachial plexus cords. We believe that having the LA "bathe" the nerves is important in predicting the success of the blocks. The U-shaped distribution of LA and the anterior displacement of the artery are often seen as a result of the correct deposition of the LA. Fascial click, however, is sometimes felt twice during the blocks. One click is always felt as the needle pierces the neurovascular sheath, and the second click is sometimes felt as it passes through the septum.

Dr. Lévesque writes, "… we need to think in terms of the diffusion compartment, instead of focusing on pure needle-to-nerve distance". We, too, feel that observing the appropriate LA spread is far more important than having the block needle near the nerve at the beginning of the block. Sinha et al.[1] have previously reported that the presence of nerve stimulation had no impact on the success of the resulting blocks. Not only does the ultrasound technology allow us to locate the target structures, it also allows us to direct the needle and to observe the real-time LA spread. Ultimately, this know-how should lead to safer and more effective blocks.

Maki Morimoto MD
Jovan Popovic MD
Jung T. Kim MD
Harald Kiamzon MD
Andrew D. Rosenberg MD
NYU Medical Center, New York, USA
E-mail: maki.morimoto@med.nyu.edu

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Hypertrophic cardiomyopathy – a matter of genes

To the Editor:

We write to comment on the *Images in Anesthesia* feature by Fayad,[1] which describes a patient with undiagnosed hypertrophic obstructive cardiomyopathy, who developed hypotension during anesthesia. Because the patient had a long-standing history of hypertension, we suggest that the cardiac condition in this patient may not have been secondary to hypertrophic (obstructive) cardiomyopathy.

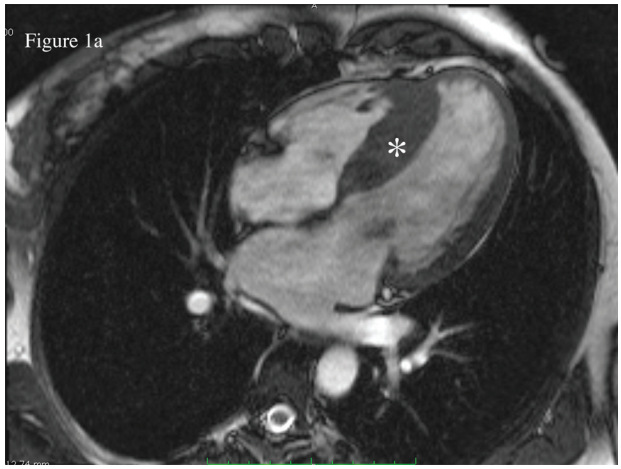

FIGURE A Diastolic image of a steady state free precession (SSFP) sequence in the four-chamber view of the above-mentioned patient. The septal hypertrophy can be clearly seen (asterisk).

Hypertrophic cardiomyopathy (HCM) is a genetic disorder. Diagnosis usually requires the exclusion of other causes of left ventricular hypertrophy — in particular, arterial hypertension, and aortic stenosis.[2] To date, some 400 mutations, in at least 11 genes, have been shown to cause HCM, with an autosomal dominant pattern of inheritance. The phenotype of HCM is highly variable, ranging from asymptomatic cases, to patients who are severely limited by dyspnea and angina. Cardiac hypertrophy in affected individuals can be restricted to the interventricular septum, or to virtually any part of the ventricle. The disease can also present as concentric hypertrophy. Accordingly, left ventricular outflow tract (LVOT) obstruction is not a mandatory feature of HCM. Many patients with HCM do not have LVOT obstruction at rest, but develop LVOT gradients during exercise (latent hypertrophy). Alternatively, dynamic LVOT obstruction may be restricted in patients with HCM to conditions involving a hyperdynamic circulation (e.g., secondary to anemia). Dynamic LVOT obstruction can also be precipitated by drugs that reduce cardiac afterload, thus increasing the gradient between the left ventricle and the aorta. However, a diagnosis of LVOT obstruction is not pathognomonic for HCM. Patients with long-standing and/or pronounced arterial hypertension may also present with LVOT obstruction. Since LVOT obstruction is a dynamic phenomenon, even patients with structurally normal hearts may develop LVOT obstruction.[3]