



Is Compartment Syndrome Risk a Contraindication to Regional Analgesia?

Stephen D. Lucas^{1*}

Acute compartment syndrome (ACS) occurs when pressure within a closed space causes ischemia to the tissues within that space [1]. It is relatively rare, with an incidence of 3.1 per 100,000, but it can be devastating if not promptly diagnosed and treated [2]. The diagnosis of compartment syndrome is a clinical one, based primarily on the presence of pain out of proportion to the clinical situation, often worsened by muscle stretch. Based on a few case reports and case series, some, particularly in the orthopedic community, have suggested that regional anesthesia should be avoided in patients at risk for ACS [3]. This is based on a concern that the analgesia from regional anesthesia may mask the hallmark sign of ACS, pain. Other case reports, particularly two excellent recent ones [4,5], suggest that regional anesthetic techniques will not mask the pain of ACS. As was pointed out eloquently in an editorial by Clark, we will likely never have more definitive evidence than case reports, which is considered level C evidence [6]. These authors have been cautious and rightfully stated that evidence-based guidelines to proceed or refrain from regional anesthesia in these patients will not be forthcoming as these types of guidelines are generally not propagated based on Level 3 evidence. Ethically, we must provide some form of analgesia for these patients with painful injuries. A review of the existing literature suggests that carefully administered and monitored regional anesthesia can and should be offered to these patients.

A brief review of the evidence to avoid regional anesthesia in patients with a risk of developing ACS is enlightening. Hyder et al. attributed the delayed diagnosis of a lower leg compartment syndrome after tibial intramedullary (IM) nailing to a post-operative 3 in 1 block [7]. Obviously the innervation of the components of the lower leg compartments should not be anesthetized by this block as it does not affect the sciatic nerve. Noorpuri et al. reported a patient who had an ankle block for a forefoot arthroplasty and subsequently developed ACS [8]. This patient initially had no pain but developed severe pain unrelieved by PO narcotics for 12 hours. Again, there is little evidence that the regional anesthetic obscured the diagnosis. In a similar vein, Uzel and Steinman reported a case of thigh ACS in a patient who underwent IM nailing of a femur fracture [9]. This patient had a single shot femoral block with Ropivacaine 0.75%, but still reported pain 4 hours postoperatively. Mar et al. excellent review of the effect of analgesia on ACS diagnosis summarized 35 cases in which epidural anesthesia was involved [10]. They report that classic signs and symptoms of ACS were present in 32 of these patients. In

the three patients for which a delay in diagnosis did take place, all had prolonged, dense motor and sensory epidural blocks.

Other analgesic modalities, such as Patient Controlled Analgesia with opiates (PCA) have been implicated for masking the diagnosis of ACS [11,12]. Interestingly, these case reports involved high doses of opiates. These authors suggest intermittent IM narcotics, mainly with the supposition that it would increase nursing interaction with the patients. A common flaw of many of these case reports is that they include scant detail regarding the type and frequency of monitoring for ACS symptoms.

The more recent case reports by Cometa and Walker are very useful because they carefully describe the monitoring methods and the timeline of developing symptoms and subsequent diagnosis. The Cometa report in particular, as it involves an inpatient being cared for by a dedicated regional anesthesiology and perioperative pain medicine service, gives us an hour-by-hour timeline of events. These reports make it quite clear that a low dose infusion (Ropivacaine 0.2% at typical volumes) delivered via a continuous perineural catheter does not delay the diagnosis of ACS. These reports also illustrate the value of careful monitoring and patient education to promptly diagnose ACS. In the Cometa report, the initial diagnosis of ACS was suspected by the acute pain physician who had the patient evaluated by his orthopedic colleagues. In the Walker case, the patient sought prompt attention at the ED because of thorough patient education.

A review of the available evidence suggests that regional anesthesia should be offered to patients at risk for ACS. Continuous techniques, particularly continuous peripheral nerve blocks appear to be safe. Dense anesthesia, particularly via the epidural route, should likely be avoided. Probably most importantly, all involved should agree on the plan and closely monitor the patient for signs and symptoms of ACS. This close attention needs to include appropriate education of the people most likely to see the patients frequently, including nurses and, in the case of teaching hospitals, house staff. In the case of outpatients, good patient education and quick, reliable lines of communication to treating physicians are imperative. Hopefully, we may someday have more rigorous evidence in the form of retrospective analysis of large registries. Until then, we need to offer these patients good analgesia while keeping them safe from the potentially devastating consequences of ACS.

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*Corresponding author: Stephen D. Lucas, Division of Regional Anesthesiology and Perioperative Pain Medicine, Department of Anesthesiology, University of Florida College of Medicine, Florida, USA, E-mail: slucas@anest.ufl.edu

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Author Affiliation

[Top](#)

¹Division of Regional Anesthesiology and Perioperative Pain Medicine, Department of Anesthesiology, University of Florida College of Medicine, Florida, USA

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