Pain Medicine

Pain Medicine 2011; 12: 823–828
Wiley Periodicals, Inc.

ACUTE PAIN SECTION

Case Reports

Did Continuous Femoral and Sciatic Nerve Block Obscure the Diagnosis or Delay the Treatment of Acute Lower Leg Compartment Syndrome? A Case Report

M. Anthony Cometa, MD,* Andrea T. Esch, DO,* and André P. Boezaart, MD, PhD*†

*Department of Anesthesiology, Division of Acute Pain Medicine and

†Department of Orthopedic Surgery and Rehabilitation, University of Florida College of Medicine, Gainesville, Florida, USA

Reprint requests to: M. Anthony Cometa, MD, Department of Anesthesiology, University of Florida College of Medicine, 1600 SW Archer Road, Gainesville, FL 32610, USA. Tel: 352-265-0077; Fax: 352-265-8013; E-mail: mcometa@anest.ufl.edu.

Presentations: None.

Financial Support: None.

Abstract

Objective. We report a case of acute lower extremity compartment syndrome that was diagnosed despite continuous regional analgesia with 0.2% ropivacaine via femoral and sciatic nerve catheters.

Setting. Academic tertiary care center.

Study Design. Report of a clinical case.

Summary. A 15-year-old boy with adolescent Blount's disease underwent elective distal femur and proximal tibia osteotomy with external fixation and stabilization of his right leg. The patient's anesthetic and analgesic management included general anesthesia with adjunctive regional anesthesia *via* continuous femoral and sciatic nerve blocks with 0.2% ropivacaine—each block initially infused at 10 mL per hour. On the first postoperative day, the patient reported no pain (0/10 on the visual analog scale, where 0 is no pain and 10 is the worst pain imaginable). However, on the second postoperative day, the patient reported severe pain despite effective blocks and oral opioid analgesic modalities. Compartment syndrome was diagnosed and treated with decompressive fasciotomy; tissue loss resulted.

Conclusion. Despite concerns of masking pain that may be secondary to compartment syndrome, this case demonstrates that compartment syndrome can be diagnosed in the presence of effective regional anesthesia. Careful clinical evaluation coupled with a high index of suspicion is essential in the timely diagnosis and effective treatment of compartment syndrome.

Key Words. Continuous Nerve Block; Femoral Nerve Block; Sciatic Nerve Block; Compartment Syndrome; Complications

Introduction

Compartment syndrome occurs when pressure within a fixed anatomical closed space rises to levels where the perfusion gradient across tissue capillary beds are decreased resulting in nerve and tissue ischemia. If untreated or treated too late, this may lead to irreversible neuromuscular ischemic damage [1]. Compartment syndrome is a clinical diagnosis characterized by pain that is out of proportion to what is expected given the clinical situation and that is worsened by passive movement of muscles within the affected compartment. Compartment syndrome is a surgical emergency with definitive treatment requiring immediate fasciotomy to relieve the pressure within the affected compartment. Delay in the diagnosis and treatment can be detrimental, as irreversible tissue damage can occur as early as 4 hours after the onset of symptoms [2].

Regional anesthesia (RA) is regularly used to provide surgical anesthesia or as an adjunct to general anesthesia for the treatment of postoperative pain and to facilitate early mobilization [3]. RA and its efficacy for postoperative analgesia have raised concerns for the possible delay in

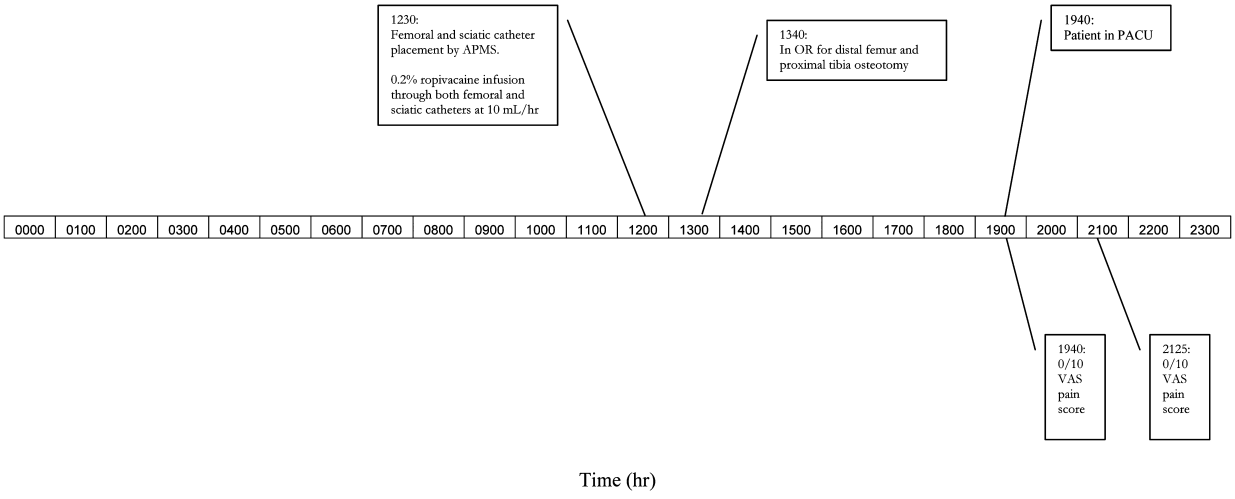

Figure 1 Course of events on day of surgery. APMS = acute pain medicine service; OR = operating room; PACU = postanesthesia care unit; VAS = visual analog scale.

diagnosis and treatment of compartment syndrome in susceptible individuals [4], and two questions need to be asked:

1. Does RA mask the chief symptom (severe pain out of proportion to what is expected)?
2. Does RA obscure the clinical picture that may result in the delay in the diagnosis and treatment of acute compartment syndrome?

This case report attempts to highlight these issues and furthermore attempts to provide some answers to these questions. By its very nature, compartment syndrome and its diagnosis and treatment would be ethically impossible to study prospectively because it cannot be reliably and safely reproduced in human subjects. We therefore must rely on anecdotal case reports to formulate and challenge conventional thinking.

Case Report

We report a case involving a 15-year-old boy with Blount's disease (localized varus deformity of the leg), weighing 150 kg, who underwent elective distal femur and proximal tibia osteotomy [5,6]. Before the operation, continuous femoral and sciatic nerve blocks were placed to facilitate continuous nerve block for perioperative analgesia using techniques described previously [7], making use of StimuCath™ catheters (Arrow International, Reading, PA). Blocks were placed preoperatively under mild sedation with propofol, and the patient received 30 mL of 0.5% ropivacaine through each of the catheters. He demonstrated complete and appropriate sensory and motor blockade. The operation proceeded without incident under combined general and regional anesthesia, and in the immediate postoperative period, the patient reported no pain (0/10 on a visual analog scale [VAS], where a number of 0 represents no pain and 10 represents the worst pain imaginable). Both nerve blocks were maintained with infusions of 0.2% ropivacaine at a rate of 10 mL/h (Figure 1).

On the first postoperative day, the patient was evaluated by the acute pain medicine service (APMS), and pain throughout the evening was reported to have ranged from 0–3/10 on the VAS, with appropriate motor and sensory blockade along the femoral and sciatic nerve distributions. The infusion of 0.2% ropivacaine through the catheter to the femoral nerve was decreased to 5 mL/h because of weak quadriceps muscle function, while the infusion through the catheter to the sciatic nerve remained unchanged at 10 mL/h. The patient's hamstring function remained strong, and the pain was appropriately controlled in both femoral and sciatic nerve distributions (Figure 2).

On the second postoperative day, both of the continuous block infusions were turned off at 06:00 in anticipation of full return of sensation and subsequent catheter removal if pain could be controlled with oral or intravenous analgesics only. The nurse caring for the patient documented that the patient reported no pain (VAS = 0/10) for 2.5 hours following discontinuation of the infusions. Three hours after the blocks had been turned off (09:00), the primary orthopedic physicians evaluated the patient and also documented no pain. During APMS rounds, 20 minutes later (3 hours and 20 minutes after discontinuation of the block infusions at 06:00), the patient reported severe excruciating antero-lateral pain (VAS = 10/10) in the area where the external fixation device was placed. On physical examination, the sensory and motor nervous systems were fully intact. Given his complaint of severe pain, the APMS physicians judged that the regional anesthesia therapy was discontinued prematurely and administered a bolus of 10 mL of 0.2% ropivacaine through

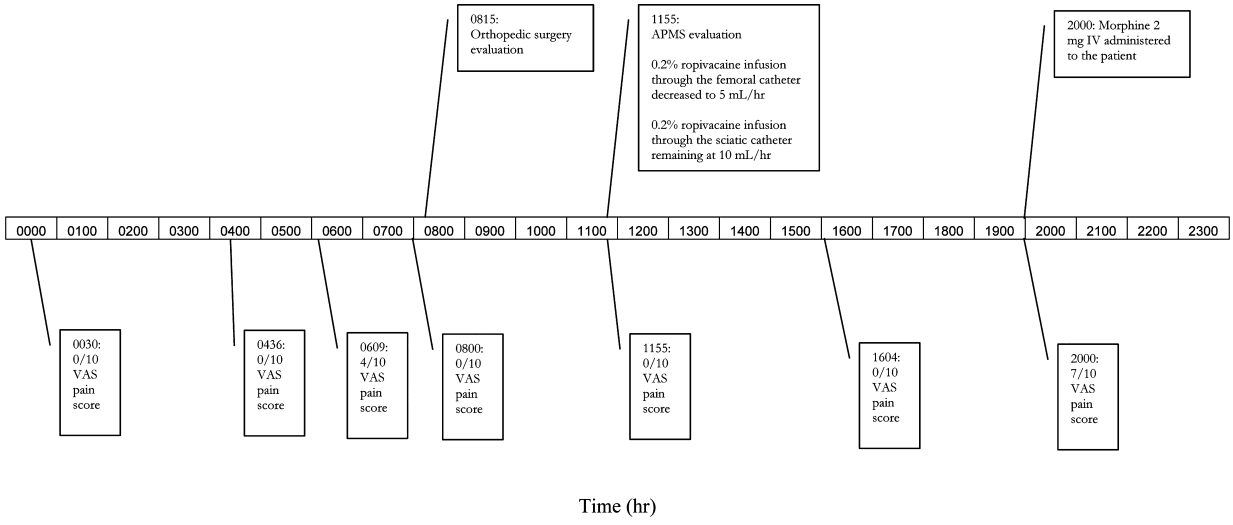

Figure 2 Course of events on first postoperative day. APMS = acute pain medicine service; IV = intravenous; VAS = visual analog scale.

each of the femoral and sciatic block catheters. This resulted in immediate relief of pain. The infusion of each block was again reinitiated with 0.2% ropivacaine at 5 mL/h.

Seven hours later, at 16:31, APMS was notified that the patient had moderately severe pain (VAS = 8/10) that was not relieved with the nerve blocks or intravenous opioids. On physical examination, sensation was intact, but the pain was hyperalgesic in nature. The patient was able to perform only limited plantarflexion and dorsiflexion. This limitation was thought to be secondary to impaired motor function caused by the sciatic nerve block or decreased patient effort due to pain. His quadriceps function was also weak, and his right dorsalis pedis and posterior tibial pulses were normal and equal to the left side. Based on these findings, a further bolus of 10 mL of 0.2% ropivacaine was administered through the femoral and sciatic catheters. Subsequent physical examination showed excruciating pain upon passive plantarflexion and dorsiflexion of the foot. Additionally, the tissue surrounding his right gastrocnemius muscle felt tense when compared with his left. Compartment syndrome was suspected, and the orthopedic surgeons were contacted. They evaluated the patient and documented severe pain (VAS = 10/10). Intra-compartmental pressure measurement within the anterior, lateral, superficial posterior, and deep posterior compartments of the right lower extremity 2 hours later were elevated, exceeding 30 mm Hg. These measurements were repeated an hour later, and similar results were obtained, and emergent decompressive fasciotomy was performed (Figure 3).

Upon intraoperative inspection, the musculature appeared "dusky," yet judged to be viable because it was contractile to electrical stimulation. Subsequently, during follow-up surgery 3 days later, the deep and superficial posterior compartments were judged healthy and viable. The anterior and lateral compartment muscles also appeared viable but were judged to be "not perfectly healthy." On the fifth post-fasciotomy day, the fasciotomy sites were closed and, on inspection, some anterior and lateral musculature of the leg was found to be nonviable and removed. The patient did not have any further local or systemic complications of this compartment syndrome. At the time of discharge, the patient's sensation over the dorsum and plantar surfaces of his right foot was intact. Active plantarflexion and dorsiflexion of his toes were normal, and plantarflexion at the ankle was normal, but the ability to dorsiflex or evert his affected foot was absent. No neurological injury could be demonstrated. The patient was discharged on the 12th post-fasciotomy day to a rehabilitation facility for directed physical therapy.

Discussion

RA by continuous nerve block techniques is becoming an increasingly popular and common practice as an adjunct to general anesthesia for orthopedic surgical procedures [3,8–10]. More recently, the benefits of postoperative analgesia provided by lower extremity regional anesthesia is becoming more popular in the pediatric population [11]. However, given the efficacy of regional anesthesia, and especially continuous nerve blocks for providing postoperative analgesia, there are concerns of masking the pain that may be the earliest or only warning symptom of impending compartment syndrome. Corrective varus tibial osteotomy is well known to carry a high risk of compartment syndrome, and the use of RA for this surgery is controversial [5,6].

The maximum time to intervention that would prevent ischemic necrosis to an affected extremity is estimated to be 4–8 hours [2,12], and delays in diagnosis and

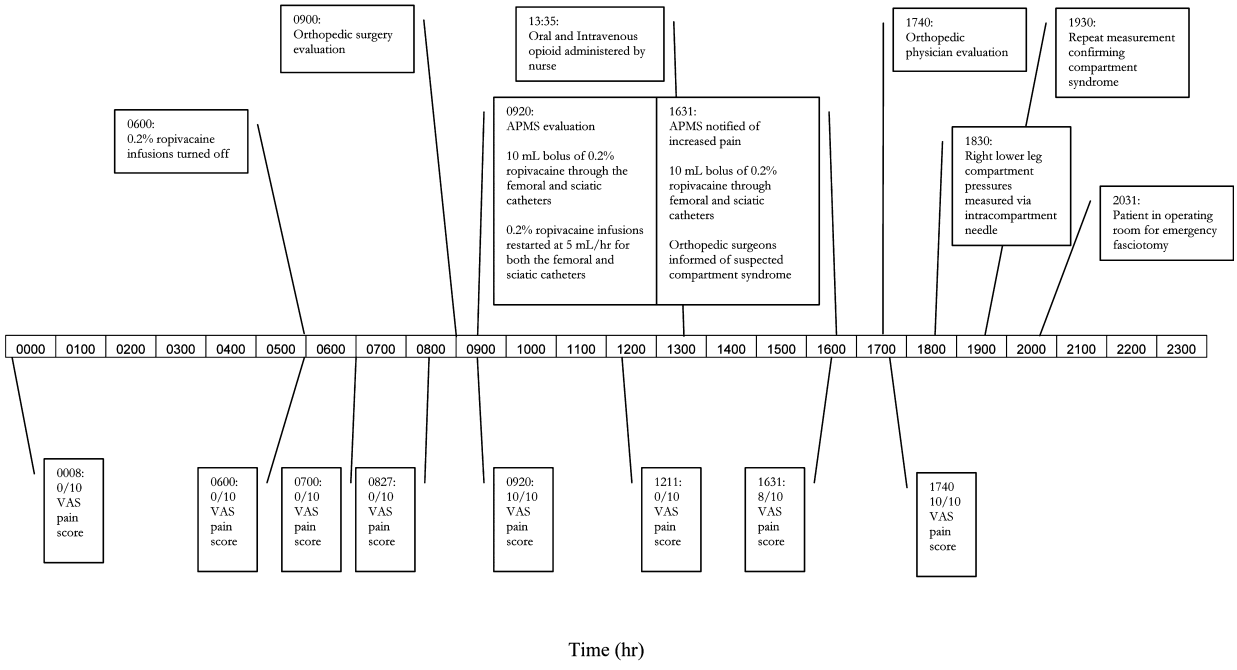

Figure 3 Course of events on second postoperative day. APMS = acute pain medicine service; VAS = visual analog scale.

intervention with fasciotomy have resulted in poor outcomes, which include devastating outcomes such as amputation, multiorgan failure, and death [13]. A recent review of the literature, however, comparing multiple modes of analgesia, which included intravenous patient-controlled analgesia with opioids and RA, showed no convincing evidence that a delay of compartment syndrome diagnosis can be attributed to such analgesic modalities [14].

The anesthetic management of this patient included combined general anesthesia and RA techniques. Given the patient's age and physical attributes that were concerning for possible respiratory obstruction, a full analgesic block using 0.5% ropivacaine to minimize the use of intraoperative systemic parenteral opioids was chosen. The patient did not require any parenteral opioid medication during his intraoperative and immediate PACU course on the initial day of the surgery. The patient started to experience severe pain that required further APMS intervention on the morning of the second postoperative day. That treatment provided adequate analgesia, but during the afternoon APMS evaluation on that day, the patient reported pain out of proportion to what one would expect with effective nerve blocks and both intravenous and oral opioids in place. Even the 10 mL boluses of 0.2% ropivacaine at that time through the nerve block catheters did not relieve the pain. The pain remained out of proportion to expectation, and physical examination showed excruciating crescendo pain upon passive stretching of the muscles. The presentation of crescendo pain with associated increased lower extremity edema should provoke a high index of suspicion for compartment syndrome. These findings seriously challenge the notion that regional anesthesia techniques might mask the telltale warning of pain caused by muscle ischemia of compartment syndrome.

The pathophysiology of ischemic muscular pain is complex and is mediated by chemical and inflammatory markers acting on nociceptors [15–19]. Myelinated Aδ and unmyelinated C fibers are sensitive to an array of inflammatory and hormonal markers that result in nociceptor activation in injured tissue [15–17]. In ischemic tissue, however, it is postulated that bradykinin, serotonin, acetylcholine, adenosine, potassium ions, and hydrogen ions are some of the substances responsible for ischemic pain [16,17]. Tissue acidosis evidently initiates the pain pathway as increasing levels of hydrogen ion concentration may act on skeletal muscle nociceptors resulting in pain impulse transmission [18]. The hormonal markers of inflammation and injury are thought to undergo tachyphylaxis after nociceptor activation [15]; however, hydrogen ion excitation, in particular, produces non-adapting activation of nociceptors [15,19]. We therefore postulate that this patient's affected compartments were ischemic such that nociceptor activation *via* hydrogen ion excitation was sufficient to render continuous regional anesthesia with 0.2% ropivacaine ineffective to provide analgesia.

We further propose that the analgesia achieved from our intervention with 0.2% ropivacaine on the morning of the second postoperative day sufficiently addressed the pain

associated with the operative injury and inflammation to the surgical site, yet it was not sufficient to treat the ischemic pain. The exact time that the compartment syndrome actually started is impossible to determine for this particular patient, but we estimate that the ischemia associated with compartment syndrome started sometime between the morning and afternoon APMS evaluation—between 09:20 and 16:30. The patient underwent a decompressive fasciotomy of the superficial and deep posterior compartments and the anterior and lateral compartments of the leg within 5 hours of the diagnosis of compartment syndrome. In spite of this, the patient suffered tissue loss. The question that remains to be answered is whether the continued nerve blocks obscured the clinical presentation enough to delay surgery. Was the onset of the ischemic process in fact when we think it was?

Given the fact that the patient had no pain upon discontinuation of the continuous blocks but subsequently developed pain that was so severe that it was unresponsive to additional interventions with local anesthetic, we believe that our assumption of the approximate onset of the ischemic process was indeed correct. We do not believe that the blocks in any way obscured the diagnosis. In fact, our model of having APMS mandatory twice daily rounds on patients may have contributed to relatively early diagnosis.

The diagnosis of compartment syndrome is ultimately clinical—taking into consideration the classic signs and symptoms of pressure, pain, paresthesia, pallor, pulselessness, and paralysis. However, these signs and symptoms of compartment syndrome have been known to be unreliable [14]. The way that this patient presented included pressure, pain, and paresthesia. He continued to have capillary refill and strong bilateral lower extremity dorsalis pedis pulses, the loss of which is uncommon and a late clinical finding [20]. Adjunctive monitoring *via* compartment measurement monitoring has been reported in the literature [21,22]. Needle manometry was used to measure the intra-compartmental pressures of the patient's affected extremity. Although the patient's intracompartmental pressures were elevated, it should be noted that this technique has been shown to be difficult to interpret and can have measurement inaccuracies [23]. Ultimately, this information should be used in the context of the entire clinical presentation. In patients at risk for compartment syndrome, careful monitoring with ongoing patient assessment, a high index of suspicion, and close communication among all individuals caring for the patient are essential [12].

Conclusion

The diagnosis of compartment syndrome can be difficult, especially in the pediatric population because children are often construed as being "immature" or not able to "handle" or communicate pain. Because compartment syndrome constitutes a surgical emergency, a high index of clinical suspicion is absolutely essential to make the diagnosis early. Further evaluation is critical when symptoms of ischemic pain are present. Time is of the essence when the diagnosis of compartment syndrome is made. The two pillars upon which the high clinical index of suspicion is based are pain out of proportion to expectations and crescendo pain upon passive stretching of the muscles in a particular compartment. Although there is no strong evidence in the literature that pain secondary to compartment syndrome can be masked by regional anesthesia, this current case report strongly suggests that ischemic pain due to compartment syndrome can be recognized early despite a known well-functioning regional anesthetic technique. Confirmatory tests in the form of repeat pressure measurements should probably be negated if a high index of suspicion exists, and pain "breaking through" an otherwise effective nerve block should immediately alert all members of the patient's health care team to initiate immediate and emergent treatment.

Conflict of Interest

Dr. A P Boezaart invented the stimulating catheter and receives royalty payment from Arrow International for the sales of StimuCath.

References

1 Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. J Am Acad Orthop Surg 2005;13:436–44.

2 Davis ET, Harris A, Keene D, Porter K, Manji M. The use of regional anaesthesia in patient at risk of acute compartment syndrome. Int J Care Injured 2006;37: 128–33.

3 Ayers J, Enneking FK. Continuous lower extremity techniques. Tech Reg Anesth Pain Manag 1999;3:47–57.

4 Hyder N, Kessler S, Jennings AG, De Boer PG. Compartment syndrome in tibial shaft fracture missed because of a local nerve block. J Bone Joint Surg Br 1996;78-B:499–500.

5 Sabharwal S. Blount disease. J Bone Joint Surg Am 2009;91:1758–76.

6 Rab GT. Oblique tibial osteotomy revisited. J Child Orthop 2010;4:169–72.

7 Boezaart AP. Atlas of Peripheral Nerve Block and Anatomy for Orthopaedic Anesthesia, 1st edition. Philadelphia: Elsevier; 2008:143–52, 186–8.

8 Grant SA, Nielson KC, Greengrass RA, Steele SM, Klein SM. Continuous peripheral nerve block for ambulatory surgery. Reg Anesth Pain Med 2001;26: 209–14.

9 Ganapathy S. Continuous nerve blocks for orthopedic injuries. Tech Reg Anesth Pain Manag 2002;6:27–32.

10 Yufa M, Kurc P, Vloka JD, Hadzic A. Lower extremity blocks for analgesia. Tech Reg Anesth Pain Manag 2002;6:60–5.

11 Simion C, Suresh S. Lower extremity nerve blocks in children. Tech Reg Anesth Pain Manag 2007;11: 222–8.

12 Kashuk JL, Moore EE, Pinski S, et al. Lower extremity compartment syndrome in the acute care surgery paradigm: Safety lessons learned. Patient Saf Surg 2009;3:11.

13 Finkelstein JA, Hunter GA, Hu RW. Lower limb compartment syndrome: Course after delayed fasciotomy. J Trauma 1996;40:342–4.

14 Mar GJ, Barrington MJ, McGuirk BR. Acute compartment syndrome of the lower limb and the effect of post operative analgesia on diagnosis. Br J Anaesth 2009;1:3–11.

15 Raja SN, Meyer RA, Ringkamp M, Campbell JN. Peripheral neural mechanisms of nociception. In: Wall PD, Melzack R, eds. Textbook of Pain. Churchill Livingston: Edinburgh; 1999:31–4.

16 Newham DJ, Mills KR. Muscles, tendons, and ligaments. In: Wall PD, Melzack R, eds. Textbook of Pain. Churchill Livingston: Edinburgh; 1999:518.

17 Mense S, Simons DG, Russell IJ. Muscle Pain: Understanding Its Nature, Diagnosis, and Treatment. Philadelphia: Lippincott Williams and Wilkins; 2001:53.

18 Issberner U, Reeh PW, Steen KH. Pain due to tissue acidosis: A mechanism for inflammatory and ischemic myalgia? Neurosci Lett 1996;208:191–4.

19 Steen KH, Issberner U, Reeh PW. Pain due to experimental acidosis in human skin: Evidence for non-adapting nociceptor excitation. Neurosci Lett 1995;199:29–32.

20 Tiwari A, Haq AI, Myint F, Hamilton G. Acute compartment syndromes. Br J Surg 2002;89:397–412.

21 Kosir R, Moore FA, Selby JH, et al. Acute lower extremity compartment syndrome (ALECS) screening protocol in critically ill trauma patients. J Trauma 2007;63:268–75.

22 McQueen MM, Court-Brown CM. Compartment monitoring in tibial fractures. The pressure threshold for decompression. J Bone Joint Surg Br 1996;78:99–104.

23 Elliott KGB, Johnstone AJ. Diagnosing acute compartment syndrome. J Bone Joint Surg Br 2003; 85-B:625–32.