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REVIEW ARTICLES

Acute compartment syndrome of the lower limb and the effect of postoperative analgesia on diagnosis†

G. J. Mar*, M. J. Barrington and B. R. McGuirk

Department of Anaesthesia, St Vincent's Hospital, Melbourne, PO Box 2900, Fitzroy, 3065 VIC, Australia

**Corresponding author. E-mail: gjpmar@yahoo.com.au*

Acute compartment syndrome can cause significant disability if not treated early, but the diagnosis is challenging. This systematic review examines whether modern acute pain management techniques contribute to delayed diagnosis. A total of 28 case reports and case series were identified which referred to the influence of analgesic technique on the diagnosis of compartment syndrome, of which 23 discussed epidural analgesia. In 32 of 35 patients, classic signs and symptoms of compartment syndrome were present in the presence of epidural analgesia, including 18 patients with documented breakthrough pain. There were no randomized controlled trials or outcome-based comparative trials available to include in the review. Pain is often described as the cardinal symptom of compartment syndrome, but many authors consider it unreliable. Physical examination is also unreliable for diagnosis. There is no convincing evidence that patient-controlled analgesia opioids or regional analgesia delay the diagnosis of compartment syndrome provided patients are adequately monitored. Regardless of the type of analgesia used, a high index of clinical suspicion, ongoing assessment of patients, and compartment pressure measurement are essential for early diagnosis.

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Compartment syndrome is a condition in which increased pressure within a closed compartment compromises the circulation and function of the tissues within that space.[65] It occurs most commonly in an osseofascial compartment of the leg or forearm, but it may occur in the upper arm, thigh, foot, buttock, hand, and abdomen. The most common cause of compartment syndrome is trauma, usually after a fracture.[11] In an audit, 4.3% of all patients with tibial shaft fractures, 3.1% of diaphyseal fractures of the forearm, and 0.25% fractures of the distal radius developed acute compartment syndrome.[36] It is seen more commonly in patients <35 yr of age[34] and in male patients.[36 42] Compartment syndrome also occurs in the context of reperfusion, ischaemia, burns, and poor positioning for prolonged surgical procedures (particularly lithotomy position)[55] and in drug-affected individuals (Table 1).[28] The incidence of compartment syndrome is up to 20% in acutely ischaemic limbs that have been revascularized.[7] Acute compartment syndrome requires prompt diagnosis and management. Delays in treatment can result in significant disability including neurological deficit, muscle necrosis, amputation, and death. The diagnosis requires a high index of suspicion and is challenging. Pain is thought to be a cardinal feature of compartment syndrome and it has been claimed that analgesia may delay its diagnosis resulting in a poor patient outcome.

The primary objective of this review was to undertake a systematic review of articles relating postoperative analgesia to a delay in diagnosis of compartment syndrome. In addition, a literature review was performed to detail the pathophysiology, clinical presentation, and role of compartment pressure manometry. The focus was on compartment syndrome of the lower limbs after trauma and surgery.

Clinical presentation, diagnosis, and monitoring

The underlying pathophysiology of acute compartment syndrome is an ischaemia–reperfusion–ischaemia cycle. Ischaemia can be precipitated by remote perfusion failure

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Table 1 Common aetiology of compartment syndrome

Orthopaedic	Fractures and fracture surgery
Vascular	Arterial and venous injuries
	Reperfusion injury
	Haemorrhage
Soft tissue	Crush injury
	Burns
	Prolonged limb compression
Iatrogenic	Arterial/venous puncture in anticoagulated patients
	Casts and circular dressings
	Pulsatile irrigation
	Surgical positioning—especially prolonged lithotomy position
	Pneumatic antishock garment
Other	Snakebite
	Muscle overuse

(vascular obstruction or trauma, systemic hypotension) or by increased resistance to flow within the compartment itself.[31] The ischaemia results in tissue membrane damage and leakage of fluid through capillary and muscle membranes. With arterial reperfusion, the damaged membrane continues to leak, increasing oedema formation and the pressure in the closed compartment. The clinical signs and symptoms of acute compartment syndrome are known to be unreliable.[1 11 27 28 35 66] The symptoms of compartment syndrome are severe pain and paraesthesia. This is difficult to assess at the extremes of age or in those with central nervous system (CNS) compromise.[28] CNS compromise can be a particular issue after general anaesthesia and in sedated patients in an intensive care setting. Difficulties with sedation or pain management may be the only clinical indicator of compartment syndrome in this group.[16] However, pain may be an unreliable symptom as it is subjective and variable. It may be absent in established acute compartment syndrome associated with nerve injury, or minimal in deep posterior compartment syndrome.[35] The signs of compartment syndrome are tense, swollen compartments, pain on passive stretching of the muscle, and sensory loss. Pulselessness is not common and generally implies a late stage.[62] In a review examining the clinical signs and symptoms of compartment syndrome, the false-positive rate was shown to be high in relation to the true-positive rate.[66] That is, clinical findings of compartment syndrome were more likely to be present in patients who do not have compartment syndrome than in those who do. A lack of clinical signs and symptoms was more helpful in excluding the diagnosis than was the presence of findings for confirming compartment syndrome. In a prospective study using a predetermined screening protocol for lower extremity compartment syndrome in critically ill trauma patients, physical examination was considered inaccurate for diagnosis. On completion of the study, it was decided not to use physical examination as part of the screening protocol.[27]

Compartment syndrome must be treated urgently as the extent of injury is mainly determined by the duration of ischaemia and the pressure in the osseofascial compartment. In a canine model of compartment syndrome, significant muscle necrosis occurs after 8 h with a compartmental pressure of 30 mm Hg.[20] In a clinical setting, it is not possible to pinpoint the precise time compartment syndrome develops. The incidence of complications is related to the time from diagnosis to fasciotomy.[37 42] Catastrophic outcomes were inevitable if fasciotomies were delayed for more than 12 h, whereas a full recovery was achieved if decompression was performed within 6 h of making a diagnosis.[11] In addition to poor clinical outcome, a delayed diagnosis has medicolegal ramifications. In a review of closed claims in a state in the USA spanning 23 yr, out of 1515 cases involving orthopaedic surgeons, 19 claims related to compartment syndrome in 16 patients. Nine cases were resolved in favour of the patient and seven in favour of the surgeon with poor surgeon–patient communication being a reason for compensation in six instances. Defence was always successful when a fasciotomy was performed within 8 h of the first presenting symptom.[5] Patients at risk of compartment syndrome are often poorly assessed. In a retrospective study of preoperative medical records of 30 consecutive patients who underwent fasciotomies for compartment syndrome, documentation was inadequate for 21 (70%) patients.[9]

Compartment pressure monitoring

Compartmental pressure measurement is recommended in high-risk patients as an adjunct to clinical diagnosis[27 35] except where the diagnosis is obvious.[28] Normal pressure in the muscle compartment is below 10–12 mm Hg.[65] The compartmental perfusion pressure is the difference between the diastolic arterial pressure and the compartmental pressure. The diagnostic pressure difference in one study was 21 mm Hg.[27] Absolute compartment pressures of 45[32] and 30 mm Hg[40] have been suggested as thresholds for compartment syndrome. Needle manometers are commonly utilized for compartment pressure measurement. They are cheap and easy to use, but have been shown to have inaccuracies and cannot be used continuously.[11] Catheter techniques are effective for continuous compartmental pressure measurement but require accurate placement of the external transducer, have more complex

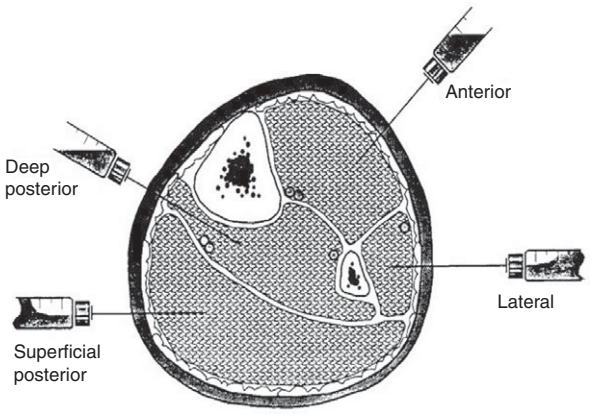

Fig 1 Osseofascial compartments of the calf.

equipment, and fragments of tissue or clots can obstruct the tip affecting accuracy. In Figure 1, the position of the various osseofascial compartments of the calf and the approach when inserting a needle manometer are demonstrated. All compartments in a limb suspected of having compartment syndrome should be measured.[28] The compartment with the highest initial pressure reading should be used for continuous pressure measurement.[11] It should be noted that neuromuscular damage is caused by ischaemia rather than elevated pressure alone.

Other monitors and investigations

Near-infrared spectroscopy (NIRS) measures tissue oxygenation and shows promise in monitoring for compartmental ischaemia. It has an advantage over needle and catheter techniques in that it measures tissue hypoxia directly using a principle similar to pulse oximetry.[14] Muscle oxyhaemoglobin (St_{O_2}) levels measured by NIRS strongly reflect compartment pressure, perfusion pressure, and loss of myoneural function. St_{O_2} was a more consistent predictor of neuromuscular dysfunction than compartment perfusion pressure.[13] It is non-invasive and can be used continuously, thus allowing duration of ischaemia to be measured. Unfortunately, the equipment is expensive and only measures to a limited depth, not reaching the deep posterior compartment of the calf. Another technique under evaluation is pulsed phase-locked loop ultrasound, which can analyse fascial displacement waveforms which correspond to arterial pulsations and change with increased compartmental pressure.[67] MRI can show the tissue changes in established compartment syndrome but is not good for diagnosing an evolving compartment syndrome.[28] Its use is limited by the time taken to perform the scan, potentially delaying management. Serum creatine phosphokinase (CK), which reflects muscle necrosis, has been used as an indicator of compartment syndrome. Significantly elevated CK levels may be useful in diagnosis where the clinical picture is not obvious and compartmental pressure measurement devices are not available.[47]

Monitoring may increase clinical awareness and aid diagnosis in the presence of equivocal clinical findings.[11] In a retrospective review of the use of compartment pressure monitoring in tibial diaphyseal fractures, the average delay from fracture manipulation to fasciotomy was 7 h in the monitored group and 24 h in the non-monitored group. The complication rate in those without monitoring (10/11) was higher compared with those in the monitored group (0/12).[35] Lack of compartment pressure monitoring and inadequate assessment and observation are the most common factors associated with a missed diagnosis.[63] Most surgeons accept that compartmental pressure measurement is important for the diagnosis of compartment syndrome,[28] and invasive arterial pressure transducers are widely available and can be attached to a saline-filled catheter placed in a compartment as a manometer.

Systematic review

A systematic review of articles relating postoperative analgesia to the diagnosis of acute compartment syndrome of the limb was conducted. The Pubmed, MEDLINE and EMBASE databases, Cochrane Library, and Google Scholar were searched from 1986 to present. We used a combination of search terms: compartment syndrome/epidural/extradural/analgesia/an(a)esthesia/an(a)esthetic/nerve block/regional/diagnosis/surgery. The search was restricted to articles published in the English language and letters of correspondence and surveys were excluded. The reference sections of relevant articles were hand searched for further publications. Reports were included if they related postoperative analgesia to the management and diagnosis of acute compartment syndrome. Two case reports described the same patient, so only the earlier report was included.[45 46] The reports were examined by all the authors.

A total of 28 case reports (n=20) and case series (n=8) were identified which referred to the influence of analgesic technique on the diagnosis of compartment syndrome. These techniques were patient-controlled analgesia (PCA; n=3), peripheral nerve block (PNB; n=2), and epidural analgesia (n=23), respectively. A large audit of epidural use in the UK and Ireland also analysed the diagnosis of acute compartment syndrome in children.[30] There were no randomized controlled trials or any other outcome-based comparative trials to include. All the evidence is Level 3.[19]

Analgesia and diagnosis of compartment syndrome

PCA was implicated in a delay in the diagnosis of acute compartment syndrome of the lower limb in three reports describing six male patients with tibial fractures. Two case reports detail patients with traumatic mid-shaft tibial fractures who had PCA morphine for analgesia after intramedullary nailing (90 mg over 24 h and 131 mg over 36 h, respectively).[21 46] The first patient complained of reduced sensation and foot movement prompting a diagnosis of compartment syndrome. The other patient had no pain observations from 6 h post-surgery and compartment syndrome was an incidental finding when the patient returned to theatre for scheduled wound closure 36 h after the original procedure. A case series of four patients who had compartment syndrome after tibial fractures where it was thought that PCA opioids delayed the diagnosis has been reported.[50] The patients in these reports had doses of 0.5–1 mg h^{-1} of PCA morphine. These are small doses,[64] suggesting that the patients did not have severe pain. The case reports provide limited detail on the clinical care provided to these patients in the lead up to the diagnosis of compartment syndrome. Other clinical features like paraesthesia and swelling were not mentioned. Two authors recommend avoiding PCA in favour of intermittent i.m. morphine injections.[21 46] The preference for this modality

was that it facilitates nursing contact with patients who can be avoided with PCA.

We did not find any case reports suggesting PNB delayed the diagnosis of upper limb compartment syndrome. In a literature review to establish whether a femoral nerve block may mask the signs and symptoms of thigh compartment syndrome, there was no evidence of an association between a femoral nerve block and a delayed or missed diagnosis.[25] However, a postoperative single shot 3-in-1 block using bupivacaine 0.5% may have led to a delayed diagnosis of calf compartment syndrome after intramedullary nailing of a tibial fracture.[23] However, femoral nerve block would not have completely removed the pain associated with a tibial injury as much of the pain will have been in the sciatic nerve distribution. In a report of foot compartment syndrome after a forefoot arthroplasty, the author suggests an ankle block delayed the diagnosis, yet pain was a significant clinical feature in the postoperative period.[44]

Many authors state that the presence of epidural analgesia did not contribute to a delay in the diagnosis of compartment syndrome.[2 10 15 22 24 30 38 58 65] There were four cases of compartment syndrome in a large multicentre prospective audit of the use of epidural analgesia in children in the UK and Ireland. Each case was diagnosed without delay, despite highly effective analgesia in two patients and less effective analgesia in the others.[30] Classic signs and symptoms were present when compartment syndrome developed in 32 of 35 patients discussed in the case reports (n=16) and series (n=7) relating to epidural analgesia. This includes 18 patients with documented breakthrough pain (Table 2). In contrast, there was a delay in diagnosis in three patients with dense bilateral motor blocks.[26 57 60] In one report,[60] the patient had 'complete anaesthesia' from the waist down in the postoperative period, implying a complete motor and sensory block, and in the others,[26 57] the patients had dense motor and sensory blocks for more than 18 h after operation. These patients did not have breakthrough pain due to their dense blocks, which is in contrast to the majority of case reports where pain was present (Table 2). Table 3 details the similarities and differences between the features of compartment syndrome and epidural analgesia.

Discussion

The importance of pain in the diagnosis of compartment syndrome is controversial. Virtually, all analgesic modalities have been linked to a delayed diagnosis of compartment syndrome; however, only Level 3 evidence is available. Reports commonly misattribute analgesia as the cause rather than an association with a delayed diagnosis. In addition, reports consistently reveal opportunities for improved clinical care including improvements in documentation and postoperative monitoring for compartment syndrome. Reference to the signs and symptoms of compartment syndrome should be in the immediate vicinity of any patient at risk. This could be on a designated orthopaedic observations chart alongside pain (including analgesic requirements), neurovascular, and vital signs. Risk assessment tools have been described which may aid monitoring patients at high risk of developing acute compartment syndrome.[27] Written protocols detailing appropriate care including the management of adverse events and triggers for medical review are important.

Pain may be an unreliable symptom as it is subjective and variable. However, in many of the case reports reviewed, pain was present but compartment syndrome not considered for a period of time.[3 10 15 18 24 43 57 59] Increasing demands for analgesia should trigger clinical review because these events have preceded neurovascular changes by 7.3 h.[1] PCA and continuous infusions of local anaesthetics may aid the diagnosis of compartment syndrome when patients analgesic requirements are observed appropriately. The view that analgesia should be withdrawn or an inferior mode of analgesia be used to facilitate diagnosis of compartment syndrome should be discouraged. Withholding analgesia to patients with acute abdominal pain for fear of masking pathology was once common clinical practice, but now it is considered safe and humane to administer narcotic analgesia to patients presenting with acute abdominal pain.[33] Analgesia is required after trauma and surgery on humane grounds alone and pain management is a core responsibility of our specialty.

There is a lack of appreciation by some authors of the importance of the pharmacology of epidural analgesia in the clinical presentation. For example, a report of four patients who developed gluteal compartment syndrome in the context of postoperative epidural analgesia does not describe the clinical examination or drugs used.[29] The fourth patient in this series was noted to have complete ankle paralysis 4 h after cessation of 43 h of continuous epidural analgesia. This suggests that either it was a new sign or the patient's motor function was not being monitored during the epidural infusion. Local anaesthetics and opioids are considered to have similar pharmacological activities by some authors. For example, a 16-yr-old male complained of discomfort and numbness in the leg after an osteotomy of the distal femur and proximal tibia which the author attributes to the pharmacological effects of an epidural fentanyl infusion.[49] Epidural opiates do not lead to numbness, paraesthesia, or motor block.[41] The symptoms may well have been the clinical features of compartment syndrome.

Dense local anaesthetic blocks can influence the assessment of pain and movement making the diagnosis of compartment syndrome difficult without invasive pressure monitoring. Dilute concentrations of local anaesthetics avoid motor and dense sensory blocks. For example, the optimal concentration of ropivacaine for epidural analgesia

Table 2 Summary of reports relating epidural analgesia to delayed diagnosis of compartment syndrome. NS, not specified; Epi, epidural; GA, general anaesthesia; CSE, combined spinal and epidural; B0625, bupivacaine 0.0625%; B1, bupivacaine 0.1%; B125, bupivacaine 0.125%; B2, bupivacaine 0.2%; B25, bupivacaine 0.25%; B5, bupivacaine 0.5%; L2, lidocaine 2%; M2, mepivacaine 2%; PCEA, patient-controlled epidural analgesia; TKJR, total knee joint replacement; THJR, total hip joint replacement; CS, compartment syndrome; Postop analgesia, indicates analgesic type and duration of use (where specified). *Time to fasciotomy from surgery or development of symptoms are estimated from case report details where possible

Report	Patient age/gender	Surgery	Anaesthetic	Postop analgesia	Drug	Significant other issues	Signs/symptoms	Classic CS symptoms despite Epi	Time to fasciotomy (h)*		Pressure monitoring utilized	Outcome
									From initial surgery	From symptoms of CS		
Hailer and colleagues[18]	43F	TKJR	Epi	Epi till fasciotomy	Ropivacaine and sufentanil (no dose details)		Paraesthesia, swelling, pain, increased analgesic requirements	Yes	48	27	No	Sensory, motor deficit
Kumar and colleagues[29]	46F	TKJR	Epi	Epi 20 h	NS	Obese (BMI 38)	Tenseness, swelling, pain once Epi removed	Yes	48	48	No	No disability
	71M	THJR	Epi	Epi 28 h	NS	BMI 28	Pain 16 h after Epi removed. Tense, firm, tender, swollen buttock	Yes	44	34	No	No disability
	55M	Hip resurfacing arthroplasty	Epi	Epi 19 h	NS	BMI 30	Pain 4 h after Epi removed	Yes	28	23	No	No disability
	72M	TKJR	Epi	Epi 43 h	NS	Ankle paralysis not noticed till Epi ceased	Foot drop, paralysis, buttock swelling	Yes	47	47	No	Limp, weak abduction
Benevides and Nochi Junior[3]	42M	Duodenal switch procedure	Epi/GA	NSAIDs/tramadol	NS	BMI 43, single-shot epidural injection	Pain, paraesthesia, tenseness, swelling. Pain on movement	Yes	No fascitomy	No fasciotomy	No	No disability
Haggis and colleagues[17]	69F	TKJR revision	Epi	Epi till fasciotomy	NS	Intraop vascular injury	No pain. Tight, swollen calf	Yes	14	NS	No	Foot drop, equinus
	53M	TKJR	Epi	Epi till fasciotomy	NS	Vascular compromise, osteomyelitis	Pain, cold, pulselessness, swelling	Yes	38	NS	No	Foot drop, equinus
	48F	TJKR	Epi	Epi 48 h	NS	Vascular compromise	Swelling, foot drop	Yes	192	NS	No	Foot drop, numbness
	49F	Bilateral TKJR	Epi	Epi 32 h	NS		Pain, foot drop	Yes	51	NS	Yes	Foot drop
	61M	TKJR	Epi	Epi 72 h	NS	Preop dalteparin	Pain, paralysis, paraesthesia, tight swollen calf	Yes	38	NS	No	Below knee amputation
Heyn and colleagues[22]	52M	Radical prostatectomy	Epi/GA	Epi till fasciotomy	NS	7 h lithotomy	Pain postop, pain passive stretch, swelling	Yes	No delay	No delay	Yes	No disability
Bezwada and colleagues[4]	60M	Bilateral TKJR	CSE	Epi 1 day	Bupivacaine and fentanyl (no dose details)	Diabetes, coronary artery disease	Weakness, paralysis, swelling, numbness	Yes	3 days	2 days	No	Reduced strength
Somayaji and colleagues[57]	39M	THJR	Epi/GA	Epi 36 h	B125 and fentanyl	Dense block at 6 and 24 h	Pain after Epi stopped, paralysis, paraesthesia	No	24	No delay	No	Reduced abduction and external rotation
Stotts and colleagues[58]	15F	Spinal instrumentation and fusion	GA	Epi till fasciotomy and PCA	Hydromorphone PCEA and PCA morphine		Cramping in recovery. Pain, tenderness, swelling	Yes	24	24	Yes	No disability

Continued

Table 2 *Continued*

Report	Patient age/gender	Surgery	Anaesthetic	Postop analgesia	Drug	Significant other issues	Signs/symptoms	Classic CS symptoms despite Epi	Time to fasciotomy (h)*		Pressure monitoring utilized	Outcome
									From initial surgery	From symptoms of CS		
Pacheco and colleagues[48]	47M	TKJR	Epi	Epi 22 h	NS	Obesity (BMI 42), dense motor block	Back pain once Epi ceased, then buttock pain	No	44	15	Yes	Gluteal pain
	71M	TKJR	Epi	Epi 43 h	NS	Dense motor block	Foot drop, paraesthesia once Epi ceased	Yes	47.5	No delay	No	Lower leg motor and sensory disability
Tang and Chiu[60]	62F	TKJR	Epi	Epi till fasciotomy	B125	Posterior dislocation noted postop, dense block	Decreased capillary return (day 2), no pain, calf swelling	Yes	2 days	No delay	Yes	Numbness, impaired walking
Dunwoody and colleagues[10]	14M	Hip osteotomy	Epi/GA	Epi 30 h	B1 and fentanyl	Developmental hip dysplasia	Pain, worse pain on movement	Yes	46	16 h	Yes	Paraesthesia, weakness
	7M	Ilazarov frame/ corticotomy	Epi/GA	Epi 1.5 days	B25 and fentanyl	Congenital short femur	Decrease pulse, calf spasm, reluctant to move foot	Yes	2 days	1 day	Yes	Decreased motion, normal sensation
Kontrbarsky and Love[26]	69M	Percutaneous nephrolithotomy	Epi/GA	Epi 18 h	B125 and fentanyl	Obesity, dense motor block	Buttock pain when Epi ceased	No	18	No delay	No	NS
	70M	Ankle fusion	CSE	Epi 48 h	B125	Obesity, sleep apnoea, dense motor block	Buttock pain	Yes	No fasciotomy	No fasciotomy	No	No disability
Goldsmith and McCallum[15]	48M	Total colectomy	Epi/GA	Epi removed postop	B5	Ulcerative colitis, 7 h lithotomy	Pain, tenseness, swelling, tenderness	Yes	14	NS	No	Bilateral foot drop
	40M	Laparotomy—rectal excision	Epi/GA	Epi till fasciotomy	B5 loading dose then B125 and fentanyl infusion	Ulcerative colitis, obese, 4 h lithotomy	Pain, erythema, tenderness (immediately postop)	Yes	No delay	No delay	No	No disability
Nicholl and colleagues[43]	65M	THJR revision	Epi/GA	Epi 2 days	Morphine	Preop enoxaparin, electrical calf stimulators	Pain, pain with passive stretch, swelling, tenderness	Yes	3 days	1 day	Yes	Decreased movement
Price and colleagues[49]	16M	Knee osteotomy	GA	Epi till fasciotomy	Fentanyl	Rickets	'Uncomfortable', tenseness, numbness	Yes	18	NS	Yes	No disability
Seybold and Busconi[54]	18M	Scapular fasciocutaneous-free flap grafting	Epi/GA	Epi till fasciotomy	NS	Obesity. 12 h procedure in lateral decubitus position. Total 4 h 48 min tourniquet time	Swelling, rigid compartment. Pain once Epi ceased	Yes	14	2	Yes	No disability
Tuckey[65]	28M	Major laparotomy	Epi/GA	Epi 4 days	B5 and fentanyl intraop, B125 and fentanyl postop	Ulcerative colitis	Bilateral leg pain, tenseness, swelling, tenderness	Yes	15	No delay	No	Bilateral foot drop
Slater and colleagues[56]	40M	Major laparotomy	Epi/GA	NS	NS	Ulcerative colitis, 11 h lithotomy	Pain, weakness	Yes	2 days	1 day	Yes	Weakness
Morrow and colleagues[39]	18M	Bilateral femoral IM nailing	GA	Epi	B2 and fentanyl	Motor bike accident	Unilateral paresis and anaesthesia, 'turgid' calf	Yes	13	No delay	Yes	NS

Beerle and Rose[2]	76F	Radical cystectomy	Epi/GA	Epi till fasciotomy	B5 and fentanyl intraop, B125 and fentanyl postop	7.5 h lithotomy	Pain, tenseness, swelling, pulselessness	Yes	1.5	No delay	Yes	No injury
Iwasaka and colleagues[24]	15F	Repair genitourinary fistula	Epi/GA	Epi till fasciotomy	M2 intraop, lidocaine in recovery	11 h lithotomy	Pain, tenseness, swelling, paraesthesia, paralysis in ankle and great toe	Yes	23	23	Yes	Slight limp
Montgomery and Ready[38]	52F	Radical cystectomy	Epi/GA	Epi till fasciotomy	L2 intraop and morphine after	10.5 h lithotomy	Numbness and muscle spasm, tightness, swelling	Yes	18.5	6	Yes	Sensory, motor deficit
	17F	Urological reconstruction	Epi/GA	Epi	B0625 intraop, morphine and fentanyl postop	11.5 h lithotomy	Numbness, swelling, pain, pain on passive stretch	Yes	16	16	Yes	Slight limp
Strecker and colleagues[59]	45M	Osteocutaneous-free fibular transfer	Epi	Epi 4 days	B125	Infected non-union tibia	Swelling, pain once Epi ceased	Yes	8 days	4 days	No	Not clear due to other injuries

Table 3 Signs attributable to compartment syndrome *vs* epidural infusions[41]

	Analgesia	Paraesthesia	Anaesthesia	Paralaysis	Swelling
Compartment syndrome	+/−	+	+	+	+
Epidural					
Low-dose local anaesthetic	+	+/−	−	−	−
Higher dose local anaesthetic	+	+	+	+	−
Opioids	+	−	−	−	−

and avoidance of motor block is 0.2%.[52] This is often combined with an opioid such as fentanyl 4 μg ml^{-1} to improve analgesia.[53] The pathological pain of compartment syndrome is unlikely to be masked by analgesia produced by dilute concentrations of local anaesthetic. One example is the report where compartment syndrome was promptly diagnosed and treated in the presence of an epidural infusion with bupivacaine 0.125% and fentanyl.[2] In contrast, the hazards of dense epidural block are highlighted in three reports of compartment syndrome in which patients had dense bilateral motor blocks.[26 57 60] Epidural analgesia provides effective pain relief after lower limb surgery, but should be supervised by an acute pain or anaesthetic service in order to derive the greatest benefit and avoid potential complications.[61] An alternative to epidural analgesia is continuous PNB (CPNB) and probably represents the gold standard for postoperative analgesia after major unilateral surgery. CPNB is associated with a reduced incidence of side-effects when compared with epidural analgesia.[12] The use of CPNB is increasing as the evidence for their efficacy increases. In a meta-analysis, perineural analgesia provided postoperative analgesia that was superior to opioids for all time periods and all catheter locations.[51] Ultrasound imaging aids precise perineural injection and may also facilitate the use of dilute concentrations of local anaesthetics for both the primary block and the subsequent infusion through the catheter in patients at risk of compartment syndrome. Local anaesthetics used with CPNB have included ropivacaine 0.2% or bupivacaine 0.25%[8] and in a comparative study ropivacaine 0.2% was as effective as ropivacaine 0.3%.[6]

A limitation of this review was that the data available were mainly from case reports and therefore statistical analysis was not possible. There may also be significant underreporting of complications like compartment syndrome, especially where medico-legal proceedings may be involved.

Conclusion

Compartment syndrome is challenging to diagnose and requires urgent treatment in order to avoid disastrous complications. This systematic review does not provide convincing evidence that PCA opioids or regional analgesia

delay the diagnosis of compartment syndrome. Whatever the mode of analgesia used, a high index of clinical suspicion, ongoing assessment of patients, and compartment pressure measurement are essential for early diagnosis.

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