A cute compartment syndrome is a common but potentially life threatening condition that occurs in the limbs and abdomen, and that requires prompt recognition and intervention. Caused by high pressure in a closed fascial space so that capillary perfusion is too low for tissue viability, it is well recognised as a potentially devastating complication of tibial shaft fractures and many other common conditions observed in emergency departments.

Diagnosis is usually made on clinical grounds following a high index of suspicion, with an excessive and unexpectedly disproportionate pain response being one of the key symptoms. Avoiding delay in recognition requires vigilance and repeated examination to avoid significant complications.

A detailed understanding of the pathophysiology involved in acute compartment syndrome contributes to understanding the seriousness of this condition and the signs and symptoms that are present. However, patients' inability to identify pain can mask its progression.

**MUSCLE TISSUE**

There are three types of muscle tissue: skeletal, cardiac and smooth (Marieb 2004). Compartment syndrome mainly involves skeletal muscles.

Like all body cells, skeletal muscle fibres are soft and fragile. The covering, or sheaths, of connective tissue support individual cells, reinforce muscle as a whole and provide it with its natural elasticity. It also provides entry and exit routes for blood vessels and nerve fibres serving the muscle.

The normal activity of skeletal muscle is absolutely dependent on a rich blood supply and its nerve supply. Unlike other muscle tissues, which can contract without nerve stimulation, skeletal muscle fibres are supplied with individual nerve endings that control their activity.

Muscle fibres use tremendous amounts of energy when they contract so they need a near continuous delivery of oxygen and nutrients by way of blood vessels. They also produce large amounts of metabolic waste that must be removed through the veins if contraction is to remain efficient. In general one artery and at least one vein serve each muscle.

**COMPARTMENT SYNDROME**

The muscles of the leg are grouped into four compartments that are formed by thick layers of fairly inelastic tissue called fascia (Fig. 1), and each compartment contains a major nerve as well as blood vessels (Table 1). Compartment syndrome is a serious condition whereby trauma or haemorrhage causes swelling within a muscle compartment. It is defined as 'an elevation of the interstitial pressure in a closed osseofascial compartment that results in microvascular compromise' (Mubarak and Hargens 1983).

If this condition goes unrecognised, it can be devastating, and delay in diagnosis can affect patients' future quality of life.

Compartment syndrome is fairly common following soft tissue injury, and young men are at particular risk especially if they have a clotting disorder or are taking anticoagulant therapy so that risk of intracompartment haemorrhage following trauma is increased (McQueen et al. 2000).

**SITES OF COMPARTMENT SYNDROME**

The most common site of compartment syndrome is the lower leg (Abramowitz and Schepsis 1994), with the anterior compartment being the most frequently affected, followed by the lateral compartment and the deep posterior compartment.
Increase in volume of the contents of the compartment can be caused by bleeding, infiltration of intravenous fluid or post-traumatic or ischaemic swelling. However, compartment syndrome can also occur in either of the two compartments of the forearm, and any of the three compartments of the thigh (Tiwari et al 2002) (Table 2). It can also occur in the abdomen after laparotomy for major trauma and can render the abdomen difficult or impossible to close (Ertel et al 2000).

Compartment syndrome can occur in an open wound if the skin laceration is insufficient to decompress the oedema or if there is haemorrhage. Even if the wound does not involve the compartment fascia, compartment syndrome is still possible. Compartment syndrome can also be seen elsewhere: the feet of patients with diabetes (Lee et al 1995) and the lower limbs after malignant hyperthermia (Johnson et al 1999).

**CAUSES AND PATHOPHYSIOLOGY OF COMPARTMENT SYNDROME**

Tiwari et al (2002) describe several ways that compartment size can be reduced (Table 3). The initial injury, whether traumatic, haemorrhagic, surgical, vascular or a complication of another condition, leads to localised swelling in the muscle compartments due to the stimulation of the inflammatory response (IR) (Huddleston 1992).

**INFLAMMATORY RESPONSE**

The IR is a systemic response that produces extensive inflammation by attracting nutrients, fluids, clotting factors and large numbers of neutrophils and macrophages to a damaged site. To attract these, damaged tissue releases mediators that are part of a chemotaxic signalling system. These mediators include histamine, prostaglandins and cytokines but the list is immense and is rapidly growing (Zuccarelli 2000). They cause a localised increase in capillary permeability, which leads to swelling, oedema and pain, and vasodilatation, which leads to redness and heat, the signs often observed at the site of inflammation (Fig. 2). The stimulation of the inflammatory process damages the endothelium of the blood vessels, which is a major contributor to the activation of the IR.

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**Table 1. The four compartments of the leg and their nerve supplies**

<table>
<thead>
<tr>
<th>Compartments of the leg</th>
<th>Nerve supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior deep peroneal</td>
<td>Fibula</td>
</tr>
<tr>
<td>Deep posterior, or medial tibial</td>
<td>Superficial posterior</td>
</tr>
<tr>
<td>Lateral superficial peroneal</td>
<td>Superficial posterior</td>
</tr>
<tr>
<td>Superficial posterior muscle compartment</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2. The compartments of the forearm and thigh**

<table>
<thead>
<tr>
<th>Compartments of the forearm</th>
<th>Nerve supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volar (anterior) musculocutanous nerve</td>
<td>Deep posterior, or medial, muscle compartment</td>
</tr>
<tr>
<td>Dorsal (posterior) radial nerve</td>
<td>Lateral muscle compartment</td>
</tr>
</tbody>
</table>

**Table 3. Compartment syndrome**

<table>
<thead>
<tr>
<th>Location</th>
<th>Nerve supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior femoral</td>
<td>Superficial posterior</td>
</tr>
<tr>
<td>Posterior sciatic</td>
<td>Deep posterior, or medial, muscle compartment</td>
</tr>
<tr>
<td>Medial obturator</td>
<td>Lateral muscle compartment</td>
</tr>
</tbody>
</table>

**Fig. 1. Lower leg compartments**

Deep posterior, or medial, muscle compartment
Superficial posterior muscle compartment
Fibula
Lateral muscle compartment
Anterior muscle compartment
Blood vessels
and nerves
Tibia
Fibula
The endothelium is not just an inert barrier between the blood and the substructure of the blood vessels and tissue (Edwards 2002a). It is an active metabolic organ, and when damaged, is responsible for stimulating anticoagulation pathways. Stimulation of the clotting cascade always accompanies injury and is closely linked to the IR. This serves to prevent excessive blood loss and isolate the injured site (Huddleston 1992).

Increased capillary permeability leads to swelling and an increase in intracompartmental pressure (ICP). Compartments have relatively fixed volumes so introducing fluid or constricting them increases pressure and decreases tissue perfusion. This results in hypertension, or an increase in hydrostatic pressure (HP), at the venous end of the capillary bed (Tiwari et al. 2002).

But further oedema occurs when there is an increase in HP at the arterial or venous end of the capillary bed (Edwards 2003a). This raises the pressure of blood in the capillary and causes an increase in filtration rate. Larger than usual amounts of fluid then move into the interstitial space and cause the collection of even more fluid in the compartments (Fig. 3).

INCREASED PERFUSION PRESSURE

Elevated perfusion pressure is the physiological response to rising ICP. As ICP rises, autoregulatory mechanisms are overwhelmed and a cascade of injury develops. A normal resting intramuscular pressure is 0-8mmHg. Pain and paraesthesia occurs at 20-30mmHg (Tiwari et al. 2002). An ICP of 30mmHg is often used as a basis for performing a fasciotomy (Cooper 1992). Whitesides et al. (1975) and later McQueen and Court-Brown (1996) however use a differential between diastolic pressure and ICP of 10-30mmHg as the threshold for doing so. Using this method severely reduces the number of patients that undergo this procedure without endangering those who do (Ovre et al. 1998).

Tiwari et al. (2002) suggest that fasciotomy should be performed when the difference between mean arterial pressure and ICP—the so-called /g98 pressure—is 40-50mmHg. But the ideal /g98 pressure threshold for performing a fasciotomy remains unknown.

If ICP exceeds 30mmHg and observations are compatible with compartment syndrome, prompt therapies to decrease the pressure such as removal or opening of casts, skeletal fixation of unstable fractures, maximising local arterial pressure, placing the limb at a level with the heart or in some instances anticoagulation to prevent complications, are necessary. If ICP exceeds 40mmHg, emergency treatment is needed because blood flow through the capillaries and therefore oxygen delivery will cease.

HYPOXIC INJURY

Eventually blood flow is so reduced that tissue is no longer viable (Phillips 1992). Increased interstitial pressure overcomes intravascular pressure of the microcirculation, causing the vessel walls to collapse and thereby impeding blood flow still further. This results in local tissue ischaemia and intracellular oedema, which in turn further increases ICP (Jobe 1992). Meanwhile, the interrupted supply of oxygenated blood results in anaerobic metabolism, loss of adenosine triphosphate (ATP) and cellular membrane disruption (Edwards 2002b) (Fig. 4).

High intracellular potassium and low intracellular sodium and calcium concentrations are maintained by active transport systems. Without sufficient ATP, the plasma membrane of the cells can no longer maintain normal ionic gradients, and the sodium and potassium, and calcium, pumps can no longer function.

<table>
<thead>
<tr>
<th>Table 3. Ways that compartment size can be reduced</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fracture (tibia or fibula fractures, usually in the middle or distal third of the leg, or supracondylar fracture of the humerus)</td>
</tr>
<tr>
<td>Crush injury</td>
</tr>
<tr>
<td>Excessive exercise of a muscle group</td>
</tr>
<tr>
<td>Surgical procedures including closure of fascial defects</td>
</tr>
<tr>
<td>Major vascular surgery</td>
</tr>
<tr>
<td>Bleeding disorders</td>
</tr>
<tr>
<td>Compression bandages</td>
</tr>
<tr>
<td>Constrictive devices such as a tight cast</td>
</tr>
<tr>
<td>Insect sting or snake bite</td>
</tr>
<tr>
<td>Burns</td>
</tr>
<tr>
<td>IV drug use</td>
</tr>
<tr>
<td>Weightlifting</td>
</tr>
<tr>
<td>Post-ischaemic swelling</td>
</tr>
</tbody>
</table>

All of the above generally lead to ischaemia due to the swelling in the compartment. Prognosis depends directly on the time interval between the onset of the intracompartmental ischaemia and the start of effective treatment.
Potassium leaks into the extracellular space, while calcium and sodium followed by water move into the cells. This causes cellular oedema and increased intracellular osmotic pressure (Edwards 2003b), which can eventually cause the cells to burst. Muscle contraction relies on the passage of electrochemical impulses down specialised pathways and requires the movement of sodium, potassium and calcium ions in and out of cells to produce action potentials. These action potentials may limit movement and contraction of muscle but these changes are reversible if oxygen supplies are restored. If interventions such as fasciotomy and oxygen administration are not started, intracellular acidaemia and cellular dysfunction become extreme. This can lead to intracellular lysosome membrane disruption, an accumulation of intracellular calcium and, if allowed to continue, irreversible cell damage, loss of limb and death. Necrotic muscle never recovers so the seriousness of this condition and the importance of recognising the warning signs should not be underestimated. The surrounding tissue suffers further damage because of increased pressure within the compartment, leading to injury, stimulation of the IR and reduced blood supply. This leads to further hypoxic damage, which stimulates the release of IR mediators, thereby developing a vicious cycle.

**MYOGLOBINURIA**

When muscle tissue suffers necrosis, it releases the intracellular muscle protein, myoglobin. Excess myoglobin after major muscle trauma appears in urine as a dark reddish brown colour. Myoglobinurea, often known as rhabdomyolysis, is indicative of severe, life-threatening muscle trauma. The renal threshold for myoglobin is low, about 0.5mg/100ml urine, so that only 200g of muscle need be damaged to cause visible changes in the urine (McCance and Huether 2003). Priorities on observing myoglobinurea include identifying and treating compartment syndrome and preventing life-threatening renal failure. Myoglobinurea from muscle death, as well as causing acidosis and renal failure, can also result in amputation of the involved extremity, sepsis and death. Early diagnosis of compartment syndrome is therefore essential.
PATIENT MANAGEMENT

Ischamia is a major feature of compartment syndrome but blood pressures are only rarely low enough to occlude arterial blood flow (Fig. 5). For early recognition, more subtle skills and knowledge are required.

EARLY RECOGNITION

Recognising compartment syndrome early is imperative (Middleton 2003a). Early symptoms are pain that is disproportionate to the apparent injury and paraesthesia. If present, they are signs of severe involvement. Pulselessness is uncommon and rarely noted as an early symptom; it appears at a much later stage and generally implies vascular injury.

Nerves that traverse the area of decreased perfusion are affected and paresthesias develop in their distribution. They may be an early complaint but, if allowed to persist, can be irreversible. Unfortunately, paresthesias are also seen after contusions that affect nerves and after vascular damage.

Nevertheless, the most reliable physical finding in compartment syndrome is sensory deficit (Moore and Friedman 1989). The loss of two-point discrimination is a sensitive sign (Tiwari et al 2002) and the distribution of sensory changes can help differentiate which compartments are affected.

However, all of these signs of vascular injury can only be identified in fully conscious patients. Emergency nurses must therefore be vigilant in clinical observation and examination.

INTRACOMPARTMENT PRESSURE MEASUREMENT

Patients with ICPs of 10-30mmHg should be admitted and followed closely with repeated pressure measurements (Table 4). If the pressure continues to increase, treatments are indicated. Due to the effect of blood pressure on the development of compartment syndrome, it may be more appropriate to monitor differential pressures such as that between diastolic pressure and ICP rather than absolute ICP (McQueen and Court-Brown 1996, Tiwari et al 2002, Whitesides et al 1975).

ADMINISTERING ANALGESIA

Drug overdose and pain deserve a special mention because they can rival or surpass limb trauma as major causes of compartment syndrome.

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Fig. 4. Cellular changes following hypoxic or ischaemic injury

Ischaemia and impaired tissue perfusion causing generalised cellular damage

Anaerobic cellular metabolism causes the formation of lactic acid and oxygen free radicals

Depletion of ATP and failure of sodium/potassium pumps

Lysosome membranes damaged, releasing toxic enzymes into the cytoplasm

Endothelial damage, cytotoxic vasodilator mediators released into the circulation

Cellular damage leading to tissue and organ death and failure

Mitochondrial calcium further impairs cellular functions

Progressive vasodilatation, myocardial depression, increase in capillary permeability and intravascular coagulation

Release of mediators such as kinins, serotonin, lysosomal enzymes and histamine leads to stimulation of the inflammatory immune response

The formation of nitric oxide

Water and sodium influx causes cellular swelling and metabolic acidosis

Cellular swelling

HP = 1mmHg

OP = 0mmHg

NFP = 20 - 25 = -5mmHg

Arterial end

HP = 1mmHg

OP = 0mmHg

NFP = 20 - 25 = -5mmHg

Venous end

HP = 43mmHg

OP = 25mmHg

HP = 21mmHg

OP = 25mmHg

HP = 1mmHg

OP = 0mmHg

NFP = 17 - 5 = 12mmHg deficit

leading to oedema formation

HP = hydrostatic pressure, OP = opposing pressure to the HP, NFP = net filtration pressure

If hydrostatic pressure at the arterial end of the capillary increases by 5mmHg, from 38 to 43mmHg, and at the venous end from by 5mmHg, from 16 to 21mmHg, filtration increases, absorption reduces and fluid accumulates in the interstitial space (oedema).
Patients who self-administer morphine regularly, for example those who use patient-controlled analgesia (PCA), do not at first perceive pain, so diagnosis of compartment syndrome can be delayed. Pain is a vital protective response; it can warn of a tight cast, or of underlying haematoma, infection, ulceration, subluxation or slipping of fixation. Conditions that mask a pain response can result in devastating consequences. Patients with compartment syndrome can present with no pain response because of high blood morphine concentrations administered in hospital for pain relief. Nevertheless, the first symptom of compartment syndrome is usually pain, which often appears late and out of proportion to initial injury. Because of the seriousness of the condition, therefore, a low level of pain or discomfort should be advocated where the risk of compartment syndrome is high. This is difficult because pain is what patients say it is, but it can be determined by pain assessment.

**TREATMENT**

Full recovery from compartment syndrome is probable when treatment is started within four and eight hours of the onset of symptoms, such as swelling or pain. Intracompartment pressure measurement (Table 4) should be considered immediately when compartment syndrome is suspected. If the ICP is not relieved by decompression of cast or temporary elevation of limb, fasciotomy is indicated to decompress the compartment and allow reperfusion of the muscle. Fasciotomy has a high complication rate because it transforms a closed lesion into an open wound (Fitzgerald et al. 2000). The open wound can be allowed to heal by second intention, where for example the skin is left open after the fasciotomy and allowed to granulate from the outside in, or undergo skin closure within four days, skin grafting or flap coverage. However, the combination of an open wound and necrotic muscle can contribute to the development of life-threatening infection and other complications such as acidosis, renal failure and loss of limb.

**CONCLUSION**

Compartment syndrome simply cannot afford to be overlooked. If it is confirmed or suspected, prompt consultation with an orthopaedic or vascular surgeon is warranted. If left unchecked, the cycle of oedema and ischaemia results in muscle infarction, nerve injury and permanent loss of function in the extremity. Diagnosis of compartment syndrome in the leg can be delayed because patients fail to show a pain response. This ultimately results in an emergency fasciotomy and possible shock. This should prompt health practitioners to question the use of PCA in any trauma-tised limb. It is suggested that ICP monitoring of the anterior compartment of the leg be carried out when analgesic manoeuvres can have masked early clinical features of the syndrome. Nurses must be ever vigilant when caring for patients with limb injuries and be aware of the physiology, signs and symptoms indicating the development of compartment syndrome, and there is clearly a need for prompt diagnosis and intervention, without which limbs and lives can be lost. There is a need for further nursing literature, better evaluation of the syndrome and the development of nurses’ skills in recognising the syndrome early and using the appropriate measuring techniques.
Table 4. Measuring ICP

<table>
<thead>
<tr>
<th>Type</th>
<th>Procedure</th>
<th>Advantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple needle</td>
<td>Uses an 18-G needle</td>
<td>A saline injection into the tissues is required while taking the reading.</td>
</tr>
<tr>
<td>Saline draw</td>
<td>Uses the same needle</td>
<td>A water draw is needed to reduce clotting problems</td>
</tr>
<tr>
<td>Wick catheter</td>
<td>Uses polyglycolic acid</td>
<td>Accurate and allows continuous measurement.</td>
</tr>
<tr>
<td>Slit catheter</td>
<td>Made from epidural</td>
<td>Uses an infusion system but easier technique to infuse 0.7ml saline.</td>
</tr>
<tr>
<td>Transducer</td>
<td>Available in specialist monitoring</td>
<td>Allows continuous measurement.</td>
</tr>
<tr>
<td>Side ported</td>
<td>Allows measurement</td>
<td>Cannot be used for continuous measurement.</td>
</tr>
<tr>
<td>Central venous</td>
<td>Uses a manometer technique</td>
<td>An 18-G needle is used. Quick, with no special equipment required.</td>
</tr>
<tr>
<td>Fiberoptic</td>
<td>Expensive and only easy to use.</td>
<td>Allowing continuous monitoring of several compartments.</td>
</tr>
</tbody>
</table>

Johnson IA et al (1999) Lower limb compartment syndrome after 'damage control' laparotomy in 311 patients with severe abdominal and/or abdominal compartment syndrome after 'damage control'