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Review article

DIAGNOSING ACUTE COMPARTMENT SYNDROME

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An acute compartment syndrome is a surgical emergency, which if not recognised and treated early, can lead to devastating disabilities, amputation and even death.

Aetiology

Compartment syndrome has been defined as "a condition in which increased pressure within a limited space compromises the circulation and function of the tissues within that space".[1] It is most commonly seen after injuries to the leg[2-5] and forearm[6-8] but may also occur in the arm,[9] thigh,[10] foot,[11-13] buttock,[14] hand[15] and abdomen.[16] It typically follows traumatic injury, but may also occur after ischaemic reperfusion injuries,[17] burns,[18] prolonged limb compression after drug overdose[19] or poor positioning during surgery.[20-24] Furthermore, subclinical compartment syndromes may explain the occurrence of a variety of postoperative disabilities which have been identified after the treatment of fractures of long bones using intramedullary nails.[25]

Approximately 40% of all acute compartment syndromes occur after fractures of the tibial shaft[26] with an incidence in the range of 1% to 10%.[26-30] A further 23% of compartment syndromes are caused by soft-tissue injuries with no fracture and fractures of the forearm account for 18%.[26] Acute compartment syndrome is seen more commonly in younger patients, under 35 years of age[31] and therefore leads to loss of function and long-term productivity in patients who would otherwise contribute to the country's workforce for up to 40 years.

Pathophysiology

Although there are a number of hypotheses regarding the impairment of the microcirculation which occurs in a compartment syndrome, the arteriovenous pressure gradient theory has gained the most popularity.[32] First introduced by Matsen and Krugmire,[1] it is based on the premise that ischaemia begins when local blood flow cannot meet the metabolic demands of the tissue. As the intracompartmental pressure rises, the intraluminal venous pressures also increase leading to a reduction in the arteriovenous pressure gradient with subsequent diminished or absent local perfusion. The resulting reduction in venous drainage causes a further rise in interstitital tissue pressure with the formation of tissue oedema. The lymphatic drainage is subsequently increased to protect against the rising interstitial fluid pressure. However, once this has reached its maximum, further increases in the intracompartmental pressure cause deformation and ultimately collapse of the lymphatic vessels.[33] It is only in the late stages of a compartment syndrome that the arterial flow into the compartment is seriously compromised and the continuing flow of blood into the compartment augments the swelling and oedema throughout the early stages of the syndrome.

Outcome

The end results of an unchecked acute compartment syndrome are catastrophic and include neurological deficit, muscle necrosis, ischaemic contracture, infection,[34] and delayed healing of a fracture.[29] It can also lead to the 'crush syndrome', marked by acute renal failure as a result of the precipitation of myoglobin released from damaged muscle in the distal convoluted tubules of the kidneys.[34] Acidic, hyperkalaemic blood is also released from the damaged muscle, resulting in cardiac arrhythmias and further precipitation of myoglobin in the kidneys. Extensive irreversible damage to muscle can also lead to amputation, and on occasion, loss of life. The incidence of these complications is strongly related to the timing of fasciotomies,[34] and therefore, it is essential that compartment syndromes are diagnosed and treated early before irreversible damage occurs.

Treatment

There is widespread agreement that the immediate removal of all circumferential dressings down to skin, followed by open and extensive fasciotomies with decompression of all

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the compartments within a limb is the treatment of choice.[31,33,35,36] This relieves the pressure and allows the return of blood flow into the injured tissue in the absence of an arterial injury.

Fasciotomies. Since the acceptance of fascial release as the definitive treatment for compartment syndrome, several surgical approaches have been tried in the leg. Following the recognition of four separate compartments in the leg,[37,38] Kelly and Whitesides[38] advocated a single lateral incision with fibulectomy. This approach only allowed limited views and adequate fascial release was difficult. The technique was modified to the parafibular approach by Matsen and Krugmire in 1978,[1] which avoided excision of the fibula and reduced the incidence of damage to the superficial peroneal nerve. Subsequently, Mubarak and Owen[39] advocated the use of two incisions, one lateral between the tibia and fibula and one medial behind the posteromedial border of the tibia. This technique allows safer access to all four compartments with good visualisation of important superficial and deep structures, and remains the treatment of choice for compartment syndrome in the lower leg.

Limited dermotomies were popular for a time as they were believed to reduce the rate of infection, cause less bleeding with less damage to the collateral circulation and leave smaller scars with a shorter healing time thus facilitating secondary wound closure. However, Cohen et al[40] found that the lengthening of the skin wounds in the leg from 8 cm (limited) to 16 cm (wide) significantly reduced the intra-compartmental pressures. Wide dermotomies and fasciotomies allow adequate inspection, reduce the risk to the superficial peroneal nerve and eliminate the possibility of the skin becoming the limiting envelope following reperfusion swelling, while adding little to the associated morbidity.

Making the diagnosis of acute compartment syndrome is often difficult and the decision to operate is frequently delayed. Despite attempts to identify consistent clinical and objective measures of an impending compartment syndrome, no reliable, clear-cut diagnostic guidelines have been established so far.

Clinical assessment

Initially, a high index of suspicion is required to make the diagnosis. It is important to remember that the syndrome can occur after any injury regardless of the aetiology, velocity or degree of comminution of the fracture. A common misconception is that a build-up in intracompartmental pressure (ICP) is unlikely to occur after an open fracture on the assumption that the haematoma has an escape route through the torn fascia and that muscle can expand without limitation.[3] However, usually the small, transverse fascial tears which occur in association with open fractures do not adequately decompress the compartment.[28,41]

Clinicians should be aware that the compartmental pressure can be raised by the various methods of immobilising a fracture. During intramedullary nailing, peaks in the pressure occur as a result of traction,[30,42] reduction of the fracture, reaming and insertion of the nail.[43] Reduction of the fracture lengthens and narrows the surrounding compartments resulting in a decrease in compartment volume and therefore an increase in compartment pressure, the so-called "finger-trap phenomenon".[44] Application of a long-leg plaster cast[45] also elevates the pressure significantly, and further changes may occur with elevation of the injured limb. Although the absolute pressure is diminished by relieving soft-tissue swelling,[46] high elevation also reduces the arteriovenous pressure gradient and renders the tissues less tolerant to raised intracompartmental pressures.[47] This may partially explain the occurrence of the syndrome after the lithotomy[23] and hemilithotomy[20,24] positions which are used in certain surgical procedures.

Currently, the clinical diagnosis is made on a constellation of physical signs and symptoms which include increasing pain out of proportion to the stimulus, altered sensation, pain on passive stretch of the affected muscle compartment, muscle weakness, and palpable tenseness of the compartment. All have inherent drawbacks in making the diagnosis. Pain is an unreliable and variable indicator. It can range from being very mild to severe and is already present in patients who have been subjected to recent trauma.[31] The pain of the injury can mask that of an impending compartment syndrome and pain alone cannot be depended upon as a reliable diagnostic symptom.[35] Mubarak et al[48] found that pain in response to passive stretching of the affected muscle compartment was also an unreliable sign and suggested that the presence of hypoaesthesia was more dependable. However, Rorabeck and Macnab[3] found hypoaesthesia to be the last clinical finding to develop as the syndrome progressed. Sensory changes may occur as a result of a simultaneous nerve injury not related to the development of acute compartment syndrome.[25] Palpable tenseness is a crude indicator of increased compartmental pressure.[48] Since only a small part of the deep posterior compartment of the leg is sufficiently superficial to be palpable beneath the skin,[44] this sign is of little value in diagnosing involvement of this compartment. Frank motor weakness is very difficult to elicit in a patient after trauma, and a demonstrable weakness may also result from an isolated nerve injury.[36] Furthermore, if an acute compartment syndrome is so advanced as to cause a detectable muscle weakness, a full functional recovery is rare.[27,36,49] In Bradley's study[2] only 13% of patients in whom paralysis was present at the time of diagnosis recovered function.

The key to the successful treatment of the acute compartment syndrome is early diagnosis. Based on the clinical studies which have been undertaken to date the symptoms and signs which appear to be the most reliable in making an early diagnosis, are increasing pain and pain on passive stretching of the muscles within the affected compartment.[3,4,50] However, since both of these symptoms are subjective they are impossible to elicit in patients who are

unconscious or unco-operative, as can occur after a head injury, or in children.

Objective methods of diagnosing the acute compartment syndrome

ICP monitoring. Following Matsen's unified concept[51] identifying increased intracompartmental pressure in all cases of the syndrome regardless of the aetiology, monitoring of intra-compartmental pressure was introduced in the 1970s to facilitate early diagnosis. Since then, many new systems of monitoring pressure have been developed to improve the sensitivity and accuracy of the measurements obtained.

Devices for monitoring pressure. Initially, Whitesides et al[52] utilised a hand-held needle manometer which could be used for intermittent recordings but this system required the injection of saline to maintain patency. Concerns were raised that this could aggravate an impending compartment syndrome and could also generate falsely high pressure readings. Currently, there are a number of commercially available needle manometer systems of this type, but all have been shown to have difficulties and inaccuracies. None can be used for continuous pressure measurement, and therefore they are not able to monitor the trend of the pressure within a muscle compartment. They require a bubble-free column of saline to prevent dampening of the recording, and both muscle and blood clot can block the needle tip, rendering these monitors useless. In a compartment syndrome model in which the accuracy of the needle manometer was compared with wick and solid-state catheters, the needle manometer performed least well, consistently giving falsely high readings at pressures less than 60 mmHg, and falsely low readings at pressures greater than 60 mmHg.[53]

The wick[53] and slit[54] catheters were developed to overcome some of these flaws. Both designs work on the basis that an increase in the surface area of the tip of the device within the tissues improves the accuracy for measuring tissue pressure. The tips are not uniform, fibrils extend out from the end of the wick catheter, and the slit catheter tip has several cuts at its end to reduce the likelihood of blockage by haematoma or surrounding muscle. Despite these improvements, blockage can occur, and both systems still rely on a bubble-free column of saline to function. Although these methods can provide continuous pressure recording for up to 24 hours,[54] the accuracy of the measurements is also dependent upon the correct placement of the external transducer dome at the level of the tip of the catheter. Rorabeck et al[54] and McQueen et al[43] among others, advocate the use of the slit catheter as the most accurate method for continuously monitoring the intracompartmental pressure over a 24-hour period.

Further attempts to reduce the potential inaccuracies associated with these devices led to the development of a solid-state transducer intracompartment catheter. This has a multiperforated tip, and incorporates a solid-state transducer within the lumen. It was hoped that it would resolve the difficulties associated with the correct positioning of the external dome transducer at the exact height of the tip of the catheter, found with the previous types of monitor.[55] However, this device also requires a pool of heparinised saline around its tip to maintain patency and has not therefore proved to be significantly better than earlier designs.

The latest device for monitoring pressure is the transducer-tipped probe. This has been shown to be easy to use and highly accurate,[56] with an excellent dynamic response to changing pressures, and without the artefacts associated with saline-column systems.[57] Although a falsely high reading can be obtained by placement of the tip of the catheter within the intramuscular tendon,[56] this can be avoided at insertion by small changes to the depth and direction of the tip. It seems likely that this new device will prove to be the best method of measuring the intracompartmental pressure.

Interpretation of measurements of intracompartmental pressure

These advances in technology are of little value unless they are accompanied by simultaneous improvements in the guidelines for interpreting their readings and appreciating their clinical relevance.

The absolute intracompartmental pressure. Many attempts have been made to identify the critical level of pressure above which the viability of the compartment is compromised. Initially, it was considered that a critical level of intracompartmental pressure could be identified, above which fasciotomy should be performed. Levels of 30 mmHg,[28,32,36,40,48,58,59] 45 mmHg[35,60] and 50 mmHg[61] have since been suggested as the critical threshold above which circulation within the muscle compartment is compromised. The lower level of 30 mmHg, which is most commonly used, is based on the view that when the tissue fluid pressure is greater than 30 mmHg, the capillary pressure is insufficient to maintain muscle capillary blood flow.[58] In addition, fascial compliance decreases sharply at an absolute ICP of 33 mmHg, as the fascia reaches its maximum limitation of stretch.[58]

Differential pressure. Whitesides et al[52] introduced the concept that the level of intracompartmental pressure at which ischaemic compromise of muscle tissue occurs is related to the perfusion pressure, and there is now a growing body of evidence to support this view. Setting an absolute pressure ignores the role of the blood pressure in maintaining an adequate blood flow within the compartment. Tissue viability is dependent on adequate perfusion, and the blood flow within the microcirculation is dependent on both the tissue and venous pressures. Heppenstall et al[62] found that the pattern of tissue disturbance at a fixed intracompartmental pressure was erratic and variable, while the damage occurring at a specific level relative to the blood pressure was more consistent. Several authors who still use the absolute level to diagnose an acute compartment syndrome have

also noted that hypotension can lower the threshold for irreversible muscle injury.[27,63-65]

The 'delta pressure' is the diastolic blood pressure minus the intracompartmental pressure, and the critical level has been found to range between 10 mmHg[66] and 35 mmHg.[67] The most commonly cited delta pressure, currently used to diagnose acute compartment syndrome, is less than or equal to 30 mmHg.[31,41,51] In a study using this delta pressure for surgical intervention it was demonstrated that many unnecessary fasciotomies were avoided, while patients who had developed an increase in compartmental pressure sufficient to cause obvious tissue compromise as seen at the time of fasciotomy were identified correctly.[41] No clinically significant complications related to an acute compartment syndrome were identified within the group with a delta pressure greater than 30 mmHg in whom fasciotomy was withheld, although such clinical parameters may not be sufficiently sensitive to exclude all sequelae which may result from a compartment syndrome.

Others have found that by using a differential pressure calculated using the mean arterial pressure minus the intracompartmental pressure, greater accuracy can be achieved. Mars and Hadley[68] found that a differential pressure of 30 mmHg or less indicated the need for fasciotomies in children, and this level was also found in dogs in which an acute compartment syndrome had been induced in the hindlimb.[69] A further study in dogs[62] indicated that if the muscle is traumatised before the introduction of a compartment syndrome, the tissue is more susceptible to the increased pressure, suggesting that a wider threshold of 40 mmHg should be used in a clinical setting.

The time factor. The observation of isolated measurements of pressure which are equal to, or greater than, the suggested threshold level for fasciotomy, does not necessarily confirm that a compartment syndrome is established. Instead, it is the trend of intracompartmental pressure or delta pressure over time which should be observed.[5,43] Transient, but significant increases in intracompartmental pressure occur during operative procedures, such as intramedullary nailing of fractures of long bones, and these relatively short peaks in pressure are not associated with an increased incidence of compartment syndrome.[43]

There is a dynamic relationship between the blood pressure, the level of intracompartmental pressure and the duration of time for which a raised pressure is maintained. With increasing pressure, greater dysfunction occurs more rapidly, but a lower pressure maintained for a longer period of time may also cause similar tissue damage. Prolonging the interval between onset and treatment by fasciotomy results in a poorer long-term clinical outcome.[44]

With the wide variety of absolute and differential pressures used in the studies performed to date, a direct comparison is difficult. However, it is clear that the higher the pressure, the faster is the damage. Hargens et al[58] found that an intracompartmental pressure of 30 mmHg maintained for eight hours in dogs in the presence of a normal blood pressure, resulted in significant muscle necrosis. In patients with soft-tissue trauma to the lower leg a poor outcome resulted from an intracompartmental pressure of 40 mmHg of six hours' duration.[61] Significant changes in muscle biochemistry and histology were also observed when a differential pressure of 20 mmHg was maintained for between four and six hours. In situations in which the differential pressure approached zero, these biochemical and histological changes were present two to three hours earlier.[69]

Examination of the velocity of nerve conduction indicated that total failure of conduction occurred after 50 minutes in the presence of a pressure of 120 mmHg, and in 330 minutes at an intracompartmental pressure of 50 mmHg.[70] The lowest threshold for nerve dysfunction was 30 mmHg, provided that it was maintained for between six and eight hours. However, at this pressure only the fast conducting fibres were affected, with sparing of the slow fibres. Rorabeck and Clarke[71] found that conduction in the peroneal nerve was completely blocked by an absolute intracompartmental pressure of 40 mmHg maintained for 150 minutes.

The longer the delay to fasciotomy, the worse is the outcome. In a group of patients with involvements of the deep posterior compartment of the leg, a delay of at least 12 hours to surgical decompression resulted in permanent disability in four of five patients, while those treated within 12 hours had no apparent clinical deficits at follow-up.[44] The findings of this study are substantiated by Sheridan and Matsen,[34] Rorabeck and Macnab,[3] McQueen et al,[31] and Mullett et al,[72] who concluded that catastrophic clinical results were inevitable if fasciotomies were delayed for over 12 hours, whereas a full recovery was achieved if decompression was performed within six hours of making the diagnosis. In the clinical situation, the exact time of onset of an acute compartment syndrome cannot be accurately identified, and therefore clinicians must depend on the confirmation of the diagnosis as the marker for delay to fasciotomy.

Sources of confusion in interpretation of the intracompartmental pressure

Several sources of error arise from studying the compartment pressure. Traumatised tissue is more susceptible to a rise in pressure since it has an increased metabolic rate secondary to the injury. This, in turn, leads to greater energy requirements and increased oxygen consumption. Clinical trials involving patients who have suffered an injury and have tissues with higher energy requirements, cannot be directly compared with animal experiments, in which a model of a compartment syndrome is used without further tissue trauma. Heppenstall et al[62] therefore suggest that the threshold of pressure used in trauma should be lower than the standard levels found in experimental animal models using non-traumatised muscle. The time scale for nerve damage is different from that of skeletal muscle[59,73] and many clinical trials are flawed in that they examined only

nerve dysfunction or muscle damage in assessing the clinical outcome. There is also considerable variability among individuals as to their tolerance to a given level of intracompartmental pressure.[48,64]

Another potential source of error is the site within the compartment at which the pressure is measured. Initially, it was believed that tissue pressure rapidly equilibrates throughout the compartment and therefore pressure could be monitored at any site with an accurate indication of the overall pressure in the compartment. This theory has subsequently been shown to be wrong, both in experimental saline injection models[74,75] and in clinical trials involving patients. Heckman et al[74] warned "that failure to measure tissue pressure within a few centimetres of the zone of maximum pressure may result in serious underestimation of the maximal ICP". They also stated that it is the highest figure that should be used in deciding the need for fasciotomies.

Matava et al[75] monitored intracompartmental pressure at various levels within the anterior compartment of the lower leg in patients with tibial fractures. This revealed that the zone of highest pressure was always within 5 cm of the site of the fracture. Outside this zone the recorded pressure could be as much as 20 mmHg lower than the true peak pressure. They therefore recommend that the pressure should be recorded as close to the site of injury as possible. This practice could, theoretically, turn a closed fracture into an open one, with all the associated risks but there have been no studies to date to confirm or refute this. It seems sensible therefore to monitor pressure within the 5 cm zone, but not directly over or connecting with the fracture.

Concern has been expressed about monitoring only one compartment in the leg when the syndrome may develop in neighbouring compartments. This is a particular problem in unconscious or unco-operative patients with injuries to the lower limb in whom monitoring of the pressure is the only diagnostic tool available. It is impractical to monitor the intracompartmental pressure in all four compartments continuously. Rorabeck[36] suggested the initial measurement of the pressure in all of the compartments in the injured limb, followed by continuous monitoring of that with the highest initial recording. Unfortunately, this compartment may not be the one in which the syndrome then develops since it can occur within hours or days of the initial injury in any of the adjacent compartments. Others rely on the clinical findings to determine the compartment with an impending syndrome, and use measurements of pressure only to confirm the diagnosis.[48] Heckman et al[74] found that the highest pressure was in the anterior or deep posterior compartments, or both, in 24 of the 25 patients whom they examined, while McQueen et al[31] found involvement of the anterior compartment in all 25 patients diagnosed with the syndrome in their study. They now recommend routine monitoring of the anterior compartment, including the other compartments only as clinically indicated.

Given such disparate results, it is not surprising that a recent survey in the UK indicated a considerable variation in the opinion and practice of consultant orthopaedic surgeons in the management of patients at risk of acute compartment syndrome.[76] Despite overwhelming support for the use of the monitoring of intracompartmental pressure, there was a wide variety of methods by which this investigation was performed and interpreted. More than half of the group did not have pressure monitoring available in their hospital, and 42% did not know at what level they would perform fasciotomies. It was concluded that "monitoring equipment should be made universally available throughout the UK", the type of which "needs to be standardised to prevent patients suffering unnecessary fasciotomies and more importantly to avoid missing a potentially catastrophic compartment syndrome".

Despite its flaws, monitoring of intracompartmental pressure is the only objective method currently available to aid the diagnosis of an acute compartment syndrome. By using monitoring, clinical awareness is increased, and it may also help to confirm the diagnosis in the presence of equivocal clinical findings. Lack of monitoring is the most common factor associated with a missed diagnosis.[5] McQueen et al[31] advocate monitoring in all patients at risk as an adjunct to clinical diagnosis, while others use it solely in unconscious patients and in those with equivocal clinical findings.[6]

Alternative methods

As an alternative to monitoring pressure, technologies from other areas of medicine have been investigated.

Near infrared spectroscopy (NIRS). This is a non-invasive method of detecting variations in the level of muscle haemoglobin and myoglobin. It is applicable to compartment syndromes on the basis that light in this spectrum passes relatively easily through both skin and bone, but is differentially absorbed by haemoglobin and oxyhaemoglobin.[77] Most studies which have used NIRS have investigated patients with the chronic exertional form of compartment syndrome (CECS) and it appears that although it has good predictive power in comparing CECS with normal patients, its diagnostic value in acute compartment syndrome is limited. It is a good method for detecting trends and changes in relative oxygenation, but of no value in the acute syndrome since changes in relative oxygenation may have already occurred.[78,79]

NIRS was found to differentiate between an induced acute compartment syndrome and both hypoxia and hypotension in pigs.[77,80] However, it should be borne in mind that loss of dorsiflexion twitch was used as the endpoint to indicate a compartment syndrome and no muscle histology or biochemistry was obtained. No cut-off point for venous oxyhaemoglobin saturation was identified in the acute syndrome. Further limitations in the use of NIRS, particularly in the leg, include the restricted measuring depth of the probe, which lies between 3 mm and 40 mm. Superficial

muscle readily absorbs the light, and therefore the deep muscles cannot be isolated.

Magnetic resonance imaging. MRI has a wide variety of diagnostic applications in modern medicine, and some encouraging results have been obtained in studies examining its ability to diagnose both chronic exertional[81] and acute compartment syndromes.[82] It is thought that the frank oedema which occurs in the compartment syndrome can be visualised on T1-weighted MRI. Studies performed to date have included only small sample numbers, and confounding factors, such as atherosclerosis, may exaggerate the changes seen on MRI in response to exercise.[83] In acute trauma, swelling and oedema will be present to variable degrees after an injury, and may not be readily distinguishable from the acute compartment syndrome by MRI. Rominger et al[82] found that the changes on MRI in an established compartment syndrome with swollen compartments and loss of normal muscle texture, correlated well with both the intraoperative findings and the tissue histology. However, MRI could not identify any changes associated with an imminent compartment syndrome with no neurological signs.

Scintigraphy. This is used to evaluate regional perfusion, in particular myocardial perfusion, and more recently, perfusion in peripheral vascular disease and popliteal entrapment. Edwards et al[84] showed that ^{99}Tcm-methoxyisobutyl isonitril (MIBI) scintigraphy had a positive predictive value of 89% in a study investigating 46 patients with suspected CECS. They also found that all four compartments of the lower leg could be distinguished in every case. It is not known if this technique is applicable to acute compartment syndrome, but as it is not a continuous measure of changing perfusion, it is unlikely that it would be of value in acute trauma.

Laser doppler flowmetry. Limited studies have been performed to assess invasive laser doppler flowmetry as a diagnostic aid in compartment syndromes. Abraham et al[85] found that there were differences in patients with and without CECS, but considered that further investigation was to be required in order to gain information regarding the sensitivity and specificity of this investigation in a larger group of patients.

Conclusions

There is still no conclusive answer as to the critical threshold of intracompartmental pressure at which fasciotomies should be performed. Since the pressure is not a direct measure of tissue damage and there is considerable variation among individuals as to their tolerance to a given pressure, it may not be possible to define the critical level more satisfactorily nor to establish the duration of raised intracompartmental pressure which results in an acute syndrome. The real breakthrough in making an accurate, early diagnosis is more likely to come from new technology which will provide more direct information about the degree of tissue ischaemia within a compartment.

Summary and Guidelines

In the meantime, we must rely on the best available clinical and experimental evidence:

1) Vigilance must be maintained in all potential cases of acute compartment syndrome. To this end the education of those caring for these patients is essential.

2) Monitoring of the intracompartmental pressure should be routine, particularly in patients in whom subjective clinical assessment is not available, i.e. in unconscious or uncooperative patients, and in those under the age of 35 years with injuries to the lower leg.

3) Emergency conservative measures should be instituted if the delta pressure approaches or drops below 30 mmHg, or clinical symptoms develop. Appropriate measures include the restoration of normal blood pressure in the hypotensive patient, the removal of all constrictive dressings, the maintenance of the limb at heart level, and the commencement of supplementary oxygen.

4) Full and extensive fasciotomies should be performed within six hours if the delta pressure remains less than 30 mmHg, and/or clinical symptoms and signs persist despite conservative measures. In the leg, this should be performed using both medial and lateral incisions and should include all four compartments. Although the morbidity of fasciotomies is significant, it is preferable to the outcome of a missed compartment syndrome.

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