Compartmental Syndromes:
When the Pressure Gets Too Great

- A compartmental syndrome is a condition in which increased pressure in a confined space compromises the circulation and function of the contents of that space.
- Compartmental syndromes most often arise when swelling of muscle occurs in a part of the arm or leg that is confined by a tough tissue known as fascia.
- Compartmental syndromes are common causes of permanent disability following injuries such as fractures of the leg, fractures of the forearm and arm, dislocation of the elbow or knee, and prolonged pressure on the arm or thigh.
- Compartmental syndromes may also arise from excessive exercise, such as prolonged running or marching.
- The most practical and sensitive method for evaluating an arm or leg for possible compartmental syndrome is repeated clinical examination looking for (1) pain out of proportion to what would be expected from the clinical situation, (2) weakness of the muscles in the compartment, (3) diminished sensation in the distribution of the nerves running through the compartment, (4) pain on passive stretch of the muscles in the compartment, and (5) tenseness on palpation of the compartment.
- If untreated, compartmental syndromes can lead to permanent deformity and loss of function, including both strength and sensation in the hand or foot.
- Because tissue has a very limited tolerance for increased pressure, the diagnosis of compartmental syndrome needs to be made promptly and surgical decompression needs to be carried out within hours of its onset.
- Once a compartmental syndrome is present, elevation of the limb above the heart further compromises the circulation to the compressed tissues.
- When a large amount of muscle dies as a result of a compartmental syndrome (rhabdomyolysis), there is a risk of kidney failure.
- In conditions where the risk of compartmental syndrome is high, preventative treatment, known as prophylactic fasciotomy is considered.

Thirty-five years ago there was a lot of confusion regarding the cause of weakness and deformity in the hands and feet after seemingly straightforward injuries, such as a fracture of the arm from roller skating or a fracture of the tibia from skiing. It was also noted that army recruits required to march long distances would develop similar problems in their legs and that sometimes this condition was complicated by failure of the kidneys. The names attached to these problems were confusing as well: march gangrene, Volkmann’s ischemia, ischemic contracture, and claw foot. Opinion on the cause was divided: some attributed the problem to spasm of the arteries and others believing blockage of the veins was the problem. The diagnosis was often missed until it was too late because physicians could not understand how there could be inadequate circulation to the leg or arm when the pulses were normal and the digit tips were pink. This seeming paradox was ultimately explained by our group as shown in Figure 2.

Kay Clawson and Ted Hansen had a substantial interest in complications of trauma. They suggested that Rick Matsen take a year out of clinical residency training to work toward a better understanding of post-fracture swelling and its effects on limb function. Funding for this year was provided by...
In the 1974-1981 era, substantial contributions to the field were made by an expanded investigative team that included Dick Krugmire, Geoff Sheridan, Bob Winquist, Anne Matsen, Keith Mayo, Rachael King, Cammy Mowery, Craig Wyss, Charles Simmons, Stan Newell and Rob Veith. These contributions can be grouped in categories as describe below.

Vocabulary and causation

We discovered that many clinical conditions shared the same underlying physiology: increased pressure within a limited space can compromise the circulation and function of the contents of that space. We defined a compartmental syndrome as any condition where this mechanism was in effect. We found that the leg had four compartments, any of which could be the site of a compartmental syndrome: the anterior, the lateral, the superficial posterior and the deep posterior (this latter was first described by our group in 1975). Compartmental syndromes of the forearm were most common in the deep and superficial volar compartments. While these compartments in the leg and forearm are limited by tough fibrous tissue known as fascia, compartmental syndromes can also occur within tight skin, tight dressings, and tight casts. One of the more interesting cases we encountered was a compartmental syndrome occurring within shrunk-fit jeans!

The increased pressure within these limited spaces can arise from a wide variety of causes, including fractures, contusions, crush injuries, surgery, arterial injury, infiltrated infusions, prolonged exercise and post-ischemic swelling (the swelling that occurs after circulation is restored following a period of poor circulation).

Pathophysiology

In each compartmental syndrome regardless of the initial cause, the mechanism of circulatory compromise is the same. Tissue is normally nourished by blood flowing through capillaries. The rate of capillary blood flow is determined by the perfusion pressure, i.e. the difference between the pressures at the arterial and venous ends of the capillaries. The pressure at the arterial end is the systemic blood pressure (unless the limb is elevated, in which case the local blood pressure is reduced by the amount of elevation). The pressure at the venous end of the capillary is determined by the local tissue pressure. In the normal recumbent person, the mean arterial pressure in the leg may be 80 mm Hg and the local venous pressure may be 10 mm Hg, so the perfusion pressure driving capillary blood flow might be 70 mm Hg. If, however, the local tissue pressure in the anterior compartment of the leg is increased by swelling in the closed space to 60 mm Hg, the perfusion pressure is reduced to 20 mm Hg, which may be insufficient to keep the tissues in that compartment functioning and even alive. Note that a tissue pressure of 60 mm Hg is not enough to cut off the arterial circulation (with its pressure of 80 mm Hg) so that the pulses at the ankle and the circulation to the toes may be completely normal. Note also that if this leg is elevated (in a vain attempt to reduce the swelling), the local arterial pressure will be reduced by the amount of the hydrostatic column between the leg and the heart, further reducing the capillary perfusion pressure. As a specific example, elevation of the leg 52 cm above the level of the heart reduces the local arterial pressure, as well as the tolerance of the leg for increased tissue pressure by 40 mm Hg! (1.3 cm
whole blood = 1 mm Hg).

Tolerance of tissue for increased pressure

We were very interested in the question of how living tissue responded to increased tissue pressure. We developed a laboratory model of compartmental syndrome that consisted of a balloon that could be placed within a compartment of a small animal and then inflated to any desired pressure while the function of the muscle in the compartment was monitored. We then turned to a human model system in which pressure was applied to the leg using an air splint while the functioning of the nerves and muscles within the leg were monitored clinically and electrophysiologically. In both the animal and human models we were able to measure the tissue oxygen tension as well, using a special catheter connected to a mass spectrometer. Using the human model we were again able to demonstrate that the susceptibility of the tissue of the pressurized leg to ischemia (poor blood flow) was significantly increased by elevation of the leg above the level of the heart.

New efforts have been focused on a non-invasive, continual and real-time monitoring system for tissue perfusion. Near infrared spectroscopy (NIRS), a new technology which uses visible light to measure the percentage of oxygenated hemoglobin approximately 3 cm below the skin, has been FDA approved for monitoring cerebral perfusion during anesthesia. At Harborview Medical Center, Garr et al. applied this technology in the diagnosis of acute compartmental syndrome. Muscle oxygenation measured by NIRS was shown to be inversely related to increased intracompartmental pressures with high specificity and sensitivity in animals as well as human tourniquet studies.

Diagnosis of compartmental syndrome

Although we pioneered methods for the continuous monitoring of intracompartmental pressures, our clinical experience taught that individuals varied with respect to their tolerance for increased tissue pressure and that no absolute or relative value for intracompartmental pressure could be considered diagnostic of a compartmental syndrome or indicative of the need for surgical decompression (Figure 3).

Instead the most sensitive and specific approach for diagnosing compartmental syndromes is the repeated assessment of the physiological status of the tissue within the compartment by documenting the presence or absence of: (1) pain out of proportion to what would be expected from the clinical situation, (2) weakness of the muscles in the compartment, (3) diminished sensation in the distribution of the nerves running through the compartment, (4) pain on passive stretch of the muscles in the compartment, and (5) tenseness on palpation of the compartment. This clinical examination is most sensitive if it is performed serially (i.e. hourly) on limbs at risk for compartmental syndromes and if the findings are well-documented in the chart, where it is accessible to all those caring for the patient.

Treatment

Once the tissue within the compartment shows signs of ischemia from increased pressure, the treatment is surgical decompression by opening the fascia, skin, and any dressings that may potentially contribute to the increase in pressure.

One of our contributions to treatment was the description of the relatively function-preserving perifibular technique for decompression the four compartments of the leg (see Figures 5 & 6). In the management of individuals with compartmental syndrome it is important to keep an eye on the function of the kidneys, making sure the patient does not develop renal failure from the load of myoglobin released into the circulation by dying muscle.

Conclusion

In the future, research needs to be directed at anticipating when limbs will be at risk for compartmental syndrome so that they can be closely monitored and that prophylactic decompression can be considered. In addition, research needs to be directed at methods for preserving muscle and nerve function as well as joint range of motion after surgical treatment of compartmental syndromes.

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Figure 4: Surgical decompression of the deep compartment of the forearm from the relatively concealed ulnar incision. (Reproduced from Matsen: Compartmental Syndromes.)

Figure 5: The perifibular approach to decompression of the four compartments of the leg: decompression of the anterior and lateral compartments. (Reproduced from Matsen: Compartmental Syndromes.)

Figure 6: The perifibular approach to decompression of the four compartments of the leg: decompression of the superficial and deep posterior compartments. (Reproduced from Matsen: Compartmental Syndromes.)

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References


http://www.orthop.washington.edu/compartmentalsyn