Diagnosis and Management of Compartmental Syndromes

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ABSTRACT: Patients at risk for compartmental syndromes challenge both the diagnostic and the therapeutic abilities of the physician. Suboptimum results may be due to delays in diagnosis and treatment, to incomplete surgical decompression, and to difficulties in the management of the limb after decompression. Although careful clinical assessment permits the diagnosis of a compartmental syndrome in most patients, we have found measurement of tissue pressure and direct nerve stimulation to be helpful for resolving ambiguous or equivocal cases. In our experience, the four-compartment parafibular approach to the leg and the ulnar approach to the volar compartments of the forearm provide efficient and complete decompression of potentially involved compartments. The skeletal stabilization of fractures associated with compartmental syndromes may facilitate management of the limb after surgical decompression.

The goals of the physician who is caring for a patient with a compartmental syndrome are early diagnosis, prompt decompression, and an uncomplicated recovery. These goals may not be realized because of problems in recognition and management of affected compartments. The symptoms and signs of a compartmental syndrome may be sufficiently ambiguous that a definite diagnosis cannot be made on clinical grounds alone. The differential diagnosis of a compartmental syndrome may be problematic as well.

Although surgical decompression is the definitive treatment of a compartment syndrome, confusion concerning the indications for such decompression may delay this procedure until it is no longer beneficial and possibly deleterious. The advent of techniques for measurement of tissue pressure has provided an objective means of evaluating the status of a compartment, although it is impractical to measure the pressures in all compartments at risk. Furthermore, the significance of a given pressure-measurement value remains open to question. Even if the need for surgical decompression is determined promptly, an inadequate release of potentially limiting fascial and cutaneous envelopes will obviate a good result. Finally, a fractured limb that has required surgical decompression often presents the combined problems of bone instability and a large incision that cannot be closed primarily.

This article presents a practical approach to the patient at risk for a compartmental syndrome which is designed to help the physician to avoid potential pitfalls in management of the condition. The approach evolved from a five-year experience gained at the University of Washington affiliated hospitals and through consultation with other physicians.

Diagnosis

Clinical Symptoms and Signs

Many compartmental syndromes can be diagnosed from clinical symptoms and signs alone. These include: (1) pain out of proportion to the clinical situation; (2) weakness and pain on passive stretch of the muscles of the compartment; (3) hypoesthesia in the distribution of the nerves running through the compartment; and (4) tense ness of the fascial boundaries of the compartment.

We examine patients who are at risk for compartmental syndromes at frequent intervals, and the results of each examination are carefully documented. Any limb showing signs of a compartmental syndrome is freed from circumferential dressings and is placed at the level of the heart to assure that local blood pressure is not compromised by elevation of the extremity. If these signs do not resolve promptly, surgical decompression usually is indicated.

Adjunctive Diagnostic Techniques

The physician may encounter situations in which diagnosis of a compartmental syndrome cannot be made or ruled out with certainty from clinical findings alone. The clinical findings may be equivocal and the patient may have head, spinal cord, or peripheral nerve injuries; be uncooperative; or have communication difficulties. In these situations, we have found the adjunctive diagnostic techniques of tissue-pressure measurement and direct nerve stimulation to be useful.

Tissue-Pressure Measurement

Although several techniques for measuring tissue
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We investigated the clinical significance of different intracompartmental pressures using the infusion technique to prospectively evaluate thirty-one compartments (thirty patients) at risk for compartmental syndromes. The patients were carefully observed for clinical evidence of a compartmental syndrome and we correlated the clinical observations with the results of tissue-pressure monitoring, particularly with the maximum intracompartmental pressure. Twenty-five anterior tibial compartments, three deep posterior tibial compartments, two volar compartments of the forearm, and one quadriceps compartment of the thigh were monitored. The duration of pressure monitoring ranged from less than fifteen minutes to seventy hours, depending on the clinical situation. Even for the longer periods of monitoring, the infusion system continued to maintain patency of the catheter. In no case did the system require flushing or discontinuance because of obstruction of the catheter. All patients in this series who had maximum intracompartmental pressures of forty-five millimeters of mercury or less did not require fasciotomy and demonstrated no residua of a compartmental syndrome at follow-up (Fig. 2). All patients having maximum intracompartmental pressures of more than fifty-five millimeters of mercury displayed significant losses of neuromuscular function attributable to a compartmental syndrome.

Perhaps the most significant observation in this series of patients was that individuals varied in their tolerance for increased tissue pressure. Thus, there was a range of intracompartmental pressures in which some patients demonstrated neuromuscular deficits while others did not. This variability in pressure tolerance was evident in our investigation of model compartmental syndromes in rabbits and humans. We have also demonstrated that the pressure tolerance of a limb may be affected by its position relative...
to the heart. Thus, the concept of a critical pressure above which surgical decompression should be performed is of limited value. If a low value is selected as a critical pressure, all patients with significant compartmental syndromes would certainly be included. Yet it is likely that surgery would be performed in a number of patients who would have no significant functional losses without such intervention. For example, thirteen patients in our series had tissue pressures in excess of thirty millimeters of mercury, yet because of their benign clinical examinations they did not undergo surgical decompression. None of these patients demonstrated residua of a compartmental syndrome at follow-up. If a high value is selected as a critical pressure, unnecessary surgery would be unlikely, but some patients who would benefit from fasciotomy would not receive it. For these reasons we have not used the concept of a critical pressure in determining the need for surgical decompression.

Currently measurement of tissue pressure is used for: (1) the evaluation of a compartment in which the diagnosis of compartmental syndrome cannot be made or ruled out with certainty, and (2) the prospective evaluation of a compartment at high risk, as in a patient with a severely swollen limb but with no neural deficits. Because it is impractical to monitor tissue pressure in all parts of all compartments at risk, the catheter is placed in the part of the limb that clinically appears most tense. Monitoring is continued until the question of a compartmental syndrome is resolved. The data from pressure measurement are integrated with all other available data on the patient in determining the need for surgical decompression.

Sheridan et al. 21, Rorabeck and Clarke, and more recently Hargens et al. 4 have demonstrated that the duration of pressure elevation is fully as important in the production of neuromuscular deficits as is the magnitude of pressure elevation. Pressures that are benign for a few hours may be detrimental if allowed to persist for longer periods. Thus, continuous monitoring of tissue pressure provides clinically useful information as to the trend of intracompartmental pressure.

Direct Nerve Stimulation

If a patient is unable to voluntarily contract the muscles of a compartment after an injury to an extremity, the physician must differentiate between a compartmental syndrome and a primary nerve injury proximal to the compartment. In this situation, an objective method for differentiating the two diagnoses can be clinically useful. We have found a small, battery-powered nerve stimulator (NS-2A peripheral nerve stimulator, Professional Instruments Company, Houston, Texas) to be of value in evaluating the integrity of the myoneural junction in patients who are unable to contract the intracompartmental muscles voluntarily. The motor nerve supplying the muscles of the compartment is stimulated just proximal to the compartment, using either surface or needle electrodes. We have found that the stimulus is best tolerated by the patient if delivered via two 3.8-centimeter (one and one-half-inch) 25-gauge hypodermic needles steriley placed one centimeter apart near the nerve in question. Because the myoneural junction is the part of the motor unit that is most sensitive to ischemia, a muscle that is paralyzed by a compartmental syndrome would be expected not to have a response to stimulation of its motor nerve. However, when paralysis is due to an acute nerve injury proximal to the compartment, stimulation of the motor nerve as it enters the compartment would be expected to produce a normal muscle response. Obviously, when the patient can voluntarily contract the muscles within the compartment in question, nerve stimulation is not necessary. However, in patients with more proximal injuries to the nerves, spinal cord, or head, the physician’s ability to diagnose compartmental syndromes on clinical grounds alone is greatly hindered. It is in these cases that direct nerve stimulation proves most useful. Nerve stimulation is not a useful technique for prospective monitoring of patients at risk for this condition, however, because of the importance of diagnosing compartmental syndromes before paralysis ensues.

Diagnostic Problems: Illustrative Case Reports

The following five cases demonstrate the usefulness of continuous monitoring of tissue pressure and direct nerve stimulation in resolving ambiguous clinical assessments in patients who are at risk for a compartmental syndrome.

CASE 1. A twenty-five-year-old male pedestrian was hit by an automobile and sustained a fracture of the third cervical vertebra and a severely comminuted fracture of the proximal end of the right tibia. On admission, he had hypoaesthesia in the distribution of the deep peroneal nerve and could not actively extend the toes. Active flexion of the toes was strong, and the sensation in the tibial-nerve distribution was intact. There was no pain on passive flexion or extension of the toes. The proximal end of the leg was swollen and bruised. Stimulation of the right peroneal nerve at the level of the fibular neck produced strong extension of the toes, indicating that the paralysis was not due to a compartmental syndrome. Anterior tibial compartment pressures reached a maximum of twenty millimeters of mercury. These data indicated that the loss of active toe extension was due to a more proximal nerve injury, probably a traction neurapraxia.

CASE 2. An eighteen-year-old man sustained an anterior dislocation of the left knee while playing football. Following reduction of the dislocation, examination revealed swelling of the proximal end of the leg, no active extension of the toes, hypoaesthesia in the distributions of the deep and superficial peroneal nerves, and a diminished dorsalis pedis pulse. An arteriogram revealed an intimal tear near the origin of the anterior tibial artery. Stimulation of the peroneal nerve at the fibular neck produced strong extension of the toes. Pressure measurements in the anterior compartment reached a maximum of fifteen millimeters of mercury. These data indicated that the paralysis was not due to compartmental ischemia, but rather to an injury of the peroneal nerve proximal to the fibular neck.

CASE 3. A thirty-four-year-old woman lay on her left side for twenty-four hours after a barbiturate overdose. After awaking, she was unable to extend her foot or toes. The anterolateral aspect of the left leg was swollen, but the compartments did not appear clinically tense. Stimulation of the peroneal nerve distal to the fibular neck elicited nor-
normal extension of the foot and toes. Anterior compartment pressures reached a maximum of twenty-two millimeters of mercury. Subsequent formal measurement of the conduction velocity of the nerve and electromyography confirmed our initial impression of common peroneal-nerve palsy caused by direct pressure. There was no subsequent evidence of compartmental or crush syndrome. There was no myoglobinuria.

Case 4. A twenty-five-year-old woman sustained a closed fracture of the distal end of the shaft of the right tibia in a fall. On initial examination, she had hypoesthesia in the distribution of the deep peroneal nerve along with paralysis of the extensor digitorum brevis muscle. Although the compartments were soft, she was unable or unwilling to extend the toes voluntarily. Stimulation of the peroneal nerve at the fibular neck produced strong extension of the toes. Anterior compartment pressure rose from an initial value of twenty-five millimeters of mercury to a maximum of fifty millimeters of mercury without change in the patient's neuromuscular status. Within one month of injury the strength of toe extension was normal, although the distribution of the deep peroneal nerve was still hypoesthetic and the extensor digitorum brevis muscle was still weak. Subsequent measurement of nerve conduction velocity and electromyography confirmed the diagnosis of injury to the deep peroneal nerve at the level of the fracture, occurring at the time of the accident.

Case 5. A sixty-year-old female pedestrian was hit by an automobile which was traveling at approximately 133 kilometers (seventy miles) an hour. She sustained multiple trauma, including a depressed fracture of the skull, a pelvic fracture, an intertrochanteric fracture of the right femur, and a spiral fracture of the right tibia with avulsion of the femoral neck. She was comatose from the head injury. Intermittent stimulation of the right peroneal nerve provided assurance that the presence of the characteristic clinical symptoms and signs of a compartmental syndrome, including deficits in neuromuscular function. Compartments demonstrating equivocal or ambiguous clinical signs in the presence of significantly elevated intracompartmental pressure are also decompressed. As a result of the data shown in Figure 2, we currently use a tissue pressure in excess of forty-five millimeters of mercury as a relative indication for surgical decompression, assuming a normal blood pressure, blood volume, and peripheral vascular system. Obviously these indications must be tempered by the patient's over-all condition and the trend of the symptoms, signs, and pressure measurements.

Technique for decompression of the volar compartments of the forearm. A. The incision extends from the medial epicondyle to the ulnarmost extent of the flexor crease of the wrist. B. The lacertus fibrosus and fascia overlying the flexor carpi ulnaris (FCU) are opened. C. The flexor carpi ulnaris is retracted ulnarily and the flexor digitorum superficialis (FDS) is retracted radially to permit opening of the fascia of the deep volar compartment. Care is taken to avoid the ulnar artery and nerve (U. a. & n.).

Techniques of Surgical Decompression

The goal of surgical decompression in compartmental syndrome is the expedient, complete opening of all tight fascial envelopes. One may be tempted to minimize this procedure either by doing a fasciotomy through limited skin incisions or by not decompressing all of the potentially involved compartments. These limitations, however, may yield poor results because of inadequate decompression. In a significantly involved limb, limited skin incisions or subcutaneous fasciotomy are not used for two reasons. First, complete decompression of all fascial and epimysial envelopes cannot be assured; and second, the postischemic hyperemia and swelling that are expected within the first hour after decompression of an ischemic compartment may well cause a secondary compartmental syndrome within the intact cutaneous envelope.

In decompressing the volar compartment of the forearm, we prefer the technique illustrated in Figure 3. This technique provides good access to all important structures through a linear ulnar incision which can be extended proximally and distally if indicated. We frequently perform a release of the carpal tunnel in conjunction with this procedure. After decompression the muscles of the forearm are palpated; if they are soft, no further surgical intervention is necessary. However, residual muscle tension may require epimysiotomy, as described by Eaton and Green.

Several techniques have been described for decompressing the compartments of the leg. Because of reluctance to use limited skin incisions or to sacrifice the fibula, we use the four-compartment parafibular approach as shown in Figure 4. Because of experience with two pa-
TABLE I

PATIENTS WITH FOUR-COMPARTMENT PARAFIBULAR DECOMPRESSION AND SKELETAL FIXATION OF ASSOCIATED Tibial Fractures

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, Sex (Yrs.)</th>
<th>Tibial Fracture</th>
<th>Compartments Involved</th>
<th>Internal Fixation</th>
<th>End Result*</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>32, M</td>
<td>Segmental</td>
<td>Anterior, superficial, and deep posterior</td>
<td>Closed intramed. nail</td>
<td>United fracture, normal function</td>
</tr>
<tr>
<td>7</td>
<td>23, M</td>
<td>Oblique (distal end of shaft)</td>
<td>All four</td>
<td>Plate</td>
<td>United fracture, normal function</td>
</tr>
<tr>
<td>8</td>
<td>22, M</td>
<td>Segmental</td>
<td>All four</td>
<td>Closed intramed. nail</td>
<td>United fracture, normal function except for moderate contracture of FHL</td>
</tr>
<tr>
<td>9</td>
<td>18, M</td>
<td>Spiral (distal end of shaft)</td>
<td>Anterior and lateral</td>
<td>Plate</td>
<td>United fracture; 4/5 strength of EHL and tibialis anterior, otherwise normal function</td>
</tr>
<tr>
<td>10</td>
<td>12, M</td>
<td>Oblique (mid-shaft)</td>
<td>All four</td>
<td>Roger Anderson pin fixation</td>
<td>United fracture, normal function</td>
</tr>
<tr>
<td>11</td>
<td>25, M</td>
<td>Oblique (distal third)</td>
<td>Anterior and deep posterior</td>
<td>Closed intramed. nail</td>
<td>United fracture, normal function</td>
</tr>
</tbody>
</table>

* FHL = flexor hallucis longus and EHL = extensor hallucis longus.

Patients who showed sequelae of a deep posterior compartmental syndrome after their anterior compartments alone were decompressed, all four compartments of the leg are decompressed if any one of them sustains an acute compartmental syndrome. To date, we have decompressed fourteen legs using the four-compartment parafibular approach. Satisfactory decompression was attained in all cases, as demonstrated by the softness of all muscle groups when palpated at operation and by the subsequent clinical course of the patients.

Management of the Limb following 
Surgical Decompression

After surgical decompression, the limb is splinted and

![Technique of four-compartment parafibular decompression of the leg. A. An incision is made from the fibular neck to the lateral malleolus. B. The lateral compartment (LC) is opened. C. Retracting the anterior skin exposes the fascia of the anterior compartment (AC), which is opened, with care being taken to avoid the superficial peroneal nerve (SPn). D. The posterior skin is retracted to expose the fascia of the superficial posterior compartment (SPC), which is opened. E. The lateral compartment is retracted anteriorly. The soleus is released from the fibular shaft and is retracted posteriorly, exposing the fascia of the deep posterior compartment (DPC), which is opened.](image-url)
passive range-of-motion exercises are performed to prevent contractures. Three to five days after decompression, the patient is returned to the operating room for a dressing change and skin closure. If delayed primary closure cannot be accomplished, we use Patman and Thompson’s progressive wound-edge approximation technique, in which sterile paper tapes are reapplied to the wound each day. On two occasions we were able to reduce a fourteen-centimeter-wide skin defect to a scar one-half centimeter wide using this technique.

After surgical decompression of a fractured limb, the surgeon may be faced with both a significant wound and an unstable fracture. The increased intracompartamental pressure that had given the fractured limb some stability (acting much like an air-splint) is no longer present. Unless the limb can be splinted in such a way that the fracture is maintained in proper alignment and the wound is available for inspection and treatment, skeletal fixation of associated fractures is considered at the time of compartmental decompression.

Rigid fixation maintains good position of the fracture fragments, protects the soft tissues, and facilitates access to the wound. The data on six consecutive patients with tibial fractures treated in this manner are summarized in Table I. Satisfactory results were obtained in all of them. In each instance, we believe that management of the limb without skeletal stabilization would have been difficult and perhaps deleterious to the eventual outcome.

References