

Acute Compartment Syndromes: Diagnosis and Treatment with the Aid of the Wick Catheter*†

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ABSTRACT: Intracompartmental pressures were measured by the wick catheter technique in sixty-five compartments of twenty-seven patients who were clinically suspected of having acute compartment syndromes. A pressure of thirty millimeters of mercury or more was used as an indication for decompressive fasciotomy. The range of normal pressure was from zero to eight millimeters of mercury. Eleven of these patients were diagnosed as actually having compartment syndromes and in these patients, twenty-seven compartments were decompressed. Only two patients had significant sequelae. In the sixteen patients (thirty-eight compartments) whose pressures remained less than thirty millimeters of mercury, fasciotomy was withheld and compartment syndrome sequelae did not develop in any patient. Intraoperatively the wick catheter was used continuously in eight patients to document the effectiveness of decompression. Fasciotomy consistently restored pressures to normal except in the buttock and deltoid compartments, where epimysiotomy was required to supplement the fasciotomy. Continuous intraoperative monitoring of pressure by the wick catheter technique allowed us to select the few cases in which primary closure of wounds was appropriate and to decide which patients were best treated with secondary closure.

The sequelae of a compartment syndrome may be devastating — Volkmann's contracture, neural deficit, and even gangrene. The common denominator of compartment syndromes is an elevated interstitial fluid pressure causing vascular occlusions in the compartments containing muscle. Many investigators have demonstrated the relationship between increased tissue pressure and ischemia of muscle^{4,11-13}. Although the critical pressure has not been definitely established, estimates^{6,10,13} have ranged between forty and fifty millimeters of mercury. Whitesides

and associates believed that a pressure above this range in a compartment is adequate reason, in itself, for surgical decompression. The measurement of intracompartmental pressure therefore may be used as an objective test to diagnose a compartment syndrome in the early stage. The wick catheter technique provides an accurate and reproducible method for measurement of interstitial fluid pressure⁸, and in the present study it was used not only to aid in the diagnosis of compartment syndromes but also in treatment, to indicate the effectiveness of the decompression and to define the specific roles of fasciotomy and epimysiotomy.

Materials and Methods

Between July 1974 and June 1976, we subjected all patients with suspected compartment syndromes who were seen at the University of California at San Diego Medical Center, Balboa U.S. Naval Hospital, and the Veterans Administration Hospital in San Diego to intracompartmental pressure measurements using the wick catheter** technique⁸. We included all patients who had painful, swollen limbs with palpably tense compartments. Motor and sensory deficits usually were present. Routine laboratory tests included a hemogram and urinalysis as well as serum potassium, creatinine, blood urea nitrogen, and creatine phosphokinase determinations. Wick catheter measurements were performed in each suspected compartment, and in most patients the pressures were compared with those in the contralateral limb.

We determined previously⁸ that the pressure in a recumbent individual averages four millimeters of mercury. Every patient with a compartment pressure equal to or greater than thirty millimeters of mercury had a fasciotomy and was included in the syndrome group. The pressure level of thirty millimeters of mercury was selected as critical for two reasons: First, normal muscle capillary pressure is from twenty to thirty millimeters of mercury as determined in cats² and dogs⁵ by direct measurement with micropipettes, and if the intracompartmental pressure exceeds thirty millimeters of mercury the intracompartmental circulation to muscles and nerves will be impeded⁵. Second, our previous experience with continuous pressure measurements in patients undergoing osteotomy⁸ showed that pain and paresthesia first appeared when the pressure reached approximately thirty millimeters of mercury.

Patients with pressures of twenty to twenty-nine millimeters of mercury as measured by the wick catheter were

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TABLE I
COMPARTMENT SYNDROME PATIENTS

Case	Age/Sex (Yrs.)	Cause	Findings	Level of Creatine Phosphokinase* (IU)	Compartments	Preoperative Wick Catheter Pressures (mm/Hg)	Fasciotomy	Skin Closure	Time from Injury to Fasciotomy (Hrs.)	Results
1	27/M	Contusion	Swollen leg, pain, motor and sensory loss, pulses intact	1,620	Ant. leg, lat. leg	50 40	+ +	Delayed 6 days	7	Normal leg
2	53/F	Aortic bypass surg.	Swollen leg, coma, pulses absent	34,200	Ant. leg, lat. leg, sup. post. leg, deep post. leg	35 36 22 38	+ + + +	—	12	Cardiogenic shock; died 7 hrs. later
3	19/M	Tibial plateau fracture	Swollen leg, pain, motor and sensory loss, pulses intact	1,740	Ant. leg, lat. leg, sup. post. leg, deep post. leg	27 30 30 13	+ + + +	Delayed 5 days	12	Normal leg
4	41/F	Drug overdose, limb compression	Swollen forearm, coma, pulses intact	2,040	Volar forearm, dorsal forearm, biceps	40 20 12	+ — —	Delayed 7 days	24	Normal forearm and hand
5	24/M	Gunshot wound and fracture	Swollen forearm, pain, motor and sensory loss, pulses intact	200	Volar forearm	35	+	Delayed 6 days	18	Normal forearm and hand
6	70/M	Contusion	Swollen thigh, pain, pulses and motor and sensory intact	2,150	Quadriceps	105	+	Delayed 6 days	24	Normal thigh and leg
7	32/M	Drug overdose, limb compression	Swollen buttock, obtunded, pulses intact	126,000	Gluteus maximus, gluteus medius, tensor	40 35 24	+ + +	Partial primary, delayed 7 days	48	Mild atrophy and peroneal sensory loss, no contracture, motor normal
8	34/M	Drug overdose, limb compression	Swollen buttock, pain, sensory loss, pulses and motor intact	156,000	Gluteus maximus, gluteus medius, tensor	32 40 25	+ + +	Primary	40	Normal buttock and leg
9	20/M	Drug overdose, limb compression	Swollen deltoid, forearm and thigh, obtunded, pulses intact	84,000	Deltoid, volar forearm, dorsal forearm, vastus lateralis	45 100 39 25	+ + + +	Forearm — mult. débrid. and skin graft; thigh and shoulder delayed 6 days	36	Forearm — Volkmann's contracture; normal shoulder, thigh, and leg
10	51/F	Burn	Swollen forearm, obtunded, pulses intact	—	Volar forearm, dorsal forearm	55 20	Elected not to treat	—	6	95 per cent burns; died 12 hrs. post-injury
11	18/M	Drug overdose, limb compression	Swollen deltoid, obtunded, pulses intact	60,000	Deltoid, biceps, gluteus medius, tensor	80 21 25 40	+ + + +	Partial primary — buttock, delayed 7 days	16	Normal arm and forearm; normal thigh and leg

* Normal upper limit, 130 international units.

considered to have impending compartment syndromes and were closely followed with compartmental pressure measurements every four hours if clinically indicated. Pressures of more than eight millimeters of mercury were considered to be elevated. Decompression was carried out only if the pressure rose to thirty millimeters of mercury or more. Continuous monitoring of pressures, although feasible with the wick catheter⁸, was not found to be practical because usually more than one compartment was involved in individual patients. In most cases once a high compartmental pressure measurement was obtained it was clear that the syndrome was present.

The wick catheter was used intraoperatively in eight patients undergoing decompression. In these patients intracompartmental pressures were monitored continuously throughout the procedure to show the effects of the incision of skin, fascia, and epimysium. In selected cases in which primary wound closure was desirable, pressure monitoring was used to determine the extent to which closure could be accomplished safely.

Results

The pressures in sixty-five compartments in twenty-

seven patients were measured. The three most frequent causes of increased pressure in those patients were fracture, contusion of muscles, and prolonged compression of a limb in a patient who had had an overdose of a narcotic drug. The four compartments of the leg were the most frequently involved, but there also were cases involving the forearm, arm, shoulder, thigh, and buttock⁹.

Twenty compartments in eleven patients were classified as the syndrome group because the pressures in them were thirty millimeters of mercury or more (Table I). The pressures ranged up to a maximum of 105 millimeters of mercury, the mean being forty-seven millimeters of mercury. Nineteen of the twenty compartments measured in this group were decompressed, the one exception being in a terminally ill burn patient. In the eleven patients the eight compartments in which the pressure was less than thirty millimeters of mercury also were decompressed because they were adjacent to compartments with pressures above that level (Table I).

The ages of the eleven patients in the syndrome group ranged from eighteen to seventy years old; eight of the eleven were men. The state of consciousness in six of these patients either was so obtunded or they were so un-

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cooperative that a proper history and physical examination could not be obtained. The cooperative patients had positive physical findings which always included tense compartments and pain when the involved muscles were stretched, and usually paresis and sensory loss. Except for one patient whose compartment syndrome was initiated by major arterial disease, peripheral pulses were palpable and capillary filling could always be demonstrated. Four patients initially were admitted to the hospital because they were suspected of having the syndrome. Their cases were classified as "impending" syndromes because of pressures of twenty to twenty-nine millimeters of mercury, but within twenty-four hours their compartment pressures had risen to thirty millimeters of mercury or more. The classification then was changed and the patients were assigned to the syndrome group. The rise in pressure in one patient was mediated by continuing hemorrhage into the thigh. In the other three patients the primary diagnosis was drug overdose with limb compression. Following hydration of those patients, there was increased pressure in the involved compartments.

When the proximal compartments of an extremity were involved (above the knee or above the elbow) a neural deficit was noted in only two of five patients, yet the injury to muscle was severe. Crush syndromes with renal failure developed in four of the five patients; their average serum creatine phosphokinase was 107,000 international units (normal, up to 130 international units).

Of the eleven patients in the syndrome group, follow-up was available for nine. Seven had no residual contracture or neural deficit. Two patients with compartment syndromes secondary to drug overdose with limb compression who were treated more than one and one-half days after the onset of coma had sequelae. One patient (Case 7) who was promptly treated after admission to the hospital for gluteal compartment syndrome⁹ had a mild peroneal sensory loss and atrophy in the left buttock at follow-up, but had no significant weakness or contracture. Another patient (Case 9) also was diagnosed and treated immediately after admission for compartment involvement in the right deltoid muscle, right forearm, and right vastus lateralis muscle. At the time of initial surgery, the muscles in the volar compartment of the forearm were necrotic; multiple procedures of débridement and skin-grafting were required. The shoulder and thigh healed without any residual deficit, but the patient was left with a Volkmann's contracture in the right forearm. Both of these patients had sought medical assistance more than a day and a half following the injury to the limb.

Of particular interest and importance were the sixteen patients who were suspected of having a compartment syndrome but whose compartment pressures remained less than thirty millimeters of mercury. None of these patients had a fasciotomy and no sequelae developed.

In eight patients undergoing fasciotomy, the wick catheter technique was also used intraoperatively. With incision of the skin the initial intracompartmental pressure decreased by an average of nine millimeters of mercury

(Fig. 1). Fasciotomy then produced a large drop in pressure (average, thirty-nine millimeters of mercury), following which the pressures in all involved compartments were in the normal range of zero to eight millimeters of mercury. Epimysiotomy in the forearm and leg then had little effect, the average decrease being only three millimeters of mercury. However, in the deltoid and gluteus maximus muscle compartments, where the fascia and epimysium blend, a decrease to normal levels occurred only after multiple incisions were made in the epimysium of the muscles.

In three of the preceding eight patients, we closed the skin while monitoring the intracompartmental pressure (Table I). Closure was discontinued if the pressure reached ten millimeters of mercury. This technique was particularly valuable in wounds located in an area likely to be contaminated, such as the buttock. In all wounds that were left open primarily, delayed primary closure was accomplished five to seven days after the fasciotomy; only one of our patients (Case 9) required any skin-grafting.

Discussion

Pathophysiology of a Compartment Syndrome

A compartment syndrome is due to increased tissue pressure in a closed fascial space (compartment) compromising the circulation to the nerves and muscles within the involved compartment. As noted in this study, the four compartments of the leg were the most frequently involved, but we also saw patients with involvement in other sites. The syndrome can be caused by a fracture, severe contusion, drug overdose with limb compression, a burn, or vigorous exercise. The initial insult causes hemorrhage, edema, or both in the closed fascial compartments of the extremities. Then there is an increase in intracompartmental fluid pressure, causing ischemia. Damage to the contents of the compartment, if permanent, may result in a Volkmann's contracture. To prevent this complication, a prompt diagnosis must be made and decompression of the involved compartments must be done quickly if the pressure in them is high.

Physical Findings in Compartment Syndromes

Swelling and palpable tenseness over a muscle compartment are the first signs of a compartment syndrome and are manifestations of increased pressure within the compartment. However, these signs are only crude indications of increased intracompartmental pressure, and other physical findings must also be sought. Pain with stretch of the muscles involved is a common finding, but it is quite subjective and unreliable because the sign may be engendered by the initial direct trauma rather than by ischemia. Furthermore, later in the course of the disease pain on stretch may be absent because there may be anesthesia secondary to ischemia of the nerve. Paresis also is a difficult sign to interpret because it may arise secondary either to neural involvement or to primary ischemia of muscle, or there may be a guarding, secondary to the pain, that simulates paresis. In our experience the most reliable

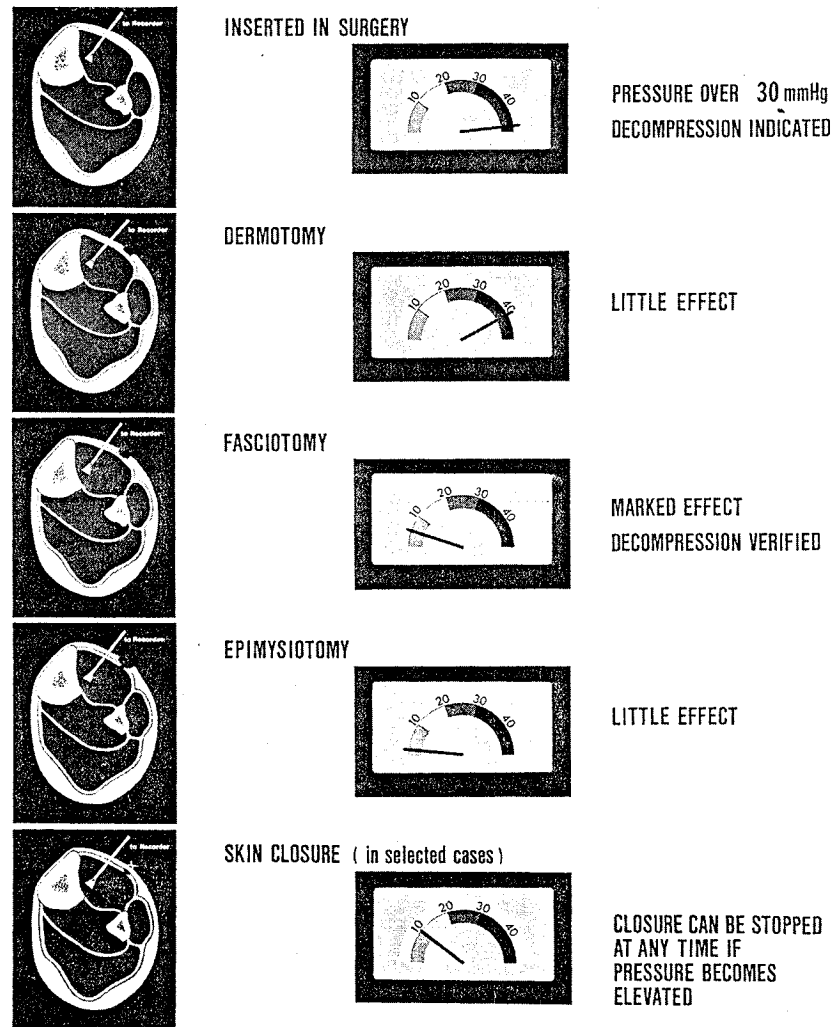


FIG. 1

Intraoperative use of the wick catheter in patients undergoing decompression, demonstrating the effects of dermotomy, fasciotomy, and epimysiotomy. Fasciotomy consistently restored pressures to normal levels. Epimysiotomy was performed in order to demonstrate its value in the decompression. It had a negligible effect except in the gluteus maximus and deltoid muscle compartments.

physical finding is a sensory deficit. Although it appears early in a compartment syndrome, it may be manifest only as a paresthesia. Significant delay in treatment may make anesthesia inevitable. Each compartment of the leg and forearm has at least one nerve coursing through it that has sensory fibers, and, with careful physical examination, selective confirmation of the compartment involved will be possible because of the sensory deficit in the area of nerve supply appropriate to the involved nerve.

Except in the presence of major arterial injury or disease, peripheral pulses are palpable and capillary filling is demonstrable routinely, even though a compartment syndrome is present. Although it may be high enough to cause ischemia of muscle and nerve, elevation of intracompartmental pressure only very rarely is high enough to occlude a major artery. Failure to appreciate this fact may lead to a false sense of security if the surgeon, palpating the pulse distally, decides that all is well.

Increased pressure more proximally, as in the arm, thigh, deltoid, or gluteal muscle compartments, makes the interpretation of the physical findings more difficult. Sen-

sory deficit in such cases is unusual because the nerves are not entirely confined in the compartments. Frequently the only findings are swelling and tenseness. An additional reason for difficulty in diagnosis when the lesion is proximal is that the patients most commonly involved are those with drug overdose-limb compression sequelae, whose consciousness often is obtunded and who therefore are unable to respond appropriately to examinations that depend on subjective responses.

The Wick Catheter Technique in Diagnosis

Most compartment syndromes can be diagnosed clinically. Then documentation of the elevated compartment pressure may only be confirmatory. However, there are three groups of patients in whom difficulties in eliciting or interpreting the physical findings are encountered. In them, the measurement of intracompartmental pressure is particularly valuable as a criterion for whether decompression should be done.

Uncooperative or unreliable patients: A young child who has sustained a fracture at the elbow, forearm, or leg.

These children may be so frightened that a careful motor and sensory evaluation is not possible. Movement of their digits may be followed by painful cries that defy accurate interpretation. Thus, without an objective criterion on which to act, the surgeon must maintain a careful vigil until the signs of a compartment syndrome become unequivocal.

Unresponsive patients: Particularly those with a diagnosis of drug overdose-limb compression syndrome. In our experience, this is a common cause of a compartment syndrome. Four of the five patients who had this diagnosis were obtunded or comatose on admission. This group included the only two patients in this series who had a residual functional deficit. Our results represent a considerable improvement over the results of treatment rendered to this type of patient before the wick catheter technique was available⁷. The severity of the residual impairment in our patients was attributable to delay in hospitalization, diagnosis, and treatment. The improvement in diagnostic assessment has played some part in the improvement in the care of patients recently, but increased interest in the problems and increased awareness by physicians of the danger of this syndrome and of the need for prompt diagnosis and treatment may have also been a factor in improving results.

Patients with fractures or contusions and nerve deficit: In whom it is difficult to differentiate whether the deficit is caused by direct trauma or is secondary to a compartment syndrome. In our series, the patients who had compartmental pressures of more than thirty millimeters of mercury (mean, thirty-four millimeters of mercury) easily could be separated from the seven patients who had neurapraxia (mean, twelve millimeters of mercury). The neurapraxia group did not have surgery and recovered fully without contractures within twelve weeks. The three patients diagnosed as having compartment syndromes attained full return of function in the first week following immediate fasciotomy.

Whitesides and associates used the needle manometer technique and Matsen and colleagues used an infusion

technique to determine intracompartmental pressures in patients with suspected compartmental syndromes. They selected, as the critical pressure level, either the diastolic blood pressure minus twenty millimeters of mercury¹³, or forty to fifty millimeters of mercury⁶; that is, levels higher than thirty millimeters of mercury, which we selected as the lowest level at which decompression is indicated. Some of our patients with pressures in the range of thirty to forty millimeters of mercury might well have recovered without a fasciotomy. There is no single correct pressure for all individuals. A spectrum of critical pressures exists depending on many variables, including the measurement technique used. Analysis of Starling's forces suggests that when the tissue-fluid pressure exceeds thirty millimeters of mercury, the capillary pressure is not sufficient to maintain muscle-capillary blood flow⁵. We believe therefore that it is prudent to use a value close to the capillary blood pressure (twenty to twenty-five millimeters of mercury) as a criterion for decompression and to do the surgical decompression before the muscle and nerve are injured by ischemia. Perhaps increased experience with compartmental pressure measurement may allow us to delay treatment in selected patients in order to define the critical pressure level better.

The Wick Catheter Technique in Treatment

The wick catheter was used continuously intraoperatively in eight patients undergoing fasciotomy in order to document the effectiveness of the surgery. Reports that the incision of skin³ and epimysiotomy¹ are important adjuncts to adequate decompression in the compartment syndrome have not been confirmed by the pressure measurements in our patients. Neither procedure significantly decreased the intracompartmental pressure except in special cases in which the deltoid and gluteal muscles were involved. Adequate and complete fasciotomy was the key. It consistently restored pressures to normal levels.

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