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Invited Review

Postoperative rehabilitation of compartment syndrome following fasciotomy

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ABSTRACT

Compartment syndrome can be defined as an increase in pressure in an anatomical compartment in the body resulting in muscle and nerve damage. Acute compartment syndrome mostly develops after crushing traumas and fractures to the extremities and is a condition that requires urgent intervention. Fasciotomy of the affected extremity segment in the early stage of the compartment syndrome is the gold standard in treatment of crush injury. Rehabilitation after fasciotomy is extremely important in regaining extremity functions. In this review, we discuss current approaches to compartment syndrome and rehabilitation principles following fasciotomy.

Keywords: Compartment syndrome, fasciotomy, rehabilitation.

Compartment is a closed area of muscles groups, nerves, and vessels surrounded by a fascia.[1] The flexibility of the fascia is limited. Although it varies according to the region, the average intra-tissue pressure ranges between 5 and 15 mmHg.[2] Compartment syndrome (CS) is an increase in the tissue pressure within a myofascial compartment that exceeds the resting pressure of the capillary system.[3] Increased pressure impedes local muscle blood flow, thereby impairing neuromuscular function of tissues within the specific compartment.[4] It was first described by Richard von Volkmann, a German physician, in 1881 as a permanent contracture of the forearm due to ischemia in the muscle compartments.[5] Although CS is rarely seen in the trunk, the extremities are much more affected. It can be acute or chronic. Chronic exertional CS is a rare diagnosis among athletes. The typical presentation is seen in running athletes who have recurrent anterior, posterior, or lateral leg pain with repetitive physical activity and alleviation of symptoms with rest.[6] On the other hand, acute CS is a medical emergency that should be treated promptly to prevent irreversible damage.[7]

ANATOMY

There are two compartments in the upper arm. The anterior compartment contains biceps, brachial muscle and ulnar, median and radial nerves. The posterior compartment has the triceps muscle. In the forearm, there is the dorsal compartment containing the wrist and finger extensors, and the volar compartment containing the flexors of the hand and fingers. In the hand, there are 10 compartments: dorsal interossei (4 compartments), palmar interossei (3 compartments), adductor pollicis, thenar, and hypothenar.[8] The lower leg is comprised of four compartments: anterior, lateral, superficial posterior, and deep posterior.[9]

ETIOPATHOGENESIS

Any event, external or internal, that increases the pressure within a compartment can cause CS. It most often occurs following a fracture or a crush injury to the extremity.[10] Accumulation of blood and other tissue fluids after tissue injury creates a mass in the myofascial compartment. Due to the non-elastic nature

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of muscle fascia, this accumulation of mass leads to an increased pressure within the compartment, which is transmitted to the thin-walled venous system, causing venous hypertension and further transudation of fluid. Increased pressure in the compartment causes collapse of vascular structures, impairing local perfusion and causing direct compression of muscles and nerves. This leads to transcellular pump failure and, ultimately, tissue ischemia.[10] The pressure increase can be acute or chronic. Common causes of CS are given in Table 1.

CLINICAL PRESENTATION

The initial complaint is often increasing pain and pressure disproportionate to the initiating event. The pain is accompanied by a feeling of pressure and stiffness and increases, when the compartment is stretched actively or passively. Paresthesia, hypoesthesia, and paralysis may develop over time. Although there are classic seven P's (i.e., pain, pressure, pain with passive stretch, pallor, pulselessness, paresthesia, and paralysis) in patients, some of these findings may occur late and lead to a delay in diagnosis. Ischemia may be at the capillary level and pulses are normal, but pallor may not be evident. The earliest sign may be burning pain and pain with passive stretching of the muscle. Pain and aggravation of pain by passive stretching of the muscles in the compartment are the most sensitive (and usually the only) clinical findings, before the onset of ischemic dysfunction in the nerves and muscles.[11] Paresthesia may be present at this time. The time to develop drop foot in the lower extremity may vary, and pain with passive hyperextension of the toes should be considered as an early finding. In chronic CS, patients complain of pain and cramps which increase with exercise, but these complaints usually decrease with rest.

DIAGNOSIS

The key to rapid diagnosis is to recognize clinical symptoms and clues in the patient's medical history. Another important factor is to have a high index of suspicion in case of trauma. If the diagnosis is clinically evident, it is not necessary to measure the compartment pressures.[4] In cases where the diagnosis is uncertain or the patient is unconscious, the measurement of compartment pressure is the gold standard for surgical decision. Currently, a solid-state transducer intracompartmental catheter (Stryker STIC) is used to measure compartment pressure.[12] Although there is no consensus on the critical level of intracompartmental pressure, the general opinion is about 30 to 50 mmHg.[13] However, experimental studies have shown that there are differences between individuals in terms of exposure to ischemia and oxygen saturation that would result in tissue damage. Another proposed method is to use the delta pressure (ΔP) obtained by subtracting the measured compartment pressure from the patient's diastolic pressure. A ΔP of less than 30 mmHg indicates critical CS.

Mühlbacher et al.,[14] in their cadaveric study, suggested that the intracompartmental pressure could be estimated with ultrasound using the tibiofascial angle in the anterior compartment; however, the findings are too preliminary to be used in clinical practice currently.

Similarly, although it has been suggested by some researchers that magnetic resonance imaging may aid in diagnosis, it is currently of secondary importance and can be used in confusing cases and for estimating surgical margins.[15] Laser Doppler flowmetry and scintigraphy have only been evaluated in chronic CS to date.

TABLE 1
Common causes of compartment syndrome

Increased compartment contents		Decreased compartment volume
Trauma	Fractures (tibia & forearm most common) Crush Injury	Tight casts Lying on limb
Vascular	Hemorrhage (vascular puncture in patients, postoperative bleeding etc.)	
Muscle overuse (increased capillary filtration & pressure)	Excessive exercise Seizures Tetany	
Burns	Thermal & electrical burns-can increase compartment contents and decrease compartment volume (circumferential eschars)	

TREATMENT

Immediately all circumferential dressings must be removed, normal blood pressure should be achieved to manage any cause of hypotension. The extremity should not be elevated, but kept at heart level, to maintain perfusion in the compartment. Supplementary oxygen, to improve the tissue oxygenation, is helpful.[4] Tissue pressure at rest over 30 mmHg or ΔP is below 30 mmHg requires urgent surgical intervention (fasciotomy). The changes that occur in the muscles within 3 to 4 h are reversible. While various damages occur in 6 h, they become irreversible after 8 h. Compression on the nerves for more than 2 h leads to loss of nerve conduction, 4 h to neuropraxia, and after 8 h irreversible changes occur. If the fasciotomy is performed within the first 6 h, complete recovery is most likely. Prognosis worsens over time. Permanent motor impairment, contracture, crush syndrome (acute renal failure with myoglobinuria, shock, and cardiac complications), and even death may occur.

Fasciotomy of the affected extremity segment in the early stage of the CS is the gold standard in treatment of crush injury.[16] It must, however, be kept in mind that the correct indication and timing of fasciotomy can be only decided on the basis of a thorough examination of the patient and the affected body parts and consultation with a surgical team experienced in traumatology.

Surgery

Fasciotomy, by definition, is the surgical incision of the skin and the underlying deep fascia of an extremity. There are several important aspects of this relatively simple intervention.

First of all, the fasciotomy incision must extend to the full length of the affected segment in a longitudinal fashion. To illustrate, effective decompression of the forearm can be only achieved by starting slightly above the cubital area and ending in the palmar area distal to the carpal tunnel. Such an extended exposure allows thorough exploration of not only the crushed muscles, but also the major arteries and nerves, which may not infrequently be injured in this type of trauma. Immediate repair of the thrombosed arteries is crucial to prevent persistent ischemia and recurrence of the CS.[17-19] Special attention must be directed to the anatomical constriction points in the course of major nerves, which may easily become compressed in the crush process. Complete opening of the carpal tunnel is a good example of preemptive decompression of such nerves; the median nerve in that case.[19]

All severely injured muscle parts must be detected carefully and debrided during the fasciotomy, otherwise, ischemic muscle segments would continue to secrete myoglobin, potassium, and other toxic compounds, maintaining the vicious cycle of ischemia-CS. Indeed, it is now well understood that previous negative reports in the literature on early fasciotomy in CS can be attributed to less effective debridement of the non-viable muscle tissue rather than the decompression itself.[20] Surgical re-exploration of the wound and additional debridement should be always considered in the cases where expected recovery of the patient or the extremity in question is not observed.

Fasciotomy wound management

In most cases, it is neither possible nor advisable to perform primary closure of the fasciotomy incision due to the persisting edema in the extremity. The open wound created by fasciotomy can easily be managed with strict sterility and wound care protocol in the ward. The vacuum-assisted closure (VAC) systems can be employed in the prolonged open wounds.[21] Most fasciotomy wounds eventually become smaller in several weeks and a skin graft applied over the healthy granulation tissue of the wound finalizes the repair phase.

REHABILITATION AFTER FASCIOTOMY

Rehabilitation steps after fasciotomy include PRICE, (P [Protection], R [Resting], I [Ice] C [Compression] E [Elevation]) restoration of range of motion (ROM) of the joint, mobilization of soft tissues, neurodynamic mobilization, stretching, and strengthening.[22]

PRICE is required in the proliferation phase. It takes about three to 20 days. During this period, the tensile strength may decrease to 15% of the normal tissue, and it is essential to protect it.[23] Fasciotomy can lead to severe edema and hematoma and, therefore, elevation is very important. Elevation and compression can reduce fluid volume in the affected extremity, reducing the amount of granulation tissue formed in the injured compartment.[24] Reducing tissue swelling with elevation also reduces postoperative pain. Cold applications also help to reduce edema and relieve pain.[25] However, it should be kept in mind that the extremity may have sensory impairment and ice burns should be avoided.

Range of motion and soft tissue mobility

In the proliferation phase, a large amount of collagen, edema, and intense stress to the healing tissue cause more inflammation and collagen deposition, resulting in dense scar tissue, which adversely affects functional results. Wound contraction usually begins on Day 5 and peaks around Week 2. The maturation phase begins after approximately Day 9 and is the longest phase. Factors such as the orientation of the collagen fibers in the maturation phase and the synthesis/lysis balance of collagen may play a role in determining the structure of the final scar tissue.[26] Collagen in scar tissue is less organized than in uninjured tissue. Internal and external stress determines the development of final scar tissue. Joint movement, muscle tension, fascial gliding, and mobilization have a positive effect. Postoperative active and passive ROM is important to develop a well-organized scar tissue. Prolonged immobilization may cause limitation of motion in the adjacent joints, as well as the joints in the affected area. Therefore, active and passive ROM of adjacent joints should be started as early as possible.

Sensory re-education

There may be short- or long-term hypersensitization due to stretching of the injured tissue. Desensitization approaches can reduce fear and anxiety of touching the scar. It starts with soft and light fabrications and, then, continues with stiffer, thicker materials. Care should be taken not to damage the injured skin.

Stretching

It is performed to obtain an antifibrotic effect and to prevent muscle shortening after surgery. The intensity, frequency, timing, and duration of stretching are critical to obtain anti-fibrotic effects. Scar tissue responds best to long-term low-intensity loads.[23]

Neurodynamic mobilizations

Inflammation and scar formation can cause decreased nerve movement within the tissue. Neurodynamic intervention may be beneficial after surgical decompression.

Strengthening

Weakness in the muscles in the affected area after fasciotomy is an expected situation. The main goal of the strengthening programs is to return the muscles to their former condition. As the intramuscular pressure at rest is elevated following eccentric muscular activity compared to concentric muscular activity, eccentric muscle strengthening should be initially avoided postoperatively.

The program, which includes the basic rehabilitation approaches mentioned above, can be carried out according to the following phases:[22]

Phase I: 2 to 3 weeks following surgery: Protection and mobility

Phase II: 3 to 4 weeks postoperatively: Light strengthening

Phase III: 4 to 6 weeks following surgery: Progression of strengthening

Phase IV: 8 to 12 weeks following surgery: Activities of daily living/return to sports

Below is the rehabilitation guideline after fasciotomy of lower extremity developed by the University of Wisconsin-Madison Health Sports Rehabilitation and the UW Health Sports Medicine Physician group for lower extremity.[27]

Phase I: Goals in this period are to protect the postoperative compartment, minimizing postoperative swelling, maintain/restore knee, ankle ROM, ability to raise the involved leg painlessly in all directions while standing, controlling the leg in the open and closed kinetic chain during walking, pain-free walking with full weight and equal stride length without assistive devices.

Precautions: Avoiding activities that increase swelling (e.g., sitting for long periods of time, tight clothing proximal to the surgical area, hot pack or bath), friction to the new scar tissue (e.g., crossing legs, tight clothing, scar massage, or friction), and activities such as running and jumping. It is recommended not to bear weight (using crutches suggested) while walking for the first two weeks, and pain score should be less than 2 according to the Visual Analog Scale (VAS) (0-10) while walking with partial weight.

Exercise: Quadriceps sets for isometric strengthening and active ankle and knee ROM exercises are initiated with 10 reps in each direction 1 to 2 times a day, and the number of repetitions is increased as tolerated. At post-operative Week 3, open kinetic chain strengthening exercises with TheraBand (1-2 level of resistance) may be started 10 reps in each direction, 1 to 2 sets daily. Hip strengthening exercises (flexion/abduction/extension) are started in supine position, lying on side, continued in prone 1 set of 10 reps in each direction 1 to 2 times daily and increased as tolerated. Muscle pumping exercises

are started at the ankle joint, while lower extremity is elevated to assist with venous return and swelling. Cardiovascular (CV) exercise using arm ergometer (begin with 5 to 10 min, 1 to 2 times daily, and progress) is also recommended.

Phase II: Goals in this period are as follows: the circumference difference between the two extremities is less than 1 cm, good healing of the incision site, minimal muscle atrophy and flexibility deficit, ability to stand on one leg for 30 to 60 sec on an unstable surface, full flexibility in the gastrocnemius muscle, maintaining strength and CV endurance in unaffected muscles, starting active and light resistance exercises on the operated extremity, painless and adequate lower extremity control during squats, and device-free and non-antalgic equal stride gait.

Precautions: Similar to Phase I, avoidance of excessive pressure and friction on the new scar tissue, prolonged standing, and eccentric loading during activities.

Exercises: Scar massage/mobility and desensitization (3 to 5 min 12 times daily), gentle stretching (30 to 60 sec 2 to 3 times daily) and nerve mobilization in the affected compartment, and lower extremity straight leg raise in supine position (peroneal nerve is stretched with ankle plantar flexion and inversion). Open kinetic chain and strengthening exercises are continued. Number of sets/repetitions and resistance increase according to tolerance. Balance and proprioception exercises are started. First bilaterally, then unilaterally on hard ground-soft ground, and then on balance board (eyes open-closed 30 to 60 sec; 2 to 3 reps, 1 to 2 times/day). The CV endurance exercises are continued. If the wound has healed, stationary bike or treadmill walking may begin (starting with low resistance 5 to 10 min daily) and progressively increased by tolerance.

Phase III: Goals in this period are to prevent recurrence of postoperative symptoms during activities, ability to complete 15 to 30 min of uninterrupted aerobic activity without symptoms, full and painless ankle muscle strength with manual muscle testing in the affected compartment, appropriate lower extremity control and alignment, and painless single leg functional movements including squats and lunges.

Precautions: No stretching until the wound is fully healed, no running until postoperative Week 6 to 8, and avoiding painful strenuous activities.

Exercise: Continue lower extremity stretching and nerve mobilization. Closed chain functional strengthening including lunges, step-backs and single leg squats. Plyometric exercises begin at Week 6.

Phase IV: Goals are to return to work/sports without pain, instability or swelling, adequate dynamic neuromuscular control and alignment with eccentric and concentric multi-plane activities.

Precautions: Avoiding pain with any exertional activity and post-activity swelling.

Exercise: For specific sports activities in addition to exercises in previous phases.

The main phases of the above rehabilitation program recommended for the lower extremity also apply to the upper extremity.[28] In the early postoperative period, it is of utmost importance to keep the hand in the functional position, as well as in elevation. The collateral ligaments of the metacarpophalangeal (MCP) joints are elongated when the joint is flexed. The MCP joints should be positioned in at least 60 degrees of flexion. The collateral ligaments of proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints are elongated in extension and should be held in this position.[28]

Volkmann's ischemic contracture (VIC) is an important complication that can develop after upper extremity CS.[29] Fibrotic proliferation after muscle injury causes cicatricial shortening, extensive fasciocutaneous and musculotendinous adhesions, and ultimately a dysfunctional contracture. According to the severity of the impact, it can be classified as follows:[30]

Mild Volkmann's ischemic contracture: Limited area and segment is affected. There is partial flexor digitorum profundus (FDP) damage and minimal sensory disturbance.

Moderate Volkmann's ischemic contracture (classic type): Involvement is near-total in FDP and flexor pollicis longus (FPL) and partial in flexor digitorum superficialis (FDS) and other superficial muscles. There is a marked disturbance of sensation and typical clawing of the fingers.

Severe Volkmann's ischemic contracture: All flexors and even extensors are damaged. There is sensory and motor loss that may be irreversible and a high probability of amputation.

Treatment of Volkmann's ischemic contracture: In cases with mild contracture, muscle stretching exercises

and splinting the affected fingers may be sufficient; however, a surgical intervention (i.e., debridement, tendon lengthening, muscle sliding) is not infrequently necessary to obtain functional recovery.

For moderate contracture, surgery is the only modality to correct the contracture by removing muscles, tendons, and decompressing nerves that are thickened, scarred, or dead. Intact muscles are advanced distally and/or shortened tendons are lengthened to obtain free musculotendinous excursion. In severe contractures, following extensive debridement of the non-viable muscle tissue there is usually not enough muscle tissue left and either free muscle transfer or amputation of the affected part is necessary.

In conclusion, ACS is a condition that requires careful follow-up and emergency surgical intervention in most cases. There is a standard postoperative rehabilitation program in the literature. However, the protocol described in the current study can be applied after fasciotomy in ACS. There is also a lack of data in the literature on postoperative rehabilitation outcomes. Further studies on this subject may contribute to the development of better programs in the future.

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REFERENCES

1. McLaughlin N, Heard H, Kelham S. Acute and chronic compartment syndromes: Know when to act fast. JAAPA 2014;27:23-6. doi: 10.1097/01.JAA.0000446999.10176.13.
2. Williams PR, Russell ID, Mintowt-Czyz WJ. Compartment pressure monitoring--current UK orthopaedic practice. Injury 1998;29:229-32. doi: 10.1016/s0020-1383(97)00193-9.
3. Matsen FA 3rd, Winquist RA, Krugmire RB Jr. Diagnosis and management of compartmental syndromes. J Bone Joint Surg [Am] 1980;62:286-91.
4. Köstler W, Strohm PC, Südkamp NP. Acute compartment syndrome of the limb. Injury 2004;35:1221-7. doi: 10.1016/j.injury.2004.04.009.
5. Volkmann R. Ischemic muscle paralysis and contractures. Zentralbl Chir 1881;8:801-3.
6. Vajapey S, Miller TL. Evaluation, diagnosis, and treatment of chronic exertional compartment syndrome: A review of current literature. Phys Sportsmed 2017;45:391-8. doi: 10.1080/00913847.2017.1384289.
7. Schmidt AH. Acute compartment syndrome. Injury 2017;48 Suppl 1:S22-5. doi: 10.1016/j.injury.2017.04.024.
8. Miranda-Klein J, Howell CM, Davis-Cheshire M. Recognizing and managing upper extremity compartment syndrome. JAAPA 2020;33:15-20. doi: 10.1097/01.JAA.0000660124.51074.e5.
9. Frink M, Hildebrand F, Krettek C, Brand J, Hankemeier S. Compartment syndrome of the lower leg and foot. Clin Orthop Relat Res 2010;468:940-50. doi: 10.1007/s11999-009-0891-x.
10. Schmidt AH. Acute compartment syndrome. Orthop Clin North Am 2016;47:517-25. doi: 10.1016/j.ocl.2016.02.001.
11. Whitesides TE, Heckman MM. Acute compartment syndrome: Update on diagnosis and treatment. J Am Acad Orthop Surg 1996;4:209-18. doi: 10.5435/00124635-199607000-00005.
12. Collinge C, Kuper M. Comparison of three methods for measuring intracompartmental pressure in injured limbs of trauma patients. J Orthop Trauma 2010;24:364-8. doi: 10.1097/BOT.0b013e3181cb5866.
13. Allen MJ, Stirling AJ, Crawshaw CV, Barnes MR. Intracompartmental pressure monitoring of leg injuries. An aid to management. J Bone Joint Surg Br 1985;67:53-7. doi: 10.1302/0301-620X.67B1.3968144.
14. Mühlbacher J, Pauzenberger R, Asenbaum U, Gauster T, Kapral S, Herkner H, et al. Feasibility of ultrasound measurement in a human model of acute compartment syndrome. World J Emerg Surg 2019;14:4. doi: 10.1186/s13017-019-0222-9.
15. Corrêa DG, Costa FM, Mendonça S, Severo A, Canella C. Magnetic resonance imaging alterations in acute compartment syndrome. J Foot Ankle Surg 2022;61:1134-5. doi: 10.1053/j.jfas.2019.10.006.
16. Williams AB, Luchette FA, Papaconstantinou HT, Lim E, Hurst JM, Johannigman JA, et al. The effect of early versus late fasciotomy in the management of extremity trauma. Surgery 1997;122:861-6. doi: 10.1016/s0039-6060(97)90099-4.
17. Reis ND, Better OS. Mechanical muscle-crush injury and acute muscle-crush compartment syndrome: With special reference to earthquake casualties. J Bone Joint Surg [Br] 2005;87:450-3. doi: 10.1302/0301-620X.87B4.15334.
18. Reis ND, Michaelson M. Crush injury to the lower limbs. Treatment of the local injury. J Bone Joint Surg [Am] 1986;68:414-8.
19. Botte MJ, Gelberman RH. Acute compartment syndrome of the forearm. Hand Clin 1998;14:391-403.
20. Farber A, Tan TW, Hamburg NM, Kalish JA, Joglar F, Onigman T, et al. Early fasciotomy in patients with extremity vascular injury is associated with decreased risk of adverse limb outcomes: A review of the National Trauma Data Bank. Injury 2012;43:1486-91. doi: 10.1016/j.injury.2011.06.006.
21. Zannis J, Angobaldo J, Marks M, DeFranzo A, David L, Molnar J, et al. Comparison of fasciotomy wound closures using traditional dressing changes and the vacuum-assisted closure device. Ann Plast Surg 2009;62:407-9. doi: 10.1097/SAP.0b013e3181881b29.

22. Schubert AG. Exertional compartment syndrome: Review of the literature and proposed rehabilitation guidelines following surgical release. Int J Sports Phys Ther 2011;6:126-41.
23. Cameron MH. Inflammation and tissue repair. In: Cameron MH, editor. Physical Agents in Rehabilitation: From Research to Practice. 2nd ed. St. Louis: Elsevier Science; 2003. p. 23-4.
24. Tsang KK, Hertel J, Denegar CR. Volume decreases after elevation and intermittent compression of postacute ankle sprains are negated by gravity-dependent positioning. J Athl Train 2003;38:320-4.
25. Block JE. Cold and compression in the management of musculoskeletal injuries and orthopedic operative procedures: A narrative review. Open Access J Sports Med 2010;1:105-13. doi: 10.2147/oajsm.s11102.
26. Wang PH, Huang BS, Horng HC, Yeh CC, Chen YJ. Wound healing. J Chin Med Assoc 2018;81:94-101. doi: 10.1016/j.jcma.2017.11.002.
27. Rehabilitation guidelines following compartment syndrome release with open fasciotomy. Available at: https://bynder.uwhealth.org/m/9e93f2ab2f20fb9f/original/Rehab-Guideline-CECS.pdf%2027
28. Miranda-Klein J, Howell CM, Davis-Cheshire M. Recognizing and managing upper extremity compartment syndrome. JAAPA 2020;33:15-20. doi: 10.1097/01.JAA.0000660124.51074.e5.
29. Stevanovic MV, Sharpe F. Compartment syndrome and Volkmann ischemic contracture. In: Wolfe SW, Pederson, Hotchkiss RN, Kozin SH, Peterson WC, Cohen MS, editors. Green's textbook. 7th ed. Philadelphia: Elsevier; 2017. p. 1763-87.
30. Tsuge K. Treatment of established Volkmann's contracture. J Bone Joint Surg [Am] 1975;57:925-9.