5-1-1937

Volkmann's ischemic contracture

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VOLKMANN'S ISCHEMIC CONTRACTURE

by

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Senior Thesis
Presented to the College of Medicine
University of Nebraska
Omaha, 1937
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INTRODUCTION and DEFINITION
In selecting a subject for a senior thesis it seems logical that the choice should fall on one pertaining to that field of medicine in which the originator is particularly interested. In addition the subject should be one whose every aspect may be adjudged. Hence, he learns about that specific condition and acquaints himself with the men outstanding in that phase. The subject Volkmann's Ischemic Contracture meets these requirements in my particular case. The useless "claw hand" known as Volkmann's Contracture, or Ischemic Paralysis, is one of the most unfortunate sequelae of fractures or traumatisms of the forearm.

The term is self-explanatory but as a means of removing any possible confusion, I am offering a definition. Prince (40) defines it as follows:

"Contracture is a condition into which a muscle passes following spasm to produce a rigid state maintained over a period of time; a decrease in the length of a muscle, which is not merely a passing state, that will disappear when the stimulus ceases."

In this state of extreme and permanent contraction, the muscle has degenerated from its function of producing motion to that of support or holding parts in position, which is the function of ligaments, and the condition of contracture.
The word ischemia means to hold back blood; a local and temporary deficiency of blood chiefly due to the contraction of a blood vessel.

Jones (27) gives us the following definition of the condition as a whole. The specific contracture, namely, Volkmann's Ischemic Contracture, or Ischemic Myositis is a deformity found mainly in children between one and fourteen years of age, the majority between six and eleven. It follows injury to the elbow and is due to pressure from within or pressure from without, or both. It is usually, but not always, accompanied with fracture.

In a typical case the wrist joint is palmar flexed, the carpo-metacarpal bones are dorsiflexed, the interphalangeals flexed, the hand is often pronated and the elbow joint flexed. As a rule, sensation is normal in the milder cases and the electric reactions are unaltered. On flexing the wrist the fingers can be straightened. There is lowered temperature in the hand and arm. The skin is blue and sometimes blistered and scars are frequently present. The muscles rapidly waste and become hard and ropy. If the nerve is sufficiently involved whole or partial loss of conduction may result.
HISTORY
In tracing the history of Volkmann's Ischemic Contracture it is interesting to note that since Volkmann's and Leser's presentation of the condition little change has been made in any phase other than treatment. The etiology, physiology, pathology and symptoms presented in articles of the past two decades merely substantiate the original work.

Keith (31) in his book "Menders of the Maimed" offers us an adequate history of the man who first described this condition. Our knowledge concerning this "ischemic" disaster which sometimes overtakes muscles dates back to Halle, during the years which followed the Franco-Prussian War. Richard von Volkmann, who served as consulting surgeon to several armies during that war, had then returned to his professional chair, a man of forty one.

As an orthopedic surgeon, he was particularly interested in the surgical disorders of muscle. He noted that in cases of fracture of the forearm or of excision of the elbow, a distinctive form of contracture of the muscles of the forearm was apt to occur.

Sever (43) offers the following information in the first work done by Volkmann. In 1869 Volkmann first described the clinical picture of a contracted wrist and hand following fracture, with atrophy of the forearm.
His first case however occurred in an acute synovitis of the knee following application of a ham splint. This led him to believe the causative factor was too tight bandaging, following fracture. He states that the paralysis and contractures usually come on together while paralysis, due to nerve pressure, comes on gradually.

Thomas (50) and Bernays (3) point out that Volkmann later published a revised edition of the book published in 1869, in 1875, in which he described ischemic paralysis. In addition Hildebrand quotes a case of Hamiltons in 1850, the original article or book was not given. The credit of calling general attention to the condition belongs to Leser. The affection is often referred to as the Volkmann-Leser Contracture. Since Leser's article in 1884 the pathology has been fairly well understood. There has been, however, considerable dispute over the mechanisms by which the process occurs.

Volkmann's description of the clinical picture was an excellent paper. The treatment, however, has been modified in the past forty years as would be expected. The second article published by Volkmann in 1881 emphasized certain points. These points are discussed in this paper and are the essential points in all of the articles read and will be found under nine headings in
the summary.

Brooks (6) who has done much work on this condition offers an excellent review of the pathology, both early and late, seen in the muscle fibre of dogs. His work is the latest done on this phase of contracture and was published in 1922.

Jepson (24) has also published an article in 1926 which confirms Brook's work.
INCIDENCE
Lewis (33) points out that the ischemic palsy is much more common than it is generally considered to be. It is certainly more common than one would be led to believe by the cases reported in the literature. In many instances the change is so slight recovery occurs without any residue. In many cases the severe degrees of palsy are not thought to be of sufficient interest to report.

Thomas (50) suggests that the lack in available case presentations is possibly due to the widespread belief that the condition is due to improper application of splints, as indeed is sometimes the case. For instance, Ward states he has seen twenty five cases in twenty five years but reports only two of them. Bernays thinks the condition frequent. Rowlands speaks of having seven cases and knowing of four more unreported cases. There are other men who state they see but one case in several years. The wide difference depends largely on the experience of the surgeon.

Stone (47) apparently believes the condition is seen more frequently in children possibly because they are submitted to more pain than adults. Adults will remove too tight bandages or complain in a more comprehensive manner.

Driver (12) gives the report of Dr. Frank P.
Stricklet, Louisville, Ky., who states that in his experience at the Kentucky Crippled Children's Commission with this type of case, he has seen ten such cases in the past year.

Pusitz (41) gives Meyerding's reports from 1910 to 1927 of one hundred and twenty eight cases of Volkmann Ischemia entered the Mayo Clinic. The number of Volkmann Ischemias is apparently on the increase since his report in 1881. This is undoubtedly due to the increased frequency of diagnosis and the knowledge of the condition being disseminated throughout the profession.

Meyerding (38) offers the following figures as to incidence in sex and age factor. Of one hundred and twenty eight cases of ischemic contracture, seventy percent were males, thirty percent females, sixty seven percent were in patients less than thirty years of age, thirty three percent in patients more than thirty years of age.

From this report we see that the disorder is seen more commonly in young males. The answer to the sex difference is possibly due to the fact that young males are exposed to greater physical exercise and chances for upper extremity injury.

Cravener (10) in citing the possibility of ischemic
contracture entities in medicine have become inseparable; interstitial keratitis and lues, quinine and malaria, liver and pernicious anemia. Similarly, the reaction word to Volkmann's Contracture is forearm.

Volkmann's Contracture need not occur in the forearm alone. We have an anatomical arrangement similar to the flexor surface of the forearm on the posterior aspect of the leg. The flexors drained by the median vein to the antecubital fossa in the forearm, and the flexors of the foot by the posterior tibial vein which passes through a window in the popliteal fascia. In view of the similarity it is logical to believe that the contracture may occur in the leg, since the accepted etiological factor is impaired circulation with muscle change. The reports however show that ninety percent of the ischemic contractures of Volkmann are seen in the forearm.

The lack of specialization of calf muscles masks the deformity and it is not so often recognized, the author suggests. A calcaneo varus deformity would be seen in the event of contracture of the leg.

The incidence is not alarmingly high but the literature is inadequate as an index, in view of the fact that some men believe the contracture due to
mistakes or mistreatment on their part. With such an erroneous idea throughout the profession the number of cases reported is limited. Knowledge of the incidence and the proper treatment is essential in such a disaster due to the poor prognosis and the disabling deformity which results.
PATHOGENESIS and ETIOLOGY
Jones (27) offers the following etiological factors. Experimental work has shown that circulatory block may occur from within or from without, due to the pressure of edema and hemorrhage or from tight bandaging.

In most fractures of the lower end of the humerus a considerable hematoma forms in the anticubital fossa. This is a circumscribed fascial compartment with unyielding boundaries. At this level the venous return from the internal epicondylar group of muscles, (pronator radii teres, flexor carpi radialis, flexor sublimis digitorum, flexor profundus digitorum, flexor longus pollicis), converge to one main vein, the profunda vein, which is easily compressible. The rapid occlusion of the vein will obviously initiate the degenerative and fibrotic changes in ischemic contracture.

Other etiological factors of importance are, the placing of the arm in acute flexion and mechanically impairing or obliterating the venous return, the failure to reduce displacement, delay in reduction and allowing kinking and compression of the veins.

Eikenbary (14) offers the information that in his experience Volkmann's Ischemia has developed only in those cases which were placed in acute flexion with the fracture unreduced. The only cases of Volkmann's Con-
tracture see by Eikenbary were those with persistent posterior displacement following supracondylar fracture of the humerus.

Sever (43) points out that the damage is always done on the flexor surface and is always a flexor deformity or contracture and never an extensor contracture. He states that the condition may be seen after ligation, rupture and contusion of the blood vessels due to an embolus or thrombus of the brachial artery.

Lewis (34) brings to our attention that the presence of arterial thrombus or embolus can rarely be expected to yield transient loss of blood flow for just the adequate period to produce death of muscle alone. In the event that the collateral circulation be established in time contracture may be avoided. We must remember that pressure on the muscle and skin sufficient to collapse the capillaries would produce marked contracture and if over sufficient period of time gangrene may set in.

Taylor (48) suggests the traumatic origin of the contracture. Experimental work and clinical observation point to traumatic myositis as the fundamental condition in causing Volkmann's Ischemic Contracture.

Volkmann in his original publication stated it was a result of interference with arterial blood supply
aggravated by venous congestion and muscle substance, deprived of its oxygen, underwent coagulation necrosis and developed localized rigot mortis with contracture. As the ischemia is not complete gangrene did not occur.

Steindler (46) points out that Volkmann's original paper mentioned the possibility of ulcerating gangrene of the palmar surface of the hand and the inflammatory or cicatrical contracture which is apparently characterized by the rapidity with which it develops and the extraordinary resistance to attempt at correction.

Brooks (6) gives Leser's impression as follows:

"The appearance of the muscle tissue shows two processes having taken place, first, degeneration of the muscle tissue, second, marked inflammatory process in the muscle. The former manifested by the absence of muscle tissue nuclei and the predominance of connective tissue. The latter by the marked cellular infiltration seen throughout the sections."

Stone (47) states that the ischemia produced due to the interference of blood supply destroys the muscle fibers in a few hours. All of the muscles are not destroyed because there is usually only partial interference with the blood supply. If the blood supply were completely obliterated gangrene would be seen. The connective tissue growth contracts and produces more ischemia so
that the complete contracture is not seen until the interstitial myositis produced by the connective tissue overgrowth is complete.

Groves (20) offers some evidence by some experimental work done on a patient that was to be operated on for an elbow injury. There was injected one ounce of sterile saline solution under the skin and deep fascia and the arm flexed. The ease with which the radial pulse was obliterated proved a point showing the possibility of circulatory obstruction with a small amount of effusion into the tissues in an area of firm fascial support. It disproved the argument of malalignment as a causative factor in ischemia contracture.

J. P. Lord (35) reports a case of Volkmann's Paralysis in a hemophilic patient. This shows chiefly that the contracture can be brought on in the absence of tight splints. The case gave a history of slight trauma to the right elbow joint. The hemorrhage was a spontaneous thing apparently as the severity of the blow was not sufficient to cause it. The boy developed a typical Volkmann's Contracture with the exception that the fingers were not so acutely flexed. The arm responded to conservative treatment fairly well. The use of splints or adjustable apparatus was cut as a hemorrhage was elicited so readily. Fibrinogen was used and
the bleeding time reduced to six minutes.

We see by the work done on the etiology of the condition that Volkmann's theory has merely been confirmed. The ischemia disaster is due to partial arterial obstruction accompanied by venous congestion. The diminished blood supply is inadequate for muscle tissue growth but sufficient for fibrotic tissue growth which occurs. The venous congestion seems to be a necessary factor in the production of the contracture.
PATHOLOGY and PHYSIOLOGY
Jones (22) refers to the experimental work on dogs done by Jepson and Brooks. It has been proven that the causative condition in contracture is a venous return blocked and a partial arterial block. In the event of such a condition the cycle of changes begins immediately. First of all there is a widespread degeneration of muscular fibers and a rapid invasion of the whole muscle by round cells so intense as to suggest an inflammatory lesion. This phase of myogenic degeneration is followed by the formation of fibrous connective tissue which helps to obliterate the remaining muscular fibers and which shows a progressive muscular contraction.

Sever (43) points out that the pathological muscle change represents typical hyaline degeneration and disappearance of muscular tissue in varying degrees according to the severity of the original process.

Steindler (46) gives us a microscopic description of the tissue. The muscle pieces removed from various areas show varying amounts of connective tissue which is fully developed, with few vessels, in every respect corresponding to scar tissue.

The arterial blood supply being diminished there is increased carbon dioxide and impaired normal muscle metabolism. In the presence of the reduced arterial blood and high carbon dioxide connective tissue grows well.
The end result is the contracture of the muscle, due to destruction of myogenic tissue.
SYMPTOMS and DIAGNOSIS
and
DIFFERENTIAL DIAGNOSIS
Jones (22) states that symptoms may begin in the space of a few hours after injury. The fingers become numb and swollen and possess little or no power of voluntary movement. The damage is often complete in forty eight hours, so that initial symptoms are very urgent. Muscular rigidity and sometimes intense pain come on early and then the contracture. After some days the swelling disappears and the altered muscles become hard and resistant. If untreated further contracture ensues and disproportional growth of bone follow.

Lewis (34) discusses the particular symptoms on the basis of experimental work on ischemic effect of muscles and nerves. The times are not precisely known, but the death of muscle fibers is probably assured when they are deprived of blood for six to eight hours and their death results in a replacement fibrosis. Nerves recover after being ischemic for twelve or even twenty hours. Longer periods lead to degeneration and the period of regeneration is a long slow process.

Thomas (50) and Ferguson (15) point out that the chief symptoms to be guarded against and watched for, are pain, muscle tenderness, swelling, pulse diminished or absent and various trophic changes as coldness, cyanosis, shiny skin, ulcers on the fingers or blebs.

L. S. Dudgeon (13) discusses the contracture from
half the ring finger. In radial nerve palsy there is a wrist drop due to extensions being paralyzed. The triceps would also be paralyzed. Anesthesia over the radial nerve distribution.

In contracture after anterior poliomyelitis the history is essential. Perhaps the most diagnostic point is the much longer time required for contracture to develop poliomyelitis.

Little's Disease is differentiated by the involvement of the lower extremities and the etiology of the two conditions.

Functional disease could be ruled out by the complete reduction of the contracture under anesthesia. The electrical reaction to faradic or galvanic currents would also differentiate.
COMPLICATIONS

and

PROGNOSIS
Taylor (48) enumerates some of the more common complications. Pressure sloughs and abscesses are common on the ulnar side of the wrist and on the flexor prominence just below the elbow. When present these abscesses burrow to the deeper structures, heal slowly and add to the cicatricial rigidity. Nerve injury is purely a complication. It occurs in about sixty percent of the cases. It may however be primary due to severing or pinching at time of the accident. It may be secondary and by far the greater amount of the sixty percent are here. The ulnar and median nerves are most often involved and are usually constricted. Ulnar nerve involvement aids in the "claw hand" deformity.

Washburn (51) brings to attention the possibility of nerve involvement and the means of differentiating. In testing nerve involvement the inability to adduct or pronate the thumb indicates ulnar nerve involvement. The inability to flex the wrist or pronate the forearm indicates median nerve.

Jones (22) states that in many cases the prognosis is unfavorably although the function of the hand can be generally improved by appropriate treatment. In cases where, first, nerve damage is severe, second, obstruction to circulation to fingers has remained pronounced, third,
the forearm is fixed in extreme pronation and there is limited movement about the elbow, fourth, and only mass movement occurs in the fingers, the prognosis.

Taylor (48) again emphasizes the fact that the prognosis is unfavorable in the majority of cases. In as much as it is impossible to determine the exact amount of damage done, and an accurate diagnosis cannot be made until proper treatment has been watched for sometime.

J. M. Jorge (30) remarks that the prognosis after operative treatment depends not only on the duration and extent of the contracture and the loss of elasticity, but also on the patient's perseverance in the exercises afterward.
PROPHYLAXIS

and

TREATMENT
Jones (22) give us the following types of treatment and prophylaxis. The treatment of elbow fractures by flexion has so many advantages that it still is the position of choice. Whatever happens, we must never subject fractures and displacements about the elbow to plaster-of-paris splints or bandages.

Prophylactic measures demand therefore, that we should:

(a) Avoid circular compression.
(b) Reduce dislocations and displaced bones.
(c) Avoid all kinds of splints, more especially if there is much swelling.
(d) Use no force on flexing the elbow.
(e) Critically watch all cases of fracture about the elbow the first few hours, guarding against pain (not always present), stiffening and swelling, cyanosis and lividity of the fingers.

Treatment in cases of already started ischemia. The arm should be released from all restraint which involves compression, and elevated. Manipulation of every kind for further reduction of displacement stopped. Murphy, in 1914, suggested that if cyanosis continued with forearm extended and elevated, a
subcutaneous division should be made on the antero-
ulnar side of the forearm. Some cases have been
treated by a subfacial incision and removal of the
subfacial hematoma with resulting ischemia disappear-
ance and the hand return to normal.

(a) Mechanical type of treatment consists of
unfolding the contracture by gradual splinting as the
contracture yields, the hand is markedly improved in
functional ability. In cases of severe contracture,
usually the conspicuous "claw hand" can be reduced some.
This splinting is accompanied with physio-therapy as
radiant heat, bathing and gentle massage.

(b) The operative treatment consists of (1) teno-
plasty, or lengthening of the individual tendons at the
wrist, (2) shortening of the radius and ulna by resect-
ion of a sufficient length of bone. In severe cases
muscle sliding has been tried, but is not so successful.
In extreme cases excision of the joint is offered. The
results of conservative splintage should be tried before
such radical procedures are even considered.

Steindler (46) offers a suggestion on mechanical
treatment. The mechanical means of treatment is
accomplished by splinting and gradual extension. The
splints are placed on the effected arm with the wrist
flexed, for the fingers can ofttimes be extended in
this position. As the fingers are straightened the wrist is brought into extension until it is hyper-extended.

Thomas (50) advocates a fasciotomy. In the severe cases, and those where there is a limitation of pronation and supination, a cutting operation seems to offer the best chance of a good result. Resection of the bones, while it corrects the deformity from contracture, must weaken the extensor muscles, there is danger of non-union, and often no increased usefulness to the crippled hand. The treatment by lengthening the tendons has given better results than resection, but has the chief objections of the possibility of infection following so many sutures, the great difficulty afterwards of breaking up the numerous adhesions within tendon sheaths, and the time element involved, particularly in children. In all cases where operative measures are decided upon the after treatment cannot be over exaggerated. Passive and active movements, massage and faradic current stimulation to nerves, must be exercised for several months following operation.

Myotomy or dissection of the effected muscles from their attachments and suture in other positions has been used by several men and seems to offer the greatest chance of good results in severe cases.
C. U. Collins, (9) states that in his experience if muscle massage and manipulation are used until the contracting process is completed then the use of the tenoplasty operation good results can be obtained.

Baughman (2) agrees with previous authors in that fasciotomy is the procedure of choice. The skin is sutured loosely, but not the fascia, in closing the incision. He states that a patient with a Volkmann's Contracture will require three months at the least for the contracture to reach it's full development. Operation for correction should not be done before.

J. M. Jorge (30) suggests hot baths, exercises, suspension of the limb, adjustable apparatus to aid in stretching the retracted muscles, and massage may succeed alone, but if the contracture shows no signs of yielding he advises not to wait too long before resecting the diaphysis or doing a tenotomy.

H. W. Meyerding (39) offers two types of treatment depending on whether the case is a recumbent one or ambulatory. In the former he advises elevation of the arm to relieve swelling. In the latter case an airplane type of splint is excellent. He advises treatment directed toward care of soft parts, defer the reduction several days if necessary. Every case requires individual judgement. Meyerding reports that forty
percent of cases of fracture associated with ischemia paralysis are malunion cases. So reduction as good as it can be done is essential also.

J. R. Girdlestone (17) states that whenever reduction has been previously and unsuccessfully attempted under anesthesia, open reduction rather than further manipulation is advocated. In the operative procedure an antero-internal approach is preferable, identifying the brachial artery and median nerve first and freeing them from pressure.

Milici (37) reports that since 1928 he has treated seven cases of Volkmann's Paralysis with elastic traction. This is not a new method, but the author has devised a rather ingenious device which gives excellent results. The splint is made of ordinary metal that is not too pliable. It consists of two parts, one proximal part for the forearm, and another distal part for the hand. These two parts are connected by a hinge, so that the distal part can be moved to any desired position. At the wrist the two parts of the splint have grooves through which a metal rod can be inserted and thus immobilize the two parts. A loop of wire spread out in banjo fashion from the end of the splint serves as a means of making elastic traction on the fingers.
Japanese finger traps are placed on each finger of the hand to be corrected. These finger traps are made of woven straw and are so constructed that they become tighter on the finger as pull is exerted. They can quickly be released by approximating the ends of the traps together. Each trap, four in number, is held to the banjo hoop by Esmarch elastic bandage, thus steady traction is made on the fingers. The fingers are taped first to avoid irritation from the traps. The traction is started, by placing the splints in the position in which the fingers lie and gradual extension over a period of time is accomplished. The immobilization is thus gradually changed into a more dorsiflexed position and traction continued until both wrist and fingers are on the same horizontal plane. The finger traction is then removed and the wrist hyper extended still further by traction between the dorsum of the arm and the distal part of the splint. It is found better here to use a plaster of paris case over the two pieced splint. The traction is maintained by Esmarch elastic bandage again. The distal part of the splint is removed and incorporated on the dorsum of the cast, and the fingers dorsiflexed to a plane with the wrist. The forearm, wrist and fingers are then placed in a cast and maintained from four to six weeks.
Immobilization after the first correction is essential or a return to the former position will occur. In the end position the wrist and hand and fingers are in a dorsiflexed position.

Carlson (8) presents a case treated with a banjo wire. He offers further information on traction of the fingers. In his cases he has used a Keith's straight abdominal needle placed through the distal phalanx and traction with an elastic bandage. He reports good results.

Dr. S. Driver (12) states that an important point in treatment, in cases where dressings are to be used, is that the dressing include the hand and fingers. If you stop your dressing at the wrist a vicious circle develops.

As to the prevention Dr. G. A. Caldwell offers the following:

"The most needed lesson in the prevention of the Volkmann's Ischemia is to watch the arm closely and frequently during the stage of swelling, to lower the forearm if necessary and to make liberal incisions, not puncture drainage, through the deep fascia when less flexion fails to relieve the tension."

M. E. Pusitz (41) points out the essentials in proceeding after having made use of longitudinal
incision in the fascia of the arm. The muscle groups must be separated by blunt dissection to allow complete drainage. In making the incisions through the vaginal fascia, it must be remembered that the forearm consists of a number of fascial compartments. Opening one compartment when another is involved does not solve the problem. The hematoma may be in the antecubital space, or the joint, or in the fascial compartments of the forearm. The many possibilities make it imperative to know the anatomical structure of the fascial compartments of the forearm. Professor Prentiss has done some remarkable work in this particular field of anatomy.

Sorrel (45) gives us a new technic of resection of the bones of the forearm. The treatment consists of a shortening of the antibrachial skeleton by a chevron-wise osteotomy of the two bones, by means of the electric saw, retention, if necessary, of the osseous extremities by a plate, disinsertion of the superficial and deep flexor tendons, or else, section or disinsertion of the round and square pronators. Before or after this operation, but at a timely distance, the arterial bifurcation, is explored, and if a segment is found definitely altered, one proceeds to its resection.
Since the condition calls for quite a marked shortening of the antibrachial skeleton, -- 4.5 cm., for instance, in one of the writer's cases, a nine year old boy -- the writer suggests a chevron-wise osteotomy, as shown in the illustration. It also stands to reason that, with this technic, it will not be so difficult to hold the shortened fragments in place, and hence, retention plates will not be needed so often. Although Pouzet reports a fine result with resection of the bones of the wrist, the writer believes that the antibrachial bones are better adapted for the shortening. Aside from the impairment of the movability of the wrist that a resection bearing on this region might entail, it also permits only a small shortening. As to the muscular reduction by disinsertion of tendons, it is a complementary measure to the skeletal shortening.

Chevron-wise osteotomy. As has been said, this osteotomy can only be effected with the aid of an electric saw.
Godoy Moreira (18) favors the operative process of shortening of the bones of the forearm. He states that the procedure has in its favor the simplicity and rapidity of execution and leaves the muscles untouched, whose structure is already altered, and which by an operation would necessarily be traumatized.

The disadvantage of this method lies in the possible delay of consolidation or even in the formation of pseudo-arthrosis.

The study of the operative technique in the shortening of the ulna and radius which the authors prefer, all other things alike is achieved by a simple division of the continuity of the bones followed by a termin--terminal osteo-synthesis.

Three steps are of essential importance:

First, resection at different levels, distal for the ulna, proximal for the radius which means that one is to resect as far as possible from the wrist; for the ulna, however it is necessary to make the resection at a distance from the elbow.

This step has the advantage of avoiding casual synostosis which might take place in consequence of exuberant calluses where the points of division are on the same level.
Second, the division must be made in such a manner as to obtain a large surface of contact for the osteosynthesis and achieve a better fixation which will permit an early mobilization without the dangers of nonconsolidation.

Third, the fingers and the wrist must be in hyperextension.

The hyperextension of the fingers and of the wrist favors the retraction of the extensors which are too long and on the other hand the elongation of the flexors which are too short.

We see from a resume of the various advocated treatments that the accepted one is the mechanical treatment. The authors agree that conservative treatment should precede radical. In cases where improvement can be seen after progressive stretching by splints it should be used.

The perfected forms of osteotomy is a second alternative. The chevron-wise method being the preferred method. The tenoplastic operation is a means of correction but not advocated by the majority of men.

The best treatment is prophylaxis which can be practiced only by having a knowledge of the condition and its onset.
SUMMARY
The following nine points gives us a composite picture of Volkmann's Ischemic Contracture.

1. Condition follows too tight bandages or soft tissue edema, particularly in the arm, rarely in the leg.

2. Paralysis is ischemic in origin and is followed early by contracture. The venous stasis hastening the general process. Coagulation necrosis setting in, in the involved muscles.

3. Paralysis and contracture always come together, or at least almost so, unlike nerve injury where contracture is latent.

4. There is great rigidity from the first.

5. The rigidity increases from contraction of scar tissue.


7. Ischemia isn't complete so there is no gangrene.

8. Prognosis depends on amount of destroyed muscle tissue.

9. Volkmann maintained that only mechanical treatment helped, advised stretching. In cases where tendon or bones would give before muscles, nothing could be done. Present day treatment gives us osteo-
tomy as an alternative in difficult cases.
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