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ISCHÆMIC CONTRACTURE*

EXPERIMENTAL STUDY

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DEFINITION.—Ischæmic contracture is a specific deformity following an injury to the extremities, and usually occurs in children between three and twelve years of age, whose blood-vessels are less mature and more easily disturbed than those of older persons. In the majority of cases the injury is in the upper extremity, at or near the elbow-joint. As it usually comes on following a fall or a blow in cases of fracture, and at times when there has been no fracture, there is an extensive injury to the soft parts in the fold of the joint with a resulting hæmatoma. Usually fixation by one or another method or tight bandaging has been applied, causing severe pressure on the structures in the region involved, and in a short time swelling and blueness of the extremity with paræsthesia followed by more or less pain. Within a varying period after these symptoms, and even after the removal of the fixation or bandage, there is swelling in the muscles, especially the flexors. These muscles gradually shorten, and there follows a severe contraction of the wrist as well as of the fingers, and in the more advanced stage, the claw-hand deformity results. In some cases there is limitation of supination and pronation. The joints are usually not affected. The fingers can be extended if the wrist is hyperflexed. The muscles are hard and rope-like, and often there is a slough on the flexor surface of the arm. Even the skin may have a hard leathery feeling. There may be disturbances in sensation in areas supplied by one or more nerves, most frequently only in this part of the hand supplied by the ulnar nerve, but sometimes in the area supplied by the median or radial nerves.

Following these usual initial symptoms the fingers become pale, cyanosed, and cold. Within a few hours the patient complains that the fingers are numb. He is usually more concerned than the surgeon. Electric irritability of the muscle is lost after about five hours of ischæmia, in some cases in as short a time as three and one-half hours. The muscles become flaccid and powerless. Muscular rigidity is then noted and the painful contracture begins. This disappears in about sixty-five to seventy hours, leaving the muscles again flaccid and very tender to pressure. After a few more days the

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swelling disappears and muscular tissue begins to be replaced by fibrous tissue. Muscles then become hard and resistant, and muscular atrophy appears.

The tension on the extensor musculature of the fingers as a result of the hypertension in the metacarpophalangeal joints is practically unavoidable even though extensor muscles are usually not involved in myositic changes.

A gross pathologic study of the condition shows that the muscular tissue is pale yellow, hard and board-like, and that the tendons are usually matted together. As a rule there is no change in the joints other than a tendency toward subluxation. The nerves appear to be flattened. Microscopically there is a loss of muscle striations, atrophy of the muscle fibres, a loss of the nuclei of the muscle fibres, and an increase in connective tissue.

Review of the Literature.—In 1869, and later in 1875, Volkmann described a deformity of the hand and wrist resulting from an interference of some nature with the blood supply of the muscles of the forearm. This condition was usually preceded by the application of splints or bandages for fracture of the humerus in the region of the elbow-joint. In his classical article published in 1881, Volkmann said that he believed the affection was due to ischæmia caused by the muscular tissue being deprived of arterial blood, in consequence of which the muscle perished from want of oxygen. He called attention to the fact that the contracture comes on sometime after the initial paralysis; that it becomes more marked as more repair tissue is laid down; and that from the onset of the condition there is considerable rigidity which is increased as more scar tissue is formed. He reported but one case in 1875 and that was in a child of sixteen. Mention of this case was made in his book published in 1869. Previous to this Hildebrand, in 1850, quoted a case of Hamilton's without giving the reference, but so far Hamilton's account has not been found.

The credit of calling attention to the condition and establishing it as a real entity belongs to Leser. In 1884 he reported seven cases. A little later he investigated the condition experimentally, using dogs in his research work. He gave a comprehensive and detailed account of the findings. Leser believed the condition was caused by a deprivation of oxygen to the muscles, but gave no definite theory as to how this lack of oxygen was caused.

Bardenheuer maintained that the pathologic change was due to a vascular disturbance such as venous stasis, and that the degeneration of the muscle fibres was caused by the retention of toxic metabolic products in the muscle. Rowlands believed that the paralysis and contracture were the result of the sudden release of pressure on the muscles, allowing the blood to congest the muscular tissue. Murphy believed that the condition was due to a pressure ischæmic myositis caused by hemorrhage and effusion into the muscles. This condition is augmented by the constriction of a splint or bandage or even by tight skin, and this in turn leads to myositis from pressure anæmia and later to contraction of muscles as a whole, resulting in a shortening of muscle tendon. He thought it was not injury to arteries but injury to veins that caused the destruction of the protoplasm of the muscle cells

Some of the cases reported by Langer and Schloffer were evidently due to embolism. Bardenheuer reported a case of fracture of the clavicle resulting in an ischæmic contracture brought about by bandaging the arm tightly to the body. Barnard, Dudgeon, and Ward reported cases in which contusion of the forearm without fracture resulted in contracture of the hand and fingers. Powers and Riedinger reported deformity following the use of elastic bandages.

Thomas contended that a mere circulatory disturbance was not sufficient in itself to cause a typical Volkmann's contracture.

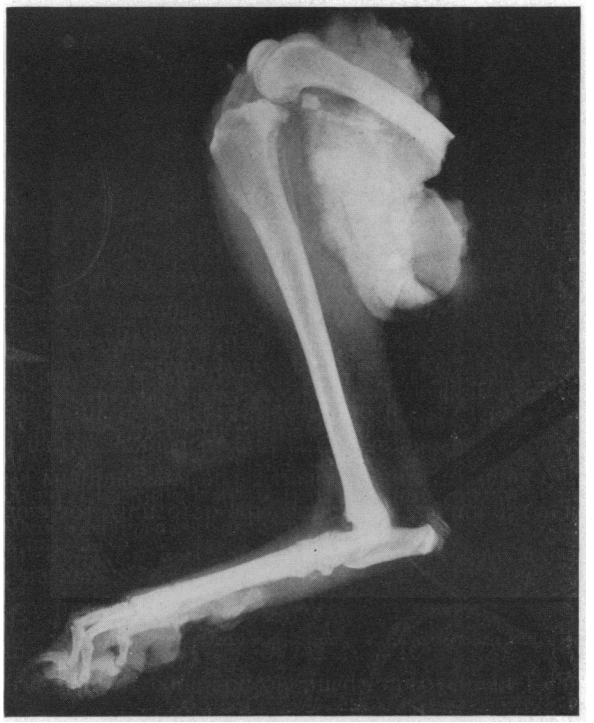

FIG. 1.—Almost complete occlusion of blood-vessels resulting from operation as revealed by injection of suspension of barium sulphate.

Thomas, Bernhardt, Köbner, and von Frey, and others believed that the paralysis following the use of the elastic bandage resulted in a flaccid paralysis with no contracture in the paralyzed muscles due to an injury to the nerves at the time of the accident, or subsequently. He was preceded in his ideas by Petersen, who, in 1888, wrote of the occlusion of the arteries and the involvement of the nerves. Wallis and Brehmann reported cases in which nerves were involved. As far back as 1850, Hamilton found the median nerve stretched over a fragment of bone and obtained improvement following the release of tension. Thomas stated that the reaction of degeneration in the small muscles of the hand was positive evidence of involvement of the nerve trunks, because with a destructive inflammatory process in the muscle itself there would be only a diminished reaction, if any. Paralysis of the muscles of the hand can be due only to involvement of nerves.

Steindler placed the location of the injury at the epiphysis of the humerus, and corroborated this statement by citing a number of cases.

Some cases have been reported with limitation of supination or pronation, and one case with involvement of the pronator quadratus only, in which the contracture of this muscle limited supination. After this muscle was explored and sectioned to relieve the tension almost complete recovery followed.

Soubeyran has explained the exclusive localization of the contracture in the flexor and pronator muscles by the anatomy of the arterial system in the arm. When there has been an injury to the arm or forearm in the region of the elbow the circulation usually stops in the ventral part but persists in the dorsal area, owing to the anastomotic plexus of blood-vessels about the elbow. The defective circulation prevents the supply of oxygen from going to the flexor and pronator muscles. Certain transformations occur which are followed by shortening and stiffening of muscular fibrils.

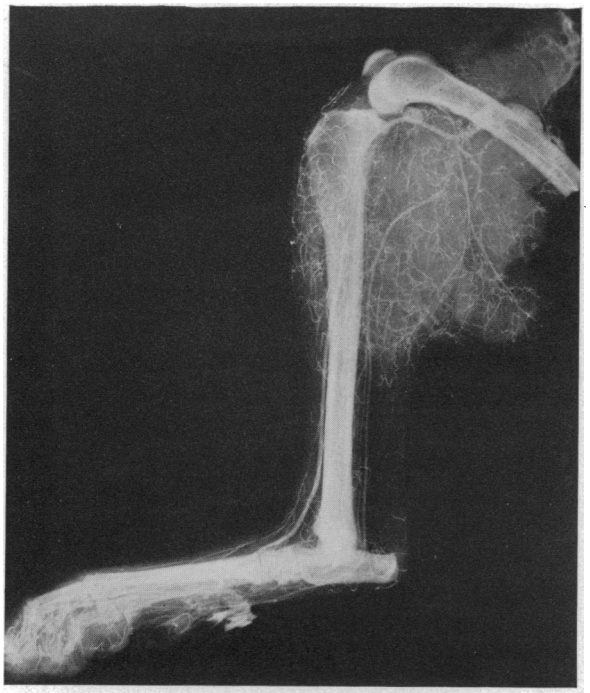

FIG. 2.—Patency of blood-vessels in control extremity of same animal as in Fig. 1, as revealed by injection of suspension of barium sulphate.

Brooks showed in his experiments that total paralysis of the muscle could not occur. In some of his experiments he demonstrated totally viable and totally necrotic muscles adjacent to each other when there was arterial obstruction, but he did not believe such a condition was due to permanent arterial obstruction. He also showed that skin was more susceptible to a continued diminished blood supply than muscles, but muscles would survive for a shorter period than skin in complete absence of blood.

Davies-Colley found both the median and ulnar nerves small and purplish below the scar tissue in one case, and many others have reported similar conditions. Bradley has recently contributed the thought that atrophic changes are brought about by chemical liquefaction of the tissue proteins acted on by enzymes. Peptoids and amino-acids are formed as end products. In some cases of atrophy, digestion of the tissue proteins is further facilitated by phagocytosis, but this, of course, is a secondary process. Bradley believed that the increase of connective and adipose tissue in atrophied muscle was a compensatory invasive process from the intermuscular septum and a phenomenon secondary to the atrophy of the muscle itself.

Some writers (Davies-Colley) have maintained that the contracture is due to the scar tissue resulting from pressure sores, but this is disproved by those cases in which the typical deformity of the Volkmann-Leser contraction developed without pressure sores.

So far as I have been able to determine the mechanism of an ischæmic contracture as seen in man has never before been reproduced in an animal for the purpose of determining, if possible, a means of preventing this deformity. For this reason it was thought worth while to report the experiments begun in 1924 at the Institute of Experimental Medicine of The Mayo Foundation.

Method of Experimentation.—Dogs weighing approximately 12 kg. were used in all the experiments. Ether was used for the anæsthetic and all operative procedures were carried out with sterile technic. The non-operative procedures were carried out with due regard to the comfort of the animal and morphin was freely used.

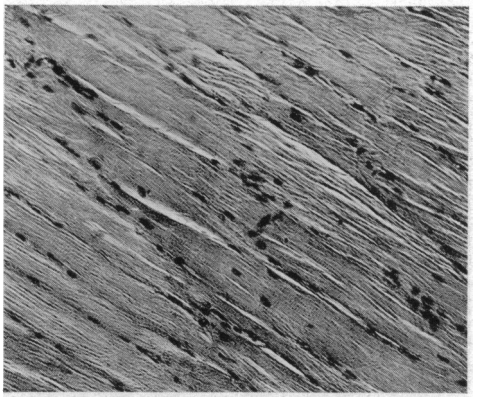

FIG. 3.—Longitudinal section of muscular tissue from ischæmic extremity of dog H90 (hæmatoxylin and eosin, × 120).

In the first series of experiments an attempt was made to produce a more or less typical contracture deformity by means of splints, casts, and bandages, but it was soon seen that it was impossible to produce a deformity lasting for any length of time by these methods.

In the second series of experiments an Esmarch rubber bandage was applied above the knee of the right pelvic limb and left on for from one and one-half to twenty-four hours. In the animals who wore the rubber bandage for from one and one-half to three and one-half hours, the deformity manifested by a main-en-griffe, disappeared within three or four hours after removal of the tourniquet. In those with the bandage applied for a longer period the deformity remained for three or more days, but at the end of this time there was normal function in the extremity.

In the next series the dogs were operated on in pairs. In one animal a simple ligation of the femoral vein was performed through a longitudinal incision over the vein on the right thigh. The vein was isolated and ligated with linen as it lay in Hunter's canal. In the second animal in this series an incision was made on the medial side of the right thigh running parallel to, and about 6 cm. below, Poupart's ligament, in such a manner as to encircle a little more than one-third of the thigh, and it was carried down through the fascia to the muscle. Within a few minutes the extremity operated on was cold and bluish. So far as could be noted there was little difference in the animal having the simple ligation and the one having the incision around the thigh. First, there was spasticity of the extremity with rather flaccid paralysis of the digits, but of such a nature as to make the animal tend to curve its toes under. This deformity simulated the main-en-griffe deformity in the human being, and was maintained for from six to nine days. The

dogs with simple ligation of the vein maintained the deformity slightly longer than those with the partially encircling incision.

In the next series these two operations were combined. Almost the same results were obtained as in the two previous series, except that the deformity lasted a few hours longer.

From the results of these experiments it was concluded that ischæmic paralysis did not depend wholly on the procedures that had been employed. Accordingly, after the wounds had healed an Esmarch bandage was applied fairly tightly above the knee at the site of the initial operation, fastened in place with adhesive, and left on for from six to twenty-four hours.

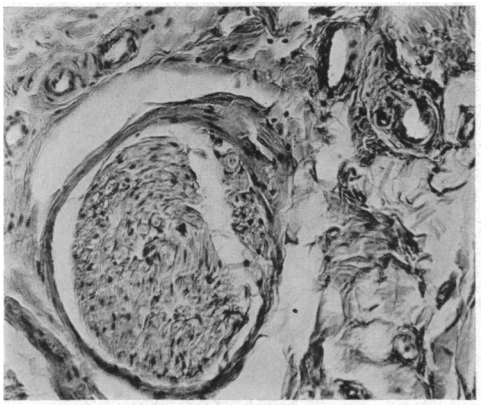

FIG. 4.—Cross-section of muscular tissue from ischæmic extremity of dog H90 (hæmatoxylin and eosin, × 120).

One of the animals was left with a deformity typical of ischæmic contracture and similar to that in man. The forefoot and distal phalanges were flexed while the proximal phalanges were extended. This simulation of the typical contracture was not persistent in all of the dogs. However, the animal which wore the bandage the longest maintained the deformity for more than eighteen months with no signs of improvement. The leg gradually wasted away and became hard and board-like as in the human being. When the animal attempted to put the foot to the ground, only the tips of the toes would touch and there was considerable resistance in the muscles. With exercise the deformity increased and the dog limped about as he did immediately after the application of the bandage. Another animal on which the bandage was kept for several hours still had the deformity at the end of two months, but after that he gradually improved.

A method of producing a lesion which appeared similar to that in man having been developed, the next procedure was to determine, if possible, a method of preventing such deformity. A dog in which the incision and ligation had been performed was selected for this experiment. An Esmarch bandage was applied above the knee and left on for eight hours. At the end of this time there was considerable œdema and other signs of a sluggish circulation, and the toes were contracted. Six hours later the wound was opened and the blood and serum were evacuated. Two rubber tubes for drainage were placed deep in the intermuscular space and sutured. The following day the swelling had gone down markedly, and four days later the dog was walking normally. This was in marked contrast to the condition of the control animals in which drainage had not been instituted. The

experiment was repeated often enough to bring out the fact that intrinsic pressure is a factor which must be dealt with in this condition.

Correlation of Former Findings with Present Findings.—In the pioneer study of the affected muscular tissue made by Leser, loss of nuclei of the muscle fibres and an increase in connective tissue is described. Petersen reported similar findings. Bernays, who was the first to report the microscopic findings, observed the loss of the nuclei and of the transverse striations of the muscles. He also found some atrophy of the muscle fibres. Most writers speak of the hardness and yellow color of the muscles and also of the tendency for the tendons to mat together.

In one dog the typical main-en-griffe deformity was produced and has been maintained for more than eighteen months with no signs of improvement. This was the typical contracture deformity. On careful examination of this animal the first thing noticed was the contraction deformity of the extremity operated on. This dog was given especial attention as he was the only animal to maintain the typical deformity for more than a year. When he was taken from his kennel he apparently walked normally, but after a dozen or more steps he began to limp, and walk favoring the right pelvic limb. The toes were curved back and seemed colder than those on the left leg. The measurements were as follows:

	Right side, cm. (side of operation)	Left side, cm.
Ball (above the toes)	6.5	7.0
Instep	19.0	22.0
Knee	22.0	24.5
Above the knee	23.0	25.7

There was noticeable wasting of the muscles similar to that seen in man. The animal was anæsthetized and when the muscles were carefully dissected the blood-vessels were found to be markedly enlarged above the site of the former operation. At the point of the old operation all the structures were matted down with scar tissue. The blood-vessels were the size of fine threads below the operative scar and the nerves were flattened and darker than those in the control leg.

The vessels of the extremity operated on (Fig. 1) were injected almost immediately, but it was impossible to get the lightest suspension of barium sulphate under high pressure through the vessels. The solution stopped at the cicatricial scar. However, when the control extremity was injected with barium sulphate the vessels were quickly filled with the suspension, as would be expected (Fig. 2).

Histologic study of muscular tissue from the ischæmic extremity showed a marked increase in the intermuscular nuclei which have arranged themselves in rows, and variation in the size of the muscle fibres and some decrease in muscle striations (Fig. 3). A cross-section shows proliferation of the endothelial cells lining the smaller blood-vessels, almost approaching the point of occlusion in some places, and definite thickening of walls of all the blood-vessels, especially the small vessels. Many cells present the characteristics of fibroblasts, an increase in connective tissue is seen, also some loss of the nuclei of the muscle fibres (Fig. 4).

Microscopic examination of the nerves from the ischæmic extremity (Fig. 5) revealed the well-marked degeneration of some of the fibres (light gray) and the intact state of others (dark gray). There was definite, but not as extensive, degeneration as in a nerve which has been actually cut. In certain areas there was slight swelling of the myelin and in other areas almost complete disappearance of it (Fig. 6).

The histologic features of muscle atrophy have long been studied. I found, as have others, an increase in the amount of connective tissue (Fig. 3). The number of intermuscular nuclei was increased. In some sections there was an increase in adipose tissue (Fig. 4). The muscle fibres were usually diminished in size, but occasionally there was an hypertrophied fibre.

Discussion.—It appears that direct injury to the nerves may frequently occur, but it has not been proved that such injury is the primary cause of the typical main-en-griffe deformity. There must be some other causative factor. It is true that the involvement of the intrinsic muscles of the hand can be explained only by direct injury to the ulnar nerve, but as a rule injury to some other tissue precedes injury to the nerves. In the forearm the ulnar nerve is the one most commonly affected. The explanation of delayed ulnar palsy is that peripheral neuritis develops some time after the injury.

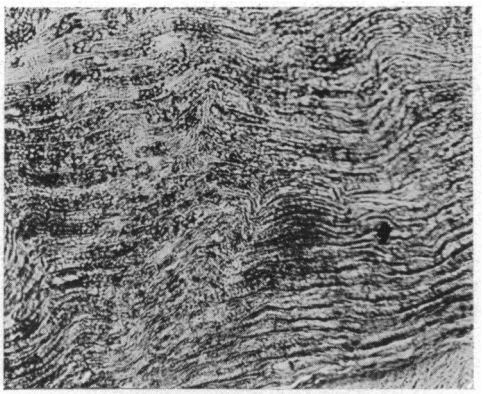

FIG. 5.—Longitudinal section of nerve from ischæmic extremity of dog H90 (Weighart-Pal, × 120).

The almost universal involvement of the flexors but not of the extensors may be explained on anatomic grounds. On the anterior surface of the forearm the soft tissues, composed of the flexor muscles, arteries, and nerves, overlie the radius and ulna, forming a good-sized pad. Besides this, the body of the anterior flexor muscles comes in direct contact with the source of the injury, whether it be the splints, cast, bandages, or direct force. The ulna is situated between the splint and the extensor muscles and protects these muscles from the injury of a splint.

No other structures are more susceptible to injury than the nerves. With their close proximity to the blood-vessels, a constriction lasting for a long time and sufficient to close the large blood-vessels of the brachial region must of necessity exert marked pressure on the median and ulnar nerves, and induce neuritis with subsequent degeneration of the nerve. The extensor muscles do not suffer because the musculospiral nerve which ultimately distributes branches to this group of muscles is deeply placed in the upper arm between the triceps muscle and the humerus. In the region of the elbow-joint the nerve is protected by its position between the long extensors of the forearm. It is, therefore, not subjected to as much trauma as the other nerves because of their superficial position in the upper arm.

Embraced as it is by the pronator radii teres muscle, the median nerve is subjected to practically the same injurious forces as the muscle. Therefore, if pressure over this muscle and its associated group is sufficiently great to cause profound ischæmia, the nerve must share the fate of the muscle

which encloses it and suffer degenerative changes to some extent. Flaccid paralysis at the first, followed shortly by the contraction deformity, would suggest primary injury to the nerves but in all probability there is also injury to the muscle, which helps to form the contracture and resulting deformity.

On the other hand, the acceptance of a primary myositis and a neurogenic origin for this type of contracture would explain cases of Volkmann's ischæmic contracture in which no fracture occurred or in which no profound changes took place in the muscles. The site of the lesion may often be indicated by the scar on the forearm.

If a fracture or injury occurs in the region of the elbow-joint, the tissues are bruised and effusion of blood and serum follows. The tension in the subfascial zone in the forearm can be so great as to cause cyanosis of the whole forearm and hand. A blood clot forms in the tissues, and inflammation follows with a deposit of the inflammatory products in the tissues. This deposit in itself will cause pressure and have the same effect as a tight splint or bandage.

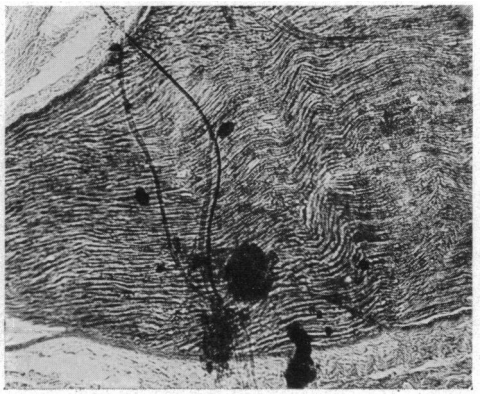

FIG. 6.—Cross-section of nerve tissue from the ischæmic extremity of dog H90 (Weighart-Pal, ×120).

Muscles completely deprived of their oxygen supply finally disintegrate, but in a typical case of Volkmann's contracture the deprivation of oxygen is not complete. There is ischæmia, the blood supply is cut down far below normal, reducing the nutrition to the muscles, and as a result the resistance of the extremity is lowered. In the specimens studied during this experiment, the calibre of the blood-vessels was so markedly reduced as to prohibit the flow of the solution used in an attempt to make a röntgenogram of the circulatory tract of the extremity.

It can be understood from the discussion that no one factor is responsible for the production of the typical deformity seen in a case of the Volkmann-Leser ischæmic contracture. It would seem from these experiments that the deformity in the human is usually produced somewhat as follows: First, there is an injury of some extrinsic nature in the region of the elbow-joint, or to the humerus or bones of the forearm, consisting of a fracture or an injury to the soft parts only, or both; or fracture of a clavicle, and so forth. Splints, casts, or bandages may or may not be applied. When splints are used pressure sores often develop, usually coming over the flexor muscles which predispose to the formation of scar tissue. But pressure sores are not required to bring about the deformity. The tissues are bruised by the trauma and extravasation of blood, and serum follows. The tension may be so great as to cause cyanosis of the entire forearm. This intrinsic pressure

causes local myositis and then a pressure on the nerves (usually the median and ulnar) and the blood-vessels. There develops a flaccid paralysis followed by swelling in the muscles. Almost immediately contraction of the flexor muscles begins, resulting in the main-en-griffe deformity originally described by Volkmann. As the condition goes on, due to the diminished blood supply, the flexor muscles begin to atrophy and the tendons become matted together. When the wrist is hyperflexed the fingers can be straightened out. If the intrinsic pressure is relieved within a short time after the formation of the hæmatoma, the patient will usually recover.

SUMMARY

The lesion of ischæmic paralysis as seen in man was reproduced in animals by bandaging one extremity and by preventing the return of the venous blood. In an attempt to prevent the development of the deformity, it was found that if drainage was instituted within a few hours after carrying out the procedures leading to the development of the lesion, contracture did not ensue, or was very slight. The results of these experiments would seem to indicate that the contracture deformity is due to a combination of factors, the most important of which are impairment of the venous flow, extravasation of blood and serum, and swelling of the tissues with consequent pressure on the blood-vessels and nerves in the involved area. If this is true, early drainage would be of value.

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