of the elbow, except extension, which was limited by the operation scar. Six months after the original injury this scar was excised and skin-grafted. At this procedure the brachial artery and its branches were seen to be of normal calibre in the previously affected region.

Observation in the above case supports the opinion that an intense arterial vasoconstriction is present in Volkmann's ischaemia, the change starting at a definite point on the brachial artery. Intense venous engorgement is also present. Operative intervention upon the artery did not here result in a speedy alteration in the state of affairs. It cannot be claimed that an operative measure which brings relief after eighteen hours in an acute vascular crisis is a factor in the cure. Similarly, as regards operative measures on the venous side, there was no haematoma to release. While decompression of the muscle bellies caused an improvement in the appearance of these, there was no immediate relief in the peripheral circulation, as seen by the state of the fingers.

It would appear that surgical intervention directed to the arteries does not contribute to a favourable termination of the circulatory changes in Volkmann's ischaemia. Reduction of the fracture and expectancy, with judicious freedom of the elbow flexure, while preserving the position of the fracture, form the first measure, assisted by elevation. It is possible, however, that decompression of the muscle bellies by division of the fascia may prevent the occurrence of permanent muscle changes during the slow process of vascular readjustment.

Discussion

Just as pathologically there are two changes in Volkmann's ischaemia—arterial and venous—so do two theories based on these changes exist. Volkmann's opinion was that the condition was due to venous congestion and deprivation of arterial blood—an opinion which supports the pathological findings of these days but does not elaborate the mechanism of such changes. Brooks's experiments show conclusively that arterial deprivation alone does not cause these changes, and numerous clinical observations on cases of damaged arterial supply of the upper limb amply confirm this opinion. Brooks's experiments, repeated by Middleton, emphasize the venous element, and there seems little doubt that the resulting cellular infiltration and subsequent fibrosis in the muscles are the result of venous congestion.

The arterial change is very striking, but, as has been shown, treatment directed to the artery does not rapidly alleviate the condition. The sudden change of calibre roughly corresponding to the fracture site might suggest that this has been caused by direct contusion of the artery by the fractured upper fragment. Garber (1939), quoting a case of Clarke's, found this constriction in an identical position two inches above the elbow-joint in a case of ischaemia associated with fracture of the upper third of the radius and ulna. This important case seems to disprove any theory of direct injury. He also points out that pinching the artery causes a local constriction at the site of pressure, but that there is no distal vasoconstriction spreading to the radial and ulnar arteries. It is therefore not possible to assume that there has been an arterial disturbance by the direct effect of the fractured bone ends. The pronounced venous changes led Middleton to suggest that the essential cause of the ischaemia was compression of the junction of the superficial and the deep venous plexus, but, as already pointed out in my case, no haematoma was found in the antecubital fossa, and it was also observed that the venous

junction shared in the engorgement. It is apparent that the vascular changes in Volkmann's ischaemia are striking on both the arterial and the venous side.

It is probable that the venous engorgement accounts for the pathological changes in the muscles and that relief of this engorgement mitigates the permanent damage. A parallel is found in the good effect of cranial decompression in cases of cerebral oedema.

The cause of the arterial and venous changes has yet to be explained. A general venous engorgement can be accounted for by the distortion and general obstruction of the normal circulation by reason of severe deformity and the accumulation of metabolites which cause capillary dilatation. It seems that the physiological response to this is a vasoconstriction of the arteries in an attempt to diminish the blood flow to a limb already soaked with fluid. The practical inference is that attention must be directed to the venous engorgement by reduction of the deformity, elevation of the limb, and, if need be, the emergency measure of decompression of the muscle bellies to limit the local damage while vascular readjustment is taking place.

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OCCLUSION OF THE BRACHIAL ARTERY AND VOLKMANN'S ISCHAEMIC CONTRACTURE

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Until recent years the most widely accepted theory of the cause of Volkmann's ischaemic contracture was that it was due to acute venous obstruction. This view was supported by clinical observations and by the experimental work of Brooks (1922) and Jepson (1926). Middleton (1928) described the anatomical details contributing to the condition.

In 1928 Leriche published a case of Volkmann's syndrome following a supracondylar fracture of the humerus on which he had operated and found complete obliteration of the brachial artery. He resected the obliterated portion of the artery, and the patient rapidly recovered. Since then many cases have been presented supporting this observation (Fontain and Kunlin, 1936; Brentano, 1935; Fèvre and Bertrand, 1935; Leveuf, 1938; Leveuf and Funck-Brentano, 1938; Garber, 1939; and Griffiths, 1938a, 1938b, 1940). Furthermore, Wilson (1932) and Brooks (1922) showed that lesions similar to those of Volkmann's syndrome could be produced by occlusion of the main artery of a limb. Finally, both Jefferson (1934) and Griffiths (1940) have reported cases in which this syndrome has followed embolism.

Although not yet widely recognized in this country the above evidence shows that some at least of Volkmann's contractures originate from an arterial lesion. The following report may be of some value in support of this theory, but is open to the criticism that, since contracture did not develop, the case may not have belonged to this group at all. The arteriectomy recorded below is, I believe, the earliest following arterial contusion with signs suggestive of Volkmann's syndrome: the arteriectomy was done early enough, in fact, to prevent the development of this dreaded malady.

Case Record

A girl aged 7 was admitted to hospital on August 28, 1939, after falling from a tree. She had a compound supra-condylar fracture of the right arm. The proximal fragment of the humerus was protruding through a grossly contaminated wound, and the median nerve and brachial artery were stretched over it. Her hand was cold and slightly cyanotic, there was no pulse, the fingers were kept in a slightly flexed position, and there was complete loss of sensation to pin-prick below the level of the elbow. On pressing the finger-nails and fingers one could get no return of circulation.

Operation was begun one hour after the accident. The wound was excised and practically all the brachialis anticus was removed because of extensive contusion and laceration. After excision of the wound the fracture was reduced and the brachial artery exposed upwards as far as two inches above the level of fracture and down to the level of the bifurcation. At the former level the artery looked and felt like a piece of string. Pulsation stopped about three-quarters of an inch above this point. There was obvious haemorrhage into the adventitia. No pulsation could be felt in the radial and ulnar arteries. One inch of the brachial artery was resected, the upper level of excision being just above the point where pulsation stopped. On opening the excised portion of the artery the intima was found to be discoloured by subintimal haemorrhage, and there was a small thrombus attached to the wall. About five minutes after the resection was completed the hand was noted to be warm. The median nerve was swollen and discoloured. The wound was closed without drainage and a collar-and-cuff applied.

At the end of the operation the hand and fingers were warm and pink. A feeble radial pulse could be felt. The following day a complete median palsy was noted, other movements and sensation having returned. The wound healed by first intention, and the arm was taken out of the collar-and-cuff four weeks after operation. The patient developed slight trophic changes such as shininess of the skin and a deeper coloration of the hand than normal. On elevation of the limb the hand became a pale pink colour, and in dependent positions it became cyanotic. At all times the hand and fingers were colder than those of the other side. A few days after operation the patient was given a woollen glove with an opponens splint incorporated in it, and she wore this continuously.

Three months after the accident she had made an almost complete recovery from the median palsy. There was no anaesthesia or paralysis, but still some motor weakness. Twenty-five degrees limitation of full extension of the elbow-joint was present. The child was attending school and playing games as usual. Six months after operation the trophic changes were not so pronounced, and extension of the elbow was only a few degrees short of normal.

Commentary

The clinical significance of this case is important. It appears to have been established that arteriotomy performed twenty-four hours after the injury does not prevent the complication of Volkmann's syndrome. But this case supports the view that early arteriotomy will do so. Griffiths's two cases were successful after five and six hours, whereas Arousseau's (1938) case was only partially successful after twenty-three hours.

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Clinical Memoranda

Granulocytopenia following Sulphapyridine Therapy

To the growing literature on granulocytopenia following sulphapyridine therapy the following case is added.

CASE REPORT

A 15-months-old male baby was transferred to the Park Hospital on February 5 suffering with meningitis. He had been admitted to another hospital two days previously with broncho-pneumonia, the meningeal signs appearing only on the morning of the 5th. At that time a lumbar puncture showed turbid fluid under increased pressure, and a meningococcus of undetermined group was grown. On admission to the Park Hospital the infant was in good general nutrition, with obvious signs of meningitis, but there was no petechial rash. No gross physical signs were detected in the chest. The diagnosis of cerebrospinal fever was confirmed by spinal puncture and the routine laboratory examinations.

Sulphapyridine was administered as follows: two 1-gramme doses on February 5, 3 grammes daily on the 6th and 7th, 2 grammes daily on the 8th, 9th, 10th, and 11th, and 1.5 grammes on the 12th—a total of 17.5 grammes over a period of eight days. The sulphapyridine content of the cerebrospinal fluid was 7 mg. per 100 c.cm. twenty-four hours and 4 mg. fifty-two hours after the initiation of treatment.

The patient rapidly responded to treatment, and on the third day from admission the cerebrospinal fluid was clear and contained 176 cells per c.mm., 50 per cent. of which were mononuclear. On the following day, however, the baby was pale, restless, and ill, with rapid grunting respirations and slight rise in temperature. The general condition became worse the day afterwards, with high fever and listlessness. Although physical examination and a radiograph failed to reveal any area of consolidation in the lungs, it was obvious that the child was suffering from a respiratory infection. There appeared to be some discrepancy between the severity of the constitutional symptoms and the gravity of this respiratory infection. As conditions remained unchanged, drug fever or agranulocytosis was suspected after a few days' observation, but there was no rash and no ulcerations had appeared in the oropharynx.

On February 13 a blood count showed 6,000 white cells per c.mm., with only 21 per cent. polymorphonuclears and no eosinophils. The following day the white cells had fallen to 4,000, with 9 per cent. polymorphs. Pentnucleotide, 10 c.cm. in two doses, was given that day by the intramuscular route and repeated for the next two days. The white cell count on February 15 was 5,400 per c.mm. with 20 per cent. polymorphs; on the 16th it was 8,800, with 40 per cent. polymorphs; and on the 18th it was 8,200, with 55 per cent. polymorphs. Eosinophils (2 per cent.) reappeared in the blood on the 18th.

The clinical conditions rapidly improved from the 16th, when the temperature fell abruptly. The child has since made an uninterrupted recovery.

COMMENTARY

This case belongs to the relatively mild group, as the granular white cells in the peripheral blood numbered 400 per c.cm. at their lowest. Necrotic ulcers in the mouth were absent throughout. A respiratory infection coming on top of a meningitis could account for the greater part of the constitutional symptoms observed, but there was some clinical indication that this might not be the whole explanation; it was this clinical impression which led to the search for some other possible explanation. The search was justified by its results, and confirms the importance of blood examination when there is a secondary rise in temperature or when not entirely accountable symptoms