means of a syringe, or by more frequent washings, as in the Carrel-Dakin treatment, will depend on various factors, such as personal predisposition, nursing facilities and idiosyncrasies of the patient. It would seem natural to suppose that quicker and surer results could be obtained by the two-hourly flushings of Carrel-Dakin than the daily or twice daily flushings by syringe when dressing the case.

Conclusions.

1. Most if not all cases of empyema can be cured without resort to such mutilating and dangerous operations as thoracoplasty and pulmonary decortication, because it is not necessary to obliterate the pleural space, but merely to render its walls sterile, and this is possible by frequent washings with hypochlorite solutions.

2. Flushing of the pleural cavity with hypochlorite solution is not dangerous provided there is a free exit for the fluid, as by the use of a double drainage tube.

3. The earlier this treatment is instituted the sooner and more surely will a cure be obtained, as there will be less formation of granulation and scar tissue, and the organisms will not have had time to entrench themselves deeply in the tissues, and will therefore be more exposed to the action of the antiseptic.

4. The drainage should be free, and the opening should not be valvular; if a Carrel tube is used it should be introduced as far as possible into the pleural cavity through a large drainage tube, so that the fluid, starting at the most remote part of the septic space, passes down over its walls and escapes freely between the Carrel tube and the drainage tube.

5. The opening is most suitably made by removing one inch of the eighth rib in the scapular line, as this is the most dependent spot for practically all positions, and there is no fear of its closure by the rising diaphragm.

PARALYSIS FOLLOWING ARTERIAL INJURIES.

BY

CAPTAIN HAROLD BURROWS, R.A.M.C.(T.F.).

AMONG the accidents of war injury of a main blood vessel is common, yet little inquiry appears to have been made into the effects upon the structures which lie within the area of distribution of the wounded vessel. Injury of a main artery—for example, the common femoral, the popliteal, or the brachial—may produce immediate and remarkable consequences in the affected limb. As the ensuing symptoms are paralytic they have been apt to be regarded either as “functional” or as arising from concomitant nerve lesions, for, owing to the close association and common anatomical relations between the vessels and the nerves, simultaneous injuries of the structures are of frequent occurrence.

As the following cases will show, extensive paralytic phenomena may follow a vascular lesion independently of any direct traumatism of the main peripheral nerves. That these sequences are not infrequent is shown by our experience at the — General Hospital, and also by knowledge gained at the other military hospitals in the neighbourhood.

FIG. 1.—Shaded area shows extent of loss of sensation to light touch, the upper limit of which is marked by 1; 2 defines the upper limit of loss of sensation to pin-pricks.

The patient complained of pain in the hand and fingers and a “tingling, horrible sensation” in the same area. He said his

“funny bone” was tender, and that pain shooting down to the fingers could be caused by pressure applied to the ulnar nerve at the elbow.

There was anaesthesia to light touch and pressure, as shown in Fig. 1, and it is notable that the loss of pressure sense was slightly greater than the loss to light touch. There was also complete loss of sense of position in the wrist and all joints below the wrist. There was absolute paralysis of all muscles below the elbow. The right hand was warmer than the left. The radial pulse could be felt, but the pressure within it was too low for accurate estimation with Riva-Rocci apparatus. A pressure of less than 20 mm. of mercury was sufficient to obliterate the pulse. The maximum systolic blood pressure in the left arm was 118.

There can be no question that the clinical symptoms in this case cannot be accounted for by peripheral nerve injury, for the musculo-spiral escaped damage, and yet the forearm muscles were completely paralysed.

CASE II.

Q.M. Sergt. W. G. D. Wounded on June 23rd, 1917, was admitted on June 25th, under the care of Captain E. T. Tatlow, R.A.M.C.(T.F.). The note from the casualty clearing station was: “Through wound of left arm and forearm, passing through bend of elbow and wounding brachial artery. Wound explored through separate incision at bend of elbow. Artery tied. Bipp dressing. Packing for serious oozing. Scalp wound cleaned.”

There was a small entry wound on the inner posterior surface of the left arm, with a small exit wound on the radial side of the anterior surface of the forearm. Both wounds had been excised, bipped, and firmly plugged with gauze. There was an oblique operation wound in front at the bend of the elbow, and this also had been sutured. The limb was fixed on an internal rectangular splint. There was no injury to the bones or to the elbow-joint, the projectile having passed anteriorly to these structures. The left hand and forearm were much swollen and colder than the right hand and forearm, and there was considerable bruising in the neighbourhood of the elbow. No radial pulse could be felt. He complained of “pins and needles” in the left hand, from the wrist to the tips of the fingers. He felt this especially if he coughed, sighed, or took a deep breath.

There was “glove” anaesthesia of the left hand (Fig. 2), including the loss of appreciation of light touch, pin-pricks, pressure, tuning-fork vibrations, and sense of position; further, there was complete paralysis of all the muscles below the elbow, except the supinator longus and extensor carpi radialis longior, which were not paralysed. In this connexion it may be noted that the blood supply of these two muscles is derived from the superior profunda artery. The paralysed muscles felt hard and inelastic. On July 9th it was noted that there had been no material alteration in the loss of power or sensation since the patient's admission. The oedema had become less, but it had not disappeared, although the limb had been elevated; the hand was now warm.

FIG. 2.—Shaded area shows loss to light touch; 2 shows upper limit of loss to pin-pricks; and 3, the upper limit of loss to deep pressure. The area of loss to light touch alone has been defined on the palmar surface owing to discomfort caused to the patient during examination of this region. The arrows show the direction of the wound.

In this case it is clear that the ulnar and median nerves may have been injured, but it is equally clear that the musculo-spiral escaped. Consequently, some factor additional to peripheral nerve injury is required to explain the symptoms.

CASE III.

Pte. S. U. F., wounded August 23rd, 1917, was admitted on August 25th under the care of Captain J. H. C. Fegan. He presented three wounds: (1) Gunshot wound of left leg, with compound fracture of left tibia and fibula; (2) a large vertical wound in the left deltoid region, tightly plugged with gauze, over which the skin was sutured; (3) a long vertical wound on the inner side of the left arm, also plugged tightly with gauze and sutured. The notes accompanying the patient from the casualty clearing station did not mention any arterial injury.

The patient had pins and needles in the left hand for the first twenty-four hours, but this symptom disappeared. He complained of a “feeling of pressure” in the hand. The radial pulse could not be felt. There was moderate oedema of the forearm and hand, and the muscles felt hard, inelastic, and tender. The fingers and thumb were flexed at all the joints.

The wrist was neither flexed nor extended. All the muscles below the elbow were completely paralysed. Sensation for light touch, pin-pricks, and pressure was lost up to the level of the wrist (Fig. 3).

FIG. 3.—Shaded area represents loss of sensation to pin-pricks; 2 marks the upper limit of anaesthesia to pressure, and 3 the upper limit of anaesthesia to light touch. On the palmar surface the loss to pin-pricks alone was investigated, as the examination of this region caused the patient to suffer pain.

CASE IV.

Pte. E. G. was wounded on May 3rd, 1917. The note from the casualty clearing station was: "Femoral vessels tied." When admitted under the care of Captain J. H. C. Fegan there was a vertical operation wound on the antero-internal aspect of the right thigh, and a similar one at the same level over Hunter's canal. A large drainage tube connected these two wounds. The note taken on May 31st recorded an extensive area in the leg and foot, as shown in Fig. 4, where sensation to light touch

FIG. 4.—The shaded area represents loss of sensation to light touch.

was lost (other sensations were not tested). There was persistent foot-drop with rectangular talipes. The peronei were hard, bulky, and weak, but not completely paralysed. The extensor longus hallucis and all the other anterior leg muscles were weak, though not completely paralysed.

In this case, imperfect as the record is, it is still clear that there is a muscular weakness and a loss of cutaneous sensibility that cannot be accounted for by any direct injury of the peripheral nerves.

CASE V.

Pte. T. P., wounded on June 13th, 1917, was admitted on June 16th, under the care of Captain Graham-Jones, R.A.M.C. The note from the casualty clearing station was: "Gunshot wounds left thigh (three wounds). Haemorrhage. No posterior tibial pulse. All wounds excised. External branch from popliteal artery ligated. Also perforating branches of the femoral. Bipp and paraffin gauze. Progress: Foot cold and white for twenty-four hours, then signs of returning circulation; can now feel toes. Dressed. Slight discoloration over instep and tips of toes. Wound healthy."

On admission to a general hospital on June 16th, under the care of Captain Graham-Jones, the toes and a portion of the dorsum of the foot were cold and discoloured. Tight gauze packing in popliteal wound. No pulsation was felt in dorsalis pedis or posterior tibial. The whole left leg was swollen and hard; the foot was warm except in the discoloured area. There was no loss of sensation to pin-pricks except in area of gangrene, but there was absolute paralysis of all muscles below the knee except the soleus and gastrocnemius, and these were capable of the slightest movement only. Amputation above the knee was performed on June 27th.

Examination of the leg showed a striking change of colour of all the muscles of the leg, which were pale, and in their lower portions had hardly a tinge of red. The gastrocnemius was affected in its lower two-thirds and the soleus in its lower third. The upper portions of these muscles were of normal colour and looked healthy, and the transition from this healthy muscle to the pale altered tissue below was gradual, the muscle in the intermediate zone having a streaky appearance, bundles of red fibres being interspersed among bundles of discoloured fibres.

The pallor of the affected muscles was striking and at once arrested attention as an abnormal condition. Portions of these muscles were sent to Captain A. W. Stott, who prepared sections of them for microscopical examination. His report is given later.

CASE VI.

Pte. H. T. was wounded on June 16th, 1917. The note from the casualty clearing station was: "Penetrating wound of right thigh just below Poupart's ligament. Artery three-quarters severed; artery ligated. Bled freely from lower part. Pulse felt in left foot."

The note made on June 24th recorded that the patient had early gangrene of the toes and the neighbouring portion of the foot. There was absolute paralysis below the knee, and the muscles felt hard and stiff. Amputation was performed above the knee. The muscles of the leg were pale and bloodless. Unfortunately no observations were made on the cutaneous sensibility in this case.

CASE VII.

Shoering-smith J. S. was wounded on July 17th, 1917. The note from the casualty clearing station was: "Shell wound of thigh, left, July 18th, 1917. Wound excised; foreign body removed; superficial femoral and deep femoral veins divided—ligatured; bipped and sutured." He was admitted on July 19th under the care of Major Moffat. The note made on the following day recorded a transverse sutured wound $\frac{7}{8}$ in. long on the front of the left thigh, 4 in. below the centre of Poupart's ligament. There were no gross signs of inflammation in the neighbourhood of the wound, and the patient seemed fit except for his pallor; he said that he had lost much blood when wounded. No pulsation could be felt in the posterior tibial or dorsalis pedis of the wounded limb, but blood was circulating in the foot and toes, and there was no gangrene or blistering.

The patient complained much of subjective sensation in his left foot and leg, which he described in these words: "My foot feels as if it were being drawn up tight in an onion net: the feeling works up into my ankle and shin, and keeps me from sleeping; it is a most unpleasant sensation; you would think my foot was coming off."

There was extensive impairment of sensibility of the leg and thigh, as shown in Fig. 5, but it was particularly noticeable

FIG. 5 (Case VII).—Shaded area represents loss of sensation to pin-pricks.

that the boundary of the anaesthetic area was not perfectly definite—that is to say, there was an intermediate zone, perhaps 1 in. wide, in which pin-pricks sometimes were and sometimes were not felt. There was a small area on the anterior surface of the thigh in its lower third where the anaesthesia was similarly incomplete. There was loss of sense of position in all joints distal to the tarsus. Deep pressure sense was lost up to a level about 2 in. above the ankle-joint. The boundary line of this loss was slightly irregular—that is to say, it was serrated, but might be described as belonging to the stocking type. There was complete paralysis of the extensor longus hallucis, and of all the flexors of the toes. There was very slight power in the long extensors of the outer four toes. The tibialis anticus and peronei were weak but not completely paralysed. The gastrocnemius, soleus, and tibialis posticus were nearly normal, though there was some obvious weakness in these also.

CASE VIII.

Sapper T. T., wounded on July 31st, was admitted, under the care of Major Moffat, on August 1st. He stated that he did not lose much blood after being wounded, and that no tourniquet was applied. There was an entry wound on the outer side of the left knee-joint, level with the lower end of the patella and situated over the biceps tendon; there was no exit wound. There was copious effusion into the knee-joint, and a systolic bruit could be heard in the popliteal space. The posterior tibial and dorsalis pedis could be felt pulsating. On August 5th the leg was oedematous. He had "pins and needles" in the foot, and there was an extensive area of loss of sensibility, as shown in Fig. 6. The gastrocnemius and soleus were active, and there was perhaps the slightest amount of activity in the tibialis anticus; but with these exceptions there was absolute paralysis of all muscles below the knee.

The knee-joint was needled and dark blood withdrawn; the pathologist (Captain A. W. Stott) reported: "No increase of polymorphs. No organisms seen."

On August 6th the popliteal artery and vein and the anterior and posterior tibial arteries were ligated. The artery had been wounded at its bifurcation. The resulting arterial haematoma communicated with the knee-joint through a rent in the posterior part of the capsule. The internal and external popliteal nerves were seen to be uninjured. On August 7th some recovery of sensation on the dorsum of the foot was noted, but sensation was still absent from the sole. Sensation in the left foot had completely returned on August 8th, and all movements had returned, though the muscles previously paralysed were still weak. By August 13th the muscles had recovered almost completely, and their contractions were strong, except that some weakness remained in the gastrocnemius and soleus. This weakness may have been reflex in origin, being merely an unconscious attempt to maintain rest in the popliteal region.

FIG. 6 (Case VIII).—The shaded area represents loss to light touch; 2 is the upper boundary of loss to light pressure; 3 the upper limit of loss to pin-pricks.

CASE IX.

Lieut.-Cpl. H. W., wounded on August 5th, was admitted on the following day. There were small entry and exit wounds in the left arm, on the inner side, just above the elbow. The projectile had passed in an antero-posterior direction close to the bone. He said that he lost a great deal of blood when he was hit—the blood “pumped out.” A tourniquet was applied over the wound, and not removed until about one and a half hours later.

FIG. 7 (Case IX).—The shaded area represents loss to pin-pricks and light touch. The dotted area marks a partial loss of sensation to pin-pricks. The wounds of entrance and exit are indicated by small circles.

The interesting point in Case IX is that the anaesthesia extended far above the level of the vascular and nerve injury. We may explain the phenomenon by referring it to a “functional” origin, if that is any explanation. Or we may regard the condition as being reflex in nature, but we can hardly attribute it to ischaemia.

CASE X.

Cpl. W. J. B. B., wounded on August 21st, 1917, was admitted, under the care of Captain J. H. C. Fegan, on August 23rd. The note taken on the following day recorded a small entry wound in the left leg on a level with the lower border of the patella and situated on the outer side of the back of the leg just behind the tendon of the biceps. There was no exit wound. The moment he was hit he lost all sensation in the leg, his first thought being that the leg had been blown off. There was considerable oedema of the leg, and he stated that if the leg were allowed to hang down it swelled and became very painful. The muscles did not feel hard. No distinct difference could be felt in the surface temperature of the two feet. An arterio-venous aneurysm had formed in the popliteal region, as shown by a palpable thrill and a loud systolic murmur and venous hum. The posterior tibial could be felt pulsating, but the amplitude of its pulse wave was small and the tension low.

The patient had no “pins and needles” or other subjective sensation in the leg while he was at rest in bed. Sensation to pin-pricks and light touch was lost over an area which, as shown in Fig. 8, extended far above the level of the wound. It

was not complete, for there were areas lower down where both light touch and pin-pricks could be appreciated. The sole of the foot and front of the leg contained such areas. The upper border of the general area of anaesthesia was indefinite, there being a zone in which some pin-pricks were felt, while others were not noticed. The muscles of the leg and foot were parietic, and apart from weak contractions of the gastrocnemius, tibialis anticus, and long extensor of the toes, there was no muscular power in the leg at all. By August 25th the oedema of the leg had disappeared, muscular power had increased, and sensation was returning.

FIG. 8 (Case X).—1 represents loss to light (shaded area), 2 is the upper limit of loss to pin-pricks, 3 the upper limit of loss to light pressure. In the dotted areas loss of sensation to pin-pricks was incomplete, deep prickings being appreciated, while only a percentage of light prickings could be felt.

SYMPTOMS.

From these cases it will be seen that the main symptoms are:

1. Subjective sensations in the distal part of the affected limb.
2. Anaesthesia, more or less of the “stocking” or “glove” type, and involving all kinds of sensation, including light touch, pin-pricks, and deep pressure.
3. Muscular paralysis.
4. In certain cases hardness and inelasticity of the muscles.
5. Oedema.

In all of the cases in which no arterial pulse could be felt distally to the injury (I to VIII) the patients complained of subjective sensations, which they described in various terms, but to which we may refer as “pins and needles.” On the other hand, in the two cases (IX and X) in which a distal pulse could be felt there were no “pins and needles.”

In all the cases which were tested except one there was loss of cutaneous sensibility; oddly enough, in the one (Case V) in which sensation was retained (except in the toes, which became gangrenous) there was not only gangrene of the toes, but most extensive myopathy was shown to be present in the muscles of the leg. In the two cases in which a distal pulse could be felt (IX and X), the area of anaesthesia was most extensive, and reached to a level considerably above the wound. In several of the cases the loss to pin-pricks was greater than the loss to light touch.

Motor paralysis was present in all. With one exception, in every case in which no distal pulse could be felt there was a feeling of hardness and inelasticity of the affected muscles which suggested that alterations in their composition had occurred. The exception (Case VIII) was of special interest, inasmuch as the muscular and sensory paralysis rapidly passed away after a wounded popliteal artery had been ligated together with the vein. In the two instances (IX and X) in which sensory and motor paralysis were present, while the distal pulse was not obliterated, the muscles were flaccid and soft, in contrast with the hard muscles felt in the cases of complete arterial lesion.

No case of paralysis was observed as a sequence of injury to the radial, ulnar, anterior tibial, or posterior tibial arteries. In three of the cases in the series (II, III, and V) the wounds were plugged with gauze at the time of the patient's admission to the — General Hospital, and the pressure of this packing may have played a subsidiary part in the causation of symptoms.

It is a little difficult to understand why ligature of an artery in one patient should lead to little or no evil consequences, while in another, seemingly as favourable at the time of operation, grave consequences follow.

Take the following case as an example:

CASE XI.

Pte. G. T. B., wounded on August 22nd, was admitted, under the care of Lieutenant C. N. Longridge, on August 25th. The note from the casualty clearing station was: “Compound comminuted fracture of lower end of left humerus. Brachial artery ligated. Great loss of muscular tissue.”

On admission he presented a through-and-through wound above the left elbow. The original wounds had been excised, and partly stitched over gauze packs. The arm was fixed on a Robert Jones elbow splint. No radial pulse could be felt. There was much oedema of the hand, forearm, and arm.

This case certainly had many of the features which we should regard as favourable to paralysis of vascular origin, for his brachial artery had been ligated, he had a fracture close above the elbow-joint, the wounds were tightly packed, the arm was splinted, and there was copious oedema. Yet there was no paralysis at all. The case is mentioned merely as a contrast with the others and to illustrate the fact that whatever the actual cause of angiotic paralysis may be, it is not clearly defined at present.

PATHOLOGY.

So far these observations have been easier to make than to explain. And the probability is that pathological explanations must remain in abeyance for the present owing to insufficiency of data. Nevertheless, it may be a matter of interest to wander a little into the field of theory.

The attractive and simple course of attributing all the phenomena to ischaemia—to an insufficient supply of blood to the tissues—at once suggests itself, and, if we follow this course, we may describe the symptoms collectively under the title of ischaemic paralysis, and be done with all further speculation. Objections, however, to this title become apparent on close scrutiny. In the first place it involves the acceptance of a hypothesis of causation which at present is not fully established. The symptoms may be due to the small quantity of blood which is circulating, to injury of the sympathetic nerves in the vessel sheath, to the low pressure of the blood stream below the injured artery, to cold, to some other cause altogether, or to a combination of causes. Moreover, although some of the cases just described show that a paralysis similar to the so-called Volkmann's ischaemic paralysis often does follow occlusion of an artery, yet it is clear that this is not the only form of paralysis which may follow a vascular lesion.

For these reasons it appears better to adopt a general title which will not implicate us in any hypothesis other than that the symptoms are consequent upon damage to a blood vessel. Accordingly the term "angiotic paralysis" may be applied with convenience to the symptoms described in these cases.

At the outset we must assume that two factors at least take part in the causation of the symptoms. On the one hand, we may have extensive degeneration of muscle and massive gangrene, without any loss of cutaneous sensibility, except in the area of mortification, while on the other there may be widespread loss of sensation and muscular power without gangrene, without gross myopathic change, and even without obliteration of the distal pulse. From this it is clear that there is no exact correlation between the neuropathic and the myopathic symptoms.

Probably ischaemia is one of the factors, the one which is associated especially with Volkmann's myopathy. In the cases just narrated this condition has been seen only in those cases (I to VII) in which the distal pulse was obliterated, which is significant. If, however, we accept ischaemia as the cause of this myopathy, there must be at least some other cause for the flaccid paralysis and sensory loss which are to be observed after certain vascular injuries in which the artery is not completely divided or obstructed, and in which the distal pulse can still be felt. The fundamental cause in these cases seems to be the actual damage to the arterial wall or to its sheath, rather than any consequent effect upon the supply of blood to the limb. Further, it appears that an incomplete injury—that is to say, an injury in which the artery is not completely severed—is more likely to be followed by extensive sensory loss and flaccid paralysis than is a complete division or ligation of the artery. There is reason to suppose that these symptoms are reflex in nature. Accepting this to be so, we arrive at the following classification of paralysis following arterial injuries:

Let us consider these two factors in detail. The first is illustrated well by Case v. Here a wound in the popliteal

region was followed by absence of pulsation in the dorsalis pedis and posterior tibial arteries. The foot became cold and white, and remained so for twenty-four hours, at the end of which time there were signs of returning circulation, except in the toes and a small portion of the foot, which became gangrenous. The whole leg up to the knee was slightly oedematous; the muscles were hard and absolutely paralysed. Inspection of the muscles after amputation showed them to have undergone a striking change of colour—they were no longer red, but quite pale and almost buff-coloured. This change affected some of the muscles more than others, and the change was more complete in the distal than in the proximal ends of the muscles. What the actual change and its cause may be we do not know, though the suggestion has been made that it is identical with rigor mortis. Sections of the muscles in this case were made by Captain A. W. Stott, who gives the following report:

Pathological Report.

"Portions of the peronei and gastrocnemius muscles were cut; each showed gross pathological changes. No normal muscle fibres were seen. In sections stained by haemalum and eosin the general impression was that given by an anaemic infarct. The muscle fibres and the interstitial tissues stained a uniform pink. The fibres showed various stages of degeneration—some had almost normal striation but no nuclei; others had completely lost their striation, and appeared as granular masses. Fibres showing different degrees of change were often found next to each other. The majority of fibres appeared swollen. Some were broken up into irregular masses; others were split into longitudinal fibrillae or transverse discs. There was no hyaline degeneration, and no gross fatty change was seen. The interstitial tissue appeared oedematous, stained a faint pink with Van Gieson, and was almost cell-less. There were few capillaries. Throughout the sections there was but little trace of inflammatory reaction. The large vessels, arteries, and veins contained blood, not clot, and appeared normal."

These cases (I to VII) represent the first main variety of angiotic paralysis. The condition is, for the most part, a muscular phenomenon; it is well known as Volkmann's ischaemic paralysis, following especially fractures about the elbow-joint, and may be seen apart from any material sensory defects. The damaged muscles do not recover, and any resumption of function which comes about is attributable to the fact that portions of the muscles, especially the proximal portions, usually escape and are capable of limited activity later on.

Summary Concerning Ischaemic Cases.

1. An arterial injury with obliteration of the distal pulse.
2. Subjective sensation of "pins and needles."
3. Muscular paralysis; the muscles being hard and inelastic to the touch.
4. Anaesthesia of a "stocking" or "glove" distribution, confined to the portion of limb which is distal to the injury, and involving all forms of sensation.

Reflex Paralysis.

The cases (VIII to X) in which the paralysis has seemed to be of a reflex nature have the following characteristics:

1. An arterial injury without complete blockage of the vessel.
2. Absence of "pins and needles" sensation.
3. Flaccid paralysis of the muscles which do not feel hard and inelastic.
4. Widespread loss of cutaneous sensibility, extending in two instances well above the level of the wound.

These cases give the impression of being less organic, if one may put the matter in that way, than the ischaemic ones. Case VIII showed almost complete recovery within two days of an operation which involved ligation of the popliteal, anterior and posterior tibial arteries and veins. Case X improved up to a certain point with no treatment other than rest in bed. Case IX was not under observation long enough for any improvement to be observed.

The hypothesis that these cases are of a reflex nature

has been adopted partly for convenience of argument, but largely because this explanation seems plausible. The fact that the anaesthesia in two of the cases extended well above the wound has led to the suggestion that it is of a functional nature. But even if we call it functional, does that carry us any further? Several controls have been tried, and patients with fractures, wounded joints, and other bad wounds of the limbs were tested, but in no case was a stocking anaesthesia discovered except in those who had arterial lesions.

Whatever the cause of the paralysis may be in these cases, it can hardly be attributed to ischaemia. In this connexion, it may be well to recall the cerebral disturbances which are apt to follow sudden obliteration, by operation or otherwise, of the lumen of the common carotid or internal carotid artery. These disturbances, severe and not rarely fatal, surely cannot be attributed to ischaemia, seeing what a liberal provision of collateral channels is present for supplying the brain with blood, and considering also the rapidity with which the cerebral symptoms develop.

CASE XII.

Pte. O. K., wounded on September 1st, 1916, was admitted on September 9th. He had been shot through the right neck. He stated that his left arm became paralysed at the instant he was hit; in fact, he thought the wound was in his arm. The projectile had clearly divided the internal carotid artery.

It is difficult to believe, in the absence of better evidence, that such an immediate paralysis could be brought about by ischaemia or embolism of the brain.

GENERAL CONCLUSIONS.

The currents of thought started by these cases are, however, not likely to lead us into any useful channel until we have a great many more clinical records to work upon, together with additional observations, including the electrical reactions of the affected muscles, the deformities produced, and the prognosis. It has not been possible to gather information on these points.

Although the importance of these paralytic effects of arterial injuries cannot at present be estimated, yet, until we are better informed as to prognosis, we may well be more reluctant in the future than we have been in the past to tie a main artery for the arrest of haemorrhage. We shall be more particular and persistent in our endeavours to find the exact source of bleeding, in the hope that it may prove to concern a branch only instead of the main trunk. And, in those instances in which the main trunk itself is wounded, we shall be more disposed to cure the leak by suture of the vessel, or failing this, to attempt an escape from the ill consequences of direct ligation by the temporary use of a Tuffier tube. Furthermore, when we are compelled by the nature of the case to tie a main artery, we shall take care to tie the main vein also; because, as Sir George Makins has pointed out, the results which follow ligation of both vessels are better than those which ensue upon a blockage of the main artery alone.

LITERATURE.

Though there is not much literature on this subject there is enough to give food for thought.

In the first place, the association of so-called ischaemic paralysis, of the type described by Volkmann, with injuries involving complete blockage of an artery, has been recorded by J. G. Sherrill,¹ Carl Hirschmann,² L. Verdelet,³ and others, although the association does not appear yet to have become a matter of general surgical knowledge. Further, H. Meige and Mme. Athanassio-Benisty⁴ have observed that the vasomotor, trophic, and secretory disturbances found in certain cases of nerve injury are not due directly to those nerve lesions, but are dependent on coincident arterial lesions, while Leriché⁵ has noted not only that so-called ischaemic paralysis may follow obliterating lesions of an artery, but has put forward the suggestion that the symptoms are the result not of the arterial wound, but of the concomitant injury to the sympathetic nerves which accompany the artery. He states, moreover, that if in such a case the whole of the obliterated segment of the artery be removed together with its nerve sheath the major part of the troubles disappear, the hand becomes warm, its colour changes, and the skin resumes its normal appearance, the fingers lose their stiffness in an appreciable measure, and, finally, the

pulse reappears at the wrist. Leriché has been able to demonstrate this in six cases, and in all the transformation was identical.

It is clear that if this pathology be established there is justification for a new title for paralysis of vascular origin, a title which shall include the two factors of ischaemia and sympathetic reflex disturbance.

Acknowledgments.—In conclusion, I should like to express my thanks to the medical officers, under whose care the cases quoted have been, for their friendly co-operation, to Colonel C. A. Ballance and Colonel Gordon Holmes for several helpful suggestions.

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A CASE OF RAT-BITE FEVER TREATED SUCCESSFULLY BY INJECTIONS OF NOVARSNOBILLON.

BY

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RAT-BITE FEVER—Sodoku of the Japanese—is a comparatively rare disease. It was recognized, quite a long time ago now, by Miyake and others in Japan as a definite entity. Cases have also been reported from East Africa (Middleton, Horder, Cruickshank), in the United States of America, in France, and in Italy (Frugoni). As far as we are aware, our case is the first reported from East Africa or the tropics proper. The finding of the causative agent is of recent date. In 1915 Futaki and his fellow workers demonstrated the presence of long spirochaetes in the lymph glands of cases on the tenth to thirteenth day of the illness. Later they discovered in man, in the wound of the rat-bite, and in the blood of inoculated mice short, thick spirochaetes, and at first were unable to determine which of these two was the cause of the disease. Recent observations have, however, shown that the long and short forms are the same species, the long ones being old and found almost exclusively in human tissues, while the short ones are the young forms, and can be found in the blood of patients with the disease and in experimental mice (Kaneko and Okuda). Futaki's work has been confirmed by Kaneko and Okuda,¹ and it seems quite clear that rat-bite fever is another spirochaetal disease. According to the authors last named, the parasite can be found in the blood during the height of the disease.

Hata, in a paper published in 1912,² refers to eight cases of rat-bite fever treated by salvarsan. The results were striking; all symptoms quickly disappeared, and most of the cases were cured. Two relapsed, but in them the dose had only been small—namely, 0.3 and 0.4 gram. Since then other cases treated in a similar manner have been reported upon, and the recent discovery that the malady is a spirochaetal one fits in well with the undoubted efficacy of salvarsan and its derivatives in curing it.

The following is the history of a case we have treated recently:

Mr. H., aged 25, on January 31st, 1917, when in East Africa, was bitten by a rat on the back of the right forearm. He was sleeping in a mosquito net, and the rat attacked him through this, inflicting a severe bite. This healed up and was practically gone when, some seven days later, pain commenced at the site and the place swelled up. Fever then appeared, and with this a rash broke out on the body. Later the glands in the right axilla enlarged, and following this a general adenitis was noticed. On the twelfth day, after onset of symptoms, the bite, which had now broken down, was lanced and some pus escaped. About the twentieth day severe joint and bone pains came on, and the throat also became inflamed and very sore. The condition improved, but quickly relapsed, and between that time and the present (January 31st, 1918)—namely, a year—patient can count nineteen relapses at varying intervals. There was a severe one in May, 1917. Complicating the real rat-bite symptoms, however, the patient had also suffered from malaria, and it is not certain which of the two infections was responsible for the different bouts of pyrexia. Malarial parasites were found in the blood in August, 1917, during an attack