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DR. MABANE studied medicine in Edinburgh, the city of his birth. After practising briefly, he is believed to have acted as Surgeon’s Mate on one of the King’s vessels. Following this experience, he sailed to America to join Amherst’s forces, landing in New York in 1758. He was at Crown Point, N.Y., 19 days before the invasion of Quebec.

A letter of introduction from Lord Elmbank to his son, General Sir James Murray probably resulted in Mabane’s remaining to practise medicine in Quebec after the conquest. When Murray became Governor in 1764, he named Mabane to his first Council and appointed him a judge of the Court of Common Pleas and of the Surrogate Court.

Mabane not only continued his medical work but also served as a Councillor and on the Bench under three Governors, Murray, Haldimand and Carleton (Dorchester), the latter of whom removed Mabane from the Council in 1767 only to reinstate him in 1774. Dr. Mabane remained on the Bench throughout, however, and his judgments were noted for clarity and regard for the common weal—a fact which won him many friends but also a few unscrupulous enemies who made strong but unsuccessful efforts to unseat him in 1783.

When American Invasion under Benedict Arnold threatened in 1775, Mabane was entrusted with many important missions and supplied lists of parishers and old officers of militia who would serve. He was Surgeon of the Garrison Hospital when Carleton arrived after fleeing from Montreal.

Although Dr. Mabane maintained his connections with the General Hospital and the Garrison Hospital while pursuing his career as a Jurist, he gave up his private practice.

Dr. Mabane was unmarried. He died on January 5th, 1792, from pneumonia due to a cold contracted on the Plains of Abraham where he lost his way in a blizzard. He had a sister Isabel, who survived him.

The example set by pioneer men of character like Dr. Mabane in helping to establish a sound foundation for the practice of medicine in Canada, inspires this organization to maintain with unceasing vigilance its policy—Therapeutic Exactness and Pharmaceutical Excellence.

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Peripheral Nerve Injuries---A Review
By Ramsay W. Gunton, '45

Incidence and Importance

Injuries to the peripheral nerves are not commonly encountered in civilian practice. During war-time, however, they assume considerable importance and more complete knowledge of them is required by the Army Medical Officer than by the civilian practitioner. In the war of 1914-1918, peripheral nerve injuries comprised two per cent of all injuries and 20 per cent of injuries to the extremities. In this war, the widespread use of land mines has increased the number of injuries to the extremities, so nerve injuries have correspondingly increased in frequency, now comprising 10 per cent of all casualties. Radial, median and ulnar nerve injuries comprise no less than 60 per cent of the total, followed in order by: sciatic, peroneal (lateral popliteal), posterior tibial, axillary, musculocutaneous and femoral nerves.

The Anatomy of the Peripheral Nerves

It will be recalled that peripheral nerves contain efferent motor fibres, afferent sensory fibres and sympathetic fibres (both afferent and efferent). Thus a transection of such nerve would result in paralysis, anaesthesia and vasomotor changes in the area of distribution of that nerve. Nerve fibres, correctly speaking, are axis cylinders of nerve cells located in the spinal cord. Upon leaving the gray matter of the cord, many of these axis cylinders receive a myelin or medullary sheath, composed of a phospholipid compound. Upon leaving the white matter of the cord, the axis cylinders are invested with a thin layer of cells known as the neurilemma sheath or sheath of Schwann. In the nerve trunk, the axis cylinders arrange themselves in groups, destined for a certain skin area or muscle. These small bundles are called funiculi. A peripheral nerve trunk is, therefore, composed of many funiculi, bound together by a delicate connective tissue containing blood vessels. The connective tissue between adjacent axis cylinders is known as endoneurium; that between adjacent funiculi as perineurium, and the outside covering of the nerve trunk as epineurium. The concept of funicular anatomy cannot be over-emphasized. When it is realized that
one funiculus has such a definite muscle group or skin area to supply, it is obvious that when nerve suture is performed, the operator must attempt to appose corresponding funicular ends in order that regenerating axones may follow the correct pathway in reaching their destination. Axial disalignment is an important cause for failure to regain function in peripheral nerve surgery.

NERVE DEGENERATION

When a peripheral nerve is divided, certain definite changes occur in both the proximal and distal ends. (a) Proximal end—The cell body of the divided axis cylinder becomes swollen; its nucleus becomes indistinct and eccentrically placed; its Nissl granules fade or undergo complete dissolution. If the division occurs very near the cell body, the latter may disintegrate completely. However, if the division is some distance along the nerve trunk, then the cell body gradually resumes normal form and begins to regenerate its divided axis cylinder. (b) Distal end. Because its trophic center, the nerve cell body, has been cut off, the distal part of the axis cylinder undergoes complete degeneration. The neurofibrils of the axis cylinder first become fragmented and then disappear. The myelin sheath is dissolved into small globules. The cells of the sheath of Schwann proliferate and assume phagocytic characteristics. They remove the fragmented neurofibrils and myelin globules and then multiply to such an extent that they form solid cords of cells occupying the position of the main degenerated axis cylinder. These cords of neurilemma cells are called band-fibres (Bandfasern) and it is believed that regenerating axis cylinders are guided along their course by them.

DIAGNOSIS AND EXAMINATION OF PERIPHERAL NERVE INJURIES

The diagnosis of individual nerve lesions is too extensive a subject to be covered in this review. It requires a detailed anatomical knowledge of muscles, nerves and cutaneous innervation. In this section we shall discuss some important points in the examination of nerve injuries.

(1) History of the Injury. This should include the type of injury (i.e. whether caused by a bullet wound, shrapnel, compound fracture, lacerating wound, bruise, etc.) as well as the time of onset of paralysis or anesthesia. If the paralysis were immediate, an anatomical interruption or division of the nerve is indicated. If the onset were gradual it indicates a progressive compression of the nerve by callus or scar tissue.

(2) Examination of the Site of Injury. This gives an idea of the nerve lesion. Careful palpation of the site may reveal presence or absence of anatomical continuity of the trunk as well as the presence of intra-neural fibrosis if the lesion be of some duration.
(3) **Motor Examination.** This tests the patient’s ability to perform various movements. It is stressed that one should beware of false movements, i.e. where one muscle group compensates for an absent one.

(4) **Atrophy and Tone of Muscles.** In peripheral nerve injuries disorders of the flaccid type exist.

(5) **Vasomotor Symptoms.** At first, interruption of sympathetic fibres is manifested by vaso-dilatation and hyperaemia. After 15 days, the cold phase commences, characterized by pallor, cyanosis and lowered temperature of the skin.

(6) **Electrical Examinations.**

(a) **Faradic response.** The response of denervated muscles to faradic stimulation is lost as soon as the nerve is divided. Of all the electrical tests this is the last to return.

(b) **Galvanic.** A normal muscle will respond to galvanic stimulation when the electrode is applied at its “motor point”. When the nerve to a muscle has degenerated, the maximum response is elicited from a point near the tendon of the muscle, for there the maximum number of muscle fibres are concentrated. This phenomenon is known as descent of the motor point.

(c) **Chronaxie.** This is defined as the length of time a current of twice the rheobase must be applied to a muscle before it responds. Chronaxie can be measured by a special instrument and is expressed in milli-seconds. It is increased 100 times in upper motor neurone lesions but in lower motor neurone lesions (e.g. peripheral nerve injuries) only four times. Diminution in the chronaxie is a valuable prognostic sign.

(7) **Sensory Examination.** This should designate the areas of anaesthesia and analgesia. As the nerve recovers, diminution in these areas should be watched for. An important feature in this examination is to determine the type of sensation lost, i.e. affective (protopathic) or discriminative (epicritic). The former perceives pain (e.g. pin-prick and temperature). The latter mediates minor variations in temperature, position sense and two point discrimination. As the nerve regenerates, affective sensation returns first. Discriminative sensation returns later or not at all. Thus, in nerve injuries involving the muscles of the hand where discriminative sensation has not returned, difficulty will be encountered in performing skilled movements such as piano playing. This difficulty will be present despite the fact that all muscular movement has returned.

(8) **Trophic changes.** These are often found where a nerve lesion has been present for a long time. Dry skin, brittle nails, ulcers and susceptibility to gangrenous change are common features.
Regeneration and Repair of Nerves. The regeneration of nerves commences at the proximal stump as an outgrowth of neurofibrils having end-buds. These outgrowths encounter scar tissue and assume a disordered whorled arrangement. By the tenth day, some neuraxes have reached the distal stump and regain a parallel arrangement. They grow down the band fibres formed by the hyperplastic Schwann cells and are thereby guided to muscles and skin areas in the periphery. If a motor nerve axone reaches a muscle, it forms a motor end-plate in that muscle but if it is guided incorrectly down a sensory nerve sheath, it fails to reestablish normal function. It must be stressed that there is no selective rearrangement of fibres from proximal or distal stump—the number of axones which find their correct sheath is governed partly by chance and partly by the exactness of funicular re-approximation. The rate of growth of regenerating neuraxes has varied in experiments from 0.05 to 2.0 mm. in 24 hours with an average figure of one mm. Clinically, the time required for regeneration from certain common sites has been noted, as follows:

(1) From the wrist—5 months.
(2) From the elbow—10 months.
(3) From the axilla—14 months.
(4) From the knee—12 months.
(5) From the thigh—15 months.

Signs of Regeneration.

(a) Where the interruption is physiological, the signs of regeneration are rapid in appearance and irregular in sequence.

(b) Where there has been anatomical discontinuity in the nerve trunks, regeneration is manifested by a definite orderly sequence of events:

(1) Vasomotor. The skin improves in colour; the temperature and texture return to normal.
(2) Muscle bellies, previously insensitive, become painful when squeezed.
(3) Muscle tone improves; the extremity feels freer.
(4) Response to galvanic stimulation changes from slow and undulating to rapid. The maximal response is again excited from the "motor" point.
(5) Voluntary movements return.
(6) Sensation returns. Affective sensation is first; discriminative sensation later.
(7) Tinel's sign. This sign is obtained by tapping the nerve trunks with the fingers. Tingling sensations in the area of distribution
of the nerve are thought to indicate the presence of young axis cylinders in those regions. The sign is said to be of questionable value.

NATURE OF NERVE LESIONS

A recent classification of nerve injuries is based upon the microscopical appearance of individual axis cylinders. This classification divides nerve injuries into three types:

(i) **Neurapraxia.** No anatomical discontinuity can be demonstrated, yet the nerve does not conduct impulses. Such an injury can be termed "physiological" block. Function returns speedily in this type of lesion.

(ii) **Axonotmesis.** The axis cylinder is divided but the neurilemma sheath of Schwann is not. This lesion gives all the signs of anatomical discontinuity but function, when regained, is almost perfect because the regenerating proximal segment of the axis cylinder grows down its correct sheath.

(iii) **Neurotmesis.** In this type of lesion all the elements of a peripheral nerve are divided, including the axis cylinder, neurilemma, medullary sheath, and connective tissue.

A more useful, if less academic, classification of nerve injuries is based upon the gross appearance:

(i) **Nerve concussion** with temporary paralysis. (Corresponds to neurapraxia in the above classification).

(ii) **Intra-neural haematoma.**

(iii) **External scar or callus** from injury to an adjacent structure.

(iv) **Intra-neural scar.** This is the commonest type of injury and is found where laceration of nerve tissue is followed by extensive fibrosis. The repair of such a lesion necessitates the removal of the fibrosed section of the nerve trunk followed by end-to-end suture of the proximal and distal stumps.

(v) **Complete division.**

TIME OF OPERATION

If a wound is known to have involved a peripheral nerve, it is permissible to explore the wound and perform primary nerve suture providing this exploration is done within five to six hours of the time of injury. During these first hours, a wound is considered surgically as contaminated—not infected. Because this procedure is not feasible under battle conditions, it is thought advisable to wait until the wound is healthy and granulating, then to employ secondary suture or repair. In this war, Russian surgeons have been advocating early operation. They
use vaccine and sulphonamide compounds locally to clean up the wound preliminary to nerve repair. Some writers maintain that sulphonamides applied locally inhibit nerve regeneration.

If the nerve lesion is left untreated, pending the healing of the soft tissue wound or fracture, the surgeon is faced with the problem of whether to operate at all for he is not sure that the nerve will not regenerate spontaneously without interference. It is generally agreed that if signs of regeneration do not appear in two to four months, surgical exploration is justified. Frazier has estimated that approximately 50 per cent of cases require operation.

TECHNIQUE OF OPERATION

Complete relaxation under general anaesthesia is imperative because the slightest movement could tear out the delicate sutures used in peripheral nerve surgery. In a recent, clean wound where complete division has occurred, primary end-to-end suture is the method employed. Stay sutures of fine chromic catgut are introduced at each end of the suture line and serve to hold the two cut surfaces in apposition while finer epineural sutures are passed. These are supposed to include a thin layer of epineurium only and are tied with forceps. The suture material may be 000 Corticelli silk, human hair, 000 chromic catgut or tantalum wire. The latter is the diameter of human hair but of greater tensile strength. It causes no tissue reaction.

Two common types of lesions are encountered in peripheral nerve exploration. The first is a bed of scar tissue or callus which has compressed the nerve; the second is intra-neural fibrosis as a result of a lacerating injury. The former is treated by neurolysis, an operation in which the nerve is freed from the compressing tissue and placed in a scar-free bed. The latter is treated by resection of the fibrosed portion followed by end-to-end suture as described above. In this latter procedure, the operator slices off sections of nerve containing scar tissue until he reaches healthy uninvaded nerve where the funiculi can be clearly defined in the cut end. It is estimated that 25 per cent of injuries require neurolysis, while 75 per cent require resection and suture.

When a segment of nerve is excised, there will be some difficulty in re-apposing the ends. The following procedures are employed:

(1) Flexion of neighbouring joints.

(2) Mobilization by careful dissection proximal and distal to the point of injury.

(3) Transplantation. This procedure is commonly employed in ulnar nerve injuries. The nerve trunk is transplanted from its position behind the medial epicondyle to the front of the elbow joint, thereby shortening its course.
(4) *Two-stage stretching operation.* This is employed where the ends of a completely divided nerve are widely separated. In the first stage the joint is flexed and the two ends roughly sewn together. When healing has occurred, the joint is gradually extended, stretching the nerve in the process. In the second stage, the nerve is re-exposed, the scar tissue about the roughly-joined ends is excised, the joint is flexed and end-to-end suture is performed. After time for healing, gradual extension of joint is again carried out.

Certain principles governing nerve suture can be cited:

1. Absence of infection.
2. Minimal damage to nerve tissue during handling.
3. Complete haemostasis.
4. Accurate approximation of nerve ends with axial re-alignment.
5. Minimal tension on suture line.
6. Scar-free bed for suture line.
7. Excision of all scar tissue from the nerve.

**OTHER METHODS OF REPAIR**

1. *Nerve Crossing.* In this procedure the proximal end of one nerve is joined to the distal end of a divided nerve. It is most commonly used for facial nerve injuries where division has occurred within the cranial course of the nerve as a result of fracture of the base of the skull. The hypoglossal nerve which is in a very available position, is sutured to the distal end of the facial nerve. It is said that after this operation the facial muscles give a mass response rather than movement of individual muscles and their movements are sometimes coincident with movements of the tongue. But the correction of the facial asymmetry more than justifies the use of this operation.

2. *Tantalum Cuffs.* A recent development in peripheral nerve surgery has been the employment of tantalum foil cuffs for the suture line. Tantalum is a metal alloy which causes no tissue reaction and possesses great elasticity and tensile strength. In the form of wire, it is used for suture material. When pressed into very thin sheets it may be used as a cuff which surrounds the suture line of a divided nerve. The proponents of tantalum foil cuffs point out three great advantages: (1) the cuff protects the nerve and suture line from envelopment and compression in an external scar; (2) the close approximation of the foil to the nerve promotes a parallel outgrowth of axis cylinders with (3) more accurate axial re-alignment.

Other writers have advocated the use of sections of an artery or vein as protective cuffs for the suture line.
Plasma Clot Suture of Nerves. In this method, the nerve ends are held together by coagulated fibrin. Originally, the plasma from certain animals was used. Recently, since the advent of blood banks with their large supplies of human plasma, fibrinogen has been separated from the other plasma proteins and has been used in neurosurgical procedures: (1) as a haemostatic agent and (2) as "nerve glue". The use of this material obviates the necessity for sutures with their destructive and irritative effects and provides a truly physiological medium for regenerating axis cylinders.

Nerve Grafting. When a large section of a nerve trunk is destroyed by injury or removed during repair, the surgeon is faced with the problem of how to bridge the gap when all the methods, outlined above, are unsatisfactory. The interposition of another piece of nerve tissue is the only solution. This procedure is known as nerve grafting.

Theoretically, nerve grafting should give good functional results. The grafted segment becomes vascularized from adjacent tissue; its neuraxes undergo normal degeneration and are replaced by Schwann cells forming band-fibres. The regenerating nerve fibres from the proximal stump then have to pass through two lines of scar tissue and it has been shown experimentally that this occasions but slight delay. Practically, the results from nerve grafting have not been encouraging. The present war has given an impetus to the investigation of the problems concerned.

Nerve grafts may be of three types:

1. Autogenous, where the graft is taken from the patient's own body.
2. Homogenous, where the graft is taken from another human being, e.g. fresh cadaver or amputation stump.
3. Heterogenous, where the graft is taken from another species.

The commonest type of autogenous graft used is the so-called "cable graft". Sections of an unimportant sensory nerve (e.g. the saphenous) corresponding in length to the defect are carefully sutured to the proximal and distal stump in such a manner that one funiculus is joined to its fellow in the other stump. Several sections of a small nerve such as the saphenous would be required to bridge a gap in the sciatic.

Homogenous grafts are now very popular. In the United States, successful grafts taken from amputation stumps have been reported. These are used fresh. Russian surgeons have advocated the use of formalinized grafts from cadavers. The purpose of the formalin is that it preserves the nerve in situ after grafting until the regenerating axis cylinders have had time to grow into it. The length of time of immersion
in formalin varies with the length of the graft. An advantage of
cadaver grafts is that a nerve of desirable size and shape can be readily
obtained. In some centres, nerves are preserved until needed by the
freezing and drying method employed for human blood plasma. These
newer methods of grafting sound attractive but until statistical results
are presented their value remains unproved.

POST-OPERATIVE MANAGEMENT OF NERVE INJURIES

This is even more important in nerve injuries than in fractures. It
should include:

(1) Prevention of the overstretching of paralyzed muscles and
contraction of antagonists by the application of suitable splints.
For example, a cock-up splint on the wrist should be worn in
cases of radial nerve injury with wrist-drop.

(2) The prevention of joint stiffness by active and passive move­
ments.

(3) The prevention of atrophy by massage and electrical stimula­
tion of denervated muscles.

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Injuries to the Principal Ligaments of the Knee

By L. N. McANINCH, '45

A REVIEW of the literature on injuries to the four principal ligaments of the knee, namely, the internal and external lateral ligaments and the anterior and posterior cruciate ligaments, reveals certain positive findings. True, there are arguments for and against some of the statements which appear below but these are the result of consultation with surgeons who have seen and treated many injured knees.

Injuries to the ligaments of the knee are relatively common, but it is still true that failure to diagnose the exact lesion and hence failure to treat the lesion properly results in many unstable knee joints. Probably the most common cause of the unstable knee following injury is a wasted quadriceps muscle. If the patient follows the instructions that he should receive, this ailment is preventable in practically 100 per cent of cases. These instructions are “active exercise of the quadriceps muscle five minutes every hour of the day during the waking hours”. This can be done without movement of the knee joint. It is explained to the patient to attempt to pull the “knee cap” or patella up his thigh and then relax. This manoeuvre is repeated for five minutes every hour. The knee joint depends on a strong quadriceps for stability and with a wasted quadriceps an unstable knee is inevitable, regardless of proper treatment of the lesion itself.

The Internal Lateral Ligament (Tibial Collateral)

Anatomy—This ligament has three attachments: Above, it is attached to the medial surface of the medial condyle of the femur; below, to the medial surface of the tibia about 1½ inches below the articular surface. The deep fibres are attached to the medial meniscus of the knee.

Function—Only in mid-flexion is the entire ligament slightly relaxed and then only sufficiently to allow about five degrees abduction. Thus it is readily seen that this ligament resists abduction of the knee at all times and hence contributes much to the stability of the joint. Its attachment to the medial meniscus contributes to maintaining this structure in its proper position.

Mechanism of Injury—The usual type of insult to the knee which results in a lesion of the Internal Lateral Ligament is a blow from the side forcing the knee into abduction. If internal rotation of the femur and weight-bearing occur at the same time, the medial meniscus will
Injuries to the Principal Ligaments of the Knee

likely be torn also, but this injury will not be discussed in this paper. Rarely is the broad tibial attachment torn; more commonly the ligament is torn from its femoral attachment or the deep fibres are separated from the medial meniscus.

**Diagnosis**—With a history as above, the knee in question is examined. If the history is short, that is, a matter of hours or days, undoubtedly the knee will be swollen. If the swelling has come up quickly (15 to 45 minutes) following injury, it is due to a hæmorrhrosis. However, if the swelling occurred gradually there is a greater chance that it is an effusion into the joint. Even in a hæmorrhosis there is still an associated traumatic synovitis with some effusion. There is pain over the medial surface of the knee and tenderness on palpation, usually at the femoral attachment. The pain is made worse when the knee is strained into Valgus (abducted). Sometimes an ecchymosis will be present on the lateral side of the knee. This is due to the contusion of the soft tissues at the time of the injury. The terminal degrees of extension are sometimes limited but this usually disappears in 10 to 14 days. These findings are enough to diagnose a torn or sprained internal lateral ligament. The next question is whether or not the ligament is completely severed. If there is a lot of pain, two per cent Novocaine can be injected in this area. The leg is next held with the knee in 15 to 20 degrees of flexion and abduction strain is placed upon it. The good knee is treated likewise and an X-ray is taken of both knees. If the internal lateral ligament is completely torn, there will be a relatively wide separation of the articular surfaces medially. This is proven by comparison with the good knee.

**Treatment**—This is divided into cases seen early and those seen late.

**Early:** (1) Traumatic Synovitis with sprain of the Internal Lateral Ligament. The knee is bandaged firmly with a crepe bandage to ½ inch above the patella. If the effusion is excessive, a posterior splint with the knee in full extension is applied. Quadriceps drill is started at once. Weight bearing is resumed in a few days and the posterior splint is discarded in 10 days. The inner border of the heel and sole are raised ¼ inch to take the strain off the ligament and usually in about four weeks the ligament is healed. The effusion is not aspirated.

(2) Hæmorrhosis—this is aspirated and a pressure bandage applied. If necessary, repeated aspirations may be performed. The existing lesion causing the hæmorrhosis will be treated as outlined.

(3) Complete tear—complete immobilization in plaster for eight to ten weeks with daily quadriceps drill is necessary. If one end of the ligament is displaced into the joint, the opening of the knee and the suturing of the torn ends will be necessary.

(4) Avulsion of bone from the tibial insertion. This is diagnosed
by X-ray whereby a flake of bone is seen to be displaced. Manual reduction and then treatment as a complete tear is usually sufficient. Failure to reduce manually will necessitate operative fixation.

_Late._ Any knee with a history of injury months or years previously, that is still causing trouble, deserves the greatest respect. The quadriceps is undoubtedly wasted and this must be corrected by daily quadriceps drill for two to three months. Often this will cure the complaint. Other lesions to look for are: (1) adhesions from an old haemarthrosis which require passive manipulation for their breakdown; (2) a complete tear in a ligament that has not been treated as such. This will require operative repair and attempts at suturing may or may not succeed. Several operations for the reconstruction of the ligament have been described by such men as Hey-Groves, Alexander Edwards and McMurray. By use of the tendons of the gracilis or semitendinosus muscles this ligament has been successfully reconstructed. The tendon is cut four to five inches from its insertion into the tibia and then fitted into a groove, made for it, on the medial surface of the condyle of the femur. The cut end of the muscle is then sutured to one of the neighbouring muscles. The leg is immobilized for four to six weeks in plaster with daily quadriceps drill.

_The External Lateral Ligament (Fibular Collateral)_

This ligament is attached above to the lateral surface of the lateral condyle of the femur and below to the lateral aspect of the upper end of the fibula. It is not attached to the lateral meniscus, being separated from this by the tendon of the popliteus.

_Function_—It causes resistance of adduction rocking movements from $150^\circ$ of extension to full extension. In all other positions it is relaxed.

_Mechanism of Injury_—A blow on the inside of the knee may result in a sprain of this ligament or avulsion of bone from its fibular attachment; rarely is the femoral attachment avulsed.

_Diagnosis_—A history of varus strain with pain and tenderness over the lateral aspect of the knee and swelling is sufficient to make one suspect an external lateral ligament lesion. In a sprain, adduction strain will increase the pain which is commonly over the fibular attachment, but rocking movements will not be possible. However, in cases where a portion of the fibula has been avulsed, rocking movements will be possible and separation of the lateral articular surfaces will be seen on X-ray.

_Treatment_—The swelling in the knee (effusion or haemorrhage) is treated as above. The remainder of the treatment for cases of sprain is adequate immobilization with faithful quadriceps exercise. Cases of
complete tear or avulsion are treated similarly to those of tibial collateral rupture or avulsion.

The lateral aspect of the knee is fairly well supported by the biceps muscle and ilio-tibial band. Rarely is operative reconstruction of the ligament necessary.

Injuries to the Cruciate Ligaments

The Anterior Cruciate ligament is attached below to the anterior intercondylar fossa of the tibia and passes backwards, upwards and laterally to attach to the medial surface of the lateral condyle of the femur. This ligament resists anterior displacement of the head of the tibia on the femur. The Posterior Cruciate is attached below to the posterior margin of the tibia and extending upward in an antero-medial direction attains its femoral attachment on the lateral aspect of the medial condyle of the femur. This ligament resists posterior displacement of the head of the tibia.

Rarely are the cruciate ligaments damaged alone; more commonly they are one of several lesions in an injured knee. They also may be sprained or torn completely across. Whether they ever cause an avulsion of bone is questionable on an anatomical basis. It is likely that any elevation of bone attached to one of the ligaments is in the form of an associated fracture.

Diagnosis—Anterior displacements of the head of the tibia while the knee is flexed to $90^\circ$ and the foot held firm is diagnostic of a ruptured anterior cruciate. However, if the ligament is only partially torn, no displacement is possible but the pain is greatly increased. Similarly, posterior displacement is indicative of a rupture of the posterior cruciate. In some cases, displacement of the head of the tibia is only possible under general anaesthesia.

Treatment—Once more immobilization and quadriceps drill are secrets to success. Treat haemarthrosis as above. If the Anterior Cruciate alone is torn, the knee is immobilized in 45 degrees of flexion with the head of the tibia pushed back. Three months will be necessary if the ligament is completely torn. In a Posterior Cruciate lesion, the knee is immobilized in nearly full extension. If both ligaments are torn immobilize the knee in about 30 degrees of flexion.

Reconstructive operations for these ligaments have been done and use made of fascia from the Ilio-tibial band or the tendons of the Gracilis and Semitendinosus; however, with no avulsion of bone early, conservative treatment will usually be sufficient and give a good result.
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The subject of Rheumatic Fever is one upon which volumes have been written in the past and upon which volumes will still be written until its aetiology has been definitely proven, its treatment more successfully carried out and the prevention of its sequelae accomplished.

In this paper I propose to review the recent literature (some still controversial) upon the subject of Rheumatic Fever.

It is loosely stated that rheumatic fever is the greatest killer of people in early adult life. It is important that we realize fully just how prevalent is this disease and to urge ourselves on to overcoming this disease that cripples life in its youth and leads to an early grave for such a large number of our fellow men. In children, as Boyd states, “rheumatism is a disease which licks the joints but bites the heart”. It is stated by Martin that one per cent of school children have rheumatic heart disease in the U.S.A.; that it causes physical incapacity in about one per cent of the wage earning population; that there are about 1,000,000 people affected in the U.S.A. and that there are 40,000 deaths a year from this disease with an average age at death of 30 years. The implications of these figures should be obvious to anyone. Ball says that there are 170,000 new cases yearly in the U.S.A. In Philadelphia in 1936, rheumatic heart disease caused more deaths under 20 years of age than did whooping cough, measles, meningococcal meningitis, diphtheria, scarlet fever and poliomyelitis combined. The mortality rate in the U.S.A. in 1936 was 17.6 per 1,000. In Great Britain, rheumatic fever causes 5.4 per cent of all deaths between 10 and 15 years of age. For many obvious reasons, then, it is vitally important that steps be taken toward lowering the incidence of this disease. The aetiology of rheumatic fever is still uncertain. Various hypotheses have been made as to the organisms having a leading rôle in its production. Ball lists these as:

1. Virus infection—this theory is losing ground.

2. Streptococcal infection—this theory is gaining favour.

Some claim that it is a generalized bacteremia while others maintain that it is a focal infection that is responsible. The mechanism whereby the streptococcal infection causes rheumatic fever is debatable and will be discussed later. It is a known fact that the general health of the patient is also vitally important. The disease shows a higher inci-
dence in the poor and in temperate zones where the incidence of acute haemolytic streptococcal sore throats are more common. The statement that rheumatic fever is unknown in the tropics must be corrected in our minds for it is now known that although the incidence of rheumatic fever is relatively uncommon in the south of the U.S.A. it is far from unknown and is much more frequent than people once realized. Numerous investigators have shown that although the incidence of recurrences was lower when the children were taken to live in the warm south, it was far from the ideal that was suggested by many people previously. Ball experimented with transporting children who had rheumatic fever to the south and investigated the incidence of recurrence while still there. He concluded that the migration of rheumatic individuals to the south is not invariably a beneficial therapeutic procedure. Ball also finds that there is no evidence that any one physical type is predisposed to rheumatic fever. It has been a common belief that the thin, underweight, freckled, red-haired child was very susceptible to this disease.

Ditkowsky and his co-workers investigated an epidemic of rheumatic fever occurring in a large hospital for sick children during the winter of 1942-43. There were 561 children in this hospital in which an epidemic of acute haemolytic streptococcal sore throat broke out and 241 of the children developed this specific type of sore throat. As an aftermath, 88 of the children with specific sore throat developed rheumatic fever, i.e., 15 per cent of the total population of the hospital. The most susceptible were ages 9-14 and it was found that sex played no part. Thus the aetiological importance of acute haemolytic streptococcal sore throat is definitely established for rheumatic fever. The mechanism whereby the lesions of rheumatic fever arise is believed to be on an allergic basis.

There are three phases in the development of rheumatic fever: 1. Acute haemolytic streptococcal sore throat or an infection which lasts only a few days and may even be so mild as to be unrecognized. 2. Incubation period of 10-14 days. 3. Rheumatic attack.

This is the course as outlined by Coburn. The similarity of the clinical manifestations of rheumatic disease and serum sickness is well recognized. Von Pirquet suggests that the characteristic incubation period is essential for the development of antibodies in serum sickness. Haig-Brown described a similar period in rheumatic fever. Coburn and Pauli state that in children and young adults rheumatic fever is more commonly polycyclic and during each febrile phase of the cycle severe inflammatory reactions take place in the vascular tissues. These reactions continue to occur so long as the respiratory pathogen (Hem. Strep.) liberates antigen and induces an abnormal antibody response in
the rheumatic individual. These inflammatory reactions in the vascular tissues of the body continue so long as the union of antigen and antibody keeps occurring and results in the usual polycyclic attack of rheumatic fever and progressive damage to the tissues of the body. The degree and duration of inflammation in the heart determines the extent of myocardial damage and chronic valvular heart disease (Coburn). Levinthal attributes rheumatic fever to the debility of the reticulo-endothelial system and its production of too few antibodies. This theory conflicts with Coburn's theory.

When we look at the incidence of permanent heart damage following rheumatic fever we find very interesting figures. Morton Brown, in a study of 214 patients with acute rheumatic fever and follow-ups for nine years, found on an average that approximately 48 per cent developed heart damage. Of these 102 cases, 11 died of the acute attack and three died of subacute bacterial endocarditis. Of those who developed permanent heart damage it was found that 68 per cent developed this damage at the time of the first attack of rheumatic fever; 16 per cent developed heart damage after the second attack and 16 per cent developed heart damage at an unknown period. Of the 214 cases 46 per cent, or 98 cases, showed no permanent heart damage and 6 per cent of the original 214 cases, or 13 cases, were discarded because of insufficient follow-up data. It should be noted that no patient without evidence of valvular damage died of acute rheumatic fever or bacterial endocarditis, while 14 of the cases with heart damage did die. They also found that the age of the patients has nothing to do with the development of heart disease, and concluded that the patient's immunity or resistance is the important factor in determining whether or not permanent heart damage will develop. When the patients developed heart disease all of the valvular damage was evident after the first attack, with the common exception that aortic stenosis commonly was late in making its appearance. If the patients show no cardiac damage after their first attack they are likely to escape permanent injury, despite further attacks. Cohn and Ling found that when the disease begins in childhood carditis is the most frequent (75 per cent of all cases) but rarely the only type of infection, and that if carditis occurred the chances were 3:1 that the infection was severe. They also found that one or more recurrences appeared in 75 per cent of the 3,129 cases studied. The recurrences were most prone to occur within five years following the first attack but were common up until the time of puberty, after which time the incidence of recurrences fell sharply.

From the prognostic standpoint it is well to look back at the incidence of deaths from rheumatic fever and its sequelae referred to earlier in the paper, and also to the figures just mentioned on the frequency of recurrences. A few more figures may be given here. Cohn and Ling found that:
in children with severe infection (carditis frequent)
less than one-half survive childhood
less than one-tenth survive adolescence
less than one-fiftieth survive the third decade.

(b) with mild infection
one-third survive to age 30
one-tenth survive to age 45.

The pathology of rheumatic fever is sufficiently well known that space will not be taken to repeat the characteristic lesions.

The symptomatology is worth repetition. The child (usually between the age of two to 15 years) may be noticed to tire easily, be losing weight, have frequent nose bleeds, increasing pallor, vague and fleeting pains in the extremities, slight fever, slight tachycardia, leucocytosis, increased sedimentation rate, or prolonged PR interval in the electrocardiogram. These signs and symptoms are all lesser signs and symptoms and only if a number are present are they significant. The usual case of rheumatic fever is not caught in this early stage when it is essential for the welfare of the child that it be treated adequately. Most of the cases are not diagnosed until the manifest signs and symptoms appear which are: polyarthritis, heart murmurs, growing pains, subcutaneous nodules, erythema nodosum, cardiac arrhythmias, abdominal pain which may simulate appendicitis, or chorea. This summary of clinical manifestations is taken from the paper by Martin. He sums up the condition stating: "The disease may be insidious in onset, protean in its manifestations, polymorphic in its behaviour patterns, unpredictable in its course and showing a tendency to be polycyclic."

The mode of treatment of the acute attack represents an old battleground. The usual method has been to put the patient at complete bed rest and to treat the symptoms as they arise. (McEwen.11) There is evidence in favour of moving the patient from his old environment where he caught the disease to a new place where conditions will not be conducive to recurrence, as for example, by bringing the patient into the hospital. There is general agreement that sulphonamides should not be given during the acute stage for there is evidence that they may even do harm. At complete bed rest, the pulse rate is reduced 10 beats per minute on an average and this, during the course of a day, will reduce the number of heart beats by approximately 10,000—a significant economy.2 The bed rest is continued along with the symptomatic therapy until all signs (clinical or laboratory) of activity have disappeared and remained so for at least two weeks after cessation of the administration of salicylates. The disease may be considered inactive, according to McEwen,11 if the temperature, pulse, white cell count, red cell count, haemoglobin, sedimentation rate and AV conduction time are all normal for at least two weeks after cessation of anti-rheumatic drugs and the
general condition of the patient is satisfactory and concurs with the laboratory findings. Then graduated exercises are begun, as for example—the patient is allowed up half an hour daily to start with and the time increased by half an hour daily as long as there is a continued absence of symptoms and signs of activity of the disease. The time required for the return to active life varies from one to nine months in the average case. On return, the majority may go back safely to full school life with unrestricted games and drill. The only exceptions mentioned are those with definite clinical evidence of cardiac enlargement or valvular damage. These can be allowed to go back to school but must not take part in games or drill.

McEwen states that if there is no heart failure the child may be allowed anything but the most strenuous competitive sports and may indeed be encouraged to be active in order to prevent the development later in life of a cardiac neurosis. He also states that if heart failure is evident the child must be treated as though in the active phase. Considerable psychotherapy and occupational therapy will be required for the rheumatic patient with valvular disease for, in most cases, they must live a sedentary life.

Once a child has had rheumatic fever there is always the fear that he may have recurrences and further damage to an already damaged heart. The prevention of the recurrences is then of no little importance. Kuttner in a paper states that the measures that may be used may be summarized as being of two main types:

General Measures—These aim at building up the general health of the patient by adequate diet, rest, vitamins, climate if feasible, tonsillectomy, etc.

Specific Measures against the streptococcal infection—Wasson believes that desensitization by the use of subcutaneous injections is valuable—generally speaking these are not feasible. Sulphonamides have been used and Thomas and Coburn and Moore in 1939 reported on their value. Kuttner used sulphonamides on 54 patients in a rheumatic fever hospital with 54 others as controls who were not so treated. It was found that 30 of the 54 not on sulphonamides developed streptococcal pharyngitis and 14 of these had rheumatic fever relapses. The dosage of sulphonamides used was 15 grains per day for a child weighing 75 lbs. and other dosages in proportion. Toxic reactions occurred in about 20 per cent of her cases. Salicylates have been recommended by several men in this field. Coombs and Schlesinger believe that salicylate therapy may reduce the incidence and severity of the cardiac lesions and recommend two grams of salicylates daily for four weeks following an upper respiratory tract infection.

Coburn and Moore give case records of the use of salicylates. This
is a report of a two-year study of the application of salicylate prophylaxis in rheumatic children in the overcrowded tenements of New York—obviously exposed to the prevalent respiratory infections. These children were in the clinic at the Presbyterian Hospital, N.Y. At the onset of pharyngitis, a physical examination was done and throat cultures taken. If streptococci, haemolytic type A, were found, the patients were placed on a daily dose of four to six grams of Salicylates for four weeks. The sedimentation rate was determined once or twice a week for two months and two throat cultures were taken at intervals of at least once a month. The results were as follows: They treated 47 quiescent rheumatic subjects with salicylates as outlined above and found that they escaped clinical manifestations of rheumatic fever. Of these, 15 showed a brief rise in the sedimentation rate within a few days after stopping the drug, and 31 maintained a normal sedimentation rate. One patient developed rheumatic fever but it was discovered that he had not taken the drug. Of the 139 controls who received no prophylaxis and developed a streptococcal pharyngitis it was found that 57 developed rheumatic fever. To eliminate the possibility that salicylate prophylaxis might mask the symptoms of rheumatic fever, the drug was discontinued four weeks after the onset of the sore throat when rheumatic activity might be expected to manifest itself, yet none was found.

The article which interested me most was one which aims at hitting the first attack with all the ammunition available and with the definite hope that the incidence of rheumatic heart disease following the acute attack may be decreased. In it, the author gave a rational explanation for the mechanism of action of the drugs used in the treatment and supported his theory by a detailed study of the cases which came before him, ran a good series of controls and ended up by giving a detailed account of his proposed method of treatment of rheumatic fever in the acute attack. The article is by Lt.-Cmdr. Alvin F. Coburn who is an old hand at rheumatic fever and is having an unusually good opportunity to study the disease since he is in the Navy where the incidence of rheumatic fever is higher than the average for the country, and where also they get the cases early, have them under close observation and with adequate laboratory facilities for the careful study of these cases. He begins: “The essential problem in the therapy of rheumatic fever is the prevention of disabling heart disease.” He refers to the theory for the causation of the typical lesions of rheumatic fever, namely: The acute attack is usually preceded by a haemolytic streptococcal pharyngitis which acts as a focus of infection and as antigen and causes the body in the next two weeks approximately to throw out antibodies to combat the infection. As long as the antigen unites with the antibody there is produced an acute inflammatory response in the vascular tissues of the body and this is the response which is responsible for the produc-
A REVIEW OF RECENT LITERATURE ON RHEUMATIC FEVER

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tion of the typical lesions of rheumatic fever. These febrile inflammatory phases must be suppressed in order to reduce cardiac damage to a minimum.

Salicylates have been used in the treatment of rheumatic fever for the last 50 years at least, and yet its use is still empirical. It is generally agreed that it has no effect on the infectious agent (haemolytic streptococci) and there is little evidence that it reduces the production of antibodies. Thus, salicylate therapy cannot be expected to alter the potential duration of the rheumatic attack. But, if the salicylate therapy modifies the sterile inflammatory reactions which occur during the activity of the rheumatic process, one may expect this effect to inhibit the development of cardiac disease. Immunological studies (Coburn and Kapp) have shown that salicylates prevent the precipitation of antigen by antibody in vitro, and that this effect is more marked as the concentration of salicylate in the system is increased. From the present paper (Coburn), it appears that salicylates act similarly in vivo.

The preliminary study was made on 64 patients ranging in age from 17 to 28 years, who were admitted to the hospital with an acute attack of rheumatic fever. They were divided up into groups and treated as follows:

Group I—43 men, all of whom were treated with salicylates only for the relief of symptoms. The daily dose ranged from intermittent doses up to six grams daily.

Results—24 had monocyclic attacks and none of them developed signs of progressive heart disease.
19 had polycyclic attacks and sedimentation rates remained elevated for an average of nine weeks and 16 of these developed cardiac valvular disease.

Group II—Seven patients with severe attacks who were treated with 10 grams of Sodium Salicylate daily until the sedimentation rate was normal for two weeks. None developed valvular heart disease and the sedimentation rate was normal in two weeks.

Group III—11 patients with severe attacks. They were treated with 10 grams of Sodium Salicylate intravenously for two weeks and then orally until the sedimentation rate was normal for two weeks. None developed valvular heart disease.

Thus 18 patients were treated with massive doses of Sodium Salicylate (10 grams daily) and none developed valvular heart disease and all showed rapid clinical recovery and a progressive fall in sedimentation rate. Fifty per cent of these patients showed electrocardiographic evidence of carditis. Thus, although doses of three to six grams
were adequate for the relief of symptoms, they failed to suppress the inflammatory reaction. Daily doses of 10 grams brought not only symptomatic relief but also modified the rheumatic reaction favourably.

Then Coburn observed another group of 58 patients with severe attacks of rheumatic fever to determine the effect of different doses and routes of administration on the plasma salicylate level and to observe the effect on the sedimentation rate when there was a high plasma salicylate level. He found that the maximal level of 400 gamma was reached in 48 hours with the oral administration of 10 grams Sod. Salicylate and in four hours on giving 10 grams Sod. Salicylate intravenously. He used a group of men who had been treated symptomatically only with daily oral doses of Sodium Salicylate varying from intermittent doses up to six grams daily. These men showed plasma salicylate levels of 0-250 gamma and had had persistently elevated sedimentation rates for an average of 49 days, and yet had been symptom-free. Thus they still manifested clinical signs of activity of the inflammatory reaction. They were then placed on massive doses (10 grams daily) of Sodium Salicylate, either orally or intravenously, and this caused the sedimentation rates to fall to normal in two weeks and the patients made rapid clinical improvements, as evidenced by gain in weight, etc. The plasma salicylate level rose to an average of 368 gamma under treatment.

He then investigated the effect of the oral versus the intravenous administration of massive doses (10 grams daily) of Sodium Salicylate:

1. Fourteen patients were placed on oral therapy.
   Results—Symptom-free in 48 hours.
   Plasma salicylate level of 368 gamma, on an average, in 48 hours.
   Afebrile in 72 hours.
   Normal sedimentation rate on an average in 17 days—these patients had an average sedimentation rate of 86 before beginning treatment. When the plasma salicylate level reached 368 gamma, all signs of carditis disappeared and all these patients escaped clinical signs of valvular heart disease. Thus it seems that the inflammatory reaction was suppressed when the plasma salicylate level reached 368 gamma.

2. Six patients, all severely ill and with sedimentation rates averaging 74 mm., were placed on 10 grams of sodium salicylate intravenously.
   Results—Symptom-free in four hours.
   Plasma salicylate level of 368 gamma in four hours.
   Afebrile in 24 hours.
   Sedimentation rate normal in 10 days on the average.
Coburn's proposed therapeutic technique for use of sodium salicylate in rheumatic fever is as follows:

First Week—10 grams of sodium salicylate in 100 cc. of normal saline given by intravenous drip in four to six hours. On the second day, if symptoms are still present, 20 grams may be used. This is rarely necessary.

Days, 7 to 30—Oral therapy replaces the intravenous method. Dose is 10 grams daily given in divided doses with NaHCO₃, eg. Sodium Salicylate 1.6 grams and Sod. Bicarbonate 0.6 grams every four hours day and night.

If by this time the sedimentation rate has remained normal for two weeks then the patient may be given a trial week of bed rest without salicylate. If he remains symptom-free and shows no signs of activity of the infection he is allowed to progressively return to the normal activities of life. But if at any time he develops frank symptoms, then another two weeks of salicylate therapy is indicated as stated for the first week's treatment and he must start over again his return to normal.

SUMMARY OF THE FOREGOING TECHNIQUE, COBURN

The factor of infection is apparently not modified by salicylate therapy. So long as this infection is active, the rheumatic patient is subject to inflammatory reactions. Observations in this article indicate that the inflammatory reaction of rheumatic fever can be suppressed to such a degree that the disease process is not detectable by clinical or laboratory examinations and suggests that the prompt suppression of the rheumatic inflammation may prevent the development of the stigmata of heart disease. The effect is parallel to and dependent upon the concentration of salicylate in the blood plasma, and relatively high plasma salicylate levels are essential (368 gamma on an average), but as high as 600 gamma may be required to break up an intense reaction at the height of the attack. None of the 38 patients treated with 10 grams of sodium salicylate daily developed valvular heart disease but 21 of 63 similar cases who received only small doses (up to six grams daily) of sodium salicylate did develop physical signs of heart disease.

In conclusion, may I state that we have not yet discovered how to control the cause of rheumatic fever but apparently we have developed means of controlling the allergic reaction induced in the host by the causative organism. Until we can control the cause we will do well if we adequately treat the reaction produced, and in a manner which will result in the lowest incidence of rheumatic sequelae. The most rational form of therapy suggested in any of the papers is that recommended by Coburn. The treatment is almost entirely without toxic reactions and the results are much ahead of the old standard method of treatment and it is thus worthy of acceptance until a better procedure is suggested.
Gas Gangrene---A Review

By J. D. Lawrence, '45

Gas gangrene is a clinical entity which develops in a certain percentage of deep puncture or lacerated wounds which extend into the muscle layers. It is especially frequent in wounds containing clothing, missiles, dirt, etc. Accordingly, it is a relatively frequent complication of war wounds (0.4 per cent of all wounds in the British Army in the Middle East Campaign). Most of the civilian cases follow compound fractures.

AETIOLOGY: Gas gangrene is caused by specific bacteria belonging to the Clostridium group. These are anaerobic, Gram-negative bacilli, which split carbohydrates and proteins with the production of a large amount of gas. These bacilli are found in excreta, both animal and human, and consequently are common in cultivated soil. In the tissues, the bacilli becomes encapsulated. A powerful exotoxin is produced, and it is the toxin which causes the damage; the bacteria themselves living only on dead and necrotic tissue. In order of frequency in causing gas gangrene, the pathogenic Clostridia are: Cl. Welchii, Cl. Oedematiens, and Cl. Septicum. It should be noted that when a wound is infected with Clostridia, the bacilli are in a resting state, and no toxin is produced unless an exciting factor is present. Bullock and Cramer have shown that the exciting agent is probably the Calcium ion.

The necessary factors in the development of gas gangrene are:
1. The presence of one of the Clostridia group of organisms.
2. A wound containing dead or necrotic carbohydrate or protein material.
3. The absence of free drainage.
4. The presence of an exciting factor, probably ionic calcium.

PATHOLOGY: The disease is confined chiefly to the muscles. A single muscle may be involved, or the toxin may spread to the adjacent muscle groups. The involved muscle becomes soft, dark red and necrotic, thus furnishing further material for the growth of the organisms. The muscle fibres are split up by the gas which is produced, and the gas spreads along the tissue planes in all directions from the original lesion. As the condition spreads, the area involved becomes oedematous and the overlying skin becomes first red, then brownish-yellow and finally black. There may be bullae on the skin surface.

Microscopically, there is disintegration of the muscle structure by gas, and necrosis of the individual muscle fibres. Large numbers of Gram-negative bacilli may be found.
SYMPTOMS: The onset of the disease is usually marked by an abrupt rise in the temperature and pulse rate. MacLennan\textsuperscript{1} claims that the earliest symptom which he noted was the sudden onset of pain in the wound. The patient is obviously seriously ill and may show mental changes ranging from acute apprehension to delirium.

SIGNS: There is progressive local swelling. The tissues become oedematous; the overlying skin becomes red and boggy, then brownish-yellow and finally frankly necrotic. Gangrene of the skin slowly advances away from the wound margins. A sanguino-purulent exudate is present in the wound. The tissues contain gas, which commonly can be felt on palpation. The gas can usually be demonstrated by X-ray before it can be palpated. It is said that there is a characteristic "mousy" odour. Jaundice may be present terminally. According to MacLennan, there is little skin discolouration unless there is an associated streptococcic infection. Acidosis always develops quickly.

THE DIFFERENTIATION OF TYPES:\textsuperscript{1} \textit{Cl. Welchii}: The incubation period averaged 23 hours in 50 cases. There is a great deal of gas present and relatively little oedema. Hyperpyrexia and a secondary anaemia are marked.

\textit{Cl. Oedematiens}: The incubation period averaged 5\frac{1}{4} days in 25 cases. There is a profuse yellow serous discharge and relatively little gas is produced. The wound is often odourless. The patient’s general condition is poor, the blood pressure is low, fever rarely over 100° F., and there is marked haemoconcentration. Mental changes, if any, are slight.

\textit{Cl. Septicum}: The incubation period averaged three days in seven cases. There is much oedema and gas production is not a marked feature.

TREATMENT: The treatment of gas gangrene is variable. There are many theories and techniques suggested. The following is the technique followed at the Medical College of Virginia Hospitals—the only one given in detail.\textsuperscript{2}

Prophylactic Measures:
1. Usual care of all traumatic cases.
2. All patients in whom the deep fascia is penetrated are considered potential cases of gas gangrene, and are treated accordingly.
3. Painstaking clean-up with sterile water, green soap and shaving of the skin, followed by ether, iodine and alcohol.
4. The field is then draped and a thorough debridement is carried out—all non-essential exposed tissue is removed. Vessels
and nerves are exposed and cleaned. The full depth of the wound is explored and opened. It is to be noted that the opposite surface of the limb should also be examined for wounds of exit. All foreign bodies and devitalized bone are removed, and then all instruments are discarded.

5. Copious irrigation with warm saline.

6. All bleeding points are ligated.

7. Ten grams of sulphanilamide are put in the wound if it is to be closed, or 20 grams if it is to be packed open.

8. If the wound cannot be closed without tension, or if a dead space will be left, the wound is packed open with vaseline gauze, taking care that the packing is not too tight.

9. If there is an associated fracture, or if the wound is near a joint, a plaster cast is applied.

10. Fifteen hundred units of anti-tetanic serum are given subcutaneously.

11. If the surgeon has any faith in gas gangrene antitoxin, it is also administered. "If there is any impairment of the circulation to the part, a sympathetic novocaine injection should be given."

12. As soon as possible, an X-ray is taken to determine the state of the fracture, if one exists, and to serve as a control to be compared later with X-ray films. At the same time, a prophylactic dose of Roentgen ray is given. Twelve and 24 hours later, a second and third X-ray are taken and Roentgen ray treatments are given. Six grams of sulphanilamide are given daily by mouth for 48 to 72 hours.

Active Treatment:

If gas gangrene develops, an immediate amputation or debridement is indicated. Roentgen ray treatments and sulphonamide therapy is continued. A red blood cell count and a haemoglobin determination should be done daily, with plasma or whole blood administered as required. Before discharge from the hospital, the patient should have an electrocardiographic examination, as experience has shown that patients with gas gangrene frequently show cardiac damage of varying degree.

Truetta calls attention to the fact that an acidosis of varying severity begins early in the disease, and should be treated at once. He recommends ten cc. of 20 per cent sodium bicarbonate solution intravenously twice a day.

RECENT WORK ON TREATMENT: There has been a great deal of experimental and clinical work done on the treatment of gas gangrene.
Penicillin is now recognized as a very valuable agent in this condition, but not much literature has been published in this connection as yet.

Much of the literature is concerned with the value, or lack of value, of gas gangrene anti-serum. There is much to be said for the use of this agent. In the following table, from an article on gas gangrene in the British Army in the present war, the value of antitoxin is shown:

<table>
<thead>
<tr>
<th></th>
<th>Established on Admission Cases</th>
<th>Developed in Hospital Cases</th>
<th>Total Cases</th>
<th>Deaths</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antitoxin</td>
<td>42</td>
<td>64</td>
<td>106</td>
<td>60</td>
<td>56.6%</td>
</tr>
<tr>
<td>No Antitoxin</td>
<td>4</td>
<td>16</td>
<td>20</td>
<td>18</td>
<td>90%</td>
</tr>
<tr>
<td>Total</td>
<td>46</td>
<td>80</td>
<td>126</td>
<td>78</td>
<td>62.2%</td>
</tr>
</tbody>
</table>

In the same article, cases are divided into three classes:

1. Those receiving treatment within six hours of diagnosis, with surgical treatment and an initial dose of at least 50,000 units of antitoxin intravenously. The mortality was 34.2 per cent of 35 cases.

2. Those receiving treatment later than six hours after diagnosis, or those receiving a smaller dose of antitoxin, or antitoxin intramuscularly. The mortality was 60.4 per cent of 58 cases.

3. Those receiving no surgical treatment and/or no antitoxin. The mortality in this class was 90.4 per cent of 33 cases.

The sulphonamide drugs have been experimented with to a great extent and certain results have been obtained. The consensus seems to be that sulphanilamide and sulphathiazole are of value in preventing the development of gas gangrene, but are of practically no use in stopping the condition once it has started. Experimental results vary greatly in different hands:

Warthin found that of 12 patients treated with sulphanilamide either locally or orally or both, there were four deaths, a mortality of 33.3 per cent. Erb and Hodes, in experimental studies on pigeons, found a mortality of 100 per cent using sulphonamides alone.

Other drugs used are very numerous. A comparison of the results obtained with sulphonamides, proflavine, zinc peroxide and penicillin in mice have been presented by McIntosh and Selbie:

<table>
<thead>
<tr>
<th></th>
<th>Number of Cases</th>
<th>Number dying on day 1.</th>
<th>Number dying on day 2.</th>
<th>Number dying on day 3.</th>
<th>Percentage of Survivals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphonamides</td>
<td>60</td>
<td>11</td>
<td>13</td>
<td>5</td>
<td>52</td>
</tr>
<tr>
<td>Zinc Peroxide</td>
<td>36</td>
<td>7</td>
<td>7</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Proflavine</td>
<td>48</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>77</td>
</tr>
<tr>
<td>Penicillin</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>
Their conclusions were as follows:

1. Zinc peroxide was an effective prophylactic, but was useless if *Cl. Welchii* got into the tissues.

2. Proflavine was not only more active than sulphanilamide in preventing infection in mice, but was at least as effective in combating a developed infection.

3. Tyrothricin (*Gramicidin and Tyrocidine*) was also tried, and while it killed *Cl. Welchii* in culture, it had practically no effect on the organism in the tissues.

4. Penicillin was the most effective of the above drugs.

Physical procedures used in treating gas gangrene include Roentgen ray therapy, simple debridement and amputation.

Roentgen ray therapy is apparently the subject of much controversy. Warthin found it to be an effective prophylactic agent in 35 cases, none of which developed gas gangrene. He also claimed it to be of value therapeutically—18 cases with a mortality of 33.3 per cent. Erb and Hodes, working with pigeons, reported a mortality of 95 per cent one week following experimental infection, using a prophylactic treatment, and of 90 per cent one week following infection using therapeutic doses. Caldwell and Cox reported a mortality of 100 per cent in guinea pigs treated with Roentgen ray alone.

With regard to surgical procedures, opinion is again divided. Amputation apparently gives the lowest mortality in developed cases—18.2 per cent of a series of 36 cases reported by Warthin. Careful debridement had a mortality of 28.5 per cent in 28 cases reported by the same observer.

It seems apparent from the above that a combination of several forms of treatment should give the best results. However, the results obtained do not entirely bear this out:

1. Erb and Hodes report a mortality of 36 per cent in pigeons using antitoxin alone, whereas a combination of antitoxin and Roentgen ray therapy yielded a mortality of 53 per cent.

2. Caldwell and Cox in their experiments on guinea pigs had 14 survivals out of 20 animals treated by debridement alone, but only five out of 20 survived by the treatment by debridement and Roentgen ray.

**SUMMARY:** The aetiology, pathology, symptoms and signs of Gas Gangrene have been reviewed. The treatment has been discussed, and may be summarized as follows:

1. All wounds involving muscles should be regarded as poten-
tial cases of gas gangrene. Such wounds should receive careful
debridement and some antiseptic such as the sulphonamides, zinc
peroxide or proflavine should be applied locally. Roentgen ray
therapy may be used locally. Free drainage of the wound should
be provided, either by drainage tubes or by packing the wound open.
Gas gangrene antiserum in the polyvalent form should be admini-
stered, preferably intravenously.

2. If gas gangrene develops, the wound should be carefully
cleansed again and further debridement carried out.

3. Probably the best drug to be used is penicillin, both locally
and systemically or both.

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3Truetta: Principles and Practice of War Surgery, pp. 117.
In this issue of the University of Western Ontario Medical Journal, it is fitting to welcome Dr. G. E. Hall, Dean of the Faculty of Medicine, and Dr. A. D. McLachlin, Professor of Surgery to the Medical School.

Dr. Hall was appointed Dean of Medicine in January, 1944, but owing to his duties in connection with the Royal Canadian Air Force, he was unable to assume the position officially until the beginning of this year. He comes to Western following a brilliant academic and postgraduate career, culminating in his appointment as Director of Medical Research for the R. C. A. F. It is understood that Dr. Hall will continue his medical research activities here. This in itself will be of immense value to the Medical School as it will foster more interest on the part of the students in research. Dr. Hall has already shown himself, to those who have met him, to be keenly interested in the students and in the development and expansion of the Faculty of Medicine.

Dr. McLachlin, who is a graduate of this school, has spent many years in post-graduate training. Prior to his appointment as Professor of Surgery he was with No. 10 Canadian General Hospital overseas. He has now taken complete control of the Department of Surgery, and already his presence has been felt, especially by the members of the senior years. He has proven himself to be an excellent teacher, who can be counted upon to raise the already high standard of the Surgery Department.

We welcome you to the University of Western Ontario, Dr. Hall, and we welcome you back, Dr. McLachlin. We should also like to officially extend a belated welcome to Dr. J. H. Fisher, who returned to his post as Professor of Pathology in June of last year. Dr. Fisher has been on leave of absence since the fall of 1942, serving with the R. C. A. M. C. and he spent most of the intervening time with No. 10 General Hospital overseas.

In the midst of our welcome, we must, perforce, think of the men who are retiring. Dr. F. J. H. Campbell was Dean of Medicine since 1934. Dr. G. A. Ramsay, Professor of Surgery since 1932, has also retired. Both of these gentlemen will long be remembered by past and present students for the work which they have done here, both for the students and for the School. During the war years, it has been increasingly difficult, owing to the shortage of Staff Doctors, to carry on medical
instruction satisfactorily, and much of the credit for successfully meet­ing the obstacles rightfully belongs to Dr. Campbell and Dr. Ramsay. Nor need we fear that these men will be lost to the School. Dr. Campbell continues as Professor of Medicine and Dr. Ramsay can be counted upon to lend his assistance and experience to the Department of Surgery.

To many of the more recent graduates and the present students, it seems that the Medical School has been marking time during the past two or three years. This may have been so, but with the anticipated return of the members of the teaching staff who are now in Military Service, and the advent of new men to the Staff, it is safe to say that the reputation of the University of Western Ontario as a medical centre will soar to greater heights than ever before.
THE UROLOGIST OF THE FUTURE
By C. E. Burford
Journal of Urology,

In this short article C. E. Burford advances his ideas of the model urologist. For 40 years he has observed the gifts and qualities which he has keenly felt to be lacking in his own experience. This permits him to make statements which smack of the depths of his convictions about the subject.

He reviews the advances of medicine and surgery and their enrichment and broadening of the field of urology. This, in his opinion, necessitates the study by future urologists of such specialties as cystoscopy, trans-urethral surgery, urology in children, etc. It is pointed out, however, that the training for concentrated efforts in a narrow field must not be correspondingly narrowed, but must be as broad as Medicine itself.

His thoughts concerning prerequisites for present-day study of medicine, are interestingly combined with prophesies regarding the demands which will be placed upon future urologists, educationally, socially and politically.

This paper is especially suitable for medical students and young doctors. Make use of his 40 years' experience. His views are sound.


POST-TRAUMATIC EPILEPSY
By Wilder Penfield, M.D., Montreal
American Journal of Psychiatry,
100:750-751, May, 1944.

Epileptic seizures may be produced by many types of brain injury involving the cerebral gray matter. In general, those local injuries which result in meningo-cerebral scarring are most apt to cause epilepsy. The usual cause is brain laceration by depressed fractures or mis-
siles, or scars from healed abscesses; less frequently, scars are due to infarction, e.g. by cerebral embolism, by phlebothrombosis occurring in meningitis or by dehydration. Closed injury to the skull, subdural haematoma, or a gradual diffuse increase in intracranial pressure rarely result in seizures.

In order to cause epilepsy, the trauma must leave a focus of abnormal neuronal activity, which periodically results in an explosive, spreading ganglionic discharge. This focus does not lie in the scar; nerve cells are absent there. It is usually to be found on the border of normal cortex, in a small marginal gyrus, partly atrophic, but containing functioning nerve cells. The blood supply here is apparently adequate to prevent immediate destruction but inadequate for constant satisfactory oxygenation; thus anoxaemia may stimulate the ganglion cells and cause a seizure.

Following depressed fractures, drained abscesses, etc., extradural vessels are prone to anastomose with cerebral vessels during healing. These extradural vessels are subject to frequent active variations in calibre due to more effective sympathetic innervation, in contrast to cerebral vessels controlled passively by functional demands of the cortex, through the CO₂ content in the tissues.

The neurosurgeon should, therefore, in treating post-traumatic epilepsy, remove by suction any gyrus that is partly destroyed. The focus is usually a narrow, tough gyrus, which may be covered by dense white arachnoid. The pia along the summit of the gyrus is incised, the gyrus is aspirated with a sucker, without injuring the pial covering of adjacent gyri. With this method, extracerebral vessels will not grow in, and the blood supply will be regulated as before solely by tissue requirements. If the focus is removed carefully, complete cure will result.

Geo. D. Wilkins, '45.
1. RUPTURE OF THE HEART IN MYOCARDIAL INFARCTION.
EXPERIENCE IN A LARGE GENERAL HOSPITAL

By SIDNEY FRIEDMAN, M.D., AND
PAUL D. WHITE, M.D., F.A.C.P.

Annals of Internal Medicine,
1, No. 5, Nov., 1944.

The material for this article was collected by the Cardiac Laboratory of the Massachusetts General Hospital. The records covered 270 cases of myocardial infarction which were included in a series of 2,967 autopsies over a seven-year period. Of the 270 cases, 165 showed old coronary occlusion with healed infarction. While in these cases many showed ventricular aneurism, not one showed cardiac rupture. Of the 105 cases of acute infarction, all within 2 weeks of death, 10 had as the immediate cause of death, rupture of the ventricle. Of these cases, eight were due to occlusion of the anterior descending branch of the left coronary artery. It is interesting to note that of the 10 cases, four received inadequate sedation. Thus, in a series of 270 cases in a large general hospital 10 or 3.7 per cent showed rupture of the ventricle.

This may be compared with a similar series observed in a large mental institution:

WORKING CAPACITY AFTER THYROIDECTOMY

By W. L. SCOTT AND J. W. PARKS
British Journal of Industrial Medicine,
1: 176-178, July, 1944.

The authors present 105 cases (67 females and 38 males) between the ages of 16 and 60 years for a comparison of the post-operative working capacity of patients who had undergone thyroidectomy between the years 1929-1943. Although all the cases were proved to have had thyrotoxicosis it was impossible to separate those with primary exophthalmic goitre from those with secondary adenomatous goitre; nor was it possible to obtain information regarding the pre-operative condition, presence or absence of complications, or the technique employed—all of which would have an important bearing upon the end-result. Furthermore, this group of patients were all employed in one particular field, namely, the Postal Service in London, England.

The largest number of cases occurred in the 35 to 40 age group. The cases were divided into two groups, those who died or retired on medical grounds and those who are still in the service or have retired. Of the 105 cases, four died, seven did not return to work, 13 resigned and 13 were invalided on medical grounds. Medical causes accounted for the death or retirement of 20.9 per cent of the cases during the five years following operation. The average annual sick rate of those remaining in the service was 9.3 days per year—a considerable drop from the pre-operative sick days. Post-operative recovery took from five to six months. Of the women retired, 47 per cent were over 40 years of age at the time of operation—the post-operative prognosis in women over 40 must therefore be considered poor. In 79 per cent of cases in which death or retirement did not take place the level of subsequent health and working capacity was not inferior to that of normal individuals.

2. RUPTURE OF THE HEART IN PATIENTS IN MENTAL INSTITUTIONS

WALTER M. JETTER, M.D., AND
PAUL D. WHITE, M.D., F.A.C.P.
Annals of Internal Medicine
1, No. 5, Nov., 1944.

This series was conducted under the supervision of a committee of the Massachusetts Public Health Service. In a series of 22 cases of recent myocardial infarction, 16 or 73 per cent died of rupture of the heart. Of these, 10 were ambulatory until the time of death. Two were ordered to bed 72 hours before death and collapsed when they went to the bathroom. Most of the cases showed left ventricular involvement. It is interesting to note such a wide variation in a mental hospital group and a general hospital group. Those in a general hospital usually arrive with the diagnosis already made and are co-operative in
following orders. They had subjective symptoms and were able to describe them. Of the mental patients, many may be desperately ill and due to perversion of symptoms may not complain until the end.

In comparing these two series the value of bed rest and adequate sedation is well illustrated. Bed rest is the best physiological method of resting a damaged heart and relieving the strain thrown on it by the pathological condition present.

Maurice Zaltz, '47.

THE USE OF PENICILLIN IN SURGICAL INFECTIONS
By John S. Lockwood, M.D., William L. White, M.D., and Franklin D. Murphy, M.D.

Annals of Surgery,
120:311-349, Sept., 1944.

The object of this paper is to present the scope and limitations of penicillin and to indicate how it may meet the shortcomings of the sulfonamides in surgical infections.

Penicillin is active against most gram-positive species of bacteria, also meningocci and gonococci, but is inactive toward most gram-negative bacilli. It is a bacteriostatic agent administered parenterally either by the intravenous drip method or by intramuscular injection. Penicillin circulates through the body fluids in an active state without any apparent toxic effects, and is rapidly excreted in the bile and urine. The results of its use in surgical infections are treated under four headings, and include case summaries to illustrate the basis for the conclusions:

(1) Cases of staphylococcic bacteremia respond readily to penicillin treatment but many cases of pneumococcic meningitis fail to recover due to secondary changes. In stubborn cases, the possibility of an infected mastoid should be investigated and drainage instituted if necessary. Lumbar cisternal injection seems to show superiority in cases of meningitis.

(2) Empyema and suppurative arthritis cured by penicillin — sensitive bacteria are often effectively destroyed by single daily injections without resorting to surgical drainage. In peritonitis, penicillin is effective in combating haemolytic streptococci but the possibilities of other cases have not been adequately studied.

(3) In localized infections of the soft tissues such as cellulitis of the face, boils, and carbuncles, penicillin is quite effective in preventing suppuration and in shortening the time of healing. In cases of pulmonary suppuration, the drug merely places the patient in a better condition for operation and reduces post-operative infection.

(4) Penicillin brings about the disappearance of evidence of disseminated sepsis in acute haematogenous osteomyelitis, but complete recovery is rarely observed without surgical treatment. In cases of chronic osteomyelitis, a high frequency of recurrence occurs unless surgery is combined with penicillin therapy.

These investigations indicate that penicillin does give promise of meeting the limitations of sulfonamide treatment in surgical infections.

EARL QUERENGESSE, '48.

ESSENTIAL HYPERTENSION—ITS SURGICAL TREATMENT
By W. P. E. Berwald and K. D. Devine
American Journal of Surgery
64:382, 1944.

An optimistic viewpoint is held by the authors in the treatment of essential hypertension by surgery, two operative techniques having been used; one a subdiaphragmatic approach, the other a thoracolumbar approach.

The cases, it is stressed, must be properly selected for the operation, the following being the foremost criteria:

(1) Age — under 50 — the physiological age being stressed rather than the chronological age.

(2) A favourable response to sodium nitrite and sodium amytal tests.

(3) Patients with advanced cardiac and renal disease are refused operation.

(4) An obese patient must lose considerable weight before being operated upon.

(5) Retinal vessel sclerosis appears to
bear no relationship to the response to sympathectomy.

(6) There is no evidence of relationship between the length of known duration of the hypertension and the operative results. The operation involves the removal of certain dorsal and/or lumbar ganglia in addition to removal of the greater splanchnic nerve with or without a segment of the coeliac ganglion. Over a period of 3 years, 29 patients have been operated upon. All but two of the patients are living at the present time, one death having occurred at operation. The other from coronary thrombosis one and one-half years after operation.

Objectively, improvement was discernable in 64 per cent of patients — as evidenced by an average drop in the systolic blood pressure of 75 mm. Hg. and in the diastolic pressure of 15 mm. (the latter being the more important). Subjectively, improvement occurred in 92 per cent of patients.

The operative risk is allegedly minimal.

W. BRUCE BARTON.

THIOURACIL IN THE TREATMENT OF THYROTOXICOSIS
By E. M. WATSON, M.D., AND
L. D. WILCOX, M.D.,

This article consists of a report on the use of thiouracil in the treatment of a group of thyrotoxic patients. Much experimental background and the findings of other workers are discussed at length. This is followed by a short note on the therapeutic application of thiouracil given orally, to treat hyperthyroidism.

The main body of the article consists of the reported records of 11 thyrotoxic patients who were treated with thiouracil. Although these case reports do not add any new information to that which already has been published by others, they do provide confirmatory evidence relative to the efficacy of the medication. With one exception all the patients carried on their treatment at home. In most instances the initial dosage was 0.6 gm., in divided doses, daily. The dosage was reduced as clinical and metabolic improvement occurred.

Judging by the results obtained, it is evident that the oral administration of thiouracil does produce a salutary effect in at least a majority of the patients with thyrotoxicosis and hypermetabolism. The notable features include relief of the symptoms commonly associated with a state of thyrotoxicosis, a reduction of the B. M. R. to normal or near-normal levels, a slowing of the pulse rate, a gain in body weight, an increase of the blood cholesterol and a reduction in the amount of circulating thyroid hormone as indicated by a return to the normal concentration of the organic iodine of the blood. No toxic effects were noted as a result of the drug in this group of cases.

The mode of action of thiouracil in depressing the functional activity of the thyroid gland is, at the present time, largely conjectural although there is evidence that it influences, indirectly, the production of thyrotropic hormone by the anterior pituitary gland.

Jos. E. Wittig, '47.

CIRCULATORY CHANGES FOLLOWING THE SUBCUTANEOUS INJECTION OF HISTAMINE IN DOGS
By I. J. DEYRUP

Following the observations by Dale and Laidlaw in 1910 of the extreme hypotensive effects of histamine, the suggested role of "H" substance in traumatic shock has been the subject of much discussion and debate. Deyrup studied some of the changes caused by histamine in unanaesthetized dogs and compared these results with those obtained by Gregerson and Root on traumatic shock in dogs.

Dosages of histamine diphosphate, about 10 mgm. per kg., were injected subcutaneously in 18 normal unanaesthetized dogs, nine splenectomized dogs and six dogs anaesthetized with ether. The following were examined: mean arterial pressure, heart rate, rectal temperature, venous pressure, plasma volume, serum, protein, hematocrit value, hemoglobin concentration, blood volume
and red cell volume. Finally, necropsies were performed.

Shortly after the injection of histamine, a characteristic circulatory disturbance resulted. However, it differed markedly from the picture seen in the traumatic shock experiments. The absence of blood volume reduction in histamine shock was in marked contrast with the blood volume reduction in traumatic shock. The change in blood volume in histamine shock was due to a change in the plasma volume (either an increase or a decrease) and to an increase in the volume of red cells mobilized from the blood depots, particularly the spleen. In the histamine shock, the hypotension was much more marked than in traumatic shock. The clinical symptoms produced were significantly different in the two sets of experiments.

Albert Schilling, '48.
To Study the Phenomena of Disease without Books Is to Sail an Uncharted Sea. —Osler.

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January 11, 1945.
TECHNIQUE IN TRAUMA—PLANNED TIMING IN THE TREATMENT OF WOUNDS INCLUDING BURNS

By F. B. GURD, M.D., C.M. and F. D. ACKMAN, M.D., C.M., MONTREAL,
J. LIPPINCOTT COMPANY, 1944

This is a remarkably complete, concise monograph based mainly on the work of F. Douglas Ackman, Fraser B. Gurd and co-workers at the Montreal General Hospital and McGill University.

The monograph is composed of three papers, an extensive bibliography and a commentary by Ralph R. Fitzgerald, M.D. One cannot help admiring the candor of the authors in their comments and criticism of cases in their own series.

The workers give a complete account of the step by step treatment of traumatic injuries, stressing particularly the factor of timing, early control of shock, occlusive bandages infrequently changed, local sulphathiazole therapy and skin grafting as early as possible. The authors support their methods by adequate data and a great many case histories. In addition there are several colour photographs, charts and diagrams to enhance the value of the book.

This work is now available in the U. W. O. Medical Library and should be regarded as a "must" by all interested in the subject.

EARL PLUNKETT.

A DICTIONARY OF ABBREVIATIONS

Compiled by HERBERT J. STEPHENSON, NEW YORK,

This book is valuable in the absolute sense and amazingly so in the relative sense. It is only slightly over 100 pages, yet the scope of its contents is such that it would more than fulfil average needs.

The greater portion is devoted to a comprehensive list, alphabetically arranged, of abbreviations in general use. This book is, however, sufficiently extensive to include, as well, less common terms. Of particular interest to professional people are the "call-letters" of the world's honour societies. The remaining pages contain material supplementary to this main list. In these more specialized terms, conveniently categorized, are made readily available as the following headings indi-
cate: Legal Literature, Geographical abbreviations, Months and Days (given in the principal languages of the world), Foreign Monetary Units.

This unique volume has the additional advantage of possessing a rather friendly appearance in contrast to the usually more forbidding aspect of dictionaries. Its compactness, accuracy and diversity of subject matter recommend it for your private library.

—MARY E. PURDY.

SURGERY OF THE AMBULATORY PATIENT

By L. Kraeer Ferguson, A.B., M.D., F.A.C.S., Montreal, J. Lippincott Company, 1942, 923 pp., $11.50.

This book was written "as an aid to the younger men and general practitioners in their everyday practice". Only subjects applicable to ambulatory patients are discussed, except for a few topics mentioned for purposes of differentiating them from the lesions which fall into the sphere of ambulatory surgery.

The volume is divided into three sections. The first is on surgical principles and lesions. It includes an excellent discussion of the methods of employing local anaesthetics; chapters on pre- and post-operative care, dressings, etc. In this section non-regional lesions are dealt with in a popular and practical manner. Operative and medical treatment is emphasized with mention of both successful therapy and the common surgical mistakes.

The second part of the book deals with regional surgery. Here are detailed the common surgical lesions of the specific parts and the method of treatment. Again, treatment is dealt with in length and with remarkable detail. Alternative techniques with their advantages and disadvantages are also given.

The third section is entitled the musculoskeletal system. It includes certain fractures and dislocations and their treatment in ambulatory patients. Naturally, fractures, e.g., of the hip requiring hospitalization are not included. The methods of splinting are discussed in detail as are the methods of strapping in sprains, strains, etc. A full description of the gelatin boot and its surgical applications is included.

For the undergraduate or graduate interne or for the general practitioner this is an excellent, practical and very useful volume. The detailed techniques with numerous diagrams (645 illustrations) and lengthy references are the outstanding features. The treatment in places, however, may appear to be somewhat at variance with accepted methods, but this would invite employment with caution and not rejection.

WILLIAM D. WILKEY.
BURMA SURGEON

By GORDON S. SEAGRAVE, LIEUT.-COL., M.C., NEW YORK,
W. W. NORTON & CO. INC., 1943, 295 pp., 23 illustrations, map, $3.00.

THE BURMA SURGEON is the story of a Doctor Missionary who
cares little for conventionality, a man who throws himself whole­
heartedly into his work and has fun doing it.

Dr. Seagrave, beginning in the jungles of Burma with a waste­
basketful of broken-down surgical instruments, in a few years was able
to show for his work a hospital, a nurses’ home and a well-trained, efficient staff. He speaks very highly of the native nurses whom he trained in the hospital and the nurses in turn adore their “daddy’ as they fondly call him. After his twenty years of splendid work, the Japanese invaded the country. Dr. Seagrave volunteered the services of himself and his staff to General Stilwell. Then for several months the party worked to the point of utter exhaustion caring for the Chinese Sixth Army. Forced to retreat from Burma to India, they trekked through the jungles with the Japanese only a few miles behind.

Written in a free and easy style this is a book that anyone will enjoy.

—NORMA COOK.

MANUAL OF MILITARY NEUROPSYCHIATRY

Edited by HARRY C. SOLOMAS, M.D.,
Professor of Psychiatry, Harvard Medical School, and
PAUL I. YAKALEV, M.D.,
Instructor in Neurology, Harvard Medical School.
Contributions by forty-five authorities.
Pp. 764.

The manual has been designed as a reference text in Topics of Clinical Neurology and Psychiatry and is based largely on experience gained in the present war.

The book is divided into six sections, each containing a number of Topics.

1. Introductory: 2 Topics. Neuropsychiatric experiences of the first World War are briefly stated and neuropsychiatric organization in the present war is discussed.

2. Induction: 5 Topics. Modern selective and screening methods are considered.

3. Administration and Dispensation: 7 Topics. Problems of handling neuropsychiatric cases are discussed, e.g. recreation, discipline, discharge, etc.

4. Clinical Entities: 17 Topics. The clinical features of the psychoneuroses, psychopathic personalities, etc., are described as well as those of numerous neurological lesions.


Electroencephalographic diagnosis of epilepsy is considered.

The manual is recommended especially for Medical Officers for use in the field and also for general practitioners as an aid in understanding of civilians and returned soldiers suffering from neuropsychiatric conditions.

KEN WARD.