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Vol. V.

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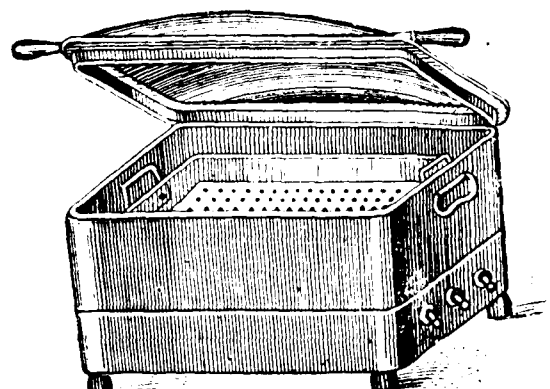
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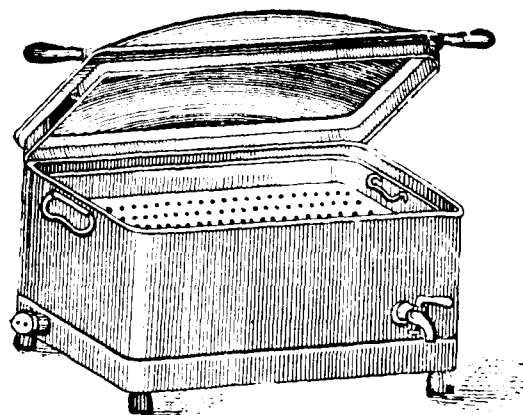
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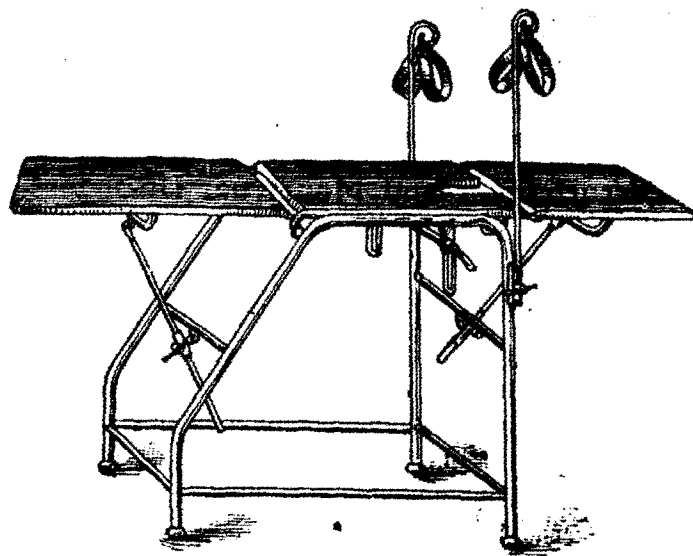
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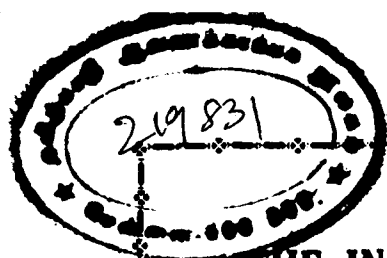
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MEDICO-SURGICAL SUGGESTIONS

A MONTHLY JOURNAL OF PRACTICAL MEDICINE & SURGERY

VOL. V

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MEDICO-SURGICAL SUGGESTIONS

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Leading Articles

TUBERCULOUS DISEASE OF BONE

BY LIEUT. COL. M. M. CRUICKSHANK, I. M. S.,
*Professor of Operative Surgery, Medical College and Surgeon,
General Hospital, Madras.*

Bone tuberculosis is a chronic inflammatory condition and an osteomyelitis with secondary periosteal involvement occurring in early life, choosing particularly the ends of long bones, the short long bones and the short bones; displaying as a rule an excess of bone destruction over bone formation and yet with a decided tendency towards limitation of spread and spontaneous healing owing to the same fibrous tissue formation, so characteristic of tuberculous disease in the lungs and other organs.

Though essentially a disease of childhood and of adolescence, it may become manifest at any age, even in senility, but in these older patients it can be proved to be a recrudescence of any affection, latent perhaps for many years.

Route of Infection.—The infection is carried to bone by the blood stream probably in the form of a tuberculous embolus from some other part of the body.

Site of infection.—Tuberculosis commences in cancellous bone, seldom in compact bone.

The most frequent sites are, ends of long bones especially the tibia and femur, the short long bones, the short bones of the tarsus and vertebrae; as all of these are in close proximity to joints, bone and joint tuberculosis are very frequently associated.

The common situation to be affected is usually the metaphysis, occasionally the epiphysis of a long bone.

Both are richly supplied with capillary loops in which an infected embolus may lodge.

The circulus vasculosus (of Hunter) containing the vessels responsible for carrying the infection lies in the reflection of the synovial membrane on to the bone. When the reflection includes the metaphysis as at the upper end of the femur, the disease will

originate in that part; when on the other hand the reflection lies entirely in relation to the epiphysis as at the upper end of the humerus and to a lesser extent at the lower end of the femur the primary focus will be in the epiphysis, but another factor is present which may well determine which bone is to be attacked. The blood vessels which supply the bone become affected with tuberculous endarteritis, due to the circulating tuberculous toxin.

The nutrient arteries of the short bones are especially so affected, and as a result the bone marrow undergoes a gelatinous or fibromyxomatous degeneration, and in such a bone tuberculous osteomyelitis readily develops.

Occasionally the diaphysis is affected.—Why? Typical tuberculous diaphysitis is seen in the affection of the phalanges, metacarpals and metatarsals and in the bodies of the vertebrae.

There are two considerations which explain the development of this form of the disease.

1. The vascular distribution in the smaller bones is peculiar; the nutrient vessel as soon as it enters the interior of the bone, breaks up into a complete network of vessels and there is therefore a tendency for any circulating tuberculous material to be held up before it reaches the metaphysis.

2. The nutrient vessel is sometimes the site of a tuberculous endarteritis and the peculiar marrow changes which appear secondary to this condition are responsible for the lodgment and development of the tuberculous lesion.

This is the influence which explains the infection of the larger long bones.

The common forms of tuberculous diaphysitis such as "spina ventosa" are practically confined to the earlier years of life, because it is during this period that the nutrient vessel is the main source of blood supply. After growth has ceased the periosteal vascular supply becomes of greater importance than the nutrient and there is a corresponding fall in the incidence of tuberculous diaphysitis.

Occasionally the Epiphysis is affected.—Why? The impression abroad is that the epiphysis like the metaphysis is frequently the site of tuberculous disease, but this is not so. The error has arisen through erroneous nomenclature and confusion of the terms metaphysis and epiphysis.

There are however conditions in which a tuberculous epiphysitis does occur.

The most common is the secondary infection which extends from a related infected joint.

The second condition is an anatomical one. If the reflection of the synovial membrane is related only to the epiphysis and does not extend on to the diaphysis, then that epiphysis may become the site of a primary tuberculous epiphysitis, because into such an epiphysis a

large number of vessels enter from the neighbouring joint and it is therefore liable to be infected from the joint itself or by a blood borne infection from other parts (Fraser). Such a statement might give one the impression that tuberculous epiphysitis is rare but this is not so in long bones.

As Greig puts it, "in long bones the initial tuberculous infection does not occur so invariably at the metaphysis as does the acute septic process; commonly it is epiphyseal and as such, may by early perforation, cause a tuberculous arthritis or more rarely spread towards the marrow cavity. When the tuberculous affection begins in the diaphysis it may be periosteal but more frequently is endosteal or medullary. Of the long bones the tibia is probably the most commonly affected."

Changes which result from the lodgment of the Bacilli. The Primary Bone lesion.

Probably the bacilli become deposited by an extravasation of blood, from a vessel, the wall of which has been previously weakened by Tuberculous endarteritis.

In the effused blood the Bacilli form, just as they do in tuberculosis elsewhere and as a result of their growth a typical tuberculous follicle is found with giant cells, epithelioid cells and lymphocytes, a central mass of endothelial cells with a surrounding zone of lymphocytes—a miliary tubercle. A mass large enough to be apparent to the naked eye, a small translucent greyish white area in the centre of the marrow. Changes appear at the centre, caseation occurs and at the periphery there is an attempt at fibrosis and localisation of the follicle the grey tubercle has now become a yellow tubercle. Independent of the growth of the miliary tubercle changes appear in the various constituents which comprise the bone.

The marrow around the focus shows tissue changes similar to those associated with tuberculosis elsewhere.

An acute irritant is at work and a leucoblastic reaction on the part of the marrow gives rise to great numbers of polymorphonuclears. The tuberculous toxins then begin to make their presence felt and lymphocytes replace polymorphs.

Later still the fibrosis which plays so great a part in the healing of a tuberculous lesion begins to develop and the fat is replaced by connective tissue which in time will become fibrous tissue and wall off the original focus.

In acute spreading forms this does not happen.

The arteries of the marrow and the bone itself show a marked endarteritis obliterans similar to that so characteristic of syphilis, save that it is a late feature of the disease and is unaccompanied by any perivascular infiltration of lymphocytes.

"The presence of tubercles, their bacilli and their toxins, within the marrow cavity or within the haversian canals, or even on the

surface acts as a foreign body and as such provokes a histamin-induced hyperaemia" (Greig.)

When this hyperaemia reaches a certain degree there is proliferation of fibroblasts, plump fusiform cells with vesicular nuclei and branching processes—*granulation tissue*; there must therefore follow a decalcification of bone.

The bone lamellae show two characteristic changes, osteoporosis or rarefaction, osteosclerosis or condensation.

Osteoporosis or caries is brought about by

- (1) action of osteoclasts.
- (2) erosive action of cellular granulation tissue, which is formed in relation to blood vessels and which enlarges haversian canals and erodes bones under the periosteum and in contact with the medulla.

Branching trabeculae of bone are left, the interspaces being filled with granulation tissue.

Osteosclerosis.—Seldom well marked in tuberculosis, is the analogous process in bone to fibrosis in marrow. By osteoblastic action fresh layers of bone are laid down on the laminae and so increase their thickness and density. When osteosclerosis occurs it is always the result of superadded sepsis plus exposure to toxins of the septic process, together with the presence of endarteritis.

"The process by which the portion of a diaphysis affected with tuberculous osteomyelitis changes from its normal shape to a thin walled fusiform expansion is not generally appreciated. There seems to be prevalent the idea that the bone is distended by pressure of tubercles from within. As bone is a rigid structure such a supposition is manifestly absurd. Bone can no more expand or dilate than it can bend without initial decalcification. Such a change in shape however presents no mystery." (Greig).

When the bony tube becomes soft as a result of the histamin-induced hyperaemia, then it can accommodate itself to its contents, with this relief the hyperaemia diminishes and at once the local excess of calcium is utilised to form new bone.

The factors in this enlargement or false expansion typified in the spina ventosa of tuberculous dactylitis, may be stated shortly so.

- (1) the presence of tubercles acting as foreign bodies.
- (2) provides a histamine-induced hyperaemia which
- (3) is followed by decalcification which
- (4) renders the affected area simply a soft pliable tube in part or in whole, allowing it to alter in shape, and
- (5) with the cessation of the hyperaemia, the local excess of calcium is utilized, new bone is laid down, recalcifying the area of bone in its altered shape, thus rendering this shape permanent.

As the periosteum reaches its limit of expansion, the stretching diminishes its vascularity and the local excess of calcium is again available for the formation of bone.

This sheath or case of new bone is formed at the periosteum because there and there only in the diseased bone can there be the necessary combination of adequate blood supply and excess of calcium salts, but it is not formed by the periosteum. Though subperiosteal it is by no means of periosteal origin.

THE QUESTION OF THE SEQUESTRUM IN TUBERCLE.

A sequestrum is not common in tuberculous disease because of the increase of peripheral hyperaemia which the tubercle provides and of the degree of periosteal hyperaemia which it excites. Should minute spicules or fragments of the trabeculae escape decalcification, they may find their way into an avascular, purulent or caseous area of fused tubercles, where they may remain as fine particles.

In severe forms of infiltrating tuberculous osteomyelitis, however, larger areas of bone may be more or less isolated and in the short long bones the whole of the body of the bone may be affected. Even then the so called necrotic bone lies in a mass of tuberculous granulation tissue and in virtue of the granulation tissue is undergoing rapid decalcification. It never forms a true sequestrum, a dead homogeneous entity, unless sepsis be superadded.

In the absence of sepsis any dead osseous tissue will be dealt with by the inflammatory process and will ultimately disappear, its decalcification providing an immense amount of excess calcium which goes to form the light porous fusiform involucrum, standing well away from the necrotic shaft, so characteristic of tuberculous dactylitis. It is misleading to speak of this granulation—impregnated necrotic osseous tissue as a "sequestrum," it is not isolated, but retains its attachment to the extremities of the metatarsal and is connected freely with the new porous bone in which it is encased. If closely examined the "sequestrum" if large will be seen to be porous.

TUBERCULOUS PERIOSTITIS.

While usually associated with tuberculous osteomyelitis, it may occur as a separate entity. It is usually found in superficially placed bones but may occur in the long bones. The pathological process is due to the presence of tubercles and their toxins, producing a hyperaemia in the periosteum with slight serous exudation with resulting rarefaction of cortical bone: this is later replaced by light porous subperiosteal bone. Excess of this porous spongy bone on one aspect of the bone as compared to another is due to posture, the exudate raising the periosteum further from the bone over the dependent parts.

Superficial bones such as clavicle, sternum, ribs, vertebrae, skull, occasionally show this new bone formation. It is much less frequently seen in the long bones of the limbs.

In *superficial bones* such as the sternum the formation of tuberculous granulation tissue in the deeper layers of the periosteum and its subsequent caseation and liquefaction is attended by the insidious development of a doughy swelling which is not as a rule painful, although tender on pressure. While the swelling often remains quiescent for a time, it tends to increase in size, to become boggy or fluctuating and to assume the characters of a cold abscess.

In *deeply seated bones* such as the upper end of the femur the formation of a cold abscess in the soft parts is often the first evidence of the disease.

THE PROGRESS OF THE DISEASE—PATHOLOGICAL VARIETIES.

Tuberculosis may behave in various ways in bones as in other organs. It is unnecessary to introduce a special series of names to denote different varieties. It is sufficient to recognise

BOYD	FRASER
1. a localised form.	1. encysted.
2. a spreading form,	2. infiltrating.
3. an atrophic form or caries sicca	3. atrophic.
	4. hypertrophic ? mixed with syphilis.

The *localised form* is much the commonest and most chronic. The defence is sufficiently strong to hold the disease in check and this form corresponds to a localised nodule in the lung. The lesion, which is about the size of a marble presents a centre which is either caseous or converted into a semi-fluid material.

Around this is a pale pink zone blending with the surrounding marrow. This is the limiting band of new fibrous tissue. The condition is essentially chronic so that the slow process of rarefaction results in complete absorption of the lamellæ. If the lamellæ are not absorbed completely, then they separate as and form small sequestra.

The *spreading form*.—Where the fibrosis is insufficient to limit the spread of the disease, it corresponds to a diffuse tuberculosis of lung. The central caseous mass is surrounded by a zone of gray infiltration representing tuberculous granulation tissue which has not yet become caseous. Around this there is a red zone of reaction.

Thrombosis of vessels which occur early greatly weakens resistance. Rarefaction is not so marked, because active processes are arrested and necrosis supervenes, with the formation of comparatively large sequestra. The disease may spread down the medullary cavity until the greater portion or the whole of the shaft is involved. It may spread to the cartilage of conjugation and involve the epiphysis and neighbouring joints. It may spread to the periosteum and form a sub-periosteal abscess. It may ultimately discharge on the skin. Where the medullary spread is marked,

expansion of bone with deposition of new bone is seen as in tuberculous dactylitis.

The *atrophic form*.—Caries sicca is a rare condition chiefly affecting the upper end of the humerus. The deeper portion of the bone becomes rarefied owing to the formation of granulation tissue and new bone is deposited on the surface which itself in turn becomes eroded. Finally the granulation tissue becomes dried and fibrosed.

With the replacement of marrow by granulation tissue, the bone lamellæ undergo a rapid absorption. The process begins in the centre of the medulla and gradually extends to compact bone at the periphery: the lacunae are increased in size and the whole appearance is one of extreme porosity.

True suppuration never occurs.

The changes may extend to articular cartilages but never involve it.

The absorption is due to the activity:

(1) of osteoclasts; (2) of granulation tissue.

A periosteal hyperaemia brings about a deposition of subperiosteal new bone which tends to become absorbed near the original shaft, so that a ring of granulation tissue lies between the new bone and the original shaft. This goes on and on giving an increased but unstable circumference.

"Tuberculous osteomyelitis as seen in the long bones and short long bones has no counter part in the bones of the cranium and face. In the membrane, bones of the skull tuberculosis is of the greatest rarity, yet tuberculoma of the brain and tuberculous meningitis are common occurrences.

There is a vast difference between the anatomical and functional relations of the pericranium on the outer table and the dura mater on the inner table, to the cranial bones, and the periosteum and endosteum in the long bones, and there is no parallelism between the diploe and the marrow cavity.

In the long bones the central canal is naturally evolved as being an essential part of the mechanism of stability under the stress and strain to which such bones are subjected by function and by muscular strain; but the diploe is a covered way whereby the venous intracranial circulation finds ready transference by an alternative route in the event of unilateral temporary obstruction, be it postural or from extraneous pressure.

In the limbs temporary obstruction to the venous return is met by the system of superficial subcutaneous veins, but in the calvaria, the physical features of the scalp and of the galea aponeurotica make impossible a similar arrangement of such superficial cisterns for venous overflow. The prime importance to the organism of the intracranial contents make it imperative that venous congestion be

avoided and for this no more efficient plan than the venous canals of the diploe could be devised. The occurrence of true intraosseous tuberculosis is practically confined to the zygomatic bone. When tuberculosis occurs the osseous changes are due to the hyperæmia it provokes and they conform to the changes found in the milder inflammatory conditions and are sooner rather than later complicated by the addition of sepsis.

When tuberculosis does occur in the skull it begins in the diploe and extends outward so as to perforate the outer table. It is found most commonly in children and in the frontal, parietal and temporal bones, and usually with the formation of a fine, worm eaten, circular sequestrum.

Two points with regard to tuberculous disease in a vertebrae must be borne in mind:

- (1) that the varieties of the disease in a vertebrae depend on the blood supply of a vertebrae;
 - (i) Medullary; (ii) Epiphyseal; (iii) Periosteal;
 - (iv) Anomalous:
- (2) that while compression of the cord with paraplegia may be due to
 - (i) oedematous membranes; (ii) tuberculous granulation tissue; (iii) abscess formation; (iv) sequestrum or tissue debris; (v) pathological dislocation;

yet paraplegia may arise apart from mechanical compression as a result of toxic and vascular changes, which often account for transitory paraplegias; this is due to repeated minor circulatory damage by chronic disease going on year after year with little outward sign.

[Lecture delivered before the Madras University Extension Board.]

Selected Articles

THE MAJOR IMPORTANCE OF MINOR INFECTIONS

BY PHILIP A. CAULFIELD, M.D.

*Associate in Surgery, Georgetown University School of Medicine,
Washington, D.C.*

The ever increasing number of industrial accidents each year calls upon the doctor to treat all types of wounds and injuries arising from these accidents. By far the greater number of these wounds fall into the category of Minor Surgery and are treated, not by the specialist, but by the general practitioner. It is important, therefore, that all doctors have a thorough knowledge of the best methods for treating these wounds. He should further know how to care for any complications that may arise in the form of infection, and it is these infections and other minor surgical infections that I wish to discuss.

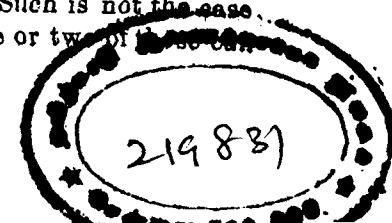
For the sake of clarity, I shall consider these infections under the following groups: (1) Wounds in general, (2) infections about the hand, (3) lymphangitis and lymphadenitis, (4) furuncles and carbuncles, and (5) erysipelas.

WOUNDS IN GENERAL

Every accidental wound should be considered as a potential reservoir of infection. However, if these wounds are properly treated most of them will heal without any evidence of infection. The greatest danger of infection in wounds is not so much from the mechanical force producing them as from the hands and instruments of the doctor treating them, since these are more likely to be contaminated with virulent organisms. Again, the mechanical force producing the wound enters it but once, whereas the hands and instruments enter it many times. This entrance usually takes place after the flow of blood has stopped, and infection is well known to be harbored better in clotted blood than in freely flowing blood. Therefore, before an attempt is made to repair any wound, all instruments to be used should be carefully sterilized and the hands carefully washed, the same as in any major surgical procedure, for it is far better for a wound to heal without any attempt at mechanical repair if it remains uninfected, than for a repair to be done and infection to be thereby carried into the wound. The following procedure should be strictly adhered to if infection of accidental wounds is to be prevented: (1) All bleeding should be stopped, the wound flushed with soapy water and its edges washed and shaved, (2) all loose and devitalized tissue should be removed, (3) all foreign bodies should be removed, (4) all cavities and recesses and excavations should be opened and the repair should be such that these are obliterated. The wound should then be flushed with 95 per cent alcohol, and if badly contaminated, it should be first cauterized with pure phenol. All accidental wounds should be drained and the drain allowed to remain in place no longer than is necessary to remove serum or liquified fat. Forty-eight hours is usually time enough to accomplish this unless infection occurs, and then the drain should remain in the wound until all pus has been removed.

No wound on the surface of the body or potentially infected wound should ever be sutured by a continuous suture. All sutures should be interrupted, since a continuous suture inhibits the flow of blood to the wound which is so necessary for the prevention of infection and the rapid repair of living tissue, and also because a continuous suture prevents free drainage. Another reason why the continuous suture is inadvisable in the repair of wounds on the surface of the body is that if the wound does become infected the entire suture must necessarily be removed and, therefore, will be followed by a wide gaping of the wound edges. Such is not the case of interrupted sutures in infected wounds. One or two of these can

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be removed and drainage inaugurated, and the remaining portion of the wound will be held together by the other sutures. For all intents and purposes, it is best to let the wound remain open without a dressing unless such a procedure is impractical. Dressings do not prevent infection and are used merely to absorb serum or blood or for an esthetic reason—to cover an unsightly scar. Most patients will object to this procedure, but from a practical standpoint—and this is particularly true in children—a wound uncovered can be washed and bathed with soap and water, and the possibility of infection being rubbed into the wound by a contaminated dressing is greatly reduced.

INFECTIONS ABOUT THE HAND

Of all the members of the body, the hand is by far the most frequently involved in accidents. This is because of its peculiar function. These infections of the hand are most serious and I am afraid that many times we fail to give them the consideration that we should. However, my work in the surgical clinics of various hospitals during the past 6 years has taught me to handle these injuries with a great deal of reverence. Kanavel many years ago realized their importance and published a classical work dealing with infections of the hand, a book which has since been revised 5 times. I will consider these infections under the following headings: Paronychia, Felon, Acute Tenosynovitis and Space Infections.

Paronychia. Commonly known as a "run around." The removal of "hang nails" and manicuring of the nails are largely responsible for this type of infection. The cuticle is broken, and the organisms lodge in the recesses of the skin overhanging the nail. The typical paronychia starts as a red, hot, painful area along one edge of the nail 3 or 4 days after the initial injury. The infection is apt to spread, however, around the base of the nail involving the entire cuticle. The swelling usually involves the entire distal phalanx of the finger causing it to throb, but point tenderness is most marked over the cuticle. It is best not to make any attempt at incision during this initial stage of inflammation but to advise hot soaks until a small abscess forms close to the edge of the nail, giving the nail a greenish yellow discoloration. At this time the dead overlying skin can be removed and the abscess aborted without resorting to anesthesia. If the pus has worked its way beneath the nail the nail will be elevated and any loose portion should be removed. Sometimes it is necessary to remove the entire nail. This is best done with sharp scissors. Anesthesia is not needed. The infection will subside in 3 or 4 days and complete healing will eventually follow, although a complete new nail will not be grown in less than 3 or 4 months.

Felon. Because of the peculiar anatomical structure of the distal phalanx of the finger, infected wounds over the anterior pad are

very apt to be followed by osteomyelitis of the bony phalanx. The reason for this can be briefly described. The periosteum of the distal phalanx is directly connected to the skin by small fascial bands, and infection in the skin works its way along these bands to the periosteum. The subsequent development of osteomyelitis is further enhanced by the fact that the nutrient artery to the distal phalanx only supplies the proximal third of the bone, the distal two thirds being supplied directly from the surrounding soft tissue. Therefore, edema occurring in the fat pad causes thrombosis and mechanical blocking of these vessels and naturally lowers the resistance of the distal end of the bone. All that is required for the treatment of this condition is adequate drainage. An incision should be made along the lateral margins of the finger, since a median longitudinal incision is apt to be followed by a troublesome scar. Without a doubt the so-called fishmouth incision is attended with the best results. This incision makes a flap of the anterior pad down to the bone and allows for free drainage and the escape of sequestered bone. A small piece of rubber tissue should be placed between the flaps for 3 or 4 days. The use of moist dressings and adequate splinting are the only after treatment necessary. A word of warning at this point may be timely. Unless the infection has spread to the middle phalanx, one should hesitate before deciding to remove the entire distal bone, because this bone regenerates very rapidly following the most severe infections, and even if it is entirely involved it will work out if the skin incision is adequate.

Acute Tenosynovitis. This is the most serious form of infection in the hand, both because of its severity and because of the disability which it often produces. The infective agent, which in serious cases is usually streptococcus, is generally carried in to the tendon sheath on the flexor surface by a pin, a needle or some other small object. The most dangerous anatomical site for these punctured wounds is the proximal flexion creases of the finger, for it is here that the tendons are most superficial. Suppuration does not immediately distend the entire sheath, so that a timely incision may prevent its spreading into the palm of the hand. Excruciating pain, tenseness, tenderness over the involved tendon which is held in moderate flexion and pain on motion of the finger following a slight wound in the above-stated region should lead to the diagnosis of a tendon-sheath infection. Many times the symptoms of tenosynovitis may not be sufficiently distinct to enable one to say positively whether pus is within the tendon sheath or merely subcutaneous. This distinction is of less importance, since in either case an incision is necessary for drainage and at the time of the incision examination of the tendon sheath can be made, which if found to be involved, can be opened widely. Incision into the tendon sheaths should always

be made along the lateral margins of the finger between the creases if possible and never on the palmar surface. One may pass an artery clamp across the digit from one lateral incision to the other and drain by rubber tissue or by a rubber band. Post-operative treatment should consist of massive wet dressings or hot hand soaks. The drains can be removed on the third or fourth day and the patient encouraged to move the involved finger. If there is extensive involvement of the sheath or sloughing of the tendon one should warn his patient that his injury is liable to be followed by considerable loss of function.

Space Infections of the Hand. There are 4 spaces in the hand and 2 in the wrist that must be considered. The source of infection of these is either a spreading infection from the fingers, usually acute tenosynovitis, or a primary wound on the, palm of the hand. Swelling is usually most marked not in the palm but on the dorsum due to the redundancy of the skin in this region. However, on careful examination point tenderness over the infected space will soon determine the location of the pus. The hypothenar space is only a potential space and seldom becomes involved; however, if infection does separate the muscles of the hypothenar eminence, a small incision directly over the eminence will be sufficient to abort the pus. If the infection is located in the thenar space the pathognomonic sign is ballooning of the thumb and marked swelling of the digital fold between the thumb and index finger. Drainage is best secured by a longitudinal incision on the dorsal surface of this fold, inserting a clamp through the incision into the thenar space beneath the thenar eminence and allowing a piece of rubber tissue to remain in place.

The mid-palmar space lies between the deep palmar fascia and the interosseous membrane of the 4 metacarpal bones. It is most often involved by infection spreading from the fingers by way of the tendon sheaths. However, it can be infected by an injury extending directly into it. The pathognomonic signs are obliteration of the normal palmar concavity, pain on direct pressure over the palmar space and marked swelling of the entire hand, most pronounced on the dorsum, as the firmness of the palmar fascia limits the amount of swelling on the palmar surface. Drainage of this space should be made through the lumbrical spaces between the third and fourth or the fourth and fifth fingers. A clamp should be inserted through the wound deep to the palmar aponeurosis and a drain inserted. The reason an incision is made in this region is because of less likelihood to injury of the important structures of the hand. The postoperative treatment should consist of splinting of the hand with the fingers in full extension to prevent flexion deformities. Massive wet dressings should be employed or periodic soaking of the hand in hot magnesium sulphate solution.

Because of the peculiar arrangement of the mucous sheaths of the hand, infection occurring in the little finger is apt to pass along the course of the mucous sheath of this finger and localize in the ulnar bursae beneath the annular ligament. This extension is sometimes difficult to diagnose. The valuable sign, however, is the presence of exquisite tenderness just proximal to the distal flexion crease at the wrist. Swelling is most marked on the dorsum of the hand, and a general fullness in the palm is found although the palmar concavity is still present. The edema and swelling are of such a character that fluctuation can seldom be elicited, and with the above signs and symptoms one should never wait until fluctuation is present, but should operate immediately, the incision being made between the tendons of the flexor carpi ulnaris and the flexor digitorum sublimis muscles. This will open directly into the ulnar bursae. Because of the close proximity of the radial bursae to the ulnar bursae in the wrist this space often becomes infected by direct extension from the ulnar bursae and this is the reason why infections in the little finger sometimes involve the thumb. However, the radial bursae can also be involved by infection originating in the thumb. The signs and symptoms of infection of these bursae are practically the same as ulnar bursitis except that the tenderness and pain are most marked along the radial side of the wrist and over the thenar eminence. An incision made directly over the proximal portion of the thenar eminence will open into this space. Drains should be inserted and the wound treated as previously stated for other infections about the hand.

Infections on the Dorsum of the Hand. These are entirely different from infections on the palmar surface. The tissues swell inordinately owing to the looseness of the skin. There is very little tendency to extension, and spontaneous localization is the rule. Adequate incision directly over the fluctuating mass will usually abort the pus.

LYMPHANGITIS AND LYMPHADENITIS

Lymphangitis. Inflammation of the lymphatic vessels is due to the entrance of bacteria through a wound or on abrasion of the skin which can be so minute as to escape detection. Some of the most serious forms of lymphangitis are due to puncture of a finger at an autopsy or an operation. The streptococcus is usually the offending organism.

The inflammation commences in the lymphatic reticulum of the skin, and passes rapidly up the collecting vessels to the nearest lymphatic glands. Some idea of the rapidity with which infection may pass along the lymphatics may be had from the work of Hudack and McMasters. Using a patent blue dye they noted that 5 minutes after an intradermal injection on the volar surface of the forearm the dye had spread 15 centimeters from the site of the

original injection and after 8 minutes had spread to the axillary lymph glands.

This evidence of the rapidity with which infection may spread into the general circulation warns us that active therapy should be started early. Usually the appearance of these red streaks up an arm or leg is presumptive evidence that pus has not yet formed. This is an important point, for most fatalities following minor infections are due to untimely probing or incision of a wound the minute lymphangitis appears. The best form of therapy at this time is to incorporate the injured part in massive dressings saturated with hot magnesium sulphate solution and institute general eliminative therapy such as hot drinks of a goodly quantity of fluid. An incision of any kind should not be made until definite evidence of localization of pus has occurred. Many times an incision is not necessary and the condition undergoes resolution without the formation of pus. If an incision is needed it should be just large enough to abort the pus and no larger, for cutting into healthy tissue will often tend to spread the infection.

Lymphadenitis. The lymphatic glands being one of the main lines of defense of infection from without, it is but natural that they should be the seat of inflammation. All degrees of inflammation may be encountered depending upon the virulence of the infecting bacteria. Two main forms may be encountered, the acute and the chronic. The chronic form is usually due to some wasting disease such as tuberculosis and syphilis and does not come under the scope of this paper. The acute lymphadenitis is usually due to infections of the skin or mucous membrane. The glands become swollen, tender and painful following lymphangitis. Sometimes it is possible to trace the course of the infection from the primary focus along the inflamed lymph vessels. Often the infection appears to pass directly from the initial lesions to the glands leaving the intervening area unaffected. This is usually the case in the more benign strains of infecting organisms. No matter how acute the inflammation may be, it usually ends in resolution if the same form of therapy as instituted for lymphangitis is employed. Should the infection be virulent, especially if it is due to streptococcus, suppuration may occur. With definite evidence of the formation of pus an incision should be made through the point of greatest fluctuation. Here again the incision should be small, just sufficient to drain the pus. Rubber tissue should be used as a drain, since gauze has a tendency to dry and plug the wound. One should not be surprised if long after the primary infection has subsided the glands remain swollen and indurated, for it sometimes takes 3 or 4 weeks before complete resolution occurs and they regain their normal size.

FURUNCLES AND CARBUNCLES

Furuncles. Best results are gained when dry therapy is resorted to for this condition, as moisture tends to macerate the surrounding skin and allows the Staphylococcus to penetrate and form another furuncle. Absolute rest, dryness, cleanliness and heat are essential in the preliminary stages. In 3 or 4 days a greenish white cap develops. This can then be lifted off without pain or discomfort and the pus evacuates itself. If another cap forms this too should be lifted off. Complete resolution usually occurs in about 1 week. There is no systemic treatment which will prevent recurrence of a crop of boils. Reduction of the carbohydrate intake and the use of autogenous vaccines has met with favorable approval by many observers. Irradiation with X-ray is an excellent form of therapy and causes rapid resolution, which favors the non-spreading of the organisms to the surrounding skin. Bacteriophages have been used with good results by some physicians.

Carbuncles. This condition is a series of communicating abscesses which discharge by separate openings on the surface. It is not a group of boils. Treatment here is general as well as local, the general treatment being supportive and eliminative. The local treatment consists in aborting the pus. Many if seen early undergo rapid resolution if irradiated with X-ray, with a minimal loss of substance of the skin. In the later stages X-ray is of little benefit, and an incision has to be made. This is usually crucial, and the skin flaps are dissected away from the deep fascia as far back as the skin is undermined by pus. Excision of skin should never be done, since it is impossible to tell at the time of incision what skin is viable and what skin is necrotic. Loss of substance of skin has been greatly reduced by adhering to this rule. As in furuncles, medicaments to aid the body defense powers have not proven their worth. Bacteriophage, however, is very popular and in time may come to be generally used.

ERYSIPELAS

This condition can no longer be regarded as an entity, since it is a form of streptococcus cellulitis and not a special type of infection as to origin, symptoms, course or outcome. There is no more need of isolating such a case than there is of isolating any other case of cellulitis. It usually lasts from 7 to 10 days, running as set a course as measles or scarlet fever, and its rash is just as easily controlled as these. Cauterants such as iodine and carbolic acid are of no more benefit and often prove harmful by devitalizing otherwise healthy skin.

The treatment is both local and general as in any other type of streptococcus infection. General treatment is supportive. Except in facial erysipelas, the original focus of infection should be located and if it fluctuates, should be opened and drained. In facial

erysipelas, discretion is the better part of valor and the original lesion should not be touched—rather let it point and rupture spontaneously. The blebs should not be opened until the third or fourth day, and care should be taken not to contaminate the skin by the serum contained in these blebs. The entire area should be covered with gauze soaked in hot magnesium sulphate solution. When the stage of desquamation is reached, itching and irritation may be relieved by dusting with zinc stearate. Erysipelas occurring at a mucocutaneous junction is extremely dangerous to life, and great care and judgment is needed in handling these cases.

Erysipelas antitoxin if administered early is of undoubted value, particularly in facial erysipelas. The dose should be 30 or 40 cc. in 100 cc. of normal saline administered intravenously. The mortality from erysipelas has been greatly reduced since the routine use of the antitoxin has been inaugurated. Transfusions of whole blood have also proven an effective weapon in combating the ravages of this disease.

COMMENT

No part of the body is exempt from accidents or infections, and it is because of the general interest to all that I have chosen to discuss these subjects. Many minor surgical conditions become major surgical problems because of neglect or ignorance on the part of the physician who first treated the injury. The same care in diagnosis, handling of tissue and treatment should be exercised in these conditions as in major surgery. A good knowledge of what can happen to a life or a member of the body following a pin prick is essential in the education of all physicians.

THE URINARY TRACT INFECTIONS OF INFANTS AND CHILDREN.

BY C. W. VINING, Leeds.

INTRODUCTION.

The bacterial infections of the urinary passages form a common and important group of diseases during the early months and years of life. The name "pyelitis" has for many years been given to these infective processes and for convenience sake the word is worth retaining, but it should be pointed out that there is no pathological evidence to show that the pelvis of the kidney is primarily at fault or indeed mainly at fault. In fatal acute cases changes are found in the kidney on both sides and in such cases the kidney pelvis may or may not show evidence of an inflammatory process. There is no doubt that the severe symptoms of the acute case are due to kidney damage.

The disease is found with greatest frequency during infancy and becomes less common as age advances. The female child is

more frequently attacked at all ages; the difference between the sexes, however, is not so marked under the age of two years. After two years, pyelitis in boys becomes comparatively infrequent. In the large majority of cases the organism belongs to the coliform group.

Pyelitis may be a primary or secondary infection. It is not an uncommon development during the course of any chronic health defect and the urinary tract is very susceptible to the influence of vitamin deficiency.

There is now a good deal of evidence to show that some of the cases of chronic pyuria are associated with some congenital abnormality usually in relationship to the ureter. In a proportion of these cases aberrant blood vessels have been demonstrated crossing the ureter. The partial obstruction which may be produced whether by kinking, imperfect formation or from direct pressure by an aberrant vessel, will act no doubt as a predisposing cause of urinary infection.

DIAGNOSIS.

The diagnosis depends most especially upon a careful examination of the urine; unless care is taken the diagnosis may be made more frequently than is justified. The essential evidence is the finding of a frank bacilluria with or without pus cells. Pyelitis may be negatived at once in the case of the urine which proves to be crystal-clear when examined in a good light in a clean tube. If the urine is turbid in the slightest degree and if the turbidity cannot be got rid of by warming after the addition of a few drops of dilute acetic acid pyelitis is likely and should be verified by the examination of a hanging drop preparation using the sixth power of the microscope with the light turned down.

The diagnosis based upon the cultivation of organisms by the bacteriologist may be misleading. The growth of a few colonies of *B. coli* from the specimen sent by post, or after standing some hours, is of little value. The same criticism holds good with the finding of a few pus cells after the centrifuge has been used. In the case of the urine which shows a frank bacilluria care must be taken to exclude the possibility of faecal contamination, especially in the case of the small infant. I have been caught out more than once by finding pyelitis in a urine which had been passed into a chamber previously used for a bowel movement and not thoroughly cleansed.

In the case of a suspected acute pyelitis a negative urine during the first few days of the illness should not be accepted as conclusive evidence that the urinary tract is not at fault. It has been pointed out by Dr. Still that in the acute cases several days may elapse before bacilli appear in the urine and I have noticed this on several occasions. This is a point which supports the theory that infection of the tract may take place via the kidney and is not in all cases an ascending infection.

The urine having been shown to contain bacilli and pus cells there are still mistakes to be avoided. A simple vulvo-vaginitis may easily produce contamination and in a child acutely ill with some extremely toxic illness such as a severe gastro-enteritis the urethral opening may be covered with a bead of pus in much the same way as the conjunctiva may be coated with a film of pus. It may, however, be here mentioned that an acute toxic gastro-enteritis may be associated with an acute pyelitis, and indeed the latter may be the more important clinical condition. In many cases of acute pyelitis there is a history of loose stools. When possible the specimen of urine examined should be obtained by catheter.

A further point to be remembered after the finding of pus and bacilli is the possibility that the primary trouble is not in the urinary tract. In the deficiency states of childhood, especially those associated with a lack of vitamins A and C, secondary infective processes may take place. In the case of infantile scurvy blood cells are frequently found in the urine, and with them bacilli and pus are usually associated. Small children are especially liable to multiple infections and one infection seems to lead on to others as the resistance becomes lowered, and in any long-standing wasting and nutritional disease a urinary tract infection may arise. This is of some importance as in treatment our attention must be directed to the individual as a whole and not only to the urinary tract. In cases of tuberculosis of the urinary tract pus cells and *B. coli* are always to be found and are clearly of secondary importance.

Looking back over a good many years it has been my impression that while I have met with a large number of ill children whose illness has been primarily and certainly due to a coliform infection of the urinary passages, I have also met with large numbers of poorly children whose illness or health defect has been only temporarily and often very fleetingly associated with the presence of coliform bacilli in the urine. No doubt some of these cases have had the urine contaminated in some way and one has been led astray by the want of care in collecting the specimen. In other cases, while I am sure these mistakes have been avoided, yet I am equally sure that the urinary infection which has been of quite a temporary nature has really been a chance finding and has had no important relationship to the actual illness from which the child has suffered.

CLINICAL MANIFESTATIONS.

From the earliest days of life, through infancy and childhood, illness may be caused by an infection of the urinary tract, usually with a coliform organism. Early recognition is important. In a considerable number of the cases there is nothing in the history to draw attention to the urinary tract and it is only by remembering the possibility that mistakes may be avoided. In the minority of the

cases the child has experienced pain in micturition or has been noticed to pass urine frequently or the mother has noticed some change in the smell or appearance of the urine. The acute cases may simulate closely disease of other parts of the body, especially meningitis and acute abdominal conditions. In the chronic cases the picture of a severe marasmus and debility may be produced. In all cases, whether acute or chronic, irritability, great fretfulness, restlessness, twitchings, and sometimes convulsions may be present. The course of the disease may be very variable. In many cases the illness seems to be quite temporary, in other cases it is intermittent, and frequent relapses are common. In the acute cases the severe symptoms may continue for several weeks and in the chronic cases there may be a prolonged nutritional disability lasting for many months.

An acute pyelitis is by no means a rare development in the newborn. At so young an age one's attention is usually drawn to the infant by the irregular temperature, which may be considerable. At the Leeds Maternity Hospital I have seen quite a number of newborn infants which were brought to my notice either because they had a considerable pyrexia for which there was no obvious reason or because they were unusually quiet or were acting abnormally in some other way. Several of these children also had loose green stools. Examination of the urine demonstrated turbidity due to a frank coliform bacilluria. With the free use of alkalies the symptoms quickly subsided.

At all ages the onset may be extremely sudden and may be accompanied by convulsions. Hyperpyrexia is not uncommon and there are few conditions which will cause such a high level of temperature as pyelitis. It is the only condition in my experience from which recovery may take place after the temperature has touched 106° or 107°. Head retraction, squinting, twitching, screaming, restlessness, pallor, and loose stools may be witnessed in the severe cases. Pain is probable and hyperæsthesia is likely. Blueness of the extremities and round the mouth is not infrequent. Pyelitis is said to be the only condition in a child during which rigors may occur. I have not myself ever witnessed a true rigor in a child but I have seen the twitching and the cold extremities and the pinched appearance. The child's appearance is sometimes that of a profound toxæmia of the type that one sees in the acute abdomen.

CHRONIC PYELITIS.

This may develop insidiously or may follow an acute phase. Wasting, debility, anorexia and fretfulness are the chief characteristics, and abdominal tenderness may be found. In some of the cases of chronic pyuria in older children the general health may be little disturbed. In such cases the possibility of a urinary tract tuberculosis should be considered.

In between the acute and chronic types there are to be met with cases of varying severity. The intermittency of the condition may be a striking feature, especially as regards the condition of the urine. Summing up my own experience I would say that pyelitis should always be suspected in any sick child for whose ill condition no physical signs are forthcoming, and especially should one be suspicious in the case of the infant or child with a continued temperature and with pronounced fretfulness or irritability and in the chronic cases when the chief symptom is wasting without obvious cause. In all cases, acute or chronic, irritability of the nervous system and fretfulness of a pronounced type is a very characteristic phenomenon, and I am in the habit of teaching that the two conditions which most frequently cause prolonged irritability of temper and fretfulness in a small child are Pink Disease and urinary tract infection.

PATHOLOGY.

There is now a considerable amount of evidence that in the fatal case the kidney is involved in the process. Wilson, Schloss and Schown and others have shown that the fatal cases have died from a suppurative interstitial nephritis. The macroscopic appearance of the kidney is not very striking. When the cortex is stripped the superficial vessels are dilated and a few points of early abscess formation may be made out. It is difficult to escape the conclusion that in the fatal cases infection has taken place via the blood stream, the kidney being first affected. It may here be mentioned that cystitis is comparatively rare during childhood, which is possibly a further evidence that the acute case is infected from above rather than from below.

It is rather a remarkable fact that there appears to be no connection between the nephritis of the text-book and the urinary tract infection. The urine in cases of nephritis is usually sterile and this even in those cases of hæmorrhagic nephritis which are believed to be related so frequently to bacterial infections of the naso-pharynx, respiratory tract, the skin and other parts. There is also no evidence to show that the urinary tract infections are the cause of later chronic nephritis. The frequency with which the coliform organism is found has led to the belief that the pyelitis is caused by an ascending infection and its greater frequency in female children seems to support this possibility. On the other hand, the infrequency of cystitis, the evidence of the seriously damaged kidney in the acute case, and the sterility of the urine during the early few days in some cases, gives force to the belief that infection is via the blood stream or possibly the lymphatic in at least a proportion of the cases. It may be mentioned here that it occasionally happens that the infectious agent is a staphylococcus. The *Bacillus coli* may or may not be present. In such rather rare cases the clinical picture

is like that of an acute focal hæmorrhagic nephritis as hæmaturia is always present. The presence or absence of casts will of course settle the point. In chronic pyelitis X-ray examination following the injection of Uroselectan B, or its equivalents, has demonstrated the frequency of some congenital anatomical defect of the ureter or the presence of an aberrant blood vessel which has led to partial obstruction, and it is believed that in some of the chronic cases this defect has predisposed the child to the urinary infection. Dilatation of the pelvis and of the upper part of the ureter is not infrequently seen. It may be mentioned that in chronic renal fibrosis of the congenital type pus cells and bacilli are usually found in the urine. In such cases a persistent low specific gravity will be of importance in coming to a diagnosis.

TREATMENT.

For the acute case the treatment of choice has for many years been the thorough alkalisation of the urine using potassium citrate and sodium bicarbonate, and so satisfying have the results been in a proportion of the cases that for the acute case the alkali treatment is likely to remain the routine method of attack. In a minority of the cases the alkali treatment fails. Either it is found that alkalinity cannot be produced or with the production of alkalinity improvement does not take place. In such cases hexamine has been suggested for a period of a week to ten days, to be followed again by another attempt at alkalisation. In my experience hexamine does not often prove successful when alkalies have failed.

Recent work in connection with the ketogenic diet has demonstrated its value in urinary tract infections. It has been shown that the improvement has been brought about by the presence of acetone rather than by any increased acidity of the urine. The ketogenic diet is not easily applied to small children and the recent introduction of sodium mandelate in conjunction with ammonium chloride seems to offer a useful alternative. In older children I have had several surprising results in chronic cases of pyelitis, but I have not yet tried it sufficiently often in the acute case in small children to decide its merits. In one case the treatment produced rather a persistent hæmaturia.

In the carrying out of the alkaline treatment sufficient alkali must be given to produce a definite alkalinity of the urine, and the alkalinity must be retained for a week or ten days. For a child of three years 60 grains daily to start with will be necessary, and the amount may have to be increased to 120 grains before alkalinity is obtained. In older infants and small children 90 to 120 grains will be necessary at the start, and over two years quantities up to 240 grains may be required. Improvement is unlikely until the urine has been alkaline for two or three days. The alkali should be given in frequently repeated small doses rather than in three large doses.

The treatment is not without danger: in some cases the urine refuses to become alkaline and the child becomes more ill. In such cases the kidney is almost certainly seriously affected and there is the possibility of alkalosis. I have seen several cases which have rapidly improved immediately the alkali has been stopped, although previous to starting the alkaline treatment the child has been quite ill and showed no improvement during its administration. As a rule doses of alkali should not be continued for more than a week or ten days in face of continued fever, prostration and toxemia.

This article is being written at the time when the treatment of the acute case is under review in the light of the results obtained in the older child with the use of sodium mandelate and I am rather of the opinion that the near future will show that good results will be obtained by its use in the case of the small infant with acute pyelitis. The administration of such large doses of alkali has always seemed to me to introduce an element of risk and possibly the decision will revolve round the problem whether the risk is greater with large doses of alkali or with large doses of ammonium chloride.

Apart from the actual drug treatment, whether by the use of alkalies, hexamine, or sodium mandelate, the child's general health must be watched. Fluids must always be given freely and the diet must be such as to supply sufficient vitamins, remembering that chronic vitamin deficiency may be an important factor in the production of a urinary tract infection.

(—University of Leeds Medical Society Magazine, May, 1936.)

Extracts

HISTIDINE TREATMENT OF PEPTIC ULCER.

(*Lancet*. 1936. i. 95).

The multiplicity of the methods of treatment devised for peptic ulcer emphasises alike the incompleteness of our knowledge and the need for caution. The chronicity of the diseases and its tendency towards relapse and remission make it an awkward subject for therapeutic investigation, and we are also handicapped by the difficulty of reproducing in animals the typical chronic ulcer found in man. Among the more interesting of the remedies now on trial is the amino-acid histidine given by injection. The work of Mann and Williamson and of Aron and Werss on dogs showed that the injections were given in the belief that the development of ulcers was in some way due to amino-acid deficiency. Further work led Aron to apply his theory to the treatment of peptic ulcer in man, and using intramuscular injections of a 4 per cent solution of histidine hydrochloride (Larostidin) he reported very good results in 17 cases. Since this paper several other records of the use of Larostidin have been published in various countries. The new form of treatment has been

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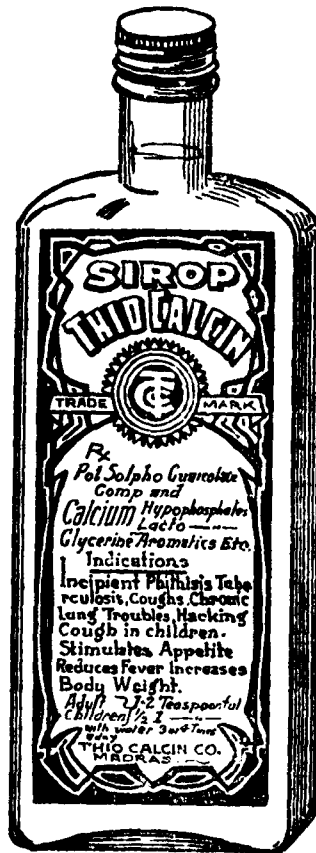
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adopted with unusual readiness because its advocates have claimed from the first that it largely, if not wholly takes the place of diet, alkalies, and rest in bed.

It will be generally admitted that whatever the treatment, medical or surgical, the ultimate prognosis as regards cure of peptic ulcer is at present none too good. Statistics of permanent cure vary between 50 and 90 per cent, but if a long enough margin is allowed, the final percentage is probably not above 75 and less for duodenal lesion. On the other hand, almost all forms of treatment seem capable of producing temporary improvement in almost every uncomplicated ulcer.

Histidine treatment differs from the others because it is based upon the view that peptic ulcer is at least partly a deficiency disease, and that injection of this amino-acid will correct part of the deficiency. It does not appear probable, however, that the diet of the patient, who afterwards gets an ulcer, is consistently deficient in protein—in fact, many are normally big meat-eaters—and if such deficiency arises through faulty digestion or absorption of proteins, no cause for the failure is yet known.

The dose recommended is 5 cc. of a 4 per cent solution of histidine hydrochloride given daily by intramuscular injection for a period up to 3 or 4 weeks and repeated as required. There are said to be no dangers, the patients need not be kept on a restricted diet, alkalies are unnecessary, and rest in bed is usually not required. Relief from symptoms follows as a rule in 2 to 6 days, and some 60 to 90 per cent of cures are recorded. But it is far too early to assess the value of this treatment. Already some disappointing relapses are known, and histidine is not a specific remedy for peptic ulcer, in the sense that it does not counteract the cause. If this is so, it must seem unwise to relax attention to dietetic and general measures while carrying out injection treatment. At the same time the many accounts of relief of symptoms, increase in weight and well-being, and removal of clinical and radiographic evidence of active ulceration point to the treatment being in some way beneficial. Judgment of its value must be formed largely on clinical experience. Whether its action will turn out to be in any sense specific or is merely (as seems likely) comparable to that of other injected substances, and whether it can lead to lasting cure or replace other therapeutic measures, are questions that can be decided only by further study. The time has not yet come for accepting an entirely new theory of ulcer causation, and for abandoning the ordinary rules of diet and management.

TREATMENT OF JOINT, GLAND, AND BONE TUBERCULOSIS.

(*Journal of A. M. A.* 1936. cvi. 1215.)

Discussing the problem of surgery in tuberculosis other than pulmonary, Dr. Max Jerusalem of Vienna said that the nature of the

disease is important, whether fibrous or destructive. In the last decade, conservative procedure has gained many adherents, particularly as the importance of heliotherapy and roentgenotherapy has been recognised. Nevertheless in recent years many Swiss specialists have rather turned to surgical treatment, which, if the indication is correct, may save much time and effect speedier cures. The guiding principles are as follows.

Treatment of bone, gland, and joint tuberculosis should be predominantly and almost entirely conservative. Selection of therapeutic measures must depend on the particular conditions presented in each separate case.

When operative treatment is indicated one should be guided by the following suggestions:—

1. Isolated, not unduly large, firm, movable glandular tumours on the axillae or neck, when refractory to radiotherapy, should be extirpated.
2. Wide electrosurgical opening of small cold abscesses on the thorax or neck should be done, evacuation without drainage (in order to avoid mixed infection), then complete suture and, after primary healing, roentgenotherapy.
3. Bone sequestrums should be removed only if spontaneously projecting outward in a necrotic state or as foreign bodies lying in the soft parts.
4. Lesion on tendon sheath that may cause liquefaction should be treated as under 2.
5. Para-articular bone areas may occasionally be removed. This requires a large amount of skill.
6. Resection of the knee joint should be done in adults presenting a shrunken fibrous capsular fungus with erosion of the cartilaginous covering.
7. Amputation should be done in cases of progressive joint processes in aged patients; otherwise treatment should be as conservative as possible.
8. After successful treatment, osteotomy and arthroplasty should be chosen as a secondary operation for the purpose of correcting posture.
9. Contra-indicated are simple incision and drainage of a cold abscess, scraping of central bone sequestrums, extirpation of multiple diffuse glands on the neck or axillae, and resection of carious ribs, as well as resection of all joints with the exception of the knee joint as indicated.

THE ELLIOTT TREATMENT IN PELVIC INFLAMMATION & DYSMENORRHOEA. (L. D. DICKEY: *Colorado Medicine*. 1936. xxxiii. 16.)

The Elliott treatment consists of the application of heat in the vagina at a controlled temperature and pressure, by a machine

which circulates hot water through a distensible rubber applicator. The average treatment starts at 115 degrees F., is increased approximately $\frac{3}{4}$ of a degree per minute to 130 degrees, where it is maintained for the remainder of an hour, with the pressure at the highest level consistent with the patient's comfort. The first 2 or 3 treatments are 30 to 40 minutes in duration with a maximum temperature of 125 degrees to accustom the patient gradually to the treatments.

This method was devised in 1909 in Seattle by Dr. Charles Robert Elliott, who conceived the idea of an internal hot water bottle for use in the treatment of pelvic inflammatory disease. The greatest amount of information concerning the Elliott treatment has come from the work of Holden at the Bellvue Hospital and Counsellor at the Mayo-clinic.

Heat applied locally acts chiefly through changes in the vascular system producing vasodilation, increased rate of surface circulation, volume flow of blood, thermal conduction, lymph formation, oxygen tension of the blood and phagocytosis. Heat as applied to the pelvic region by the Elliot method gives rise to an average increase of 17 per cent in the leucocyte count in 87 per cent. of the series of cases studied by Holden and Gurnee, the production of a profuse discharge from the cervix and vagina which subsides after the first few treatments, a local elevation of temperature in the vagina, rectum, bladder, and peritoneal cavity of from 5 to 7 degrees F. and a reduction in the R. B. C. sedimentation rate.

The patient with pelvic inflammation who is subjected to treatment with heat by this method is surprisingly relieved of her pain, chronic fatigue, and exhaustion. There is marked resorption of exudates, diminished tenderness on bimanual examination, definite destruction of bacteria, and marked increased activity of the cervical glands, which at first pour out large quantities of secretion. Improvement continues after treatments are discontinued, with the gradual and continual return to normal of the sedimentation rate. Any burns which may be found on speculum examination are not significant and would heal satisfactorily by continuing daily treatment at a lower maximum temperature. This prevents the folds of the vagina from healing together. These burns are usually due to incomplete distension of the applicator. The efficiency of this method is based on the fact that the vaginal mucosa is less sensitive to heat than is the vulva and perineum.

Under the former conservative regime patients with pelvic inflammatory disease were usually discharged symptom and pathology free but not organism or pathology free. By the use of the Elliott treatment we are now able more often to discharge our patients organism and pathology free. During the past 2 years the author has used the method in 20 cases, which include acute salpingitis, acute peritonitis of pelvic origin, chronic salpingitis,

leukorrhoea, cervicitis, gonorrhoea, puerperal infection, post-abortal infection and dysmenorrhoea. As a guide to therapy more value is placed in the sedimentation time than the white count for, when the sedimentation time remained stationary, it is a fairly good indication that the case would not respond to treatment by this method; in those in which the sedimentation time dropped and the white cell count becomes elevated, results are obtained.

Reviews.

JOURNAL OF THE ANDHRA MEDICAL ASSOCIATION. Vol. I, No. 1, April 1936. Subscription: Re. 1 8 for members and Rs. 2-8 for others:—We have received the first number of this quarterly Journal published from Bezwada in the interests of the medical profession of Andhradesha. Major, M.G. Naidu of Hyderabad (Dn.) is given the first place of honour with some thoughts on "the trend of modern medicine." Dr. T. S. Tirumurthi of Vizagapatam describes "the present state of public health in India" after going into the care of the problem. Other articles which hold the interest of the reader are "non-specific protein therapy" by Dr. C. Suryanarayanamurti of Bezwada, "x-ray of the lung" by Dr. M. Kesava Pai of Madras, and "a few thoughts on longevity" by Canon D. Anantam. These and a few case reports and extracts together go to form a promising medical periodical, which is bound to prove worthy of the medical profession in Andhradesha.

THE NAIR HOSPITAL DENTAL COLLEGE, BOMBAY. The prospectus for 1936-37 of this College, which we have received due to the kind courtesy of the Dean, Dr. V. M. Desai, D.D.S., shows that a thorough and scientific training in dentistry is obtainable therein. The course of studies extends over four years embracing the fundamental medical sciences as well as dentistry proper, both theoretical and practical, but only two years for qualified medical men. The College is recognised by University Dental Colleges in some foreign countries, but authorities in India have been very hesitant in doing so. It is very gratifying to note that the future programme of activities of the College will be the inauguration of a public dental health service, education of the public in preventive dentistry by means of dental health exhibitions and children's clinics. The College also runs a post-graduate course. We wish the College a long and useful career in a much neglected field, and hope that the products of the College will carry the torch of modern dental science throughout this country.

ANDHRA MEDICAL CONFERENCE. The sixth Andhra Medical Conference was held on the 4th and 5th July at the Museum Hall, Rajahmundry. Dr. S. V. Rama Rao, Chairman of the Reception Committee, welcomed the delegates and said that the establishment

of the Indian Medical Council had created great disharmony in the medical profession by the exclusion of medical licentiates from its purview. He deplored the non recognition of the Vizagapatam Medical College by the Indian Medical Council. Dr. A. Lakshmi-pathi, B.A., M.B., C.M. of Madras declared the Conference open. Dr. T. S. Tirumurthi, M.B., C.M., D.T.M. proposed Dr. B. C. Roy, M.D., M.R.C.P., F.R.C.S. to the chair. Dr. Roy referred to the high death rate in this country and said that the members of the medical profession had a great duty to perform in the prevention of disease. He said that in India rich and middle class people paid amply to a lawyer when there was a dispute over a trivial sum, but paid grudgingly to a medical man who had saved them and their families from death, and that these people are now resorting to hospitals intended for poor men due to unwillingness to pay medical bills. He appealed to the medical men to produce a synthesis of the eastern and western systems of medicine. He said that there were more doctors in towns than in rural areas due to mal-distribution and not excessive production of medical men, as shown by the fact that there was one medical man for every 15,000 people in this country, as compared to one for every 880 persons in Austria, one for every 900 in America, and one for every 1490 persons in England. The second day's deliberations of the Conference were presided over by Dr. D. S. Ramachandra Rao, M.A., M.D. president of the Andhra Medical Association. Among the papers read were those on "the relative value of chemical analysis and clinical experience" by Dr. A. Lakshmi-pathi; "impotence" and "honorary medical service in the mofussil stations" by Dr. T. Kanakaraju of Ramachandrapuram; "pitfalls in the practice of midwifery by male doctors" by Dr. V. V. Krishnayya of Cocanada; "a case of fatal fever during a menstrual period" by Dr. C. Suryanarayanamurthi of Bezwada; "arthritis" by Dr. Y. V. Subba Rao of Rajahmundry; "complications in a cataract operation" by Dr. Ramakrishna; "infantile liver" by Dr. G. V. Subba Rao; "modern gas warfare" by Dr. P. Visweswara Rao; and "eclampsia" by Dr. Allen of Pithapuram. Several resolutions on current medical topics were also passed. The annual meeting of the Andhra Medical Association was also held, when Dr. D. S. Ramachandra Rao was re-elected president, and Drs. M. V. Krishna Rao and P. V. Brahmanandam were elected joint secretaries.

RAMNAD DISTRICT MEDICAL ASSOCIATION. A monthly meeting of the above association (which was formerly known as the Chettinad Medical Association) was held on the 24th May at the Ramnad High School under the presidency of Dr. B. V. Varadachari, B.A., M.B., C.M. District Medical Officer. After tea and reading of the minutes, the president showed several interesting cases. Dr. T. S. Shetty showed the bits of thyroid cartilage he had removed after a preliminary

tracheotomy in a case of accidental traumatic rupture of the cartilage with subcutaneous emphysema and severe dyspnoea from blocking of the air passages; he also showed maggots removed from the middle ear in a case of middle ear suppuration.

TUTICORIN MEDICAL UNION. The second Anniversary of this Union was held on the 6th June at the S. A. V. High School. Dr. I. M. Orr of Neyyoor with some of his assistants and also some members of the Tinnevely District Medical Association were present. After tea and music, the meeting commenced under the presidency of Lt. Col. T. S. Shastri, I.M.S., District Medical Officer of Tinnevely. The annual report was read and adopted, Dr. J. M. Orr, M.D., F.R.C.S. delivered a lecture on "the relationship between medicine and surgery in the treatment of dyspepsia", Lt. Col. Shastri congratulated the Union on its good work. Dr. K. S. Iyengar, L.M.S., president of the Union proposed a vote of thanks. The meeting came to a close with a dinner at night.

TINNEVELLY DISTRICT MEDICAL ASSOCIATION. A meeting of the above association was held at Papanasam on the 27th June with Lt. Col. T. S. Shastri, I.M.S., District Medical Officer, in the chair. Dr. K. Rama Aiyar, M.B., B.S., secretary, read the minutes of the previous meeting. Dr. U. Rama Rau's proposed Bill for establishing a College of Physicians and Surgeons was discussed, and the opinion expressed was decidedly against the introduction of the Bill. The rules of the Tamil Districts Medical Federation were then considered and passed. Dr. K. Padmanabha Aiyar of Tenkasi read a paper on "constipation". After discussion the meeting terminated with a vote of thanks to Dr. Janardana Rao and his co-workers for arranging the meeting. A health exhibition was also held at Ambasamudram under the auspices of the Association, which was opened by Mr. M. D. T. Kumaraswami Mudaliar, President of the District Board. The exhibits were explained to the gathering by Dr. Lakshmana Aiyar of Kalladaikurichi.

TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION. A monthly meeting of the above Association was held on the 27th June in the E. R. High School, Teppakulam, Trichinopoly, when about 25 members were present. Dr. R. Srinivasan of Kulitalay was "At Home" to the members and Capt. Sangham Lal, I.M.S., F.R.C.S., District Medical Officer, Trichinopoly, presided. After the demonstration of a few X-Ray films and discussion of interesting cases, Dr. R. Kalamegham, B.A., M.B., B.S. delivered a lecture on "Diphtheria". A discussion followed.

GUNTUR DISTRICT MEDICAL ASSOCIATION. A meeting of this association was held on the 27th June at the Headquarters Hospital with Major A. Krishnamurthy, B.A., M.B., B.S., A.I.R.O., District Medical Officer, in the chair. After tea, Dr. P. H. Vittal Rao, M.B., B.S., D.S.O. read a paper on "Salpingitis."

PROFESSOR LUDWIG ASCHOFF. Of the University of Freiburg in Breisgan, celebrated his seventieth birthday on January 10, says the *Journal of the American Medical Association*. Aschoff's work embraces nearly all fields of general pathology and pathologic anatomy. He will probably be chiefly remembered for his contributions on thrombosis and on nephritis, and for his studies on the origin of inflammations. Among his many other achievements was his explanation, in collaboration with Jawara, of the bundle of His and his work on pulmonary tuberculosis, on the functional organisation of the stomach, and on the reticulo-endothelial system. He has made it possible to formulate a general theory of disease. Hand in hand with his manifest ability as a scientist go a lively technic of presentation and an extra-ordinary mastery of language, gifts all of which combine to place the name of Aschoff among the most illustrious in his field.

IVAN PETROVICH PAVLOV. The Contributions of Professor Pavlov to contemporary medical science have been of such a high order that words fail in their adequacy of description, says *American Medicine*, March 1936. Every physician is making daily use of knowledge gained through the efforts of Pavlov. Probably of outstanding importance are his contributions to the physiology of digestion gained by the technique of the "Pavlov pouch" and the working knowledge of the physiology of the nervous system which Pavlov's methods have supplied. The Soviet Government granted unusual recognition to Pavlov's work, when although not himself a communist, and frequently a critic of the government, he was made a free citizen and permitted to do his work unmolested. Medicine, science, and the world lost a commanding figure when on February 27th 1936 Ivan Petrovich Pavlov died at the age of 86.



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I think I can safely recommend your LAXANTHINE ASTIER & NEO-RIODINE for Rheumatism and other infections.

The Urine was also cloudy and full of sediments which disappeared after three or four day's use of Laxanthine Astier. It became clear and free from Phosphate and other sediments.

Yours Sinc,
DR. P. KRISH

Treatment of a case of Elephantia

By Prof. NOCUER MORE.

Chief of the Dermatologic Service.

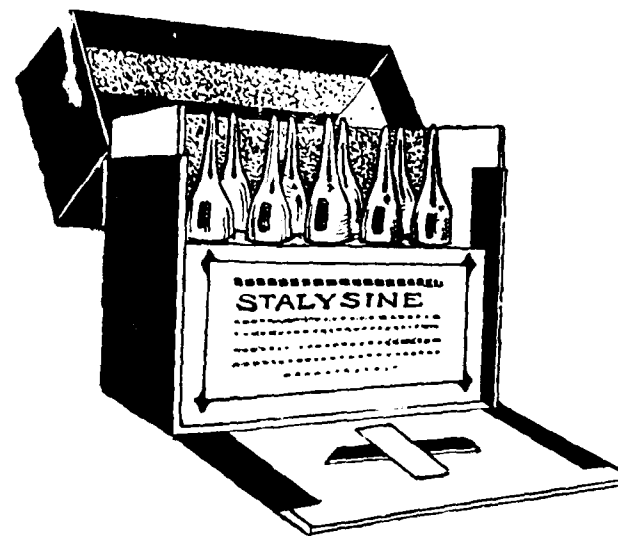
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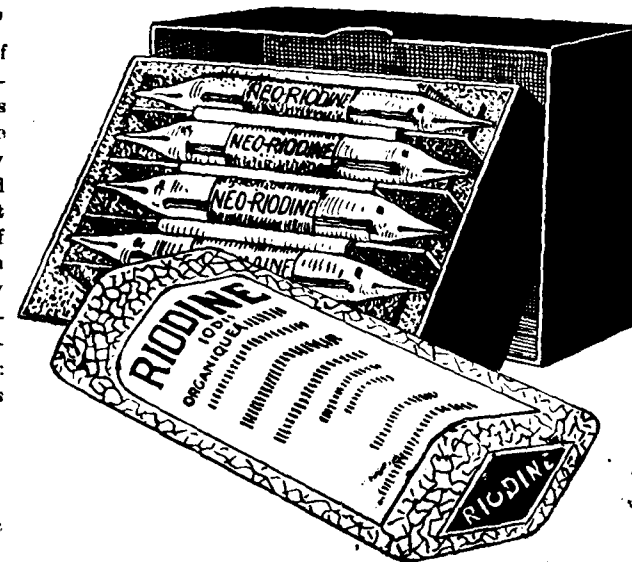
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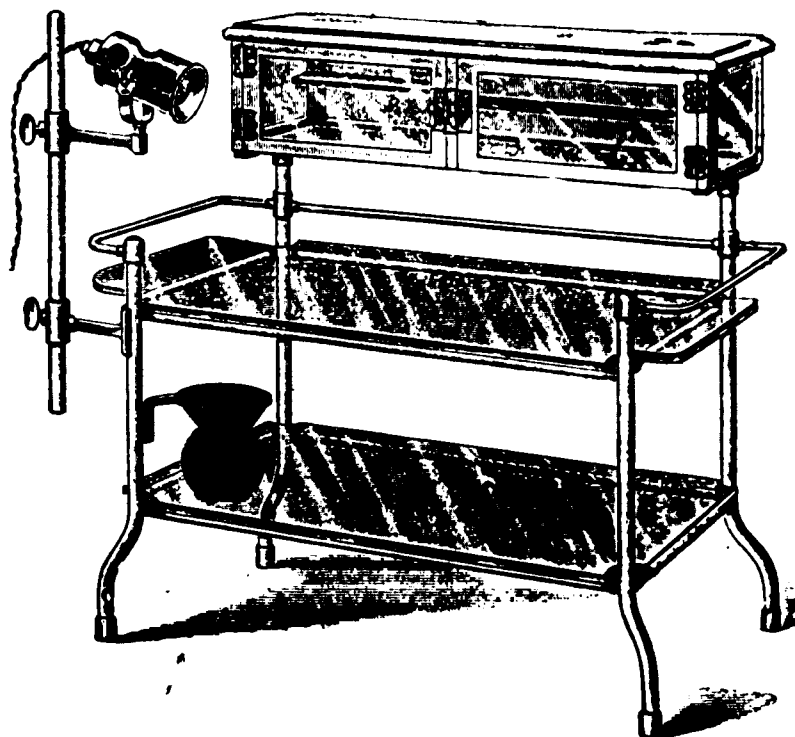
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