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(Detailed contents on page 2) (Index to Advertisements on page 4)

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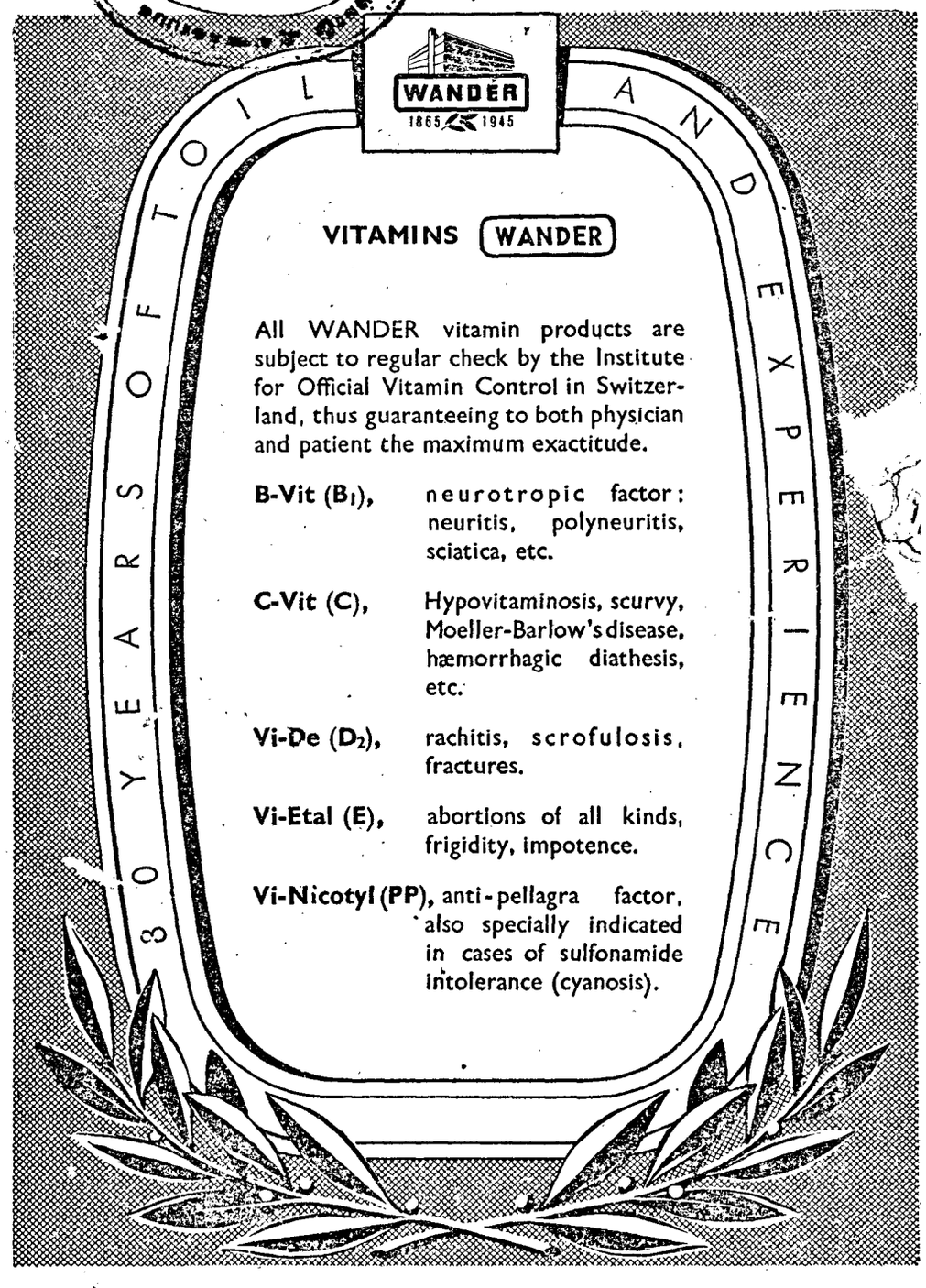

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CONTENTS

INDEX TO ADVERTISERS

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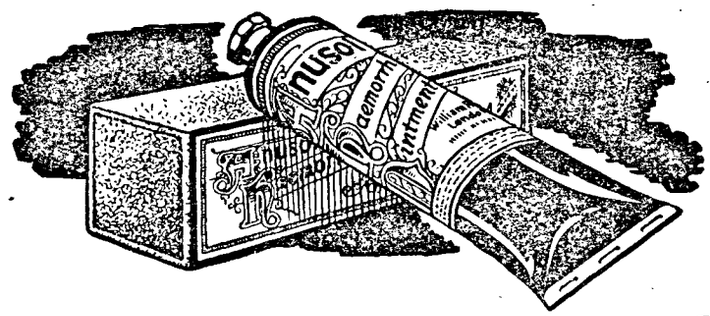

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Original Articles

Artificial Limbs*

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I. **Introduction.**—In India the manufacture and use of artificial limbs have not progressed much. All classes of people, particularly intellectual classes, of all religions, are averse to amputations where necessary partly because of the belief that no person, who dies without all his limbs intact, dies a "natural" death. As it is, amputations are being performed on the poor and the lower middle classes. The very poor use their disability for begging which they resort to as a source of livelihood, the middle classes are not able to pay for artificial limbs.

The causes of this aversion are many. One is the superstitious belief mentioned above, another is a dislike of a deformity. They would rather face death than go about with a deformed or mutilaed limb. A third cause is perhaps the fear that the wage-earning capacity will be diminished whatever be the deformity. It is worthwhile emphasizing here that with the modern types of artificial limbs, especially of the DeSoutter type the average wage-earning capacity is diminished only in the case of those engaged in certain types of occupations, such as those engaged in heavy industries or certain other trades and that the diminution of earning capacity does not depend entirely upon the site of amputation or of the length of the stump.

II. **Amputations and working capacity.**—In many cases the capacity for work is good when the amputation is comparatively high and in many low amputations the capacity is poor. Of course men who have had *double amputations* or *amputations near hip*, who previously did hard manual labour, would not be able to return to their previous occupations and they must be taught a trade which could be done in a seated position.

Amputations above knee:—It is difficult for a poor agriculturist to continue his occupation. But office and desk work, work of a supervisory nature are easily performed.

* Specially contributed to THE ANTISEPTIC.

Amputations below knee :—Even with a kneeling leg it is easy for a person to carry on occupations and trades such as driving a bullock cart or tilling the soil.

Syme's :—A Syme's amputation gives an end bearing stump and if the amputation is not performed for trench foot or frost bite or peripheral vascular diseases, most occupations can be managed.

Lisfranc's :—Disarticulation at the tarso-metatarsal joint is a good operation; the foot is plantigrade and there are patients who have worked hard barefooted for over twenty-five years.

Upper limb amputations :—Little finger and thumb should be spared wherever possible. Cases of congenital deformities with no digits and metacarpals are able to use their wrist for miscellaneous purposes. A wrist enables supination and pronation to be performed. A long forearm is useful, but special training is required in cases of upper limb amputees to make themselves useful.

III. **General remarks on artificial limbs.**—It must be stressed in the very beginning that though appearances are very important, they count for little if there be comfort, stability, efficient progression and lightness in artificial limbs. *Simple appliances* are very valuable and a thorough training for amputees in their use is important. With a *simple wrist strap* a handless man is able to dress himself and feed himself with a spoon and do most of his daily ordinary acts. A *peg leg* with a broad base for farm labourers and for others who must be on their feet for most part of the day has proved most practical; it is stable under the stress of rough life and can be readily repaired by the local carpenter and blacksmith.

In recent years there has been a great improvement in the manufacture of artificial limbs. They are made lighter and simpler in mechanism. The majority are made of either wood or duraluminium. The wooden buckets are usually made of best seasoned black English willow or of lime. They should not present any flaw or knot which can be seen on the uncovered inner surface. The wood will not split and will be strong when the outer surface is covered with leather.

The average life of both the wooden and the metal types of artificial limb is about 2 years.

Artificial limbs so far designed are for lower limbs and no really useful and satisfactory artificial limb has been designed for the *upper extremity*. (Cineplastic amputations for upper limbs).

IV. **Preparation of a stump to fit an artificial limb.**—For sometime after an amputation, the stump progressively shrinks in size partly due to the subsidence of œdema both venous and lymphatic and partly atrophy of the muscles.

Hence any artificial limb fitted on soon after an amputation will be found to be improperly fitting after some time. It generally takes about 3 months for a stump to shrink to its ultimate size and, hence, one has to wait for this period before fitting a permanent limb. This process of shrinking is known as "consolidation". In the meantime the stump should be kept bandaged either by a simple bandage or by application of elastoplast *from above downwards*. If applied from below upwards there would be a roll of flesh at the upper part of the stump. Meanwhile the patient must be encouraged to use the muscles of that extremity and stump

xercises consisting of movements of flexion, extension, adduction and bduction *against resistance* are given two or three times a day, for twenty inutes each time.

At the end of about three months, measurements for an artificial limb an be taken provided the following conditions, which constitute the requiites of an *ideal stump* are satisfied, *viz.*, the scar should not be tender or dherent; there should be no tenderness or excoriation of the skin of the tump; œdema must have completely disappeared; nerves must not be ainful. It should be possible for the stump to be handled fairly freely vithout any pain. If the patient says it jumps it does not matter. The hantom limb feeling should not be a painful feeling. So much for general rinciples. The principles concerned in making an artificial limb for lower nd upper limbs are briefly considered below.

V. **Artificial limbs for the lower extremity.**—1. *The essential principles* of an artificial limb in the lower extremity are:—

(*a*) The points upon which weight can be taken. These are known as "bearing points".

(*b*) The proper method of fitting the stump with a 'bucket'.

(*c*) Principles of securing stability and methods of "suspending" the limb.

(*d*) Mechanism of knee, ankle and metatarso-phalangeal joints.

A. Three bearing points are possible: (i) at the base; (ii) upon the surface; and (iii) upon the end of the stump.

(i) *Bearing upon the case*, for example, on the tuberosity of the ischium for an amputation through the thigh and on the head of the tibia for an amputation through the leg, is good and is now generally used.

(ii) *Bearing upon the surface* of the stump possesses no advantages.

(iii) *Direct end bearing*, for example, on the remaining sole of an amputation through the foot is quite good and is only used in this situation. To take the pressure upon the end of the stump it is necessary to stretch across the bucket at the right height a piece of material covered with felt. If the bucket is made of leather the support is taken upon a circular band of metal fixed to the lateral steels.

B. The proper method of taking a mould for the bucket from the stump is considered later at the end of the section. The bucket must be made for each patient, hollows being made for each bony point which must be marked out and felt with the fingers.

C. The principles of securing stability and method of suspending the limb will be considered later.

D. *The mechanism of the* (i) *knee*; (ii) *ankle*; *and* (iii) *metartarsophalangeal joints*:

(i) *Knee joints*:—A normal joint is a polycentric joint. It is not possible to reproduce the normal joint. The fixed axis joint is situated at the junction of the anterior three-fourth with the posterior fourth at the level of the lateral and crucial ligaments at the centre of the patella and it has been the level of the artificial knee joint for nearly a century. They are now trying a polycentric joint. With a polycentric type of joint the

amputees are not tired out; no chaffing of skin occurs or no puckuring of the skin on the back of the calf occurs.

The type of knee joint should be simple. An essential to the mechanical construction of the joint is the location of its axis posterior to the centre of gravity of the anatomical joint. If this demand is not complied with, the patient loses all sense of security, because the artificial leg tends to bend at the knee under the patient's weight. If the mechanical joint lies posterior to the normal, then the body weight tends to lock the joint.

(ii) *Ankle joint*:—This, like the knee, should be of the simplest type, allowing merely flexion and extension. It works on a simple hinge with roller bearings, movements being checked in front by rubber buffers. If there is a tendency for the knee to shoot forwards, increase of the thickness of the anterior buffer is helpful.

(iii) *Metatarso-phalangeal joint* allows of a very limited movement of gliding to keep up the spring of the foot.

2. *Disarticulation of the hip*:—In the majority of cases where a disarticulation of the hip has been performed, the patient will not wear an artificial limb.

3. *Amputations near the hip joint*:—A "tilting table" is the name applied to the artificial limb worn for amputations at this level. A stump, to enable a tilting table to be worn, should have the head of the femur in it, as the presence of the head prevents the stump from "sinking in". An amputation below the level of the lesser trochanter is better as the ilio-psoas flexes the stump and the hip will thus be in the sitting position. The retention of the great trochanter and the flexed stump provides points to which the socket of the artificial limb can be fitted closely and the stump is thus prevented from slipping out of the socket.

"*Tilting table*":—This weighs about 8 lbs. It consists of a *socket* in which the pelvis rests, and below this *the leg* is attached by a *simple hinge* capable of being locked or released by a catch easily operable through the clothes. The catch is released *for sitting* and is fixed with the leg in extension for *walking*; in this latter position the hip is rigid and progress made by a swing of the pelvis.

The tuber ischii bears the weight in this limb.

Method of fitting the stump with a bucket:—The success or failure of the artificial limb depends upon an accurate fit at the ischial tuberosity. It is important to realise that the tuberosity *does not* slant from above downwards and forwards but in the reverse direction, *viz.*, from below upward and forwards.

Further the tuberosity of ischium is situated in the posterior part of the perineum, the anterior part of which is unable to stand pressure. It is necessary therefore to clear this part by cutting down the inner border of the bucket in its anterior part, forming a perineal concavity, which rises posteriorly against the ischium.

In applying the steel uprights which support the body or in the case of a wooden limb, in joining the thigh piece with the calf, it is advisable to give the calf about 5° of genu valgum position as this adds markedly to the stability of the artificial limb.

Mechanism of knee, ankle and metatarso-phalangeal joints have been considered already.

4. *For amputations through the thigh*:—

(*a*) *General principles*:—A stump measuring less than five inches from the top of the great trochanter cannot be fitted with a limb giving hip control. A stump should not be longer than four inches above abductor tubercle, as a longer stump interferes with the mechanism of the artificial knee joint and is ill-nourished.

The ideal stump is nine to eleven inches according to the height of the patient, measured from the top of the great trochanter. The ideal flap is a long anterior and short posterior flap, the scar being placed at the back. The scar should be transverse posterior and one and a half inches above the cut end of the bone. Scar must not be in front because there is forward movement of hip and it should not be terminal because on account of piston action it will split. Hamstring muscles should be cut short to prevent them adhering to the scar, for if these are adherent to the scar the necessary action of bracing the stump back in socket when walking is painful. All femur stumps grow spurs. If the spur is situated half an inch from the edge of the femur on the postero-internal aspect an inch long, it has no pathological significance. This spur does not interfere with the artificial limb.

(*b*) *Wooden artificial limb of the American model*:—In the wooden artificial limb of the American model, a sling which passes over the shoulders is attached to the leg below the knee in such a way as to act as a mechanism for extending the knee; the leg portion is manufactured out of a single piece of wood; the tendo-achilis action for the ankle is abolished and is replaced by one of ankle with movement limited by India-rubber buffers. The knee mechanism should lock in any degree of flexion when a strain is put upon it so that the wearer does not necessarily fall when his weight comes upon the limb when the knee is flexed. The ischium should not slip inside the bucket, otherwise the inner border will come in contact with the perineum. Therefore the diameter of the bucket must be less than that of the limb, so that the ischium may rest upon its inner edge. If the bucket is too large, the patient abducts the stump, so as to lower the inner border and prevent pressure on the perineum; he carries the leg away from the side as he walks and this is both unsightly and fatiguing.

If the bucket is narrow there is a tendency for the limb to rotate inwards.

The front of the upper border of the bucket slopes downwards and inwards at an angle of about 45° when as a result of its weight the bucket turns inwards as the limb is swung, the base of the stump will come against a higher part of the bucket; but when the pressure of the weight of the body returns, the stump, being forced into the bucket, will descend again along this slope, that is to say, a passive external rotation of the artificial limb will be brought about correcting at every step the tendency to internal rotation. If the top of the bucket is narrower than the circumference of the top of the limb, measured below the ischium, it may be circular and still give support to the ischium which will not slip into it. If the ischium does slip into the bucket, the result will be that it no longer serves as

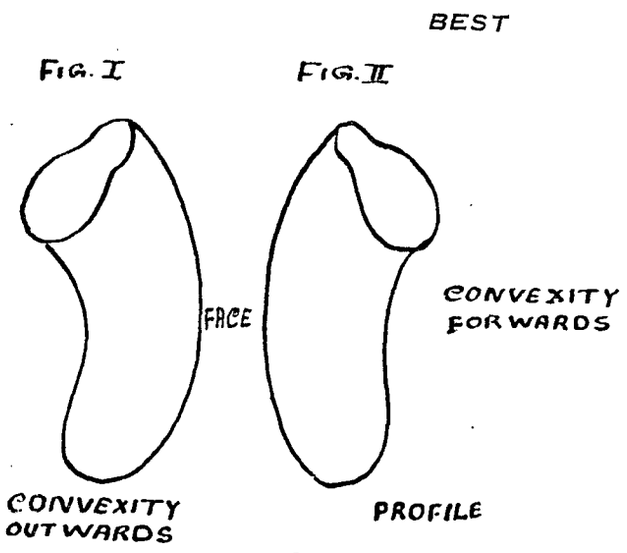
the support, the pressure coming instead upon the ischiopubic ramus and upon the perineum which are unable to stand the pressure of an artificial limb.

The constriction thus exerted upon the top of the stump may easily become insupportable. The correct solution of the problems is to cut down the upper border of the bukcet opposite the perineum, letting it rise again posteriorly beneath the tuberosity of the ischium, and gain a good support there.

Further from *the front it is convex outwards*; from the side convex forwards. This is the form that is generally used.

The *curve outwards* by drawing away the soft parts from it, frees the region of the ischium and allows the tuberosity of the ischium to press upon the bucket.

If the thigh *piece is curved forwards* and particularly if the limb is built with a very slight flexion of the knee, the stump remains flexed at the hip and the patient feels as if he is sitting in the apparatus. But if the thigh piece is straight, an uncomfortable pressure is produced by the edge of the bucket against the ischium. If the thigh piece is straight, the short stump cannot follow the movement of extension necessary in walking.



Mode of suspension:—Suspension of thigh piece is essential and is all the more important when the stump is short and consequently more liable to slip out of the bucket. For this purpose support may be taken either from the waist, upon the prominence of the iliac crests or from the shoulders by means of braces.

In the case of a long stump (amputation below the middle of the thigh) braces are quite good.

Suspension by means of braces:—Leaves the pelvis free; patient does not feel the pull of the hip piece; if the belt is used, when the patient sits down, the buttock on the side of the stump is raised to an extent corresponding to the thickness of the bucket, an obliquity of the pelvis which is both uncomfortable and unsightly. The braces being relaxed in the sitting posture, the patient can avoid this inconvenience; for the stump may be slipped partly out of its bucket, the upper extremity of which is

then beyond the level of the edge of the chair. The position is very comfortable, because it is normal but the patient must replace his stump in the bucket whenever he stands up.

This form of suspension is essential for those artificial limbs with a free knee joint in which the braces serve to extend the joint.

If the stump is short:—Combined method of suspension by braces and by belt is required. Braces should be 5 to 6 cm. wide.

Belt consists of a pelvic plate which is attached to the hip steel, below the iliac crest. A belt attached to the extremities of this plate surrounds the pelvis and passes above the iliac crest on the other side. The thigh piece is attached to this support, on the other side by articulation of the outer femoral steel with the hip steel and on the inner side by a perineal strap. The axis of the metal joint between the outer femoral steel and the lower end of T piece should be directly above the great trochanter.

The femoral steel often breaks in the neighbourhood of this joint; this difficulty is got over by adding immediately beneath it a joint which allows abduction. A perineal strap limits this movement.

(c) *The above knee type of modern limb—DeSoutter type*:—Thighs are pressed in one piece from thin sheet metal, the sockets are of metal enamel finished and perforated to allow ventilation, knee joint mechanisms are built internally to give a smooth appearance, and are designed to control the swinging of the limb and produce a natural action.

The socket is so shaped that the greater part of the patient's weight is supported on the tuberosity of the ischium. The limb is supported by a special form of pelvic band with no chest or shoulder harness which allows free movement of the stump in all directions.

(*d*) *The use of pylons*:—Pylons are used in the above knee amputations. They are temporary supports made of plaster of Paris to which are attached wooden ends similar to the ends of crutches. A patient should not wear a pylon till up to three months after the amputation and the stump is in a condition to permit the fitting of a permanent limb. The pylon was used to allow the limb to shrink so that he can be saved a socket or two. The pylon is not advocated now. As there is no knee joint the patient is forced to swing the pylon out to the side at every step. It is impossible to cure this habit when a pucca limb is fitted.

A pylon tends to develop a roll of flesh at the top of the stump. This gives trouble to the limb maker.

(*e*) *Walking on a peg leg*:—The peg leg is a rigid rod ending in slight enlargement, which transmits the weight of the body, resting by means of the isonium upon the top of the bucket, directly to the ground. Erect position is secured and stability in walking is very good. To raise the limb from the ground and carry it forwards, the patient uses at the same time both flexion of the stump at the hip and movements of pelvis, elevation and then rotation inwards, varying to some extent, with his proficiency and length of stump.

Rigid peg leg has two defects:—There is no appearance of leg and foot; further when the patient is sitting the rigid peg is unsightly and inconvenient to him and his neighbours.

5. *Disarticulation through the knee joint* :—Disarticulation through knee joint is indicated in one group of cases, *e g.*, when the patient has been accustomed for long to bear his entire weight upon the anterior surface of a fully flexed knee, *e.g.*, in congenital deficiency of the entire tibia.

Though it is discarded operation now, there is another indication for this operation *i.e.* when a life may be saved in out of the way and ill-equipped dispensaries. The stump does not suffer from any circulatory changes because the anastomoses is left behind. A neat knee joint cannot be fitted. They can walk on a kneeling leg.

6. *Below knee amputation* :—With a well fitting artificial limb a man with a satisfactory below knee amputation may expect to be able to walk five to ten miles in comfort.

(*a*) *Bearing points* :—The socket takes weight on the heads of tibia and fibula and the lower edge of the patella. This has not been found quite successful as it is found that the limb becomes intolerant and requires re-amputation at the end of about fifteen years. This is especially so in growing children in whom there is in addition the danger of hyperextension deformity at the tibial epiphysis. To avoid this, the recent limbs have a high fitting of leather so that some of the weight may be borne on the tuber-ischii.

(*b*) The bucket limb is now universally adopted in place of the old knee rest when the knee was permanently flexed.

The bucket limb requires an extended stump, the end of the stump should not touch the bottom of the bucket.

(*c*) The Farabeuf amputation at the seat of election is not designed for a bucket, but to be flexed on a knee rest ; it is short and will kick over the bucket in front on extension of the knee.

(*d*) *Amputees below the knee for thrombo-angitis obliterans can* only wear a kneeling limb.

(*e*) Kneeling leg was popular in France until the World War of 1914–18. The peg leg was common in Germany following the Franco-Russian War. The so-called "American leg" became popular in 1914.

A kneeling leg on which the patient knelt was of great use for a long time. This is still the only method of obtaining a good natural method of weight bearing end to a stump. For this stump three inches of tibia should be left.

(*f*) *Modern methods of amputation through the leg* :—A tibial length of seven inches was regarded as ideal since the war of 1914. It was discovered that as the patient grows older, the stump became ill-nourished and that a tibial length of five and a half inches is better. The head of the fibula should always be retained ; the socket can be fitted closely to it, and its presence helps to retain the stump therein.

Even with short stumps fibula should not be removed completely. This alters the shape of the stump from triangular to circular. The triangular shape is extremely useful in helping to prevent an artificial limb from rotating. Also the scar left after the excision does not bear pressure.

The scar must not be on the anterior aspect ; an antero-posterior scar n gets drawn up between the cut ends of tibia and fibula ; a transverse

terminal scar as much posterior as possible with a slightly longer anterior flap is good.

If there is post-operative anæsthesia, delay the artificial limb till it is gone.

For below knee amputations in which the stump is very hard and healthy a prosthesis consisting of a socket, a knee hinge, and thigh corset is given. The socket takes the weight on the heads of tibia and fibula and the lower edge of the patella. This has not been found possible and it is better to take some of the weight on the pelvis by fitting a limb with a bearing on the tuberischii, especially in growing children, to avoid the danger of hyperextension deformity at the tibial epiphysis.

The ankle works on a simple hinge with roller bearings, movement being checked in front and behind by rubber buffers. If there is a tendency for the knee to shoot forward, increasing the thickness of the anterior buffer is of help.

Sockets are made of duralnmin or wood.

(*g*) *Modern artificial limb* :—*The below knee type.* The skin is pressed in one piece from a thin sheet metal, and supports a leather socket which is moulded from a plaster cast of the stump. The ankle mechanism is selfcontained and the working clearance between foot and skin is imperceptible. Ball bearing side steel joints attached to a soft thigh corset give support and freedom from lateral strain to the knee joint.

7. *Syme's amputation* : (*a*) *Original Syme's operation*—The heel flap was too voluminous ; an artificial ankle joint could not be fitted in.

The weight bearing was on the pad which acts as a sponge ball ; it wobbles about and it gets displaced and weight is taken on cut ends of tibia and fibula. There is traction on the anterior horizontal scar. The piston action splits the scar ; there are callosities immediately beneath the scar ; there are soft callosities on the under aspect of the pad. In old age and in long standing cases after 8 years in cases of amputation for trench feet or frost-bite, there are circulatory disturbances. Bone atrophy of tibia and fibula is seen with sometimes periostitis of anterior border of tibia and fibula. In a case of Syme's amputation done 40 years ago the leg is wasted and thin, the heel stump is loose ; but the man is able to carry on the work of an ice man at the hospital. Original Syme's amputation is still a valuable operation in this country and poor people can carry on their work in the roads and agricultural fields with a leather support.

With a Syme's circular boot (diagram) the patient can do his work satisfactorily.

(*b*) *Modified Syme's operation* :—It is intended that the stump should be end bearing. It leaves enough room for a mechanical ankle joint. There is less loose flesh and therefore less likelihood of the end bearing pad loosening. Even if no artificial support is worn, the patient can get on well.

The terminal increase in the stump is still of value in holding the artificial limb in place.

8. *Lisfranc's disarticulation* is a very valuable operation in this country and patients have been doing very good work for over thirty years.

VI. **Artificial hand and arm.**—1. The artificial hand should be detachable so that it may be replaced by other appliances. Arms are of two

types, *viz.*: (*a*) Mechanical purposes (Worker's French type); and (*b*) show arms.

No really useful and satisfactory artificial limb has been designed in the upper extremity.

2. *Forequarter amputation*:—No appliance is possible. A limb maker supplies a light protective covering which will square the shoulder and support the coat.

3. *For above elbow amputations*:—For above elbow amputations, shoulder harness is used. The elbow is flexed by moving the shoulders forward and so pulling on a cord. There is also a mechnanism which enables the patient to lock the elbow at any angle and release the catch at will. This is operated by the patient forcing the stump down into the socket and so pulling on a second cord attached to the shoulder harness.

In above elbow amputations, the forearm prosthesis is bisected; this arrangement allows of pronation and supination of the hand and is placed as near the elbow as possible so that the strain on the elbow flexion cord is at a minimum. Instruments are snapped into this short forearm prosthesis. The dress arm is of normal length.

Instead of a single cord looped round the opposite shoulder and used to open the spring thumb at least 3 such cords are used, actuated by: (1) rounding the back; (2) by expanding the upper part of the chest; (3) by raising or lowering the shoulder on the side of the amputation. These may be used for (1) flexing the artificial elbow, (2) working the elbow lock and (3) actuating the thumb, fingers or appliances used by the hand.

The hinge joint at the elbow with an upper arm cuff should not be employed since it gives no opportunity for pronation and supination. A better method of attachment is the figure of 8 strap, which passes just above the condyles of the humerus and crossing the posterior surface of the humerus, descends again over the anterior surface.

4. *Forearm amputations—Keller-artificial hand*:—For amputations of the forearm, Keller, himself an amputee, devised this for the use of the farmers. It consists of a leather socket cuff reinforced by two longitudinal steel bars, held in place by a figure of 8 strap which passes just above the elbow. The handpiece made of wood, may be removed from a socket, if desired. Inserted into the wooden handpiece are three strong steel hooks. Small objects can be inserted between them; they furnish the leverage for larger instruments. To hold the larger instruments firmly in place, a leather strap attached to the anterior portion of the apparatus is made to take a double turn about the handle of the article used and then passing backward between the hooks, is fixed to the posterior aspect by means of a steel pin.

Those possessing an elbow joint have a great advantage over those amputated above the elbow. Amputations of the forearm and of the upper arm if not more than two or three inches above the elbow do not debar a man from becoming a carpenter or farmer or some type of mechanic. An amputation near the shoulder joint would debar a man from doing his previous occupation.

For many men the stump itself is the best prosthesis. This is so in a particularly long forearm stump. This can be used for filing, almost as

effectively as the normal hand; for hammering, the handle is gripped in the elbow between the upper arm and the stump. At the turning lathe, the stump can easily be trained to turn the adjusting swivel.

For those who have lost both hands, if the stumps are long enough to allow them to be approximated, they should be encouraged to train themselves very assiduously to use the stumps together. They will regain a good deal of function and they have to be trained for non-manual labour such as teachers.

Where no mechanical hand is to be fitted, forearm amputations are provided with a hinge at the elbow and a simple corset round the arm. The patient is supplied with a "dress" hand having a thumb working on a spring, with which, a book or paper can be held. All appliances are made to be snapped in place of the hand.

A C hook is very useful as it can be used for pulling, pushing, or torsion. Where the arm is weak it is advisable to fit an adjustable lock at the elbow to enable the joint to be fixed at any angle desired and to avoid strain.

5. *Fingers and hand*:—Artificial hands operated by the hand have proved unsatisfactory. They are used occasionally and are moved by the action of appendages and cords attached to the shoulders.

The possession of one perfect digit is preferable to the best artificial arm, as a prosthesis can be made to which this digit is opposable.

VII. **Method of taking a mould of the stump for the bucket.**—A very accurate mould of the stump in plaster of Paris should be obtained.

The following materials are required to obtain a plaster mould:—

(*a*) Vaseline and vaseline bandages.
(*b*) Plaster bandages two to four inches wide.
(*c*) A strip of lead about one inch wide.
(*d*) A sharp knife, scissors and a tape measure.

The stump is rubbed over with vaseline to prevent hairs being caught in the plaster and to facilitate removal of the plaster mould. Place the strip of lead down the front of the stump and bandage the stump with plaster bandage quickly and evenly, keeping the lead in position. The surface of the bandage is smoothed out and another bandage is applied. The bony landmarks are carefully moulded over the plaster as it is still pliable. Lines are marked across the front of the casing with an indelible pencil in three places to enable the mould to be correctly joined again. Lateral, medial, front and back sides are correctly marked. The plaster is cut over the centre of the lead piece to within about two inches of the end of the stump before the plaster gets hard. The mould is removed only after it has hardened.

The mould is removed without disturbing the impression and it is pressed together to appose the pencil marks and to prevent any disturbance of the position; a plaster bandage is applied over the mould till it is firmly joined.

Fitting the mould.—The following materials are required: (1) liquid soap made by dissolving two teaspoons of soft soap to ten ozs. of boiling water; (2) plaster of Paris made into consistency of liquid cream; (3) $\frac{1}{4}$ inch iron square rod 6 to 8 inches in length varying according to the

length of the stump or a piece of one inch square wood; and (4) a wire hook, knife, brush, spoon, basin.

Method of filling the mould.—Some liquid soap is poured into the mould and turned all around with a brush till it is well coated with the soap. Liquid plaster is poured in and out of the mould till there is a uniform coating of the plaster. Than the plaster is made into a consistence of cream and this is poured into the mould upto the top. Before it hardens, a hook is inserted at the edge and an iron rod in the centre which protrudes about three to four inches outside. The use of the iron rod is to fix into the vice to hold the cast steady.

When the plaster is quite hard, the line of the original split is cut down with a sharp knife and the cast is removed. The cast is smoothened out and when it is quite dry, a thin even coating of wax is applied by dipping the cast into a pan containing melted paraffin wax which is applied to prevent the cast from sticking to the bucket.

A cast of the sound limb is required by the instrument maker so as to make the artificial limb look similar to the sound one and is taken in the same manner as is described above.

Measurements are to be supplied to the makers of artificial limb in addition to the casts on the charts supplied by them and no opportunity should be lost to make oneself familiar with the correct methods of taking the measurements.

Medical Relief in Famine Areas*

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DARKENING clouds of famine that were threatening in the azure skies to inundate this subcontinent as well as China and the European countries overrun by Germany are slowly receding away from the Indian horizon, as ships loaded with cereals and other food articles sail from all parts of the world towards the Indian ports, as stringent governmental measures are undertaken unflinchingly and with forethought in respect of food production, procurement and equitable distribution among the poor, half-naked, semistarving rural population, and as schemes to quell corruption and black marketing are carried out relentlessly. The only silver lining in the otherwise dark clouds is the fact that, if the famine were to occur this time, it will be certainly not *man-made* at all. There has been a bit of easing of the tight Indian food situation quite recently. While it is for the Governments and the public health staff to do the spade work in attempting to prevent the advent of famine, in trying their best to ameliorate the conditions if it were to occur unfortunately, as it may possibly in Madras, and in organising relief measures in the famine areas, the medical profession also has its own onerous and responsible duties by the nation of treating the victims of such mishap should the Governmental measures fail and fall short of the requirements. What then is their role and function? How then could they best serve the nation? The bitter lessons learnt from the sad experience of the woeful tragedy of Bengal Famine of

*Specially contributed to THE ANTISEPTIC.

1943 during which about 2 million people died of starvation and epidemics, the post-mortem notes published by Woodhead Famine Enquiry Commission, and the Report of the Committee of Enquiry under the ægis of Indian Research Fund Association—all these should serve as an eye opener and should be made full use of both by the Governments and the medical profession during this impending crisis in formulating their relief measures and policy.

World food position and organisation.—The present food shortage exists not only in India but all the world over and this would make relief schemes in India more difficult. There was a big gap between the available world cereal supplies (of rice and even wheat) and the minimum requirements of the people. This is being slowly bridged up and mass starvation in many countries is being prevented by systematic planning of supplies, etc., though the situation in India is still critical. The world crisis will not be solved by the harvest of 1946; the emergency will continue but with improvement throughout the first half of 1947. The burden of relief has fallen on the shoulders of U. S. A. Canada, S. E. Asia, Australia, Brazil and Argentina. This work of allocating and diverting the world's marketable food supplies to the deficiency areas with reference to the emergency present is now being done by UNRRA and the International Emergency Food Council. It is but proper and essential, however, that a *World Food Board* to organise a world pool of food reserve should be established early.

India's requirements.—It should be borne in mind that India covers an area of 18,08,679 sq. miles, that there are 7 lakhs of villages and that there is a population of 400 millions which is increasing at the rate of 10 millions every year. While India's food requirements are 65 million tons the annual productions are only 51. This deficit which used to be made up even in normal times only by imports from abroad became unavailable totally during the war years and partly during the later years. There has been a further shortage of 7 million tons due to failure of crops in 1945. By means of rationing so that each adult got 12 oz. a day the total deficit for 1946 was narrowed to 6 million tons ($1\frac{1}{2}$ millions rice). So at least $\frac{1}{2}$ million ton of cereals must be imported from abroad every month till the next harvest if India is to stave off widespread famine conditions. As against this demand India has been allotted for 1946 only $1\frac{1}{2}$ million tons of which $\frac{1}{2}$ million will be rice. It has not been possible to import all this even. And the total All-India outturn of crops at the end of this year 1946 also will be only upto average as the rice crops in Assam and the maize crops in Bihar have suffered appreciable damage by floods and as the rice crops of the Punjab have failed to a large extent because of drought. Thus India will not be out of the woods for another 6 months and the shadow of famine will continue to lurk on her horizon.

Requirements of and food position in Madras.—Madras Province is suffering the largest deficit in food. There is a population of 50 millions and the monthly consumption is more than 2 lakhs of tons of cereals, major part of which is *rice*, which is the staple food of the people. A buffer stock for 6 weeks namely 3 lakhs of tons is to be provided if the province-wide rationing system is not to break down. The total deficit in Madras during the current second half-year is estimated to be 5 lakhs tons, after taking into account the six lakhs of tons which will become

available by internal procurement before the end of December. To meet this deficiency the Central Government has allotted 2½ lakhs of tons by way of imports from foreign countries. To create the buffer stock, to conserve the existing stock of rice up to the end of January 1947 when the main harvest would be expected to become available in the market, and to anticipate and provide against any further food shortage during the coming lean months the Provincial Government has been compelled to cut the ration in February 1946 to 12 oz., in May 1946 to 10 oz. and in August to 8 ounces of rice though the remaining 4 oz. is given as wheat, wheat products and millets. With all this, there will be an over all deficit of 3 lakhs tons of food grains up to January. Thus the food position in Madras is critical. The latest reports are neither encouraging nor heartening. Although there is no cause for alarm and despair there is no ground for complacency or for loosening our efforts. After all there may be no widespread famine as such, but famine conditions in scattered areas and ill health, under nutrition and semi starvation are bound to occur, unless the efforts of both the Central and Provincial Governments to get over the crisis bear fruit within these three critical months, and unless the citizens are ready in a public-minded spirit of self-sacrifice to co-operate and share what they have with others.

Causes.—During the second World War the imports from abroad especially South-East Asia declined very greatly; this has not improved much even after the cessation of hostilities due to various causes. The International Food Council has made allocations of food grains which fall much short of India's demands from abroad; and the import of even these inadequate allotments has not been possible due to the political situation in Burma, Siam and Java as well as the physical and transport difficulties such as the American Maritime strike; (2) Food production is not keeping pace with the growing demand for food stuffs and with the increase in the population which is growing at an alarming rate of 10 millions every year; (3) The failure of the N. E. monsoon last year, the great drought in the early months of 1946 and the Cyclone in the Northern districts of Madras coupled with the insufficient irrigational water-supply in the Cauvery Delta have caused a major failure of both crops; (4) The recent strikes, communal strife as well as the floods in various parts have affected the food supplies. Thus a variety of these adverse natural causes has led to an acute and grave food shortage in India, which is faced with a drastic famine.

And this food shortage is bound to have its repercussions on the already semi-starved, teeming millions of India and have disastrous consequences on their impoverished health which is on the verge of breaking. Already majority of the people are poor and even in normal times do not get enough to eat, while a large portion of the rest eat only an unbalanced diet made up of carbohydrates chiefly rice or millets with little or no supplementary nutritive foods like milk, ghee, dhalls, eggs and meat which they cannot afford to buy. This cereal-food could only satisfy their appetite and just keep the body and soul together. Thus the average villager has been on the bare margin of subsistence, has been under and malnourished for a number of years and has lived and toiled without sufficient nutritive food factors in his diet. And now his cereal ration on which alone he depends for his food has been cut down to 12 oz. which is a starvation allowance as it amounts to less than 1,200 calories. Another

important cause that will hasten the advent of famine conditions in South India is that the people are not taking to wheat or maize which are available, as they are accustomed to eat only rice; they are prepared to forego their 4 oz. cereal ration and subsist only on the 8 oz. of rice. This quantity is utterly inadequate to sustain any person in normal health; if this is to be continued for a few months more or if it is further cut down due to failure of supplies from abroad the nutritional state of majority of the people is bound to collapse ending in subacute starvation, ill-health, epidemics and high mortality. Thus the lurking spectre of Famine would be allowed to prey on these semi starving millions; and we would have to face a very grave food situation and a real threat of famine. It is essential to bear in mind these causes for food shortage and famine conditions while planning the famine relief measures.

Relief measures.—The problem of famine is purely a question of economics (food supplies, etc.), public health (nutrition, etc.) and medical welfare and thus rests as a tripod on these 3 legs; as such it has to be dealt with under three subheads. (1) Relief measures by the Government; (2) By the Public Health department; and (3) By the Medical profession. This problem as of any other in India is complicated by the twin evils of ignorance and poverty and because 89% of the people live in the scattered 7 lakhs of villages, the relief measures must be concentrated in the rural areas, where sanitation is poorest and epidemics are likely to spread like wild fire.

I. By the Government.—As the catastrophe of famine does not come suddenly like a bolt from the blue, it is possible to avert it by prudence, precision and proper planning. The Government must undertake to carry on well-conceived and bold measures in time, with a resolute heart for controlling and promoting food production, procurement, movement and supplies; they must ensure proper and equitable distribution of the available supplies by a system of efficient and rigid rationing. They should prevent speculation, black-marketing and hoarding of food grains by the producers as well as the rich and also should check corruption with an iron hand. They should control and stabilise the prices of essential commodities and should see that there is a free flow of supplies of even unrationed articles to the consumers at fair price and at controlled rates through licensed dealers or co-operative societies with the help of Village Committees of non-officials. The government must take measures to prevent those living in plenty from wasting; must endeavour to infuse public confidence, mobilise public opinion and enlist public sympathy, cooperation and support, which are quite essential for the efficient working of any of their measures and also must undertake *to increase the terribly low purchasing power and standard of living of the masses.*

1. *Food procurement.*—The problem of finding food for all the citizens is the first charge on any Government, and it is obliged to spend the last rupee in saving human life from starvation. Food must be purchased wherever available and whatever the cost and given to the people at fair prices and in famine areas to the destitutes even free of cost. The food procurement must be from internal as well as external sources. The entire produce of the food grains and all the quantity available locally must be bought under government procurement and monopoly systems, *i.e.*, the scheme of state trading in food stuffs must be worked out. The procurement schemes must be tightened both in the surplus and deficit areas to

mobilise all available supplies. The government by offering bonus for immediate deliveries would bring more food into the open market from under ground. Efforts must be redoubled to get rice from the other maize and wheat eating provinces of the north in exchange for the wheat and millets allotted to Madras. The Ministry should tackle the *transport problem* and establish a system of planned movement of supplies from surplus to deficit areas by requisitioning all vehicles or by even nationalising the Transport Industry. Last but not the least every effort to secure additional supplies immediately from overseas must be made, though it would be a folly to depend on them entirely.

2. *Food control.*—The available food must be arranged to be distributed evenly and equitably by an efficient rationing system, which must be extended to the surplus areas as well.

3. *Food production* must be intensified; but this may not serve any useful purpose for the immediate needs to meet or avert the impending crisis. However it should be undertaken as a long term policy. Of the 420 million acres of land cultivable in India, 150 lie fallow and food grains are grown in 210 million acres. 80% of total acreage depends upon timely rains for their cultivation. Though the resources in India are immense the yields at present are low because of bad soil, bad seed, bad cattle, bad implements, bad methods of cultivation and bad economics and marketing. India can and must become self-sufficient and self-supporting as far as the staple foods are concerned but that can be achieved only by modernising her agriculture, by well-planned economic development and industrial organisation, by improvement of irrigational facilities, by improvement of fertilizers, by measures designed for the protection of crops from pests and diseases, by coordination of Agricultural and Nutritional studies and research and last but not the least by reclamation. By all these more fallow lands could be made cultivable. The people must be taught to shake off and abandon their primitive methods and be persuaded to adopt modern methods of agriculture.

However as a short term policy the *grow more food compaign* must be made intense and practical. During the crisis the government must grant liberally concessions in the form of land revenue remissions, bonuses for deliveries of food grains, subsidies to sink wells and gratuitous reliefs in the shape of cash to the cultivators; they should arrange for free supply of manure and open fodder depots to relieve fodder scarcity. All these measures are bound to increase food production, though they may not break the edge of the scarcity. The government must further subsidise and promote the cultivation of *short term and substitute food crops*, such as sweet potatoes and tapioca, vegetables, roots, tubers and fruits that could be grown in a short time must be raised in the areas where paddy cannot be grown; edible cannas must be cultivated on a wide scale in the gardens or even the backyards of houses. Thus in this present acute shortage of food every possible source of supply of substitutes must be diligently explored. The Government must take steps to popularise these substitute diets by educating the public how to cook the unfamiliar food stuffs such as maize, ragi, sweet potatoes, tapioca, etc., into good and wholesome delicacies by distributing printed leaflets, by arranging for mobile kitchens and propaganda vans and by actual demonstration.

4. *Other relief measures:*—The exodus and influx of starving sick destitutes and other semifed people in quest of work and food from villages

to towns and cities is the crux of the problem. As this movement is bound to spread panic as well as epidemics, it must be controlled and even checked. So the government have to set up *camps* at selected points where such destitutes deserving free supply of food could be collected, fed, clothed at State cost and protected against infections and communicable diseases. It may become necessary to distribute grains to the decrepit and infirm in the affected areas. Local charity must be mobilised to start free kitchens. Government must also give subsidies for milk for children; powdered or condensed milk must be purchased and distributed to infants, expectant and nursing mothers. In addition the government must purchase lakhs of tons of protective foods to supplement their diet. These schemes must be made applicable to rural areas where it must be worked by village organisations and Panchayat Committees.

II. **By the Public Health Department.**— Public Health measures should be taken at once to check the breaking out of epidemics which are the inevitable sequelæ of famine conditions and combat them if they break out due to food scarcity and exodus of sick destitutes. Arrangements should be made to secure ample stocks of mepacrine, anti-cholera vaccine, influenza vaccine, smallpox lymph D.D.T., tropical chloride of lime, bleaching powder, multivitamin tablets and milk powders and keep them at each Heâdquarter station as Epedemic Reserve. The Health authorities must train lay persons and other volunteers to carry out distribution of mepacrine and other drugs, to chlorinate wells and disinfect water supplies, to use D.D.T. in the control of malaria, to do preventive inoculations and to carry on propaganda work from village to village in favour of substitute diets. Voluntary associations who come out to do relief work must be assisted and encouraged. The Health department must tackle the *problem of nutrition*, publish balanced food recipes that could be made from the substitute food articles and carry on extensive propaganda by pamphlets, lectures, films, etc., regarding their use. The improvement of nutrition must form an essential part of Public Health programme at least as a long term policy and scheme.

Nutrition.—The problem of famine relief is essentially one of nutrition and in India the latter is complicated by poverty, ignorance, superstition and social customs. The health of a nation depends on the judicious choice of its food and in that lies our fault and weakness. As has already been pointed out the diet of the vast majority of Indians is extremely ill-balanced and insufficient, the deficiency being both quantitative and qualitative of high class proteins, vitamins and minerals; it is quite inadequate to maintain them in normal health. Thus millions of them suffer from subclinical deficiency states that are chronic and latent manifestations of defective nutrition; they are in a border line state of nutrition manifesting none of the well-known signs and symptoms of deficiency diseases as described in text books. Many suffer from hypovitaminosis manifested by xerodermia, bleeding gums, night blindness or from nutritional œdema, low standard of general health impaired resistance to infection and chronic illness attributable to defective diet and nutritional errors. A few of course suffer from frank clear-cut avitaminosis as manifested in beri beri, rickets, scurvy, xerophthalmia, pellagra and from marasmus. The deficiency states are either—

A. "*Primary*" in which the chronic and latent condition of subnormal nutrition is accentuated and made manifest by intensification of the

deficiency, the cause of which may be (1) due to lack of protective food factors such as proteins, vitamins or minerals in the diet, (2) due to decreased energy intake in the food, (3) due to faulty absorption of the proximate principles in chronic gastro intestinal diseases, (4) due to defective assimilation and utilization as a result of illness and (5) due to increased demand as in pregnancy, lactation, growth, infancy and convalescence *or B. Conditioned* in which intercurrent diseases like diabetes, chronic dysentery, malaria, leprosy, fevers, etc., would disturb the previously just-balanced nutritional state and make the deficiency apparent. These factors are bound to operate when famine conditions set in as a result of insufficient rations and cut asunder the delicate and flimsy thread by which the poor health of the average villager hangs. Thus we are confronted with this medical problem of the highest magnitude, the *prevention and treatment* of *nutritional deficiencies*. With this end in view a Nutrition Commission must undertake a comprehensive survey of the actual state of nutrition of the people, an analysis of the common diet articles consumed by the general public and investigate into their composition, caloric and nutritional values; it must make also a scientific study of the food requirements in terms of quantity as well as quality from different points of view so as to suit the energy needs of the active men, the labourers, the aged, the women, the expectant and noursing mothers and the infants and recommend well-planned diets suitable for each category. The duty of discovering suitable cheap methods of improving the diet nutritionally also will devolve on this Commission.

Food.—It is necessary to go into details about *the composition of food, its sources, its nutritive value and the daily standard requirements of man.* It is vast study in itself and so I shall indicate only the outlines.

The essential food elements are:

(1) *Proteins* which are indispensable for tissue growth and repair. (Caloric value is 4·1 per gram.

(2) *Fats* which are reserve sources of heat (caloric value is 4·1 per gram.)

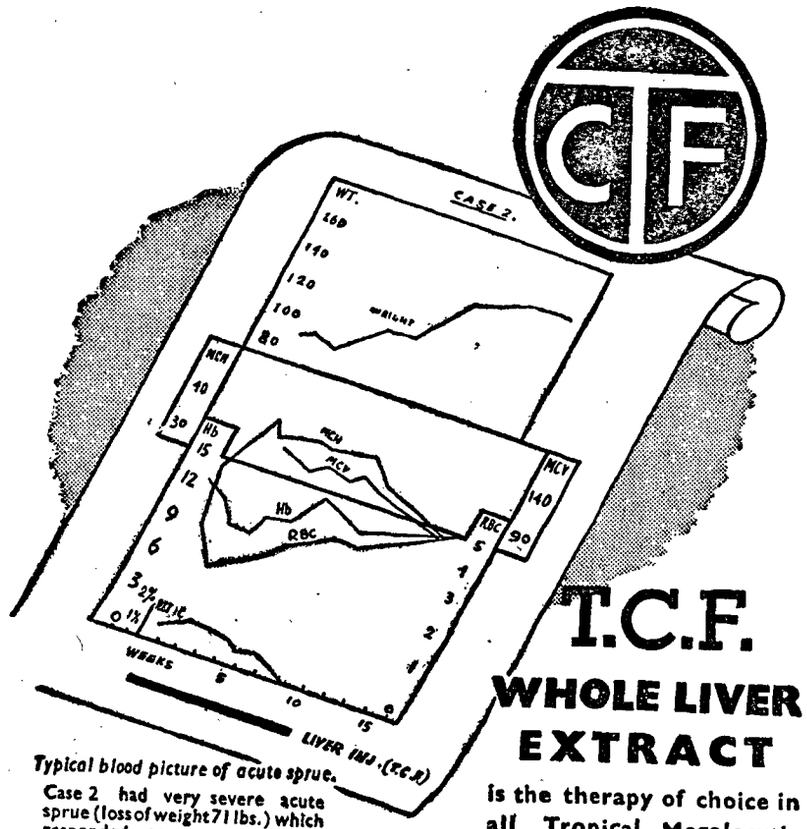
(3) *Carbohydrates* which are the cheapest sources of heat and enargy (caloric value is 9·3.)

(4) *Minerals* such as Na, K, Ca, Fe, I, P, etc.

(5) *Vitamins* which are necessary accessory food factors that maintain health and are essential for the normal growth and physiological functions of all living cells of the tissues. A food value chart (*vide pages 83 and 84*) is reproduced here from "Food" by ROBERT MCCARRISON for the benefit of the readers.

From the following 2 tabular statements it will be evident that the common sources of proteins are milk, eggs, fish and meat and their vegetable sources are dhall, nuts and seeds; and the sources of carbohydrates are rice, millets, wheat, sugars, tubers (potatoes, tapioca), banana and mangoes. The fats could be derived from vegetable or animal sources and the latter contain vitamin A and D in addition. The minerals (especially calcium are supplied from milk, vegetables and fruits. The chief sources of :—

(1) *Vitamin A* are Cod liver oil, Halibut liver oil, Shark liver oil, eggs, butter, liver, colored vegetables such as carrot and tomatoes and leafy vegetables like spinach, amaranth and lettuce.



Typical blood picture of acute sprue.

Case 2 had very severe acute sprue (loss of weight 71 lbs.) which responded to parenteral liver therapy (T.C.F.).

Reproduced from "A Study of the Onset and Cyclic Development of the Sprue Syndrome" by K. D. Keele, M. D., British Medical Journal, July 27, 1946.

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Composition of some common food materials used in India.

No.	Food stuffs.	Proteins in gms. per ounce.	Fats. gms./oz.	Carbo-hydrates gms./oz.	Calories %	Fe. mgm. %	Calories per oz.
	CEREALS.						
1	Home pounded rice ...	2.3	0·85	22·3	0·01	2·8	99
2	Milled rice ...	1·79	0·13	26·1	0·01	1·0	113
3	Wheat flour (Atta) ...	3·9	0·54	20·35	0·04	7·3	100
4	Ragi ...	2·78	0·46	23·35	0·33	5·4	109
5	Cumbu or Bajri ...	3·4	1·3	18·6	0·05	8·8	102
6	Maize ...	3·6	1·23	19·9	0·01	2·1	97
	SUGARS AND STARCHES.						
7	White Sugar ...	Nil	Nil	28·3	—	—	113
8	Sago ...	2·2	0·04	22·0	...	...	97
	TUBERS.						
9	Tapioca ...	0·05	0·04	11·2	0·05	0·9	45
10	Sweet potatoes ...	0·06	0·06	9·1	0·02	0·8	47
11	Potatoes ...	0·7	0·04	8·15	...	...	36
12	Dhall ...	6·5	0·99	16·2	...	...	100
	NUTS AND SEEDS.						
13	Ground nut ...	7·3	10·9	6·9	0·05	1·6	155
14	Cashew nut ...	5·4	13·35	7·43	...	...	172
15	Almonds ...	5·26	15·96	4·3	...	...	182
	MILK PRODUCTS.						
16	Milk (cow's) ...	0·94	1·02	1·36	600 mgm. per pint.	...	18
17	Buttermilk ...	0·85	0·14	1·36	...	...	10
18	Curds ...	1·4	1·00	0·80	...	...	18
19	Butter and ghee ...	Nil	23·1	Nil	—	—	208
20	Vegetable oils ...	Nil	28·00	Nil	...	...	252
	FLESH, EGGS, ETC.						
21	Lean mutton ...	5·97	1·98	Nil	...	...	42
22	Fish ...	5·15	0·2	Nil	...	...	22
23	Eggs ...	3·79	2·97	Nil	...	...	42
24	Chicken ...	6·74	0·38	Nil	...	...	30
25	Food yeast ...	11·6	0·86	1·02	0·01	20 mgm.	58

Vitamin content of common Food Stuffs.

No.	Food article.	Vit. A.	Vit. B_1.	Vit. B_2 (Ribo).	Nicotinic acid.	Vit. C.	Vit. D.	Vit. E.
	CEREALS.							
1	Parboiled rice, whole ...	...	+ +	...	...	...	...	...
2	,, polished...	...	+ +	...	...	...	...	...
3	Raw rice polished ...	O	O	...	...	O	O	V
4	Rice polishings ...	+	+ + +	...	...	...	O	...
5	Wheat whole grains ...	+	+ +	+	...	...	...	+
6	Millets ...	+	+ +	+	...	O	...	...
	VEGETABLES.							
7	Cabbage, green fresh ...	+ +	+ +	+ +	...	+ + +	...	+
8	Cabbage cooked ...	+	...	...	...	+	...	...
9	Carrot, raw ...	+ +	+ + +	+	...	+	...	...
10	Carrot, cooked ...	+	+	...	...	+	...	...
11	Lettuce, green ...	+ +	+ +	+	...	+ + +	...	+ + +
12	Pumpkin ...	+ +	+	...	...	+	...	...
13	Spinach (cooked) ...	+ +	+ + +	...	...	+ +	...	...
	NUTS, ETC.							
14	Ground nut ...	+	+ +	...	...	O	...	+
15	Almond ...	+	+	...	...	..	...	...
	MILK PRODUCTS.							
16	Human milk ...	+	+	+	...	+ + +	0 to +	...
17	Cow's milk, fresh ...	+ varies	+	+ +	+	+	0 to +	V
18	Milk, whole (condensed).	+ +	+	...	...	+	+	+
19	Skimmed milk ...	+	?	...	...	+	...	...
20	Buttermilk ...	+	+	...	...	O to +	...	...
21	Butter ...	+ +	O	...	...	O	+ +	+ +
22	Ghee ...	O to +	—	...	...	...	—	...
23	Groundnut oil ...	O to +	...	...	...	...	O	+
24	Red palm oil ...	+ + +	...	...	...	...	+	+
	FISH OILS.							
25	Cod liver oil ...	+ + +	...	...	...	...	+ + +	+
26	Halibut liver oil ...	+ + +	...	...	...	...	...	...

Vitamin content of common Food Stuffs—*Contd.*

No.	Food article.	Vit. A	Vit. B_1.	Vit. B_2 (Ribo).	Nicotinic acid.	Vit. C.	Vit. D.	Vit. E.
	FISH OILS—*Contd.*							
27	Egg (yolk) ...	+ +	+ +	+	+ +	O	+ + +	+ +
28	Fish liver ...	+ + +	...	...	...	...	+ + +	...
29	Sheep liver ...	+ + +	...	+	+ +	...	O	+
30	Kidneys ...	+ +	+ +	+ +	...	+	+	...
	FRUITS							
31	Banana ...	+ +	+	+	...	+ +	?	+ +
32	Papaya ...	+ +	+	...	...	+ + +	...	...
33	Oranges ...	+	?	?	...	+ + +	...	+
34	Tomato ripe ...	+ +	+	+	...	+ + +	..	...
35	Apple ...	+	+	+	...	+ +	...	...

(2) *Vitamin* B_1:—Food yeast, embryo of cereals and seeds, unpolished or germinated grains, rice polishings, fresh fruits, green vegetables, eggs and kidneys.

(3) *Riboflavine.*—Liver, kidney, heart, germinated grains, milk, and eggs.

(4) *Nicotinic acid.*—Germinated grains, milk, eggs, meat, liver and cheese.

(5) *Vitamin C.*—Fresh fruits (orange, tomato, lemon, grapes), green vegetables and gooseberry.

(6) *Vitamin D.*—Cod liver oil, eggs, butter and milk.

(7) *Vitamin E.*—Wheat germ oil, egg-yolk, milk, green vegetables and mutton.

Nutritive value of certain food stuffs.—1. *Cereals and millets :*—The food value of rice is low compared to wheat, maize and millets in respect of proteins and mineral salts. Wheat is richest in protein, maize comes quite close to it ; the latter is very rich in fat (twice that of wheat). Ragi is several times richer than all others in calcium.

2. *Sweet potatoes and tapioca* are edible starchy root vegetables with high calorific value and are of considerable supplementary food value in these times of scarcity of staple grains. Highly palatable and nourishing preparations can be made with flour from these or the roots can be boiled and eaten.

3. *Groundnut* is an edible nut of high food value containing 40% fat, 27% protein and adequate amount of minerals and vitamins. The cakes obtained after extracting the oil are very cheap, nutritious and suitable for human consumption in the place of pulses when they are in short supply. As large quantity of groundnuts will cause indigestion, it is

necessary to advise against an intake of more than *one ounce daily* to supply the protein and fat deficiencies.

4. *Food yeast* is a very cheap and a chief source of vitamin B; it has a high protein content and can be consumed as a nutritious food article.

5. *Milk* is a nutritive digestible diet article of high caloric value supplying many of the constituents that are deficient in the other ordinary foods (calcium, phosphorus, vitamin B and A); but it lacke in some factors such as iron, and cannot be relied upon as a source of vitamin D or C. The special nutritive value lies in its proteins and mineral content. If *only to the deficient diets every one could add one pint of milk a day, these defects are removed.* But the milk, when raw, can act as a potent vehicle for the spread of diseases. Boiling the milk will destroy the vitamins, precipitate the salts and make the proteins undigestible; and so it must be rendered safe by *pasteurisation i.e.* being kept at 145°F. for 30 minutes and then immediately cooled to a temperature of 55°F. *Skim milk* contains more proteins and minerals but much less fat than whole milk and hence is of considerable value as food for infants with diarrhœa, for premature babies and for those who do not tolerate whole milk. Milk powders, condensed milk, dried whole milk, skim milk may be reconstituted and given to children, expectant and nursing mothers and other sick people when and if fresh milk is not available. They are all good substitutes.

6. *Eggs* are good protein—supplements to either the South Indian or average Bengali diet and are better than soya bean, grams or groundnuts. Their vitamin content is also high.

7. *Meat* is a rich source of high class proteins, nicotinic acid and riboflavine.

8. *Vegetables and fruits* (except mangoes) supply very little of protein, fat or carbohydrate but are rich sources of minerals and vitamins except riboflavine.

It is necessary to remember that amaranth is a rich source of iron, that drinking water taken by each person daily supplies 75 mgm. of calcium which is 1/10 of man's daily requirement, that the arrowroot made from tapioca or edible cannas is wholesome and can be prescribed as food for children and invalids, that milk and ghee are not good sources of vitamin A and that sour butter-milk is better than curds. It should be borne in mind that provitamin A content (carotene) of the coloured leafy vegetables is reduced considerably by cooking as also the vitamin C content of milk and vegetables by heat, that thiamine is lost in the cooking process especially if baking soda is also used, that the vitamin B_1 content is reduced by keeping foodstuffs as tinned food and by the milling of the grains, that overcooking and long storage of cooked food have a tendency to decrease the vitamin value and as such edible substances must be as fresh as possible, that the rejection of water in which the vegetables are boiled takes away their minerals and that the washing of rice and removing the kanji water removes away 60% of its thiamine. Whole meal bread contains 10 times vitamin B_1 as white bread and twice as much as brown bread. Parboiled rice even after milling and washing may contain enough vitamin B_1 to prevent beriberi but raw rice loses 100%.

Standard daily requirements.—The food requirements depend on age, sex, body weight, occupation, climate, season and conditions of health.

The diet must supply a the average on minimum of *30 calories for kilogram body weight* (2·2 lbs) per day *at rest.* But the establishment of a minimum world food target of 2500 calories a day is necessary. 50% of the caloric value must be derived from CH, 30% from fat and 20% from proteins; the heat value of the diet must satisfy the *nutrient radio* P : CH + FX $2\frac{1}{4}$ = 1:8 *i.e.* the diet must consist of 320 gms. of CH, 80 gms. fat, 60 gms. proteins, salt $\frac{1}{2}$ oz and water 50 oz. for a man at rest. It is absolutely necessary that at least part if not the whole of proteins and fats mnst be of animal origin; the diet should contain liberal amounts of vitamins and minerals. The minimum amounts required are :—proteins $\frac{1}{2}$–1 gram per lb body weight, fat 2/3 gram and CH 3–4 gms per day. Expectant and nursing women require extra proteins, vitamins especially A and B and K, calcium and iron. The children and adolescents also require greater amounts of vitamins for growth; undess they get these in time, adequate supply later on will not remedy the mischief already done.

Daily standard requirements.

For	Proteins gms.	Calcium mgm.	Fe. mgm.	Vit. A I. U.	Vit. B_1 I.U.	Vit. B_2 mgm.	N. Acid mgm.	Vit. C mgm.	Vit. D I.U.
Infant ...	...	...	..	2,500	166	0·6	4·0	30	500 to 1,000
Child 6 years ...	50	1,000	8	2,500	300	...	5 to 10	50–75	400 I.U.
Woman ...	60	800	12	5,000	500	...	15	75	...
Pregnant or lactating woman.	100	1,200 to 2,000	15	8,000	1,000	2-3	25	100 to 150	800 to 1,200
Male adult ...	70	800	12	5,000	500 to 1,000	2-3	15 to 20	75 to 100	500 I.U.
Therapeutic dose ...	...	...	...	...	3,330 to 16,650	10 mgm.	50 mgm. daily.	...	4,000 (for infants.)

Pantothenic acid :—Daily need is 5-10 mgm. and the therapeutic dose is 20-40 mgm. orally.

Pyridoxine (B_6) :—Daily requirement is 2 mgm. or 600 gammas. Vitamin A requirement is 50 I.U. per kgm. body weight. Vitamin B_1 requirement can be found out by the formula vitamin in I.U.=caloric intake × wt. in kgms. × ·0284. Vitamin D requirement is greatest during the periods of infancy and also pregnancy and lactation. The normal requirements of vitamins are greater when there are co-existent and intercurrent diseases.

The daily requirement of calcium (1,200 mgm.) is supplied by one quart of milk; that of vitamin A (5,000 I.U.) by 2 pints of milk, 2 eggs, 2 oz. of butter and green vegetables; of vitamin C (50 mgm.) is assured by 6 oz. of orange juice.

South Indian Diet—what it is and what it ought to be in the light of the above mentioned facts. The average S. I. diet consists mostly of

cereals (90% is cooked rice). 3 per cent pulses, 4 vegetables and 1 per cent meat, fish, etc., and thus contains 40 grams of proteins (of which 15 per cent only is of animal origin and 80 per cent from the cereals), 25 gms. of fat and 500 gms. of carbohydrates. It lacks in animal proteins and protective food factors such as vitamin A and C as the consumption of milk, meat, eggs and fruits by the common man is negligible; it lacks in vitamin B due to the fact that milled rice is used and even that is washed; it lacks in some minerals as they are lost by the throwing away of the water in which the vegetables are cooked. Thus the diet is ill balanced and is not enough to furnish energy for work nor to maintain normal health, much less to allow for normal growth. A proper reorientation of our dietary regime is therefore essential; the whole must be recast, remoulded and modified in the light of the remarks made. The protective foods are expressive and beyond the means of even some of the middle classes and all the poor classes. Milk, eggs, and fresh fruits have become articles of luxury with a majority of the people. It is impossible to improvise or purchase a balanced diet with protective factors with a per capita income of 2½ annas a day. As it is essential that the diet that must provide optimum nutrition and lead to vigour of body and mind must be improvised at cheap cost and within the easy reach of the man in the street, the improvement in the dietary of the nation could be achieved only if the economic status of the average man and his purchasing power are increased and augmented and only if measures are taken at once to produce the protective foods in large quantities and at cheap prices.

The first step towards improving the S.I. diet will be to make use of this present emergency of rice scarcity and replace a portion of the rice in the diet by wheat, maize or ragi which are easily available and are certainly more nutritive. The people must be educated to get accustomed to such a *mixed diet*. It should be seen that the cereals are handpounded and not milled; the rice water should not be thrown away. Also food yeast and ground nuts—one ounce a day—may be added to the diet with advantage. Soya beans and more of pulses and grams must be added on with effect. The improvised diet should contain abundance of fresh leafy and colored vegetables which should be properly cooked. Milk and milk products must form an integral and essential part of any diet and must be made available to all (1 to 2 pints a day especially to children and mothers). Eggs or fish or meat must be consumed twice a weak at least. Thus a cheap modification of the present S.I. diet which would be nutritious and balanced is cooked parboiled hand-pounded rice 50 per cent, ragi cooked dhall 20 per cent, sprouted pulses 5 per cent, green leafy vegetables, amaranth 10 per cent and in addition peas, beans, bananas, papaya and food yeast. This will be well within the means of the poor class; and the middle class people will do well to add a pint or two of milk, 1 or 2 eggs, an ounce of butter, fruits like oranges and apples together with eggs or fish or meat occasionally.

III. **Medical welfare.**—The relief measures by the medical profession entirely depend upon what manifestations of famine conditions and starvation in human beings they are called upon to treat. It is necessary to know in detail the *effects of famine starvation on health*. The onset of famine conditions will first affect in one or two ways the poor classes (among them the decrepits and destitutes) who are already under-nourished and semi-starved. These are: (1) *Acute starvation:* rarely occurs in famines

and; (2) *Chronic starvation* commonly occurring in the famine-stricken areas and leading on to hypovitaminosis, hypoprotinæmia, inanition, marasmus, intercurrent diseases and death.

1. *Acute starvation* is sudden, total deprivation of food. The symptoms and signs are:—

(*a*) *Hunger* which is most acute within 24 hours and then it tends to disappear in another 24 hours. Pain and discomfort in the stomach at first relieved by pressure but gradually becoming intense. Hunger pain disappears on the 5th day.

(*b*) *Body weight* is lost rapidly at the rate 1/24 weight daily. The muscles waste, fat disappears and exhaustion and emaciation appear. Prostration gradually increases.

(*c*) The voice becomes weak; tongue dry and coated; mouth dry and saliva scanty; the breath becomes foul.

(*d*) *Pulse* becomes first quick, then slow and later very rapid and weak.

(*e*) *Temperature* after a preliminary rise falls to subnormal.

(*f*) *The gums* become spongy and the mucous membranes red, inflamed and excoriated.

(*g*) Skin is scurfy and putrescent, smell comes from the body.

(*h*) The eyes become wild and sunk; pupils dilated.

(*i*) Facies hippocratica sets in; delirium may and coma often dose precede death. Death occurs when 40 per cent of body weight is lost. An adult can live for 10 to 12 days even without water.

But if water alone is given life may be prolonged to a maximum of 2 months. Death is due more to loss of heat than that of nutrition. The onset of death depends on (1) *Age*: older patients stand starvation better. (2) *Sex*: females bear it worse. (3) *Adeposity* more the fat, longer the survival. (4) *Amount of exertion*, greater it is, quicker is death. (5) *Temperature* colder it is, death occurs earlier. Many assume that after the hunger pain disappears on fifth day in starvation, death is a gentle fading away, not entirely unpleasant; but this is wrong.

2. *Chronic starvation.*—Due to famine conditions and scarcity coupled with the high cost of protective foods, many people will have nothing but unbalanced diets. The qualitative deficiencies in the diet already present become accentuated greatly and lead first to deficiency states such as hypovitaminosis and hypoprotinæmia—manifestations of *malnutrition*.

Malnutrition is essentially a clinical conception; it is revealed by the state of the skin, lustre of hair, appearance of the eyes, colour of mucous membranes, alertness and attitude of the person. This malnutrition is seen in many children even in normal times and it is bound to affect even the elders as famine conditions set in. But the symptoms of under and malnutrition in early stages are not marked enough to attract the attention of the victims or drive them to doctors; they can however be discerned by the experienced clinician who makes a thorough examination. Mostly moderate deficiencies leading to subclinical states may be encountered in famine areas. The children especially of the growing age, expectant and nursing mothers and the sick are first liable to suffer from the deficiencies in the diet.

I. **Hypovitaminosis.**—Is bound to flare up in the famine areas due to lack of the vitamins in the diet. It may also occur commonly as complication of other diseases. The vitamin deficiencies are rarely single and uncomplicated and in most cases several factors are involved. In the early stages and in mild deficiencies the clinician must depend on indefinite signs and laboratory diagnostic tests. The gross deficiencies of the vitamins leading to well developed specific diseases give obvious signs but are rare even in famine areas. As vitamin B is not stored in the body and a continuous supply must be constantly ingested, its lack is more quickly manifested clinically than those of lack of vitamins A, D or C.

Lack of vitamin A.—The subclinical deficiency of this is manifested by functional changes prior to the pathological changes. (1) *Defective or poor dark adaptation.* The subnutrition is assayed by dark adaptation test by using a biophotometer; (2) The earliest manifestation is *night blindness*; next is decreased resistance to infection especially respiratory; and (3) the earliest sign is *peculiar smokiness of the conjunctiva.* These are danger signals which put one on guard to avert its advancing to the stage of keratomalacia. The latter manifestations of vitamin A deficiency are xerophthalmia, keratinisation of epithelium of respiratory, urinary, alimentary passages and skin, coreal opacity, general lowering of body's resistance and retardation of growth in young.

Lack of vitamin B_1.—Loss of appetite, atonic condition of gastro-intestinal muscles causing intestinal stasis and autointoxication, derangement of carbohydrate metabolism which causes polyneuritic symptoms. However gross deficiency is manifested by beri beri (wet or dry types; infantile types).

Lack of riboflavine which plays an important role in cellular activity in the formation of the yellow enzyme and in its oxidation function. The earliest indication is *typical glossitis*—tongue clean, pappilæ flattened, colour purplish or magnetta.

(2) Next is *corneal vascularisation* (due to anoxia) symptoms of which are fatigue, aching and burning of eye, intolerance to reading, decreased visual acuity in weak illumination and photophobia.

(3) *Cheilosis.*—Reddening, cracking and maceration of lips and *perleche* or angular stomatitis.

(4) A fine *seborrheic follicular dermatitis* scattered at nasal folds, eyelids and ears.

(5) *Phrynoderma* comes later on.

Lack of nicotinic acid causes pellagra, when other vitamins of B complex are also deficient. In the prepellagrous condition which is common; the early clinical picture is one of gradually developing lassitude, anorexia, vague digestive symptoms, retarded mental processes, mental torpor or apathy, emotional instability and irritability, dizziness, and later superficial glossitis, angular stomatitis and typical skin lesions.

Lack of vitamin C.—Its gross deficiency causes scurvy. The early manifestations of scurvy which must be recognised are loss of appetite, weakness, and swelling of and sponginess of the bleeding gums; the early diagnosis of of latent scurvy is by the X-ray evidences in long bones and in the failure to excrete an optimum amount of ascorbic acid in urine. Dental decay is also seen when there is deficiency of calcium and vitamin D also. Later the characteristic signs of scurvy such as purpura occur.

Lack of vitamin D grossly results in dental decay and rickets in children.

II. Another effect of food deficiency on health is **hypoprotinæmia** which is symptomatically manifested by *nutritional œdema.* This is caused by insufficient proteins in a diet low in calories (as in people of famine areas who are forced to live on roots and vegetables). This limited amount of proteins is diverted to produce energy and plasma becomes deficient in proteins; its osmotic pressure therefore falls and fluids leak into the tissues causing œdema. In children there is no typical œdema but they become pale and doughy-looking. Salt in the diet will increase the swelling.

III. **N. Diarrhœa.**—The most noteworthy effects of starvation are on the alimentary system; the Hcl of the stomach decreases; absorption lessens and intestinal ulcers develop. These account for the intractable diarrhœa and colitis simulating dysentery in malnutrition. When this combined with nutritional œdema the patient is in grave danger.

IV. **The immunity reactions** are lost by the starving and so they must be protected against infectious diseases during the famines when epidemics are likely to break out.

V. **Avitaminosis** is due to gross deficiency of the various vitamins in the diet; however this is of rare occurrence in famines. The diseases are xerophthalmia, beri beri, pellagra, scurvy, and rickets.

VI. In most cases of starvation where there is no gross deprivation of food, the victim lingers for a long time before some *intercurrent diseases* like broncho-pneumonia, dysentery, nephritis, etc. set in. These may mark the true nature of illness and we may miss the underlying malnutrition.

VII. **Marasmus** is a nutritional disorder of infancy and childhood caused directly and primarily by dietetic errors or food deficiency (quantitative or qualitative) and indirectly by some conditioning diseases such as malaria, chronic dysentery, helminthiasis, etc. Marasmus may occur either due to mother's defects (during pregnancy and lactation the mother should have a balanced diet of maximum nutritive value) or due to defects in the child itself (impaired intake, absorption and utilisation of food by which a vicious circle is created). The condition is characterised by stunted growth, progressive wasting, loss of weight, extreme emaciation; muscles become thinned out, skin loose and bones stand out prominently. The children suffer from digestive disturbances; enlargement of liver and spleen occur; some suffer from recurrent attacks of respiratory catarrh, low fever, and œdema; and in some there are manifestations of the vitamin deficiency such as xerophthalmia, rickets and scurvy.

VIII. **Inanition.**—Large percentage of these cases give a history of malaria while most others of bowel upset. Many have severe diarrhœa or colitis which is neither amœbic nor bacillary; some of the children have prolapse of rectum. The patients are very thin, almost reduced to a skeleton, cheek bones being very prominent; emaciation of the limbs may be often masked by presence of œdema. Subcutaneous fat is completely absent. All organs and tissues are shrunken. Skin is dry and cold.

Temperature is subnormal; in a few it is raised, while in others the sub normal temperature increases as improvement occurs. They show the characteristic mental picture of inanition—a marked apathy. Blood pressure is greatly reduced. One great peculiarity is the presence of great craving for food which subsides with improvement of the general condition

IX. **Post-mortem appearances.**—Emaciation and wasting of the body. Absence of fat even in the omentum. Organs are pale and atrophied. Stomach and intestines are thin, empty and contracted; their mucous membranes are ulcerated. Liver, lungs, heart and kidneys are destitute of blood. Gall bladder is full. In most cases there are evidences of serious complications such as pneumonia, nephritis, dysentery, anæmia and ulceration of the gut. Death from starvation may be simulated by death from exhaustion or from diabetes, sprue, tuberculosis, Addison's disease and chronic dysenteries. Death from starvation can only be proved negatively by excluding death from wasting diseases.

In main the starving sick destitutes requiring treatment come under 3 groups:

I. Those suffering from inanition due to starvation.

II. Those suffering from inanition due to a combination of starvation and intercurrent diseases.

III. Those showing relatively little inanition but suffering from acute or chronic diseases or from infectious diseases.

Those of GROUP I are seen to be in one of the following four stages:

1. *Collapsed cases*:—Last stages of acute or chronic starvation; either severely dehydrated or œdematous and semicomatose; are incapable of taking even simple liquid diet by mouth. Likely to die without parenteral feeding and therapy.

2. *Less markedly collapsed cases*:—Anorexia and failure of thirst present and even milk may not be retained in the first few days. But they are capable of recovery by oral feeding.

3. Cases capable of taking simple milk diet.

4. Cases usually able to obtain nourishment from gruel.

GROUP II:—Diagnosis is more often difficult. The presence of the disease may be masked by the inanition (patients may show malarial parasites in blood but no temperature; may have infection but no fever; may have amœbic dysentery but no diarrhœa). Such cases are mistaken and treated for inanition but when their general condition improves with treatment, they develop the symptoms of the disease.

GROUP III:—The typical manifestations of the particular diseases are present and so easily diagnosed. The method of handling and management of these necessarily varies and differs for each type.

Treatment.—The Government must, in addition to tackling the nutritional problem mentioned previously, spread medical relief far and wide and within easy reach of every person in need of it. They should organise *Emergency Camp Hospitals* with free kitchens attached, in the scarcity areas for dealing with the starving or sick destitutes. There should be *acute hospitals* of 20 beds each at every 5 miles and *chronic hospitals* of 100 beds each in the main sticken areas. These should be well equipped

and supplied with all essential medicines to treat influensza, malaria, pneumonia, helminthiasis, dysentery, scabies etc. The Acute Hospitals must serve to treat acute starvation, inanition and acute diseases. The hospitals must have a full complement of disinfectants such as tropical chloride of lime, lysol, D.D.T. as well as yeast, multivitamin tablets, milk powders and other hospital necessaries such as bedding, clothing etc.; *protein hydrolysates and glucose saline* must be readily available in large quantities in all hospitals for the treatment of inanition. Within hundred yards of each hospital there should be an *infectious diseases shed* adequately provided with water supply, cooking facilities and sanitary arrangements. There should be ambulance vans attached to each hospital to serve as epidemic vans for picking up starving destitutes from the road side. As part of these famine relief hospitals there should be *Mobile Health Units* consisting of touring medical parties in mobile vans to tour the villages to render first aid as well as medical relief in rural areas and to distribute milk, multivitamin tablets etc. to the needy peasants.

The line of treatment depends upon the deficiencies present and upon the amount and severity of starvation and upon the conditioning and other diseases existing.

Treatment of hypovitaminosis:—The most important element in the treatment is fundamentally the establishment of a sound dietary regimen which is balanced and complete in itself based on the individual's requirement. It is essential that the diet must be one that the patient can purchase, eat and assimilate. The specific food factors recommended must be available in the locality. This is important as the deficiency state will recur if the faulty diet is not rectified besides the therapy instituted. However it is inadvisable and impractical to rely on diet therapy alone and so that deficient vitamin must be substituted either orally or better parenterally. But as vitamin deficiency is definitely multiple in nature a policy of a *mixed multiple vitamin therapy* must be adopted in the treatment of these nutritional deficiencies in addition to the specific vitamin. At the same time any factor such as gastro-intestinal diseases, toxæmia of pregnancy, dysphagia, intestinal parasites and carcinomata which interferes with proper assimilation of food factors must be treated and parenteral administration of the vitamins would be required in those cases. Other co-existing diseases and other conditions causing excessive loss or requirements of these factors must be attended to.

In treatment of frank deficiency diseases (*Avitaminosis*) such as xerophthalmia, beri beri, rickets or scurvy, the institution of massive doses of the vitamin A, B_1, D or C, parenterally in the beginning and later orally, along with large doses of multivitamin tablets (6 a day) is indicated. In addition a suitable diet containing vitamins in large amounts and consisting of milk, eggs, fresh fruits, vegetables and liver should be consumed for months and even years. Complicating diseases should be cured as otherwise vitamin therapy would be of no avail.

Treatment of partial starvation:—These patients have been undernourished for some months but are not acutely ill. The treatment is essentially dietetic and they respond promptly to a diet of easily digestible foods such as milk or better skimmed milk, butter-milk, fresh ripe fruits, vegetables and cereals. Dehydrated potatoes, dried milk, dehydrated fish and dried eggs even may be added later on with benefit. These patients

suffer from diarrhœa and so excess of fat is contraindicated. Pepsin and Hydrochloric acid may be needed to help digestion. The patients should have absolute rest in bed for at least 1 to 2 weeks till they are able to take any ordinary diet. As an adjuvant to the diet, two or three multivitamin tablets should be given daily. (1 tablet supplies 2500 I.U. of vitamin A, 500 I.U. of vitamin D, 26 mgm. of vitamin C, 2 mgm. of Thiamine, 2 mgm. of Riboflavine, 20 mgm. of Nicotinamide). Any one of the following preparations is handy and beneficial. (1) Vitaminets (Roche); (2) Abidol--with capsules (P.D. & Co); (3) Esdairte Pearles (Sharp & Dohme); (4) Wyamin capsules or (5) Vigran capsules (Squibb). These tablets are stable, readily assimilated, 'valuable, economical and convenient. Prognosis is good in these cases except when the patient is very old.

Treatment of inanition and starving sick destitute:—This depends on the group and the stage to which the case belongs.

GROUP III. Primarily the disease must be treated along general lines either in acute or the infectious diseases hospital. Because their condition is weak and they are starved, they require hospitalisation and good nursing. Feeding should be done with digestible and balanced diet supplemented by multivitamin prophylaxis.

GROUP II. The inanition must be treated first in the lines to be mentioned under group I. Dietetic treatment is all the more important in this group II *i.e.*, in cases of diseases superimposed on starvation or *vice versa*. A starved person suffering from acute infection has no reserves and so the diet must be kept at the maximum possible level unless there is gastro-intestinal disorder. Thus a highly nutritious fluid diet is of vital importance. Glucose water, fresh lemon or orange juice, barley water, whey and butter milk are used. Multivitamin tablets 3 or 4 have to be given daily. When improvement occurs the patients could be given gruel. The intercurrent disease, if acute, must be treated simultaneously along with the dietetic treatment.

GROUP I cases. The immediate problem thus confronting the doctors in the Emergency Famine Relief Hospitals is to revive the patients who are brought in a state of collapse by some parenteral therapy and parenteral feeding so as to prevent high mortality. Thus in the severe cases that belong to stage (*a*) the collapse must be treated efficiently at once by *I. V. protein hydrolysate* containing glucose and B complex as described in detail below. If this preparation is not available or is contraindicated then 5% Glucose saline may be given. Besides the general treatment for collapse by *rest, warmth and Coramine* must be given. As many of these are destitutes who have been exposed to cold, they should be provided with clothing and blankets. As these patients recover from the collapse, dietetic treatment should be instituted. In the beginning it may be necessary to resort to *nasal feeding* even. Pass a Ryle's tube into the stomach by nasal route, leave it in the stomach and attach it to protein hydrate infusion apparatus. A suitable fluid diet fit for administration by this nasal method besides the protein hydrolysates is :—

Fluid diet I.—Consisting of thin gruel prepared from 4 oz. of flour (rice, barley or wheat) to which is added sugar (4 oz.) salt ($\frac{1}{4}$ oz.) as well as $\frac{1}{2}$ oz. yeast, one multivitamin tablet and $\frac{1}{2}$ oz. of shark liver oil. This yields only 800 calories. Many patients will be able to take this fluid diet by mouth when they recover from the collapse. Some may require and

tolerate in addition fruit drinks prepared from 2 or 3 oranges, 2 oz. Glucose and water. The caloric value of this diet I is too small to support, life indefinitely and so this is only an interim treatment designed to carry over the patient to the next stage. As the patient progresses his diet is to be changed to *Milk diet II*. This consists of the above thin gruel or conjee and in addition milk—fresh if tolerated or tinned milk two pints, thus supplying a caloric value of 1,200. This diet is also insufficient and so it is increased to *Gruel diet III* as soon as the patient becomes fit to consume it. This consists of a gruel made of 5 oz. of rice, 5 oz. of atta, 3 oz. dhall, $\frac{1}{2}$ oz. salt, 4 oz. green vegetables, and onions $\frac{1}{2}$ oz. yielding 33 calories per oz. In addition the diet consists of 1 pint of milk, 4 oz. sugar, $\frac{1}{2}$ oz. shark liver oil and one multivitamin tablet. Fruit drink also may be given. The total caloric yield is 1,900. Thus the patient's diet is changed from one category to the next as his condition improves. Patients of stage (*b*) can take fluid diet I, of stage (*c*) can tolerate milk diet II and of stage (*d*) can utilise gruel diet III straight away.

The use of protein hydrolysates in the treatment of starvation and of inanition.—

It is necessary to know in detail about *protein metabolism and synthesis*. Proteins are vital ingredients necessary for the life and growth of every cell in the tissues. Digestion of the ingested proteins begins in the stomach where pepsin and Hcl convert them into simpler proteoses and again to peptones, which are however still complex to be absorbed and need further to be broken down in the small gut. Trypsinogen converts these peptones into aminoacids which can be absorbed into the blood. Then the main site for the synthesis of the plasma proteins from these aminoacids in the liver which produces them for body nutrition and use. The tissue proteins are elaborated by the cells of the body from the aminoacids to suit their particular requirements and peculiar to their structure and function. Portion of aminoacids not required by the tissues is taken by the liver and deamination occurs and the amino part is converted into urea which is returned to the blood to be excreted by kidneys. The amino free part is also sent to blood to be used up in energy production. In addition other amino acids are continually being produced by the breaking down of the proteins in the body cells in their normal activity. These endogenous aminoacids are also treated similarly by the liver. There is an increase of destruction of body proteins as well as poor ingestion and assimilation of proteins in starvation causing hypoprotinæmia.

Historical.—HENRIQUE and ANDERSON (1913) showed that proteins hydrolysed into its constituent aminoacids by some suitable means could safely be given I.V. to protein starved animals and could maintain them in nitrogen equilibrium and support life. Due to increasing demand for large quantities of transfusion material for combating shock and due to the high cost and other difficulties attended with the preparation of plasma and serum, various other cheaper substitutes had to be tried. CHEVILLARD pointed out that it was possible to supply the nitrogen needs of man by administration of a mixture of aminoacids. But is not feasible to use a pure aminoacid mixture as was done by MADDEN and others (1943) as it is ineffective as well as expensive. In 1939 ELMAN and WEINER had reported that an acid hydrolysate of casein fortified by tryptophane and cystine could be used for I.V. alimentation of human subjects with favourable

results. ELMAN and LISCHER (1942) demonstrated that solutions of protein hydrolysates could be administered I.V. to man in fair amounts in cases of shock, hypoprotinæmia and inanition and maintain him in nitrogen equilibrium when he was not able to take food by mouth. It was observed that these pH lost none of their nutritive value, that these could be metabolised I.V. as well as proteins in the diet of normal individuals and that therefore they could be used as temporary expedients to supply the nitrogen requirements of the body when alimentary tract could not be used. In America *Amigen* (a pancreatic hydrolysate of casein) was recently used; others used acid hydrolysates. But many of these pH gave rise to allergic or severe reactions, so preparations of pH in a form better suited were investigated at the All India Institute of Hygiene, Calcutta, during the Bengal Famine of 1943 with the result that papain digest of meat had been found useful. Quite recently in Holland where the food situation is grave, research work is going on under the ægis of Medical Research Council about the efficacy of serum, plasma and pH in starvation. Attempt is being made there to administer an enzymic partial digest of casein or meat with addition of glucose and vitamins by slow intra-gastric drip and to note the effect. The results are awaited.

Rationale of therapy :—Just as glucose is given I.V. as a means of supplying predigested CH, so also pH can be injected for supplying the nitrogen needs of a patient which are more urgent in cases of starvation, as the tissue proteins are being rapidly broken down. Glucose alone will supply the caloric need but is insufficient. Proteins and even pH can be given by mouth but they need to be digested or absorbed in the gut, which is almost impossible in cases of advanced inanition or collapse because of intestinal ulcers, diarrhœa and damage to the liver. Hence for quick and sure effect parenteral administration of pH has to be resorted to, if life is to be saved. If along with hydrolysed proteins (easy of assimilation), glucose and vitamins are given I.V. as in the preparations of All India Institute of Hygiene, the starving tissues receive their urgent and essential requirements in a form ready for utilisation. Can plasma or serum I.V. be used instead? What is its therapeutic value in inanition? In starvation the need for tissue proteins is more urgent than that for serum proteins and as the utilisation of serum depends on the proper functioning of the enzyme systems responsible for splitting up of the proteins into animo-acids and for reconverting them into tissue proteins and as these systems are deranged in inanition, serum does not act as well as pH.

Use of protein hydrolysates :—It could be said with certainty that a number of deaths by inanition could be prevented by using pH parenterally. The results in advanced inanition have been satisfactory and beneficial. The immediate benefit from this is chiefly due to its glucose saline content rather than the hydrolysates and this effect is maintained for another 12 hours. The effect due to the aminoacids in pH comes on only 3-4 hours after administration. The glucose acts by reducing liver damage and by increasing the functional efficiency of the liver. The amino-acids and pepticles reach all the tissues simultaneously and are synthesised into tissue proteins in 3-4 hours and into serum proteins in 6 hours. The vitamin B complex helps in the metabolism of glucose. In most cases within 3 days there is resuscitation to the point at which fluid or milk diet may be taken. Thus the pH has a definite therapeutic value in hypoproteinæmic conditions such as inanition, starvation and malnutrition by helping in the synthesis of essential body and plasma proteins. The intractable diarrhœa which is a prominent feature of these conditions responds dramatically to this I.V. therapy.

The intra-gastric drip method also may prove to be a satisfactory alternative form of administration. Thus the pH may solve not only the immediate problem of combating collapse in inanition but also may serve for treatment of all conditions of malnutrition associated with impaired absorption or ingestion from the alimentary tract.

It has been, however, found that its value in the treatment of shock—surgical shock or in extensive burns would come only midway between that of serum and glucose saline, because the real benefit from pH could come only 6 hours later when the aminoacids begin to be converted into serum proteins and because pH do not maintain blood pressure at high level. So unless it is further reinforced with suitable colloids, it can occupy only a second place to serum in the treatment of surgical shock.

The pH can be used in *infective hepatitis* with benefit. A sense of wellbeing and improvement in general condition occurs within 3 days; the sedimentation rate begins to rise in 2 weeks and is normal in 6 weeks; the liver comes to normal size very rapidly. The marked improvement is due to the direct supply of protein digests that have a protective action on the undamaged portion of the liver and due to the re-establishment of nitrogen equilibrium. Frequent feeds of pH can be given for treatment of *peptic ulcers*.

Methods of preparation.—pH can be prepared in one of 2 ways :—

(1) *Acids hydrolosis* :—This is an easier method and presents no great technical difficulties. But the hot acid used destroys the essential tryptophane, which has therefore to be added later. This aminoacid is difficult to obtain, is also costly. These pH are fairly satisfactory though not absolutely safe in all cases (ELMAN).

(2) *Enzyme hydrolysis* is an easy and cheap method, but the hydrolysis is not so complete so that the resultant mixture contains polypeptides ac well, derived from the substrate and the enzyme. These cause allergis reactions and so have to be removed. Trypsin was the enzyme used; but it had 2 drawbacks :

(*a*) Optimum temperature of its action was 37°C and at that temperature it was difficult to avoid bacterial contamination. Its pH was toxic and pyrogenic.

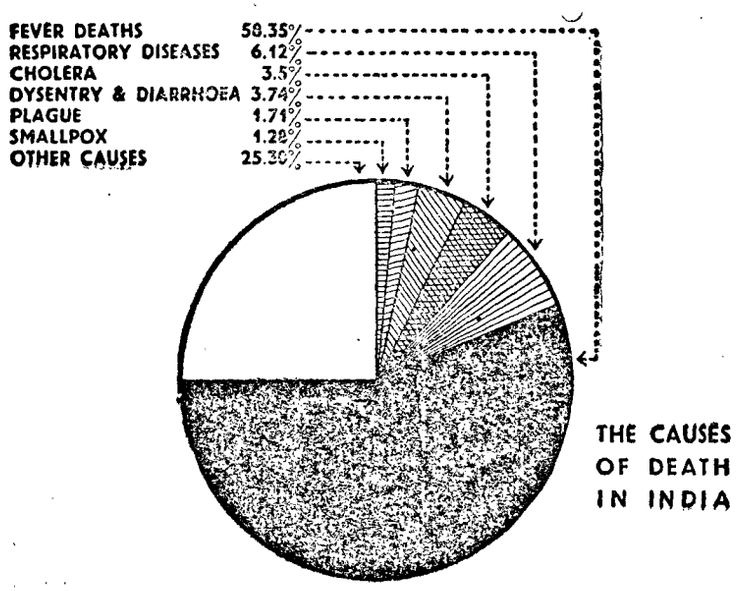
(*b*) Histamine or histamine-producing enzymes may be present in the Trypsin and cause reaction. One advantage of Trypsin was that its hydrolysis was extensive. Yet an enzyme without these two drawbacks had to be found out for producing hydrolysis. Papain, the cheap vegetable proteolytic enzyme, containing both proteinase and polypeptidase, was selected, as its optimum temperature of action was 50°C and pH = 5 – 7 and its reaction products were free from histamine. Papain can be produced easily by collecting the latex of unripe fruits of carica papaya after scarifying the fruits while still on the tree and by mixing the later with acetone and drying repeatedly till a powder is got. The hydrolysis by papain is not complete but thereby the therapeutic properties of the pH is not diminished. Meat is chosen as the substrate, as the reaction of papain on casein or vegetable proteins is limited. The pH prepared at the All India

Institute of Hygiene is a papain digest of meat. Meat which is freed from all fat and bone is minced to a fine consistency. About 1/40 by weight of papain dissolved in water is added and digestion is conducted at 50°C for 24 hours. The formal titre, total solids and total nitrogen are determined to note the extent of digestion. Then the undigested proteins, metaproteins and polypeptides which act as an aphylactogens are removed by repeated heat coagulation at pH=7. By an aseptic technique and addition of a preservative (toluol), bacterial contamination is avoided. The final product is mixed with Glucose and Sodi Chloride so as to give a mixture of 5% pH, 5% Glucose and ·85% Nacl; the mixture is then sterilised and tested pharmacologically on cats for absence of toxicity, bacteriologically for sterility, and immunologically for freedom from allergic reactions. It is seen that this mixture also contains vitamins B complex which increase its therapeutic efficiency. The mixture is supplied in screw-capped, blood transfusion bottles, each of 200 or 400 c.c., it is of port wine colour, free from precipitate or turbidity. The bottles are best stored in cool, dark place in a cup board at room temperature. Storage tends to deepen the colour but this is of no importance. The qualities are retained for a long period and as such no date of expiry is fixed. Some bottles may contain few fine suspended particles; then they should be filtered through sterile gauze before use. Bottles showing turbidity or precipitates or giving a foul odour on opening or whose corks are found to be loose should be rejected. Once a bottle is opened it must be used up within two hours. After use the transfusion set should be immediately washed thoroughly in clean water and resterilised for use; the fluid in it may decompose with formation of toxic substances.

Technique.—The I.V. injections are best given using Haye's pattern transfusion set. The screw cap of the bottle is removed and the sterilised cork of the transfusion set is fitted on to the bottle which is inverted and hung up. The flow through the tube is adjusted at *60 drops per minute* and the needle is introduced into the cubital vein. This rate is important, as more rapid administration is not tolerated. It takes 1 hour for the 200 c.c. to be given. The maximum daily dose is 400 c.c., this can be given as a single dose or in 2 doses b.d. Most of the collapsed patients require one dose daily for 3 consecutive days but rarely it may be necessary to continue for another 3 days. The effect is often immediate and lasts for 24 hours. Improvement in pulse, respiration, blood pressure and general condition follows very rapidly. There is marked improvement in 3 days and as one dose supplies only 100 calories, it is quite essential to supplement it by nasal feeding or intra-gastric drip method and later on by fluid or milk diet. These pH may be given by mouth also in small quantities or by a stomach tube.

Pronutrin (Herts) is a casein hydrolysate; it is palatable and taken as a solution in water. If the inanition cases are also suffering from pneumonia, dysentery etc, the administration of pH parenterally is not contra-indicated but it should be supplemented by specific therapy.

Contra-indications.—These are *nephritis and liver damage.* So it is always essential to examine the urine for albumin and casts. But in advanced cases of inanition, these pH can be given though there may be a trace of albumin. But if there is generalised œdema, kidney or liver damage, other methods of treatment should be adopted.



The starving destitutes who are brought to the hospitals may not be suffering from much of inanition but may be suffering from other systemic diseases or infectious diseases. Those that show a high incidence, are malaria, bowel diseases (dysentery, diarrhœa), œdema, nephritis, anæmia, pneumonia, generalised scabies, tropical ulcers (Naga sore) and epidemic diseases such as cholera, pox and measles. These diseases have to be treated on general lines with the specifics either in the acute or the chronic or the infectious diseases hospitals. It is necessary to give them in addition a balanced nutritions, easily assimilable diet, fluid or ordinary.

Thus it is the bounden duty of the medical practitioners to serve the nation in her distress and they would be relieving the misery in famine areas to a considerable extent by their devotion and service.

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Flatfoot*

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Introductory.—The characteristic gait of Charlie Chaplin has always been a very amusing sight for the filmgoers. Nevertheless there is no controversy over the fact that flatfoot is a great disadvantage and curse to one who has it.

As we stand and walk all our bodyweight is supported by our feet. Further, when we jump from a height or carry heavy loads, as many of us very often do, extra strain is thrown on our feet. All this would have been very difficult if nature would not have modified our feet to meet our requirements satisfactorily. In the anatomy of our feet an elastic structure has been supplied which is made up of a good number of small bones held together by ligaments, tendons and muscles. In a normal adult foot there are transverse and longitudenal arches. These arches of foot may be looked upon as angles which are concave downwards and convex upwards. When these angles are straightened out, in other words, the arches are slackened, a clinical condition, the name of which is self explanatory—the flatfoot—develops. (*Vide page 100 Fig. 1.*)

Varieties and mechanism of causation of flatfoot.—1. Flatfoot is rarely congenital. It is said to be a dominant feature in certain families and is occasionally so seen in Jews.

2. The spasmodic variety of flatfoot is usually the result of painful spasm of the peroneal muscles.

3. The paralytic form usually occurs due to infantile poleomyelitis.

4. It is associated with telepes and is accompanied by coldness, blueness and wasting of the limb. The deformity is corrected easily by manipulation but recurs at once.

* Specially contributed to THE ANTISEPTIC.

5. The inflammatory variety is a result of inflammation. Due to inflammation the ligaments yield rapidly and are accompanied by swelling, redness, edema and pain. This is usually due to the gonorrhœa but may also be rarely due to septic inflammation, acute rheumatism, tuberculosis, etc.

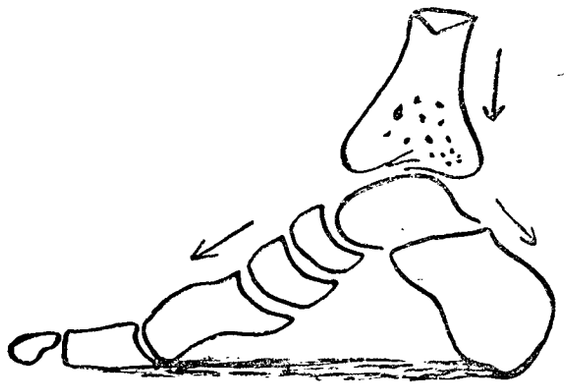


FIG. 1
The arch of the foot.

6. Traumatic form is also common and serious. Injuries in the neighbourhood of foot and ankle usually cause it. It is sometimes seen as a result of malunited Pott's fracture in which fibula has been allowed to be united with the foot in the everted and valgus position. Similar condition may develop in badly treated fractures of tibia and fibula or both. It is also sometimes caused by fractures of astragalus or os calcis or rupture of the ligaments supporting the arch.

7. Rickety form of the disease is also seen.

8. In some cases it occurs as a compensatory deformity secondary to the malposition of the limb above, such as coxa vera, jenu valgum or carvature of tibia.

A patient long in bed may also experience flatfoot. This is due to loss in tone of the muscles due to lying in bed for a long period.

Although a fully developed congenital flatfoot is a rare finding yet in certain persons a tendency towards flatfoot is usually observed. There is some inherent weakness of the ligaments and other supports of the arches and this, at times, has a hereditary trait. In these persons-unless good precautions are taken early - flatfoot is very frequently seen.

It is also common in long and narrow feet and is the result of the insufficient supporting power of the body for the bodyweight. It is normal in very small children in whom the ligaments and muscles of the foot have not adapted themselves for standing on the feet. However, cases of flatfoot are often seen in rapidly growing children and adults of subnormal health especially when they begin to do some job which requires long periods of standing or carrying weight.

Faulty methods of standing are also responsible for causing flatfoot Many people who have to keep standing for long periods adopt an usual position which throws the least strain on the muscles. They usually stand with their feet apart and abducted, knees slightly flexed and toes everted

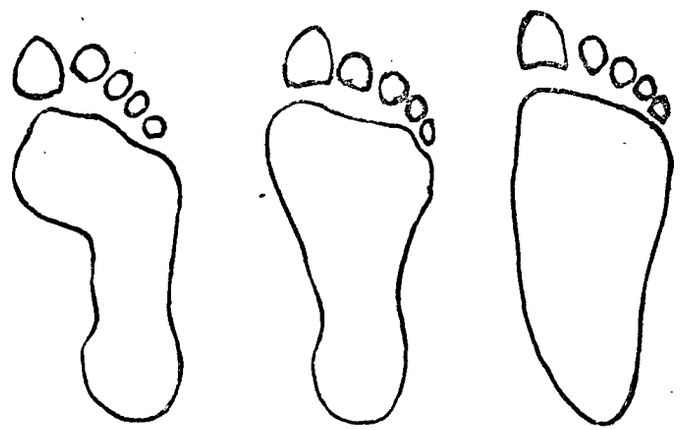
The parts of the foot which normally bear the body weight and transmit it to the ground are the tuberosity of calcaneous, head of the first metatarsal bone, and the head of the fifth metatarsal bone. The talus is the keystone of the arch, it receives the body weight and transmits it to the arches below. The position described above is very bad and unsuitable since the entire bodyweight is thrown on the ligament and bone of

he foot causing them to stretch. This may be aggravated by weakness of he muscles.

When the ligaments especially the inferior calcanes scaphoid, and the endons supporting the arch become in efficient, the head of the talus is no nore supported and it drops down. It forces itself between the internal nalleolus and the tubercle of the scaphoid with the result that the forefoot is deflected laterally and into valgus position. Also the inner border of the foot which should normally show a slight concavity is replaced by a convexity.

Symptoms.—The symptoms complained by the sufferers of flatfoot are varied. Before pain comes, early tiredness by standing or walking. Pain follows next and is the most marked feature before any deformity is developed. In the beginning the pain is most severe just behind and below the tuberosity of the scaphoid, where it is due to the stretching of the spring ligament. Pain may radiate all over the foot and to the calf. It may also be experienced across the instep due to the stretching of the interosseous ligaments. Cramps in the foot at night are common. When the arch of the foot has completely given way the pain may altogether disappear. With this come the arthretic and inflammatory changes in the little joints followed by adhesions. This makes the deformity permanent.

FIG. 2



Normal. Moderate. Severe flatfoot.
FOOT PRINTS.

Clinical signs.—As the name denotes, the foot is flat, coming in contact with the ground for almost the whole of its extent.

The foot spreads out due to stretching of the arches and therefore appears longer and broader. Its inner border instead of being concave may be convex.

The sustentaculum tale and the tubercle of the scaphoid are not well felt and the internal malleolus is unusually prominent. The muscles of the leg may be found flabby and weak and the movements of the foot are not easily performed by the patient.

Due to the increase in the length of the foot the tendons may be shortened and toe may be contracted.

The foot is usually found cold und perspiring. The patient walks in a slouching and slip-sod fashion when the foot is fairly deformed. Footprints give a great clue and so do the worn boots of the patients. The footprint will be seen to vary greatly from the normal and in the boots the instep

will be seen to bulge towards the sole, which is now away more on the inner side than on the outerside.

The following conditions are also caused by, or associated with, a flatfoot—(1) Hallux valgus; (2) Hallux rigidus; (3) Hallux flexus; and (3) Hammer toe.

Prevention of flatfoot.—Flatfoot can be prevented to a large extent. From the very start when the children are young and learning to walk they should be taught to walk properly with toes pointing forwards and not outwards. In those, where a tendency towards flatfoot is observed properly fitting shoes should be used. These subjects should be taught to stand on tip toes as much as possible. If much standing is required then the optimum position is to stand deliberately on the outer edge of the feet.

Exercises.—Certain exercises also prevent the flatfoot and cure it in early stages; a few of these useful exercises are given below :—

(1) Rise up and down on tip toes, with toes touching and heels separated and feet turned in.

(2) In excrcise No. 1 when on tiptoes bend knees and squat on the heels. Rise up again without heels touching the ground.

(3) Sit on a chair with one knee crossed over the other. Rotate and circumduct the ankle to its fullest possible degree.

(4) While standing with the feet parellel lift the.inner borders of the feet together off the ground and walk on the outer side of the foot.

These excerises aim at strengthening the ligaments and muscles responsible for keeping up the arches of the feet.

Treatment.—For the purpose of treatment the flat foot may clinically be divided upto four degrees :—

First degree—when the arch reappears and deformity disappears if the patient takes off weight or stands on tiptoe.

Second degree—in which the deformity can be corrected by surgical manipulations.

Third degree—when the deformity can be corrected only under an anæsthetic.

Fourth dergee—when anklyosis and body deformity have occurred os that the deformity can only be corrected by operation.

As already referred before, while discussing the symptoms of flatfoot that pain and disabality are worst felt in the early stages of flatfoot. Pain is sometimes so severe that it is sometimes called as acute flatfoot. The first thing required is rest for a few days; legs and feet can be given light massage and general health duly attended. This will enable the ligaments and muscles to recover their power and tone. When pain and tenderness has passed off or in milder casse where they are less, the patient must be instructed properly with regard to excercises already referred to. Long standing and long walking ought to be avoided for sometime. He should learn to rest occasionally on the outer edge of the feet and upon the tiptoes. In walking he should keep his feet parallel and rise well upon the balls of his toes.

In the *second degree* the treatment is similar. It is advisable in this stage to place a metal plate to support the arch of the foot *e.g.* Whitman's spring. To fit this satisfactorily the metal spring must be moulded on a plaster cast while the foot is in its proper position. If more support is required an outside iron to the knee with a valgus T starp is fitted.

In the *third degree* the deformity will have to be rectified forcibly under an anæsthetic and the arch restored. Thomas' wrench may have to be employed. Foot should be moved in all the directions to break the adhesions. The foot is placed in plaster of Paris for six to eight weeks, after which properly fitting boots are given and then the treatment becomes that of first degree. In some cases tenotomy may also be performed before or after the forcible rectification.

FIG. 3

A metal spring suitable to the arch of the particular foot when fitted inside the boots with the metal spring inside under the instep the foot adopts a normal shape.

In the *fourth stage* the symptoms can be relieved by carefully fitting an instep pad. Excision of the dislocated head of talus may have to be done. In some cases other complicated and elaborate procedures may have to be resorted. This should be followed by fixing the feet in plaster of Paris, excercise and massage.

In spasmodic variety—if the rest does not cure correction should be done under an anæsthetic. Tenotomy of peroenal tendons and crushing of the peroneal nerve have also been recommended.

The paralytic variety may be treated by tendon transplantation, tendon fixation or artherodesis of the joints.

In inflammatory form rest and specific or non-specific therapy should be carried out.

In traumatic form it always defends on the extent of damage done. In mild cases rest and Faradic baths may cure, and in severe cases œsteotomy of bones of the legs may have to be done.

Summary.—The common deformity of flatfoot or slackening of the arch of the foot is caused in various ways. It gives discomfort in continued walking or standing and makes the gait look coarse and ugly. Flatfoot can be prevented to a large extent by the knowledge of right methods of walking and by doing proper exercises to keep up the tone of the muscle and ligaments. The treatment of flatfoot essentially aims to convert all the severe degrees into the first degree.

Perforation of Hard Palate by Maggots

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G., a Mohammadan, male, aged 44, of filthy habits was brought to the dispensary for the treatment of offensive discharge from the nose.

Previous history.—Duration 12 days. The illness began with nasal catarrh. The discharge was watery for the first four days. It then became thicker and finally mucus and blood were discharged. The attendants were frightened at the sight of blood and probably this made them attend the dispensary.

General condition.—The man was fairly built, though of filthy habits, no history of having suffered from syphilis, tuberculosis or any other specific disease. No history of traumatism. Solid food could not be taken owing to perforation of the hard palate, milk or water had to be taken with the help of another man by lying down with head thrown well back. This partly escaped through the nose and partly swallowed.

Signs and symptoms.—The fluid taken into the mouth was escaping through the nostrils. Articulation was so indistinct that it was impossible to understand what he said. The intonation of voice was peculiar. Offensive smell from the nose. Pain over the eyebrows. Intense feeling of indisposition. Feeling of stuffiness in the nose. Temp. 100·2°. The patient seemed to be acutely ill.

On examination of the mouth the hard palate was found to be perforated. The aperture was of the dimension of an eight-anna piece situated anteriorly.

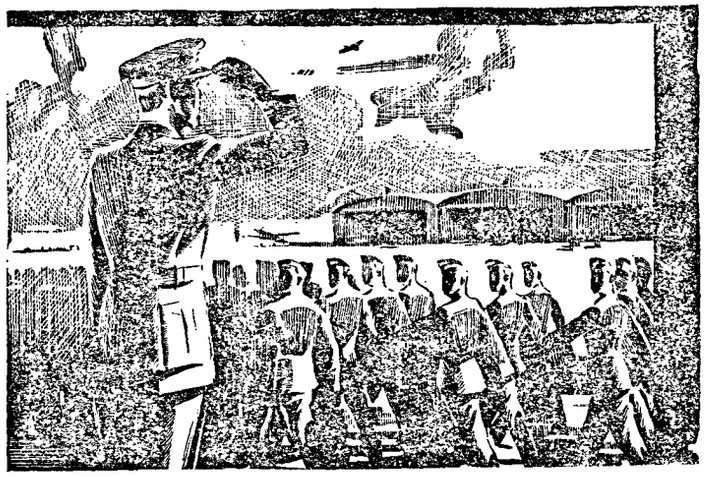
Treatment.—After general cleansing of the face, the nose was douched with warm Condy's lotion, Chloroform Turpentine lotion (Oleum Turpentine ʒ i; Chloroform ʒ i; Aqua Distil ad ℥ i.

Plugs were inserted in both the nostrils and the patient was kept under observation. After an hour the plugs were removed and the nose was again douched. Another plug of the same lotion was inserted in one nostril only. The patient was asked to remove this plug after 3 hours and the nostrils be plugged alternately after every three hours. Warm weak Condy's lotion gargles frequently. The condition remained the same.

Next day when the nose was being douched 6 maggots, both dead and alive, were discharged and in 24 hours about 1½ dozen maggots, alive and dead, were discharged. For 5 days the discharge of maggots continued but their number went on decreasing every day. The discharge of mucus and blood also ceased after the cavity was cleared of maggots. On the 7th day he was given Cibazol orally two tablets t.d.s. for 2 days. He was also given 2 ampoules 2 × 5 c.c.—5% of Urea Sulphazide (U.D.C.) intramuscularly.

Further treatment was stopped except warm weak Condy's gargles 4 times a day for about a fortnight.

The patient was seen by me a few days after enjoying sound health but two troubles persisted, one the intonation of the voice and the other escape of fluid from the nostrils but both these troubles did not seem to affect his health much. The aperture in the hard palate coalesced after



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about six months though the union is not bony but muscular and the escape of fluid was also checked. His articulation became distinct and he felt comfortable as ever.

POINTS OF INTEREST:—1. The perforation of hard palate by maggots is not mentioned in text books.

2. Innumerable live maggots were discharged from day to day. How they gained their foothold, multiplied and caused perforation is really strange.

3. The live maggots remained in the nasal cavity for about 2 weeks without doing any other serious harm except the perforation of the hard palate.

4. The perforation coalesced automatically after the nasal cavity was cleared of maggots and stoppage of discharge without any operation. No plate obturator or sheet of India rubber was required over the aperture.

5. The patient is enjoying sound health. The aperture coalesced completely though the union is not bony but muscular. There is neither any escape of fluid through the nasal cavity nor any intonation of the voice now.

I am highly thankful to our worthy D. M. O. H. Karnal, Dr. WAZIR SINGH, D.P.H., for his kind permission to send the above case notes for publication.

A Case of Miliary Tuberculosis

K. P. S. NAMBIAR, L.M.P.,
Cantonment Hospital, Wellington.

MILIARY tuberculosis though a comparatively rare condition in general practice has to be kept in view when dealing with a case of undiagnosed fever, especially in children. The following case was first thought to be one of malaria and then enteric until the X-ray examination of the chest disclosed the real nature of the illness.

Master V, student, aged about 18, was admitted in the Cantonment Hospital, Wellington, on 12-8-1946 complaining of fever and loss of appetite for the past nearly a fortnight. He had malaria about a year ago but no other illness of any importance. Family history was irrelevant.

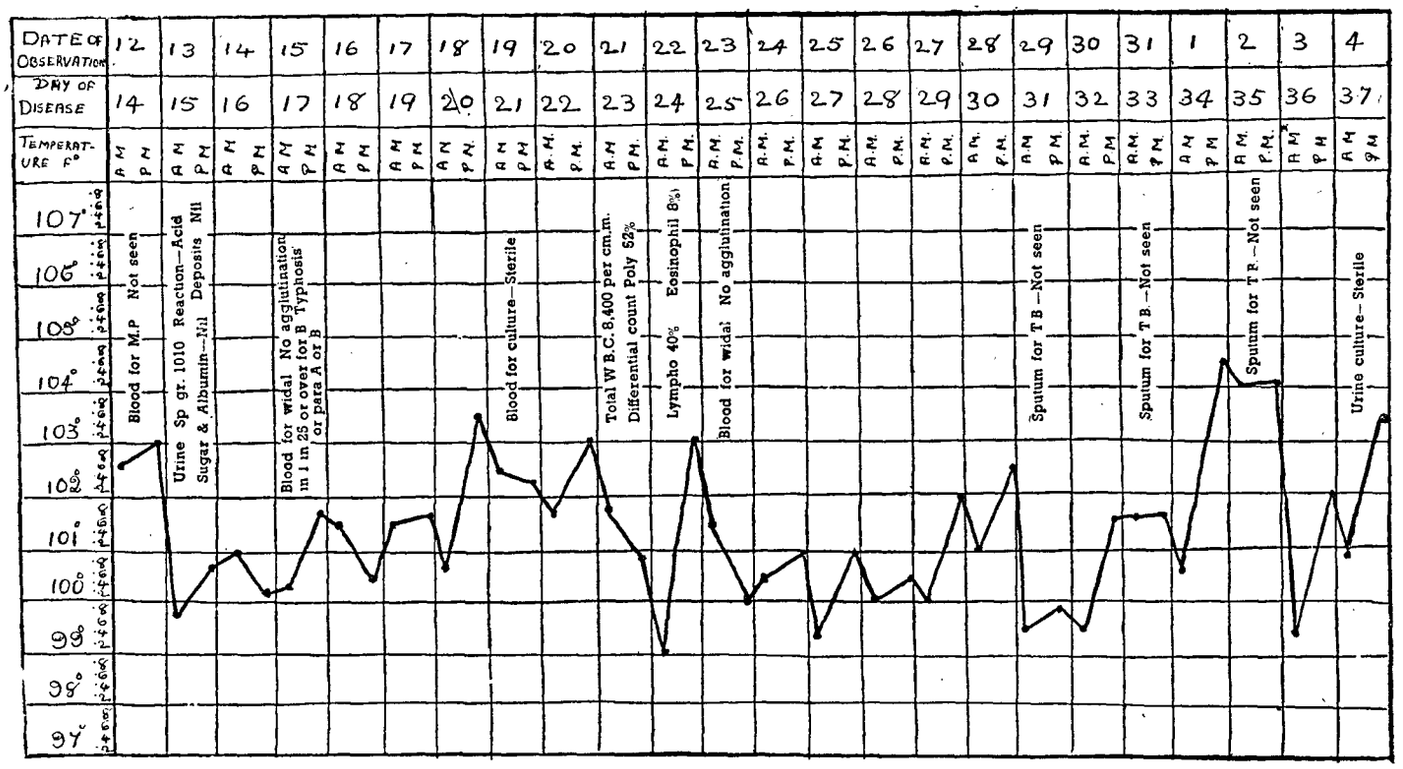
On examination.—Patient fairly well nourished, looked more ill than what the temperature suggested, tongue clean, bowels regular, spleen enlarged to 2 fingers breadth below costal margin and slightly tender, liver not palpable, heart and lungs nil abnormal detected. Blood slides for malarial parasites were negative but in view of the enlarged spleen and old history of malaria, the patient was put on anti-malarial treatment. The temperature continued to be quite irregular usually swinging between 100° and 103°F. Pulse and respiration were comparatively rapid. Total W.B.C. 8400 per cm.m. and differential count polymorphs 52% lymphocytes 40% and eosinophiles 8%. Widal and Weil Felix were repeatedly negative. Blood culture sterile. Urine showed slight albumin but culture was sterile. Sputum was examined several times with negative results.

The condition of the patient gradually deteriorated. His appetite was poor, abdomen slightly tender and there was tendency to diarrhœa. His hearing was defective and he seemed to lose interest in his surroundings. His chest which was clear on admission showed evidence of generalised

CLINICAL CHART I

CANTT. HOSPITAL AT WELLINGTON

Name: Vincent. Age 18. *Disease*: Miliary Tuberculosis. *Date of Admission*: 12-8-'46.
Date of Discharge: 28-9-'46. *Result*: Discharged otherwise.



CLINICAL CHART II

CANTT. HOSPITAL AT WELLINGTON

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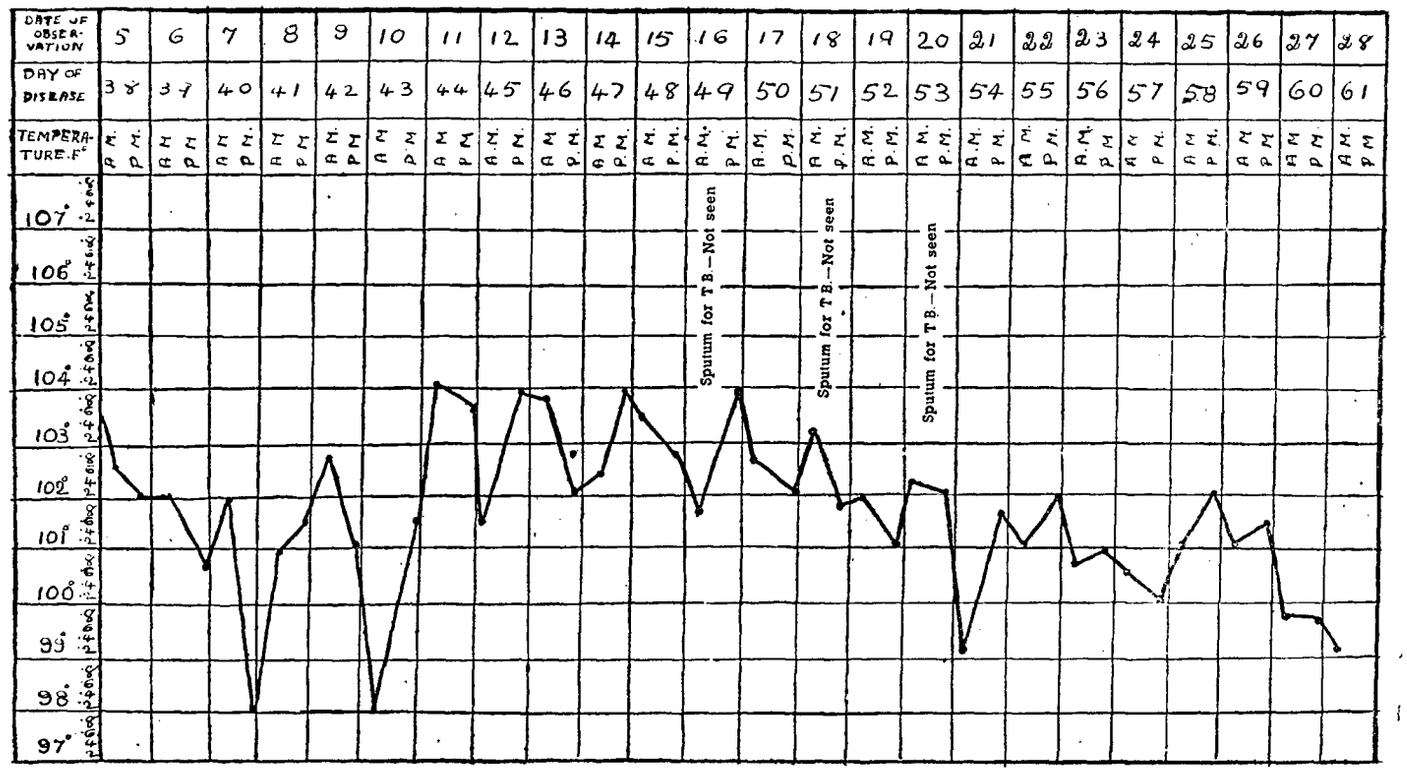
Date of Discharge: 28-9-'46. *Result*: Discharged otherwise.



bronchitis. An X-ray at this state 4 weeks after admission showed that both lungs were thoroughly infiltrated with small ill-defined foci of consolidation which were all of same size and uniformly distributed all over.

The usual 3 conditions which can bring about an X-ray condition similar to this are: (1) Eosinophilic lung, (2) anthracosis; and (3) miliary tuberculosis. Number 1 is excluded by the absence of leucocytosis and the comparatively low eosinophilia in the differential count. Number 2 is essentially an occupational disease and hence can be ruled out. The severe toxæmia, enlarged spleen, the negative blood and urine cultures and agglutination tests together with the vague physical signs iu the chest and X-ray findings were definitely in favour of the diagnosis of miliary tuberculosis though sputum examination till the last was negative.

Meanwhile the condition of the patient gradually became worse and his anxious parents removed him from hospital on 28-10-'46 to his house where he died 2 days later.

I am deeply indebted to Capt. E. G. H. KOENIGSFELD, M.D., Medical Officer, Cantonment Hospital, Wellington for his valuable help and advice in treating the case as well as in writing up this report. I must also express my thanks to Col. M. L. AHUJA, M.D., D.P.H. Director, Pasteur Insititute, Coonoor, for the laboratory investigations.

Psycho-Therapy in Neurotic Cases

HARIDAS DEY, L.M.F.,
Late Senior House Physician, Campbell Hospital, Calcutta,
Medical Officer, Chalmohar Charitable Dispensary and Auxiliary Govt. Hospital, Pabna.

I READ with much interest the article "Magical treatment of the psycho-neurotic cases" written by my fellow brother DR. KAMAL KRISHNACHATTERJEE, L.M.F., in the Dec. '46 issue of the popular medical journal, the *Antiseptic.* I have had also occasions to treat such cases and I venture today to publish two of my such case notes with the idea that my readers will find some sort of interest in them.

Case 1.—A lady, aged about 45 years, wife of a weaver of Pabna Dist. of Bengal, mother of 5 children, having been tried of spining thread from cotton asked her husband to take her to Calcutta as she wanted rest in life. Her eldest son 30 years old told his mother that she should not worry about spining thread anymore in life as his wife would now spin and he took her mother to Calcutta to take rest in a relation's house residing in a Bustee of Calcutta. One day while that lady was walking with her son at some part of Calcutta she noticed some big buldings containing cotton, when the mother asked her son with curioisty what the big buildings contained. Her son in good faith told his mother that those were cotton packed up in storage in these buildings. At this all on a sudden her mother told, "still I am to spin thread of so much cotton wool. Then I shall never get rest in life". Her son asked his mother why she was telling that. But his mother did not reply to his word and fell on the footpath and fainted. Her son cried out for help when men came at the spot and his mother was then sent to a hospital by an ambulance called by some kindhearted person. After few days, his mother was discharged from the hospital and her son brought his mother back home. After coming home his mother became dull and did not speak to anybody. She only ate and used to sit alone idle murmering something. The husband of that

lady called some unpassed doctors who treated her but the mental condition of the lady did not improve. She was then put under some "*Ojhas*" or "*Mahats*" with the idea that she was over powered by some ghost. But all the best attempts of *Ojhas* or *Mahats* proved abortive. One day I was going to see a case of mine by the side of the house of that lady when her husband called me in and requested to treat her wife. I examined her thoroughly but could not find any abnormality anywhere. I was rather puzzled to diagnose the case and told her husband that the case was very serious and I would come next day to see her with my books. After returning home, I consulted many books but found no clue for the diagnosis of the case. However with a few books I again went to see her next morning and watched her carefully. I put her many questions but she could not give me any answer. She looked towards me with a dull apathetic face. After an hour I suddenly heard that with a deep sigh that lady was murmering "still I am to spin thread of so much cotton. I will take whole of my life to spin it". I heard it distinctly and asked her relations why she was murmering that. Her eldest son gave me the history in detail about the Calcutta incident.

From the tale by her eldest son I came to a definite conclusion that at the sight of huge cotton in a cotton godown his mother fainted for which she was taken to hospital and treated for shock and collapse and got cured and was discharged from the hospital and after that the lady became apathetic and dull and spoke to nobody. From the history of the incident and absence of any positive signs and symptoms of any disease I came to the conclusion that the patient was an eurotic one. To gain her confidence and the confidence of the relations I told her relations that the patient must be cured by few injections though the case seemed to be a serious one as my previous physicians and '*Ojhas*' had failed to cure her despite their best attempts. I injected her 2 c.c. redistilled water intramuscularly and asked her son to come to my dispensary to take medicine along with his father. When her eldest son and the husband of the lady came to my dispensary to take medicine, I gave them Rose Syrup water 4 doses and advised them to give to the patient every 4 hours. I further told them that when I would go to see the patient next morning, they would say that they also have heard what I would tell them, otherwise the patient won't be cured. Next day I with my daily *Armita Bazar Patrika* went to see the patient and while examining the patient told her husband and son that "Have you heard or read in newspaper that all cotton godowns of Calcutta were gutted by fire." They also told that, yes, they have heard from a Pradhan of their village that all cotton godowns of Calcutta were completely gutted as the news had been published in newspaper. At this all on a sudden that patient called out "what did you say". I then told her boldly that all the cotton godowns of Calcutta had been burnt out by fire. She again asked me in anxious mood. "Is it true". I told her that it was true that all cotton godowns had been burnt to ashes. She then with a sigh of great relief told me "I am saved". Henceforth she answered all questions and narrated herself how at the sight of such a huge stock of cotton in godown, suddenly she felt uneasiness in the head and after that she could not remember anything. I again injected her 2 c.c. of redistilled water and returned back home with cheerful mind. After that her cure was uneventful and she is quite O.K. at present and taking Livogen (B D H) as she had become very weak. Her husband also gave me good remuneration and is ever grateful to me.

Case II.—One female, Hindu, aged 26 years came to me that a 'kara', a kind of insect entered her right ear while she was asleep at night and built nest inside the skull and so she is always hearing noise and feeling pain in the head. I examined her ears very carefully and found no abnormality anywhere. I thought it was simply a neurotic case. I asked her to come to me next day to my dispensary. When she came I caught hold of a 'kara' and held it with a forcep and kept the forcep *in secret* with my compounder. When she showed me her ear, I posing to examiue her ear very carefully told that there was a 'kara' inside the right ear. I asked her to keep quiet and shut her eyes. I then put another forceps inside the ear and after sometime, I drew out the forcep and showed her the "kara" caught by the forceps which I kept with my compounder. She was very glad and from that day she never heard any noise nor did she feel any pain in her head. She is quite O.K.

A New Cure for Opium Poisoning

RATILAL K. MODI, L.M.P.,
Assistant Chief Medical Offieer, State Hospital, Jaora State, C.I.

On the 16th November '46 a young Hindu, aged 30 years, was brought to the hospital by his relatives, suspected to have taken a big dose of opium at about 7 p.m.

This man was working as a cashier in one of the Marwari's shops. In addition to his duties, he was given to speculation, in which he had lost a considerable amount of money and had got into heavy debt. It was therefore surprised that on account of getting into debt which he could ill afford to repay, that he thought of ending his life by taking opium.

When he was admitted into the hospital, he showed the following symptoms:—Lethargic condition. Drowsiness. Responding to questions when they were shouted in his ears. Pupils contracted to pin-points. Insensible to light. Smell of opium present in his breath. Pulse quick at 120 per minute. Breathing deep and shallow.

Immediately after his admission into the hospital, his stomach was washed out with a weak solution of Potassium Permanganate 3 or 4 times. During this operation his breathing was becoming slow and laboured. He was now completely in comatose condition—the corneal reflex was absent. At this juncture, one ampule of Coramin plus Atropine was injected.

I waited watching further development of symptoms and the type of turn he might take. After half an hour, *i.e.*, at about 8 p.m., signs of failure of breathing and cynosis supervened though the heart continued to beat very fast. At once I began to give him artificial respiration and started giving oxygen inhalation also. I gave him another injection of Coramin 5 c.c. in the gluteal region and continued artificial breathing for 45 minutes with oxygen inhalation; but there was no sign of normal restoration of breathing.

I lost all hopes of his coming round. He was lying like a dead person, with eyes half open—no corneal reflex, legs completely immobile, heart continuing to beat at such a speed that rendered beats imperceptible.

Thinking the case to be entirely hopeless, as a last resort, I thought of giving him Lexin injection (a snake-bite cure) of Great Bengal Pharmacy Mihijam, E.I. Rly. I remembered having read about its use in opium poisoning though I had never had occasion to try it before. I injected one 2 c.c ampoule of Lexin (intravenous) slowly, while artificial respiration was being continued all the time. After 2 or 3 minutes of Lexin injection, to my surprise and of all those prerent, the patient opened his eyes and cried for water, while only a few minutes ago, I had given up all hopes of his recovery. A little water was poured into his mouth. Gradually his breathing was restored to normal. Oxygen was stopped and within 10-15 minutes of Lexin injection he regained his memory. He began to recognize his relatives and friends who were assembled round him. The expression of his face became quite normal.

Personally I felt as if God had descended from heaven and infused new life into the dead body which lay before us motionless and without breathing for an hour on the table.

Encouraged by the wonderful effect which one ampoule of Lexin had produced, I administered half an hour later, another dose of Lexin, this time subcutaneously. I was struck with wonder to see the magic effect of Lexin within 2-3 minutes in a completely lost case of opium poisoning. As the pulse was slow and weak I gave another injection of 5 c.c. of Coramin in the gluteal region at 10 p.m. After this the condition of the patient improved considerably. At 11 p.m. I left the patient leaving instructions to his attendants to keep him awake by flicking a wet towel on his face or by making him walk about well-supported. Next morning when I saw him, he looked perfectly sober and went home quite cheerful.

The purpose of my writing about this case, is to find out whether any of my brothers-in-profession have had an experience of using this drug (Lexin) in cases of opium poisoning; if not I would like to request them to give it a trial, and let me know personally and through *Antiseptic* the results.

Many ignorant and illiterate mothers, especially of the labouring class, are in the habit of giving opium to their children. Sometimes they give them a heavy dose in order to put them to sleep for a long period, and very often such doses of opium have proved to be fatal. When the parents are asked, they say that while playing the child accidentally got hold of the box containing opium and put the contents into its mouth. In such cases if 1 c.c. of Lexin is injected immediately sub-cutaneously, and repeated as necessary, in my opinion, many deaths can be averted—Experience alone will prove it.

An Interesting Case of Malingering

DR. B. L. SHARMA,
C.M.O., Sitamau, C.I.

Medically or surgically there is nothing important or interesting in this article. I simply write this in the interest of junior M.Os., who in dispensaries have to deal single-handed with medico-legal cases who seldom miss chance to bluff. In the jail, police and army, I have seen expert malingerers feigning—galloping T.B. with hæmoptysis, paralysis due to fracture of spine due to fall from horse back, snake bite, etc. Cases feigning total blindness and deafness are common. The snake bite case was so complete that I was easily bluffed in the beginning and had to inject Antivene. But the case of

this article - being of a girl with little experience of the wide world seems more interesting.

Once at 8 p.m., a constable brought an urgent letter to me that in a village scuffling took place between two women and the one who received more hammering was lying unconscious in a precarious condition and my presence was urgently required there. I at once started on a camel back and reached the village, 8 miles off at 11 p.m.

What I found and saw.—A woman, aged 18, robust and healthy. married, was lying flat on the back on a cot in the courtyard of her small house with closed eyes, speechless and still with stiff body (apparently quite unconscious).

She was said to be in such a condition since 4 p.m., when the scuffling took place.

The other woman who was alleged to have inflicted the injuries was the next door neighbour-aged 50, very weak and crippled.

The weapon used in the fight was—fist blow, used 4—5 times only. The part which received the fist-blows, was the back only.

The only family members present in the house and in attendance on the injured girl were old father and mother-in-law, who were bitterly weeping and crying for their faithful daughter-in-law whom they thought to be unrecoverable and dying.

Corneal reflex was present, pupils—normal, breathing quite natural and normal, pulse, heart and lungs—all normal.

All my efforts and tacts failed to bring the girl round to her senses as well as to pacify the old in-laws. Now the problem before me was whether a healthy and strong girl of 18 could be made so unconscious under 4-5 fist blows from a weak and crippled woman who could hardly stand and walk erect and specially when the blows were given on the back only and not on any vital part and with the force leaving no mark of any injury—redness, swelling,etc. I tackled the abdomen, soles of feet, armpits of the girl, so much so that I thrust my thumb nail under her thumb nail, but failed to get any response—even winking of eye-lids or twitching of any face muscle could not be seen. Yet my innerself denied to accept her as a genuine case of unconsciousness due to the injuries. I also resolved firmly not to be easily bluffed and defeated by the young girl malingering under support and guidance of the worn and weather beaten old foxy in-laws. Hence I determined to try the last drastic tact. I asked the in-laws to give me a cup of milk and get out of the house. The police was also put behind the court yard wall about a couple of yards from the girl and me, from where they could see us without being seen. Thus when apparently alone with the girl, I put my right hand on her one leg and spoke thus firmly into her ear: "Come to senses at once, speak and open the eyes or I push my hand up to the extreme." With these words I also gave to and fro rapid movements to my hand as if really pushing up.

Before I myself could realise the magic effect of my tact, I heard a roar of laughter from the police, because the girl was sitting erect on bed with gasping mouth and wide open eyes-staring at me furiously. I gave her the cup of milk which she gulped easily in one draught and then narrated her whole history and complaints to me. The old in-laws were called in, and after handing over the charge of their dear daughter in-law, with full satisfaction of her safe recovery and of full justice in the Court of Law, I left the village and reached the dispensary at dawn, quite tired and sleepy but with one interesting addition to the stock of my experience.

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A Century of Progress

IN October of last year the world celebrated the centenary of the advent of surgical anæsthesia. In England it was celebrated by exhibitions at the Royal College of Surgeons and at the Welcome Historical Medical Museum illustrating the history of anæsthesia through the ages; by celebrations organised by the Society of Anæsthetists; by meetings of the Royal Society of Medicine; and by our distinguished contemporary the *British Medical Journal*, bringing out a special number on the subject. From its pages one can well learn the development of anæsthesia. We are told that the Assyrians and Egyptians used pressure on the carotid arteries to secure temporary loss of consciousness. The Egyptians were also said to have used the fumes of Indian hemp to stupefy the patient before operations. The Chinese were said to have used wine of unknown composition as an anæsthetic. The Arabian practitioners were supposed to have used certain plant infusions for this purpose. In the middle ages most of these anæsthetics were administered by mouth. Later alcohol, opium and other narcotic beverages were used. But in all these, though success was achieved in certain cases, there could be no guarantee that the method would be effective. In certain countries mesmerism and hypnotism were also used for producing sleep and preventing pain during operations. But all these were not true inhalation anæsthesia. Medicine had to await the development of chemistry before any true anæsthesia could be thought of. It is said that the works of JOSEPH BLACK and JOSEPH PRIESTLEY paved the way for these new discoveries. SIR HEMPHRY DAVY's experiment of breathing pure Nitrous Oxide could really be said to have been the starting point in the inhalation anæsthesia. To CRAWFORD LONG, HORACE WILLS and W. T. G. MORTON must, however, it is said, go the credit for the real advance in inhalation anæsthesia. It was in 1846 that the last named MR. MORTON gave his historic exhibition of inhalation anæsthesia at Boston. LISTON was the first to do a major operation in England under general anæsthesia. JAMES SIMPSON of Edinburgh was however the first to use Ether as our anæsthetic in obstetric practice.

As our distinguished contemporary, the *British Medical Journal*, puts it in its editorial, the subsequent history of anæsthesia was dominated by the

Ether-chloroform controversy. A lot of progress has also been made in the apparatus used for the production of anæsthesia by this inhalation method.

In a very learned article in the Special Number of the *British Medical Journal* mentioned above on the subject of recent advances in anæsthesia LANGTON HEWER discusses briefly the advances made in this speciality during the last quarter of a century.

In the beginning of this period pre-operative starvation and purging were common. Premedication was confined to subcutaneous Morphine and Atropine administration, basal narcosis being unknown. The anæsthetiess in common use were Nitrous Oxide, Chloroform, Ether and Ethyl Chloride. Short procedures such as dental extractions were done under Nitrous Oxide. For minor operations in children the usual anæsthetic was Ethyl Chloride. For major surgery the two commonest standard techniques were the Nitrous Oxide—Ether—Chloroform sequence and the Nitrous Oxide—Ether—open ether. Inhalation with a face piece was the predominant technique, although the insufflation endotracheal method had been developed for plastic repair of facial wounds. Local analgesia was practised considerably and Cocaine was used as a surface analgesic and Procaine for injection purposes. Spinal analgesia was confined to hyperboric solutions usually of Stovaine or Tropacocaine.

Things have considerably changed in recent years. The preparation of the patient was directed towards putting him in the best possible condition immediately before operation. It may include the administration of extra Glucose or possibly a blood transfusion. Purging was reduced to the minimum. Premedication was no longer a routine. It became selected and was calculated to each patient. Sedation was achieved with a Morphine derivative and Scopotamine or with an oral barbiturate. Complete basal narcosis was indicated for highly strong and nervous patients. Rectal paraldehyde for children and Avertin for adults have been found to be useful. Various new volatile anæsthetics have been introduced since the last war—Ethylene, Acetylene, Propylene and Cyclopropane. Of these the last Cyclopropane has gained popularity. It is potent and non-irritating. But it is costly. Certain ethers besides the usual diethyl variety have been investigated but the only one to be popular is the divinyl ether. Trichlorethylene is said to be another volatile agent that is in general use.

Local analgesic techniques have advanced a great deal to cover a variety of field and nerve blocks. Very many new drugs have been tried as local analgesia, but, apart from the original Cocaine and Procaine, Nupercaine and Amethocaine have attained any real popularity. The refrigeration of limbs has proved useful for amputations in patients in very poor condition. The tissues in this case are simply cooled to about 5°C

Spinal analgesia has undergone many changes. The mechanism of distribution of the injected drugs is now more fully understood. The large number of serious complications that have been recorded even with the best technique has, in the opinion of LANGTON HEWER, been the chief cause of the growing unpopularity of this type of anæsthesia.

The introduction of Curare is perhaps the greatest advance in recent years in anæsthesia. The isolation of the pure Alkaloid Tubocurarine Chloride by King in 1935 has enabled the use of a preparation which is free from undesirable side action. It is a pure muscle relaxant and has been proved to be very useful in upper abdominal surgery, where it is given in divided doses by the intravenous route, the patient having been rendered unconscious by very light inhalation or intravenons anaesthesia.

There is no doubt that anaesthesia has become increasingly safe in the hands of experts. "Medicine's greatest gift to suffering humanity" has been a real boon to the sick and injured. The world must ever remember with gratitude the work of the pioneers.

Gleanings From Medical Press

MEDICINE and THERAPEUTICS

Penicillin in treatment of putrid lung abscess.—Stivelman and Kavee administered penicillin to 13 patients with putrid lung abscesses. Six of these patients, 3 men between the ages of 21 and 49 years and 3 women between the ages of 28 and 37, had an acute putrid lung abscess, and 7 patients had a chronic putrid lung abscess. Twenty-five thousand units of sodium salt of penicillin was given intramuscularly every three hours. Sulfadiazine was employed concurrently, although used by itself it had no appreciable effect on a small series of patients so treated. All patients with acute putrid lung abscess became free from local and constitutional symptoms. Improvement in weight and strength was evident. In 5 patients there was a complete recovery from the disease as judged from the rœntgenograms. In one a previously observed encapsulated exudate remained symptom free and unchanged. In 5 of the patients with chronic putrid lung abscess the treatment with penicillin and sulfadiazine was followed by decided improvement of the toxic manifestations. Serial chest rœntgenograms revealed definite regression of the lesion in 2 patients and pronounced improvement in 1. There were no septic or metastatic complications while under treatment and all 7 patients became better surgical risks after penicillin-sulfadiazine treatment than they were before the treatment was instituted. In chronic putrid abscess this method of treatment should be started preparatory to surgical intervention and continued after operation until all toxic symptoms disappear.—*J.A.M.A.*

Gonococcic peritonitis.—Stanley reports 3 cases of gonococci perihepatitis, all of which were treated with penicillin, with prompt recovery. Women in whom the syndrome develops invariably have had an antecedent gonococci infection, sometimes as long as five years before. The onset is usually dramatic, with a sudden excruciating pain in the upper part of the abdomen, often referred to the shoulder. It is of the pleuritic type, exaggerated by deep breathing, coughing or laughing or by bending or walking. Trauma of inflamed pelvic organs, in the form of surgical procedures, gynecological examination, intercourse or douching, may precipitate the development of this complication. Culture of the cervical or urethral secretions yields gonococci. Gonococcus vaccine, hyperpyrexia and the various sulfonamide compounds have been used with varying success in the past, but at present penicillin is the drug of choice. Doses of 20,000 units administered every three hours intramuscularly usually relieve the symptoms completely within twenty-four hours. The disease occurs more frequently than is generally believed and should be considered in the differential diagnosis of young women in whom an acute pain develops in the upper part of the abdomen, particulary on the right side. It is often misdiagnosed as acute cholecystitis. The importance of correct diagnosis is obvious, since surgical intervention not only is not necessary but may be harmful.—*J.A.M.A.*

Anemias associated with nutritional disorders.—All morphologic types of anemia are found in patients with nutritive failure. Some of these result from an inadequate intake in specific nutrients. A deficiency in extrinsic factor results in macrocytic anemia, of iron in microcytic anemia, and occasionally of ascorbic acid or other factors in normocytic anemia. Among patients with microcytic anemia Vilter and Spies detected a considerable number, all female, who seemed to have the Plummer-Vinson syndrome, or sideropenic dysphagia. These patients, in addition to evident pallor, complained of burning of the tongue and difficulty in swallowing. Some had been grinding their food for years. The tongue usually showed atrophy of the papillae and appeared smooth, pale and glistening. Sometimes there was an associated cheilosis or angular stomatitis. Anorexia, flatulence and constipation were usual. Spoon nails were occasionally observed and also a spotty but coalescent brown pigmentation over the face. Fluoroscopic studies of the upper part of the esophagus frequently revealed a semilunar membrane partially obstructing the lumen and an

adjacent narrowing of the esophagus. Hypochlorhydria was usual in patients with anemia and dysphagia. There are two theories about this syndrome. On the one hand the dysphagia can so alter the dietary that there is iron deficiency and anemia develops; on the other, it is possible that the alterations in the esophagus are secondary to the anemia or the iron deficiency. Iron alone is effective in this microcytic, hypochromic anemia. Liver factor deficiency (macrocytic anemia) may be treated with liver extract or synthetic folic acid, iron deficiency with ferrous compounds and the anemia of scurvy with ascorbic acid. Therapeutic combinations of these specific substances for the treatment of anemias are economically wasteful and have been used only as the result of misconceived advertising and indifference in diagnosis.—*J.A.M.A.*

Effect of vitamine in coronary heart disease.—"While studying the effect of high-dosage vitamin E (a-tocopherol acetate) on purpura, the good influence of this factor upon coronary heart disease became apparent. A study of a series of cardiac patients, carried out with the help of Mr. Floid Skelton and Dr. Wilfrid Shute, has suggested: (*a*) vitamin E in large dosage (200-600 mgm. Ephynal-Hoffman-LaRoche) has no apparent effect upon normal hearts, even after administration for many months on end; (*b*) its effect upon patients having congestive heart disease and the anginal syndrome is marked; it increases exercise tolerance and diminishes or abolishes anginal pain during the period of its administration; its diuretic effect is pronounced.

"The effect of vitamin E upon coronary pain may be produced by a direct action on the coronary vessels or by influencing the metabolism of the heart muscle. The first possibility is suggested by an older observation on the effect of vitamin E in dilating the local capillaries in senile vulvitis. Our work on the purpuras raises another possibility too. Small hemorrhages into the walls of the coronaries or into the heart muscle itself may produce such pain. It is now clear that such extravasations may be either prevented or reabsorbed by means of vitamin E.

"When vitamin E was given in large doses over long periods of time, some patients complained of cardiac irrigularities. These were relieved by reducing the medication to low levels."—Vogelsang, A, and Schute, E. V.: Effect of vitamin E in coronary heart disease. Nature 157: 772, June 8, 1946.—(*Nab. Med. Bull.*)

Status thymico-lymphaticus. — Jesse L. Carr, J. *Pediat.* 27: 1 (July) 1945.—In reviewing 521 cases of sudden death of persons under the age of 12 years recorded in the San Francisco City and Country Corner's Office, all cases in which death could be ascribed to causes other than those associated with a thymus lymphatism, changes in the cortical regions of the adrenal glands or lymphatic infiltration around the bronchioles were discarded. This left 43 deaths distributed into three categories: 1, acute asphyxia from tracheal compression and distortion of the intrathoracic contents by a greatly enlarged thymus gland; 2, thymic hyperplasia and hypertrophy, with adrenal atrophy and insufficiency; 3, an asthmatic status which is characterized by a definite peribronchial lymphocytic infiltration, bronchial musculature of the hypertrophic type, mucous plugs within the bronchial channels and also a diffuse lymphatism of greater or lesser degree associated with changes of the thymus, usually hyperplastic in nature.

The term status thymicoasthmaticus is proposed as applying a proper anatomic and clinical term to sudden death associated with this type of anotomic and pathologic change which is believed to be congenital. These changes have been observed in babies taken in utero, as well as in those recently born and those who have lived several months.

Also tabulated and commented on were cases of sudden and dramatic involution of the thymic tissue, both cortical and medullary, in the presence of cachexia, inanition, starvation, electric shock and wasting diseases.

Sudden death in children resolves itself as a problem into many components. There is of course no one cause, and in our series of well over 500 autopsies on children dying suddenly, the problem of thymus enlargement is a secondary one. Strangely, however, enlargement of the thymus gland, lymphatism, hypoadrenalism and similar related conditions have been displaced even from secondary positions of importance as causes of disease and death and have been thrown into disrepute by articles in the medical literature which are based more on precedent and fashion than on observation.

The now popular attitude that there is no such thing as status thymicolymphaticus can be quickly dissipated by studies at autopsy in any corner's office where children are studied, and the widespread belief that an enlarged thymus in itself will not cause asphyxia by tracheal compression and distortion can be disproved at necropsy wherever studies are done on cases of sudden death in children.

On the other hand, when once accepted, status thymicolymphaticus and related terms become far too easy to use. Instead of being reserved for specific cases to which they are properly applied, such diagnoses are generally used to explain many sudden deaths in children for which there is no other obvious cause. It may be that such diagnostic abuses have in the main been responsible for the disrepute of the thymic status.

We now feel that orderly proceedings at necropsy will segregate the various causes of asphyxia in infants, asphyxia being by far the commonest cause of sudden death under the age of 2 years, and that such studies will in their natural evolution reestablish the relationship of enlarged thymus glands, lymphatism, adrenal hypoplasia and such conditions, either isolated or related, as veritable, tenable, direct and sole causes of death.—Author's Abstract.—*A. J. D. C.*

The management of diabetic accidosis and coma.—Leslie W. Swanson, M.D., Mason City.—A patient with uncomplicated but uncontrolled diabetes mellitus may have polydipsia, polyuria, polyphagia, weight loss, unusual fatiguability, loss of muscular strength, apprehensiveness, visual disturbances and pruritus. In a patient presenting a history of such symptoms, or one known to have diabetes, development of the following additional manifestations should lead to prompt consideration of acidosis: unusual weakness, muscular aching (sometimes erroneously ascribed to "influenza"), nausea, vomiting, Kussmaul respirations, mental dullness. flushed facies, acetone odour on the breath, restlessness or delirium, abdominal pain, dehydration, or actual coma.

Patients with uncontrolled diabetes will invariably present glycosuria and hyperglycemia, while those with acidosis will show in addition acetone in the urine, and frequently albumen and casts. Laboratory determination of reduced CO_2 combining power of the blood is a more exact method of diagnosis. The symptoms and signs of acidosis vary with the severity of the acidosis—the greater the loss of alkali reserve, the greater the tendency toward complete coma. A mild acidosis of short duration will often show nothing but acetonuria, whereas a mild one of long duration may have headache, muscle aching, weakness, and restlessness. An acute severe acidosis may produce complete coma with Kussmaul respirations and depress the carbon dioxide combining power of the blood to below twenty volumes per cent. Serve acidosis of longer duration may be accompanied by azotemia and even depletion of the sugar stores to such an extent that the blood sugar is only moderately elevated.

Consideration of the causes and antecedent circumstances of acidosis helps in the diagnosis and evaluation of the history and findings. Unrecognized diabetes will lead to acidosis if it is severe enough. However, a child may develop diabetes in such severe form that it leads very quickly to diabetic acidosis. A second cause and a common one is neglect of diabetes; that is, failure to follow a diet, failure to adjust properly the insulin dosage with the aid of urinalysis, or by refusal to take insulin. A third cause, seen commonly in practice, is the precipitation of acidosis by the development of an acute severe infection which throws the diabetic far out of control before he is fully aware of the seriousness of the situation. This is especially true when vomiting accompanies the infection.

The fundamentals of the treatment of acidosis are the correction of dehydration, restoration of adequate dextrose oxidation, and replacement of the electrolytes lost by excretion. Correction of dehydration may be accomplished by administering 3000 to 5000 cc. of fluid in the firsl twenty-four hours. If vomiting. coma, or aeute dilatation of the stomach is present to prevent oral administration, the initial portion may be given intravenously or subcutaneously. Normal saline or Ringer's solution will help to restore electrolyte balance; Hartman's sodium lactate solution may be used as well. Restoration of adequate dextrose oxidation involved administration of glucose and insulin. Those who consider acidosis to be an acute insulin insufficiency may attempt to calculate initially the total insulin need and after the injection of this dosage begin glucose administration.

Under ordinary circumstances regular or unmodified insulin is simpler to use because of its greater flexibility due to shorter action. If the patient has been taking protamine zinc insulin prior to the development of acidosis, however, it is usually advisable to continue the same dosage and supplement it with regular insulin as needed.

Mild acidosis usually can be corrected by the simple administration of glucose solution orally in the form of fruit juice, perhaps with added sugar. accompaniod by insulin in dosage estimated by ratio of approximately one unity of insulin to each two grams of glucose, as for example, 10 units for 200 cc. of orange juice. This may be repeated at intervals of one to two hours for perhaps six or eight hours until the acidosis disappears. However, the severely acidotic patient is likely to be vomiting, or if not vomiting he may fail to absorb fluids from oral administation because of an acute dilation of the stomach. In this instance it is advisable initially to give 1000 cc. of 10 per cent glucose solution in normal saline intravenously very slowly. This may be followed by isotonic glucose solution subcutaneously or per rectum. When given subcutaneously, 5 per cent glucose in distilled water is ordinarily tolerated quite well. Repeated intravenous injections may then be alternated with subcutaneous administration if necessary. Here again it is advisable to administer with each two grams of glucose one unit of insulin, which may be given subcutaneously rather than intravenously in the average case. From one hundred to three hundred grams of glucose given in this fashion per twenty-four hours, depending upon the severity of the acidosis, is ordinarily sufficient. Progressive doses may be varied according to the urgency of the situation based upon repeated blood sugar determinations and urinalyses.

Too much parenteral fluid must be avoided in order not to embarrass the cardiovascular system, but at the same time sufficient quantity should be given to provide the kidneys with adequate output. Ordinarily a safe maximum intravenous limit should be 2000 cc. per twenty-four hours, while two or three thousand more may be given safely subcutaneously if absorbed properly.

The advantages of this method of therapy include simplicity to the point of usability without complicated laboratory procedures.

It may be carried out in the home if necessary and controlled adequately by ordinary urinalyses for sugar and acetone.

If the patient presents an indication for immediate surgical treatment requiring general anesthesia, it is advisable to introduce at least 100 gr. of glucose into the patient before surgery and, if at all feasible, to achive freedom from acetonuria.

Following recovery from acidosis, the administration of large amounts of glucose for a few days is in order to help build up body stores of glycogen before rigorous regulation of the diabetes is begun. This helps avoid a period of instability which often follows acidosis and prolongs the time needed for regulation.

During the recovery period, a troublesome complication may be the rentention of nitrogenous waste products in the blood. One of our patients who gave no history suggessive of previous renal disease developed fatal uremia. This complication has been previously described as an extrarenal azotemia, probably the result of electrolyte imbalance induced by diminished alkali reserve. Prompt restoration of electrolyte balance and urrinary output appears to be the best preventive of such nitrogen retention.—*Jour. Iowa State Med. Soc.*, Dec, '46.

Primary atypical pneumonia.—Benjamin F. Wolverton, M.D., Cedar Rapids.

Recognizing that the term "primary atypical pneumonia" may apply to a group of diseases rather than to a single entity, the evidence in favor of a virus of viruses as a cause may be summarized as follows: The incubation period of about ten to twenty-six days is similar to that in other diseases known to be due to viruses. Viruses which caused pneumonia in susceptible animals have been obtained from nasopharyngeal washings and from the blood of patients. These viruses obtained by several workers in different parts of the world have differed from all known viruses, but in several instances they have resembled those of psittacosis, lymphocytic choriomeningitis and lymphogranuloma venereum. The temperature curve in some cases is biphasic. The pathologic changes in the lungs are similar to those in pneumonias of known virus origin. On the other hand, there has been a striking failure of all workers to find a virus consistently or to reproduce the disease in more than a few experimental animals. The disease in many respects resembles "Q" fever, which is due to Rickettsia, but those bodies have not been found in primary atypical pneumonia. Until further evidence accumulates, therefore, the question of causation by a specific virus or viruses remains unsettled.

The disease occurs both sporadically and in epidemics, the latter having occurred in many military installations. It affects males and females equally, and by far the highest incidence is among young adults. It occurs in all seasons, apparently being conveyed by contact. A succession of cases in a family or household permits a fairly accurate estimate of the incubation period as about ten to twenty-six days in contrast with one to two days for influenza.

The onset of the disease is usually insidious, the early symptoms being not unlike those of a common cold. Chilliness, headache, malaise, fever, and dry unproductive cough appear and increase during the first few days. The temperature may range as high as 104 and 105 F., there usually being an evening peak and a fall of two or three degrees in the morning. Profuse sweating is common during the febrile course of the disease, which may be as short as six days or as long as six weeks. The temperature may return to normal for a few hours or a day only to rise again. The headache and malaise are out of proportion to the objective appearance of illness of the patient.

The physical signs on the whole are inconspicuous as compared to the temperature chart. In the milder cases the patient looks only slightly ill. Dyspnea and tachypnea are absent or slight except in the most severe cases. Cyanosis is usually absent but does occur. The pharynx is usually not swollen and only mildly injected. Cervical lymphadenopathy is uncommon. There may be no abnormal physical signs in the chest during the first few days or at any time. Usually, however, a few crepitant rales are heard with or without one or more small areas of dullness. There may be suppression of breath sounds. The physical signs, if any, tend to increase as the disease progresses, and commonly they are much more marked after the fever has subsided than during its height. The appearance of diffuse sibilant rales throughout one or both lungs with or without fine and medium moist rales occurs frequently, usually after the first week. These rales, which are indistinguishable from those of bronchial asthma, occur in non-asthmatic individuals. The same finding was common in the pneumonias complicating influenza in 1918. Severe cases may show dyspnea, moderate tachypnea, and considerable cyanosis, requiring oxygen administration for a few days or a week or two. The pulse is slow, relative to the temperature, and early, but in the second and third week it tends to increase as the temperature comes down. Movement of the alae nasi is rare, as are herpes and abdominal distention. Occasionally the spleen is palpable for a day or two. There may be scanty mucoid sputum, increasing somewhat as the disease progresses, and it may be occasionally blood streaked.

The total leukocyte count is within the normal range in two-thirds of the cases. The remainder show a slight leukocytosis or a leukopenia. A definite leukocytosis with an increase in the neutrophils suggests secondary bacterial infection. There appears to be no tendency toward the development of anemia. As the disease progresses, the neutrophils and monocytes tend to decrease, while the lymphocytes and eosinophils increase. The late appearance of eosinophilia is of interest in view of the similar late appearance of asthmatic rales. The urine may show febrile albuminuria, but little else. Blood cultures are sterile in the absence of complicating bacteremia. The sedimentation rate of the erythrocytes is moderately increased. The X-ray changes are always much more striking and extensive than one would suspect from the physical signs and may furnish the only proof that pneumonia exists.

From the clinical standpoint complications are few, but with rœntgenologic aid more complications are apparent.

Treatment consists primarily of bed rest and adequate nursing care until the temperature has been normal for several days. Probably it is desirable not to let the patient up and about until all physical signs have disappeared, although several have been sent home with residual rales to complete their convalescence at home with no apparent ill effect. Steam inhalation may be of some benefit, particularly if laryngitis is present. Codeine is usually needed to ease the hacking cough. The sulfonamide drugs and penicillin are of no value unless secondary infection due to susceptible bacteria occurs. Serum or blood from patients convalescing from the disease have been used in a few cases with apparently good rsults. Aminophylline has been suggested by Roberts, oxygen by mask, nasal catheter, or tent is indicated is present.—*Jour. Iowa. St. Med. Soc.* Nov. '46.

Diuresis in congestive failure —By Mark D. Altchule, M.D., "*The Bulletin of the New England Medical Centre*", June, 1946.

In the diagnosis and treatment of heart disease, physicians are accustomed to examine the skin and lungs for œdema. However, examination may be misleading, for a considerable amount of œdema may be present without causing abnormal physical signs. The presence of œdema in the skin is rarely uncomfortable, but relatively small increases in fluid content of the lung may give rise to symptoms. The treatment of œdema in patients with heart disease is important not only for the restoration of swollen limbs to the normal diameter, but also for the control of dyspnœa.

The blood and tissue fluids in œdematous cardiac patients contain sodium in normal concentration, but the concentration of bicarbonate tends to be a little low, while that of chloride is correspondingly elevated. However, the total amounts of sodium and chloride in the body are greatly increased and are in proportion to the amount of œdema fluid present.

Possibly the simplest type of diuresis in patients with heart failure is that which follows the administration of digitalis. The drug restores most of the circulatory derangements of heart failure toward normal and its action includes an increase in renal blood flow. The urine volume is increased, and since the concentration of urinary chloride is elevated, the total excretion of chloride is very large. Base, chiefly sodium, is similarly excreted, but in lesser amounts. Because of the reduction in blood and tissue chlorides, the bicarbonate concentration increases.

A similarly simple diuresis is occasionally observed as a consequence of oxygen therapy.

Studies of the mercurial diuretics indicate that these drugs cause no primary change in blood flow or in blood volume nor do they mobilize salt or water from the tissues directly. Their one striking demonstrable action is inhibition of the reabsorption of chloride in the tubules of the kidneys.

The xanthine diuretics are believed to act by causing a local increase in blood flow in the kidneys. The chemical changes which occur following their administration, though less marked, resemble those caused by the injection of mercurials.

The importance of ammonium chloride as a diuretic in the treatment of heart disease has been exaggerated. Its acts by giving up a part of its ammonium radicle which is converted to urea, so that the body has to give up sodium in order to excrete the chloride thus made available. The diuretic effect is scarcely significant unless the daily dose is 5 Gm. or more, but in such amounts various harmful effects may occur. One reason why these harmful effects are not more commonly observed is because the enteric coating of the tablets prevents absorption of much or all of the drug. Large doses are useful when given for one or two days to patients who, because of lowering of the plasma chloride, have become irresponsive to the injection of mercurial diuretics. In such instances, 8 or 9 Gm. given daily for two days with mercurial diuretic being injected on the second day usually restores responsiveness.

Salt restriction may at times play an important part in diuresis. The effect of restriction of sodium chloride by itself cannot be expected to cause active diuresis in patients with heart disease; each Gram of sodium chloride retained in the body holds with it approximately 150 c.c. of water, so that reducing the salt intake from its normal level of 5 Gm. a day to 1·5 or 2 Gm. results in a maximum diuresis of only about 0·5 litres. On the other hand, restrictlon of salt does demonstrably retard the re-accumulation of œdema after diuresis. It should be borne in mind that lowering salt intake under 2 Gm. a day results in a most unappetising diet and one which of necessity must be deficient even in regard to nitrogen and thiamin The combination of a low salt intake with forcing fluids or 6 to 8 litres a day appears to be more efficacious in retarding œdema formation than salt restriction alone.

Thiamine is occasionally of value as a diuretic. Some patients who are unresponsive to the usual methods for the treatment of cardiac œdema, regain their responsiveness after receiving large amounts of thiamin parenterally.

It is clear from the above discussion that the treatment of cardiac œdema requires individualization rather than a routine approach.—*G.P.* Dec. 15, '46.

SURGERY

Fractures of the head of the radius (D. M. Meekison, in "*Journal of Bone and Joint Surgery*, Jan., 1945).—A damaged or tilted head is a detriment to the function of radiohumeral joint and should be removed. We therefore advocate excision within a week or so of injury; the excellent results justify the treatment when the patient is beyond the growth period. The old fracture, with organized transudate within the capsule, with limited movement, and with pain at the extremes of the arc of movement, is a real problem. One may correct the mechanical disadvantage of an irregular or eccentric head, but, so far as I am aware, there is no solution for the problem presented by changes that have taken place in the capsule.

Active movement is commenced as soon as possible following operation. Slings are not allowed. A good result may be anticipated in a fresh fracture in from 3 to 6 weeks. Our experience with 110 cases of fracture of the head of the radius led to the conclusion that all of them require surgical interference.—*G.P.*

Local implantation of gelatin in wounds.—Sinclair and Douglas (*Archives of Surgery*), have studied the problem of gelatin implantation in wounds in the Department of Surgery at Vanderbilt University since June, 1942. It appears that the implantation of both gelatin and the closely related collagen results in their utilization in experimental sutured wounds in such a way that the strength of these wounds is greatly increased over that of untreated control wounds. Application of gelatin to fresh,open wounds. from extractions of teeth and to soft parts of ulcers appears to hasten the process of fibroplasia and to produce more rapid, stable healing than occurs in similar wounds not so treated.

The experiments were of two types: In series one, wounds of equal length were made vertically on the backs of dogs in the middorsal region and later separately excised. In series two, similar wounds were made, but were arranged parallel to each other on opposite sides of the spine, so that a transverse strip of skin of measured constant width including both could be excised later. In series one, the wounds frosted with gelatin before being sutured were 33·9 per cent stronger than the controls at the end of one week. Too much gelatin, however, may act as a foreign body. In series two, 90 per cent of the gelatin-treated wounds easily withstood tensions which ruptured the control wounds.

There was no sign of irritation or reaction of the tissues to the gelatin implantation. In fact, the gelatin-treated wounds appeared to be drier and to heal with less signs of inflammation than nearby control wounds. Sulphonamide compounds and gelatin possess no chemical or physiologic incompatibility and have therefore been mixed and implanted advantageously in contaminated wounds.

Promising results have also been obtained with the use of powdered gelatin in the chronic leg ulcers. By the local implantation of gelatin in such wounds in scarred areas or in patients with hypoproteinæmia or lowered vitamin C reserves, intra-cellular substances may readily be supplied and therefore fibroplasia.—*G.P.*

Painful feet syndrome.—Page directs attention to a condition known as painful, electric, hot or happy feet among prisoners of war in the Far East. It is ushered in by severe burning and aching of the feet associated with hyperesthesia, raised skin temperature and vasomotor changes in the feet, with general body wasting. The condition was first seen from three to five months after the Japanese occupation in the various countries in the Far East where large prison camps were established. No definite cause could be attributed at first, but protein, fat or vitamin deficiencies were considered as possible causes. A factor contained in grain was regarded as important when it was observed that men fed for months on rice with a rich admixture of barley did not develop painful feet, whereas their comrades fed on a poor grade of frequently musty rice did. The syndrome appears to be the result of a deficiency of factors in the vitamin B complex closely allied to the PP factor but primarily affecting the peripheral arteries of the lower extremities, or it might possibly be due to a toxic factor in old musty polished rice associated with a low protein intake and reduced vitality. The appearances in the early stages are similar to those of erythromelalgia. It is not an angioneurosis and is not associated with a neuropathic diathesis. The disease is amenable to treatment by replacement of the deficient elements in the diet; otherwise it is rapidly progressive, ending in death or serious crippling deformities.—*J.A.M.A.*

Intravenous procraine hydrochloride in surgery.—Berthelemy (in *Progress Medical*, Paris, 73, Dec. 10, 1945), reports the results of intravenous injections of procaine hydrochloride in 80 cases of frostbite of the foot in the French army during the winter campaign of 1944. Seventy-five of the cases were mild, while the remaining 5 were considered grave. In the 5 grave and in 15 mild cases, the injection was made into the femoral artery. The remaining 60 soldiers were given an intravenous injection of 20 cc. of a 1 per cent solution. Patients who suffered extremely were given another injection the same night and almost all of them slept. Sixty patients were able to walk the second day after admissian, and three days later, 70 patients returned to the front line. Only 5 patients, in addition to the 5 with gtave frost-bite were exacuated to a base hospital. Both intra-arterial and intravenous injections appeared effective, but the effect of the intravenous injections seemed to be more satisfactory. An intravenous injection proved effective after the failure of an intra-arterial injection. Intravenous procaine hydrochloride was used also in the treatment of hundreds of indolent wounds with torpid cicatrization. Complete cicatrization occurred within twenty days to one month of the start of the treatment. Intravenous injections proved superior to the intra-cutaneous or subcutaneous infiltration of the area adjacent to the wound. Three daily intravenous injections were made in many cases of tibiotarsal sprain, and the results were as satisfactory as those with local infiltration and there was less pain. Good results were obtained in several cases of dislocation of the shoulder, the elbow and the fingers after reduction of accomplished. Function was restored rapidly. In fractures with slight displacement, *e.g.*, fractures of the tuberosity of the humerus and in fractures of the wrist, the injections were continued daily for ten days. Painless mobilization became possible within twenty-four hours of the first intravenous injection. Union occurred rapidly and without complications. The results from this treatment in arthritis, hydrarthrosis and for pain of gastric ulcer were encouraging.—*G.P.*

Missue of morphine.—Evidence is mounting that morphine has a single legitimate use; namely, the relief of severe pain, a view that is widely accepted, but too little put into practice. Causes of morphine misuse or overdosage, are:

1. Failure to appreciate that nearly the safe maximum analgesic effect of morphine can be produced by ¼ grain and that ½ grain can produce serious depression in small persons or those whose tolerance may have been lessoned by wounds. An otherwise modest dose may become an overdose in a given case.

2. Failure to realize that subcutaneous injections of morphine will be only poorly absorbed, if at all, when the peripheral circulation is sluggish or inactive, as a result of cold or low systemic blood pressure. Failure to get pain relief from the unabsorbed morphine leads to the administration of a second or third dose, all of which may be absorbed at one time, when the peripheral circulation is re-established as a result of resuscitative measures.

3. The incorrect belief that extensive wounds are inevitably associated with severe pain, often leads to needless use of morphine. At Anzio, only a quarter of the severely wounded patients said, in response to a direct question, that they were having enough pain to want pain relief medication:

4. Administration of morphine for conditions that will not respond satisfactorily to the agent, however large the dose. is a common error. Specifically, the use of morphine to treat the restlessness associated with bleeding, with anoxia, or with hysteria, cannot be soundly defended. The use of morphine or with hysteria cannot be soundly defended. The use of morphine in shock, except for the treatment of severe pain, is contra-indicated. Morphine is occasionally administered when other agents (barbiturate sedation for example), are far better.—Beecher, H. K.: *Surg., Gynec., and Obst.*, Oct., 1945.—*G.P.*

Surgery of infections.—Kinsella uses the term "pad infections" for the subcutaneous infections whichtend to become localized in the palmer fatty pads. Pad infection I, the so-called felon, involves the distal phalangeal pad. Pad infections II and III involve the middle and proximal phalangeal pads respectively. Suppurative tenosynovitis may occur as a later complication. Pad infection IV involves the pads in the distal part of the palm und may spread to the web. It is usually due to infection occurring beneath a callosity, whereas infection in the first three pads is usually due to a puncture wound. Pad infections II and III may be distinguished from acute suppurative tenosynovitis; in acute suppurative tenosynovitis the swelling of the finger is uniform and the finger is sausage shaped; in pad infections the swelling is most pronounced over the affeeted pad, and the finger is spindle shaped. Incisions required to evacuate pus in pad spaces II, III or IV may be C shaped or cross shaped. The author uses for postoperative treatment the method which imitates the Winnett Orr technic. Dressings are changed infrequently, usually once a week He regards the sulfonamides and penicillin as important additions. Erysipeloid infections occur in those handling fish and pigs. In these cases an ill defined area of inflammation spreads diffusely and irregularly from a slight injury. Operation is not indicated, for pus does not form. This infection is self limited.—*J.A.M.A.*

Acute Pancreatitis—Diagnosis and treatment—John A. Ganshorn, M.D., M.Sc. (in Surgery.)

Three conditions are included in the diagnosis acute pancreatitis.

1. Acute haemorrhagic pancreatitis or acute pancreatic necrosis.

2. Acute interstitial pancreatitis or acute pancreatic oedema.

3. Acute suppurative pancreatitis (a relatively rare condition.)

The immediate cause of acute pancreatitis is the escape of normal pancreatic secretion into the substance of the pancreas. The lipase of the secretion digests fat giving the typical fat necrosis seen as greyish white spots on the fat. The trypsin, resulting, as has now been proved, from activation of the trypsinogen by intercellular juices without the presence of enterokinase, causes a digestion of the tissues and vessel walls with necrosis and haemorrhage. The mildness or severity of the disease depends upon the amount and nature of the escaping pancreatic juice.

Etiology.—There are two main theories as to what causes the pancreatitic juice to escape into the pancreas:

Less common causes:

1. Trauma—severe accidental trauma or on rare occasions surgical trauma, has given rise to acute pancreatitis. It was not often seen in war injuries of the pancreas.

2. Infection—this is a rare cause and gives rise to a suppurative pancreatitis. The infection may be blood borne or may arise from direct or indirect extension from neighbouring organs.

3. Vascular accidents—on rare occasions it is believed that arterial occlusion from emboli or thrombosis may give rise to the disease.

Diagnosis:—The signs and symptoms parallel in severity the pathological process. The onset of acute pancreatic necrosis and acute pancreatic œdema may be very similar but the clinical course of the two are very different.

The condition is most frequently misdiagnosed as perforated peptic ulcer, acute cholecystitis or acute intestinal obstruction and at time is mistaken for coronary occlusion. In its typical severe form the disease has a sudden onset with very severe and usually crampy pain. The pain is in the epigastrium. Nausea and vomitting follow immediately. The condition when most severe is an upper abdominal catastrophe, the patient is prostrate with the severity of the pain, he becomes shocked and has a greyish cyanonis, The pain may radiate through to the back, to the left or right scapula and there may be retrosternal pain suggestive of coronary occlusion. The patient usually tends to obesity and will often have had a good meal or drunk considerable alcohol before the onset of the pain.

On examination there is upper abdominal tenderness and rigidity. The rigidity is never a true board-like rigidity as in a perforated ulcer. A mass may be felt in the epigastrium, this has been taken as an acutely distended gall bladder more than once. There may be abdominal distension, though this usually follows after 24 hours, and is first noted in the upper abdomen. There is only a slight elevation in temperature (99°) and a fast pulse. This disproportion between temperature and pulse is fairly constant in the early stages, and helps to rule out inflammatory conditions of infectious origin. The temperature never tends to run very high unless infection occurs; usually 100°-101°. The leukocyte count is high; 15-20 thousand with an increase in polymorphs.

The condition may be so severe that the patient expires within 24–48 hours, of toxaemia. If the condition is not so severe the patient continues to have severe abdominal pain which radiates to the flanks and often down the right side. Distension from ileus developes as the pancreatic secretion and blood spread over the peritoneal cavity. Nausea and vomiting continues and the picture suggests in some respects an acute intestinal obstruction. No peristaltic sounds will be heard. The patient remains very ill for a considerable time, with a moderately high fever and marked leukocytosis. There is usually an albuminuria, 11% of cases show sugar in the urine with a elevated blood sugar and jaundice may be seen early or later in the disease. Some series put the incidence of jaundice as high as 30%. Cullen's and Grey Turner's sign, ecchymotic discoloration about the umbilicus or in the flanks is a relatively late sign occurring three days or more after the onset. The discoloration is from blood tracking out from the retroperitoneal space.

In about 50% of cases there will be a history of previous indigestion. This is usually of a type associated with chronic cholecystitis, a chronic gaseous indigestion, occasionally with more severe attacks suggestive of colic. This has often been present for years. In some there is a history of previous attacks to sudden severe epigastric pain, the illness lasting only 2 or 3 days, and suggestive of a previous milder attack of pancreatitis.

In the less severe type of disease, the onset may be less acute with gradually increasing crampy or constant epigastric pain and nausea and vomiting. It may be on the 2nd or 3rd day that the doctor is called because the pain has become very severe, and nausea and vomiting have continued. In these cases there is abdominal tenderness, but the abdomen is not tense, and palpation of a tender swollen pancreas may be quite possible. Peritoneal irritation is much less pronounced.

Treatment.—The treatment of acute pancreatitis, both the mild and the severe types, is now considered by the majority of authors to be medical and not surgical. Operation is indicated only in the later stages of the disease, when complications occur. The treatment of acute interstitial pancreatitis has always been considered medical, but because of the difficulty of diagnosis, many cases were and still are being operated upon to make a diagnosis. The older teaching was that in cases of acute haemorrhagic pancreatitis operation was indicated as soon as the diagnosis was made. There is an immediate mortality in these cases of about 10%. The patient dies of an apparent overwhelming toxaemia in 24 to 48 hours from onset and to date no treatment has been of avail in these cases. Many are never fit for on operation.

The operative treatment which is still advised by some surgeons is directed.

1st—at draining the necrotic pancreas and the peretoneal cavity.

2nd—at draining the biliary tract.

Medical treatment:—This is aimed primarily at putting the pancreas completely to rest.

1. Continuous gastric suction by Levine tube.

2. Intravenous fluids—there are several warnings in the literature about the use of glucose intravenouly. Glucose by mouth stimulates pancreatic secretion. I have read nothing to contra-indicate amino-acids by intravenous injection.

3. Reserved Trendelenburg position.

4. Morphine sedation.

5. Atrophine sulphate. This was used in some of Dr. Payne's cases. In pancreatic fistula cases atropine sulphate decreases the amount of the pancreatic secretion and the lipase content of the secretion.

As long as symptoms persist, the temperature remains elevated or the disease is above normal the patient cannot be considered well.

Surgery is indicated later in the disease if suppuration occurs. This will be necessary in all cases of the rarer acute suppurative pancreatitis.—*Vancauver Medical Journal.*

Some observations on the treatment of fibrositic conditions—A. Gordon Watson, M.D.

"Fibrositis may be defined as a condition in which acute or chronic inflammatory changes involve the fibrous tissues of the body, such as the subcutaneous tissue, the superficial and deep fascia, the muscle sheaths and tendons, the fibrous portions of the joint capsules and ligaments, the bursæ and the fibrous sheaths of the nerves. The affection gives rise to pain and impairment of movement. It may be subdivided broadly into three types according to the nature of the structures primarily attacked.

1. Panniculitis: Inflammation of the subcutaneous tissues and fat.

2. Inflammation of the muscle sheath and the fibrous tissue between the muscle fibres, the aponeuroses, the tendons and the superficial and deep fascia.

3. Peri-arthritis: Inflammation affecting principally the fibrous portions of the joint capsules, ligaments, bursæ and intra-articular cartilages.

4. Peri-neuritis: Inflammation affecting primarily the nerve sheath and/or the fibrous tissue between the nerve fibres.

All cases are due to some form of toxæmia or trauma. The former may be traced to local infection or disturbed metobolism, e.g., menopausal or gouty, while the latter may be due to direct injury to the tissue or prolonged strain or exposure to cold.

The differential diagnosis is often clinically easy but can be very difficult—the symptoms of pain on movement, tenderness on pressure, elicitation of painful areas on palpation, dull aching pain at rest, referred pain, stiffness and restricted movement may lead to a correct diagnosis, but it is often necessary to confirm by all the laboratory and X-ray findings at our disposal. A high blood uric acid suggests gout, a steep sedimentation rate suggests the possibility of rheumatoid arthritis, malignancy or severe anæmia. An allergic history, climateric, etc., endocrine therapy; a specific infection appropriate treatment for the disease. The results of treatment help, e.g., manipulation, massage.

Panniculitis.—This is much commoner in women than men; it can be divided into the generalised type when the small, shot-like-nodules can be detected practically all over the body, and the more localised condition. The former is usually found in women approaching or during the menopause, and, as would be expected, is benefited by ovarian extracts, sometimes with the addition of thyroid. These patients tend to be obese and a strict vegetarian diet, e.g, orange juice for a week or so, with daily intestinal washouts, will sometimes cause the little nodules to miraculously disappear, only to return when the treatment is stopped. The same occurs during treatment at a radioactive spa. These facts, however, can be utilised in outlining the modified home treatment diet, hot baths followed by skin friction, careful attention to evacuation of the bowels with an occasional wash-out and suitable medication to correct obvious defects.

For the more localised conditions, and for adiposis dolorosa, radiant heat and massage, ultra-violet radiation and ultra short-wave therapy are all useful. There are also various local applications, of which one of the best is 10 to 12 per cent salicylic ester of ethylene chlorhydrine in a lanoline ointment; it is clean to use and, if well rubbed in, will not soil the most delicate underwear. Estorsil, a proprietary linament, is equally useful. An electric pillow also helps many, as it does for other forms of fibrositis. Exercise and exercises of all kinds help to keep fibrositis in abeyance.

2. *Inflammation of the muscle sheaths, etc.*—Treatment here varies with the particular tissue involved. Having located the site one should deal with predisposing causes, e.g., focal sepsis, repeated strain, metabolic dysfunction, etc., which often necessitates very careful examination, including that of the blood with differential blood count, Arneth count, Blood uric acid and sedimentation rate.

The treatment advised is firstly, the correction of any errors disolosed. As a result of the tests, and the stimulation of elimination by the skin, bowels and kidneys. Baths of various kinds, coupled with skin friction, certainly have a marked, not only curative effect, but preventive effect as well. in people prone to relapses, as though the condition may be limited to one painful spot in say, the supraspinous muscles, it is often part of a general tendency towards fibrositis which, in this particular case, has been localised by, perhaps, some train.

For the local condition rest of the affected part, e g., strapping the lumbar region for lumbago; the ribs for pleurodynia; light plaster splingting for joints where this treatment can be carried out, followed by a broad strip of plaster round the limb proximal to the painful area. If deep-seated, deep massage or injections with a solution of novacan, or pin-point firing of the skin over the area, all have their uses.

Infra-red radiation and ultra-violet radiation, alone or together, are often curative. Ultra short-wave therapy, static electricity, surging currents, ionisation, infiltration of the painful area by the injection of analgesics, oxygen, salt solution, etc., have all proved efficient methods of treatment.

Peri-arthritis and perineuritis.—As regards treatment these may be considered together. Special care is required in accurate diagnosis of any underlying bony deformity, especially

in regard to the vertabræ in sciatica and where symptoms point to damage of inter-articular cartilages and intervertæbral discs. Corrections of the causes of strain and/or methods of giving physiological rest to the area affected are one of the first considerations, e.g., many acute sciaticas of the peri-neuritic type do well when the leg is slung from a Balkan Beam with slight flexion at the hip joint and to a right-angle at the knee. Intervertæbral discs will require a spinal plaster bed as for spondylitis ankylopoietica, followed, possibly, by a Goldthwaite brace. Brachial neuritis requires rest—a sling may be sufficient. Any of the treatments mentioned in the preceding paragraph may be helpful, especially ionisation, using a 1 per cent. K. I. Electrolyte and a galvanic continuous current of 5 to 12 m.a. for 15 minutes, either daily or on alternate days; soda salicylate may be substituted for K.I. Operative measures may be necessary, e.g., ruptured meniscus of the knee joint, pressure on nerve roots from deformity or disease of the spine or protusion of a intravertæbral disc.—*Medical World.*

Ulcerative colitis.—To a questioner the following answer is given in the *B.M.J.* Dec. 14, 1946:—The ætiology is still uncertain. It is probable that the condition should be regarded as a syndrome, different cases having a different aetiology. Every patient should have a careful sigmoidoscopy examination with, if possible, a barium enema.

Medical treatment should be tried first. If there is an acute flare-up the patient should be in bed. Diet must be highly nutritious and completely free from all roughage. There is no specific remedy, Sulpha suxidine retention enemata 2 % with full doses by mouth, supported by penicillin, may be successful in some cases, in others, there may be symptomatic improvement only following control of the secondary invading organisms. Local treatment may consist of retention enemata of silver nitrate, 5% tannic acid (2 gr. to the ounce) or of bismuth subgallate (2 gr.) is a suspension of mucilage. If diarrohœa is frequent and painful, releif may be obtained. For a starch and opium enema, recently some dramatic responses, have been reported after the ingestion of saw pig intestine. It should be given in dose equivalent to ½ lb. daily and must be continued for a month. Any anæmia should be dealt with by repeated transfusions of fresh blood.

Surgery is best kept until medical treatment has failed. A transverse colostomy or an ileostomy is the operation of choice, but in any case, the artificial anus should be retained for at least a year, and may have to be kept in definitely. For strictures or polyposis a colectomy may have to be advised. —*B.M.J.* Dec. 14, '46.

OPHTHALMOLOGY

The ocular changes of primary diffuse toxic goitre.—Woods, A. C. *Medicine*, 1946, v. 25, May, pp. 113-154.

This comprehensive review discusses, in great detail, the usually noted ocular changes of primary toxic goiter. In the first section, all of the lid signs are described, and the author explains each one, in terms of the relationships between relaxation of the orbicularis muscle, overaction of the levator muscle and increased tone of the palpebral smooth musculature. The last mentioned factor is a logical one, in view of other evidences of sympathicotonia in toxic goiter.

Under the heading of external ocular changes, the author discusses weakness of convergence (Moebius' sign), abnormal pigmentation of the skin of the lids (Jellinek's sign), adrenalin-sensitized pupils (Loewi's sign), and excessive lacrimation.

Two types of patients with extra-ocular muscle palsies are described. The patients in the first group have isolated or multiple pareses, with no definite pattern of involvement. These occur with exophthalmos, and disappear when the thyrotoxicosis is controlled. Organic changes analogous to those of myasthenia gravis are sometimes found in these muscles. However, there is no favorable response to prostigmine. In the other type ocular-muscle palsies occur in "malignant exophthalmos", or "exophthalmic ophthalmoplegia". The muscle changes progress even after thyroidectomy. The muscles show hypertrophic myositis, fibrosis and edema. This condition has been related to postoperative changes in pituitary function, and treatment must include thyroxin and iodine.

Exophthalmos is seen in 65 per cent of patients with primary diffuse toxic goitre. Reports are cited to indicate that the exophthalmos does not regress after the thyrotoxicosis is controlled. The pathogenesis of the exophthalmos is discussed and the importance of the following factors is evaluated.

Hypertrophy of orbital contents, particularly orbital fat is considered important. Relaxation of the rectus muscles, allowing the eye and orbital contents to protrude is probably not an important factor, since the role of the obliques is not satisfactorily explained. The importance of contraction of smooth muscle of the orbit is difficult to accept, since the orbital smooth muscle is vestigial in man. Edema of the orbital contents has been substantiated as a factor in exophthalmos. Orbital vasodilation has not been proved to be a causative factor. Degenerative myositis, with increase in muscle size, can be at best only an inconstant cause of exophthalmos, since it is not uniformly present.

The action of the anterior pituitary hormone probably plays a part. The work of Mulvany is discussed. The author concludes that although there appears to be a relationship between the exophthalmos and the anterior pituitary, its exact nature is as yet unknown: Benjamin Milder.—*A.J. Opth.*

The etiology and treatment of blepharitis: a study in military personnel.—Thygeson, Phillips, *Military Surg.*, 1946, v. 98, March, pp. 191-203.

Blepharitis was the most common external eye infection seen in military personnel. Laboratory and clinical studies of 350 cases of blepharitis indicate that the three important types are the staphylococcic, the seborrhœic blepharitis, and the diplobacillary. Staphylococcic blepharitis is often unilateral, and is often associated with chalazion, hordeolum, and ulceration. It is characterized by small hard scales, which are tenacious and difficult to remove. An associated keratoconjunctivitis is almost the rule. Temporary or permanent loss of cilia, and associated otitis externa, impetigo, sycosis barbe and acne vulgaris are common. There is burning, itching, and photophobia.

With seborrhoeic blepharitis, there is seborrhea capitis, and usually an associated seborrhoeic dermatitis of brow and ears, the lesion is always bilateral, nonulcerative, and squamous, the scales are greasy and easy to rub off, and associated conjunctivitis is minimal if present. There are no complicating inflammations. The skin gives evidenee of increased seborrhea, discomfort is minimal.

Diplobacillary blepharitis is characterized by maceration of the skin at the inner and outer canthus, and associated conjunctivitis. The infection is superficial, there is no ulceration, and no inflammation of the Meibomian glands.

The finding of budding yeast forms, pityrosporum ovale, was considered diagnostic of seborrhoeic blepharitis. Routine bromicroscopic examinations of the lid margin in chronic conjunctivitis revealed a high incidence of mild and subclinical blepharitis, usually staphylococcic. There is no evidence that vitamin deficiency, refractive error or allergy play significant roles. I. E. Gaynon. —*A.J. Opth.*

Ophthalmic penicillin ointments.—L. Von Shallmann, M.D., Anne E. Crosso, Ph. C., and Mary Glyde Marsh, B.A., *Arch. Ophth.* 36:284 (Sept., 1946.)

1. Of the series of nine ointment bases which were tested, two were irritating to the eye. No irritation could be attributed to any of the four penicillin compounds which were incorporated in the ophthalmic ointments.

2. The potency of the penicillin in the salves was relatively stable in one hydrous and two anhydrous bases after twelve to sixteen weeks in the refrigerator. Storage at room temperature resulted usually in a rapid loss of protency except for two ointments prepared with an ahhydrous base. No difference in the stability of the various ointments could be ascribed to the penicillin compound in the salves.

3. The penetration of penicillin into the aqueous of rabbits was substantial from one hydrous and two anhydrous bases. After the application of two ointments with the calcium salt, the content of penicillin in the aqueous exceeded that after the use of salves with the sodium and ammonium salts. The resorption of penicillin was unsatisfactory from the one ointment which contained the free acid of penicillin.

The Schering Corporation supplied the ammonium salt of penicillin and the ointments containing the free acid and the ammonium salt in the S base, and the Lederle Laboratories, Inc., the ointment containing the calcium salt of penicillin in the L base.—*E.E.N.T.*

EYE, EAR, NOSE & THROAT

Concussion deafness.—J.V. Stewart and D. W. Barrow, *Arch. Otolaryng*, 44:274 Sept. 1946.

1. The noise of gunfire produces a definite loss of hearing, beginning in the hightones (2,048 to 11,584 cycles per second), among gunnery instructors. When severe, all tones are affected. There was wide variation in individual susceptibility in this group.

2. The average loss of hearing for both ears in the gunnery instructors in this group was 20 decibles. The length of exposure necessary to produce damage also varies widely from person to person. After ten to twelve months of service on the range, further deterioration occurred slowly if at all.

3. Tinnitus of varying degree was a complaint in 50 per cent of the subjects examined and was the most annoying symptom noted.

4. Since this damage occurs insidiously and since damage to the conversational range is late, few persons appreciate their handicap until it is far advanced and fail to request medical attention. Routine prearranged surveys are necessary.

5. Cotton plugs in the external auditory canal have not been effective in preventing loss of hearing due to the noise of gunfire.

5. There has been no significant improvement six months after cessation of exposure to the noise of gunfire, and it is believed that the loss of hearing will be permanent.

7. The efficacy of various protective devices is being investigated and will form the basis of a subsequent report—*E.E.N.T.*

OBSTETRICS and GYNÆCOLOGY

Heartburn in pregnancy.—Daniel B.Roth, M.D., Teaneck, N.J.

Heartburn is a common complaint in the last trimester of pregnancy and one which occasionally causes real discomfort to the patient. It is due to the regurgitation of gastric contents and has no relation, at all, to the heart. It is essentially a neuromuscular phenomenen.

Clinical picture:—The clinical picture is familiar to all who treat pregnant women. It appears in the last trimester and seems to be worse at night. Many women experience it only in a recumbent position and in some, the symptoms are severe enough to interfere with sleep. Once the patient has

delivered there is rapid disappearance of the symptoms. It is usually not associated with organic disease and has been treated with many remedies of varying effectiveness.

Of prime etiologic importance is the hypomotility which is found at the later stages of pregnancy and involves both the cardiac sphincter and the lower end of the oesophagus.

Treatment :—A reduction of the fat in the diet with the elimination of fried foods is frequently beneficial. The diet should be rich in proteins and milk

Prostigmine has been reported as a very efficient agent in this condition. This synthetic cholingeric drug acts well on the distured neuromuscular function of the stomach.

Large doses of B complex are also efficacious. Hart reported a series of cases in which he administered yeast, thiamine chloride and nicotinic acid to a group of women complaining of heartburn. The use of thiamine in heartburn is based on the observation that dysphagia and cardiospasm in nonpregnant persons can be relieved by this vitamin.

While it has been shown that these patients have lowered gastric motility and secretion, many women who have heartburn have obtained relief by using alkalis, such as bicarbonate or block magnesia. The effect of these alkalis must be largely psychic, a fact to be borne in mind in the evaluation of other remedies.—*Jour. Med. Sc. New. Jessy.*

Hæmolytic disease of the newborn (erythroblastosis petalis); clinical analysis of 55 cases with special reference to pathogenesis; prognosis and therapy.—Martha F. Leonard, *J. Pediat.* 27: 249 (Sept.) 1946.

This is a review of the cases of 55 patients with hemolytic disease of the newborn treated by members of the pediatric service of the New York Hospital from 1938 to 1944. The infants showed the typical signs: jaundice, progressive anæmia, hepatosplenomegaly, edema, hemorrhage and erythroblastemia. For 84 per cent of the 33 infants tested there was the typical Rh pattern—the father and baby had Rh-positive blood and the mother Rh-negative blood; the other 16 per cent had mothers also with Rh-positive blood. Two multiple pregnancies were included as well as 2 infants of a diabetic mother, in whom the typical picture, including Rh factors was present. Clinical signs varied greatly in degree. There was no correlation between the severity of the anemia and the degree of jaundice or the number of circulating erythroblasts. Jaundice often occurred in the absence of or before the development of anemia, which casts some doubt on the usual explanation that jaundice and erythroblastemia are secondary to hemolytic anæmia. Hemorrhagic manifestations were freqnent, especially in infants with low prothrombin levels. Parenteral administration of vitamin K often had little effect on prothrombin levels. This fact, together with the poor correlation between the jaundice and anæmia and the pathologic conditions in fatal cases, is evidence of poor hepatic function. The cause of hepatic damage is still unknown; perhaps it is arrested development of the liver in an immature phase or a direct effect of the antigen-antibody reaction. Patients with more impairment of hepatic function have deeper jaundice; those with better functioning livers may have little or no jaundice in spite of pronounced destruetion of blood.

Complications were nonspecific diarrhea in 19 pationts, obstructive jaundice in 3, septicemia in 2 and spasticity in one. The mortality was 35 per cent, with hemorrhage, atelectasis or bronchopneumonia frequent observations, as weil as extramedullary hemopoiesis and hepatic necrosis. .In only 2 cases did anemia appear to be a contributing cause of death. Edema and severe or increasing erythroblastemia indicated a poor prognosis.

Transfusion was the chief method of treatment. Comparison of results following use of Rh-positive and Rh-negative blood showed a similar immediate effect in raising the blood level and in improving the clinical condition, but Rh-negative blood resulted in fewer transfusion reactions and in a better sustained rise in hemoglobin. The necessity for immediate transfusion in nonanæmic infants was questidned. In babies with good general condition it was recommended that frequent blood counts be done, and Rh-negative blood transfusions be given only if the hemoglobin fell below 11 Gm. In the absence of anæmia, occasional infusions of plasma may be used for supportive effect and to prevent hemorrhagic tendencies. In critically ill infants, small transfusions of freshly drawn Rh-negative blood are advised. Vitamin K should be given frequently until the prothrombin level is normal. Additional therapeutic measures used in these cases—a high protein, high carbohydrate, and low fat formula (powdered half-skimmed milk with added carbohydrate), and administration of choline and liver extract—have a rational basis and warrant further trial. Administration of vitamin K to Rh-negative donors before drawing their blood was suggested as a means of raising the prothrombin level of affected infants and preventing hemorrhage. —*A.J.D.C.*

DERMATOLOGY

Treatment of leprosy with "solutiazamide".—Lima L. de S. & Ceroqueira, G. de C. Terapeutica experimental da lepra pela solutiazamine, *Rev. Brasileria Leprologia.* S. Paulo, 1945, June, v. 13, No. 2, 97–100.

"Solutiazamide" is stated to be p. (y-phenyl propyl amino) phenyl sulphamidothiazole-x-y-disulphonate of sodium and prepared directly from sulphathiazole, and is very readily soluble in water. A solution of 45·3 per cent. corresponds to a 20 per cent. sulphtathiazole base. It was administered daily (except Sundays), intravenously in doses starting with 1 c.c. and increasing up to 5 c c., or, for adults, 10 cc. After three weeks an interval of one week is allowed to elapse before the treatment is continued. The treatment is controlled by a blood count and hæmoglobin estimation every 10 days, examination of the urine and any deposit, and an estimation of the drug in the blood. If the red cells fall below 3½ million per cmm., the dose of the drug is reduced and liver preparations are given; the urine is examined for blood and albumin or any indication of nephrosis, and if it appears treatment is suspended.

The new drug was tried in 50 cases of moderately developed lepromatous forms, and others more advanced, 50 cases of lepromata wlth skin involvement and lesions of eyes and nose and some in whom chaulmoogra had been tried ineffectually. The authors publish their results in order that others may be led to make trial of the drug. So far reported these results sound almost too good to be true. The authors report: rapid cicatrization of leprous ulcers, cicatrization of fusing lepromata, disappearance of lepromatous infiltrations to the level of the skin, softening and disappearance of cutaneous nodules; perforating ulcers are sometimes benefited; the ocular lesions improve and do not relapse, nasal crust disappear, obstruction is relieved and dyspnoea subsides. —*Leprosy* abstracted by H. Harold Scott.

The therapeutic effect of promin in leprosy.—Faget, G. H. and Pogge, R C., *Pub. Healtn Rep.* Wash. 1945, Oct. 5, v. 60, No. 40, 1165–71, 8 figs. on 2 pls.

This paper records further trials of promin in leprosy in continuation of those reported in Nov. 1943 [this *Bulletin*, 1944, v 41, 494].

The present report is based on the treatment of 137 patients at the U.S.A. National Leprosarium by means of 32,000 intravenous injections amounting to a total of 126,961 gm The average daily dose, including days of rest, varied from 0·4 gm. to 4·6 gm. The usual dosage is 1 gm. daily increasing, to 5 gm. but in some patients who showed toxic reactions the maximum daily dose did not exceed 2 gm. The present routine consists of 2 to 5 gm daily by the intravenous route on six days each week, in courses up to two weeks with a week's rest between the courses, which allows of restoration of the red corpuscles lost during a course. The longer the duration of the treatment and the larger the doses, the greater the benefits, as shown by tables of data. The percentage of tho total 137 patients treated who improved was 58·4; the figures for treatment of less than six months respectively being 25·6 and 71·4 per cent. All of six cases treated for 3 to 4 years had improved. A total dosage of less than 500 gm. only produced improvement in 25·6 per cent., but with 500 to 1,000 gm. the percentage rose to 72·5 per cent or nearly three-fourths of the patients. Only two patients became worse while under treatment; both were advanced cases with involvement of the larynx. The improvement was objective and not only subjective; this is confirmed by photographs of patients with advanced lepromatous disease before and after treatment; the benefits are not limited to action against secondary infections. In over 10 per cent of 62 patients treated for more than one year, positive bacteriological findings have changed to negative on several consecutive monthly examinations. Serological reactions may also become negative coincidently with improvement in or arrest of the disease, as shown by tables of data. The cost of the drug is not given. Promin is not claimed to be a specific remedy for leprosy, but there is evidence that it has at least an inhibitory effect on the progress of the disease and that it even causes retrogression in some lesions. There is, therefore, some hope that continued chemotherapeutic research may produce a still more effective remedy. Preliminary studies suggest that diasone has a similar action to promin and that it may prove even more effective.—*Leprosy* abstracted by L. Rogers.

The frequency of neuritis in leprosy. Its symptomatic treatment with intravenous calcium gluconate. Pogge, R. C. *Internat. J. Leprosy* Cleveland, Ohio. 1944. Dec., v. 12, 31–7.

At the U.S.A. National Leprosarium, distressing neuritis was fouud to have occurred in the course of the disease in 56 per cent. of 203 unselected ambulatory patients. It is mainly due to pressure on nerve trunks produced by a lymphocyte infiltration, which may either destroy the function, or result in painful irritation of the nerves, especially the ulna, median, radial. personal and superficial cervical, which become enlarged, painful and tender to touch. In 73·7 per cent. of the patients the symptoms persisted for a year or more, so that in the remaining 26·3 per cent. improvement had taken place within 12 months. A greater proportion of improvements should therefore be obtained to indicate good results from any treatment under trial. Routine treatment with either chaulmoogra preparations or with promin did not, within 12 months, have any demonstrable effect on the course of the nerve pains.

Treatment trials showed that mild cases improve on vitamin B, orally, or on heat treatment. In moderate cases about half improve on vitamin B_1, but slightly less under heat; in severe cases it is necessary to inject vitamin B_1, which is then effe tive in about half the time. In persistent cases soluble calcium was tried for its sedative influence in decreasing the irritability of nerves, but absorption on oral administration is variable. The author therefore injected 1 gm. of calcium gluconate in 10 cc. of distilled water intravenously (given rather slowly) daily for a week, and subsequently gave one to three injections a week until the nerve pain became moderate in degree over several weeks. During 1943, 35 patients were so treated without any other medication, with improvement in some 90 per cent. of them. This treatment thus proved palliative in neuritic complications.—*Leprosy* by L. Rogers.

Correspondence

Infantile Hepatic Cirrhosis

To The Editors, THE ANTISEPTIC, Madras.

Sirs,

The events of death of Mr. Devendra, son of Dr. Rajmal Nandecha (*The Antiseptic*, Dec. 46, page 792) moved me to note the following comments with the hope that you will give them space in the next issue of the *Antiseptic*.

CAUSES.—(1) Chronic malaria ; (2) abuse of quinine or mercury ; (3) sedentary life ; (4) excessive eating or drinking that bring more blood to the liver ; (5) residence in tropical climate ; and (6) exposure to a cold current of air from wind or punkha.

SYMPTOMS.—*In early stage* :—Fever (irregular), pain in liver, coated tongue, loss of appetite and clay coloured loose stools.

In later stage:—Tenderness of liver, nausea-constipation or diarrhœa and enlargement of liver, coloured urine.

In the final stage :—The liver, either contracts (cirrhosis) or abscess developes into the cavities of the chest and bowels, from which there is no escape.

TREATMENT :—*Merc. Sol.* (6-30-200) :—It is very frequently indicated for inflamed and enlarged liver, with severe pain and ascites with indurated liver.

Merc. Daelcis (6 x) :—It is valuable in diarrhœa of infants, with scarcely any tenesmus, stools grass-green, excoriating, liver enlarged, mouth pale with offensive odour, swollen glands and burning pain in anus. (Allen).

Podophyllum (3-30) : — In acute hepatitis with constipation, liver enlarged, bilious liquid evacuations.

Leptandra (3x–3):—Tongue yellow, copious brownish or putrid smelling evacuations, dysentery, fever and clay coloured motions. Dropsy.

Bryonia (3x, 6 and 30):—Liver enlarged and hard, obstinate constipation and slight jaundice. (To be used alternately with Mec. Sol. in acute hepatitis).

DIET.—Bland easily nourishing and gentle exercise.

"According to Moore, in India, there are several causes at work, that act through the bowel resulting in cirrhosis of the liver. The absence of accurate diagnosis of the ailment in the very beginning developed into the complications with abuse of quinine. Strong doses loaded on the system one by one led the child to death."

Rewari, Gurgadi, 14-1-'47. S. L. BHARADWAJ, M.B.H.

News and Notes

The 2nd Behar Provincial Medical Licentiates' Conference, Darbhanga

Dr. L. C. Sinha, Secretary, Darbhanga Branch. Saraswati Dispensary, Darbhanga, writes as follows :

A scientific session is going to be held along with the conference on 16th March '47.

Interesting scientific papers are cordially invited from our members to be read in the Conference. The papers should be sent within the 1st week of March '47 positively to the Secretary.

STALLS FOR EXHIBITION WILL BE AVAILABLE. Medical firms may apply to the Secretary for advance booking because of the high pressure of demand for the stalls due to the presence of Darbhanga Medical College here.

The Gujarat & Kathiawar Provincial Branch of the Indian Medical Association, Ahmedabad

Hon. Gen. Secretary, Gujarat & Kath. Prov. Branch, I.M.A., writes as follows :

The Conference of the above Association will be held during the last week of March, 1947 at Ahmedabad. Members from branches of Gujarat and Kathiawar are earnestly requested to participate in the ensuing Conference and thus make this function a success.

Khandwa Medical Union

Secretary, Medical Union, Khandwa, writes as follows :

A meeting of the Medical Union, Nimar, was held on 12-1-'47 evening at the residence of Dr. D. C. Jain, Private Practitioner, Khandwa. Khan Sahib S. M. A. Rahman, Civil Surgeon, Nimar, was in the chair. 19 medicos out of 22 attended the meeting.

1. The meeting resolved to hold meetings on the 1st Sunday of every month regularly. The regular absentees would be penalised for their absence from the meeting.

2. The meeting resolved that each and every medico of Khandwa should enlist himself as a regular member of the Medical Association. The minimum subscription of the membership would be As. 8/- only.

3. It was further resolved that a liberal donation be collected from the members for subscribing foreign and home medical literature, so that everybody may get the advantage of recent development in the medical science.

4. An interesting lecture was delivered by Dr. T. K. Kulkarni, on his special branch of radio-therapy wherein he explained adequate treatment regarding different kinds of diseases with ultra-violet rays, infra-red lamps, diathermy, ionised and galvanised current with its advantages and disadvantages, indications and contra-indications.

5. Dr. Khan Sahib explained the advantages of the Association in view of the medical profession. He narrated one interesting case of typhoid fever of his personal experience showing how the present day medical science is deficient.

In the end the meeting came to a close after a light refreshment.

Decline and Fall of the Undescended Testis

See The Lancet, p. 999, May 2nd, 1936. *A Report on 25 treated cases*

GROUP	TYPE	CASES	RESULT
I Adherent Normal Testis Normal Child	Unilateral - -	5	Failed
II Subnormal Testis Subnormal Child	Bilateral - -	6	Cured
	Bilateral with hypospadias -	2	
	Unilateral - -	3	
III Subnormal Testis Normal Child	Unilateral - -	3	Cured Improved
	Bilateral - -	3	

"The injections were given once a week and the dose was 500 rat units given intramuscularly into the buttock."

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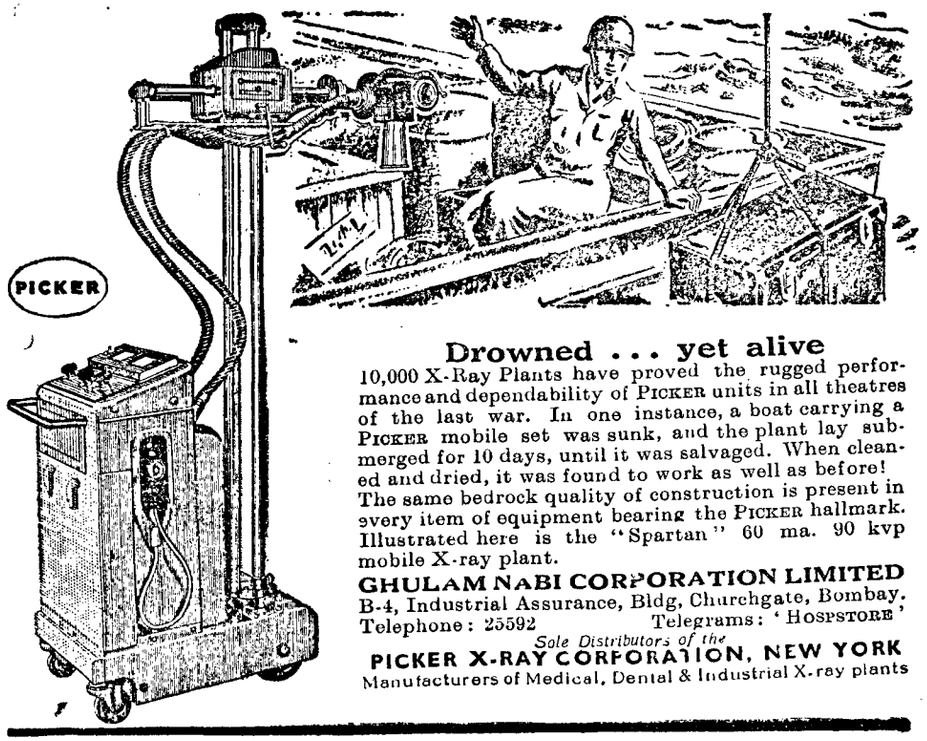

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W.