The absence of addisin in the gastric juice in pernicious anaemia is the cause, we believe, for the reversion to a megaloblastic marrow in this disease. The lack of a satisfactory test for the presence of addisin in concentrated gastric juice, save its injection into a patient with pernicious anaemia, is a handicap, as it has been in the case of liver extract and ventriculin. Experiments are under way, both biological and chemical, to try to overcome this difficulty. The observations we have made up to the present time indicate the probability that addisin is the physiological hormone which maintains the normal state of the blood in health.

The therapeutic effect of intramuscular injections of addisin in secondary anaemias is under investigation.

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AN AMBULATORY DISTRACTION SPLINT FOR LEG FRACTURES

BY

E. ROBERT, F.R.C.S.

LATE SURGEON, GENERAL HOSPITAL, JOHORE, MALAYA

The splint illustrated consists of two lateral distending rods, bearing on an upper transfixion pin passed through the tibial tuberosity, and a lower one passed through the tibia and fibula just above the ankle-joint. Each rod has a central segment, the ends of which are turned with right- and left-hand screw threads respectively, which, engaging in corresponding sheaths, can lengthen or shorten the whole rod.

The terminal parts which engage the transfixion pins are connected with the rod by universal joints to rectify any deviation in the axes of the pins. A rectangular cross-bar, with movement in two planes, connects the upper segment of one rod with the lower segment of the other rod, and so corrects the internal rotation of the lower bone fragment, which is produced by the transfixion pins lying in different horizontal planes; the lower pin passes from without inwards and forwards, whereas the upper pin passes directly from side to side. The steel pins, about 3/16 inch in diameter and 7 inches long, are "square-tapered" at one end to facilitate penetration of the bone. Care is taken that the points are not softened by overheating on the lathe, as the cortex of the fibula is very hard and is apt to turn the steel point, while a long taper, about 3/4 inch, is less likely to split the brittle fibula.

TECHNIQUE AND RESULTS

The leg is *x*-rayed in two planes. The patient is then taken to the theatre and, preferably, given a general anaesthetic: local anaesthesia does not control the jarring at the site of fracture. The lower pin traverses the fibula first, driven through with an ordinary heavy carpenter's hammer. The distraction splints are then threaded over the pins and extension secured by turning the middle

segments with a key. Correction of lateral angulation and final adjustment of the bone fragments is done under the *x*-ray screen on the following day, after which the patient is made to walk. The internal-rotation deformity of the lower fragment is corrected by clamping on the cross-bar, while antero-posterior angulation is controlled

FIG. 1.—The splint applied.

FIG. 2.—X ray showing position.

by stretching a well-padded bandage between the two splints. Although early full correction of bone displacement is desirable, such manipulations can be repeated as often as is found necessary during the course of the first four or five weeks, no anaesthesia being needed. In the three cases—one Tamil and two Chinese coolies—treated by this method (all closed fractures), union between the fragments occurred suddenly—that is, within the space of two to four days at about the end of the fifth week. The pins are removed and the splint discarded after six weeks (actually forty-three days in the above cases). The method is suitable for any case in which the line of fracture does not encroach upon the terminal two inches of either end of the tibia.

Ulceration of the superficial tissues occurs around the pin-holes, more so above, where it may reach the size of a sixpence, and where a small slough of deep fascia may separate, after which the small cavity so formed fills up with clean granulations while the patient continues to walk about on the pins. It must be admitted that early progressive ulceration with a purulent discharge caused by a dirty-looking pin transfixing the leg rather strains one's confidence in the capacity of the body tissues to survive surgical insults; but provided the

Diagram of apparatus.

pin is passed through healthy skin and tissues in the first place, no serious bone infection seems to occur. The punctures have generally healed within ten days of removal of the pin, and in the case of a persisting sinus curettage has effected healing.

WALKING

It is undoubtedly a terrifying experience for the average patient to commence walking on a recently broken leg, and, as in all unpleasant physical ordeals, firmness on the part of his mentor is the quickest way to banish the fears of a nervous individual. While fully realizing that we bear another's pain with more fortitude than we bear our own, experience in ambulatory treatment in fracture work convinces me that the pain in early walking is by no means unbearable. All of us have an instinctive fear of leaning upon a visibly broken prop, in spite of reason telling us that the prop itself is adequately supported. It is just the proportion of calm reason to instinctive panic in any one individual that determines his response to early ambulatory treatment.

Although the splint described here may seem rather brutal, a little reflection shows that any splint which separates the bone fragments, and keeps them apart, removes the one source of pain in a fractured limb—namely, repeated slight trauma to the sensitive periosteum at the line of fracture, produced by muscle spasm or misguided handling of the limb. It is generally agreed that skeletal traction is both efficient and painless for this very reason, and as the transfixion pin, with attached weight bearing on the bone, causes no pain in recumbency, it is illogical to suppose that the same principle applied in the upright posture can differ markedly in its sensory

effects. I found no difference in this respect from cases treated in the "Delbet" plaster splint.

OEDEMA

This seems to be a source of anxiety to patient and doctor alike, and Bohler describes a special zinc-gelatin bandage which he applies to the leg for several weeks after union of the bone has occurred. But surely this question of oedema is exaggerated as a disability in fractured legs, and, apart from causing some stiffness at the ankle, its presence is of little import. The oedema is due to damage to lymphatics and small blood vessels around the broken bones, and it will persist and recur with dependency of the limb until new channels of absorption are opened up by centripetal pressure, as in massage or walking.

The objection to massage is that at best it is a poor substitute for natural function—that is, walking; also, it centres the patient's attention on his disability as such. In contrast to this, the exercise of walking causes progressive elimination of the idea of disablement. It must be admitted that early walking greatly increases the oedema, but on the other hand, if persisted in, it is probably the quickest way of permanently reducing it. In the first case upon which this splint was used swelling around the ankle was so marked in the second week that pus was suspected in or around the joint, yet within three months from the date of accident difference in size between the two legs was barely noticeable.

One of the advantages of the splint is that the whole limb is exposed to view, thus giving a rough idea of the position of the bones at a glance; also, apart from the pin-holes, there is no apparatus to cause pressure sores.

THE PROBLEM OF LYMPHOID TISSUE

BY

J. M. YOFFEY, M.Sc., M.D., F.R.C.S.

LECTURER IN ANATOMY, UNIVERSITY COLLEGE OF SOUTH WALES
AND MONMOUTHSHIRE

The designation "lymphoid*" is applied to a complex of tissues scattered throughout the body. Since the classical work of His¹ in 1860, much has been written concerning this tissue aggregate, yet there has been little real advance in our understanding of its functions. For this there have been two important reasons. The lymphoid tissues are not concentrated into one organ, but are widely diffused throughout the body. Consequently the methods of direct experimental attack, involving complete extirpation, are inapplicable. Indirect methods of extirpation, such as exposure of the animal to x rays, are not sufficiently selective to be of value. Failing the experimental approach we have been compelled to fall back upon a clinical and pathological study of the problem. Here, unfortunately, both the clinician and the pathologist, looking at the lymphoid tissues from their own special angle, have combined to draw a particularly bright red herring across the trail.

THE "BARRIER" FUNCTION

The clinician and the pathologist have repeatedly observed that lymphoid tissues are often the seat of secondary inflammation or malignant deposits. They have therefore assumed that lymphoid tissue exists for that very purpose, serving as a barrier which holds up—for a time, at any rate—the spread of injurious matter.

* In recent years Aschoff, Ehrlich, and others have endeavoured to distinguish between "lymphatic" and "lymphoid" tissues. The grounds for this distinction are somewhat arbitrary, and hence the older term "lymphoid" is here retained.

Yet even a brief consideration will serve to show that there are serious flaws in this interpretation.

It is undoubtedly a striking phenomenon to observe a mass of lymph glands enlarged as a result of inflammation or malignant deposits. But while it is true that the lymph glands act as temporary barriers against the spread of disease, it is equally true that the lymphatic vessels greatly facilitate this spread. Thus, quite a small epithelioma on the back of the hand may cause metastases in the axillary lymph nodes. Once in the lymph nodes the metastases are then held in check, albeit only for a time. Had it not been, however, for the lymphatic vessels affording these metastases an easy and rapid pathway, they would never have been able to travel as far as the axilla, but would have remained strictly localized to the hand. The gain from the barrier action of the axillary glands is more than offset by the dangerously easy dissemination made possible by the lymphatic vessels. Similar considerations apply, *mutatis mutandis*, to the lymphatic spread of acute inflammation. The lymphoid tissue found in the wall of the alimentary canal has been thought to be even more obviously defensive in function, lining as it does a hollow tube full of micro-organisms. On this hypothesis one would expect that the removal of tonsils and adenoids would leave the nasopharynx weak and defenceless, which is not the case. It would appear that the picture of countless hordes of lymphocytes lying in wait in the tonsils to pounce upon passing bacteria may be attractive, but does not fit in either with our knowledge of what happens when they are removed, or with the fact that the lymphocyte is not an actively phagocytic cell.

The same arguments apply, but with added force, to the lymphoid tissue present in the lower end of the ileum and the appendix. In old age the lymphoid masses (Peyer's patches) in the terminal ileum have, as a rule,