BRITISH MEDICAL JOURNAL

LONDON: SATURDAY, MAY 9th, 1936

ADHESIONS OF JOINTS AND INJURY*

BY

R. WATSON JONES, M.Ch.Orth., F.R.C.S.

LECTURER IN ORTHOPAEDIC PATHOLOGY, LIVERPOOL UNIVERSITY; ORTHOPAEDIC SURGEON,
LIVERPOOL ROYAL INFIRMARY

The conditions under which adhesions form round joints constitute one of the fundamental problems of fracture treatment, and a problem which is frequently misunderstood.

On what grounds can early active movement of joints be urged with the object of preventing stiffness, when at the same time absolute immobility of the joints near the fracture is insisted upon? Why in some circumstances may joints be immobilized without becoming seriously stiff, whereas at other times dense adhesions form even although the joints have not been completely immobilized? Why can some joints be immobilized for a year with impunity, and yet others become permanently stiff within a few weeks? Why does the wrist-joint become so much more stiff when immobilized in a cock-up splint than when immobilized in a dorsal plaster splint? Why do the stiff knee- and the stiff shoulder-joint sometimes react well to manipulation and sometimes badly? Why does massage and passive stretching always aggravate stiffness of the finger- and elbow-joints? These are some of the many questions which arise in considering the relative responsibility of immobility, of injury, and of other factors in promoting adhesion formation, and in determining the probable response of adhesions to massage and passive movement, to manipulation, and to active exercise.

The Nature of Joint Adhesions

The surgeon who recently admitted that he had explored an elbow-joint in order to divide the adhesions was obviously less familiar with the elbow than with the abdomen. It is true that bands of fibrous tissue may sometimes be seen between the surfaces of a joint where infection has destroyed the articular cartilage, but healthy articular cartilage is rarely if ever the site of adhesion formation. The joint adhesions associated with injury are almost entirely peri-articular, and they involve mainly the plications of the joint capsule. The term "adhesion formation" conveys a less accurate picture of the pathological process than the term "adherence of capsular plications." The adhesive substance which is responsible for the gumming together of adjacent tissues is provided by any sero-fibrinous exudate. In the early days it is the fibrinous deposit which is responsible; in the later stages the fibrin is replaced by a young connective tissue and ultimately by a fully formed fibrous tissue. All of these stages may be described as "adhesion formation."

The impression that adhesions can only form as the result of injury or infection of a joint is quite erroneous. It is true that when the joint capsule is torn exudate is poured out from the torn edges. Moreover, when there is a traumatic or infective synovitis the inflam-

matory exudate involves the capsular tissues and causes adhesion of the plications. But other factors may be responsible. Quite frequently the joint itself is perfectly normal, and the source of the exudate is entirely extra-articular. There may have been a spreading exudation from a distant injury or infection; there may have been recurrent oedema of adjacent tissues; or simply venous stasis and congestion due to the muscular inactivity of immobility and disuse. The potency of these factors in promoting adhesion formation depends upon the degree of exudation and the fibrin content of the exudate. It also depends upon the frequency with which the soaking is repeated. In fact it is the recurrence and persistence of sero-fibrinous exudation which provides the key to the problems of adhesion formation. If the tissues of a joint are soaked day by day in exudate adhesions will form whether immobility is complete or incomplete and whether the joint is injured or normal. Even a severe fracture of the elbow-joint where the injury is not repeated will cause less adhesion formation than the relatively trivial injury of passive stretching repeated day after day by an over-enthusiastic masseuse. Adhesions form less readily round the ankle-joint which is immobilized in a walking plaster than round the same joint when it is left free to move but is subject to recurrent oedema.

The Adhesions of Immobility

Immobility in itself, therefore, is not a major factor in the development of adhesions. It is not true to go so far as to say that "rest of a normal joint in a normal position cannot give rise to adhesions."¹ When an upper limb plaster spica is applied for an abnormality of the shoulder and includes the normal elbow- and wrist-joints, even though functional activity is maintained so far as possible by active movement of the fingers, the wrist-joint has only half of its normal range of movement on the day that the plaster is removed. There has been some degree of venous stasis with engorgement of the tissues, and the capsular plications which have been in constant contact have become slightly adherent. In such cases the adhesions are so light that they rapidly stretch with the patient's own exercise. Even if the joint has been immobile for many months a normal range of movement may be expected within a few weeks. Only when the immobilization is associated with other factors do the adhesions become dense and resistant.

The Adhesions of Immobility and Injury

When a joint has been injured there is an obvious source of fibrinous exudate in the effusion and haemorrhage from the synovial membrane, the fractured bone, and the torn capsule and periosteum. A much greater

* A British Medical Association Lecture delivered to the North Wales Branch at Bettws-y-Coed, October, 1935.

degree of adhesion formation might be expected from the immobilization of such a joint than from the immobilization of a normal joint. Actually, however, the initial exudate rapidly absorbs, and provided it does not recur through infection, through the institution of movement at too early a date, or through the practice of passive stretching movement, we still find that the adhesion formation is not of serious degree.

It is constantly found in fractures of the carpal scaphoid bone where the wrist has been immobilized for six or twelve months or even longer that if functional activity has been continued throughout that time the wrist-joint can be painlessly moved through at least one-third of its normal range on the day that the plaster is taken off. Similarly, when even a severe fracture-dislocation of the ankle-joint is treated in a walking plaster so that there has been no recurrent oedema, no functional disuse, and no other factor than a single injury and immobility, a very free range of movement can be demonstrated in the ankle- and foot-joints immediately the plaster is removed.

The stiffness in such cases is only slightly greater than it is in the case of a normal joint which has been immobilized. Moreover, the adhesions that have developed are still fairly light, and there is complete recovery of movement within a few weeks by the patient's own active exercise.

The Adhesions of Immobility and Injury with Other Factors

What then are the factors which convert the slight adhesion formation of immobility into serious adhesion formation? Under what circumstances does the recoverable stiffness of an injured immobilized joint become an irrecoverable stiffness? The factors are those responsible for continued or recurrent exudation, and several may occur together in the same case: (1) Disuse with continued venous stasis. (2) Recurrent oedema. (3) The recurrent trauma of daily passive stretching or repeated manipulation. (4) The constant trauma of immobilization in a position of strain. (5) Continued infection near a joint. (6) The continued irritation of foreign bodies near a joint.

The Influence of Disuse

There can be no doubt that the addition of functional disuse to immobilization is an important factor in promoting adhesion formation, whether the joint has been injured or not.² The slight adhesions of simple immobilization are due to the tendency to venous stasis. If in addition to immobility of the joint there is complete immobility of the muscles of the limb, it is obvious that the delay in venous return and the stasis and engorgement of the tissues will be of much greater degree. It is for this reason that the wrist-joint immobilized by means of a cock-up splint in the palm of the hand which interferes with functional activity becomes more stiff than the same joint immobilized for the same period in the same position by means of a dorsal plaster cast which allows the patient to go on using the hand. The difference in the circulation of the wrist may be confirmed by comparing the decalcified bones of the first case with the normally calcified bones of the second. Such disuse decalcification of the bones of a joint is always associated with considerable adhesion formation; the more severe the degree of decalcification the more severe is the stiffness of the joint.

In commenting on the recent report³ of the British Medical Association Committee on Fractures, one writer⁴ criticized the suggestion that in upper limb fractures stiffness of the fingers could result from immobility and disuse alone, on the grounds that in the same report it was urged that the joints near the fractured bone should be completely immobilized for a long period. The reply,

which is quite obvious, is that adhesions form round all joints in which there is immobility and disuse, whether the joint is near to the fracture or distant from it, and the fact that in the treatment of a forearm fracture temporary stiffness of the elbow and wrist may be inevitable is no justification for unnecessary stiffness of the finger-joints. But the obvious reply does not go far enough. Active movement of the fingers has an effect not only on the finger-joints themselves but also on the forearm muscles and the circulation of the limb. In contrast with passive movement, which is relatively useless, active exercise involves contraction of the muscles, acceleration of the circulation, and diminution of the venous congestion of the wrist- and elbow-joints. If stiffness of the fingers is prevented, stiffness of the wrist is reduced and minimized. On the other hand, no patient is ever seen with severe post-immobilization stiffness of the fingers and normal movement of the wrist. It is the fact that muscular activity of the forearm can still be maintained through movement of the fingers, even although the wrist and elbow are completely immobilized, which allows these joints to be immobilized for long periods without becoming seriously stiff.

When there is a fracture of a phalanx or a dislocation of an interphalangeal joint and the finger itself must be immobilized the position is quite different. It is no longer possible to immobilize completely and at the same time to maintain sufficient functional activity for a normal circulation through the finger. This is at least one of the reasons for the particular susceptibility of the finger-joints to stiffness. The adhesions can be minimized by retaining full movement of the other fingers, but, unlike the wrist-joint, the adult finger-joint cannot be immobilized for twelve or eighteen months without becoming permanently stiff. The duration of immobilization must be minimized, and fortunately it is seldom necessary to delay active movement for more than a few weeks.

The Adhesions of Recurrent Oedema

Swelling of a limb has so long been acknowledged as one of the signs of injury that it has been accepted as inevitable, and the serious and harmful influence of oedema when it is associated with disuse and immobility of joints has passed almost unrecognized. And yet there is no more potent factor in adhesion formation, for oedema is the "glue" of which adhesions are made.

Fractures of the lower forearm are sometimes associated with severe swelling of the fingers. The natural instinct of the alarmed patient is to maintain absolute immobility of the fingers and hand. Frequently the practitioner shares the patient's fear; he accepts the swelling as an index of the severity of the injury and prescribes rest until it has subsided. Exactly the opposite treatment is urgently indicated. Adhesions are forming hourly, and the more swollen the fingers the more imperative is the indication for active movement. The fracture must be reduced and immobilized at once, so that finger movement may be practised painlessly, and the patient must be urged to bend the fingers as fully as possible into the palm of the hand, and to continue the active exercise for not less than ten minutes hourly throughout the day. If the swelling is of more than minimal degree, in addition to continued active exercise, complete recumbency with the forearm and hand elevated between pillows should be insisted upon for a day or two, in order to reverse the effect of gravity. Cutting the plaster down the sides is rarely of value, because in such cases the oedema is a reaction to trauma and to gravity, not to pressure, and it will appear wherever the surface of the limb is unsupported.

It is well known in lower limb injuries that the sudden removal of the rigid external support of a plaster cast is

followed by a recurrent gravitational oedema of the leg, which gradually increases during the day and subsides during the night. The tendency to oedema only disappears when the distended tissue spaces are obliterated, and when the muscle tone of the limb has recovered through active exercise. This recovery may be delayed for several months, or even indefinitely, if the limb is allowed to swell. The vessels remain dilated, the tissue spaces remain patent, every swelling of the limb encourages further swelling, and a vicious circle may be established. This is the type of case in which adhesions always develop round the ankle-joint. The daily soaking of the tissues follows a period of immobility and disuse, and both the disuse changes and the weight of the oedematous limb limit active exercise. The ankle-joint, which is already slightly stiff, becomes steadily stiffer. On the other hand, if an elastic dressing is applied immediately the plaster is removed, and is kept in position while the patient walks and practises exercises until in about six weeks' time the tendency to oedema disappears, the slight stiffness of the joint rapidly goes.

It is obviously wrong to manipulate any joint for adhesions while the limb is still subject to recurrent oedema. Even with the relatively slight and more localized oedema which gives rise to the sensation of "woody thickening" of the ankle, and especially of the knee-joint, manipulation is always followed by recurrent adhesion formation. Not only does the oedema produce fresh adhesions, but, since the adhesions themselves are oedematous, there is a greater exudation when they are torn than if the circulation is first restored to normal. The swelling must be controlled by external pressure and exercise, and if any adhesions still remain a manipulation may then be successful.

Unnecessarily prolonged disability is frequently seen in lower limb injuries where the toes are allowed to become rigid in the clawed position through the adhesion formation of recurrent oedema. Flattening of the transverse arch with correctable clawing of the toes is a common posture of relaxation in the adult foot. If such a foot is put in a plaster which does not extend beyond the metatarsal heads the toes remain at rest in the clawed position, and, being unsupported, become oedematous, and adhesions develop. The patients are frequently seen after many months with a united fracture of one leg, symptomless mobile transverse flat-foot on the opposite side, and painful rigid transverse flat-foot on the same side. The pain beneath the metatarsal heads, the painful hammer-toes, and the secondary disuse oedema account for several months of further disability, and operative treatment may be necessary. The complication may be avoided by extending the plaster to the tips of the toes on both dorsal and plantar surfaces with the toes in the neutral position, so that although there is immobility there is no recurrent oedema. Alternatively, the plaster may be extended beyond the toes on the plantar surface but cut away on the dorsal surface, so that regular toe exercises may be practised.

Two years ago there was a vigorous discussion in the correspondence columns of this *Journal* on the treatment of sprained joints and torn ligaments. Some writers,⁵ impressed with the frequency of adhesion formation after simple ankle sprains, urged immediate deep massage and energetic movements. Others⁶ pointed to the teaching of Hilton and of Robert Jones, and appealed for rest of the torn structures. The truth clearly lay in a combination of the two views. The factors responsible for adhesion formation after an ankle sprain are oedema and immobility. Both factors remain if there is no treatment except rest. On the other hand, the traumatic exudation and oedema may be increased by immediate passive stretching and vigorous massage. The first step, therefore, is to prevent

swelling and oedema, and firm elastic strapping or bandage with adhesive felt pads occupying the joint hollows is more effective than massage. The second step is to prevent immobility, but by the painless *active* exercises of the patient himself, and not by the forcible passive stretching of a masseuse.

Adhesions due to Massage and Passive Stretching

"Massage and movements" is frequently prescribed as a treatment for stiff joints; actually it is a very common cause of stiff joints. When the adhesions that have already formed round a joint are violently stretched there is a reactionary exudation and oedema which is capable of producing fresh adhesions. If this treatment is repeated day after day a continually recurrent traumatic exudation results, and although each day some adhesions are stretched many more develop. Such treatment delays the recovery of movement in every joint. The weight-bearing lower limb joints being less susceptible to trauma suffer the least, and they may ultimately regain their movement despite it. On the other hand, the upper limb joints, which are designed for mobility and not for stability and which are far more susceptible to injury, become steadily stiffer so long as the treatment is continued.

The elbow-joint is the commonest site for adhesions due to physiotherapy. It is generally recognized that passive stretching of the elbow may cause myositis ossificans, but it is less well known that the treatment *always* defeats its own object, and that it increases the very stiffness for which it was prescribed. A patient sustains a slight elbow injury followed by stiffness, which slowly recovers until massage is begun; the recovery at once ceases and the joint again stiffens. Week after week such cases are sent to consultants: the only advice given is to stop the massage, to stop the carrying of buckets of water which may have been recommended, and in fact to stop all treatment. Improvement immediately follows. If the treatment is not stopped there is not only delay in recovery but limitation of movement may remain permanently.

Cases are still to be seen, even in the hands of qualified masseuses, of the reprehensible practice of forcing elbow-joints day after day. It is no solution to the problem to be told that this is an illustration of the abuse and not of the use of massage,⁷ for lesser degrees of force are applied by even the best-trained masseuses, who, in their enthusiasm, seem to be quite incapable of resisting the temptation to "push" an elbow-joint which is slow in recovering movement. Six years ago I⁸ reported the results of 450 elbow injuries which showed that the slightest degree of passive movement aggravated the stiffness, and that any treatment, even massage alone, delayed recovery. It is illuminating to find that the unqualified bone-setter and manipulator will usually refrain from treating an elbow which is stiff after injury, and will frequently refer it to a qualified surgeon. The only safe procedure, therefore, is to allow no patient with a stiff elbow to go near a masseuse or a massage department. Movement must be allowed to recover at its own rate, and beyond urging the patient to practise his own active exercise no attempt must be made to accelerate recovery.

All that has been said of the elbow applies with equal force to the finger-joint. Massage, passive movements, and stretching have no place whatever in the treatment of the stiff finger, and if such treatment is continued for a few weeks the finger will remain permanently stiff. Similarly, if accurate measurements are recorded, it will be found that the stiff shoulder recovers more rapidly if the patient's own exercise is relied upon than if it is treated by massage and passive stretching.

Joint Manipulation as a Cause of Adhesions

It is too generally assumed that if there are adhesions round a joint a manipulation will cure them. Manipulation is a two-edged sword. The procedure will undoubtedly break down the adhesions, but that very process gives rise to a new exudation from the torn fibres, which can produce fresh adhesions. The new adhesions cannot be prevented by vigorous and forcible after-treatment, for, as we have shown, the trauma of such treatment may produce still further adhesions. Indeed, the balance between adhesions cured and adhesions produced may be a very delicate one.

The very localized adhesions of certain joints which cause discomfort and weakness rather than limitation of movement represent almost a different disease, so far as treatment is concerned, from the diffuse adhesions which actually limit the range of movement. In the tennis-elbow, the sprained back, the twisted knee and ankle, and the painful flat-foot the adhesions are so light that there is usually no demonstrable limitation of movement. In such cases the reaction to manipulation is minimal, and nothing but good results from the treatment. At the other end of the scale are the adhesions which cause actual stiffness of the elbow- and finger-joints. It is probably not an overstatement to claim that no stiff elbow or stiff finger has ever been improved by a manipulation under anaesthesia. The practice is only sustained by surgeons who do not actually measure the range of joint movement. If accurate records are kept it is found that the stiff elbow and the stiff finger always become much stiffer after manipulation, that many weeks elapse before the joint recovers the movement it had before manipulation, and that if the manipulation is repeated several times there is actual loss of movement and permanent stiffness.

The shoulder- and knee-joints are less susceptible to trauma than the elbow- and finger-joints, and manipulation sometimes increases the range of movement. The response to manipulation depends on the nature of the adhesions (whether localized or diffuse), the type of manipulation (gentle or forcible), and the after-treatment (active or passive). If a stiff shoulder is manipulated and there is no audible snap of localized adhesions but a gradual giving way of a solid mass of diffuse adhesion the manipulation will do no good and will probably aggravate the stiffness. If, on the other hand, the adhesions are heard to snap and are obviously localized, the prognosis is good, provided that too much is not done at one time. It is far better to perform two or three very gentle and incomplete manipulations at intervals of not less than six weeks than to perform one severe manipulation which must be followed by a severe reaction. Finally, the beneficial result of a manipulation may be neutralized if it is followed by repeated passive and forcible stretching.

Effect of Immobilization in a Position of Strain

Joints stiffen more readily if they are immobilized in a position of strain than if they are immobilized in a position of rest.⁹ If the fingers are badly splinted so that the metacarpo-phalangeal joints are continually forced into hyperextension the stiffness which results is very intractable. This is another example of the factor of recurrent exudation, for the strained position is the equivalent of a constantly repeated traumatic synovitis. This does not apply, however, to any position which is within the normal range. There is no reason at all why certain fractures of the elbow should not be treated in extension, some fractures of the ankle in equinus, or fractures of the spine in full extension. The factor is probably not of very great importance, but whenever possible the position of immobilization of a joint should be a few degrees within the normal limit of movement.

Continued Infection near a Joint

The commonest example of adhesion formation round joints which though not themselves infected are near to a focus of infection is seen in the septic hand. All the factors of adhesion formation are present. The infection causes a spreading serofibrinous exudation, there is recurrent oedema of the whole hand, the pain and swelling warn the patient not to move his joints, and frequently the principle of immobilization is applied with far too much enthusiasm. Too often the stiffness of the finger which has actually been infected represents a very minor part of the ultimate disability, and the real crippling has arisen from the general adhesion formation in fingers which originally were quite normal and had never been infected. It is obviously essential in such cases to control the infection as rapidly as possible by suitable drainage, and when a finger is likely to be irreparably damaged by early rather than by late amputation. It is equally important to control swelling and oedema by elevation of the limb and by avoiding frequent dressings and antiseptic baths for prolonged periods. Above all, if immobilization is used at all it must be confined to the finger actually infected, so that active movement of the other fingers may be practised throughout.

Continued Irritation of Foreign Bodies near Joints

Metallic foreign bodies frequently give rise to irritative changes in adjacent tissues associated with a recurrent exudation. This is particularly the case where the foreign body penetrates the skin so that there is also the possibility of a low-grade infection of the track. Skeletal extension pins which are left in for many weeks are associated with this danger. The infection may be of minimal degree, and may show no external evidence, or at the most a few granulations with slight serous discharge. Even this degree of infection accounts for continued exudation in neighbouring tissues, and if the pin has been driven through the supracondylar region of the knee-joint at the level of the synovial reflection dense adhesion formation may result. The adhesions that develop are much more resistant than the adhesions of simple immobility. Not infrequently the stiffness remains permanently, and this is true even in cases where the supracondylar skeletal extension has been associated with early mobilization of the knee-joint. Even if right-angled movement has been retained from the beginning the remaining movement never recovers, whereas in cases in which there has been no mobilization but no supracondylar infection, although the joint is very stiff three months after the fracture, the ultimate recovery is complete.

The bigger the wound at the site of insertion of the pin the greater is the danger, and adhesion formation occurs most commonly with the ice-tongue calliper, less often with the Steinmann pin, and least commonly with Kirschner wire. An extension pin driven through the lower part of the tubercle of the tibia is relatively safe, for even if slight infection does develop it is too remote from the periarticular tissues to cause joint adhesions. If an extension pin is left for many weeks in the heel, above the os calcis or through the bone, a similar adhesion formation may develop round the subastragaloid joint, and the recovery of inversion and eversion movement is delayed or even permanently limited. Probably the most dangerous situation of all is the olecranon. The elbow-joint is particularly susceptible to adhesion formation, and full extension very rarely recovers after several weeks of traction by an olecranon pin.

The same factors account for the frequency of permanent stiffness of joints when articular fractures are treated with wire and screws. Fractures of the olecranon may be

perfectly reduced by operation, the wound may heal without obvious infection, and passive movement and manipulation may be avoided in a blameless after-treatment, and yet, if wire is used for internal fixation, there is usually permanent limitation of extension movement. The wire is associated with a low-grade inflammatory reaction, and the continued irritative serofibrinous exudation gives rise to periarticular adhesion formation which is particularly resistant. In these fractures it is quite unnecessary to use anything stronger than catgut, which absorbs so rapidly that it does not give rise to adhesion formation. Similarly in the replacement of epicondylar fragments to the humerus, of malleolar fragments to the tibia, and in the repair of comminuted fractures of the lower end of the humerus and upper end of the tibia, plates, screws, and wire should always be avoided. The rustless and stainless metals recently introduced produce less reaction than other metals. Nevertheless, even these metals should be used with caution, because some reaction does occur after two or three months. In fractures of the neck of the femur the effect of a stainless steel nail is minimized by its distance from periarticular tissues. Even so, it appears advisable to remove the nail in all cases as soon as the fracture is firmly united.

Conclusion

I would repeat, then, that fractures should be perfectly immobilized by complete fixation of adjacent joints, so that active movement of distant joints may be practised painlessly. Patients must be definitely instructed in joint mobilization; briefly, as follows:

- Colles's fracture: Fix wrist; actively move fingers, elbow, and shoulder.
- Fractured shafts of the radius and ulna: Fix elbow and wrist; actively move fingers and shoulder.
- Fractures of the elbow: Fix elbow; actively move fingers, wrist, and shoulder.
- Fractured shafts of the humerus and shoulder: Fix shoulder and elbow; actively move wrist and fingers.
- Pott's fracture: Fix ankle; actively move toes, knee, and hip.
- Fractured shafts of the tibia and fibula: Fix ankle and knee; actively move toes and hip.
- Fractured shaft of femur: Fix hip and knee; actively move ankle and toes.
- Fractured neck of femur (with nail): Actively move hip, knee, ankle, and toes.

Summary

1. Joint stiffness after injury is due to the adhesion of capsular plications, and this may result from the organization of any exudate in the periarticular tissues.
2. The adhesions of immobilization, uncomplicated by other factors, are temporary, and recover by the patient's own exercise.
3. The factors which convert the recoverable stiffness of injured immobilized joints into irrecoverable stiffness are those responsible for a continued or repeated serofibrinous exudation: (a) disuse with continued venous stasis; (b) recurrent oedema; (c) repeated trauma of passive stretching; (d) continued infection near a joint and irritation of foreign bodies near a joint.
4. Fractures should be perfectly immobilized by complete fixation of adjacent joints, so that active movements of distant joints may be practised painlessly.

REFERENCES

- ¹ *British Medical Journal*, 1932, i, 542.
- ² *Ibid.*, 1932, i, 1073.
- ³ *Supplement, British Medical Journal*, 1935, i, 53.
- ⁴ *British Medical Journal*, 1935, i, 616.
- ⁵ *Ibid.*, 1923, i, 135; 1933, i, 391.
- ⁶ *Ibid.*, 1933, i, 247; 1933, i, 340.
- ⁷ *Lancet*, 1932, ii, 1217.
- ⁸ *Proc. Roy. Soc. Med.*, 1930, xxiii, Sect. Orthop., 23.
- ⁹ *British Medical Journal*, 1931, ii, 1019.

TREATMENT OF PARALYTIC ILEUS

BY

R. L. HOLT, F.R.C.S.

HONORARY ASSISTANT SURGEON, CHRISTIE HOSPITAL; CHIEF
ASSISTANT, SURGICAL UNIT, MANCHESTER ROYAL
INFIRMARY

The object of this paper is to outline a treatment of paralytic ileus which I have used as a routine during the past two years with consistently good results. In the past a variety of therapeutic measures have been recommended in the treatment of this condition and many of them have enjoyed a certain measure of success. But paralytic ileus still remains one of the most harassing post-operative complications which the surgeon is called upon to treat. The condition begins as an inhibition of intestinal tone and motility as a result of injury of the neuro-muscular mechanism or of inhibitory impulses from the spinal cord and coeliac plexus. In the early stages the intestinal muscle is capable of contracting if suitably stimulated. In the later stages, perhaps as a result of overstretching and circulatory disturbances, the intestinal muscle loses its contractile power and does not readily regain it.

The common method of treatment is to use some drug or measure which under normal conditions will augment intestinal peristalsis. Many cases of intestinal paresis may be resolved by the timely use of such drugs as pituitrin, eserine, acetylcholine, etc., but this is not surprising when the many grades in which this condition occurs are considered. The fact remains that many other cases do not respond to "stimulant" measures, and frequently the conclusion cannot be escaped that the continued use of such measures has an ill effect on the progress of the patient. The too literal adoption of the old dictum "Get the bowels open and the patient will live," which has long formed the basis of the treatment of paralytic ileus, is likely to kill more patients than it will save.

Physiopathology of Paralytic Ileus

Whatever the cause of the condition, the essential clinical features are a distension of the intestinal tract—usually most marked in the small intestine—and a complete or almost complete absence of intestinal movements. Vomiting is not so marked a symptom in paralytic ileus as in mechanical obstruction, and unless the patient has been given large amounts of fluid by mouth the intestinal distension in paralytic ileus is largely due to gas; there is an absence of those typical splashing sounds which are obtained on abdominal palpation in cases of mechanical obstruction. There seems little doubt that in paralytic ileus there is a depression of more than one function of the intestinal tract; muscular activity, absorption, and perhaps the most important of all, gas exchange, are all affected. But what is the effect of intestinal distension on the body as a whole? What are the lethal factors in paralytic ileus?

The results which may be attributed directly to distension of the small intestine are very imperfectly understood. It is generally believed that toxic products are absorbed from the contents of distended loops of intestine in sufficient amounts to cause death. Some recent articles^{1,2} have cast doubt on the truth of this view, and I have just completed a series of experiments in which I failed to find any evidence whatever to support this theory, except, of course, in the presence of strangulation.

The most noticeable changes, apart from distension, presented at operation in the obstructed loops of gut are the cyanotic tinge, the engorgement of the blood vessels, and the thick oedematous intestinal wall. Several writers