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.

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TREATMENT OF PARALYTIC ILEUS

BY

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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

have drawn attention to the large accumulation of fluid in the splanchnic area in patients dying of paralytic ileus. I have found experimentally that the most striking change which takes place in loops of intestine in which distension develops gradually is the large amount of tissue fluid which fills the distended tissue spaces of the various layers forming the intestinal wall. I believe that the most serious factor in cases of ileus is the circulatory failure which results from the stagnation of blood and tissue fluid in the distended loops of gut and in the peritoneal cavity. The circulation is further embarrassed by the amount of fluid lost by vomiting. The logical treatment of paralytic ileus, therefore, would appear to consist in:

1. Relieving the distension of the small intestine.
2. Maintaining the circulation.

Effects of Peritonitis

The cases of paralytic ileus commonly seen by the surgeon can be divided into two groups, according to whether a peritonitis is present or not. Obviously in the group associated with peritonitis the results of treatment cannot be expected to be as good as those of the other group, since the peritoneal infection adds a complicating factor which may be sufficient in itself to cause death. Often the peritoneal infection is not such as to cause death by itself, and in these cases it is the combined effects of peritonitis and paralytic ileus which prove fatal—often the latter is the more important factor. In cases of peritonitis I merely claim that the effects of any associated paralytic ileus can be controlled by the methods to be described.

Prophylaxis and Drug Therapy

Paralytic ileus is frequently the result of a faulty surgical technique. Many cases can be traced to excessive packing off and manipulation of the small intestine, which should be avoided, especially in the presence of a peritonitis. The incidence of ileus can be lessened by adopting the following post-operative measures in cases in which there is believed to be any danger of this condition developing.

The importance in such cases of keeping the intestinal tract as completely rested as possible cannot be too strongly emphasized. Purgatives, enemata, etc., are definitely contraindicated.

No fluid whatever is allowed by the mouth, which is kept fresh by frequent mouth-washes, and by chewing-gum, or, better still, by raw pineapple.

Normal saline is given in sufficient amount (usually 6 to 8 pints) by any of the available routes, but preferably intravenously. Morphine is given in doses of 1/6 grain every six hours.

In the absence of the development of intestinal distension water may be given by mouth on the third day, beginning with small quantities, say, one ounce hourly.

The usual outcome of this treatment is a very smooth post-operative period, and almost without exception the bowels are moved naturally on the fourth day.

I do not intend to discuss the use of drugs at any length. It has already been indicated that the timely use of such drugs as pituitrin, eserine, acetylcholine, etc., may produce good results. Therapeutic measures designed to stimulate intestinal peristalsis may be tried once, but if they fail they should not be repeated. Some three years ago I had occasion to operate on a case of paralytic ileus due to obstruction of the terminal ileum by a band. A spinal anaesthetic was used, and at no time during the operation was there any evidence of peristalsis, nor could any muscular contraction be evoked by the usual methods of stimulation. These findings have since been confirmed on several occasions. It seems hardly

credible that any result can be expected from the use of drugs in such cases.

Relieving Distension by Decompression

A few words might be inserted here about the vexed question of enterostomy. Drainage is often limited by kinking of the gut, and a virulent peritonitis is not an unusual sequel to this operation. There is no doubt that enterostomy has saved many lives, but too often it is unsatisfactory. In paralytic ileus an enterostomy drains very poorly, and I believe that its use has been entirely superseded by the measure described by American writers^{3, 4, 5} under the term "intestinal decompression." This method is more effective than an enterostomy and has none of its risks.

The principle of the method is the application of continuous mild suction to an indwelling tube in the stomach or duodenum. It has been shown experimentally that the intestinal tract can be drained as far as the ileo-caecal valve by such methods; I have been able to verify this at operation in one case.

CONTINUOUS SUCTION APPARATUS

The apparatus used is shown in the accompanying diagram; it is somewhat simpler than that described by Wangenstein and Paine.⁶ It consists of a duodenal tube which is passed by mouth or through the nares. This is attached to a

T-piece, one limb of which is attached to a reservoir (A) containing tap-water and placed at a height of about 2 feet above the patient; to the other limb is attached a piece of rubber tubing, which drains into a bottle (B) on the floor by the bedside.

It is important that the end of this tube should be below the level of the fluid in the bottle (B). The difference in height between the open end of the duodenal tube and the level of the fluid in the bottle (B) supplies by siphon action the continuous mild suction necessary. It has been found in practice that the best results are obtained when the difference in these two levels is about 2 feet to 2 feet 6 inches.

Air is removed initially from the apparatus by filling it with water from the reservoir (A). The clip (C) is then closed, clip (D) is left open, and the stomach contents begin to siphon off. The fact that siphonage is actually taking place should be verified frequently, since the duodenal tube from time to time gets blocked with mucus and debris. It is usually unnecessary to remove it, for it can generally be washed clear by water, to which sodium bicarbonate may be added, from reservoir (A). In this way the intestinal con-

tents are removed, and it will be found that the abdominal distension gradually decreases. As a rule the patient will be found to tolerate the indwelling duodenal tube very well; nevertheless I prescribe as a routine 1/6 grain morphine six-hourly.

The patient obtains prompt relief from vomiting and from the discomfort due to abdominal distension. It is possible, and indeed preferable, to allow fluids by mouth as soon as the apparatus is functioning. It gives the patient a good deal of comfort and helps to keep the mouth fresh, and at the same time it does no harm, since the fluid is promptly removed from the stomach by the indwelling tube. This "intestinal decompression" is kept up until the abdominal distension has completely disappeared and the bowels have been opened naturally, an event which usually takes place about the fourth day after the treatment is begun.

Maintaining the Circulation

The circulation is best maintained by giving normal saline intravenously by the continuous drip method. About six to eight pints a day are usually sufficient for an adult, but the amount can be regulated by the quantity of urine passed, which should not be less than one and a half pints in twenty-four hours. The saline should be made up from treble distilled water, in which case it will be found that the drip can be kept up without any reaction or local phlebitis for a week or more, if necessary.

Once the bowels have moved naturally and the distension has disappeared the duodenal tube can be removed and the patient may be allowed to take small drinks by mouth, beginning with one ounce of water hourly for the first twenty-four hours. This is gradually increased and at the same time the amount of saline given is decreased. Any return of distension or vomiting is unusual, and is treated by reinserting the duodenal tube.

Results of Treatment

The immediate improvement in the patient's condition following the institution of this treatment is not spectacular. Vomiting is at once relieved and the discomfort due to the abdominal distension is lessened as the intestinal contents are siphoned off. The distension gradually diminishes, but the pulse rate does not fall appreciably during the first two or three days. In the majority of cases the condition is clearing up satisfactorily by the fourth day, but in other cases the period is prolonged, and two cases in my experience have gone eight and twelve days respectively before improving. It is apparent that the intestinal muscle does not recover its function rapidly, hence the importance of resting the intestinal tract as completely as possible during the recovery phase. For this reason I believe that purgatives, enemata, "stimulant" drugs, etc., are contraindicated in the treatment of the established case of paralytic ileus.

The results of this treatment in a short series of cases have been most encouraging. In ten cases of paralytic ileus not associated with peritonitis there has been one death; this occurred after the paralytic ileus had cleared up, and was due to a pulmonary embolus. The opportunities of treating cases associated with peritonitis have not been so numerous: five cases have been treated with one death—this patient, a girl with general peritonitis due to acute appendicitis, died with all the signs and symptoms of an acute peritonitis. Abdominal distension, which had been marked at operation, had almost disappeared under treatment.

Many other cases, not included in the above series, have been treated by my colleagues and by the senior residents at the Manchester Royal Infirmary with equally encouraging results.

Conclusion

A routine is described for the treatment of paralytic ileus which is based on the physiopathology of this condition.

The results have been a great improvement on those obtained by any other method which I have seen tried.

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CHRONIC MENINGOCOCCAL SEPTICAEMIA

BY

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Since Gwyn in 1899 first established meningococcaemia in a case of meningitis with arthritis, and Warfield and Walker described a case of meningococcal endocarditis with septicaemia in 1903, it has been gradually recognized that, in addition to the short initial invasion of the blood by the meningococci which probably precedes infection of the meninges in most if not all cases of meningococcal meningitis, meningococcal septicaemia may also occur in a subacute or chronic form lasting many weeks or months, in which the meninges may or may not be involved, the septicaemia being the striking clinical feature. This type is well recognized by writers about the war period, more particularly in the French literature. Sir Humphry Rolleston, in his Lumleian Lectures in 1919, said that intermittent meningococcal fever due to septicaemia might (a) last for weeks or months without any meningeal symptoms developing; (b) follow meningitis; (c) show transient meningitic symptoms; or (d) might terminate in meningitis. He stated that a correct diagnosis was seldom made in the absence of meningeal symptoms.

In spite of these descriptions it is felt that the more chronic forms of meningococcal septicaemia with their characteristic clinical features are not sufficiently often recognized, and do not at the present time receive generally the attention they merit, seeing that no fewer than three unconnected cases, which are described here, have occurred in the London Hospital during the last two years. These follow two further cases of a similar nature in the same wards, reported by Lawson Stote in 1929.

Case Reports

CASE I

A printer aged 25 was admitted to the London Hospital on March 31st, 1934, under Dr. Hutchison, with three weeks' history of an illness characterized at the onset by headache, and followed later by fever, pain in the legs, and tender, red cutaneous nodes on his limbs.

History.—Three weeks previously he had awoken in the early morning with severe frontal headache, which passed off after a few hours. He vomited once, felt weak, and stayed at home. During the next two days he developed spasms of gripping pain in the right heel, which occasionally radiated up the leg and back to the nape of the neck. He tried to work during the two succeeding days, but had a constant frontal headache, felt lethargic, and commenced to have pains in his knees and ankles. Since then he had been confined to bed with fever, profuse sweats, especially at night, pains in his legs, and severe headache during the first few days, the