Miss D., aged two or three years over 70, had sclerosed arteries and a mitral and aortic systolic murmur. She was having recurring headache, attacks of disordered vision which made her temporarily unable to read, and passing attacks of aphasia and of unilateral disturbances of sensation. The pulse resistance measured from 200 to 210 and 220 mm. By means of rest, careful dieting, and suitable medicinal management, aided by the understanding watchfulness of the lady's local doctor and her own intelligent co-operation, the pulse resistance was stabilized to round about 170 mm. But it took ten months to effect this.

Mr. S., aged 68, came to me in November last saying that he had "high blood pressure." He complained of listlessness and somnolence so great that whenever he began to read he fell asleep. The arteries were sclerosed and tortuous, and the pulse resistance to the armlet 210 mm. The heart apex was one and a half inches beyond the mammary line. The aortic second sound was accentuated. The urine was normal. He had always been of costive habit and took a daily laxative. He was not a big eater, but he drank burgundy, and suffered somewhat from gastric hyperacidity. Wine was stopped, the acidity was checked, and the laxative modified. The result was that the somnolence passed off, mental activity returned and he felt quite well, and the pulse resistance fell to 180 mm. He has continued to follow out instructions and the arterial condition remains stable.

It will be noted that in these three cases a stable pressure of 160 to 170 to 180 respectively—which was a lowering of 50 to 30 mm.—removed symptoms, and the patient kept well so long as instructions were followed. The arteries being sclerosed no attempt was made to lower the pressures further. Treatment was directed to removing and checking the hypertonia. The structural resistance of the thickened arterial wall could not be removed.

The foregoing cases illustrate the limitations of treatment when dealing with sclerosed vessels. The following case illustrates the progressive character of sclerosis when its early manifestations are not understood.

Mr. B. was seen by me in 1913 for digestive disorders. He was then 44 years of age. His pulse resistance to the armlet was 170 mm., the artery was thickened, the heart and urine normal. I did not see him or hear of him again until the end of February, 1924, when he was again sent to me. He was by this time 54 years of age. He complained of apathy and drowsiness; he could not remember what he had read in the morning newspaper half an hour after he had read it. The radial artery was small and calcareous. The pulse wave was moderate. The brachial artery was hard and tortuous, the pulse resistance was 240 mm. The sounds of the heart were faint. There was a trace of albumin in the urine. Such was the advance of the arterial changes, with their accompaniments, in the ten years between the ages of 44 and 54. The immediate prognosis, of course, was very bad.

CONCLUSION.

I have mentioned Trunczek's definition of vascular hypertension. He recognizes, as I have long preached, that the raising of aortic pressure results from constriction of the arterial wall and consequent diminution of the content of blood in the constricted arteries. He further recognizes that the true blood pressure is determined by the response of the left heart to the increased aortic pressure and that the left heart may become embarrassed and commence to fail, so that the arteries may be constricted without elevation of the blood pressure. The arterial constriction he attributes to blood composition, regarding it, as I have suggested elsewhere, as due to acidosis, traceable to "la dyspnée cellulaire." I frequently find that the application of the doctrine of acidosis is effective in some of the clinical manifestations of arterial hypertonia, both when the arteries are sclerosed and when they are normal. Whatever the precise nature of the blood impurity, it is more than a vaso-constrictor, for it leads to obliterative endarteritis in the kidneys and to capillary occlusion.

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C

ACUTE APPENDICITIS AND INTESTINAL OBSTRUCTION.

BY

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THE occasional occurrence of intestinal obstruction as a complication of acute appendicitis has long been recognized, and there must be very few abdominal surgeons who have not had to face this sinister association many times. It is difficult to estimate with any accuracy the frequency of the complication: if we examine the terminal causes of death in fatal cases of acute appendicitis, as revealed in the published lists of individual surgeons, we cannot fail to notice a considerable discrepancy in the incidence of ileus and general peritonitis, though the mortality rates in these lists differ but slightly; nor is it possible for us to obtain any clear notion as to the type of ileus, whether mechanical or paralytic, in many of these cases. The same uncertainty in regard to ileus is encountered when we examine the non-fatal complications of acute appendicitis.

In the light of my own experience I believe ileus, and that frequently of a mechanical type, is associated with acute appendicitis much more frequently than the literature would lead us to believe, and that some, if not many, of the deaths ascribed to general peritonitis or to intestinal paralysis are due primarily to mechanical ileus. McWilliams,¹ in describing a series of 687 cases of acute appendicitis (1906-9), states that ileus, mechanical or paralytic, developed to such a degree in 18 cases as to require secondary operation, and of these 14 died. On the other hand, Beekman,² in describing a more recent series of 500 cases of acute appendicitis with 34 deaths, ascribes the cause of death to ileus in only 2 of these 34; but in the same paper stated, "It must be assumed, however, that some of our cases of diffuse peritonitis had a paralytic ileus, and therefore the above statistics may be misleading" (18 of the 34 deaths were stated to be due to diffuse peritonitis). McNeill Love³ has recently reviewed a series of 1,500 cases at the London Hospital (1919-22), and in a set of 421 of these, in all of which the operation was performed through the gridiron incision and drainage established through this, he records 102 complications, and of these 17 were instances of ileus or intestinal obstruction (10 fatal) and 14 of general peritonitis.

Furthermore, we must not lose sight of the occasional and very unwelcome appearance of acute obstruction as a remote sequel of appendicitis; this may occur months or years afterwards, and is particularly prone to follow operations for acute appendicitis that have required drainage. It is noteworthy because the patient and even his doctor may be disposed to minimize the gravity of the trouble during its early stages, knowing that the most common cause of acute abdominal disease—the appendix—has been previously removed; yet many of these late attacks, two, three, or more years after the appendix operation, are due to strangulation of the bowel and call therefore for immediate recognition, whilst those developing some weeks or months afterwards are more commonly due to adhesions. McGlannan⁴ has recently recorded 276 cases of acute intestinal obstruction in 25 of which there had been a previous operation for acute appendicitis, and in 23 of these drainage had been employed.

It is, however, with intestinal obstruction as an immediate complication of appendicitis that I am concerned here, and I will now describe the pathological conditions, as seen at operation, responsible for obstruction. At least three distinct types can be recognized.

1. Adhesion by Lymph Exudate of Lower Ileum to Pelvic Wall or, less frequently, to Neighbouring Loops of Small Intestine.

This I believe to be the commonest cause of intestinal obstruction occurring before or soon after operation for acute appendicitis. This condition is most commonly set up by perforation or gangrene of the pelvic appendix; in

either case a lymph-forming exudate is poured out in the right posterior quarter of the pelvis; by this means a barrier, more or less complete, against the spread of infection is created, and in the formation of this barrier neighbouring viscera, particularly the terminal ileum, inevitably participate.

Sometimes a perforated appendix lying above the pelvis may by gravitation of its contents give rise to the same state. A loop of the terminal ileum at a point about 6 to 18 inches above the ileo-caecal valve is fixed to the posterior wall of the pelvis just to the right side of the rectum and at a varying depth in the pelvic cavity. The acute angulation—and rotation also in some cases—of the bowel at this fixed point can usually be readily revealed to the eye or, according to the depth of the point in the pelvis, appreciated by a cautiously inserted finger. The bowel above the fixed point is usually well filled, and may be distended if actual obstruction has already begun, and the efferent loop can be seen rising steeply out of the pelvis to reach the caecum; this loop is usually of normal calibre and empty. A typical example of this is described later (see Case II).

In other cases where there is diffuse peritonitis the terminal ileum may be found to be stuck to other loops of small intestine in such a way as to produce acute kinking at one or more points.

It is not suggested that lymph adhesion and angulation of the bowel of this type will, if unrelieved, invariably produce obstruction of noteworthy degree, but it is suggested that this is a common cause of obstruction, particularly if other related factors come into play; many patients with such a condition may weather through the post-operative stage with nothing more than a certain degree of "paralytic ileus," but a few of these will surely return to the surgeon with acute or subacute obstruction within a few weeks owing to the organization of the lymph coagula and a consequent increase in the distortion of the bowel.

2. Ileal Band.

The presence of a well marked ileal band is responsible, though rarely, for intestinal obstruction in acute appendicitis; the band contracts and becomes more rigid owing to acute inflammatory changes, so that its normal action in tethering down the bowel to the posterior abdominal wall, and in causing a rotation of the bowel, is so intensified that it holds the terminal ileum in a contracted and fixed condition; the freely mobile bowel above this is, in the presence of a spreading peritonitis, more or less distended and clearly demarcated from the contracted portion in the grip of the ileal band; the junction of the two under these conditions is often marked by a kink. The following case is an example of this type of obstruction:

CASE I.

A man, aged 50, was admitted to a nursing home in September, 1921, and operated upon the same day for localized suppurative appendicitis of five days' duration—appendix, behind mesentery of small intestine, removed: drainage. Four days after the operation he presented the typical picture of acute intestinal obstruction—abdomen distended; visibly distended coils of intestine showing some peristaltic movements; one small faecal-coloured vomit; no proper expulsion of flatus since operation despite enemata and use of tube; face pinched and dried up in appearance. Re-operation. Marked distension of small intestine, particularly of jejunal coils, which were like inert water bags; no evidence of peritonitis. Obstruction located at lower ileum and due to angulation produced by well marked ileal band; the distension ceased abruptly at the ileal band. Lateral ileo-colostomy (transverse colon), ileum drained before establishing anastomosis. The patient made a good recovery and required no aperient medicine; he is in good health to-day.

3. Ileus Duplex.

This condition of the terminal ileum was described by Sampson Handley⁵ in 1915 in his Hunterian lecture on "ileus duplex." This is due to involvement of the lower ileum in a pelvic peritonitis. "The coils of the pelvic ileum, bathed in pus, show evidence of acute inflammation of the bowel wall." "The bowel wall is swollen and oedematous. The affected coils are empty, passively contracted, and motionless, and between adjacent coils are

sharp angular kinks sometimes fixed by flakes of sticky lymph." Victor Bonney noted in an earlier description that the bowel is flattened like a strap.

I have recognized the condition of ileus duplex on two occasions at least, and the appearance of the terminal ileum in both at once arrested attention and created a profound distrust in the capacity of the affected bowel to transmit its contents; it appears to me that the lesion I have described in Section 1 is due essentially to the same processes as is the ileus duplex of Handley, and that between the two distinct types various intermediate forms occur in which the obstruction is in part due to fixation and in part to paresis from inflammatory infiltration of the bowel wall. When the condition of ileus duplex is encountered the second lesion, affecting the pelvic colon, must be kept in mind.

Handley's work on this subject has been of the greatest service to the surgeon in directing attention to the importance of the obstructive element in appendicular peritonitis, to the need for careful inspection of the terminal ileum, and thus to the possibility at the primary operation of preventing obstruction.

CLINICAL TYPES AND FEATURES.

Intestinal obstruction may occur at any period in the course of an attack of acute appendicitis except within, approximately, the first twenty-four hours, and we can conveniently distinguish, according to the time relationship, the following groups—pre-operative; post-operative; convalescent; and remote.

Pre-operative Group.

The distinctness of the clinical picture of intestinal obstruction varies greatly and depends on the duration and type of appendicitis. In some cases the evidence of obstruction may be so slight that the examiner may easily overlook it (though a careful consideration of the history should afford some indication), whilst in others the signs of obstruction predominate to such an extent that the primary and underlying disease may be overlooked.

The cases that present a fully developed picture of intestinal obstruction are those with a well shut-off abscess, usually in the pelvis; the history in these, therefore, extends over one or two weeks, and is occasionally marked by an interval of improvement, or apparent recovery, some three or four days after the onset and which lasts until the obstructive symptoms begin.

Some degree of pre-operative obstruction is commonly seen in pelvic appendicitis after the first forty-eight hours. It is important to bear this in mind, because the condition, if recognized, can be remedied at the primary operation. The following is a typical example.

CASE II.

A man, aged 50, was admitted to a nursing home in June, 1922. History: Onset forty-eight hours previously with severe abdominal pain and repeated vomiting. About eight hours after onset began to improve, pain became much less, and vomiting ceased; improvement continued for about twelve hours, then vomiting began again with pain of a gripping character and eructation of wind. Repeated enemata at home without result.

On examination the abdomen was distended and tense; no rigidity nor any tenderness; pulse 84; temperature 99.4°. Operation fifty hours after onset. Pelvic appendix with minute perforation at its tip sealed by plate of lymph; considerable turbid exudate in pelvis, lower ileum at a point 12 inches above ileo-caecal valve, fixed down to posterior wall of pelvis by lymph adhesions; bowel above this point distended and empty below it. The efferent loop filled up rapidly after setting free the ileum. The patient made an uninterrupted recovery.

Post-operative Group.

The patient in this group never makes really satisfactory progress in the first few days after operation, though during this time there may be nothing to create much anxiety. The abdominal distension gradually increases; purgatives produce no result except an increase in the discomfort of the patient. Enemata are not followed by satisfactory expulsion of flatus. Vomiting usually ceases after the anaesthetic nausea has passed off, but begins again about the third or fourth day and is commonly small in amount and infrequent at first. About the fourth to the sixth day the evidence of intestinal obstruction increases and the general

picture becomes unmistakable; the patient's facial appearance alters noticeably; the pulse shows little acceleration but its volume is diminished; the vomit, at first bilious, becomes intestinal or even faecal in character; the abdominal distension becomes marked, and in thin or moderately nourished patients the outline of distended coils of bowel can be seen. Frequent griping pains and sensations of wind rolling about the abdominal cavity disturb the patient. A secondary operation, under depressing conditions, appears inevitable, however loath the surgeon may be to perform this.

Convalescent Group.

In this group obstruction develops, approximately, between the tenth day and the eighth week. The group cannot be sharply distinguished from the post-operative or remote groups, but in general I may say that there is a certain degree of liability to obstruction during convalescence, and that the clinical and pathological features of obstruction in this period differ from those of obstruction in the other periods. Obstruction in this period is due essentially to an increase in the fixation and kinking of the bowel owing to organization of lymph deposits or to the shrinkage of an abscess cavity after drainage. Abscesses in the pelvic and hypogastric regions provide most of the examples of the last type; abscesses outside the small intestine area—for example, retrocaecal or retrocolic abscesses—very rarely give trouble of this kind.

The onset of obstruction during convalescence is sometimes distinctly subacute and gradual, and at such times the significance of the early symptoms may be overlooked. The prognosis is decidedly better in this than in the post-operative group.

TREATMENT.

(a) Primary Operation for Appendicitis.

The whole of this article constitutes an additional reason, if one were needed, for the early operation—that is, within the first twenty-four hours, when the diagnosis is clear and the circumstances reasonably favourable. After twenty-four hours have elapsed since the onset, or, more precisely, after perforation and spread of infection have occurred, then, if operation is decided on, the case, and particularly the history, should be examined for any evidence of the presence of an obstructive element. The incision employed should be one which will give easy access to the terminal ileum and pelvis. A vertical incision through the outer third of the lower rectus muscle, splitting the rectus muscle or displacing it medially, answers the purpose best in my own experience. After removal of the appendix and careful cleansing of its bed, the mobility of the last 12 inches or so of ileum should be examined, particularly in the presence of a pelvic peritonitis.

When the ileum is fixed in the pelvis as previously described, a finger should be passed gently down to the apex of the angle and the intestine slowly disengaged by pressure from below; this done the bowel can be readily lifted up into the wound, and the plate of lymph on its walls, which marks the site of the previous adhesion, is apparent. No bleeding at all should arise from this mobilization. The fixation of the bowel should not be overcome by pulling on the afferent or efferent loops. When the terminal ileum is stuck to neighbouring coils, it should be gently separated at points where there is obvious kinking; lymph coagula should not be removed, but long trailing strands of lymph should be lifted out.

In the more uncommon cases of obstruction by a well developed ileal band relief may be obtained by its division, but if this is inadequate, or if such a step appears unwise in the presence of advanced peritonitis, then ileo-colostomy should be performed. The lesion of the terminal ileum described by Handley in "ileus duplex" urgently calls for the performance of ileo-colostomy and probably also for a caecostomy (by a self-closing method), the operative procedures recommended by Handley.

The treatment after operation for acute appendicitis has some influence in determining the incidence and severity of obstructive troubles. That the administration of purgative

medicines is unnecessary I feel sure; that it is also very harmful, by producing intestinal turmoil and distension, I believe. For the past three years I have abandoned the use of purgative medicine in such cases and rely on the administration of a simple enema, daily or on alternate days, to procure an evacuation of the bowels. Morphine in small hypodermic doses is given during the first day or two if necessary.

A satisfactory action of the bowels is a very favourable indication of the patient's progress in peritonitis, but it must not be regarded as the cause of the improvement, and I think it possible that we defeat our purpose by trying to force the appearance of this welcome sign. A suggestive paper on this subject by Le Roy Long⁶ treats the matter in more detail than is possible here.

(b) Post-operative Obstruction.

This, I believe, will rarely arise if the treatment already outlined is carried out.

The management of a case of post-operative obstruction presents one of the most anxious and difficult tasks with which the surgeon has to deal. The following plan of treatment, which I believe should in all cases be adopted in the first instance, is based on the fact that the obstruction is rarely a complete one and that the intestine will frequently recover its function if relieved of some of its load.

(1) All nourishment and drinks by mouth are stopped, but chewing of fresh fruit (orange, grape pulp), etc., is encouraged.

(2) Subcutaneous infusion of saline solution.

(3) Evacuation and lavage of stomach by stomach tube.

Morphine, too, in many instances will be found beneficial, despite the apparent contraindication to its use, in producing rest and sleep.

For the subcutaneous infusion of saline solution Souttar's apparatus answers admirably; the rather stout needles fitted to this can be painlessly introduced by previously injecting novocain through a hypodermic needle at the selected site. About 3 pints should be introduced in the first twelve hours.

The passage of the stomach tube is an unpleasant task with a very poorly patient, especially with a child, but its use is of the greatest value. By retaining the tube in a position for a few minutes after the stomach has been emptied it is often possible to withdraw several ounces more of intestinal contents. The drainage of the stomach may need repeating in twelve or twenty-four hours.

The following is an instance of the value of this treatment.

CASE III.

P. H., aged 13 years. Onset of acute appendicitis fifty hours before operation. Abdomen distended but not tense; hypogastric tenderness. Pulse 140 (after repeated counts, patient mentally tranquil), temperature 104.4°. Operation revealed a greatly distended appendix, almost entirely gangrenous but unruptured and lying in the pelvis. Lower abdominal peritoneum everywhere red and inflamed but no effusion. Appendix inevitably ruptured during removal. Terminal ileum noticed to be tethered down into pelvis, but was not interfered with owing to the child's toxic condition and technical difficulties arising from fatness of abdominal walls. Pelvic drainage tube.

Four days later the patient had vomited 3 pints of intestinal fluid in the past twelve hours—the later vomit faecal in appearance. No flatus at all after enema or tube for past forty-eight hours. Belly globular in shape from extreme distension and as hard as a well inflated motor tyre. Face pallid and shrunken as compared with appearance a few days previously. Lips cyanosed. Pulse 140 but fair volume.

Stomach tube withdrew 2½ pints of faecal-coloured fluid. Subcutaneous infusion instituted and all drinks stopped. Morphine 1/8 grain. Marked improvement within twelve hours, as shown by decrease in the tenseness of abdomen, improved facial appearance, and action of bowels after enema. Made progressive improvement afterwards, and was discharged home one month after admission, healed and without having had any aperient beyond paraffin.

If treatment on these lines is not followed by definite improvement in the course of one to two days then operative interference will be necessary. The scope of the operation depends entirely on the condition of the patient, but drainage of the small intestine above the obstruction either

by ileo-colostomy or enterostomy (using a self-closing method for tube insertion) is the essential objective of the operation. Separation of the adhesions at this stage will prove insufficient, and will involve more exposure and handling of distended coils than a simple drainage operation.

Obstruction during Convalescence.

Operative treatment is necessary in almost all cases in this period. Separation of organized adhesions about the lower ileum, followed by a gentle milking down of the intestinal contents through the liberated bowel, will be sufficient in the majority of cases, but in cases developing early on in this period or in cases presenting widespread adhesions an ileo-colostomy or even an enterostomy may be necessary.

GENERAL REMARKS.

As some basis for the substance of this paper I ought to refer to my own experience of acute appendicitis. Taking a series of 126 consecutive cases (since January, 1921), which includes an average proportion of desperate cases, there have been four deaths. The cause of death was determined after a *post-mortem* examination in each case, except the first.

1. Delirium tremens following pronounced alcoholism: a case of fulminating perforative appendicitis.

2. Cardiac muscle failure in a man of 63, whose arteries were rigid and tortuous: a case of diffuse peritonitis in which there appeared no possibility of recovery without operation. *Post-mortem* examination revealed no evidence of spread of peritonitis.

3. General peritonitis in a woman of miserable physique.

4. General peritonitis and ileus duplex: enterostomy performed at primary operation, and this acted freely up to the time of death on the third day after operation. *Post-mortem* examination showed general peritonitis; there was an absence of intestinal distension and a definite diminution in swelling of the intestinal wall at site of the ileal lesion.

In this series one patient required re-operation during the first week for intestinal obstruction due to ileal band (Case 1); two patients required re-operation during convalescence—on the twenty-fourth and forty-second days after primary operation. All recovered.

Very little mention has been made in this paper of paralytic ileus; that some degree of paralytic ileus occurs in bowel which is exposed to acute peritonitis cannot be doubted, and I believe it to be a factor in the production of the types of obstruction already described. The presence of a kink or of an angulation, which in normal circumstances would be insufficient to produce obstruction, will in the case of bowel already sluggish and inactive (from paralytic ileus) lead to acute intestinal obstruction. This contention is borne out by the observation that treatment directed to the reduction of intra-intestinal tension above the obstruction, but which ignores the actual site of the obstruction, is usually effective in affording complete relief; on the other hand, the liberation at the primary operation of a fixed and kinked terminal ileum will lead to the speedy disappearance of ileus in a case where this is a conspicuous feature before operation, and will, moreover, materially reduce the patient's liability to ileus during the post-operative and convalescent periods.

Finally, in regard to the influence of a drainage tube in the production of intestinal obstruction, I believe a drainage tube may be a factor in cases of pelvic peritonitis where no attempt is made to release an adherent terminal ileum; under such conditions a tube, passing from a wound on the right side straight down to the floor of the pelvis, will lie over the point of angulation in the ileum and must tend to intensify the effects of this by pressure and irritation. If, however, the terminal ileum is free, or is mobilized at the operation, then I believe the risk of obstruction from adhesions around the tube track is a very small one and is much less than that attaching to the abandonment of drainage in cases of purulent pelvic peritonitis.

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ACUTE INTUSSUSCEPTION IN CHILDREN: FIFTY CONSECUTIVE CASES.*

BY

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ACUTE intussusception is comparatively common in the first year of life, especially from the fourth to the eighth month; it is more common in boys than girls. The most important features are: (1) periodic attacks of crying because of acute abdominal pain in a previously healthy child; (2) a swelling in the abdomen; (3) vomiting, which may become persistent; (4) passage of blood and mucus once or several times from the rectum; (5) emptiness of the right iliac fossa; and (6) the abnormal quietness of the child.

The persistent periodicity of these attacks of abdominal colic with the associated crying is one of the most important features. Fifty consecutive cases were observed and all gave this typical history; many displayed it during their short sojourn in the ward prior to operation.

In all the cases save four blood had been passed by the rectum. The cases where no blood was passed were ileal intussusceptions, two of which required resection. In four cases blood was passed soon after the commencement of the attack, then not for a period of almost a day, and then more blood was passed; all four were on operation seen to be two intussusceptions—one, the original, an ileo-ileal which had proceeded to the ileo-caecal valve, and, sticking there, was replaced by an intussusception which had for its apex the ileo-caecal valve.

In some cases a tumour cannot be felt, partly because of the position of the intussusception; it may be tucked up under the ribs at the splenic flexure, or the patient's abdominal wall may be too resistant to permit its palpation. In these cases the tumour can readily be felt under light anaesthesia. If deep anaesthesia be induced, only the mass swelling of an advanced intussusception will be detected, as the spasm of the intestinal musculature, to which the early tumour is due, will not then occur, or, if present, will disappear.

In some cases the "signe de Dance"—that is, emptiness of the right iliac fossa—can be made out; this will obviously only be present when the intussusception has progressed beyond the caecum, and when the caecum is mobile.

The abnormal quietness of the child may be commented upon by the mother. It is usually a grave sign, and may mean that gangrene has occurred or that severe toxæmia is present. The possible explanation is that the toxins have paralysed the nervous mechanism of the musculature of the sheath, which no longer attempts to pass on the enclosed bowel. This is probably the reason why the intussusceptum, in every case that is left to itself, does not present at the anus. The toxæmia which kills the child has previously paralysed the bowel.

At the onset there may be no rise of temperature, but later on, especially after twenty-four hours, the temperature may be anything from 100° to 105°. In this series there is one in which the temperature was 108.2° prior to operation. Intussusception is associated with an abnormally mobile colon, and all are agreed that it occurs after intestinal upset with succeeding irregular peristalsis, but there is probably some other important factor. Intestinal upsets are frequent, yet few develop intussusception.

A pedunculated tumour growing in the lumen of the gut would, of course, tend to give rise to an intussusception; this does occur sometimes in the adult, but I have never seen such a condition in the child. Neither have I seen or palpated anything in the lumen which was capable of giving rise to this condition. An enlarged Peyer's patch is said to cause intussusception, and that is theoretically possible, but cannot be asserted as the cause, for had it been normal when it got in with the apex it would soon become enlarged and congested.

Latterly I have been looking out for possible causes outside the lumen, and I have noticed the presence of enlarged

* A post-graduate lecture delivered at the Royal Liverpool Children's Hospital, December, 1923.