or subinfection are to be detected. I would, however, in all meekness, object that the intoxication theory has been tried, and, save as regards a singularly limited series of symptoms, has so far been found wanting when subjected to the test of experiment; while, on the contrary, it is possible to adduce experimental evidence in favour of each of my pleas in favour of the infective nature of the conditions discussed.

CONCLUSIONS.

To epitomize:

1. It is more rational to regard the evil effects of intestinal stasis as, in the main, a result of conditions favouring subinfection and low forms of infection than as a result of chronic intoxication.

2. The term "gastro-intestinal auto-intoxication" is pernicious and not to be employed by any self-respecting member of our profession, save for so limited a set of conditions that for ordinary purposes it may safely be wholly expunged from the medical vocabulary.

3. While the symptoms and diseases enumerated by Sir Arbuthnot Lane may follow intestinal stasis, at least a large proportion of them may originate independently of such stasis.

4. Before recommending the operation of short-circuiting it is necessary, therefore, to make the fullest studies, so as to discover, if possible, the nature of the organism responsible for the disturbance and its probable seat of entry.

5. A discovery of the cause of the symptoms is calculated to suggest the appropriate means of treatment by means other than short-circuiting. Only when these have been tried and found wanting is removal or short-circuiting of the colon justifiable.

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UNDER the will of the late Mrs. Sarah Berry of Wimbledon the Royal Hospital for Incurables at Putney receives a bequest of £1,000.

IN a pamphlet entitled, *What Town Planning Means*, published by the Garden Cities and Town Planning Association (post free, 3d.), the author, Mr. George Pepler, F.S.I., in addition to discussing what local councils and private owners of property may do under the Act, and the possibilities that the Act opens up, gives a list of corporations and urban district councils which have already prepared, or are considering the preparation of, schemes of improvement. They number not far short of a hundred. In a good many localities schemes for dealing with several different areas are in view.

An Address

ON

CHRONIC INTESTINAL STASIS.

DELIVERED BEFORE THE DUBLIN UNIVERSITY BIOLOGICAL ASSOCIATION ON NOVEMBER 22ND, 1913.

By SETON S. PRINGLE, F.R.C.S.I.,

PRESIDENT OF THAT ASSOCIATION, AND SURGEON TO MERCER'S AND CORK STREET HOSPITALS, DUBLIN.

ALL of us have had the experience of treating and some of us of operating on cases with more or less indefinite abdominal symptoms—symptoms which may have suggested gastric or duodenal ulcer, gall stones, chronic appendicitis, ovaritis, etc. But at the operation none of these lesions have been found, or if found, their appropriate treatment has failed to permanently relieve our patients. Looking back over such cases we recognize that in many the abdominal trouble has been accompanied by general debility, chronic constipation, a sallowness or duskiness of the skin, headaches, bad appetite. In fact, the patients were toxæmic, and in treating the lesion we have merely dealt with the result and left the cause untouched. The root of the trouble in these cases is chronic intestinal stasis, or, in other words, delay in the passage of the contents through the alimentary tract, and consequent excessive absorption of toxic products. We are indebted chiefly to Sir Arbuthnot Lane for enlightenment on this subject, and no communication such as this would be complete without heartily acknowledging the excellent pioneer work he has accomplished in the face of much prejudice and keen opposition. Many other workers have thrown light on one or other aspect of the problem, and I wish to mention in this connexion the names of Coffey, Wilms, Flint, Mayo, Jackson, Connell, Rovsing, Goldthwait, Bainbridge, Eastman—and last, but not least, Mr. Gray. Personally, I have been feeling my way in the treatment of these most interesting cases for the last five years. As my own ideas have become crystallized, I have absorbed and adopted many of the ideas put forward in the first place by some of the above writers, so here and now I wish to express my obligation.

There are, to my mind, two distinct factors in the production of stasis: (1) Ptosis of some part of the gastro-intestinal tract, and (2) kinking and stenosis of the bowel by bands and membranes; in other words, we may speak of "stasis of gravity" and "stasis of obstruction." These two conditions are as a rule associated; but in such cases one condition generally preponderates and overshadows the other.

ANATOMICAL CONSIDERATIONS.

In the study of ptosis the first matter to consider is the manner in which the various parts of the gastro-intestinal tract are maintained in position against the force of gravity which is constantly tending to produce downward displacement in the erect position assumed by man. If we glance at the longitudinal section of the abdomen we are at once struck by the fact that the shape of the abdominal cavity proper, as distinct from the pelvis, is that of an inverted cone. It will also be seen that the posterior abdominal wall forms a shelf inclined at an angle of about 50 degrees, on which all the heavy organs rest. The majority of the organs are supported on this shelf by peritoneal folds and mesenteries, by pads of fat, and by the abdominal pressure maintained by the anterior abdominal wall. In the case of the duodenum and the ascending and descending colon we also have direct attachments to the fascia of the posterior abdominal wall—a state of things brought about by the fusion and disappearance of the peritoneal coverings of the gut and parietes where these come in contact. If one of these means of support is lessened, equilibrium is disturbed and ptosis results. Thus ptosis is in the early stages localized, the commonest condition being either mid-line ptosis where the stomach and transverse colon are affected, or right-sided ptosis where the caecum is the organ at fault. In many cases the condition does not progress further; but on the other hand, if stasis is caused by the ptosis, we get distension behind the affected part, with general ill-health and resultant absorption of fat and loss of muscle power.

This combination of factors brings about general ptosis of the intestinal tract and finally of the solid viscera; in other words we get the condition known as Glenard's disease.

Mid-line Ptosis.

For practical purposes we may look on the stomach as being suspended at either end—on the left by the oesophagus and gastro-phrenic ligament, on the right by the strong free edge of the gastro-hepatic omentum containing within its layers the bile duct, the hepatic artery and the portal vein. The remainder of the gastro-hepatic omentum attached to the lesser curvature is a weak peritoneal fold and cannot provide material support. In the same way the transverse colon is slung from the firmly fixed splenic and less fixed hepatic flexure and is suspended from the posterior abdominal wall by its mesocolon and from the greater curvature of the stomach by the gastro-colic omentum. If in the course of development the two anterior layers of the great omentum fail to fuse with the two posterior layers (thus obliterating the omental bursa) and with the anterior surface of the colon, this part of the intestine loses a material support. It is also to be noticed that neither the body of the stomach nor the transverse colon rest on the shelf which I have described. From these facts it is evident that the stomach and transverse colon depend largely on abdominal pressure for their support. If for any reason the musculature of the anterior abdominal wall should be weak, and especially if this is associated with constipation and loading of the colon, ptosis must inevitably occur, and if the omental bursa has not been obliterated as mentioned, the colon will prolapse to even a greater degree than the stomach. As soon as any sagging takes place the fixed points, namely, the first stage of the duodenum and the hepatic and splenic flexures, will be dragged on and a certain amount of kinking result; thus stomach and bowel will have greater difficulty in emptying, there will be retention of their contents, more weight will be thrown on their supports, causing an increase in the sagging, and so a vicious circle is set up and symptoms produced.

Right-sided Ptosis and Mobile Caecum.

Right-sided ptosis and mobile caecum are one and the same condition. Normally the ascending colon is firmly fixed on the shelf of the loin by peritoneal fusion, but in as many as 20 per cent. of subjects this fusion is incomplete or does not occur at all. As a result the caecum tends to sag, frequently bringing with it the right kidney. When this occurs the caecum is suspended between the fixed hepatic flexure and the last stage of the ileum. If the fixation at the former site does not stretch, the flexure is dragged on and a sharp angle is formed, while, if there is not a long mesentery to the terminal ileum, here also a kink and consequent stasis are produced. The attachments of the hepatic flexure, however, frequently do stretch, and thus we get prolapse of the caecum, ascending colon, and right half of the transverse colon.

These conditions of local ptosis are frequently the first steps in the production of general sagging of the intestinal tract. I have seen cases with the stomach well below the umbilicus, the dependent part of the third stage of the duodenum resting on the promontory of the sacrum, the small intestines in the pelvis, and the whole of the colon provided with such a long mesentery that it was possible to bring it, including even the splenic flexure, which is the last part to give way, completely out of the abdomen through a laparotomy incision. In such cases the bowel is dilated, its wall is markedly atonic, and serious stasis results.

Bands and Membranes.

We have now to consider the presence of the various bands and membranes which have been described in connexion with the alimentary tract. No one who carefully explores a number of abdomens can doubt the frequent presence of one or other of these bands or so-called "adhesions"; and when we find that their presence is in many cases associated with a certain set of symptoms which are always alleviated and frequently completely abolished by the division of these adhesions, I think we are justified in looking on them as the cause of much abdominal discomfort.

A great deal of controversy has raged, and will still rage, around the etiology of these conditions. From amongst the many conflicting and often contradictory opinions I have, as regards each condition, adopted and frequently modified that explanation which fits in best with my personal observation and experience. I do this in the hope that it will stimulate other surgeons to a closer study of the intra-abdominal conditions found during operation, and likewise the physicians to re-examine many of their patients with indefinite abdominal troubles, in the light which recent research has thrown on the subject. I believe that we shall arrive at the true cause and correct treatment of these conditions by means of the work not of one, but of many. It will assist us in our consideration of the etiology of these bands, etc., if at the outset I state that I am not dealing with the very varied adhesions which may result from inflammation, although undoubtedly these also frequently produce stasis. I am only referring to those bands, membranes, or peritoneal folds which are not adhesions in the ordinary sense of the term. They are too regular and constant in their position and extent and the arrangement of their blood vessels, and lack the opaque scarred appearance of inflammatory adhesions.

I propose to deal with these bands, etc., seriatim, describing them briefly. In every case I hope to be able to prove that these bands are embryonic in origin, though undoubtedly they may become thickened and otherwise altered by the strain thrown on them in pathological conditions of the bowel. I also believe that in many cases they produce no symptoms, and that it is only in those cases in which they are well developed or associated with more or less marked ptosis that they cause trouble.

1. Gall Bladder-Duodenum Band.

The first band to be considered is the fold of peritoneum which frequently extends from the under surface of the gall bladder to the first stage of the duodenum (Fig. 1), and sometimes down unto the anterior aspect of the transverse colon. In common with other observers I have found this band in the fetus, and it is undoubtedly due to a persistence of the fold of peritoneum which the gall bladder as it buds out of the common duct draws out of the ventral mesogastrium. If a band such as this is associated with ptosis of the stomach, it is obvious that the kinking of the first stage of the duodenum will be more marked.

Fig. 1.—Diagram showing cholecysto-duodenal band.

2. The Mesocolic Band.

The mesocolic band, first described by Mayo, which suspends the antimesenteric border of the first few inches of the jejunum from the under surface of the transverse mesocolon (Fig. 2). This band also I have found in the fetus. It is, I believe, due to fusion between the peritoneum of the mesocolon and that covering the jejunum. In cases of ptosis of the small intestine secondary to obstruction further on in the tract, I have often seen a sharp angle produced at the duodeno-jejunal flexure causing stasis in the duodenum, as evidenced by its dilated condition. Where, however, this mesocolic band is present, it tends to keep the angle open and thus obviate the formation of such kink. Personally, I have never seen a case in which I considered this band as assisting in producing duodenal stasis, and I believe its action to be

mainly conservative. Lane describes other bands which "run upwards and outwards from the commencement of the outer aspect of the jejunum to the peritoneum lining the adjacent abdominal wall," and considers that they are adhesions acquired in an effort of Nature to support a sagging small intestine.

Fig. 2.—Diagram showing mesocolic band.

face of the ileum to the peritoneum at the brim of the pelvis; not only does it in this way tie down the terminal ileum, but it causes a rotation of the bowel at the point of its attachment, and when it is divided the

3. Lane's Ileal Kink.

The general appearance of this condition is best understood by reference to the drawing (Fig. 3) originally published by Mayo, and accepted as correct by Lane. This band, found in connexion with the last few inches of the ileum, is generally triangular in shape, and extends from the free surface

peritoneum lying in contact, normally occurs in many situations in the evolution of the adult condition from that of the embryo, so that it is but a small step to the supposition that a similar process may occur at the lower end of the ileum. Lane believes that this band is due to "the crystallization of lines of force" developed by

Fig. 3.—Shows Lane's ileal kink, with the band of peritoneum which produces it. (Mayo, *Surg. Gynaec. and Obstet.*, March, 1911.)

Nature in the endeavour to hold the caecum out of the pelvis. I have, however, found this band unassociated with mobile caecum, and vice versa have demonstrated its absence in many cases of marked prolapse of that organ. If it were developed as an accessory suspensory ligament of the caecum we would expect it in connexion with the upper leaf of the mesentery, as has been pointed out by Gray and others.

In whatever way this fold is formed, there is, at any rate, no question that kinking is frequently produced, especially if ptosis of the caecum be present and that the lumen of the bowel is reduced by this kinking as well as by the rolling in of itself on its mesentery, as I have already described.

4. "Jackson's Membrane."

We have now to consider the bands and membranes which are found in connexion with the caecum and ascending colon, and to which the generic term "Jackson's membrane" is given. Lane has for years described adhesions tying the prolapsed caecum to the right lateral abdominal wall, but Jackson in 1909, under the title of "membranous pericolicitis," described a thin vascular veil extending over the ascending colon. Since then many surgeons have described various forms of membranes or veils in this region, till at present, I think, that we may apply the term "Jackson's membrane" to any membrane filmy or in parts fibrous which extends over whole or part of the ascending

Fig. 4.—Photograph (and tracing) of caecal region in a fetus, 28 cm. long, showing terminal ileal kink (A), and the fold (X) producing it.

ileum, as it were, rolls out of it. I have examined this region in a number of infants and embryos. In 20 per cent. I have found some abnormal peritoneal attachments. In some cases the appearances were identical with that described as Lane's kink in the adult, and such a condition in the fetus is illustrated in Fig. 4. In others there was a more or less extensive fold which corresponds to that described by Reid, and called by him the genito-mesenteric (Fig. 5). This fold is attached above to the mesentery of the terminal ileum, and frequently extends on to the lower aspect of the circumference of the bowel. Below it passes downwards to be attached to the posterior abdominal wall along the line of the right spermatic or ovarian vessels, often being continuous below with the suspensory ligament of the ovary. Again, I found in an embryo 20 cm. long in which the caecum had not yet descended, the under-leaf of the ileal mesentery firmly adherent to the posterior parietal peritoneum well up in the iliac fossa (Fig. 6), and it was easy to see that as the intestine descended this bend might be pulled out into the fold resembling Lane's kink. From these observations I consider that the condition is always congenital in origin and due either to persistence of Reid's fold or to an excessive peritoneal fusion. The adhesion, fusion, and sometimes subsequent absorption of two layers of

Fig. 5.—Photograph (and tracing) of caecal region in a fetus 26 cm. long, showing Reid's genito-mesenteric fold (X), extending into suspensory ligament of ovary (ov.). a, Slips of paper underneath fold and ovary; ut., uterus. Note puckering of ileum at b.

colon and caecum, attached externally to the parietal peritoneum, and blending internally with the peritoneum covering the anterior aspect of the colon, and sometimes even continued on into the great omentum. This condition

also I consider congenital. In common with many others I have found fine veil-like adhesions extending to the lateral abdominal wall from the surface of the colon in the fetus, and it is easily understood if such peritoneal fusion occurs while the caecum is incompletely descended and rotated,

Fig. 6.—Photograph (and tracing) of caecal region in a fetus 20 cm. long, showing undescended caecum and fold of peritoneum (X) fixing terminal ileum in right loin and producing angulation. *a*, Appendix; *b*, ileum; *c*, inner surface of reflected flap of abdominal wall; *d*, slip of paper underneath peritoneal fold.

that as the bowel descends and rolls inwards they will be pulled out into just such a membrane. In fact, such a condition was described years ago by Jonnesco and Juvara, who called it the "parieto-colic fold," and it has been found by different observers in from 10 to 20 per cent. of fetuses after the sixth month. It has been suggested by Gray and Anderson and by Cottee that this membrane is due to a prolongation of the right edge of the great omentum out over the colon, with fusion to the anterior aspect of the bowel and to the parietes, and I have found in one fetus an arrangement strongly confirmatory of this view.

In many cases where stasis is present these membranes are in places distinctly fibrous. This thickening, in my experience, is found most frequently opposite the crest of the ilium and just below the hepatic flexure. This condensation is, I believe, due to secondary inflammatory change, and this inflammation may extend so as to bind the ascending colon to the right half of the transverse colon in cases in which the latter is ptosed, bringing about the condition known as double-barrelled colon. When the ordinary veil-like type of membrane is divided, peritoneum is found underneath; but if the secondary inflammatory changes, with thickening, have taken place, such a division leaves a raw surface, which requires to be covered in. There can be no question that in many cases these membranes cause a narrowing of the bowel. I have often been astonished at the way in which the ascending colon will unroll and expand as they are divided. The densest bands are found associated with mobile caecum, but, just as in the case of the ileal kink, I have seen a well-marked Jackson's membrane on a colon in perfect position.

5. The Splenic Kink.

This is due to the fact that the phrenico-colic ligament is very dense, and is the last normal attachment of the bowel to stretch in cases of ptosis; in fact, here the increased drag in such cases brings about a strengthening of this ligament, as pointed out by Lane. The result is that there is a very sharp angle produced, and the more the transverse colon is ptosed the greater the obstruction to its emptying itself. The acuteness of the angle is also increased if the descending colon should happen to have a mesentery. In advanced cases also the two limbs of the angle are bound together by more or less dense adhesions, due to secondary inflammation.

6. Pelvic Colon Kink.

Lastly, we come to the kink in the pelvic colon called by Lane the "last kink." This part of the colon should normally have a free mesentery, but frequently the meso-colon just opposite the brim of the true pelvis is shorter than that above or below. On examining this we find that

it is due to a shortening of the under-leaf of the mesentery, and on this shortened part we find one of those "white lines" which have been pointed out by Toldt as signifying the limit of fusion between two peritoneal surfaces. This fusion is embryonic in origin, and has taken place between the outer fold of the meso-sigmoid and the parietal peritoneum at this point. I have found a very beautiful example in a fetus 20 cm. long (Fig. 7). The attachment of this shortened mesentery to the pelvic wall is very close to the ovary, and any drag is easily communicated to that organ, especially in those cases in which secondary inflammation has caused condensation and contraction with implication of the ovary in the fold. This binding down of the colon causes a very distinct kink, and if the bowel above is very loose it, when loaded with faeces, will fall over into the pelvis, thus tending to aggravate the kink.

SYMPTOMS.

As regards the symptoms produced by these ptoses and kinks a whole book might be written, for with lesions in such different parts of the intestinal tract it is obvious that the predominating symptoms will vary with the site of the chief trouble. Clinically, however, I would divide the symptoms into (*a*) toxic, and (*b*) obstructive.

(a) Toxic.

This group of symptoms is best marked in cases with extensive general ptosis when a very characteristic picture is produced. The patient, generally a woman, has a muddy complexion and dark rings under the eyes. The skin is harsh, dry, and pigmented, especially in the axillae and groins; the thorax is long and the lower ribs depressed; the breasts are small and nodular; the upper abdomen is narrow and contracted, the lower protuberant, and the abdominal muscles are atrophied. She complains of headaches, bad appetite, a feeling of depression and wretchedness and general lack of energy. Constipation is

Fig. 7.—Photograph (and tracing) of left lower abdomen in fetus 20 cm. long, showing fusion (X) of under-leaf of mesentery of pelvic colon (P.C.) to peritoneum pelvic brim (*a*), producing the sigmoid kink. *b*, Inner surface reflected flap of abdominal wall; *c*, left iliac fossa; *d*, slip of paper underneath fold.

marked as a rule, and there may be mucus in the stools. She complains of abdominal discomfort, a sense of fullness and distension, with perhaps nausea and at times vomiting. Such is the picture of an extreme case, but we find all gradations in proportion to the amount of stasis present, until, in cases of localized ptosis, the toxæmia is overshadowed by the obstructive symptoms.

(b) Obstructive Type.

Here the symptoms vary with the site of the local trouble. In kinking of the duodenum from mid-line ptosis or of the duodeno-jejunal flexure we get a train of symptoms which are suggestive of gastric or duodenal ulcer—for example, pain after food, distension and vomiting, with tenderness to the right of the middle line, and sometimes hæmatemesis. This class also includes many cases which are sent to the surgeon to have gall stones removed,

or are treated by the physician as suffering from nervous dyspepsia, chronic gastritis, etc. Rest in bed immediately relieves such cases, and if accompanied by a course of massage and fattening treatment, relief may be experienced for a considerable time.

If a Lane's kink is present the symptoms are very similar to those of chronic appendicitis. I have operated on several such cases in which the appendix had already been removed. These patients complain of fullness and pain some time after food, associated with constipation and tenderness in the right iliac fossa. I have found in several cases marked distension of the left abdomen some hours after a meal, due to its retention in the small intestines. At times attacks of afebrile colic are complained of.

In mobile caecum and in constriction by bands of the ascending colon or hepatic flexure there is tenderness in the right iliac fossa, a distended caecum can be made out and a succussion splash can frequently be elicited. Many of my patients of this type have complained of burning pain in the right lower abdomen, especially in the evenings or middle of the night. The pain may culminate in a sharp attack of colic, and in my experience patients with trouble in this region suffer more pain than those with any other form of localized ptosis.

Splenic kink is generally associated with mid-line ptosis and causes much the same symptoms. It is frequently accompanied by a dragging, aching pain with a sense of distension in the left hypochondrium, and the patient often states that such pain is relieved as flatus is felt to pass on.

The last, or sigmoid kink, may be associated with a chronic pain in the left iliac fossa which is often relieved by the passage of a motion. In females this dragging pain is often worse at the menstrual period, a fact which is easily explained by the close connexion to the ovary.

All these bowel symptoms are associated with chronic constipation, or in some cases with diarrhoea due to the irritation of the retained faeces. In all the toxic symptoms above described are more or less marked. Frequently, of course, we have several kinks in the same subject, so that we get the symptoms grouped in very different ways, but with experience it is generally an easy matter to differentiate. Of late, since the number of these cases which I have seen has reached a respectable total, I have experienced little difficulty in arriving at a correct diagnosis from a study of the clinical symptoms combined with a careful physical examination. The greatest help can be gained from a study of a series of x-ray photographs, taken at different periods after a bismuth meal, and I strongly advocate the routine adoption of this method of confirming the diagnosis. In many cases, as I have stated above, these kinks, etc., may be present without producing stasis, and in a case with several of these conditions present—some producing stasis and some not—the bismuth meal shows us clearly which part of the intestine is at fault.

A consideration of the symptoms of chronic intestinal stasis would not be complete without some reference to what Lane terms "end results." He believes that gall stones, gastric and duodenal ulcers, cancer of the stomach and intestines, chronic mastitis, goitre, atheroma of the arteries, chronic arthritis, and tuberculosis, all arise from stasis. Perhaps we may not agree with him entirely, but at any rate it is obvious that stasis must lead to the presence of micro-organisms higher up in the intestine than normal, and in this way cause gastric or duodenal ulcer and infection of the bile ducts. There is also an obvious connexion between chronic irritation at the flexures and kinks and the production of cancer at these sites.

TREATMENT.

The treatment of these cases of chronic stasis is a subject of lively controversy. For example, I may mention the two extremes: On the one hand, Sir Arbuthnot Lane advises ileo-colostomy or complete colectomy in all severe cases, while, on the other hand, orthopaedists such as Goldthwait claim to be able to cure even most advanced cases by belts, exercises, etc. Personally, after treating many of these cases, I consider we may divide the treatment into (1) prophylactic, (2) medical, (3) local operative, (4) radical operative.

1. Prophylaxis.

I would like to impress on the profession my belief that the abdominal muscles are the most important voluntary muscles in the body. The abdominal organs largely depend for the maintenance of their positions on the support of these muscles, and if they are properly developed by suitable exercises the possibility of ptosis occurring is reduced to a minimum. The other prophylactic measures I would urge are the acquiring in early life of a regular habit of emptying the bowels and the avoidance of the routine use of aperients. If there is any costiveness it should be combated early by the regulation of the diet and the use of liquid paraffin.

2. Medical Treatment.

A great many cases of stasis, if seen in the early stages, can be rendered comfortable by the use of an abdominal belt, the practice of exercises which develop the abdominal muscles, and the internal administration of paraffin. The latter can be taken in doses of 1 to 4 drachms three times a day in the form of liquid paraffin (*British Pharmacopoeia*), flavoured with one of the essential oils, such as cinnamon or peppermint. A few patients find it impossible to take it in this form, and then one or other of the proprietary preparations may be ordered.

3. Local Operations.

The local operative procedures are legion in number, and I cannot do more here than specify those I have employed and found satisfactory.

In mid-line ptosis I, as a rule, perform Rovsing's gastropexy, together with shortening of the gastro-hepatic and gastro-colic omenta, but in some cases I have employed Coffey's omentopexy. If the liver has also prolapsed and would endanger the success of the gastropexy, I first fix it up by shortening the falciform ligament and suturing the free edge of the liver to the anterior parietal peritoneum. If the upper abdomen is very contracted I have enlarged it after the manner advocated by Coffey and Rovsing.

Should the splenic flexure be slung very high, I divide the costo-colic ligament and open up the angle by suturing the descending colon to the left lateral abdominal wall after dividing any adhesions which might bind the two limbs of the angle together.

In kinking at the duodeno-jejunal junction I fix the jejunum to the under-surface of the transverse mesocolon in such a way as to open out the angle; in other words I reproduce the mesocolic band.

For Lane's ileal kink I divide the band parallel to the line of the bowel and suture the resultant rent vertically, employing if necessary an omental graft to assist in covering in the raw area.

In mobile dilated caecum unassociated with Jackson's membrane I have employed many methods of fixation, and have also enfolded it in cases of marked dilatation. Recently, however, I have used the method of Coffey, which is a combined fixation and plication. If a mobile caecum is associated with Jackson's membrane, I divide this prior to fixation. When Jackson's membrane is found without ptosis of the caecum, but constricting the colon, I merely divide the membrane, and if any raw surface is left, which occurs in cases of secondary inflammatory thickening, I use an omental graft to prevent adhesion formation.

If a double-barrelled colon be present, or if the bands in the neighbourhood of the hepatic flexure are very dense, and I consider them likely to recur, I do a lateral anastomosis between the caecum and the transverse colon, or—and I think this the better procedure—excise the caecum, ascending colon and right half of the transverse colon, and restore the continuity of the intestine by implanting the end of the ileum into the remaining part of the transverse colon. In all cases of trouble in the caecal region I also remove the appendix. Where the last or sigmoid kink is causing trouble I divide the shortened leaf of the mesentery transversely and suture it vertically. Many times I have found it necessary to do two or more of these conservative operations at the same time; for example, I have divided an ileal kink and Jackson's membrane, fixed the caecum in the loin, freed the splenic and sigmoid kink, and finally performed a Rovsing's gastropexy with expansion of the upper abdomen.

4. Radical Operations.

The radical procedures are those so strongly advocated by Lane, and are two in number. The first consists in short-circuiting the intestinal flow by ileo-colostomy. This procedure he employs in all cases of stasis which require operation, and in which the colon is not loose nor the abdominal wall lax. The ileum is divided about six inches above the ileo-caecal valve, and its proximal end implanted into the lateral aspect of the pelvic colon as low down as possible, and in all cases below the site of the last kink. Lane claims that "the last kink," if well developed, obviates in these cases the reverse flow of the intestinal contents up along the colon, which was the troublesome sequela of many of his earlier operations. If the last kink is not well developed, he produces one by suturing the mesentery of the colon above the anastomosis to the peritoneum over the pelvic brim.

The second and most radical procedure of all is complete colectomy. This operation Lane performs in all cases in which the whole colon is loose. In such cases the entire colon, including the hepatic and splenic flexures, is provided with a mesentery, and can be lifted out of the abdomen, making the performance of the operation safe and astonishingly easy. Personally I reserve these radical procedures for the cases in which I consider that multiple conservative operations (of course carried out at the one sitting) would not be effective, or in which the length of time necessary for their performance would endanger the life of the patient. My own opinion is that ileo-colostomy is not a good surgical procedure, and I believe that where conservative operations are contraindicated the best operation is complete colectomy.

In the performance of colectomy I use an extra broad crushing clamp (Fig. 8) specially made for me by Messrs.

Fig. 8.

Fannin. This instrument has blades 1 in. wide, and presses out laterally the muscular and mucous coats of the bowel, leaving the serous intact. A ligature is placed round either end of the crushed part and the bowel divided between. In this way the possibility of infection is reduced to a minimum and the invagination of the ends of the severed gut by purse-string sutures is rendered easy.

The patients after complete colectomy make good recoveries, put on flesh and gain better health than they have experienced for years; in fact, they derive the greatest benefit from the removal of their great bowel, which had lost its original function and degenerated into a cesspool, as tersely expressed by Lane.

From the foregoing it will be seen that in my treatment of these cases I have gradually felt my way up from the performance of small conservative operations to the adoption of radical procedures in suitable cases. The majority of my cases are too recent to allow me to judge of the final outcome, but the immediate results have been most satisfactory and encouraging. In those cases which have been operated on one, two, or three years ago, the improvement, which in some was very startling, has been maintained. The result of my experience has been to convince me of the importance of intestinal stasis, and I believe that here a great opportunity is presented to the surgeon for the relief and cure of much unnecessary suffering.

In conclusion, I would like to express my deep sense of indebtedness to Mr. Gray for coming over to address us to-night, and also to Professor Dixon for the facilities he most kindly gave me for the study of the intra-abdominal conditions in infants and fetuses.

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Remarks

ON

CHRONIC INTESTINAL STASIS.

MADE AT THE MEETING OF THE DUBLIN UNIVERSITY BIOLOGICAL ASSOCIATION, NOVEMBER 22ND, 1913.

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IN the consideration of the various aspects of intestinal stasis it will be of great advantage if we continually keep in mind their bearing on the question of treatment, which is after all the most important question—how are we to turn our knowledge, our discoveries, to the best advantage? We should always be asking ourselves, "Why, why?" or "How, how?" However hackneyed a subject is, however highly developed the technique of an operation is, we should always have the attitude that something more may be learned, some improvement can be made, which will redound to the better comfort or safety of our patients. I am glad that during recent years the feeling has been gaining ground that surgeons have, in the past, been doing too little for many of their abdominal cases, that the primary cause has been neglected in the presence of an assertive effect.

If the symptoms are not perfectly clear, it is due to the patient, when once he has submitted to operation, that a thorough intra-abdominal investigation should be made, and, if it is thought advisable, a sort of "spring cleaning" carried out, multiple operations should be done, so as thoroughly to break up the vicious circles which, as all of us know, abound in the abdomen. At the same time, the surgeon must be discriminating; there must be clear indications for interference; our desire to help must not allow our itching fingers to maltreat perfectly harmless conditions. What I say can be applied with great force to our subject to-night, and I think that there are still many points to be settled in connexion with intestinal stasis. For example, the treatment of this condition is by no means so straightforward or systematized as we are led to believe by Lane and his followers.

We must, in following up what has been mapped out so admirably by Mr. Pringle, discard from discussion all cases of stasis due to new growth, to the immediate or remote direct effects of inflammatory processes such as tubercle, dysentery, diverticulitis, and so forth, and to those other pathological conditions which cause constipation by a reflex influence, such as appendicitis, gall-bladder disease, or pyloric stenosis, and which are most satisfactorily dealt with by suitable and thorough surgical procedures.

There is also a small number of cases in which, either before or after death, no satisfactory explanation of the stasis is forthcoming. There is no stenosis, or angulation of, or other interference with, the bowel, so far as apparent to us, and yet the bowel has that dilated and possibly also hypertrophied appearance characteristic of cases in which real organic obstruction exists. Till better diagnosis and treatment are available, apparently nothing other than surgical remedies are of any lasting use in these cases, and even they sometimes fail to be permanent in their effects.

The discussion, therefore, is confined to the consideration of that large residue of cases of stasis caused by the presence of certain bands or membranes which interfere with the full action of the bowel. It is curious how many surgeons are still sceptical about the presence of these membranes. All one can say is, although their presence