were given to the radiologist which were manifestly based on insufficient knowledge. He noted with satisfaction Dr. Williamson's explanation that adverse developments after x-ray treatment were not necessarily the results of such treatment, and suggested that a more formidable list could be cited as occurring after operative treatment. The fact was that our knowledge of the biological effect of x rays was still very vague, just as until comparatively recently we were ignorant of the very nature of x rays, and, moreover, our methods were subject to so much change and advance, to say nothing of improvement in tubes and other apparatus, that it was quite impossible at present to standardize technique.

A Post-Graduate Lecture

ON

CLINICAL MANIFESTATIONS OF THE MOBILE PROXIMAL COLON.

DELIVERED AT ANCOATS HOSPITAL, MANCHESTER.

BY

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In any endeavour to elucidate the question of the clinical importance of abnormal mobility of the caecum and ascending colon, it is necessary briefly to recall the light thrown upon the subject from the different angles of human embryology and of comparative anatomy.

Embryological Evidence.

In early intrauterine life the mid-gut loop, extending from the duodenum at the opening of the common bile duct to the splenic flexure of the colon, is suspended from the middle of the spine by a single dorsal mesentery, of which the superior mesenteric artery forms the axis. (Fig. 1.) During the fourth and fifth months of development this U-shaped mid-gut loop undergoes a counter clockwise rotation round its axis. The caecum, originally a diverticulum on the posterior limb of the loop, swings round through three-fourths of a circle until it lies in the right hypochondrium under the liver. Here it may occasionally remain as an "undescended caecum," a condition that gives rise to those rare but puzzling cases in which an inflamed appendix is found in close relation with the gall bladder. Usually, however, during the later months before birth a gradual descent of the caecum into the right iliac fossa follows, and as a result the ascending colon and hepatic flexure become recognizable parts of the large intestine. (Fig. 2.) This process is contemporaneous with the descent of the testis or ovary, and probably results from the same mechanical causes.

Meanwhile a separate and very remarkable process of peritoneal fusion has been going on. First the great omentum grows down over the front of the transverse colon and adheres to it. Then the primitive mesenteries of the ascending and descending colon disappear. (Figs. 3 and 4.) These segments of the colon fall back against the posterior parietal peritoneum in the right and left sides of the abdomen, and become permanently fixed in position by fusion of the mesocolon and peritoneum covering the posterior surface of the bowel with the parietal peritoneum. The great omentum at this stage takes a far more active part in the posterior fixation of the ascending colon than has been generally recognized, and one reminder of the part it plays is often seen in the structure known as Jackson's pericolic membrane.¹

Evidence of Comparative Anatomy.

The explanation of this phase of intestinal development is obvious from a study of comparative anatomy. If we examine a series of mammalian types, in which the mid-gut loop undergoes rotation closely analogous to that in man, we find a lack of this peritoneal fusion, and a consequent persistence of the original mobility of the ascending and descending colon, in the pronograde animals only. Thus the dog and the lower monkeys have a colon that is completely mobile. But as we trace the series up through the lower pronograde monkeys to the higher anthropoids, we find the fixation of the colon progressing *pari passu* with

the adoption of the erect posture, until, in an orthograde animal such as the chimpanzee, the fixation differs in no material degree from that in the normal human type.

We are thus led to the inevitable conclusion that fixation of the ascending and descending colon is evolved as a necessary adaptation to the upright posture.

In the course of evolution individuals with the more mobile type of ascending colon (for mobile descending colon is an excessively rare anomaly) have been in some way handicapped in the struggle for existence and have tended to succumb. Let me try to explain some of the various ways in which this lack of adaptation to the upright posture may show itself in man, for it is a very common and, I believe, a very important developmental defect. In some 20 per cent. of individuals of all ages you will find some degree of abnormal mobility of the caecum and ascending colon.

MANIFESTATIONS IN ACUTE ABDOMINAL EMERGENCIES.

1. Ileo-caecal Volvulus.

In October, 1918, I was asked to see a lady, aged 68, who had been suddenly taken ill three days before, with acute abdominal pain and vomiting. The pain had attacked her in intense spasms for the first twenty-four hours, but for the last two days it had been more continuous. She had absolute constipation and had passed no flatus, in spite of repeated enemata. She was a thin woman, with sunken features and a foul, dry tongue. The pulse was 110 and thin. In the mid-line of the lower abdomen was a large rounded tumour, standing out visibly through the thin abdominal wall. It was rather tense on palpation, and gave a deep tympanitic note on percussion. No caecum could be palpated in the right iliac fossa. A diagnosis of volvulus was made, and an operation performed. On opening the abdomen through a median incision below the umbilicus, a huge dilated caecum popped out. It was found that the distended parts consisted of the lower five inches of the ileum, the caecum, and the lower half of the ascending colon. They were extremely mobile, and mounted on a long mesentery continuous with that of the small intestine. They had undergone clockwise rotation to the extent of one and a half turns. There was no actual gangrene, but the tension of gas in the caecum was so great that the serous coat was beginning to split. The whole volvulus was uncoiled with ease, the appendix removed, and appendicostomy performed by stitching a rubber catheter into its stump and fixing the stump of the appendix to the parietal peritoneum. This procedure both relieved the enormous distension and fixed the caecum so as to prevent recurrence. Her recovery was uneventful. The catheter was removed on the seventh day, and the wound healed without any leaking of caecal contents.

Ileo-caecal volvulus is a rare condition as compared with volvulus of the pelvic colon, and it has a very high mortality. It is obvious that an essential predisposing cause is failure of the normal posterior fixation of the caecum and ascending colon, for without this abnormal mobility the rotation that obstructs the bowel could not take place.

2. Intussusception.

In all the discussions that have taken place on the etiology of intussusception attention has tended to be concentrated on the mechanism by which the process starts. Is the invagination started by a localized spasm of a segment of the bowel or by a localized paresis? It is an interesting pathological problem, and I believe the evidence is in favour of spasm as the exciting cause. But far too little attention has been given to a predisposing cause of intussusception that is almost always present. I refer, of course, to lack of peritoneal fixation of the proximal colon. It is obvious that this factor does not influence the rare enteric form of intussusception. But in the common form in which the ileo-caecal valve forms the apex, or the ileo-colic form in which the lower ileum first prolapses through the valve until it jams, and then the ileo-caecal region travels along the colon, we find, invariably in my experience, a definitely abnormal mobility of caecum and ascending colon, with persistence of the ascending mesocolon, and very often a definite Jackson's pericolic membrane. It is sufficiently plain that in the presence of firm fixation of the back of the caecum and ascending colon to the parietes, an extensive degree of

intussusception could hardly take place. I believe that the much maligned ileal band of Lane (or genito-mesenteric fold of Douglas Reid) is also, when present, a very effective safeguard against the common forms of intussusception.

I need hardly remind you of the classic symptoms of acute intussusception—the abdominal pain, at first in intermittent spasms, with intervals of repose; the vomiting and constipation with the passage of blood and mucus by the rectum; and very often the typical sausage-shaped tumour in the abdomen. But it may be well to point out that the symptoms of intussusception are not always classical. I was recently asked to see a female child, aged 14 months, who had been perfectly well until seven days before, when she began to scream with pain for a few moments at intervals of thirty minutes or so, hyper-extending her back and legs during the attacks. She had vomited once slightly on the first day, but not since. The bowels had been opened daily, and no blood had been seen in the motions. The spasms of pain had recurred day and night at fairly short and regular intervals during the whole seven days. The child was well nourished and in fair condition. The abdomen was held too rigidly for satisfactory palpation, but no caecum could be detected in the right iliac fossa. In some faecal material on a napkin there was an appreciable excess of mucus. A diagnosis of intussusception was made. At operation a large ileo-caecal intussusception was found with its apex as far down as the splenic flexure. It was reduced easily and completely. The proximal colon was not only extremely mobile, but very wide in diameter, and this accounted for the lack of complete obstruction. After reduction I sutured the caecum and ascending colon back into the right loin as a precaution against a possible recurrence. The child made a normal recovery.

The operation of colopexy and caecopexy, anchoring the colon to the posterior parietal peritoneum, should, I consider, be performed as a routine after reduction of the intussusception, for a recurrence of the condition is by no means uncommon without it.

CHRONIC MANIFESTATIONS.

Far more common than these acute catastrophes are the chronic manifestations of the mobile proximal colon. In the more frequent type the patient complains of chronic constipation and pain in the right iliac fossa. The pain is usually worse after food, on exertion, and when the bowels are particularly constipated. It is described as a dull ache or a dragging pain, and the patient gives no history of a previous attack of acute appendicitis. There is often some associated sensation of flatulence and discomfort in the epigastrium, though this is more variable in its relation to the taking of food than in gastric or duodenal ulcer. Some tenderness on palpating the right iliac fossa is found on examination, and often palpable splashing and gurgling in a dilated caecum. X-ray examination after a barium meal and enema affords valuable confirmatory evidence of the condition. When screened in the upright position the caecum and ascending colon are prolapsed, so that the hepatic flexure lies down on the false pelvis, and the caecum may pass over the pelvic brim into the true pelvis. Screened in the left lateral position the loaded

mobile proximal colon sags across the mid-line to the left, and the caecum may even be seen in the left iliac fossa. In this left lateral position the extent of attachment of the hepatic flexure can be most readily estimated.

On exploring the abdomen in such cases by an incision through the right rectus, the peritoneal attachments are found to be so lax that the caecum and the greater part of the ascending colon can be lifted out of the wound well above the level of the abdominal wall. More often than not the appendix shows no signs of inflammation. The attachments of the hepatic flexure vary. In some cases a firm, broad peritoneal band containing some fibrous tissue fixes it to the lower pole of the right kidney (the nephro-colic ligament) or to the parietes close to the kidney or to the gall bladder. In other cases the hepatic flexure is so singularly devoid of firm fixation that it can be readily drawn out of the wound along with the ascending colon. The doctrine that abnormal mobility of the right kidney is invariably associated with mobility of the proximal colon is by no means in conformity with my experience, though the association is undoubtedly frequent.

Less commonly the patient complains, in addition to the symptoms already described, of sudden acute attacks of pain in the right iliac fossa, with a sense of swelling in that region, and often nausea or actual vomiting. These attacks seldom last longer than an hour or two, and may subside in a few minutes. When seen during such an attack the caecum may be found greatly distended, and tender on palpation. The explanation of such sudden crises is to be found in a partial volvulus of the mobile ileo caecal region, or an acute kinking at the hepatic flexure.

But how are we to explain the more chronic symptoms—the constipation and the aching pain in the right iliac fossa? It is one thing to affirm that the mobile proximal colon is ill adapted to the upright position. It is quite another to explain the precise mechanism by which the symptoms are produced.

We must be willing to admit that there are large gaps in our knowledge of the physiology and pathology of the colon, and we must avoid an undue craving for a simplicity of explanation that may not be reconcilable with all the clinical and pathological data. The colon, as is now well known, propels its contents, not by unceasing peristaltic movements like those of the stomach and small intestines, but by strong short thrusts, lasting only a few seconds, and occurring in normal conditions only after the principal meals. In a normal individual this gastro-colic reflex works smoothly, and causes no subjective sensation. But in a patient with the developmental abnormality that we are considering the ascending colon is hampered, in its task of pushing on its semi-solid contents against gravity, by the lack of the purchase it should derive from firm fixation to the posterior abdominal wall. Ineffectual contractions fail to force all its contents round the hepatic flexure, but the ileum passes more fluid contents through the ileo-caecal valve despite this failure. Distension of the caecum results, and is responsible for the pain in the right iliac fossa. When fixity of the hepatic flexure is combined with a very mobile ascending colon there is probably a tendency, when the colon swings into certain positions allowed by its lax attachments, for an acute angulation at the flexure to occur, amounting to a transient intestinal obstruction.

FIG. 1.—Diagram of U-shaped mid-gut loop before rotation. S., Stomach; C.D., common bile duct; V.A., vitelline (superior mesenteric) artery; V.I., vitello-intestinal duct; C., caecum.

FIG. 2.—Mid-gut loop after rotation and partial descent of caecum. S., Stomach; V.I., vitello-intestinal duct; C., caecum; A.C., ascending colon; H.F., hepatic flexure; S.F., splenic flexure.

FIG. 3.—Diagram of transverse section through abdomen, showing peritoneal arrangement before fixation of colon (after Huntington). A.C., Ascending colon; S.I., small intestine; D.C., descending colon; K., kidney.

FIG. 4.—Transverse section showing peritoneal arrangement after fixation of ascending and descending colon (after Huntington). A.C., Ascending colon; S.I., small intestine; D.C., descending colon; K., kidney.

The epigastric pain so often felt is probably referred from the downward drag of the overloaded caecum on the ascending mesocolon. When operating on cases under spinal anaesthesia I have often noticed that if any traction on the caecum was exerted the patient complained at once of sharp pain in the upper epigastrium, although the abdominal incision had been completely painless.

Putrefactive changes in the stagnant caecal contents and a chronic catarrhal inflammation of the mucosa no doubt play a part in producing both the local pain and the general toxæmia associated with the condition, and as a result of this inflammatory process one frequently sees a calcareous lymph gland along the ileo-colic artery in these cases.

It is a fact of some clinical importance that the more extreme degree of mobility, in which the hepatic flexure is so loosely attached that it drops on to the false pelvis in the erect posture and can be lifted well out of a right rectus incision, often produces less severe symptoms than those resulting from a mobile caecum and ascending colon, combined with a fixed hepatic flexure. The following case is a striking illustration of this:

Lieut.-Colonel D., aged 39, had suffered from an extreme degree of general enteroptosis for many years, with attacks of flatulent pain in the epigastrium, and sometimes in the right iliac fossa, which came on one to two hours after food. He had never been particularly constipated. In August, 1919, about an hour after lunch, he was suddenly seized with severe central abdominal pain and urgent vomiting, and presented every symptom of high intestinal obstruction. After eight hours the pain suddenly and spontaneously passed off, and when I saw him in consultation an hour later there was little to be made out beyond a very marked *habitus enteroptoticus*, with easily elicited splashing in both stomach and caecum. X-ray examination three days later showed a low-lying stomach and extremely mobile proximal colon, with the hepatic flexure lying down well below the iliac crest in the erect posture. No ulcer in the duodenum was detected by the radiologist. Exploratory operation confirmed the x-ray findings as regards the extreme enteroptosis, but revealed a chronic ulcer in the first part of the duodenum, with a flake of recently deposited fibrin on its peritoneal surface. I took the view that his previous symptoms had been due rather to the ulcer than to the enteroptosis. Accordingly, posterior gastro-enterostomy was done, the duodenal ulcer infolded, and the appendix removed. Nothing at all was done for the mobile proximal colon, in view of the very general splanchnoptosis associated with it. The soundness of the procedure adopted was demonstrated by his subsequent progress. All the indigestion and flatulence, which had been for many years attributed to his enteroptosis, at once left him, and he has lived an active life without restrictions as to diet ever since.

In such a case, where the whole proximal colon with the hepatic flexure lies down on the pelvis, the bowel has more chance of propelling its contents normally than if the ascending colon and caecum are mobile and the hepatic flexure fixed and kinked. The case may be used also to point another moral. It should not too readily be assumed that a patient with a mobile proximal colon has no other lesion. Such patients are far more prone to develop appendicitis, gastric or duodenal ulcer, and gall stones than those whose alimentary canal has not had a lifelong struggle against gravity by reason of its abnormal mobility. We do not yet know the precise manner in which gastric or duodenal ulcers or gall stones result from the caecal stagnation and catarrh associated with the mobile proximal colon. Nor for that matter do we entirely understand how hyperchlorhydria and gastric or duodenal ulcer result from chronic inflammation of the appendix. In both cases the clinical fact of their frequent association is well established, but many gaps in our knowledge need to be filled by further pathological investigation. I think, however, that we know enough to reject quite confidently the mechanical theory recently advanced by Waugh,² who suggests that the downward drag of the mobile ascending colon through its mesocolon may produce destruction of tissue in the fixed second part of the duodenum and cause duodenal ulcer. Alternatively he suggests that where the duodenum and pyloric end of the stomach give way under the strain and become prolapsed, there is a tendency for the line of strain to be transferred to the lesser curvature and cardiac end, producing an ulcer there. Without attempting to justify his very questionable hypothesis that a gradual drag exerted on the peritoneal aspect of the duodenum or stomach would produce ulceration of their mucosa, he asserts that the second part of the duodenum is "the site that is most liable to ulceration." But the experience of most surgeons shows that ulcer is very

common in the first part and relatively very uncommon in the second part of the duodenum. Mr. Waugh further remarks that "if the mode of formation of these ulcers has been rightly assumed . . . a mobile ascending colon should be present in all cases." To one who can recall, as I can, a large number of cases of both gastric and duodenal ulcer in which it has been most difficult to expose the appendix through the ordinary upper rectus incision owing to normal fixation of the ascending colon, it will hardly appear that the mode of formation has been rightly assumed by Mr. Waugh.

OPERATIVE TREATMENT.

The ideal method of dealing with the mobile proximal colon is to fix it back in the right loin in the position that we have seen is the normal one for the orthograde primate. This may be done by the method of Coffey,³ or by the method recently described by Waugh.² A considerable experience of the former method has taught me that it is often very difficult through the ordinary right rectus incision to fix the colon in a way that approximates to the normal, and there is the further difficulty that fixation by firm and unyielding scar tissue is a poor substitute for the firm but elastic support afforded by the normal peritoneal fixation of the colon. Accordingly I have latterly adopted the alternative course of reducing the load of the overburdened caecum and ascending colon by the simpler method of plication. After removal of the appendix, a continuous suture of fine thread picks up the anterior and lateral longitudinal taeniae from the base of the appendix, and is carried as far up the ascending colon as the dilatation appears to require. By this method the lumen of the caecum and ascending colon is permanently narrowed without in any way hampering the contraction of the bowel, and the clinical results have been in my experience very satisfactory.

The drastic operation of colectomy, either partial or total, with its considerable mortality, is one that should be reserved for cases with the most extreme disability, and should only be undertaken when the procedures devoid of any appreciable danger have failed to give relief.

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Remarks

ON

A CASE OF HEPATIC CIRRHOSIS ALLIED TO HANOT'S DISEASE.

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THIS case, though a single one, is perhaps worth publication on account of the failure to arrive at a correct diagnosis during life, the histological appearances of unilobular and intralobular cirrhosis, and the light recently thrown on such cases and on the problem of biliary cirrhosis by the experimental work of Peyton Rous and Lavimore, of the Rockefeller Institute for Medical Research.

A boy, aged 11 years, had influenza in December, 1918, and since then had never been well. In April, 1919, he became drowsy, and in May came under observation at the Victoria Hospital for Children. On May 23rd he was shown at the Section for the Study of Disease in Children, Royal Society of Medicine, as "a case for diagnosis," by Dr. E. A. Cockayne.¹ At that time there was slight jaundice with a trace of albumin and of bile pigment in the urine, which was high coloured but free from excess of urobilin. The faeces were normal in colour and consistency. The blood count showed 6,100,000 red blood corpuscles per cubic millimetre, 6,400 leucocytes, with a differential count of polymorphonuclears 54 and mononuclears 46 per cent. The Wassermann reaction was negative. There was an evening pyrexia of 100° to 101° F. The boy was somewhat wasted, with a slightly dark skin, which was free from any haemorrhages or spider-angiomas. There were no enlarged glands. The abdomen was distended, but there were no dis-