T H E S I S

on the subject of

"CHRONIC INTESTINAL STASIS WITH SPECIAL REFERENCE TO ITS SURGICAL TREATMENT"

Presented for the Degree of M.D.

by

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May 1923.

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INTRODUCTION. - Since Metchnikoff and Lane first aroused interest in the large intestine as being not only a useless but a pernicious structure, perhaps no subject more than chronic intestinal stasis has so arrested the attention of thoughtful medical minds or been the subject of so much controversial discussion. A wealth of suggestions as to treatment, both medical and surgical, has been advanced; but, as Pauchet says, what we still lack is a knowledge of indications - when to treat medically and when to operate and what to do when we do operate. And so we look for an infallible sign and thus far we have not found it.

I used to regard chronic intestinal stasis as a somewhat rare disease. Since I began to interest myself in the condition four years ago and to do a considerable amount of medical and surgical work connected with it I have come to regard it as one of the commonest conditions

with which we have to deal. The multiplicity of pathological conditions for which it is held responsible and the extreme surgical measures which may be called for in its advanced degrees have convinced me of the necessity for its earlier recognition and treatment; the early indications may be trifling certainly, but they are no more to be disregarded than the flitting rheumatic pains of the child.

Although the term chronic intestinal stasis should really be used merely to describe a condition of stasis in the intestinal tract, yet it is more generally used to apply to the clinical picture which is considered to result from a state of stasis. Sir Arbuthnot Lane recognises chronic intestinal stasis as that condition where there occurs an abnormal delay in the onward passage of the contents of the alimentary tract at one or more points, leading to the absorption into the blood-stream of poisonous substances in excess of what can be dealt with by the normal protective agencies of the body, and thus causing a general poisoning or, as it is called, alimentary toxaemia. The starting point is thus a delay in the passage of the contents of the alimentary tract.

But at the same time it must be noted that the existence of stasis is not determined merely by the frequency of stools. We recognise that type, described by Sir James Goodhart as the 'greedy colon' type, that may have an evacuation no oftener than once in every three or four days and yet be in apparently perfect health. Possibly they have an intestinal flora of a benign type, or they may have immunised themselves against it, or possibly retribution may yet overtake them. A few months ago I came across one of the opposite type and a markedly toxaemic type. This was the case of a woman who for nearly three months had had a troublesome diarrhoea which I discovered was resulting from a chronically overloaded colon.

Much as has been written and stated on the subject of stasis, general agreement regarding its causation and effects as well as treatment is still far from being secured. By not a few its very existence is doubted. If these would but look inside an abdomen occasionally they would probably see and believe.

It is my intention, keeping in the foreground my

own findings and results, to discuss in this thesis the present day views regarding chronic intestinal stasis, citing illustrative cases from personal experience and to deal more fully with the operative measures that have been put forward for the cure of the condition. I wish to deal more particularly with stasis occurring in the coecum and ascending colon, for it is here that I consider the condition most frequently originates. In doing so, I am not laying myself open to the accusation of not taking a wide and comprehensive view of the subject, for I am well aware that it is in this portion of the colon that indications of stasis occurring further down the intestinal tract may first show themselves. Indeed, I shall draw attention to the part which pelvic conditions in the female may play in the production of stasis, for I believe that such have not been sufficiently emphasised.

In order that our treatment of the condition should be rational and sound we should, if possible, have a clear understanding of the manner of its production. And it would be well in the first place to make a brief reference to comparative anatomy and embryology in view

of the structural anomalies which may be present in certain individuals and of the part which such may play in the production of stasis.

SOME POINTS IN THE COMPARATIVE ANATOMY, EMBRYOLOGY & PHYSIOLOGY OF THE ALIMENTARY TRACT. -

Lamarck and Darwin considered the postural adaptations of the human body peculiar to man. Modern anatomists, as Keith[1] tells us, regard these adaptations as the culmination of a series of evolutionary phases which are to be traced back in the bodies of the orthograde primates. Structural modifications both of brain and body occurred - not only adaptations in the muscular mechanism which maintains the body erect and lends support to the abdominal viscera, but elaboration and expansion of the various spinal and higher nerve centres presiding over postural and vasomotor tone, and of the centres for automatic control of the muscular system.

Fixation of the abdominal viscera has also been evolved, and, from the complete mobility of the ascending and descending colons which exists in four-footed animals, we come to the first stage of sealing down of

mesenteries in the gibbon which represents the first (or hylobatian) stage in man's posture. In the great anthropoids (troglodytian stage) is observed a sealing down of mesenteries approaching to that found in man (plantigrade stage), in whom there occurs complete fixation of both ascending and descending colons.

It is well known that the caecum, which in the course of foetal development rotates and comes to lie under the liver, descends during the later months of intrauterine life along the posterior abdominal wall to its normal situation in the right iliac fossa. Meanwhile, a series of peritoneal adhesive processes take place. 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 colons along with the peritoneal coverings of their posterior surfaces become fused with the peritoneum lining the posterior abdominal wall, so anchoring these portions of the colon firmly in position. There may be failure of this normal adhesive process to occur on the right side - it is rare in the case of the descending colon - and so arise variations in structure which make it more difficult for

the individual to react normally to his environment. For it is on the proper adjustment of structure and environment that the efficient working of the alimentary system depends. And thus we have a considerable group of individuals of all ages provided with the mobile type of ascending colon, and these are handicapped in some degree in the struggle for existence.

It is in this group of individuals in my experience that ascending colon stasis is especially liable to occur. It is remarkable what a degree of enteroptosis may occur without the appearance of symptoms. Some time ago I came across a boy of seven who had a large right inguinal hernia, the contents of which I found were the terminal portion of ileum, caecum and appendix (containing two large coproliths) and entire ascending colon; also a man who for seventeen years had had a large scrotal hernia which proved to contain over two feet of colon, part of which had become intimately adherent to the scrotal wall. In neither of these cases was even constipation present. The great majority of these individuals, then, may lead a healthy and vigorous life; but once function gives out, and given that they cannot alter the conditions of life that are

proving physically detrimental to them and remembering that stasis is a progressive condition, then their digestive downfall is assured.

What I seek to show here is that structure may be defective and that the nerve mechanism regulating muscular and postural tone may be defective. In some individuals the nerve mechanism may be so inadequate that it becomes readily exhausted by the demands which an orthograde posture makes upon it.

Let us consider a few points regarding the normal working of the alimentary tract. We are aware from the researches of Keith that the myenteric (Auerbach's) plexus contains, in addition to ganglion cells and a network of fine fibres, intermediate cells (nodal tissue) which link up the ganglion cells with muscle cells. The development of Auerbach's plexus varies in different parts of the intestinal tract and whereas it occurs in fair abundance in the distal half of the transverse colon and descending colon it shows comparatively poor development in the caecum and ascending colon. In addition to the power of initiating contractions, Auerbach's plexus has a conducting function and conveys impulses coming through

the vagi and sympathetic to the intestinal wall. Localised concentrations of nodal tissue occur in various situations dividing the gastro-intestinal tract into zones. Each zone has its own rhythm presided over by its own nodal tissue. Thus, whereas we have continuous peristalsis occuring in stomach and small bowel, peristalsis in the case of the colon is limited to those few massive waves which pass over it three or four times daily after the principal mealtimes. We recognise a series of sphincters regulating the onflow of the gastro-intestinal contents, chief among which are the cardia, the pylorus, the ileo-coecal valve and the pelvi-rectal sphincter (at the junction of the pelvic colon and rectum). Others are situated in the second part of the duodenum and in the middle of the transverse colon. Besides the muscular fibres situated directly at the ileo-coecal orifice the musculature of the ileum for an extent of about four inches above is endowed with special tonic functions. In such animals as rodents there is a well marked sphincter marking off caecum from colon, and it is possible that in man there may be a close functional co-ordination between the caecum

and ascending colon.

In the course of evolution man must have immunised himself against those bacteria which have selected the alimentary tract as their normal habitat. There they find a medium rich in various food stuffs with plenty of moisture and warmth. Free oxygen is largely absent, and in the colon where there is no free oxygen pure anaerobes can flourish. The normal inhabitants of the alimentary canal are facultative anaerobes and some of them have adapted themselves to live in the presence of bile and bile salts. The chief groups of organisms are the coli group, various streptococci, and certain anaerobes. In addition, certain others such as Bacillus proteus and Bacillus pyocyaneus may occur. In the mouth we find chiefly the streptococcus salivarius. The stomach and duodenum are practically sterile, but as we pass down the small intestine bacterial growth begins again and here we find coli bacilli and scanty streptococci, the latter being of the non-pathogenic 'short-chain' variety. It is in the caecum and colon where the intestinal contents are delayed to allow absorption of the last remnants of nutrition that bacterial growth is

most abundant. Here we have B. coli and streptococcus faecalis in almost equal numbers - though the former usually predominates - and putrefactive proteolytic anaerobes such as B. aerogenes capsulatus.

VIEWS on CAUSATION & EFFECTS of CHRONIC INTESTINAL STASIS. -

What precise factor or factors determine the occurrence of chronic intestinal stasis are still far from being understood.

That stasis is a product of modern and artificial life - strain, stress and unsuitable diet - would appear to be evident from the absence of its manifestations amongst the uncivilised races as reported by some competent observers, notably McCarrison.

Probably many cases of stasis begin in the early years or months of life. Certainly one can often obtain a history of chronic constipation and recurring bilious attacks from an early age. In 1901 Dr. Eric Pritchard came to the conclusion that overfeeding during the first year of life was responsible for over-

development in length of the bowel with consequent delay in the passage of its contents due to impeded peristalsis. I have been struck by the frequency with which manifestations of stasis first show themselves in young adults, and especially in young women of from seventeen to twenty-five years of age. Many of them give a history of having been engaged for some months or years at some straining occupation the demands of which they found themselves increasingly unable to meet. They invariably show poor general and vascular tone with low blood pressure. A large proportion in whom operative treatment became necessary showed varying degrees of coloptosis. Walton[2] considers this fact as largely due to the neglect of static muscles by young women at the present day.

Probably what occurs in the first instance in these subjects of enteroptosis is a neuro-muscular exhaustion causing diminished tone of the musculature of both bowel and abdominal wall with consequent diminution of intraperitoneal pressure. It is probable that the weakened pelvic and abdominal muscles cause dyschezia which leads to loading up of the pelvic colon so

increasing the difficulties of the colonic musculature. As a result of the impairment of its normal supporting mechanism the bowel, weighed down by its contents to the limits of its normal movement, compels the mesenteries to assume a suspending function for which they were not intended. Tension exerted on the mesenteries beyond a certain degree produces pain. It is probable, furthermore, as Tyrrell Gray[3] suggests, that the constant stimulus to the sympathetic fibres within the mesenteries inhibits intestinal movement while compression of the veins by the undue tension leads to venous stasis which further impairs muscular action.

There is a growing tendency to regard some cases of stasis as probably part of a general nutritional disorder arising from a deficient or ill-balanced diet, the effect of which is felt by the colon in common with other organs, more especially the endocrine system. McCarrison has recently shown that feeding animals on a diet deficient in Vitamin B. produced a marked dilatation of the intestine, necrosis of Auerbach's plexus, and decrease in the adrenalin content of the suprarenal glands. Gross[4] is still carrying out

experimental work on the subject, but his results so far are similar to those of McCarrison and he shows that the intestinal stasis resulting from Vitamin B. deficiency is chiefly iliac and colonic. The intestinal lesions described by these two observers bear a strong resemblance to those described by Keith in his examination of colons removed by operation for chronic intestinal stasis and which will be mentioned later. If we could show that bowel changes such as these are produced in young children as a consequence of a deficient diet I believe that it might go far to explain why tubercle should gain a foothold in the intestinal tract.

I believe that it is only by careful observation of the intestinal tract at laparotomy, noting any variations in the mobility of the bowel, the state of its walls, and the presence of adventitious bands in various situations, and correlating these findings with the early history and symptomatology, that we will attain to an earlier recognition of stasis.

The caecum and ascending colon which have to propel their contents against gravity in the erect

posture, have great demands made upon them. This part of the colon is doubtless intended to extract the last food remnants from its semi-fluid contents; and so it is as if nature has provided for a physiological stasis by arranging that its contents should have to be propelled along a channel against gravity. A mobile ascending colon is further hampered in its movements by lacking the purchase afforded by firm fixation to the posterior abdominal wall. There is some degree of strain also on the transverse colon, the distal half of which has an uphill course. The descending colon has an easy downhill course and is firmly fixed, but there is some strain again at the beginning of the sigmoid where the colon passes over the pelvic brim. We are aware that a meal reaches the terminal ileum in from four to five hours, but that owing to contraction of the ileo-coecal sphincter a physiological stasis occurs to permit of adequate digestion of the ileal contents. After a meal, when normally the ileo-coecal sphincter relaxes and the caecum and ascending colon become filled with more or less fluid contents, the weight of this column of

fluid in the erect posture exerts a downward pressure on the caecum. As a consequence of a break-down in the neuro-muscular mechanism there occurs a tiring and stretching of its muscular walls, a dilatation of its cavity and ultimately a displacement downwards into the pelvis. Owing to its lack of propulsive power it may act very much like a paralyzed urinary bladder from which only the surplus escapes. Rotation of this loaded caecum from right to left tends to occur. This may give rise to a severe ileal torsion (as Jordan has pointed out) as well as occasional attacks of volvulus. Though there may be marked coecal stasis occurring I have found that constipation is not always present; indeed, occasional attacks of diarrhoea may occur due, no doubt, to irritation of the bowel by its putrefying contents. The hydrostatic pressure exerted by the coecal contents may cause obstruction to the ileal efflux and possibly at times actual regurgitation may take place owing to forcing of the ileo-coecal valve, thus leading to an ascending infection of the ileum. I believe that spasmodic obstruction may occur at the ileo-coecal sphincter as a result of the heavy prolapsed

caecum pulling on and partially kinking the more or less fixed terminal ileum, and I would compare the mechanism to the pylorospasm often induced by a dilated ptosed stomach which is causing partial kinking of the fixed pylorus. The weight of such a loaded caecum as felt in the hand is often remarkable, and the downward pull which it exerts may account for the drooping of the hepatic flexure and the elongation of the ascending colon so frequently present. Prolapse of the transverse colon may become so marked that acute angulation occurs at the fixed splenic flexure. The theory put forward by Waugh[5] that the downward drag by a mobile ascending colon causes duodenal or gastric ulcer is interesting but its correctness is doubtful.

Effieient musculature in the ascending colon is of the first importance, for it is there that most of the peristaltic waves arise, and colonic muscle which ceases to function properly probably tends to atrophy.

Associated with this condition of the proximal colon I frequently encounter bands of adhesions in certain situations. Jackson's pericolic membrane - a thin veil-like structure with small vessels coursing

over it - passes from the parietal peritoneum of the posterior abdominal wall in a downward and inward direction to be attached to the anterior surface of the ascending colon often extending as far up as the hepatic flexure. This membrane is often very dense about the middle of the ascending colon and may form a definite constricting band. Other bands may be present at the hepatic flexure pulling it up and occasionally causing obstruction there; and where the caecum is prolapsed obstruction is more liable to occur at a fixed hepatic flexure than a ptosed one. Lane's ileal band is a fan-shaped band on the lower surface of the mesentery of the ileum, three to four inches from the ileo-coecal valve and attached to the antimesenteric border of the ileum and is designed, according to Lane, to retain the end of the ileum in position and to oppose the downward displacement of the caecum.

Other bands may be present at the beginning of the sigmoid, at the splenic flexure, and below the gall bladder, where a band from the gall bladder, liver, duodenum and pylorus passes down to be attached to the

transverse colon.

Wilms[6] described the mobile caecum as congenital and the primary cause of chronic intestinal stasis, but he failed to observe the presence of bands; while Jackson[7], who in 1908 drew attention to the pericolic membrane, regarded coecal stasis as secondary to obstruction produced by this membrane.

Regarding the origin of these bands there is still much controversial discussion and three theories have been advanced - the inflammatory theory, the congenital theory, the acquired evolutionary theory.

Those who support the inflammatory theory consider these membranes to be the result of recurrent inflammatory attacks such as occur in the course of appendicitis, colitis, cholecystitis, duodenal ulcer etc. Against this theory of inflammatory origin it has to be stated that these bands commence at a point most distant from the intestine and then gradually approach and secure the bowel. Also they occur only on one side of the peritoneum and the side on which strain is exerted. Moreover, the constancy of their position and direction, as well as their occurrence along with a perfectly normal

appendix etc. are strongly opposed to this theory.

With regard to the congenital theory there is no doubt, as Keith[8] and others have shown, that these or similar bands may be present at birth or in late foetal life. This theory regards these bands as resulting from variations in the normal peritoneal adhesive processes - both as regards the time and extent of their occurrence. These adhesive processes are most variable in extent in the region of the iliac colon and the ileo-coecal valve. The lower portion of the ileum with the ileo-coecal valve region and caecum may be completely fixed down to the posterior abdominal wall at an early stage. Later on in life when the terminal ileum becomes mobilised by drawing away from the posterior abdominal wall the original adhesion remains and constitutes Lane's ileal band. Non-descent of the caecum is regarded as due to abnormal entanglement by the right margin of the omentum. If the caecum eventually overcomes this obstruction to its descent the affected part of the omentum becomes drawn out into Jackson's pericolic membrane. Keith, however, does not claim that all adhesive structures found in

adults are embryological and normal. Those who support this theory consider that these bands may lead to constipation.

The third theory as to the causation of these bands is Lane's, namely, that they are acquired evolutionary structures and are identical in origin, function, and structure, and are due to an effort to prevent prolapse of the abnormally loaded bowel. Lane recognises two extreme types, and has pointed out that those of poor vitality and resisting power make no attempt to prevent elongation and prolapse of the bowel; whereas individuals of considerable vigour respond by the formation of bands which at first support the bowel, but which may later contract and lead to obstruction. According to Lane,[9] the first band to develop - the so-called "first and last kink" - is that occurring at the beginning of the sigmoid and is an effort to support the loaded pelvic colon. The effect of this band is to cause an accumulation of contents in the entire colon which becomes supported by various bands along the "crystallised lines of strain." Contraction of the bands next occurs in order to lend better support, and

so angulation and kinking may result, especially of the lower end of the ileum. The ileum proximal to the kink becomes distended. This distended ileum drags on its mesentery and so kinks the duodeno-jejunal flexure, perhaps also causing partial torsion of the beginning of the jejunum, with the result that the duodenal effluent is obstructed and the duodenum dilates. This causes pylorospasm which leads to dilation of the stomach which tends to prolapse, and so leads to further "crystallised lines of strain" in the shape of adhesions between the pylorus and gall bladder and liver.

Such is Lane's theory of a drainage system completely at fault, the large bowel forming a cess-pool which cannot be properly emptied, and infection and stagnation extending up all along the gastro-intestinal tract.

I have encountered these bands in a considerable number of cases and I have met Lane's ileal band and Jackson's pericolic membrane in quite young children. What has struct me is the frequency with which some

degree of Jackson's membrane occurs in most abdomens. I have frequently found it co-existing with definite inflammatory pericolonic adhesions - occasionally the right margin of the omentum has been firmly involved in them - and the two types of adhesive bands are readily distinguishable.

It appears to me a matter of indifference what view we hold regarding the causation of these bands. Whether they actually play any considerable part in producing mechanical obstruction is the subject of much controversy. They are to be observed on many occasions where they have been causing no trouble whatever. It is common experience that a remarkable degree of matting and adhesion of coils of bowel may occur in tubercle and after general peritonitis where there is no obstruction to the onflow of the bowel contents. Probably the musculature and its innervation are the factors which count most. I am bound to admit that, in my own cases where X-rays showed the existence of an ileal stasis, and where at laparotomy a Lane's ileal band was found, in no case so far have I been able to satisfy myself that such stasis was resulting from actual mechanical

obstruction, even though definite kinking and rotation of the terminal ileum existed. I believe that the obstruction in these cases was due to spasm of the ileo-coecal sphincter, for, as Hurst reminds us, the ileal efflux is of such fluid consistence that it can find its way through the narrowest aperture. I consider that a "controlling" appendix may also frequently produce ileal stasis by a spasmodic rather than a mechanical action. Probably the mechanism is similar to that at the pylorus where peripyloric adhesions may induce pylorospasm.

Within the last few weeks I have operated on two cases of supposed appendicitis where the appendix was perfectly normal but where a well-marked ileal band was rotating the terminal ileum. In both cases the predominant symptom was pain, referred to around the umbilicus, occurring immediately after food and lasting for a variable time. In one case this symptom had been constantly present for over six months. Probably the pain was due to the accumulating ileal effluent striving to make its way through a spasmodically contracted ileo-coecal sphincter. That such an explanation would

appear to be correct is shown by the fact that since division of the bands the pain has disappeared.

Looking broadly at the subject of chronic intestinal stasis, I find it useful to regard it as really a chronic intestinal obstruction which may be mechanical, spasmodic, or due to atony of the musculature of the colon either in its entire extent as in advanced degrees of visceroptosis or in a localised portion, particularly the caecum.

I have already discussed the special liability for stasis to occur in the proximal colon and have attempted to give some reasons for such. At the same time it has to be stated that an obstruction further along the colon - whether it be due to a band at splenic flexure or sigmoid, a tonically contracted descending colon or some intra-pelvic cause - may first manifest itself at the caecum. The changes might be compared to those that occur in a cardiac chamber behind a stenosed valve. Certainly the blind end has the chief strain thrown upon it.

I would like to emphasise here the part which pelvic conditions in the female, and especially the retroverted uterus, frequently play in the production

of colonic stasis. I believe that the 'neurasthenic' and other symptoms frequently accompanying such a displacement are in many cases due to intestinal toxaemia. There are those who regard retroversion of the uterus as a result of chronic intestinal stasis. Doubtless where there is much fat absorption due to toxaemia with consequent lack of pelvic support this is often so. I believe that it is also frequently the cause.

Just the other week I was asked to fix up the uterus of a woman who, I was informed, had had a retrodisplacement for several years. A colpo-perineorrhaphy performed seven years previously had failed to give her any relief. She had been severely constipated during that time and she looked definitely toxaemic. There was no lack of pelvic fat, but the uterus was enlarged and heavy and impacted against the rectum. In the course of my usual routine examination of the bowel, I discovered in the region of the sigmoid a faecal concretion which had ulcerated through a diverticulum and which was surrounded by inflammatory adhesions. There were no adhesive bands present in the sigmoid or elsewhere.

Another case which I would mention was that of a woman who complained of severe pain on defaecation and pain 'low down' at her menses. The condition had troubled her for over three years. She had consistantly disregarded the normal call to defaecate because of the almost intolerable pain. She showed the typical facies of severe alimentary toxaemia. Pelvic examination revealed the presence of a cystic left ovary - the result of some old gonorrhoeal mischief - which at operation proved to be intimately adherent to the rectal wall. Removal of the cystic ovary resulted in complete cure of the constipation and of the alimentary toxaemia.

Nearly two years ago I attended a lady upon whom Jones[10] operated in 1916. At that time she was suffering from rheumatoid arthritis - there being considerable stiffness and swelling of her fingers and both knees - as well as a serious cardio-renal condition. A band was found on rectal examination occluding the lumen of of the bowel. Removal of the band led to complete disappearance of the rheumatoid condition and the

renal condition improved very remarkably. From being a chronic invalid she regained almost normal health within a month or two. When I saw her I found the joints perfectly normal, but the damage that had been inflicted on the kidneys was again showing evidence.

I have considered how stagnation of the contents of the bowel may arise. What then occurs as a result of such stagnation and how are the normal conditions of the alimentary tract affected by it? Delay favours increased bacterial activity; but as yet we know little regarding the nature of the poisonous substances which on absorption give rise to general poisoning, or alimentary toxaemia. We certainly do not appear to have the power of immunising ourselves against them.

There is no evidence that exo- or endo-toxins of the normal bacteria of the bowel are a cause. It is probably to the products of protein decomposition that we have to look. We know that proteins in the course of digestion are split up into amino-acids which are carried to the liver, where they are reconstituted for body requirements. As a result of putrefactive activities of certain bacteria, especially the strict

anaerobes in the colon, these amino-acids may be changed into other highly poisonous substances, many of which are still unknown. Thus, as Prof. Dixon[11] has shown, tyrosine may be changed into tyramine (para-hydroxy-phenyl-ethylamine); histidine into a poisonous histidine base; tryptophane into indolethylamine; arginine into agmatine. Cadaverine, putrescine and sepsin are also derived from putrefaction of the corresponding amino-acids. All these substances have a definite physiological action. Indol is formed from bacterial putrefaction of tryptophane and certain other amino-acids and represents the activity chiefly of coli bacilli. Indican and skatol seem to be more or less inert substances and do not indicate the degree of alimentary toxaemia present. Indeed a severe degree of such may occur without their presence in the urine. Intestinal poisons are normally dealt with by the neutralising action of the bowel mucosa, the antitoxic action of the liver and probably certain internal secretions. Where stasis occurs, an overwhelming amount of poison is formed and the protective mechanism breaks down. The first line of defence, the bowel

mucosa, weakened by chronic catarrh, gives way, and the remaining protective mechanism in time gives way also. It is in the caecum and ascending colon, where the contents are more or less fluid, that putrefactive changes are greatest, and stagnation here may possibly cause an upward bacterial spread and so lead to increased bacterial activity in the lower end of the ileum. It is to an ascending infection of the small bowel as a result of ileal stasis that Lane ascribes the severe degrees of auto-intoxication. Alterations in the bacterial flora of the ileum have been demonstrated at operations for colectomy, Mutch[12] showing that the predominating streptococcus was of the pathogenic "long chain" type. It is possible that the behavour of the ileo-coecal sphincter depends on variations in reaction of the chyme produced by such bacterial infection. I believe it is of importance to be able to discover alterations in the flora where stasis is occurring, and I shall later describe how this may be achieved in the case of coecal stasis in the course of operative treatment.

Let us consider briefly the effects of these poisons

on the body. That they are virulent is evident from the remarkable deteriorative changes that may take place in the tissues where severe alimentary toxaemia is occurring. The toxins generally show a selective action and seem especially to attack those organs and tissues that are normally engaged in excreting toxins. The skin, kidneys, and liver suffer early, but sooner or later every tissue and system suffers, and the patient gradually lapses into a state of chronic invalidism.

The cutaneous structures show early changes. The facies is so absolutely characteristic that from it one can invariably tell these patients most of their symptoms. The healthy glitter disappears from the face. The complexion becomes sallow and pasty and may even be slightly icteric. Patches of brown pigment appear round the eyes and may extend over the face and neck and to other parts of the body. The eyes are heavy and dull and often sensitive to light. The inelastic skin throw the face into furrows which give the face a troubled and anxious expression. They look the picture of misery. There may be considerable anaemia.

The hair may lose its healthy lustre and the nails may become brittle. The breasts tend to atrophy and may show cystic degeneration. There is often profuse offensive perspiration. They invariably become markedly thin owing to absorption of fat from the body. The occurrence of fat absorption within the abdomen coupled with the flabby wasted state of the abdominal muscles favours the occurrence of visceroptosis. Moveable kidney is frequently present. Muscular and vasomotor tone is generally poor. These patients become readily fatigued and peripheral circulation is poor, as shown by the livid cold extremities and the frequent occurrence of Raynaud's phenomenon. In some cases the heart muscle suffers and various irregularities of rhythm may occur owing, no doubt, to toxic action on the cardiac muscle and its nerve regulating mechanism as frequently pointed out by Sir James MacKenzie.

The muscular wasting is noticeable on palpation of the abdomen which is lax and doughy. On going through the abdominal wall one notices in marked cases the extraordinary wasting of the various structures. The toneless skin causes the wound edges to invert rather

than gape. There may be practically no adipose tissue and one comes down directly on fascia which is soft and atrophied. There is frequently little or no bleeding. The muscles are pale and atrophied. The parietal peritoneum may resemble a thin membrane and frequently tears on stitching. From the poor quality of all the structures one can readily see how ineffective is the support afforded to the bowel by such an abdominal wall.

The nervous system tends to suffer. They become nervous and depressed. The memory becomes impaired. 'Neuritis' and 'neuralgia' and altered sensations are frequent. Headaches are often severe and sleep may be troubled. There is exaggeration of the reflexes. These individuals are generally labelled "neurasthenia" and too often we are content to accept the term without seeking to discover its underlying cause.

There is often marked hepatic inadequacy. Many of these patients come complaining of 'liver' - blessed appelation too often used as a cloak for our ignorance!

Nephritis may occur as a result of implication of the kidneys. I even observed this complication in a child of little over a year in whom there had been an

extremely obstinate constipation since birth (it was not a case of megacolon). There was a severe colitis present. Treatment - colon lavage, etc., eventually brought about an absence of albumen from the urine.

There are those who go much further and ascribe practically every known disease to the results of auto-intoxication. Thus, upward spreading infection of the small bowel leads to infection of the pancreatic and biliary ducts and gall bladder with production of calculi and finally carcinoma of pancreas and gall-bladder. Infection of duodenum and stomach leads to duodenal and gastric ulcer. Reaction of the thyroid gland to the toxaemia causes enlargement - simple adenomatous or exophthalmic in type. Arteriosclerosis and degeneration of the kidneys lead to hyper trophied heart and high blood pressure. Involvement of the articular structures may lead to the occurrence of rheumatoid arthritis and Still's disease may occur in children.

The toxaemia by its selective action presents different symptoms according to the organ or system affected. Thus, in many of my operation cases, the

typical cutaneous changes were present; in others they did not occur. Some again exhibited chiefly nervous symptoms, others thyroid affection and two or three albuminuria. One girl who had taken very frequent epileptic fits since she was a child was cured by a course of medical treatment. And it may be that, although there is no obvious toxaemia or interference with health in many individuals, slow degenerative changes are nevertheless occurring in the body. In many of these cases the large bowel is bluish, inelastic and toneless with the most marked changes occurring in the mobile proximal colon which generally shows considerable dilatation and atrophy of its walls. Very commonly the chief changes are to be observed in the caecum which may be so dilated that all its muscular elements are gone, and rarely its fibrous walls may be little thicker than tissue paper. Where there has been prolonged catarrhal inflammation of the mucous membrane of the bowel with bacterial invasion of the neuro-muscular tissues, the colon may be thick and soggy, and frequently this degenerative change is confined entirely to the caecum. Stercoral

ulcers have been found post mortem in such a caecum. It is invariably the proximal colon that shows the most marked degenerative changes.

Portions of colon removed by operation for chronic intestinal stasis have been examined by Keith[13] who classifies them into two groups. The first group were of the soggy type showing intense inflammatory infiltration of all the coats. The chief changes were found in the mucous coats, the interglandular retiform tissues of which had become crowded by proliferation of its constituent elements, and Auerbach's plexus showed degeneration in areas. The caecum and lower end of ileum showed greatest involvement.

The second group showed only a slight degree of chronic inflammatory change, but was characterised by the presence of pigmented cells in the retiform tissue of the mucous coat. The muscular coats and Auerbach's plexus showed structural changes very similar to those described by McCarrison in deficiency diet in animals, of which mention has been already made. Around the pigmented masses were numerous mononuclear lymphocytes apparently engaged in digesting the pigmented debris.

Salkowski[14] and also Brahm and Schmittmann[15] regard this pigment as a waste product derived from metabolism of fat and of the same nature as melanin. There is, in the opinion of these writers, some close relationship between the adrenal function, the motor nerve plexus of the bowel, and pigment formation.

It has been suggested that these changes in the colon may be due to an increased adrenalin content of the blood leading to constriction of the lymphatics passing through the muscularis mucosae layer and so to lymph stasis. It has still to be discovered whether a bowel belonging to either of these two groups could recover its efficiency or whether removal by operation is the only method of cure. What we are in need of is a clinical test for determining the efficiency of the colon just as we test for renal or gastric efficiency.

The question of diagnosis I need do little more than touch upon.

It is well to begin, as Hurst suggests, by making a rectal examination 24 hours after a bismuth meal. In some cases the delay will be shown to be occurring

in the rectum and to be due to dyschezia. Where the patient is a woman a pelvic examination under anaesthesia should be performed in nearly every case. Some intra-pelvic obstructive factor may thus be discovered or a prolapsed loaded caecum palpated. I even go so far as to suggest that every woman suffering from colitis should be given the benefit of such an examination. At the same time one has the opportunity of performing a thorough abdominal examination.

The sigmoidoscope may reveal the cause of stasis low down, and a barium enema may prove of value. From X-ray examination of the gastro-intestinal tract after a barium meal definite information may be gained as to where stasis is occurring; but in my experience X-rays are not always satisfactory and are frequently fallacious. Probably they are only useful where stasis is very marked, and in affording information as to the state of the bowel wall and the position of the various portions of the colon.

I have found the charcoal test of considerable help. A quantity of charcoal is given immediately after breakfast and the time of its appearance in the stools

is noted. The total time taken by the gastro-intestinal contents to pass along is thus obtained.

Examination of the gastric contents after a test meal invariably shows marked diminution or complete absence of free hydrochloric acid. I have derived no help from examination of the faeces.

I have come to rely chiefly on the clinical appearances and signspresent. There is this definite clinical type which as a result of observation during surgical treatment I can now readily recognise. In some cases the evidences of auto-intoxication are most prominent; in others the obstructive element predominates. In the latter group there is a type whose symptoms are chiefly gastric and which may reproduce almost faultlessly the classical symptoms of gastric or duodenal ulcer. This type is invariably appendicular or coecal. There are no symptoms of stasis which are pathognomonic of the condition; collectively they cannot readily be mistaken for anything else. It cannot be too strongly emphasised that the multiplicity of symptoms frequently is the most important diagnostic point, as it is only the presence

of a circulating poison which could produce changes in so many organs.

PRINCIPLES of TREATMENT. -

(a) Non-operative:- Before proceeding to the treatment of a case of stasis every clinical method at out disposal should, if necessary, be used to determine the cause and site of the stasis, and where a definite pathological obstructive factor exists surgical treatment should not be delayed.

I believe that it is our frequent failure to recognise or deal early with apparently trifling conditions in those possessing an over-responsive nervous system that so frequently superadds a mental state to the physical condition. Eventual surgical interference may fail to eradicate the nervous superstructure and even the abdominal symptoms may fail to be relieved. Too often these individuals become the possessors of an imposing display of abdominal scars, and of many it might truly be said that their last state is worse than the first.

In a large proportion of cases medical measures may be sufficient to effect a cure. Such treatment should be directed towards preventing the formation of poisons in the bowel, aiding their elimination and repairing the destruction wrought by the toxaemia. At the same time we may have to correct ptosis of varying degrees and restore an atonic bowel musculature.

Simple laxatives, a suitable diet, and possibly an abdominal support may be all that is necessary in early stages. Where the condition is more advanced and where visceroptosis may be a feature the horizontal or Trendelenburg position for a period may be necessary. Render them into pronogrades for the time being. The Trendelenburg position, by taking strain off mesenteries, may entirely relieve the pain, and particularly the pain which many of them get at certain times every day due to loading up of the caecum; but I have occasionally seen no relief and even aggravation of symptoms from this position in cases where the caecum was so prolapsed or tied down in the pelvis that it was being doubled back by the pelvic brim, which was thus acting as an actual obstructive factor.

Having obtained thorough unloading of the bowels, I generally begin treatment with three days of alimentary rest, when measured quantities of clear soup or tea are given four hourly with abundance of sterile water or soda water in the intervals; no solids of any kind being given. Practically no nitrogenous residue, therefore, reaches the ileo-coecal region and so toxin formation is prevented or restricted, thus favouring the recovery of the protective agencies of the body. Possibly the increased power of utilising sugar which invariably follows alimentary rest in the treatment of diabetes is due in some measure to this recovery of hepatic and possibly pancreatic functions.

The diet then given should be a light nourishing non-distending one free from red meats, and it is better to avoid sloppy milk foods. Buttermilk is frequently of great advantage. Many of these patients are extremely thin and so a "fattening cure" may also be necessary.

Laxatives should be mild. The hurrying on of an imperfectly digested ileal effluent may lead to the contents of the entire colon being fluid instead

of solid, thus favouring putrefactive changes. I rely principally on an infusion of senna pods - which has the advantage of acting on the entire colon - mild saline aperients and liquid paraffin, the last being given preferably before the principal meals so as to precede the passage of food.

Most of these cases, especially where colitis exists, derive great benefit from a course of colon lavage with normal saline. I continue its daily use for a short period, but omit those occasional days which are set aside for alimentary rest. I have found inflation of the colon with oxygen of definite therapeutic value; presumably the growth of certain putrefactive anaerobes becomes inhibited. I generally have the oxygen administered two hours after a colon douche. The technique is rather important: the rectal tube should not be introduced until the requisite amount of oxygen is coming, and the latter should not be turned off until after removal of the tube, i.e. the key of the cylinder should not be turned while the tube is in position in case of accidents.

Abdominal and general massage, vocal and respiratory and gymnastic exercises, and electrical treatment all play an important part in restoring muscular tone. Endocrine gland preparations and preparations of vitamins, such as metagen, have proved to me of some value. Where the nervous system is irritable bromides should not be withheld.

Hydrochloric acid after meals is of benefit where there is a diminution or absence of this normal antiseptic element of the gastric secretion and is probably no less important than intestinal antiseptics and intestinal vaccines. I sometimes advise a large dose of bismuth at weekly or fortnightly intervals and it is noteworthy that it seldom increases an existing constipation. Probably, as Hurst pointed out years ago, it produces a constipating effect only where there is bowel irritation owing to the excessive formation of sulphuretted hydrogen.

A Curtis belt may be necessary in the subjects of visceroptosis once they begin to get about again. I believe that this belt acts, not by actually holding up the bowel, but by increasing intraperitoneal

pressure, and chiefly by providing a firm resisting surface from which the bowel can derive purchase just as it does from the posterior abdominal wall. The belt should be removed at times to permit of the carrying out of abdominal exercises and massage. Rest in the prone position after meals for a short time is also of importance when we consider the physiological happenings around the ileo-coecal region at these times.

That psychotherapy is of value in some cases is undoubted. Is it possible that the influence of the mind can prevail over obstructive bands and kinks? But should we not expect it to overcome at least certain types of primary rectal stasis? Many of these patients need much encouragement, and the importance, for successful treatment, of an understanding of the mental outlook of such cannot be sufficiently emphasised.

On a system of treatment such as I have briefly indicated the majority of these cases do very well; but I am finding more and more that, by the time one sees many of them, there is an obstructive factor which only surgical treatment relieves.

(b) OPERATIVE TREATMENT. -

At the outset one is bound to confess that the surgical treatment of chronic intestinal stasis is still largely in the melting-pot. There is no established basis of treatment, and practically no type of operative treatment has given certain success. Whatever our method of treatment, we should set out with the fixed object of securing efficient drainage of the bowel. The question largely resolves itself into dealing with a chronic obstructive factor whether mechanical, spasmodic, or one due to muscular inefficiency of the colon in general or of the caecum in particular. My results have been best where I have been able to demonstrate a definite mechanical obstruction. As I have already indicated we may have to deal only with some intrapelvic obstructive factor. I contend that every case should be judged and treated on its own merits; treatment by one unvarying operation appears to my mind irrational and unsound, and particularly if that operation be a severe one. I think that all operative interference should be as simple as we can make it, so that the existing condition may not be replaced by another with

perhaps more serious possibilities. I have relied chiefly on a policy of reconstruction of the alimentary tract; and the results of the large amount of work I have had opportunities of observing and of carrying out during the last few years have amply justified such procedure and have brought me to the conclusion that extensive surgical measures are seldom called for in the treatment of stasis.

Where the cause of the stasis is on the right side I use a large pararectal or paracentral incision - I believe much is often missed by operating through a small gridiron incision - and it is of the first importance to preserve, if possible, the nerves going to the rectus muscle.

There is no divergence of opinion regarding the treatment of an inflamed or adherent appendix which may be holding up an ileal effluent by inducing spasm of the ileo-coecal sphincter or producing a mechanical block by controlling the terminal ileum. I shall later describe a case where severe ileal stasis was resulting from a mesenteric cyst - an ileal diverticulum - which was causing flattening together of the ileal walls. This

was dealt with by resection of the portion of ileum containing the diverticulum and performing a lateral anastomosis.

The division of bands may give striking results as I have already pointed out. Where bands are producing kinking or rotation of the bowel I divide them and carefully cover in the raw edges; where an ileal band is producing no obvious kinking and where there co-exists a prolapsed caecum, fixation of the latter into the right iliac fossa will invariably be sufficient to take any strain off this so-called internal lateral ligament of the caecum. I think it is important to allow for drainage after division of a fairly extensive ileal band because of the danger of peritonitis resulting from the septic organisms present in the lymphatics of the band.

Very extensive ileal bands or dense adhesions around the terminal ileum may call for an ileo-sigmoidostomy, as it is doubtful whether permanent relief is afforded by simple division; and, moreover, there is the risk of inducing a severe post-operative ileus. It is generally recognised that the mortality of short -

circuiting operations is always greatly increased if these adhesive bands are divided at the same time. Where extensive ileal bands exist, Paterson performs the operation of colonic exclusion in preference to ileo-sigmoidostomy. According to his method, the divided proximal end of the ileum is anastomosed laterally to the sigmoid colon. The latter is then divided proximal to the anastomosis. The operation is completed by performing appendicostomy, or, if the appendix is not available, caecostomy, in order to allow for drainage of the excluded colon. He also performs this operation for severe cases of atony of the colon.

Appendicostomy as an isolated operation for stasis need hardly be mentioned here. It is simple and free from risk, but only a make-shift operation after all. I have known it prove of benefit in those cases of stasis in which there is superadded a severe ulcerative or haemorrhagic colitis - cases in which even colectomy so frequently fails. I do not regard colon lavage through an appendicostomy opening as probably any more effective than intestinal lavage per rectum; and as has been abundantly shown in appendicostomy cases such douches reach right round to the caecum.

The more cases of stasis that I see the more do I find the cause to be a mobile atrophic caecum and ascending colon. Only very exceptionally do I find the same atrophic changes existing on the left side of the colon. I have already made mention of the state of affairs on opening the abdomen in these cases - the dilated toneless prolapsed caecum, often blue and soggy in type; the ascending colon usually similarly affected, but occasionally constricted in its middle or upper portion by a dense pericolic membrane; the appendix generally the seat of some chronic inflammatory change. At first I used to wonder why attention to the appendix alone in such cases failed to relieve their symptoms, or relieved them only until they began to get about again. Now I also deal with the proximal colon and there has been a corresponding improvement in results. Treatment is designed to secure firm fixation of this portion of the colon as well as a diminution in its calibre, on the principle that the walls of a small cavity will have more purchase on their contents than those of a large dilated one, the amount of muscular tissue in the two cases being approximately equal. Since 1919, the procedure which I have generally adopted

in these cases has been briefly as follows: Appendicectomy is first performed and the pericolic membrane, if very dense, is removed. A continuous catgut suture beginning at the base of the appendix and proceeding a variable distance up the ascending colon picks up the anterior and external longitudinal bands so invaginating the segment of bowel between them. Where the ascending colon shows much dilatation I carry the plication nearly up to the hepatic flexure. The circumference of the bowel is thus considerably diminished. It is a good plan first of all to rub vigorously with a piece of gauze the surface of bowel wall to be invaginated so as to favour adhesions. I then proceed to fix the ascending colon and caecum into the right flank and right iliac fossa as Wilms[16] advised in 1908 and also Waugh in 1920. The method of fixation which I have found most satisfactory consists in the shortening of the mesocolon by puckering it up in two or three situations; so that it is the junction of the ascending colon with its mesocolon which becomes fixed to the posterior abdominal wall. I also fix up a ptosed hepatic flexure and a markedly dropped transverse colon.

For some time I have been performing another operation for marked atonic dilatation of the caecum and I believe this method to be much more reliable than caecoplication. By this method, the segment of caecum instead of being invaginated is removed. Briefly, the technique is as follows: The abdomen is opened by a long pararectal incision. The caecum and part of the ascending colon are brought well outside the wound which is well protected by cloths wrung out of normal saline. The appendix is taken away and Jackson's membrane removed or pushed aside. A pair of bowel clamps is then applied longitudinally along the caecum (and sometimes including the beginning of the ascending colon) in such a way that the segment between the anterior and external longitudinal bands is grasped. Another pair of bowel clamps is now applied external, parallel and close to the first pair and the segment of caecum is removed by cutting between the clamps with a knife. The clamp first applied is therefore still in position. Having cauterised the cut edges with pure carbolic, a running suture of catgut is inserted from end to end passing through all the coats. The bowel clamp is now removed. An occasional bleeding point may require

control. Tissue forceps are now applied at either end of the coecal wound and held well apart by an assistant. Another running catgut suture is now inserted picking up the sero-muscular coats and invaginating the first line. It is my custom to insert a third line of very fine catgut picking up the serous coat. When this has been completed, the calibre of the caecum and beginning of ascending colon is not much greater than that of the ileum. The operation is completed by fixing the ascending colon and caecum into the right flank and right iliac fossa. No drainage is necessary and recovery is as uneventful as that of an ordinary appendicitis. I have used this method in a number of occasions and the more I use it the more I become struck by its effectiveness. I am aware that many do not regard the plication method as giving in all cases a permanent diminution in calibre. This operation has at least three advantages:-

1. The calibre of the greatly dilated caecum is permanently diminished and we may expect stasis here to be permanently relieved.

ii. An opportunity is afforded for determining the bacterial flora at the site of stasis and a vaccine can be prepared. I believe it is the bacteria invading the mucosa or present in the mucus adherent to it that really matter. Cultivating from the faeces is likely to prove misleading. I have had a vaccine prepared in two or three of these cases, and, although in the majority vaccine treatment is unnecessary, in certain others I believe it is a valuable adjunct to operative treatment.

iii. The piece of coecal wall can be examined macroscopically and microscopically and in doubtful cases such examination might reveal a degree of degeneration requiring hemicolectomy or colectomy.

I believe that by these reconstruction methods a dilated prolapsed and mobile caecum and ascending colon which might hitherto have been acting as a veritable cess-pool can be made to perform their physiological function and send on their contents normally. I am so convinced of the liability to ascending colon stasis where a loosely attached ascending colon exists that when I encounter such at any laparotomy I invariably fix it firmly to the posterior abdominal wall. In view of the very satisfactory results I have obtained in actual stasis cases I believe that in so doing I am helping

certain individuals to meet conditions which might otherwise overcome them.

Amputation of the caecum is a good operation where the caecum has become so ballooned that all its muscular elements have disappeared or where the coecal head shows actual cystic formation.

I have never performed a colectomy nor an ileo-sigmoidostomy in a case of chronic intestinal stasis, though I have had opportunities for observing the after-results in a few cases; I have not so far met with a case where I considered either of these methods justifiable.

Ileo-sigmoidostomy has given brilliant results in certain cases. The best results probably are obtained by an end to side union as advised by Lane, the divided proximal end of the ileum being implanted into the sigmoid or rectum; at the same time the angle between the iliac colon and sigmoid is artificially increased to lessen the possibility of regurgitation of faeces into the colon. Appendicostomy or caecostomy may be performed at the same time to allow for drainage and colon lavage. Afterwards it is desirable that three evacuations daily be obtained to prevent loading up of

the pelvic colon. In a considerable proportion of cases a secondary colectomy becomes a necessity owing to faecal regurgitation; even in many apparently successful cases it is probable that the colon still continues to act, the contents passing back from the rectum along the colon to its coecal extremity.

Ileo-sigmoidostomy, in short, is an unscientific operation; its mortality is probably nearly as high as that of colectomy; and there is a fairly general feeling that its results are not so good.

The operation of colectomy cannot be regarded other than a serious one, and one not to be undertaken lightly, attended as it is under the best conditions by a mortality of about 16.5%; and while it may have brilliant successes its frequent failures may, as Lord Dawson says, be "such dire failures as to mean to the patients misery and suffering little capable of alleviation." It is difficult to regard the colon as a useless structure; our knowledge of its functions, and it has probably many important ones, is still too fragmentary and incomplete. Keith tells us that its glandular structure put into a mass would form a gland larger than the pancreas and its

muscular tissue a muscle bigger than the biceps of a blacksmith. Moreover its powers of recovery as seen in cases of chronic dysentery are remarkable. What we strive to know is what degree of degeneration of its structure should exist before colectomy becomes a necessity. When we have a completely degenerated organ with muscular tissue absent - afibrous sac in other words - then colectomy offers the only prospect of a cure. In the hands of a brilliant surgeon such as Lane, colectomy may be a good operation, but it is an operation which, so far from not gaining in general favour, is being performed increasingly less. I observe that whereas 34 colectomies were performed at Guy's in 1915, and 13 in 1918, only one was performed in 1920 and none in 1921. These are rather significant facts. Apart from the immediate mortality and the danger of obstructive complications due to adhesions of small bowel to omental tags, we have remote results which are far from uniform. There are the favourable cases, the incurable diarrhoeas, the obstinate constipations where stasis continues as marked as before, and those in whom the pre-operative symptoms persist.

Where the degenerative changes are confined to the

caecum and ascending colon - and they are frequently confined to this portion of the colon - hemi-colectomy is probably preferable to a total colectomy. The caecum, ascending colon and proximal half of transverse colon are removed and the divided proximal ileum anastomosed to the transverse colon. Here also, as in colectomy, although omentum may be used to cover up raw surfaces, there is the risk of intestinal obstruction from peritoneal adhesions. I think there is also this to be said against the operation that we have the distal ileum exerting through the anastomosis a downward pull on the gastro-colic omentum, and so on stomach.

By the operation of colectomy, partial or complete, we remove the normal protective mechanism of the small bowel - the ilio-coecal valve - and the loss of this important structure permits of an ascending bacterial infection of the ileum the fluid contents of which tend to undergo excessive fermentation and putrefaction. It is interesting to know what changes occur in the bowel muscle after colectomy. Tyrrell Gray[3] has shown that the anastomosis enlarges afterwards to from two to three times its original size and becomes thinned and atrophied

like the adjacent bowel. In hemi-colectomy this atrophy involves the small bowel adjacent to the anastomoses; whereas, in complete colectomy, the small intestine progressively hypertrophies and dilates. We can thus understand why, although the immediate results of hemi-colectomy may be brilliant, the remote results have proved so frequently disappointing; and also why colectomy for the loose toneless colon of visceroptosis gives such poor results.

It has occurred to me, if necessity should arise to perform a colectomy, to try and arrange for the preservation of the ileo-coecal sphincter. Du Pury, in his operation of caeco-sigmoidostomy - which can only be performed where the sigmoid is sufficiently mobile to be brought over to the caecum - certainly achieves this, and he claims good results. But I cannot see how we would, by this method, be any more likely to escape the possibility of faecal regurgitation into the colon than with an ileo-sigmoidostomy.

In conclusion I would emphasise that chronic intestinal stasis is a condition met with exceedingly commonly in every day practice. The slight degrees of toxaemia

to which it gives rise are frequently overlooked and the advanced degrees may occasionally occur as conditions generally regarded as separate clinical entities, such as albuminuria, exophthalmic goitre, rheumatoid arthritis and neurasthenia. Where these conditions occur, a careful investigation of the intestinal tract should be carried out, and in female subjects an examination should be made for possible intrapelvic conditions causing partial bowel obstruction. One of the commonest varieties of stasis is that occurring in a mobile proximal colon, and the direction in which prevention will be achieved here may prove to be the recognition of the type at an early age and placing the individual later on under conditions of life which will throw least strain on the defective structure.

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A P P E N D I X.

The following are a few selected cases illustrating the main contentionsof the above thesis:

Case I. A case illustrating the conscious abdomen and neurasthenia.

Mrs G - aged 48, whom I first attended in 1919, had suffered from severe constipation all her life, and for years had experienced a sickening, dragging pain in her back. Her health had been miserable, she had always been extremely nervous and at intervals had had to lie up for spells of weeks or months because of her abdominal condition. Her appendix had been removed seven years previously with no benefit and she had been twice curetted with only slight temporary advantage. For a few years she had at frequent intervals received treatment for colitis, and all that time she was under excellent medical advice. Menstruation had ceased three years previously.

She was of the thin, anxious, neurasthenic type and she presented the features of a marked alimentary toxaemia. The complexion was muddy, the face furrowed, the skin inelastic and the general tone extremely poor. The tongue was thickly coated. There was no oral sepsis. The appetite was poor and she was having great difficulty with her bowels and was passing varying quantities of mucus. The abdomen was lax, doughy and tender on palpation

especially over the proximal colon. There was a movable right kidney. The horizontal position relieved her pain in some measure.

I attended her at intervals for over two years and during that time every conceivable method of treatment was used with no improvement. Latterly her abdominal troubles became increasingly marked and she was really a total nervous wreck. On examination at that time I made out a retroverted uterus. X-rays showed a marked degree of coloptosis with definite delay occurring in the proximal colon. I advised operation to which she gladly consented.

At operation in March, 1922, I corrected the uterine displacement, plicated the greatly dilated and prolapsed caecum, and fixed the excessively mobile ascending colon and caecum into the right flank and iliac fossa. I also stitched up the dropped hepatic flexure and transverse colon. The pelvic colon showed considerable elongation. There were no bands present at any part.

She began to improve rapidly after the operation. Her colour became healthy, and nervous symptoms diminished remarkably. In the first few weeks she put on over a

stone weight. And now a year after her operation she looks fat, has practically no abdominal discomfort, and constipation is no longer a source of trouble though a mild laxative is still necessary. She is able to attend to her household duties and she says that she feels better now than she has ever done.

Case II. A case of marked ileal and coecal stasis simulating duodenal ulcer.

Mr Samuel M - aged 36 years, came to see me first in August, 1922, complaining of epigastric pain, occasional vomiting and persistent constipation. He gave a history of an attack of appendicitis just before leaving for Canada twenty years previously and of two subsequent similar attacks, unaccompanied by vomiting, several years later. He was very well until he returned to this country in 1918, when he began to experience more or less constant epigastric pain which continued up till the occasion of my seeing him. Ingestion of food relieved the pain for a half to one hour when it returned as before. Vomiting, which also gave relief, occurred only rarely, but he frequently brought up sour mouthfuls. He

frequently took bicarbonate of soda and magnesia because of the temporary cessation of pain which they afforded. He had to live chiefly on milk diet as meats always aggravated his symptoms. Constipation had been a marked feature for over twenty years and he had continually to take large doses of purgative medicine. His colour at times he described as almost jaundiced. There had been no haemorrhage from stomach or bowel.

When I saw him he was miserably thin and poisoned looking. The tongue was thickly coated. There was no sepsis from teeth, nose or throat. No localised epigastric tenderness was present. Although he had no appendicular pain I was able to make out definite tenderness on deep pressure over this region as well as marked splashing. Nothing abnormal was present in the various other systems. There was marked indicanuria and the charcoal test revealed a delay of 72 hours. I recognised the case as one of the ilio-coecal type and advised operation.

I was not greatly surprised to find that instruments for a gastro-enterostomy had been put out. On opening the abdomen I found a chronically thickened appendix

adherent well up under mesentery of the ileum. The ascending colon which was dilated and somewhat elongated was provided with a mesentery. Jackson's pericolic membrane was well-marked and was particularly dense about the middle of the ascending colon. The caecum was prolapsed and dilated to nearly the size of one's fist. The pull which it was exerting on the appendix was causing the latter to act like a sling-band controlling the terminal ileum. The small bowel was distended and the duodenum markedly so. Careful examination revealed no duodenal or gastric ulcer.

I removed the appendix, and, having removed the pericolic membrane, I plicated the caecum and most of the ascending colon and fixed these portions of the colon to the posterior abdominal wall.

The symptoms as well as the toxaemia entirely cleared up after operation. His colour became healthy and he gained $1\frac{1}{4}$ stones in a month. Five months later he told me that he had enjoyed excellent health with complete freedom from gastric symptoms; his constipation was quite relieved and he had put on $2\frac{1}{2}$ stones in three months.

Case III. A case illustrating ileal stasis due to a mesenteric cyst.

I was first called to see Miss N - , aged 19, in November 1920, because of severe abdominal pain. She gave a history of life-long trouble with stomach and bowels, and life had been a constant misery to her. She had always experienced a dull deep pain in her abdomen after meals, and there was the feeling as if "something were pressing against something." Consequently she had refrained from food as far as possible. Even between meals there was vague abdominal discomfort. No vomiting had ever occurred but headaches had been frequent and severe. There had been persistent constipation and it was usual for several days to elapse without an action of the bowels. There were occasions when she had been confined to bed for two or three days with "liver" attacks. Apart from measles she had had no other troubles as a child. Menstruation had been normal. When I saw her she was having attacks of colicky abdominal pain which heat partly relieved. There was much tenderness and resistance to pressure in the lower abdomen and especially in the right iliac region where undue fulness

was evident on palpation, The tongue was thickly coated, but she had not vomited. The temperature was 100° and pulse-rate 92.

Operation on the following day revealed the presence of a mesenteric cyst - an ileal diverticulum about the size of a cricket ball - incorporated with the ileal wall about 1½ feet from the ileo-coecal junction. The manner in which the ileal walls were flattened together caused one to wonder very much how any ileal contents could have passed through at all. A portion of ileum containing the cyst was resected and a lateral anastomosis performed.

The pathological report was as follows:-

"This is a large spherical mesenteric cyst attached to a piece of small intestine. It is about the size of a cricket ball. The vessels supply its outside wall and are in direct and normal communication with those of the mesentery. Internally it has no communication with the intestine, but the inner wall of the gut is grooved at right angles with mucosal folds for a length of about two inches. The interior of the cyst is velvety and smooth, similar in all its parts. The contents were a

thick pink glairy fluid, which coagulated at once on boiling. Microscopically the fluid contains large numbers of undamaged red corpuscles and crystals of cholesterin.

"Sections of the wall show externally vessels, under which are muscular coats regularly disposed and exactly like those of its ileum.

"The inner lining is in parts necrotic, but shows a great many gland cells and in some cases the outlines of intestinal glands.

"There are patches of inflammation here and there on the mesenteric surface.

"There can be no doubt that this cyst represents a diverticulum which has been shut off but continued to expand because there was no outlet for its secretions. Eventually the mucosa itself was digested."

This was a case of marked ileal stasis. She is now quite well and her symptoms have been entirely relieved, though she still requires a mild laxative on occasion.

Case IV. A case exhibiting exophthalmic goitre as the main feature.

Mrs Mc F - , aged 54, first consulted us in November 1921 because of prominence of the eyes, severe palpitation, nervousness and digestive disturbances. She stated that for many years she had suffered from flatulence and indigestion, attacks of severe distension, and oppressed breathing. Constipation had been a trouble for over 30 years. There had been no abdominal pain. In March 1921, she began to experience palpitation, nervousness and irritability, and her friends noticed prominence of her eyes. She had become very thin and her face had assumed what she called a 'dirty pale' colour. She received medical treatment during the period from March until we saw her first in November, 1921.

She presented the features of a well-marked case of exophthalmic goitre, and she exhibited an extreme degree of thinness and palor. There was marked exophthalmos. The right lobe of the thyroid gland was definitely enlarged. The heart, which was not enlarged, was rapid and irregular, the rate being invariably around 130. There were no cardiac murmurs audible. Dr. Jones and

I, who both saw her, formed the opinion that this was a case of ileal and coecal stasis. This was confirmed at operation when an adherent appendix was removed and a mobile caecum, which was atrophic, dilated and prolapsed, was plicated and fixed into the right iliac fossa.

Within a few weeks the enlarged right lobe of the thyroid gland diminished to almost normal size, palpitation ceased to trouble her and her digestive symptoms cleared up. There occurred a marked improvement in her general condition. She is now very well and has displayed no further evidence of hyperthyroidism. She has put on two stones in weight since her operation.

Case V. A case illustrating asthma as the chief symptom.

Robert M - , aged 28 years, a miner by occupation, was first seen by me in May 1922, because of asthma and severe intercostal pains. He stated that his health had been good up till six years previously when he had an attack of pleurisy after which the asthmatic condition began. What he called a/"severe paroxysm" had occurred

nearly every night. He had expectorated abundant clear sputum which had been examined on a few occasions for the presence of tubercle bacilli with negative results. Indigestion had invariably troubled him. The bowels had tended to be rather loose, but latterly he had begun to experience constipation for which he took frequent doses of castor oil as he "thought it helped the asthma." There had been no abdominal pain. He had been able to work only for occasional short spells during those six years, and he had been having fairly constant medical attention.

When I saw him he looked thin and sallow. There was no elevation of temperature and the pulse-rate was normal. There was much intercostal fibrositis present. He exhibited the signs of an ordinary bronchial asthma. The sputum was abundant and clear. There was no sepsis from teeth or nasal sinuses. Abdominal examination revealed marked splashing over caecum and ascending colon, but there was no tenderness on pressure. I put him on treatment for the chest and bowel condition, but with little benefit. The charcoal test revealed a delay of 50 hours and there was marked indicanuria. On

August 15th. he complained for the first time of severe crampy pain in the right iliac region. I diagnosed a chronic appendix and advised operation.

At laparotomy I found a prolapsed ballooned caecum and an adherent appendix which showed evidence of chronic inflammatory change. There was a well-marked ileal band 3" from the ileo-coecal junction rotating and kinking the terminal ileum. I removed the appendix, divided the ileal band, plicated the caecum and fixed it into the iliac fossa.

The asthma began to clear up in a wonderful manner even before he left hospital. No further paroxysms occurred, and in the course of a few weeks it practically ceased to trouble him. His general condition improved remarkably and he put on weight. Although I advised him to get other work he has been earning his living as a miner for several months without interruption and lately he told me he had never felt better in his life.

This was a definite coecal case with probably some ileal stasis due to the ileal kink present. The case is of interest in the light of Prof. Dixon's investigations on the Histidine group of decomposition products which he

showed cause increased tonus of plain muscle, especially that of the bronchioles.

Case VI. A common type of stasis occurring in the proximal colon.

Miss B - , aged 27, first consulted me in November 1921, because of increasing digestive troubles and marked failing of general health. She had enjoyed excellent health up till nearly four years previously when she took up the duties of a nurse in a military hospital. She very soon began to feel her health suffer from the long hours and prolonged standing to which she had been previously quite unaccustomed. She became readily tired and exhausted and began to suffer from an occasional dragging pain in the right side. There developed more or less continuous epigastric discomfort amounting to actual pain at times, and this was relieved for a short time by the taking of food. She lost her healthy colour which became sallow and pasty and she was troubled with a profuse acue eruption. The bowels had acted with fair regularity.

When I saw her she displayed the features of a well-marked alimentary toxaemia. The complexion was

pasty and sallow and the tongue thickly coated. There was no oral sepsis present. X-rays revealed a marked degree of enteroptosis with delay occurring in the proximal colon. She was put on medical treatment for fully three months but with little relief from her symptoms. Operation in February 1922, revealed a chronically inflamed appendix and confirmed the marked degree of enteroptosis revealed by X-rays. The appendix was removed and the prolapsed caecum, ascending colon and hepatic flexure stitched up after performing coecoplication.

Immediate relief from her symptoms followed and she quickly regained her former good health.

This is a type of case one meets with very frequently and the history of the mode of onset is also very typical.
