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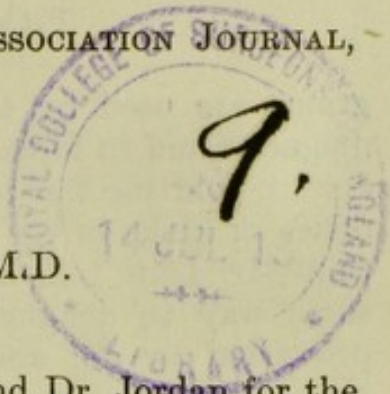
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ILEAL STASIS

By LEWIS GREGORY COLE, M.D.

New York



WE are indebted to Sir Arbuthnot Lane and Dr. Jordan for the interest which has been elicited in this country and abroad in the question of intestinal stasis. It cannot be denied that such a condition exists and is the cause of a certain group of symptoms; and it is also true that in many instances practitioners and surgeons have failed to recognize it as such. The extreme theories of Mr. Lane and some of his disciples have served their purpose in exciting the antagonism of conservative observers, and have resulted indirectly in the discussion of this subject at the present meeting.

While I may have a very definite opinion as to whether or not intestinal stasis is a cause or *the* cause of a large group of ills to which humanity is subject, such as cancer, tuberculosis, nephritis, senility, insanity, lack of sexual desire, etc., my discussion must be limited to a consideration of the subject from the roentgenologic standpoint. The whole subject of intestinal stasis centres around the roentgenologic findings. If it were not for this method of examination intestinal stasis would still be discussed under the term constipation. Therefore it is essential that the roentgenologic foundation be sound, solid, and scientific or the whole superstructure will fall.

The indirect or continental method of examination, based on the detection of symptom complices, is as the name suggests, simply the observation of various groups of symptoms by an unusual method and not direct evidence of the lesion itself. Each roentgenologist compiles his groups of symptom complices, corresponding to the different diseases he has to diagnose, with the result that they are as varied as the observers who describe them. The direct method, on the other hand, is based on the detection of morphologic changes in the wall of the gut, or direct evidence of spasms of functional origin, by means of roentgenograms or serial roentgenograms.

Roentgenology may become a two-edged sword. The damage and danger of its use cannot be overestimated, when roentgeno-

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grams are used by the surgeon to demonstrate a preconceived diagnosis and to force the patient to submit to surgical procedure. Jordan told me that at first he seldom found roentgenologic evidence of kinks in the various portions of the small intestine, but that Mr. Lane told him they were there, and that he must find some way to demonstrate them roentgenographically. This he proceeded to do, and very graphically described among other phenomena the writhing duodenum, due to obstruction at the duodeno-jejunal junction. Now this condition does exist, and I have demonstrated it roentgenocinematographically, but it is not found with anything like the frequency that some observers would lead us to believe. A writhing duodenum may be obtained in any case, as Holzknicht has shown, simply by exerting pressure on the duodeno-jejunal junction and pressing it against the spinal column. Great care must therefore be observed to differentiate between real and artificial obstruction at this point. There are likewise cases supposed to be suffering from kinks of the terminal ileum and ileal stasis, which show no roentgenologic evidence of delayed evacuation at this point. The roentgenologic diagnosis in such cases is based on mobility under deep palpation of certain localized areas of the terminal ileum during fluoroscopic examination. Personally, I believe that this is insufficient evidence upon which to base a diagnosis of ileal stasis requiring surgical relief.

In other instances the diagnosis of intestinal stasis has been based on the length of time during which bismuth was observed in the terminal portion of the ileum after its ingestion. For this consideration the fundamental question is,—what is the normal time for the bismuth to remain in the terminal ileum? Jordan, in his recent tour of this country, illustrated cases of "ileal stasis requiring surgical procedure" by demonstrating slides showing retention in the terminal ileum six, six and one-half, and seven hours after the ingestion of bismuth. When I put the question to Sir Arbuthnot Lane, he stated that he "felt uneasy about ileal stasis after the seventh hour following the ingestion of bismuth." According to Lane, therefore, seven hours is the limit of time during which chyme may normally be found in the ileum after the ingestion of bismuth-impregnated food. I habitually make roentgenograms of the entire gastro-intestinal tract during the seventh hour after ingestion and in more than 98 per cent. of all the cases examined bismuth has been found in the ileum at that time. In fully one half the cases there is a trace of chyme in the stomach, cap or duodenum during the seventh hour. In other cases, particularly if the stomach is of the cow-horn type, complete gastric

evacuation takes place in two or three hours. Here is an important source of error in reckoning ileal stasis. If ileal stasis is reckoned from the time of ingestion, the problem becomes a complex one, that of ileal stasis plus gastric retention. To determine the degree of stasis in the ileum one may reckon the period that elapses between the ingestion of food and the time when the first bismuth-impregnated food passes into the cæcum, except in cases of stasis due to insufficiency of the ileo-cæcal valve. These results may be checked up by noting the time that elapses between the complete evacuation of the stomach and the evacuation of the ileum. The first is the more accurate and convenient method of determining true ileal stasis, because when the ileum is dilated one usually observes an accompanying functional gastric retention, which may be prolonged for eight, ten, twelve, or fourteen hours without any organic obstruction of the pylorus or cap. This of course prolongs the ileal stasis, because the chyme is four to eight hours late in arriving at the ileo-cæcal junction.

Roentgenologic evidence indicates that ileal stasis or rather ileal dilatation may be caused by (1) incomplete evacuation or atony of the cæcum and ascending colon, (2) various types of membranes and veils involving the colon, (3) kinks of the terminal portion of the ileum, (4) insufficiency of the ileo-cæcal valve, (5) chronic appendicitis (primarily or secondarily from adhesions, either before or after the appendix has been removed). All of these conditions may be recognized and differentiated from each other with a remarkable degree of accuracy by a thorough roentgenologic examination, preferably in conjunction with a serial roentgenographic examination of the stomach and duodenum.

The symptoms of ileal stasis are rather varied because sometimes the symptoms of the cause prevail, and sometimes the symptoms of the effect prevail, particularly those referable to the stomach and cap. These symptoms will be considered under the discussion of each cause.

Colonic stasis is perhaps the most common cause of ileal stasis. Since the time when man dispensed with forefeet and assumed the erect posture, it has been up-hill work for the ascending colon to evacuate itself. Overdistention of the cæcum and ascending colon constitutes a large proportion of colonic stasis. The dilatation and atony of this region is partly compensated for by the active peristalsis of the terminal portion of the ileum. In proof of this, bismuth-impregnated fæces will be observed in the cæcum much longer after a bismuth meal followed by a period of fasting,

than after a bismuth meal followed by the ingestion of food or possibly water. If the stasis (or constipation) in the ascending colon is persistent, unusual energy is demanded from the peristalsis of the terminal ileum. When its strength is not sufficient to break the blockade, delayed evacuation results, accompanied by a chain of symptoms, perhaps referable directly to the exciting cause, "cæcal constipation," or perhaps referred to the stomach and cap. To overcome this obstruction, bismuth-impregnated chyme may be seen frequently forcing its way up through the fecal accumulations in the cæcum and ascending colon, or the next meal succeeding the bismuth meal may be observed working up through the ascending colon. If in such cases the cæcum and ascending colon can be evacuated by properly applied manipulation, massage or even catharsis, previous to the ingestion of more food, stasis of food in the terminal ileum will thus be diminished, and often the gastric or duodenal symptoms referred to the right hypochondrium will be relieved. Moreover lesions in this region of a much more serious character may perhaps be prevented.

The various types of veils and membranes involving the cæcum, ascending and first portion of the transverse colon undoubtedly cause an ileal stasis, either directly by affecting the colon or terminal ileum or indirectly by the associated constipation which blocks ileal evacuation. These lesions may be diagnosed with a great degree of certainty by roentgenography, preferably combined with roentgenoscopy. The drawing up of the cæcum, the irregular filling defects in the colon, and particularly the "double-barrel-shotgun" appearance of the ascending and first portion of the transverse colon, referred to by George, are very characteristic roentgenographic findings. Such conditions are undoubtedly important factors in the cause of obscure symptoms referred to the stomach and cap, particularly those associated with hyperchlorhydria, and should be searched for when the right hypochondrium fails to reveal the seat of the trouble. Sometimes the direct symptoms of constipation are so severe that the presence of gastro-duodenal symptoms is entirely overlooked.

I fear that the treatment of these conditions is not so simple as some surgeons would lead us to think. One group of surgeons will split these membranes, remove the veils, and watch the colon pop out of its cage, believing that the patient is freed from symptoms forever. Unfortunately they are likely to return, renewed by the conditions which originally caused them, or by the trauma of manipulation from surgical procedure. In other cases where there

is atony and dilatation, some surgeons attempt to reduce the size of the colon and hold it in position by artificially producing the conditions which the aforementioned group of surgeons try to destroy. The most successful method of surgical treatment for such cases could be determined by post-operative roentgenologic investigations in conjunction with careful clinical observations.

Kinks in the terminal portion of the ileum, either with or without mobile cæcum, undoubtedly do occur and cause the group of symptoms described by Lane and recognized by others. On several occasions I have been able to recognize them with an accompanying dilatation of the proximal ileum, and I have been able to demonstrate them to the most sceptical observer. But these kinks are a rare rather than frequent cause of ileal stasis. Where a kink really exists and causes ileal stasis and dilatation, especially if it is associated with symptoms, surgery for its relief is undoubtedly indicated. But operative procedure intended for the relief of such kinks will not cure all cases of ileal stasis, nor relieve the accompanying symptoms. Furthermore if the stasis is not caused by a kink, operative procedure may aggravate the symptom.

Insufficiency of the ileo-cæcal valve is a condition which I first recognized in 1902. Out of the cases examined since that time, I have observed about two hundred and fifty cases of this irregularity, and have used every effort to arouse the interest of surgeons and practitioners in the subject without success. Even now in cases where the clyisma passes all the way to the duodenum, and the patients present marked abdominal symptoms, surgeons and practitioners refuse to attribute any significance to this finding. I find that the only way I can interest them is to ask if they would expect symptoms, if they fed their patients fæces removed from the colon. I have failed to report this group of cases, at first because the question of tuberculosis occupied my attention and later because my efforts were concentrated on gastro-duodenal lesions. Kraus, Schwartz, Holzkecht, and more recently Groedel and Dietlan, have reported roentgenographic observations of ileo-cæcal insufficiency. Case has published an article, and on several occasions demonstrated slides, showing the frequency with which this lesion manifests itself. Its clinical aspects have been described by Kellogg, who has recently devised an operation for repairing an incompetent valve, and a method of constructing an artificial one, which will undoubtedly create a great interest in the whole subject.

Some of my cases of insufficiency of the ileo-cæcal valve, particularly the first ones that I recognized as such, where the bismuth had passed a long distance up the small intestine, were associated with periodical attacks of nausea, vomiting, fever, prostration and headache, and with cramping abdominal pain, especially marked in the right quadrant of the abdomen. This is the group of symptoms which is attributed variously to bilious attack, migraine, auto-intoxication, and frequently appendicitis. Sometimes, if the symptoms are sufficiently indefinite, the patient is permitted to retain his appendix, at least temporarily, in case it has not been removed already.

Reflecting on the roentgenologic evidence, it occurred to me that this group of obscure symptoms was very likely due to the influx of large quantities of fæces, loaded with bacteria and their products from the colon, where they are normal, into the small intestine, which is relatively sterile compared with the colon. To increase my confidence in this theory, I found that the severity of the symptoms was proportionate to the length of the small intestine which the cæcal contents traversed. Many cases were observed, presenting a mild degree of insufficiency, and the accompanying symptoms were only slight, or if acute, the attacks occurred at long intervals. Kellogg and Case have already referred to the ileal stasis, caused by or associated with this lesion, and my experience corroborates their observations.

The importance of insufficiency of the ileo-cæcal valve justifies a communication limited to that subject alone, but it is impossible to consider it further in this communication, where it has been introduced merely as one of the potent factors in ileal stasis, and consequently in spasmodic and organic lesions of the stomach and cap.

The roentgenologic findings of chronic appendicitis are of immense diagnostic importance. The appendix, partially or completely filled, has occasionally been found by many roentgenologists, and exhibited at meetings or perhaps reported as a monstrosity. But Case and George deserve the credit of observing the appendix roentgenographically often enough to justify them in drawing conclusions as to the significance of its roentgenographic appearance. Adhesions accompanying an involved appendix which flatten the side of the cæcum are frequently observed both before and after an appendectomy, and in my opinion are of more significance than the actual demonstration of the appendix itself, unless it retains the bismuth content for many days.

In summarizing the foregoing remarks it may be said that ileal stasis does occur, but not as frequently as some observers claim to find it. Its presence is due to or associated with colonic stasis and dilatation of the ascending colon, pericolonic veils and membranes, kinks of the terminal ileum, insufficiency of the ileo-cæcal valve, and chronic appendicitis, before or after the removal of the appendix. The common error in reckoning true ileal stasis, together with the custom of operating for the relief of stasis which does not show roentgenographically, has been sowing to the wind, and even conservative roentgenologists throughout the country are reaping the whirlwind of criticism. The use of roentgenograms as a weapon with which to urge surgical procedure for some preconceived diagnosis should be vigorously condemned.

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