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Spastic colon

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THE SPASTIC COLON.

SENIOR THESIS.

EDWIN MYRON KAHN.

UNIVERSITY OF NEBRASKA. 1933.

INTRODUCTION.

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How familiar to every physician in the patient who comes to the office with the following complaints: Abdominal pain, diffuse or localized in the right lower quadrant; varying degrees of discomfort, fulness, belching, or nausea after meals, headache, nervousness, and other more or less vague and non-specific complaints. In many instances, the physician makes a physical examination in which he fails to discover anything significant. The usual laboratory procedures are equally unproductive. The physician then shakes his head, or rubs his hands together, depending on his temperament, and tells the patient that he has chronic appendicitis, and that an operation is indicated. The operation is performed, and the appendix examined. The surgeon reports that the appendix shows no evidence of gross pathology, but that there is, perhaps, a suggestion of constriction at the base, which may account for the symptoms. The patient makes an uneventful recovery. A month after operation, he reports that his symptoms are much improved, or have entirely disappeared. Six months or a year later another physician receives a call from the same patient. The patient reports that he is suffering from abdominal pain, diffuse or localized in the right lower quadrant; varying degrees of discomfort, fulness, belching, or nausea after meals with headache, nervousness, and other more or less vague and non-specific complaints. The second physician suggests that perhaps the patient is suffering from chronic appendicitis, whereupon the patient informs him that he has been relieved of his symptoms for a time following an appendectomy, but that they have since returned as badly as ever. The physician shakes his head, mutters something about a neurotic, writes a prescription for some sort of a placebo, and another dissatisfied patient is started on his round of the irregular practitioners.

The next patient has a similar list of complaints, but in addition states that fatty foods seem to disagree with her. She weighs over one-hundred and fifty pounds, and has had three children. The radiologist reports that although there appear to be no stones in the gall bladder, it does not empty as rapidly as it should. Whereupon the physician tells the patient that she is suffering from a chronic gall bladder infection, and should have an operation before matters become worse. The patient goes to the hospital, loses her gall bladder which appears quite normal at operation, makes a nice post-operative recovery, feels better for a while, and is then surprised, and dissatisfied to find that her symptoms have returned. But she still has faith in her physician, and returns for further advice, and treatment. This time, he informs her that it must be the appendix that is causing her difficulties. She goes to the hospital again, and returns minus her appendix, but plus the same old syndrome. And so another patient goes to the chiropractor, the osteopath or the christian science practitioner, each in turn getting credit for a cure.

The next patient is a man. His symptoms do not differ greatly from the others except that he thinks that his symptoms come on about an hour or an hour and a half after meals. Although the gastric examination and x-ray examinations show nothing, and occult blood cannot be demonstrated in the stools, his ~~physician~~ feels that the clinical picture is so typical of peptic ulcer that it is advisable for the patient to enter the hospital for Sippy management. For reasons that will appear later, the patient responds very nicely to this management. His physician is greatly pleased, and chooses to think that his diagnosis is now fully confirmed. Perhaps he even kids the radiologist about the case when he meets him at lunch. At any rate, the patient is discharged from the hospital and placed under ambulatory management. As his diet is gradually

augmented, he discovers that his symptoms are returning. From here on, anything may happen from a gastroenterostomy to a session with the psychoanalyst. Eventually the patient becomes disgusted and joins the great body of people who believe that doctors are a ~~crew~~ of thieves, swindlers, and nincompoops.

It is the purpose of this paper to present a disease, which the writer believes to be much more common than generally supposed. This disease, or rather syndrome, has been given various names such as: Spastic Colitis; Spastic Colon; Enterospasm; Functional Colon; Colospasm; and Irritable Colon. The importance of the condition lies chiefly in its differential diagnosis, for in itself it leads to no fatalities or serious morbidity. As suggested in the preceding paragraphs, it may closely simulate a number of common gastric or intestinal conditions, and is a common cause of much unnecessary abdominal surgery. The writer believes that the profession would do well to direct their attention to this syndrome, and to consider it in the differential diagnosis of all doubtful cases of abdominal distress.

HISTORY:

So far as may be determined from the literature, Spastic Colon was first described in 1830. In that year, John Howship, Surgeon to St. George's Infirmary, London, published a small book entitled, "Discrimination and Successful Treatment of Spastic Strictures of the Colon." (53). Apparently, this passed unnoticed by the profession for a matter of sixty years, for the next mention of this condition proceeds from a meeting of the International Medical congress at Paris in 1890. At this meeting, Spastic Colon was said to have been discussed in some detail, although this report is not well authenticated.

After this follows a period of seventeen years in which no reference to the condition can be found in medical literature. In 1907, Max Einhorn again called the attention of the medical fraternity to a condition which he termed enterospasm, and which presented a similar clinical picture to the present disease (38). Although Howship is given credit for the discovery of Spastic Colon, almost equal credit is due Cherewsky who in 1883, three years later than Howship, independently observed and described the condition (47).

Of late years, the condition has been receiving the attention which it deserves, and we find many articles in modern medical literature dealing with the subject. Even so, the interest shown in the disease by modern medical writers is in no way commensurate with the diagnostic importance of the disease or with its frequency of occurrence. Also, it is worthy of note that most of the work done so far on Spastic Colitis has been along clinical lines. The writer is unable to discover that adequate scientific research has been carried out on this condition. Therefore, it is obvious that the various etiological factors to be mentioned later are based on supposition, and are by no means proved. It is

equally patent that the treatment is largely empirical, and as such not entirely successful. It is to be hoped that further research along physiological lines will eventually lead to a better understanding of the etiology, symptomatology, and intelligent treatment of this condition.

INCIDENT:

The incident of Spastic Colon in respect to frequency, Age, sex, Color, nationality, and constitutional type has been variously estimated by different investigators and clinicians. Morgan (38) believes it to be present in two-thirds of all young adults, thereby almost bringing into the classification of a normal physiological state. Jordan and Kiefer(27), on the other hand, find it present in only one-third of all patients exhibiting gastro-intestinal disorders from all causes, while McLoone(33) believes that half of his patients with gastric disturbances are suffering with colospasm. Spriggs(50) is of the opinion that it occurs in five per-cent of the general population, and in five to twelve and a half percent of diseases of the colon.

In regard to sex, most writers state that spastic colon is more frequently observed in members of the female sex. Eggleston(9) states that the ratio of female to male sufferers is seventy-five to twenty-five. Drueek(8) also thinks that it is of more frequent occurrence in women, as does Gauss(14), who concurs in Eggleston's estimate of a three to one ratio. McMillan(34), and Ryle(47), on the other hand, fail to observe any difference of incident as between the two sexes.

In regard to age, there is a more or less general agreement among writers that spastic colon occurs most frequently in middle age. This view is taken by Morgan(38), McMillan(34), Ryle(47), and McLoone.

No investigators have observed any difference in respect to color or nationality.

ETIOLOGY:

There is no single etiological factor that has been as yet found to be responsible for spastic colon. In this respect it must be considered as a symptom rather than a definite disease entity. There are, however, a large number of factors which have been frequently enough found associated with spasm of the colon to entitle them to consideration as possible or even probable etiological agents. For the sake of unity, it has been deemed expedient to classify these factors under the following general heads: 1, Psychic Causes; 2, Reflex Causes; 3, Local Pathology; 4, Local Irritation; 5, Deficiency Disease; 6, Intoxications; 7, Infectious Causes; 8, Neurological Causes; 9, Allergy; 10, Endocrine Disturbances; and, 11, Metabolic Disorders.

It is logical to consider first the local conditions which may give rise to spasm of the colon. Machechrie (32), and Hurst (23) believe that colospasm is sometimes due to the presence of chronic appendicitis, believing that the mechanism is either that of local irritation or due to reflexes originating in the diseased appendix. Machechrie also believes that the cells of Aurbach's Plexus may become irritated under other conditions, and produce a spasm. However, he fails to mention what specific irritants may produce the effects mentioned. Jirasek (24) also looks to the plexus of Aurbach for the pathology of this condition, and states that specific degenerative lesions occur in the plexus, and result in this syndrome. He fails to state what factors are responsible for this degeneration.

The condition has been found to be associated with Enterocolitis due to various causes, and this is stated as an etiological factor by Emery(10), Ryle(47), Drueek(8), and Graves(17). Drueek(8) thinks that adhesions of the gastro-intestinal tract sometimes cause colospasm by mechanical irritation. He also believes that intestinal diverticulæ may sometimes act as a cause of spastic colitis. McLoone(33) has found Proctitis to be an occasional cause of spastic colon.

Local irritation has been cited by many writers as being a frequent cause of functional spasm of the colon. This local irritation may proceed from many and varied causes. McMillan(34), Emery(10), Hurst(23), Milligan(37), and Hunt(21) believe that many cases of spastic colon are due to dietary errors. Among the various specific errors stated by these writers are Irritant foods, Fermentation due to excess ingestion of carbohydrate foods, Intestinal putrefaction due to constipation, Irregularity in meals, Coarse foods, Rapid eating and imperfect mastication. A number of writers are of the opinion that excess use of cathartic measures, including both drugs, enemata, and colonic irrigation is responsible for a large number of cases. Hurst(23), Ryle(47), Drueek(8), and Jordan(25) concur in this view. Irritation due to the presence of fecal masses is emphasised by Hurst(23), and Ryle(47). Hurst(23), and Graves(17) find occasional cases due to the presence of intestinal parasites such as *Ascaris* or Amebic infestations. Hunter(22) is the only

writer who mentions chemical irritation as a possible cause of colonic spasm. He believes that the too extended use of Sippy powders by peptic ulcer patients may result in spastic colon.

A number of clinicians believe that spastic colon may proceed from reflexes originating in other organs, and due to pathological processes present in those organs. Machechrie believes that these splanchnic reflexes originate in other organs sometimes remote from the colon, and are relayed by way of the coeliac plexus. Hurst(23) and Morgan(38) are of the opinion that such reflexes may originate in the gall bladder in cases of cholecystitis or cholelithiasis, or in the stomach or duodenum in cases of peptic ulcer. Gardner(13), and Milligan(38) think that Visceromotoric organs give rise to reflexes which may result in colospasm, but Eggleston(9) takes an opposite view, and states that the visceromotoric is a result of the colospasm rather than a cause of it. Reflexes from the pelvic organs are mentioned as causes by Hurst(23) who believes that reflex disturbances may arise in cases of salpingitis, and Ryle(47) who believes that similar reflexes may accompany disturbances of menstruation. Ryle(47) also states that reflexes proceeding from jolting, cold and fatigue may reflexly cause spasm of the colon.

A few writers mention psychic conditions as causes of spastic colon. Neuroses, particularly of the anxiety type, are mentioned by Hurst(23), and Eggleston(9), while Fenwick(11) cites two cases of colospasm following shell-shock.

Milligan(37) states that spastic colitis may be due to nervous irritation. Gauss(14) thinks that the habit of ignoring the impulse to defecate is responsible for some cases.

Spastic colitis is thought by some investigators to be due to neurological causes. Morgan(38) states that it may be due to a disturbance of the balance between the Vagus and Sympathetic nerves, and that this disturbance of balance operates at certain special nerve centers or nodal regions located as follows: 1, The upper end of the Oesophagus; 2, The lower end of the Oesophagus; 3, The Pylorus; 4, An area located just below the ampulla of Vater; 5, The Ileo-Cecal junction; 6, The middle third of the transverse colon; 7, The recto-sigmoid junction; 8, The anal sphincter. Emery (10) holds a similar view, and states that colic spasm is frequently due to vagotonia. Jirasek(24) thinks that there is a specific degeneration of the cells of Aurbach's plexus in some cases of colospasm. Steindl(51) believes that he has found degenerative lesions in the medulla and pons near the dorsal nucleus of the Vagus nerve in cases of Spastic Colon. Hurst(23) thinks that spasm of the colon sometimes accompanies Tabes Dorsalis.

Spastic Colitis is thought by some to be due to certain specific and non-specific intoxicating agents. Hurst(23) thinks that it may be due to lead poisoning. Along with Ryle(47) he also states that the nicotine poisoning due to excess smoking

may cause spastic colon. Hurst also believes that Caffeine may cause the condition. Gilliland(16) and Emery(10) suggest that intestinal auto-intoxication may be sometimes a cause.

A few writers suggest an infectious origin for spasms of the colon. McWhorter describes a specific epidemic type, which he believes is of influenzal origin, while Morris(39), and Emery(10) think that focal infection may play a part in the production of spastic colon.

Allergy has been suggested as a possible cause of some otherwise unexplained cases of colospasm. Bisset(5) was able to obtain a personal or family history of allergy in thirteen out of fifteen patients with the disease, and was able to elicit positive food reactions in fifteen of the patients in his series. Gardner(13) has found spastic colon frequently associated with Migraine. McLoone(33) and Kantor(28) have found cases of spastic colon presumably due to wheat, eggs, cheese, Banannas, Rice, Milk, Cocoa, Oatmeal, Potatoes, Chocolate, Strawberry, and Shell Fish susceptibility.

Kantor(28), and Jordan(25) think that avitaminosis is sometimes a cause of spastic colon, and Kantor believes that a deficiency of vitamin B. is a specific factor.

Drueek(8) states that endocrine disturbance or at times faulty calcium metabolism may cause spastic colon.

SYMPTOMATOLOGY:

In the discussion of symptoms, laboratory or radiological data will not be discussed, and the account will be limited to those abnormalities of which the patient makes complaint, or which are elicited by the physician either in the course of history taking or physical examination. Laboratory findings and x-ray findings will be discussed later under the heading of diagnosis.

As the majority of patients come to the physician with some complaint or complaints referable to the gastro-intestinal tract, it is proper that this group of symptoms should be discussed first.

The most common symptom which leads the sufferer from spastic colon to consult a physician is pain or discomfort located somewhere in or near the abdomen. However, its location may vary greatly, which, as we shall see, leads to some interesting problems in the differential diagnosis of this condition. There seems to be no constant location for this pain. Machechrie(32), and Smith, Miller & Fowler(49) report cases in which the pain is in the epigastric region, simulating peptic ulcer. Turner reports umbilical localization of the pain in some cases. Graves and other writers(17) think that the pain is usually localized along the course of the colon, while Bailey(2) finds that it is more frequently confined to the Lumbar or Illiac regions. Milligan(37) and others have reported cases in which the pain has been localized in the right lower quadrant, simulating an attack of acute appendicitis, and Condry(?) has seen cases in which a diagnosis of angina pectoris has been made, due to occurrence of precordial pain.

Various writers comment on the nature of the pain which occurs in colospasm. It is described by Milligan(37) as a feeling of fullness, pressure or weight in the abdomen. Wolfe(59) refers to it merely as a vague feeling of discomfort, while Kantor(28) and Graves(17) employ the term "Belly Consciousness" to describe it. On the other hand, some writers have described instances of very acute pain in connection with attacks of spasticity of the colon, and Morgan(38) states that it is entirely out of proportion to the physical findings.

Most of the writers have reported no definite time of onset in connection with the pain of spastic colitis. But Machecrie states that it sometimes occurs one to two hours following the ingestion of food. Jordan(25), on the other hand observes that it occurs immediately following or during the meal, while Wolfe(59) thinks it has a closer relationship to defecation, occurring during or immediately following that act.

Relationship of the pain to other factors has been observed by a few clinicians, while others fail to make observation of such relationships. Turner(53) observes that the pain tends to disappear upon rest. The same writer notes that it is relieved also by defecation, and in this view he is concurred in by Graves(17). Graves also notes that the pain is relieved by alkalies, but states that such relief occurs only after belching, thus attempting to differentiate this from the relief obtained in peptic ulcer which occurs whether the patient belches or not.

Bailey reports cases in which the pain appeared to be precipitated or aggravated by apples, pears, cabbage or leafy vegetables, while Kantor(28) reports similar aggravation due to jars or pressure on the abdomen.

Saper(48), Turner(53), Jordan(25), and many other writers note that in most if not all cases of spastic colon, the colon is palpable in all or a portion of its course. It is usually felt as a cord-like or rope-like mass along the course of the descending colon, sometimes the ascending colon, and less frequently the transverse portion. On such palpation, tenderness is noted along the entire course of the colon Saper(48); Jordan(25), or in localized portions of it, Turner(53). This may or may not be accompanied by abdominal rigidity, Jordan(25, Graves(17)).

It seems thoroughly consistent with the pathology to expect constipation to be one of the outstanding features of this disease. Machechrie(32), Turner(43), Milligan(37), and Graves(17) report constipation as one of the principal features accompanying the condition. Hunter(22) and Turner(53) note that a condition of spastic colon is not inconsistent with perfectly normal bowel movements, while Graves(17) observes that diarrhea alternating with constipation may be a feature of the disease.

The occurrence of abnormal stools seems frequently to be a feature of spastic colon. Rowe(45) reports that the stools of spastic colitis are apt to be ribbon-like due to the narrowing of the lumen of the colon by spasm. Saper(48) notes that they are

frequently fragmented due to the same mechanism. Graves(17) on the other hand reports the occurrence of mushy stools. Most writers fail to note the occurrence of unusual amounts of mucous in the stools of spastic colon sufferers, but Drueek(8) notes that in some cases there is an excess of mucous, and that the stools are apt to be frequent and incomplete.

Nausea is cited by most writers on the symptomatology of spastic colon. Jordan(25) and Turner(53) state that the nausea is usually unaccompanied by vomiting, but Gardner(13), Graves(17), Smith, Miller & Fowler(49), and Gilliland & Sigallof(16) find the nausea of colospasm frequently followed by vomiting.

These are the principal symptoms referable to the gastro-intestinal tract in cases of spastic colitis. There are, however a large number of miscellaneous symptoms reported by various writers on the disease. Kantor(28), Graves(17), Machechrie(32), and Drueek(8) report varying degrees of meteorism occurring in this condition. Machechrie(32), Jordan(25), Graves(17), and Bailey(2) observe that patients with spastic Colitis frequently suffer from loss of appetite. Machechrie(32) and Graves(17) note that the tongue is frequently coated. Machechrie(32), Milligan(37) and Gilliland & Sigallof(16) that belching occurs frequently as a manifestation of the disease. Flatulence is reported as a feature by Milligan(37), Russ(46), and Smith, Miller & Fowler(49), while the occurrence of unpleasant or metallic taste in the mouth is noted by Graves(17), and Bailey(2). Russ(46) states that

fetid breath is often present, while hyperacidity and pylorospasm is reported by Barker(3).

In connection with spastic colitis, there are a surprisingly large number of symptoms referable to the central and peripheral nervous mechanisms. This is perhaps not so surprising when considered in the light of the nervous theory of the etiology of this condition. Some of the writers on the subject feel that these symptoms are due to the presence of toxins of gastro-intestinal origin as a result of stasis, while others feel that they are of reflex origin. In the absence of definite proof, it may be permissible to surmise that there is always the possibility of both factors operating in equal or varying degrees to secure these results.

Headache is perhaps the most prominent symptom in this classification. Wolfe(59), Machechrie(32), Milligan(37), and Bailey(2) along with others, report its occurrence. Its most prominent feature is its prompt disappearance after defecation which points to a reflex rather than a toxic origin. Graves(17), Russ(46), Drucek(8), and Bailey(2) find that general depression is a frequent accompaniment. Like the headache, it frequently disappears promptly after defecation.

Paraesthesias like numbness of the fingers, or tingling of the toes have been reported. Ryle(47), and Barker(3) report cases in which this has been a prominent feature.

Insomnia has occasionally been reported as an unpleasant symptom of spastic colon. Russ(46), and Drucek(8) report its occurrence in cases seen by them.

Russ(46) reports anxiety, Vertigo, Neuritis, Neuralgia, and loss of memory as occurring in patients found by him to be suffering with spastic colitis. Vertigo is also reported by Barker (3). Dreuuk reports loss of libido, and Barker transient diplopias in sufferers with colospasm.

A few symptoms referable to the circulatory system occur in patients with spastic colon. In this case also the question arises as to whether these are toxic or reflex in origin, or indeed whether they are a result of the same etiological factors that are responsible for the primary condition.

Drueek(8) reports tachycardia as accompanying spastic colon, but fails satisfactorily to rule out other etiological agents which might have been responsible for tachycardia. On the other hand, Barker(3) reports cases in which Bradycardia was the cardiac manifestation. He also notes arrhythmias and extra-systoles occurring in his cases.

Russ(46) is the only writer to report peripheral circulatory manifestations, and notes hypotension, and cold extremities.

The remaining symptoms may possibly be due to metabolic changes. Drueek reports lowered basal metabolism in some of his cases, though this is by no means constant. Graves(17) and Jordan (25) note weakness as a symptom, and Jordan(17) finds occasional loss of weight. However, he fails to eliminate other causes of lost weight in considering this symptom. Russ(46) believes that

some of his patients present brittle hair and nails, and this well fits in with the lowered basal metabolic rates reported by Drueek.

DIAGNOSIS:

As in many other diseases, considerable information may be derived from a careful study and analysis of the symptoms as they present themselves, particularly their relationship to various influences.

The pain of spastic colitis presents certain characteristics which help to differentiate it from the pain of other important gastro-intestinal conditions. Jordan(25) calls attention to the fact that its onset may be associated with an attack of coryza, irritating foods, and frequently with the abuse of cathartics. Less frequently it begins after some strenuous physical effort. He also points out that it is relieved by hot water taken by mouth, defecation, belching or flatus. Turner(53) finds the relief of the pain of spastic colon by belladonna or hyoscyamus useful in distinguishing it from the pain caused by organic disease, but Beams(4) finds that the nitrites are more useful in making this differentiation. He states that the members of the atropine family sometimes relieve the pain due to organic pathology, a fault that is not shared by the nitrite group.

Emery(10) and Graves(17) call attention to the observation that the pain of colospasm is relieved by the alkalies only after belching has occurred, and point out that in peptic ulcer, the pain is relieved by alkalies irrespective of whether belching has taken place or not. Jordan(25) and Emery(10) note that the pain of colonic spasm is relieved by heat, a phenomenon that is not present in many diseases presenting similar symptoms, while Smith, Miller & Fowler(49) report a corollary to this, namely that the pain is aggravated by cold drinks.

The location and nature of the pain presents a further opportunity for comparison with other conditions. Jordan(25) comments on the shifting character of the pain and its lack of localization, while Gauss(14) notes that it covers a larger skin area than in most other abdominal diseases.

In relation to the nausea sometimes present in this condition, Jordan(25) states that it is not accompanied by vomiting, but as already noted in the section on symptomatology, other writers do not hold the same view, believing that vomiting is a rather frequent accompaniment of the nausea of spastic colitis.

Some clinicians have called attention to abnormalities of the stools in spastic colitis. Emery(10) calls attention to the characteristic hard stools evacuated by the patient, but as already reported under symptomatology, other writers state that the stools are frequently soft. Saper(48) points out the frequency of fragmented stools in this condition, while Jordan(25) notes that undigested food is often noticed in the stool.

Jordan(25) uses a test enema to differentiate spastic colitis from other conditions. He uses an enema of six pints of tap water, and notes that by this method pain is elicited which is identical in character to that which the patient ordinarily experiences in typical attacks of spasm.

Physical examination may reveal peculiarities which may be useful in arriving at a diagnosis. As reported by McWhorter(35) and others (See symptomatology), the colon itself may be palpated as a hard, ropelike mass along its course. This palpation is usually accompanied by tenderness, Saper(48). Emery(10) calls attention to a splashing noise which may be elicited by massage over the colon in cases of spastic colitis. Saper believes that proct-

oscopic examination is useful in making a diagnosis, but Bailey(1) warns that proctoscopic examination may be entirely negative in marked cases of colospasm due to the fact that the examination may take place during a period of quiescence.

Bailey(2) and Graves(17) state that the occurrence of vague intestinal and gastric symptoms in the asthenic type of individual should cause the physician to consider Spastic Colon in his differential diagnosis.

The radiologist may be of some assistance in establishing a diagnosis of spastic colon. The barium enema is particularly useful. Wolfe(59) notes a spastic type of peristalsis observed under the fluoroscope, while Jordan(25) notes that the barium enema fills the colon with greater smoothness and speed in the presence of spasm. He also calls attention to the onset of the characteristic pain of spastic colitis when the barium enema is given. McMillan(34) observes that the patient is, however, free from pain during the time that the barium is in the bowel. Barker(3) suggests that the barium

enema can be made to yield more definite information by making comparative studies before and after a dose of atropine sulphate, but Beams(4) finds that nitrites are more valuable for this purpose, as they relieve the spasm when functional, and fail to do so when organic in nature.

The flat plate yields less information than the barium enema in these cases. However McMillan(34) frequently finds the greater part of the barium in a dilated cecum and ascending colon with the remaining bowel exhibiting varying degrees of contraction. He also reports that the haustral markings are found to be obliterated in cases of spastic colon.

A note of warning in regard to the use of x-ray in the diagnosis of spastic colon is sounded by Ryle(47) and Saper(48) who find the x-ray un dependable due to the fact that the barium may overcome the spasm and lead to negative results. Ryle(47) also points out that the time of x-ray examination may fail to coincide with a period of spasm and in this way a false negative result may be obtained. Gilbert(15) believes that examination by barium enema is unphysiological and thinks that

the study should be made after normal feeding. Drueek (8) believes that an incompetent illeo-cecal valve may often be demonstrated in spastic colon.

The laboratory contributes little to the diagnosis of spastic colon except in a negative way, such as demonstrating the absence of blood in the stool, Bailey(2), which may assist in ruling out peptic ulcer, neoplasm etc; the absence of abnormal urinary findings, which may assist in ruling out ureteral colic, Wolfe(59); the absence of leukocytosis, which may assist in ruling out appendicitis, McWhorter(35); and the Wasserman reaction which may be used to rule out the gastric crises of tabes dorsalis.

The positive findings are limited to the discovery of undigested food in the stools, Jordan (25); a preponderance of gram positive intestinal flora, Morris(39), and the presence of hyperacid stomach contents. All of these are more co-operative than specific, and their absence does not invalidate the diagnosis any more than their presence makes it.

DIFFERENTIAL DIAGNOSIS:

The somewhat protean manifestations of spastic colitis render it easy to confuse with other diseases, particularly those involving the gastro-intestinal tract. However, as will presently be made plain, many conditions involving areas more or less remote from the intestinal tract present features that make it possible to confuse them with spastic colon. It is this ability of spastic colitis to simulate other diseases that has led to so many errors in diagnosis involving expense, discomfort, unnecessary, or even harmful medication, and all too frequently, futile surgery. It is therefore of the utmost importance for the general physician and the specialist to be familiar with the differential diagnosis of spastic colitis.

Acute appendicitis stands foremost in the list of diseases that are commonly confused with spastic colitis. McLoone(33) reports a series of two-hundred cases of spastic colon in which twenty-six percent had been previously operated on for appendicitis, acute or chronic, without relief of their symptoms. Kantor(28) reports a larger series of six-hundred and fifty-four cases in which twent-

-y-two percent had undergone operations for appendicitis without relief. Rowe(45), Jordan(25), McWhorter(35), McLoone(33), and Kantor(28), along with many others, report cases in which the clinical picture of spastic colitis has been mistaken for that of acute or chronic appendicitis. Chronic appendicitis seems to be a particular offender as there seems to be a distinct tendency for the average physician to label any syndrome presenting vague gastro-intestinal symptoms as being due to chronic appendicitis. The actual differentiation is not always easy. As suggested by Jordan(25), the onset of spastic colitis is frequently associated with colds, irritating foods, and abuse of cathartics, factors which are rarely the concomitants of appendicitis. Then again, McWhorter(35) points out that the pain of spastic colitis is apt to lack specific localization with a tendency to be generalized, while appendicitis typically commences with general pain, followed by localization at McBurnie's point. A white blood count is of the utmost value in this differentiation, suggests the same writer, but Bailey points out that an increase in temperature and leukocytosis may occas-

ionally be present in spastic colitis. Also, it is well known that occasionally, the leukocyte count in appendicitis is not increased. It must also be borne in mind that appendicitis and spastic colitis may be present concurrently, the colospasm being secondary to the irritation arising from the inflamed appendix. Here is a condition in which the clinical experience of the physician must be exercised to its utmost that no case of acute appendicitis fail to receive the benefit of surgery, and no case of uncomplicated spastic colon undergo unnecessary surgery.

Cholecystitis and cholelithiasis are conditions which, particularly in their milder forms are often mistaken for spastic colitis, though the error is usually in the other direction. Rowe(45), and Jordan(25) with others, have reported cases of spastic colitis which have undergone cholecystectomy without relief, only to be demonstrated later as cases of spastic colitis. Again, Jordans(25) observation of the onset of typical cases of spastic colitis with colds, irritating foods, and the abuse of cathartics is useful. Milligan(37) observes that gall bladder disease

is apt to be characterized by distinct attacks with free periods between. While this is apt to be true of the more acute varieties, too much confidence must not be put in this feature, as some of the more chronic cases lack this clean-cut history of definite attacks. If the patient is of the female sex, near the age of forty, and a multipara, a diagnosis of gall bladder disease is favored, but obviously an individual of this type may also be a victim of spastic colitis. The Graham-Cole test is very useful in making the differential diagnosis.

Although not the usual finding, an occasional case of spastic colitis may show an apparent relationship of symptoms to the taking of food which may lead to a mistaken diagnosis of peptic ulcer. Rowe(45), and Morgan(38) have reported cases in which this error was made, and urge the consideration of a diagnosis of spastic colitis in doubtful cases. Carstens(6) has even reported cases of spastic colon in which the pain and rigidity was sufficiently marked to lead to a diagnosis of perforated peptic ulcer. Of course the x-ray is of the utmost value in making a diagnosis, as is the finding of occult or gross blood in the stool,

providing that the patient has been put on a meat-free diet for three days preceeding the stool examination. Gauss(14) notes that the pain present in spastic colitis is apt to cover a much larger area than that exhibited in peptic ulcer cases. Emery(10), and Graves(17) mention an interesting differential point. They state that the pain of spastic colitis is relieved by soda only if and after belching has occurred, while that of peptic ulcer is relieved whether belching occurs or not, and occurs before the belching takes place. Nevertheless, the writer saw a case of peptic ulcer, proved by x-ray and stool examination, who was very positive that he received no relief from soda unless belching took place.

The attacks of pain in spastic colitis may be of such location and severity as to simulate an attack of renal colic. Rowe(35), Wolfe(59), and Carstens(6) report cases in which this error in diagnosis was made. However, the pain of spastic colitis is rarely as well localized as that of renal colic, nor does it often exhibit the typical radiation down the thighs or into the

scrotum. Also, urinary findings may be present in renal colic and make the diagnosis. Pyelography, and cystoscopy may be useful diagnostic aids.

Sometimes, the pain of spastic colitis has a rather low localization, suggestive of pelvic disease. Rowe(45), and Ryle(47) report cases in which this error was made. Pelvic examination should reveal the presence of pathology in these organs, but it must be borne in mind that the presence of pelvic pathology does not rule out the presence of colospasm as a secondary or entirely independent condition.

Bailey(2) calls attention to the confusion between spastic colitis and dyspepsia, gastric neuroses, hyperacidity, and hypoacidity. This is especially confusing as spastic colitis may be secondary to and concomitant with a chronic gastritis due to the long continued ingestion of irritating foods, and either hypo- or hyperacidity may be present in spastic colitis. As for gastric neurosis, it is all too easy for the physician to brand a group of vague gastro-intestinal symptoms as a neurosis, when a little investigation would unearth the true pathology. The answer is to keep the possibility of spastic colitis in mind

and seek the aid of physical diagnostic procedures, the proctoscope, and the x-ray in unexplained cases of digestive disturbances.

Sometimes, in cases of spastic colitis for which no apparent cause can be determined, a careful microscopic examination of the patient's stool will reveal the presence of intestinal parasites. Bailey(2) calls attention to this possibility. These parasites may be Amebae, Ascaris, Pin worms, Seat Worms, the ordinary Tape worm, or other less common intestinal invaders. Obviously no therapeutic measures will be effective which do not remove the worms.

Chronic ulcerative colitis is sometimes accompanied by secondary spasm of the colon(2). The presence of gross blood in the stools, and the findings on proctoscopic examination make the diagnosis. Rectal and proctoscopic examination should never be omitted in cases of suspected colospasm.

Ryle(47) calls attention to the presence of symptoms resembling neoplasm in cases of spastic colon, and in these cases, it is very important that a differential diagnosis be made. The spastic colon may resemble a tumor mass in the abdomen, and if the patient happens to be in

the cancer age, serious mistakes may be made in the way of prognosis and treatment. The tumor formed by a spastic colon is apt to vary in size. It is apt to be present at one examination and absent at another. The typical location along the course of the colon is helpful. In making the differentiation, proctoscopic examination and the x-ray are of the utmost value. Of course the two conditions may coexist.

Ryle(47) also reports cases of spastic colitis of such a fulminating character, and accompanied by such marked degrees of vomiting and pain that a diagnosis of intestinal obstruction was made. Fortunately this is rare, but it is almost inevitable that many such patients will be brought to operation. Again, the x-ray may be of assistance.

Barker(3), Condry(7), and Hunt(21) report cases in which the pain of spastic colitis was referred to the precordium, and the attacks resembled angina pectoris. Hunt(21) states that pain in the precordial region which is relieved by the passing of flatus, change of position or massage is apt to be due to a spastic colon. Of course, the electrocardiograph is of great assistance in making this differentiation. Also, anginal attacks are more frequent in males whereas spastic colitis is predominantly a disease of

the female.

Attacks of spastic colitis resembling pancreatic or hepatic disease have been recorded by Jordan and Kiefer(27).

Parsons(41) states that spastic colitis may be confused with intercostal neuralgia. He suggests that differentiation may be made by exerting pressure on the abdominal wall, first with relaxed, then with tense muscles. If the pain is intra-abdominal in origin, it will be elicited only with relaxed abdominal wall, while if it is of local origin, it will be elicited both with relaxed and tense walls in equal degree.

Hunt(21) reminds us that the pain in pleurisy, with or without pneumonia may be referred to the abdomen, and should be considered in the differential diagnosis of all abdominal disease.

TREATMENT:

There are two salient principles in the management of a case of spastic colitis. They are the removal of the cause and the control of the spasm. While measures to control spasm may appear to be purely symptomatic treatment, it is reasonable to suppose that in many cases a so-called "vicious circle" may be so broken up, for it is conceivable that, some outside factor long since inoperative having established a state of spasm, the spasm perpetuates the irritation, and the irritation the spasm. Thus, by interrupting the spastic condition for a time, the bowel may be allowed to return to a normal condition with complete and permanent relief of symptoms. This perhaps may explain the cases where a condition of enterospasm is present, but where no assignable etiological factor can be demonstrated. However, physicians must not fall into the error of prescribing symptomatic measures without first making every effort to discover the cause, as all symptomatic measures are sure to result in failure in those cases in which the colospasm is due to perfectly definite causes. For example, no amount of symptomatic treatment would produce permanent cure in an intestine

infested with ascarides.

We shall first consider the forms of treatment directed toward the removal of the cause, as this is the most rational attack, and offers the most hope for rapid and permanent cure.

To the extent that dietary errors may be the cause of spasm of the colon, or may contribute to the continuation of the spasm once it has been established, they should be corrected. Various writers have made note of specific articles of diet which they have believed to be responsible for irritation, and recommended their elimination from the diet of the sufferer from spastic colitis. These articles include Fried or greasy foods(17), Raw fruits(17), Coarse vegetables(17), (38), Condiments(17-8), Bran(38), Alcohol(38-8), and cold foods or drinks(28). Morgan(38) particularly deplors the widespread misuse of bran by the laity and by physicians, and thinks that it has no place in the diet of normal human beings. Emery(10) believes that irregular meals may be a cause of spastic colitis, and recommends that meals be taken at regular specified hours.

Since the abuse of cathartics appears to be a frequent cause of spastic colitis, their inter-

diction in an important measure in its management. In this view Gardner(13),Morgan(38),Hurst(23), Ryle(47),Drueak(8),andJordan(25) concur.Certainly the public is overphysiced, the abuse of advertising being largely responsible.

The eradication of infectious and other pathology is an important measure in the management of spastic colitis.It is well known that reflexes from organs both within and without the intestinal tract may cause spastic colitis, and diligent search for such conditions as appendicitis, cholecystitis, peptic ulcer, hemorrhoids, and rectal fissures should be made with the object of their elimination.Machecrie(32) reports two cases of spastic colitis which cleared up rapidly after appendectomy, and other clinicians have reported similar cures after eradication of analagous pathology.Morgan(38) believes that the removal of focii of infection such as diseased teeth and tonsils may assist in clearing up cases of colospasm. Where local intestinal infection exists, intestinal antiseptics may be tried.Spriggs(50) recommends Iothyol gr.2,Morgan(38) suggests argyrol or acetylsalicylic acid enemata, while Wilson(57) uses

Salol gr.X T.I.D.

Since it is thought that excess irritability of the central and peripheral nervous systems may play a part in the production of enterospasm, all stimulants of nervous tissue should be avoided. On this basis Morgan(38), Ryle(47), and Spriggs(50) recommend the interdiction of tobacco in all forms in colospasm. Drueek(8) believes that tea, coffee, and cocoa should be eliminated from the diet.

In some cases putrefaction, and in others fermentation may be responsible for irritative conditions resulting in spastic colon. It is therefore rational therapy to attempt correction of these conditions when and if present. Where the condition is characterized by a putrefactive intestinal flora, correction by a high carbohydrate diet(8), administration of lactose (1), (21), or administration of acidophilus milk two glasses T.I.D.(16) are indicated. Where the intestinal flora is excessively fermentative, the diet should be low in carbohydrates.(38), (3), (16), (39). Differentiation between these two states may be made by a gram stain of a stool specimen.

Since it has been suggested by Barker(3), and Gilliland & Sigaloff(16) that abnormal conditions of gastric acidity may exert an indirect or direct effect on the colon, it is logical therapy to attempt correction of gastric acidity in cases where this abnormality is demonstrated by gastric analysis. The therapy would be directed toward the addition of dilute hydrochloric acid to the diet in cases of hypoacidity, and of alkali therapy or secreto-inhibitory drugs in the cases of hyperacidity.

To the extent that allergy may be demonstrated to be a part of the mechanism of production of colonic spasm, elimination diets are in order, both for the purpose of discovering the substance or substances to which the patient is sensitive, and for the purposes of either desensitizing him or permanently eliminating them from his diet(5).

A number of writers have suggested a toxic etiology of colospasm, in some cases. Whether these toxins be due to faulty elimination, infectious processes, faulty metabolism, or from exogenous sources, a thorough search should be made for their

presence, and steps taken to eliminate them. In this connection, special note should be taken of the vocations and avocations of the patient with a view of determining the probability of his exposure to exogenous toxins such as lead, arsenic etc.

As has been previously stated, deficiencies in calcium, vitamins or both have been suggested as causes of spastic colitis. Therefore, Steindl (51) suggests ten grains each of calcium carbonate and calcium phosphate three times daily as therapy in those cases of enterospasm in which calcium deficiency is suspected. Gardner (13), and Gilliland & Sigaloff (16) believe that the calcium deficiency may be associated and due to a lack of the parathyroid hormone, and suggest that calcium lactate be given in two gram doses three times daily with fifteen units of Parathormone given intramuscularly every three or four days.

In some cases, the irritability of the colon is thought to be brought about and perpetuated by contact with hard or impacted fecal matter. In these cases, regulation of the bowels, chiefly for the purpose of keeping the stools soft and

non-irritating has been suggested by a number of clinicians. Enemata of warm olive oil, two to three ounces, are suggested by Rowe(45), Gardner(13), Morgan(38), and Drueek(8). Jordan & Kiefer(27) however, are of the opinion that such enemata are irritating to the colon, and are strictly contraindicated in this condition. Among other methods of keeping the stool soft are Warm liquid parafin enemata, ounces four at night (47), Mineral oil by mouth (17), (3), (16), Agar(3), (16), Psylla(7). Hurst(23), and Gauss(14) believe that normal stools and bowel movements may best be established by the formation of regular habits of going to stool.

As mentioned above, control of the spasm of spastic colitis is important in the symptomatic and etiological management of enterospasm. Four measures are of importance in this connection. They are: 1. Antispasmodic drugs; 2. Hydrotherapy; 3. Diathermy; 4. Rest.

The last three mentioned need no explanation or elaboration. Hydrotherapy has been suggested by Gilliland & Sigaloff(16) and Drueek(8). These are general measures. In addition there are a few local methods of controlling spasm. One of these is the

local application of magnesium sulphate in saturated solution. This may be done by means of an applicator through a proctoscope on alternate days(48), or by means of ~~of~~ enemata of approximately three ounces. It is claimed that the first method brings about permanent relief in eight to ten days in an appreciable number of cases. Nevertheless this method is criticised by Jordan & Kiefer(27), who say that magnesium sulphate is irritating to the lower bowel, and by Parsons(41) who believes that enemata or irrigations of any sort are contraindicated in colospasm. Massage over the course of the colon is suggested by Eggleston(9) and Hunt(21). Gilliland and Sigaloff(16) believe that demulcent substances such as Bismuth or Kaolin may be useful in reducing spasm. Rowe(45), Eggleston(9), Drueck(8), Barker(3), Emery(10), Gardner(13), and Graves(17) believe that heat applied over the abdomen assists in controlling spasm.

The antispasmodic drugs used in an attempt to control spasm in spastic colitis are many. Of course the ones most frequently mentioned are atropine and belladonna. They are used by Gauss(14), Wolfe(59), Turner(53), Gardner(13), Graves(17),

Morgan(38), Drucek(8), and Lurje(31). However, the last mentioned writer, finds atropine only effective when administered by the intravenous route, and believes that both atropine and belladonna are without sufficient effect when administered orally. Other anti-spasmodics suggested are: Barbiturates (13), (9); Tincture of Valerian (38); Bromides (13), (47); Eumydrine gr. 1/60 (Atropine Methyl Nitrite) (8); Benzyl Benzoate (M. 15-30 t.i.d.) (8); Novatropine (Hoatropine Methyl Bromide), Hyoscyamus(53), (50) ; Magnesium Perhydrol (3).(12) ; and Sodium Nitrite (Grains 1. t.i.d.). Beams, who suggests the last named drug, finds it much superior to atropine or its derivatives, and believes it extremely valuable both for diagnostic and therapeutic purposes.

CASE REPORTS:

CASE NUMBER 1.

University of Nebraska College of Medicine
Dispensary case number 51896. Ruth C. . . . White,
female, age twenty-five, married. Entered the dis-
pensary on June twenty-fourth nineteen hundred
and thirty-one, complaining of severe headache
and dizziness which of late has been accompanied
by nausea and vomiting. She also complained of in-
termittant headache of two or three years durat-
ion. An appendectomy had been performed in August,
nineteen hundred and thirty without relief of sym-
ptoms. She still experiences severe pain in the lo-
cation of the scar occasionally. She has had hemm-
orrhoids since childhood, accompanied by bleeding.

Her past illnesses, in addition to the above,
include chicken pox, small pox, scarlet fever, meas-
les, chorea, and congestive heart failure with edema.

Previous operations, in addition to the append-
ectomy noted above, include removal of tuberculous
cervical lymph glands, and removal of tonsils and
adenoids.

PHYSICAL EXAMINATION.

THROAT: Pharyngitis with post-nasal discharge.

HEART: Systolic murmur at apex, increased by exer-
cise.

ABDOMEN: Tenderness in region of umbilicus. No oth-
er tenderness nor masses.

WEIGHT: 113 PULSE: 80 BLOOD PRESSURE: 110/70/

LABORATORY FINDINGS:

URINALYSIS: Negative.

X-RAY: Studies of colon show poor definition of haustrations and occasional spasm in descending portion. Rectal ampula small. Patient failed to retain enema fully.

ADDITIONAL HISTORY:

Later questioning elicited the following: The patient vomits whenever bananas or onions are ingested. She has acquired the habit of eating very rapidly. She belches considerably after meals, and this is usually followed by relief. She complains of continuous pain, both during the day and night. This pain is referred to the epigastrium and right lower quadrant. There is some relief after vomiting. No flatus. Stools are hard, dry, and black. Patient does not use laxatives. Also complains of muscae volitantes at times.

ADDITIONAL PHYSICAL EXAMINATION:

Tenderness elicited over the entire course of the colon. Proctoscopic examination shows the mucous membrane of the rectum and sigmoid portion of the colon to be injected and vascular.

DIAGNOSIS:

Proctosigmoiditis. Spastic Colitis.

TREATMENT:

1. Enemata of acriflavin 1:10,000 on alternate days.
2. Luminal Gr. ss.
Ext. Belladonna Gr. 1/6 B.I.D.

RESULT:

Patient states that she has been relieved of all symptoms.

CASE NUMBER 2.

University of Nebraska Dispensary patient number 53432. Josie G..... A married, white female, aged 44. Entered the dispensary clinic on November 2, 1931, complaining of flatulence, belching after meals, a feeling of oppression in the epigastrium, spells of vertigo, nervousness, weakness, and a feeling of cold over the entire body. These symptoms had been present since July, 1931. In addition, she has had occasional attacks of pain referred to either the right or left scapula, dyspnea on exertion and constipation.

PAST ILLNESSES:

Small-pox, Measles, Influenza, Tonsillitis.

OPERATIONS:

Tonsillectomy.

ADDITIONAL HISTORY:

Appetite is usually good. The symptoms described above ordinarily appear immediately following a meal. Sometimes the pain is in the left hypochondrium. Patient frequently belches one to two hours after eating. The belching is accompanied by a bitter taste like bile. These relations hold for all of the symptoms except the pain, which appears at no definite time. It is not ac-

accompanied by nausea or vomiting. Sometimes it seems to be precipitated by nervousness. It is relieved by defecation. The patient is in the habit of taking cascara about every other day. Her stools are small and soft.

LABORATORY FINDINGS:

URINE: Negative.

GASTRIC ANALYSIS:

FREE	TOTAL.	
15	30	
0	12	
12	28	
25	42	
32	43	
32	44	
30	42	
22	37	
20	31	
17	30	
12	30	Mucous 4 Plus.

ROENTGENOGRAM OF GALL BLADDER: Negative.

BLOOD COUNT: Hb.: 93% R.B.C. 5,270,000

W.B.C. 9,000 Polys 62% Lymph. 27% Monos. 9%.

Eosinophiles 2%.

PHYSICAL EXAMINATION:

This was negative except that on abdominal

examination a tender, definitely spastic descending colon was palpated.

TREATMENT:

1. Dilute Hydrochloric Acid. m.XV.
Elix. Peptenzyme. Dr.1. T.I.D.
2. Fat free diet.
3. Fifteen hundred cubic centimeters of water between meals.
4. Mineral oil.

RESULT:

Relief of symptoms and reduced spasticity observed on physical examination.

CASE NUMBER 3.

University of Nebraska Dispensary patient number 54254. Anthony G....., a white, single male aged 25 entered the dispensary January twenty-third, nineteen thirty-two complaining of epigastric distress occurring two to three hours after meals. This distress is relieved by food, and is not accompanied by vomiting. It has been becoming increasingly worse during the last ten months. There is also weakness and fatigue, headaches, constipation, and a history of tarry stools. The symptoms are aggravated by cabbage or greasy foods.

PAST ILLNESSES

Pneumonia, Undiagnosed attack of jaundice.

OPERATIONS.

Tonsillectomy.

PHYSICAL EXAMINATION.

NOSE: Anterior discharge. Left septal deviation.

TEETH: Poor teeth.

PHARYNX: Atrophic pharyngitis with posterior drainage.

TONGUE: Injected and enlarged.

ABDOMEN: Asthenic habitus. Spasticity of descending colon.

SKIN: Acne Vulgaris.

LABORATORY FINDINGS.

URINE: Negative.

GASTRIC ANALYSIS:

	FREE	TOTAL.
	5.5	17
	8.0	27.5
	28	54
	35	62
	38	59
No occult blood.	40	57

BLOOD COUNT:

R.B.C. 4,550,000 W.B.C. 5,000 Hb.90%.

Polys. 66% Lymphos. 32% Monos. 2%

ROENTGEN: Reports dysfunction of gall-bladder

REMARKS:

This case is incomplete, inasmuch as no therapy nor results of therapy is given. Apparently, the patient failed to return to the dispensary. Of course, the diagnosis is not entirely clear, the question being to what extent the patient's symptoms are due to the spastic colon, and to what extent, if any, to the dysfunctioning gall bladder, and associated pathology. Also, there is the possibility that the spastic colon may be secondary to the gall-bladder condition.

CASE NUMBER 4.

Joseph C. . . , dispensary patient number 39038 entered the University of Nebraska dispensary on February the sixteenth, nineteen-thirty two. He was a white male, age forty-seven.

His complaint was of a nagging feeling of discomfort in the epigastrium, which usually woke him up about eleven p.m., impelling him to take food for its relief. This was of two years duration, during the last year of which, he had experienced considerable belching following his meals. No history of any particular food aversions, nor of any relation of particular kinds of food to his symptoms was obtained. There was no nausea not vomiting at any time. There was no report of relief of symptoms by alkalies. The patient has been in the habit of taking laxatives to relieve a moderate degree of constipation. The stools have been small and soft, and have never been remarkably dark nor tarry.

Physical examination was entirely negative, and the usual laboratory procedures revealed no evidence of disease.

Roentgenological examination of the lower intestinal tract by means of a barium enema disclosed redundancy of the sigmoid colon. The descending colon and splenic flexure showed a loss of haustrations. A diagnosis of spastic colitis was made.

REMARKS:

This case was rather incompletely worked out, but serves as an illustration of simulation of duodenal ulcer by a spastic condition of the colon.

CASE NUMBER 5.

Carl N., White male, married, age 22. Dispensary patient number 51940, entered the University of Nebraska dispensary clinic on June twenty-seventh, nineteen-thirty one, complaining of abdominal soreness of two years duration. This was worse in the morning and after exercise. It was relieved by eating, only to return in about two hours. It was present most of the time except as noted above.

His past history was inconsequential except for the repair of a right inguinal hernia a few years before.

Physical examination was negative except for an area of tenderness about three-quarters of an inch above the umbilicus in the midline, and a palpable, spastic descending colon.

A complete blood count was taken, and disclosed a hemoglobin of 95%, 8,000 whites, 5,270,000 reds. The differential showed polymorphonuclears 70%, Lymphocytes 25%, Monocytes 5%.

A blood wasserman was taken, and reported as negative.

Gastric analysis was done by the fractional method, and revealed an essentially average curve for both free and total acidity. No occult blood was found.

Stool examination failed to reveal anything significant.

Roentgenological examination by means of a barium enema showed a smoothing out of the descending colon with considerable spasm of the ascending colon.

A diagnosis of spastic colitis was made.

REMARKS:

This case is somewhat better worked up than the preceding one. It illustrates a rather peculiar symptomatology, that of abdominal soreness. As may be seen, it might easily have been confused either with an abdominal myocitis or even with peptic ulcer due to the typical relation to meals. It is rather unfortunate that, both in this case, and the preceding one, no follow up or record of treatment, or response to same was kept.

CASE NUMBER 6.

Christie M.....White, female, single, age 52
dispensary number 37494. This patient entered the
University of Nebraska dispensary clinic on Feb-
ruary thirteenth, nineteen-thirty two.

She stated that since the preceeding June,
she had been troubled with constipation, and
stated that the stools were small, grey and hard.
There had been an unexplained loss of twelve
punds in weight since April. She also complained
of weakness, pain in the left lower quadrant, noc-
turia three to four times nightly, dizziness, muscae
volitantes, and a swollen face in the morning. The
abdominal pain was aggravated by the ingestion
of pickles or saurkraut. Also stated that sour
foods caused headaches. There was occasional nau-
sea and vomiting, but no belching. There was cons-
iderable flatus.

Physical examination was negative except for
tenderness in the gall bladder region, infected
tonsills and an atonic rectal sphincter.

Laboratory findings included two to three
leukocytes per high power field in the urine,

but the record fails to state as to whether the specimen was catheterized or not. No casts nor red blood cells are reported. No kidney functional test, nor blood pressure is recorded.

The blood count showed a moderate relative lymphocytosis, and moderate eosinophilia(11%)

X-ray studies showed the findings consistent with an irritable colon.

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