

fracture of the adjacent bone. Infectious arthritis and synovitis may simulate sprained wrist, but the history and progress of the disease soon makes the diagnosis clear.

The treatment of sprained wrist depends somewhat upon the location of the injured ligament. When the dorsal or lateral ligaments are injured, a satisfactory dressing consists of strips of adhesive plaster crossing at the back of the hand and extending from the metacarpophalangeal joint to about the mid-forearm, being so applied as to limit flexion at the wrist. When the anterior ligaments are injured, which occurs only very rarely, rest and massage is the treatment of choice.

When the condition is such that the injury cannot be differentiated from fracture, it is better to consider the lesion as a fracture and to treat it accordingly. A plan which has been found satisfactory in the treatment of fractures of this type (fractures near joints without gross deformity) is fixation combined with early massage and passive motion. As such treatment is equally beneficial in sprained wrist it merits description in some detail. The wrist is fixed by means of a posterior splint extending from the knuckles to the upper third of the forearm and the patient told to return daily for massage and passive motion. The massage is begun on the second day with very gentle manipulations, the arm being stroked gently and superficially from the fingers toward the elbow, according to the method of Lucas-Championnière. Care should be taken to cause little or no pain, and to massage constantly in the same direction. As the pain diminishes, the fingers, and later the wrist joint, should be moved, at first passively and later actively, the amount of movement depending upon the subsidence of the pain and swelling. Usually ten to fifteen minutes' massage during the first day is sufficient, but the period may be lengthened to twenty-five or thirty minutes after a few days. Under this treatment there is a rapid subsidence of tenderness and swelling, if the case is sprain, but should fracture be present the improvement is more gradual and the tenderness persists even after several weeks.

In conclusion, attention should again be drawn to the importance of the early recognition of fractures near the wrist joint, and emphasis should be placed upon the fact that "sprained wrist" is a diagnosis which often serves as a cloak to hide the ignorance of the physician. The belief that "a bad sprain is worse than a break" is based upon the fact that a "bad sprain" is usually a fracture, and it is worse than a break because it is treated as a sprain and neglected as a fracture.

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A STUDY OF PEPTIC ULCER FROM THE DIAGNOSTIC POINT OF VIEW.*

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IN this paper I have grouped gastric and duodenal ulcer under one heading, that of peptic ulcer, since physiologically, clinically, and pathologically, the first part of the duodenum is identical with the stomach.

In browsing through the literature, which is voluminous, I was surprised at the alleged frequency of ulcer. Mumford has stated "that of all the lesions giving rise to digestive disorders, ulcer of the stomach and duodenum holds the first place."

W. H. Welch estimated that about 5% of mankind suffer from gastric ulcer, figures founded on findings at autopsy of open or cicatrized ulcers. Some writers place the percentage much lower, while others claim much greater frequency. Mumford believed that the true percentage was probably higher than Welch's estimate.

Careful analysis shows that gastric ulcer is as common in men as it is in women, though duodenal ulcer is perhaps more frequent in males.

The most frequent age given is twenty to forty years, but an analysis of a long series of cases shows that ulcer is more liable to occur between forty and fifty. Frequently ulcer patients are over fifty, but the records show few cases under twenty. Ulcer has been found in young children.

The *conjugal condition, occupation and family history* seem to play little part, but ulcer seems to be a trifle more frequent in the negro race; and if reports from Teuton sources are correct, the Germans seem more prone to ulcer than other nationalities.

In looking over the previous history of ulcer cases, in a few instances there is a definite history of trauma. It is generally conceded that burns may lead to ulcer, especially of the duodenum, but I could find no satisfactory explanation of the circumstance. There is no doubt that arteriosclerosis, by predisposing to thrombosis, plays some part in the pathogenesis of ulcer. Syphilis may have a place in the etiology through the production of a local specific endarteritis. Endocarditis may give rise to embolism and should be considered. One investigator found, in a series of ulcer cases,

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valvular disease present in 9%. It is agreed that any chronic disease which lowers the vitality, such as tuberculosis, increases the liability to ulcer. Alcoholism may predispose. A certain percentage of cases give a previous history of chlorosis, and in a very large majority there is a definite history of "stomach trouble" persisting for months before the onset of symptoms suggesting ulcer. The majority describe it as "indigestion," as shown by discomfort after the ingestion of certain articles of food, eructations of gas and pyrosis. A study of peptic ulcer shows that the previous history of "stomach trouble" verges into the present illness without any line of demarcation. In other words, if we go back to the onset of dyspeptic symptoms, we shall know when the ulcer or ulcers started.

How do peptic ulcers form? This is a much debated question. W. J. Mayo seems to lean toward the theory of mechanical injury plus excessive acidity, while Leube says "A weak constitution, chlorosis, and anemia predispose to ulcer." Riegel states that local trophic changes take place, necrosis ensues and the dead tissue is naturally digested by the gastric juice. He believes that hyperchlorhydria (if present) is a manifestation of abnormal irritability on the part of the secretory glands of the stomach. His is the most reasonable hypothesis from my point of view.

The cardinal symptoms of ulcer are pain, vomiting and hematemesis. In Greenough's and Joslin's series of 187 cases, 96% had vomiting, 92.5% had pain and 79% had hematemesis. Pain is the predominant subjective symptom. In 50% of a series of 82 cases, pain was the chief complaint. In 47.7% "stomach trouble," dyspepsia, or vomiting were the symptoms for which relief was sought. In only 6% was the vomiting of blood voluntarily mentioned.

Only a few of the cases in this series could be termed acute, the large majority being examples of chronic ulcers existing from two months to twenty years. The usually accepted time limit, however, is from three to five years.

Pain is usually the first indication of the existence of ulcer, and is its most constant and distinctive feature, although, as we all know, pain is sometimes difficult to estimate. In ulcer cases it is usually epigastric, sometimes referred to the back, sometimes to the left shoulder blade, and rarely to either hypochondrium. In passing, it is a mistake to believe that the location of pain gives any clue to the site of the ulcer, for in 27 cases where the location of the ulcer was determined at operation or autopsy, the site of pain seemed to bear no relation to the location of the ulcer.

The pain is usually severe in character, though it may not exceed that of hyperchlorhydria; it is rarely continuous and, if so, usually denotes some complication such as localized peritonitis, adhesions or hyperchlorhydria. Pain is variously described as sharp, dull, aching, burn-

ing, colicky, gnawing or grinding, also as cutting or tearing. It is often influenced by position, usually increased by eating, but may be relieved or be entirely independent of food. Gerhardt makes the point that, while the patient would like to eat, he is often afraid to do so on account of the suffering which may ensue.

Vomiting is about as common a symptom as pain. In most cases it is frequent, in a few constant, and in others periodic. In a certain number of cases it is only occasional, following paroxysms of pain, or the ingestion of certain food. The quantity of vomitus varies, but is profuse only when there is pyloric obstruction or hypersecretion. Analysis of a series of 22 cases shows there is less liability to vomiting and pain when the ulcer involves the lesser curvature. In Howard's series pain was practically constant, except in ulcer of the pylorus or lesser curvature. Vomiting is also less frequent where ulcer is duodenal. Indeed it is stated by Moynihan that pain is the only symptom of duodenal ulcer. Ulcers involving the greater curvature or either orifice produce more constant vomiting.

Hematemesis occurred in 76% of 82 cases. Two-thirds of these had coffee ground vomitus, and one-third bright red. Of course the coffee ground vomiting occurs when the blood stays in the stomach long enough to be acted on by the gastric juice. Hematemesis often occurs more than once, and sometimes gives immediate relief to pain and vomiting.

Severe hemorrhage from an ulcer almost invariably gives bloody or tarry stools. This may not be accompanied by hematemesis when the ulcer is in the duodenum or near the pylorus. Patients are not reliable observers as to melena. The color of the blood varies according to the quantity, freshness and length of time taken to pass through the intestinal tract.

Nausea is noted in a relatively small percentage of cases, which seems natural, as vomiting is generally the result of pain, and is an attempt on the part of the stomach to get rid of irritating material. It does not usually require much effort.

There is almost invariably discomfort after eating, eructations, pyrosis, headache, palpitation and meteorism. This may be due to chronic gastritis, or attendant hyperchlorhydria.

Patients with ulcer are usually constipated, probably on account of the small amount of solids taken and retained. On the other hand, a small percentage of cases show looseness if not actual diarrhea.

There is almost invariably a loss of strength and some loss of weight, due to pain and inability to eat. Sometimes the loss of strength is due to hemorrhage. Marked loss of weight suggests cancer but it must not be forgotten that pain, coffee ground vomitus and marked loss of weight can all be present in benign ulcer of the stomach.

Tenderness is the least important of the physical signs. Its absence or presence is responsible for many errors in diagnosis. In a series

of 187 cases, it was noted in 69.5%, and the degree varied from one extreme to the other. It is usually located in the epigastrium, but may be in either hypochondrium, in the right iliac fossa, or the umbilical region.

Authorities seem to differ widely as regards demonstrable mass or resistance. In Osler's and McCrae's series, it was noted in three-fourths of the cases. In Howard's series in about one-third. Gerhardt says it may be found in any one of four conditions, namely, (1) Thickened base and hard margin of the ulcer itself; (2) functional hypertrophy of musculature; (3) localized exudate or abscess from perforation; (4) adhesions between neighboring organs.

It is estimated that the stomach is dilated in 20% of ulcer cases. Dilatation would signify pyloric obstruction; directly from the ulcer, resulting scars, or perigastric adhesions; or indirectly, from pyloric spasm.

The determination of free HCl is of value in the analysis of gastric contents, but too much importance should not be attached to it. In Howard's series, analysis of gastric contents, after a test meal, showed HCl diminished in 26%, practically normal in 27%, and increased in only 18%. It is valuable to note the contrasting results in a series of cancer cases where free HCl was absent in 92%. The relative infrequency of hyperchlorhydria in ulcer cases, as shown by analyses, is somewhat contrary to the generally accepted belief, but it fits in nicely with Riegel's explanation.

Roentgenologists are, of course, enthusiastic over the advantages of their method of diagnosis and claim the ability to diagnose a large percentage of ulcer cases. Doubtless, in many instances, regional x-ray photographs, after the ingestion of a bismuth soup, would show characteristic depressions in the gastric or duodenal mucosa as well as the cicatricial contraction which follows extensive ulcers.

There is but one other laboratory test of value and that is the finding of occult blood in the stools or in the gastric contents.

The alterations in the blood are those of secondary anemia due to hemorrhage.

The urine shows nothing of interest.

Fever is present in about one-third of the cases, but does not exceed one or one and one-half degrees. It is usually not continuous and, if irregular, signifies a complication.

Of 76 cases collected by Howard, 5 had definite pyloric obstruction and two had duodenal stenosis.

In Greenough's and Joslin's series, there were 7 fatalities from hemorrhage and in Howard's cases 7.

Perforation is rather rare, Howard noting 6 cases out of 82.

Parotitis is not a rare complication. I suppose it is due to lowered resistance to bacterial invasion.

There may be almost any associated condition. As regards diagnosis, it is not easy. Sava-

reud estimates that 20% of ulcers do not produce symptoms. M. C. Millet believes that every other case is undiagnosed in life.

Osler says, "The condition may be met with, accidentally, post-mortem. In other cases again, for months and years the patient has had dyspepsia, and the ulcer may not have been suspected until the occurrence of sudden hemorrhage."

H. D. Niles, in a series of 75 cases, found that in 88% the initial symptoms might easily be attributed to any mild digestive disturbance. In his series the average time from the first symptom to operation was 8 years. He believes that if a stomach ailment is of long duration, or recurs without apparent cause, it is probably due to ulcer rather than to a functional disturbance.

Moynihan says that, "hyperchlorhydria is the medical term for the surgical condition called duodenal ulcer."

The recognition of duodenal ulcer is probably more difficult than that of gastric ulcer, for there is usually less symptomatology. The patient usually states that attacks of pain have recurred for years past; sometimes daily, perhaps only once a year. During intervals patients are usually perfectly well, although they sometimes complain of persistent acid dyspepsia, or burning distress some hours after meals, often at night. Relief is obtained by taking food, or some simple antacid, like soda bicarbonate. With duodenal ulcer there is seldom any vomiting, and the physical signs are usually negative. There may be tenderness during the attack or immediately after.

In gastric ulcer troublesome dyspepsia is more likely to be persistent between attacks. The pain is usually of the same character, but is more liable immediately to follow eating. Following a test meal the stomach contents may show occult blood. In duodenal ulcer occult blood might be found in the feces, but seldom in the stomach contents. There is usually more tenderness in gastric ulcer.

In general it is very difficult to differentiate between ulcer of the duodenum and ulcer of the stomach, and it is hardly necessary. It is far more important that we recognize or differentiate between a functional disturbance of the stomach, such as acid dyspepsia and ulcer, no matter whether gastric or duodenal.

There is no single symptom or laboratory test upon which we can rely. We must depend largely on groups of symptoms, and, where after careful study the diagnosis is doubtful, resort to exploratory laparotomy.

Where the diagnosis rests between ulcer and cancer, we must remember that practically all cancer cases show absence of free HCl, accompanied by loss of appetite, and rapid emaciation, symptoms most unusual in ulcer. Differentiation is not ordinarily difficult.

It is more difficult to rule out attacks of hepatic colic. There are the same recurring seizures, possibly the same chronic dyspepsia, and in the

interval the patient is perfectly well. However, the pain is different in character, being colicky, with remissions and exacerbations coming on more suddenly and ceasing more abruptly than in ulcer. It usually starts in the epigastrium but radiates quickly to the right costal border, and around into the back underneath the right shoulder blade. The pain is not relieved by food, alkalis or vomiting. Patients with biliary colic often feel chilly, sweat profusely, are almost invariably nauseated, and vomit frequently.

In about half the cases, jaundice follows the attack. The tenderness and rigidity following the attack is at the right costal border. Occult blood does not occur in the feces unless complicated by some other condition.

If we bear in mind the frequency of peptic ulcer, and are diligent in the study of our chronic "dyspepsia" cases, we may be spared humiliation and remorse. Undoubtedly in many instances ulcer is the true diagnosis.

THE FIRST CASE IN WHICH ABDOMINAL SURGERY WAS SUGGESTED FOR THE RELIEF OF EPILEPSY.

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WITH NOTES ON THE OPERATION.

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ON August 7, 1913, there was published in this JOURNAL an article entitled *The Role of Gastric and Intestinal Stasis in Some Cases of Epilepsy*. This was a report of what was probably the earliest employment of the bismuth x-ray in the study of epilepsy, and in conclusion the writer stated that he felt reinforced in his belief that in so-called idiopathic epilepsy the essential lesion was not in the nervous system. All assertions made in that article have since then been verified by others. In one of the cases, after consultation with Dr. Frank H. Lahey, it was decided to advise the patient to submit to an abdominal operation, not for the cure of constipation, but for the relief of the epilepsy. Sufficient time having elapsed, we now feel that the case may be reported.

The following description of the case and the illustrations are from the original article:

"CASE 2. Male, age 23, occupation clerk. First entered clinic Feb. 15, 1911. Two paternal uncles

had epilepsy. His father's grandparents were first cousins. He is a brunette like his father's family. At fifteen he had measles and shortly afterward his first epileptic seizure. The attacks have occurred about once in two or three weeks since then. They are preceded by tremor of the facial muscles, sometimes twenty-four hours beforehand. They are of grand mal type, lasting a few minutes and followed by stupor for a half hour and drowsiness for the remainder of the day. Examination was negative except for a fine tremor of the outstretched fingers. Salt-free diet and sodii bromidi gr. x t.i.d. were prescribed. Later, because of a bromide rash, Bromotone gr. v t.i.d. was prescribed. Under such treatment there was no change in the severity or frequency of the attacks. On Oct. 9, 1912, when first seen by the writer, he reported that while in New Hampshire during the summer he had been very much worse. He was then instructed in abdominal exercises and advised to use laxatives more frequently. On Jan. 27, 1913, he reported only one attack since the last visit in October, and that was on Christmas Day after dinner. On Jan. 28, 1913, the first radiograph was taken. Plate I shows the stomach much dilated and extending down into the pelvis. Plate II, after five and one-half hours, however, shows the stomach quite empty and the bismuth meal occupying part of the ileum and the colon as far as the splenic flexure. The transverse colon is far below the iliac crests and the hepatic and splenic flexures are thereby much accentuated. Plate V, after forty-eight hours, shows the transverse colon still occupied by the bismuth meal, which would be normal after twenty-eight hours. There is none in the caecum or hepatic flexure and this points to the very low transverse colon as the cause of the stasis. Plate VI, after fifty-four and one-half hours, shows the descending colon and rectum still full, which would be normal after thirty-two hours. *In this case the possibility of relief through surgery suggests itself and this will be attempted with the patient's consent.*"

The patient at that time was a typical epileptic as described in the text-books: heavy in body and mind, unfit for any employment and discouraged. After eight years of treatment by the various bromides and by the salt-free diet, the milk and vegetable diet, *et cetera*, he had not enough confidence left in medicine to begin another course of treatment, and, after the radiographs were taken, he did not return to the hospital until September, 1914. He was then willing to submit to an operation, and on Sept. 17, 1914, a colectomy was performed by Dr. Lahey. After his recovery from the operation, he was unwilling to be placed upon a régime, but ate everything that he cared for and, after his old habit, he bolted and gormandized. Notwithstanding this, he remained free from attacks until Dec. 14, 1914. In March, 1915, he had another attack. He was then willing to submit to treatment and was placed upon a diet list published by the writer in the *Interstate Medical Journal* for December, 1914, and for some time in use in the Boston City Hospital. This diet is not vegetarian or salt-free and in it the abuse of milk is avoided. It may be briefly described as one excluding fried food, fresh white bread, pastry, beans, milk, except in moderate quantities only with meals, and uncooked fruit except oranges, figs and dates. The diet list bears a footnote directing the patient to chew thoroughly, eat slowly, never hurry after eating, never to eat too much and not to eat