low upward. If too much encroachment be made upon the skin a mucous-producing surface will always remain outside the sphincter. If too little is removed the skin margin may be drawn within the grasp of the external sphincter. The membrane which has retracted upward is now pulled down, the longitudinal fibers cut near its margin, and the circular fibers forming the internal sphincter pushed upward, when it is stitched to the margin of the skin. For this I use a continuous suture of catgut passing each time through the loop, as in making buttonholes. The epidermal layer should not be pierced that there may be less tenesmus and discomfort. I usually fortify each quadrant by an independent suture. Aseptic dressing is then applied, and one-quarter grain of morphin is administered. After twenty-four hours the wash should be begun and used two or three times a day, as after the other operation described. It promotes comfort. From one to two weeks subsequent to operation any irritable projections around the anal margin may be smoothed off with the scissors. Two or three times, at intervals of about a month, the sphincters may need to be dilated.

I have found it necessary to do this operation in only about 5 per cent. of the cases operated upon. The result in every case so far as I know has been satisfactory, except in one case, in which there is not as good control of the gas as might be wished. Its cause is probably a break in some of the fibers of the external sphincter at the posterior commissure, which I knew existed at the time, but which I did not think of sufficient importance to repair. In some cases they do not have complete control of the gas for two or three months. I have found this true even in cases where the action of the sphincters appeared normal, and have ascribed it to defective sensation, the gas passing too far down before its presence is recognized.

This is a slight modification of Whitehead's operation, of which the London *Lancet*² says: "In the treatment of internal hemorrhoids the authors advocate ligature and tell us that they never employ any other method. Their objection to every one of the clamp methods is on the ground that with the hemorrhoids some healthy mucous membrane must be removed also, while they consider that Whitehead's operation leads to a serious loss of blood, is tardy in healing and is liable to cause ultimate contraction of the lower part of the rectum. All these objections are imaginary and none of these evils result when operations are carefully performed."

Dr. Walter Whitehead³ of Manchester, in commenting on the above editorial, makes the following, among other statements: "My experience with excision, which now covers a vast number of cases and extends over a period of twenty-five years, has completely convinced me that when the operation is performed as directed and common intelligent principles of general surgery are observed, there is no excessive hemorrhage, and I can absolutely deny that the slightest contraction of the lower bowel ever can or does take place unless some of the skin at the verge of the anus is sacrificed—which would be a distinct violation of the instructions given for the excision. Contraction depends entirely upon the removal of skin, and the contraction bears almost a direct relation to the amount of skin removed."

In my work on the rectum I wish to acknowledge my indebtedness to Drs. Allingham, Mathews, Pratt, Ball, Gross, Quain, Gant and others.

2. Editorial, Jan. 26, 1902, reviewing "Diseases of the Anus and Rectum," by D. V. Goodsall, F.R.C.S. Eng., and W. Ernest Miles, F.R.C.S. Eng.

3. London *Lancet*, February 5.

THE CAUSAL RELATION OF BLOOD POVERTY TO GASTRIC ULCER:

WITH REPORT OF AN ILLUSTRATIVE CASE WITH ATYPICAL SYMPTOMS.*

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As there is perhaps no subject the etiology of which has been so thoroughly thrashed over as that of gastric ulcer, and with so little final satisfaction in the way of a definite conclusion, it may not prove uninteresting to consider the condition with reference to a case that presents strong evidence of an actual causal systemic condition. Although traumatic as well as tubercular and syphilitic ulcers, and similar lesions of the stomach, are infrequent phenomena, when they do occur their etiology is clear because of known existing conditions. The characteristic peptic ulcer, or, as it might be well named, the pyloric ulcer, from its frequent occurrence in this position, gives, however, only a hint as to its causation; and the best writers on the subject satisfy themselves with the statement that in all probability a lesion of the mucous membrane has been present, followed by self-digestion of the gastric wall. In studying the etiology of the pyloric ulcer we have at hand the following undisputed facts gathered from clinical and portmortem observation.

The typical peptic ulcer occurs in about 5 per cent. of all hospital cases (Welch, Bramwell, etc.), and in probably a slightly lower percentage of the general population. Statistics are by no means fair means of arriving at such figures, since few cases are autopsied outside the walls of hospitals, and yet without question many of the general population are carrying unrecognized gastric ulcers into their daily life. On the other hand, hospital subjects show a greater tendency to lesions of all kinds than the general population. Statistics are, however, our only guides to an approximate knowledge of the frequency of the condition. The age at which gastric ulcer most frequently occurs seems at the present time to be between the 16th and 30th years, although Brinton, in his classic monograph (1857), stated that of the cases studied by him the greatest number occurred in the second half of adult life and in old age.

It seems also to be agreed generally that the peptic ulcer occurs in a large percentage of all cases at or near the pylorus, on the posterior wall, and near the lesser curvature. (Mayo Robson's recent statement that latent gastric ulcer is usually found at the lesser curvature near the cardia is not borne out by general experience. He states that such a position easily explains the latency of the lesion, since food is taken in the erect posture and is less likely to irritate the eroded gastric wall at this point. We know, however, from the autopsy table that latent ulcers outnumber the open variety two or three to one; and as Robson himself admits that the greatest number of peptic ulcers are lodged near the pylorus it seems difficult to follow his reasoning in arriving at these contradictory conclusions.) We know, also, that its character is that of a funnel, with the smaller opening toward the serosa and the larger in the mucosa itself, while its base is usually clean and its walls punched out and sharply defined; also, that it usually occurs in single formation, though as many as five are frequently found and thirty-four were noted by Berthold in a case cited by nearly every writer on the sub-

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. Frank A. Jones, George Dock and J. M. Anders.

ject. Even when multiple these ulcers have usually been found near the pylorus, and often at points in apposition with one another. It is also generally conceded that the female sex is especially involved, and in many instances the lower classes, including seamstresses and housemaids; also, that associated with the ulcer there is usually, if not always, a marked condition of anemia. Finally, the theory seems to meet with general approval that for one reason or another the mucous membrane of the stomach loses its resisting power to the acids of the gastric juice, and that self-digestion takes place. There is a tendency,¹ especially at the present time, to ascribe gastric ulcer, with every other pathologic condition, to microbic infection, but it seems safe to say that as yet this is not the preponderant opinion. In fact, it seems to be the most difficult one to explain.

Further than the foregoing we may not venture, and the authorities in considering the features of peptic ulcer lead us further only into conjecture. Just what causes tend to a loss of resistance to the acid gastric juice have never been satisfactorily determined. Arterial and venous thrombosis have been suggested, and undoubtedly do occur, but rarely, and still more seldom in the conditions that are associated with gastric ulcer. Embolism has been a favorite experimental theory, but emboli of the gastric mucosa almost invariably occur at the cardia, while peptic ulcer is much less often found at this point. Moreover, thrombi and emboli have only very rarely been found in the postmortem stomach, and are difficult to cause experimentally. Still further, cardiac disease and other conditions of the circulatory system that tend to the formation of emboli do not furnish a sufficient number of gastric ulcers to form more than an incident in the entire number, though gastric disturbance is frequent in these conditions and almost the rule. Mechanical injuries, contact with corrosive substances, compression (as from tight belts and corsets), microbic infection, have all been suggested, and in isolated instances all undoubtedly play their part, but to no such extent as to account for such a percentage morbidity as one-twentieth of all hospital patients.

As early as 1857, however, Brinton remarked of gastric ulcer that "careful clinical study leaves me . . . little doubt with respect to the remarkable influence of poverty and intemperance." And Gempt, in 1886, wrote that "it is often necessary to remove the ultimate cause, i. e., the anemia or chlorosis before the healing of a gastric ulcer can be accomplished." In order to bring about this result he gave the albuminate of iron, although other authorities at the time condemned the use of iron at this stage on account of its irritant effect. Gempt did not even look on hematemesis as a contraindication to the use of iron. Osler in his text-book says "anemia and chlorosis predispose strongly to gastric ulcer"; Ewald and Leube both suggest a causal connection between anemia, hyperchlorhydria and gastric ulcer; and finally the case reported in this paper lends evidence of the strongest kind to the theory that the necessary condition of lowered resistance to the digestive action of the gastric juice is best furnished by a gastric poverty of blood—including in the term a lack of one or all of its constituent parts.

Mrs. —, a physician's wife, 25 years of age, was first seen by the writer in May, 1901, at which time she was pale and chlorotic in appearance, but felt well except for a constant wearied feeling of which she spoke as nothing new in her experience at that time of year. Her blood was examined and showed hemoglobin 55-60 per cent (Fleischl), red corpuscles

4,650,000 and leucocytes 8200; no poikilocytosis; no other pathologic blood changes. She had always been pale but no other blood record had ever been obtained. She was then placed upon a course of iron treatment by her husband, which was followed only irregularly. The patient, however, felt well throughout the summer.

Her father is alive and well. Her mother died of carcinoma of the mamma after a very late fertility (48). Two brothers and one sister of the patient have the same pale, anemic appearance, though presumably to themselves and others, perfectly well. The patient was always well and strong during childhood, and had no serious illness until her sixteenth year, when she went through an attack of typhoid fever, complicated with severe cerebrospinal symptoms. She recovered perfectly, but has had since that time a tender spot over one spinous process (the fourth cervical) that has always been sensitive to the touch and to pressure.

The menses began about the sixteenth year and have been regular; they last over a period of five or six days, and considerable blood is lost. This was also the case with her mother, and is now with a sister who is still living and well. There is some functional pain on the first day, but with the appearance of the flow this disappears. No history of edema or headaches, but a slight tendency to occasional bilious attacks, during which she has suffered from headache, constipation, nausea and sometimes vomiting, and then again has felt perfectly well. Often with these bilious attacks there was nosebleed, though the latter has not occurred for more than a year. Never any bleeding from the bowel or stomach. Patient has always craved sour things, and especially during the last year. Appetite always good, bowels regular. Patient has always looked upon her stomach as her strong point.

When the writer was asked to see the patient on Nov. 19, 1901, she had felt well since last examined, six months previously. Three days ago she felt dizzy and nauseated while in the street, and several times since then has been so dizzy as nearly to faint. Yesterday she had an attack of vertigo and again to-day, and was barely able to reach home. There was repeated vomiting of greenish bilious matter. The bowels moved thoroughly yesterday owing to fractional doses of calomel prescribed by her husband. No nourishment has been retained for two days, vomiting taking place within a few minutes after the ingestion of food. To-day she is unable to stand or even look at the light, on account of immediate vertigo, nausea, and vomiting. No blood at any time in the vomitus or stool. No history of pain in any part of the body up to this time, except for some soreness in the epigastrium after vomiting. Menses appeared four days ago, and have disappeared to-day. No difficulty in micturition.

The physical examination showed a rather slender but symmetrically built woman, skin very sallow, conjunctiva and mucous membranes suggest a slight icteroid tinge. No real jaundice. Skin warm, neck normal, no glandular enlargement. Pharynx normal, tongue clean but dry. Temperature normal, pulse 85 and of good character. Arteries soft. Lungs normal with full and equal expansion giving no pain. Cardiac sounds all clear at valve areas. Over the pulmonary region a suggestion of a faint systolic murmur (hemic), not transmitted. Cardiac action regular but rather feeble. Abdomen scaphoid; rather tense condition of recti; and very slight tenderness over the epigastrium. Pain does not seem localized to one point. Liver and spleen normal; no appendiceal tenderness or rigidity.

Blood—Hemoglobin, 70 to 75 per cent.; red blood count, 4,880,000; leucocytes, 4000. No poikilocytosis. No other pathologic changes. Urine—1034, acid, amber, turbid (urates), sediment very abundant, pink, semisolid, albumin faintest trace, sugar none. Full of amorphous urates, many clusters of large uratic crystals. Many long, thin, uratic casts; few hyaline casts. No renal epithelium. Much mucus. Few leucocytes. Feces—Liquid, light yellow, no blood noted. No parasitic segments. Vomitus—Small quantity, pale, green color, odor natural. Not tested chemically. Patient was given a capsule of bismuth, cocain and small (gr. 1/24) doses of calomel.

Nov. 20, 1901, next day. Marked change for the better. This morning is able to retain liquid food with relish. Dizzy

1. Böttcher, and more recently, Box, Brit. Med. Jour., Feb. 8, 1902, et al.

only when on feet. Skin seems much clearer and whole expression brighter. Tongue clean. No vomiting since noon yesterday. Abdominal walls more relaxed and abdominal tenderness nearly gone. At this time the attack was looked upon as one of vertigo and gastric distress in chlorosis, and rest in bed was ordered with iron and strychnia, and careful overfeeding with milk, according to tolerance.

November 23, three days later, patient is better in most respects, but now complains of a burning pain excited almost at once upon the ingestion of food, and localized over a small area just below the ensiform cartilage. This pain is transferred acutely and constantly to the fifth, sixth and seventh dorsal vertebrae. Bowels opened with an enema yesterday and passages contained no blood. No further vomiting to-day and no headache. On examining the abdomen a slight tendency to rigidity was met with over the epigastrium, but no distention. Just below and slightly under the ensiform cartilage there is a distinctly circumscribed and very tender area, not larger, if as large as a silver dollar. No tenderness at any other point over the abdomen. Appendix negative. Marked pain on pressure over the fifth, sixth and seventh dorsal vertebrae, transferred to and from the epigastric area of tenderness. Gastric ulcer was suspected at this time, though the diagnosis was not believed to be fully warranted.

November 24. Patient this morning seems bright and well, all dizziness being gone. Dressed herself and bathed superficially. Slight quantities of food tasted good, but invariably started the same burning pain over the same circumscribed area. Distinct spot of tenderness present over the fourth dorsal spinous process, and pain is referred to this from the epigastrium. Is sometimes burning, sometimes dull and heavy, and not constant. Tongue quite clean. Epigastric pain on gentle palpation is much aggravated. No tenderness in surrounding parts. Pain last night came one-half hour after eating. This morning is more like a dull weight, after the test breakfast. No nausea or vomiting. Test meal of one roll and glass of water. One hour and a half later 8 c.c. of pale gray fluid were withdrawn. No blood evident macroscopically, fresh or clotted. No lactic acid. HCl markedly increased (0.65 per cent.). Microscopically: full of starch granules, considerable number of red blood corpuscles, few leucocytes, few long thread-like bacilli, no sarcinae. Urine (a. m.): 1013, acid, straw color, clear, sediment abundant, white, flocculent; albumin faint trace; sugar none; chlorids slightly diminished. Many squamous epithelial cells, few leucocytes, no casts, no crystals, considerable mucous; p. m., 1030, acid, amber, clear, sediment scanty, white, flocculent; albumin none, sugar none; urea 2.5 gm. per 100 c.c.; chlorids markedly diminished; few small hyaline casts, much mucus, no renal epithelium, many leucocytes, no crystals.

Nov. 25, 1901. Seen in consultation with Drs. Frederick A. Packard and A. D. Smith. Same symptoms as at time of last note. Pain in epigastrium about the same, sometimes burning, sometimes dull, but nearly constant, localized over the pyloric region, and in the same circumscribed area. Pain still referred to the dorsal spine. Diagnosis of gastric ulcer concurred in by consultants.

A further description of the case may be omitted except to state that the treatment consisted of rectal nutrient enemata of peptonized milk, predigested beef, and egg albumin, all mouth feeding being discontinued. Absolute rest in bed was insisted upon, and a pill of nitrate of silver and opium (gr. 1/12) given q. i. d. On the following day the patient showed positive signs of opium poisoning, and belladonna was substituted in the pill for the former drug. The pain and burning were controlled by carbonated water and at times by a little champagne, though both continued to some degree until the localized tenderness disappeared on the 30th, when for the first time palpation elicited no pain. Beef juice and peptonized milk were then gradually fed by the mouth, the rectal enemata being dropped one by one until at the end of another week the feeding was entirely *per os*. The patient at this time was much emaciated, but there was at once a rapid and steady gain. Gradual overfeeding with milk and eggs and whisky was cautiously attempted and for two weeks the patient in-

gested from two to three quarts of milk and from six to eight raw eggs daily. She sat in a chair, December 16, for the first time, one month from the time she was placed in bed. The gain in condition continued on iron, strychnia and a careful, overful diet until January 4, when she went out for a short drive. On January 22 she sailed for Europe seemingly perfectly well, and free from subjective and objective symptoms.

The blood picture in May, 1901, when first seen (six months before attack) was hemoglobin, 55 to 60 per cent.; red blood count, 4,650,000; leucocytes, 8200. On Nov. 19, 1901 (third day of attack), hemoglobin, 70 to 75 per cent.; reds, 4,880,000; leucocytes, 4000. Dec. 8, 1901, hemoglobin, 73 per cent.; reds, 4,216,000; leucocytes, 6100. Jan. 18, 1902, hemoglobin, 82 per cent.; reds, 4,360,000; leucocytes, 5200 (sailed for Europe next day). At this time the patient's appearance was better than at any time before or since her illness, and in all probability her blood condition was better than at any previous time in her existence. She was in Europe and Asia, sightseeing moderately, for three months, and returned in what seemed to be first-class condition on April 15, 1902.

On May 6 last her blood was examined, and after several confirmatory repetitions of the hemoglobin estimation, it was recorded as follows: Hemoglobin, 70 to 72 per cent.; reds, 5,056,000; leucocytes, 4000. The pulse was good. There were no subjective or objective symptoms. Patient felt a little tired after the work of rearranging her house for occupation. She was immediately placed on a diet of pure beef juice, squeezed from the beef after a few hours mild heating (not cooking) in addition to her ordinary food. On May 22, sixteen days later, and the day following the cessation of her usual long menstrual period, the blood picture was: hemoglobin, 82 per cent.; reds, 4,500,000; leucocytes, 4800. On June 4 the hemoglobin reading was 87 to 88 per cent., the highest point that had been reached at any time.

The above history has been cited somewhat in detail because it appears to illustrate an occurrence which, to my mind, can not have been an infrequent one in the history of gastric ulcer, and one in which the anemia was known to have preceded by months and probably years what was either a sudden ulcer formation at or near the pylorus, or the sudden exacerbation of a chronic ulcer that had previously given no symptoms. It does not seem possible that the diagnosis could have been a mistaken one, as the symptom complex was recognized at the time as incomplete and rather atypical. There was present, however, on the third day and thereafter, a very typical burning pain in a sharply circumscribed and very tender area, excited almost at once by the ingestion of food, and transferred in a characteristic way to the dorsal spine. The abdomen was scaphoid and the recti rigid, especially over the epigastrium. There was also a marked chlorotic blood picture, a marked gastric hyperacidity, the presence of red corpuscles in the gastric contents and a diminution of the chlorids of the urine, all of which, with the age of the patient, made only one diagnosis probable. The absence of hematemesis is seen in nearly 50 per cent. of all cases, and Boas has recently reported ten cases of gastric ulcer in which there had been no hematemesis, and in the gastric contents of which blood had never been seen with the eye, but was recognized as an "occult" hemorrhage, by means of the chemical tests. The blood, he states, in some of these cases (four out of ten) was repeatedly but not constantly found.

Granting, then, that the case was one of gastric and pyloric peptic ulcer, with atypical, or rather aclassical symptoms (for I do not believe that in this case the two words are synonymous), we are driven to the choice of character of the ulceration between 1, a small symptomless lesion of long standing, suddenly induced in a deeper and wider growth; 2, an embolus, or thrombosis,

and a consequent ulceration of the infarcted area; 3, a hemorrhagic erosion due to the rupture of some small capillary in the mucous membrane, and 4, an acute peptic ulcer, caused by localized digestion of a gastric mucous membrane, whose powers of resistance to the gastric juice had become so depleted by long standing local and general anemia that it finally gave way to digestive action. In the latter event the severe onset of the symptoms could easily be explained by the nervous shock of the first ulceration, while the later appearance of the typical pain may be readily traced to the gradual invasion of the deeper and more sensitive tissues.

With regard to the first condition, that of a symptomless chronic ulcer, with a violent acute exacerbation, we are not disposed to deny the possibility of its occurrence. Welch found that of 11,888 autopsies studied by him from the literature of the Prague hospitals, 164 (1.4 per cent.) showed open gastric ulcers, and 373 (3.1 per cent.) cicatrices or healed ulcers that had not been recognized prior to death. Grünfeld examined 1,150 bodies for gastric ulcer, and found it present in 124 cases (11 per cent.). In 700 cases in which an examination was made, but not with a view particularly to the discovery of gastric ulcer, the latter was found in 4.8 per cent.; and in 450 cases carefully searched for cicatrices ulcer was found in 92 cases (20 per cent.). All of these were found in Copenhagen, where gastric ulcer seems to be a frequent condition. Jaksch (quoted by Brinton) in 2,330 autopsies found 113 ulcers, of which 57 were open sores and 56 cicatrices; Willigk, in 1,600 autopsies, 139 ulcers, of which 74 were open and 65 cicatrices. Finally Barker, in February of the present year, showed in his clinic nine cases of chronic ulcer of the stomach, all of which had had typical symptoms for many months, and in several cases he verified his diagnosis at operation. He speaks of the condition as "a very common form of gastric disease such as you may often see treated in the outpatient department or the medical wards." All of these cases were seen within a short time.

And yet in spite of the frequency of chronic gastric ulcer I hesitate to believe that the symptoms of the case under discussion point to it as a probability here. If symptomless before, why the sudden outbreak with no known cause; and still more cogent, why the prompt healing and seemingly perfect convalescence of what is often and even usually an obstinate condition? These reasons are neither conclusive nor final, but they are forceful and accord with our knowledge of the usual behavior of gastric ulcer and must be considered. I do not, therefore, exclude the possibility of a chronic ulcer, though I do not consider it the most likely condition.

Upon the second possibility, that of an acute ulcer of embolic or thrombotic origin, I must look with still less favor, since there was no condition known to be present that would suggest an embolus; while the only known possibility of a thrombus rested in the anemic condition which was of long standing but of a temporarily less degree than usual owing to treatment. We must, moreover, again consider the rarity of thrombosis and embolism at this point, and the important fact that of the few times that the latter condition occurs by far the most numerous are at the cardia. Walsh states that no one has ever experimentally produced a solitary embolus of the pylorus. Hemorrhagic infarct has, however, been observed postmortem in the stomach, both alone and with ulcer formation; and in such cases thrombosis may have been present, either as a causal or consequent condition. Rindfleisch reports such a case, and v. Reckling-

hausen another. Both state, however, that the condition is a great pathologic rarity; and this very rarity prevents our considering thrombosis as a probable condition, in the presence of a more satisfactory explanation.

The third suggestion is that of a hemorrhagic erosion. All authors mention this as a possible early stage of gastric ulcer, and all pathologists refer to it as a frequent postmortem finding. No one can deny that a small hemorrhage may have occurred as a result of the vomiting during the first days of the attack. And yet there was a gastric condition present at the earliest stage that needs more of an explanation than that of an erosion, and which possibly, and to my mind probably, was due to a superficial ulcer already present; this in view of the later symptoms. If a hemorrhage occurred, its immediately surrounding area might easily have offered a susceptible focus for gastric digestion; but this would not have explained the early violent attack (which in some features suggested gastric perforation) as well as an ulcer newly formed, still superficial and due to causes that will be discussed immediately.

We are led finally to the theory which I consider the most rational, and which I believe may be the explanation of many if not the majority of acute and chronic peptic ulcers. It has already been intimated that the most likely condition appears to have been an acute peptic ulcer, located as usual, at or near the pylorus, and due to digestion of a gastric wall rendered susceptible to such action by a long-standing anemia, a consequent poverty of blood and inanition of the part, in the presence of a hyperacid gastric juice. My reasons for arriving at these conclusions are as follows: We were fortunate to have examined the blood of the patient six months previous, and knew that it was markedly deficient in hemoglobin and to a lesser extent in red corpuscles. The appearance of the patient had from childhood suggested a chlorotic condition. During this entire time and until the attack occurred gastric digestion had been apparently faultless, and certainly was symptomless. We therefore find ourselves in the possession of two important facts; first, that the patient for months and perhaps years had been lacking in hemoglobin and red corpuscles; and secondly, that up to the time of her final onset of symptoms nothing had occurred to even attract attention to the stomach. This, we may add, is never the case in a recognized chronic gastric ulcer, which has its own succession of dyspeptic, and finally ulcerative symptoms, that are always present, though not always ascribed to the correct cause.

It has been repeatedly shown (Ritter u. Hirsch et al.) that with the chlorotic condition is often associated a hyperacidity of the gastric juice, and as if to form a combination of conditions most liable to cause self-digestion of the gastric wall, Pavy, Silbermann and others have shown that in all anemic conditions the alkalinity of the blood is more or less decreased. We have, then, in long-standing chlorosis or anemia an abnormal power in the digestive action of the gastric juice, while the main influence of resistance against this acid content, viz., the alkalinity of the blood that fills the capillaries, is removed.

There were present in our case the ideal conditions for the formation of a peptic ulcer at some point in the stomach, the entire wall of which was suffering primarily from a lack of blood in sufficient quantity and quality, and secondarily from the lack of protection ordinarily afforded by the alkalinity of a normal blood supply.

The reasons for the frequent involvement of the pylorus in digestive lesions have been repeated so often

that they need not be cited here. I wish, however, to call attention to a fact with reference to the circulation of the pylorus to which I find no reference in the literature, and which to my mind lends a still greater emphasis to the theory that gastric ulcer is often if not usually dependent on pyloric anemia, or the results of circulatory disturbance. While the blood supply of the stomach is a free one, especially to the cardiac end (gastric, left epiploic and the brevia), and a sufficient one to the pylorus (pyloric and the right epiploic), the venous outlet is free only from the cardiac end of the stomach. The latter is drained by the large coronary vein which passes along its lesser curvature, by the left gastro-epiploic and the vasa brevia. The pyloric end of the stomach, however, has a much smaller outlet in the pyloric and the right gastro-epiploic veins, both of which are small, while the former enters the portal vein almost at a right angle, and the latter joins the superior mesenteric under the same angular disadvantage. In normal conditions, and with normal blood pressure, this venous exit is ample and able to discharge the pyloric blood at a rate prompt enough to avoid stagnation or congestion. In anemic conditions, however, in which the blood pressure is often so low that it seems impossible for the heart to force a drop from the deeply pricked finger, an outlet that is ordinarily sufficient may easily become inadequate and the cardiac activity too slight to force the pyloric blood into and against the rectangular stream of the portal system. In short, a venous congestion of the pylorus will in all likelihood be added to an impoverished arterial blood supply, a lowered alkalinity of the blood and a hyperacid condition of the gastric juice.

It must be frankly admitted that peptic ulcer sometimes occurs in cases in which there is no hyperacidity; but under such conditions as have been pictured hyperacidity seems hardly necessary to self-digestion, and a normal or even subacid condition might easily erode the depressed gastric wall. This actually occurs postmortem, as soon as the protective circulation of the gastric wall ceases to flow, and is often seen in the cadaver.

As to the frequency of association of chlorotic and other forms of anemia there is no lack of evidence. V. Limbeck states that either the chlorotic state may precede or that the stomach impairment may cause the blood condition. Welch suggests that amenorrhea, chlorosis and anemia favor the development of gastric ulcer. He seems to intimate, however, that the anemia follows (p. 487, *Pepper's System*) rather than precedes the ulceration. Osler goes a step farther in stating that "anemia and chlorosis predispose strongly to gastric ulcer," and both Ewald and Leube speak of a possible causal connection between anemia, hyperchlorhydria and the condition in question. Strümpell, however, believes that the influence of chlorosis may have been exaggerated. Gempt (to whom reference has already been made) seems to have been one of a very few writers to express the conviction that the anemia and the inanition consequent on it, particularly of the pyloric end of the stomach, was the primary predisposing condition.

Many interesting experiments have been attempted in the study of the pathogenesis of gastric ulcer, such as the ligation of the arteries supplying the stomach (Pavy), the introduction of multiple emboli into the gastric arteries (Panum and Cohnheim), the ligation of the portal vein (Müller), trauma through the abdominal wall (Ritter), and more recently, the cutting of the vagi (Saitta, Yzeren). In the last year experiments have been reported by Fütterer of Chicago that led him to conclusions much in line with my own theory of

peptic ulcer formation. Fütterer found that in animals in whose stomachs ulcers had been artificially produced, there was a marked tendency to healing as long as the hemoglobin was present in full proportion. As soon as he destroyed the hemoglobin he found that this tendency to heal disappeared and was followed by the characteristic appearance of peptic ulcer as seen in man. From this he concludes that peptic ulcer depends mainly on the lack of hemoglobin, and states that he feels warranted in asserting "the dictum that an ulcer of the stomach can form only when there is a certain amount of hemoglobin lacking, and that it must heal when the hemoglobin percentage is increased to a certain amount." His conclusions hardly seem fully warranted, however, in the light of clinical results. There are on record innumerable instances of gastric ulcer, treated with success, and yet in the handling of which the anemia was totally neglected, because it was not considered the causal condition. Cornils cites the occurrence of a gastric ulcer in his own person, that was cured without any other treatment than rest and a purgative medication. He also notes other similar cases from his experience. Pariser in the present year in his study of gastric ulcer dwells at length on the local treatment by remedial agencies, and barely mentions the treatment of the anemic condition. In fact, few only of the text-books emphasize this need, and their authors have all of them cured gastric ulcer perhaps many times by simple and primarily local methods. We must not forget, however, that all modern forms of treatment include absolute rest in bed and that such a provision means less of a drain on and therefore a gradual bettering of the blood supply, though probably not an increase of the percentage of hemoglobin. It seems necessary to believe that in men both local and systemic treatment are required; but it is equally certain that peptic ulcers occur in individuals with a high hemoglobin reading (cases cited by Cabot, Greenough and Joslin, etc.), and that often they heal without any change in the hemoglobin percentage. The latter fact is evidenced by the large number of cicatrices (i. e., healed ulcers) found postmortem in cases that have had no treatment whatsoever, and have died as the result of another condition.

We must agree with Fütterer, notwithstanding, that the hemoglobin percentage is an important matter, both in the causation and in the treatment of the condition, and only differ from his belief that it is the only or the main factor in the case. Cabot in 1897 reported 28 cases of gastric ulcer, of which 13 showed less than 50 per cent. hemoglobin, and only 4 over 80 per cent.; while of the 21 cases in which the red corpuscles were counted 5 had under 3,000,000, and the average red cell count was 3,800,000. As a rule the color index was low, though Cabot quotes Ostertag to show that it may be high. He also quotes Grawitz in citing a case of peptic ulcer in a very pale girl whose hemoglobin registered 90 per cent. and later 91 per cent., and in whom the red corpuscles numbered 4,140,000, and later, 4,340,000. In this case, however, the dry residue of the serum was only three-quarters of the normal amount, and the girl was really anemic, the contradictory blood picture being due to anemic concentration. Oppenheimer has also called attention to the great pallor in certain cases with normal counts and hemoglobin readings. He cites six cases of gastric ulcer, however, of which the highest reading was hemoglobin 75 per cent., with 4,100,000 red corpuscles, and the lowest, hemoglobin 25 per cent., with 1,200,000 red corpuscles, while the other cases ranged between these figures. Greenough and Joslin

have studied the cases of gastric ulcer treated in the Massachusetts General Hospital for the years 1888-98, and out of 13,097 patients treated found 187 cases of gastric ulcer (1.4 per cent.). There was pallor in 131 out of the 164 patients regarding whom any note of this condition was made. Hemoglobin and blood estimations were made in 73 cases, in 34 of which the hemoglobin was below 50 per cent., and in 64 below 80 per cent. The red blood cells were counted in 43 cases and in 24 the count was below 4,000,000. The writers seem to look on the anemia rather as a symptom than as a cause. DaCosta reports 20 cases of gastric ulcer in all of which the hemoglobin was below 80 per cent., in 8 below 50 per cent.; the series averaging 57.8 per cent. The red blood cell count averaged 4,017,000. Nearly all authorities note an absence of leucocytosis, though this is not an invariable rule.

A number of interesting cases somewhat similar to the one reported in this paper have appeared in recent medical literature, among which Maunsell reports one of a servant girl who had suffered from anemia for some months, and for some weeks from uneasiness in the stomach. Perforation occurred with a sudden onset of pain. Death followed the operation and at the autopsy "a single ulcer of the acute type" was found.

Shettle reports a case of "an anemic girl aged 17 years" who "had suffered from anemia with dyspeptic symptoms for some time, but had none of the typical symptoms of gastric ulcer." Suddenly she had pain, fainted, vomited, and then ensued general abdominal pain. Operation was performed, and an ulcer was found near the cardia.

Goodhue cites two cases, one of a woman 24 years old and six months pregnant, "anemic and chlorotic since puberty, with amenorrhea, dysmenorrhea, and metrorrhagia, at different times." Miscarried on Sept. 14, 1893. At this time gastralgia, but no other symptoms than those of dyspepsia. On November 21 of the same year she experienced nausea and coffee-ground vomit; there was blood in the vomit and feces, and burning and boring epigastric pain. The case finally recovered.

His second case was noted in an anemic and neurotic woman of 32 years, a nurse who had had amenorrhea since puberty. For four or five years she had had digestive troubles. Suddenly perforation took place with pain and vomiting of blood, though the case was not properly diagnosed until a late consultation, and death followed. The autopsy showed an ulcer in the pyloric region.

Finally Stratton, in March, 1902, reports a case in which the pain was more severe when the stomach was empty, with spells of pain coming and going every four or five hours. The attacks lasted a week or ten days, and were induced by exposure to wet and cold rather than by food. A tender spot always localized over the epigastrium in the same area was present, but the patient never vomited blood, food, or gastric secretions. The tenderness entirely disappeared between attacks. Finally, under an unusual exertion, there was sudden intense pain; then none for 10 minutes; and again a violent pain in the epigastrium. Operation was performed and perforation found in the anterior wall near the pylorus. This case is cited especially because of its many features resembling those of the one now reported by myself. Before summing up our conclusions it will be well to note that Saitta, in 1900, in treating two cases of gastric ulcer (one following long-standing gastrosuccorhea, and the other succeeding digestive disturbances, with nervous involvement in both cases, as

well as chloro-anemia, and neuralgia), thought of the vagus as the cause of the trouble, and as furnishing a predisposed soil. He severed the nerve on both sides in 16 rabbits, and fed them on a weak solution of hydrochloric acid. By this means he induced multiple ulceration, with general gastritis; in control animals, with the pneumogastries intact, the procedure had no effect.

Yzeren, in 1901, reported a series of experiments, also in rabbits, to justify Talma's theory that gastric spasm is the underlying cause of chronic peptic ulcer. By cutting the vagi below the diaphragm he found that he could produce ulcers corresponding absolutely with those occurring in the human stomach. They appeared several days after the operation, and showed no tendency to spontaneous healing. Usually only one ulcer appeared, and this in the pyloric region close to the lesser curvature. It was preceded by necrosis of the mucosa, while the structures more remote from the pylorus were not affected. Gastro-enterostomy or incision of the pylorus seemed to prevent or delay the ulcerative process. After the operation the stomach was distinctly harder, indicating spasm, this spasm of the gastric muscle helping to produce anemia of the mucosa, and resulting in necrosis. As the muscle is thickest around the pylorus, the lesion is most likely to be situated here. Both of the latter observers, it will be noted, produced, and studied the effects of, a local pyloric anemia.

Ewald, in his paper before the German Congress on Internal Medicine, held during the present year, considers Yzeren's theory very improbable, and believes that both conditions (ulcer and spasm) are the result of hyperacidity. His own figures, however, would rather convince the unbiased observer that in at least 76 per cent. of the cases some other cause than hyperacidity was at work, since in just this percentage of cases he found hyperacidity absent. He states that he has seen over 1080 cases of gastric ulcer in the last 10 years of which 34.1 per cent. showed hyperacidity, 66.8 per cent. normal acidity, and 9 per cent. subacidity. Blood was frequently found in the contents, and hematemeses was present in 203 out of 364 patients (54.5 per cent.).

Our conclusions with regard to the causation of peptic ulcer of the stomach are as follows:

1. In many, and perhaps in most cases, a high grade of anemia precedes the appearance of peptic ulcer, and this anemia usually assumes the chlorotic form.
2. With few exceptions, gastric ulcer is attended by the symptoms of marked anemia. In certain cases in which the blood picture fails to show a reduction in the hemoglobin percentage and in the number of red cells the fact seems to be due to concentration of the blood dependent on anemic causes.
3. General anemia implies anemia also of the pylorus, and consequent inanition of its mucous membrane.
4. The venous outlet from the pylorus is one that in anemic conditions would predispose still further to an unhealthy condition of the muscular and membranous coats of the pyloric wall.
5. Hyperacidity is usually present in chlorosis and often in other forms of anemia; and when present in any condition it is an influence predisposing to gastric ulcer, when associated with an anemic gastric wall.
6. Hyperacidity is not essential to self-digestion of the stomach wall, provided that wall lacks the proper nourishment and stimulation of a healthy or sufficient blood supply.
7. Any form of hemorrhage, menstrual, operative, or anemic, may either predispose to, or accentuate an already present anemic condition of the gastric wall, and

such hemorrhages are *de facto* often followed by gastric ulcer.

8. Finally, the foregoing facts, in association with the general one that gastric ulcers are seen almost invariably in anemic subjects, warrant the conclusion that blood poverty is a leading, and the usual, predisposing cause of peptic ulcer.

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DISCUSSION.

DR. FENTON B. TURCK, Chicago—This subject is one of great interest and importance. The first part of the paper has dealt with well-recognized etiologic factors. He did not call attention to the recent observations made with special reference to the muscular activity of the stomach in which it was determined that the lack of motility was one of the chief factors in cases of gastric ulcer. Recently Kocher called attention to the ulcers found in the jejunum following a gastro-enterostomy; here a true peptic ulcer was found. The conclusions reached were that such cases, in which there was a lack of motility and a lack of the muscular contraction together with disturbances of the splanchnic circulation, especially the portal circulation and circulation in the intestines, resulted in true peptic ulcers of the jejunum. In my own observations carried out one year ago I made a large number of experiments with guinea-pigs and rabbits, keeping them in small cages in which they scarcely had room to turn around, confining them in the dark; at the end of a certain time only seven or eight were alive, some showing the chlorotic type of blood, and in two were found ulcers which had the appearance of peptic ulcers which failed to heal and showed other evidences of true gastric ulcers. The evidence adduced seemed to show that, clinically, there were two important factors in their causation: 1, disturbance of the circulation; 2, a lack of gastric tone, or motility, or gastric myasthenia. Bramwell has shown that most cases have occurred in girls coming from the country leading sedentary and indoor lives. It has been noted that girls with poor circulatory disturbances since early childhood at puberty acquire these ulcers and it is stated that the early environment and personal hygiene were the direct factors at play, causing a disturbance of the circulation in the splanchnic area; this interference with the muscular tone of the stomach accounts for a large number of the lesions.

DR. JAMES J. WALSH, New York—With regard to the causation of gastric ulcers it is pretty well recognized that the basis

for the development of the lesion is an anemic condition, and I think that nearly every general practitioner has realized that all such cases have a traumatic element which may occur in many ways as, for instance, in cooks, from swallowing hot food; in seamstresses, from the vibration of the sewing machine against which they lean; in those who use the knitting machine, from the same kind of vibrations; in typewriters, from vibration of the machine against which they lean; and in clerks, from leaning against the desks. I have seen two cases occurring in men. One was a carpenter who leaned heavily against his chisel while smoothing boards. The other was a shoemaker who cut leather while holding it firmly pressed against his stomach. Among the poorer classes, the wearing of old corsets poorly made may have a causative effect, because of inequality of compression that interferes with the circulation of the abdominal wall and reflexly the stomach wall and because of the traumatic effect through a thin abdominal wall on the stomach itself. The prophylaxis of gastric ulcer would seem to demand the warning of anemic patients against all such possibilities.

DR. H. B. FAVILL, Chicago—I think we all agree with the general importance of this subject and its general interest and, also, with the general clinical conditions preceding the trouble. But the most interesting point in regard to it is the active determining factor to be found in the deep ulceration, the underlying factor being acknowledged. I am of the opinion that the recent view advocated, and becoming well established, that there is a lack of hemoglobin, and that this lack of hemoglobin is an essential factor, without which such ulcerations will not take place, is to be kept in mind. It is reasonably well established in a considerable wandering literature that, experimentally, defective hemoglobin can be shown to be an adequate determining cause. Without going further into the point for the sake of argument I only want to say that this bears on the subject of therapeutics enormously. If it is true that defective hemoglobin is the essential element in such ulcers, it certainly is true that the restoration of the hemoglobin is the most essential element in restoring the integrity of the mucous surface. Rectal feeding and rest in bed, then, is not the rational method of dealing with ulcers of the stomach if their genesis is as it is supposed to be. This is all the suggestion I care to make, although the whole subject, viewed from this point, seems pregnant with importance, bearing on the management of the condition.

DR. WILLSON, in closing—I am sorry that in the short time at my disposal I could not make reference to the enormous amount of experimental work that has been done in this line. All of these experiments have led to the conclusion that there is either a local or general anemia present in every case of gastric ulceration, and that when this condition of anemia is corrected there is a marked tendency to heal. There seems also to be no question, clinically, that the great number of cases of gastric ulcer occur in patients who have suffered from an evident anemia, and that this anemia usually assumes the chlorotic type. Many of these ulcers follow hemorrhage of one form or another. Welch has referred to the frequency of cases that occur at or about the menstrual period, to which the name of "menstrual ulcer" has been given. On the other hand, gastric ulcer may occur in persons with a perfectly normal blood count, who seem to be in a state of good bodily nutrition. Moreover, for many years gastric ulcer has been treated without reference to the blood condition, and many cases have been completely cured (to say nothing of those that have cicatrized and escaped recognition until the postmortem examination). I have already shown that these facts call into question the correctness of such sweeping conclusions as those arrived at by Fütterer and others. I do not, therefore, wish to take the ground that gastric ulcers occur only in patients who have an evident blood poverty; or that they heal only when the blood supply has again been perfected. But that when blood poverty is present, whether local or general and whether of quality or quantity, ulceration is likely to occur; and that both the occurrence of the condition, and the likelihood of its healing, bear an intimate though not an invariable relation to the quality and quantity of the pyloric blood supply.