severe headache, injection of conjunctiva, lachrymation, the sensation of constriction of the chest which soon passes off, bloody urine, indicanuria, transitory glycosuria, increase of the daily amount of urine, nervousness and disturbed slumber, a rash, and last and most serious and which is nearly always the final and fatal complication, pneumonia. It is especially dangerous to give the drug when there are kidney complications. When there is high temperature it does no good. With the complication of pneumonia it is absolutely dangerous. Pulmonary hemorrhage is rapidly improved. Prolonged administration causes severe and harmful anemia. If the test for blood or indican in the urine shows either to be present the drug should be discontinued at once. The treatment is of little avail when there is sapremia due to absorption from large cavities that have become infected with pyogenic bacteria. Small dosage with creasote is not beneficial; the only positive results obtained have been from a very liberal administration. Hopefulness led me to think many of my cases cured, but in nearly all, the disease has recurred. It has been impossible to follow all of these cases though a few report to me at stated intervals. I doubt that a single case has thus been cured. In comparison with other drug treatment creasote has given me the only satisfaction, however scant, obtained.

Large dosage with creasote given constantly at the point of toleration is dangerous for the following reasons: After continued administration for a great length of time, the patient becomes very anemic, from which trouble he rarely recovers; secondary infection of healthy lung tissue takes place from detached necrotic tissue producing a rapidly fatal pneumonia. Many of my cases on the large dosage have improved very rapidly and were thought to be nearly well. They have been exhibited to medical friends with much enthusiasm with the hopefulness that the cure would be permanent. Many of these patients would rapidly succumb to pneumonia probably a short time afterward. For a long time I thought the pneumonia to be only an accidental complication but I now feel convinced that the treatment is the cause, due to secondary infection with necrotic tissue as mentioned above. Finally I must state that I believe that no well marked case of tubercle of the lungs recovers with the creasote or any other drug treatment without the very best and most favorable climatic conditions coexisting with the treatment.

ON THE DIAGNOSIS OF GASTRIC ULCER.

Read before the Chicago Medical Society, Oct. 7, 1895.

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A disease that causes no less than 5 per cent. of all deaths and that can only be "approximately" diagnosed in the absence of hematemesis is still deserving of the closest study. The following case is presented as a text for a discussion upon the hitherto known signs of gastric ulcer.

N. H., aged 27, first came under my observation Nov. 29, 1893, through the kindness of Drs. Lyman and Ward, after a profuse hematemesis. Neither the family history of the young woman nor that of her early life presented anything of special note. Having somewhat of a neurotic type of constitution, she nevertheless has always borne the appearance of fairly good health, being well nourished and well developed. She has always been of a studious turn of mind and devoted much time to music and literature. She is fond of outdoor sports and is an expert bicyclist. Except for a slight degree of paleness, one would never look upon her as in any way an invalid. There were no pulmonary or cardiac symptoms. Menstruation began at 15 and has always been regular, painless and scanty. Amenorrhea occurs at times. There is a tendency to constipation. About eight years ago she began to be troubled with dyspepsia. At that time Dr. Birdsall, of New York, treated her for anemia, which treatment she continued irregularly for five years. For her dyspepsia she consulted Dr. Vaughan, of Ann Arbor, quite a while ago, and he assured her then that she did not have gastric ulcer. Several physicians have diagnosed her case at various times as nervous dyspepsia and she has been almost continuously under treatment for this condition. Her appetite had always been good but she learned the necessity of being careful in the selection of her food. Near the cardiac end of the stomach she has had at times a slight gnawing or boring pain which shot through to the back and was felt near the angle of the left scapula. Sometimes after partaking of solid food the pain would become very acute and definitely localized so that she could easily cover it with a half-dollar coin. There was almost always more or less pain in the left hypochondrium, and its site never shifted anteriorly or posteriorly. Sometimes, she says, it feels as though a grape seed or other small, pointed object had gotten fast in the left side of the stomach. In the intervals between the taking of food, she feels as though the stomach were contracting or that its lining were forcibly drawing together. She suffers from occasional headaches, due probably to the anemia, but otherwise she is perfectly free from neuralgic pains. Three or four severe "fainting spells," about a year apart, have caused her considerable alarm. In them she became decidedly cyanosed and remained semi-conscious for a brief time. They always left her very weak. In one of these "spells" she fell completely dazed from off her bicycle. I can obtain no more definite history in regard to these attacks of syncope, as they occurred mostly when she was alone and closely engaged in some occupation. Their after-effects were vouched for by friends and relatives. It never occurred to her to notice the character of the stools immediately after them, as she supposed they were due probably to "biliousness" or "nervous weakness." There is a possibility that they may have been the result of small internal hemorrhages, which gave no other indication than that of mild shock. She does remember, however, that right after these spells the stools were somewhat looser than usual. She has frequent attacks of nausea, and though the appetite is good, food frequently produces a feeling of repugnance. There has never been any vomiting.

On Nov. 27, 1893, a severe hematemesis occurred, the first she had ever had. Members of the family declared it amounted to nearly two quarts of blood, probably an exaggerated estimate made in the excitement of the moment. The blood was described as very dark in color and full of small clots. This so prostrated the patient that she fell to the floor and was put to bed in a dazed condition, nauseated and completely exhausted. Menstruation began upon the following day and still more weakened her physically. The pain in the hypochondrium suddenly became much worse and was experienced most keenly on the left side and under the angle of the left shoulder blade. There was an intolerable headache. The temperature was not elevated and the pulse became subnormal. Just before the hematemesis the bowels had moved very freely, almost to the extent of diarrhea. They were noticeably dark in color. She had just been taking for her dyspepsia ten drops of dilute hydrochloric acid. Dr. Ward saw the case immediately after the hemorrhage and administered ergot, cracked ice, cold drinks and one-sixtieth of a grain of strychnia every four hours. Such was the history of the case related to me when I first saw it two days later, Nov. 29, 1893, at the urgent request of Dr. Ward, who was preparing to leave for California. I found the girl in bed, pale, with an anxious expression and complaining of the sharp burning pain in the stomach. Lying upon either side made the pain more acute. Not knowing what diagnosis had been given, I at once told the parents that, in my opinion, it was a case of gastric ulcer. Absolute quiet in bed was enjoined and all visitors positively forbidden. A drachm of milk and lime water or matzoon was allowed every hour. There was no vomiting when I saw her, so that I had no opportunity of examining the contents of the stomach and the passing of a tube to secure a sample of the gastric juice was entirely out of the question. A week later patient was still very dizzy and nauseated when she injudiciously attempted to sit up in bed. The bowels had moved twice, once normally and once after an enema. Both times they were quite normal in color. The headache continued to be dull and constant. The localized pain in the left hypochondrium did not abate but radiated

up along the left side and behind the sternum. It was also very acute posteriorly. At this time one ounce of matzoon, or milk and lime water, or a little of Leube's beef solution was taken every hour, according to the patient's taste. Three times a day she also took a drachm of the juice of pressed, slightly broiled beef, as well as one-sixtieth of a grain of strychnia. Beyond the dietary mentioned the slightest increase brought on sharp pain, nausea and a tendency to vomit.

On December 7 the strychnia was withdrawn and one drop of Fowler's solution of arsenic administered three times a day.

Up to December 20, when for the first time I permitted the patient to sit up in bed for a few minutes, there was nothing of special importance in the progress of the case. Once or twice a slight excess of nourishment came near precipitating an attack of vomiting. Pain and nausea continued but were very noticeably diminishing. Hemoglobin compound (P. D. & Co.) was added to the treatment to restore the loss caused by the hemorrhage. On December 27 the patient began to dress and recline awhile each day in a large arm chair. On Jan. 4, 1894, she commenced taking solid food in small quantities. There was no nausea now, very slight pain and only an occasional ache in the region of the cardiac end of the stomach. There was still much weakness with some shortness of breath. The strychnia was again added to the treatment. After January 7 she began to go downstairs each day and cautiously resumed her usual occupation. Convalescence was rapid and satisfactory. Carlsbad salts and ferruginous tonics terminated the special medication.

In February the patient went to Florida, spending several weeks there, greatly to her benefit, eating heartily, bathing in salt water and living an outdoor life. She returned April 3, the picture of health and having gained twenty pounds in weight. She was then quite free from all suffering. In August, 1894, she was married, and has since given birth to a healthful child. During pregnancy there was no vomiting of any consequence but considerable nausea. Occasionally now some indiscretion of diet will bring on an attack of nausea and revive the acute lancinating pain which shoots through to the back. It does not last, however, and it quickly disappears with a return to liquid or semi-solid food. There is a marked tendency to constipation, but carelessness in regard to eating precipitates a sharp attack of diarrhea. In all other respects the patient appears and feels perfectly well.

There are two kinds of ulcer of the stomach, the diagnosis of which is either impossible or unimportant. The one is the latent ulcer which runs its course without symptoms, and is recognized merely as a post-mortem curiosity; the other is the ulcer which remains latent until a sudden, rapidly fatal hemorrhage renders its diagnosis of retrospective interest only. As clinicians, the form of ulcer which concerns us most is the variety having more or less definite symptoms. Of this variety there are the *suspected* ulcers, the diagnosis of which can never be more than approximate and the *recognizable* ulcers, of which we can form a reasonably positive diagnosis.

The cardinal symptoms of gastric ulcer are, in the order of their importance, gastrorrhagia (hematemesis), pain of a peculiar sort, dyspepsia, hyperacidity and vomiting. Given all these symptoms in any one case and we may be assured that ulceration of the stomach is present. Of these five cardinal symptoms gastrorrhagia is by far the most important, for until there occurs a hemorrhage, large or small, from the stomach, the other symptoms can only lead us to a suspicion of ulcer. The variable importance attributed to these other symptoms is the ground upon which clinicians differ most in their diagnosis of Cruveilhier's malady. It is extremely desirable that more unanimity of opinion should obtain in regard to the value of these symptoms or that new ones should be discovered which would foster uniformity of diagnoses by reason of their greater exactness. These symptoms arranged in the order of their frequency are epigastric pain, vomiting, indigestion, hyperacidity and gastric hemorrhage. Constipation, anemia, chlorosis, amenorrhea, the red tongue of Niemeyer, the local hyperthermia of Peter and Hayem, the gastric contraction and dilatation of Gerhard and the associated cachexiæ are all so variable as to be of less importance as diagnostic signs than the etiologic factors of age, sex and occupation. I will consider, therefore, for a few brief moments the relative value of the cardinal symptoms of gastric ulcer, namely, the pain, indigestion, hyperacidity, vomiting and gastrorrhagia.

Everybody knows that a dull, lancinating, burning and gnawing pain, strictly localized near the epigastrium, shooting through to the back and appearing with peculiar distinctness near the angle of the left scapula, occurring at times in severe paroxysms, increased usually by food and external pressure, and more or less modified by posture is almost pathognomonic of gastric ulcer. I can not recall a case diagnosed ante-mortem as ulcer, in which pain was entirely absent. It is a marvelously uniform and correspondent symptom. Pain, however, is always an unsatisfactory indication upon which to base a diagnosis, and in gastric ulcer it would probably be one of the least valuable, were it not for its constancy. Its fixed nature and relative constancy are its preëminent characteristics. Even the steady discomfort of gastrodynia can usually be temporarily relieved by a small blister over the site, but not so the peculiar pain of gastric ulcer. I believe, therefore, that its constancy with one or two peculiar features, which it always manifests, raises it to the position of a most valuable diagnostic sign. It is very exceptional for this pain to be continuously absent throughout the entire course of the disease, and a careful study of its character ought, it seems to me, to furnish, when present, the strongest kind of a suspicion of ulcer. Occasionally the pain is modified so as to render a differentiation of ulcer from gastralgia, cancer, chronic gastric catarrh and the gastric crises of Charcot a matter of some difficulty. But if one has an opportunity to observe his case for a time, I think he will be able to determine the cause of the pain. Gastralgia is met with in chlorotic and hysteric females, is independent of the ingestion of food and is not fixed; it is not relieved by vomiting, but is often caused to vanish by external pressure; it is entirely absent in the intervals between the paroxysms and it is benefited more by electricity and general tonics than by the regulation of the diet. Other well-known symptoms usually accompanying cancer, chronic gastric catarrh and the gastric crises of certain organic nervous affections are adequate for a differentiation. It should be remembered, however, that ulcer is always accompanied by chronic catarrhal gastritis. Thus the pain of ulcer during a paroxysm may resemble the pain of gastralgia and in the intervals between the paroxysms, resemble that of chronic catarrh; but in such a dilemma it is safer to conclude that the disease which can give rise to both gastralgic pains and catarrhal soreness is present than that the patient is the victim at one and the same moment of two such dissimilar affections as chronic gastritis and nervous dyspepsia. I do not believe it is possible to diagnose the site of the ulcer, as Brinton teaches, by the location of the pain, by the manner in which it is affected by posture or by the mere ingestion of food. Peripheral pain is never uniformly situated when due to an uncertain, deep-lying lesion, and on the other hand patients assume all sorts of bizarre attitudes in their efforts to get rid of their agony.

Next to pain, vomiting is probably the most fre-

quent symptom of gastric ulcer, but unfortunately it is so commonly associated with other troubles that it is perhaps the least important in this. Its only value here lies in its association with other signs. Unlike pain it alone would never lead us to suspect ulcer. There are certain cases of ulcer in which vomiting is the most marked and distressing symptom, but unless the vomited matter contains blood or it is associated with several other characteristic symptoms, it is a perfectly useless indication of ulcer. It has been said that the vomiting of "mucus or of a thin fluid unmixed with food" is indicative of only chronic catarrhal gastritis, and that "alimentary vomiting" is always suggestive of gastric ulcer. I think I have seen alimentary vomiting more than once in chronic catarrhal gastritis, and moreover, catarrhal gastritis is almost invariably a complication of ulcer. Not infrequently the victim of ulcer experiences an intensely sour taste from the matter vomited, and this leads me to the consideration of the next symptom, hyperacidity.

In regard to the pathogenesis of gastric ulcer, the theory of the self-digestion of the gastric mucous membrane has had, and still has, many advocates. Whether the theory be true or false, it seems to be a fact that the hydrochloric acid of the stomach is in excess in a large percentage of the cases of ulcer. According to Korczynski and Jaworski,[1] it is greatest at the time of hemorrhage. Some declare that the excess of digestive fluid results from the irritative action of the ulcer itself, others that it precedes and originates the ulcer. Riegel, who has studied this symptom most exhaustively, says that the proportion of hydrochloric acid in the stomach of a case of chronic gastric ulcer is 0·4 to 0.6 per cent. as against 0.1 to 0.2 per cent. in health. Ewald's words are: "I regard the demonstration of increased acidity as a marked advance toward the recognition of this condition and it enables us to make an early diagnosis." Hence, hyperacidity, when present, becomes a valuable sign of gastric ulcer. I say advisedly "when present," because it sometimes happens that in the progress of the ulcer the acidity of the gastric juice diminishes to normal or even below normal. This has frequently been demonstrated by Ewald, Ritter and Hirsch and Jaworski. According to Lenhartz the acid may even be deficient in gastric ulcer. After reporting a case in which the percentage of acidity was far below the boundary line, Ewald remarks "that hyperacidity is not an absolute attribute of ulcer of the stomach and that a negative result is accordingly not decisive in establishing a diagnosis."[2] Furthermore, as ulcer is not infrequently associated with cancer, it may happen that the more pronounced manifestations of the ulcer will be accompanied by total absence of hydrochloric acid. Such association of ulcer and cancer occurs, according to Dietrich, in 5 per cent., according to Rosenheim in 8 per cent. of all gastric carcinomas. In these cases hyperacidity may continue with the growth of the cancerous tumor, or it may diminish. Unfortunately, we can not always obtain this valuable support to our diagnosis, the symptom of hyperacidity, even when it may be present. In many cases of ulcer there is no vomiting, not even nausea; and as soon as ulceration is suspected the passing of a tube into the stomach for the purpose of securing a sample of the gastric juice is regarded by most clinicians as too hazardous to pay for the meagre information likely to be obtained. For this reason Leube omits the acid test when he thinks he has a case of gastric ulcer. Germain-Sée disapproves of lavage and the use of the tube in such conditions since Cornillon and Daguet have both reported fatal hemorrhages from such maneuvers.

It has been shown by Schreiber and Pick that catheterization of the stomach, however gently performed, invariably produces an immediate and rapid outpouring of secretion from the gastric glands and that only after many trials does the viscus become tolerant enough to retain during such manipulation its wonted production of digestive fluid. In addition to this it must also be remembered that we can only diagnose hypersecretion, according to Vierordt, when by a rapid and careful procedure at least 200 c.c. of acid gastric secretion are obtained. All of this goes to show that those cases of ulcer in which the symptom of hyperacidity, however valuable it may be, becomes available are not very many. It is of some practical use, however, to notice that the state of the urine is more or less complementary to that of the gastric juice; for with the increase of acidity of the latter there is in the former an increase of specific gravity, diminution in quantity and loss of acidity. Alkalinity of the urine and a decrease or absence of its chlorids are bad prognostic signs.[3] With the increase of hydrochloric acid in the stomach there are also such general symptoms as thirst, heartburn, pain at night and acid vomiting. As a matter of differentiation it is to be remembered that inacidity occurs in gastric cancer, dilatation produced by an ulcer scar at the pylorus, in severe anemia, various fevers and simple nervous dyspepsia with or without gastralgia.

Indigestion on account of the associated chronic gastric catarrh, must obviously be a frequent symptom of gastric ulcer. As the indications of catarrhal dyspepsia are so familiar to all, they need not be detailed here. With all its train of objective and subjective manifestations, indigestion is not an altogether worthless symptom of ulcer, because in the first place it is almost constantly an accompaniment of ulcer and in the second place when constituting the general background of the picture in which appear one or more special symptoms of ulcer, it immensely strengthens the diagnosis. There is nothing peculiar about this indigestion except its persistency and conjunction with the other suspicious signs of gastric ulcer.

I have thus far referred to the more ordinary signs of ulcer, and I fully believe that if they are carefully studied in every case they will afford us more than a mere suspicion of the disease. The symptom, however, which completes the picture and gives precision to the diagnosis in the majority of well-defined cases at least is gastric hemorrhage. Out of seventy-two cases of hematemesis, forty resulted from gastric ulcer, according to Handfield Jones,[4] Lebert noted gastric hemorrhage in four-fifths of his cases, and in three-fifths of them the hematemesis was profuse. Brinton estimates that large hemorrhages occur in about one-third of all cases; according to Müller's analyses it occurs in one-fourth of the cases. Gastrorrhagia is sometimes the only symptom of ulcer, such cases being of the latent variety and quickly terminating fatally. They are the ulcères foudroyantes of the French authors. I believe that gastric hemor-

[1] Deut. med. Zeit., Berlin, April 28, 1892.
[2] Ewald, Diseases of the Stomach, 1894.
[3] Korczynski and Jaworski. Krakow, 1890, quoted by Sajous' Annual.
[4] Med. Chi. Transactions, vol. XLIII.

rhage occurs more often than is generally supposed, but that it is so slight as to pass off unnoticed by way of the bowels. I imagine that something of that sort may have caused the various fainting spells referred to in the case which I have reported.

Some authorities state that in gastric ulcer a hemorrhage sometimes occurs regularly as vicarious menstruation. Aside from their being but little foundation for such an opinion, I believe it would be dangerous for the patient and confusing to the diagnosis to so regard such hemorrhages. The irregular catamenia, especially the amenorrhea so common in these cases, are not the cause of the hemorrhages so much as the result of the debilitated constitution brought about by the loss of blood. A periodic hemorrhage in gastric ulcer is not menstrual but rather a gastrorrhagia accompanying the ulcer and provoked by the monthly disturbance of the system. If the hemorrhage is large and recent the blood will be bright red in color, alkaline, fluid and mixed with food and mucus. More frequently, however, it is retained long enough in the stomach to be acted upon by the gastric juice. It will then be more or less clotted, having the appearance of coffee grounds, changed in color to dark brown by the changing of hemoglobin into hematin, acid, unaerated and minutely intermingled with particles of food and sour mucus. Hematemesis occurs in many diseases and must always and especially be differentiated from hemoptysis.

In conclusion then, I believe that gastric ulcer may be strongly suspected where there is the peculiar pain already described and hyperacidity; and if to these be added gastrorrhagia, the diagnosis may be made with gratifying certainty. I have purposely refrained from considering the indications of the site of the ulcer, which in cases of perforation may be surgically important. My only object has been to emphasize and assign the proper valuation to each of the cardinal symptoms of gastric ulcer, symptoms upon which alone anything like a positive diagnosis may be based.

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OBSERVATIONS AND STATISTICS UPON THE USE OF ANTITOXIN IN ONE HUNDRED CASES OF DIPHTHERIA.

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The following observations, statistics and histories are offered as a contribution toward clearing up obscure and unsettled points relative to diphtheria and antitoxin.

It has long since been experimentally proven that the Klebs-Löffler bacillus produces a specific toxin giving rise to all the classic signs of diphtheria. The pseudo-diphtheria bacillus is supposed to be a non-virulent, attenuated or modified form of the former. The latter, the streptococcus longus, streptococcus pyogenes and staphylococcus are associated with the Löffler bacillus and cause pathogenic conditions, respecting which there is much to learn. For instance, such cases of croup, necessitating even intubation, in which these non-specific germs only could be grown in spite of repeated culture trials, have been relegated to the list of anomalous cases. Such an explanation, however, no longer satisfies the scientific world. More extended biologic research and study of serum-therapy will doubtless change the nomenclature of a disease having such a multiple genesis and pathology. The minuter chemico-physiologic reactions of the diphtheria toxin and antitoxin upon the human cell and organism still require elucidation. Before proceeding to the tabulation of cases as observed by me in eight weeks' service in the health department, I will formulate the points that particularly impressed themselves upon my attention and later emphasize them by a recital of interesting histories. They are:

1. The marvelously rapid improvement, especially in the laryngeal or most dangerous form of the disease when antitoxin is properly administered, viz., early enough, in large enough doses and in frequently repeated doses in severe cases where but little improvement is noted within eighteen hours.

2. The necessity for early cultural diagnosis.

3. The clinical relation of the pseudo-diphtheria bacillus to the Klebs-Löffler bacillus and their mutual interchangeable attributes, such as virulence, benignancy, transmission, etc.

4. Bacterial, aborted or modified diphtheria without clinical manifestation.

5. Persistence of the Klebs-Löffler bacilli in the throats of these subjects in spite of rigorously applied antiseptic treatment.

6. Menace to the community as contagion bearers of these subjects; hence the need of isolating them.

7. Relative absence of post-diphtheritic paralysis despite the severe character of the epidemic.

8. Period of, and positive and partial immunity conferred by the use of antitoxin.

9. Contraction of diphtheria after immunization due to the tardy use of antitoxin.

10. Rashes and sequelæ consequent to the use of antitoxin.

11. Failure to demonstrate the Löffler bacillus in some undoubted cases of diphtheria.

These inferences have been reached by actual observation and care of the greater number of cases comprised in the following table:

Cases visited or seen, 137; curative antitoxin doses given, 102; curative antitoxin doses given by me and assistants, 72; curative antitoxin doses given by other physicians in my district, 30; recoveries after antitoxin, 95; deaths after antitoxin, 7; physicians asking treatment for their patients, 9; physicians giving this treatment, 20; cultures made for 250; laryngeal cases, 50; mild laryngeal (seen within first sixty hours) cases, 28; severe stenotic cases, 22; cases immunized, 166; cases completely protected, 150; partially protected, 16; bacterial not clinical cases, 28; deaths twenty-four hours after injection, 3; deaths later, 2; deaths in which antitoxin was not used, 5; cases of paralysis within twenty-four hours after use of antitoxin, 2.

	1st day.	2d day.	3d day.	4th day.	Later.	Unknown day.
Number of cases injected.	16	34	21	10	14	7
Recovered	16	33	20	8	11	7
Died	0	1	1	2	3	0

Intubations by others, 4; intubations by me, 1; total 5; tracheotomies, 1; cases of rashes consequent to use of antitoxin, 12.

The death, as reported after the use of antitoxin upon the second day of the disease, was in a laryngeal case of probably longer duration than reported. That reported as having received an injection upon the third day, received, to my knowledge, too small an initial and second dose, and besides had not been freely enough stimulated. One of the cases of paralysis was in my practice and was, I think, due to the profound toxemia and could not be ascribed to the action of the antitoxin, for the diagnosis was not made until the fifth day of the disease, or until stenosis set in and