Post-Mortem Examination.—There was found to be great distention of the lower two feet and a half of the small intestine and of the large intestine as far as the junction of the descending colon and sigmoid flexure, but there was no stricture at this point. The sigmoid flexure and the rectum were empty. There were many ulcers laying bare the muscular coat of the large intestine and of the lower few inches of the small intestine. There were no tubercles.

In discussing the treatment of the first two cases one has to bear in mind the difficulty in diagnosing the condition during life. If there is good reason to suspect it, the chief means to be adopted are kneading or massage of the lower part of the abdomen, the exhibition of laxatives, such as large and repeated doses of olive oil or castor oil, and copious enemata administered by the long tube. I agree with Dr. Bristowe that the use of drastic purgatives should be avoided.

One must admit the probability that such measures as I suggest may be without avail, and that the urgency and obstinacy of the symptoms may call for abdominal exploration. Provided that the diagnosis be established (and this, of course, is the difficulty), the best means to adopt would be to expose the distended sigmoid flexure by an incision in the middle line below the umbilicus, to rectify any misplacement, and to endeavor to force through the anus part, at least, of the fecal contents, or, failing in this, to make an artificial anus.

GASTRIC ULCER.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY CHARLES G. STOCKTON, M.D.,

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GENTLEMEN,—The patient is a physician, forty-four years old, and since the age of twenty-one he has practised his profession. His father died at seventy of what is believed to have been gastric ulcer of that form which has been described to you under the name of round or perforating ulcer or the ulcer of Cruveilhier. The patient's mother is living, at the age of seventy. He has one brother who suffers from indigestion, and four sisters, one of whom has some heart-difficulty and two have slight dyspepsia. When a student at this college, the patient suffered intensely from indigestion. He subsequently practised medicine, and, although he has been active and able to work, he has been rather delicate and has suffered with painful dyspepsia during all this time. About four years ago he had a very severe hæmatemesis, while suffering from pain and distress in his stomach, with vomiting. Subsequent to the hæmatemesis he vomited mucus and had great pain in the epigastrium and right shoulder. After this severe attack he became better than he had been for some years, but from time to time his trouble has recurred, with slight vomiting of blood and mucus and with pain, inability to eat, loss of sleep, and it became necessary for him to take anodynes. He came to me about three weeks ago suffering intensely from pain in the epigastrium and right shoulder; he was vomiting persistently, the vomited matter containing large amounts of mucus. Three or four months ago he had vomited a small amount of blood. He was digesting little, and was very much excited from his long-continued pain, and his nervous condition was not improved by the use of morphine, of which he was taking one-quarter grain hypodermically every six hours. Ice-cream he had found to be about the only nourishment that he could retain, and often his stomach would not tolerate even that.

The diagnosis had been made of gastric ulcer and dilatation, the dilatation being supposed to depend upon contraction of the pylorus which often accompanies the cicatricial change of ulceration. On examination, there was a moderate succussion-sound to be heard, and by the usual methods of percussion and bimanual palpation I found that the lower border of the stomach came about to the umbilicus. Although this could not be considered to indicate an actual dilatation, it did represent a relative dilatation, since the doctor had not taken food in any quantity for a long time. The stomach, though relaxed, had sufficient power to expel the mucus which was in it, and which, to my mind, proved that there was present gastric catarrh, the result mainly of excessive vomiting.

Now, on making light percussion, with the patient on his back and without using the pleximeter finger, I am compelled to say that the stomach is dilated, since the lower border reaches at least one and a half inches below the umbilicus. Not only is the percussion-note clear to this point, but the succussion-sound also. At a point half-way between the ensiform cartilage and the umbilicus and two inches to the right of the median line there were formerly induration and great tenderness, but these signs have disappeared. The patient is taking more food now than before, and the signs of dilatation have probably developed as a result. For breakfast this morning he took beefsteak, farina pudding, bread and butter, and a glass of milk, and he has since had a second half-pint of milk. He suffers no pain nor tenderness, he is rapidly gaining flesh and color, and he is feeling well. He has taken no morphine since his entrance here, three weeks ago.

We must admit the diagnosis of gastric ulcer with dilatation. The patient was *in extremis* when he entered, suffering intense pain, almost starving,—for he had been obliged to nourish himself by the rectum when it was not possible for him to retain a little ice-cream. Let us see if we can account for his condition and for his improvement.

The round or perforating ulcer, which was beautifully described by Cruveilhier many years ago, has been accounted for by some investigators as a result of thrombosis or embolism in some small vessel near the pylorus, for the lesion usually is found near the pylorus, along the lesser curvature, and on the posterior wall of the stomach, though it may elect other points. To my thinking, this theory is not satisfactory. I believe the ulcer is to be explained on the basis of some neuropathy, like that of hæmatoma auris in the insane or that of symmetrical gangrene in young people,—Reynaud's disease. Again, if herpes selects certain points for its appearance on account of some

neuropathy, I think we may similarly explain gastric ulcer, which selects some point of the stomach where there is a local weakness, as due to a devitalization, so to speak, of the tissues, which are then attacked by the gastric juice and digested, leaving the characteristic ulcer with abrupt walls, and having, therefore, the peculiar punched-out appearance. The ulcer does not extend to surrounding tissues, it may be present without catarrh of the stomach, and it may occur singly or in groups of two or three. Moreover, the neuropathic theory is probable, because, as a rule, gastric ulcer occurs in neurotics, and especially in young females; although occasionally it is seen later in life, as in this man's case and in that of his father. For these reasons I think it is fair to suppose that the disease is due to a nervous defect rather than to a thrombus or an embolus. Besides, we do not usually find evidences of thrombosis occurring in other parts of the body coincidently with the ulcer; and it seems to me extraordinary that thrombosis or embolism should occur so uniformly in certain individuals of a certain age, at a certain spot, and produce such constant results.

The doctor has suffered from an excess of hydrochloric acid for many years, and this history belongs to gastric ulcer for two reasons: and first, because any distinct local irritation in the stomach which does not cause catarrh of the stomach usually excites an excessive secretion of hydrochloric acid. The passage of the stomach-tube, the use of the gastric electrode, or almost anything which will excite the mucous membrane, will do this. But I am satisfied that the hydrochloric acid is there for other reasons, for when the ulcer is healed and there is no longer any irritation from it you will still find the excess of hydrochloric acid in such patients. I believe that gastric ulcer is almost uniformly associated with that form of dyspepsia which now goes under the name of hyperhydrochloric acid dyspepsia. This is a form of indigestion which gave a great deal of trouble before we understood its nature. To-day it is comparatively easy to control.

When the doctor has not been suffering from the hemorrhage which the ulcer occasioned and the catarrh to which the vomiting gave rise, he has suffered from hyperhydrochloric acid dyspepsia. He is now in perfect comfort, in spite of the abrupt stoppage of the morphine.

Believing that there was catarrh besides the ulcer, the catarrh being due to vomiting, I thought it best to give the stomach complete rest for a time, and the patient was nourished by the rectum for two or three days,—not on account of the ulcer, but because of the catarrh. If he had been vomiting blood without signs of catarrh, I should still have given his stomach rest for the sake of the ulcer,—in other words,

to prevent the hemorrhage. He was given at the same time local sedatives and antacids, which I thought would soothe the stomach and neutralize the excess of hydrochloric acid, which was not simply surmised, but was demonstrated by an examination of the gastric contents. The mixture was

℞ Cerii oxalatis, 10;
Bismuthi subcarbonatis, 20;
Magnesii carbonatis levis, 40.
M. et S.—Half to one teaspoonful every two to four hours.

Under the effect of the magnesia his bowels, which had been constipated, soon became loosened. The mixture was then modified to

℞ Cerii oxalatis, 10;
Bismuthi subcarbonatis, 20;
Cretæ preparatæ, 20;
Carbonis ligni pulv., 10.
M. et S.—Half-teaspoonful every two to four hours.

This had a quieting effect on the bowels and corrected the previous prescription. One might have given, in place of this, five or ten grains of black oxide of manganese (C. P.), either alone or in combination with bismuth and cerium. These agents are gastric sedatives, while chalk and magnesia are antacids. Using these mixtures,—one when the bowels were constipated and the other when they became loose,—the evacuations were regulated, the stomach became quiet, and his pain passed off so that he got along without morphine, although he was still very nervous.

Then he was fed, and the feeding once begun was continued regularly, steadily, frequently, and bountifully. He was given peptonized milk every two hours, first four ounces, then six, then eight. When you begin to feed a patient with gastric ulcer, you must feed him freely, because the presence of even a little food will stimulate the secretion of hydrochloric acid in excess, and thus cause greater irritation of the ulcer. When the feeding is begun the stomach must not be allowed to become empty. After having fed the patient on peptonized milk for a while, I began giving him meats and egg albumen. These were given rather early to this patient, and were continued at short intervals because the albuminoids are very soon changed by the gastric juice into an acid peptone, with a proportionate decrease of hydrochloric acid and thereby diminution of the irritation to the stomach. Theoretically this practice is right, and practically it is right sometimes, as it was in this case; but you must remember that these patients have a tendency to the

over-secretion of hydrochloric acid, and the stimulation of the secretion by the albuminoid food is not always properly counterbalanced by the formation of peptone.

After peptonized milk, I began with eggs in this case, and later added meats, and when I was satisfied that the hydrochloric acid secretion was being controlled and that the ulcer was healed, I began giving starches. Now, some of the French advise starchy food to the exclusion of albuminoids in gastric ulcer, and for the reason that with meats they found the acid secretion increased. I find that while starches do stimulate less the secretion of hydrochloric acid, they are not well digested, and consequently they give rise to fermentation, which, in turn, causes distress and the stomach has no rest. For their digestion starches require a faintly acid or alkaline medium. I believe, therefore, in giving starches late, not until the excess of hydrochloric acid has been controlled. The patient has now come to the point where quite a general diet is taken, and without producing distress. He still has occasionally pain in the right shoulder, but it does not arise from the stomach; it is simply a habit of pain. An analogy may be drawn from trifacial neuralgia produced by a decayed tooth which may be awakened by exposure to cold weeks or months after the removal of the tooth.

For the future we must endeavor to control the hydrochloric acid excess and to prevent the return of the peculiar condition which gives rise to the ulcer. To meet the former indication, he must have recourse to a diet which is most likely to agree with him, which will give him strength and yet not over-stimulate the secretion of hydrochloric acid. That is to say, his diet must be much like that on which he is at present, amplified somewhat, but he must deny himself fruits, which, by fermenting, are apt to cause disturbance, and other substances whose digestion is difficult or which excite the stomach, such as condiments and sauces. This diet, which maintains the quality of the blood and the tonicity of the nervous system and which keeps the body as a whole well nourished, meets in part the second indication also. In addition the patient should be careful not to overdo, should take the proper amount of rest, and if, in spite of all precautions, his blood falls below par,—that is, if he suffers from anæmia,—this also should receive appropriate treatment.

Now, there is another aspect of this case, for the doctor has also a dilatation of the stomach. What brings that about? It is due sometimes to stenosis of the pylorus. We once thought this the usual cause, but we know now that it is comparatively an infrequent cause of dilatation

in proportion to the whole number of cases. I do not think that stenosis, if present at all, is marked in this case, for the patient empties the stomach fairly well. You may suggest that his dilatation is probably owing to relaxation of the stomach, the result of his neurotic condition. This is possible, but there is another way in which I prefer to account for it here. Anything that gives rise to local irritation in the neighborhood of the pylorus tends to produce a spasmodic closure of the pylorus. So, any food coming against the sensitive area of ulceration would tend to shut up the pylorus and in this way produce virtually a stenosis, that we may call a functional stenosis. I think it is in this way that his dilatation has been caused, for it has long given him more or less pain for his stomach to empty itself. Now if we keep the contents of the stomach bland and unirritating and not over-acid, it will be possible for them to pass downward without causing much irritation, and if we keep the stomach free from ulceration it will be able to empty itself comfortably. The use of internal faradization, lavage, or massage will probably not be called for here.

The case is of great interest in showing the heredity of the disease, by occurring in a man instead of in a woman, and in one of advanced years instead of in a young person. It illustrates the tendency of the disease to continue for a long time, the tendency of dilatation to occur with ulcer, and I think it shows also the tendency of dilatation to occur as a result of irritation at the pylorus.

FATTY DEGENERATION OF THE HEART ASSOCIATED WITH A SLIGHT DILATATION.

CLINICAL LECTURE DELIVERED AT THE PHILADELPHIA HOSPITAL.

BY J. M. ANDERS, M.D.,

Professor of Medicine in the Medico-Chirurgical College, and Visiting Physician to the Philadelphia Hospital, etc.

J. H., white, Irish, aged fifty-five years; occupation when young, a farmer, later, a blacksmith. Family history: father died of typhoid fever; mother died at the age of ninety-seven of natural causes. Has one brother, in Scotland, living and well; one sister, well. No family history of rheumatism. Was always healthy as a child. At blacksmithing he worked thirty years, and during that time he was in perfect health excepting for colds, which were generally very severe and caused much coughing. When younger he drank pretty heavily, and then became quiet and steady. He states that his sprees used to cure his colds. Has had five children, three living and two dead. The three living children have no heart-trouble. Has never had rheumatism; had malaria in 1888, lasting several months, after which he had swollen feet for two weeks; since that time, if he catches cold, they always become slightly swollen. Denies emphatically all venereal history. During the spring and summer of 1887 was in a Pittsburg hospital for five months. Symptoms then were palpitation, dyspnœa, slight œdema of legs, anginoid attacks, and feeling of faintness. Was admitted for the first time to medical ward of the Philadelphia Hospital, June 1, 1889; condition then was as follows:

Dyspnœa on slight exertion, especially when going up-stairs or carrying weights, with marked cardiac palpitation, and the return of anginoid attacks, the pain radiating towards the right shoulder; great depression, pulse 50 and irregular; on exertion heart's action showed gallop rhythm. Remained in house until September; then went out, and returned in November. Left again April 15, 1890, and returned August 7, 1890. After admission, was in bed for five weeks; symp-

toms: great dyspnœa, palpitation, and considerable œdema of feet and legs. Physical signs on admission were those of slightly dilated right heart and probably fatty heart. Impulse very feeble, and area of impulse not increased. Pulse was slow, exceedingly weak, irregular, and intermittent. Dulness extended towards the right, a little beyond the normal boundary-line. Œdema disappeared and condition of heart slightly improved; no anginoid attacks since date of last admission. Present condition, December 13: impulse scarcely perceptible, though it can be distinctly felt. Area of percussion dulness slightly lessened, owing to moderate emphysema. On right side percussion dulness extends slightly beyond normal limit; the upper boundary-line is in the fourth interspace. On auscultation very weak, short, distant, first sound; no murmur present, heart-action very irregular and sometimes intermittent.

The history of this case, together with the present signs and symptoms, points to the probable existence of fatty degeneration with occasional slight dilatation. The diagnosis of fatty degeneration, however, can never be made to a certainty. You should examine carefully into the different elements of causation in each case. Let us here glance hastily at the different varieties of this affection and at their well-recognized causes.

Fatty degeneration is frequently associated with valvular disease, especially in those cases in which the walls have become hypertrophied. Now, the condition under which fatty degeneration is developed and the reasons for these changes have been given you when discussing the subject of dilatation. But fatty degeneration also attacks the normal heart, and it is to this variety that the present case apparently belongs, since you will recollect that neither valvular disease nor hypertrophy has been discovered.

The causes, for practical purposes, are divided into two classes,—namely, general and local. Among general causes I may mention chronic alcoholism, profound anæmia, certain poisons, such as phosphorus, arsenic, etc., wasting diseases, failing nutrition of old age, prolonged fevers producing parenchymatous degeneration, etc. Of the local causes the most important are pericarditis and sclerosis of the coronary arteries. In this connection I would have you remember that disease of the coronary arteries is very generally associated with general atheromatous changes, hence its existence or non-existence is in the majority of instances to be judged of by the condition of the radials. From the history of this case it will be seen that three general causes must have been at work,—namely, general atheroma, chronic alcoholism,

and anæmia. Most probably one local cause has been operative,—namely, atheroma of the coronary arteries,—for the reasons which I have before mentioned. Now, malarial anæmia is very apt to lead to fatty degeneration of the heart, according to my own observation.

Apart from the history of the case, we have among the most reliable points on which to base a diagnosis a permanently feeble impulse, a weak, irregular, and at times intermittent pulse,—either very rapid or very slow,—a fatty arcus senilis, the presence of angina pectoris, or, as in this case, of anginoid attacks; at times œdema of the feet, Cheyne-Stokes breathing, and pseudo-apoplectic attacks; additionally we have the physical signs which we previously mentioned in detail.

When chlorosis or chronic anæmia has preceded the coming on of the manifestations of this disease, there ought to be no difficulty about the diagnosis. The latter disease being associated in the present case corroborates strongly our diagnosis. You should always distinguish fatty degeneration from fatty overgrowth of the heart, when possible. Fatty accumulations around the heart occur in very stout persons. Dyspnœa is apt to be constant, though much increased on exertion; cough and asthma are common. The pulse is rapid, weak, occasionally slow; it becomes irregular and intermittent at times, and in my opinion this is evidence of the fact that infiltration has led to fatty degeneration of the muscular fibres. The physical signs do not differ materially from those found in fatty degeneration. The area of percussion dulness, however, is apt to be somewhat increased. Syncope frequently occurs.

The prognosis of fatty degeneration of the heart is, as a whole, very grave, especially in those instances in which the disease is associated with advanced atheroma; it is less grave, on the other hand, when due to anæmia or chlorosis, causes that are frequently in part or wholly removable. Hence your prognosis should be guardedly favorable under these circumstances.

Treatment.—Endeavor to remove the cause. Another leading object of treatment is to sustain the integrity of the heart-muscle. The remedies best calculated for this purpose are iron, arsenic, and strychnine. Such patients should lead very quiet lives, making no straining efforts whatsoever; especially is straining at stool to be avoided. In most other respects the treatment is the same as that indicated for cardiac dilatation. Where digitalis, however, is indicated on account of associated dilatation, it must be given very cautiously.