in a raised blood sugar curve, although the fasting blood sugar may be normal.

When is diabetes mellitus diagnosed in association with hyperthyroidism?

John, in his discussion on this subject, says that no single blood sugar curve is of value in the diagnosis of diabetes. “The diagnosis of diabetes in hyperthyroidism can only be made after long study of the patient, determining the persistence of defective carbohydrate metabolism.” Joslin and Lahey consider “a true diabetic glycosuric as one who has at least some of the characteristic symptoms of diabetes, and shows hyperglycaemia above 0.13% before, or above 0.16% after a meal and with glycosuria varying with diet,” but with this definition, there would be too many cures of diabetes following operation and so they “raised the standard for the diagnosis of diabetes in hyperthyroidism to a blood sugar of 0.15% fasting or 0.2% or more after meals, in addition to glycosuria.”

A convenient definition of diabetes mellitus is, “that condition in which there is hyperglycaemia which is not transient, associated with glycosuria.” The patient has a raised resting blood sugar, a rising blood sugar curve above normal limits, which falls slowly within three hours after taking the glucose.

Using this definition one may classify hyperthyroidism with glycosuria as:

1. Those cases with a lowered renal threshold; i.e., the resting blood sugar is normal, the blood sugar curve rises no higher than normal limits but glycosuria is present.
2. Those cases with a mild, true diabetes; the blood sugar curve is slightly above normal and the resting blood sugar raised; glycosuria is present.
3. Those cases with a severe, true diabetes, the resting curve is markedly raised and the glycosuria is heavy; there are other signs and symptoms of diabetes mellitus present.

Diabetes may occur with primary or secondary hyperthyroidism.

Treatment—

Insulin, though given in large quantities, has little effect in diminishing glycosuria.

Iodine treatment is of little benefit, and the patient’s condition rapidly becomes worse. From the literature, it is apparent that the only treatment is surgical.

Careful pre-operative and post-operative treatment is essential for the success of the operation. Insulin, insulin and carbohydrate must be given before operation, and as soon as possible after it. Large quantities of saline and glucose are of value in averting post-operative crises, as diabetic coma and “thyroid storms.”

Joslin and Lahey have found that the mortality rate has been lowered by doing two stage operations and more conservative operative approach if the mortality is to be kept low. As Dr. Lahey puts it: “The important warning I would like to utter to medical men, is that they should urge immediate surgery in cases of diabetes and hyperthyroidism.”

“The complication of hyperthyroidism and diabetes is a serious one and will be attended with high mortality unless these cases are approached with care.”

REFERENCES:

Annals of Surgery, October, 1934.
Bulletin, Johns Hopkins Hospital, September, 1934.
Lancet, November 4th, 1933.
American Journal Medical Sciences, July, 1928.
Journal American Medical Association, August, 1932.
American Journal Medical Sciences, December, 1934.

A CASE OF SYPHILIS OF THE STOMACH

MAX SEGAL and WERNER WEINBERG.

An unusual case was recently admitted to Ward 21 under Dr. Bloom. The features of this case were so interesting both from a diagnostic and clinical viewpoint, that a preliminary report was considered to be warranted.

The history is as follows:

G. M., age 15, scholar, admitted 10/8/36.

Complaint—

(1) Pain in Stomach.
(2) Severe vomiting.
(3) Extreme loss of weight.

History of present condition—

Until 3 months before admission the patient was perfectly well and attended school normally. Pains then suddenly commenced in the epigastrium on taking food of any nature, liquid or solid. This pain commenced 10-30 minutes after every meal, followed by severe vomiting. This vomiting was not preceded by any nausea. Gaseous eructations were frequent, but there was no flatulence. The pain was knife-like in character, did not radiate, was relieved by the vomiting, and
was absent if he abstained from food. This vomiting never occurred during sleep, was projectile and consisted of the food taken during the preceding meal. It contained no "coffee grounds" or fresh blood.

This state of affairs continued for 3 months during which period the patient lost 28 lbs. At O.P.D., 10 days previous to admission, he was given an alkaline mixture which, he states, occasionally relieved the pain and the vomiting unless he was constipated when the vomiting recommenced.

Appetite was fairly good but the fear of vomiting kept him off food during the period of his complaint. The bowels were constipated during the previous three months, acted once in 3 days only on taking a purgative and the stools were of a dark green colour. There was no occult blood in the faeces.

Past History—
Measles, mumps and whooping cough as a child. Mastoiditis, 1935. Was operated on for this and after the operation developed pneumonia, from which he recovered completely.

Habits—
Plenty of exercise before complaint began. Tobacco, nil.

Family History—Parents both alive. Father has phthisis. 4 brothers and 1 sister alive and well. 2 sisters died of infantile alimentary trouble (exact disease not known) at the age of 8 months and 13 months respectively. The mother states that these had been perfectly normal children. The mother has never had any miscarriages.

Physical Examination—
On admission to the ward, patient looked very pale, emaciated and had a senile facies. Weight 83 lbs., Temperature 97°, Pulse 96, Respiration 24, B.P. 80/40.

Examination of abdomen—
On palpation there was voluntary guarding and tenderness in the epigastrium. Liver and spleen were not enlarged. No tumour was detected in the abdomen.

Examination of other systems revealed nothing abnormal.

Laboratory Findings—
Wasserman tests in the blood, + + strongly positive. (It must be noted that this WR. was taken to confirm the finding of the patient's own doctor whose note to the O.P.D. reported a strongly positive WR. on a previous occasion).

Urine Analysis—
At O.P.D. albumin and acetone were found. In the ward, one week later no abnormal constituents were detected.

Faeces—
No occult blood.

Blood Count—
Rbc., 5,240,000; Hb., 95%; C.I., 0.98. White cell count, 8,400.

Differential Count—
Neutrophiles, 78%; Lymphocytes, 21%; Eosinophiles, 0.5%; Large Mononuclears, 0.5%; Basophiles, 0.

Blood Urea—
21 mgs. per 100 cc. of blood.

X-Ray Reports—
12.8.36—The stomach is somewhat dilated and pylorospasm is present. A small niche is shown on the lesser curvature in the antral portion of the stomach which is suggestive of an ulcer here. The duodenum is normal except for a mild degree of ileus. 3 hour examination shows 1/2 the amount of barium passed through and a narrowed pylorus again shown.

17.8.36—Very narrow pylorus is outlined and there is very definite delay in emptying of the stomach. No ulcer crater is visible. 6 hours after the meal the stomach is still 3/4 full of barium, the normal limit for emptying being 6 hours. This indicates a marked obstruction at the pylorus. 24 hours after the meal; there is a 24 hour retention in the stomach, the patient having vomited during this time. Remainder of the barium has passed to the colon normally.

Gastric Analysis—
This was attempted on two occasions, on both of which, though the tube entered the stomach, it was vomited up by the patient.

Differential Diagnosis—
At the O.P.D, the following conditions were discussed in order of likelihood:
1. Gastric ulcer.
2. Pyloric obstruction due to either gastric ulcer or neoplasm.
3. Pylorospasm.
5. Chronic duodenal ileus.
6. Acidotic vomiting.
7. Worm in the stomach.
After the X-Ray reports were received, the diagnosis was narrowed down to:

(1) Gastric ulcer with obstruction.
(2) Pylorospasm.
(3) Chronic duodenal ileus.

**Treatment of Case**

Patient was first given Mist. Gent. Alk. 5ss. t.d.s. and for first day or two remained on full diet. During this time there was no improvement. Similarly on a Sippy's diet for 7 days there was no remission of vomiting. It was then decided to give the patient a course of antisyphilitic treatment together with a modified Sippy's diet. This was commenced on the 21/8/36 with Pot. Iod. V.D.C. No. 1, 5ss. t.d.s., N.A.B. intravenously commencing with 0.15 G. and Bismetal 1 M.I. 5cc. at weekly intervals. With this treatment, almost instantaneous improvement occurred. The vomiting stopped, the patient ate heartily and within 4 days (25/8/36) patient had gained 4½ lbs. His weight on the 14/9/36 was 96 lbs., a gain of 13 lbs. above his weight on admission. Today the patient has no complaints at all, no tenderness in his abdomen, looks very well, and is active about the ward.

**Syphilis of the Stomach**

A review of the literature reveals that, clinically, syphilis of the stomach is not as infrequent as its rare discovery at autopsy would indicate. At autopsy, Singer and Meyer found no cases of gastric syphilis in 5,000 instances with almost 10% syphilitics, and Symmers found, in 4,880 autopsies with 314 syphilitics, only 1 case of gastric syphilis. At autopsy the findings are probably modified by previous antisyphilitic treatment, the difficulty of finding the spirochaetes in such lesions as are present and by the fact that in proved syphilis as Windholz demonstrated after a study of the autopsies in Vienna, that changes in the stomach of syphilitics are not more frequent that in non-syphilitics. Hence there is a great likelihood that many cases are overlooked.

Clinically, the features of gastric syphilis are varied. A study of cases described by Singer, Meyer, Hartwell, Eustermann, Einhorn, Morton and Coelho and other authors show that there are no characteristic symptoms or findings which are pathognomonic of syphilis of the stomach. All authors, however, agree that in a case with the following features, a clinical diagnosis of gastric syphilis can be made:

1. Symptoms simulating any gastric disease, particularly gastric ulceration and neoplasm.
2. WR. + positive.
3. Gastric anacidity or markedly reduced HCl content.
4. Less marked cachexia, emaciation and anaemia than would be found with the radiographic changes due to lesions other than those of syphilis.
5. Improvement after antisyphilitic treatment as opposed to non-improvement with other medical measures.
6. According to some authors e.g., Boehm rapid onset of the condition.

X-Rays do not furnish definite evidence of gastric syphilis (Groedel and Haudeck), though Hartwell states that an X-Ray picture showing marked deformity of the stomach, particularly of the dumb-bell type with an abrupt margin between healthy and diseased portions of the stomach would give a certain amount of evidence of syphilis of the stomach. Most authors, however, do not rely on the X-Ray pictures.

The same clinical picture is also found in the much rarer cases of congenital syphilis of the stomach. In this respect, it is interesting to note that Huber described a case of congenital syphilis of the stomach in an 8½ years old boy, the features being almost exactly similar to those of the case presented in this paper.

**Discussion**

From the above, the diagnosis in the case we have reported would appear to be syphilis of the stomach. The reasons for this are:
1. The history indicated a gastric lesion.
2. The X-Ray picture showed a pyloric obstruction.
3. The WR. was positive.
4. The response to antisyphilitic treatment was instantaneous and marked.
5. There was no response to other therapeutic measures.

A question which arose was, whether this was an acquired or a congenital condition. The WR. of the mother was found to be negative. She also gave no history of any luetic infection. Unfortunately, efforts to obtain the WR. of the father were not successful. As the patient, moreover, presented none of the stigmata of congenital syphilis, the view taken is that this is a case of acquired gastric syphilis.
In conclusion, we would like to suggest that in all gastric cases not responding to treatment, a WR. be done, as this may throw light on the condition. Perhaps we may go further in suggesting that in all gastric cases the WR. be routine, as well as a study of the gastric wall for spirochaetes in gastric cases coming to post mortem, except, of course, where the diagnosis is obvious.

**Acknowledgments**—

We are very much indebted to Dr. A. Bloom for the permission to publish this case, and to Dr. P. Menof for the interest which he has taken in this work.

**LITERATURE:**