The kidneys weighed 300 grams and were frankly negative macroscopically and microscopically. In the bladder just above the trigonum there was a definite patch of hemorrhagic cystitis, and I presume that was the source of the blood in the urine. The prostate, seminal vesicles and testes were negative.

DR. CABOT: Were there any abscesses in the kidneys?

DR. RICHARDSON: No. We obtained a profuse growth of the staphylococcus from the heart blood.

DR. CABOT: Would you hazard a guess as to where the process entered or started?

DR. RICHARDSON: I always do, and I think you take the other one. It is quite difficult in this case to know whether to think of his arms first, or whether to think, as some do, that these processes come by the genitourinary tract or by other roads. He has a cystitis. I think on the whole his arms came first.

DR. CABOT: It is not a clear case for any one place. The lungs certainly were secondary?

DR. RICHARDSON: One would think so. The whole thing inside looks secondary.

DR. CABOT: It is interesting that apparently he did not say a word about his arms. He was complaining about his chest.

DR. RICHARDSON: Against the arms as a starting point, of course, is the fact that the cellulitis seemed to be deeply seated.

DR. YOUNG: I do not believe it started in the bladder. I think the absorption from the urinary bladder is very slight.

DR. CABOT: In general this organism starts in the skin or subcutaneous tissue as often as anywhere?

DR. RICHARDSON: Yes.

A PHYSICIAN: The symptoms in his arms are not referred to until five days after his pulmonary symptoms.

DR. CABOT: Yes, that sticks in my crop a good deal.

Therapeutically we do not know anything to do for these things. People recover from slighter degrees of staphylococcus sepsis, boils, carbuncles, which represent this infection in slighter form. The point that chiefly interests me is whether we ought to have followed up our urine cultures harder, so that we ought to have said at once, this is staphylococcus sepsis.

DR. YOUNG: I think there is enough in it so that we should follow it up. Apparently the staphylococcus does come through in a fair proportion of cases at least, if not in a large proportion. Whether that would be of value I don't know.

A PHYSICIAN: Can you tell me how often cases of staphylococcus septicemia recover?

DR. CABOT: No. I was not speaking just now of staphylococcus septicemias but of staphylococcus infections of the body. I should suppose that almost all carbuncles are staphylococcus. Of course we know that many of them recover. My guess is that there are a good many staphylococcus processes in the kidney with recovery.

DR. YOUNG: Yes, I know there are. The so-called coccus kidney, which is a hematogenous infection, is seen not infrequently, and the great majority get well.

DR. CABOT: And often without operation. Whether we should call that septicemia or not I don't know. In the carbuncle, for instance, I think it is in the blood before it is in the neck. So I suppose with the kidney, it is always started in the blood. But if a septicemia is meant which is proved during life by a culture, I never knew a case that lived.

A HOUSE OFFICER: We had a case a few weeks ago in the wards where we recovered the staphylococcus from the blood. It started with an abscess in his neck; he developed a pneumonia and an abscess in his foot, and we recovered the staphylococcus from his blood about four times.

DR. CABOT: That was quite like this, a lung infection and then a skin infection. Was this a yellow staphylococcus?

DR. RICHARDSON: Yes. We presume it is yellow unless it is otherwise stated.

DR. CABOT: I suppose it often depends on how long the culture is kept whether it is called yellow or not.

## CASE 9453

AN American of seventy-three, formerly a deep-sea fisherman, was sent from the Out-Patient Department August 22, 1923, complaining of pain in the epigastrium of eight years' duration.

F. H. His mother died of tuberculosis, one brother of "stone cutters' consumption." Another brother was now ill, possibly with the same disease.

*Habits.* Good.

P. H. He was strong, healthy and active until the beginning of his gastric trouble eight years ago. Since the onset of this illness he had had dyspnea on exertion, and an occasional attack of sharp precordial pain localized in an area about the size of the end of a lead pencil just below the left nipple. Five years ago he had an attack of urgency and frequency, D every half hour. Recently the stream had

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lessened in caliber and force.

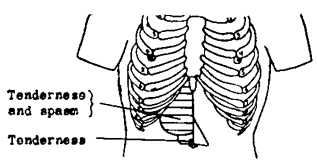
P. I. For eight years he had had attacks similar to the present one, occurring every three or four months and lasting five or six weeks. Be-

tween the attacks he had felt well. Since the beginning of this trouble his bowels had been very constipated.

Six weeks before admission he began to have epigastric pain, occurring half an hour to an hour after eating, partially relieved by food and sometimes by soda. The pain was constant, dull most of the time, and often kept him awake at night. Sometimes it was sharp and knife-like and radiated to both shoulders. He was nauseated and vomited four or five times a day, vomiting almost everything he ate. Two weeks ago and again the day before admission he vomited coffee ground material. Since the beginning of this attack he had lost about thirty pounds and considerable strength. He had eaten very little. His bowels had not moved for days. He was troubled with belching of gas. The present attack was much severer than any previous attack.

*Records of the Out-Patient Department* May 21, 1915. showed a history of the first attack, then of four months' duration,—gradual onset of gastric distress and sharp epigastric pain an hour and a half after eating, relieved by food or vomiting. He had much gas and occasional vomiting of food recently eaten. He had lost 13½ pounds. Examination showed spasm of the right upper rectus. May 27, 1915, the fasting contents of the stomach showed free HCl, guaiac negative; test meal, a few blood clots, free HCl 0.31%, total acid 0.4%. June 8, 1915, he was weaker, knee-jerks increased, Romberg positive.

P. E. Evidence of loss of weight. Teeth poor. Moderate pyorrhea. *Lungs* normal. Apex impulse of the *heart* felt in the fifth space. Measurements not recorded. $A_2$ accentuated. Soft blowing systolic murmur at the apex. Radials slightly palpable. B.P. 180/88. *Abdomen.* Definite rigidity and spasm in the right upper quadrant, tenderness on light pressure over the




right upper quadrant and down the midline as far as the level of the umbilicus. No definite masses felt, although there was a suggestion of a mass in the region of the muscle spasm. (See diagram.) *Genitals, extremities, pupils,* and *reflexes* normal.

Before operation *chart* not remarkable, *urine* cloudy and alkaline at the single examination, otherwise negative, *blood* not recorded, *Wassermann* negative. *X-ray.* Stomach empty in normal time. Peristalsis active, suggesting irritation. No irregularity of gastric outline noted. Sphincter questionable in its regularity. Barium seen to pass through it with some difficulty. First portion of duodenum constantly irregular in filling. Tenderness in this region. Remainder of tract not remarkable.

August 25 operation was done. Next day the general condition was good, but the patient vomited and continued to vomit during the next two days. August 28 the temperature rose to 102.2°, the pulse to 136, the respirations to 32. There was purulent sputum, and râles were heard. The patient became worse, grew dyspneic and cyanotic; the lungs filled with râles. September 1 he died.

## Discussion

### BY DR. HUGH CABOT

The description of this man's present illness seems to coincide rather strikingly with the only suggestive fact in his previous history: namely, the dyspnea on exertion and the precordial pain. These attacks are certainly suggestive of angina pectoris, and would lead one to suspect a fairly high degree of arteriosclerosis. The attacks of epigastric pain which have characterized his present illness are quite clearly different from his precordial pain and dyspnea on exertion. We have a pretty satisfactory account of his first attack in 1915 owing to the description of their findings in the Out-Patient Department. This attack, though not typical, is strongly suggestive of duodenal ulcer, though the examination made at that time is insufficient for a diagnosis. It is not easy to account for his other symptoms, namely increased knee-jerks and positive Romberg, except upon the assumption of arteriosclerotic changes in the nervous system.

It is on the whole a little surprising that he has continued to live as long as he has if one takes their description of his condition in 1915 at its face value. Clearly if he has had attacks of this kind every three or four months they must have been of fairly short duration and he must have picked up very considerably between times. It is noticeable that the history of the present attack is slightly different from the first one in that the pain is constant and but partially relieved either by food or by soda.

The physical examination suggests arteriosclerosis, though we have no positive evidence of hypertrophy and dilatation of the heart. His blood pressure is definitely above normal, and though we have no positive evidence of a lesion of the kidney, it is not improbable that he has some degree of arteriosclerotic nephritis. We shall I think be justified in assuming that he has a lesion of the stomach or duodenum, and that this accounts for the symptoms of his present attack. It is also fairly clear that the lesion from which he is now suffering must be the same

from which he has suffered for eight years, and we are therefore at liberty to exclude cancer as a primary lesion, though we must consider it as a possibility as secondary to chronic ulcer.

The x-ray examination seems to be positive as to a lesion of the duodenum quite typical of a chronic ulcer. Their description of the pyloric sphincter raises the question of whether or not he might perhaps have a lesion both of the duodenum and of the stomach. This is of course unlikely, and we are probably safe in excluding it.

In this region we are pretty safe in excluding malignant disease secondary to ulcer, and I therefore cannot avoid the diagnosis of chronic duodenal ulcer.

In regard to treatment, this is not the sort of lesion likely to be benefited by dietetic measures, since it is clearly a chronic ulcer of the indurated type. In a younger man one would not hesitate to advise operation and to give a favorable prognosis. In this case one might properly hesitate to advise operation, but simply on the ground that his age and probable arteriosclerosis make him a poor risk, and that he might live longer in reasonable comfort on a carefully adjusted diet. I think I should have been willing to advise operation if he clearly understood the risk, but should certainly not have urged it, and should not have quarreled with anyone who believed a careful trial of dietetic measures was desirable.

In regard to the methods which might minimize the risks of operation: It is quite clear that prolonged general anesthesia, particularly with ether, would greatly increase the risk. Spinal anesthesia for operations at this level and at his age appears to me distinctly too dangerous to be risked. My own choice in such cases has been local anesthesia by infiltration and then a short general anesthesia with gas and oxygen if the lesion proved unmanageable under local. In a number of cases it has been possible to carry out the whole operation under local anesthesia in a perfectly satisfactory way.

### DR. CABOT'S PRE-OPERATIVE DIAGNOSIS

Chronic duodenal ulcer.
Arteriosclerosis.

### PRE-OPERATIVE DIAGNOSIS

Duodenal ulcer.

### OPERATION

Twenty cm. right rectus incision. Prior to the operation the needle was introduced beneath the twelfth on either side along the body of the first lumbar vertebra and just in front of it. When the needle was entered about 9 cm. 20 c.c. of 1% procain was injected. The direction of the needle was then changed to slightly downward and another 25 c.c. was injected. This procedure was repeated on each side. The skin and subcutaneous tissues were infiltrated with novocain locally but rather scantily in the usual manner. The peritoneum was opened without incident and without causing pain to the patient. Exploration revealed an ulcer in an indurated area in the first portion of the duodenum. The omentum was adherent apparently to an old appendix. The gall-bladder was negative. The jejunum was mobilized and a loop measured off. A hole was made through the mesocolon and the edges of it sutured to the stomach with silk. A segment of jejunum and one of stomach were each picked up and clamped with intestinal clamps. The patient was then redraped and ordinary gastro-enterostomy was done. The patient complained of discomfort when traction was made on the mesentery. The parietal peritoneum seemed to be almost anesthetic. Snaps were placed upon it without pain, but it was closed with difficulty owing to the patient's straining. The patient did not appear to be uncomfortable and made no complaint at the close of the operation.

*Surgeon's Notes.*—In the first portion of the duodenum extending from the pylorus downward for one inch there was marked thinning and narrowing of the duodenum. No further exploration was made. The only unusual fact was that the omentum was adherent in the lower right quadrant, making it difficult to turn the stomach upward. The omentum was tied and cut to relieve the tension.

### FURTHER DISCUSSION

The block anesthesia which they elected to use seems to have worked pretty satisfactorily, and the difficulty which they had in closing the wound is not more than is commonly experienced under local anesthesia supplemented by gas and oxygen. The evidence that the lesion was in fact a chronic ulcer of the duodenum seemed satisfactory and the indication for a gastro-enterostomy clear. I think they were well advised in this case not to make any attack upon the ulcer.

The history after operation strongly suggests that his death was due to a respiratory infection such as is not uncommon in these patients. Whether the process was a purulent bronchitis or actually went on to a bronchopneumonia cannot be decided on the evidence. I see no reason for assuming that there was any important infection below the diaphragm.

Necropsy should show arteriosclerosis, ulcer of the duodenum, hypertrophy, and dilatation of the heart, probably definite coronary sclerosis with areas of myocardial degeneration, possibly arteriosclerotic nephritis, purulent bronchitis, possibly bronchopneumonia.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Duodenal ulcer.
Operation, posterior gastro-enterostomy.

DR. HUGH CABOT'S DIAGNOSIS

Ulcer of duodenum.
Arteriosclerosis.
Hypertrophy and dilatation of the heart.
Probably definite coronary sclerosis with areas of myocarditis and degeneration.
Probably arteriosclerotic nephritis.
Purulent bronchitis.
Possibly bronchopneumonia.

ANATOMICAL DIAGNOSIS

1. *Primary Fatal Lesion*

Ulcer of the duodenum.

2. *Secondary or Terminal Lesions*

Gastro-enterostomy.
Focal pneumonia of the lower lobes of the lungs.
Fibrinopurulent pleuritis.
Acute degeneration of the myocardium.
Soft spleen.
Arteriosclerosis.
Arteriosclerotic degeneration of the kidneys.
Hypertrophy and dilatation of the heart.
Edema of the lungs.

DR. RICHARDSON: We were not permitted to examine the head.

In the anterior abdominal wall there was a linear operation wound 16 cm. long closed with sutures. The peritoneal cavity, appendix, and esophagus were negative. Between the stomach and the first portion of the jejunum an anastomosis was established by the posterior route. This was patent and in good condition. The mucosa of the stomach generally was rather flat but otherwise negative. The pyloric ridge formed the upper margin of a loss of substance in the posterior wall of the duodenum 1½ cm. by 12 mm. The margins of the ulcer were rounded and descended rather abruptly to a smooth grayish intact base resting over the head of the pancreas and extending slightly into the postero-lateral wall of the duodenum to the right. From the peritoneal aspect of this latter situation old adhesions extended to and along the lesser omentum. The small intestine elsewhere was negative. The large intestine showed several small intact diverticula in the sigmoid region, and in the region of the rectum there were two very small papillomata. The intestine otherwise was negative.

The mesenteric and retroperitoneal glands were negative.

In each pleural cavity there was a small amount of fibrinopurulent semi-fluid material. There were no pleural adhesions.

The lungs in the apical regions were negative. The upper lobes showed no areas of consolidation and the tissue was pale reddish, spongy, and yielded a large amount of thin, dirty, reddish, frothy fluid. The tissue of the lower lobes was dark red, and spongy to a little leathery. In each lower lobe about midway there were several smaller and larger foci of pneumonia, some of which rested just beneath the pleura, which was coated with a thin layer of fibrinopurulent material.

The heart weighed 400 grams, slightly enlarged. The myocardium generally was brown red and lax to a little soft. The cavities were slightly dilated. The valves were negative. The coronaries were free but showed a slight to moderate amount of arteriosclerosis, with no definite diminution of their lumina. The aorta and great branches showed marked fibrous and fibrocalcareous sclerosis, and were rather capacious.

The spleen was rather small and the tissue somewhat soft.

The combined weight of the kidneys was 213 grams, rather small. The capsules stripped and left pale brown red surfaces, which were rather thickly studded with small areas of depression. The tissue showed a slight increase of consistence, and the cut ends of the vessels were rather prominent. The markings were made out. The cortex measured 3-5 mm. The whole picture was that of arteriosclerotic degeneration of the kidneys, not of sufficient extent to be called arteriosclerotic nephritis.

NOTE BY DR. CABOT

The necropsy findings coincide closely with what one would have expected. The statement that the appendix is normal is a little hard to reconcile with the adhesions which they describe.

## Book Reviews

*The Urethra and the Urethroscope.* By F. CARMINOW DOBLE, M.R.C.S., L.R.C.P. (Lond.). London: Henry Frowde and Hodder & Stoughton. Pp. 120.

This treatise on urethroscopy covers very thoroughly the practical side of the inspection of the urethra and the intra-urethral treatment of the lesions found therein. The author describes in detail the leading types of urethroscopes, and gives many helpful suggestions for their manipulation. It is evident that he is thoroughly conversant with the procedures which he describes. Naturally, the book tends to