nute to small discrete and confluent tubercles. These tubercles extended pretty well down into the substance of the middle portion of the lobe. Just beneath the pleura on the upper part of the anterior lobe there was a very small fibrocalcareous nodule. The tissue of this lung elsewhere was generally leathery, dark red, and yielded a moderate amount of dark reddish, frothy fluid.

The pericardium contained a considerable amount of pale, cloudy fluid supporting fibrinous shreds and masses. The vascular and parietal layers were coated with a thin layer of membranous fibrinous material which weakly bound the two layers together. Here and there on the pericardium there were discrete and confluent dark reddish hemorrhagic spots and areas.

The heart weighed 257 grams, enlarged. The myocardium was negative. The cavities showed some enlargement. The valve circumferences were: mitral 9 cm., aortic 6 cm., tricuspid 10½ cm., pulmonary 6½ cm. The mitral curtain showed scattered along its free margin an irregular row of reddish coxcomb-like granulations. The curtain elsewhere showed but little if any fibrous thickening. The aortic cusps presented a moderate amount of diffuse fibrous thickening which slightly contracted and deformed at least two of the cusps. Along the free margin of the cusps there was an irregular band of grayish granulations. The other valves were not remarkable. The coronaries were free and negative. The aorta, however, showed in a few places fibrous streaks and areas in an extent not to be expected at this age. The great branches were not remarkable.

The spleen showed a dark red, elastic tissue with prominent follicles.

The kidneys weighed 252 grams and showed passive congestion. The gastro-intestinal tract was negative except that the solitary and agminated follicles showed marked prominence.

The retroperitoneal and mesenteric lymphatic glands were moderately enlarged, up to 2 cm. in greatest dimension.

DR. CABOT: It is of interest to note that there was a fibrous and therefore chronic endocarditis on the aortic valve in addition to the acute process there and on the mitral. This means that at previous time, very possibly in the attack of chorea three years ago, the aortic valve was involved. The chronic pleuritis also gives us reason to believe that the tuberculous process had extended beyond the bronchial lymphatics at an earlier time. Indeed this seems to be proved by the presence of obsolete foci of tuberculosis in the lungs themselves.

One may speculate a little perhaps upon the question whether this old tuberculosis, presumably acquired in her second year, was lighted up by the attack of chorea, or whether the chorea itself represents an infection made possible by diminished resistance, itself a result of tuberculosis. I remember another case with exactly this association of an acute fatal chorea and acute pulmonary tuberculosis in a patient of this age, when pulmonary tuberculosis is relatively rare. It would be interesting to collect cases of this association and see if one could trace the order of development so as to determine whether the chorea determines the outbreak of tuberculosis, or *vice versa*.

What relation, if any, has the condition of status lymphaticus found here at necropsy to the rest of the lesions? We have ordinarily supposed, on rather insufficient evidence, I think, that the presence of status lymphaticus makes any other disease or injury, such as operative insult, much more serious. But one needs to trace further the relation between this mysterious condition and the two infections which were present in this case.

Since no organisms were demonstrated in this case except those of tuberculosis in the lung, it might be argued that the lesions in the heart and pericardium were really manifestations of tuberculosis. Against that we can merely say that from their morphology it is reasonable to suppose that they represent another infection, since we often see such lesions in connection with streptococcus disease, and do not so far as I know ever see them demonstrated to be of tuberculous origin. That is, we never find tubercle bacilli in the vegetations on the valves in such case. Until such are demonstrated we may well refuse to believe that tuberculosis is ever a cause of endocarditis.

CASE 9433

AN American barber of sixty-two entered July 21, 1923, complaining of pain in the stomach.

F. H. Unimportant.

P. H. He had always been well and strong except for gonorrhea at twenty-two and tape worms at thirty-two.

P. I. A year and a half before admission he began to have pain under the ensiform radiating to the back, steady, and "burning like fire." It occurred every day, sometimes in the middle of the forenoon or the middle of the afternoon, sometimes at night. Because of it he gave up working altogether two months and a half ago. It was severe enough to make him curl up or walk the floor. It was relieved by pressing his hand to his stomach, eating a bowl of corn flakes and milk, taking baking soda, which gave immediate relief for several hours, or applying Sloan's liniment or a hot towel. During the

attacks he frequently vomited with temporary relief clear fluid, extremely bitter and very acid; no blood. He immediately regurgitated most of the water he drank and all the lemonade. Grapefruit gave more heartburn than anything else and caused watery eructations. For over a year he had been limiting his diet, first omitting pork, then all meats. For the past month he had eaten nothing but corn flakes and milk. For seven months he had had heartburn daily, relieved by lime water. It was impossible to draw out any history of the hemorrhage. Three weeks ago his bowels, usually regular, did not move for three days. As a result of $MgSO_4$ he had a movement as "hard as stone," he thought gray. He had grown pale and had lost about thirty pounds in the past year and a half. His best weight was 196 pounds, his present weight probably 170.

P. E. Rather obese. Mucous membranes pale. Septum deviated to the right, causing slight obstruction. Throat and tonsils reddened. *Lungs* clear. Apex impulse of the *heart* not found. No enlargement to percussion. A systolic murmur at the apex. B. P. 160/85. *Abdomen.* Resistance in the upper abdomen. Liver felt on the right. Under the upper end of the rectus liver, spasm or a mass was felt—impression, spasm. *Genitals, extremities, pupils* and *reflexes* normal.

Chart not remarkable until August 4; August 4-8 *T.* 98°-101.8°, *P.* 71-106. Before operation amount of *urine* not recorded, sp. gr. 1.010-1.022, alkaline at four of five examinations, a very slight trace to a slight trace of albumin at two, 2-3 leucocytes to a high power field at one; *blood*—hgb. 60 per cent. to 75 per cent., leucocytes 10,800-25,000, polynuclears 72 per cent.; reds 4,230,000-5,960,000, at the first examination (the only stained smear) marked achromia, poikilocytosis and anisocytosis, tending toward microcytes, tailed forms, many oval and sausage-shaped cells, platelets markedly decreased and small. *Wassermann* negative. *Gastric analysis. Fasting contents.* 35 c.c., slightly yellow. Free HCl, 1. Total acid, 12. Guaiac negative. *Test meal.* 20 c.c., normal. Free HCl, 41. Total acid, 59. Guaiac negative. *Lumbar puncture.* 10 c.c. of clear colorless fluid. Pressure, 160; after withdrawal of 5 c.c., 120; after withdrawal of another 5 c.c., 110. Three cells. Total protein 30. All tests negative. *X-ray.* July 25. (See illustration.) Stomach contained a small residue at six hours. Peristalsis active. Outline irregular along the lower portion of the lesser curvature, presenting at this region a large projection. This appeared to interfere with the peristaltic wave, and the region never filled well. The sphincter appeared wide and questionable in regularity, although there was no marked delay in the passing of the barium. Tenderness was present in this region. First portion of duodenum never filled with good outline, perhaps because of an insufficient amount of barium entering it, or very possibly because of a second lesion in this region. These abnormalities were the same at two observations on successive days. Remainder of gastro-intestinal tract not remarkable. Left diaphragm high and limited in excursion.

Outline irregular along the lower portion of the lesser curvature, presenting at this region a large projection. This appears to interfere with the peristaltic wave; the region never filled well. The sphincter appears wide and questionable in regularity. The first portion of the duodenum never filled with good outline.

Orders. July 21. Five meal gastric diet. Sodium bicarbonate gr. x t.i.d. p.c. July 23. Atropin gr. 1/120 by mouth, repeat in an hour if necessary. July 24. Morphia gr. 1/6 s.c. Hyoscin hydrobromide gr. 1/200 s.c. Sodium bicarbonate gr. xv. July 26. Hot Dobell's gargle t. i. d. July 27. Magnesium oxide and sodium bicarbonate each gr. x t. i. d. July 28. Morphia gr. 1/6 s.c., repeat once if necessary. July 29. Morphia gr. 1/6. August 1. Sodium bicarbonate gr. xv. August 4. Force cool fluids; no ice. Veronal gr. x at bed time. August 6. Two per cent. cocain ten drops in half a glass of water. Nothing by mouth. Rectal glucose 5 per cent. ℥ iv every three hours (not given). August 6. Hyoscin hydrobromide gr. 1/200 s.c.

Before July 25 the patient had one attack of hiccups. Surgical consultants advised operation, to which the patient was much opposed. It was therefore planned that he should go home with orders for a five meal diet and alkali as needed.

The night of August 3 he complained of sleeplessness. Next day he had epigastric discomfort and no appetite, and in the evening the temperature rose to 101.8°, the pulse to 102, the respirations to 26. The leucocyte count was 19,600. There was some dullness at the left base posteriorly with loss of tactile fremitus and

bronchial voice sounds. The night of August 6 he again had hiccups. August 7 he had acute retention, requiring catheterization. The prostate was felt by rectum to be enlarged. August 8 he was definitely weaker and hiccuping. The sclerae were icteric.

August 8 operation was done. The next day the patient was slightly irrational, but objectively better. August 12 the temperature was down. There was little drainage and he looked and felt better. August 16 he was hungry and comfortable.

August 18 after walking across the ward he fainted. A few minutes later he was dyspneic, pale and in moderate shock, with pulse 120 and blood pressure 118/75. He was treated for shock. Soon afterwards he vomited (no blood). At first there was no pain, and the abdomen was soft. Ten minutes later he complained of pain over the right chest, became very dyspneic and cyanotic, and died a respiratory death.

Discussion

BY DR. EDWARD L. YOUNG, JR.

There are two things in this history that stick in my mind: (1) the comparatively recent onset of pain, which may be suggestive of stomach trouble; and (2) the fact that this pain comes on in the middle of the forenoon or night,—in other words, when the stomach is presumably empty.

We have another thing that is of considerable importance, and that is that this pain is relieved by food, by alkali, and by getting rid of the acid contents of the stomach. I think we have enough in the history here to throw us into one of the two big divisions we always have to consider,—that is, has this thing an organic basis which demands serious attention in the line of surgery, or is it a so-called functional condition?

Here is a man with definite symptoms, although not all typical, with loss of weight. His pain is real enough so that I think we have to believe in it, and it is relieved by those things which we know often relieve organic stomach conditions of ulcer, whether simple or malignant. As I say, the one thing that seems to impress itself on my mind is the comparatively recent onset of symptoms in a man of cancer age.

"Spasm or a mass was felt—impression spasm." That is the best description of that sort of thing that I have read in any record. It describes what it might have been from the feeling, and the impression of the man who felt it.

Alkaline urine is natural with his diet and the amount of soda he is taking.

The hemoglobin is a little low, which again suggests malignancy.

They were fishing for the possibility of a gastric crisis, I suppose.

I do not think it is possible to get back of the story this man gives as to its being an organic condition. The fly in the ointment is the lack of blood in the vomitus and the negative guaiac on examination of the stomach contents. If this is a cancer of the stomach of one and a half years' duration, there should have been in that time some evidence of blood in the vomitus. Is it possible that this is trouble in the biliary tract? It certainly is not possible to rule it out. But it is not the typical story or anywhere near the typical story that we should expect. It is notorious that gall-bladder disease has the relation to food which causes the "atypical" indigestion, that certain things cannot be eaten, and there will be attacks called "bilious," or indigestion, after eating certain food. It is not necessarily a harsh food that causes trouble; it may be simple food, and very seldom does it go to the point where the pain is relieved consistently by vomiting or by alkalis.

It seems to me, however, that in spite of the lack of blood there is enough to say that there is some organic condition near the pylorus.

Dr. Cabot: You mean cancer?

Dr. Young: Yes.

Dr. Cabot: One point, I should say, in favor of your diagnosis is that he has anemia and yet he has not lost any blood. He cannot have a secondary anemia from ulcer unless he has lost blood.

Dr. Young: There is no evidence except that he has been so shy of food.

Dr. Cabot: There is no evidence that one can get anemia from starvation alone. It concentrates the blood.

Dr. Foster: Is that a very great anemia?

Dr. Young: No, it is not, and yet it is below 70 per cent. Down to 70 I always think is debatable ground.

Dr. E. L. Oliver: It says that he has achromia at the first examination.

Dr. Cabot: That is much more important, I think. Those are things we don't get just for the fun of it,—when people are run down, etc.

A Physician: Does the continuity mean anything in favor of its being cancer—the lack of periodicity?

Dr. Young: It is more suggestive to me of cancer.

A Physician: Do you think the loss of weight is in favor? He weighs 170 pounds, and the cancer has been going on for a year and a half.

Dr. Young: He has not been going downhill all that time. His first symptoms were a year and a half ago, but I think we have all seen cancer that goes on for a considerable time without any more definite loss of weight. This is thirty pounds, which is a considerable loss, of course.

DR. MERRILL: If we knew just how recently he had begun to lose that weight, shouldn't we have more basis?

DR. YOUNG: It says during the last year and a half.

A PHYSICIAN: Do you think the loss of weight can be attributed, if he had ulcer, to the fact that he had not eaten much food?

DR. YOUNG: He has done pretty well on diet. Corn flakes and milk, if he has eaten enough of it, is a good diet. This is not like some of the stories we read here about a patient who has not had anything but liquids for a month or two months, and has vomited everything eaten. Under these conditions—even though we know of course they do not vomit everything—it is inevitable to lose a lot of weight.

Dr. Merrill, will you comment on the x-ray plate?

DR. MERRILL: I think there is no doubt, as Dr. Young says, there is something to indicate that the man has some organic disease. So far as the value of the history to us is concerned, I think that most of our demonstrable gastric cancers give us a briefer history than a year and a half, and this man says it has been a year and a half since his acute symptoms began with pain. That usually suggests to us that the history may really be longer than that before the acute things began, or until they were precipitated by some change in his gastric condition. But presuming the history is true, that is longer than the average story which we get from gastric cancer cases. His age is in favor of cancer. His loss of weight, assuming that it was very recent, is also in favor. If it had been gradually falling off for a year and a half it might be, so far as we know, either cancer or ulcer. The gastric findings were so questionable that we made a second observation and found practically the same thing both times. The stomach contained a small residue at six hours. By "small residue" we mean twenty-five per cent. or less of the meal, and that does not signify to us actual obstruction. The peristalsis was active. The activity of the stomach may account for the finding of such a small delay even in the presence of a carcinoma, but usually gastric hyperactivity is inconsistent with gastric malignancy. It is more suggestive of an inflammatory condition. So far as we have gone we have a stomach which has some delay in spite of gastric activity, and the outline of the stomach as seen after giving a full meal is irregular along the lower portion of the lesser curvature. As an ordinary thing projections from the gastric outline mean ulcer craters rather than carcinomatous tumors, although there is a small class of malignancies which from their irregular outline or perhaps by an ulcerative process in the malignancy itself will show an apparent projection simply because we do not see the whole gastric outline. Of course the portion of the stomach occupied by the tumor is invisible. In this small class of cases, which has been noted by every observer, it may very well be malignancy and is often found to be. As in infiltrating conditions, the wave stopped and did not go by that point. There was no marked delay in the passage of the barium during the observation. Coincident lesions in the duodenum and the lesser curvature of the stomach are not at all uncommon. The findings are suggestive of an ulcerative lesion on the lesser curvature, and I believe that the deformity was in part due to adhesions about the antrum following the perforation of the ulcer, although malignancy cannot be excluded.

The more I think of this case the more uncertain I become. But I think that after following this discussion I should be tempted to say that considering all the possibilities,—the significance of the gastric symptoms, as Dr. Cabot has explained, having so little bearing on the question of malignancy or ulcer,—after all, malignancy may be the most probable conclusion.

We see here on the lesser curvature, rather low down, a projection, and below that the stomach does not fill well. That may be and probably is due to the presence of a tumor on the lesser curvature, preventing its free filling. It might also be due to adhesions about this end of the stomach in consequence of a large perforating ulcer.

DR. YOUNG: Dr. Merrill speaks of that as being a short time for symptoms of cancer of the stomach as the roentgenologists see it. On the other hand, I believe that the ordinary story of ulcer will extend over a much longer period, and very often there will be the intermittent story of pain and freedom from trouble.

The orders are essentially attempts to lessen discomfort in the stomach by diet, by alkalis. The atropin was given the day of the x-ray to lessen any pyloric spasm. The hiccups always suggest attacks of trouble with the kidneys. Of course it can be due to plenty of other things. The icterus may be nothing but infection.

Does this medical picture mean long infection of definite nature, or can it go with congestion?

DR. CABOT: It may perfectly well all be below the diaphragm.

DR. YOUNG: The possibility of a leak from a perforating ulcer, subacute perforation, so called, might account for the story. The acute retention with a large prostate to be felt by rectum suggests obstructing prostate. But in a man who is definitely weak it does not necessarily mean a severe grade of that. But that, plus his hiccupping, again makes us wonder whether there is trouble there. I think we have to remember that we do not see the patient

here. On the evidence here I do not see what they were operating for, unless for a subacute perforation.

DR. HARMER: I don't think the description is complete. He was seen by the surgical people about the fourth or fifth of August, and at that time he showed spasm and tenderness on the right side, which is not mentioned here, although it is in the record.

DR. YOUNG: In other words a very definite suggestion of a subacute perforation to go with the story of increase of pulse and temperature.

DR. HARMER: There was a very distinct change, both in his appearance and in his chart.

DR. YOUNG: So that the belief then is that this process is perforative and that it is necessary to go in and repair the damage. That is what I assume from what is added.

DR. HARMER: He was seen in the medical wards by Dr. Davis, operation advised and refused. He was kept a little longer, and during these few days he apparently perforated. Then he was seen again by Dr. Jones, who thought he had a perforation. The man was in wretched condition. He was cold, his forehead was sweaty, his nose was cold. The abdomen was not board-like but was resistant; but here we are considering a man in very poor shape. There was, however, definite resistance on his right side, extending down to the lower part of the abdomen. It was thought then that he had probably ruptured. Dr. Jones suggested operation under local anesthesia, if nothing else was done to put a drain into that side.

DR. CABOT: I should like to know what the diagnosis was.

DR. HARMER: The diagnosis was ruptured gastric ulcer.

DR. LINENTHAL: I think ulcer is the most probable.

DR. CABOT: I am betting on cancer.

DR. YOUNG: How often proportionately do ulcer and cancer perforate?

DR. CABOT: I have seen cancer perforate a number of times, but nothing like so often as ulcer.

DR. YOUNG: Isn't it later in the disease, isn't it more obviously a cancer?

DR. CABOT: Yes, I should say so.

DR. YOUNG: I hate to give up the diagnosis of cancer myself, and yet I think the story for ulcer is very strong.

DR. HARMER: The point you mention is the one mentioned by the surgical service, that is, that he apparently was perforated, and if it was perforation it was probably ulcer rather than malignant.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Perforated gastric ulcer.

PRE-OPERATIVE DIAGNOSIS

Perforated gastric ulcer.

OPERATION

Local novocain. Four-inch high right rectus muscle-splitting incision. The peritoneum was opened without incident except for adhesions of the omentum to the anterior abdominal wall. There were also many adhesions between the duodenum and the liver, most of them of old standing. These were cautiously separated, and a recent inflammatory mass was encountered. A cigarette drain was placed to the foramen of Winslow. There was no free fluid in the abdominal cavity. Abdomen closed in layers to wick.

FURTHER DISCUSSION

DR. YOUNG: In other words, according to this—Dr. Harmer will amplify it as necessary—they did not find anything. It does not say anything about the stomach here at all.

DR. CABOT: They did not find any ulcer or cancer or anything else?

DR. HARMER: Oh yes, we did. I did the operation. We did it under novocain and it worked very well, and we were able to get a good exposure. There were adhesions to the front wall inside. He had considerably more fat than one would think from his loss of weight. He had a mass which was in back of his duodenum. We could see the front wall of the duodenum. There were a few fresh adhesions, but most of them seemed to be old, as if he had had an old process which in the past had started to perforate and had walled itself off. There was no free fluid. In other words, he had no definite perforation, although he might have been trying to perforate, because there were some fresh adhesions present. The under surface of the liver showed no malignant nodules. The lower end of the stomach was felt—it could not be well seen—and did not feel remarkable. So a drain was simply placed to the foramen of Winslow.

DR. FOSTER: Why did they put in a drain if there was no fluid?

DR. HARMER: Here was a man *in extremis*. He had a few fresh adhesions. He was apparently on the point of perforating. The chances were he was more likely to perforate than not. So the drain was put in simply as a precautionary measure.

DR. CABOT: With what diagnosis did the surgeon come out of the operation?

DR. HARMER: Duodenal ulcer.

DR. YOUNG: You thought that mass was ulcer?

DR. HARMER: We did not think it was cancer.

DR. YOUNG: That is an operation that cures. There are two things I should like to settle. (1) I think we have as much evidence as we can have that he died at the last of one of the surgical accidents, of pulmonary embolus. But I should like to know what you think Dr. Rich-

ardson is going to find. Dr. Harmer did not find anything in the pylorus in feeling of it suggestive of cancer or ulcer such as we have been describing. He thinks there is a lesion in the duodenum, which means of course ulcer,—because cancer of the duodenum is too rare to be considered,—with subacute perforation, which I think still has got to be assumed as being there. Have his symptoms all been due to that, so that we can on that basis explain his low hemoglobin in some other way than by cancer? I am going to put myself on record as believing he had carcinoma. That is rather a foolish thing to do because a good deal of the evidence since I discussed it seems to me to swing the other way. Dr. Harmer believes that he has duodenal ulcer. Dr. Linenthal believes he has ulcer.

DR. CABOT: I think he has a cancer.

DR. YOUNG: How many for cancer? How many for ulcer? About half and half.

DR. FOSTER: Have we any stool examination?

MISS PAINTER: Guaiac was constantly negative at five examinations.

DR. YOUNG: Of course it is possible that there was nothing there. Every now and then Dr. Richardson assures us that there was nothing.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Perforated gastric ulcer with localized peritonitis.
Pulmonary embolus.
Coronary block.
Operation, laparotomy with drainage for perforated gastric ulcer.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Gastric cancer.
Pulmonary embolus.

ANATOMICAL DIAGNOSIS

1. *Primary Fatal Lesion*

Ulcer of the duodenum.

2. *Secondary or Terminal Lesions*

Pulmonary embolism.
Arteriosclerosis.
Arteriosclerosis of the vessels of Willis.
Wet brain.
Partial arteriosclerotic occlusion of the coronary arteries.
Slight fibrous myocarditis.
Hypertrophy and dilatation of the heart.
Edema of the lungs.

3. *Historical Landmarks*

Operation wound.
Chronic localized peritonitis.
Cholelithiasis.

DR. RICHARDSON: This man was well enough developed, but rather poorly nourished. The body looked as though weight had been lost,—that is, the skin was loose over the subcutaneous tissues and fat, and on section the fat seemed to be in fairly reasonable amount at first glance, but on closer inspection we saw that the connective tissue was quite prominent, so that there was quite a reticulum of fibrous tissue, indicating that previous to that there had been more fat there.

There was a wet pia and a wet brain. The vessels of Willis and the branches showed marked sclerosis, in places slightly calcareous.

The skin and mucous membranes were pale.

The stomach was considerably enlarged and contained much opaque fluid and food-like material; the mucosa was rather pale,—a good stomach. In the duodenum however, on the right side posteriorly and extending around to the anterior lateral wall on the right, was a loss of substance 4½ x 3 cm., a frank ulcer, the upper margin of which was practically the pyloric ridge. The ulcer rested far enough above the gastroduodenal artery, which runs across and back of the duodenum, so that the patient escaped what probably would have happened if lower down,—death from hemorrhage. The ulcer on the right was adherent, as Dr. Harmer said, to the inferior surface of the left lobe of the liver. Removal of those adhesions tore away a thin filmy veil of tissue which spread across the base of the ulcer; so that there was practically a perforation at some previous time.

The intestinal tract below that was out of the picture.

The heart weighed 482 grams,—enlargement due to sclerosis, which was well marked in the aorta and great branches. In one branch of the aorta, that is the left coronary artery, just beyond its orifice, there was considerable fibrous and fibrocalcareous sclerosis with marked diminution of the lumen,—in other words, partial occlusion of that coronary. The right coronary, while not showing such a marked diminution, showed some, and was the seat of well marked arteriosclerosis. In the pulmonary artery there was a V-shaped embolus. On the left side, while the first great branches were frankly negative, in remoter branches there were small bits of embolic material. When we first opened it we thought we were not going to find an embolus.

The portal veins and radicles, the liver, pancreas, and ducts were out of the picture except that the common bile duct, which was slightly dilated, contained three small concretions. The gall-bladder contained one coke-like stone. The spleen was negative. The kidneys showed no definite changes. In the myocardium there were a few spots; it was a question in my mind whether they were myocarditis or not. I thought they were, and the microscope showed fibrous myocarditis.

A PHYSICIAN: What was the origin of the emboli?

DR. RICHARDSON: We did not find it.

ABBREVIATIONS USED IN CASE RECORDS

F.H.	family history	cm.	centimeters
P.H.	past history	c. c.	cubic centimeters
P.I.	present illness	gm.	grams
P.E.	physical examination	mgm.	milligrams
T.	temperature	mm.	millimeters
P.	pulse	gr.	grains
R.	respirations	t.i.d.	three times a day
B.P.	blood pressure	p.r.n.	when required
A_2	aortic 2nd sound	s. c.	subcutaneous
P_2	pulmonic 2nd sound	p. c.	after meals
hgb.	hemoglobin	tb.	tubercle, tuberculosis
cta.	catamenia	K. L.	bacillus diphtheriæ
sp. gr.	specific gravity		

DIAGRAM REPRESENTING THE CARDIAC BORDERS

$\frac{4 \mid 9 \quad .3.5}{}$ The upright line represents the midsternum. The figure at the left, 4, indicates the distance to the right border as found by percussion: the sum of those at the right, 9 + 3.5, shows the distance to the apex impulse, or if that is not recorded, to the left border by percussion. The large dot (not the decimal point) indicates the mammary line. In certain cases the substernal dullness corresponding to the aortic arch is indicated by a second transverse on the midsternal line.

$\frac{7}{4 \mid 9 \quad .3.5}$

$\frac{D\ 5\text{-}6}{N\ 4}$ = MICTURITION, day five or six times, night four times.

THE COMMON CHEMICAL ABBREVIATIONS, as HCl, KI, etc.

INQUIRY FROM A SUBSCRIBER

Will you please give me, through the medium of the *Cabot Case Records,* the method used at the Massachusetts General Hospital in determining the non-protein nitrogen of the blood. I should like the technique in detail.

A detailed account of the technique of the non-protein nitrogen test, with instructions for making the necessary reagents, would require too much space for the limits of this publication. Instructions for the whole procedure may be found in Folin's Laboratory Manual of Biological Chemistry, edition of 1922, or later (Appleton, N. Y.) or in an article by Folin and Wu in the *Journal of Biological Chemistry,* 1919, page 81. The Du Boscq colorimeter for the test may be obtained from Eimer and Amend, Third avenue, 18th to 19th streets, New York.

The Massachusetts Medical Society

PROCEEDINGS OF THE COUNCIL

STATED MEETING, OCTOBER 3, 1923

A STATED meeting of the Council was held in John Ware Hall, Boston Medical Library, on October 3, 1923, at 12 o'clock, noon. The President, Dr. E. H. Bigelow, of Framingham Center, was in the chair and the following 113 councilors present:

BARNSTABLE,
W. D. Kinney.

BERKSHIRE,
A. P. Merrill.

BRISTOL NORTH,
F. A. Hubbard.

BRISTOL SOUTH,
A. I. Connell.
E. F. Curry.
D. J. Fennelly.
C. J. Leary.
W. A. Nield.

ESSEX NORTH,
R. V. Baketel.
J. Forrest Burnham.
W. W. Ferrin.
T. R. Healy.
A. M. Hubbell.
G. E. Kurth.
F. W. Snow.
W. D. Walker.

ESSEX SOUTH,
F. W. Baldwin.
W. T. Hopkins.
P. P. Johnson.
J. F. Jordan.
W. G. Phippen.
A. N. Sargent.
R. E. Stone.

FRANKLIN,
G. P. Twitchell.

HAMPDEN,
G. H. Janes.

HAMPSHIRE,
A. J. Bonneville.

MIDDLESEX EAST,
C. R. Henderson.

MIDDLESEX NORTH,
W. B. Jackson.
J. H. Lambert.
G. A. Leahey.
J. A. Mehan.

MIDDLESEX SOUTH,
E. A. Darling.
E. H. Bigelow.
F. B. M. Cady.
I. J. Fisher.
F. W. Gay.
F. J. Goodridge.
C. E. Hills.
L. H. Jack.
S. F. McKeen.
Edward Mellus.
C. E. Mongan.
C. F. Painter.
W. D. Ruston.
J. W. Sever.
C. H. Staples.
E. H. Stevens.
A. K. Stone.
G. L. West.
W. S. Whittemore.

NORFOLK,
W. W. Howell.
E. H. Baxter.
D. N. Blakely.
W. L. Burrage.
F. J. Callanan.
D. G. Eldridge.
A. H. Hodgdon.
F. C. Jillson.
G. W. Kaan.
Bradford Kent.
J. S. H. Leard.
Edward Martin.
E. T. Rollins.
Victor Safford.
Lucia F. Vickery.
H. F. R. Watts.

NORFOLK SOUTH,
C. A. Sullivan.
C. S. Adams.
O. H. Howe.
G. M. Sheahan.

PLYMOUTH,
F. H. Burnett.
W. C. Keith.
N. K. Noyes.
Gilman Osgood.
F. G. Wheatley.

SUFFOLK,
J. S. Stone.
S. H. Ayer.
J. T. Bottomley.
M. E. Champion.
David Cheever.
Loretta J. Cummins.
G. B. Fenwick.
Channing Frothingham.
J. E. Goldthwait.
J. C. Hubbard.
H. T. Hutchins.
F. T. Lord.
Donald Macomber.
G. B. Magrath.
T. J. O'Brien.
W. H. Robey.