

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL



Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*BETTY U. KIBBEE, *Assistant Editor*

CASE 45331

PRESENTATION OF CASE

A sixty-five-year-old retired asbestos worker entered the hospital because of dyspnea.

Ten weeks previously he experienced a sudden episode of collapse, with dyspnea and weakness, that lasted for an hour. Two weeks later he noticed swelling of the legs, dyspnea on exertion and anorexia. His physician prescribed a low-salt diet, and digitalis therapy was begun. One month before admission the edema and dyspnea increased and persisted despite Diamox therapy. During the two weeks before entry Mercuhydrin therapy was given, without effect. There was no chest pain or hemoptysis.

The patient allegedly had rheumatic fever when he was twenty-four years old and was admitted to a hospital for three months. There were no known cardiac murmurs. His dental hygiene had been poor for many years. He had been an asbestos worker for thirty-five years, and had retired one and a half years before entry after sustaining accidental injuries. He was studied at a sanatorium two years before admission, and was told that he had fibrosis of the lungs.

Physical examination showed a slightly cyanotic man who was dyspneic after exertion. Clubbing was present. The teeth were very carious. Examination of the chest revealed moist rales at both lung bases and dullness with diminished breath sounds at the right-lung base. The left border of cardiac dullness extended 3 cm. beyond the mid-clavicular line in the fifth intercostal space. The pulmonic second sound was greater than the aortic; no murmurs were audible. The abdomen was normal. There was + + + pitting edema of the legs and thighs.

The temperature was 99°F., the pulse 76, and the

respirations 22. The blood pressure was 140 systolic, 85 diastolic.

The urine gave a + + + test for albumin; the sediment contained 3 to 5 white cells and a moderate number of hyaline and coarsely granular casts per high-power field. Examination of the blood revealed a hemoglobin of 17.4 gm. per 100 ml. and a white-cell count of 7400. The nonprotein nitrogen was 33 mg., the bilirubin 1 mg., the calcium 7.2 mg.,

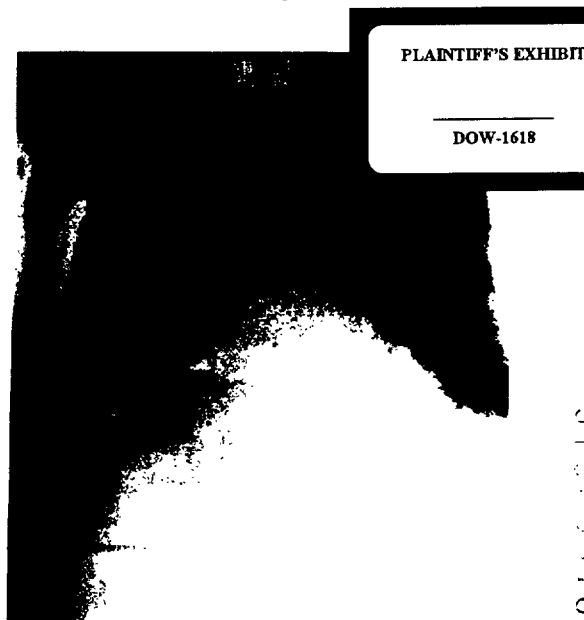


FIGURE 1. Posteroanterior Film of the Chest, Showing Cardiac Enlargement, Pulmonary Congestion and a Pleural Reaction, Together with Fluid at the Right Base.

the phosphorus 3 mg., the protein-bound iodine 6 microgm., and the total iodine 8.4 microgm. per 100 ml. The sodium was 136 milliequiv., the potassium 4 milliequiv., the chloride 92 milliequiv., and the carbon dioxide 36 milliequiv. per liter. The prothrombin content was 65 per cent, and the alkaline phosphatase was 7 units. The transaminase was 25 units. A sputum culture grew out a normal flora and a few colonies of pneumococci; cytologic examinations for malignant cells were negative. The tuberculin skin test (second-strength PPD) was positive. X-ray films of the chest (Fig. 1) demonstrated moderate, nonspecific cardiac enlargement; there was a moderate right-sided pleural effusion, with some pleural thickening around the lower portion of the thorax bilaterally, more pronounced on the right; the pulmonary vessels were congested; there was no hilar lymphadenopathy. An electrocardiogram (Fig. 2) showed low voltage and atrial fibrillation, with a very regular ventricular rhythm, probably of nodal origin; there were small Q waves in Leads 1 and aVL and a QS pattern in Leads V₁ through V₃.

A thoracentesis on the right side yielded 1100 ml. of amber fluid; it contained 500 white cells and no change from the previous tracing. On the next

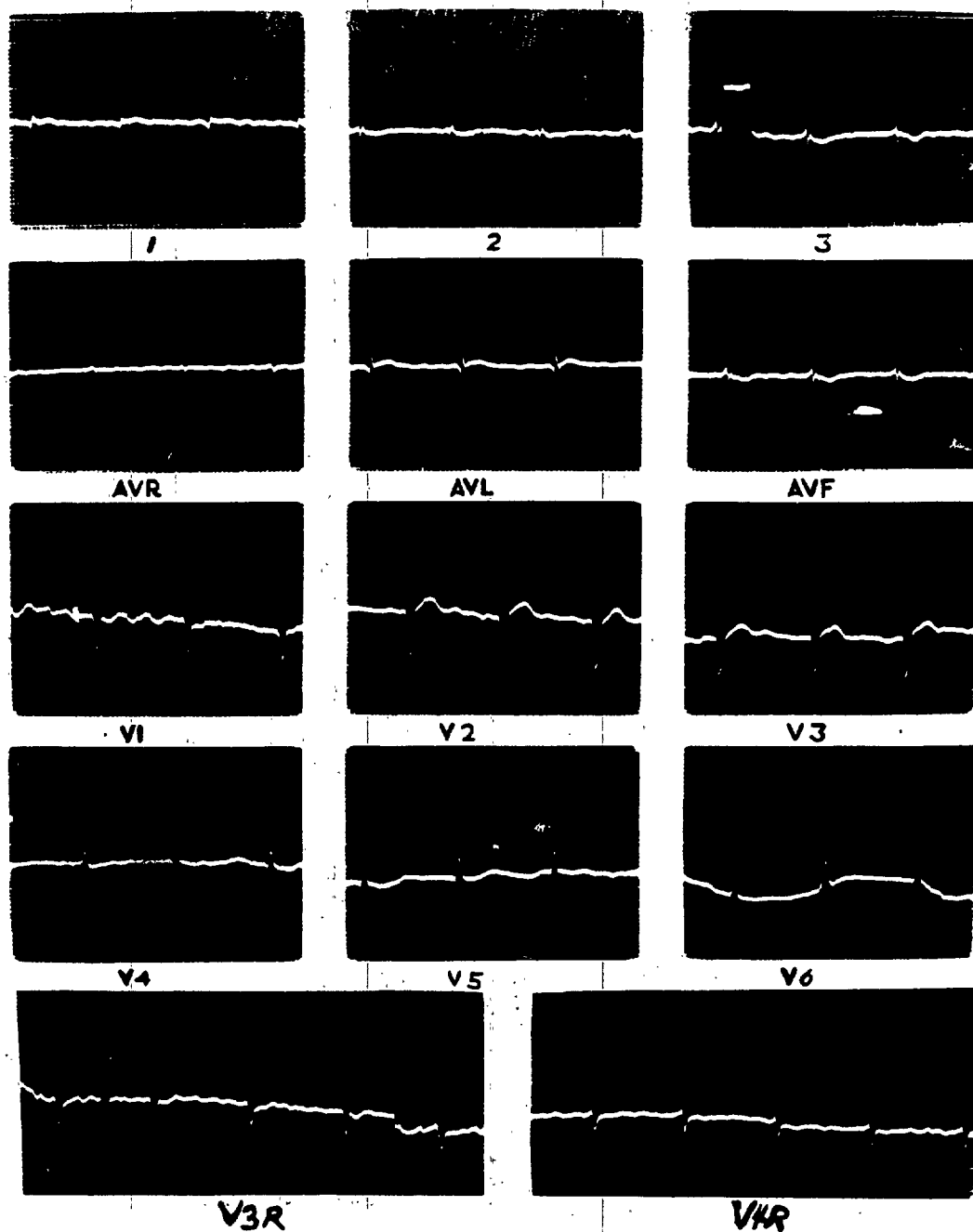


FIGURE 2.

7300 red cells per cubic millimeter; the specific gravity was 1.008; the total protein was 0.43 gm. per 100 ml.; cytologic examination for tumor cells was negative. Ammonium chloride therapy was begun.

day there was marked diaphoresis. A protodiastolic gallop was present for several days. Three times the patient complained of generalized abdominal pain that occurred after a bowel movement and

mucus, with a few small red blood cells. Physical examination showed the neck veins to be distended 5 cm. above the level of the clavicles when he was raised 45°. The skin was cold, clammy and dusky. The pulse was 88 and thready, and the blood pressure was unobtainable. There were signs of a pleural effusion on the right side and rales at both lung bases. The heart sounds were distant, and there was a gallop and a normal sinus rhythm. The abdomen was diffusely slightly tender and rigid, with hypoactive bowel sounds; there was no rebound tenderness. The femoral pulses were good. An electrocardiogram showed no change except for non-specific ST-segment changes. Despite Aramine and Levophed given intravenously the blood pressure remained unobtainable. An abdominal paracentesis yielded no fluid, and an x-ray film of the abdomen was normal. The patient died seven hours later.

DIFFERENTIAL DIAGNOSIS

DR. MYLES P. BAKER*: In summary, this is the problem of a sixty-five-year-old man who entered the hospital in congestive heart failure. Treatment with the standard measures achieved some improvement, but the progress was punctuated by episodes of abdominal pain and of apparently small hemoptyses, and was finally brought to an abrupt and disastrous close by severe abdominal pain and circulatory collapse. We must find the answers to several questions. Regarding the etiology of the heart disease, is there evidence in support of pneumoconiosis with chronic cor pulmonale, rheumatic heart disease or coronary thrombosis and myocardial infarction? Had there been pulmonary embolism and infarction? Finally, what explanation best fits the terminal episode?

This patient certainly had an occupational history suggestive of pneumoconiosis. He was exposed to the danger of pulmonary asbestosis, and pulmonary fibrosis was reported on an x-ray examination two years before admission to this hospital. However, the x-ray films obtained on entry apparently did not indicate the type of pneumoconiosis that tends to be a precursor of right-sided heart failure because there is no mention of large agglomerates with fuzzy borders, although pleural thickenings were noted. I think that the x-ray examination has much information to offer in this case, and I should like to see the films now.

DR. STANLEY M. WYMAN: On the films of the chest taken on admission (Fig. 1) cardiac enlargement can be fairly well seen, and the left ventricle in particular seems prominent. The right costophrenic angle is obliterated, probably by fluid, although there may be underlying pleural changes.

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The aorta is tortuous, the hilar vessels are prominent, and the prominence of the pulmonary vessels extends well out into the lungs. The question of underlying pulmonary fibrosis has been raised, but such changes are minimal, if present, and I am unable to make a positive statement in view of the pulmonary congestion. I see no evidence of a pulmonary infarct or other localized active process. The left leaf of the diaphragm is elevated, but that is probably not a significant finding.

In the films taken four days later, after the patient had received diuretic therapy, the pulmonary vessels are definitely less engorged. Although 1100 ml. of fluid had been removed from the right pleural cavity, the obliteration of the costophrenic angle demonstrates that a fair amount remained. Trying again to answer the question of an underlying fibrosis, I think that minimal discrete linear shadows may be present, but I cannot interpret them as frank fibrosis. The heart has not changed greatly in size. Overlying the left heart border I see a little area of calcification that intrigues me, and I wonder if it might represent calcium in a coronary artery or even in the wall of the left ventricle.

DR. PAUL D. WHITE: Is there a round shadow near the right base?

DR. WYMAN: I think that is an optical illusion, and is probably a superimposition of shadows.

One more chest examination, done about ten days later, shows very little alteration. The pulmonary vessels may be a little more engorged, but this change is certainly minimal.

DR. BAKER: The patient was in acute circulatory failure at that time.

DR. WYMAN: I see no evidence of pulmonary edema other than a very questionable little patch in the right-middle-lung field.

DR. WHITE: Does the calcification remain in the same place?

DR. WYMAN: The calcification is still present, overlying the left heart border. I wish I could say exactly where it lies, but I can't.

The films from the barium-enema and the gastrointestinal examinations were normal. Those films of the abdomen that were taken just before the patient died are poor because of his precarious condition. The film taken with the patient in a supine position shows no significant distention of bowel, and that taken with the patient lying on the left side with the right side elevated shows no free air in the abdominal cavity.

DR. BAKER: I think the negative findings in the x-ray films are most important — the absence of marked pneumoconiotic processes, of mitral configuration of the heart and of pronounced widening of the pulmonary conus. This man's pulmonary function could have been severely limited by pneumoconi-

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osis, although we have little evidence of pulmonary emphysema in either the films or the physical findings, and there is no indication in the history that he had been a pulmonary cripple before the onset of his troubles two months before admission. However, he did have clubbing of the fingers, which is otherwise unexplained, and a high blood carbon dioxide level, which may accompany chronic pulmonary disease.

Before attacking the problem of the structural nature of this patient's heart disease, we should note that pulmonary and pleural tuberculosis were looked for and within reason excluded, and also that bronchogenic carcinoma was considered and not found to be the factor responsible for the bloody sputum and the pleural effusion. The findings pertinent to the heart at the time of admission include its large size, atrial fibrillation and the absence of hypertension. The conspicuous absence of murmurs virtually rules out rheumatic heart disease. The laboratory data conformed to the usual pattern of congestive heart failure in a patient with pitting edema of the legs, presumably a large liver, basal rales and a right-sided pleural effusion, although the electrolyte pattern was not as disturbed as one might expect in a waterlogged patient who had failed to respond to treatment with diuresis in the previous month. Indeed, the congestive heart failure did react to standard therapy in a somewhat encouraging fashion, although the patient's legs apparently remained edematous. However, three threatening symptoms intervened: profuse sweating on the second hospital day followed by a gallop rhythm, indicative of a dilated heart; abdominal pain after defecation, with a suggestion of gastrointestinal-tract bleeding; and the production of bloody sputum. Each of these new features has a bearing on the cause of this man's heart failure.

May we review at this time the electrocardiograms that were taken on admission and two weeks later?

DR. CHARLES S. KEEVIL, JR.: The initial tracing (Fig. 2) shows low voltage in the limb leads. Atrial fibrillation and a regular ventricular rate are demonstrated, indicating complete atrioventricular block, with the ventricular rhythm of nodal origin. In Leads I, AVL, V_2 and V_3 there are Q waves, which are suggestive of an anterior infarction, although of uncertain age. In the electrocardiogram done on the fourteenth hospital day (Fig. 3) a normal sinus rhythm has appeared, with a prolonged PR interval, constituting evidence of first-degree atrioventricular block. In Lead V_1 there is a definite small R wave that becomes smaller in Lead V_2 , and gives way to Q waves in Leads V_3 through V_5 . This pattern gives additional weight to the possibility of an anterior infarction, although the changes between this and the initial tracing may be merely due to electrode position and not necessarily the result of an extension of the process.

DR. BAKER: Will you comment on the height of the P waves in Leads 2 and V_1 ?

DR. KEEVIL: The P waves in Leads 2 and V_1 are certainly large enough to attract attention, although they are not above 3 mm. They suggest atrial hypertrophy, although they are not nearly as striking as those often seen in that condition.

DR. BAKER: They are not as high as the P waves that are sometimes seen in chronic cor pulmonale.

DR. KEEVIL: The P waves in chronic cor pulmonale are often more peaked and prominent.

DR. BAKER: The developments shown by the electrocardiograms, particularly the second tracing after the return to a normal sinus rhythm, impressed me with the likelihood of a previous anterolateral myocardial infarction. If we accept this diagnosis we must make the following assumptions: that the initial collapse, with dyspnea but no chest pain, ten weeks before admission was due to either pulmonary embolism or myocardial infarction, probably the former; that congestive heart failure, both left-sided and right-sided, developed on the basis of coronary-artery disease, possibly triggered by pulmonary embolism; and that the profuse sweating on the second hospital day was the expression of another vascular accident, probably myocardial infarction. The prolongation of the PR interval that appears in the tracing made a few days before death I find more difficult to explain on the basis of an infarct involving the lower part of the septum because such a change ordinarily accompanies right-coronary-artery occlusion.

We come to a consideration of the possible explanations for the hemoptyses in this case. A chronic bronchiectatic infection would account for the bloody sputum in the absence of pleural pain, but we have no reason to suppose from his symptoms before admission that this man had been suffering from bronchiectasis. However, if repeated lower-lobe pulmonary embolism and infarction had occurred, the infarcted areas could easily have been tucked away behind the basal shadows, particularly in view of the probable fluid on the right side. Therefore, I am inclined to attribute the hemoptyses to pulmonary infarction rather than to bronchiectasis, since the previous story did not point to infection and the x-ray picture was not characteristic of diffuse, severe pneumoconiotic changes. One might consider the prominent P waves that appeared on the second tracing a straw of evidence in favor of right-sided heart strain in the face of repeated pulmonary embolism. The character of the pleural fluid, with the large number of red blood cells, is also consistent with pulmonary infarction.

This line of thought is abruptly halted by the development on the seventeenth hospital day of severe abdominal pain, which is our next diagnostic problem. The studies done at the time of the ter-

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terminal episode yielded no evidence of rupture of the abdominal aorta intraperitoneally, acute small-bowel obstruction, rupture of a viscus, abdominal aortic dissection or a new myocardial injury. There were no new signs of trouble in the chest. The distended neck veins raise the question of cardiac tamponade from hemorrhage into the pericardial sac, but this condition would scarcely be compatible with the abdominal pain, and the venous distention in the neck can equally well be explained by left-sided and right-sided heart failure. The sudden character of the pain suggests another embolism. The fact that it again followed defecation implies that the lesser attacks of similar pain that had been occurring for about two weeks were more ominous than the cramps of muscular irritability due to an unexplained hypocalcemia, which apparently interested the House Staff at the time.

Several explanations of the terminal episode come to mind. First of all, was it embolism of the superior mesenteric artery, at first with small emboli and finally with either a large embolus or sufficient small ones to make collaterals ineffectual? In that event, symptomatic infarction of the small bowel either did result or would have resulted if the patient had lived longer. The source of such an embolus could have been either a mural thrombus in the left ventricle consequent to the myocardial infarct that I believe this patient had, or the previously fibrillating atria. It is also possible that the advent of abdominal pain not long after defecation was due to a smashing pulmonary-artery embolism. Under such conditions no new signs in the chest would be expected, the examination of the abdomen might be undiagnostic, as in this case, and the signs of heart strain would be similar to those described. It should be emphasized that the pain of lower-lobe pulmonary embolism, in particular, may be referred to the upper abdomen.

My diagnoses, therefore, are coronary-artery disease, with myocardial infarction and congestive heart failure, pulmonary embolism and infarction, particularly of the right lower lobe, and terminal embolism of the superior mesenteric artery. Some evidence of pneumoconiosis may have been found, but I don't believe that this man had chronic cor pulmonale.

DR. BENJAMIN CASTLEMAN: Would anyone like to offer any comments?

DR. WHITE: I agree that this man probably had extensive myocardial disease, and certainly coronary-artery disease is a more likely diagnosis than the other rare conditions that might simulate it. I was recently in Africa, and saw some of the patients with endomyocardial fibrosis, which is frequently found in East Africa and can give rise to extraordinary myocardial changes, abnormalities in the electrocardiogram and even calcification in the myocardium, but I have never seen in this part of the

world, a case of "EMF," as they call it, that resembled the ones that I saw in Kampala. However, that disorder does produce extensive myocardial disease, not on a coronary basis, with some calcification. Calcification is also seen with chronic pericardial disease, but that diagnosis wouldn't be consistent with this patient's history. The calcification in this case could have been in an area of myocardial infarction or possibly in a coronary artery.

The electrocardiogram seems consistent with severe myocardial disease of coronary origin, without the addition of something like cor pulmonale. The low voltage is prominent in the limb leads, a finding that is more common with extensive myocardial disease due to coronary thrombosis than any other cause. One sees it sometimes with pericardial disease and with myxedema, but I think those conditions are very unlikely in this case. The original attack ten weeks previously could have been a coronary attack because most of the cases of painless coronary thrombosis that I have seen have been associated with collapse. Therefore, this wasn't necessarily pulmonary embolism, although embolism is a strong possibility, and for that reason I tried to find a small infarct in the lung. Dr. Wyman doesn't agree, but I am still a little suspicious of that area. I believe that the terminal event was probably embolism.

DR. CASTLEMAN: Dr. Hardy, will you comment on the possibility of asbestosis?

DR. HARRIET L. HARDY: This patient could have had asbestosis, and yet not as a prominent part of the hospital episode. Asbestosis would account for the clubbed fingers and the relatively high hemoglobin, and could even have been responsible for the calcification because the inhalation of asbestos characteristically causes marked pleuritis and even calcification. In this case there may have been some fibrosis that developed over a long period and thus wasn't remarkable on the x-ray films. Asbestos bodies were probably present throughout the lung, but these merely reflect asbestos exposure and are not correlated with the pulmonary fibrosis of asbestosis, which is disabling. Since this patient had a sedentary job he might well have had asbestosis that was of no importance in his clinical course.

DR. CASTLEMAN: Dr. Wyman, do you think that streak of calcification could be in the pleura?

DR. WYMAN: It's in an odd place to be construed as in the pleura. Calcification in the pleura would lie in a plane perpendicular to the x-ray and not tangential, and therefore would not cast a thin linear shadow, as this does, unless an anomalous fissure was present. I'd like to agree with Dr. White and Dr. Baker about the probability of infarcts in this case, but I just can't recognize any evidence for infarction in view of the many pleural changes.

DR. CASTLEMAN: Is there considerable pleural thickening?

DR. WYMAN: I think the changes are primarily due to pleural fluid, but it is impossible to differentiate thickening in the presence of so much fluid.

DR. RICHARD A. FIELD: Dr. Hardy, is this case at all reminiscent of the man with asbestosis and a hidden carcinoma of the lung whom you reported?¹ He ended up with cardiac involvement from carcinomatous infiltration into the interstices of the myocardium.

DR. HARDY: Yes, it is, and I committed myself in the record that even with a negative sputum examination we might find a carcinoma of the lung in this man. We have seen 5 patients with asbestosis at this hospital, and 3 of them have been examined at autopsy and found to have bronchogenic carcinoma. The present patient had the same history of exposure as one of our patients.

DR. WHITE: I thought of suggesting that a malignant process in the heart must not be forgotten, but I don't believe that it would give this electrocardiographic pattern.

DR. HARDY: Since Dr. White has brought Africa into the discussion, I might add that I've just been abroad and learned about a high incidence of pleural mesotheliomas in asbestos workers in Africa, a report of which is about to appear.²

DR. CASTLEMAN: We have seen 1 asbestos worker who had a mesothelioma.

DR. KURT J. ISSELBACHER: How extensive was the exposure of the patient under discussion?

DR. HARDY: This man worked with asbestos for three years in a shipyard down in the holds of ships, spreading asbestos powder in a paste to cover pipes. He had previously worked in a variety of companies applying mixtures of asbestos, magnesium and other ingredients as linings for the boilers of ships. The asbestos is dry and powdery, and is mixed by the worker when he is ready to apply it. The present patient worked with asbestos alone and in the mixture described for thirty-five years, and since some of the work had been in confined spaces he certainly had been thoroughly exposed.

DR. WALTER W. POINT: Wouldn't it also be possible to explain terminal bowel infarction without implicating an embolus in the superior mesenteric artery? It should be pointed out that some patients in failure have been examined at autopsy and found to have bowel infarction with no embolus at all — merely arteriosclerotic narrowing.

DR. CASTLEMAN: That is the usual finding in patients in congestive heart failure.

DR. KENNETH T. BIRD: From the pulmonary viewpoint, the persistence of a loculated right-sided pleural effusion, with apparent clearing of the peripheral edema, is certainly a reasonable indication for a needle biopsy of the pleura, particularly in view of the known high incidence of associated carcinoma of the lung with pulmonary asbestosis. This

is an easy procedure, and I think it should often be employed in conjunction with the initial thoracentesis except in patients in overt congestive heart failure or with an obvious systemic disease causing a pleural effusion. When a pleural effusion persists in this age group one has to beware of an underlying carcinoma or even of a pleural membrane that has formed around a contracted lung, leading to the chronic recurrent effusion described by Stead et al.³ In any event, a needle biopsy of the pleura in this patient might have been helpful.

DR. BAKER: Would you do a needle biopsy even if the edema hadn't cleared up?

DR. BIRD: Probably not, although the biopsy findings might still be helpful in the management of the case.

DR. WHITE: Of course, one has to remember that right-sided hydrothorax in congestive heart failure is the last congestion to clear.

DR. BIRD: Yes, but I am impressed with the known association of asbestosis with carcinoma.

DR. JOHN H. KNOWLES: As a matter of cold, hard fact, I did a pleural biopsy on this patient, although with tongue in cheek. It was negative.

From the time of admission he had a very loud, persistent diastolic gallop rhythm, and he was never free of congestive heart failure. I thought that the principal problem could be summed up purely on the basis of heart disease and not asbestosis or bronchogenic carcinoma, and pulmonary embolism seemed a useful way of explaining the terminal episode.

DR. JOHN DENOVIAN: Dr. White, isn't it unusual for a fibrillating heart in a patient in severe failure to revert suddenly to normal sinus rhythm?

DR. WHITE: We frequently see paroxysmal fibrillation in a very sick patient, with a spontaneous return to a normal rhythm.

CLINICAL DIAGNOSES

Coronary-artery disease.
Congestive heart failure.
Infarction of small bowel.

DR. MYLES P. BAKER'S DIAGNOSES

Coronary-artery disease.
Myocardial infarction.
Congestive heart failure.
Pulmonary embolism and infarction, particularly in right lower lobe.
Embolism of superior mesenteric artery, terminal.
? Pneumoconiosis.

ANATOMICAL DIAGNOSES

Coronary-artery disease, with myocardial infarct, old, left ventricle and interventricular septum.
Mural thrombosis, left ventricle and atrium.

Endocardial fibroelastosis, left ventricle.

Arteriosclerosis of aorta, severe, with mural thrombus occluding orifice of superior mesenteric artery.

Infarction of small bowel, terminal.

Pulmonary infarct, organizing, right lower lobe.

Pulmonary asbestosis, with pleural and subpleural fibrosis, marked, and pulmonary fibrosis, slight.

Cor pulmonale.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: Professor Harrison, of the Postgraduate Medical School in London, is pathologist pro tempore this week, and I have given him the assignment of explaining this case.

DR. C. VICTOR HARRISON (London, England): This is a great privilege if a little of an embarrassment. I will put up on the blackboard so that I don't stray from the trail myself the problems that I think ought to be answered. There was first of all the incident of ten weeks before admission, followed by congestive cardiac failure; then there were the pleural effusions, the hemoptyses and finally the fatal collapse.

The final collapse was due to infarction of the small intestine. The patient had an infarct that started about 1 meter below the end of the jejunum and ended about 1 meter above the ileocecal valve. There was no embolus, but he had what must have been a fantastically rare lesion — a dreadful atherosclerosis in the aorta with a mural thrombus on top of it that had grown and flapped over the origin of the superior mesenteric artery. A completely fabulous thing, but there it was. I am a little disturbed because the extent of the infarction did not exactly correspond to the anatomical distribution of the whole of the superior mesenteric artery. It should have gone farther around, but possibly there was some collateral circulation through the inferior mesenteric artery. Histologically, the infarction was extremely fresh, fitting very well with the short duration of the final episode.

The heart was not very heavy, weighing only 350 gm. The size was, I think, due very largely to dilatation. Two possibly dissociated lesions were found — a cor pulmonale, with a right ventricular hypertrophy up to about 6 mm. in thickness at the conus, as well as a big, dilated left ventricle with a magnificent endocardial fibroelastosis! Although this man had had rheumatic fever and was in the hospital for three months, it doesn't seem to have done him much harm because the chordae tendineae of the mitral valve were of decent appearance and were quite slender, and the commissures of the aortic valve were not fused. I think we can forget the rheumatic story. There was, however, a remarkable fibroelastosis, with dilatation and relative thinning of

the left ventricular wall. Considering that the heart weighed 350 gm., there must have been considerable stretching to make the wall so thin. Among the chordae carneae were some ante-mortem thrombi. Histologically, elastic stains showed a greatly thickened layer of fibrous tissue interlaced layer by layer with elastic tissue (Fig. 4). Presumably, it had been present quite a long time. I don't know the cause of the fibroelastosis, but it was associated with diffuse ischemic heart disease. This man had calcification with about 30 per cent reduction in the lumen of the right coronary artery. In the two left coronary arteries there was very gross narrowing producing "pinhole" lumens with 80 or 90 per cent reduction.



FIGURE 4.

A large area of scarring was irregularly distributed beneath the thickened endocardium. The precise correlation of the chronic ischemic heart disease, the old myocardial infarct, the left ventricular dilatation and the fibroelastosis cannot be specified, but it is possible that they were related.

An explanation of the cor pulmonale is equally difficult. The right lung weighed 900 gm., and there was a fair bit of edema in it. Infarction of the greater part, nearly the whole, of the right lower lobe was found, as well as a reddish-brown thrombus in the lower-lobe arteries corresponding to it. The left lung had a few little scars, but no apparent

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infarcts. Both lungs were fairly firm to the touch, but the prosector did not describe any chronic interstitial fibrosis of the type one would expect in asbestosis. About 1 liter of fluid was present in each pleural sac. The visceral pleura was considerably thickened, but showed only patchy adhesions to the



FIGURE 5.

parietal pleura on each side. I think we can assume that the pleural fibrosis was due to asbestosis. Microscopically, we did find asbestos bodies, but they were not at all frequent — they had to be looked for. There were also odd little spots of rather nonspecific

fibrosis, which quite honestly I would not have recognized as due to asbestosis if I had not found the scanty asbestos bodies.

When we examined sections of the lung, we found that the impacted embolus in the right lower lobe had organized to an appreciable depth. I think there is little doubt that the embolus had been there for some time, and it seems fair to say that the original attack ten weeks previously was due to this embolus (Fig. 5). The rest of the lung was macroscopically free from infarcts, and the vessels of dissectable size did not show emboli, but microscopically in a few arteries there were recently organizing emboli.

DR. WHITE: Where did the pulmonary emboli come from?

DR. HARRISON: The legs were examined as far as the popliteal space, Dr. White, but nothing was found. My own suspicion is that the source was in the calf because the pulmonary emboli were such little chaps.

The only other findings were passive congestion of the liver, chronic cholecystitis with some calculi, and a little right adrenal adenoma.

DR. WHITE: Did you find pulmonary or pleural calcification?

DR. HARRISON: We found calcification only in the coronary arteries. It is described as severe.

DR. WHITE: That linear calcification shown in the x-ray film may have been in a coronary artery.

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The New England Journal of Medicine

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MEDICAL EDUCATION AND THE WMA

THE Second World Conference on Medical Education, the first having been held in London in 1953, has already had a preliminary bout of advance publicity in the *Journal* of April 16, 1959. In this second such conference to be held, after a decent interval of six years, medical educators and others interested in medical education from fifty countries are expected to join forces in Chicago's Palmer House from August 29 to September 4, under the sponsorship of the World Medical Association, the World Health Organization, the Council for International Organizations of Medical Sciences, and the International Association of Universities. President Eisenhower, who helped organize the National Fund for Medical Education during his presidency of Columbia University, is not only patron of the Conference, according to a recent release, but has been invited to attend it.

No fewer than 125 speakers are scheduled to participate in the Conference, as are delegations from both the Union of Soviet Socialist Republics and Poland. Neither of these countries is represented in the World Medical Association.

The Conference, which, through the wizardry of modern technics of communication, will be carried on simultaneously in English, French and Spanish, will be divided into four sections. Each section will devote two and a half days to its allotted subject — Section I to Basic Clinical Training for All Doctors, Section II to Advanced Education for General and Specialty Practice, Section III to The Development of Teachers and Investigators, and Section IV to Continuing Medical Education. Various social events will reach their zenith, although presumably in the evening, at an official Conference dinner.

The President of the Conference will be Dr. Raymond B. Allen, chancellor of the University of California in Los Angeles. He will be aided by two deputy presidents: Dr. Ray F. Farquharson, Sir John and Lady Eaton Professor of Medicine, University of Toronto, and Dr. Victor Johnson, director of the Mayo Foundation for Medical Education and Research of the University of Minnesota's Graduate School of Medicine. According to Dr. Allen, "It is the purpose of this Second World Conference on Medical Education to pass on to the young in mind and heart the experience and learning of the ages" — certainly a commendable objective.

At the end of the Conference those who are capable of further intellectual absorption will move on to Montreal, possibly by water, since the passage can now be accomplished without an overland portage, to reconvene as the Thirteenth General Assembly of the World Medical Association at the Queen Elizabeth Hotel, across the border. Here, on Monday afternoon, September 7, the Assembly will open with the tenth annual medical editors' conference, which

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