CLINICO-PATHOLOGICAL CONFERENCES OF THE MOUNT SINAI HOSPITAL

Edited by Fenton Schaffner and Hans Popper

Clinico-Pathological Conferences of The Mount Sinai Hospital

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Preface

Knowledge in clinical medicine and in pathology has developed on the basis of careful observation with initially tentative correlations. These correlations have been strengthened by two approaches; one is accumulation of incidents and coincidences selected empirically, and the other is the gradual development of medical knowledge based on experimentation as well as on deductive reasoning. The scientific method in medicine attempts to elevate correlations from tentative ones to certainties. However, the nature of the techniques makes absolute certainty virtually impossible when applied to the individual case. Thus, judgment of the significance of symptoms and signs based on subconscious reasoning and then called the art of medicine tempers the scientific approach.

The clinical-pathologic conference represents a concentrated effort of this scientific method in medicine both by the clinician and the pathologist. Clinicians have the privilege of considering their observations unhurriedly in relation to the available literature, which cannot be done at the bedside, while the pathologist in addition has the opportunity to apply any diagnostic method in his field. The clinical pathological conference therefore becomes an exercise in reasoning, judgment and correlation, a tool in teaching and a stimulus to research exerted by the illness of one patient.

At present, the scientific method of medicine stimulated by observations and clinical pathological correlations in individual patients is challenged by the fundamental approach of the human-disease–oriented biologist, who prefers to start from a simple abnormal biological process not necessarily studied in man. This has produced an increasing amount of information useful in clinical management, and progress in medicine will probably depend on balanced utilization of case-oriented medicine and basic science initiated by a study of biologic processes. While the latter approach has not been neglected at the Mount Sinai Hospital, emphasis on the former, which includes the clinical pathological conference, has led this institution to its present position in medicine.

As new grafts on this old institution, we choose to refer to one of the architects of this development for the history of research at the Mount Sinai Hospital before presenting the recent conferences which have attempted to live up to the standards set by the previous generations. This is also the occasion to express our appreciation to the large number of staff members who not only conducted and participated in the conferences but also cheerfully edited their remarks.

The Editors

Introduction

By GEORGE BAEHR, M.D.

With the rise of pathology in Europe during the 19th century as one of the fundamental medical sciences, conferences of clinicians with pathologists around the autopsy table became routine. Both found that they had much to learn from one another by mutual reexamination and reevaluation of clinical observations made during life with the findings uncovered at necropsy.

Toward the latter part of the 19th century, enterprising American physicians who had been trained in our inferior medical institutions of that day began to flock after graduation to the great European centers of medical education in order to acquire knowledge of the rapidly developing sciences of chemistry, bacteriology, physiology, and pathology. Upon their return, they established modern laboratories in our hospitals and medical schools and revolutionized clinical medicine in this country. Some, like William Henry Welch at Johns Hopkins, created the modern American medical school which today leads the world.

In this country Richard Cabot of Boston deserves the credit for employing the clinico-pathological conference as an effective tool for the education of undergraduate medical students. The conferences which he initiated shortly after the turn of the century were soon imitated by medical schools throughout the country.

In 1919, soon after the conclusion of World War I, we began to conduct a somewhat different type of weekly clinico-pathological conference at the Mount Sinai Hospital in New York. Whereas the conferences popularized by Cabot consisted essentially of a more or less impromptu colloquy between the clinician and the pathologist and were conducted as a teaching exercise for undergraduate medical students, the conferences at the Mount Sinai Hospital were carefully prepared in advance and conducted in a more sophisticated fashion. They were designed primarily for the continuing education of experienced physicians, not only for the Hospital's medical staff but for the benefit of practicing physicians throughout the metropolitan area who could find time to attend.

Held at first in the autopsy room of the Hospital, the conferences attracted so many physicians that it soon became necessary to enlarge the room by the addition of a small amphitheatre. In succeeding years, physicians from the community outside the Hospital attended in such numbers as to incommode the Hospital's own staff, and by 1929 it became necessary to move the conferences to a large auditorium which could hold the crowd of weekly visitors.

Today, clinico-pathological conferences are no longer concerned solely with arriving at an unbiased inventory of errors and omissions in diagnosis and therapy and a concluding critique of how they might be avoided. The rapid advances in medical technology and the growing multitude of specialties and subspecialties of medicine and pathology often require the participation of highly qualified experts in special fields of research to supplement the knowledge and experience of the pathologist and the reporting clinician. The discussion of each case has become a comprehensive review of existing knowledge concerning the disease process and the values of alternative methods of therapy.

The careful reader of these reports of selected clinico-pathological conferences will be rewarded with a wealth of information of practical value which cannot be found in any single monograph. They summarize some of the most significant clinical and therapeutic experiences of a major teaching hospital.

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Pulmonary Disease in Mother and Daughter

A 31 year old white married female commercial artist was admitted to The Mount Sinai Hospital on October 28, 1957 with shortness of breath, fever and a cough.

In 1952, during a routine examination, a pulmonic diastolic murmur was heard. Fluoroscopy revealed a large pulmonary artery. While in Colorado a year later, the patient first noted shortness of breath and palpitations. At that time a left vocal cord paralysis was discovered. During the next year, the patient did well and underwent a tonsillectomy for recurrent tonsillitis. Two years prior to admission, her shortness of breath gradually increased. She coughed persistently and on one occasion coughed up about an ounce of bright red blood. Later that year her liver and spleen became palpable and a transient homonymous hemianopsia developed. In May 1956, spontaneous ecchymoses were seen. At that time a platelet count was normal but a tourniquet test was positive. The patient complained of chest pain and ankle edema three months before coming to the hospital. The diastolic murmur was louder and an apical systolic murmur had appeared. On October 1, 1957 she experienced fever, muscle aches, increasing dyspnea and ankle edema. She had a positive hepatojugular reflux and was treated with penicillin, Mercuhydrin® and, for the first time, digitalis. She improved until three days before admission when she again noted fever, muscle pains, cough productive of yellow and bloody sputum, severe shortness of breath and weakness. She was treated with penicillin, streptomycin and mercurial injections without improvement. Throughout this febrile illness, examination of the lungs was normal.

Family history: In 1952 the patient's mother was admitted to The Mount Sinai Hospital because of shortness of breath, edema and abdominal swelling. The mother's symptoms dated back four years when she first experienced shortness of breath on exertion. These symptoms progressed and by the time of her admission she had anasarca. Six paracenteses were performed during the months prior to her admission. Two Papanicolaou smears of the ascitic fluid were reported positive for malignant cells but cell blocks were repeatedly negative. On admission the mother had distended neck veins with a positive hepatojugular reflux and systolic pulsations of neck veins and liver. Her heart was markedly enlarged. P2 was accentuated. A2 and M1 were normal. Faint systolic murmurs were heard over the pulmonic and aortic areas. The abdomen was tense with ascitic fluid. After paracentesis the liver was felt a handbreath below the right costal margin. No other organs or masses were felt. Pelvic examination was normal. The significant abnormal laboratory data during the mother's hospitalization were as follows: A/G ratio 3.0/3.5, bilirubin 1.7 mg%, urine urobilinogen 1:80, alkaline phosphatase 32 KAU, cephalin flocculation 2+, prothrombin time 17.5/13 seconds (patient/control), thymol turbidity 8.0 units, BSP 43 per cent,

and venous pressure 240 mm with a rise over the top of the manometer with right upper quadrant pressure. Circulation time was 23 seconds arm-to-tongue. Electrocardiogram showed right ventricular hypertrophy. Chest x-ray revealed the heart to be considerably enlarged and globular with a prominent pulmonary artery, the configuration suggesting the possibility of an inter-atrial septal defect. Paracentesis fluid was negative for tumor cells. The mother experienced chest pain and hemoptysis, leading to shock and death five days after admission.

Physical examination: The patient was a well developed, well nourished, young white female in acute distress owing to a persistent brassy cough, shortness of breath and orthopnea. Temperature 103°; pulse 104, regular but weak; respirations 32; blood pressure 85/70 in both arms. Cyanosis of the lips and nail beds was present. The head, eyes, ears, nose and throat were normal. The neck veins were distended. A markedly positive hepatojugular reflux was seen with systolic pulsations of the neck veins. The chest wall and diaphragms moved well. The lungs were clear to auscultation and percussion. The PMI was palpated in the fifth intercostal space 2 cm. left of the midclavicular line. A forceful systolic impulse was felt midway between the apex and the left parasternal border. There was a gallop rhythm. P2 was markedly accentuated; M1 and A2 were normal. A grade III blowing apical systolic murmur, a grade II soft prolonged pulmonic diastolic murmur, and a grade II soft pulmonic systolic murmur were heard. The liver was felt one fingerbreadth below the right costal margin. All peripheral pulses were palpable and no edema was seen.

Laboratory data: Hgb. 14.6 G., WBC 21,600, segmented neutrophils 57%, bands 16%, lymphocytes 21%, monocytes 4%, atypical lymphocytes 2%, BUN 42 mg.%, blood sugar 95 mg.%, A/G ratio 3.2/3.4, antistreptolysin-O titer 166 units. Influenza hemagglutination titers were as follows:

Group A	NY-3 (1953)	1:160
	Denver (1957)	Neg.
	MSH-1 (1957)	1:160
	Far East (1957)	Neg.
	R.E. MSH	Neg.
Group B	B-G1	Neg.

Electrocardiogram showed marked right ventricular hypertrophy. X-ray of the chest showed an increase in the prominence of the pulmonary vessels. At the level of the third right interspace, there was a band-like density. The heart was enlarged with rounding of the ventricular contour. The aortic knob was small. The right atrium seemed prominent as in an interatrial septal defect.

Course: The patient was treated with Chloromycetin®, streptomycin, penicillin and digoxin. She continued febrile. Her respiratory distress and cyanosis increased and she ceased breathing 48 hours after admission.

Dr. Charles Friedberg:* This is apparently a two-in-one case. The major prob-

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lem is concerned with a 31 year old white married female commercial artist who was admitted with cough, shortness of breath and fever. The date, October 28, 1957, may have some bearing because of the prevalence of respiratory diseases, especially influenza.

During a routine examination at the age of 26, a pulmonic diastolic murmur was heard and fluoroscopy revealed a large pulmonary artery. Some findings have a bearing out of proportion to anything else that is stated. Such a finding is the one about a pulmonic diastolic murmur, which may imply pulmonic valvular insufficiency in the sense of intrinsic valvular disease, but is extremely rare. As a rule, this finding denotes pulmonary insufficiency associated with pulmonary hypertension.

We then have to determine the nature of the pulmonary hypertension and whether it is primary, or secondary to congenital heart disease with left-to-right or combined shunts, or to intrinsic pulmonary disease such as sarcoidosis or to pulmonary vascular disease.

In 1953 a left vocal cord paralysis was noted. While that can be due to a variety of causes, in this instance where we already have reason to suspect pulmonary hypertension, we have to assume that a very large pulmonary artery is compressing the recurrent laryngeal nerve supplying the left vocal cord.

She noted a persistent cough and on one occasion coughed up about an ounce of bright red blood. In the absence of anything to suggest a febrile infectious disease, this could possibly denote a pulmonary infarction or some other pulmonary vascular obstructive lesion. Recurrent thrombosis of the pulmonary vessels or recurrent embolism also may be responsible for pulmonary hypertension. Furthermore, thrombosis indirectly can be part of the syndrome of pulmonary hypertension and narrowing of the vessels.

In 1956 spontaneous ecchymoses were seen and one asks whether that denotes some hematologic disease for which we have no additional data. Pulmonary hypertension can occur as a result of thrombosis due to schistosomiasis in the lungs. We have no evidence that this possibility was seriously entertained or investigated.

Three months before admission, the patient complained of chest pain and ankle edema. Examination revealed that the diastolic murmur was louder and an apical systolic murmur had appeared. We might then begin to wonder whether we are now dealing with heart failure with a functional tricuspid regurgitation accounting for the systolic murmur.

On October 1, 1957 she experienced fever, muscle aches, increasing shortness of breath and ankle edema. We now come to the final illness of a few weeks before her admission to the hospital. She was treated with penicillin, Mercuhydrin® and, for the first time, digitalis; in other words, a combination of drugs for infection and for congestive heart failure.

She was admitted with what sounds like some pulmonary infection, possibly pulmonary infarction, at a time of the year when pneumonia is prevalent, in addition to a suggested pulmonary hypertension.

The family history is of interest. We have a story that the mother had evidence

of right heart failure and with an accentuated P2, perhaps something related to the type of disease we are encountering in the daughter.

The significant abnormal laboratory data during the mother's hospitalization were hypoalbuminemia and moderate hyperglobulinemia with reversal of the A/G ratio, elevation of serum bilirubin and urinary urobilinogen, increased alkaline phosphatase, BSP retention and an elevated venous pressure which rose over the top of the manometer with right upper quadrant pressure.

All this evidence of right heart failure and abnormal liver findings theoretically could mean intrinsic hepatic disease. In the presence of advanced right heart failure, there is no reason to invoke liver disease because all these findings could result from an extremely congested liver in a patient who has sufficient heart failure to cause tricuspid insufficiency.

The heart was described as considerably enlarged and globular with a prominent pulmonary artery, suggesting the possibility of an interatrial septal defect.

We must assume that this is only a suggestion because the configuration of the heart associated with an atrial septal defect can look very much like that associated with other conditions. The point of interest here is that again we have a picture which suggests to us that the same kind of pulmonary hypertension which we are predicating in the daughter was present in the mother.

The ascitic fluid was negative for tumor cells. The mother experienced chest pain and hemoptysis, leading to shock and death five days after admission.

I am not clear whether shock occurred as a result of the paracentesis, in which case we might not attach any specific diagnostic importance to it. On the other hand, sudden death as a result of very minor procedures is almost characteristic of primary pulmonary hypertension.

The patient, the 31 year old daughter we started with, had acute distress with a persistent brassy cough, shortness of breath and orthopnea, and not only a low blood pressure but a low pulse pressure. Whether this is due to the acute pulmonary infection or whether this is an additional evidence of the type of disease we are postulating in the pulmonary artery is difficult to say.

There was cyanosis of the lips and nail beds. Ordinarily we would have to lay more stress on the significance of the cyanosis because in cases with intrinsic or primary pulmonary hypertension, if cyanosis occurs at all, it is more likely to be of the peripheral type due to the diminished blood flow and tissue anoxia, and not to a shunting of blood as occurs in congenital heart disease with severe pulmonary hypertension. In this instance, however, the clinical history strongly suggests that the patient may have had pneumonia and, therefore, may have had some cyanosis of the lips due to that disease.

The positive hepatojugular reflux with systolic pulsation of the neck veins indicates tricuspid regurgitation due to right-sided heart failure, and such pulsations may occur in cases of pulmonary hypertension.

The fact that the lungs were clear to auscultation and percussion is quite a surprise in the light of the rest of the story because, on the basis of her cough and expectoration of bloody sputum and a fever, one would ordinarily think that at this time of the year the patient had some respiratory infection and, more specifically, pneumonia.

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If these findings are correct, she probably had a lesion which was chiefly or exclusively interstitial, and perhaps this accounts for the absence of more findings.

The forceful systolic impulse midway between the apex and the left parasternal border also suggests an increase in the size and pulsation of the right ventricle, a finding associated commonly with pulmonary hypertension. The gallop rhythm is not specific but may very well indicate failure of the right ventricle, a finding encountered frequently in cases of primary pulmonary hypertension.

The white blood count was 21,600. At this time of the year, with influenza prevalent, I would have predicated that she had a viral pneumonia, which usually we expect to be associated with a normal or a low white count. This high white count makes us wonder if this is just an exceptional finding or if some complication exists such as a secondary staphylococcus or streptococcus infection.

The electrocardiogram showed marked right ventricular hypertrophy with the sharp peaked P waves, so-called pulmonary P waves, which are commonly found in cases of right ventricular hypertrophy and congenital heart disease. Lead V_1 shows a very prominent R, also indicative of a right ventricular hypertrophy.

The suggestion has been made, at least with the mother and perhaps also in this patient, that the x-ray film suggested the presence of an interatrial septal defect. The electrocardiogram associated with this condition is characterized by a pattern that looks like an incomplete right bundle branch block, that is, with an RSR' pattern, which is absent in this case.

X-ray films of the chest showed an increase in the prominence of the pulmonary vessels. At the level of the third right interspace, there was a band-like density.

Dr. Rabin will say a word about it.

Dr. Coleman Rabin:* In the axillary region there is a triangular density which seems to occupy a small part of the lateral part of the anterior segment of the right upper lobe.

The heart is enlarged with rounding of the ventricular contour. Most striking is a prominence of the left pulmonary artery. There is apparently an increase in the vascular markings that extends almost to the periphery. Most of this is probably due to venous congestion. The periphery of the lung is not highly vascular as one might expect in instances of interatrial or interventricular septal defect. The aortic knob is small. The right atrium seems prominent.

Dr. Friedberg: In summary, the case is that of a young woman in her 20's and early 30's with pulmonary hypertension of a severe degree. That this pulmonary hypertension is not secondary to some other cause seems apparent from the absence of other findings. Interatrial or interventricular septal defects, which might give this type of x-ray finding, may be associated with concommitant pulmonary hypertension, do not seem to be likely here because of the other physical findings. For example, we might have expected that long before there was a pulmonary diastolic murmur, she would have a murmur suggesting a ventricular septal defect. It was not heard early or late although before death additional murmurs had appeared. Some of the evidence indicating that this was probably not an interatrial septal defect was discussed. There was no murmur to suggest a patent ductus although, at the stage in which the shunt is reversed,

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the murmur may be atypical. Nothing is present to suggest that this patient had sarcoidosis, nor is there anything to indicate a long history of intrinsic pulmonary disease. On the other hand, we do have a woman in the age group in which this disease is most common, with a history that is very suggestive of primary pulmonary hypertension.

We do not have a story to suggest primary embolic disease which then produces pulmonary hypertension. Perhaps many of the cases we call primary pulmonary hypertension are of this kind.

Now, for the more immediate history, I think that some kind of a viral pneumonia with perhaps secondary infection to account for the marked leukocytosis was present, despite the absence of pulmonary findings on physical examination. Interstitial pneumonitis is a possibility, but that is not what the x-ray film denotes.

Before we go on with the pathology, would you say a word, Dr. Aronson, about the findings on bacteriological or immunological examination?

Dr. Betty Aronson:* This patient had a hemagglutination-inhibiting antibody titer of 1:160 against the New York 3 and the Mount Sinai 1 strains of Influenza A. These are strains which have been present in the population since 1953, and approximately 65 per cent of the adults who have been tested during the current epidemic have antibody titers against these particular strains. Therefore, she is entitled, on the basis of her age and length of residence in this area, to have antibodies against the New York 3 strain.

We also received a fairly fresh lung specimen from this patient and we failed to isolate influenza from her lung. All the evidence seems to indicate that, within 24 hours after death due to influenzal pneumonia, the chances of recovering the virus from lung is very high, and this was, I believe, a 10-hour post mortem specimen. I must add that her lung suspension appears to be pathogenic in tissue culture and we have not yet been able to identify the source of this cytopathic tendency. It is non-bacterial. Even if we isolate a virus that can be identified, whether that has any etiological relationship to the disease, I cannot say.

Dr. Friedberg: Does the 1:160 antibody titer with New York 3 and Mount Sinai influenza strains mean that there was infection with these viruses?

Dr. Aronson: Yes. The patient at some time in the past four years had experience with these strains.

Dr. Friedberg: Do you find these increased antibodies in many cases?

Dr. Aronson: Yes. About 65 per cent of adults have demonstrable antibody titers.

Dr. Friedberg: Dr. Rabin, did this patient have pneumonia on the 8th day?

Dr. Rabin: In the x-ray film made at that time, the heart was seen to be enlarged to the left, but much of this is caused by the right ventricle. The pulmonary artery is large and some branches are still large, but toward the periphery there are no large pulmonary branches. On the left side, except for the main artery, all other vessels are small. This is what is seen in patients who have hypertension of the pulmonary circuit and who have diminished blood flow through the lungs.

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The remainder of the picture, as far as the vessels themselves near the root are concerned, is identical with that which are found in interatrial septal defect.

The triangular lesion in the right upper lobe is largely due to atelectasis with perhaps some consolidation of the lung. This indicates that the patient had a bronchopulmonary infection.

Dr. Richard Bader:* I think that this patient had pulmonary hypertension, the cause of which is unknown. The cyanosis can be explained on one of two bases, first, on a peripheral basis, and, secondly, and only theoretically, because of the right-sided hypertension, on a right-to-left shunt. Patients can experience chest pain with enlargement of the pulmonary arteries, as occurs in pulmonary emphysema, asthma, and any congenital lesion which enlarges the pulmonary artery.

Dr. Hans Popper†: At the autopsy we are interested first to examine the skeletal muscles to see if vascular changes could be demonstrated, and the vessels in the skeletal muscles were entirely normal. We saw no significant change in the bone marrow.

To paint the background for the actual disease which we are discussing, we turn first to the abdominal organs. In the colon an extensive circumscribed hemorrhage was seen which may or may not have been the result of congestion or some manifestation of shock.

Grossly, the kidney appeared entirely normal except for severe passive congestion. The vessels in the medullary portion showed acute congestion. The spleen was enlarged to more than 200 gms. with congestion and slight degenerative vascular changes.

The liver was moderately enlarged to almost 1600 gms. On the surface, congestion was seen which was not severe. The lobular architecture in general was still well preserved. Liver cells had disappeared around the central vein because of marked passive congestion but in the portal areas not too much congestion was present.

Death occurred from cardiac failure with acute and not yet chronic congestion. The heart weighed 420 gms. It had a globular appearance with a soldier's patch anteriorly. In the left heart some blunting of the apex was noted but the valves appeared entirely normal. No septal defect was found and the aorta contained no arteriosclerosis. In the myocardium there was some circumscribed or interstitial fibrosis. The right heart was markedly enlarged, thick walled and dilated. The heart muscle was of extreme thickness with flattening of the papillary muscle. Histologically, leukocytic infiltration was seen and a few cells accumulated in the vessels and were in the process of escaping from it. This is called a nonspecific type of an interstitial myocarditis in a hypertrophic heart. The right atrium was tremendously dilated. The auricular appendage was large and the tricuspid valve incompetent. The right and the left ventricles were almost equal in thickness. The pulmonary artery was dilated and revealed arteriosclerosis. On section it was very fatty with excessive fibrosis, narrowing of the muscular layer and some disappearance of elastic fibers, and appeared as though it were taken from an arteriosclerotic aorta.

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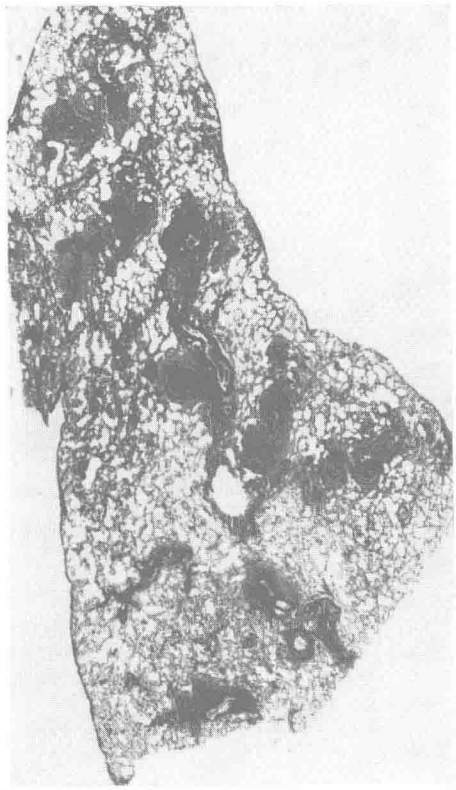

FIG. 1. Nodules in the lung parenchyma produced by arteritis (8×—chromotrope aniline blue stain).

The lung showed few changes grossly. The pleura was normal. Some emphysema was seen in the periphery. The tracheobronchial lymph nodes were somewhat enlarged. Some fat was apparently deposited within, having been drained from something which had broken down. On cut section of the lung, distinct nodularity was seen (Fig. 1). The nodules varied in size and we suspected that most of the nodules were related to vessels. In the left upper lobe, two old and probably tuberculous scars were found. The lesions in which we were most interested were the vascular ones. The round nodules were extremely thick, muscular pulmonary arteries. Smaller arteries, but still muscular, were completely fibrosed (Fig. 2).

Some arteries showed intraluminar proliferation with distinct inflammation, a severe arteritis (Fig. 3). In any other organ this would be called a polyarteritis nodosa. However, the only organ in which periarteritic changes were found was the lung and, therefore, this is pulmonary polyarteritis.

The arterial muscle fibers were disintegrated. The whole wall of the pulmonary arterial branch in some areas had fallen to pieces. Around these arteries were edema and cellular infiltration. The inflammation and fibrosis extended outward

FIG. 2. Fibrotic occlusion of small pulmonary arteries with surrounding emphysema (60×—H & E).

from the arteries into the interalveolar spaces. Such a pulmonary polyarteritis is a counterpart of polyarteritis in other organs not only in its similar histologic appearance but possibly also etiologically. We know that some instances of polyarteritis are unquestionably hyperergic, but in these instances there is involvement of the peripheral arteries and veins (1). We separate this hypersensitivity angiitis in the periphery from the real periarteritis or polyarteritis nodosa, which we now assume is not the result of hypersensitive reaction but something else. Some polyarteritis in the periphery is doubtlessly the result of increased systemic arterial hypertension.

We may have the same situation in the pulmonary circulation. In proven increased pulmonary hypertension, as in a mitral stenosis or secondary pulmonary hypertension, this picture can be produced (1, 2). In this case we have a primary pulmonary hypertension because we were unable to find any changes in the heart to explain it, and it, therefore, must originate in the lungs.

In a later stage of the lesion, near a bronchial artery, many vessels were seen between the pulmonary arteries and the bronchial arteries. These vessels came partly from granulation tissue and were probably not newly formed around the

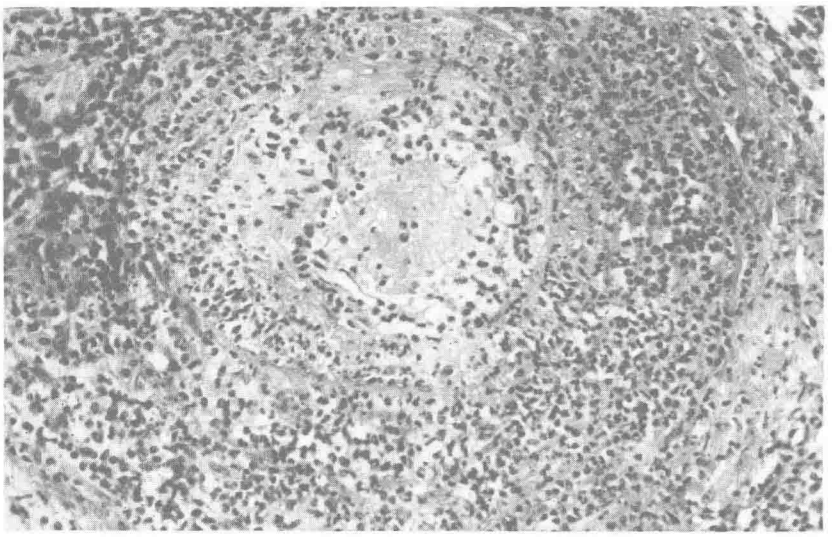

FIG. 3. Acute pulmonary arteritis similar to peripheral polyarteritis with periarterial inflammation (160×—H & E).

artery but were dilated bronchial artery branches (3, 4). Therefore, part of the inflammation became a collateral or compensatory hyperemia of bronchial vascular origin.

In the branches of the original pulmonary artery, these vessels were markedly proliferated and they have been described under the name of plexiform capillaries with glomus-like formation because they resemble the glomus in the periphery (4, 5).

In summary, the following changes were present in the vascular system: an arteritis in the pulmonary arterial branches, dilatation of pulmonary veins, dilatation of the bronchial arterial branches, and changes in the bronchial artery vascularization.

In the polyarteritic granulation tissues, new vessels from the bronchial artery grew around and into pulmonary vessels, and capillaries connected with the pulmonary artery. The extensive arterial anastomoses may have been the result rather than the cause of pulmonary hypertension (3, 5).

In the literature, some claim that the anastomoses are arteriovenous (6) while others state that they are between the pulmonary and bronchial arteries (3, 4, 5). The multitude of vascular anastomoses partly obstructs the blood flow and partly leads to shunts within these vessels. As the process becomes clear, severe fibrosis of vessels develop which makes the pulmonary hypertension permanent. The arterial elasticity is destroyed. In the presence of this pulmonary hypertension, arteriosclerosis in the middle-sized pulmonary branches and even in the larger pulmonary branches is secondary to the increased pressure.

As to the cause of death, in the base of the right upper lobe there were yellow areas of consolidation. Some central necrosis was found in the consoli-

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