JAUNDICE FOLLOWING PULMONARY INFARCTION IN PATIENTS WITH MYOCARDIAL INSUFFICIENCY: *I. A Clinical Study*

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JAUNDICE FOLLOWING PULMONARY INFARCTION IN PATIENTS WITH MYOCARDIAL INSUFFICIENCY

I. A Clinical Study

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INTRODUCTION

Jaundice occurring in myocardial insufficiency is a well recognized condition. It is seen most frequently in patients with long standing heart failure, particularly in patients with mitral or tricuspid disease and auricular fibrillation. The studies of van den Bergh and Snapper (1913), Lepehne (1921), Eppinger (1923), Feigl and Querner (1919), McNee (1922–23), Fishberg (1923), Andrews (1924), and others, have shown that jaundice in myocardial insufficiency is due to an increase of the bilirubin content of the blood serum. The subject we wish to discuss is the sudden appearance of jaundice or the marked increase in the jaundice, which occurs after pulmonary infarction in patients suffering from myocardial insufficiency.

The only references in the literature that we have been able to find in which a specific relationship between jaundice and pulmonary infarction has been suggested have been those of Schottmüller (1914), Eppinger (1923), and Libman (1923). There are many other instances in which pronounced jaundice has been associated with pulmonary infarction, but the significance of the association has evidently not been appreciated; at least, it was not mentioned or discussed. Among those are reports by Feigl and Querner (1919), Oertel (1910), and Mann (1907). Of especial interest in this regard is the statement of Matthes (1925), that one of the characteristic features of jaundice in patients with myocardial failure, is its sudden appearance, although no reference is made to the possible dependence of the jaundice on pulmonary infarction.

Schottmüller (1914) recorded a case in which jaundice occurred after a pulmonary infarction and he felt that the jaundice was due to the presence of hematin in the serum. However, he did not state whether or not the patient had myocardial insufficiency or whether he had an increased bilirubinemia as well.

There are other instances in patients with myocardial insufficiency in which jaundice may *seem* to appear suddenly. These instances occur most frequently during convalescence or during a period of marked improvement. This condition is not to be confused with the sudden appearance of jaundice that occurs after pulmonary infarction; because it is not at all unusual for the jaundice in cardiac patients to become more distinct when the cyanosis disappears, since the cyanosis tends to mask the icterus. Another explanation for this phenomenon may be the wide fluctuation of the bilirubin content of the blood plasma which may occur from day to day even while the patient is improving. This type of jaundice may be easily differentiated from the type that appears following pulmonary infarction since, in the one case the patient continues to improve, while in the other he usually becomes progressively worse.

We wish to report in this first communication, ten cases in which jaundice appeared for the first time, or became more intense, after the occurrence of pulmonary infarction in patients with myocardial insufficiency; and later we also wish to record the results of some experiments which deal with the underlying mechanism of jaundice in such cases.

REPORT OF CASES

Case 1. A man with severe myocardial insufficiency; condition unimproved with usual therapy; three days after pulmonary infarction sudden appearance of jaundice and urobilinuria; progressive myocardial failure; death sixteen days after pulmonary infarction.

History. A colored man, aged 52 years, with no previous history of syphilis, entered the Johns Hopkins hospital complaining of shortness of breath and swelling of the legs. For one year he had had symptoms of myocardial insufficiency, these symptoms having become considerably worse during the past month.

On examination, he was found to have the physical signs of aortic insufficiency together with the usual evidences of moderately severe congestive heart failure. The liver was enlarged. The sclerae were not icteric. There was no anemia and the white blood count was 10,800 per cmm. The urine contained neither bilirubin nor urobilin.

Course before pulmonary infarction. Following the usual forms of therapy the patient's condition remained stationary. He had numerous attacks of paroxysmal dyspnoea.

Course following pulmonary infarction. On the fourth day after admission, the patient was seized with a pain in the chest, which was followed by tachycardia, increased respiratory distress, paroxysmal coughing and the expectoration of bloody sputum. The temperature rose a degree and one-half above the previous average temperature (99° to 101°F.). *Three days following the infarction the sclerae, became jaundiced and urobilin appeared in the urine.* The jaundice deepened, myocardial insufficiency increased and death occurred sixteen days after the development of the infarction.

Necropsy findings. The important necropsy findings were: syphilitic aortitis extending to the vessels of the aortic arch; scars in the myocardium; cardiac hypertrophy; mural thrombi in all the heart chambers; infarct of the spleen; *embolic thrombi in the pulmonary arteries with multiple infarctions of the lungs; bronchopneumonia; extreme chronic passive congestion of the liver.*

Case 2. A man with severe myocardial insufficiency; temporary improvement; two days after pulmonary infarction appearance of jaundice, bilirubinuria and urobilinuria; hemorrhages into skin; progressive myocardial failure leading to death seven days after pulmonary infarction.

History. A white man, aged 37 years, with a previous history of acute rheumatic fever, entered the hospital complaining of shortness of breath and swelling of the legs. He had had symptoms of myocardial insufficiency for six months.

On examination, he was found to have the physical signs of aortic valvular disease, together with the usual signs of severe myocardial insufficiency of the congestive type. There was deep cyanosis but no jaundice. The liver was enlarged. There were frequent attacks of coughing with the expectoration of blood tinged sputa. There was no anemia and the white blood cells were 12,400 per cmm. The urine did not contain any bilirubin.

Course before pulmonary infarction. For three days after admission, there was definite improvement in his condition; the edema diminished, the blood tinged sputa disappeared, and cyanosis became less marked.

Course following pulmonary infarction. Three days after admission the patient had a chill, a severe attack of coughing and he expectorated a small amount of blood. His respiratory rate increased, the pulse rate became more rapid, and cyanosis deepened; these symptoms were unaccompanied by pain or fever. On the following day a petechial eruption appeared on the chest, abdomen and the upper part of the legs and arms.[1] The severity of myocardial insufficiency in-

[1] The eruption was part of the hemorrhagic phenomena occasionally seen in patients with myocardial insufficiency and particularly in patients with myocardial insufficiency and jaundice. These hemorrhages may be due in part to extensive liver damage.

creased. *Two days following the pulmonary infarction the sclerae were yellow and on the next day the skin was definitely jaundiced and bilirubin was found in the urine.* The petechial eruption became more extensive and purpuric spots appeared over the back. There was marked chemosis with hemorrhages into the conjunctivae. Jaundice continued to deepen. Blood cultures were negative. The temperature remained normal. Death occurred seven days after infarction.

Necrospy findings. The important findings at necropsy were: chronic aortic stenosis and insufficiency; marked chronic passive congestion of the viscera; mural thrombi in the right auricular appendix; ascites; *multiple old and fresh hemorrhagic infarcts in the lungs; marked chronic passive congestion of the liver with necrosis of the cells in the central portion of the lobules; jaundice.*

Case 3. A girl with severe myocardial failure; probable history of pulmonary infarction two days before admission; on admission, slight jaundice and bilirubinuria; temporary improvement with disappearance of bilirubinuria; sudden deepening of jaundice on day following another pulmonary infarction; reappearance of bile in urine; progressive myocardial failure; death three days later.

History. A white girl, aged 20 years, with a previous history of acute rheumatic fever, entered the hospital complaining of shortness of breath, palpitation, and edema of the legs. For six years, there had been symptoms of myocardial failure which, following the birth of a child, increased so markedly that she was obliged to enter the hospital. Two days before admission, she had had severe pain in the chest, followed shortly by cough, blood tinged sputum, and an exacerbation of her general symptoms.

On examination, she was found to have the physical signs of mitral, aortic and probably tricuspid valvular disease, associated with the usual evidences of severe congestive heart failure. The liver was enlarged, and *there was a slight icteric tinge to the sclerae.* There was a moderate secondary anemia (hemoglobin 65 per cent), and the white blood cells were 21,000, per cmm. The urine contained a trace of bilirubin. The stools contained bile.

Course before pulmonary infarction. Following the usual forms of therapy, there ensued a period of slight improvement, during which the bile disappeared from the urine.

Course following pulmonary infarction. On the twelth day after admission, she suffered from sudden pain in the chest, the pulse became more rapid, cyanosis deepened, and she developed paroxysms of coughing, productive of bloody sputum. There was no elevation of temperature above the previous level (99° to 101°F.). *On the following day, there was a striking increase in the degree of jaundice and bilirubin reappeared in the urine.* These symptoms were associated with marked increase in the severity of myocardial failure, which progressed up to her death, three days later.

Necropsy findings. The important necropsy findings were: cardiac hypertrophy; chronic endocarditis of mitral, aortic and tricuspid valves; chronic passive congestion of the lungs; *multiple hemorrhagic infarcts of the lungs;* bronchopneu-

monia; ascites; fibrinous pleurisy over both lungs; *extreme chronic passive congestion of the liver with necrosis of the central part of the lobules and fatty infiltration of the midzone of the lobules; jaundice.*

Case 4. A man with moderately severe myocardial insufficiency and slight jaundice; probable pulmonary infarction two days before admission; temporary improvement; intensification of jaundice three days following pulmonary infarction; appearance of bilirubinuria; hemorrhages into skin; slight fever; death eleven days later, due to progressive myocardial failure.

History. A white man, aged 39 years, whose previous history was unimportant, entered the hospital on account of shortness of breath and edema of the legs. He had had symptoms of myocardial insufficiency for nine months. Two days before admission all of his symptoms had been exaggerated following the occurrence of pain in the chest that was followed by the expectoration of bloody sputum.

On examination, the striking features were the physical signs of a dilated aorta with aortic insufficiency associated with a moderate degree of heart failure of the congestive type. The sclerae had a yellowish tinge but the urine did not contain bilirubin. There was neither anemia nor fever.

Course before pulmonary infarction. Following the usual forms of therapy, the patient's general condition improved for a period of five days. Edema diminished and cyanosis became less marked.

Course following pulmonary infarction. Five days after admission the patient was seized with pain in the chest, associated with extreme respiratory distress. The pulse rate increased in frequency, the respirations became more rapid and labored, cyanosis deepened and edema increased. There were frequent attacks of coughing with expectoration of bloody sputum. *Three days after infarction, the jaundice, which had been very faint, became intense,* bilirubin appeared in the urine and a few petechial hemorrhages appeared about the knees. The jaundice was most marked over the upper half of the body and not so conspicuous over the legs.[2] The temperature rose to 101°F. three days after infarction. Death occurred eleven days later.

Necropsy findings. The necropsy revealed syphilitic aortitis with involvement of the aortic valves; chronic passive congestion of the viscera; *extensive hemorrhagic infarcts of both lungs; jaundice; marked chronic passive congestion of the liver with necrosis of cells about the center of the lobule and increase in the connective tissue.*

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[2] We have observed upon several occasions that the jaundice in patients with myocardial insufficiency is most conspicuous over the upper portions of the body where the edema is less marked. It is most marked in the sclerae and mucous membranes, in the face and arms and over the chest and abdomen but to a less extent over the edematous legs. Occasionally it is seen best upon the abdominal wall, where jaundice is not masked by cyanosis and the tissues are not very edematous. This distribution of jaundice in myocardial insufficiency has also been noted by Meakins (1925).

Case 5. A man with moderately severe myocardial insufficiency; no improvement; two days following pulmonary infarction sudden appearance of jaundice with increase in bilirubin content of blood; appearance of bilirubinuria; progressive myocardial failure; death eleven days after pulmonary infarction.

History. A white man, aged 45 years, with a previous history of syphilis, entered the hospital complaining of abdominal pain and spells of dizziness. He had had attacks of paroxysmal dyspnoea and substernal pain for ten months. He had had one previous attack of congestive heart failure from which he had recovered.

On examination, there were present the physical signs of syphilitic aortitis with aortic insufficiency, associated with signs of moderately severe myocardial failure. The liver was enlarged. There was no jaundice and the urine was free from bilirubin. There was no anemia and the white blood cells were 8,600 per cmm.

Course before pulmonary infarction. The patient's condition remained stationary for seven days. He had irregular fever, numerous attacks of paroxysmal dyspnoea, and substernal pain.

Course following pulmonary infarction. Seven days after admission the patient's temperature rose to 104°F., he complained of pain in his chest and expectorated bloody sputum. On this day the amount of bilirubin in the blood serum was two units (van den Bergh), giving the indirect reaction. *Two days later the sclerae and skin were visibly jaundiced* and bilirubin appeared in the urine. The amount of bilirubin in the blood on this day was four units and the reaction was direct.

On the fifth day following infarction the blood gave the direct reaction and contained five units of bilirubin, and the skin was now deeply jaundiced. He failed progressively and died eleven days after infarction.

Necropsy findings. The important necropsy findings were: syphilitic aortitis with aortic insufficiency; cardiac enlargement; chronic passive congestion of the viscera; *old and fresh pulmonary infarcts; jaundice; terminal pneumonia; chronic passive congestion of the liver with necrosis of the cells about the central vein.*

Case 6. A man with severe myocardial failure; slight jaundice and urobilinuria on admission; temporary improvement; on day following pulmonary infarction sudden deepening of jaundice with appearance of bilirubinuria; progressive heart failure leading to death fifteen days after pulmonary infarction.

History. A white man, aged 41 years, with a history of recurring attacks of acute rheumatic fever, entered the hospital on account of edema of the legs and a cough without the production of sputum. There had previously been several attacks of myocardial insufficiency, each time with temporary recovery. His present attack was of four months duration.

On examination he was found to have the signs of mitral valvular disease, auricular fibrillation and severe myocardial insufficiency of the congestive type. *There was definite jaundice of the skin and sclerae.* There was a moderate secondary anemia (hemoglobin 70 per cent); the leucocytes were 7,640 per cmm. There was urobilinuria and moderate retention of nitrogen in the blood.

Course before pulmonary infarction. There was a definite improvement in the patient's condition for four days. As cyanosis disappeared, icterus became more distinct.

Course following pulmonary infarction. Four days after admission the patient had severe pain in the chest, the respirations became accelerated, the heart rate increased, the pulse deficit became more marked, cyanosis deepened but the temperature did not rise above the previous level (100° to 100.6°F.). There was neither cough nor bloody expectoration. *On the following day there was a marked increase in the degree of jaundice, bilirubinuria appeared,* and a friction rub was heard over the lower lobe of the right lung. The myocardial insufficiency increased in severity and the patient died fifteen days later.

Necropsy findings. The important necropsy findings were: chronic endocarditis of the mitral and tricuspid valves; *marked chronic passive congestion of the liver; infarct in the right median lobe;* right hydrothorax.

Case 7. A man with severe myocardial failure and slight jaundice; temporary improvement; sudden increase of jaundice two days following pulmonary infarction; appearance of bilirubinuria; hematemesis and melena; progressive myocardial failure; death six days after pulmonary infarction.

History. A white man, aged 37 years, with a history of acute rheumatic fever, entered the hospital on account of swelling of the extremities and breathlessness. He had had symptoms of myocardial insufficiency for four months.

On examination he was found to have the physical signs of aortic and mitral valvular disease, associated with rather severe myocardial insufficiency of the congestive type. The sclerae and skin had a yellowish tinge; the urine did not contain bilirubin. There was no anemia and the white blood cells were 10,400 per cmm.

Course before pulmonary infarction. Following the usual forms of therapy there was a definite improvement in the patient's condition. The edema diminished, the liver became smaller and cyanosis was less marked.

Course following pulmonary infarction. Four days after admission, the patient suddenly became more dyspneic, the pulse became more rapid, cyanosis deepened and the signs of congestion increased. There was no fever, pain, cough nor bloody expectoration. *Within two days the jaundice deepened,* urobilinuria and bilirubinuria appeared. The patient had a hematemesis and the stools contained occult blood. Death occurred six days following pulmonary infarction.

Necropsy findings. The necropsy revealed chronic mitral and aortic stenosis; cardiac hypertrophy; chronic passive congestion of the viscera; mural thrombi in the right auricular appendage; *multiple hemorrhagic infarcts of the lungs; jaundice; chronic passive congestion of the liver with necrosis of the cells about efferent vein of the lobule;* multiple superficial gastric and duodenal ulcers; diphtheritic colitis.

Case 8. A man with severe myocardial failure and slight jaundice; temporary improvement; two days following pulmonary infarction deepening of jaundice and

appearance of bilirubinuria; progressive myocardial failure. Death nine days following pulmonary infarction.

History. A colored man, aged 41 years, with a history of acute rheumatic fever, entered the hospital on account of swelling of the extremities. He had had symptoms of myocardial insufficiency for three months and for one month they had been severe.

On examination, he was found to have the signs of mitral valvular disease and moderately severe myocardial insufficiency. The sclerae had a yellowish tinge; there was no bilirubinuria. There was no anemia and the white blood cells were 8,400 per cmm.

Course before pulmonary infarction. Following the usual forms of therapy there was a definite improvement in the patient's condition for a period of four days.

Course following pulmonary infarction. Four days after admission the patient had pain the chest which was followed by tachycardia, increased respiratory rate, cough and bloody sputum. The temperature did not rise above the former level (99° to 100°F.) until three days after the infarction took place when the temperature was 102°F. The white blood count was 8,040 per cmm. The symptoms of myocardial insufficiency increased; *four days after infarction the jaundice deepened* and bilirubin appeared in the urine. The patient failed rapidly and died nine days after pulmonary infarction.

Necropsy findings. The necropsy revealed: chronic mitral endocarditis; adhesive pericarditis; cardiac enlargement; scars in the myocardium; *multiple hemorrhagic infarcts of the lungs; jaundice marked chronic passive congestion of liver with necrosis of central and mid zones of the lobules;* bronchopneumonia.

Case 9. A man with severe myocardial failure; sudden appearance of jaundice two days following pulmonary infarction; temporary improvement for sixteen days; deepening of jaundice and appearance of bilirubinuria following second pulmonary infarction; hematemesis; death due to progressive myocardial failure seven days after second pulmonary infarction.

History. A white man, aged 59 years, whose past history was unimportant, entered the hospital on account of swelling of the abdomen. He had had one previous attack of myocardial failure five months before admission. He had had symptoms of moderately severe myocardial insufficiency for two months.

On examination, the patient was found to have signs of aortic valvular disease, with a dilated aortic arch. associated with the usual evidences of myocardial insufficiency of the congestive type. There was no jaundice. The liver was enlarged. The urine did not contain any bilirubin and there was no anemia. The white blood cells were 5,880 per cmm.

Course before pulmonary infarction. Following the usual methods of therapy the patient's condition improved for a period of two days.

Course following pulmonary infarction. Two days after admission the patient began to have attacks of coughing and expectorated bloody sputum. The respiratory and heart reates increased, but there was no fever. Two days later, *there*

was definite jaundice of the sclerae and skin and urobilinuria but no bilirubinuria. Following this attack his general condition improved for a period of sixteen days when another pulmonary infarct occurred. This was followed by an increase in myocardial insufficiency, jaundice became more intense and bilirubin appeared in the urine. The patient had a hematemesis two days before death and died seven days after the second pulmonary infarction.

Necropsy findings. The important necropsy findings were: syphilitic aortitis; aortic insufficiency; aneurysm of the arch of the aorta; chronic adhesive pericarditis over the aneurysm of the ascending arch of the aorta; chronic passive congestion of the viscera; *multiple infarcts in the right lung; hemorrhagic erosions of the mucosa at the cardiac end of the stomach; jaundice; chronic passive congestion of the liver with necrosis of the cells about the central vein.*

Case 10. A man with severe myocardial insufficiency; slight jaundice on admission; temporary improvement; two days following pulmonary infarction increase in intensity of jaundice; appearance of bilirubinuria; death twelve days later due to progressive myocardial failure.

History. A colored man, aged 45 years, was admitted to the hospital on account of swelling of the extremities. He had had symptoms of progressive myocardial failure for one year and these symptoms had all become more severe recently.

On examination, he was found to have an enlarged heart without evidences of valvular disease, moderate arteriosclerosis and the usual signs of myocardial insufficiency of the congestive type. There was a slight yellowish tint of the sclerae. The urine did not contain bilirubin. The liver was enlarged. The amount of bilirubin in the blood at this time was 1.5 units (indirect).

Course before pulmonary infarction. Following the usual forms of therapy, his general condition *seemed* to improve. His liver remained large, however, jaundice of the sclerae deepened in degree, the amount of bilirubin in the blood rose to 3 units (indirect) and urobilin appeared in the urine.

Course following pulmonary infarction. Eight days after admission he became more dyspneic, he had paroxysmal attacks of coughing with the expectoration of bloody sputum. The symptoms of myocardial insufficiency increased and auricular fibrillation appeared. Three days later, *jaundice became more marked,* the amount of bilirubin in the blood increased to five units (direct and bilirubin appeared in the urine. The myocardial insufficiency and jaundice continued to increase in degree, a friction rub appeared over the lung. The temperature was not elevated above 101°F. The leucocyte count was 7,800. Two days before death there were 10 units (direct) of bilirubin in the blood. Death occurred twelve days after pulmonary infarction.

Necropsy was not obtained.

ANALYSIS OF CLINICAL CASES

a. Clinical picture before pulmonary infarction. Of the ten cases studied in this group, five had syphilitic, four rheumatic and one ar-

teriosclerotic heart disease. One-half of these patients had had previous attacks of myocardial insufficiency varying in duration from two months to one year before the illness that brought them to the hospital. All had had symptoms of heart failure from one to three months before coming under observation. Four of the ten patients gave a history of having had symptoms that might be interpreted as being due to previous pulmonary infarctions. On examination, seven of the ten patients had severe heart failure, while the other three were moderately ill. Six of the patients had slight jaundice of the sclerae and skin, while four showed no evidence of jaundice whatever. The liver was enlarged in eight cases and not palpable in the other two. Bilirubin was present in the urine in only two cases. The leucocyte count was normal in all of the cases except one in which there was a leucocytosis of 21,000 per cmm. Following the usual forms of therapy six of the patients improved, three remained stationary and one patient became progressively worse.

b. Clinical picture following pulmonary infarction. Following the development of pulmonary infarction, the entire clinical picture changed, suddenly and often without warning. The characteristic features and course were: 1, increased severity of the manifestations of myocardial failure; 2, the appearance of jaundice; 3, the symptoms dependent on the local process in the lungs; and 4, the appearance of hemorrhagic phenomena. The first one was constant; aside from the appearance of jaundice, the most characteristic feature was the sudden exacerbation of the symptoms of myocardial insufficiency, rapidly progressive, and leading to the death of the patient within three to fifteen days. Marked cyanosis was present in every case. In one patient transient auricular fibrillation developed.

The symptoms usually considered characteristic of pulmonary infarction were not always striking, and were, in fact, sometimes absent. In practically all cases, there was increase in the respiratory rate and also in the severity of dyspnea; but ordinarily, these symptoms alone are not necessarily an indication of the development of pulmonary infarction. Thoracic pain was present in six cases, cough and bloody expectoration in eight. In only four instances did a friction rub appear over the affected lung, in these cases it was heard within two to five days after infarction occurred. Fever was present in only four

cases, and was not associated with marked increase in the number of leucocytes.

The most striking and conspicuous manifestation of pulmonary infarction in these cases was the sudden appearance of jaundice, or, if jaundice was already present, its sudden deepening. This symptom occurred within one to four days, usually on the second or third after infarction. About the same time, bilirubin was found in the urine of all but one of the cases, and in this exceptional instance, there was a large amount of urobilin in the urine.

In two cases hemorrhages occurred into the skin; in two cases hematemases occurred; and melena and hematuria developed, each in one instance.

The important necropsy findings in nine of our cases were pulmonary infarctions, usually multiple and extensive chronic passive congestion of the liver with necrosis of the cells about the central veins. The bile passages were free and patent in all of the cases. The source for the pulmonary emboli was the right auricular appendage in five cases and in the other four cases the source for the emboli could not be determined with certainty.

CLINICAL PICTURE

Clinical picture. With these data, one may construct the following picture. The patient suffers from fairly severe heart failure, usually of considerable duration. At first he may show slight jaundice; visible jaundice may however be entirely absent. Under treatment, definite improvement may take place. Suddenly, a distinct change in the clinical picture occurs, dependent on the development of pulmonary infarction. Dyspnea becomes more intense, cyanosis deepens, myocardial failure increases and progresses rapidly. Usually, on the second or third day after the occurrence of pulmonary infarction, jaundice suddenly makes its appearance, or it becomes markedly intensified if it has been present previously. At the same time, bilirubin and urobilin are found in the urine. Hemorrhagic phenomena occasionally develop. These symptoms increase, advance steadily, and within a relatively short time, death ensues. It is important to bear in mind the fact that the entire course of events may take place in the absence of characteristic signs of pulmonary infarction. *The*

sudden appearance of jaundice or the sudden increase in the severity of myocardial insufficiency, should make one suspect that pulmonary infarction has occurred, even when outspoken evidences of pulmonary infarction cannot be obtained.

Undoubtedly, the above description applies only to a certain group of patients. There are certainly instances of pulmonary infarction which bring about lesser degrees of jaundice, perhaps detectable only by use of a method such as the van den Bergh test; and it is altogether probable that in these cases, the clinical course is not so stormy as we have pictured it, and that recovery frequently takes place.[3] Indeed, in our own cases, there were several instances in which the slight jaundice that was first seen was probably due to previous infarctions (possibly multiple); and recovery, at least temporary, might have ensued, had not further infarction taken place, this time leading to irreparable damage and death.

SUMMARY AND CONCLUSIONS

We have reported 10 cases of myocardial insufficiency in which the sudden appearance of visible jaundice, or the sudden increase in jaundice that was already present seemed directly dependent on the development of pulmonary infarction. The jaundice was due, in all instances, to bilirubinemia. In all these cases, the clinical course was very similar, for death, due to progressive myocardial failure, occurred within a relatively short time.

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[3] Since this paper was written, we have observed four more patients in whom jaundice developed following pulmonary infarction. Three of the patients recovered and one died. We should like to emphasize the fact, therefore, that pulmonary infarction occurring in patients with myocardial insufficiency and followed by jaundice does not lead to a fatal outcome in all instances.

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