qualities of the ideal bronchographic agent described in the introduction. It is promptly eliminated within eight hours of its introduction to the bronchial tree. Satisfactory bronchial mucosal detail without alveolarization or deleterious histologic effects in the experimental animal have been demonstrated. It appears that tantalum possesses excellent potential as a bronchographic medium for clinical usage.

It is obvious that the additional disadvantage of iodide preparations with respect to subsequent thyroid studies is eliminated with the use of this medium.

With the method of tantalum administration described above, satisfactory filling of the bronchi only in the lower lung fields could be demonstrated. Further studies with different methods of administration are in progress, both in the experimental subject and in patients.

REFERENCES

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Fatal Hemoptysis in Mitral Stenosis*

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Fatal massive hemoptysis is described in a patient with moderate mitral stenosis treated supportively. Hemodynamic studies and postmortem examination supported bronchial vein anastomoses as the source of hemorrhage. Review of previous reports emphasizes the necessity of prompt surgical intervention in this syndrome.

Although hemoptysis is a common symptom of mitral stenosis, it is rarely life-threatening. Occasional cases of fatal hemoptysis have been reported, and in recent years emergency mitral valve surgery has proved to be effective therapy. A case

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of fatal hemoptysis in mitral stenosis is described and the pathologic anatomy is presented, reinforcing the concept of early surgery for this condition.

Case Report

A 40-year-old white man with no history of rheumatic fever was first noted to have a heart murmur in 1961, but he was asymptomatic until 1965, when he noted mild dyspnea on exertion. He had a transient episode of hemoptysis in 1966. Two days prior to admission the patient developed extensive hemoptysis of two to three cups per day.

On admission, the patient was afebrile. With a normal blood pressure but a sinus tachycardia of 120 beats per minute and a respiratory rate of 25. Chest examination demonstrated dullness, splitting, and rales with decreased breath sounds on the left side. Mitral stenosis was manifested by an accentuated first heart sound, a closely split opening snap, and a long rumbling diastolic murmur. There was no right ventricular heave and no evidence of right ventricular failure, but pulmonary hypertension was suggested by an accentuated pulmonary closure sound. Laboratory results showed a hematocrit of 37 percent, and white blood count of 13,700 per mm³. The electrocardiogram revealed right axis deviation, left atrial enlargement, and changes suggestive of right ventricular hypertrophy. His chest x-ray films revealed fine calcifications, pulmonary outflow enlargement and a new left lingual infiltrate.

Over the next three days the patient improved with digitalization, sedation and codeine to suppress his cough. He developed a tender, hot left saphenous vein, and received a single dose of heparin for suspected pulmonary embolus, but persistent hemoptysis with his hematocrit falling to 30 led to prompt discontinuation of this therapy. An emergency right heart catheterization demonstrated a mean right atrial pressure of 5 mm Hg, right ventricular pressure of 50/5 mm Hg, pulmonary artery pressure of 50/15 mm Hg, and pulmonary capillary wedge pressure (mean) of 20 mm Hg. Pulmonary angiogram did not demonstrate a pulmonary embolus (Fig 1). Bronchoscopy confirmed bronchial bleeding limited to the right lung, without locating an active bleeding site. No specific organic lesion was identified.

Over the next 24 hours the patient improved on oxygen therapy and sedation with morphine, with decreased hemoptysis. Possible mitral valvulotomy was contemplated, but the patient again developed increasing hemoptysis and his temperature rose to 40.9°C (105.8°F), with tachypnea and tachycardia. He died five days after admission despite vigorous supportive therapy with sodium nafcillin, penicillin and kanamycin in addition to blood transfusions. Sputum cultures prior to death grew penicillin-resistant Staphylococcus aureus.

Postmortem examination demonstrated a heart weighing 390 gm, with a thickened right ventricular wall of 7 mm. The mitral valve was fibrotic and markedly stenotic. Microscopic sections showed endocardial and valvular fibrosis compatible with rheumatic heart disease. The right lung weighed 1,000 gm, the left 900 gm. There was no evidence of pulmonary arteriosclerosis present on microscopic section. The left bronchi were filled with blood clots. The bronchial veins were prominent but no specific bleeding point could be found. There was no evidence of pulmonary infection. The remainder of the examination was normal.

Discussion

Hemoptysis is a common symptom in mitral stenosis, the incidence varying from 9.5 percent to 36.5 percent in reported series.1-4 Death from massive hemoptysis, however, is distinctly rare. There were no fatalities in 300 cases of hemoptysis reported by Wood,6 and Oppenheimer and Schwartz7 noted only three deaths in a review of 1,000 patients with hemoptysis due to mitral stenosis. In addition, several case reports8-10 describe isolated instances of severe hemoptysis. The case reported by Barnum,8 with moderate hemoptysis amounting to approximately one liter, survived with supportive therapy. A fatal case described by Ginsberg8 died despite repeated transfusions. At autopsy, the large bronchi were filled with blood clots and the heart showed right ventricular hypertrophy. The mitral valve was stenotic but not calcified. The patient described by Issacs and co-workers10 presented with fever, tachypnea, tachycardia and hypotension. Despite intensive supportive therapy, including morphine, oxygen, digitalis, antibiotics and blood transfusions, the patient died. His autopsy revealed large bronchi filled with blood clots and alveoli filled with extravasated blood. No specific bleeding point was found. The mitral valve was markedly stenotic and heavily calcified. The resemblance between the fatal cases described above and our current case is apparent.

Hemoptysis in mitral stenosis usually is not a late manifestation of the disease, as Wolf and Levine6 and Wood6 emphasized. A variety of etiologies has been postulated: diapedesis of blood cells through alveolar capillaries;7 pulmonary arteriolar sclerosis with rupture;5 or necrotizing arteriolitis due to the

Figure 1. Pulmonary angiogram demonstrating no clots in the pulmonary arterial supply to the left lung. The left lung density represents blood in the bronchi with resulting atelectasis, and parenchymal infiltration in the clotted blood.
FATAL HEMOPTYSIS IN MITRAL STENOSIS

There has been little pathologic support for these hypotheses. A hypothesis better supported by pathologic and hemodynamic studies postulates that varicose bronchial veins are established by flow from the pulmonary veins to the ayzygos and hemiazygos systems. The amount of flow is dependent upon the pressure gradient between the left atrium and pulmonary veins and the right atrium and ayzygos and hemiazygos veins. This gradient decreases as right ventricular failure develops with a concomitant rise in right atrial pressure. In addition, the increasing pulmonary vascular resistance often accompanying advanced mitral stenosis may further decrease anastomotic flow resulting in decreased hemoptysis. The patient reported had a 15 mm Hg gradient, quite adequate to create bronchial vein anastomoses, and although pulmonary hypertension was present, there was no evidence of right ventricular failure.

The development of saphenous vein phlebitis complicated the management of this case. There is an increased incidence of pulmonary infarction in mitral stenosis. Wood reported an 8 percent incidence in his series. Wolf and Levine noted that 50 percent of his series had pulmonary infarction. Both authors emphasize, however, that pulmonary infarction is a late complication of mitral stenosis, usually occurring after the onset of right ventricular failure. The incidence of pulmonary emboli is small with superficial thrombophlebitis. Moreover, the massive hemorrhage prohibited anticoagulation. The pulmonary angiogram performed showed no embolus, and autopsy confirmed the absence of emboli.

Several case reports discussing treatment of this complication by emergency valve operations are now in the literature. Fulton reported a successful result in a 19-year-old pregnant woman with predominant mitral stenosis and minimal aortic stenosis and regurgitation. She became critically ill with massive hemoptysis and anemia developing collapse of the right lung, not responding to morphine, phenobarbital, and antibiotics. At operation, she had extreme stenosis, with a mean left atrial pressure of 35 mm Hg, and a pulmonary artery systolic pressure of 65 mm Hg. A lung biopsy showed mild pulmonary arteriolar hyperplasia. After mitral commissurotomy her bleeding promptly stopped and follow-up cardiac catheterization demonstrated a drop in the pulmonary arterial systolic pressure to 35 mm Hg, and return of the left atrial pressure to normal. Lunger and associates described a 33-year-old woman with mitral stenosis who presented with massive hemoptysis. Despite vigorous supportive therapy with morphine, barbituates, antibiotics and repeated blood transfusions, she deteriorated. At operation, a heavily calcified mitral valve was noted with a valve area of 0.5 sq cm. The pulmonary artery was strikingly enlarged. Bleeding stopped shortly after mitral valvulotomy. No hemodynamic data was described. The 23-year-old white man described by Schwartz and co-workers had mitral stenosis and insufficiency with a past history of multiple episodes of hemoptysis. He presented with massive hemoptysis, tachycardia and apprehension, and the left lower lobe was collapsed by a bronchial blood cast. He was treated with morphine, conjugated estrogenic hormone (Premarin), Pituitrin, nine units of packed red blood cells and repeated bronchoscopies. Right heart catheterization revealed the following pressures: right atrium (mean) 10 mm Hg; right ventricle 60/10 mm Hg; pulmonary artery 55/30 mm Hg. Replacement of the mitral valve resulted in prompt cessation of hemoptysis. Ramsey and others described a 22-year-old woman with mitral stenosis who presented with symptoms of pulmonary congestion and physical findings consistent with right ventricular hypertrophy. Right and left heart catheterization gave the following data: right atrium (mean) 2 mm Hg; right ventricle 71/3 mm Hg; left atrium (mean) 12 mm Hg; mitral valve area 0.8 sq. cm; and pulmonary vascular resistance 835 dynes/sec/cm². Because of active rheumatic carditis the operation was postponed, but after readmission two months later, she developed massive hemoptysis and cardiac arrest. After successful resuscitation and blood transfusion, hemoptysis continued intermittently requiring several additional units of blood. Therefore, a closed commissurotomy was performed with immediate clinical improvement and cessation of hemoptysis within 24 hours. A 39-year-old woman with silent mitral stenosis was described by Nennhaus and Hunter. Over a four-day interval this patient expectorated 6,000 ml of blood despite sedation, tracheostomy, and repeated bronchoscopies which removed bronchial casts of clotted blood. The hemorrhage ceased abruptly following mitral commissurotomy after surgical exploration confirmed the presence of tight mitral stenosis.

The five cases treated with emergency valve surgery had virtually identical presentations to the fatal cases described above. Each of the patients was relatively young, with evidence of moderate mitral stenosis and, on occasion, pulmonary hypertension, but without right heart failure. The hemodynamic data as well as the clinical material correlates well with the fatal case described in this report. All failed to respond to intensive medical therapy. In a general discussion of therapy for he-

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suffocation. Support these upon surgery and a conclusion. Dalen and associates monitored a group of patients after mitral valve replacement, noting a drop in left atrial pressure, pulmonary pressure, and pulmonary vascular resistance promptly after surgery in the initial postoperative period. The drop in left atrial pressure should result in a decreased bronchial vein flow and cessation of hemoptysis. In an analysis of hemoptysis in relation to mitral stenosis, Greenwood and co-workers reported upon 13 cases with preoperative hemoptysis. Ten of these patients had total cessation of hemoptysis, and two of the three patients who later developed recurrent hemoptysis gave evidence of restenosis. Both clinical experience and hemodynamic data support early valve surgery in the patient with mitral stenosis who develops life-threatening massive hemoptysis.

References

Seventh Annual Cardiology Seminar
Arrhythmias and related topics will be discussed at the Seventh Annual Cardiology Seminar, sponsored by the Rogers Heart Foundation, to be held at the Tides Bath Club, Redington Beach, Florida, October 23-26. Members of the visiting faculty will include Drs. Stephen M. Ayres, New York City; J. Willis Hurst, Atlanta; David P. Lauber, Boston; Rashid A. Massumi, Washington, D.C.; Jane Somerville, London, England; Bernard Tabatnik, Baltimore, and Andrew G. Wallace, Durham. Dr. Henry J. L. Marriott is the seminar director. Further information may be obtained by writing the Rogers Heart Foundation, St. Anthony's Hospital, St. Petersburg, Florida.