Spontaneous Hemopneumothorax.
Report of Two Cases*

WILLIAM H. KASTL, M.D., F.A.C.S.
Alexandria, Louisiana

Spontaneous hemopneumothorax, not due to trauma, tuberculosis, or any other active pulmonary disease, is essentially a rare condition. Deliss, Gale and Brown,1 in a recent article, found 61 reported cases to which they added two others. Subsequently, five additional cases were reported3,4 bringing the total to 68. In this series, there were 14 deaths (20 per cent).

The pathological and clinical aspects of this condition have been adequately described by Hartzell.2 It is accepted that the responsible mechanism is the rupture of an emphysematous bleb with the formation of a bronchopleural fistula. This may be accompanied by the tearing of one or more richly vascularized adhesions with bleeding from either the visceral or parietal sides. Symptoms arise as a result of: (1) the pulmonary collapse and mediastinal displacement due to the pressure of the blood and air, and (2) the associated blood loss.

Treatment must be directed at the restoration of the normal physiological and anatomical states. This can be accomplished by (1) removal of the accumulated blood and air from the pleural cavity with subsequent re-expansion of the lung, and (2) by restoration of the normal blood volume with whole blood transfusions. It was formerly believed that immediate removal of the blood, through lowering the intrapleural pressure, might cause increased bleeding. This concept was proved to be invalid by Sellors5 who advocated immediate and repeated aspirations. He also emphasized the fact that since delayed aspiration of blood results in fibrin deposition on the pleura, this condition could eventually cause mediastinal fixation, fibrothorax, empyema, and inexpandable lung with depression of pulmonary function. Blood must be completely removed either by frequently repeated aspirations, or by the insertion of a catheter intercostally. This catheter should be attached to a water seal and suction system6 with a negative pressure of 15 to 20 centimeters of water. Myers, Johnston, and

*From the Surgical Service, Veterans Administration Hospital, Alexandria, Louisiana.
Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

226
Bradshaw\(^4\) reported a case in which emergency thoracotomy was necessary because of massive, uncontrolled bleeding. If the fluid becomes loculated, infected, or if the lung fails to expand, then pulmonary decortication or even resection or thoracoplasty may become necessary. Read and Berry\(^5\) recently reported the successful use of streptokinase-streptodornase in a case of hemopneumothorax. The action of these agents is fibrinolytic, so that they must be used with caution in the presence of a bronchopleural fistula, since, as Read warned, they can cause the reopening of an established fistula even if they cannot primarily produce one.

Once the prompt evacuation of the fluid is effected, the air is usually absorbed, allowing the lung to expand. If the bronchopleural fistula remains open, a tension pneumothorax can develop. Decompression must be obtained by the insertion of a needle or catheter intercostally, attaching it to a water seal with mild suction. However, even as low a negative pressure as 15 centimeters of water can re-open a sealed fistula, so that allowing the air to bubble into a water seal without suction may be sufficient to maintain slow expansion without air leakage. Persistent bronchopleural fistula indicates need for surgical closure.

Two cases of spontaneous hemopneumothorax are presented which we have seen on our service and which illustrate the extremes of treatment that may be necessary in the same pathological state.

**Case 1** is a 27-year old white male admitted August 5, 1949, with severe pain in his left anterior hemithorax. Thirty-six hours prior to admission, he had been awakened from sleep by sharp pain in his chest. This increased in severity and he became increasingly dyspneic. There was no history of tuberculosis, but since childhood he had had occasional minor pains in the left side of his chest.

On admission, he did not appear acutely ill, but was moderately dyspneic. His temperature was 99.4 degrees Fahrenheit (orally), blood pressure was 120/70, pulse rate was 132 and respirations 32 per minute. The trachea was in the midline. There was decreased expansion, absent breath sounds and vocal fremitus over the left hemithorax. Marked hyper-resonance over the upper half and dullness to flatness over the lower half of the left chest was found. The heart sounds were muffled and rapid.

Examination of the blood revealed 9.75 grams of hemoglobin per 100 cubic centimeters, 3,400,000 erythrocytes and 17,200 leukocytes per cubic millimeter, with a differential count showing 87 per cent neutrophils, 8 per cent lymphocytes, and 4 per cent monocytes. A series of five sputum examinations and one gastric washing failed to reveal acid-fast bacilli. A roentgenogram on admission showed pneumothorax on the left side about 80 per cent complete. In addition, a fluid level was present to the tip of the third rib. The heart shadow deviated slightly toward the right.

Immediately following admission, 850 cubic centimeters of fluid were
aspirated from the left pleural cavity. This appeared to be blood but did not clot. An additional 425 cubic centimeters of the same type of fluid together with 385 cubic centimeters of air were aspirated on August 9, 1949. A total of 1000 cubic centimeters of whole blood were given by transfusion and he received 400,000 units of penicillin parenterally daily.

A low-grade fever subsided after four days. A roentgenogram on August 16, 1949, showed the left lung to be almost completely re-expanded with a minimal amount of pneumothorax at the extreme left apex. His blood count on August 17, 1949, revealed 12.8 grams of hemoglobin per 100 cubic centimeters, 4,400,000 erythrocytes per cubic millimeter, with a differential count of 66 per cent neutrophils and 34 per cent lymphocytes. He was discharged from the hospital August 18, 1949. A follow-up 16 months after his discharge showed that he had no pulmonary symptoms or disease since the initial episode and was in good health.

Figure 1, Case 1: Hemopneumothorax on admission.
Figure 2, Case 1: Complete expansion of lung prior to discharge.

Figure 3, Case 2: Hemopneumothorax on admission.
Figure 4, Case 2: Following thoracoplasty to obliterate empyema space.
This case illustrates the ease with which hemopneumothorax may be treated providing it is seen early after onset and that the bronchopleural fistula is small and becomes sealed off well. There was no further leakage of air or blood following prompt aspiration.

Case 2 is a 32-year old white male admitted October 4, 1950, with pain in the left side of the chest and shortness of breath. On October 1, 1950, he developed sudden, sharp pain in his left hemithorax which was accompanied by marked dyspnea. This episode occurred while he was engaged in carpentry at home, under mild physical exertion. Treatment by his family physician consisted of parenteral penicillin and roentgenograms. No other therapy was received. There was no history of tuberculosis or other pulmonary disease.

On admission, the patient appeared to be acutely ill. His skin was ashen and he was moderately dyspneic. The veins of his forehead were prominent and dilated. His temperature was 98 degrees Fahrenheit (orally), his blood pressure was 110/70, pulse rate was 92 and respirations were 17 per minute. His trachea was in the midline. There was decreased expansion, absent breath sounds and vocal fremitus over the left side of his chest. Hyper-resonance over the upper half and marked dullness to flatness over the lower half of the left hemithorax were noted. There appeared to be no cardiac displacement and the heart sounds were normal.

Examination of the blood showed 9.75 grams of hemoglobin per 100 cubic centimeters, 2,840,000 erythrocytes and 8900 leukocytes per cubic millimeter, with a differential count of 77 per cent neutrophils, 19 per cent lymphocytes, and 4 per cent monocytes. His sedimentation rate was 18 millimeters per hour. A series of five sputum examinations failed to reveal acid-fast bacilli. A Mantoux test using Old Tuberculin was positive. Skin tests for histoplasmin, blastomycin and coccidioidin were negative. A roentgenogram on admission showed complete collapse of the left lung with a fluid level below the tip of the fourth rib. The mediastinal structures deviated to the right.

Because of the mediastinal shift, the extent of the pulmonary collapse, and the possibility of tension pneumothorax, decompression was effected by insertion of a catheter into the left pleural cavity through the fifth intercostal space laterally. About 1500 cubic centimeters of bloody fluid were aspirated, following which the catheter was attached to a water seal. Suction was not immediately applied to the water seal lest the bronchopleural fistula be re-opened. He received 1500 cubic centimeters of whole blood by transfusion and parenteral penicillin was given. The next day, 15 centimeters of water suction was started. A roentgenogram taken that day showed satisfactory expansion of the left lung, but with some residual mediastinal displacement and fluid at the base. From then on, expansion of the lung was slow. On October 20, 1950, two fluid pockets were evident on roentgenograms. Both presented fluid levels, one being anterior, the other posterior, at the level of the eighth thoracic vertebra. On October 23, 1950, after fluoroscopic localization, the posterior pocket was aspirated and 70 cubic centimeters of turbid, bloody fluid were removed. This fluid, on culture, was positive for staphylococcus aureus hemolyticus and was penicillin-sensitive in vitro. Re-aspiration two days later yielded 20 cubic centimeters of the same type of fluid. Subsequent roentgenograms indicated lack of further expansion and persistence of the loculated empyema. Decision to perform left pulmonary decortication
was made. At operation on November 3, 1950, many visceral-parietal adhesions were found. The lower lobe was densely adherent to the diaphragm by fibrous bands and the remainder of the lung was covered by moderately thick fibrinous peel. A small amount of fibrin was present in the pleural cavity. The bronchopleural fistula could not be found, but several small, emphysematous blebs were present on the upper lobe. These were left undisturbed. The pleural peel was removed and several resulting air leaks were sutured. Two intrapleural catheters were inserted through two intercostal spaces prior to closure. These catheters were attached to a water seal with a suction of 15 centimeters of water. His postoperative course was marked by difficulty in maintaining patency of the catheters. The lung began to re-expand slowly.

On November 20, 1950, an excessive amount of bubbling in the water seal indicated re-opening of the bronchopleural fistula. Suction was removed from the system with prompt subsidence of the bubbling and slight increase in expansion. Repeated roentgenograms failed to reveal further expansion. Therefore, on December 6, 1950, and again on December 11, 1950, streptokinase-streptodornase was instilled into the left pleural cavity through the catheter. Each time, 300 cubic centimeters of thick, plasma-like fluid were obtained, but without any appreciable lung expansion. On January 11, 1951, thoracotomy was done with de-recortication in mind. However, the lung was covered by a thick, highly vascular peel which bled excessively with the least manipulation. The fixation of the lung was such as to render consideration of lobectomy technically impossible; therefore, it was considered expedient to collapse the cavity by a modified Sauerbruch type of thoracoplasty, resecting portions of the third to eighth ribs. An intrapleural catheter was left in place and attached to a water seal. His postoperative course was smooth, with slow obliteration of the residual space about the catheter following instillations of penicillin. He was discharged from the hospital on March 14, 1951.

This case illustrates the extreme difficulty which may be encountered in the management of hemopneumothorax. The initial delay of four days prior to the first definitive treatment was instrumental in preventing complete expansion of the lung because of the formation of pleural peel. Following de-recortication, the difficulty in maintaining patency of the catheters again prevented re-expansion. By then, it was impossible to remove the peel without endangering the patient’s condition; thus, the only alternative was thoracoplasty to obliterate the cavity. Streptokinase-streptodornase was ineffective, probably due to the excessive fibrous tissue formation.

SUMMARY

1) Two cases of spontaneous hemopneumothorax illustrate how the treatment for the same condition can vary.

2) Emphasis is placed on the prompt evacuation of blood from the pleural cavity to enhance the expansion of the lung, and the use of whole blood transfusions to combat the lowered blood volume.
RESUMEN

1) Dos casos de hemotórax espontáneo ilustran cómo el tratamiento del mismo síndrome puede variar.

2) Se hace énfasis sobre la evacuación pronta de la sangre de la cavidad pleural y el uso de transfusiones de sangre para contrarrestar la pérdida de volumen sanguíneo.

RESUME

1) Deux observations d'hémopneumothorax spontané montrent combien le traitement d'une telle affection varie selon les cas.

2) L'auteur insiste sur la nécessité d'évacuer rapidement le sang de la plèvre pour permettre la réexpansion du poumon, et sur l'utilisation de transfusions sanguines pour combattre la diminution du volume sanguin circulatoire.

REFERENCES