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Simple Spontaneous Pneumothorax

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I. SIMPLE SPONTANEOUS PNEUMOTHORAX*

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Riolan[1] (1648), Littre[2] (1713), Meckel[3] (1759) and others in the 18th and 19th centuries reported air in the pleural cavity following trauma. In 1803 Itard[4] called attention to five cases found at necropsy. He coined the term pneumothorax. Laennec[5] (1819) discussed the clinical aspects of this condition and its recognition by physical examination. The writings of these authors alerted physicians and many reported cases. In fact just a century after Itard's thesis was published Emerson[6] listed 358 literature references to this condition. During the past 50 years a vast bibliography has accrued with ever increasing knowledge of frequency, etiology, diagnosis and treatment.

Air may reach a pleural space through the parietal pleura as in stab or bullet wounds or disease which penetrates the chest wall. Perforation of esophegeal or gastric ulcers and subphrenic abscess may be responsible. Most frequently air is found in the pleural cavity as a result of willful induction in the treatment and diagnosis of disease.

Air may also enter a pleural cavity through the visceral pleura. Anything that establishes a communication between bronchial ramifications or air cells and the pleural space permits this endogenous origin of air in the cavity. Injuries to the chest wall sometimes result in rib fragments penetrating the visceral pleura. Small objects passing through the chest wall such as bullets may penetrate the visceral pleura. Needling the chest for any purpose such as aspiration of fluid may puncture the visceral pleura. Resuscitation of newborn infants, passing a bronchoscope, administering intratracheal anesthesia etc. are sometimes responsible.

Hewson[7] (1767) apparently was first to mention the possibility of entry of air into a pleural space through perforation of pulmonary diseases. It is now known that both acute and chronic disease may so damage the visceral pleura as to permit the escape of air into the pleural space.

The term spontaneous pneumothorax has been reserved for those cases in which disease or defect of the pleura permits air from the lung to enter the pleural space. Earlier in the evolution of our knowledge of spontaneous pneumothorax this condition was usually recognized in persons who had pulmonary disease demonstrable during life or at necropsy. More cases were reported due to pulmonary tuberculosis than to any other disease, probably because of its prevalence and inadequacy of differential diagnostic procedures so that many non-tuberculous pulmonary conditions were classified as tuberculous. Biach[8] (1880) collected 986 cases of spontaneous pneumothorax from the literature and from the records of three Vienna hospitals and found tuberculosis reported as responsible in 77.8 per cent. As late as 1931, Palmer and Taft[9] stated that among adults from 80 to 90 per cent of the cases of spontaneous pneumothorax were due to tuberculosis.

Observations were made to determine frequency of spontaneous pneumothorax among persons with pulmonary tuberculosis. Biach[8] reported that it had been recognized in 1 per cent of 58,731 cases. Weber[10] stated that at necropsy spontaneous pneumothorax was found in 10 per cent of tuberculous persons. With more extensive use of x-ray inspection Barlow and Thompson[11] called attention to the

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frequency of small pneumothoraces which without this diagnostic advantage were usually not detected during life.

For the first two or three decades of this century it was still generally believed that spontaneous pneumothorax nearly always indicated the presence of pulmonary tuberculosis. Even if this disease could not be located after expansion of the lung the individual was strongly advised to have at least one year of bed rest. So certain were physicians that pulmonary tuberculosis must be present that in many instances spontaneously collapsed lungs were kept collapsed artificially for a year or more.

As time passed there was an increasing number of persons reported with spontaneous pneumothorax who did not react to tuberculin, tubercle bacilli were not recovered and no evidence of tuberculosis was then or later obtainable. Since the cause was not determined this condition was referred to as idiopathic spontaneous pneumothorax.

This was frequently seen in young adult males who remained in good health so there were few opportunities to make post mortem examinations. Moreover thoracoscopy was not available to aid in diagnosis. However, in 1932 and 1933 Kjaergaard[12,12a] reported five of his own cases and six others from recent literature in which he described the etiology. In one type a valve is formed by emphysematous tissue, in another by congenital valve vesicle and in the other by scar tissue. He designated the condition spontaneous pneumothorax simplex. Kjaergaard pointed out that frequently spontaneous pneumothorax in an apparently healthy person has no relationship to tuberculosis. He expressed the opinion that individuals with this condition should be spared loss of time and expense of sanatorium treatment as well as fear of tuberculosis.

Kjaergaard's observations have been adequately confirmed and it is now recognized that simple spontaneous pneumothorax is usually due to congenital or acquired defects in the lung or pleura resulting in emphysematous blebs (bullae, air vesicles, etc.) which rupture in one or more places so that air gains admission to the pleural space. Adhesions between the parietal and visceral pleurae may also result in rents in the latter.

In 1934 Hamman[13] described spontaneous interstitial emphysema of the lungs. He believed it was due to rupture of pulmonary alveoli with the escape of air into interstitial tissues. The cause for this condition was not known, but he suggested that an alveolar wall here and there may become attenuated and weakened without known reason. This could be due to developmental defects, previous disease or present disease too slight to be detected. In any event, when a large amount of air escapes in this manner it may dissect along the connective tissue bands surrounding blood vessels and form blebs on the pleura, which in turn may rupture and cause spontaneous pneumothorax.

In 1934 we[14] reported 31 cases of spontaneous pneumothorax, 10 of whom had apparently responsible pulmonary disease. Two had bronchial asthma and 19 were classified as the simplex type. We have since seen many persons with spontaneous pneumothorax presumably caused by various pulmonary diseases including malignancies, silicosis, tuberculosis etc. In our group there have been those with traumatic pneumothorax produced by bullets, inadvertent opening of pleura during surgical operation, bronchoscopy, pitchfork tine, chest injury in automobile accidents, etc.

In this paper only cases of simple spontaneous pneumothorax are presented. This group includes 123 persons of whom 18 were preliminarily reported in 1934. Of the 123 eight were disqualified because of incomplete or lost records.

The ages of these 115 persons at the time of the first diagnosed attack are shown in table 1. Only one infant was seen (not included in table 1 because of incomplete record) and the etiology of the pneumothorax was not determined. Pneumothorax has been reported in several infants including new born babies. The first newborn case was described by Rugi

TABLE 1

Age at Time of First Diagnosed Attack

15-19	13	40-44	5
20-24	55	45-49	0
25-29	16	50-54	1
30-34	15	55-59	2
35-39	7	60-64	1

in 1878. Factors interfering with normal respiration causing emphysema and too vigorous resuscitation appear to have been the chief causes.

The highest incidence in our group occurred between the ages of 20 to 24 years with 55 (47.8 per cent) cases. In fact 73 per cent of our cases first occurred between the ages of 15 and 29 years. This age incidence has been a common observation of numerous authors but no entirely satisfactory explanation has been offered. Only 31 in our group occurred after the age of 29 years and only 16 in persons older than 34. The oldest was 64 years.

Numerous authors have observed much more frequent occurrence of simple spontaneous pneumothorax among males than among females. In this group 98 (85 per cent) are males and 17 are females. This sex difference has not been satisfactorily explained.

Among the 115 cases here reported spontaneous pneumothorax occurred on the left side in 64, the right side in 42 and bilaterally in nine.

Diagnosis

Symptoms at onset vary from those unrecognized by the individual to those of extreme severity. In two of our cases pneumothorax was found while they were being examined for other reasons with no symptom having been experienced when the collapses occurred.

In 78 (67.8 per cent) individuals the first symptoms were severe enough to impress them with the exact time when the condition occurred. Most frequently the initial symptom was sharp pain. This occurred over various parts of the involved side of the chest including upper and lower axillae, precordium, shoulder and in some cases substernal regions. Not infrequently the pain radiated down the arm and in a few the arm had felt numb. In some cases the initial pain was excrutiating and in one it was localized over the epigastrium.

Usually pain preceded dyspnea. In some cases severe dyspnea did not occur. In others shortness of breath was the first symptom with or without subsequent pain. One stated that he felt his air was suddenly shut off - pain followed.

A man of 39 years was watching a parade. As he turned to leave sudden excrutiating pain on the right side of his chest made him believe he had "been shot." Shortness of breath followed and by the time he reached his hotel one block away he was unable to speak to the clerk, who immediately placed an emergency call for a physician.

In one patient the pain and dyspnea were so severe that he suddenly fell to the floor in shock. In another the pain in the right axilla extended down the right arm which became numb, he perspired profusely and became weak and shaky. A third also described symptoms of shock.

In only two cases were chills and fever present immediately preceding or following the attack. Although spontaneous pneumothorax has accompanied pneumonia, no evidence of this disease was found in these persons.

In two cases, positive intrapleural pressure developed so rapidly that the individuals were near death at the time they were first seen, with respiratory rates approximately 50 per minute, pulses practically imperceptible and marked cyanosis.

In most of our cases the severe symptoms began to subside within a few hours and soon disappeared. However, occasional chest pains occurred for several weeks to a few months and in some cases for years following the initial attack.

In the remaining 35 the onset was gradual. The first symptoms experienced were mild such as slight aching in a shoulder, mild chest pain on changing posture or occasionally on respiration, a feeling of oppression or slight pressure on the involved side. One experienced only dizziness. Three described gurgling in their chests. A considerable number of these persons did not consult physicians for several days or more than a week after the first mild symptoms appeared.

No symptom is pathognomonic for simple spontaneous pneumothorax. However, in every person who presents the symptoms above enumerated whether with gradual or sudden onset, this condition should be considered.

Activity of the individual at the time of onset affords no help in diagnosis. Table 2 reveals a large variety of activities at the time the first symptoms were noticed. In 26 the onset was so gradual it was not associated

TABLE 2

Activity at Time of Attack

No unusual activity	26	Washing hands	1
Sleeping	10	Working on scaffold	1
Time unknown	2	Hooking casting	1
Arising in morning	4	Running	3
Eating breakfast	4	Lifting	7
Sitting	5	Climbing hill, stairs, ladder	4
Standing quietly	4	Immediately after	
Riding	4	bicycling	1
Doing laboratory experiment	1	physical ed. class	1
Painting stencil	1	wrestling	1
Developing photo print	1	playing trombone	1
Studying in bed	1	Coughing, laughing sneezing,	
Walking	12	asthmatic attack	5
Fixing furnace 2 AM	1	Not recorded	11
Shaving	1		
Brushing teeth	1		

with any particular activity. Two did not know when the attack occurred. These individuals could think of nothing unusual they were doing at the time of the initial attack. Ten were awakened from sleep with pain in the chest. In fact, 31 were engaged in slight activity and only 25 were doing or immediately before were engaged in more strenuous work such as lifting, climbing and coughing. In 11 cases our records do not contain this information.

The findings on conventional physical examination depend upon such factors as degree of collapse and how soon the individual is seen. In severe cases examined within an hour or so after onset, inspection reveals evidence of cyanosis, anxious expression, and in some signs of shock. Respiratory rate is accelerated and the movements of the chest wall on the involved side are definitely limited. If intrapleural positive pressure is present, the intercostal spaces may bulge. At the opposite extreme when only a small amount of air has reached the pleural space the only finding on inspection may be slight limitation of movement of the chest wall.

Percussion of the chest with an underlying extensively collapsed lung usually reveals a resonant or even tympanitic note. When there is marked tension the chest wall acts like an overstretched drum and cannot vibrate. Thus, the percussion note becomes muffled and at times approaches dulness. In small pneumothoraces there may not be enough air

present in the pleural space to alter the percussion note.

When the lung has collapsed to 50 per cent or more of its volume breath and voice sounds are usually faint or entirely absent on auscultation. In small pneumothoraces all auscultatory findings may be normal.

The conventional physical examination is of almost no aid when small amounts of air such as 200 to 300 cc. are present in the pleural space.

Fluoroscopic and x-ray film inspection of the chest play an important role in detecting unsuspected and confirming questionable cases and determining the degree of collapse as well as any appreciable displacement of the mediastinum and diaphragm. X-ray inspection reveals a mantle of decreased density at the periphery of the lung with absence of lung markings and demarcated from the atelectatic lung margins. When only a small amount of air is in the pleural space it may be completely overlooked by x-ray inspection. Again air may be pocketed in front or back of the lung so it is not visualized on the usual posterio-anterior exposure. All of the cases here reported were plainly in evidence on x-ray films.

Among our 115 cases in the initial attack the degree of collapse was less than 25 per cent in 15. (Table 3) In seven the collapse was approximately 25 per cent; in eight 33 per cent; in seven 40 per cent; in 33, 50 per cent, in 11 from 60 to 75 per cent; and in 34 the collapse was complete.

TABLE 3

Degree of Collapse

Number	Per Cent
15	Less than 25
7	25
8	33
7	40
33	50
11	60-75
34	100

The diagnosis of simple spontaneous pneumothorax is made by correlation of history, conventional physical signs and x-ray shadows. In some cases one or more of these is misleading and therefore differential diagnosis may be difficult. From symptoms alone simple spontaneous pneumothorax has been diagnosed as pneumonia, intercostal neuralgia, pleurisy, coronary disease, acute heart failure, pericarditis and even perforated gastric lesions. All of these conditions were diagnosed or considered in some of the persons of this group before the final diagnoses were made.

The findings on conventional physical examination may be confused with those of large pulmonary cavities, marked upward displacements of one side of the diaphragm, with large collections of gas in the digestive organs, diaphragmatic hernia, gaseous sub-diaphragmatic abscess, pulmonary aplasias and pulmonary cysts.

Various x-ray procedures aid in differentiating between some of these conditions. However, there are those, particularly lung cysts, that cannot be differentiated from simple spontaneous pneumothorax by this method. In such cases thoracotomy is necessary to determine the actual condition.

The prognosis in uncomplicated simple spontaneous pneumothorax is excellent from the initial attack since the lung usually re-expands from within a few days to a few weeks. The prognosis can become exceedingly grave when extensive and prolonged bleeding occurs within the pneumothorax cavity or when positive air pressure develops. Subsequent to simple spontaneous pneumothorax, five of our patients died from other causes. One, from cerebral hemmorrhage at the age of 22 years. One died in an accident at the age of 19 years, two from malignancy both at the age of 48 and one, a United States marshall, was shot at the age of 47 by a prisoner (Reported by A. A. Wohlrabe, Minn. Med. 15:182, 1932). Of all of our patients traced to date, none has died from spontaneous pneumothorax or its complications.

Treatment

In the earlier years of our work many physicians still entertained the belief that most persons with spontaneous pneumothorax were tuberculous even though pulmonary lesions could not be demonstrated; therefore bed rest for at least one year was recommended. This was employed for our first patient in 1923 (Table 4).

TABLE 4

Treatment

	No.
No hospital or bed rest	41
Hospital or bed rest 1 week or less Average 4.7 days	26
Hospital or bed rest more than 1 week Average 19.9 days	35
Hemothorax Average 26 days	9
Bed rest one year	1
Sanatorium and artificial pneumothorax	1
Artificial pneumothorax at home	1
Surgery	1

Forty one had no hospital or bed rest but were advised to reduce physical activity to a minimum until their lungs reexpanded. Twenty six were in bed in hospitals or homes for one week or less. The average was 4.7 days. Thirty six were on bed rest in hospitals or homes for more than one week. The time ranged from eight to 50 days with an average of 19.9 days. In nine with hemopneumothorax the period of confinement to bed ranged from four to 60 days with an average of 26 days. One of our early cases was on bed rest for a year (1928). Two had bed rest and artificial pneumothorax at home (1928 and 1930). One was sent to a sanatorium where the collapse was continued by artificial pneumothorax (1943) and one had surgery (1951).

Many cases of simple spontaneous pneumothorax require no treatment except sedation at the beginning and reduced physical activity. For others bed rest at home or in a hospital is advisable for a few days to a few weeks depending upon degree of collapse and rate of reexpansion. The pleural rent soon closes and the lung reexpands without incident in most cases. As the lung reexpands activity may be increased gradually and the majority of cases treated by bed rest are able to resume normal living in a few days to a few weeks. Throughout the reexpansion period the individual should be instructed to report promptly the appearance of any symptom such as pain, shortness of breath or pressure. Frequent fluoroscopic or x-ray film inspections should be made until the lung appears to be completely reexpanded.

In uncomplicated cases aspiration of air either by needle or catheter is neither indicated nor advisable. The creation of abnormal negative pressure in the pleural cavity may keep the pleural rent open.

Table 5 shows the approximate time required for lungs to re-expand following first attack of simple spontaneous pneumothorax. The time was not determined in four because pneumothorax was continued artificially or surgery was performed. The time is not known in five because we were unable to trace them or x-ray films of the chest were not made at sufficiently short intervals. Of the remaining 106, 10 days was the shortest time and the longest was three months.

TABLE 5

Time Required to Reexpand

	No.
10 days	3
2 weeks	14
3	34
4	29
6	15
7	3
2 months	7
3	1
Surgery	1
Cont'd. on pneumothorax	3
Unknown	5

Complications

Pleural Fluid. In the 115 cases no pleural fluid was seen in 72. Enough accumulated to be detected by x-ray inspection in 43 of whom 39 had only a small amount. There were three with moderate accumulations which absorbed within a few days without removal. One had a large effusion. This man of 21 years developed simple spontaneous pneumothorax in January, 1951 which was soon followed by chills and fever. Eight days later 1500 cc. of cloudy yellow fluid was removed from the left pleural space (reported sterile). A catheter attached to a Stedman pump was introduced one week later and continued for approximately a month. During this interval streptokinase and streptodornase were introduced on three different occasions. He was hospitalized 51 days. The only residuals now in evidence are obliteration of the left costophrenic angle and thickened pleura over the apex of the left lung.

When fluid in small or larger amount does not absorb promptly it should be removed by needle or catheter as frequently as necessary to keep the pleural surfaces as dry as possible. Otherwise the lung will not re-expand promptly and fibrin is likely to deposit on the pleural surfaces greatly delaying and even preventing reexpansion after which decortication may be necessary.

Hemopneumothorax. Occasionally when the visceral pleura ruptures blood vessels are torn with bleeding into the pleural cavity. This may be in small or large quantities. In all cases of hemopneumothorax blood should be aspirated although it usually does not result in pleural changes requiring decortication. If the hemorrhage persists unduly long or a dangerous amount of blood is being lost, bleeding points should be closed surgically and at the same time the rent in the pleura repaired. In severe cases blood transfusions may be necessary.

Spontaneous hemopneumothorax occurred in nine of our cases as follows:

1. A man of 24 years had spontaneous hemopneumothorax discovered January 2, 1930. Needle aspiration was performed on several occasions. He was hospitalized 50 days. The collapse was artificially maintained for several months. The only residual now in evidence is obliteration of the left costophrenic angle.

2. This man of 34 years was admitted to a hospital November 14, 1930 with tension hemopneumothorax. Large amounts of blood and air were aspirated. He was transferred to a veterans hospital but complete study revealed no evidence of tuberculosis. He had no further chest trouble but died March 14, 1948 from carcinoma of the prostate with metastases.

3. A man of 23 years was found to have spontaneous hemopneumothorax on April 26, 1941. Blood was aspirated on three successive days - a total of 2500 cc. He was on bed rest at home for two weeks. His chest now appears normal.

4. A man of 39 years was admitted to a hospital on October 23, 1941 with spontaneous hemopneumothorax. Bloody fluid was aspirated 10 times. He was hospitalized 26 days. His chest now appears normal except evidence of slight thickening of the pleura over the entire left side.

5. A man of 31 years developed spontaneous hemopneumothorax September 12, 1944. Two days later 1100 cc. of bloody fluid was removed from the right pleural cavity. On September 27, 1600 cc. of dark brown thin fluid was aspirated. Oxygen was prescribed for dyspnea and one blood transfusion was given. He had a mild recurrence in 1946 and has since remained well.

6. A girl of 17 years developed spontaneous hemopneumothorax on January 22, 1948. Blood was aspirated on three occasions a total of approximately 2000 cc. She was hospitalized 26 days and her lung was reexpanded in about three months. Her chest now appears entirely normal by all phases of the examination. (This case was reported by Dr. G. J. Kertesz, Journal-Lancet, 70:143, 1950.)

7. A man of 20 years was found to have spontaneous hemopneumothorax on March 31, 1948. Blood was aspirated from the left pleural cavity on two occasions, a total of 700 cc. He was hospitalized 12 days. His chest now has an entirely normal appearance.

8. This man of 34 years developed spontaneous hemopneumothorax on July 11, 1949 on the right side. One thousand cc. of sanguineous fluid was aspirated. He was hospitalized 18 days and the lung was expanded in three weeks. The only demonstrable residual is an area of thickened pleura in the right costophrenic angle.

9. A man of 31 years developed right spontaneous hemopneumothorax on August 31, 1951. He was treated surgically September 14 and has remained well.

Tension Pneumothorax. Occasionally when pleural rupture occurs, a flap remains which acts as a one-way check valve. Thus air enters the pleural space on inspiration, but closure of the valve prevents its escape on expiration with the development of positive pressure. This situation immediately becomes an emergency as pressure may result in marked mediastinal displacement with severe embarrassment of respiratory and cardiac function. If the condition is not recognized death can occur in a short time.

If diagnosis is made in time, prompt relief is observed by thrusting an 18-gauge needle through the chest wall. Enough air escapes to reduce remarkably the pressure in a few seconds. It may then be necessary to pump air from the pleural cavity but high negative intrapleural pressure should be avoided. After the initial aspiration the patient must be kept under constant observation for pressure symptoms. If these occur other aspirations may be necessary. In some cases, after the needle is removed air accumulates so fast and aspirations are required so frequently that an indwelling needle or catheter and check valve are advantageous to provide continuous escape of air until it no longer accumulates sufficiently to cause pressure. At all times high negative intrapleural pressure should be avoided.

If the lung does not re-expand and positive pressure continues to develop after aspiration is discontinued it may be necessary to close the opening in the visceral pleura surgically. This also applies to lungs that do not re-expand with reasonable promptness in the absence of positive intrapleural pressure. In such cases the pleural openings may be so large that they will not close spontaneously and chronic pneumothorax results.

Serious tension with the initial attack occurred in only two cases in our group. One had hemopneumothorax and the other simple spontaneous pneumothorax. The pressure symptoms were so extreme that passage of a needle into the pleural space apparently was life saving. In two other cases air was removed when mild pressure symptoms appeared. The degree of collapse is not an indication for removal of air. In our 34 cases of complete collapse of one lung air was aspirated in only four because of evidence of positive intrapleural pressure. The remaining 30 re-expanded satisfactorily. However, blood was aspirated in all who had spontaneous hemopneumothorax and fluid was removed from one with a large volume of nonsanguineous accumulation.

Tuberculosis. In our 115 cases, 41 had tuberculosis as manifested by the tuberculin reaction. However, in none of them was there evidence of clinical lesions at the time of or immediately after the lungs had re-expanded. There was no reaction to tuberculin in 71 indicating total freedom from tuberculosis. The result of the tuberculin test was not recorded in three.

Three of the group of 115 subsequently developed clinical tuberculosis. One at the age of 20 years in 1923 had spontaneous pneumothorax on the left side. There was a history of exposure to tuberculosis in the family and he was a reactor to tuberculin. When the lung re-expanded no evidence of clinical disease could be detected. Nevertheless he was placed on bed rest for one year after

which he gradually resumed complete activity. Five years after pneumothorax had occurred, he was found to have moderately advanced disease in the upper lobe of the left lung with tubercle bacilli in his sputum. Apparently adhesions had not formed following the spontaneous pneumothorax and an excellent collapse of this lung was obtained artificially. He made an excellent recovery. Later a lesion appeared in the upper lobe of the right lung. This was treated successfully. He has remained well, worked regularly and in 1952 he was accepted for life insurance.

A man of 24 years who had spontaneous hemopneumothorax in 1930 and was treated successfully presented no evidence of clinical tuberculosis although he reacted to tuberculin. In 1935 he was found to have moderately advanced tuberculosis in the right lung which was promptly treated successfully.

A man of 19 years had spontaneous pneumothorax on the left side in February 1928. He did not react to tuberculin and no evidence was found of disease after his lung had re-expanded. In the fall of 1942 he shared a hotel room for about three weeks with a man who was later found to have active tuberculosis. X-ray film inspection in 1945 revealed no evidence of disease. The tuberculin test was not administered until 1946 when he was found to be a reactor at the age of 37 years. He then had active tuberculosis in the apex of the lower lobe of the left lung. His sputum contained tubercle bacilli. Artificial pneumothorax was instituted with satisfactory collapse. He was in government work and was treated in a federal institution. He was discharged in 1948 but continued to receive pneumothorax refills on an outpatient basis. He worked for one year.

In June 1949 he developed spontaneous pneumothorax on the right side while the left lung was still collapsed artificially. In 10 days after the spontaneous collapse, surgery was performed on the right side and the lung completely re-expanded. In August 1949 he developed pleurisy with effusion on the right side. Tubercle bacilli were recovered from fluid. He was again hospitalized for three months.

The left lung failed to re-expand when artificial pneumothorax was discontinued in January 1950. Decortication was done in May with complete expansion of the lung. Since that time his chest condition has remained satisfactory.

Occasionally in persons receiving artificial pneumothorax treatment for tuberculosis, spontaneous pneumothorax occurs on the opposite side in the absence of any demonstrable disease in the lung or of mediastinal herniation as in the case just cited. This condition may occur at any time during treatment intervals.

In one case (not included in this study) who had been on artificial pneumothorax without complication over a considerable time spontaneous pneumothorax began to appear on the same side within an hour or so after refills. There was no likelihood that the lung had been punctured by the pneumothorax needle and it was assumed that a rent in the visceral pleura occurred at the attachment of an adhesion. At first one or two needle aspirations sufficed but finally this was not adequate and continuous aspiration became necessary for many hours. These attacks became so alarming that artificial pneumothorax was abandoned.

Bilateral Pneumothorax. Occasionally bilateral simple spontaneous pneumothorax is present when the first examination is made. This was found in one of our cases. This man of 28 years had this condition when his first symptoms occurred in November 1931. The right lung was completely collapsed and there was a pocket of air over the apex of the left lung. Both lungs re-expanded satisfactorily and no evidence of pulmonary disease could be found. (This case was reported by Dr. R. B. Hinckley, Journal-Lancet, 52:330, 1932.) This man had many bilateral recurrences during the next 20 years and died from malignancy in 1951.

Among persons who have recurrent

simple spontaneous pneumothorax some have alternating attacks on the two sides and occasionally both lungs are found partially collapsed simultaneously. Bilateral collapse has occurred in nine of our 100 traced cases.

Recurrence. Brock has reported the largest and best presented series of cases of recurrent and chronic spontaneous pneumothorax from which he draws excellent conclusions from the standpoints of diagnosis and treatment.

We have kept in touch with or have recently traced 104 of our 115 cases. Only 100 are here considered as the remaining four have had six months or less observation. In 71 of the 100 there has been no recurrence. The number of years since the initial episode is shown in Table 6.

TABLE 6

No Recurrence

No. Persons Traced	No. Years Since Attack	No. Persons Traced	No. Years Since Attack
1	29	3	12
2	27	4	11
2	24	3	10
5	22	3	9
2	21	1	8
2	20	3	7
1	19	3	6
2	18	1	5
1	16	8	4
4	15	3	3
2	14	7	3
2	13	6	1

Howey 6 mos.
Lochrem 3 mos.
Dotray 2 mos.
Striano 1 mo.

Seventeen have had one recurrence. However in five of them the second attack appeared within two months or less after the first thus it is probable that the original rent reopened. In any event the 17 cases are shown in Table 7 with the time intervals between the first and second attacks, between the last episode and the total time that has elapsed since the first attack. In 15 of these cases the second collapse occurred on the same side as the first.

Twelve have had more than one recurrence. The numerals in columns two and three, Table 8, indicate the number of attacks on the same or the opposite side. All had recurrences on the same side and six also on the opposite side. Four with bilateral recurrences had so many attacks that they do not remember the number. On occasions both sides were partially collapsed simultaneously.

Two who have had many attacks on both sides never had bed rest for more than occasional brief intervals. They stated that they became accustomed to the condition and continued with their work. One had his last recognizable attack in 1947 and died from malignancy in 1951. The other has not had an attack during the past six years and is leading an active life.

A man of 39 years who began having frequent bilateral attacks in 1941 became so incapacitated that finally mild irri-

TABLE 7

Recurrent Spontaneous Pneumothorax

One Recurrence

	Same Side	Opposite Side	Interval Between First and Second	Time Since Last Attack	No. Years Since First Attack
Engstrom		x	21 years	5½ years	24
Wallace	x		5 months	20 years	21
Schweppe	x		9 years	12 years	21
Lewis	x		2 months	15 years	15
Van Dusen	x		1 yr. 9 mos.	11 years	13
May	x		6 weeks	10 years	10
Ambrose	x		3 years	6 years	9
Jacobsen	x		6 years	3 years	9
Kalinoff	x		1 yr. 9 mos.	8 years	9
Paulson	x		4½ years	3 years	8
Teska	x		6 weeks	7 years	7
Raycroft	x		2½ years	3 years	6
Nolan		x	4½ months	4 years	4
Sorlie	x		2 months	3 years	3
Hill	x		2 years	1 year	2
Meagher, T.	x		6 weeks	2 years	2
Doe	x		7 months	11 months	1

TABLE 8

Recurrent Spontaneous Pneumothorax

Two or More Recurrences

	Same Side	Opposite Side	Interval Since Last Attack	Number Years Since First Attack
Brock	Many bilateral		6 years	20
Cole	Many bilateral		Chronic	16
Brumfield	Numerous		6 years	15
Lally	Many bilateral		6 years	13
Russell	5		2 years	13
Wood	Many bilateral		9 years	11
Kennedy	4	4	2 years	10
Thorson	3		7 years	9
Larson	4		5 years	8
Sandeen	2	1	4 years	8
Fenell	3		1 year	6
Lundgren	2	1	2 years	6

tants were placed in the pleural cavities (lipiodol on one side and glucose solution on the other) to produce adhesions between the visceral and parietal layers of pleura. This was so effective that he has had no attack in the past nine years. However he had a severe cardiac attack in 1952 which proved to be due to

cor pulmonale.

A woman of 23 years had complete collapse of the right lung when first seen in 1937. No aspiration or surgery was done at that time. Later she was in another state where air was aspirated from time to time. In 1946 pneumonectomy was performed when a large cyst was found communicating with a bronchial ramification with a check valve. It was not determined whether on previous occasions the cyst had ruptured into the pleural cavity or whether each collapse of the lung was due to pressure within the cyst. The latter seems probable. Since 1946 she has remained in reasonably good health.

A man of 21 years had an attack of spontaneous pneumothorax in November 1936. In subsequent years he had recurrences involving both sides. In 1944 in military service he was told that 10 per cent permanent pneumothorax was present at the apex of each lung. He was later discharged from service with a diagnosis of cystic disease. For several years he has resided in another state where his working capacity has been only slightly limited. The partial collapse of the apex of each lung has remained unchanged since 1949. It seems probable that his acute episodes were due to ruptured cysts and that the present apical conditions are due to the cysts themselves.

Treatment of recurrent simple spontaneous pneumothorax depends upon a number of factors such as frequency and severity of attacks. In our 100 traced cases 17 have had only one recurrence. Among the 12 who have had more than one recurrence eight have had more than three attacks. Apparently an attack of simple spontaneous pneumothorax usually does not result in symphysis of the pleurae. When spontaneous pneumothorax has recurred several times we have usually recommended procedures to prevent further attacks. In the earlier years of our work attempts were made to adhere the visceral and parietal layers of pleura by introducing into the pleural space 25 to 50 cc. of a mildly irritating substance, such as hypertonic glucose solution (30 to 60 per cent), lipiodol, or mineral oil. Brock[15] has found the production of pleurodesis by applications of silver nitrate the most satisfactory method he has employed. Recently we have recommended that chest surgeons explore and close rents in the pleura, remove pleural blebs in evidence and produce slight irritation of pleural surfaces by gentle friction with a sponge to insure pleurodesis.

Prevalence

Our observations lead us to believe that simple spontaneous pneumothorax occurs far more frequently than the literature indicates. In discussing the subject with physicians it is not unusual to learn that many have seen from one to several cases which have not been reported. Occasionally persons are found to have this condition during examinations for other purposes without symptoms or other evidence of its presence. Some individuals with mild or moderate symptoms at onset do not report to physicians. A considerable number of our cases were not examined for from several days to more than a week after symptoms appeared. They probably would not have reported had symptoms been of shorter duration. Others are not diagnosed by their physicians but are treated for pleurisy, pneumonia, cardiac disease, etc.

The possibility of previous attacks having occurred among our cases was obtained from histories of symptoms. Two had spontaneous pneumothorax definitely diagnosed on one occasion each (one patient three years, another one year) before our first examination. Five stated that they had similar attacks of symptoms on from one to several occasions but never reported for examination before we saw them. It is not possible to be sure that they had spontaneous pneumothorax. However, one who had three such previous attacks described symptoms of shock on one occasion. On December 26, 1952 a nurse in our group wrote, "I have often thought that I probably had these attacks long before the diagnosis was made in 1939. While I was in training, 1925 to 1928, I had several attacks of so-called pleurisy. They were similar but less severe than later."

In five other cases attacks had occurred from a few months to five or six years before which they described as almost identical as far as symptoms were concerned. They reported to physicians and four were treated for pleurisy and one for pericarditis but no x-ray inspection of the chest was made.

Prevention

As yet no method has been devised to determine in whom this condition may occur since in most persons who have it x-ray inspection fails to reveal evidence of pleural blebs either before the attack or after the lung is re-expanded. In an occasional person who has not had an attack, however, pleural blebs or bullae may be sufficiently large to be detected on x-ray films. This does not necessarily indicate that such persons will have spontaneous pneumothorax. However, the potentiality might be reduced by removal of such blebs as can be found.

It is not known what causes blebs to rupture. We have seen persons who have had spontaneous pneumothorax onsets when engaged in most strenuous physical work and others while they were relaxed in sound sleep as well as those engaged in various intermediate degrees of activity. It appears that in some cases the visceral pleura becomes so thin or the pressure so high in the blebs that the increase in negative intrapleural pressure on quiet inspiration is adequate.

Litigation has frequently resulted from spontaneous pneumothorax. The person who is uninformed concerning this condition is likely to attribute sudden intense pain and dyspnea to his activity at the onset or to some strenuous work during the past few days. In such cases the physician is hard pressed to state with any certainty whatsoever that the condition would not have occurred even during sleep.

In persons who have had one or more attacks of spontaneous pneumothorax one should always issue a warning of the possibility of subsequent attacks and the advisability of prompt contact with the physician with the onset of symptoms. Even a minor degree of shortness of breath may indicate that a check valve is present and positive intrapleural pressure is developing. Delay can prove fatal in a short time. On a few occasions when the onset of recurrences have been reported on the telephone we have asked that no time be wasted in calling an ambulance but that a member of the family or an associate promptly rush the individual to the ambulance entrance of the nearest hospital. An intern or resident physician is called and asked to be in readiness to aspirate air on the patient's arrival, if symptoms warrant. It is further requested that hospital authorities dispense with social service records, financial status, etc. until adequate treatment has been administered and the emergency has passed. In one of our cases who was promptly brought to a hospital by his wife on the appearance of the first symptoms, cyanosis and dyspnea were so extreme on arrival that the physician had to carry him from the automobile to the emergency room. The intrapleural pressure was so positive that when a needle was thrust through the chest wall there was momentarily a whistling of air rushing through the needle like that of a toy steam engine. Just as every physician is advised to have with him at all times tracheotomy equipment it is equally important that he always be prepared to promptly remove air in spontaneous pneumothorax emergencies.

Anyone who has had an attack of spontaneous pneumothorax should be advised against high altitudes especially in airplanes except in pressurized cabins and where oxygen can be administered. Persons recovering from spontaneous pneumothorax but in whom the lung is not completely expanded, no matter how small the amount of air still demonstrable, should also be warned against airplane travel except in pressurized cabins. The volume of air in a pleural cavity increases with altitude. Three thousand cc. in a pleural cavity at sea level assumes the volume of 3720 cc. a mile above. Therefore, a person with pneumothorax may be in distress at one mile and his life jeopardized at higher altitudes. At 18,000 feet the volume of air

is doubled and at 34,000 feet, quadrupled.

Many physicians recall emergencies that were created early in World War II by transporting spontaneous pneumothorax cases by airplane. The problem was of such magnitude that in July 1944 the National Research Council issued an excellent special pamphlet prepared by J. J. Waring, Denver, Colorado on the diagnosis and management of spontaneous pneumothorax[16]. This was made available to our military medical officers everywhere.

SUMMARY and CONCLUSIONS

1. One hundred and fifteen cases of simple spontaneous pneumothorax are reported ranging in age from 15 to 64 years. Nearly one-half occurred between the ages 20 and 24 years. Eighty-five per cent of the entire group were males. The condition occurred on the left side only in 64, the right side only in 42 and bilaterally in nine.

2. In 78, initial symptoms were severe consisting mainly of pain and dyspnea. In the remainder the onset was gradual. Activities of the individuals when attacks occurred varied from strenuous work to sound sleep.

3. History of onset, physical signs and x-ray inspection were employed in diagnosis but fluoroscopy and x-ray films were most valuable.

4. Various degrees of collapse were observed. In 34 cases it was complete.

5. Approximately one-third of these patients were treated ambulatorially while the remainder received bed rest, ranging from a few days to two months. Air was not aspirated except when positive intrapleural pressure developed. Thirty nine had small fluid accumulations which promptly disappeared without aspiration. Only one presented a large effusion which was removed. The nine cases of spontaneous hemopneumothorax were aspirated until all evidence of blood disappeared.

6. Serious tension pneumothorax occurred in only two cases. Air was removed promptly and as long as positive pressure continued to develop. From two other cases, air was removed with the first manifestation of positive pressure.

7. Among the 115, there were 41 who had tuberculosis as manifested by the tuberculin reaction but no evidence of clinical disease was found. Two developed clinical tuberculosis five years after pneumothorax occurred. Another who did not react at the time of the initial pneumothorax developed clinical pulmonary tuberculosis 18 years later.

8. Contact has been maintained or recently re-established in 104 of the 115 patients. Four have been observed for six months or less. The remaining 100 have been observed from one to 29 years.

9. Among the 100 cases, 71 have had no repetition and 17 have had one recurrence all on the original side except two. Twelve have had more than one recurrence, ranging from two to many. In six of these, all recurrences were on the original side.

10. Since 29 of this group of 100 traced cases have had one or more recurrences, everyone who has had an initial attack should be advised of the possibility of others and how to proceed in the event symptoms of tension pneumothorax begin to appear.

11. From these observations and the management of recurrent spontaneous pneumothorax, the procedure now recommended after two or more attacks consists of surgical closure of the rent, removal of blebs in evidence and producing slight irritation of the pleural surfaces by gentle sponge friction.

12. In all initial attacks as well as recurrences, accumulations of fluid, large or small, should be removed if they do not absorb within a few days to avoid deposits of fibrin on the pleural surfaces.

13. In all cases of spontaneous hemopneumothorax, blood should be removed as

often as necessary, transfusions administered when indicated and ligation of the vessel and closing the rent if copious bleeding persists unduly long.

14. Apparently simple spontaneous pneumothorax occurs more frequently than the literature indicates since not all physicians report their cases and many persons whose symptoms are mild do not consult physicians.

15. No method has been devised for the prevention of simple spontaneous pneumothorax but many recurrences can probably be prevented by surgical removal of blebs or producing symphysis of the pleurae.

16. Persons who have blebs or bullae demonstrated by x-ray inspection as well as those who have had one or more attacks of simple spontaneous pneumothorax, should avoid high altitudes except in pressurized cabins and where oxygen can be administered or a needle can be introduced into the pleural cavity in the event of emergency.

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II. MEDICAL SCHOOL NEWS

Coming Events

April 27-29	Continuation Course in Gastroenterology for General Physicians
April 28	Clarence M. Jackson Lecture; "Gastro-Intestinal Symptoms with Particular Reference to Motor Disturbance"; Dr. Chester M. Jones, Boston; Owre Amphitheater; 8:00 p.m.
April 29	Family Doctors' Day; Heart Hospital Theater; 1:30-5:30 p.m.
April 30	Medical Six O'Clock Club Dinner; Coffman Memorial Union Main Ballroom; 6:30 p.m.
May 4	Seminar on History of Medicine; "The History of Colon Surgery"; Dr. William C. Bernstein, Minneapolis; Todd Amphitheater; 7:45 p.m.
May 7	E. Starr Judd Lectureship; "The Endocrinology of Mammary Cancer"; Dr. Charles B. Huggins, Chicago; Owre Amphitheater; 8:15 p.m.
May 7-9	Continuation Course in Surgery for General Physicians
May 11-13	Continuation Course in Arthritis and Allergy for General Physicians
May 12	Duluth Clinic Lecture; "Some Aspects of Antibiotic Therapy"; Sir Alexander Fleming, London; Owre Amphitheater; 8:00 p.m.
May 13	Symposium on Antibiotics; Sir Alexander Fleming, London; Owre Amphitheater; 2:00 p.m.

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Dr. Huggins to Deliver Judd Lecture

Dr. Charles B. Huggins, Professor of Surgery, University of Chicago Medical School, will visit our campus on May 7 at which time he will deliver the annual E. Starr Judd Lecture. He will speak on "The Endocrinology of Mammary Cancer" at 8:15 p.m. in Owre Amphitheater.

Dr. Huggins' visit will coincide with a continuation course in Surgery for General Physicians which will be presented at the Center for Continuation Study from May 7 to 9. The course will deal with the management of certain malignant diseases as well as some common problems in general surgery. The visiting faculty for the course will include Dr. Harry Southwick, Assistant Professor, Department of Surgery, University of Illinois College of Medicine, Chicago, and the program will be presented under the direction of Dr. O. H. Wangensteen, Professor and Chairman of the Department of Surgery. The remainder of the faculty will include clinical and fulltime members of the staff of the University of Minnesota Medical School and the Mayo Foundation.

* * *

St. Joseph's Hospital Celebrates Centennial

The Medical School and its faculty salute St. Joseph's Hospital, St. Paul, which will observe its centennial celebration on May 16 and 17. As a part of this celebration a scientific session, with many outstanding participants, will be held in the Theatre Section of the St. Paul Auditorium on Saturday, May 16, All physicians wishing to attend are cordially invited. The features of the scientific program are listed on the following page.

(Continued on next page)

MORNING SESSION
Presiding: Justus Ohage, M. D.

9:00 a.m.	Rev. Francis W. Curtin	Invocation
	O. J. Campbell, M.D.	Message from the Minnesota Medical Association
	Philip F. Donohue, M.D.	Message from the Ramsey County Medical Society
9:30 -	Edward A. Boyden, M.D.	"Humoral vs. Nervous Control of the Gallbladder"
9:50 -	Byrl R. Kirklin, M.D.	"The X-ray Diagnosis of Gallbladder Disease"
10:10 -	Thomas J. Dry, M.D.	"Coronary Disease and the Gallbladder"
10:30 -	Warren H. Cole, M.D.	"Indications and Precautions in Cholecystectomy"
10:50 -	William Boyd, M.D.	"Tissue Permeability"
	Presiding: O. J. Campbell, M.D.	
11:20 -	Richard Trail, M.D.	"Experiences in Mass Photofluorography"
11:40 -	Armand J. Quick, M.D.	"Coagulation of the Blood"

AFTERNOON SESSION
Presiding: Eugene M. Scott, M.D.

2:00 p.m.	Rev. Joseph Kuncl	Invocation
2:00 -	Chevalier L. Jackson, M.D.	"The Development of Bronchoscopy"
2:20 -	Francis J. Braceland, M.D.	"Psychiatry and Religion"
2:40 -	Sir Alexander Fleming, M.D.	"The Discovery of Penicillin"
	Presiding: Wesley W. Spink, M.D.	
3:20 -	Burgess Lee Gordon, M.D.	"Women in Medicine"
3:40 -	Philip S. Hench, M.D.	"The Discovery of Cortisone"

A banquet will be held Saturday evening at 7:00 p.m. in the Continental Room of the St. Paul Hotel, and a brief program will be held at St. Joseph's Hospital on Sunday, May 17, at 2:30 p.m. with the Rev. Monsignor C. A. Towell and the Rev. John J. Flanagan, S. J., participating.

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Alumni News

Dr. Albert M. Snell, Med., '18, Palo Alto, California, has been selected as Chairman and Dr. Harold G. Scheie, Med., '36, Vice-Chairman of the Veterans Administration Council of Chief Consultants. Dr. Snell is a former professor of medicine at the Mayo Coundation of the University of Minnesota, and Dr. Scheie is Associate Professor of Ophthalmology at the University of Pennsylvania Medical School.

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Distinguished Visitors on Campus

Dr. Isadore Lampe of the Department of Radiology, University of Michigan, has been visiting the Department of Radiology this past week. He participated in several of the regularly scheduled meetings of the Radiology Department and delivered some special lectures.

(Continued on next page)

On Thursday, April 23, we were privileged to have as a visitor Dr. A. Bradford Hill of London who is visiting the University as a guest of the School of Public Health. Dr. Hill is Professor of Medical Statistics in the London School of Hygiene and Director of British Medical Research Council's Statistical Research Unit. Dr. Hill spoke on "The Philosophy of the Clinical Trial" at 3:30 p.m. in Owre Amphitheater.

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Faculty News

Dr. F. W. Hoffbauer, Associate Professor, Department of Medicine, and Dr. W. L. Williams, Associate Professor, Department of Anatomy, participated in the program on "Liver and Protein Metabolism" at the annual meeting of the American Society for Experimental Pathology in Chicago in early April. Dr. Williams also attended the Ohio State Meeting of the American Association of Anatomists in Columbus in March where he presented a paper.

A lecture entitled, "A Critical Survey of Current Approaches in Quantitative Histo- and Cytochemistry," was presented by Dr. David Glick, Professor of Physiological Chemistry, in New York on April 1 at the invitation of the American Chemical Society. Dr. Glick also presented a paper along with Mr. Som Nayyar on "The Determination of Protein in Microgram Quantities of Tissue" at the meeting of the Histochemical Society in Chicago April 5.

On April 9, Dr. Reynold A. Jensen, Professor, Department of Pediatrics and Psychiatry, attended a Forum on Mental Health and Education in Childhood sponsored by the Children's Hospital of Buffalo, New York, where he spoke on "Psychology and the School-Age Child."

The following members of the Department of Surgery attended the American Association for Thoracic Surgery Meeting in San Francisco, California, on March 27, 28, and 30: Doctors Richard L. Varco, E. John Lewis, Ivan D. Baronofsky, and C. Walton Lillehei. The American Surgical Association Meeting in Los Angeles on April 1, 2, and 3, was also attended by Dr. Owen H. Wangensteen, Richard L. Varco, F. John Lewis, and C. Walton Lillehei.

Dr. Berry Campbell, Associate Professor, Department of Anatomy, spoke on "The Dynamics of Early Brain Disease with Particular Reference to Multiple Sclerosis" at the Conference on the Status of Multiple Sclerosis, sponsored by the New York Academy of Sciences and the National Multiple Sclerosis Society, New York City, April 17 to 18.

The Department of Physiological Chemistry was well represented at the Meeting of the American Society of Biological Chemists in Chicago on April 6-9. Those who attended were: Doctors W. D. Armstrong, Leon Singer, William O. Caster, David Glick, C. P. Barnum, and Saul L. Cohen. Dr. Singer attended the meeting of the International Association for Dental Research in Philadelphia, Dr. Barnum, the meeting of the American Association for Cancer Research in Chicago, and Dr. Charles Carr the meetings of Electrochemical Society in New York City.

Doctors William G. Kubicek and Wesley D. Anderson of the Department of Physical Medicine and Rehabilitation attended the Federation of American Societies for Experimental Biology in Chicago recently.

Dr. Stella M. Sikkema, Physician, Students' Health Service, attended the American College of Physicians Postgraduate Course in Internal Medicine at the Mayo Clinic recently.

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III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

April 27 - May 2, 1953

Monday, April 27

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom; Todd Amphitheater, U. H.

11:30 - 12:30 Physical Medicine Seminar; Evaluation of Kinesiology of Occupational Therapy; Ruby Overmann; 132 Chemical Engineering Bldg.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:30 Physiology and Physiological Chemistry Seminar; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Atrial Septal Defect in Infants and Children; Robert Disenhouse; Sixth Floor West, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

4:30 - Public Health Seminar; 15 Owre Hall.

4:30 - 6:00 Physiology 114A and Cancer Biology 140 -- Research Conference on Cancer, Nutrition, and Endocrinology; Drs. Visscher, Bittner, and King; 129 Millard Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Ancker Hospital

8:30 - 10:00 Tuberculosis and Chest Conference; Auditorium.

2:00 - 3:00 Surgery Journal Club; Classroom.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Eldon Berglund; Newborn Nursery, Station C.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Erling Platou; Station K.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

2:00 - Pediatric Rounds; Robert A. Ulstrom; Stations I and J.

Monday, April 27 (Cont.)

Veterans Administration Hospital

1:30 - Cardiac Rounds; Drs. Ebert and Berman, and Richards.

4:00 - Cardiac Conference; Drs. Ebert, Berman, and Simonson.

Tuesday, April 28

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

12:30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Conference; Presentation of Cases from General Hospital; Drs. Lipschultz and Blank; Eustis Amphitheater, U. H.

* 8:00 - Clarence M. Jackson Lecture; Gastro-Intestinal Symptoms with Particular Reference to Motor Disturbance; Chester M. Jones, M.D., Clinical Professor of Medicine, Harvard University Medical School; Owre Amphitheater.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

9:00 - 10:00 Medical X-ray Conference; Auditorium.

Minneapolis General Hospital

9:00 - 10:30 Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall.

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:00 - Cardiac Rounds; Paul F. Dwan; Classroom, Station I.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

12:30 - Grand Rounds; Fractures; Willard White, et al; Sta. A.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall

1:00 - Tumor Clinic; Drs. Eder, Cal, and Lipschultz.

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Tuesday, April 28 (Cont.)

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Surgery Staff Seminar; A Study of the Effect of PAS on the Prothrombin Time and the Coagulation Time of Blood in Patients with Tuberculosis; John Linner; Medical Conference Room, Bldg. I.

9:30 - Infectious Disease Rounds; Drs. Hall and Zinneman.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery-Tumor Conference; L. J. Hay, J. Jorgens; Conference Room, Bldg. I.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

Wednesday, April 29

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

12:30 - 1:30 Medical Journal Club; Library.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

Wednesday, April 29 (Cont.)

Minneapolis General Hospital (Cont.)

12:15 - Pediatric Staff Meeting; Classroom, Station I.

1:30 - Visiting Pediatric Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Conference Room; Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Nesbitt, Zieve, Hay and Goodnow.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg. I.

2:30 - 4:00 Psychosomatic Rounds; C. K. Aldrich; Conference Room, Bldg. I.

4:00 - Combined Medical Surgical Conference; Drs. Flink and Hay; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics, Conference Room, Bldg. I.

Thursday, April 30

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

12:30 - Physiological Chemistry Seminar; Introduction to Scientific Research--Design of Experiments; Robert Bahn; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 Radiology Seminar; Medical Teaching Mission to India; Leo G. Rigler; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Ancker Hospital

8:00 - 10:00 Medical Grand Rounds; Auditorium.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

Thursday, April 30 (Cont.)

Minneapolis General Hospital (Cont.)

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - House Staff Conference; Station I.

2:00 - 4:00 Infectious Disease Rounds; Classroom.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Classroom.

Veterans Administration Hospital

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

1:00 - Metabolic Disease Conference; Drs. Flink, Heller, and Jacobson, and Bolin.

Friday, May 1

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 1:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; University Hospitals' Use of Nursing Homes; Annie Laurie Baker, Helen L. Knudsen; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Friday, May 1 (Cont.)

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Pediatric Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I, Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

2:00 - Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Saturday, May 2

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:00 Infertility Conference; Louis L. Friedman, David I. Seibel, and Obstetrics Staff; Eustis Amphitheater, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - Anatomy Seminar; Cerebral Cortical Activity Produced by Afferent and Antidromic Stimulation; Nathaniel A. Buchwald; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - 11:15 Hematology Rounds; Drs. Goldish and Bolin, and Howard.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide, Conference Room,

* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.