tion and correct interpretation.

In our patient there seemed to be two groups of R-R intervals between the paced beats, the shorter ones at 800 msec (in accord with the preset rate of 75 per minute) and the longer ones at 1,160 msec. The difference between the two is 360 msec, which is the interval between the pacing spike before each QRS complex and the peak or nadir of the ensuing T-wave (Fig 1A). This constant delay would indicate that the demand generator was intermittently recycling off a prominent T-wave. The latter was unusually sharp and of large amplitude, especially in the unipolar endocardial electrogram recorded from the tip electrode where it was associated with a contact current and the distance between the beginning of the QRS complex and the inflection point of ST-T junction measured 360 msec (Fig 1B). When pacing became again regular at 75 per minute 12 hours later (Figure 1C), the endocardial electrogram no longer depicted a contact current (Fig 1D). Obviously the electrode must have gradually moved away from its original juxta-endocardial position during the ensuing 12 hours, as evidenced by a significant difference in the configurations of the R and T waves between the two electrocardiograms (Fig 1A and 1C).

The most important consideration in producing effective demand pacing by transvenous route is intimate endocardial contact by the electrode. When the contact is poor, the demand pacemaker fails to sense the R-wave and thus will not function properly. On the other hand, if the contact is too close between the electrode and the ventricular endocardium, too large a contact current and too sharp a T-wave will trigger the inhibitory circuitry and thus cause, as in our case, a slower discharge rate of the pacemaker than it was set for.

Prevention and treatment of this unusual cause of pacemaker malfunction is, first, to advance the transvenous electrode to the apex of the right ventricle till a contact current is recorded from the distal electrode and then to back off till there is no longer or very little ST elevation on the unipolar intracardiac electrogram. If marked variation of pacing rate persists and cannot be overcome by either increasing the pacer rate or decreasing the pacemaker sensitivity, then either a new endocardial site must be chosen or the electrode system be converted to a unipolar configuration.

This maneuver of electrode placement is of far greater importance when the electrode is passed "blindly" than when under fluoroscopic control. Correct positioning of the float-in pacemaker-electrode is determined by recording a distinct contact current on the intraventricular electrogram from the tip electrode.1 On the other hand, since it is difficult to ascertain without fluoroscopic control the relative position of the electrode with reference to the endocardial surface in the absence of a contact current on the intracardiac electrogram, placement of pacing electrode by the float-in technique is far less exact and reliable than under direct fluoroscopic control.2,3

It is the purpose of this communication to report this unusual cause of pacemaker irregularity, not only because it may stimulate further reports from other workers in this field so as to assess its frequency of occurrence, but because once recognized its "cure" is a relatively simple matter.

ACKNOWLEDGMENT: The authors wish to thank Mr. John Maney, Product Specialist, Medtronic, Inc., Minneapolis, Minnesota for his valuable suggestions.

REFERENCES

Reprint requests: Dr. Cheng, D.C. General Hospital, 19th and Massachusetts, SE, Washington, D.C. 20003

Calcification in Chickenpox pneumonia*

Louis Raider, M.D., F.C.C.P.

The relationship between pulmonary calcifications and chickenpox pneumonia is reviewed. A case is presented in which pulmonary calcification developed over a seven-year interval following Varicella pneumonia in a patient who had a previously negative chest radiograph. After accidental death, an autopsy was performed and the pulmonary findings are presented. It is felt that an etiologic relationship has been established between the patient's Varicella pneumonia and her subsequent pulmonary calcifications.

During the past ten years many papers have appeared describing calcification following chickenpox pneumonia. The occurrence of this type of calcification in one of my colleagues and her death and the autopsy results were felt worthy of report. Negative films prior to the acute infection were available as well as annual radiographs during the subsequent seven years while calcification developed. Since there was no evidence of histoplasmosis or tuberculosis this seemed to qualify as the ultimate proved case which prior authors have envisioned.

REVIEW OF LITERATURE

Much of the credit for making the profession aware of focal calcifications in the chest as a result of chickenpox pneumonia goes to authors from Australia and New Zealand. Of these Mackay and Cairney2 are generally credited with the first description of this entity in 1960. Review of the

*From the Department of Radiology, Providence Hospital, Mobile, Alabama.
literature, however, reveals that an earlier description of calcification following Varicella pneumonia appeared in a paper published by Buechner in the Annals of Internal Medicine in October of 1950. During the 1960s, a series of papers by Drs. Kowtew, Abrahams and Stringer was published which presented many cases and convincing evidence of the association of Varicella pneumonia and pulmonary calcification. More recently papers on this subject have appeared in the British, Scandinavian and continental literature, as well as in the American publications.

Case Report

Our patient was a 29-year-old woman physician who on March 1, 1950, became acutely ill with chills and fever. After approximately 72 hours a rash developed. She had pain in her left upper quadrant and a hacking cough as well as expiratory wheezes. The cough and rash appeared simultaneously. Her temperature was 99.8°F but within 24 hours went to 104°. The patient had generalized rales and a rash typical of chickenpox. She was severely dyspneic. The blood count was consistent with a viral infection. Agglutinations were negative except for 1 to 20 para typhoid A. Blood cultures were negative. Sputum culture grew alpha Streptococcus. Result of complement fixation study for histoplasmosis was negative.

A radiograph of chest taken on March 10 during the acute illness revealed a rather classic alveolar pneumonia process consistent with Varicella pneumonia. It contained
diffuse nodular densities throughout the lung fields with some confluency particularly in the mid portions of the lungs.

A film taken ten days following the initial study (Fig 1) revealed even more pronounced changes in the alveoli and more diffuse distribution than was noted on the initial film. Vital capacity was 30 percent of normal. There was gradual clinical improvement but regression of radiographic findings was slow. On June 10, three months after the onset of illness, the patient's chest radiograph had returned to normal.

Following her residency this doctor established an office for the practice of internal medicine in Mobile and reported at regular intervals for chest films. A film taken in September, 1960, showed findings interpreted as minimal fibrosis. By February, 1963, we began to see more significant fibrosis throughout both lung fields and on the left, nodular foci of increased density. In 1964 (Fig 2) these foci were more distinct and had become faintly calcific. Her last examination in November of 1966 just prior to her death (Fig 3) revealed even more pronounced and widely distributed calcific foci. Shortly after the last radiograph was taken the patient died as a result of an accident and autopsy was performed. We were able to obtain the viscera, and a radiograph of the lungs after they were fixed (Fig 4) demonstrates the fibrotic and calcific changes in the lungs.

Autopsy Findings

The left lung weighed 750 gm and the right 675 gm. Each was rather firm in consistency and maintained its shape when the thorax was opened. The pleura of each lung was normal in color and texture, translucent, without scarring or abnormal pigmentation. No free fluid was present. On section one could see fine nodular densities throughout the lung fields. These lay approximately 2 cm apart consisting of 1 to 2 mm white calcified central collections, surrounded by areas of induration approximately 5 to 6 mm in diameter. The intervening lung tissue was relatively normal, although a suggestion of induration could be detected. A rather striking aspect of the pattern was the uniform distribution of findings throughout all lobes of each lung. The tracheal and bronchial mucosa was clear and no lesions were noted therein. The mediastinal nodes showed scattered calcific lesions similar to those noted in the lungs. No lymph node enlargement was found.

Microscopic sections showed discreet foci of dense fibrous connective tissue with central portions calcified, which stained deeply purple with homogenization of the central areas. Centrally in the nodules were calcific bodies, tiny and discreet but of varying size. About these was homogeneous collagenous connective tissue, and surrounding this a relatively cellular zone of fibroblasts. A few mononuclear giant cells of Langerhans' type were present. Numerous alveolar macrophages were noted and a protein rich edema fluid was seen in the alveolar spaces. Considerable autolysis was noted in the lungs with loss of architecture. Abundant hemosiderin was present associated with the nodules. The central portions of many of the latter had fallen out leaving central vacuoles in which calcific debris was found.

Results of stains for acid-fast bacilli and fungi were negative.

DISCUSSION

Pathologic Process

The acute process in Varicella pneumonia occurs in the alveoli where one sees swelling, proliferation and desquamation of septal cells together with infiltration by mononuclear cells. In many areas necrosis and hemor-

rhage occur. Bacteria are rare. Type A inclusion bodies may be present. The process is diffusely distributed and may be seen throughout the lung and on the pleural surface. Many of the lesions heal by resolution. The more severely affected areas go on to fibrosis and where there is sufficient necrosis caseation takes place which may be followed by calcification. Calcification becomes visible on the radiograph after an interval of two to seven years.

Clinical Picture

Varicella pneumonia is manifest in a variety of ways. Some patients are virtually asymptomatic but others are very ill with fever, cough, dyspnea beyond expectation and even cyanosis and hemoptysis. Pulmonary symptoms appear simultaneously with the rash in some patients but in others they occur several days later. The intensity of rash is not necessarily correlated with the severity of the pneumonia. Symptoms are often more severe than might be anticipated from the patient's radiograph. Some patients die in the acute phase. The disease is particularly hazardous to pregnant women. It was formerly thought that recovery was complete but recent research suggests that permanent damage may ensue. The majority of cases have been in adults but recent publications indicate that calcification may also follow Varicella pneumonia in childhood.

Roentgenologic Features

The radiographic features of acute chickenpox pneumonia were first described by Waring and his co-authors in 1942 and have been elaborated in many excellent papers since that date. The subject was admirably covered by a publication in 1965 by Drs. Knyvet, Stringer and Abrahams.

During the acute phase one sees a diffusely distributed alveolar process in all segments of the lung with confluence in many areas. The focal quality becomes obscured by superimposition and confluence. The nodular densities resulting from the infiltrating process vary in size from 1.0 mm to 1.0 cm. Hilar adenopathy is frequently present. Fluctuation may take place with increase in findings in some areas while in others the findings decrease. The course of resolution varies. In some pa-
tients, resolution is rapid and is complete within two weeks while in others it is slow and takes several months. Still others have persistent findings even after years.

The chest may remain normal following recovery but in some patients delayed changes occur. Following an interval varying from two to seven years focal nodularity may appear and progress to calcification. In these patients one sees diffusely distributed miliary type of focal fibrosis and calcification. Usually the lesions vary from 1 to 5 mm in diameter but larger lesions have been described.

**Comment**

Varicella pneumonia should ultimately prove to be one of the more frequent causes of diffuse calcific foci in the lungs. In a recent publication Brunton and Moore surveyed 16,894 people and found 463 or 2.7 percent with a history of chickenpox in adulthood. Among these there were eight patients who had diffuse calcification presumed to have been caused by chickenpox pneumonia. This reflected an incidence of 1.7 percent who underwent subsequent calcification. Since eight occurred among approximately 16,000 people in the survey they found approximately one among each 2,000 people in their general population. The average radiologist even in areas where histoplasmosis is endemic does not see more than one patient with diffuse focal calcification per thousand chests examined. It behooves him to check each of these patients to determine whether there was a history of chickenpox in adult life and if the history is positive to rule out other causes. Recently I saw such a patient and confirmed a severe case of chickenpox pneumonia 14 years earlier. This patient had negative tuberculin tests and a negative complement fixation test for histoplasmosis.

Our patient was a heavy smoker prior to and after her Varicella pneumonia. Knyvett and his associates have raised the question of the relationship of smoking to the formation of calcification in patients who have had Varicella pneumonia. Since smoking is one of the major causes of pulmonary fibrosis it is reasonable to conjecture that the combination of the two processes would be more apt to produce calcification. The vast majority of patients described in the literature who have pulmonary calcification following chickenpox pneumonia have been smokers. It does not, however, seem necessary to have the combination.

The late effects of chickenpox pneumonia on pulmonary physiology have recently been investigated by Dahlstrom and his associates who performed pulmonary function studies on patients who had calcification following Varicella pneumonia. They found signs of increased shunt in the pulmonary circulation in many of their patients and concluded that pulmonary involvement in Varicella may be extensive enough to cause lasting though moderate impairment of respiratory function.

The differential diagnosis includes many diseases. Histoplasmosis is the most frequent cause of calcification indistinguishable from those of Varicella pneumonia. Histoplasmosis, unlike Varicella, frequently results in calcification in the hilar nodes, spleen and occasionally in the liver.

Tuberculosis was once thought to be a frequent cause of diffuse calcification in the lungs but evidence in favor of miliary calcification in the lung has become less convincing in recent years and is considered extremely rare or nonexistent by many authorities. Coal miners' pneumonoconiosis results in miliary calcifications. These are usually associated with a background which has only minimal fibrosis. Schistosomiasis can cause a similar picture but is associated with cor pulmonale and esophageal varices.

Other conditions which cause parenchymal calcification include Caplan's syndrome, scleroderma, amyloidosis, paragonimiasis, armillifer, metastatic neoplasm, pulmonary ossification, pulmonary alveolar microlithiasis and mitral stenosis.

**Acknowledgment:** I am grateful to Dr. Earl B. Wert and Dr. Thomas D. Davis for their help with the pathology in this case and to Dr. Samuel Eichold for the interesting clinical history. Mrs. Norma Breazeale is commended for her help in the preparation of this paper.

**References**


Reprint requests: Dr. Raider, 1720 Spring Hill Avenue, Mobile, Alabama 36604

**Intrathoracic Lipomas**

Richard F. Rosenberg, M.D., Berta M. Rubinstein, M.D.,** and Neil H. Messinger, M.D.†

Two cases of thoracic lipomas are reported, one subpleural and the other both intra- and extrathoracic. Some of the more important roentgen and clinical features are stressed. Lipomas should be considered in the differential diagnosis of pleural tumors.

**Intrathoracic Lipomas**

Richard F. Rosenberg, M.D., Berta M. Rubinstein, M.D.,** and Neil H. Messinger, M.D.†

**Two cases of thoracic lipomas are reported, one subpleural and the other both intra- and extrathoracic. Some of the more important roentgen and clinical features are stressed. Lipomas should be considered in the differential diagnosis of pleural tumors.**

*From the Department of Radiology, Montefiore Hospital and Medical Center, Bronx.

**Associate Professor of Radiology, Albert Einstein College of Medicine.

†Assistant Professor of Radiology, Albert Einstein College of Medicine.