heart rate or in impulse duration of the spike potentials of each of the standard limb leads. The radiologic position of the electrode indicated that it was not in close proximity to the phrenic nerve. The fact that diaphragmatic pacing was abolished by deep inspiration further suggests that it followed direct contact between the perforated pacing electrode and the diaphragm, and was not due to phrenic nerve stimulation.

The important complications of myocardial perforation are failed pacing and hemorrhage with subsequent cardiac tamponade. Although the latter is frequently mentioned in the literature as a possible complication, it rarely occurs.\(^1\)\(^-\)\(^12\) It has been recommended that a perforated electrode should be retracted and repositioned, or left in situ and a new one inserted.\(^5\) Since our patient had paced normally with myocardial penetration probably of more than one year's duration, we elected to leave the electrode where it was. Although this was a departure from conventional management, we made this decision in view of the radiologic stability of the electrode, and because of the only slight rise in the pacing threshold.

Because the electrode remained in a radiologically stable position with uninterrupted cardiac pacing for more than one year, we considered further change unlikely. The rise in pacing threshold to 2.9 volts was well within the capabilities of the power unit. Furthermore, the diaphragmatic pacing did not trouble her, and repositioning of this electrode might thus have been simply meddlesome. Indeed, one might argue that after a year a track may well have formed around the perforated electrode and re manipulation in these circumstances would be more likely to cause bleeding than to prevent it. This is supported by the view of Kramer et al\(^2\) who prefer to leave a perforated electrode in situ rather than to reposition it.

Careful close monitoring of the patient over one month, following discovery of perforation, revealed no abnormality in pacing. Although we only saw her at four to six month intervals at our clinic because she lived 1,000 miles distant, she was under the care of an internist in her home town, who was fully aware of her condition and who examined her regularly. Follow-up of this patient, once the diagnosis was known, was thus frequent, with due attention being given to all possible criteria of electrode movement and pacing failure. Any interruption in cardiac pacing would have been rapidly noted, and have been regarded as an indication to urgently change the pacing electrode.

We have thus shown with follow-up of our patient that cardiac pacing had not failed over a period of at least 19 months and that the electrode was unchanged in position during this time.

This case of uninterrupted cardiac pacing together with intermittent diaphragmatic pacing following perforation of a pacing catheter is unique in our experience.

**References**


**Bronchial Asthma with “Crossed Splitting” of the Second Heart Sound**

Toshitami Sawayama, M.D.; Hiroshi Katsuume, M.D.; Masaru Tohara, M.D.; and Shozo Nezu, M.D.

“Crossed splitting” of the second heart sound was recognized in the polygraphic tracing of a 75-year-old woman during recovery from an attack of acute bronchial asthma. This auscultatory finding, not previously reported, is discussed in relation to probable hemodynamic changes associated with bronchial asthma.

The aortic component (A2) of the second heart sound (S2) normally precedes the pulmonic component (P2). Reversal of this relationship is well recognized in the presence of left ventricular malfunction, but has not been previously described in human subjects during primary disturbances of the pulmonary circulation, although it has been described during acute cor pulmonale.
BRONCHIAL ASTHMA

**FIGURE 1.** Polygraphic tracing, consisting of simultaneous tracings, from top to bottom: finger plethysmogram, indirect carotid pulse, medium frequency phonocardiogram (50-150 CPS), electrocardiogram, and high frequency phonocardiogram (300-600 CPS). Paper speed 100 mm/sec. Both phonocardiograms recorded from the left sternal border at the third interspace. Note that the aortic component of S2 (A), occurring 10 msec before the dicrotic notch of the carotid pulse tracing, shifts its relationship to P (the pulmonary component of S2) during the respiratory cycle ("crossed splitting"). P clearly precedes A in the last two beats. insp = inspiratory phase; exp = expiratory phase.

**CASE REPORT**

A 75-year-old woman has had repeated attacks of bronchial asthma since the age of 20. In early May, 1971, she was hospitalized with symptoms and signs of severe bronchial asthma and cor pulmonale, temporarily improved by the administration of oxygen and digitalis. A right atrial gallop was heard, and S2 was single. Her asthma exacerbated in late May at which time both paradoxic splitting and fixed splitting of S2 were suggested at various times. These auscultatory findings were further investigated by polygraphic method which clearly revealed (Fig 1) that the relationship of the aortic and pulmonic components of S2 actually reversed during successive respiratory cycles so that the splitting of S2 was exaggerated during both phases of the respiratory cycle. As indicated in Figure 2, large variations of the timing of the pulmonary component were chiefly responsible for this "crossed splitting." Variations of cycle length (RR) tended to vary with and slightly precede the variations of the timing of P2. Variations in ejection time tended to follow the variations of A2 (Fig 2). Somewhat less consistent was the tendency for reciprocal relationship between ejection time (ET) and the pre-ejection period (PEP).

**FIGURE 2.** Graph of various time interval measurements for 31 consecutive cardiac cycles in our patient. Inspiratory phase labelled "in." Q-II = time interval from Q wave to S2 (A = aortic component; P = pulmonic component); RR = cycle interval; ET = ejection time; PEP = pre-ejection period. Note that P precedes A during the expiratory phase of the respiratory cycle.
These auscultatory findings were variably observed during the course of recurrent asthmatic attacks in the patient in this report. Her polygraphic tracing during clinical improvement is represented in Figure 3, which shows only slight physiologic splitting of S2. Even then, however, left ventricular systolic time intervals were abnormal: ET was 210 msec and PEP was 125 msec at a heart rate of 71/min (abnormally abbreviated, and prolonged respectively).2

The asthmatic attacks abated, allowing discharge of the patient in July of 1971, although respiratory difficulty was still encountered with slight exertion.

**DISCUSSION**

Abnormal splitting of S2 in the patient of this report was noted by simple auscultation, but required special analysis of polygraphic tracing for clear delineation. Careful observation of the relationship of each component of S2 of the phonocardiogram to some landmark such as the carotid dicrotic notch was essential in the discovery of "crossed splitting" in the present patient.

Abnormal "crossed splitting" of S2 is compared with normal physiologic splitting in Figure 4. It is evident that in each type, the interval between Q and P2 is augmented during the immediately following expiratory phase of the respiratory cycle, although the normal relationship (A2 preceding P2) obtains during inspiration. These results suggest large reductions of right ventricular stroke volume during expiration during attacks of acute bronchial asthma in the present patient. It is presumed that venous return was markedly impaired due to increased intrathoracic pressure secondary to expiratory bronchial obstruction. The presumptive changes in right heart filling might have been expected to produce even more dramatic changes in S2 during the period of maximal respiratory impairment by asthma. However, auscultatory observations were extremely difficult because of loud respiratory artifacts when asthma was maximal, so that observations of this report were made during the immediate recovery period.

As indicated above, paradoxic splitting of S2, that is, widening during expiration, was part of the "crossed splitting" phenomenon. Although the latter has not been previously described, rare cases of paradoxic splitting

**Figure 3.** Polygraph tracing recorded in the same patient when free of asthma. Key as for Figure 1. Note that A precedes P throughout the tracing, as in normal subjects.

**Figure 4.** Diagram of relationship of aortic and pulmonic components of S2 (A and P respectively) in a normal subject (panel A), and in our patient (panel B). Key as in Figure 2.
have been reported in patients with right sided heart
disease, and shortening of left ventricular systolic ejection
time. One patient reported by Gray had mitral
stenosis, while another reported by Benchimol1 had
Eisenmenger syndrome.

Following the discovery of “crossed splitting” of S2 in
this patient, further patients with bronchial asthma have
been evaluated by the polygraphic method, as well as by
precise auscultation during the early phase of recovery
from acute bronchial asthma. Crossed splitting has so far
not been found in any other patients, and must therefore
be considered quite uncommon, even though not unex-
pected in view of the hemodynamic abnormalities at-
tending acute bronchial asthma.

REFERENCES
1 Boyle J III, Little RC: Study of hemodynamic factors
which alter the sequence of the second heart sound. Am
Heart J 68:91-97, 1964
2 Sawayaama T, Ochiai M, Marumoto S, et al: Influence of
amy1 nitrite in halation on the systolic time intervals in
normal subjects and in patients with ischemic heart dis-
3 Gray IR: Paradoxical splitting of the second heart sound.
Br Heart J 18:21-28, 1956
4 Benchimol A, Dimond EG: Correlation between hemo-
dynamics and phonocardiographic findings. Am J Med
28:347-356, 1960

Scleroderma, Pleural Calcification
and Reticulum Cell Sarcoma
of the Lungs*

Roy Romey, M.D.** and Myron Moskowitz, M.D.†

A case of scleroderma with calcified pleura, an exceed-
ingly rare finding, is documented. The patient died of
acute fulminating reticulum cell sarcoma, and the radi-
ographs and clinical course mimicked an acute infectious
process.

Scleroderma, or progressive systemic sclerosis (PSS),
commonly affects the lungs. It less frequently pro-
duces pleural changes,1-2 and exceedingly rarely results
in pleural calcification. The patient we describe here is
of interest not only because of the heavily calcified
pleura, which developed under observation and without
other definite etiology, but also because of the interesting
clinical-radiographic events of his terminal illness.

CASE REPORT

A 56-year-old man had been under close medical observa-
tion since 1938, primarily because of Raynaud’s syndrome.
Ulceration of fingers and toes required lumbar sympathe-
ectomy in 1942. Scleroderma was verified by biopsy of the skin
in 1950. His skin became progressively thickened and tight.
Lower esophageal peristalsis was absent. A chest roentgeno-
gram in 1954 (Fig 1) showed pleural thickening at the right
base without concomitant parenchymal disease, and some
blunting of the left costophrenic angle as well. He was
transferred to the Daniel Drake Memorial Hospital for
chronic care in 1954 and he remained there continuously
until his death in 1970.

In 1957 the chest film again revealed the obliteration of
both costophrenic angles and now right pleural calcification
was seen for the first time. By 1960, his chest x-ray film
showed what was interpreted as chronic diffuse interstitial
disease predominantly located in both bases (Fig 2). The
pleural calcification had increased. The roentgen changes
remained stable through June of 1970.

The patient became acutely ill in late September, 1970. He
was severely weak, short of breath, and had a low grade
fever. Roentgenogram of the chest showed a new infiltrate in
the lingula (Fig 3). In view of his respiratory symptoms,
mildly elevated WBC (11,000) and yellow sputum produc-
tion, pneumonia was diagnosed. Sputum culture yielded a
moderate growth of Klebsiella and one colony of Staphy-
lococcus. He was treated with multiple antibiotics with no
apparent improvement. Chest films a few days later (Fig 4)
demonstrated rapid progression of the alveolar process and
digoxin was added to the regimen.

The white cell count rose to 23,000, dyspnea and fever

*From the Department of Radiology, University of Cincin-
nati Medical Center, Cincinnati.
**Resident in Radiology.
†Associate Professor of Radiology.
Reprint requests: Dr. Moskowitz, Department of Radiology,
Cincinnati General Hospital, Cincinnati 45229

CHEST, VOL. 64, NO. 3, SEPTEMBER, 1973

Figure 1. Bilateral blunting of the costophrenic sulci, more
on the right than left.

Figure 2. Bibasilar interstitial disease is shown, as well as the
pleural calcium.