Acquired Continuous Murmur
Associated with Acute Pulmonary Thromboembolism*

Richard L. ZuWallack, M.D.;** Joseph P. Liss, M.D.;† and Bimalin Lahiri, M.D., F.C.C.P.;‡

Two cases of a continuous murmur following an acute pulmonary embolic episode are presented, and eight previously reported cases with an acquired postembolic continuous murmur (found in a review of the literature) are discussed. This finding is present in both chronic and acute pulmonary embolism and is suggestive of significant embolic obstruction. Although the continuous murmur is an unusual sign in patients with pulmonary embolism, its auscultation is often quite distinctive, and its appearance may lead to more definitive diagnostic studies when the presentation or associated clinical findings are nonspecific.

The physical findings in patients with pulmonary thromboembolism without infarction are usually nonspecific in nature and are often not very helpful in establishing the diagnosis. A relatively unusual finding, an acquired continuous murmur, has received some attention in the American literature since 1959.1-3 Interestingly, the cases reported up to this time show the continuous murmur associated with chronic symptoms and often with histories suggestive of recurrent pulmonary emboli. We report two cases of acute pulmonary thromboembolic disease with both systolic and early diastolic murmurs.

Case 1

A 20-year-old white woman was admitted to St. Francis Hospital on July 9, 1974 with a two-day history of pain in the left side of the chest and cough. Other than a three-month history of using an oral contraceptive, the remainder of her history was noncontributory. On examination, her respiratory rate was 16/min, her pulse was 76 beats per minute, and her temperature was 38.2°C (100.8°F). There was evidence of a pleural effusion on the left. A grade 2-3/6 midsystolic

*From the Cardiopulmonary Section, Department of Medicine, St. Francis Hospital, Hartford, Conn.
**Senior Fellow, Section of Pulmonary Diseases.
†Assistant Professor of Medicine, University of Connecticut Health Center, and Chief, Exercise Laboratory.
‡Assistant Professor of Medicine, University of Connecticut Health Center, and Associate Director, Section of Pulmonary Diseases.
Reprint requests: Dr. ZuWallack, Section of Pulmonary Diseases, St. Francis Hospital, Hartford 06105
murmur was noted in the back between the scapulae and over the entire anterior chest, and a soft early diastolic murmur was present anteriorly. A pulmonic-ejection click was evident, and the second pulmonic sound was accentuated. Thrombophlebitis was not clinically present.

A chest x-ray film confirmed a pleural effusion on the left. An electrocardiogram was normal. Arterial blood gas analysis with the patient breathing ambient air revealed a pH of 7.435, an arterial oxygen pressure (PaO₂) of 80 mm Hg, and an arterial carbon dioxide tension (PaCO₂) of 32.7 mm Hg. A phonocardiogram (Fig 1) done in the third left interspace showed a mid and late systolic and an early diastolic murmur.

Table 1—Reported Cases of Continuous Murmurs Associated with Pulmonary Embolism

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age (yr), Sex</th>
<th>Symptoms and Findings*</th>
<th>Murmurs</th>
<th>Murmur Location</th>
<th>Pressures (mm Hg)</th>
<th>Pulmonary Angiograms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levine and Harvey (1963)</td>
<td>25, M</td>
<td>Chronic phlebitis; recurrent pulmonary emboli; oor pulmonic</td>
<td>Continuous</td>
<td>Right lung base</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Levine and Harvey (1969)</td>
<td>50, M</td>
<td>Thrombophlebitis; recurrent pulmonary emboli</td>
<td>Grade-3 continuous (late systolic accentuation)</td>
<td>Pulmonic area</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Goodwin et al (1963)</td>
<td>29, M</td>
<td>Dyspnea (6 mo); phlebitis; hemoptysis; pleural pain</td>
<td>Faint continuous</td>
<td>Right upper chest and right axilla</td>
<td>PA, 114/51 (mean, 69)</td>
<td>No filling, left lower zone; sparse filling, left upper zone; right lung vessels, tapered peripherally with little filling at apex</td>
</tr>
<tr>
<td>Moer et al (1965)</td>
<td>41, M</td>
<td>DOE (7 mo); phlebitis, 2-3 mo earlier</td>
<td>Holoystolic, extending through 2nd sound; increased intensity with inspiration</td>
<td>Third left intercostal space and back</td>
<td>Mean PA, 35</td>
<td>Filling defect, distal RPA; no filling of vessels of RML, RLL, and LUL</td>
</tr>
<tr>
<td>Moer et al (1965)</td>
<td>42, M</td>
<td>DOE (about 1 yr); history of RLL pulmonary infarction</td>
<td>Grade-3 systolic extending through 2nd sound, increased intensity with inspiration</td>
<td>Below left scapula</td>
<td>RV, 35-40/3-5</td>
<td>No filling, RPA</td>
</tr>
<tr>
<td>Perloff (1967)</td>
<td>38, F</td>
<td>&quot;Embolic pulmonary hypertension&quot;</td>
<td>Continuous</td>
<td>Left axilla</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Claudio et al (1970)</td>
<td>48, M</td>
<td>DOE (about 10 mo); PND; orthopnea</td>
<td>Continuous</td>
<td>Right midsternal border</td>
<td>RV, 79/-1-6; RPA, 84/25; PRLLPA,84/25; DRLLPA,54/23</td>
<td>Complete occlusion of branch to LLL; right lung, normal</td>
</tr>
<tr>
<td>Fraser and Lynne-Davies (1974)</td>
<td>38, F</td>
<td>Exhaustion; DOE (3 yr); episodic chest pain</td>
<td>Grade 2-3/6 systolic, followed by 2/6 diastolic</td>
<td>Maximum 2nd right intercostal space, radiating to right side of back</td>
<td>PA, 69/23 (mean, 37)</td>
<td>Partial or complete occlusion of many segmental vessels</td>
</tr>
</tbody>
</table>

*DOE, Dyspnea on exertion; RLL, right lower lobe; and PND, paroxysmal nocturnal dyspnea.

**PA, Pulmonary artery; RPA, right pulmonary artery; RML, right middle lobe; RLL, right lower lobe; LUL, left upper lobe; RV, right ventricle; PRLLPA, proximal right lower lobar pulmonary artery; DRLLPA, distal (to dot) right lower lobar pulmonary artery; and LLL, left lower lobe.
A saddle embolus in the right main pulmonary artery and a questionable filling defect in a branch to the left lower lobe were found on a pulmonary angiogram obtained on July 10, 1974 (Fig 2). Pressures obtained were: right ventricle, 35/5 mm Hg; proximal right pulmonary artery, 36/10 mm Hg; and distal right pulmonary artery, 20/8 mm Hg.

The patient was treated for 14 days with heparin and was discharged on oral therapy with warfarin. At a follow-up visit on Aug 7, 1974, the interscapular bruit was gone, and only a soft grade 1/6 midsystolic murmur in the pulmonic area was recorded. When the patient was seen again on Aug 13, 1974 all murmurs were absent.

CASE 2

A 20-year-old white woman was admitted to St. Francis Hospital on April 23, 1975 with a history of exertional dyspnea for approximately 1½ months and recent nonpleuritic pain in the right side of her chest. The remainder of her history was noncontributory. On admission, her respiratory rate was 18/min, her pulse was 92 beats per minute, and her temperature was 38.3°C (101.1°F). The second sound was palpable in the pulmonic area, and the pulmonic component was accentuated. There was a grade 2/6 systolic murmur and a very early short diastolic murmur heard over both pulmonary fields posteriorly, and anteriorly below the clavicles. Thrombophlebitis was not clinically evident. A chest x-ray film showed a small pleural effusion on the right side. An ECG had inverted T waves in leads 3, aVF, and V1 to V3. Arterial blood gas analysis with the patient breathing ambient air showed a pH of 7.42, a PaO2 of 77 mm Hg, and a PaCO2 of 35 mm Hg. A pulmonary angiogram was performed on April 24, 1975 (Fig 3) and showed filling defects in the main pulmonary artery, a large branch to the left lower lobe, and to a small branch to the left upper lobe. Pulmonary arterial pressure was 42/17 mm Hg (mean, 25 mm Hg).

The patient was treated with intravenous administration of heparin for 14 days, followed by oral therapy with warfarin. The murmurs decreased in intensity in the hospital and were absent during a follow-up visit two weeks later.

DISCUSSION

The cardiac auscultatory findings in patients with pulmonary thromboembolism can be divided into two categories: (1) those secondary to the associated pulmonary hypertension and cor pulmonale, and (2) those secondary to the effects of the embolism itself. The findings of an accentuated second pulmonic sound, pulmonic ejection murmur, pulmonary ejection click, diastolic decrescendo murmur of pulmonary regurgitation, murmur of tricuspid insufficiency, and right ventricular S4 and S5 gallop rhythms are well-known and useful signs in helping to establish the diagnosis; however, they all are nonspecific indices of the pulmonary hypertension and right ventricular failure produced by the embolism and can be seen in a wide variety of other conditions that cause similar hemodynamic changes in the pulmonary circulation.

Both systolic and continuous murmurs have been described in patients with pulmonary embolism. Their postulated mechanism is either from turbulent flow around a partially obstructing clot or by increased bronchial arterial collateral blood flow in the area of the embolism. The acquired systolic murmur, distinct from the rather common and nonspecific flow murmur in the pulmonic area, was first reported in the American literature in 1961. This murmur has been described over the anterior chest but characteristically is loudest in the back between the scapulae. After the patient recovers, the murmur may disappear or persist.

The two cases presented had an acquired continuous murmur from pulmonary embolism, and they raise to ten the number of cases reported with this particular finding. The eight cases previously reported are summarized in Table 1. Of note, the findings on catheterization or angiographic studies, when done, showed severe pulmonary hypertension or significant obstruction, testifying to the severity of the obstruction when this sign is present. Our two cases were both previously healthy patients with acute thromboembolic disease with no historic or clinical evidence for chronic recurrent pulmonary embolization or chronic cor pulmonale and, thus, differ from the previous cases cited in the literature.

Despite recent reviews on pulmonary embolism, the murmurs caused by peripheral pulmonary arterial stenosis from the clot have received scant recognition. This finding is undoubtedly unusual but, when present, is often quite distinctive on auscultation and may provide the needed impetus for further diagnostic studies when the symptoms or other physical findings are nebulous.

REFERENCES