Pulmonic Regurgitation Following Staphylococcal Endocarditis

An Intracardiac Phonocardiographic Study

By Hugh S. Levin, M.D., Vincent Runco, M.D., Charles F. Wooley, M.D., and Joseph M. Ryan, M.D.

That congenital or acquired disease may directly affect the pulmonic valve causing clinically recognizable regurgitation has been known for some time. \(^1\)\(^{-3}\) Until recently, however, the diagnosis in the vast majority of cases was made only at autopsy. \(^3\) \(^{4}\) In recent years, greater awareness of this uncommon cardiac affection as well as refined diagnostic technics have made possible the antemortem recognition of a steadily increasing number of cases. In some, pulmonic insufficiency was associated with other cardiac lesions, \(^5\) \(^6\) while in others it was found to be the sole abnormality. \(^7\) All but one \(^8\) of the reported cases in the latter category were thought to have a congenital origin. In addition, a significant number of patients have been studied in whom regurgitation appeared after pulmonary valvulotomy for congenital stenosis of the valve. \(^9\) \(^{-11}\) It has become increasingly apparent that this uncommon hemodynamic derangement presents rather characteristic physical findings as well as typical pressure phenomena at the time of right heart catheterization whether deformity of the pulmonic valve is congenital or acquired.

Recently we have had the opportunity to study a patient who had the typical clinical and catheterization findings of isolated pulmonic incompetence following an episode of bacteriologically proved and successfully treated staphylococcal endocarditis. The technic of intracardiac phonocardiography was extremely valuable in confirming the diagnosis and in shedding light on the genesis of the unusual murmur that is characteristic of this malformation.

Case Report

A 30-year-old Negro man had no history of rheumatic fever or congenital heart disease and no knowledge of a cardiac murmur in spite of several physical examinations. He was in excellent health until September 1956, when he developed intermittent episodes of chills, fever, diaphoresis, nausea, vomiting, and pleuritic-like chest pain. The persistence of these symptoms for 2 weeks in addition to a 10-pound weight loss prompted admission to University Hospital.

His blood pressure was 120/70 mm. Hg, and his temperature was 101°F. There was no detectable cardiomegaly. Both systolic and diastolic murmurs were heard along the left sternal border, and the rhythm was regular. No pulmonary abnormalities were detected. The abdomen was soft and nontender; neither the liver nor spleen was palpable. Several splinter hemorrhages were seen in the fingernail beds. There was no edema.

The white-cell count was 18,000/mm. \(^3\) with 80 per cent neutrophils. The hemoglobin was 12.2 Gm./100 ml. A urine specimen contained 40 mg. per cent of protein as well as 5 to 10 white cells and 2 to 5 red cells per high-power field; urinalysis several days later was normal. *Non-hemolytic staphylococcus aureus* was isolated from the first two of four blood specimens drawn following admission and from the oropharynx. The chest x-ray and electrocardiogram were normal.

With a working diagnosis of staphylococcal endocarditis involving the aortic valve, the patient was treated intensively for 6 weeks with penicillin and streptomycin. After 1 week of therapy, he became afebrile and asymptomatic. Subsequent blood cultures were negative.

Although the patient was completely free from symptoms during the next 5 years, periodic examination in the outpatient department continued to show the murmurs detected originally during the acute illness. It became increasingly apparent that the diastolic murmur was distinctly unusual and not characteristic of aortic regurgitation. In

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In order to establish an accurate diagnosis, the patient was readmitted to the hospital for diagnostic studies in May 1961.

Physical examination was entirely normal except for the heart. The blood pressure was 130/80. There was no evidence of cardiomegaly, and the rhythm was regular. A grade-II (grade-VI scale) ejection-type systolic murmur was heard best in the second left intercostal space. The pulmonic component of the second sound was not identified but the aortic component was of normal intensity and was followed by a grade-IV harsh, low-pitched, crescendo-descrescendo murmur that occupied the first half of diastole and was loudest in the third and fourth intercostal spaces, immediately to the left of the sternum. The intensity of the murmur increased during inspiration and decreased during expiration (case 7, ref. 12).

The electrocardiogram was normal. Although no abnormalities were found on the chest x-ray, fluoroscopy showed a vigorously pulsating main pulmonary artery. Phonocardiogram confirmed the auscultatory findings (fig. 1).

Right heart catheterization was performed with an Allard-Laurens variable inductance micromanometer mounted in a no. 8F cardiac catheter. Intracardiac pressure and sound as well as an external phonocardiogram were recorded simultaneously (table 1). Normal oxygen saturations and indicator-dilution studies gave no evidence of intracardiac shunt and there was no indication of pulmonary hypertension or pulmonic stenosis. The end-diastolic pressures in the pulmonary artery and right ventricle were essentially equal and the diastolic limb of the pulmonary artery pressure descended steeply to the diastolic level (fig. 2). A systolic ejection-type murmur was recorded from the pulmonary artery (fig. 2A) and a diastolic murmur similar in timing and configuration to that heard externally was most intense in the right ventricular outflow tract and in the main pulmonary artery immediately distal to the pulmonic valve (fig. 2A and B). It was also present, but less intense, in the distal portion of the main pulmonary artery and in the right and left pulmonary arteries, and it was barely discernible in the right ventricle near the tricuspid valve and at the apex.

The patient was discharged with the recommendation that he receive appropriate antimicrobial prophylaxis at the time of dental or surgical procedures and that he seek medical advice promptly at the first signs of respiratory infection. The final diagnosis was organic pulmonic regurgitation secondary to staphylococcal endocarditis involving the pulmonic valve.

**Discussion**

Isolated pulmonic regurgitation occurring in the absence of other cardiovascular abnormalities or pulmonary hypertension may be due to congenital or acquired lesions. Malformation or absence of the valve cusps, supernumerary cusps, and idiopathic dilatation of the pulmonary artery are examples of the congenital variety. Acquired diseases that may affect the pulmonic valve include carcinoid, bacterial endocarditis (especially gonococcal endocarditis), syphilis, and rheumatic fever, although this last disease almost never attacks the pulmonic valve alone. An increasingly common cause of organic pulmonic insufficiency at the present time is prosthetic valve endocarditis.

### Table 1

<table>
<thead>
<tr>
<th>Location</th>
<th>Oxygen saturation (per cent)</th>
<th>Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>78</td>
<td></td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td>Right atrium</td>
<td>78-81</td>
<td>5 (mean)</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
<td>20/4</td>
</tr>
<tr>
<td>Main pulmonary artery</td>
<td>78</td>
<td>27/6</td>
</tr>
<tr>
<td>Right pulmonary artery</td>
<td>78</td>
<td>21/5</td>
</tr>
<tr>
<td>Left brachial artery</td>
<td>97</td>
<td>116/66</td>
</tr>
</tbody>
</table>

*Figure 1*

External phonocardiogram recorded from the pulmonic region. Paper speed is 75 mm./sec.
time is pulmonic valvulotomy for congenital stenosis of the valve.9–11

Heretofore, the antemortem diagnosis of organic pulmonic insufficiency has been largely based on finding at cardiac catheterization a pulmonary artery pressure curve characterized by a dicrotic limb steeply descending to the diastolic level, which in end-diastole is equal to right ventricular pressure.5–7 Some authors have placed reliance on indicator-dilution technics9,11 and on angiography.20,21 Since regurgitation of radiopaque material can be artificially caused by deformity of the pulmonic valve by the catheter,22 we sought other methods for confirming the existence of pulmonic insufficiency. The use of the phonocatheter proved to be a valuable technic for localizing the source of the unusual diastolic murmur in the case reported herein and in two others that we have had the opportunity to study.23,24 Other authors13,25 have stated that the murmur of relative pulmonic insufficiency is readily detected by the intracardiac microphone posi-
tioned in the right ventricular outflow tract, whereas the murmur of aortic insufficiency is not. Our experience has supported this view.

We have been unable to detect aortic diastolic murmurs of grade-V intensity in any of the vessels or cardiac chambers approachable during right heart catheterization. Recently, other reports16,26 have confirmed the usefulness of the phonocatheter in the diagnosis of organic pulmonic regurgitation.

The simultaneous recording of a conventional external phonocardiogram and of intracardiac pressure and sound with the catheter tip micromanometer has helped to explain the unusual nature of the murmur frequently encountered in organic pulmonic insufficiency. McKusick17 and other authors7,10,11,16,21,27 describe a low-pitched, rough, short, crescendo-decrescendo, early diastolic murmur sometimes separated from the second sound by a short but definite silent interval. Phonocardiograms published in case reports of organic pulmonic regurgitation not uncommonly show murmurs of this nature.

**Figure 2**

Simultaneous recording (from above downward) of external phonocardiogram, intracardiac pressure, intracardiac sound, and electrocardiogram with the micromanometer in the main pulmonary artery (A) and right ventricular outflow tract (B). Panel C shows superimposition of the pulmonary artery and right ventricular pressure tracings (obtained by redrawing the curve from A over that in B) with simultaneous representation of the external and intracardiac phonocardiograms. Paper speed is 50 mm/sec.

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even though they are not so described in the text of the article. In our case, the pulmonary artery and right ventricular pressure curves are strikingly similar except that the latter dips significantly below the former for a brief period in early diastole (fig. 2). This same phenomenon can be observed in many of the tracings published in the literature. Superposition of the tracings (fig. 2C) shows that the onset of the murmur occurs precisely at the moment when the pressure curves diverge. The murmur reaches its peak at the time when the pressure gradient is the greatest and ends in mid-diastole with the disappearance of the gradient. The low-frequency composition of the murmur can readily be ascribed to low-velocity regurgitation of blood from the pulmonary artery into the right ventricle at a time when the pressure in both chambers is extremely low and the gradient between them relatively small.

Since the right ventricle must eject with each systole blood regurgitated through an incompetent pulmonic valve as well as that normally entering it from the right atrium, prolongation of right ventricular ejection time may occur, causing, in turn, an exaggeration of the physiologically asynchronous onset of left and right ventricular diastole. This resultant retardation of right ventricular relaxation coupled with the absence of a discernible pulmonary closing sound in some cases probably accounts for the silent interval between the well-heard aortic component of the second sound and the onset of the murmur. In patients whose regurgitant flow is small, this delay may be absent, so that the murmur begins immediately after the second sound. Our patient appears to fit this latter category.

Because of the configuration, brevity, timing, and pitch of the murmur, Price suggested that it represents a ventricular filling murmur as did Segel et al. who considered its mechanisms similar to that of the Austin-Flint murmur. Some support for this theory was put forth by Sloman and Wee, who were able to record with a phonocatheter a mid-diastolic murmur from the inflow tract of the right ventricle. As presented in their illustrations, however, the timing and configuration of the intracardiac murmur did not correspond to that of the murmur recorded from the chest; it may represent a separate filling murmur of such low intensity as to be imperceptible externally. The demonstration by intracardiac phonocardiography that the murmur in the three cases we have studied was most intense in the right ventricular outflow tract and main pulmonary artery and only weakly audible at the apex and immediately distal to the tricuspid valve incriminates the insufficient pulmonic valve and not right ventricular inflow as the source of the murmur.

Boussvacos and Deuchar also attributed the unique characteristics of the murmur of organic pulmonic valvular insufficiency to the early diastolic gradient between the pulmonary artery and the right ventricle peculiar to this defect. They contrasted it to the high-pitched, blowing, decrescendo-pandistolic murmur heard in aortic insufficiency or pulmonic insufficiency associated with pulmonary hypertension, where the gradient is large and extends throughout diastole. However, they pictured a slowly dropping pulmonary artery diastolic pressure descending to meet the slight elevation of the right ventricular end-diastolic pressure incident to atrial contraction. Figure 2 shows that in our case the pulmonary artery pressure dropped rapidly in diastole but a more precipitous drop in the right ventricular pressure in the form of an early diastolic "dip" led to the pressure gradient, which was obliterated well before atrial contraction.

There can be little doubt about the diagnosis of isolated organic pulmonic insufficiency in the case herein presented. Likewise, the diagnosis of bacterial endocarditis 5 years previously seems unquestionable. The possibility exists that organic pulmonic insufficiency was present from birth or that endocarditis localized to a congenitally stenotic pulmonic valve led to the hemodynamic situation which we have described. It seems unlikely, however, that the usually loud murmur
of pulmonic stenosis or the presently audible grade-IV diastolic murmur could have remained undetected through numerous army and insurance physical examinations. It is, therefore, reasonable to assume that staphylococcal endocarditis attacked a normal pulmonic valve or one with a congenital deformity that was clinically inapparent and hemodynamically insignificant (e.g., a bicuspid or quadricuspid pulmonic valve). Any of these theories of pathogenesis is, of course, speculative without pathologic confirmation.

Summary

Isolated pulmonic regurgitation was found in a 30-year-old man 5 years after an episode of bacteriologically proved and successfully treated staphylococcal endocarditis. Intracardiac phonocardiography was useful in confirming the diagnosis, and simultaneous intracardiac pressure and sound recording afforded a plausible explanation for the unusual murmur of organic pulmonic insufficiency.

References

Science, A Learning Profession

Science, the fourth learned profession, is by its very nature a learning profession, and scientists are committed to factual and theoretical inquiry concerning the nature of the world. The so-called "Laws" of science (Newton's and Kepler's are familiar examples) are constantly subjected to study, reformulation, and specification as they are applied under new conditions. A scientist becomes celebrated when he discovers a new and revolutionary fact or law, the acceptance of which transforms the activities of the scientific community like an accepted judicial decision transforms the political realm. Between such revolutionary discoveries scientists carry on their practice of science within the conceptual framework of past achievements, not unlike practitioners of medicine, or lawyers practicing "lawyer's law."—Introduction, Edward D. Churchill, M.D. Listen to Leaders in Medicine. Edited by Albert Love and James Saxon Childers. Atlanta, Tupper and Love, Inc., 1963, p. 7.
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