

Gallavardin Phenomenon in Aortic Stenosis

A Possible Mechanism

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In 1925, Gallavardin reported that the harsh murmur of aortic valvular stenosis could change in quality and become musical at the apex.¹ He postulated that the high-pitched musical components of the murmur of aortic stenosis were preferentially transmitted to the apex through solid tissues, whereas the lower-pitched components were transmitted to the neck vessels via the flow of blood. Subsequently, Gallavardin's findings have been noted by others.²⁻⁵ Thus, the Gallavardin phenomenon and its proposed mechanism have become well accepted.

While recently studying a patient with aortic stenosis and the Gallavardin phenomenon, it occurred to us that the high-pitched musical murmur might be secondary to the development of papillary muscle dysfunction. The following evidence is presented to support this hypothesis.

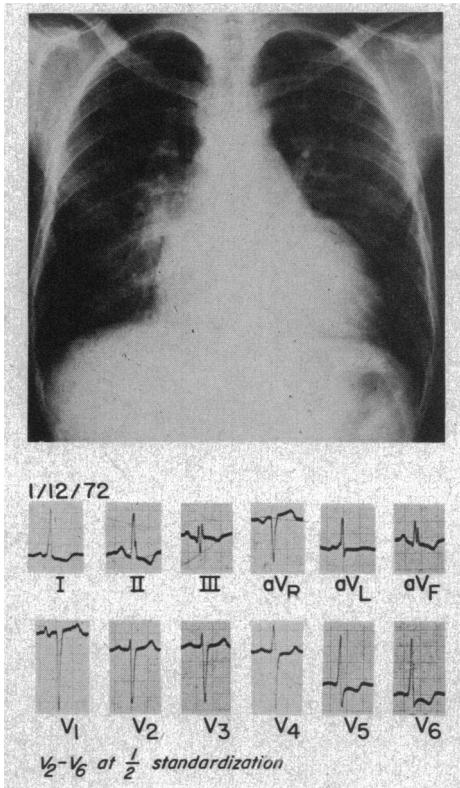
Patient Summary

A 30-year-old man was admitted to the Tulane Cardiology Service at the Charity Hospital of Louisiana at New Orleans for evaluation and treatment of severe aortic stenosis that was first diagnosed when the patient was 19 years of age. On admission, he complained of progressive dyspnea on exertion, orthopnea, ankle edema, and productive cough followed by hemoptysis.

Physical examination disclosed the following values: blood pressure, 92/70 mm Hg; pulse rate, 108 beats per minute; respirations, 16 per minute; and body tempera-

ture, 36.8°C (98.4°F). The carotid pulse was of low amplitude and had a delayed upstroke. A sustained apical thrust was felt in the sixth intercostal space at the anterior axillary line. A rough, harsh, grade 4 ejection-type systolic murmur was heard over the second intercostal space at the right sternal border, over the carotid arteries, and at the left sternal border. This harsh murmur could be faintly heard at the cardiac apex. A musical, well-localized grade 2/4 "diamond-shaped" systolic murmur was heard predominantly at the cardiac apex. The second heart sound was narrowly split. A faint decrescendo early

Fig 1.—Teleoroentgenogram of chest and ECG of patient with aortic stenosis and Gallavardin phenomenon, showing left ventricular hypertrophy and anterolateral papillary muscle disease.



blowing diastolic murmur was heard at the left parasternal area and cardiac apex.

An electrocardiogram (ECG) showed a sinus tachycardia, left atrial and left ventricular hypertrophy, and ST-segment and T-wave changes typical of anterolateral papillary muscle disease (Fig 1). Teleoroentgenograms of the chest showed left ventricular and left atrial enlargement with venous congestion and Kerley B lines (Fig 1). Cardiac fluoroscopy and tomograms of the aortic valve showed calcification of the aortic valve cusps.

Phonocardiographic Studies

Phonocardiograms were recorded with the patient in the recumbent position. Simultaneous tracings were recorded from the aortic and apical areas. The pass bands for recording the murmurs were 50 to 105 hertz for the aortic area and 250 to 1,250 Hz for the apex. With these pass bands, seemingly identical murmurs were recorded from both areas (Fig 2, left).³

After control tracings were recorded, the patient inhaled amyl nitrite for approximately 20 seconds. Continuous strip recordings were made for one to two minutes. Approximately 20 seconds following inhalation of amyl nitrite, the systolic murmur at the apex gradually decreased in intensity until it disappeared at the peak of the sinus tachycardia (Fig 2, center). During this period, the systolic murmur recorded over the aortic area decreased relatively little, the decrease probably being a result of the reduced vibrations recorded from the mitral valve murmur. At approximately 60 seconds following inhalation of the amyl nitrite, an increase in intensity of the murmur over the aortic and apical areas was recorded (Fig 2, right). These findings at the base are important in that they are not characteristic of aortic valve stenosis.

When the pass band for recording the aortic murmur was changed to 250-1,250 Hz, similar diminution in the intensity of the murmur was also observed following inhalation of amyl nitrite (Fig 3, left and center). A late increase in the intensity of the murmur was shown again (Fig 3, right).

Postmortem Findings

The patient died during an attempt

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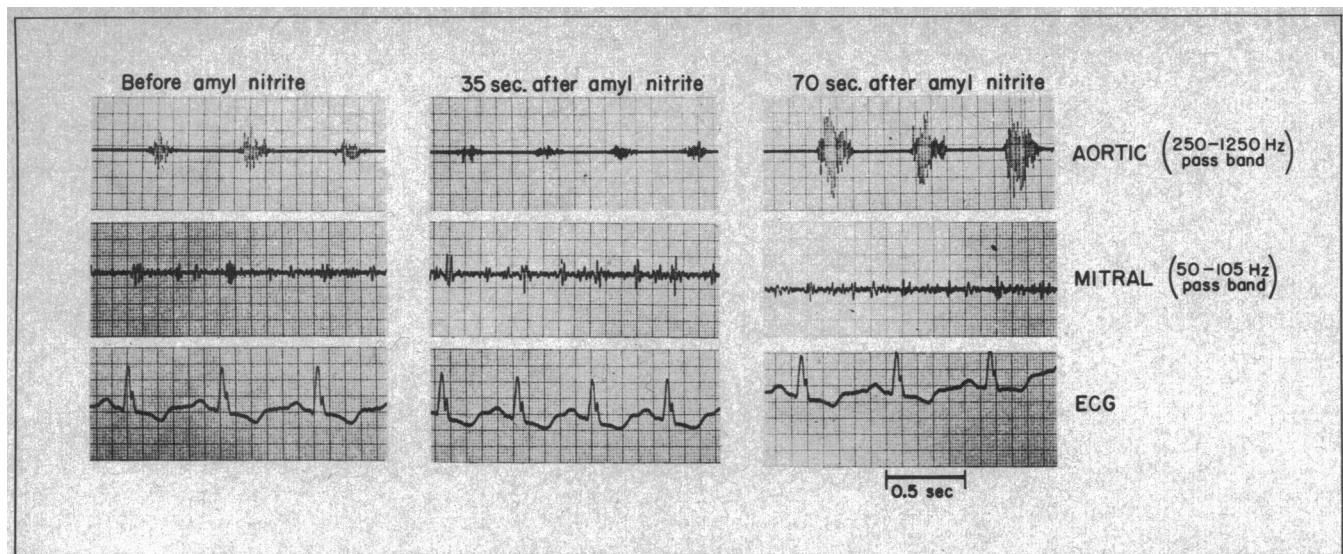


Fig 2.—Phonocardiograms of aortic and mitral valve sounds and ECG recorded simultaneously from patient with aortic stenosis and Gallavardin phenomenon. High-pitched murmur disappeared at mitral area 30 seconds after inhalation of amyl nitrite and after sinus

tachycardia developed, but changed little at the base (center). Sixty seconds following inhalation of amyl nitrite, high-pitched mitral murmur returned in greater intensity (right) than control (left). Pass bands for recording murmurs are shown.

at aortic valve replacement. Postmortem examination disclosed a severely calcified aortic valve. There was considerable left ventricular hypertrophy, and scarring was evident throughout the left ventricle including the left ventricular papillary muscles. The mitral valve leaflets were normal and the left atrium showed minimal dilatation. Microscopical examination demonstrated hypertrophy and fatty infiltration of myocardial fibers and diffuse fibrosis.

Comment

When the Gallavardin phenomenon was first described, the concept of papillary muscle dysfunction did not exist. When Gallavardin examined three patients who had classic murmurs of aortic valvular stenosis at the base and a high-pitched musical murmur at the apex, he postulated that the patients had either simple aortic lesion or organic mitral regurgitation associated with aortic stenosis. Whether or not the papillary muscles were examined is not known. Autopsy findings in two of Gallavardin's reported patients demonstrated only disease of the aortic valves. Therefore, he concluded that the murmur was of aortic origin and that it changed in quality (timbre) and became musical in apical propagation.

Gallavardin proposed that the separate pathways of sound transmission utilized by two different groups of vi-

brations originating at the aortic valve were responsible for the apparent presence of two distinct murmurs. He proposed that the rough noise produced at the valvular orifice was transmitted to the base of the heart by the flow of blood, while the musical sound composed of different harmonics was transmitted primarily by solid tissues to the cardiac apex. Thus, the two murmurs were considered to be different vibrations emanating from the aortic valve that were separated by preferential transmission rather than one murmur that was modified in quality as it was being transmitted to the apex.

Although this interpretation has been generally accepted,²⁻⁵ it is possible that midsystolic murmurs of such musical quality and shape could be due to mitral regurgitation secondary to papillary muscle dysfunction. The clinical manifestations of papillary muscle dysfunction have been described.⁶ The murmur may frequently be diamond-shaped, high-pitched, and well transmitted to the base, and may be caused by ventricular hypertrophy and dilatation and scarring of the papillary muscles. Thus, papillary muscle dysfunction is found in patients with aortic stenosis. Also, papillary muscle dysfunction is often associated with characteristic electrocardiographic changes.^{7,8} Therefore, the coexistence of papillary muscle dysfunction in our pa-

tient was supported by the clinical data, the murmur, the ECG, and the pathologic changes noted in the papillary muscles at necropsy.

Finally, the change in the musical murmur following inhalation of amyl nitrite suggests that papillary muscle dysfunction was present. The value of amyl nitrite in differentiating murmurs of aortic stenosis from those of mitral regurgitation has been reported.⁹ Furthermore, the value of amyl nitrite in distinguishing murmurs of mitral regurgitation secondary to papillary muscle dysfunction from the murmur secondary to aortic stenosis has been emphasized.⁷ Generally, amyl nitrite diminishes the intensity of the murmurs of mitral regurgitation early, whereas it increases the intensity of the murmur of aortic stenosis later.

The effect of amyl nitrite on the murmur of mitral regurgitation secondary to papillary muscle dysfunction is variable.⁵ However, such murmurs usually decrease in intensity early after inhalation of amyl nitrite, as was observed in our patient. Thus, the noticeable diminution in intensity of high-pitched murmurs recorded at the cardiac apex and base in our patient suggests that the murmur was produced by mitral regurgitation. Mitral regurgitation in our patient would almost certainly be secondary to papillary muscle dysfunction. It is difficult to understand how the physi-

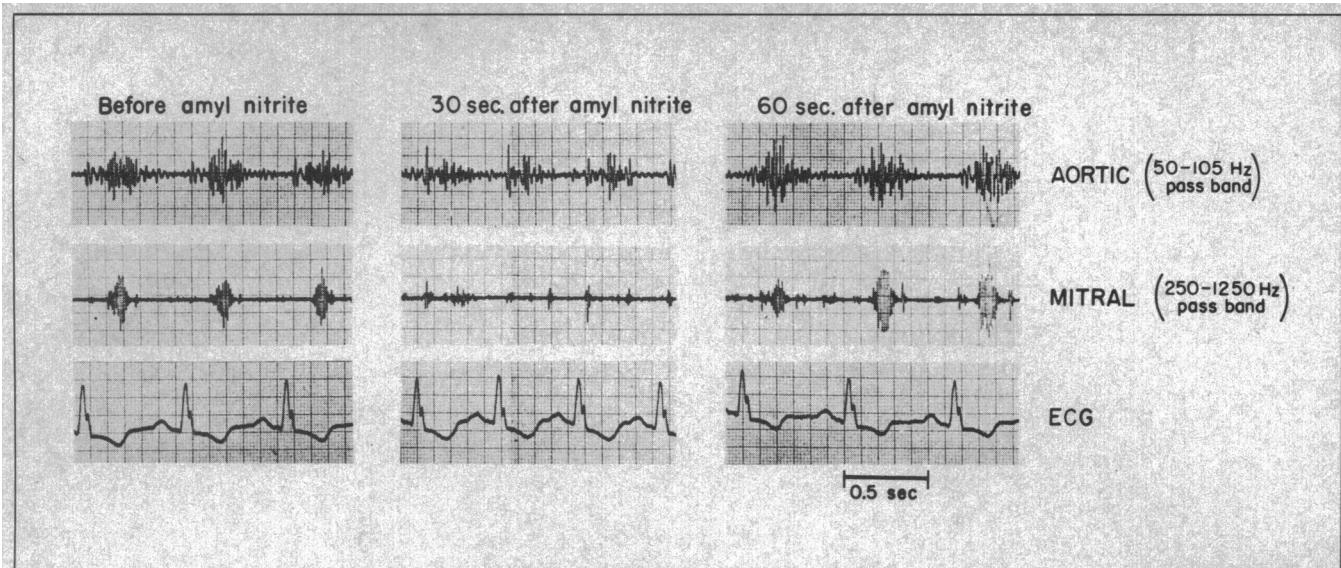


Fig 3.—Phonocardiograms of patient with aortic stenosis and Gallavardin phenomenon in which pass bands for recording murmurs

from aortic and mitral valve areas are reversed with respect to those in Fig 2 (see text for details).

ologic effects of amyl nitrite could diminish a murmur originating at the aortic valve. It is possible, of course, that the increased flow across the stenotic orifice could change the quality of the murmur.

It is possible that the patients included in Gallavardin's original report had papillary muscle dysfunction. Although not yet described at that time, the entity certainly existed. Such a mechanism should be considered, particularly for the apical murmur heard in the second of Gallavardin's three patients. That particular patient was a 60-year-old woman with syphilitic heart disease, aortic regurgitation, and chronic congestive heart failure. At autopsy, aortic lesions histologically characteristic of syphilitic aortitis were found. Aortic stenosis was considered to be produced by the calcareous adhesions of one of the commissures that fixed it against the aortic wall.

Thus, we consider that the "Gallavardin phenomenon" may be due to the association of aortic stenosis with mitral regurgitation secondary to papillary muscle dysfunction. We are not proposing that papillary muscle dysfunction explains all occurrences of the phenomenon, but rather that it may be one mechanism for the production of this interesting clinical finding. It is difficult for us to accept the explanation of Gallavardin based on the differences in blood and solid tissue transmission. Gallavardin's explanation needs study to learn whether or not it is plausible.

Summary

A 32-year-old man had classic, severe, calcific, aortic valvular stenosis, with a rough systolic murmur at the base and a high-pitched, midsystolic, "diamond-shaped" musical murmur at the apex—findings that meet the criteria for the Gallavardin phenom-

enon. However, concomitant mitral regurgitation secondary to papillary muscle dysfunction was considered in this patient. Clinical data supported this diagnosis. When the patient inhaled amyl nitrite, simultaneous phonocardiographic recordings from the aortic and mitral areas showed evidence of concomitant aortic valvular stenosis and mitral regurgitation. Necropsy findings supported the diagnosis of papillary muscle dysfunction as the cause of the mitral regurgitation. Therefore, the Gallavardin phenomenon in our patient was apparently due to severe aortic stenosis associated with mitral regurgitation secondary to papillary muscle dysfunction.

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