Unusual left ventricular wall motion and a loud added sound during the isovolumic relaxation period in a patient with hypertensive heart disease

Yuichiro Mishiro, Takashi Oki, Nobuo Fukuda

Abstract
A 69 year old woman with hypertensive heart disease had a loud added sound which coincided with a sudden interruption of the early diastolic motion of the left ventricular posterior wall, as visualised by M mode echocardiography, and came just before early diastolic transmirtal flow, as measured by a pulsed Doppler echocardiogram. Early diastolic motion velocity from the base to the middle of the posterior wall, assessed by pulsed Doppler tissue imaging, was markedly high and sharp, and its peak coincided with the sound. A notch, similar to that in the posterior wall motion, occurred in the left ventricular pressure curve during early diastole. No intraventricular flow signal was detected during the isovolumic relaxation period, as measured by pulsed and colour Doppler imaging. The added sound was probably produced by impact between the dilated heart, with a relaxation abnormality, and the extracardiac structures during the isovolumic relaxation period.

Keywords: added sound; abnormal relaxation; hypertensive heart disease; echocardiography

Added (extra) sounds during early diastole include a mitral opening sound and a pericardial knocking sound, as well as a third heart sound. However, it is very rare to find these heart sounds during the isovolumic relaxation period.1,2 We report here a patient with hypertensive heart disease, left ventricular systolic dysfunction, and complete left bundle branch block in whom an added sound was heard during the isovolumic relaxation period.

Case report
The patient was a 69 year old woman who had been treated for hypertension by her family doctor. In May 1995, she began to experience precordial chest pressure and she was treated with sublingual glyceryl trinitrate. When this symptom became frequent and was accompanied by dyspnoea, she again consulted her doctor and a diagnosis of congestive heart failure was made, based on a finding of pulmonary congestion on a chest radiograph. She was referred to our hospital for further investigation.

Physical examination showed a heart rate of 88 beats/min and regular, and blood pressure of 170/78 mm Hg. On auscultation, reversed splitting of the second heart sound and an added sound during early diastole, similar to a third heart sound, were heard at the cardiac apex. A systolic ejection murmur, grade 2/6 by the Levine classification, was also heard at the left sternal border in the third and fourth intercostal spaces. The liver was not palpable, and no lower extremity oedema was noted. The heart appeared enlarged, with a cardiothoracic ratio of 62%, and pulmonary congestion was noted on chest radiograph. Echocardiogram showed normal sinus rhythm with slightly prolonged PR interval (200 ms), poor R wave progression in leads V1–V3, and complete left bundle branch block with left axis deviation (−40°) and a prolonged QRS interval (150 ms).

Phonocardiography showed a decrease in the first heart sound, reversed splitting of the second heart sound, and a low pitched added sound at the cardiac apex during early diastole (fig 1). This added sound did not change dur-
ing respiration. A systolic ejection murmur, consistent with Levine grade 2/6, also was recorded from the cardiac apex to the left sternal border in the third intercostal space.

An M mode echocardiogram showed left ventricular enlargement (end diastolic diameter 7.0 cm, end systolic diameter 5.7 cm), a decrease in percent fractional shortening of the left ventricle (23%), and left atrial enlargement (5.0 cm). There was no evidence of heart valve or pericardial disease. On simultaneous recordings using phonocardiogram, M mode, and pulsed Doppler echocardiogram, this added sound coincided with a sudden interruption of the early diastolic motion of the left ventricular posterior wall and an initial phase of abnormal forward movement of the anterior leaflet of the mitral valve during early diastole, and came just before the early diastolic transmitral flow, consistent with the isovolumic relaxation period (fig 2).

Pulsed Doppler tissue imaging showed that the early diastolic motion velocity from the base to the middle of the left ventricular posterior wall was markedly high and sharp, and its peak coincided with the added sound (fig 3, top). A coronary angiogram showed a 50% stenosis of the circumflex branch of the left coronary artery. On left ventriculography there was diffuse hypokinesis of the left ventricular wall with a left ventricular ejection fraction of 45% and abnormal inward movement of the left ventricular posterior wall during early diastole, as detected by M mode echocardiography. The left ventricular pressure curve showed a gradual decline from the isovolumic relaxation period to mid-diastole, indicating a left ventricular relaxation abnormality, and a notch during early diastole, probably corresponding to the occurrence of the added sound (fig 3, bottom). The left ventricular end diastolic pressure was 17 mm Hg. During her hospital admission, the patient's symptoms and pulmonary congestion were improved with diuretics and vasodilators, but the added sound did not disappear on follow up examinations.

Discussion

Added sounds during early diastole have been reported in patients with various types of heart disease.1-9 The third heart sound and mitral opening sound are representative examples of such sounds. Added sounds are very rare in patients without heart valve disease or pericardial disease, and are most likely to occur with dilated failing hearts.1 In the patient described here, this sound occurred before the opening of the mitral valve, that is, during the isovolumic relaxation period, and was distinct from the third heart sound or mitral opening sound.

Added sounds during the isovolumic relaxation period may be caused by abnormal blood flow10 or by extracardiac factors.1-3 Lee et al11 have reported a patient with an added sound caused by intraventricular blood flow during the isovolumic relaxation period, and speculated that the mechanism for this sound was a prolongation of the isovolumic relaxation period with asynchronous relaxation. Sasson et al12 proposed that asynchronous contraction of the left ventricular wall at end systole may result in regional pressure differences within the ventricular cavity during early diastole, and thus lead to intraventricular blood flow during the isovolumic relaxation period and produce added sounds. Our patient had impaired left ventricular systolic function, similar to that reported by Lee et al11 with prolongation of the isovolumic relaxation period accompanied by a curious pattern of left ventricular posterior wall motion and an abnormal pressure curve. However, no distinct intraventricular blood flow signal was detected during.

Figure 2. Simultaneous recordings of phonocardiogram, M mode, and pulsed Doppler echocardiograms. The extra sound (ES) coincides with a sudden interruption (arrows) of the early diastolic motion of the left ventricular posterior wall (PW) (top), the initial phase of abnormal forward movement (arrows) during early diastole of the anterior mitral valve (AMV) (middle), and just before the early diastolic flow (E) of the transmitral flow velocity (TMF) (bottom). IVS, interventricular septum; 1, the first heart sound; 2, the second heart sound; PCG, phonocardiogram; A, atrial systolic wave of the transmitral flow velocity; ECG, electrocardiogram.
electrocardiogram; Aw, imaging high early diastolic motion derivative of notch (bottom, arrow) and ECG ES sharp, represents and --- im ventricular velocities pressure curve was its curve. atrial - and its from peak coincides with the systolic (LVP) the isovolumic diastole. The isovolumic relaxation period. This sound may have been produced by impact between the heart and the extracardiac structures during the isovolumic relaxation period. This sound may have been produced by impact between the dilated failing heart, with a ventricular relaxation abnormality, and the extracardiac structures during the isovolumic relaxation period.

In summary, we described a case with hypertensive heart disease producing unusual motion of the left ventricular posterior wall and a loud added sound during the isovolumic relaxation period. This sound may have been produced by impact between the dilated failing heart, with a ventricular relaxation abnormality, and the extracardiac structures during the isovolumic relaxation period.

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