Isovolumic Relaxation Flow in Patients with Left Bundle Branch Block and Normal Coronary Arteriogram

Zehra GÖLBASI, MD, Hüsamettin ATASEVER, MD, Nurten AKYUREK,1 MD, and Mevlüt ÇAPANOĞLU, MD

SUMMARY
The purpose of this study was to clarify the presence of isovolumic relaxation flow in patients with left bundle branch block and normal coronary arteriogram. Twenty-four patients with left bundle branch block and normal coronary arteriogram were examined by pulsed Doppler echocardiography and were compared with 20 age- and gender-matched healthy subjects. Impaired left ventricular relaxation was found in patients with LBBB. All 24 study patients showed isovolumic relaxation flow, but only 4 healthy subjects had isovolumic relaxation flow (p < 0.05). Peak velocity of the isovolumic relaxation flow ranged from 20–42 cm/s. In the study group, left ventricular systolic function was normal in 17 patients, and reduced in the remaining patients. At the end of this study, the presence of isovolumic relaxation flow which may be due to an abnormal septal motion was found in patients with left bundle branch block and normal coronary arteriogram. (Jpn Heart J 1998; 39: 653–657)

Key words: Left bundle branch block, Diastolic function, Pulsed Doppler, Intraventricular flow

The isovolumic relaxation period is an ideal phase for evaluating relaxation. Recently, Doppler echocardiography has been used to provide a detailed noninvasive estimate of intraventricular flow direction and velocity during isovolumic relaxation. This flow has been observed in patients with hypertrophic cardiomyopathy, ischemic heart disease, aortic stenosis, hypertension, undergoing right ventricular pacing, and in normal subjects.1 4) The basis for isovolumic relaxation flow (IRF) has been ascribed to an intracavitary gradient in the left ventricle. The abnormal anterior motion of the interventricular septum during ventricular ejection may be an important hemodynamic consequence of left bundle branch block (LBBB). As expected, the abnormal septal motion during ejection alters overall ventricular function. The purpose of this study was to clarify the presence and the mechanism of the isovolumic relaxation flow in

---

From the Department of Cardiology and 1Department of Internal Medicine, Ankara Numune Hospital, Ankara, Turkey.
Address for correspondence: Zehra Gölbasi, MD, Yuva sok. No: 20/2, Küçükesat-Ankara, 06660 Turkey.
Received for publication April 6, 1998.
Revised and accepted July 6, 1998.

653
patients with left bundle branch block and normal coronary arteriogram.

METHODS

This study was performed prospectively over a 6 month period and included 24 patients (16 women, 8 men, mean age 62 ± 12 years) with complete LBBB referred for elective cardiac catheterization for the evaluation of chest pain. No patient had a coronary artery lesion. Eleven patients had systemic hypertension. None of the patients had valvular heart disease, primary myocardial disease or left ventricular hypertrophy. Two dimensional, M mode, and continuous and pulsed wave Doppler echocardiograms were performed on each subject by 2 expert echocardiographers using commercially available echocardiographs with 3,75 MHz transducers. Mitral inflow velocity was recorded from the apical four-chamber view. The sampling volume was placed at the level of the mitral leaflet tips. The peak early diastolic inflow velocity (E), peak late diastolic inflow velocity (A), and the ratio of peak early to late velocities (E/A) were measured. The deceleration time of E and isovolumic relaxation time were also assessed. Deceleration time of E was defined as the time required for the E velocity to recline from its peak to baseline value, and the isovolumic relaxation period defined as the interval between aortic valve closure and mitral valve opening. The velocity and location of isovolumic relaxation flow in the left ventricle was ascertained by pulsed wave Doppler on the apical long axis view. Global left ventricular function was assessed by 2-dimensional echocardiography and was visually graded as normal, reduced, or hyperdynamic. Also, abnormal septal motion was evaluated on M mode and on 2-dimensional images. Paradoxic septal motion was defined as the motion of the interventricular septum in the anterior direction toward the ultrasound transducer during early ejection. As a control group, 20 age- and gender-matched healthy subjects (mean age 59 ± 11 years) were also evaluated.

Comparisons between two groups were performed by Student’s unpaired t test. Values are expressed as the mean value ± SD.

RESULTS

All 24 study patients showed isovolumic relaxation flow, but only 4 healthy subjects had isovolumic relaxation flow (p < 0.05) (Figure). The site of origin in most patients was at the centre of the left ventricle and along the middle one-third of the interventricular septum. In 15 patients the flow was directed toward the apex and in 9 patients it was bidirectional. Peak velocity of the isovolumic relaxation flow ranged from 20–42 cm/s (mean 29 ± 6 cm/s). In the study group,
17 patients had normal and 7 patients had reduced left ventricular systolic function. Although the early systolic downward dip of the septum was present in all patients, paradoxic septal motion was only present in 16 patients. In the study patients, peak early filling velocity (E) was $38 \pm 19$ cm/s, peak atrial filling velocity (A) $59 \pm 19$ cm/s, and deceleration time of E $184 \pm 23$ ms. In healthy subjects, these values were $62 \pm 21$ cm/s, $58 \pm 33$ cm/s, and $168 \pm 41$ ms, respectively. In the study patients, the left ventricular isovolumic relaxation time was longer than that of control subjects ($136 \pm 32$ ms, $82 \pm 24$ ms respectively; $p < 0.05$). In the study patients, values of IRF velocity were not statistically different in patients with or without left ventricular systolic dysfunction and also were not different in patients with or without abnormal septal motion ($31 \pm 9$ ms vs. $28 \pm 9$ ms and $29 \pm 9$ ms vs. $28 \pm 7$ ms, respectively). All healthy subjects had normal systolic function.

**DISCUSSION**

The degree to which LBBB itself impairs left ventricular contractility, as measured by cineangiography, is controversial. Furthermore, the effects of
LBBB on diastolic left ventricular function have not been investigated in detail. One study assessed the effects of pacing-induced left bundle branch block on left ventricular systolic and diastolic performance and demonstrated that alteration in the intraventricular conduction itself could induce impairments of left ventricular systolic and diastolic functions. In another study which assessed the systolic and diastolic left ventricular function in a patient with an echocardiographically normal left ventricle and rate-dependent LBBB, it was concluded that left bundle branch block can cause a striking deterioration of ventricular filling. In a study with dogs, pacing induced left ventricular asynchrony caused a decrease in the global and regional indexes of the left ventricular relaxation rate that was not closely related to the left ventricular loading conditions or shortening. Isovolumic relaxation period is an ideal phase for evaluating relaxation. Because there is no blood flow into the left ventricle through the mitral and aortic valves, intraventricular redistribution of flow during this period should reflect active chamber relaxation. In this study, impaired left ventricular relaxation was found in patients with natural LBBB (a decrease in peak early filling velocity (E), E/A ratio < 1, and abnormally long isovolumic relaxation time). IRF is associated with impaired left ventricular relaxation. The present study indicates that patients with left bundle branch block had IRF.

In a study in which IRF was detected in patients undergoing right ventricular pacing, it was reported that segmental early relaxation appears to be the basis for isovolumic relaxation flow in these patients. Despite the fact right ventricular pacing has conventionally been used as an experimental model of left bundle branch block, in natural LBBB the activation of the left ventricle seems to take various pathways. The different patterns of septal motion found in patients with artificial LBBB and in those with natural LBBB could be explained by differences in activation of the heart. The mechanism of paradoxical septal motion in LBBB is uncertain. A better explanation may be an initial septal activation on the right side of the septum as occurs in LBBB causing septal contraction first when the rest of the left ventricle is quiescent. When the rest of the ventricle contracts, the septum is relaxed and pushed anteriorly away from the posterior wall because of rising left ventricular pressure. In patients with natural LBBB, abnormal septal motion appears to be the mechanism of the IRF.

Although Sasson et al. reported that isovolumic relaxation flow was associated with normal and hyperdynamic left ventricular systolic function and cavity obliteration, subsequent studies have shown that it could also occur with left ventricular dysfunction. In the present study, none of the patients had hyperdynamic left ventricle or cavity obliteration. Seven patients with left ventricular dysfunction had isovolumic relaxation flow.

In conclusion, independent of left ventricular systolic function, isovolumic
relaxation flow is frequently detected in patients with left bundle branch block and normal coronary arteriogram.

REFERENCES