Letters to the Editor

Resolution of rate-related left bundle branch block after nitrate therapy

Intermittent rate-dependent complete left bundle branch block (CLBBB) is rare and may not be associated with underlying coronary artery disease (CAD)[1]. Only one report has shown that drug treatment may be beneficial[2].

A 48-year-old woman was admitted to our department with hypertension and hyperglycaemia. Her health had otherwise been good, with no cardiovascular symptoms. On admission, physical examination showed severe obesity, tachycardia, a grade 2 mid-systolic murmur, and supine blood pressure of 180/108 mmHg. Blood biochemistries confirmed the presence of hyperglycaemia (323 mg . dl^{-1}), showed high levels of total cholesterol (265 mg . dl^{-1}) and triglycerides (241 mg . dl^{-1}). The electrocardiogram taken while at rest showed signs of an old anteroseptal infarction (Fig. 1(A)). This was confirmed by echocardiography which showed mild left ventricular hypertrophy, decreased septal motion and thickening, with an ejection fraction of 70%. Myocardial scintigraphy showed the presence of an anteroseptal myocardial perfusion defect.

When the patient performed the Valsalva manoeuvre, heart rate acceleration occurred (phase 2), associated with the appearance of a CLBBB that disappeared when the heart rate decreased (phase 4) (Fig. 1(B)). Testing with Valsalva was repeated several times, always with the same results. Average onset and offset heart rates were calculated at 100 beats . min^{-1} and 79 beats . min^{-1}, respectively. Holter ECG monitoring was normal. Treatment with isosorbide 5-mononitrate was begun at a dose of 20 mg b.i.d. After ten days the 'resting' electrocardiogram showed no conduction abnormalities. The Valsalva manoeuvre (maximum heart rate: 106 beats . min^{-1}) and handgrip test (maximum heart rate: 104 beats . min^{-1}) induced no changes in the electrocardiogram. An exercise stress test was then performed with a staged protocol that was interrupted because of fatigue when the heart rate was 130 beats . min^{-1} and blood pressure 210/124 mmHg. No echocardiographic abnormalities were observed. A re-challenge was performed 10 days after withdrawal of nitrate therapy and the Valsalva manoeuvre again showed rate-dependent left bundle branch block. Nitrate treatment was repeated and the patient has now undergone electrocardiographic follow-up for 8 months without evidence of recurrence of CLBBB.

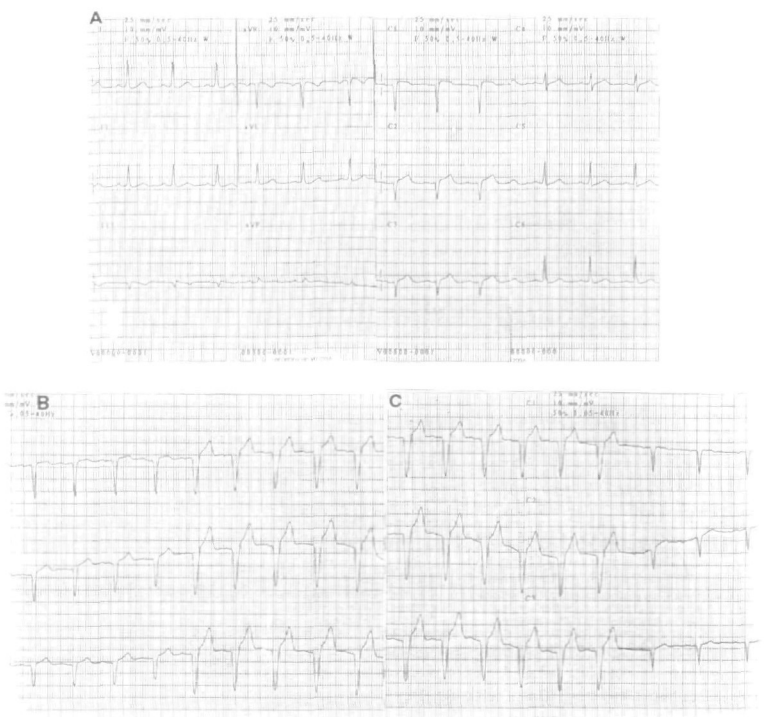

Figure 1 **(A) 12-lead rest electrocardiogram. QS complexes were present in V_1–V_3 indicating an old anteroseptal infarction. (B and C) Rate-related left bundle branch block. (B) Onset. (C) Offset.**

CLBBB due to increased heart rate and induced by exercise is rare and its clinical significance controversial. Some studies suggest that a significant proportion of patients with rate-related CLBBB have underlying cardiac abnormalities such as left ventricular hypertrophy, myocardial infarction or other conduction system abnormalities[3]. Similar evidence was suggested in our patient by echocardiography and myocardial scintigraphy. Some investigators attempted to define parameters which might suggest the presence of CAD in subjects with rate-related CLBBB. Vasey *et al.* suggested that the rate of onset of CLBBB may be a marker of CAD[1], and follow-up studies have been performed to evaluate the prognosis of rate-related CLBBB. In contrast, other studies have not reported a close association between frequency-dependent CLBBB and CAD[4–6].

In distinct contrast to the adverse prognostic significance attributed to patients with persistent CLBBB, an excellent prognosis with no progression of the conduction disturbance was reported in subjects with intermittent, rate-related CLBBB without evidence of CAD[4].

To our knowledge, only two reports have demonstrated that rate-related CLBBB can be resolved by treating underlying CAD. Puleo *et al.* described the resolution of exercise-induced CLBBB after successful angioplasty of proximal left anterior descending coronary artery lesion[3]. Garcia Pascual *et al.* described the resolution of exercise-induced CLBBB after nifedipine therapy in a patient with angina, pathological exercise stress testing and normal coronary arteriograms[2]. Our report demonstrates that rate-related CLBBB can benefit from treatment with nitrates. This might be the consequence of improved coronary blood flow or, alternatively,

the result of left ventricle unloading due to venodilatation.

L. A. SECHI
S. DE CARLI
L. ZINGARO
E. BARTOLI
Hypertension Unit, Department of Internal Medicine, University of Udine, 33100 Udine, Italy

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How does a continuous coronary perfusion catheter work in coronary artery dissection? Assessment by intravascular ultrasound

Symptomatic, flow-limiting dissections occur in a certain percentage of patients after percutaneous transluminal coronary angioplasty (PTCA), depending mainly on lesion and procedural characteristics[1]. We have shown recently that prolonged balloon inflations by means of a coronary perfusion catheter (CPC) are beneficial for treating symptomatic, flow-limiting dissections after PTCA[2]. The exact mechanism underlying the successful management of dissections by perfusion catheters, is, however, not fully understood, mainly because angiography provides limited information about the vessel wall. Thus, intravascular ultrasound (IVUS), a catheter-based method providing on-line, cross-sectional images of the vessel wall, has been accepted as an additional diagnostic tool. However, until now, no study has addressed the morphological characteristics of flow-limiting dissections after PTCA by IVUS, or analysed the effects of prolonged inflation by perfusion catheters. Therefore, we studied a 43-year-old male patient with unstable angina, by means of angiography and IVUS following insufficient standard balloon angioplasty and after successful manipulation with a CPC.

The patient's angiogram revealed a >90% proximal LAD stenosis. After two balloon inflations (45 s and 60 s with 8 and 12 bar, 3 mm monorail balloon), the angiogram revealed an unsatisfactory result and the patient reported chest pain (see Fig. 1). To obtain more information, a mechanical, 30 MHz, 3·5F IVUS catheter

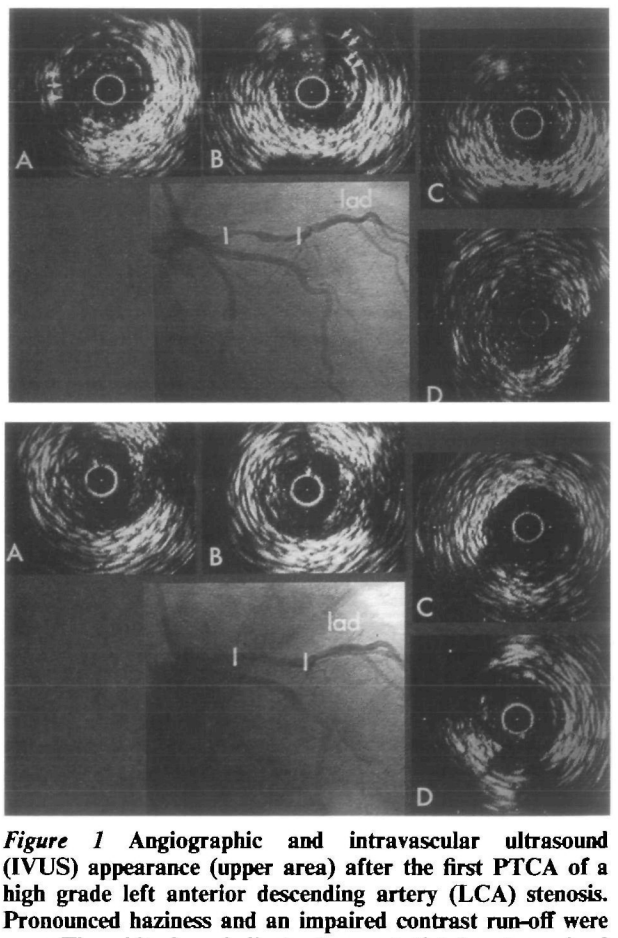

Figure 1 **Angiographic and intravascular ultrasound (IVUS) appearance (upper area) after the first PTCA of a high grade left anterior descending artery (LCA) stenosis. Pronounced haziness and an impaired contrast run-off were seen. The white bars indicate the most distal and proximal position of the IVUS catheter; letters A to D refer to the proximal and more distal positions. White arrows in the IVUS images indicate the contrast material after its injection by hand. The IVUS images after the first PTCA depict a rough surface, small luminal diameters, and a subintimal dissection with flow and mobile structure within the lesion. The distance between two dots equals 1 mm. The lower area shows the angiographic appearance after prolonged inflation with a 3·5 mm perfusion balloon catheter. A satisfactory angiographic result with normal run-off of contrast material is seen. IVUS images showed an increase in luminal area, a smoother surface, and 'disappearance' of the dissection membrane.**