References

[1] Connolly SJ, Sheldon R, Roberts RS, Gent M. The North American Vasovagal Pacemaker Study (VPS). A randomized trial of permanent cardiac pacing for the prevention of vasovagal syncope. J Am Coll Cardiol 1999; 33: 16–20.

[2] Benditt DG. Cardiac pacing for prevention of vasovagal syncope. J Am Coll Cardiol 1999; 33: 21–3.

[3] Atiga WL, Rowe P, Calkins H. Management of vasovagal syncope. J Cardiovasc Electrophysiol 1999; 10: 874–86.

[4] Passamani E. Clinical trials, are they ethical? N Engl J Med 1991; 324: 1589–92.

[5] Sutton R, Petersen MEV. The clinical spectrum of neurocardiogenic syncope. J Cardiovasc Electrophysiol 1995; 6: 569–76.

[6] Santini M, Ammirati F, Colivicchi F, Gentilucci G, Guido V. The effect of atropine in vasovagal syncope induced by head-up tilt testing. Eur Heart J 1999; 20: 1745–51.

[7] Morillo CA, Leitch JW, Yee R, Klein GJ. A placebo-controlled trial of intravenous and oral disopyramide for prevention of neurally mediated syncope induced by head-up tilt. J Am Coll Cardiol 1993; 22: 1843–8.

[8] Ector H, Reybrouck T, Heidbüchel H, Gewillig M, Van de Werf F. Tilt training: a new treatment for recurrent neurocardiogenic syncope and severe orthostatic intolerance. PACE 1998; 21 (II): 193–6.

[9] Ector H, Reybrouck T, Heidbüchel H, Ramaekers D, Van de Werf F. Tilt training in malignant neurocardiogenic syncope: results of follow-up in 42 patients (Abstr). PACE 1999; 22 (part II): 756.

European Heart Journal (1999) **20,** 1687–1688
Article No. euhj.1999.1776, available online at http://www.idealibrary.com on IDEAL®

New approach to the diagnosis of myocardial bridging by intracoronary ultrasound and Doppler

See page 1707 for the article to which this Editorial refers

Myocardial bridging is anatomically defined by the intramyocardial course of portions of the coronary arteries, mainly the midportion of the left anterior descending coronary artery[1]. Its typical angiographic presentation is the systolic 'milking' effect due to transient myocardial vessel compression[2]. Since Portmann and Iwig's[3] description of angiographically visualized systolic narrowing of the left anterior descending coronary artery, many subsequent reports have described the milking effect of this artery. A large discrepancy exists between pathological series, in which the incidence has varied from 15% to 85%[4], and angiographic series, in which it is reported as being between 0·51% and 2·5%[5]. There is also considerable controversy regarding the clinical, haemodynamic and prognostic significance of myocardial muscle bridges. Although various reports are available describing myocardial ischaemia, myocardial infarction[6], conduction disturbances[7] and sudden death[8] in association with this anatomical variation and otherwise normal coronary arteries, myocardial bridges are generally considered harmless clinical anomalies.

Ge *et al*. were pioneers in the potential clinical application of intravascular ultrasound to detect myocardial bridging[9], and provided unique information concerning wall morphology in this condition. Intravascular ultrasound is a unique technique that visualizes the coronary wall and therefore enables changes in its shape and structure with particular reference to coronary segments with myocardial bridging, to be analysed. This analysis demonstrated that vessel compression within the bridge is not a purely systolic event, but persists throughout large periods of diastole.

Ge *et al*.[10] present a new description of morphological signs characteristic of myocardial bridging by intravascular ultrasound. For the first time, they describe a specific morphological sign, a 'half-moon' like echolucent area surrounding the bridge segment. The presence of this sign seems to be highly specific for the existence of myocardial bridging, as it can only be found in the bridge segment, and not in the proximal and distal segments and other coronary arteries. It is important to note that when the specific half moon phenomenon is demonstrated by intravascular ultrasound, the milking effect can be provoked by intracoronary administration of nitroglycerin, although the milking effect was not initially revealed by angiography.

In the present study Ge *et al*.[10] nicely highlight the potential use of intravascular ultrasound in the assessment of myocardial bridge and its clinical

importance. In the largest group of patients to have been studied so far, coronary flow velocity was measured in order to calculate coronary flow reserve. The patients with myocardial bridges have a reduced coronary flow reserve[9–11]. This may be due to a moderate increase in resting flow velocity and a limited hyperaemic response, as a result of a reduced mid to late diastolic flow velocity and a marked reduction in systolic flow velocity. These results are again a worthwhile observation. Similar findings were obtained by Schwarz *et al.*[11] in a smaller series of patients. The functional characteristics of myocardial bridging, associated with reduced coronary flow reserve, enhanced by rapid pacing, support the concept of haemodynamically significant obstruction of coronary flow due to myocardial bridging in selected patients. The authors[10] report an important aspect regarding clinical implications in myocardial bridging: they show a high incidence of atherosclerosis in the proximal segment to the bridge, with no plaque formation in the bridge segment. The coronary flow velocity reserve in the proximal normal segment seems to be impaired in patients with severe coronary arteriosclerosis in comparison to the patients without. This indicates that myocardial bridging-induced haemodynamic disturbance is the main reason for myocardial ischaemia. The subsequent atherosclerosis in the segment proximal to the bridge may worsen myocardial ischaemia. Furthermore, this may explain the previous observations that this syndrome is associated with myocardial ischaemia, myocardial infarction, and conduction disturbances.

Ge *et al.*[10] also reported a typical Doppler flow pattern: an early prominent diastolic peak in coronary flow velocity in myocardial bridge segments. A sharp acceleration of flow was seen in early diastole, followed immediately by marked deceleration with a velocity plateau in mid to late diastole and a decreased or no systolic antegrade flow (finger-tip). This flow pattern has been previously described[9,11] as a characteristic of myocardial bridging. It is important to note the absence of a finger-tip phenomenon in 13% of the patients. This finding indicates that morphologically the half-moon phenomenon is more sensitive than the functional finger-tip phenomenon in identifying myocardial bridging. This is a new important observation.

Identification of the presence and evaluation of the severity of myocardial bridging in symptomatic patients without another co-existing coronary artery disease is of clinical importance[12]. Clinical symptoms are related to disturbed coronary blood flow. Improvement in coronary haemodynamics can be assessed by normalization of the coronary flow patterns. Ge *et al.* offer new concepts in myocardial bridging. Further studies are necessary to evaluate which therapeutic method (if always needed) is the best to release the compression of the bridging in symptomatic patients and its clinical implications.

V. SÁNCHEZ
J. ZAMORANO
Hospital Clínico San Carlos,
Madrid, Spain

References

[1] Geiringer E. The mural coronary. Am Heart J 1951; 41: 359–68.
[2] Noble J, Bourassa MG, Petitclerc R. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal or obstruction? Am J Cardiol 1976; 37: 993–9.
[3] Portmann W, Iwig J. Die intramurale Koronarie im Angiogramm. Fortschr Rontgenstr 1960; 92: 129–32.
[4] Polacek P. Relation of myocardial bridges and loops on the coronary arteries to coronary occlusion. Am Heart J 1961; 61: 44–52.
[5] Irving GI. The angiographic prevalence of myocardial bridging in man. Chest 1982; 81: 198–202.
[6] Tauth J, Sullebarger JT. Myocardial infarction associated with myocardial bridging: case history and review of literature. Catheter Cardiovasc Diagn 1997; 40: 364–7.
[7] Den Dulk K, Brugada P, Braat S, Heddle B, Wellens HJJ. Myocardial bridging as a cause of paroxysmal atrioventricular block. J Am Coll Cardiol 1983; 1: 1965–70.
[8] Desseigne P, Tabib A, Loire R. Myocardial bridging of the left anterior descending coronary artery and sudden death. Apropos of 19 cases with autopsy. Arch Mal Coeur Vaiss 1991; 84: 511–6.
[9] Ge J, Erbel R, Hans-Jürgen R *et al.* Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging. Circulation 1994; 89: 1725–32.
[10] Ge J, Jeremias A, Rupp A *et al.* New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J 1999; 20: 1707–16.
[11] Schwarz ER, Klues HG, vom Dahl J, Klein I, Krebs W, Hanrath P. Functional, angiographic and intracoronary characteristics in symptomatic patients with myocardial bridging: effect of short term intravenous beta-blocker medication. J Am Coll Cardiol 1996; 27: 1637–45.
[12] Alfonso F, McKenna WJ. Milking in Hypertrophic Cardiomyopathy. J Am Coll Cardiol 1999 (in press). Letter.