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New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler

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Background Large discrepancies exist concerning the incidence of myocardial bridging. This has been reported to be 0·5%–2·5% following coronary angiography but 15%–85% following autopsy. The purpose of the study was to use intravascular ultrasound and intracoronary Doppler to study the morphology and flow characteristics of myocardial bridging in order to find feasible parameters of this syndrome.

Methods and Results Intravascular ultrasound was performed in 62/69 patients in whom typical angiographic 'milking effects' were present. In 48 patients, intracoronary Doppler was performed. A specific, echolucent 'half moon' phenomenon surrounding the myocardial bridge was found in all the patients. The thickness of the half moon area was $0{\cdot}47 \pm 0{\cdot}19$ mm in diastole and $0{\cdot}52 \pm 0{\cdot}23$ mm in systole. There was systolic compression of the myocardial bridge with a lumen reduction during systole of $36{\cdot}4 \pm 8{\cdot}8\%$. Using intracoronary Doppler, a characteristic early diastolic 'finger tip' phenomenon was observed in 42 (87%) of the patients. All patients showed no or reduced antegrade systolic flow. Coronary flow velocity reserve was $2{\cdot}03 \pm 0{\cdot}54$. After intracoronary nitroglycerin injection, retrograde systolic flow occurred in 37 (77%) of the 48 patients, with a velocity of $-22{\cdot}2 \pm 13{\cdot}2$ cm . s^{-1}. Intravascular ultrasound revealed atherosclerotic involvement of the proximal segment in 61 (88%) of the 69 patients, with an area stenosis of $42 \pm 13\%$. No plaques were found in the bridge or distal segments in the 62 patients in whom it was possible to introduce the ultrasound catheter throughout the bridging segment.

Conclusion Myocardial bridging is characterized by the following morphological and functional signs: a specific, echolucent half moon phenomenon over the bridge segment, which exists throughout the cardiac cycle; systolic compression of the bridge segment of the coronary artery; accelerated flow velocity at early diastole (finger-tip phenomenon); no or reduced systolic antegrade flow; decreased diastolic/systolic velocity ratio; retrograde flow in the proximal segment, which is provoked and enhanced by nitroglycerin injection.

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Key Words: Myocardial bridging, coronary artery disease, intracoronary ultrasound, intracoronary Doppler, atherogenesis, coronary flow.

See page 1687 for the Editorial comment on this article

Introduction

Myocardial bridging occurs when a segment of a coronary artery or its major branch travels through the myocardium instead of on the surface of the myocardium. The first description of this phenomenon was given by Grainicuanu in the early 1920s[1]. Patients demonstrating this phenomenon may suffer from myocardial ischaemia[2–5], myocardial infarction[6–9], conduction disturbances[10], and even sudden death[11–13]. This phenomenon is angiographically characterized by a systolic 'milking effect'[14]. However, a large discrepancy exists between pathological series, showing an incidence between 15% and 85%[15,16], and coronary angiographic series, showing an incidence of between 0·51% and 2·5%[2,17,18].

Intravascular ultrasound and intracoronary Doppler are new methods, superceding coronary angiography,

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Table 1 Demographic data of the patients

Age (years)	52 ± 10
Gender (males)	54 (78%)
Unstable angina	5 (7%)
Hypertension	23 (33%)
Diabetes mellitus	4 (6%)
Hypercholesteraemia	39 (57%)
Current smoking	30 (33%)
Diameter stenosis >50%	12 (17%)

for visualizing arterial wall and flow characteristics in detail. We have described an early observation of myocardial bridging using intravascular ultrasound and intravascular Doppler catheter[19]. We now report new and characteristic signs of identifying myocardial bridging using intravascular ultrasound and intracoronary Doppler which may have an impact on patient management.

Methods

Study patients

The study group consisted of 69 patients (54 males, 15 females, 53 ± 9 years) with an angiographically typical milking effect in the left anterior descending coronary artery. Intravascular ultrasound was performed in all the 69 patients and intracoronary Doppler was performed in 48 patients. The coronary arteries were scanned from distal to proximal segments with a 20 or 30 MHz intravascular ultrasound catheter. All the patients (five of whom had unstable angina) presented with chest pain and underwent a clinical diagnosis by angiography. A diagnosis by thallium myocardial scintigraphy was available in 43 patients.

All patients gave written informed consent for coronary angiography, intravascular ultrasound and intracoronary Doppler flow mapping. Intravascular ultrasound and intracoronary Doppler were performed directly after diagnostic coronary angiography.

Intravascular ultrasound

The intravascular ultrasound imaging system used in this study has previously been described in detail[19,20]. A 3·2 F (Microview, CVIS, Boston Scientific Co., Watertown, MA, U.S.A.) 3·5 F or 4·8 F (Sonicath, Boston Scientific Co., Watertown, MA, U.S.A.) imaging catheter was used with a 20 MHz or 30 MHz single element transducer at the tip. The transducer is mechanically rotated within the catheter at 600 to 800 rpm to provide cross-sectional images via an ultrasound diagnostic imaging console (CVIS, Scimed, Boston Scientific Co., Watertown, MA, or Hewlett Packart, Sonos 1000, Andover, or Diasonics, Milpitas, CA, U.S.A.). The gain setting was adjusted to provide the best gray scale

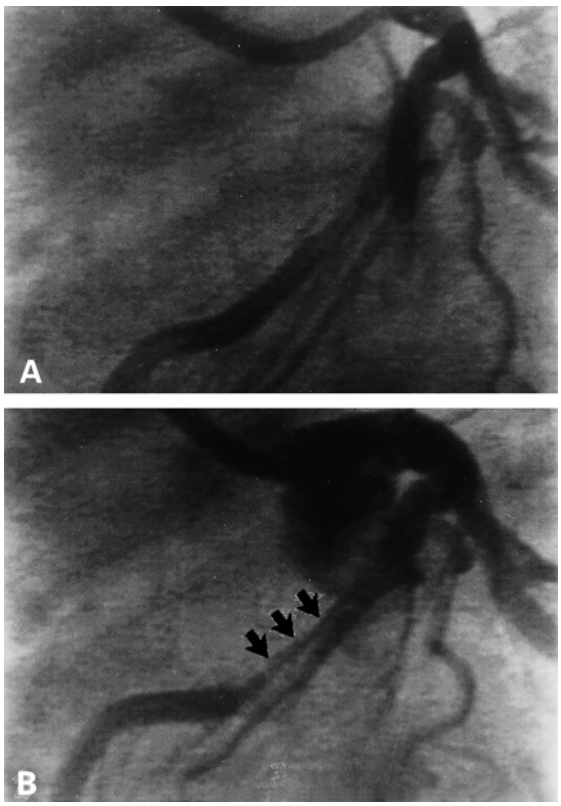

Figure 1 **Coronary angiograms in a patient with myocardial bridging in the left anterior descending coronary artery, in the cranial left anterior oblique projection. A significant 'milking effect' (arrows) can be seen during systole (B) which is released in diastole (A).**

differentiation, in order to obtain optimal morphology and structure visualization. The images were stored on a half inch s-VHS videotape for off-line analysis. An ECG was recorded simultaneously.

Intracoronary Doppler

For intracoronary Doppler examination, a 0·014 inch, 15 MHz Doppler wire (FloWire®, Cardiometrics, Mountain View, CA, U.S.A.) was used. Coronary average peak velocity ($cm \cdot s^{-1}$), and maximal peak flow velocity ($cm \cdot s^{-1}$) were recorded. For assessment of coronary flow velocity reserve, intracoronary adenosine (18 μg) was used to produce a hyperaemic reaction. Coronary flow velocity reserve was registered as the ratio of average peak velocity between hyperaemia and baseline. The diastolic/systolic velocity ratio was also documented.

Ultrasound procedure

Each intravascular ultrasound study was carried out as previously described[19–21]. After diagnostic coronary

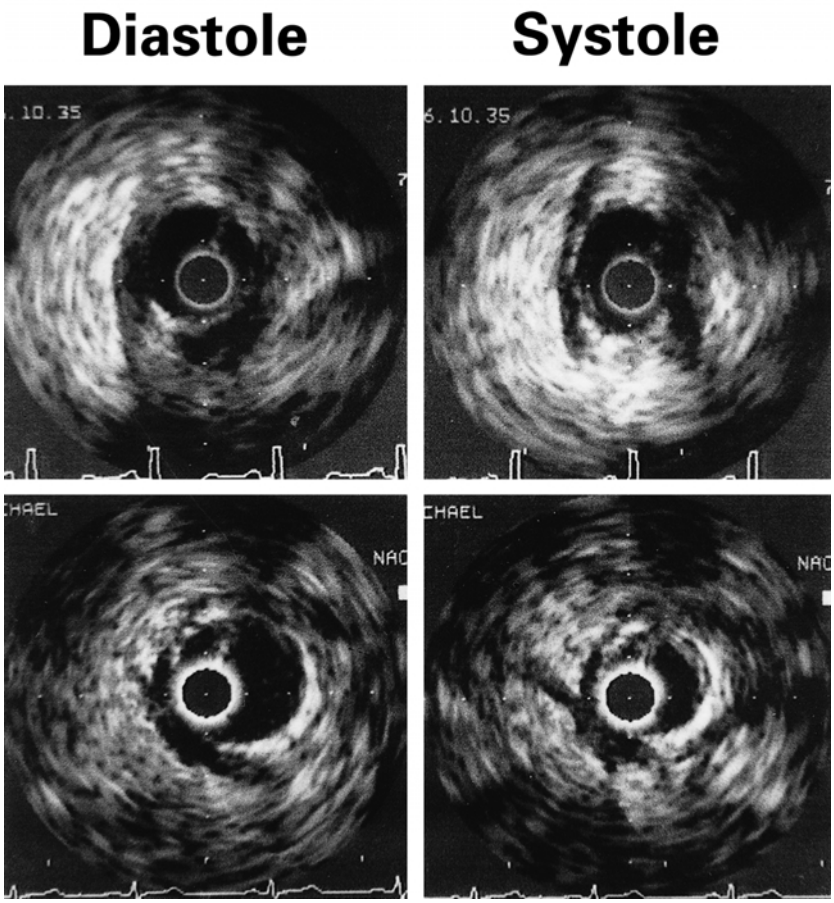

Figure 2 **Intravascular ultrasound imaging of the myocardial bridging during diastole (left) and systole (right). A 'half-moon' like, echolucent area surrounding the bridge is seen during the whole cardiac cycle. The distance between two calibration marks is 1 mm.**

angiography, an additional bolus of 3000 IU heparin was given intracoronarily. An 8F giant lumen guiding catheter (Medtronic, Denvers, MA or Cordis, Miami, FL, U.S.A.) was positioned in the ostium of the left main coronary artery. A 0·014 inch (0·36 mm) floppy guide wire (ACS, Timicula, CA, U.S.A.) was inserted into the coronary artery. The intravascular ultrasound side-saddle catheter was guided in a monorail fashion into the left coronary artery. The catheter was advanced as far as possible into the target coronary artery. Serial cross-sectional images of the vessel were obtained by slowly pulling back the intravascular ultrasound catheter with stop frames at each 2–3 mm interval. The position of the intravascular ultrasound probe was documented on X-ray film at every stop frame in order to have precise matching between the numbered intravascular ultrasound images and the angiograms. Contrast medium was injected in order to define the luminal border of the vessel wall and in order to ascertain whether there is communication between the lumen and the plaque, such as occurs after plaque rupture.

Intravascular ultrasound image analysis

The intravascular ultrasound images were reviewed off-line by independent observers. Optimal images at end-diastole were selected for analysis, using the software in the HP Sonos Intravascular machine. Interpretation of the intravascular ultrasound images was based on the previous studies[19–21]. In brief, lipid deposits were defined by echolucent regions within areas of intimal thickening or plaque that were not attributable to attenuation behind dense reflections. Calcific deposits were defined by bright reflections with acoustic shadowing. Fibrotic plaque was defined by hyperechogenic reflections of the lesion presented by dense echoes without acoustic shadowing.

The cross-sectional area of a vessel was defined as the area inside the echo-dense perimeter of the adventitia, and included lumen, plaque and media[19,20]. The luminal area was determined using planimetry of the ultrasound leading-edge interface between lumen and plaque. The percent area stenosis and diameter stenosis were calculated.

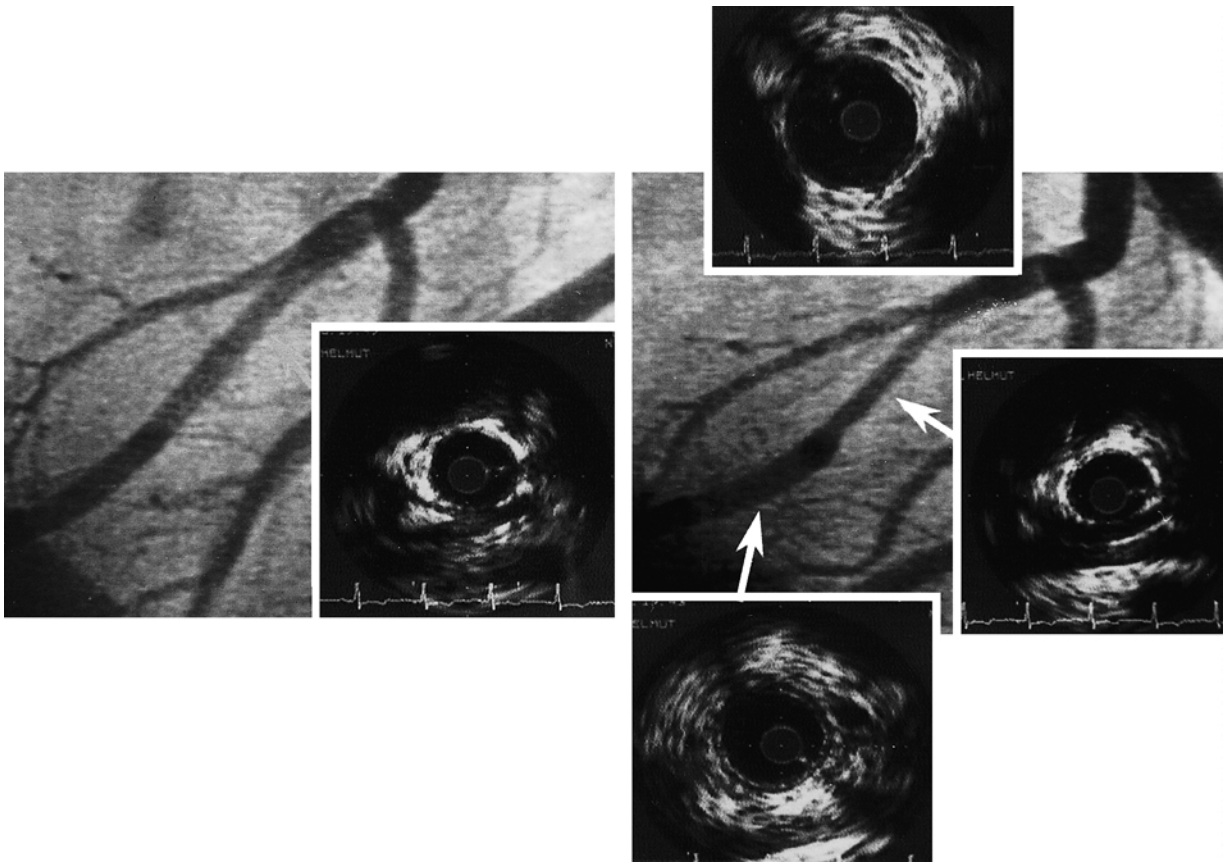

Figure 3 **Coronary angiogram and intravascular ultrasound images of bridge segments during diastole and systole. The specific 'half moon' area can only be seen in the bridged segment. The half moon does not exist in the proximal and distal segment. The distance between two calibration marks is 1 mm.**

The cross-sectional area variation of the MB segment during the cardiac cycle was analysed as previously reported[19]. In brief, 90 consecutive frames of videotaped intravascular ultrasound images of the myocardial bridge segment and of the normal segment proximal to MB were digitized into a 256×256 pixel computer. Semi-automatic software based on PV-wave (Precision Visuals, Inc., Boulder, Colorado, U.S.A.) had been developed for the evaluation of the lumen area; PV-wave is a computer program for analysing and displaying scientific data. The evaluation was performed on a personal computer (Mountain View, CA, U.S.A.). The program compensates for catheter movement due to heart beat by centring each image on consecutive frames. A region of interest comprising both the lumen and the vessel wall is defined interactively. Both the vessel wall and lumen were segmented by thresholding. To control the segmentation process, the determined contours of the lumen were superimposed on the original images. In case of mismatch, due for example to signal dropout, the procedure was repeated after correcting for the threshold or the region of interest. The area of the segmented lumen was measured after the removal of the catheter and guide wire artifact. The pulsatile variation of the cross-sectional lumen area of at least three cardiac cycles was displayed. For each cardiac cycle, the maximum area, the minimum area, and the end-diastolic area, as identified by ECG, were determined.

Coronary angiography and angiogram analysis

Siemens HICOR biplane catheterization equipment was used for coronary angiography (Siemens, Erlangen, Germany). All the angiograms were stored on CD-ROM. Coronary angiograms were assessed by two operators during coronary angiography and documented on hard copies. A diameter stenosis of more than 50% was considered as significant.

Statistical analysis

All the measurements and calculations were analysed using Microsoft's Excel Statistic Program. All the data were expressed as mean ± SD. A Student t-test analysis was used to assess the differences between the groups. A P value $<0{\cdot}05$ was considered statistically significant.

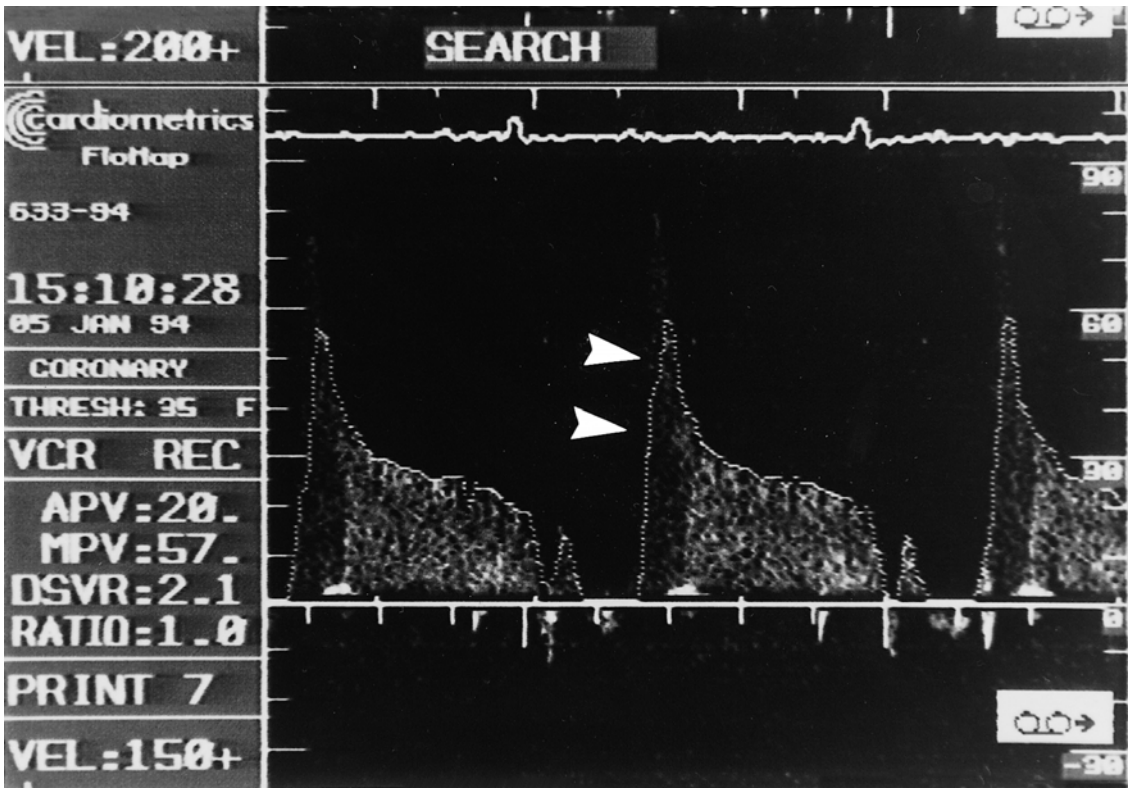

Figure 4 **Characteristic flow pattern in the bridge segment demonstrated by intracoronary Doppler FloWire®. A steep rise in the flow velocity at early diastole followed by a sharp deceleration and subsequent plateau ('finger-tip' phenomenon, arrows). No antegrade flow is observed during systole. APV = average peak flow velocity (cm . s^{-1}); DSVR = diastolic/systolic velocity ratio; MPV = maximal peak flow velocity (cm . s^{-1}).**

Results

The clinical characters and risk factors of the patients are listed in Table 1. In 18 of the 64 patients (28%) there were ischaemic ECG changes during exercise stress testing in the region supplied by a 'bridged' coronary artery. Thallium stress testing revealed signs of ischaemia in the anterior septal region in 14/43 (33%) patients and in the inferior wall in seven (16%).

Intravascular ultrasound was successfully performed in all cases (n=48) with 3·2 French or 3·5 French ultrasound catheters, i.e. the MB segment as well as the proximal and distal segments were scanned. The MB segment could be passed in only 14 (67%) of the other 21 patients using a 4·8 French intravascular ultrasound catheter.

In 12 (18%) of the 69 patients, significant stenoses were found in the proximal segment and coronary intervention was performed. Among these 12 patients, six had a positive exercise ECG. In two patients, no significant stenosis was found, despite a positive exercise ECG. All patients presented an angiographic milking effect in the mid to distal left anterior descending coronary artery (Fig. 1). Coronary spasm in the bridge segment occurred in two (2·9%) of the 69 patients during the intravascular ultrasound examination, which recovered after nitroglycerin injection.

Intravascular ultrasound

All the patients had systolic compression of the bridge segment. Of the 62 patients in whom the intravascular ultrasound catheter was able to pass through the bridge segment, 55 (89%) showed eccentric compression and the other seven (11%) concentric compression. A specific, half-moon like echolucent area surrounding the bridge segment could be found in all the patients (Fig. 2). This half moon area was located between epicardial tissue and the bridging coronary segment but not in a normal segment (Fig. 3). The thickness of the half moon area is $0{\cdot}47 \pm 0{\cdot}19$ mm (0·2 to 0·8 mm) in diastole and $0{\cdot}52 \pm 0{\cdot}23$ mm (0·2 to 1·2 mm) in systole.

A total of 61 (88%) of the 69 patients had plaque formation in the segment proximal to the bridge, with an area stenosis of $42 \pm 13\%$ (16% to 69%). The cross-sectional area of the plaque was 10 ± 4 mm^2 (6–17 mm^2). The majority of the atherosclerotic lesions (n=48) were eccentric (78%) and the others (n=13) concentric (22%). Calcification was found in 41 (67%) patients. Coronary

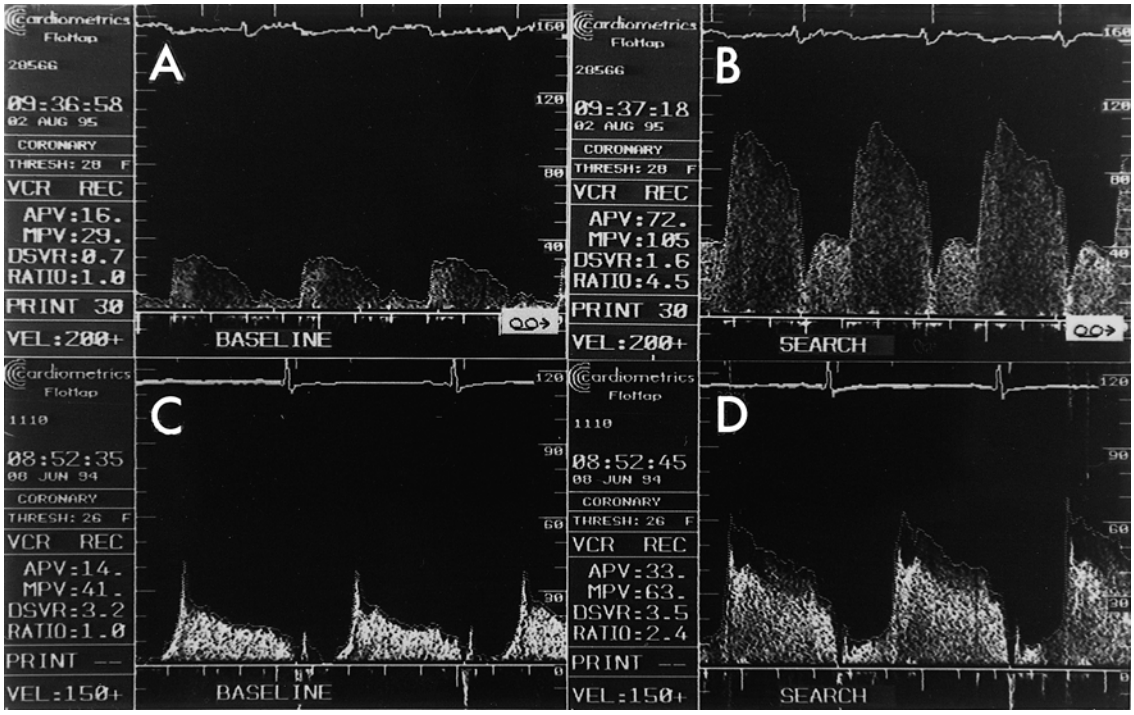

Figure 5 **Comparison of the coronary flow pattern in a reference vessel at baseline (A) and in hyperaemia (B) to the flow pattern in the bridge segment at baseline (C) and in hyperaemia (D). The flow pattern in the bridge is characterized by an early diastolic 'finger tip' phenomenon and reduced systolic antegrade flow. The diastolic/systolic velocity ratio is increased both at baseline and in hyperaemia in the patient with myocardial bridging.**

interventions (PTCA in four, stenting in eight) were performed in 12 (20%) of them. No plaque were found in the bridge and distal segments in the 62 patients in whom the intravascular ultrasound catheter was able to pass. The lumen area of the bridge segment decreased during systole to 36·4 ± 8·8%, with a range from 15·9% to 67%. The minimal cross-sectional lumen area was 4·87 ± 1·63 mm^2 (3·16–7·12 mm^2) and the maximal cross-sectional lumen area 6·33 ± 2·02 mm^2 (4·52–9·62 mm^2) ($P<0·001$). The maximal vessel area was found in early diastole.

Intracoronary Doppler

With intracoronary Doppler wire, a characteristic early diastolic, finger-tip like antegrade flow was seen in 42/48 patients (87%) (Fig. 4). This finger-tip flow pattern is characterized by a sharp acceleration of coronary flow velocity at early diastole followed by a subsequent steep deceleration with velocity plateau in mid to late diastole (Fig. 4). In addition, a decreased or absent systolic antegrade flow was seen in all patients in comparison to the flow pattern in normal segments (Fig. 5). Table 2 lists the flow velocity measured by intracoronary Doppler. The coronary flow velocity reserve was 2·03 ± 0·54. The diastolic/systolic velocity ratio was 2·91 ± 1·41 at baseline and 2·02 ± 0·79 at hyperaemia ($P<0·001$) (Fig. 6). The average peak velocity was 19·0 ± 7·9 cm . s^{-1} (8–46 cm . s^{-1}) at baseline and 37·3 ± 15·1 cm . s^{-1} (11–73 cm . s^{-1}) with hyperaemia (Fig. 7). The maximal peak velocity was 43·2 ± 18·7 cm . s^{-1} (15–78 cm . s^{-1}) at baseline and 66·7 ± 27·9 cm . s^{-1} (17–119 cm . s^{-1}) with hyperaemia. The coronary flow velocity reserve was 1·67 ± 0·68 in the eight patients with severe stenosis and 2·14 ± 0·65 in comparison to the 40 patients without ($P>0·05$).

After intracoronary injection of 200 µg nitroglycerin, a biphasic flow velocity reaction was seen: (a) abolishment of the finger-tip phenomenon, with an acceleration of flow velocity within seconds which lasted for up to 60 s, followed by (b) reduction of systolic antegrade flow velocity with an enhanced finger-tip phenomenon; and (c) early systolic retrograde flow was observed

Table 2 Coronary flow parameters measured by intracoronary Doppler

	Baseline	Hyperaemia
APV (cm . s^{-1})	19·0 ± 7·9	37·3 ± 15·1
MPV (cm . s^{-1})	43·2 ± 18·7	66·7 ± 27·9
DSVR	2·91 ± 1·41	2·02 ± 0·79
CFVR	—	2·03 ± 0·54

APV = average peak velocity; MPV = maximal peak velocity; DSVR = diastolic/systolic velocity ratio; CFVR = coronary flow velocity reserve.

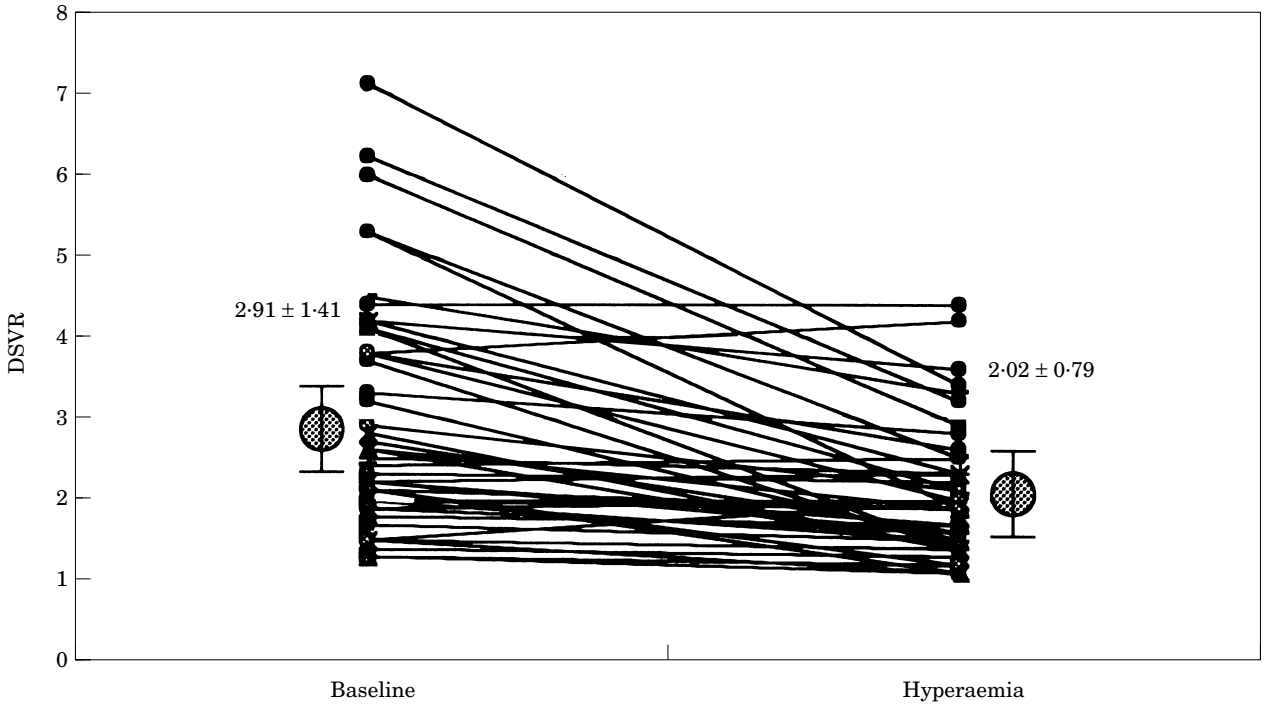

Figure 6 **Diastolic/systolic velocity ratio (DSVR) at baseline and in hyperaemia after intracoronary injection of 18 µg adenosine.**

in 37 patients (77%) in the segment proximal to the bridge segment with a mean flow velocity of $-22{\cdot}1 \pm 13{\cdot}2$ cm . s^{-1} (Fig. 8).

Discussion

New findings of myocardial bridging

In recent decades, angiography's demonstration of the milking effect, has been the only method to detect this phenomenon in vivo. The pathophysiological significance of this sign was not well understood. Intravascular ultrasound imaging is a new technique, allowing accurate assessment of vascular anatomy. Preliminary reports have shown that this technique is able to detect myocardial bridging and provide unique information concerning wall morphology in this condition[19–23]. Using intravascular ultrasound and the Doppler technique, we examined 14 patients with the angiographic milking effect and found a characteristic systolic compression of the bridge segments[19]. In this study, we have found, for the first time, a specific morphological sign (half moon phenomenon) surrounding the bridge segment (Fig. 2). The presence of the intravascular ultrasonic half moon phenomenon 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 or other coronary arteries. Sometimes, the typical angiographic milking effect is not obvious in patients with slight myocardial bridging. However, when the specific half moon phenomenon is demonstrated by IVUS, the milking effect can be provoked by intracoronary administration of nitroglycerin[22].

A reduction in coronary flow velocity reserve was demonstrated in patients with myocardial bridging using an intracoronary Doppler catheter[19] and intracoronary Doppler wire[23]. However, the flow velocity reserve in the proximal normal segment was significantly higher than that of distal[23]. Coronary flow velocity reserve seems to be impaired in patients with severe stenosis, in comparison to the patients without, but it failed to reach statistical significance. This indicates that myocardial bridging-induced haemodynamic disturbance is the main reason for myocardial ischaemia. The subsequent atherosclerotic involvement of the segment proximal to the bridge may worsen myocardial ischaemia.

With intracoronary Doppler wire, a characteristic flow pattern, showing an early diastolic prominent coronary flow velocity spike (sharp acceleration of flow in early diastole followed by immediate marked deceleration) was detected in most of the patients. This flow pattern mimics a finger tip, and hence, was regarded as an early diastolic 'finger-tip' flow pattern. Because of the high diastolic flow and reduced systolic flow, the diastolic/systolic velocity ratio is increased in comparison to the normal coronary arteries. These flow patterns, such as the diastolic finger-tip flow pattern no or decreased systolic antegrade flow, and increased diastolic/systolic velocity ratio can be explained by the systolic compression of the bridge segment and release

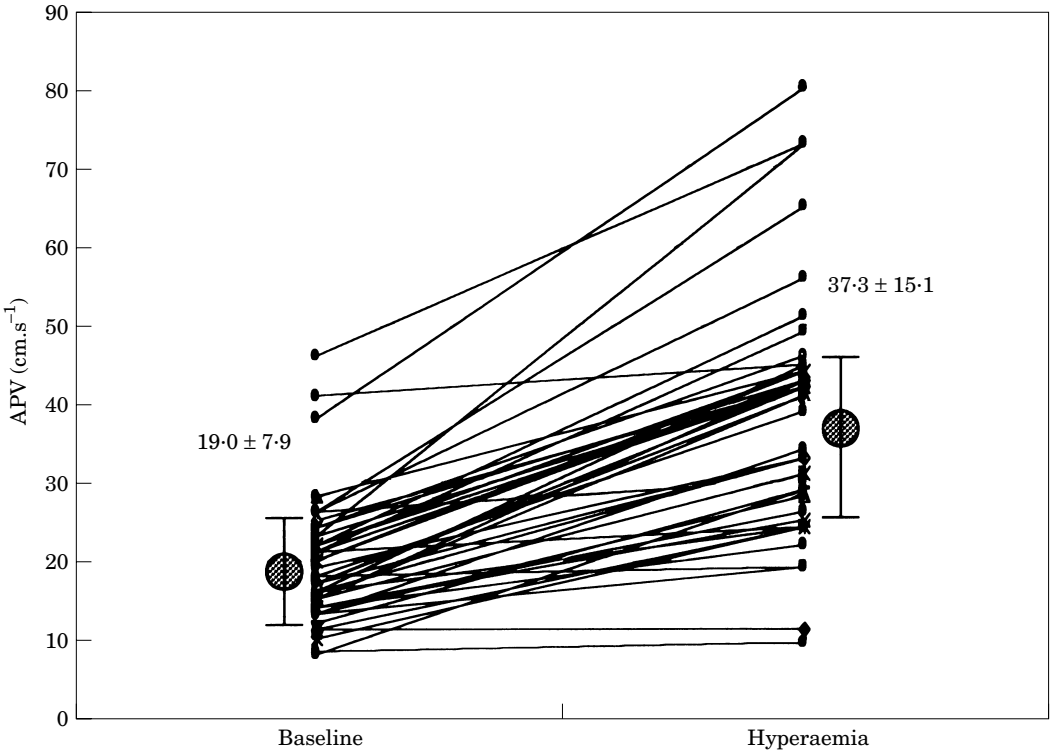

Figure 7 **Average peak flow velocity (APV) in the bridge segment at baseline and in hyperaemia. The ratio between APV in hyperaemia and at baseline is the coronary flow velocity reserve (CFVR). In this group of patients, CFVR is 2·03 ± 0·54.**

of the vascular lumen during early diastole. Due to compression of the vascular lumen during systole, this results in no or decreased antegrade flow that subsequently leads to an increased diastolic/systolic velocity ratio. At early diastole, release of the resistance vessels by intraventricular 'throttling' leads to reduction of resistance, as in normal subjects, resulting in an increase of antegrade flow. The early diastolic spike is most probably due to the antegrade coronary flow meeting the still compressed (delayed relaxation) narrow bridge segment. The subsequent sharp deceleration in coronary flow velocity results from compression release and an increase in the vascular lumen. After the release of the compression, the lumen of the bridge segment remains unchanged in the second half of diastole and therefore, corresponds to the plateau of the flow pattern at this phase[21,24].

Because of systolic squeezing of the bridge segment, reversing antegrade flow may occur during this phase. Therefore, systolic retrograde flow in proximal segments may register. This retrograde flow is induced or enhanced by nitroglycerin or orciprenalin, due to increased contraction of the myocardium covering the coronary artery[22,25]. In addition, the effects of nitroglycerin and orciprenalin are mainly in epicardial coronary arteries, causing the epicardial artery to dilate; a more obvious pressure drop in the proximal segment may also contribute to the retrograde flow. However, absence of the finger tip phenomenon was found in 13% of the patients. This absence might be because compression of the bridging segment was not severe enough to induce the haemodynamic disorder that leads to the formation of the finger-tip. This, from another aspect, indicates that the morphological half-moon phenomenon, as detected by intravascular ultrasound, is more sensitive than the finger-tip phenomenon, as demonstrated by intracoronary Doppler, in identifying myocardial bridging.

A glimpse into atherogenesis from myocardial bridging

In our intravascular ultrasound study, there was a high incidence of atherosclerotic involvement (88%) in the segment proximal to the bridge, but plaque formation was found in none of the bridge segments. Myocardial compression induced flow disturbance in this study and pressure disturbance in our previous study[24]. This strongly supports the hypothesis that endothelial injury is the main reason for atherosclerosis in these patients. The lower pressure in the bridge segment may have had the effect of preventing endothelial cell injury and therefore there was no plaque in the bridge or distal segments. These findings are consistent with the pathological observations that atherosclerotic lesions seldom involve the bridge segment itself[26–28].

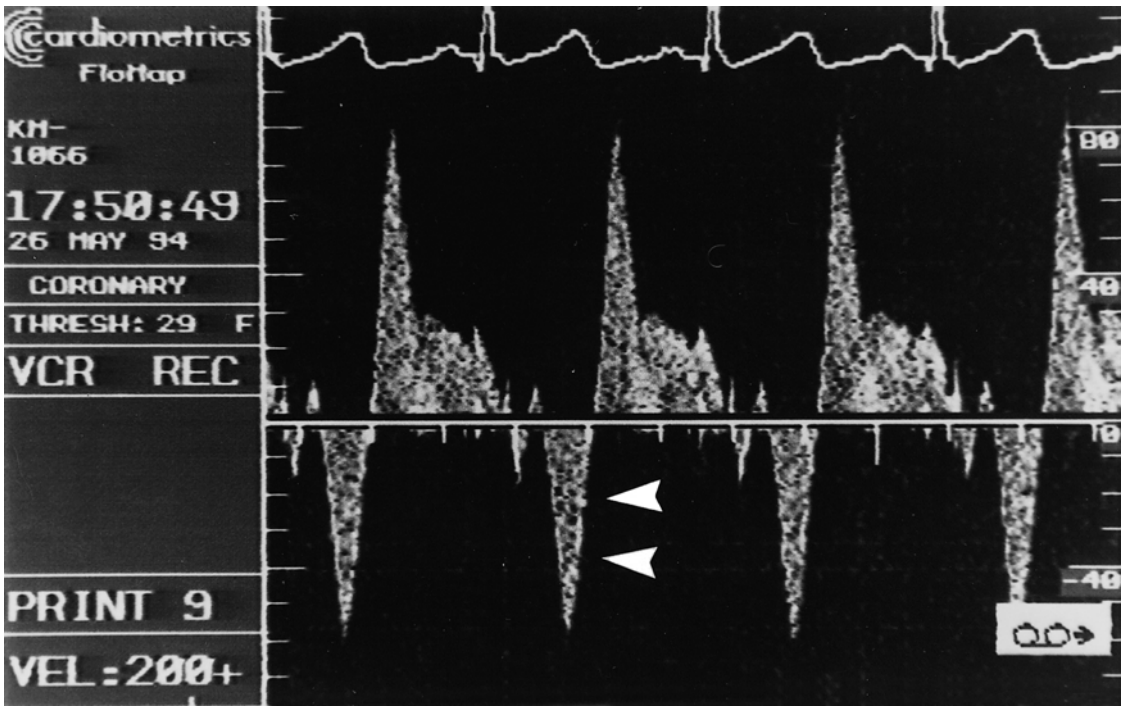

Figure 8 **Retrograde flow during systole (arrows) in the proximal segment of the bridge after nitroglycerin provocation. A typical diastolic 'finger tip' phenomenon is also seen during diastole. Scale in cm . s^{-1}.**

Clinical implication

As indicated above, myocardial bridging may induce myocardial ischaemia[2–5], myocardial infarction[6–9], conduction disturbances[10], and sudden death[11–13]. Identification of the presence and evaluation of the severity of myocardial bridging is of clinical importance. Studies have suggested that tachycardia may worsen ischaemia because of the reduction of the duration of diastolic coronary filling[2,29,30]. Agents such as nitroglycerin and dipyridmole will enhance the pressure gradient between the bridge and proximal segments by vasodilation and reduction of the proximal pressure, resulting in enhancing retrograde flow. In addition, an increase in heart rate may lead to a reduction in the duration of diastolic filling. A beta-agonist, such as orciprenalin, will enhance vessel compression, in addition to increasing heart rate. Both effects will lead to myocardial ischaemia. Therefore, it has been suggested that a beta-blocking agent maybe of help in these patients[31] and coronary vasodilators should be avoided[32]. Schwarz *et al.* demonstrated that short-acting beta-blockers, during atrial pacing, can alleviate anginal symptoms and signs of ischaemia[30]. Recently, it has been shown that intracoronary stenting may be a new approach in improving flow disturbance in patients with myocardial bridging[33]. However, we were frustrated by the long-term result of a patient in whom a stent was implanted in the bridge segment because of a severe dissection after PTCA of a stenosis in the segment proximal to the bridge[34]. The patient was re-admitted 6 months later because of recurrent angina. Coronary angiography and intravascular ultrasound showed a restenosis in the previous bridge segment, which was supposed to have been protected by the presence of myocardial bridging[26–28]. Because of the implantation of the stent, intima hyperplasia had occurred over the stent.

Clinical symptoms relate to disturbed coronary blood flow. Improvement of intracoronary haemodynamics can be assessed by normalization of the coronary flow profile[33], namely the disappearance of retrograde flow in the proximal segment, abolition of the finger-tip phenomenon, and normalization of DSVR. As to which method (medical treatment, bridge stenting, or surgical release of the bridge) is the best method of releasing bridging compression remains to be studied by long-term follow up.

Conclusion

Myocardial bridging is characterized by the following signs, which can be identified by intravascular ultrasound and Doppler: ultrasonic, a specific echolucent half moon phenomenon around the bridge segment which exists throughout the cardiac cycle; systolic compression of the bridge segment, using intracoronary FloWire®, and the diastolic finger-tip phenomenon; reduced antegrade systolic flow or retrograde systolic flow in the proximal segment, and reduced DSVR.

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