Coronary arteries that tunnel through the myocardium are seen in as many as 40% to 80% of cases on autopsy; however, functional myocardial bridging is less commonly observed on angiography (0.5% to 16.0%) and can range from 4 to 80 mm in length (1–4). Although myocardial bridges can be found in any epicardial artery, 67% to 98% occur in the left anterior descending coronary artery (LAD) (5,6). Bridges have been described as superficial or deep on the basis of 3 observations: 1) they range from 0.3 to 28 mm in depth (4,5); 2) anatomically they consist of either superficial myocardial fibers that traverse over the LAD or deep fibers that encircle the LAD (5,7); and 3) bridges >5 mm deep are less amenable to surgical myotomy (8). The hemodynamic impact of myocardial bridging depends on the thickness and length of the bridge, the orientation of the bridge relative to myocardial fibers, and the presence of loose connective or adipose tissue around the bridged segment.

**Pathophysiology**

Autopsy and intravascular ultrasound studies have shown that the intramural and distal segments of bridged vessels remain free from atherosclerotic disease while the proximal segment of the vessel is prone to developing atherosclerosis (9,10). Biomechanical forces may explain these observations. At the entrance of a myocardial bridge, fluid mechanics play an important role in plaque formation because disturbed near-wall blood flow patterns are a central factor in the spatial distribution of atherosclerosis (11,12). Low and oscillatory wall shear stress (WSS) are associated with increased expression of vascular cell adhesion molecule 1 (11,13) and reactive oxygen species production (14) as well as the development of a proatherogenic endothelial cell phenotype (12). Indeed, autopsy studies have shown that coronary segments immediately proximal to myocardial bridges, where WSS is low, have structurally dysfunctional, flat and polygonal endothelial cells, whereas endothelial cells lining bridged segments, where WSS is physiologic or high, are structurally intact (15). Clinical studies in patients with mild atherosclerosis but without bridging have shown greater plaque progression in segments with low WSS compared with physiological or high WSS (16). In a case-control series comparing patients who had bridging with control patients (17), the wall shear rate, which is the velocity gradient perpendicular to the wall, was found to be lower proximal to the bridge compared with within the bridge.

**Figure 1** shows a computational fluid dynamics model at end-systole of the LAD in a patient with a symptomatic myocardial bridge revealing an area of relatively low WSS proximal and distal to the bridge and high WSS within the bridge. Enhanced myocardial compression at the bridge...
entrance also results in abrupt breakage of the propagating antegrade systolic wave, disrupting blood flow patterns, exacerbating the low WSS, and intensifying endothelial injury and the stimuli for plaque formation (18). Another proposed mechanism of plaque formation proximal to a myocardial bridge involves solid mechanical forces that result from the motion and deformation of the coronary tree and myocardial material properties. Specifically, compression within the bridge and severe vessel angulation at the junction of the bridge result in a heterogeneous stress field in the proximal segment. The induced stresses are hypothesized to be conducive to plaque development and possible fissuring in the proximal segments (18).

Within the bridge, increased mechanical loads likely contribute to constrictive vascular remodeling as an attempt to restore loads to homeostatic levels (19). These mechanisms are amplified with diastolic dysfunction that occurs with left ventricular hypertrophy. In addition, separation of the bridged segment from perivascular adipose tissue in the epicardium that is associated with proinflammatory cytokines and adipokines may be a protective mechanism against the development of atherosclerosis (20). These factors likely contribute to plaque formation proximal to myocardial bridges and exert an atheroprotective role within the bridge. The relative lack of atherosclerosis observed distal to myocardial bridges despite the presence of low WSS is not well understood. Clearly, complex and dynamic biomechanical factors influence the blood flow within and at the exit of the bridge that in aggregate appear to attenuate the proatherosclerotic stimulus of low WSS observed distal to the bridge.

Additional pathophysiological changes can induce symptoms of myocardial ischemia in previously asymptomatic patients (Fig. 2). First, increasing left ventricular diastolic dysfunction associated with aging, hypertension, and coronary atherosclerosis can exacerbate the supply-demand mismatch imposed by the bridge. Second, development of left ventricular hypertrophy can increase compression and reduce the coronary microvascular reserve. Third, coronary vasospasm, microvascular dysfunction, or endothelial dysfunction related to cardiovascular risk factors combined with the bridge can result in myocardial ischemia. Fourth, plaque development proximal to the bridge...

![Figure 1](https://example.com/figure1.png)

**Figure 1** Relative WSS Profile of the LAD in the Context of Myocardial Bridging

Relative WSS profile of a 3-dimensional angiographically reconstructed LAD during systole from a patient with myocardial bridging. Coronary segments proximal and distal to the myocardial bridge show relatively low WSS compared with the bridged segment. LAD = left anterior descending coronary artery; S1 = first septal branch; WSS = wall shear stress. Image created by Craig Skaggs.

![Figure 2](https://example.com/figure2.png)

**Figure 2** Schematic Diagram of the Effects of Aging on the Myocardial Bridge

(A) Heart with myocardial bridging, early stage. (B) Longitudinal view of the bridged vessel. (C) Cross-sectional view of the vessel in the middle of the myocardial bridge. (A') Heart with myocardial bridging, late stage, with ventricular hypertrophy and diastolic dysfunction. (B') Longitudinal view of the bridged vessel, with hypertrophied muscle and plaque progression proximal to the bridge. (C') Cross-sectional view of the vessel in the middle of the myocardial bridge showing hypertrophied muscle and negative remodeling of the vessel with decreased lumen diameter. Images created by Clare Wang and Craig Skaggs.

### Abbreviations and Acronyms

- **BMS** = bare-metal stent(s)
- **CABG** = coronary artery bypass grafting
- **DES** = drug-eluting stent(s)
- **FFR** = fractional flow reserve
- **LAD** = left anterior descending coronary artery
- **MSCT** = multiple-slice computed tomography
- **PCI** = percutaneous coronary intervention
- **TVR** = target vessel revascularization
- **WSS** = wall shear stress
can augment coronary obstruction by the bridge. Fifth, the negative remodeling within the bridge can reduce myocardial flow. Each of these factors can contribute to a varying degree to the development of symptoms in patients with myocardial bridging.

More recently, it has been recognized that myocardial ischemia is not purely related to systolic vascular compression. Indeed, systolic vessel compression has been shown to persist into mid-to-late diastole (2). The hemodynamic disturbance imposed by this persistent diastolic luminal narrowing was corroborated by increases in both average peak flow velocity and average diastolic peak flow velocity, with only minor changes in systolic blood flow within the bridged segment of the coronary artery. These data suggest that both systolic and diastolic flow impairment contribute to myocardial supply-demand mismatch in patients with myocardial bridging.

Clinical Presentation

Although myocardial bridging can be an incidental finding on angiography or autopsy, symptomatic patients who have

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Diagnostic Modalities for Myocardial Bridging</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnostic Technique</strong></td>
<td><strong>Diagnostic Sign</strong></td>
</tr>
<tr>
<td>Quantitative coronary angiography</td>
<td>Milking effect</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Intravascular ultrasound</td>
<td>Half-moon</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracoronary Doppler measure (with pharmacological infusion)</td>
<td>Fingertip</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Intracoronary pressure measure (with pharmacological infusion)</td>
<td>Hemodynamic limitation (FFR &lt;0.75 to 0.8)</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple-slice computed tomography</td>
<td>Completely or partially surrounded coronary segment by myocardium on axial and multiplanar reformatted images</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Contrast stress echocardiography</td>
<td>Reversible perfusion defects in angiographically normal arteries</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**FFR** — fractional flow reserve.
myocardial bridges as their only cardiac abnormality may present with myocardial ischemia (21), acute coronary syndromes (22–24), coronary spasm (21,25), exercise-induced dysrhythmias such as supraventricular tachycardia (24), ventricular tachycardia (26,27) or atrioventricular conduction block (28), myocardial stunning (29), transient ventricular dysfunction (30), syncope (24,27), or even sudden death (31,32).

Diagnosis

A number of diagnostic modalities have been used to investigate the anatomic and physiological significance of myocardial bridging (Table 1). Because of the lack of a true gold standard for diagnosing myocardial bridging, the reported diagnostic accuracies are variable.

**Noninvasive diagnostic techniques.** Multiple-slice computed tomography (MSCT), stress single-photon emission computed tomography, and stress echocardiography have been used in the diagnosis of myocardial bridging. MSCT defines bridges as segments surrounded by myocardium (33). Recent developments allowing for physiological assessment by MSCT may enhance its diagnostic utility for identifying hemodynamically significant bridges. Stress single-photon emission computed tomography can detect reversible myocardial perfusion defects in patients with myocardial bridging and relate the amount of ischemia to the degree of systolic luminal narrowing (34,35). Contrast stress echocardiography has been used for detection of myocardial bridging but is not as well validated (36).

**Invasive diagnostic techniques.** On angiography, diagnosis depends on the change in diameter between systole and diastole within the bridged coronary segment. A significant “milking effect” (Fig. 3) is present when there is ≥70% reduction in minimal luminal diameter during systole and persistent ≥35% reduction in minimal luminal diameter during mid-to-late diastole (2). Systolic narrowing at the bridge can be accentuated by intracoronary injection of nitroglycerin by vasodilating adjacent nonbridged coronary segments (Fig. 4) (9,37).

Adjunctive intravascular imaging and physiology can contribute to our clinical evaluation and understanding of the complex pathophysiology of bridging. On
intravascular ultrasound the characteristic finding is the “half-moon” sign, an echolucent area present only between the bridged coronary segment and epicardial tissue that persists throughout the cardiac cycle (Fig. 5) (9). Additionally, intravascular ultrasound can characterize subangiographic atherosclerosis proximal to bridges.

Coronary physiological measurements across a myocardial bridge during pharmacological infusion can be valuable for:

Figure 6 Fingertip Phenomenon During Intracoronary Doppler Measurements

(A) Example of a normal flow pattern. (B) Example of the “fingertip” phenomenon, a characteristic velocity profile showing abrupt early diastolic acceleration, rapid mid-diastolic deceleration, and mid-to-late diastolic plateau.

Figure 7 Intracoronary Hemodynamics From a Patient With Myocardial Bridging

Coronary blood velocity (blue tracing), proximal pressure (Pa) (red tracing), and distal pressure (Pd) (yellow tracing) measurements from a patient with myocardial bridging. Blue circles indicate the portions of the tracings magnified above each circle. (A) Adenosine (140 μg · kg⁻¹ · min⁻¹); FFR = 0.83, APV = 28 cm/s; HR = 96 beats/min. (B) Dobutamine (60 μg · kg⁻¹ · min⁻¹); FFR = 0.82, APV = 38 cm/s; HR = 122 beats/min. APV = average peak blood velocity; FFR = fractional flow reserve; HR = heart rate.
1) evaluation of the hemodynamic significance of fixed obstruction associated with the bridge; 2) simulation of dynamic myocardial obstruction that could contribute to ischemic symptoms; and 3) unmasking concomitant endothelial dysfunction or coronary vasospasm within the bridged segment that could also be clinically relevant. The bridged segment produces a distinctive flow velocity called the “fingertip” phenomenon (Fig. 6). The abrupt acceleration in velocity in early diastole results from a decrease in distal microvascular resistance as the myocardium untwists during isovolumetric relaxation concomitant with continuing myocardial compression of the bridged coronary segment. A rapid deceleration in velocity ensues as the bridge muscle relaxes and the cross-sectional area of the lumen increases. The velocity plateaus when the artery has fully reopened (1,18).

For the evaluation of hemodynamically significant stenoses, fractional flow reserve (FFR) can be measured (38). A patient with a myocardial bridge with an FFR <0.75 likely has ischemia associated with that bridge. As in nonbridged patients, there is a gray zone of ischemia with an FFR of 0.75 to 0.80. For a patient with an abnormal but nonischemic FFR (>0.80), intravenous administration of dobutamine can lead to higher pressure gradients (and sometimes ventricularization of the distal pressure tracing) and reproduction of angina symptoms, which would then suggest a clinically significant myocardial bridge (39). Higher average peak velocity and greater pressure gradients with infusion of dobutamine compared with adenosine suggest a hemodynamically significant myocardial bridge (Fig. 7A and B). Finally, vasoconstriction with intracoronary acetylcholine infusion (off-label use) can unmask concomitant endothelial dysfunctional or coronary vasospasm.

**Classification.** The Schwarz classification (Table 2) can serve as a guide for directing therapy for patients with myocardial bridging because it has been linked to clinical outcomes after pharmacological and invasive interventions (40). Patients with Schwarz type A need no treatment, whereas patients with types B and C show significant symptomatic improvement with beta-blockers or calcium channel blockers at 5-year follow-up. Patients with Schwarz type C refractory to medical therapy may be considered for revascularization of the myocardial bridge.

### Management

Treatment of symptomatic patients with myocardial bridging consists primarily of pharmacological therapy, although percutaneous coronary intervention (PCI), myotomy, or coronary artery bypass grafting (CABG) can be considered for selected patients refractory to maximal medical therapy.

**Pharmacological therapy.** Aggressive risk factor modification is advocated and antiplatelet therapy should be considered in patients with myocardial bridging because they are at increased risk for developing atherosclerosis. One approach to individualizing the need for antiplatelet therapy would be to perform MSCT to identify subclinical atherosclerosis. For symptomatic patients, beta-blockers remain the mainstay of treatment and relieve the hemodynamic disturbance caused by the myocardial bridge by decreasing the heart rate, increasing the diastolic coronary filling period, and decreasing contractility and compression of the coronary arteries (2,41). Calcium channel blockers are also frequently used and, in addition to the aforementioned pharmacological effects of beta-blockers, may have vasodilatory effects that might be beneficial in

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Schwarz Classification for Myocardial Bridges and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schwarz Type</td>
<td>Criteria</td>
</tr>
<tr>
<td>A</td>
<td>Incidental finding on angiography</td>
</tr>
<tr>
<td>B</td>
<td>Ischemia on stress test</td>
</tr>
<tr>
<td>C</td>
<td>Altered intracoronary hemodynamics (quantitative coronary angiography/coronary flow reserve/Doppler)</td>
</tr>
</tbody>
</table>

**Table 3**

<table>
<thead>
<tr>
<th>First Author, Year (Ref. #)</th>
<th>Study Cohort</th>
<th>Intervention</th>
<th>Follow-Up Period</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Klues et al., 1997 (42)</td>
<td>MB (n = 3)</td>
<td>BMS</td>
<td>7 weeks</td>
<td>No ISR or MACE</td>
</tr>
<tr>
<td>Haager et al., 2000 (45)</td>
<td>MB (n = 11)</td>
<td>BMS</td>
<td>2 yrs</td>
<td>45% ISR (7 weeks)</td>
</tr>
<tr>
<td>Kurshaligoli et al., 2004 (46)</td>
<td>MB (n = 12)</td>
<td>BMS</td>
<td>6 months</td>
<td>ISR 67% in MB vs. 28% in non-MB</td>
</tr>
<tr>
<td>Kunamneni et al., 2008 (47)</td>
<td>MB (n = 12)</td>
<td>4 BMS 8 DES</td>
<td>1 yr</td>
<td>ISR 75% in BMS vs. 25% in DES</td>
</tr>
<tr>
<td>Tsujita et al., 2009 (48)</td>
<td>MB (n = 70)</td>
<td>4 BMS 66 DES</td>
<td>1 yr</td>
<td>MB stent group: 33% MACE Non-MB stent group: 11% MACE</td>
</tr>
<tr>
<td>Ernst et al., 2013 (43)</td>
<td>MB (n = 15)</td>
<td>DES</td>
<td>5 yrs</td>
<td>1 perforation during stent implantation 19% ISR (6 months)</td>
</tr>
</tbody>
</table>

BB — beta-blocker; CCB — calcium channel blocker.
patients with concomitant vasospasm. Head-to-head comparisons of beta-blockers and calcium channel blockers or randomized clinical trials of outcome benefits of beta-blockers are not available.

In contrast, pure vasodilating agents such as nitroglycerin should be used cautiously in patients with myocardial bridges. Although nitrates have antispasmodic properties and can decrease pre-load, they can worsen symptoms by intensifying systolic compression of the bridged segment and vasodilating segments proximal to the bridge (Fig. 4), thereby exacerbating retrograde flow in the proximal segment and reducing the myocardial ischemic threshold (9,37). Vasodilators should thus be avoided unless there is significant coexisting coronary vasospasm.

**Percutaneous coronary intervention.** Stent implantation in symptomatic patients with myocardial bridges can ameliorate peak intracoronary systolic pressure and vessel compression, normalize flow, and abolish symptoms (42); however, concerns regarding perforation during stent deployment (21,43), stent fracture (44), in-stent restenosis (44–48), and stent thrombosis (49) have limited their use in this condition. Investigations focusing on in-stent restenosis are summarized in Table 3 and suggest 2 conclusions: 1) stent implantation in patients with symptomatic myocardial bridges results in high rates of early in-stent restenosis that may be related to bridge-associated decreased lumen area; and 2) compared with PCI with bare-metal stents (BMS), PCI with drug-eluting stents (DES) has lower rates of target vessel revascularization (TVR).

Higher rates of restenosis were shown in patients undergoing PCI with BMS for symptomatic isolated myocardial bridging in one prospective study of 11 patients that reported early in-stent restenosis requiring TVR in 4 patients (45) and in another investigation comparing a similar cohort of 12 patients with 39 patients who underwent implantation of BMS for atherosclerotic lesions in the LAD (46). Although implantation of DES results in lower TVR rates than implantation of BMS, restenosis still occurs more frequently with PCI for symptomatic myocardial bridging than with PCI for atherosclerotic lesions. A small study that compared implantation of DES (n = 8) with BMS (n = 4) in symptomatic patients refractory to maximal medical therapy reported lower TVR rates in the DES group than in the BMS group, but both groups had higher rates than historical controls (47). Another investigation evaluated PCI with predominantly DES in 70 patients with both myocardial bridges and LAD lesions and divided them into 2 cohorts depending on whether the implanted stents ended proximal to a myocardial bridge or extended into the bridged segment. The TVR rate was significantly higher in patients with stents extending into the bridge compared with patients with stents ending proximal to the myocardial bridge (29% vs. 3%) (48). Interestingly, the minimum stent cross-sectional area was also significantly smaller for stents extending into the bridged segment as opposed to those that ended proximal to the bridge (4.8 mm² vs. 5.8 mm²). A recent prospective study of PCI with DES for symptomatic isolated myocardial bridging reported 3 of 15 patients requiring revascularization within 6 months post-procedure but no further complications (43).

Any rationale for PCI in selected patients with a myocardial bridge would be to treat plaque proximal to the bridge as well as the negative remodeling and dynamic obstruction within the bridged segment. Although...
Table 4  Studies of Surgical Interventions for Myocardial Bridging

<table>
<thead>
<tr>
<th>First Author, Year (Ref. #)</th>
<th>Study Design</th>
<th>Procedure</th>
<th>Follow-Up Period</th>
<th>Immediate Post-Operative Results</th>
<th>Follow-Up Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iversen et al., 1992 (50)</td>
<td>Retrospective</td>
<td>Myotomy</td>
<td>In-hospital</td>
<td>2 patients with right ventricular perforation All patients survived operation Post-operative studies showed flow restoration</td>
<td>None</td>
</tr>
<tr>
<td>Rezayat et al., 2006 (51)</td>
<td>Retrospective</td>
<td>Myotomy</td>
<td>7-81 months (mean 34.2 months)</td>
<td>1 patient had post-operative angina with angiography showing narrowing in the left anterior descending coronary artery and subsequently underwent CABG with LIMA graft</td>
<td>No MACE</td>
</tr>
<tr>
<td>Wan and Wu, 2005 (54)</td>
<td>Retrospective</td>
<td>4 PCI with BMS 8 CABG 16 myotomy</td>
<td>6-75 months (mean 23.5 months)</td>
<td>No complications</td>
<td>2 of 4 patients who underwent PCI had ISR; one subsequently underwent CABG No MACE in the surgical groups</td>
</tr>
<tr>
<td>Wu and Xu, 2007 (4)</td>
<td>Retrospective</td>
<td>16 CABG 15 myotomy</td>
<td>3-115 months (mean 31 months)</td>
<td>1 patient with right ventricular perforation was successfully converted to CABG</td>
<td>21 of 31 patients (11 CABG, 10 myotomy) underwent follow-up angiography showing restoration of flow No MACE</td>
</tr>
<tr>
<td>Huang et al., 2007 (55)</td>
<td>Retrospective</td>
<td>8 CABG with LIMA graft 3 myotomy</td>
<td>6-120 months (median 35.3 months)</td>
<td>1 patient with right ventricular perforation was successfully converted to CABG</td>
<td>2 patients experienced atypical chest pain and were treated medically No MACE</td>
</tr>
<tr>
<td>Sun et al., 2012 (82)</td>
<td>Retrospective</td>
<td>CABG with LIMA graft</td>
<td>24-55 months</td>
<td>No complications</td>
<td>Patients were Canadian Cardiovascular Society class 0 or 1 7 patients underwent cardiac computed tomography angiography at 1 yr, no stenoses No MACE</td>
</tr>
<tr>
<td>Bockeria et al., 2013 (53)</td>
<td>Retrospective</td>
<td>CABG 19 with SVG 20 with LIMA graft</td>
<td>LIMA graft: 6-23 months SVG: 2-26 months</td>
<td>2 patients underwent repeat sternotomy for bleeding 2 patients required inotropes</td>
<td>6 of 39 patients had recurrent angina Angiography at 12 months showed occlusions in 12 LIMA grafts and 3 SVGs No mortality</td>
</tr>
</tbody>
</table>

CABG — coronary artery bypass grafting; LIMA — left internal mammary artery; PCI — percutaneous coronary intervention; SVG — saphenous vein graft; other abbreviations as in Table 3.

contemporary metallic stent platforms can provide sufficient scaffolding to achieve adequate diastolic and systolic flow, sustained stress over time may result in stent fracture, restenosis, or thrombosis. Concerns have also been raised about the radial strength of bioabsorbable stents. Future bioabsorbable scaffolds could be designed with sufficient radial strength to safely achieve greater acute luminal gain in the intramyocardial artery while withstanding the systolic compression pressure during the bioabsorption phase, which after resorption could leave behind a much larger lumen supported by a residual thin fibrous endoluminal layer. Whether scaffolds with these biomechanical properties can be developed and withstand the scrutiny of angiographic and outcome studies remains to be seen.

Taken together, although there are no randomized controlled trials of optimal medical therapy versus optimal medical therapy and contemporary PCI with DES, medical therapy appears to be superior to PCI. Ischemia-guided revascularization using DES may be considered on a case-by-case basis for symptomatic patients refractory to maximal medical therapy and who are not optimal surgical candidates.

Surgical treatment. Surgical intervention involves either supra-arterial myotomy or CABG. In a typical myotomy case (Fig. 8), the cardiac muscle is dissected carefully and completely. Potential complications of myotomy include wall perforation, ventricular aneurysm formation, and post-operative bleeding. Conversely, the major concern of CABG with regard to myocardial bridges is graft failure.

Studies investigating the effectiveness of myotomy or CABG in patients with symptomatic bridging refractory to medical therapy are summarized in Table 4. Two retrospective studies of myotomy described overall successful operations; however, 1 series reported accidental right ventricular wall perforation in 2 of 9 patients (50), and the other study reported that 1 of 26 patients underwent CABG for post-operative angina with LAD narrowing (51). Regarding CABG, one investigation reported no complications (52) and the second described 6 of 39 patients with recurrent angina and 15 of 39 patients with...
graft occlusions on follow-up (53). Grafting with the left internal mammary artery was more likely to result in occlusion compared with grafting with the saphenous vein (12 vs. 3 patients), leading the investigators to conclude that grafting with the saphenous vein was preferable. This is in contrast to a previous report recommending the left internal mammary artery as the preferred graft for CABG (4).

Investigations comparing the effectiveness between myotomy and CABG in patients with symptomatic myocardial bridges consist of 1 study of 31 patients (4,54) and an even smaller series of 11 patients (55). In the first investigation, 1 myotomy case was converted to CABG after accidental right ventricular wall perforation. Twenty-one of 31 patients (either myotomy or CABG) who underwent follow-up angiography had restoration of distal coronary blood flow (4). In the second study, 2 of 11 patients experienced atypical chest pain and were managed medically (55).

Although both myotomy and CABG are reasonable initial choices, it is unclear which procedure is superior. On the one hand, because myotomy attempts to correct the underlying pathology, it may be the treatment of choice for patients who have symptomatic myocardial bridging refractory to medical therapy, ≥75% systolic coronary compression on angiography, or evidence of myocardial ischemia or infarction (4). On the other hand, CABG is favored over myotomy in cases of extensive (>25 mm) or deep (>5 mm) myocardial bridges (the risk of myotomy can be considerable) or when the bridged coronary segment fails to decompress completely in diastole (myotomy is unlikely to correct the persistent diastolic compression) (4,8). Importantly, there are no randomized clinical trials comparing intensification of medical therapy with surgical intervention. These limited data suggest that surgical therapy, either myotomy or CABG, appears safe and effective in symptomatic patients with myocardial bridging refractory to medical therapy.

Conclusions

Patients with myocardial bridging are commonly encountered clinically and may present with exertional symptoms of myocardial ischemia, syncope, and even sudden death. An array of noninvasive and invasive diagnostic modalities that have shed light on the pathophysiology of myocardial bridging can be deployed to evaluate symptomatic patients. Medical therapy with beta-blockers and calcium channel blockers remain the mainstay of treatment. For select patients refractory to intensified medical therapy, surgical intervention, or less preferably PCI with DES, can be considered. Larger registries and randomized clinical trials are warranted to shed light on optimal strategies for patients with myocardial bridging refractory to medical therapy.

Acknowledgments

The authors thank Bill Gogas, MD, for his editorial suggestions.

Reprint requests and correspondence: Dr. Habib Samady, Emory University School of Medicine, 1364 Clifton Road, Suite F606, Atlanta, Georgia 30322. E-mail: hsamady@emory.edu.

REFERENCES


Key Words: coronary wall shear stress • intracoronary Doppler velocity and pressure • intravascular imaging • myocardial bridge • myotomy • percutaneous coronary intervention.