

MYOCARDIAL BRIDGING IN CHILDREN WITH HYPERTROPHIC CARDIOMYOPATHY — A RISK FACTOR FOR SUDDEN DEATH

ANJI T. YETMAN, M.D., BRIAN W. MCCRINDLE, M.D., CATHY MACDONALD, M.D., ROBERT M. FREEDOM, M.D.,
AND ROBERT GOW, M.B., B.S.

ABSTRACT

Background Myocardial bridging may cause compression of a coronary artery, and it has been suggested that myocardial ischemia may result. The clinical significance and prognostic value of myocardial bridging of the left anterior descending coronary artery in children with hypertrophic cardiomyopathy are unknown. We sought to determine the prevalence and clinical effects of myocardial bridging in children with hypertrophic cardiomyopathy who underwent cardiac catheterization.

Methods Angiograms from 36 children with hypertrophic cardiomyopathy were reviewed to determine whether myocardial bridging was present and, if so, to assess the characteristics of systolic narrowing of the left anterior descending coronary artery caused by myocardial bridging and the duration of residual diastolic compression. We also reviewed clinical data on these patients.

Results Myocardial bridging was present in 10 (28 percent) of the patients. Compression of the left anterior descending coronary artery persisted for a mean (\pm SD) of 50 ± 17 percent of diastole. As compared with patients without bridging, patients with bridging had a greater incidence of chest pain (60 percent vs. 19 percent, $P=0.04$), cardiac arrest with subsequent resuscitation (50 percent vs. 4 percent, $P=0.004$), and ventricular tachycardia (80 percent vs. 8 percent, $P<0.001$). On average, the patients with bridging had a reduction in systolic blood pressure with exercise of 17 ± 27 mm Hg, as compared with an elevation of 43 ± 31 mm Hg in those without bridging ($P<0.001$). The patients with bridging also had greater ST-segment depression with exercise (median, 5 vs. 0 mm, $P=0.004$) and a shorter duration of exercise (mean, 6.6 ± 2.4 vs. 9.1 ± 1.4 minutes, $P=0.008$). The degree of dispersion of the QT interval corrected for heart rate on the electrocardiogram was greater in patients with bridging than in those without bridging (104 ± 46 vs. 48 ± 31 msec, $P=0.002$). Kaplan–Meier estimates of the proportions of patients who had not died or had cardiac arrest with subsequent resuscitation five years after the diagnosis of hypertrophic cardiomyopathy were 67 percent among patients with bridging and 94 percent among those without bridging ($P=0.004$).

Conclusions Myocardial bridging is associated with a poor outcome in children with hypertrophic cardiomyopathy. Our observations suggest that bridging is associated with myocardial ischemia. (N Engl J Med 1998;339:1201-9.)

©1998, Massachusetts Medical Society.

SYSTOLIC compression of the left anterior descending coronary artery is a well-recognized angiographic phenomenon,¹⁻¹⁶ with a prevalence of 0.5 to 1.6 percent in the general population.^{2,3} Myocardial bridging with compression of an epicardial coronary artery^{7,17} occurs in 30 to 50 percent of adults who have hypertrophic cardiomyopathy.^{2,3,12-14,18,19} Descriptions of bridging in children are rare,^{8,20-22} and consequently its prevalence in normal children and those with hypertrophic cardiomyopathy is unknown.²⁰

The clinical significance of isolated myocardial bridges in the general population^{6,7,17,19,23-26} and in patients with hypertrophic cardiomyopathy^{12-15,27} remains controversial. Myocardial bridging in adults with hypertrophic cardiomyopathy may be associated with a higher incidence of sudden death from cardiac causes, cardiac arrest with subsequent resuscitation, myocardial wall-motion abnormalities, and perfusion defects on thallium-201 scintigraphy.^{13,28} The possibility that ischemia can lead to sudden death in children with hypertrophic cardiomyopathy has been suspected^{20,29,30} on the basis of findings of transmural myocardial infarction at postmortem examination,²⁸ the presence of ST-segment depression immediately before ventricular fibrillation on ambulatory monitoring,³¹ and the documentation of a higher incidence of perfusion defects on thallium scans in those with a history of serious cardiac events.^{20,32,33}

We investigated the prevalence and clinical significance of myocardial bridging of the left anterior descending coronary artery in children with a diagnosis of hypertrophic cardiomyopathy who had undergone cardiac catheterization and angiography.

METHODS

Patients

Between 1956 and 1997, 99 children with hypertrophic cardiomyopathy were identified at a single tertiary pediatric center. Hypertrophic cardiomyopathy was defined as the presence of a hypertrophied, nondilated ventricle in the absence of underlying cardiac or systemic secondary causes.³⁴ Fifty of these children underwent cardiac catheterization for various reasons, 36 of whom had angiograms available for review that were considered adequate for an assessment of myocardial bridging.

From the Department of Pediatrics, Division of Cardiology, Hospital for Sick Children, University of Toronto Faculty of Medicine, Toronto. Address reprint requests to Dr. Gow at the Children's Hospital of Eastern Ontario, 401 Smythe Rd., Ottawa, ON K1H 8L1, Canada.

Measurements

The medical records of all 50 children who underwent cardiac catheterization were reviewed to obtain data on demographic characteristics, symptoms, family history, management, and outcomes. The characteristics of the 36 patients with adequate angiograms were compared with those of the 14 patients without adequate angiograms. The 36 patients with adequate angiograms were the focus of our study. We reviewed the results of 12-lead electrocardiograms, Holter ambulatory recordings, and exercise tests performed at the last follow-up assessment before the patient received medical or surgical therapy. For each patient, the mean QT interval was corrected for heart rate (QTc) and QTc dispersion. QTc dispersion is the difference between the longest and the shortest QTc intervals measured on all 12 leads of the electrocardiogram and is a variable believed to correlate with the risk of sudden death.³⁵ All available Holter ambulatory recordings were reviewed for the presence of ischemic changes and ventricular ectopic activity. Associated symptoms were noted.

The data recorded for 24 patients during treadmill testing according to the Bruce protocol³⁶ included symptoms, peak heart rate and heart rate as a percentage of the predicted value, peak and change in systolic blood pressure, absolute duration of exercise and duration of exercise as a percentage of the predicted value,³⁶ and ST-segment changes. A subgroup of 10 patients underwent thallium scanning during exercise for the evaluation of angina, syncope, and resuscitation after cardiac arrest. Echocardiographic measurements of the dimensions of the left ventricular free wall, interventricular septum, and end-diastolic chamber, made within two months after angiography, were available for 31 of the 36 patients.

Angiographic Assessment of Myocardial Bridging

The reasons for cardiac catheterization included evaluation of symptoms, ventricular outflow obstruction, and the response to medical management and preoperative assessment. In all but three patients, cardiac catheterization was performed before we began to suspect the potential clinical effects of myocardial bridging.

For the purpose of this study, all angiograms were assessed retrospectively by two independent reviewers who were unaware of the patients' clinical status. When bridging was detected, the location of the compression and the maximal degree of dynamic narrowing were determined. The cross-sectional diameter of the narrowed segment in the projection that showed the most severe luminal reduction was compared with the diameter of the normal segment of the same vessel.¹³ A diagnosis of systolic compression of the left anterior descending coronary artery required the finding of a transient reduction of at least 50 percent in the diameter of the segment during systole. The percentage of diastole during which the left anterior descending coronary artery was compressed was calculated by dividing the number of angiographic frames during which the left anterior descending coronary artery remained narrowed by the total number of frames showing diastole.

Statistical Analysis

The characteristics of the study population are expressed as frequencies, medians with ranges, or means (\pm SD), as appropriate. The characteristics and outcomes of patients with myocardial bridging were compared with those without myocardial bridging with Fisher's exact test, Student's t-test, and Kruskal-Wallis analysis of variance. Kaplan-Meier estimates of the length of time from the diagnosis of hypertrophic cardiomyopathy to death or resuscitation after cardiac arrest were calculated and plotted for the total population. The log-rank and Wilcoxon tests were used to compare the time to death or to resuscitation after cardiac arrest in patients with myocardial bridging and those without myocardial bridging. All P values were two-sided, and P values of less than 0.05 were considered to indicate statistical significance.

RESULTS

Characteristics of the Patients

Of the 36 patients with angiograms judged adequate for the diagnosis of myocardial bridging, 29 (81 percent) were male and 7 (19 percent) were female. The mean (\pm SD) age at diagnosis of hypertrophic cardiomyopathy was 7.1 ± 5.8 years.

Symptoms at presentation and during follow-up included life-threatening cardiac events in 7 patients (19 percent), 6 of whom were resuscitated after cardiac arrest; syncope in 7 patients (19 percent); and chest pain in 11 patients (31 percent). There was a family history of hypertrophic cardiomyopathy in 15 patients (42 percent) and of sudden death in 9 patients (25 percent).

There were no significant differences between the 36 patients included in the study and the 14 patients who were excluded with respect to the incidence of symptoms (79 percent vs. 67 percent, $P=0.51$) and the incidence of cardiac arrest with subsequent resuscitation (17 percent vs. 7 percent, $P=0.66$), chest pain (31 percent vs. 21 percent, $P=0.73$), syncope (19 percent vs. 7 percent, $P=0.42$), and death (14 percent vs. 14 percent, $P=1.0$).

Myocardial Bridging

Myocardial bridging of the left anterior descending coronary artery was found on angiography in 10 of the 36 patients (28 percent). In all 10 patients the site of systolic compression was in the middle third of the left anterior descending coronary artery, just distal to the origin of the first diagonal branch. The degree of systolic narrowing was greater than 90 percent in all patients. The mean proportion of diastole during which the vessel remained compressed was 50 ± 17 percent. In three patients the bridging had been diagnosed during a clinical workup and was confirmed during blinded review for the study. In the other seven patients bridging was not diagnosed until the angiograms were reviewed for this study.

Factors Associated with Myocardial Bridging

Table 1 summarizes the characteristics of the study population. As compared with the patients without myocardial bridging, patients with myocardial bridging were significantly older at the time of diagnosis of hypertrophic cardiomyopathy ($P=0.04$) and were more likely to have chest pain ($P=0.04$) or cardiac arrest with resuscitation ($P=0.004$) than patients without myocardial bridging. The mean age at catheterization did not differ significantly between the groups (11.5 ± 3.2 vs. 8.3 ± 5.8 years, $P=0.11$), nor did the mean age at which they last underwent standard 12-lead electrocardiography before receiving any medical or surgical treatment for their cardiac condition (9.9 ± 4.0 vs. 7.8 ± 5.4 years, $P=0.27$).

The results of standard 12-lead electrocardiography

TABLE 1. CHARACTERISTICS OF THE PATIENTS ACCORDING TO THE PRESENCE OR ABSENCE OF MYOCARDIAL BRIDGING.*

VARIABLE	BRIDGING ABSENT (N=26)		BRIDGING PRESENT (N=10)		P VALUE
	VALUE	NO. OF PATIENTS†	VALUE	NO. OF PATIENTS	
Age at diagnosis of HCM — yr		26		10	
Median	3.3		11.2		0.04
Range	0.003–17.3‡		3.9–15.8		
Symptoms — no. (%)		26		10	
Cardiac arrest with resuscitation	1 (4)		5 (50)		0.004
Syncope	5 (19)		2 (20)		1.0
Chest pain	5 (19)		6 (60)		0.04
Family history — no. (%)		26		10	
HCM	9 (35)		6 (60)		0.26
Sudden death	6 (23)		3 (30)		0.69
QTc — msec§		26		10	
Maximal	449±45		484±40		0.10
Dispersion	48±31		104±46		0.002
Holter ambulatory ECG findings — no. (%)§		26		10	
Supraventricular tachycardia	5 (19)		3 (30)		0.66
Ventricular tachycardia	2 (8)		8 (80)		<0.001
ST-segment changes (ischemia)	9 (35)		7 (70)		0.08
Echocardiographic findings¶					
Ventricular septal thickness — mm	17±11	20	19±6	9	0.68
Ratio of ventricular septal thickness to LV-wall thickness	2.39±1.56	20	2.21±0.63	9	0.75
Systolic anterior MV motion — no. (%)	17 (74)	23	6 (67)	9	0.69
LV outflow gradient — mm Hg		25		9	0.92
Median	12		6		
Range	0–149		0–102		
Ejection fraction — %	77±16	16	72±7	6	0.41
Exercise testing					
Duration of exercise		13		7	
Minutes	9.1±1.4		6.6±2.4		0.008
% of predicted value	71±11		53±24		0.04
Maximal heart rate — % of predicted	92±8	15	84±8	7	0.10
Change in systolic blood pressure with exercise — mm Hg	+43±31	16	-17±27	7	<0.001
ST-segment depression with maximal exercise — mm		16		7	0.004
Median	0		5		
Range	0–4		0–10		
Cardiac catheterization					
LV end-diastolic pressure — mm Hg	14±7	20	19±7	8	0.09
LV outflow gradient — mm Hg		25		9	0.99
Median	2		0		
Range	0–149		0–10		

*Plus-minus values are means ±SD. HCM denotes hypertrophic cardiomyopathy, QTc QT interval corrected for heart rate, ECG electrocardiogram, LV left ventricle, and MV mitral valve.

†Data for some patients were missing.

‡The disease was diagnosed in one patient at one day of age.

§The data were obtained at the last follow-up assessment before any medical or surgical treatment was given.

¶Echocardiography was performed within two months after cardiac catheterization.

showed that patients with bridging had a tendency toward longer QTc intervals, with significantly greater QTc dispersion (P=0.002). A receiver-operating-characteristic curve showed that QTc dispersion above 60 msec distinguished patients with myocardial bridging from those without bridging with a sensitivity of 92 percent and a specificity of 77 per-

cent (Fig. 1). Ventricular tachycardia was noted on Holter ambulatory electrocardiographic monitoring in 10 patients. Eight of these patients had nonsustained monomorphic ventricular tachycardia (symptomatic in four patients), and two had symptomatic sustained monomorphic ventricular tachycardia. All six patients with symptomatic ventricular tachycardia

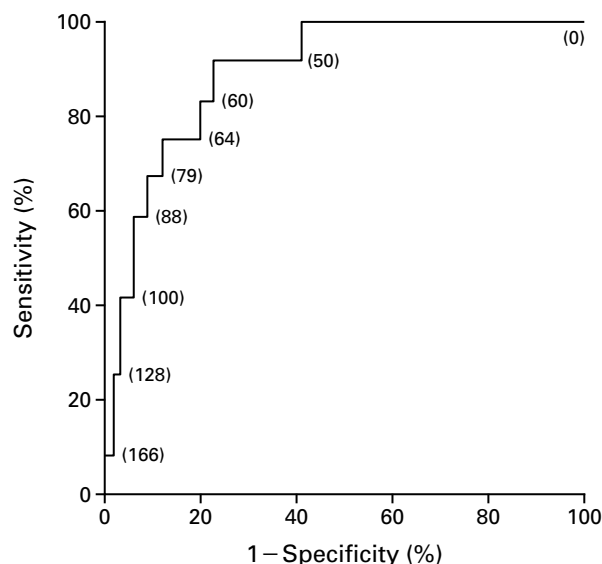


Figure 1. Receiver-Operating-Characteristic Curve for the Ability of the Dispersion of the QT Interval Corrected for Heart Rate (QTc) to Predict the Presence of Myocardial Bridging.

The numbers in parentheses represent the cutoff values of QTc dispersion in milliseconds at each point in the curve. QTc dispersion is calculated as the difference between the longest and the shortest QTc intervals on all 12 leads of the electrocardiogram.

had myocardial bridging. Echocardiographic features noted near the time of cardiac catheterization did not distinguish patients with myocardial bridging from those without bridging.

Patients with myocardial bridging did not differ significantly from those without myocardial bridging in terms of the mean age at which they last underwent exercise testing before receiving any medical or surgical treatment for their cardiac condition (12.6 ± 3.9 vs. 13.1 ± 3.2 years, $P=0.76$). In patients with bridging, however, the duration of exercise during exercise testing was significantly shorter ($P=0.008$), and the mean decrease in peak systolic blood pressure was associated with greater ST-segment depression.

All six patients with a decline in peak systolic blood pressure during exercise had myocardial bridging. In one patient with myocardial bridging, marked ST-segment depression and angina developed, followed by symptomatic ventricular tachycardia requiring cardioversion (Fig. 2). In addition, 6 of 10 patients who underwent thallium scanning during exercise had reversible perfusion defects. Four of these six patients had myocardial bridging, and in all four patients the distribution of the perfusion defect was consistent with the occurrence of compression of the middle third of the left anterior descending coronary artery. In contrast, the two patients with perfusion defects who did not have bridging had

more diffuse abnormalities in the inferior, anterior, and apical regions.

Mortality

The patients were followed until death or 19 years of age. One patient was lost to follow-up. The mean follow-up from the time of diagnosis of hypertrophic cardiomyopathy was 7.1 ± 5.4 years (range, 0 to 19.4). Two patients without bridging died suddenly. One of these patients had previously been resuscitated after cardiac arrest. Three patients with bridging died suddenly, one of whom had previously been resuscitated after cardiac arrest. Four other patients with bridging had episodes of cardiac arrest followed by resuscitation. Thus, 2 of the 26 patients without myocardial bridging (8 percent) and 7 of the 10 patients with myocardial bridging (70 percent, $P<0.001$) died or were resuscitated after cardiac arrest. One other patient with myocardial bridging presented with syncope associated with rapid atrial fibrillation, ST-segment depression, and elevated cardiac-enzyme levels consistent with the presence of ischemia.

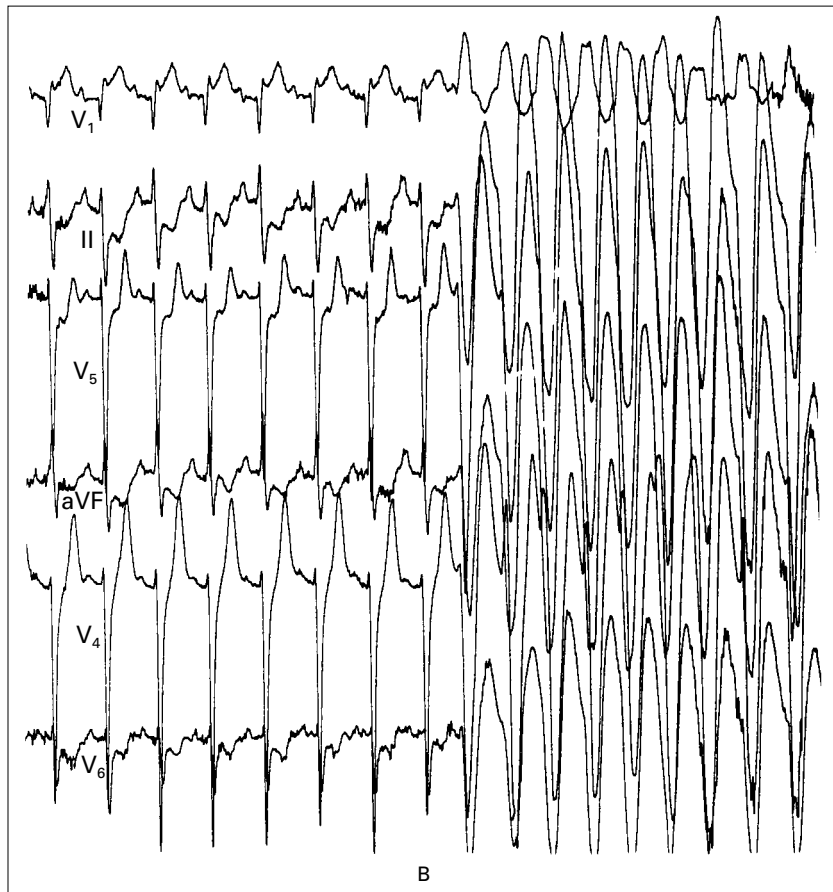
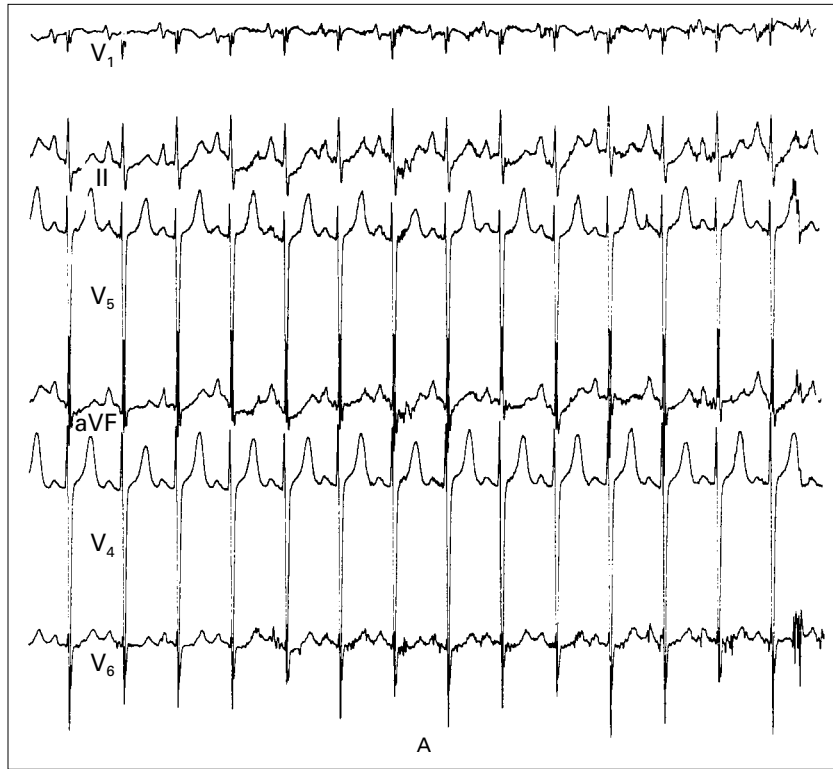
Of the nine patients who died or were resuscitated after cardiac arrest, five had an event that prompted investigation and diagnosis of hypertrophic cardiomyopathy (four patients with bridging and one without). When these five patients were excluded from the analysis, the Kaplan-Meier estimates of the proportions of patients who did not die or have cardiac arrest with subsequent resuscitation from the time of diagnosis of hypertrophic cardiomyopathy differed significantly between patients with bridging and those without bridging (Fig. 3). The five-year estimates were 67 percent in patients with bridging (95 percent confidence interval, 29 to 100 percent) and 94 percent in patients without bridging (95 percent confidence interval, 83 to 100 percent; $P=0.004$ by the log-rank test; $P=0.006$ by the Wilcoxon test).

Surgical Treatment

Nine patients underwent left ventricular myectomy, five received an automatic implantable cardioverter-defibrillator, and two underwent left and right ventricular myectomy. Three patients with myocardial

Figure 2. Electrocardiogram Obtained during Exercise Testing in a Patient with Myocardial Bridging and Induced Myocardial Ischemia and Ventricular Arrhythmia.

After nine minutes of the treadmill exercise test, the ST segments were normal (Panel A) and the patient's heart rate was 131 beats per minute. After 9 minutes and 36 seconds, the patient's heart rate was 141 beats per minute and the electrocardiogram showed acute elevation of the ST segment in lead V_1 and depression of the ST segment in leads II, aVF, V_5 , and V_6 , followed by rapid ventricular tachycardia and ventricular fibrillation (Panel B).



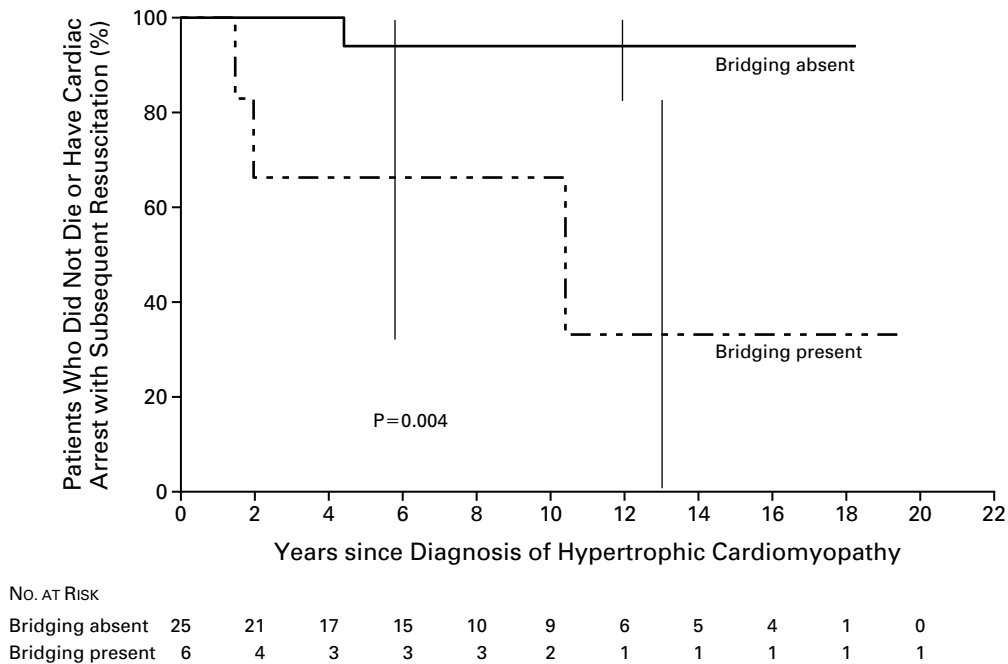


Figure 3. Kaplan–Meier Estimates of the Proportions of Patients Who Did Not Die or Have Cardiac Arrest with Subsequent Resuscitation from the Time of Diagnosis of Hypertrophic Cardiomyopathy, According to the Presence or Absence of Myocardial Bridging.

Five patients who had an event at presentation that led to the diagnosis of hypertrophic cardiomyopathy have been excluded (four with bridging and one without bridging). Vertical lines represent 95 percent confidence intervals. The P value was calculated with the log-rank test.

bridging underwent surgical division (unroofing) of the coronary artery. Unroofing was performed in conjunction with the implantation of a defibrillator in one patient, several years after the implantation of a defibrillator in one, and with a left ventricular myectomy in one. In two of these patients, thallium stress exercise tests showed reversible perfusion defects in the distribution of the middle third of the left anterior descending coronary artery before unroofing and a normal pattern of perfusion after unroofing. The third patient had a preoperative exercise study without perfusion imaging that showed ischemia-induced ventricular tachycardia (Fig. 2).

Before unroofing was performed, these patients had had numerous episodes of cardiac arrest with subsequent resuscitation. Two of these patients were asymptomatic six months and five years after unroofing was performed. The third patient had had numerous defibrillator discharges before unroofing was performed. After unroofing, the patient had one defibrillator discharge for either ventricular or sinus tachycardia. The results of an exercise perfusion study and coronary-artery angiography were normal in this patient after unroofing. Ventricular tachycardia was not induced during the electrophysiologic study, and prolonged high-rate atrial pacing did not unmask ischemia.

DISCUSSION

Myocardial bridging is marked by systolic compression of an epicardial coronary arterial segment by the overlying myocardium.³⁷ Angiographic evidence of compression of the left anterior descending coronary artery is found in 30 to 50 percent of adults with hypertrophic cardiomyopathy.¹⁴ The prevalence of this anomaly in our population of children with hypertrophic cardiomyopathy who underwent cardiac catheterization was similar to that seen in adults: 28 percent had near-oblivation of the left anterior descending coronary artery during systole. It is not clear why, on average, patients in our study who had bridging were older than patients without bridging when their hypertrophic cardiomyopathy was diagnosed. However, the two groups of patients were similar with respect to the age at which they underwent cardiac catheterization and received the potential diagnosis of bridging. Longitudinal angiographic studies would need to be done to determine whether the myocardial bridging was congenital or acquired and what factors are linked to the development of bridging. In this study, echocardiographic features associated with the severity of disease or with its complications were not associated with myocardial bridging.

Myocardial Ischemia

The association of myocardial ischemia with sudden death in young patients with hypertrophic cardiomyopathy has been recognized.^{20,29,31,38} The cause of the ischemia may be intramural abnormalities of the small vessels, abnormal myocellular architecture, or massive hypertrophy.²⁰ The role of myocardial bridging in ischemia and sudden death in patients with hypertrophic cardiomyopathy is controversial,¹⁴ because coronary perfusion normally occurs during diastole, and systolic compression is expected to have little effect on coronary flow. Our results, as well as data from adult patients with hypertrophic cardiomyopathy,¹³ demonstrate the existence of a diastolic time lag in which the previously compressed coronary vessel remains underfilled (up to 30 to 75 percent of diastole). The prolongation of the compression well into diastole, when the largest proportion of coronary blood flow normally occurs,³⁹ is likely to compromise myocardial perfusion. This prolongation may have a greater effect in young children, because the heart rate is faster and the diastolic filling time is shorter, especially during exercise. Our study provides evidence that ischemia associated with myocardial bridging is one cause of sudden death in children with hypertrophic cardiomyopathy. It should be noted that two of our patients who did not have myocardial bridging died suddenly or had episodes of cardiac arrest with subsequent resuscitation.

Ventricular Arrhythmias

Previous studies^{40,41} that demonstrated a low incidence of ventricular arrhythmias on ambulatory monitoring in children with hypertrophic cardiomyopathy have concluded that primary ventricular arrhythmias are probably not an important cause of death in this population. Myocardial damage resulting from chronic ischemia may cause diffuse fibrosis and increasing disarray of the myocardial fibers, which may secondarily create an arrhythmogenic substrate. We found a higher incidence of ventricular tachycardia on Holter ambulatory monitoring in patients with myocardial bridging, an observation that supports the role of ischemia in ventricular tachycardia. In addition, we documented ischemic ST-segment changes that culminated in ventricular tachycardia in a patient with myocardial bridging (Fig. 2).

QTc Dispersion

Variability between the electrocardiographic leads in measurements of QTc, known as QTc dispersion, is thought to reflect regional variations in myocardial recovery and excitability.⁴²⁻⁴⁴ Adults with hypertrophic cardiomyopathy have a greater degree of QTc dispersion than control subjects,⁴⁵ and patients who have ventricular arrhythmias or who die suddenly have a much greater degree of QTc dispersion

than patients without arrhythmias.³⁵ Increased QTc dispersion is believed to be an important risk factor for sudden death.⁴⁵ Regional variation in the time needed for myocardial recovery is increased in patients with ischemia after myocardial infarction and is linked to reentrant ventricular tachyarrhythmias.⁴⁶ The degree of QTc dispersion at rest has been reported to be greater in women with documented coronary artery disease than in those without coronary artery disease.⁴⁷ Exercise accentuated this difference, thus increasing the sensitivity of stress testing for detecting coronary artery disease in women. Our study found a greater degree of QTc dispersion in patients with myocardial bridging. These patients had a greater incidence of ischemia, which may lead to an arrhythmogenic substrate in association with abnormal and variable repolarization.

Exercise-Induced Hypotension

An abnormal blood-pressure response to exercise has been shown to be significantly associated with sudden death in adults with hypertrophic cardiomyopathy.⁴⁸⁻⁵⁰ Increasing left ventricular obstruction on exertion, altered diastolic filling, and abnormal systemic vascular tone have been implicated as potential causes.⁵¹⁻⁵³ More extensive myocardial perfusion defects have been documented in adult patients with abnormal blood-pressure responses to exercise.⁵⁴ We noted an abnormal blood-pressure response in all patients with myocardial bridging, an observation implicating ischemia as one cause of this hemodynamic derangement.

Surgical Treatment

Surgical unroofing of a myocardial bridge in patients with hypertrophic cardiomyopathy may lessen the incidence of ischemia and subsequent sudden death. Our experience suggests a beneficial effect of this surgical procedure, since unroofing reduced ischemia postoperatively in a small group of patients.

Limitations of the Study

The results of our study must be interpreted in the light of its limitations. The study sample was small, consisting of less than 50 percent of the children with hypertrophic cardiomyopathy presenting to our institution over a 41-year period. This sample may have been biased toward patients with systolic compression of the left anterior descending coronary artery, since such patients have a greater incidence of symptoms of ischemia (angina, syncope, and cardiac arrest) and therefore are more likely than others to undergo angiography. However, if we assume that all patients who did not undergo cardiac catheterization had no myocardial bridging, then the incidence of compression of the left anterior descending coronary artery due to myocardial bridging in our group of children with hypertrophic

cardiomyopathy would be 10 percent — still substantially higher than that reported in the general population (0.5 to 1.6 percent).^{2,3}

Conclusions

Among the children with hypertrophic cardiomyopathy who underwent cardiac catheterization in this study, the incidence of myocardial bridging was 28 percent. Myocardial ischemia caused by systolic compression of the left anterior descending coronary artery may be one of the primary causes of sudden death in these young patients and may have a key role in the development of ventricular arrhythmias. An increased degree of QTc dispersion on the 12-lead electrocardiogram may help identify the patients who are most likely to have myocardial bridging. The relation between QTc dispersion during exercise testing and the presence of myocardial bridging needs to be studied. Coronary angiography provides valuable information about children with hypertrophic cardiomyopathy who have angina, who have been resuscitated after cardiac arrest, who have ventricular tachycardia or an increased degree of QTc dispersion on the electrocardiogram, or who have an abnormal blood-pressure response during exercise.

We are indebted to Editorial Services, Hospital for Sick Children, for assistance with the preparation of the manuscript.

REFERENCES

1. Porstmann W, Iwig J. Die intramurale-Koronarie im Angiogram. *Fortschr Roentgenstr* 1960;92:129-33.
2. Noble J, Bourassa MG, Petticlerc R, Dyrda I. Myocardial bridging and milking effect of the left anterior descending coronary artery: normal variant or obstruction? *Am J Cardiol* 1976;37:993-9.
3. Ishimori T, Raizner AE, Chahine RA, Awdeh M, Luchi RJ. Myocardial bridges in man: clinical correlations and angiographic accentuation with nitroglycerin. *Cathet Cardiovasc Diagn* 1977;3:59-65.
4. Edwards JC, Burnside CH, Swarm RL, Lansing AI. Arteriosclerosis in the intramural and extramural portions of coronary arteries in the human heart. *Circulation* 1956;13:235-41.
5. Geiringer E. The mural coronary. *Am Heart J* 1951;41:359-68.
6. Kramer JR, Kitazume H, Proudfit WL, Sones FM Jr. Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending artery. *Am Heart J* 1982;103:283-8.
7. Juilliere Y, Berder V, Suty-Selton C, Buffet P, Danchin N, Cherrier F. Isolated myocardial bridges with angiographic milking of the left anterior descending coronary artery: a long-term follow-up study. *Am Heart J* 1995;129:663-5.
8. Colleran JA, Tierney JP, Prokopchak R, Diver DJ, Breall JA. Angiographic presence of myocardial bridge after successful percutaneous transluminal coronary angioplasty. *Am Heart J* 1996;131:196-8.
9. Ge J, Erbel R, Rupprecht HJ, et al. Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging. *Circulation* 1994;89:1725-32.
10. Schwarz ER, Klues HG, von Dahl J, Klein I, Kregs W, Hanrath P. Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication. *J Am Coll Cardiol* 1996;27:1637-45.
11. Irvin RG. The angiographic prevalence of myocardial bridging in man. *Chest* 1982;81:198-202.
12. Voelker W, Schick KD, Karsch KR. Myocardial bridges at multiple sites over the left coronary artery in a patient with hypertrophic cardiomyopathy. *Int J Cardiol* 1989;23:258-60.
13. Navarro-Lopez F, Soler J, Magrina J, et al. Systolic compression of coronary artery in hypertrophic cardiomyopathy. *Int J Cardiol* 1986;12:309-20.
14. Kitazume H, Kramer JR, Krauthamer D, El Tobgi S, Proudfit WL, Sones FM. Myocardial bridges in obstructive hypertrophic cardiomyopathy. *Am Heart J* 1983;106:131-5.
15. Angelini P, Trivellato M, Donis J, Leachman RD. Myocardial bridges: a review. *Prog Cardiol Dis* 1983;26:75-88.
16. Vogt PR, Dubach P, Turina MI. Muscle bridging of the left anterior descending coronary artery. *Circulation* 1996;93:614.
17. Iversen S, Hake U, Mayer E, Erbel R, Diefenbach C, Oelert H. Surgical treatment of myocardial bridging causing coronary artery obstruction. *Scand J Thorac Cardiovasc Surg* 1992;26:107-11.
18. Pey J, de Dios RM, Epeldegui A. Myocardial bridging and hypertrophic cardiomyopathy: relief of ischemia by surgery. *Int J Cardiol* 1985;8:327-30.
19. Morales AR, Romanelli R, Boucek RJ. The mural left anterior descending coronary artery, strenuous exercise and sudden death. *Circulation* 1980;62:230-7.
20. Dilisizian V, Bonow RO, Epstein SE, Fananapazir L. Myocardial ischemia detected by thallium scintigraphy is frequently related to cardiac arrest and syncope in young patients with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1993;22:796-804.
21. Visscher DW, Miles BL, Waller BF. Tunneled ("bridged") left anterior descending coronary artery in a newborn without clinical or morphologic evidence of myocardial ischemia. *Cathet Cardiovasc Diagn* 1983;9:493-6.
22. Reig J, Ruiz de Miguel C, Moragas A. Morphometric analysis of myocardial bridges in children with ventricular hypertrophy. *Pediatr Cardiol* 1990;11:186-90.
23. Feld H, Guadanino V, Hollander G, Greengart A, Lichstein E, Shani J. Exercise-induced ventricular tachycardia in association with a myocardial bridge. *Chest* 1991;99:1295-6.
24. Noble J, Grondin P, Bourassa MG. Successful periarterial muscle resection for myocardial bridging and milking effect of the left anterior descending artery. *Am J Cardiol* 1977;39:267. abstract.
25. Nair CK, Dang B, Heintz MH, Sketch MH. Myocardial bridges: effect of propranolol on systolic compression. *Can J Cardiol* 1986;2:218-21.
26. Greenspan M, Iskandrian AS, Catherwood E, Kimbiris D, Bemis CE, Segal BL. Myocardial bridging of the left anterior descending artery: evaluation using exercise thallium-201 myocardial scintigraphy. *Cathet Cardiovasc Diagn* 1980;6:173-80.
27. Achrafi H. Hypertrophic cardiomyopathy and myocardial bridging. *Int J Cardiol* 1992;37:111-2.
28. Maron BJ, Epstein SE, Roberts WC. Hypertrophic cardiomyopathy and transmural myocardial infarction without significant atherosclerosis of the extramural coronary arteries. *Am J Cardiol* 1979;43:1086-102.
29. Botvinick EH, Dae MW, Krishnan R, Ewing S. Hypertrophic cardiomyopathy in the young: another form of ischemic cardiomyopathy? *J Am Coll Cardiol* 1993;22:805-7.
30. Fananapazir L, Epstein SE. Hemodynamic and electrophysiologic evaluation of patients with hypertrophic cardiomyopathy surviving cardiac arrest. *Am J Cardiol* 1991;67:280-7.
31. Gardin LL, Nanton MA, Hanna BD. Ambulatory monitoring of the sudden death of an adolescent with hypertrophic cardiomyopathy. *Can J Cardiol* 1994;10:548-50.
32. Lazzeroni E, Picano E, Morozzi L, et al. Dipyridamole-induced ischemia as a prognostic marker of future adverse cardiac events in adult patients with hypertrophic cardiomyopathy. *Circulation* 1997;96:4268-72.
33. von Dohlen TW, Prisant LM, Frank MJ. Significance of positive or negative thallium-201 scintigraphy in hypertrophic cardiomyopathy. *Am J Cardiol* 1989;64:498-503.
34. Wigle ED, Rakowski H, Kimball BP, Williams WG. Hypertrophic cardiomyopathy: clinical spectrum and treatment. *Circulation* 1995;92:1680-92.
35. Buja G, Miorelli M, Turrini P, Melacini P, Nava A. Comparison of QT dispersion in hypertrophic cardiomyopathy between patients with and without ventricular arrhythmias and sudden death. *Am J Cardiol* 1993;72:973-6.
36. Cumming GR, Everatt D, Hastman L. Bruce treadmill test in children: normal values in a clinic population. *Am J Cardiol* 1978;41:69-75.
37. Vongpatanasin W, Willard JE, Hillis LD, Lange RA, Landau C. Acquired myocardial bridging. *Am Heart J* 1997;133:463-5.
38. Cannon RO III, Rosing DR, Maron BJ, et al. Myocardial ischemia in patients with hypertrophic cardiomyopathy: contribution of inadequate vasodilator reserve and elevated left ventricular filling pressures. *Circulation* 1985;71:234-43.
39. Braunwald E, ed. Heart disease: a textbook of cardiovascular medicine. 5th ed. Philadelphia: W.B. Saunders, 1997:1169.
40. McKenna WJ, Deanfield JE. Hypertrophic cardiomyopathy: an important cause of sudden death. *Arch Dis Child* 1984;59:971-5.
41. McKenna WJ, Franklin RCG, Nihoyannopoulos P, Robinson KC, Deanfield JE. Arrhythmia and prognosis in infants, children and adolescents with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1988;11:147-53.

42. Day CP, McComb JM, Campbell RWE. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J* 1990;63:342-4.
43. Cowan CJ, Yusoff K, Moore M, et al. Importance of lead selection in QT interval measurement. *Am J Cardiol* 1988;61:83-7.
44. Day CP, McComb JM, Campbell RWE. QT dispersion in sinus beats and ventricular extrasystoles in normal hearts. *Br Heart J* 1992;67:39-41.
45. Miorelli M, Buja G, Melacini P, Fasoli G, Nava A. QT-interval variability in hypertrophic cardiomyopathy patients with cardiac arrest. *Int J Cardiol* 1994;45:121-7.
46. Perkiomaki JS, Koistinen MJ, Yli-Mayry S, Huikuri HV. Dispersion of QT interval in patients with and without susceptibility to ventricular tachyarrhythmias after previous myocardial infarction. *J Am Coll Cardiol* 1995;26:174-9.
47. Stoletniy LN, Pai RG. Value of QT dispersion in the interpretation of exercise stress tests in women. *Circulation* 1997;96:904-10.
48. Frenneaux MP, Counihan PJ, Caforio ALP, Chikamori T, McKenna WJ. Abnormal blood pressure response during exercise in hypertrophic cardiomyopathy. *Circulation* 1990;82:1995-2002.
49. Spirito P, Seidman CE, McKenna WJ, Maron BJ. The management of hypertrophic cardiomyopathy. *N Engl J Med* 1997;336:775-85.
50. Sadoul N, Prasad K, Elliott PM, Bannerjee S, Frenneaux MP, McKenna WJ. Prospective prognostic assessment of blood pressure response during exercise in patients with hypertrophic cardiomyopathy. *Circulation* 1997;96:2987-91.
51. Counihan PJ, Frenneaux MP, Webb DJ, McKenna WJ. Abnormal vascular responses to supine exercise in hypertrophic cardiomyopathy. *Circulation* 1991;84:686-96.
52. Nihoyannopoulos P, Karatasakis G, Frenneaux M, McKenna WJ, Oakley CM. Diastolic function in hypertrophic cardiomyopathy: relation to exercise capacity. *J Am Coll Cardiol* 1992;19:536-40.
53. Lele SS, Thomson HL, Seo H, Belenkie I, McKenna WJ, Frenneaux MP. Exercise capacity in hypertrophic cardiomyopathy: role of stroke volume limitation, heart rate, and diastolic filling characteristics. *Circulation* 1995;92:2886-94.
54. Nezu S, Inoue S, Kawahara Y, et al. Clinical significance of abnormal postexercise systolic blood pressure response in patients with hypertrophic cardiomyopathy. *J Cardiol* 1996;27:65-71.