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REVIEWS

Left Main Coronary Artery Originating From the Right Sinus of Valsalva and Coursing Between the Aorta and Pulmonary Trunk

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Findings are described in five patients who at necropsy were found to have origin of the left main coronary artery from the right sinus of Valsalva and coursing of the anomalously arising artery between aorta and pulmonary trunk to reach the left side of the heart. Three of the five patients were boys and died suddenly at ages 13, 14 and 19 years, respectively: two of them had had one or more episodes of syncope and the third had an abnormal electrocardiogram. The fourth patient, a 64 year old woman, died of chronic congestive heart failure 1 year after an acute myocardial infarction. She had insignificant coronary atherosclerosis. The fifth patient, an 81 year old man, died of chronic alcoholism, having been free of symptoms of cardiac dysfunction during life.

Additionally, clinical and necropsy findings are summarized in 38 previously reported necropsy patients with the coronary anomaly. Of these 38 (34 male [89%]), 23 (61%) died suddenly in the first two decades of life; death in 6 others (16%) appears to have been related to coronary atherosclerosis and 9 patients (24%) died from noncoronary causes. Thus, this anomaly is life-threatening. Why it frequently causes fatal cardiac arrest in some young individuals and allows a normal life span in others remains unclear.

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Although once considered a "minor" coronary anomaly, anomalous origin of the left main coronary artery from the right sinus of Valsalva is now well recognized to cause fatal or nonfatal myocardial ischemia when the left main artery courses between the aorta and pulmonary trunk to reach the left side of the heart (1,2). Symptoms of myocardial ischemia from this anomaly usually develop before the age of 20 years. This report describes findings in 5 patients in whom this anomaly was observed at necropsy and it summarizes observations in 38 previously reported necropsy patients.

Patients Studied

Clinical features. Pertinent findings in the five patients are summarized in Table 1. Three were teenagers and each died suddenly: Patient 1, shortly after running home from school, Patient 2, shortly after mowing the lawn and Patient 3, while jogging. Patients 1 and 2 had one or more episodes of syncope during or shortly after exertion 7 to 12 months before sudden death and Patient 2 had had transient substernal chest pain with exertion on two occasions. Patient 2 had rest and stress electrocardiograms and an echocardiogram after syncope while running 1 month before death. No abnormalities were observed. Patient 3 had ventricular premature complexes and left anterior hemiblock on electrocardiogram 1 year before death. A precordial systolic ejection murmur, grade 2/6, was present in Patient 2.

Patient 4 died at age 64 years. She had been asymptomatic until age 57 when exertional dyspnea and easy fatigability developed. The chest radiograph showed cardiomegaly and the electrocardiogram showed left bundle branch block. She was treated for congestive heart failure with digoxin and diuretic drugs. At age 63 years she had prolonged chest pain and acute myocardial infarction was diagnosed. Three months before death she was hospitalized with chest pain, congestive heart failure and atrial fibrillation. Several days before her death she was again hospitalized with worsening congestive heart failure. She developed ventricular tachycardia followed shortly by fatal ventricular fibrillation. Patient 5 died at age 81 years from chronic alcoholism and was without symptoms of cardiac dysfunction during life.

Necropsy findings. The epicardial coronary arteries in Patients 1 to 4 were free or virtually free of atherosclerotic plaques. Patient 5 had cross-sectional area narrowing by atherosclerotic plaques up to 75% in the right, 50 to 75% in the left circumflex and less than 50% in the left main and

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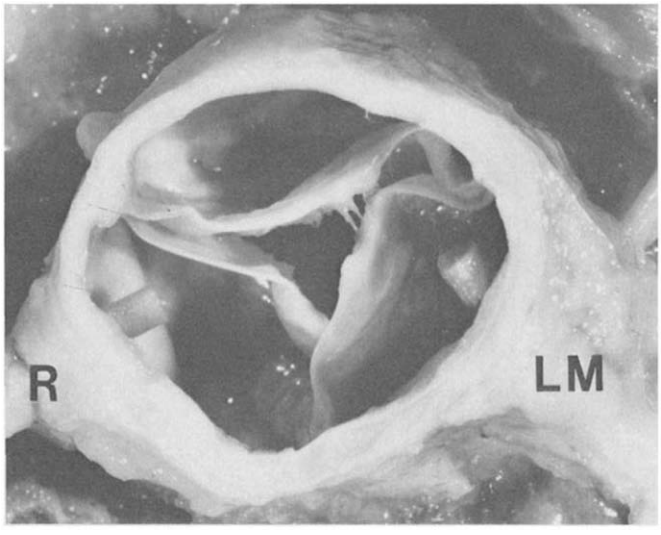
Table 1. Certain Clinical and Necropsy Cardiac Observations in Five Patients in Whom the Left Main Coronary Artery Arose From the Right Sinus of Valsalva and Passed Between the Aorta and Pulmonary Trunk

		Clinical Findings							Time of Death			
Case	Age (yr) & Sex	AP	AMI	S (no.)	D	Duration of Symptoms (mo)	ECG	Mode of Death	During Exertion	Shortly After Exertion	HW (g)	LV Fibrosis
1	13F	0	0	+ (1)	0	12	—	Sudden	0	+	210	0
2	14M	+	0	+ (3)	0	7	Normal	Sudden	0	+	370	+
3	19M	0	0	0	0	0	VPC, LAH	Sudden	+	0	325	0
4	64F	0	+	0	+	84	LBBB	CHF	0	0	500	+
5	81M	0	0	0	0	0	—	Alcoholism	0	0	420	0

AMI = acute myocardial infarction; AP = angina pectoris, CHF = congestive heart failure; D = dyspnea; ECG = electrocardiogram; F = female; HW = heart weight, LAH = left anterior hemiblock; LBBB = left bundle branch block, LV = left ventricular; M = male; S = syncope (no of episodes), VPC = ventricular premature complexes, + = present; 0 = absent; — = no information

left anterior descending coronary arteries. In contrast to normal (Fig. 1), in each of the five patients small wooden sticks placed into the ostium of the left main and right coronary arteries (Fig. 2) were at right angles to each other when viewed from the aorta. The ostium of the right coronary artery was oval in shape with the largest diameter located in a right to left direction with the ostial lumen facing the central portion of aorta. The artery coursed away from the aorta more or less at right angles to it. In contrast, the ostium of the left main coronary artery in Patients 1 to 4 was slit-like with the largest diameter located in a cephalad to caudad direction; the artery coursed parallel to the aortic wall with the ostium pointing directly toward the aortic wall rather than toward the central portion of the aorta. The ostium of the left main coronary artery in Patient 5 was a narrow oval rather than slit-like and the proximal left main artery had almost circumferential calcified plaque that did not actually cause significant luminal narrowing (Fig. 3). Patient 2 had scarring of one left ventricular papillary muscle (Fig. 4), and Patient 4 had a large healed apical infarct that was aneurysmal and contained a thrombus (Fig. 5). All five patients had a dominant right coronary artery circulation.

Figure 1. The aortic valve seen from above showing normal origin of the right (R) and left main (LM) coronary arteries. Wooden sticks have been placed in the ostium of each of the normally arising arteries demonstrating the almost perpendicular relation of the proximal portions of these arteries to the center of the aortic lumen.



Discussion

Classification of the anomaly. Anomalous origin of the left main coronary artery from the right sinus of Valsalva can be classified into four major groups according to the course taken by the left main artery in relation to the aorta and pulmonary trunk en route to the left side of the heart. The left main artery may course anterior to the pulmonary trunk (1,2), posterior to the aorta (3), within the ventricular septum beneath the right ventricular infundibulum (4) or, as in our five patients, between the aorta and pulmonary trunk. When the left main artery passes anterior to the pulmonary trunk over the right ventricular infundibulum, symptoms of myocardial ischemia have not been reported unless significant coronary narrowing due to atherosclerotic plaque was present (1,2). With the exception of the 12 year old girl with this anomaly described by Murphy et al. (3), symptoms of myocardial ischemia have not been reported when the left main artery arises from the right sinus of Valsalva and courses posterior to the aorta.

Review of previous reports. At least 38 necropsy cases have been reported with origin of the left main coronary artery from the right sinus of Valsalva with coursing between the aorta and pulmonary trunk (Tables 2 and 3) (1,5–18). In nine patients, death was unrelated to the anomaly and symptoms of myocardial ischemia were absent during life (Table 2). In the other 29, death was of coronary origin (Table 3). Of these 29 patients, 23 (79%) died before age 20 years (mean 15) and the other 6 (21%) from age 49 to 82 years (mean 61). Of the 23 patients who died young, 22 (96%) died suddenly during or shortly after vigorous exertion and 1 died of acute myocardial infarction, having

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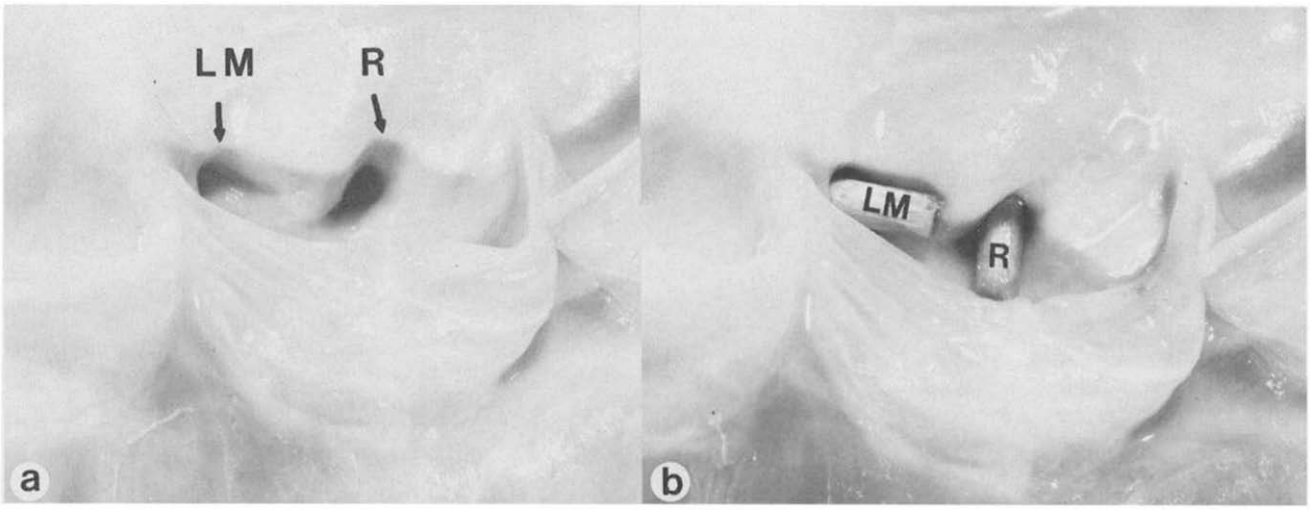


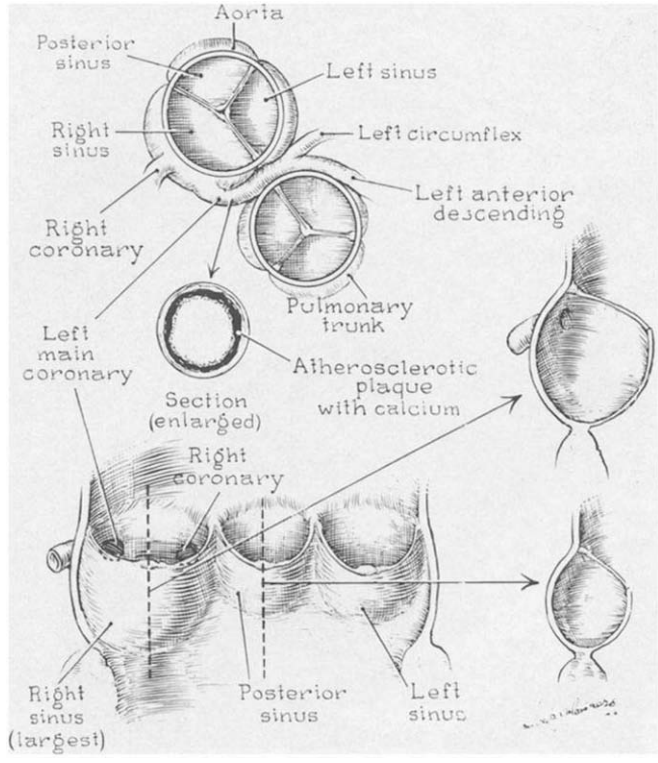
Figure 2. The ostia of the left main (LM) and right (R) coronary arteries in the right sinus of Valsalva viewed from the aortic lumen without (**a**) and with (**b**) wooden sticks in their proximal lumens demonstrating the perpendicular relation of the sticks to each other.

survived for 19 hours after initial collapse, which likewise occurred shortly after exertion. In the 11 cases in which information was provided, 8 patients had had symptoms before the final collapse: exertional syncope in 4, angina in 3 and exertional dyspnea in 1. Of the 23 young patients, 21 were male. At necropsy, 5 of the 23 patients had histologic evidence of myocardial necrosis; 1 patient apparently had atherosclerotic narrowing in the anomalous left main coronary artery.

Mechanism of death in older patients with the anomaly. All six patients who died after age 20 years were men. Three died of acute myocardial infarction and one died suddenly; the mode of death in the other two was not described. Symptoms of cardiac dysfunction were not described in any of the six patients. Histologic evidence of myocardial necrosis was present in four and not discussed in two patients. All six older patients apparently had significant coronary narrowing by atherosclerotic plaque in the abnormal or normal coursing arteries, or both. One patient lacked the circumflex branch of the left main artery.

Although it is clear from current necropsy information that this anomaly can cause sudden death at a young age, the role the anomaly plays in those who have survived past age 20 years is less clear. Our older patient (Case 4) appears to be the only one thus far reported in whom fatal myocardial ischemia could be attributed entirely to the coronary anomaly. Why this patient had fatal myocardial ischemia late in life after decades without symptoms is unclear. Possibly, the excessive cardiac weight (500 g) may have been a factor. Why Patient 5 survived 81 years free of symptoms of cardiac dysfunction is not clear either. Whereas the left main coronary artery arose from the aortic lumen at an acute angle in a manner similar to that in the other four patients, the ostium was not slit-like. This may be due in part to the presence of almost circumferential calcified plaque in the left main artery that caused less than 25% cross-sectional area narrowing but made the artery relatively rigid and less susceptible to dynamic compression. This would, however,

Figure 3. Patient 5. Schematic drawing of the anomalously arising left main coronary artery from the right sinus of Valsalva demonstrating the circumferential calcified atherosclerotic plaque in the proximal left main coronary artery.



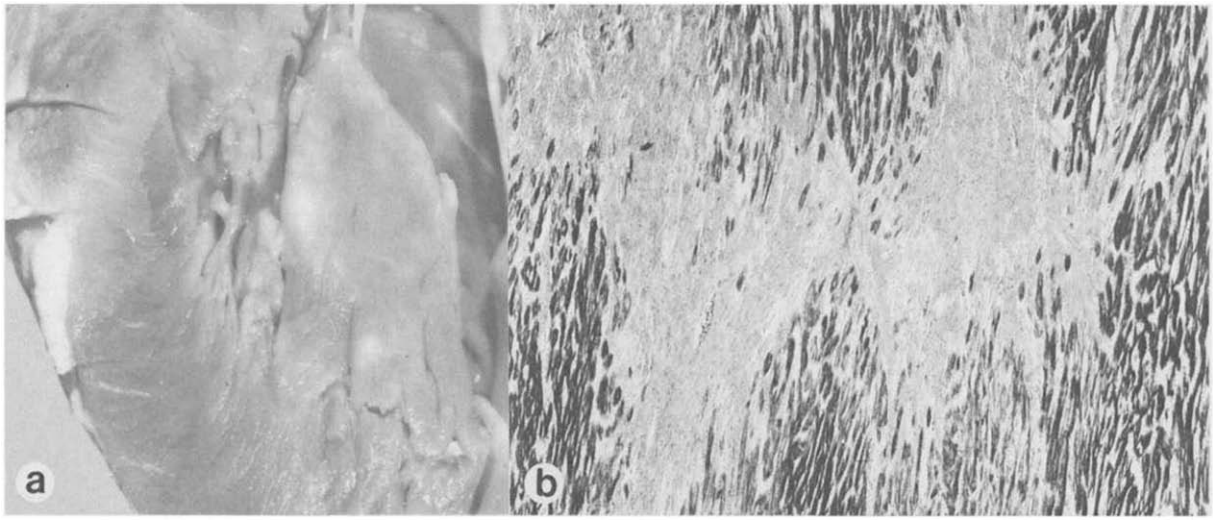
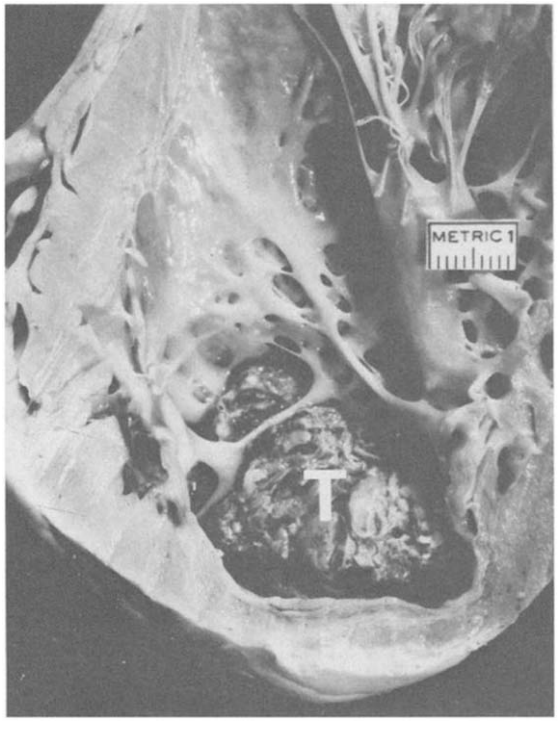


Figure 4. Patient 2. Patchy fibrosis of the anterolateral papillary muscle. **a**, The cut surface of the papillary muscle. **b**, Photomicrograph of the papillary muscle stained with the phosphotungstic acid method demonstrating scar (magnification × 40, reduced by 20%).

not explain why the anomaly did not cause symptoms earlier in life before the plaque would have been present.

Pathogenesis of myocardial ischemia. The precise mechanism by which anomalous origin of the left main coronary artery from the right sinus of Valsalva with coursing between the pulmonary trunk and aorta causes myocardial ischemia is unclear. In each of our five patients the normal right coronary artery arose at an angle more or less perpendicular to the center of the aortic lumen, and it coursed directly away from the aorta; in contrast, the left main coronary artery arose from the aortic lumen at roughly a 180° angle to the center of the aorta. After takeoff, the anomalously arising left main coronary artery was adherent to the wall of the aorta for roughly 1.5 cm as it coursed to the left side of the heart between pulmonary trunk and aorta. Additionally, the ostium of the anomalously arising left main artery was slit-like in Patients 1 to 4 with the largest diameter in a cephalad-caudal direction; in contrast, the ostium of the normally arising right coronary artery was circular and larger. The firmly anchored root of the pulmonary trunk appears to present a potential barrier against which the left main artery could be compressed by expansion of the aortic root that occurs during increased intraaortic pressure associated with exertion. The left main coronary artery of course is equivalent to two arteries in the sense that it is responsible for supplying coronary blood flow to the major portion of the left ventricle and, therefore, significant reduction of flow through it is particularly perilous. Although it is likely that the narrowed left main coronary orifice has the potential to diminish flow through it, an actual reduction appears to be significant only during or immediately after exertion in those individuals without associated atherosclerosis.

Figure 5. Patient 4. Cut section of the severely dilated left ventricle demonstrating the healed apical infarct with aneurysm containing thrombus (T).



Association of death with exertion. The association of death with exertion can be explained by three factors: 1) myocardial oxygen requirements increase with exertion and therefore any obstruction to coronary flow is more likely to result in myocardial ischemia; 2) it is likely that outward expansion of the roots of both aorta and pulmonary trunk during exertion causes further compression of the ostial lumen of the left main artery (1,13); and 3) the left main artery, as it courses between the aorta and pulmonary trunk,

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Table 2. Published Reports of Anomalous Origin of Left Main Coronary Artery From Right Sinus of Valsalva With Coursing of the Left Main Artery Between the Pulmonary Trunk and Aorta: Nine Necropsy Cases With Noncoronary Cause of Death and Without Significant Coronary Atherosclerosis

Case	References		Age (yr) & Sex	Cause of Death	LV	
	First Author	Year			Fibrosis	Necrosis
1	Sanes (3)	1937	68F	PE	+	0
2	Nicod (6)	1952	77M	Senility	0	0
3	Alexander (7)	1956	67F	Tuberculosis	0	0
4	Alexander (7)	1956	53M	Hemorrhage	0	0
5	Cheitlin (1)	1974	48M	Cancer	0	0
6	Cheitlin (1)	1974	40M	Endocarditis	0	0
7	Cheitlin (1)	1974	66M	Cirrhosis	—	—
8	Cheitlin (1)	1974	67M	Cancer	0	0
9	Cheitlin (1)	1974	42M	Cardiomyopathy	—	—

PE = pulmonary embolus; other abbreviations as in Table 1.

could be compressed against the root of the pulmonary trunk where it is firmly anchored to the infundibular septum when the aortic root and pulmonary trunk dilate during exertion. The fact that sudden exertional death and nonfatal myocardial ischemia have been seen in persons in whom the left main artery originates from the proximal right coronary artery and then courses between the great arteries (1,6), and who therefore do not have the abnormal oblique takeoff of the left main artery or the slit-like ostium in the right sinus of Valsalva, suggests that hemodynamic compression (hemodynamic vice) of the left main artery between the great arteries cannot be excluded as an additional mechanism of myocardial ischemia. Davia et al. (19) described a 14 year old boy who had two "exertionally related myocardial infarctions" due to this anomaly; he underwent surgical enlargement of the narrowed left main ostium and is asymptomatic during heavy labor 9 years later. This case lends further support to the theory that the primary mechanism causing myocardial ischemia is related to the narrowed left main coronary ostium.

Usefulness of exercise electrocardiography in diagnosis. The usefulness of stress electrocardiography in identifying the ischemic nature of this anomaly in young persons has received little attention. The fact that one of our necropsy patients, the 14 year old boy (Case 2), had a normal stress test 1 month before death prompted us to review previous experience with stress electrocardiography in young patients with isolated anomalous origin of the left main artery from the right sinus of Valsalva with coursing of this artery between the aorta and pulmonary trunk. Results of stress electrocardiography in seven patients are summarized in Table 4 (1,13,20,21). Of these seven, three had an abnormal stress electrocardiogram (evidence of ischemia or ventricular arrhythmia) and four had a normal stress electrocardiogram. In three of the latter four, however, the test was not a maximal effort. Two of the seven, both of whom had a normal submaximal stress electrocardiogram, subsequently died suddenly. The other five patients underwent surgery. It is clear that the stress electrocardiogram, particularly one that is submaximal in effort, is not a reliable screening test for this anomaly in young patients who present with exertional syncope, angina and even acute myocardial infarction.

Differential diagnosis and means of diagnosis. Young patients with this anomaly often present with exertional syncope, dizziness and angina, but other cardiovascular abnormalities, such as hypertrophic cardiomyopathy and aortic valve stenosis, can present in a similar manner and therefore must be distinguished. The diagnostic approach taken with these young patients must take into account the fallibilities of the various noninvasive tests in identifying this potentially fatal coronary anomaly. The rest electrocardiogram is normal in almost all young persons with this anomaly and therefore it is not helpful. Physical examination may provide important clues to the noncoronary causes of these symptoms, that is, murmurs and peripheral pulses characteristic for aortic stenosis or hypertrophic cardiomyopathy. Echocardiography also is helpful in assessing for or confirming aortic stenosis or hypertrophic cardiomyopathy. Liberthson et al. (22) reported a 54 year old woman with angina pectoris in whom cross-sectional echocardiography identified anomalous origin of the left main coronary artery from the proximal right coronary artery with coursing of the left main artery between aorta and pulmonary trunk. Echocardiography, while potentially useful, has not been reported to have successfully identified origin of the left main artery directly from the right sinus of Valsalva. Continuous ambulatory electrocardiography may identify significant arrhythmias, but provides no help in identifying the cause of the arrhythmias. If the noncoronary causes of exertional syncope and angina are not identified, then more extensive evaluation is dictated to exclude the possibility of anomalous origin of the left main coronary artery.

As has been demonstrated, stress electrocardiography does

Table 3. List of Published Reports of Clinical and Necropsy Findings in 29 Patients With Origin of Left Main Coronary Artery From the Right Sinus of Valsalva With Coursing of the Left Main Artery Between the Pulmonary Trunk and Aorta: Death Due to Coronary Disease

Case	Reference: First Author	Year	Age (yr) & Sex	Clinical Findings: AP	AMI	S (no.)	D	Duration of Symptoms (mo)	Abnormal ECG	Mode of Death	Time of Death: During Exertion	Time of Death: Shortly After Exertion	HW (g)	LV: Fibrosis	LV: Necrosis	CAD
1	Jokl (8)	1962	14M	0	0	0	0	0	—	Sudden	0	+	350	0	0	0
2	Jokl (9)	1966	16M	0	0	+(1)	+	48	—	Sudden	+	0	—	0	0	0
3	Cohen (10)	1967	11M	0	+	0	0	0	+*	AMI	0	+	280	0	+	0
4	Benson (11)	1968	13M	0	0	0	0	0	—	Sudden	0	+	260	0	0	0
5	Benson (11)	1968	13M	0	0	0	0	0	—	Sudden	0	+	370	0	0	0
6	Benson (12)	1970	54M	+	0	0	0	3	0	Sudden	0	0	460	0	0	+
7	Cheitlin (1)	1974	17M	0	0	+(1)	0	—	—	Sudden	0	+	—	0	0	
8	Cheitlin (1)	1974	14M	—	—	—	—	—	—	Sudden	0	+	—	—	—	0
9	Cheitlin (1)	1974	18M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
10	Cheitlin (1)	1974	17M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
11	Cheitlin (1)	1974	18M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
12	Cheitlin (1)	1974	22M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
13	Cheitlin (1)	1974	20M	—	—	—	—	—	—	Sudden	+	0	—	—	+	0
14	Cheitlin (1)	1974	22M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
15	Cheitlin (1)	1974	49M	—	+	—	—	—	—	AMI	—	—	—	—	+	+
16	Cheitlin (1)	1974	49M	—	+	—	—	—	—	AMI	—	—	—	—	+	+
17	Cheitlin (1)	1974	64M	—	—	—	—	—	—	—	—	—	—	—	—	+
18	Cheitlin (1)	1974	69M	—	+	—	—	—	—	AMI	—	—	—	—	+	+
19	Cheitlin (1)	1974	82M	—	—	—	—	—	—	—	—	—	—	—	—	+
20	Pedal (13)	1976	10F	+	0	+(9)	—	26	0	Sudden	0	+	145	+	+	0
21	Liberthson (14)	1979	1M	0	0	0	0	0	—	Sudden	0	+	—	0	0	0
22	Liberthson (14)	1979	11M	+	0	0	0	<1	—	Sudden	+	0	—	0	+	0
23	Liberthson (14)	1979	17M	0	0	0	0	0	—	Sudden	+	0	—	0	+	+
24	Lynch (15)	1980	20M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
25	Lynch (15)	1980	19M	—	—	—	—	—	—	Sudden	+	0	—	—	—	0
26	Tsung (16)	1982	14M	—	0	—	—	—	—	Sudden	+	0	450	0	0	0
27	Tsung (16)	1982	18M	—	0	—	—	—	—	Sudden	+	0	480	0	0	0
28	Betend (17)	1983	16M	+	0	+(3)	0	32	+†	Sudden	0	+	—	+	0	0
29	Topaz (18)	1985	15F	—	—	—	—	—	—	Sudden	+	0	—	—	—	0

*Supraventricular tachycardia, Q wave in lead V_1 and ST segment depression in lead V_2; †second degree atrioventricular block. CAD = atherosclerotic coronary artery disease; other abbreviations as in Table 1

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Table 4. List of Published Reports of Stress Electrocardiography in Six Young Patients With Isolated Anomalous Origin of the Left Main Coronary Artery From the Right Sinus of Valsalva With Coursing of the Left Main Between the Pulmonary Trunk and the Aorta, Who Died Suddenly or Had Symptoms of Myocardial Ischemia

First Author	Year	Age (yr) & Sex	S	AP	AMI	SD	Normal Rest ECG	Time Before Death (mo)	Type of Stress Test	Response	Follow-up
Cheitlin (1)	1974	14M	+	0	0	0	+	0	Treadmill†	Normal	Surgery
Pedal (13)	1976	10F	+	+	+	+	+	<36	15 Knee bends	Normal	SD
Mustafa (20)	1981	12M	0	+	0	0	+	0	Treadmill	Normal	Surgery
Donaldson (21)	1983	*M	+	0	0	0	+	0	Treadmill	VT	Surgery
Donaldson (21)	1983	*M	0	+	0	0	+	0	Treadmill	Ischemia	Surgery
Donaldson (21)	1983	*M	0	+	0	0	+	0	Treadmill	Ischemia	Surgery

*Ages of 15, 21 and 32 years in this series could not be identified to individual patients; †submaximal SD = sudden death; VT = ventricular tachycardia, other abbreviations as in Table 1

not always identify myocardial ischemia in young patients with this coronary anomaly who are at risk for sudden death, but it should be done and it should be carried to maximal effort if evidence of myocardial ischemia is not identified at lower levels of exercise. If stress electrocardiography is abnormal, then angiographic assessment of coronary anatomy is dictated. If maximal stress electrocardiography is normal, then coronary angiography must still be considered in boys with clear-cut exercise-induced symptoms. The usefulness of thallium stress electrocardiography for diagnosis of this anomaly is uncertain. Patient 3 (Table 4) had a normal thallium stress test (20). Finally, in the specific case of frank exertional syncope at a young age, particularly in a male patient, coronary angiography should be performed after a second episode of syncope. Angiography should be considered after a single episode if no other clear cause of exertional syncope is evident by noninvasive testing.

Contrast to origin of both coronary arteries from left sinus. In contrast to origin of both coronary arteries from the right sinus of Valsalva with coursing of the left main artery between the great arteries, origin of both left main and right coronary arteries from the left sinus of Valsalva with coursing of the right coronary artery between the great arteries is usually, but not always, a benign congenital anomaly (23). Among 10 necropsy patients with the latter anomaly reported by Roberts et al. (23), the anomaly was an incidental necropsy finding in 7 patients and had nothing to do with their death, but in the other 3, it appeared to have caused fatal cardiac arrest with exertion.

Management of patients with the anomaly. Once the origin of both left main and right coronary arteries from the right aortic sinus with coursing of the left main artery between the great arteries has been diagnosed, at least in younger individuals, operative therapy appears warranted for the prevention of sudden death and for relief of exercise-induced symptoms of myocardial ischemia. Various operative approaches for revascularization have been described. Aortocoronary conduits, either saphenous vein or mammary artery, or both, to the left anterior descending and left circumflex coronary systems have resulted in relief of symptoms and in relief of objective evidence of myocardial ischemia in several patients (22,24,25). Other surgical approaches also have been successful. Davia et al. (19) described a 14 year old boy who had two myocardial infarcts and underwent surgical enlargement of the narrowed left main coronary ostium by extending an incision from the ostium through the common wall of the aorta and the anomalous artery over the intercoronary commissure. He was free of symptoms and active 9 years later, despite the presence of mild aortic regurgitation due to the procedure. Four patients, aged 12 to 36 years, have undergone an operation of a similar nature to reestablish the normal anatomic location of the left main artery origin to the left sinus of Valsalva by incising along the course of the common wall between the aorta and anomalous artery to the region of the left sinus of Valsalva and joining the intima of the vessel to the aorta, resulting in a new ostium (20,21). No evidence of ischemia was present in a follow-up period of 10 to 36 months.

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