was administered directly into the graft with complete relief of pain and resolution of ST segment elevation. Angiography of the graft (Fig. 2, *C*) now revealed the graft to be patent; there was normal distal runoff from the graft with a 75% obstructive lesion of the native RCA just distal to the site of graft insertion. Subsequently, pain again developed associated with ST segment elevation. Graft spasm and complete occlusion was again demonstrated. NTG was administered without subsequent relief of pain or graft spasm. Nifedipine 10 mg was administered sublingually, also without any improvement. The catheterization was terminated at this point. During repair of the brachial artery, ventricular fibrillation developed which was successfully cardioverted. Subsequent observation demonstrated evolution of an inferior wall acute myocardial infarction (AMI).

Patients with CAS have been reported to have less satisfactory results following CABG than patients with fixed obstructive disease alone.[3] Spasm of the bypassed native vessel or of additional non-bypassed native vessels has been postulated to be the mechanism for the less beneficial results with CABG in CAS patients. The current case demonstrated spasm of the bypassed native vessel and two additional mechanisms responsible for recurrent anginal symptomatology; development of a significant lesion at the site of CABG insertion and spasm of the bypass graft itself. Spasm of the graft was transiently reversed by NTG but subsequently recurred resulting in AMI.

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Walk-through angina phenomenon demonstrated by graded exercise radionuclide ventriculography: Possible coronary spasm mechanisms

H. Roger Rizi, M.D., Robert C. Kline, M.D., Myrwood C. Besozzi, M.D., Victor Kalf, M.D., Mark Rabinovitch, M.D., William Chan, M.D., James H. Thrall, M.D., and Bertram Pitt, M.D.
Ann Arbor, Mich.

From the Department of Internal Medicine, University of Michigan Medical Center.

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Reprint requests: H. Roger Rizi, M.D., The Harriman Jones Medical Clinic, 211 Cherry Ave., Long Beach, CA 90802.

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The phenomenon of "walk-through" angina can be inferred from Heberden's note in the early nineteenth century.[1] This entity was later described by Osler,[2] Wenckebach,[3] Gallavardin,[4] Price,[5] and Leftwich.[6] Although the existence of this clinical entity has been challenged by some,[7] MacAlpin and Kattus[8] provided objective ECG data supporting the presence of this phenomenon in 1966. We report a patient with walk-through angina who demonstrated scintigraphic left ventricular (LV) regional wall motion abnormality and decreased LV ejection fraction (EF) at the time of exertional angina, which became normal at peak exercise with disappearance of ischemic chest pain.

A 57-year-old white female presented with episodes of increasing angina pectoris for 1 year. Four weeks before admission her anginal frequency progressed to where it was induced by minimal exertion despite 160 mg propranolol and 60 mg isosorbide dinitrate daily. Off medications for 48 hours, she was asymptomatic at rest with normal ECG prior to radionuclide ventriculography (RNV). Resting RNV LVEF was 0.73 with normal wall motion (Fig. 1, *left panel*). At the first stage of exercise (150 kpm/min), she developed typical angina with decline in LVEF to 0.64 with inferoapical hypokinesis (Fig. 1, *middle panel*). At higher workloads, chest pain disappeared and exercise (third stage, 450 kpm/min), terminated by leg fatigue, RNV wall motion returned to normal with increased LVEF to 0.72 (Fig. 1, *right panel*) despite higher systolic pressure · heart rate product (25,100 versus 9,000 at stage one and 6,200 at rest). The ECG remained normal throughout exercise. Postexercise, angina recurred which was promptly relieved by sublingual nitroglycerin. Subsequent cardiac catheterization revealed normal resting right and left ventricular hemodynamics; resting LVEF was 0.71 without wall motion abnormality. Coronary arteriograms revealed 80% proximal fixed obstruction of the right coronary artery (RCA) (Fig. 2, *left panel*) without significant stenosis in the left coronary artery system (Fig. 2, *right panel*).

During exercise testing, most patients with obstructive coronary artery disease (CAD) are asymptomatic during early exercise and experience angina at higher workloads. Occasional CAD patients, however, may have great adaptability in exercise tolerance. A common form of this adaptability is known as the "walk-through" angina phenomenon. Patients with this phenomenon commonly experience angina at early exercise and as the exercise is continued at the same workload angina gradually subsides. Some patients can walk-through their initial angina and remain free of chest pain even at higher workloads.[8] Our patient demonstrated the latter phenomenon. In some patients with historical walk-through phenomenon, angina cannot be reproduced at the time of formal exercise testing. Others, as demonstrated by the present patient, demonstrate walk-through angina for the first time during formal stress testing.[8] The reason for such variations is unclear. The protocol used during formal exercise testing may be contributory. During daily physical activity, the walk-through phenomenon is commonly

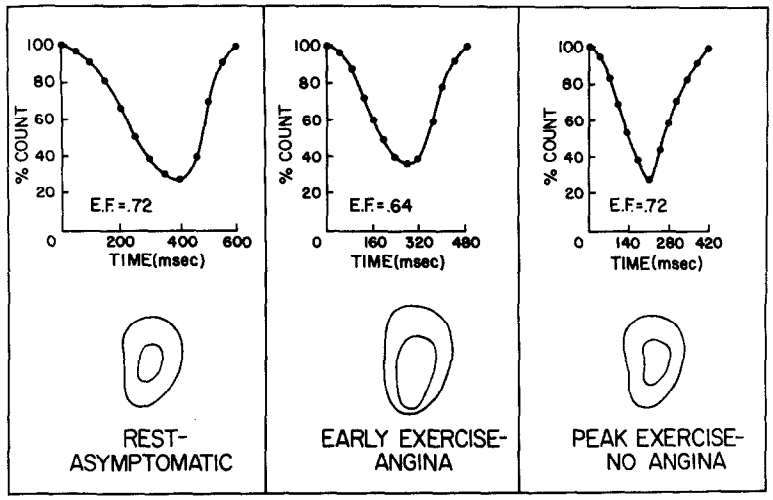

Fig. 1. Left ventricular time-activity curves *(top)* with corresponding superimposed end-diastolic and end-systolic left ventricular outlines *(bottom)* at rest *(left panel)*, during early exercise *(middle panel)*, and at peak exercise *(right panel)*. There is inferoapical hypokinesis associated with angina and decline in left ventricular ejection fraction during early exercise *(middle panel)*. *EF* = left ventricular ejection fraction.

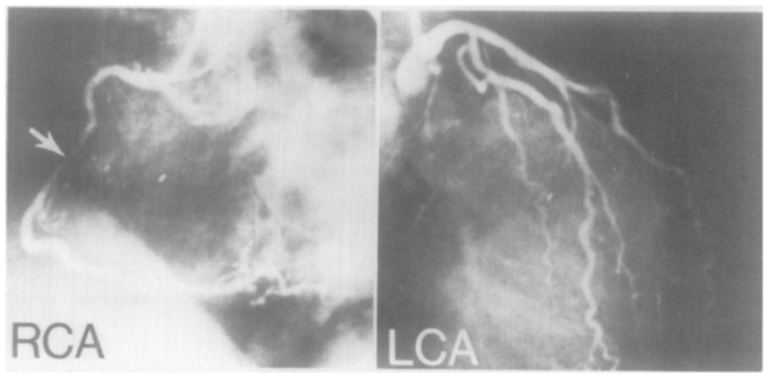

Fig. 2. Right *(RCA) (left panel)* and left *(LCA) (right panel)* coronary arteries viewed from left and right anterior oblique projections, respectively. An 80% obstruction *(arrow)* is visualized in the proximal third of the dominant RCA.

observed when the workload is relatively constant (walking and jogging). In almost all exercise laboratories the patient is subjected to graded increase in workload. This may reduce the opportunity of reproducing walk-through angina in most patients with a convincing clinical history of the phenomenon.

The underlying mechanism of walk-through angina is not well understood. Myocardial ischemia is thought to be the result of an imbalance between myocardial blood supply and demand. In some patients with walk-through angina, the heart rate · blood pressure product (PRP) has been found to be higher at early exercise before the patient develops angina than at peak exercise,[8] probably due to anxiety or abnormal neurohumoral response to initiation of exercise. Since PRP is a relatively reliable estimate of myocardial oxygen demand, this finding may explain the presence of the walk-through phenomenon in a subgroup of CAD patients. However, as indicated above, in our patient the initial PRP at the time of angina (Fig. 1, *middle panel*) was lower than during peak exercise (Fig. 1, *right panel*). It is therefore unlikely that the presence of angina at the lower workload could be explained by increased myocardial oxygen demand as compared to peak exercise. We postulate that transient coronary spasm superimposed on high-grade atherosclerotic obstruction might explain the walk-through phenomenon in this particular subgroup. Alternatively, walk-through angina might be explained by development of collateral flow in the ischemic region during later stages of exercise. It has been demonstrated that patients with coronary spasm may have exercise-induced angina early in the day that cannot be reproduced later.[9] This latter observation[9] may in part explain the marked variability in induction of chest pain in patients with walk-through angina. Direct ventriculographic data in patients with walk-through angina has not been previously reported. The appearance of inferoapical hypokinesis in our patient with high-grade RCA obstruction and decline in LVEF at the time of angina provide strong objective evidence of the walk-through phenomenon.

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Phonographic-echographic documentation of systolic honk in tricuspid prolapse

Chuwa Tei, M.D., Pravin M. Shah, M.D., and Hiromitsu Tanaka, M.D. *Los Angeles, Calif., and Kagoshima, Japan*

Tricuspid systolic whoop[1] or honk[2] has been seldom described in comparison with mitral systolic whoop[3] or honk.[4] The cause of tricuspid honk has remained unclear. Mitral honk has been reported with mitral valve prolapse.[5,6] We report a case of tricuspid systolic honk associated with tricuspid valve prolapse.

A 52-year-old male patient was initially admitted to the hospital in April, 1979, with complaints of palpitations and dyspnea. The first heart sound was loud and the second heart sound was followed by an opening snap with mid-diastolic rumble; there was no systolic murmur. Rheumatic mitral stenosis with left atrial enlargement was documented by echocardiography. Because the same symptomatology remained, the patient was readmitted in May, 1979. The jugular veins were distended with prominent V waves; a systolic thrill was present at the lower left sternal border; and a systolic honk was audible even without a stethoscope along the right and left lower sternal borders with radiation to the entire precordial surface. ECG showed paroxysmal atrial tachycardia (240/min) with varying block (ventricular rate 140 bpm) and Q waves in leads II, III, and aV_F indicative of inferior myocardial infarction. Phonocardiography revealed typical systolic honk at frequency of 80 Hz (Fig. 1, *A*) and simultaneous M-mode echocardiography documented pansystolic bowing of the tricuspid leaflet (Fig. 1, *A*) and fluttering of the anterior tricuspid leaflet at the same frequency of the honk. Real time two-dimensional echocardiography depicted prolapse of the anterior tricuspid leaflet, as well as mitral stenosis.

The patient improved with digitalis and diuretics, and atrial tachycardia changed to atrial fibrillation (AF). The intensity of the systolic honk varied from beat-to-beat

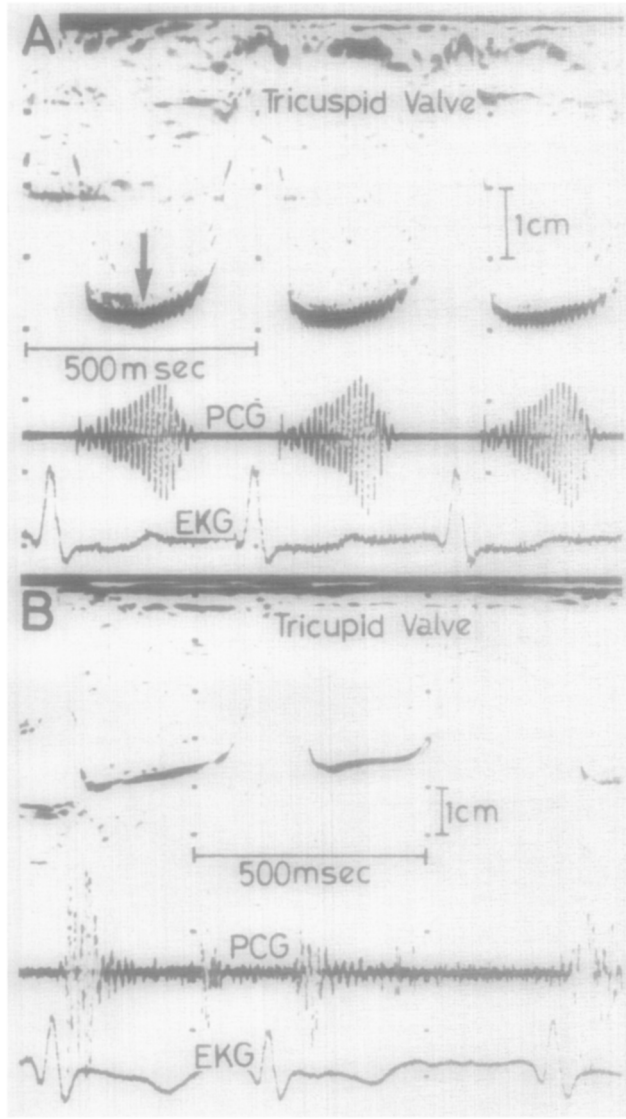

Fig. 1. Simultaneous tricuspid echocardiogram and phonocardiogram at paper speed of 100 mm/sec. *Panel A,* Phonocardiogram *(PCG)* shows systolic honk with frequency of approximately 80 Hz. The echocardiogram reveals pansystolic bowing (prolapse) of the tricuspid leaflet with fluttering during systole *(arrow).* The frequency of fluttering of the tricuspid leaflet is the same as that of the honk. *Panel B,* Neither PCG honk nor echographic fluttering is shown. The tricuspid leaflet does not show prolapse; instead the tricuspid leaflet gradually moves anteriorly during systole.

From the Departments of Medicine, University of California, Los Angeles; Wadsworth VA Medical Center; and Kagoshima University.

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Reprint requests: Chuwa Tei, M.D., Cardiology (691/111E), Wadsworth VA Medical Center, Wilshire & Sawtelle Blvds., Los Angeles, CA 90073.

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