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 reported.
NTG was administered without subsequent relief of pain
or graft spasm. Nifedipine 10 mg was administered sublin-
gually, also without any improvement. The catheteriza-
tion 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 myocar-
dial infarction (AMI).

Patients with CAS have been reported to have less
satisfactory results following CABG than patients with
fixed obstructive disease alone. 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 cur-
cent case demonstrated spasm of the bypassed native
vessel and two additional mechanisms responsible for
recurrent anginal symptomatology; development of a sig-
ificant 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
radio nuclide ventriculography: Possible
coronary spasm mechanisms

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The phenomenon of "walk-through" angina can be in-
ferred from Heberden's note in the early nineteenth
century. This entity was later described by Osler, Wencke-
bach, Gallavardin, Price, and Leftwich. Although the exis-
tence of this clinical entity has been challenged by some,
MacAlpin and Kattus provided objective ECG data sup-
porting the presence of this phe-
nomenon in 1966. We report a patient with walk-through
angina who demonstrated scintigraphic left ventricular
(LV) regional wall motion abnormality and decreased LV
jection 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 propra-
nolol and 60 mg isosorbide dinitrate daily. Off medications
for 48 hours, she was asymptomatic at rest with normal
ECG prior to radionuclide ventriculography (RNV). Rest-
ing 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 (35,100 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. Sub-
sequent cardiac catheterization revealed normal resting
right and left ventricular hemodynamics; resting LVEF
was 0.71 without wall motion abnormality. Coronary
angierograms 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 adap-
tability in exercise tolerance. A common form of this
adaptability is known as the "walk-through" angina phe-
nomenon. Patients with this phenomenon commonly
experience angina at early exercise and as the exercise is
continued at the same workload angina gradually sub-
sides. Some patients can walk-through their initial angina
and remain free of chest pain even at higher workloads.
Our patient demonstrated the latter phenomenon. In
some patients with historical walk-through phenomenon,
angina cannot be reproduced at the time of formal exer-
cise testing. Others, demonstrated by the present
patient, demonstrate walk-through angina for the first
time during formal stress testing. The reason for such
variations is unclear. The protocol used during formal
exercise testing may be contributory. During daily physi-
ical activity, the walk-through phenomenon is commonly

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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, 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. This latter observation 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

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Tricuspid systolic whoop1 or honk2 has been seldom described in comparison with mitral systolic whoop3 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, indicative of inferior myocardial infarction. Phonocardiography revealed typical systolic honk at 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.

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