favor of CABG may be linked to the use of the left internal mammary artery as a graft.

Despite the use of either revascularization technique, patients remain prone to progressive atherosclerotic disease with the potential to form plaque at previously unaffected sites. This necessitates aggressive long-term medical therapy and risk factor modification to achieve the lowest possible risk of symptomatic progression or MI. Retreatment with CABG is possible but is fraught with higher risk, and the outcome of repeat stenting for in-stent restenosis is never as good as for de novo lesions.

In a small group of patients, PCI and/or CABG fails and the patient has refractory angina. Once medical therapy has been maximized, few truly effective options remain. Transmyocardial laser revascularization in areas of ischemia has been used to reduce symptoms, but this technique is now of uncertain value. External counterpulsation is a technique whereby blood pressure cuffs are placed on each leg, inflated during diastole and deflated during systole. Patients typically have a 1-hour session that may be repeated 35 times. Angina relief has been reported with this procedure and may reflect some beneficial effect on endothelial function. Spinal cord stimulation using electrodes placed in the C7-T1 dorsal epidural space can reduce anginal symptoms in the short term, although the long-term role needs definition.

Other Anginal Syndromes

Variant Angina

Whereas typical angina pectoris is usually triggered by physical or emotional stress, some patients experience a syndrome termed *variant angina.* Variant angina was first described in 1959 by Prinzmetal and colleagues, who observed patients with chest discomfort at rest, not triggered by physical or emotional stress, and associated with ST segment elevation (Fig. 8-4). Episodes of AV block and ventricular ectopy were observed, but MI was not a common feature. These patients typically did not have the common CAD risk factors other than smoking. Coronary angiography demonstrated these patients to be experiencing transient coronary vasospasm. The vasospasm tended to occur in an area of atherosclerotic plaque, but some patients had spasm in angiographically normal segments of coronary artery.

In the course of investigating the pathophysiology of variant angina, a number of provocative tests were developed to induce coronary spasm in susceptible individuals. Intracoronary ergonovine or acetylcholine can induce spasm in patients with variant angina, probably as a result of underlying endothelial dysfunction. Other spasm-inducing provocations include the cold pressor test (placing a hand in an ice bath), the induction of alkalosis (hyperventilation or intravenous bicarbonate), and histamine infusion. Provocative testing to induce coronary vasospasm has fallen out of favor in the routine assessment of patients with angina.

Coronary vasospasm usually resolves promptly with the administration of nitroglycerin (sublingual, intravenous, or intra-arterial). The combination of oral nitrates and calcium channel blockers is often used to prevent spasm. β-Blockers may aggravate coronary spasm by inhibiting the action of vasodilating β_2-receptors, allowing for unopposed α-receptor induced vasoconstriction. Rare patients do not respond to vasodilator medical therapy and may benefit from coronary stent placement in spasm-prone atherosclerotic lesions.

Microvascular Angina with Normal Coronary Arteries

Angina can occur in some patients in the face of normal-appearing coronary arteries and no provocable spasm. Decreased endothelium-dependent vasodilation may be the underlying pathophysiology of microvascular angina. Patients with this condition may demonstrate an increase in coronary resistance and an inability to increase coronary blood flow sufficiently when challenged by increases in myocardial oxygen demand. Women are more likely to be affected with microvascular angina, and the symptoms not uncommonly occur at rest or with emotional stress. Exercise can also trigger angina.

A host of diagnostic tests can detect the presence of ischemia in patients with microvascular angina. In the case of stress testing, ST changes of ischemia can be detected as well as nuclear

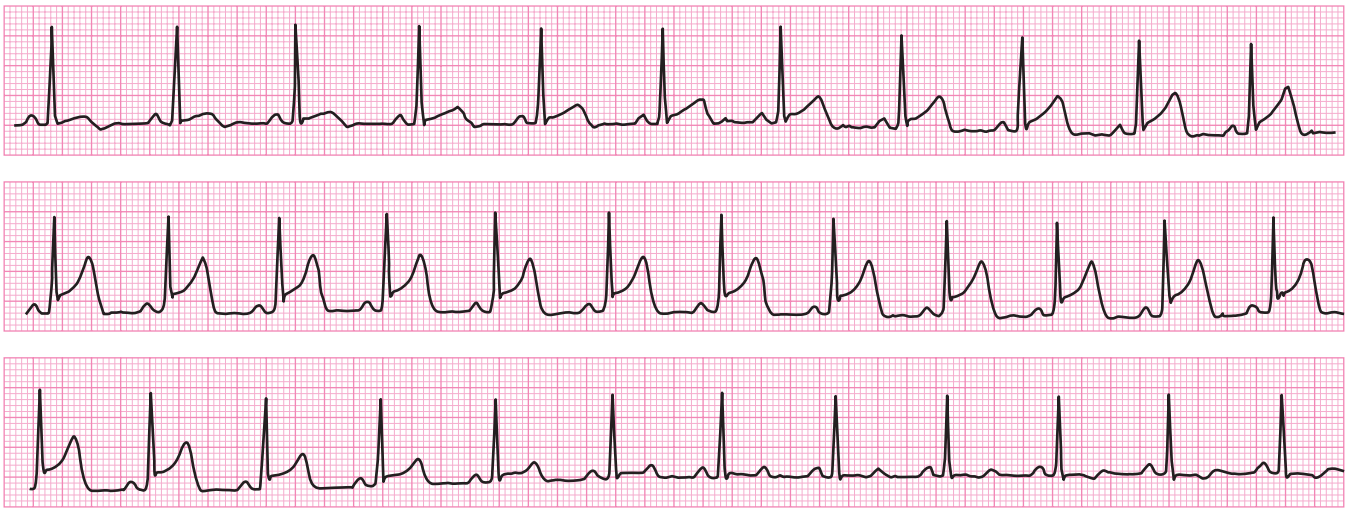

FIGURE 8-4 Continuous electrocardiogram recording in a patient with Prinzmetal (variant) angina. The spontaneous onset of chest discomfort began during the *top strip,* accompanied by transient ST-segment elevation. By the *bottom strip,* several minutes later, both discomfort and ST-segment elevation had resolved.