in mitral valve disease. In a series of 839 cases, 18.7 percent had systemic emboli. Conclusions from this large series were: 1) atrial fibrillation is the main cause of systemic embolism; 2) embolism is as common with mitral regurgitation as with mitral stenosis; 3) mitral valve calcification predisposes to operative emboli; 4) atrial appendage size has no influence on the occurrence of systemic embolism.

Review of the literature has not revealed a reported case of peripheral migration of the mitral valve. That this may occur with endocarditis and aneurysm formation of the mitral valve is easily appreciated when the pathogenesis of valve aneurysm formation is reviewed. As a result of valvulitis, granululation and scar tissue are exposed to a constantly maintained intraventricular pressure with dilatation and protrusion of the valve into the left atrium. The aneurysm may obstruct the mitral orifice producing mitral stenosis or perforate, causing incompetence. Perforation of the aneurysm allows embolization of valve fragments, vegetations and clots. The aneurysms characteristically involve the anterior leaflet of the mitral valve, particularly in the presence of aortic regurgitation.

The serious hemodynamic changes attendant with acute mitral regurgitation due to bacterial endocarditis demand judicious medical management directed to control of congestive heart failure and sterilization of the infective process. Prompt surgical intervention must be considered if response to the measures is not achieved. The prudence of removing the nidalus of infection as well as hemodynamic correction by surgical means is well documented.

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Right Coronary Arterial Spasm Causing Prinzmetal's Variant Angina

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Severe spasm of the right coronary artery was documented during the second of three injections during coronary arteriography in a 42-year-old man with a five-year history of Prinzmetal's variant angina. Only mild atherosclerosis (<25 percent narrowing) of the artery before and after the spasm was noted. Repeat coronary arteriography two years later confirmed the minimal disease in the right coronary artery, despite continued occasional severe disability from angina. Nitroglycerin invariably relieved the pain and isosorbide dinitrate taken prophylactically has been very effective in preventing episodes of nocturnal angina. These observations support the concept that coronary spasm is one etiology of ischemic pain in variant angina.

The pathophysiology of Prinzmetal's variant angina remains unclear since he initially proposed increased tonus of a large narrowed coronary artery as the precipitating cause. The significant features of this variant form of angina include its occurrence at rest, frequently on a cyclical basis during the day or night, and not precipitated by exercise. The ECG during chest pain shows marked ST elevation rather than ST depression. Hemodynamic observations in a small patient group have confirmed that increased myocardial work is not the apparent etiology for these transient anginal attacks which are accompanied by a reversible myocardial infarction pattern on the electrocardiogram.

Review of recent studies of coronary arteriography in variant angina pectoris patients shows two apparent patient groups: one group with severe proximal occlusive coronary artery disease and the other with normal or minimally diseased coronary vessels. The purpose of this report is to document a case of Prinzmetal's variant angina secondary to diffuse spasm of the right coronary artery with longterm follow-up and repeat arteriography. Its possible relationships to other forms of rest angina with and without ST elevation will be discussed.

CASE REPORT

A 42-year-old Negro man was admitted to Stanford University Hospital in May, 1972, because of increasing severity and frequency of crushing substernal chest pain. He had noted occasional pain, primarily nocturnal, since 1966. The pain usually resolved spontaneously after awakening and had never occurred with exertion. In the several months prior to admission he had noted an increasing number of attacks while asleep, ranging from two to ten per night, in addition to episodes while resting. Several episodes of chest pain oc-

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occurred while upright and were accompanied by profuse diaphoresis and near syncope. He was admitted after a particularly severe bout of chest pain; an ECG in the emergency room showed ST elevation consistent with a myocardial injury. Coronary risk factors included smoking one pack of cigarettes per day for 20 years. There was no family history of cardiovascular disease. The blood pressure and pulse were normal and no other abnormalities were detected.

Initial electrocardiogram showed diffuse ST elevation in the inferior leads consistent with the hyperacute phase of a diaphragmatic infarction, but which resolved ten minutes later (Fig 1 and 2). ECG monitoring in the CCU showed numerous arrhythmias which were only partially associated with chest pain. Additionally, ST elevation occurred with and without pain during the monitoring period. The arrhythmias occurring most commonly included abnormalities of AV conduction, sinus bradycardia and arrest, atrial and ventricular tachycardia.

Chest x-ray film showed normal findings. Results of routine laboratory tests were within normal limits, with a serum cholesterol of 200 mg percent and triglycerides of 101 mg percent.

**Hospital Course**

The hospital course was remarkable because of recurring

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**Figure 1.** Marked ST elevation in the inferior leads during chest pain (8:30 am).

**Figure 2.** Remarkable resolution of the "myocardial injury pattern" with residual nonspecific abnormalities of the ST-T wave (8:40 am).
arrhythmias with and without chest pain. Nitroglycerin was always effective in relieving the pain, usually within 30 seconds. A submaximal treadmill exercise test was performed, which was negative for chest pain or ST depression at a heart rate of 160. Coronary arteriography was performed on hospital day 3. It demonstrated a dominant right coronary system (Fig 3). The second injection into the right coronary artery showed diffuse spasm and narrowing accompanied by chest pain and ST elevation (Fig 4). Nitroglycerin produced prompt relief, and a third injection documented return to normal (Fig 5).

In an effort to determine whether this apparent coronary arterial spasm, which responded so dramatically to nitroglycerin, could be prevented, further studies were undertaken. Long-acting isosorbide dinitrate every eight hours for one day was alternated with a placebo. There was complete prevention of arrhythmias, ST elevation and chest pain during the two 24-hour periods on active therapy. The patient was therefore discharged to his home, taking long-acting oral isosorbide dinitrate.

Follow-up

The patient continued to work as a janitor with only occasional episodes of pain, which may have been related to changes in, or forgetting to take, his medication. The only change in the patient's electrocardiogram was some loss of R wave in V2-V5, raising the question of a silent anteroseptal myocardial infarction. In February, 1972, the patient developed increasing disability from chest pain. Ambulatory ECG monitoring documented periods of first and second degree block, as well as ST elevation during ischemic chest pain.

Re-admission and repeat coronary arteriography demonstrated no significant change in the coronary arteries. Left ventricular angiography showed normal contractility. Review of his past course indicated that noncompliance with regard to taking his medication may have been an important factor in the change in his clinical status. However, he continued to have at least one severe episode nightly, between 2:00 AM and 6:00 AM. Sublingual isosorbide dinitrate, taken after being awakened with pain, would rapidly relieve the pain and prevent any episodes during the rest of the night. There has been no significant chest pain during the day.

DISCUSSION

The possibility of catheter-induced coronary spasm cannot be completely ruled out, although this seems unlikely in the case reported. The precipitation of the patient's typical chest pain and typical ST elevation on ECG leads 2, 3 and aVF make it likely that we had observed a spontaneous episode of transient coronary spasm. There have been several reports in the literature relating spasm of a coronary vessel to introduction of the catheter into the coronary artery. In our experience, the area of spasm is almost always near the tip of the catheter which is presumably irritating the intima. This is particularly true of the Judkins catheter, whose tip may be longer than the proximal segment of the right coronary artery. The extensive length of the segment of spasm in our reported case, beginning several cen-

**Figure 3.** Essentially normal dominant right coronary artery is seen in the LAO projection.

**Figure 4.** Marked extensive narrowing of the mid-portion of the right coronary artery in the RAO projection during the second injection.

**Figure 5.** RAO projection approximately five minutes after the second injection shown in Fig 4. Note slight irregularities in the area where spasm occurred, but no significant occlusion.
timeters from the catheter tip also makes it unlikely that the spasm was iatrogenic in origin.

Coronary arterial spasm as one etiology for precipitation of ischemic chest pain in the small patient group with Prinzmetal's variant angina seems almost a certainty. Supportive evidence for this viewpoint is as follows:

First, the findings in the case presented support those in at least three other cases in recent medical literature, arteriographically documenting coronary arterial spasm during an episode of chest pain in a patient with Prinzmetal's angina. Additionally, there are at least five reported cases of essentially normal findings on coronary arteriography in variant angina patients. Coronary spasm can be highly suspect as the etiology for the myocardial ischemia in this patient group as well. In our patient, repeat coronary arteriography two years later helped to confirm the relative absence of significant occlusive atherosclerotic disease of the coronary arterial system, which could have been missed in the first study.

Second, our patient had symptoms for at least seven years without electrocardiographic and arteriographic evidence of an infarct in the diaphragmatic area where the marked ST elevation occurred. The chronicity of symptoms of these patients has also been noted by other investigators.

Third, the typical syndrome of variant angina as reported by Prinzmetal et al emphasized the cyclic nature of the recurrent unprovoked pain, frequently at the same time daily. This, in addition to the lack of pain on exertion, would suggest that a changing coronary flow pattern, such as transient focal or diffuse coronary arterial spasm, must occur in these patients to precipitate the myocardial ischemia without a change in myocardial work load and oxygen consumption.

Fourth, the remarkable response to the prophylactic use of short- or long-acting vasodilators also supports the concept of transient "unprovoked" spasm as the cause for variant angina. The observations during initial hospitalization, comparing 24-hour periods on active medication and placebo, were convincing and were confirmed by the patient's response over the following year. Sublingual or chewable isosorbide dinitrate, taken when the patient is awakened with the first episode of chest pain, will prevent further chest pains the rest of the night, which otherwise occur.

There appear to be two generally observed patient groups with Prinzmetal's variant angina as distinguished by coronary arteriography. Patients in one group have essentially normal coronary arteriograms and in several cases have now been documented to have spasm as the etiology of their pain. Patients in the other group have severe proximal coronary atherosclerosis similar to the patients described by Prinzmetal and reported recently by several authors. These patients are more likely to have exertional chest pain and positive treadmill exercise tests as well. Additionally, their history of angina is shorter and the pain may not be cyclic or recurrent. This distinction by history is not entirely reliable, however, since Prinzmetal's original report assumed that severe atherosclerosis was the etiology on the basis of several pathologic reports in his patient group. Despite this observation, he suggested that "... variant type of angina pectoris results from temporary occlusion of a large diseased artery with a narrow lumen. ..."

Coronary spasm could therefore be the underlying cause for the unprovoked nature of the chest pain in both patient groups. This hypothesis is strengthened by the observation of Guazzi et al that continuous electrocardiographic and hemodynamic monitoring in four patients with variant angina (two with electrocardiographic evidence of old myocardial infarctions) did not detect circulatory changes preceding the anginal attack. The inability to document a change in myocardial work load and oxygen requirements therefore also supports the proposed transient episodes of coronary spasm. This lack of preceding circulatory change is in contrast to a report by Roughgarden that "rest angina" secondary to atherosclerotic heart disease may be associated with preceding rises in blood pressure which could account for precipitation of the myocardial ischemia. We have also documented premonitory rises in heart rate or blood pressure during continuous hemodynamic monitoring in some patients with unstable or "preinfarction" angina.

Documentation of coronary arterial spasm does not necessarily explain the observed marked J point elevation during chest pain in either patient group. These electrocardiographic findings are commonly associated with myocardial injury of a more permanent nature, rather than with transient ischemic changes seen as ST depression on ECG. The reasons for this remain unclear, although one hypothetical explanation could be based on the very proximal or diffuse nature of the occlusion observed when spasm occurs in a normal or diseased proximal segment, resulting essentially in cessation of blood flow which in turn results in true, but temporary, cell and membrane injury to that area of the myocardium. Only the rapidly reversible spasm allows such immediate return of coronary flow that cell death does not follow. Support for this hypothesis actually comes from Prinzmetal's original report documenting transient ST elevation in the experimental dog during proximal occlusion of a large coronary artery. The ST elevation may represent a more severe form of "ischemia" when the cause is transient or infarction than when the cause is not transient. The etiology of the coronary arterial spasm in a normal vessel or increased tone in a diseased vessel is unclear. We suspect that it always is an area of atherosclerotic disease, whether mildly or severely occlusive.

Finally, we are in agreement with recent reports indicating that coronary bypass surgery may be an inappropriate consideration for certain patients with Prinzmetal's variant angina. Our experience suggests that aggressive prophylactic and therapeutic use of short- and long-acting vasodilators can provide dramatic results over significant periods of time.

References

Rupture of the Right Coronary Artery Due to Nonpenetrating Chest Trauma

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We describe a case of traumatic myocardial infarction due to rupture of the right coronary artery. Isolated rupture of a coronary artery is rare and often recognized only at autopsy. Absence of cardiac tamponade due to laceration of the pericardium was partly responsible for the relatively long survival time.

Injury to the heart and the great vessels after nonpenetrating trauma to the chest is a frequent occurrence and an inevitable penalty of modern traffic. Myocardial damage has been reported in 18 to 76 percent of the cases after blunt chest trauma.1 Myocardial contusion is mostly of minor importance and does not bear consequences.2 Among the rapidly fatal lesions, myocardial rupture and laceration of the great vessels are by far the most frequent.3

With the improvement in first aid and prompt evacuation, more patients with severe cardiac injuries now reach the hospital emergency room sooner and benefit from prompt recognition of the nature of their injuries and subsequent adequate treatment. This eventually proves life-saving.4

The following patient is presented because the exceptional concurrence of injuries was demonstrated at autopsy. As the patient survived for a few hours after the accident, interesting conclusions could be formulated as to diagnosis and treatment of such lesions.

CASE REPORT

A 62-year-old man suffering from mild diabetes, but without previous history whatsoever of ischemic heart disease, was involved in a car accident and sustained blunt trauma to his chest. On admission to the emergency room, half an hour after the crash, he was in severe shock. On inspection there were only a few skin lacerations. Blood pressure was 70/50 mm Hg, pulse rate was regular at 50 beats per minute. He was conscious, had tachypnea and complained of dyspnea and severe thoracic pain.

An electrocardiogram showed an acute inferior myocardial infarction with complete atrioventricular block (Fig 1). On chest roentgenogram, four rib fractures at least were visible, as well as closure of the left costodiaphragmatic sinus. The heart was slightly enlarged.

Significant laboratory findings included a hematocrit of 46 percent and moderately increased levels of SGOT and CPK (73 U and 158 U respectively).

![Electrocardiogram on admission showing acute myocardial infarction with complete atrioventricular block.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20951/ on 04/05/2017)