RELATION OF CORONARY EVENTS TO SPASM OF CORONARY ARTERIES,
PRECARIOUSNESS OF OBSTRUCTIVE LESIONS AND AVAILABILITY OF
COLLATERAL CHANNELS

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Sudden death, myocardial infarction and prolonged anginal pain without infarction are considered coronary events. As such the coronary event becomes one of the most lethal diseases to afflict mankind since over half of its victims are dead before they reach the hospital (1). In the hope of increasing understanding of the natural history of coronary obstructive lesions so that coronary events might be more accurately predicted and perhaps more reliably prevented, several clinical observations are presented that appear to be pertinent to the understanding of coronary events.

The first of these observations indicates that coronary artery spasm is a clinical reality that is, in all likelihood, the mechanism that produces angina arising out of rest or deep sleep with ST segment elevation in the ECG (Prinzmetal's variant angina) (2). The second presents evidence that coronary arteries totally obstructed and fed distally by collaterals are non-precarious lesions associated with good prognosis while local stenotic lesions are, by their nature, precarious since they are almost sure to progress and the more there are of such lesions the greater is the likelihood of a coronary event. Finally it will be shown that coronary collateral channels may be present prior to the development of coronary obstructive disease and may be rapidly activated to carry blood to an ischemic area of myocardium thus averting myocardial infarction in certain cases.

MATERIAL AND METHODS

The observations have arisen from clinical studies carried out over the last 12 years on patients referred to UCLA Medical Center,
Fig. 1. ECG strips taken from monitor scope during spontaneous attack of angina arising out of deep sleep. Lead is a transthoracic modified lead 2. Regression of changes after sublingual nitroglycerin.

Fig. 2. Left - ECG strips recorded during treadmill test during which patient walked for 3 minutes at 2, 2.5, 3, 3.5, and 4 miles per hour without developing anginal distress. Right - ECG strips recorded from Holter tape recording during nocturnal anginal episode during which the pain and ECG abnormalities arose out of a slow heart rate. Periods of 2nd degree heart block were noted.