A 59 year old man developed paroxysms of pain beginning in the right arm, and spreading to the right shoulder and the right lower jaw. During three of these attacks electrocardiograms were recorded that were entirely normal and identical to the tracing (October 22, 1952) made five years before the attacks began. No electrocardiograms had since been made in the absence of pain. Accordingly, when he was first seen by one of us (M.G.) an interim tracing (December 19, 1957) was made. Later on the same day a paroxysm of pain occurred during which another electrocardiogram (December 19, 1957 pain) was recorded. On the following day another interim record was made. On December 21, 1957 another attack of pain occurred.

In this case the electrocardiograms were normal during attacks of pain, and abnormal in the interim. Viewed superficially this is the opposite of what occurs in most cases of angina pectoris so that this phenomenon has been dubbed "paradoxical." Actually it is not paradoxical at all. The explanation is rather simple. Several attacks of angina had left a degree of "ischemia" presumably in the epicardial layers of the anterior wall of the left ventricle, as evidenced by the terminal inversion of the T waves in the precordial leads of the interim tracings. During an attack of angina, if and when the classical injury effects occur in the epicardial layers of the anterior wall of the left ventricle, the RS-T segments (and usually also the T waves) of the precordial leads are displaced upward. In this case, as they not infrequently do, they reverse the direction of the previously inverted T waves (of the precordial leads). In short, as in any case, the electrical effects of the current of injury are in a direction opposite to that of the electrical effects of ischemia in the same zone. In most cases of angina the affected zone seems to be endocardial.
Under this circumstance the current of injury results in downward shifts of the RS-T segments in the precordial leads and if a degree of ischemia is left after the attack the T waves are increased in height (in contradistinction to the effect of epicardial involvement). Since even large increases in the height of the T waves of the precordial leads cannot often be recognized as abnormal, the electrocardiogram is "normal" between attacks and shows abnormality (RS-T shifts) only during the attacks. Thus the so called "paradoxical" events shown in Figure 1 are simply determined by the fact that the affected area is in the epicardial zone rather than in the more usual endocardial zone. The smaller R waves in the anterior and left precordial leads in the last record suggest that some infarction has occurred but this was not confirmed by other laboratory findings. Variation in placement of the electrodes may account for this change.

The important practical point that is emphasized by this case is the following. In the investigation of pain in the chest it is important to record electrocardiograms during the attack, but it is also important to record the electrocardiogram between attacks. Actually, having succeeded in gaining an opportunity to make tracings during or shortly after an attack of pain, whatever the appearance of that tracing, one should administer nitroglycerin and continue to make records at short intervals until the tracing ceases to change. It is the series of changes that is diagnostic.

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