Although credit has been given to accurate diagnosis as one of the causes for the apparent increase of myocardial infarction, one must point out that important errors are still made respecting the diagnosis of myocardial infarction. These errors are especially important in industrial medicine. In any "heart attack" of industrial nature one must consider angina pectoris, coronary occlusion, coronary insufficiency, contusion of the heart muscle and, of course, other rare conditions affecting the myocardium.

1. Angina pectoris. Angina pectoris is believed to be a transient state of coronary insufficiency and pain without permanent myocardial change. This pain practically always appears in conjunction with physical or nervous strain and is believed to be due to the inability of the blood supply of the heart muscle to cope with the exertional demands. This direct relationship between exertion and angina is so well known to the medical and lay public that, by analogy, the erroneous idea that all heart pain is precipitated by strain has thoroughly embedded itself. The fine differential point which is not usually considered is that angina pectoris and coronary occlusion are manifestations of sclerotic coronary artery disease. However, coronary occlusion is not a complication of angina pectoris. This point is the basis for widespread confusion because of the similarity that exists between the pain of angina pectoris and that of coronary occlusion. The fact that persons may sometimes die of acute myocardial infarction due to coronary insufficiency without occlusion leads to further confusion. Angina pectoris by definition causes no permanent damage of the heart, and I do not believe it is a factor in the precipitation of coronary occlusion. In a legal sense one could state that an individual attack of angina pectoris which lasts for a period of two to three minutes may have been precipitated by the actual work being carried out by the individual at the time of the attack. However, as soon as this attack is concluded the effect of that work is terminated and industrial liability is also terminated. The employer should not be held responsible for future attacks of angina or possible coronary occlusion which might occur at some future date as a complication of the existing arteriosclerosis.

2. Coronary occlusion. Coronary occlusion is due to the occlusion of one of the branches of the coronary arteries. Horn and Finkelstein [4] and others have discussed the pathological nature of coronary occlusion. They point out that vascularization of the intima of the coronary arteries was found only in the presence of arteriosclerosis, and it was regarded as a sequela rather than the cause of the arteriosclerosis. As this vascularization increases, intramural hemorrhage of the sclerotic artery may occur. Such a hemorrhage may produce an acute occlusion or may serve as a site for thrombosis. The coexistence of recent and organized changes of some thrombi suggest that in some cases the process of coronary occlusion may be slow. In other words, the actual intramural hemorrhage which ultimately produces the occlusion may commence several days or weeks before the final occlusion of the vessel and subsequent myocardial infarction.

Osler's statement [5] in 1910 that "coronary occlusion was a disease of the upper bracket individuals, principally affecting persons engaged in professional life," made a profound impression. In the light of the discussion above, persons of the upper bracket would now receive better medical care and the cause of their deaths would

4. Horn, H., and Finkelstein, L. E.: Arteriosclerosis of the Coronary Arteries and the Mechanism of Their Occlusion, Am. Heart J. **19**:655 (June) 1940.

5. Osler, W.: The Lumleian Lectures on Angina Pectoris, Lancet **1**:697 (March 12) 1910.