with, especially if one does not fail to notice the often associated decrease in T-wave voltage in leads I and aVL, which are often present in these cases. I am in complete disagreement, however, with the interpretation of myocardial repolarization with reference to T-wave inversion as given by Dr. Nikolic, and stated as representing the opinion of most cardiologists. His assumption regarding both are incorrect. T-wave inversion does not “connote subendocardial rather than subepicardial ischemia.” By way of an abbreviated explanation, I offer the following: Normally, the blood supply to the subepicardium is greater than that of the subendocardium, and in the latter area, it is greatly influenced by the transmyocardial pressure gradient, thereby producing ischemia in the subendocardial layers more easily, and at times, more extensively.

In the intact heart, myocardial repolarization proceeds from epicardium to endocardium and depolarization from endocardium to epicardium. Since repolarization is mainly influenced by intact subepicardium, the recovery process is not delayed by subendocardial ischemia, resulting in a prolonged QT interval, and increased magnitude of the T wave (tall, upright T waves) resembling at times, those of hyperkalemia. Contrarily, repolarization in subendocardial ischemia is considerably delayed so that the repolarization process in the subendocardium begins before the usual direction is able to take place (reversed repolarization). The reversal of repolarization in subepicardial ischemia is typified by negative (inverted) T waves, a primary change in contrast to those that are classified as being secondary. If both, subepicardial and subendocardial ischemia are present, the changes produced by the former generally predominate.

Both ST segment elevation and horizontal depression are indicative of myocardial injury. Subendocardial injury is manifest by ST segment depression and subepicardial injury by ST segment elevation. When both areas are injured, it is the pattern produced by the subepicardial injury that predominates, but a mixture of both can occur depending on the extent of each as well as their location.

Admittedly, there is still much debate concerning the pathophysiology of reciprocal ST segment change, and it is now believed that these are of greater significance than formerly thought. The persistence of transient subendocardial injury changes (ST segment depression) more strongly predicts the presence of subendocardial infarction, since subendocardial injury is also transitory rather than persistent. Blocked (delayed) depolarization is one mechanism considered to be responsible for the production of subendocardial injury ST segment depression.

Transient subepicardial injury is much less frequent than subendocardial injury. The former is typified in cases of Prinzmetal’s angina. In contrast to subendocardial injury, subepicardial injury usually progresses to acute myocardial infarction; the exception being the cases of properly detected and treated Prinzmetal’s angina with little or no significant coronary occlusive disease.

Electrophysiologically, the following facts must be realized in order to properly address ST and T-wave deviations: The current of injury vector is directed toward the area of injury—from healthy tissue toward the injured tissue. Depressed ST segments will occur in the ECG leads overlying the area of subendocardial injury whenever the subendocardial layer of the ventricle is injured—the vector being directed from the normal epicardium to the injured subendocardial area. In cases of subepicardial injury, the vector is directed from the electrical center of the heart to the area of injured muscle, and the appropriate ECG leads will reveal an elevated ST segment. The electric potential of regions opposite to the injured area are reversed, and reciprocal changes may be present in these areas. The T vector always points away from an area of ischemia toward the nonaffected myocardium. Less appreciated by clinicians is the fact that persistently inverted T waves in the precordial leads that become upright or “normalized” following exercise actually indicates that subendocardial ischemia is occurring and not actual improvement in the pathophysiologic state.

In conclusion, the excellent studies by Sodi-Pollares and colleagues demonstrated that ST segment depression will be recorded in cases of subendocardial injury, and with subendocardial ischemia tall upright and peaked T waves will be present. If, however, a subendocardial infarction is present and it extends beyond the electrical endocardial surface, a Q wave will be produced.

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REFERENCES


To the Editor:

I thank Dr. RuDusky for his interest in my remarks. I find it difficult to envisage subepicardial ischemia or injury without much greater concomitant insult to the more hemodynamically challenged subendocardium. Whatever clinical markers exist for electrophysiologic models of epicardial ischemia, in practice they mean transmural ischemia. If T-wave inversion is ascribed to an ischemic process, the likely site of greatest damage will be subendocardial, whatever the genesis of the ECG pattern may be.

I have not seen or heard the term subepicardial ischemic pattern used to describe T-wave inversion in coronary artery disease, let alone pulmonary embolism. T wave is the most labile part of the ECG, its changes lacking specificity for the almost unlimited number of conditions in which they occur.

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Recommend Caution in Defining Risk Factors For Barotrauma in Divers

To the Editor:

We read with interest the report by Tetzlaff and colleagues titled “Risk factors for pulmonary barotrauma in divers” and the accompanying editorial by Bove. We would like to suggest these results and the recommendations contained therein should be interpreted with great caution. Of the six spirometric values measured in these subjects postinjury, only one purportedly