lar dysfunction with Dr. Goffredo Gensini and his associates and described my forthcoming editorial on this subject. At this meeting, Dr. Gensini kindly called my attention to a paper with a similar title that he had published several years ago, of which I was not aware.

Dr. H. E. Cohen of the Presbyterian–University of Pennsylvania Medical Center, Philadelphia, has, after reading our editorial, suggested to me another clinical setting in which prolonged, postischemic ventricular dysfunction may be operative. Dr. Cohen proposed that precipitation or worsening of cardiac power failure after a prolonged but successful resuscitation might be an example of global stunning, similar to that which occurs after cardiac surgery, which we described. This is a logical suggestion.

I am grateful to Drs. Gensini and Cohen for their interest, information and suggestions.

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References

More Stunned Myocardium

To the Editor:

When most have been absorbed in the measurement of infarct size and various means to reduce necrosis and salvage myocardium, it is significant that Braunwald and Kloner have added needed perspective, although not new, by defining the “stunned” myocardium. Previous studies3-5 have attempted to focus attention on the functionally deranged tissues that survive experimental myocardial infarction. Although such tissue can be considered to be salvaged as a result of reperfusion or development of collateral flow, its functionally abnormal properties can serve as the pathophysiologic basis for ectopia with the potential for sudden arrhythmic death. Recently, we described the electrophysiologic counterpart to the studies cited by Braunwald and Kloner. In the dog heart in which 30 minutes of occlusion are followed by reperfusion, ventricular tachycardia/fibrillation can be induced by stress pacing of the ventricles. Before occlusion or after 20 minutes of occlusion and reperfusion, such malignant arrhythmias cannot be induced by the same pacing regimen. In addition, 24 hours after the transient ischemic episode 40% of the dog hearts still respond to pacing with rapid sustained ventricular tachycardia leading to ventricular fibrillation. However, minimal or no necrosis can be shown.6

These studies may have important clinical implications, since the majority of subjects resuscitated from sudden arrhythmic death do not show acute infarction but do have severe coronary artery disease.7 8 The various clinical means to revascularize ischemic myocardium, such as coronary artery bypass and streptokinase thrombolysis, should consider the consequences of salvaging not only jeopardized, normal tissue but also provoking new potential for ectopic activity in ischemically damaged myocardium.

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Addendum

In the Appreciation in the December 1982 issue (Circulation 66: 1351, 1982), the name of Barry Greenberg, M.D., of Portland, Oregon, was omitted. Dr. Greenberg reviewed two manuscripts for Circulation from October 1981 through September 1982.
More stunned myocardium.
B J Scherlag

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