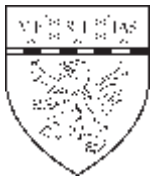
Harvard Medical School Department of Continuing Education and the Cardiovascular Division of the Department of Medicine, Brigham and Women's Hospital



Cardiology Rounds
August/September 2004

Glucose-Insulin-Potassium (GIK) for Acute Myocardial Infarction: Mechanisms of Action and Current Status

By Carl S. Apstein, M.D.

Objectives:

Physicians are well aware that myocardial ischemia is produced by an imbalance of coronary O_2 supply relative to the metabolic demands of the myocardium. In this issue of *Cardiology Rounds,* the metabolic basis of the energy production engine of the heart is reviewed. The reader will gain an understanding of the relationships between oxygen requirements and the use of glucose or free fatty acids as substrates of high energy phosphates. An improved understanding of myocardial substrates for myocardial adenosine triphosphate (ATP) synthesis will provide a rationale for potential clinical therapies for myocardial ischemia.

Test:

1. The heart synthesizes and utilizes approximately 5 kg of ATP per day.

   True ❑ False ❑

2. The heart contains enough ATP to function for only a few seconds if ATP synthesis ceases.

   True ❑ False ❑

3. Plasma glucose and free fatty acids (FFAs) are the major substrates for myocardial ATP synthesis.

   True ❑ False ❑

4. Ischemia markedly decreases ATP synthesis.

   True ❑ False ❑

5. An IV regimen of GIK increases myocardial glucose utilization, decreases plasma FFA levels, and decreases myocardial FFA uptake.

   True ❑ False ❑

6. In experimental animal studies of myocardial ischemia and reperfusion, GIK has been shown to decrease tissue damage.

   True ❑ False ❑

7. Results of clinical trials definitively support the use of GIK for all patients with acute MI.

   True ❑ False ❑

To receive AMA category 1 credit, you must correctly answer 60% of the test questions.

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This program was issued in August/September 2004.
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