In the dysrythmias shown in our figure 1, two appeared before ST-segment elevation reached its acme and therefore were classified as representing an occlusive type of dysrythmia. Some of the electrical abnormalities produced during the occlusive phase (i.e., progressive delay of subepicardial activation), are maintained during early phase immediately after coronary reperfusion. These may be an important factor explaining the persistence of dysrythmias after coronary flow resumes. 

NICHOLAS Z. KERIN, M.D. 
MELVYN RUBENFIRE, M.D. 
WALDEMAR J. WAJSZCZUK, M.D. 
Sinai Hospital of Detroit 
Detroit, Michigan

References

Exercise Capacity in Patients with LV Dysfunction

To the Editor:
The conclusion of Drs. Benge et al. that exercise capacity is normal in many patients with severe left ventricular dysfunction (Circulation 61: 955, 1980) depends on the definition of a "normal" exercise capacity. The authors define this as an exercise duration of greater than 11 minutes, based on a reference to Bruce et al. Their subjects, however, performed a Sheffield protocol, with two 3-minute stages preceding stage 1 of the Bruce test. Six of the 11 minutes of their protocol were thus occupied by stages 0 and ½ of the Sheffield protocol. This is equivalent to 5 minutes of a Bruce protocol, which is definitely not normal. Even their best subjects exercised for only 15 minutes, equivalent to 9 minutes on the Bruce protocol. The duration of exercise by itself need not correlate with exercise capacity. A constant, very light exercise can obviously be endured for a long time even in patients with reduced cardiovascular capacities. Bruce used the duration of exercise because it correlated well in his test with the maximum oxygen uptake (VO2), an objective and accepted measure of the exercise capacity.

Some simple calculations based on the Fick equation indicate internal inconsistencies in this paper. If the maximum VO2 is 34 ml/min-kg, the normal value Bruce gives for a sedentary male greater than 45 years old, and the maximum arteriovenous difference is 160 ml/l, the cardiac output would be 13.6 l/min. If the maximum heart rate were 170 beats/min, which is higher than the mean of 134 beats/min quoted for these subjects, the minimum stroke volume would be 80 ml/beat. If the ejection fraction for the subject exercising 15 minutes were truly 7%, as shown in figure 3 of Benge's paper, the end-diastolic volume would be 1140 ml. Even an estimate of VO2 two standard deviations below the predicted value yields an end diastolic volume in excess of 600 ml. Two factors may account for this absurd calculated end-diastolic volume: (1) the duration of exercise overestimates the max-VO2, especially if subjects leaned on the rail during the test, (2) the ejection fractions of 7% and 14% in the subjects who exercised for 15 minutes must be underestimated, especially because we are told that the cardiothoracic ratio was 0.46 in the patient with an ejection fraction of 14%.

Another problem with this study is semantic and concerns the definition of "severe" left ventricular dysfunction. VO2 correlates well with cardiac output, so subjects who exercise for 12-15 minutes must have obtained at least a moderate level of VO2, and thus at least a moderate cardiac output. Despite a low ejection fraction, can this really be called "severe" left ventricular dysfunction?

Finally, exercise capacity defined in terms of max-VO2 can sometimes appear to be normal in subjects with considerably decreased myocardial function because they may have started with a very high capacity. Thus, an athlete who once had a max VO2 of 60 ml/kg/min could lose 50% of his peak cardiac output and his max VO2 would be 30 ml/kg/min — still within normal range. A simple history will identify such subjects.

In conclusion, we feel that Benge's paper has failed to document a "normal" exercise capacity in patients with "severe" left ventricular dysfunction, but rather simply demonstrates that radiouclide ejection fractions are a poor predictor of the peak cardiac output and exercise capacity.

SHELDON A. MAGDER, M.D.
JOSEPH HUNG, M.D.
Cardiology Division
Stanford University Medical Center
Stanford, California

References

The authors reply:
To the Editor:
The major questions or comments put forth by Drs. Magder and Hung are:
(1) What is normal exercise capacity?
Bruce reported that the duration of exercise with the Bruce protocol for 117 normal subjects (mean age 51 years) varied from 4-15 minutes. The median value was 9 minutes. Four minutes of exercise using the Bruce protocol is equivalent to 10 minutes of exercise with the Sheffield protocol. Since about half of our patients with left ventricular ejection fractions less than 30% could exercise 11 minutes or longer (Sheffield protocol), their exercise capacity was within the normal range established by Bruce. It is important, however, to emphasize a point that we made concerning these normal values. We stated "normal exercise capacity in untrained individuals as established by Bruce is a relatively low level of oxygen consumption compared with the maximal oxygen consumption reached among trained athletes." The difference is semantic and concerns the definition of "severe" left ventricular dysfunction.

(2) Simple calculations by Drs. Magder and Hung indicate "internal inconsistencies" within our data.
One should hesitate to base substantive criticism on simple calculations. Rather than use estimated values to derive cardiac output and stroke volumes, we recently made direct measurements of hemodynamics and oxygen consumption in six patients with left
Exercise capacity in patients with LV dysfunction.
S A Magder and J Hung

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