
Meaning of Ejection Fraction After Subendocardial Resection

Dr Nath and coworkers' otherwise excellent contribution, "Regional wall motion analysis predicts survival and functional outcome after subendocardial resection in patients with prior anterior myocardial infarction" (Circulation. 1993;88:70-76), contains an error that is commonly made by the practitioner as well as by research workers, namely, the assumption that the ejection fraction in isolation has meaning. It is a truism that no fraction has meaning unless the whole is known.

Thus, simple inspection of Fig 3 indicates that the stroke volume of B is larger than that of A, provided that the diastolic volumes are equal. If the volumes are not equal, the two stroke volumes can be identical or differ widely. The stroke volume will also be affected by the heart rate.

Significant aneurysmectomy does not change the basal cardiac output because this depends on the metabolic needs of the peripheral tissues. After the operation, the efficiency of the heart will increase because it no longer is required to carry the load of the aneurysm and its contents. The following data from two patients attending our cardiac clinic illustrate both the lack of meaning of an isolated ejection fraction and a simple physiological method to assess the safety of aneurysmectomy.

A 50-year-old man suffered a myocardial infarction on September 10, 1970. He remained asymptomatic until April 1971, when he developed shortness of breath after walking up one flight of steps and also after walking two city blocks. Cardiac catheterization revealed a huge ventricular aneurysm occupying approximately the distal half of the left ventricle. The global ejection fraction was 14%. The cardiac output was normal. Because the cardiac aneurysm contributed very little or nothing at all to the cardiac output, there was no hesitancy in performing aneurysmectomy. After operation the ejection fraction rose to 58%, and there was no change in the basal cardiac output. After convalescence, he returned to work and is still working. He is asymptomatic. His echocardiogram indicates that left ventricular function has deteriorated during the past few months. He was last seen on July 19, 1993.

A 36-year-old man, an amateur tennis champion, suffered a myocardial infarction on August 12, 1969. Except for repeated bouts of Dressler's syndrome over a 14-month period, he remained asymptomatic and continued playing tennis until 1985, when a routine chest film showed a curvilinear calcification on the lateral film. The cardiac silhouette was mildly enlarged. Cardiac catheterization revealed a huge aneurysm occupying the distal half of the left ventricle. The global ejection fraction was 18%. The basal cardiac output was normal. A first-pass radionucleotide study revealed a global left ventricular fraction of 14%. The contractile portion was estimated to be 58% and its volume approximately 180 mL. An electrocardiographic stress test, using supine bicycle protocol, was entirely normal. He developed no pain or arrhythmias. He stopped exercising at 80% of the predicted peak heart rate because of leg cramps, and his blood pressure rose from 140/90 to a maximum of 205/108 mm Hg. He continued playing tennis until May 3, 1989, when he developed a prolonged episode of ventricular tachycardia documented at a local hospital. The ventricular tachycardia terminated spontaneously before electric shock could be applied. He was transferred to Temple. Pharmacologic electrophysiological studies failed to prevent inducible ventricular tachycardia. A first-pass radionucleotide study showed no change compared with the study done in 1985. The overall ejection fraction was 14%. Dr Frank Marchlinski of the University of Pennsylvania was called in consultation and decided that a patient with such excellent cardiac function should be treated medically in order to avoid a surgical mishap. He continued playing tennis until July 5, 1993, when he developed a cough and noted a slight decrease in effort tolerance while playing tennis. His local physician thought that the patient had a cold, but I thought it was important to determine whether cardiac function had deteriorated or not. A radionucleotide study revealed an overall ejection fraction of 10%. The ejection fraction of the contractile portion of the ventricle was 55%. The regional stroke volume was 94 mL per cycle, and his cardiac output was estimated at a little over 6 L per minute.

The authors' number is an average result of an unknown fraction of the global ejection fraction and an assumed similar fraction of the global cardiac volume. The method described above is more physiological and is applicable for the individual patient. I do not have sufficient patients to determine minimal function activity of the contractile part of the ventricle that is compatible with recovery from aneurysmectomy. It probably can be determined by a simple stress test. The authors may be able to provide a clue if they supply the readers with not the average but the individual cardiac outputs of all their patients and an estimate from their angiographic studies of the ejection fraction of the contractile part of the ventricle.

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Reply

We would like to thank Dr Soloff for his interest in our article.1 Our data support his argument that global ejection fraction is not the best measure of ventricular function in the presence of an aneurysm. Dr Soloff makes the point that calculating the ejection fraction of the aneurysmal portion of the left ventricle is a more physiological measure of ventricular function. In fact, as we mentioned in the "Discussion" section of our article, Garan et al2 reported that the ejection fraction of the nonaneurysmal segment calculated as an "excess ejection fraction" was a significant predictor of operative survival after subendocardial resection. However, a significant limitation of this technique is that it requires the presence of a visually discrete aneurysmal segment to be an accurate measure of regional left ventricular function. A significant number of patients in our series had a dysskinetic anteropapical segment in which the visual boundaries between the dysskinetic and nondysskinetic segments of the ventricle were difficult to define (as illustrated in Fig 3B of our article). We therefore did not measure the ejection fraction of the contractile portion of the left ventricle in our series but rather used centerline chord motion analysis to calculate regional wall motion. This method is a validated and relatively easy technique to measure regional left ventricular function that can be used in all patients.3

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References


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