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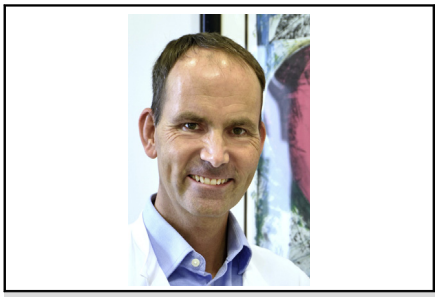
ADULT

Commentary: The wisdom of a Nobel laureate and surgical ventricular reconstruction

Torsten Doenst, MD



Torsten Doenst, MD

All it takes is just "one good experiment."

—Hans A. Krebs (1900-1981)

Hans Krebs made this statement when explaining that a convincing treatment effect (he suggested insulin-induced glucose uptake of cells) can be witnessed with a single experiment. and that there is little need for endless repetition until statistical significance is reached. In clinical practice, where causal relationships are difficult to establish, such repetition is often needed; however, we may have significant treatment effects that are difficult to explain (as for the survival impact of surgical revascularization[1]) or a suggested mechanism that is convincing but for which the treatment effect appears clinically irrelevant (as for volume reduction in surgical ventricular reconstruction [SVR][2]).

CENTRAL MESSAGE

Surgical ventricular reconstruction, although ineffective according to STICH, may be helpful in selected patients, possibly those with classic aneurysms or high relative posterior wall thickness.

BUT WHAT IF WE HAVE "ONE GOOD OPERATION"?

Let us look at the case of a 59-year-old woman, referred for ventricular assist device implantation after myocardial infarction from a left anterior descending coronary artery occlusion and an ejection fraction of 10% to 15%. She was in New York Heart Association class 4 (Intermacs class 4), with mild mitral regurgitation. Except for the occluded left anterior descending coronary artery, the coronaries looked normal. Her ventricle revealed a large anterior aneurysm, with dyskinesia in the anterior wall, the apex, and significant parts of the interventricular septum.

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Instead of implanting an assist device, my team performed SVR (ie, modified aneurysmectomy with volume reduction) as an isolated procedure. The patient was discharged 6 days after surgery in New York Heart Association class II, with an ejection fraction of 40%. This was "one good operation."

The Surgical Treatment for Ischemic Heart Failure (STICH) trial, however, demonstrated an absence of beneficial effects of SVR in patients with dilated ischemic cardiomyopathy.[2] Since then, SVR has nearly disappeared from operating rooms worldwide. But if that is so, why was the effect of our operation so clear-cut?

The underlying mechanism of SVR is supposed to be the reduction of wall stress on the remaining viable myocardium, allowing it to pump more efficiently.[3] In this issue of the *Journal*, Fantini and colleagues[4] demonstrate, in an outstanding analysis of 43 patients with restrictive filling patterns before SVR, that half of their patients had a sustained benefit of SVR on ventricular function and symptom relief. The suggested mechanism thus may be correct. They also demonstrate, however, that the other half of this patient group did not benefit from SVR. They did not further specify the morphologic characteristics of the reconstructed ventricles, but it is tempting to speculate that those patients with higher relative wall thickness and better postoperative function may have been those similar to our patient with the classic aneurysms and rather normal remaining myocardium. The volume reduction then relieved wall stress and allowed more efficient and sustained contractile function, whereas in patients with thinned (and possibly already fibrosed) myocardium, volume reduction does not help. It is striking to note that in STICH, patients with classic aneurysms were a minority.[2,5] Is it possible that we did not assess the right patients in STICH? If we combine the wisdom of Hans Krebs with the results of Fantini and colleagues[4] and our own "one good operation," we may actually need "one new trial" after STICH, but this time with more specific indications for SVR.

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