

Selection for Ventricular Restoration: A Caution

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SNOWMASS, COLO. — Many cardiothoracic surgeons are not exercising due care in selecting patients for surgical ventricular restoration—and the result is a disturbingly high operative mortality, Dr. Andrew S. Wechsler said at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

Surgical ventricular restoration (SVR)

entails reshaping the failing, distorted, spherical left ventricle into a normal elliptical shape by excluding noncontractile scar tissue and inserting a synthetic patch.

A worldwide experience totaling thousands of patients shows that when SVR is applied to the correct population with advanced ischemic cardiomyopathy, it results in excellent restoration of ventricular function with an operative mortality of 3%-6% and a 5-year survival in excess of 80%.

That is particularly impressive in a pop-

ulation of heart failure patients with otherwise dim prospects because they are failing medical therapy and realistically have only a tiny chance of receiving a heart transplant, said Dr. Wechsler, who is professor and chair of cardiothoracic surgery at Hahnemann University Hospital, Philadelphia.

But a different set of outcomes was noted in a recent series comprising about 800 patients in a Society of Thoracic Surgeons registry who underwent SVR.

In a manuscript that Dr. Wechsler had culled from the STS database, the operative mortality in a very contemporary series was 13%. "I was incredibly concerned about this completely out-of-place high operative mortality voluntarily reported by the surgeons doing these operations," commented Dr. Wechsler, who is editor of the *Journal of Thoracic and Cardiovascular Surgery*.

In going over these "rather sobering" data in search of an explanation, he found that one factor jumped out: poor patient selection. Nearly 40% of patients had SVR within a few days following a large MI. Most operative deaths occurred in this subgroup.

"I can't stress enough that this operation is designed for the chronically remodeled ventricle and not as a treatment for profound heart failure shortly after an MI; those patients shouldn't be referred for this operation. I think that we've done a bad job of getting this message out, when we find so many patients in that series fell into this very, very adverse category," he continued.

Experience has shown that the best candidates for SVR

have akinesia or dyskinesia over the anterior ventricle, viable myocardium over much of the high basilar part of the anterior ventricle, and an end-systolic volume index of 90 mL/m² or less.

The ideal candidate also has

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a pulmonary artery pressure of less than 40 mm Hg, Q waves on the anterior ECG leads, demonstrable remote myocardial viability on magnetic resonance imaging or dobutamine echocardiography, and no mitral regurgitation.

"As you move away from the ideal patient, operative mortality increases and the efficacy of the operation decreases substantially," the surgeon cautioned.

Until recently, surgeons thought the ideal SVR candidate had isolated left anterior or descending coronary artery disease. But this conviction predated current improved methods of myocardial protection and technical operative advances. In 2005, surgeons at Johns Hopkins University reported the successful extension of SVR to a population of patients with advanced heart failure and evidence of multiterritory infarction—and with an acceptably low operative mortality.

Surgeons who do surgical ventricular restoration place much less emphasis on preoperative left ventricular ejection fraction than on careful determination of ventricular volume and shape.

A strong candidate for SVR might have an ejection fraction of 17%-18%, often climbing to 38%-40% postoperatively, which is sufficient gain to remove the indication for an implantable cardioverter defibrillator. ■

DEFINING AQUARESIS

Because of the role AVP plays in regulating sodium and water levels, blockade of AVP receptors would clear excess water without contributing to electrolyte imbalance. Such an approach may be advantageous compared with current therapies and could result in a new treatment modality: "aquaresis," the electrolyte-sparing excretion of free water.^{2,4}

The effects of AVP are mediated by three receptor subtypes: V_{1a}, V₂, and V₃.

The V_{1a} receptors are located primarily in vascular smooth muscle cells and play an integral role in vascular tone. V₃ receptors are expressed by cells in the anterior pituitary and are involved in the regulation of corticotropin release. The V₂ receptors are located in the

collecting ducts of the kidneys and regulate free-water reabsorption.⁹ Blocking the V₂ receptors promotes aquaresis and thereby normalizes serum sodium levels.⁴

The imbalance of sodium and water levels remains a serious condition in hospitalized

patients—a condition that is often underdiagnosed and that can exacerbate underlying disease.^{2,7} Current therapies for hyponatremia do not directly

address AVP, one of the hormones responsible for regulating sodium and water levels.²

However, research is now being conducted into AVP-receptor blockade, with the potential to arrive at a more efficient and effective means of managing sodium and water imbalance.

"AQUARESIS": THE ELECTROLYTE-SPARING EXCRETION OF FREE WATER

References: 1. Verbalis JG. Disorders of body water homeostasis. *Best Pract Res Clin Endocrinol Metab.* 2003;17:471-503. 2. Goldsmith SR. Current treatments and novel pharmacologic treatments for hyponatremia in congestive heart failure. *Am J Cardiol.* 2005;95(suppl):14B-23B. 3. Nielsen S. Renal aquaporins: an overview. *BJU Int.* 2002;90(suppl 3):1-6. 4. Wong LL, Verbalis JG. Vasopressin V₂ receptor antagonists. *Cardiovasc Res.* 2001;51:391-402. 5. Janicic N, Verbalis JG. Evaluation and management of hypo-osmolality in hospitalized patients. *Endocrinol Metab Clin North Am.* 2003;32:459-481. 6. Goh KP. Management of hyponatremia. *Am Fam Physician.* 2004;69:2387-2394. 7. Movig KLL, Leufkens HGM, Lenderink AW, Egberts ACG. Validity of hospital discharge International Classification of Diseases (ICD) codes for identifying patients with hyponatremia. *J Clin Epidemiol.* 2003;56:530-535. 8. Miller M, Morley JE, Rubenstein LZ. Hyponatremia in a nursing home population. *J Am Geriatr Soc.* 1995;43:1410-1413. 9. Verbalis JG. Receptor antagonists. *J Mol Endocrinol.* 2002;29:1-9.