Ventriculectomy. A Direct Application of Laplace's Law

At the dawn of the 19th century, M. Lapace presented his theory of capillary attraction to the Institute of France, in which his law relating wall tension of a thin-walled sphere or tube to radius and cavity pressure first appeared. Nearly two centuries later, this law appears to have found its most direct application in a novel surgical therapy for congestive heart failure initially described by Batista et al[1], called ventriculectomy. In this issue, Bellotti et al[2] report on cardiac chamber mechanics in 11 patients with severe dilated cardiomyopathy (DCM), before and after partial removal of a portion of their ventricular wall. This is not performed as a means of aneurysm resection, since the excised wall behaves no differently from any other portion of the chamber left behind. Rather, this procedure aims at directly reversing a degree of chronic chamber remodeling and dilation associated with DCM. Since chamber volumes are reduced without directly altering wall thickness or cavity pressure, wall tension is diminished. Furthermore, if cardiac output is maintained, then by ejecting from a smaller starting (diastolic) volume, both fractional shortening and ejection fraction will increase. Chamber volumes are also inversely related to Emax, an index of chamber contractility, so that this index might also increase after ventriculectomy. All these changes were indeed observed by Bellotti et al.

There were several changes which are less predictable on the basis of simply removing a portion of the LV wall. For example, chamber end-diastolic pressure and elastic stiffness were both reduced. In addition, wall thickness was intriguingly increased. The mechanisms for these change need further exploration. Reducing chamber size alone generally **increases** measures of chamber diastolic stiffness - assuming no actual change in material porperties. Indeed, in an ACE-inhibitor atrial, reverse remodeling was associated with a slight increase in chamber diastolic stiffness[3]. Furthermore, one would not anticipate wall thickness to rise by 30% simply by removing part of the chamber and sewing it back together again. Potential roles of post-operative therapy, including pericardial release and volume status need to be sorted out.

The concept that reversing dilation and remodeling of the failing heart should be an important therapeutic goal has growing support[4]. Dilation is generally viewed as an adaptive response to reduced cardiac performance, but it quickly becomes self-sustaining, and contributes to the decline in chamber function as chamber stresses outstrip the capacity of the muscle to match them. Limiting or reversing this process is likely an important element of sucessful therapy. In a recently reported substudy of the SOLVD trial, Greenberg et al[5], presented data from 301 patients randomized to placebo or enalapril therapy, showing that ACE-inhibitor treatment prevented the ongoing chamber dilation and hypertrophy observed in the placebo group. This supported earlier data from a smaller substudy employing nuclear or contrast ventriculography[3]. While encouraging, the effects on chamber remodeling were very small, and the drug failed to reverse the remodeling process, but rather acted to inhibit further progression. Furthermore, the role of angiotensin II inhibition or kinin activation due to ACE-inhibitors on the remodeling process remains unanswered.

There are other mechanical methods for reversing chronic chamber remodeling. Cardiomyoplasty is one such procedure, in which the heart is wrapped by a latissimus dorsi muscle that is then electrically conditioned and stimulated so as to assist chamber contraction. Recent preliminary data have suggested that a major mechanism of benefit from this procedure is it passive constrait on the heart and ability to *reverse* chronic chamber remodeling[6]. Another method currently undergoing chronic clinical trials is the left ventricular assist device (LVAD), which near totally unloads the left ventricle. When measured at time of explant, these unload hearts (3-5 months of treatment) demonstrated normal diastolic pressure-volume curves at normal chamber volumes, indicating a substancial reversal of chamber remodeling[7].

Ventriculectomy provides arguably the most direct approach to reversing chamber dilation and remodeling, by removing a part of the muscle wall. Preliminary data are intriguing, but many important issues need to be resolved. The acute and sub-acute mechanical changes reported in this issue, and in a prior study of seven patients[8] are helpful, but are in many ways predictable based on the direct intervention that reduces cavity volume. Long term (at least 1 year) data using similar measurements, and more importantly, on morbidity and mortality are needed to establish the utility of this approach. As with other surgical therapies, it will be very difficult to define the placebo effect; thus, function class and subjective symptoms must be viewed cautiously. Objective data on survival, metabolic exercise capacity, and repeat hospitalizations, as well as having hemodynamic data evaluated by individuals blinded as to patient status, will be critical in order to assess the efficacy of this procedure. Only with such carefully performed studies will we learn if theacute mechanical results nicely described by Bellotti et al translate into a sustained clinical benefit, and if Laplace's law proves to be as therapeutically important as we had postulated.

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