cape the vagaries of septal motion, we also attempted to arrive at a filling index which embraced the total intracardiac dimension, and again were frustrated because of lack of simultaneous satisfactory imaging of the right ventricular (RV) anterior wall endocardium and left ventricular (LV) posterior wall endocardium. Thus, the results we reported were the most practical index we could find. As Sanderson and Adesanya note, it is subject to error if excessive swinging of the heart occurs independent of cardiac filling. Just how important this will prove to be awaits the test of time as other researchers in this area report on constriction. We note that the index reported by the correspondents contains a fair range of overlap between their normal group and construction when $\pm 2 SD$ are considered.

In response to Dr. Shubrooks, he correctly notes that three of our patients with constriction did not have elevated wedge pressures. It is well-known that left- and right-heart filling pressures in constric-
tive pericarditis are volume-dependent. All of our patients were referred because of ascites and edema; hence, their basic problem was not occult. Many had been successfully diuresed before arriving at our institutions. In all of these patients, the right-heart pressures were clearly abnormal and not "slightly elevated," as he suggests. A fluid load was not necessary to produce abnormal pressure waveforms, as is the case in "occult" disease. The patient (PC) with the lowest wedge pressure (8 mm Hg) and a right atrial pressure of 12 mm Hg was referred to us after extensive diuretic therapy. After his pericardietomy the right atrial pressure fell to 2 mm Hg as confirmed by our figure 4.

We agree that WB is the most atypical of our patients; while he had right-sided diastolic pressure equilibration, his LV end-diastolic pressure was higher than the right-sided pressures. The discrepancy is in part related to reporting procedures — our right-sided pressures were recorded from a right-heart pullback. The left-heart pressure was recorded later in the case. Simultaneous RV/LV pressures at that time indicated RV diastolic pressures to be lower, but always within 4 mm Hg of LV diastolic pressures. This is an accepted difference by published standards. Because of the presence of pericardial calcification and the equilibration of right-heart diastolic pressures, he was felt to have constriction despite the somewhat deviant LV end-diastolic pressure. This patient was the only one in our series whose LV posterior wall endocardial movement fell within the normal range. Elimination of this patient, if anything, would strengthen, rather than weaken, our observations. We did not feel that eliminating him based on the one pressure observation was justified.

The problem of restrictive cardiomyopathy is discussed in the last paragraph of our paper. Patients with this diagnosis are easy to talk about but hard to find. We think our data has been presented accurately and clearly. In our attempts to minimize "voluminous medical literature" to please "most readers," we have omitted some of the considerations Dr. Shubrooks raised.

A. GENE VOELKEL, M.D.
DAVID A. PIETRO, M.D.
EDWARD D. FOLLAND, M.D.
MICHAEL L. FISHER, M.D.
ALFRED F. PARISI, M.D.
VA Medical Centers
Baltimore, Maryland and West Roxbury, Massachusetts

The authors reply:

To the Editor:

In our article1 there is an unfortunate typographical error (see Corrections in this issue) in table 1 for the pre-pericardiectomy end-diastolic subaortic interventricular septal thickness: 1.7 cm should read 2.7 cm. Therefore, the interventricular septal thickness decreased by 1 cm after septectomy. Dr. Shubrooks states that 1 mm is "no change" and 3 mm is a "questionable change" in interventricular septal thickness. Although this can be a matter of judgment, the authors point out that an article by Popp et al. documents that changes of 1 mm or greater by M-mode echo in wall thickness is significant. The data clearly demonstrate group changes for the interventricular septal-to-posterior wall ratio, systolic anterior motion and mitral valve E-F slope with significant $p$ values ($< 0.001$, $< 0.025$, $< 0.001$, respectively). We believe that statistically assessing changes for the group is the most appropriate way to analyze such data.

The authors believe this study does add new information to the medical literature in that 1) previous reports have, in fact, not shown the expected consistent postoperative thinning of the interventricular septum as found in our patients and 2) there no other published studies examining post-pericardiectomy interventricular geometry with two-dimensional echocardiography.

Dr. Shubrooks may not realize that the editorial board of Circulation reviewed our manuscript very critically, that our study had three reviewers and went through two revisions before it was accepted for publication. The authors share Dr. Shubrooks' concern for "inaccuracy and overenthusiasm" in the medical literature; however, the same concerns may also apply to criticizing the medical literature. If it is true, as Dr. Shubrooks points out, that "most readers cannot review each reported study in detail," they have good reason to trust the review process and the broad conclusions reached by the authors. Dr. Shubrooks' letter itself bears the marks of overstatement which we have tried to avoid.

JAY N. SCAPIRA, M.D.
RICHARD L. POPP, M.D.
Cedars-Sinai Medical Center
Los Angeles, California 90048
and Stanford University
Stanford, California 94305

References


LAD Graft vs GCV Flow

To the Editor:

The article by Pepine et al. regarding comparison of the left anterior descending (LAD) graft flow and the great cardiac venous (GCV) flow provides useful information, because their method of using the LAD graft flow as a substitution of the LAD flow in the patients with an obstructed LAD seems to be the only available one in man as an in vivo validation study. When we analyze the partitions of the GCV blood, it is not an only drainage from the LAD graft but it includes the part of the drainages from the septal arteries and the diagonal branches of the LAD which take off before the occlusive lesion, and about 70% of the LAD graft flow.2

The amount of contribution from the first two components depends on the site of the graft at the LAD of which information was not available in the article. Therefore, 5–12 ml/min flows in the GCV flow even during the LAD graft occlusion may represent the drainage from these arteries rather than collaterals. If these values are subtracted from the GCV flow, it can be concluded that GCV

References

LAD graft vs GVC flow.
H Nakazawa

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