g/m² for group A patients (good result) (p < 0.05). Both groups had significantly greater left ventricular masses than our normal subjects (97 ± 4 g/m²). Thus our data are directly opposite to those of Dr. Razzolini and his colleagues; our patients with a poor result had the highest left ventricular masses, whereas their patients with the poor result had the lowest left ventricular masses. Dr. Razzolini's data suggest that his patients had relatively acute mitral regurgitation. In such patients, left ventricular mass would not be expected to increase⁵ and left ventricular function should be relatively normal. Their findings of a "normal" ESS/ESVI ratio in their nonsurvivor group in fact suggests that left ventricular function was not severely impaired in this group.

We maintain that patients with chronic symptomatic mitral regurgitation usually have left ventricular dysfunction. The amount of this dysfunction is frequently underestimated by standard ejection phase indexes of left ventricular function, such as ejection fraction. We believe that the relationship of volume and afterload of end-systole is a more precise way of evaluating ventricular function in patients whose disease process imposes abnormal loading conditions upon the left ventricle.

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References

Valsalva Termination of Ventricular Tachycardia

To the Editor:

Waxmann et al.¹ reported Valsalva termination of ventricular tachycardia (VT). We would like to describe two additional patients whose VT was terminated by Valsalva maneuver (VM).

The first patient, a 28-year-old male, was admitted to the hospital in July 1963 because of viral myocarditis. He had moderate cardiomegaly and the ECG during sinus rhythm revealed left bundle branch block. He developed sustained VT terminated repeatedly but transiently by VM (fig. 1). Approximately 2 hours after i.v. digoxin administration, VT was abolished. The second patient, a 66-year-old male, was admitted to the hospital in June 1981 because of acute subendocardial infarction. The ECG during sinus rhythm showed left bundle branch block and left-axis deviation. He developed sustained VT that was terminated transiently several times by VM; the last attempt abolished VT completely.

The diagnosis of VT was established by the presence of fusion beats in both cases, and in the second case it was further confirmed by electrophysiologic study. In both instances, termination of VT occurred during the second phase of VM. The first patient had one episode of transient atrial tachycardia shortly after completion of VM, which might have acted as overdrive mechanism (fig. 2). All 15 patients reported by Waxman et al. had VT with a left bundle branch block morphology; in contrast, both of our patients had VT with a right bundle branch block morphology.

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Figure 1. Lead I of the ECG (continuous tracing). Upper strip shows ventricular tachycardia (VT). Middle strip shows transient termination of VT during phase 2 of the Valsalva maneuver (VM). A transient atrial tachycardia with rate faster than VT developed shortly after completion of VM and is followed by sinus rhythm. The bottom strip shows sinus rhythm changed to VT. f = fusion beat.

Figure 2. Lead V₁ of the ECG. The upper strip showed transient termination of ventricular tachycardia by the Valsalva maneuver. The lower strip showed complete termination of ventricular tachycardia by the Valsalva maneuver.
To the Editor:

The recent excellent article of Borow and co-workers (Circulation 64: 878, 1981) regarding the abnormal response to afterload stress of the systemic ventricle of postoperative patients with d-transposition of the great arteries (TGA) as compared to the normal response of this ventricle in similar patients with tetralogy of Fallot (TOF) and ventricular septal defect (VSD) has been read with great interest by us. Their data showed that in response to an increased afterload there was a smaller increase in the minute work index for a similar increase in the end-diastolic pressure in TGA than in VSD or TOF, indicating a decreased pump function of the systemic ventricle in TGA, as the authors suggest. This is supported by the greater control end-diastolic volumes of the systemic ventricle in TGA patients compared with those with VSD or TOF.

We would like to suggest, however, that depressed systemic pump function may not be the only hemodynamic deficiency in patients with TGA.

In recent publications from this laboratory,1,2 the normal function of the left ventricular mechanoreceptors (stretch or baroreceptors) has been shown to be the continuous, reflex matching of left ventricular myocardial contractility and the peripheral resistance, e.g., the increase in contractility during exercise decreasing the peripheral resistance, a function useful in preparing the systemic vascular bed to receive the augmented left ventricular output. Whereas the systemic ventricle in TOF and VSD is the anatomic left ventricle, in TGA it is the anatomic right ventricle, which has been shown experimentally to be unable to cause appreciable alterations in the peripheral resistance when stimulated in the same manner as the left ventricle,3 apparently because of a paucity of mechanoreceptors. In TGA, the anatomic left ventricle, which would be expected to be well supplied with mechanoreceptors, leads to the low-resistance pulmonary circuit and is therefore not exposed to changes in the systemic afterload. This anatomic mismatching of the left ventricular mechanoreceptors with the pulmonary instead of the systemic vascular bed would deprive these patients of the normal left ventricular contractility–peripheral resistance matching, and may be a factor in the abnormal response to the methoxamine afterload of postoperative patients with TGA as compared with those with TOF and VSD, reported by Borow and colleagues, as well as in the abnormal response to exercise reported in TGA patients.4

Thus, we would expect patients in whom the anatomic right ventricle is connected to the systemic bed (as in TGA) to be functionally compromised to a greater degree than patients in whom the anatomic left ventricle is connected to the systemic bed (as in VSD and TOF) for both intrinsic (i.e., pump function) and extrinsic (i.e., reflexive) reasons.

With regard to the clinical implications of the anatomic mismatch in TGA, the Jatene, or "switch," operation would, at first thought, be preferable from both of the above standpoints. However, this procedure, which involves severing and reimplanting the great vessels to their appropriate ventricles, may result in transection of nerves carrying the mechanoreceptor reflex, thus abolishing it. Two considerations should be mentioned. First, comparison of pre- and postoperative manual compression of the systemic and pulmonary ventricles at both types of surgery for TGA may be informative. Absence of a systemic hypotensive response from compression of the systemic ventricle preoperatively supports our hypothesis, as manual left ventricular compression in patients on cardiopulmonary bypass for coronary arterial surgery has consistently elicited a systemic hypotensive response (Leonard JJ, Nicoloff DM, Fox: unpublished data). The ability to obtain a systemic hypotensive response from compression of the systemic ventricle after surgery may augur a favorable exercise response and, therefore, a favorable prognosis.

Second, postoperative evaluation of the response to exercise of TGA patients, with the above physiologic considerations in mind, may help to clarify the pathophysiologic mechanisms and appropriate treatment of this cardiac abnormality.

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