

## LETTERS TO THE EDITOR

### Pulsus Alternans: A Systolic Phenomenon?

Hess and colleagues have reported valuable observations on pulsus alternans in severe aortic valve disease (1). However, they perhaps made a too sweeping conclusion in describing pulsus alternans as "a systolic phenomenon." Their cases all had severe aortic valve disease (with or without associated lesions) and, therefore, perhaps their conclusions should be restricted to just this group. Diastolic as well as systolic abnormalities have been reported in pulsus alternans in mixed groups of patients and in animals.

Using noninvasive methods (relatively crude compared with those of Hess et al., nevertheless precise), we demonstrated that total diastolic time for strong and weak beats was statistically equal, although the apparent "filling" period preceding the weak beat was shorter owing to encroachment by the isovolumic relaxation period of the strong beat (2). More recently, Carlson and Rapaport (3) demonstrated that the strong beat of pulsus alternans was due to augmented inotropy, but the weak beat was due to reduced diastolic volume—a Frank-Starling mechanism; prolongation of systole of one beat delayed diastole of the next beat such that the next systole prematurely terminated rapid filling. In echocardiographic studies, D'Cruz et al. (4) observed alternation of the diastolic position of the left ventricular posterior wall. Finally, the occurrence of precisely the same cycle length for strong and weak beats may also be a feature of Hess's particular patient group. We (2) and others (mostly recently Schuster and Nanda [5]) noted small and consistent, though not necessarily statistically or physiologically significant, cycle length alternation.

These remarks are made not in direct criticism of the work of the distinguished authors, but rather to focus more carefully the conclusions of their excellent study.

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### Reply

Spodick points out that pulsus alternans not only is "a systolic phenomenon," but also is associated with changes in diastolic time intervals. We fully agree because it is obvious that the length of diastole as taken from mitral valve opening to end-diastole is likely to change when systole gets shorter or longer during pulsus alternans. In Table 1, we have added some additional data on systolic and diastolic time intervals in the 12 patients with aortic valve disease described in our report. These data show that the cycle length in pulsus alternans changes slightly although not significantly; the systolic ejection and isovolumic relaxation time become longer during the strong beat than during the weak beat, whereas the diastolic filling period—as pointed out by Spodick et al.—is shorter after the strong beat and longer after the weak beat (Ref. 2 of Spodick and Table 1). Even when these time intervals are alternating during pulsus alternans, one has to be very careful to draw conclusions as to the basic process of relaxation and diastolic function. For instance, the prolongation of the isovolumic relaxation time during the strong beat does not indicate an impaired relaxation. It is just due to the higher aortic closing pressure in this beat at an unchanged rate of pressure decay (Table 3 of our report). The same arguments are also true for the diastolic filling period: a decrease in diastolic filling time after the strong beat is compensated by an increase in the speed of diastolic filling (Table 1). The increase in filling rate can be explained by the decrease in end-systolic volume which leads to a higher elastic recoil. Hence, the improved systolic function during the strong beat appears to be responsible for the improved diastolic filling. We have, however, no indication that active relaxation as assessed from the rate of left ventricular pressure decay is altered during pulsus alternans. Moreover, the passive diastolic properties of the left ventricle

**Table 1.** Systolic and Diastolic Time Intervals in Patients With Aortic Valve Disease and Pulsus Alternans

	Strong Beat	Weak Beat
RR interval (ms)	622 ± 74	617 ± 76
ET (ms)	289 ± 27	272 ± 32
	p < 0.005	
IVRT (ms)	65 ± 23	57 ± 47
DFP (ms)	234 ± 64	240 ± 47
fm (ml/m <sup>2</sup> ·s)	306 ± 122	255 ± 97
	p < 0.05	

DFP = diastolic filling period from mitral valve opening to end-diastole; ET = systolic ejection time; fm = mean diastolic angiographic filling rate; IVRT = isovolumic relaxation time.