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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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References

1. Hess OM, Surber EP, Ritter M, Krayenbuehl HP. Pulsus alternans: its influence on systolic and diastolic function in aortic valve disease. J Am Coll Cardiol 1984; 4:1–7.
2. Spodick DH, Khan AH, Pigott VM. Systolic and diastolic time intervals in pulsus alternans: significance of alternating isovolumic relaxation. Am Heart J 1974;87:5–10.
3. Carlson CJ, Rapaport E. Postextrasystolic pulsus alternans and heart rate. Am J Physiol 1984;246:H245–9.
4. D'Cruz I, Cohen HC, Prabhu R, Glick G. Echocardiography in mechanical alternans. With a note on the findings in discordant alternans within the left ventricle. Circulation 1976;54:97–102.
5. Schuster AH, Nanda NC. Doppler echocardiographic features of mechanical alternans. Am Heart J 1984;107:580–3.

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^2·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.

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remained uninfluenced by the changing rate of left ventricular filling.

In summary, the systolic and diastolic time intervals do not allow conclusions to be drawn as to the underlying mechanism responsible for the alternating contractions in patients with pulsus alternans. Spodick is right that our observations were made in patients with servere aortic valve disease, and should, therefore, be restricted to this group of patients.

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Transient Entrainment and Interruption of Ventricular Tachycardia With Rapid Atrial Pacing—I

In the report by Waldo, et al. (1), one minor point of interpretation was confusing. The authors suggested that the area of slow conduction in the ventricle was probably functionally present only during ventricular tachycardia because of the shortening of the stimulus to right ventricular electrogram interval by 335 ms immediately after interruption of the tachycardia and because of the return of the QRS morphology to the "normal" right bundle branch block pattern.

Several observations from the figures in their report in combination suggest that the opposite conclusion—that is, the continued presence of the slow conduction—would be the more likely: 1) the tachycardia has a left bundle branch block morphology, 2) entrainment at a rate of 165 beats/min resulted in a narrow QRS complex with relatively normal morphology and, most importantly, 3) the first paced beat after the interruption of the ventricular tachycardia showed a right bundle branch block morphology.

These three observations suggest that the right bundle branch was the area of conduction delay. The electrical circus movement thus conducted slowly down the right bundle branch in an anterograde direction, returned retrograde via the left bundle branch and reentered the right bundle. This would yield a left bundle branch block morphology to the ventricular tachycardia. During entrainment, the atrial paced beats conducted down both bundle branches, although more slowly down the right bundle branch. With increasing pacing rates, there was increasing fusion from the paced beat conducting down the left bundle branch and the previous paced beat conducting slowly down the right bundle branch. At termination of ventricular tachycardia, this fusion phenomenon ceased; that is, there was no longer any ventricular depolarization occurring via the right bundle branch from the antecedent paced beat, resulting in the right bundle branch block morphology of the "normal" QRS. This strongly suggests that the right bundle branch was the area of slow conduction. Were the right bundle branch not involved, one would not expect this sudden widening of the QRS morphology in association with termination of the reentrant loop.

That the stimulus to right ventricular electrogram interval decreased by 335 ms is entirely consistent with the fact that the right ventricle is now depolarized via the faster conducting left bundle branch. It need not imply that slow conduction has ceased over the right bundle branch. The suddenness of the termination of the ventricular tachycardia also need not imply that the slow conduction has ceased, only that an exit block is now present. One blocked conduction down the right bundle branch was all that was needed to allow retrograde penetration of the right bundle branch via the left bundle branch, resulting in persistence of the exit block and preventing reinitiation of the tachycardia after cessation of pacing.

One can also postulate that initiation of ventricular tachycardia can be easily achieved by a single ventricular premature beat. A properly timed ventricular premature beat can find the right bundle branch still refractory, but conduct retrograde up the left bundle branch. This could 1) allow it to enter the right bundle branch and set up the circus movement, or 2) block a sinus beat from conducting down the left bundle branch, thus allowing conduction through the right bundle setting up the circus pathway. It would seem that this case report was an excellent example of sustained ventricular tachycardia due to bundle branch reentry.

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Reference

1. Waldo AL, Henthorn RW, Plumb VJ, MacLean WAH. Demonstration of the mechanism of transient entrainment and interruption of ventricular tachycardia with rapid atrial pacing. J Am Coll Cardiol 1984;3:422–30.

Reply

We suggested that the area of slow conduction in the ventricles was only functionally present during ventricular tachycardia. Tchou concludes that the opposite is more likely. Actually, independent of whether we agree with Tchou's conclusion, we believe his point really is semantic. Perhaps it would have been better to have said "functionally operative" instead of "functionally present." Our point really is that the area of slow conduction is not demonstrable except during the period of the tachycardia and, if we understand Tchou's letter, we believe he would accept that.

Tchou's suggestion that the ventricular tachycardia represents a case of bundle branch block reentry is certainly intriguing. However, we would prefer not to speculate beyond that modest statement for several reasons. First, bundle branch block reentry as a cause of sustained ventricular tachycardia is very rare, if indeed