5. Beder S, Gillette P, Garson A, Porter C, McNamara D. Symptomatic sick sinus in children as the only manifestation of cardiac abnormality or associated with unoperated congenital heart disease. Am J Cardiol 1983;51:1133–6.

Reply

I must disagree with Phibbs' statements in the strongest possible way. There are no flagrant misquotations in our article. We did make a mistake in Reference 1. We intended to refer to the work of Van der Hauwaert and Ector (1) rather than to the article by Fox et al. We must have inadvertently transposed references when preparing the manuscript.

Phibbs' computer-assisted reviews must be faulty because there are at least two other articles that describe children who died after having documented sick sinus syndrome. One is by James et al. (2) and the other is by Bharati et al. (3). The patient of Bharati et al. had a ventricular septal defect in infancy that closed spontaneously, and death did not occur until the child was 16 years of age. I have concluded that that patient did not have significant congenital heart disease anywhere near the time of death. These last two articles also give histopathologic correlation to the electrocardiographic and electrophysiologic findings and indicate that there is pathologic basis for sinus node dysfunction.

Phibbs does not seem to understand how we are using the response to atrial pacing. Probably everyone knows that it is normal for a patient to develop type I block during atrial pacing, and we certainly do. In pediatric patients who are sedated with Demerol and Phenergan before study, the pacing rate at which type I block occurs is higher. Normal values have been clearly developed and are published in our book (4). I agree that it is very difficult to predict future onset of atrioventricular (AV) block using this test; however, we have been very successful in predicting nononset of AV block using a rate greater than 120 beats/min. We feel that the AV node will continue to conduct normally for some length of time, probably at least 10 years, and we find it useful to insert an atrial pacemaker. If second degree block develops at a lesser rate while the patient is sedated with Demerol and Phenergan, we feel there is a possibility that AV block might develop at a later time and therefore we use a dual chamber pacemaker. We have not subjected this idea to random study because we feel that it might be dangerous to the patient to do so. We also feel that the difference between a dual and a single chamber pacemaker is not too great in cost or trauma to the patient and therefore prefer to use a dual chamber pacemaker if there is any question of AV conduction.

I think if Phibbs had thought his third point out carefully he would agree that the heart rates described are very conservative. These rates are intended for patients who are awake and they occurred for 6 seconds. When one is dealing with patients, one has to develop some criteria for action. I have developed these criteria over a long period of time working with pediatric patients. I have no concern that these rates are too high; I have some concern that they may be too low. When one is dealing with patients after the Mustard operation for example, in whom many centers have found a 5% or greater incidence of sudden death, it is important to establish criteria for pacemaker implantation to try to prevent such sudden death. I do not think that we can afford to wait until we have absolute statistical evidence that our criteria are exactly right before we can begin to use our common sense.

The main point of my article was that most causes of sudden death in children are now treatable and thus preventable. If we take Phibbs' point of view, we will not prevent many of these sudden deaths.

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References

1. Van der Hauwaert LG, Ector H. Sick sinus syndrome in childhood. Br Heart J 1980;44:684–91.
2. James TN, Froggatt P, Marshall TK. Sudden death in athletes. Ann Intern Med 1967:1013–21.
3. Bharati S, Nordenberg A, Bauernfeind R, et al. The anatomic substrate for the sick sinus syndrome in adolescents. Am J Cardiol 1980;46:163–72.
4. Gillette PC, Garson A Jr. Pediatric Cardiac Dysrhythmias. New York: Grune & Stratton, 1981.

Correction

Table 1 on page 532 of the September issue of the Journal was printed incorrectly (Blaustein AS, Risser TA, Weiss JW, Parker JA, Holman BL, McFadden ER. Mechanisms of pulsus paradoxus during resistive respiratory loading and asthma. J Am Coll Cardiol 1986;8:529–36).

The following is the correct version of the table:

Table 1. Baseline Hemodynamic Variables

Case	Heart Rate (min^{-1})	Blood Pressure (mm Hg)	Pulsus Paradoxus (mm Hg)	Esophageal Pressure (mm Hg)		LVEF(%)		RVEF(%)	
				I	E	I	E	I	E
1	75	129/88	1	−4	0	73	72	41	50
2	70	96/57	4	−5	−1	71	63	39	36
3	80	128/81	7	−12	−4	69	73	54	37
4	61	117/68	6	−4	0	85	85	40	38
5	76	144/97	6	−5	−1	85	95	31	32
6	81	150/103	3	−3	+2	71	66	37	36
Mean	73.8	127/82	4.5	−5.5	−0.7	76	76	40.3	38.2
± SEM	3.3	8.5/7.6	1.0	1.5	0.9	3.2	4.9	3.4	2.8

E = expiration; EF = radionuclide ejection fraction; I = inspiration; LV = left ventricle; RV = right ventricle.