are needed to elucidate the mechanisms of action of these agents in hypertrophic cardiomyopathy.

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References

**Pulsus Alternans**

To the Editor:

The report on pulsus alternans by Hada, Wolfe and Craig1 is an excellent contribution, using noninvasive methods to elucidate this phenomenon that has fascinated physicians and biologists for 110 years. Unfortunately, they have not been as thorough in their review of animal studies on this subject. The first animal study on this subject was by Gaskell2 100 years ago, who reported pulsus alternans in the frog heart when the vagus was stimulated. In 1969, we reported alternation in healthy dogs3 with no myocardial disorders in which cardiac performance was temporarily compromised by fast pacing, with or without vagal stimulation. A major part of our report in 1969 dealt with the inadequacy of the Frank-Starling relationship as the mechanism of pulsus alternans. The Frank-Starling relationship is simply the determination of the ventricular output by the filling, all other factors being constant. Assuming a fixed venous return rate and equal diastolic filling periods, the effect of a single ventricular premature complex (VPC) can be calculated with a pencil and paper (fig. 1). (Hada and his colleagues found that VPCs frequently precipitated pulsus alternans, and were the source in 13 of 14 patients studied prospectively.) Although the VPC will have an abbreviated filling interval and a weakened contraction, the beat following the premature beat will have an enhanced stroke volume, as is well demonstrated in the echocardiographic studies of Hada et al.

However, the stabilizing Frank-Starling relationship actually serves to quickly dampen out the alternation. A simple linear relationship such as the Frank-Starling relationship, even if modified with a curvilinear input/output proportionality, will simply not produce the complicated instability seen as pulsus alternans. In fact, in both animals and in man,4 there is no consistent relationship between stroke volume and diastolic volume in pulsus alternans, and in some cases there is actually an inverse relationship.

I believe that the data from Hada, Wolfe and Craig support our conclusion that a change in myocardial contractility was involved in pulsus alternans. Although these authors do not list results for the entire series, their figures 5 and 6, which provide measurements of a control beat preceding a VPC, reveal that the prejection period divided by the ejection time lengths for the VPC, shortens for the postextrasystolic beat, and then alternates thereafter. This ratio of prejection period to ejection time not only alternates, as the authors have pointed out, but the

![Effects of diastolic volume on stroke volume](image-url)

**Figure 1.** Graph of the calculated effect of a ventricular premature complex (VPC) on stroke volume and ventricular filling. The calculations are based on the following linear equations:

\[
\text{Ventricular diastolic volume (VDV)} = \text{residual volume (RV)} + c_i t
\]

\[
\text{Stroke volume (SV)} = \frac{\text{VDV}}{\text{VDV}_c} \times \text{SV}_c\]

\[
\text{RV} = \text{VDV} - \text{SV}
\]

\[\text{SV}_c\] represents the control stroke volume (100%), \(c_i\) is a constant indicating a constant filling rate, and \(t\) is for diastolic interval. \(\text{VDV}_c\) is the control diastolic volume. Reproduced from Guntheroth et al.5 with permission of the American Heart Journal.
average for alternating pairs is also remarkably close to the ratio of the control beat. In other words, one of the paired beats is actually greater than average in its index of performance, and one is less. This observation in dogs, plus the widely recognized effects of potentiation in the postextrasystolic beat, make a strong case for potentiation alternating with deletion as the mechanism of pulsus alternans. This averaging has some clinical consequences. Paired, pulsed stimulation is no longer used for increasing cardiac performance, but it should be apparent from inspection of figures 5 and 6 that the patient does not actually deteriorate from alternation, although alternation is likely to occur in patients with compromised myocardium. The alternation is not a dangerous condition, and a specific intervention is not required to eliminate it, although the clinician may wish to treat the underlying myocardial disorder. Again, the separation between the cause and effect should permit the clinician to consider the long-term health of the patient’s myocardium, rather than rushing to abolish a phenomenon that is not particularly disadvantageous.

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AHA Diet Recommendations

To the Editor:

In a recent American Heart Association (AHA) Committee Report, Grundy et al.1 wrote: “For the first time, the AHA has taken the position that a diet recommendation for the healthy U.S. population is warranted.” I believe this statement is in error. On June 8, 1964, the AHA issued a three-page National Press Release entitled: ‘Heart Association Recommends Reduced Fat Consumption to Lessen Risk of Heart Attacks and Strokes.”

The following are quotations from that release: “The action taken by the Board of Directors broadening the application of the fats statement to the general public should be placed on the record as soon as possible. The Board acted favorably on a proposal to extend to the general public the dietary recommendations of the American Heart Association’s 1961 statement, ‘Dietary Fat and its Relation to Heart Attacks and Strokes.’ It was made clear that a major purpose of the dietary recommendations is to reduce blood levels of cholesterol. Evidence from many countries suggests a relationship between the amount and type of fat consumed, the amount of cholesterol in the blood, and the reported incidence of coronary artery disease. The Association’s recommendations are thus aimed at lowering blood levels of cholesterol in the belief that ‘reduction in blood cholesterol may lessen the development or extension of atherosclerosis and hence the risk of heart attacks and strokes.’”

In 1965, the Los Angeles Heart Association, in response to the above, obtained 1,300,000 educational brochures to dramatize “Reduce the Risk of Heart Attack and Stroke” for distribution “into every home,” which must have included some of the healthy U.S. population.

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