“Ad>120 msec” or “LP2<3.5 µV" should have been written: “predictive accuracy.”

We are also aware of the importance of the prevalence in the general population to draw a positive or negative value as a diagnostic tool. In this matter, we agree with them. However, we chose as our target a hospital population mainly to differentiate the disease from the normal but not the general population, which makes it possible to diagnose it based on the prevalence of the disease, because it is very difficult to detect patients with paroxysmal atrial fibrillation during sinus rhythm.

Another study on the prevalence may be needed to screen the general population. However, the prevalence itself of paroxysmal atrial fibrillation seems very difficult to obtain, especially in the case of asymptomatic paroxysmal atrial fibrillation or even symptomatic paroxysmal atrial fibrillation without stroke. Consequently, from the present study, the sensitivity and specificity are at least valid because these do not depend on the prevalence. Also, the reason of having presented the predictive accuracy is that the positive predictive value is more heavily affected by the prevalence than the predictive accuracy.

The meaning of the screening is “the presumptive identification of an unrecognized disease or defect by the application of tests which can be applied rapidly” (cited from Reference 2). “A screening test is not intended to be diagnostic.” However, they appear to think that a positive result of the test indicates a need of anticoagulant therapy for prevention of stroke. In fact, we are now using this method in clinical situations as a screening test to do further examination (but not as a diagnostic tool) in patients suffering from paroxysmal palpitation.

As they stated in their letter, atrial fibrillation, whether it is chronic or paroxysmal, is a potential risk of systemic thromboembolism.\(^2\)\(^3\) Thus we previously demonstrated\(^4\) that atrial fibrillation itself may be more important than factors predisposing to atrial fibrillation in the development of intracardiovascular clotting, which was compatible with the results in this study in terms of independence of the presence of underlying organic heart disease. Therefore, such a screening test is thought to be more important in patients without organic heart disease who visit a hospital only because of palpitation or chest discomfort.

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References

Life Expectancy and Coronary Artery Disease
Tsevat et al\(^1\) have reported that elimination of mortality from coronary artery disease (CHD) from the U.S. population would increase average life expectancy at 35 years of age by 3.1 years for males and 3.3 years for females. These findings are of significant interest, particularly in the public arena, in that they provide some basis for future decisions affecting public health, including patterns and trends of funding for various areas of biomedical research. Thus, this research effort has received broad exposure in the press and other public media.

However, the main conclusion of the study by Tsevat et al\(^1\) is neither unexpected nor novel. Nearly 30 years ago, Robert R. Kohn\(^2,3\) utilized curves for age-specific death rates to estimate the beneficial increase in human life span if deaths from several selected diseases were to be eliminated. Kohn concluded that elimination of all deaths from arteriosclerosis (as arteriosclerotic heart disease, most vascular diseases of the central nervous system, and the majority of chronic renal diseases) would result in an increase in life expectancy at birth of approximately 7 years. This value is well within the range generated by others who model life extension following modification of risk factors for CHD, as discussed by Tsevat et al.\(^1\) Because deaths from CHD between birth and 35 years are of little concern in terms of public health, Kohn's estimate is remarkably similar to the benefit generated by Tsevat et al\(^1\) when adjusted for the well-discussed decline in mortality from CHD that has occurred over the past 25 years. Therefore, it is clearly evident to us that Kohn should be credited for his pioneering studies in this area of endeavor carried out a full quarter of a century before present efforts.

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Reply
We appreciate the comment by Drs. Baird, Massie, and Hough, who indicated that the findings in our report\(^1\) were of significant interest. We also appreciate the additional citation of Dr. Kohn's 1963 article. In our article, we cited several previous reports that were consistent with ours. Since our article was submitted, an analysis by Olshansky and colleagues\(^4\) corroborated our findings by projecting that eliminating ischemic heart disease would increase life expectancy at birth by 3.0 years for females and 3.55 years for males. It should be noted, however, that the focus of our paper was on gains in life expectancy from risk factor modification rather than from eliminating coronary heart disease (CHD) per se (an impossible goal).

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