hypothesis that reduced left ventricular filling, which is the result of diminished right ventricular output, leads to the abnormal STI in patients with COPD. For these reasons, STI are unreliable indicators of left ventricular function in patients with COPD and cannot be used to detect subclinical left ventricular dysfunction in such individuals.

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References

The authors reply:
To the Editor:
Abnormalities of systolic time intervals (STI) demonstrated in patients with chronic obstructive pulmonary disease (COPD) are indicative of changes in the electromechanical events of the left ventricle (LV) in these patients. As the selection process in our study eliminated patients with clinical evidence of valvular, myocardial, hypertensive, or coronary artery disease, we concluded the most likely explanation was LV dysfunction. Alpert et al. propose an alternate possibility which was suggested by the work of Dr. Alpert. The abnormal STIs in COPD may result from a reduction of LV filling as a consequence of reduced right ventricular output. Early work by Weissler et al. demonstrated a direct relationship between LV ejection time and LV stroke volume. Indirect interventions causing a reduction in systemic venous return have been shown to shorten the LV ejection time and also to prolong the pre-ejection period. In view of this information the possibility of a reduced stroke volume as an explanation for the abnormal STIs must be given serious consideration. It should be noted that while a reduced cardiac output and stroke volume may occur in patients with COPD, most patients will maintain normal or increased cardiac outputs even in the face of severe airway obstruction. Our patients with abnormal STI represent a select group with significant but not severe airway obstruction. Patients with COPD who have abnormal STI obviously require further study to define the mechanism involved. We welcome data which would clarify this problem and support one or both hypotheses. On the other hand, while Alpert et al. may have data to support their hypothesis, the abstract to which they refer is certainly inconclusive. Hopefully, they will see fit to report their observations more fully where we may all review them in depth.

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References

Rheumatic and Non-rheumatic MV Disease
To the Editor:
I must take exception to several comments made by Shulman et al. in their article "Differences in Antibody Response to Streptococcal Antigens in Children with Rheumatic and Non-rheumatic Mitral Valve Disease" (Circulation 50: 1244, 1974). Dr. Shulman states that pure aortic regurgitation is recognized to be infrequently due to rheumatic heart disease. He uses as his reference Dr. Robert's article published in 1970 (Am J Med 49: 151, 1970). Dr. Robert's statement was meant to cover pathological mitral valve changes which were found in patients at
Letter: Rheumatic and non-rheumatic MV disease.

H H Kloth

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