In Hypertension and Angina, the Cardiac Times Are Out of Joint

The left ventricular (LV) systolic time intervals (STI) are important for two reasons. First, as integral components and distinguishing characteristics of cardiac performance, they provide unique information about it. Second, their precise and accurate measurement can be obtained easily and repeatedly without trauma.

In patients with LV dysfunction, the STI relative to heart rate are often abnormal. In this issue (see page 51), Dodek and associates have reported that they found the pre-ejection period (PEP) and LV ejection time (LVET) relative to heart rate to be abnormal, on the average, in a group of 28 patients who had chronic systemic hypertension without clinical evidence of congestive heart failure. The PEP was long and the LVET short. Eleven of the 17 patients without evidence of LV hypertrophy had abnormal PEP/LVET ratios. These systolic temporal abnormalities may, in part, reflect the elevation of aortic pressure. The findings also imply, however, that some hypertensive patients lack the usual clinical criteria for heart disease, yet have preclinical, as well as symptomless, LV dysfunction, which determinations of the STI can detect. The revelation of such dysfunction in a patient would heighten the urgency of lowering his arterial pressure. Dodek and colleagues found, indeed, that when hydrochlorothiazide and a-methyldopa were administered on a long-term basis and arterial pressure fell, the STI improved.

This well-done study raises important questions. To me, the following three seem foremost. First, what is the true incidence of abnormal STI in the enormous population of hypertensive patients who lack evidence of LV hypertrophy? In hypertensive patients without clinical heart failure, previous studies (summarized in reference 1) have shown the Q-SI interval to be lengthened, with the PEP and the PEP minus the Q-SI interval variously reported as normal or mildly prolonged. In their 28 patients, Dodek and associates found a high incidence of PEP prolongation (28 percent) and LVET shortening (48 percent), but did not report the separate incidence of these two abnormalities in the 17 patients without LV hypertrophy. Second, how do the abnormal STI relate, in time and mechanism, to the later emergence of more overt heart disease? Third, in attempts to identify the hypertensive patient with preclinical LV dysfunction, would not responses of the STI to a standardized stress, eg, exercise, increase the sensitivity and specificity of these measurements? When done properly, determinations of the STI and their responses to stress may well deserve a place alongside sphygmomanometry in the clinical evaluation and followup of hypertensive patients. However, precise definition of the need for, and role of, obtaining STI measurements in this multitude of patients requires more extensive study.

Also in this issue (see page 56), D’Angelo and associates show that diastolic time intervals, studied much less frequently than the STI, may be disturbed in certain diseases. Most strikingly, the isovolumic relaxation time (IVRT) was abnormally prolonged in patients with moderate, severe, or unstable angina, most of whom were studied close to, but not during, an ischemic attack. Myocardial hypoxia and ischemia can lead to both a slower rate of ventricular relaxation after contraction and a decreased diastolic compliance, ie, increased resting stiffness. Both abnormalities would retard the fall of LV pressure after aortic valve closure and, consequently, prolong the IVRT.

In health, several well-demarcated time intervals, each having its optimal duration, are mortised together to compose systole and diastole. In certain illnesses, some of these cardiac times are “out of joint.” Measurement of these times provides the physician with unique information, which can be important to making decisions about a patient.

Willard S. Harris, M.D.*
Los Angeles

---

*Professor of Medicine, University of California, and Department of Cardiology, Cedars-Sinai Medical Center.

Reprint requests: Dr. Harris, Cedars of Lebanon Hospital, 4833 Fountain Avenue, Los Angeles 90029

REFERENCES

6 Shakespeare W: Hamlet, Act 1, scene 5, line 188