Relationships of Left Ventricular Systolic Time Intervals to Hemodynamic Variables in Intact and Failing Hearts

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In order to evaluate the significance of left ventricular systolic time interval (STI) determination in assessing cardiac performance, the relationships between STI and hemodynamic variables were studied in dogs and human subjects with or without myocardial dysfunction.

METHODS

The left ventricular ejection time (ET) and the pre-ejection period (PEP) were measured from external recordings in normal subjects and patients with congestive heart failure of NYHA classification II due to ischemic heart disease and primary myocardial disease (CHF patients) and those without congestive heart failure (IHD and PMD patients). The changes in STI were corrected for the changes in heart rate by the normal regression equations obtained previously from 122 normal subjects (PEP = -0.31 x heart rate + 122, ET = -1.4 x heart rate + 384 msec). STI and hemodynamic variable were measured at rest and immediately after exercise accomplished by use of a bicycle ergometer in the supine position for 4 minutes at a load of 1 watt/Kg of body weight. Cardiac output was measured by dye dilution method using an ear piece.

The experimental study was performed in open chest dogs, in which with the aid of an extracorporeal blood circuit, cardiac output, blood pressure and heart rate were capable to be controlled at any desired levels. The effects of changing hemodynamic variables on STI were studied in intact heart as well as in failing hearts induced by sodium pentobarbital administration.

RESULTS

1. Relationships between hemodynamic variables and STI.

The effects on STI of the changes in each of heart rate, cardiac output, diastolic pressure and myocardial function were studied in dogs while the other hemodynamic variables were kept constant. The increase of heart rate, stroke volume and diastolic pressure resulted in considerable shortening of ET and minimal shortening of PEP, prolongation of ET and proportional shortening of PEP, and shortening of ET associated with prolongation of PEP, respectively. Deterioration of myocardial function prolonged both ET and PEP. In human subjects, the elongation of PEP and shortening of ET relative to heart rate were observed in CHF patients, and these changes were found to be relatively well correlated with the reduction of cardiac output and independent of the levels of diastolic pressure.

2. Changes in hemodynamic variables as determinants of STI induced by exercise.

As a general, exercise resulted in moderate increases of heart rate and cardiac output accompanied by minimal changes in stroke volume and diastolic pressure. It was noted that the increase of cardiac output was less in CHF patients than in normal subjects.

3. Effects on STI of exercise or an increase

Key Words:
Ejection Time
Pre-ejection Period
Cardiac Function
Blood Pressure
Cardiac Output
Heart Rate

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* This paper was presented at the Symposium on “Hemodynamics in the Cardiovascular System” on the 36th Annual Meeting of the Japanese Circulation Society in Kanazawa, April 10, 1972.

Japanese Circulation Journal Vol. 17, July 1973 711
of stroke volume in various states of the heart.

In dogs, changes in STI were studied as the stroke volume was doubled while keeping heart rate and diastolic pressure constant. As shown in figure 1, so far as the heart remained intact, the increase of stroke volume resulted in shortening of PEP and prolongation of ET, but, by contrast, when the heart was deteriorated, it caused prolongation of both PEP and ET. Figure 2 shows the changes in STI after exercise in human subjects. Immediately after the exercise, ET lengthened and PEP diminished in normal subjects while both PEP and ET lengthened in CHF patients.

**DISCUSSION AND CONCLUSION**

At rest, PEP was prolonged, ET was shortened and, consequently, PEP/ET was high in CHF patients. The present experimental study and the results of cardiac output determination in CHF patients suggest that this may be attributed to a reduction of cardiac output associated with impaired myocardial function. After exercise, CHF patients demonstrated prolongation of PEP, slight prolongation of ET and an increase of PEP/ET. These changes in STI were found to be almost identical to those observed as the stroke volume was increased in experimental failing hearts. Since the present study also suggests that the hemodynamic effect of exercise can be regarded as an augmentation of stroke volume at a high level of heart rate, the changes in STI observed in CHF patients after exercise can be interpreted as a result of a limited increase of cardiac output and the impairment of myocardial function unmasked by an increase of flow load on the heart even though of minimal degree. Thus, the measurement of STI, particularly its response to exercise, revealed to be valuable in the differentiation between subjects with normal and those with impaired myocardial function.