Simultaneous Changes of Left Ventricular and Left Atrial Size and Function in Normal Subjects during Exercise

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SUMMARY

We performed dual M-mode exercise echocardiography, which records left ventricular (LV) and left atrial (LA) echocardiograms simultaneously, in 10 normal subjects to investigate mechanisms of cardiac adaptation to dynamic exercise. The LV end-diastolic dimension (EDD) increased significantly as exercise increased from mild to moderate (3–5%, p<0.05 and 0.001). At peak exercise the EDD was slightly increased, but it returned to the baseline during recovery. The LV end-systolic dimension (ESD) showed a progressive decrease with the severity of exercise (5–13%, p<0.05 and 0.001). It decreased further immediately after exercise (17–20%) and then returned to the resting value. The LV stroke dimension and fractional shortening increased as exercise changed from mild to moderate and reached a plateau at peak exercise. During recovery, they showed a transient increase and then decreased. By contrast, the LA dimensions, both maximum and minimum, increased significantly as exercise changed from mild to moderate (15–16% and 16–19%, p<0.01 and 0.001, respectively), but they were lower at peak exercise (12 and 14%). They returned rapidly to the resting values immediately after exercise.

Thus, during exercise, LV function is augmented by the Frank-Starling mechanism in combination with increased contractility, while the LA is enlarged to receive the increased venous return accompanying dynamic exercise. These exercise-induced changes in LV performance return gradually to the resting state, but LA size recovers rapidly after the cessation of exercise.
EXERCISE-induced changes in cardiac chamber volume and function have long been a subject of great interest. Although many investigations\textsuperscript{1-7} have been performed, the mechanism of human cardiac adaptations to dynamic exercise has not yet been entirely clarified. Recent studies\textsuperscript{8-14} in normal individuals provide conflicting evidence regarding changes in the end-diastolic volume of the left ventricle. The left ventricular end-diastolic size or volume is a major factor which regulates cardiac performance through the Frank-Starling mechanism. Not only does the left atrium act as a reservoir for returned pulmonary venous blood, but its systole augments the end-diastolic volume of the left ventricle and thereby enhances cardiac performance.\textsuperscript{15}

However, as far as we know, there have been no investigations of the changes of both left ventricular and left atrial volume and function during exercise or the correlation between these two cardiac chambers in their adaptation to dynamic exercise. In this study, we performed dual M-mode echocardiography in normal subjects and recorded left ventricular and left atrial size and function during exercise and recovery.

**Subjects and Methods**

*Subjects:* Ten healthy male volunteers, aged 17 to 35 years (mean age 26 years), were studied. All had a normal cardiovascular history, physical examination and 12 lead electrocardiogram at rest. They were not conditioned to exercise by any regular athletic program.

*Exercise equipment and stress protocol:* The volunteers exercised in a supine or slight left-lateral decubitus position, which appears to provide an optimal acoustic window for echographic viewing of the heart. An electromagnetically controlled bicycle ergometer (Siemens-elema 380B) with a pedaling frequency of approximately 60 revolutions/min was used. Work loads were determined separately for each subject, based on a preliminary study and graded exercise was performed. The initial work load was 50–75 watts, and was increased by 25 watts every 3 min. The duration of exercise was 12 min, and the maximum work load was 125–150 watts.

*Echocardiographic recording procedure and measurements:* A 78 phase array sector scanner (Toshiba SSH-11A) with a 2.4 MHz transducer interfaced with a Honeywell Visicorder was employed in this study. Two-dimensional echocardiographic images of the long axis of the left ventricle were recorded from
Fig. 1. Two-dimensional echocardiograms of the left ventricular long axis during exercise and recovery. These echocardiograms were obtained from the parasternal approach. The two white lines in each echocardiogram represent the directions from which we recorded dual M-mode exercise echocardiograms: 1) conventional left ventricular echocardiogram, 2) aortic root and left atrial echocardiogram.

a left parasternal position in the third or fourth intercostal space (Fig. 1). Dual M-mode echocardiograms were obtained in two directions: one direction for conventional left ventricular echograms and the other for the aortic root and left atrial echograms. An electrocardiogram, a phonocardiogram, and a carotid arterial pulse tracing were recorded simultaneously at a paper speed of 100 mm/sec with a strip chart recorder (Fig. 2). All these recordings were performed at rest and in the last 30 sec of each exercise level during unforced held expiration. During recovery, echocardiograms were recorded 0.5, 1, 3, 6, and 10 min after exercise. The subjects were carefully instructed to avoid the Mueller maneuver. Systolic blood pressure was determined by auscultation.

We measured the following dimensions of the left ventricle and the left atrium. The left ventricular end-diastolic dimension (EDD) was measured at the R wave of the QRS complex of the electrocardiogram and the end-systolic dimension (ESD) at the aortic component of the second heart sound. Then we calculated the left ventricular stroke dimension (LVSD: EDD—ESD).
and the fractional shortening (FS) of this dimension during systole:

\[ \text{FS(\%)} = \frac{(\text{EDD} - \text{ESD})}{\text{EDD}} \times 100 \]

In addition, the left atrial maximum dimension (LADmax) was measured immediately after the second heart sound and the minimum dimension (LADmin) at the R wave of the QRS complex of the electrocardiogram. Then we calculated the left atrial dimensional change (LADC: LADmax - LADmin) and the fractional emptying index (EFI) of the left atrium during diastole:

\[ \text{EFI(\%)} = \frac{(\text{LADmax} - \text{LADmin})}{\text{LADmax}} \times 100 \]

**Statistical analysis**: Student's paired t test was used to compare differences in individual measurements at rest and during exercise and recovery. A probability value of 0.05 was considered significant. The values shown represent the mean ± standard deviation.

**RESULTS**

**Heart rate and systolic blood pressure**: The heart rate increased substantially with the severity of exercise and increased stepwise from rest to peak exercise (67±11 to 150±9 beats/min). During recovery, it decreased sharply immediately after exercise and then gradually returned to the resting value (3 min 97±13; 10 min 87±10). Systolic blood pressure also rose with exercise from rest to peak exercise (122±12 to 204±18 mmHg) and returned to
the resting value within 6 min (124±7 mmHg) after exercise.

**Left ventricular dimensions** (Fig. 3): The EDD increased significantly by 0.2–0.3 cm (4–5%) from mild to moderate exercise (p<0.05 and 0.001, respectively), and did not increase further, or even decreased, at peak exercise (0.1 cm, 3%). During the recovery period, however, the EDD was not significantly different from the EDD at rest. The ESD decreased progressively by 0.2–0.4 cm (5–13%), from mild to peak exercise (p<0.05 and 0.001, respectively). Immediately after exercise, it decreased further by 0.5–0.6 cm (17–20%) and then returned to the baseline. However, the ESD had not recovered completely by 10 min after exercise.

**Left atrial dimensions** (Fig. 4): Both the LADmax and LADmin dimensions increased significantly by 0.4–0.5 cm (12–16%) and 0.3–0.4 cm (14–18%), respectively, from mild to moderate exercise (p<0.05 and 0.01). At peak exercise, they even decreased slightly. Within 1 min after exercise they returned to the resting values and did not change significantly during recovery.

**Left ventricular stroke dimension** (Fig. 5): The LVSD also increased by
Left atrial dimensions during exercise and recovery. The left atrial dimensions, both LADmax and LADmin, increased significantly from mild to moderate exercise, but decreased slightly at peak exercise. They dropped abruptly to the baseline values immediately after exercise.

0.3–0.5 cm from mild to moderate exercise, but did not increase further at peak exercise. During recovery, there was a transient increase immediately after exercise (0.1–0.2 cm) and then a return to the resting values.

Left atrial dimensional change (Fig. 5): The LADC showed a slight, but not statistically significant, increase during and immediately after exercise (0.1–0.2 cm).

Left ventricular fractional shortening (Fig. 6): The LVFS increased by 6–10% from mild to moderate exercise and reached a plateau at peak exercise. However, it increased again by 2–3% within 1 min after exercise and then gradually decreased to the baseline.

Left atrial fractional emptying index of left atrium (Fig. 6): This index did not change significantly during or after exercise, although there was a transient, slight increase immediately after exercise.

DISCUSSION

An exercise test is one of the most important and valuable non-invasive diagnostic tests in the clinical evaluation and management of patients with

Fig. 4. Left atrial dimensions during exercise and recovery. The left atrial dimensions, both LADmax and LADmin, increased significantly from mild to moderate exercise, but decreased slightly at peak exercise. They dropped abruptly to the baseline values immediately after exercise.
suspected or documented cardiovascular disease. Many investigators\(^{16-18}\) have designed symptom-limited multistage exercise protocols, in which exercise must be terminated at the patient’s request when significant symptoms, particularly severe chest pain, marked dyspnea, dizziness, and severe fatigue, are produced. Except for chest pain, these symptoms would be caused by a transient deterioration of left ventricular function, including both forward and backward failure induced by exercise. Recent non-invasive studies,\(^{9-14}\) using radionuclide angiography or echocardiography, have been performed to investigate exercise-induced changes in left ventricular forward function. However, little attention has been paid to backward failure. In this study, we recorded both left ventricular and left atrial dimensions using dual M-mode echocardiography, and investigated function of both cardiac chambers during exercise in normal subjects.

Dynamic exercise increases venous return and thereby produces volume overload in the heart.\(^{19}\) However, in this study, we showed a difference in the amount of dilatation during exercise: EDD (3–5%) and LADmax (12–16%). Conflicting data have been published on the changes in the end-
Fig. 6. Left ventricular fractional shortening (LV-FS) and left atrial fractional emptying index (LA-FEI) during exercise and recovery. The LV-FS increased from mild to moderate exercise and reached a plateau at peak exercise. It increased further within 1 min after exercise and then gradually decreased. The LA-FEI showed no change during exercise and a transient increase immediately after exercise.

diastolic size or volume of the left ventricle. Some have shown variable increases,\textsuperscript{2,3} others a decrease,\textsuperscript{8} and still others, no change.\textsuperscript{4,9} In this study, which employed graded exercise, the EDD increased significantly from mild to moderate exercise. At peak exercise, it decreased somewhat, but was still slightly but significantly above the resting value. During the entire recovery period, it was not significantly above the resting value. Our previous\textsuperscript{20} and Keul’s\textsuperscript{19} studies demonstrated that the EDD did not increase and sometimes even decreased during submaximum or maximum exercise. Weiss et al\textsuperscript{11} reported that the EDD increased with severe exercise, and Stein et al\textsuperscript{9} found a transient increase in the EDD within 1 min after exercise. We cannot explain these conflicting data, but the exercise protocol, and the age, sex, or physical conditioning of the subjects may have been different.

No reports have been published on the left atrial volume or function during exercise, although some studies\textsuperscript{21,22} have been done on its performance at rest. The maximum left atrial dimension, which might be thought of as an index of its reservoir function, increased by 12–16% during exercise. The stress of exercise substantially raises the heart rate, while the diastolic filling period decreases significantly. Our previous findings\textsuperscript{23} indicated that this
decreased diastolic filling time during exercise was associated with marked shortening of the slow filling time. At a heart rate of more than 120 beats/ min, the slow filling time becomes less than 50 msec. This shortening of the slow filling time might be responsible for the decrease in the conduit volume, accounting for 20–40% of the total filling volume of the left ventricle at rest. However, the left atrium must receive the increased venous return accompanying dynamic exercise. These phenomena would cause an increase in the left atrial size during exercise. This is supported by the fact that left atrial dimensions return abruptly to the baseline values immediately after exercise, when the heart rate falls. Thus, the left atrial reservoir function may increase in normal subjects during exercise.

Most previous studies have shown that left ventricular end-systolic size or volume decrease during exercise. Our previous20,23) and current studies have also shown a progressive decrease of the ESD with the severity of exercise, which might reflect enhanced contractility by exercise-induced release of catecholamines.24) Immediately after exercise, there is a further decrease in the ESD, which is thought to be due to an abrupt reduction of the afterload, while contractility remains high. These results might be responsible for a transient augmentation of left ventricular performance immediately after exercise. In contrast to the progressive decrease of the ESD of the left ventricle, the minimum left atrial dimension increased during exercise. These results suggest that contractile performance may differ between ventricular and atrial muscle. The ventricle is more strongly affected than the atrium by sympathetic fibers which are activated with dynamic exercise, so that left atrium cannot alter its performance to compensate for increased venous return. However, in this study, we did not perform the phasic analysis of the left atrial function, so it is unknown whether its contractile function is augmented during exercise.

The stroke dimension and fractional shortening of the left ventricle increased progressively during exercise. The dimensional data in this study indicate that both the Frank-Starling mechanism and enhanced contractility work to augment left ventricular performance: the Frank-Starling mechanism mainly in from mild to moderate exercise, and contractility progressively as exercise becomes more strenuous. The atrial dimensional change and fractional emptying index, however, did not change significantly during and after exercise. The minimum left atrial dimension increased during exercise, resulting in an unaltered atrial dimensional change and fractional emptying index. However, there are potential methodological errors when a single beam M-mode echocardiogram is used. Assuming the geometry of the left atrium as a sphere, the atrial volume change, calculated by the cube of its
dimension, would increase during exercise. Therefore, the Frank-Starling mechanism should operate in the left atrium during exercise, which has been reported in man.25)

Thus, dual M-mode echocardiography, even with the single beam method, permits us to study cardiac events in two different sites simultaneously.26)-28) As shown in our current study, this method used in exercising subjects can help to clarify the mechanisms of cardiac adaptation to exercise. Further investigations in patients with heart disease are necessary to demonstrate pathophysiological responses to dynamic exercise.

REFERENCES

15. Katz AM: The heart as a muscular pump. in Physiology of the Heart, ed by Katz AM,