

Assessment of Cardiologic Systole and Diastole Duration in Exercise Stress Tests with a Transcutaneous Accelerometer Sensor

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Abstract

A system for non-invasive and automatic assessment of systolic and diastolic duration is presented. The method is based on a accelerometer sensor which was positioned in the mid-sternal precordial region. The acceleration signal, together with an ECG signal, was recorded by a laptop PC and systole and diastole durations were computed for each cardiac beat. The system was tested in 103 patients who performed semi-supine bicycle exercise (71M/32F, age 57 ± 14 years, 17 healthy people 86 patients with cardiovascular diseases). A consistent signal was obtained in 98% patients and systolic/diastolic time ratio as a function of the heart rate was computed. At higher heart rates (for example 100bpm), this ratio was lower in the 17 control subjects (0.74 ± 0.12) than in the patients with systemic hypertension (0.94 ± 0.12), coronary (0.88 ± 0.11), valvular (0.93 ± 0.14) or dilated heart (0.86 ± 0.10) disease.

1. Introduction

The duration of systole and diastole as a function of heart rate provides important information on heart functionality. In fact, patients with heart failure are characterized by a prolongation of left ventricular systole

and an abnormal shortening of diastole. The systolic-diastolic mismatch is accentuated during exercise and has the potential to impair the cardiac reserve in these patients by restricting ventricular filling and perfusion [1,2].

Physiological systole lasts from the start of isovolumic contraction to the peak of the ejection phase, so that physiological diastole commences as the LV pressure starts to fall [3]. In contrast, cardiologic systole is demarcated by the interval between the first and the second heart sounds, lasting from the first heart sound to the closure of the aortic valve. The remainder of the cardiac cycle automatically becomes cardiologic diastole [3]. Thus cardiologic systole is demarcated by heart sounds rather than physiological events and includes: major part of the isovolumic contraction; maximal ejection; reduced ejection. Cardiologic diastole includes second heart sound - first heart sound interval, filling phases included.

At rest condition, first and second heart sound can be recorded by phonocardiography [4], which is a well known and established technique. In exercise stress tests, however, the artifacts that arise from the movements of the patient can severely limit the use of this method.

Recently, a cutaneous operator independent force-frequency relation recording system, which is based on first heart sound amplitude variations at increasing heart

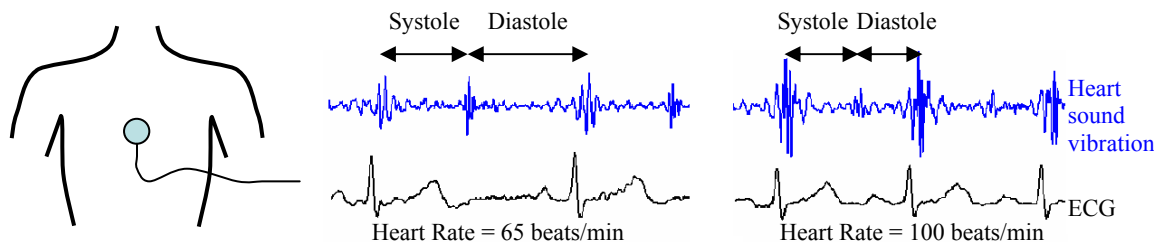


Figure 1: Heart sound vibration and ECG signal

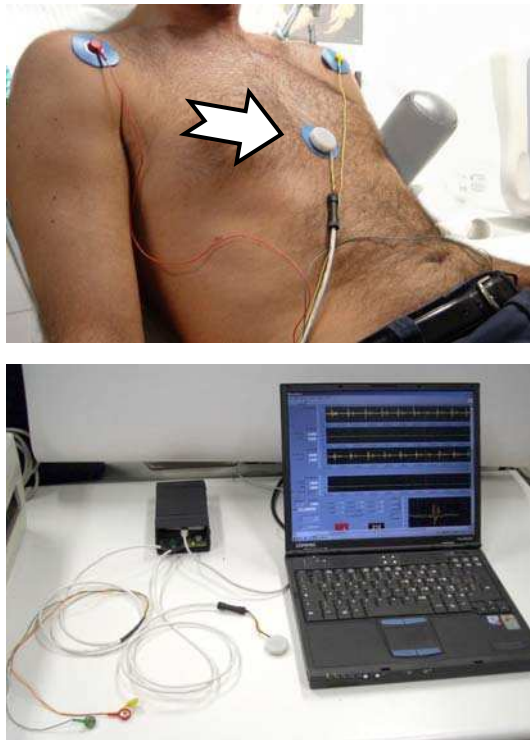


Figure 2: photos of the system. The arrow shows the acceleration sensor

rates, has been validated in our institute [5,6]. Second heart sound can be easily simultaneously recorded by the same sensor and this can be utilized to automatically quantify cardiologic systole and diastole duration in exercise stress tests (Fig 1).

2. Methods

The system is based on a micro-electro-mechanical acceleration sensor with a full scale of $\pm 2 \cdot g$ ($g = 9.8 \text{ m/s}^2$) and a resolution of $0.0005 \cdot g$. We housed the device in a small case which was positioned in the mid-sternal precordial region and was fastened by a solid gel ECG electrode (Fig 2). The acceleration signal was converted to digital and recorded by a laptop PC, together with an ECG signal (obtained by a 3 leads, single channel device). The data were analyzed by using a special purpose software developed in Matlab. A peak detector algorithm, synchronized with the ECG, scans the first 150 ms following the R wave to record the first heart sound vibration and then the interval before the following R wave to record the second heart sound vibration. Cardiologic systole and diastole duration are then computed for each cardiac beat.

The system was tested in 103 patients who performed semi-supine bicycle exercise in stress eco lab, starting with an initial workload of 25 watts, increasing up to the

maximum capacity (an increment of 25 watts every 2 minutes). Patients were 71M/32F, age 57 ± 14 years, 17 healthy people 86 patients with cardiovascular or pulmonary diseases: systemic hypertension (15), coronary heart disease (25), valvular heart disease (13), dilated cardiomyopathy (17) and chronic obstructive pulmonary disease (16).

The local Ethical Committee approved the study protocol.

3. Results

Consistent first and second heart sound signals were obtained in 98% patients at rest and during stress. In 2 patients data were discarded because of a low signal to noise ratio which was related to both small amplitude of the signal and the presence of several artifacts due to heavy movements and/or speaking of the patient.

The diastolic time decreased from $541 \pm 143 \text{ msec}$ at rest to $250 \pm 59 \text{ msec}$ at exercise peak; the systolic/diastolic time ratio increased from 0.64 ± 0.15 to 1.00 ± 0.23 (Fig 3). At higher heart rates (for example 100bpm),

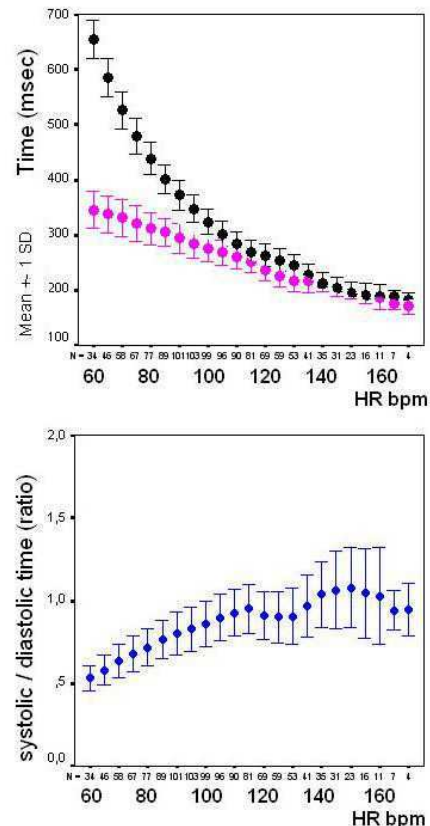


Figure 3: Upper panel: plot of systolic (pink symbols) and diastolic (black symbols) mean \pm SD time values at increasing heart rates. Lower panel: plot of systolic/diastolic time ratio as mean \pm SD time values at increasing heart rates.

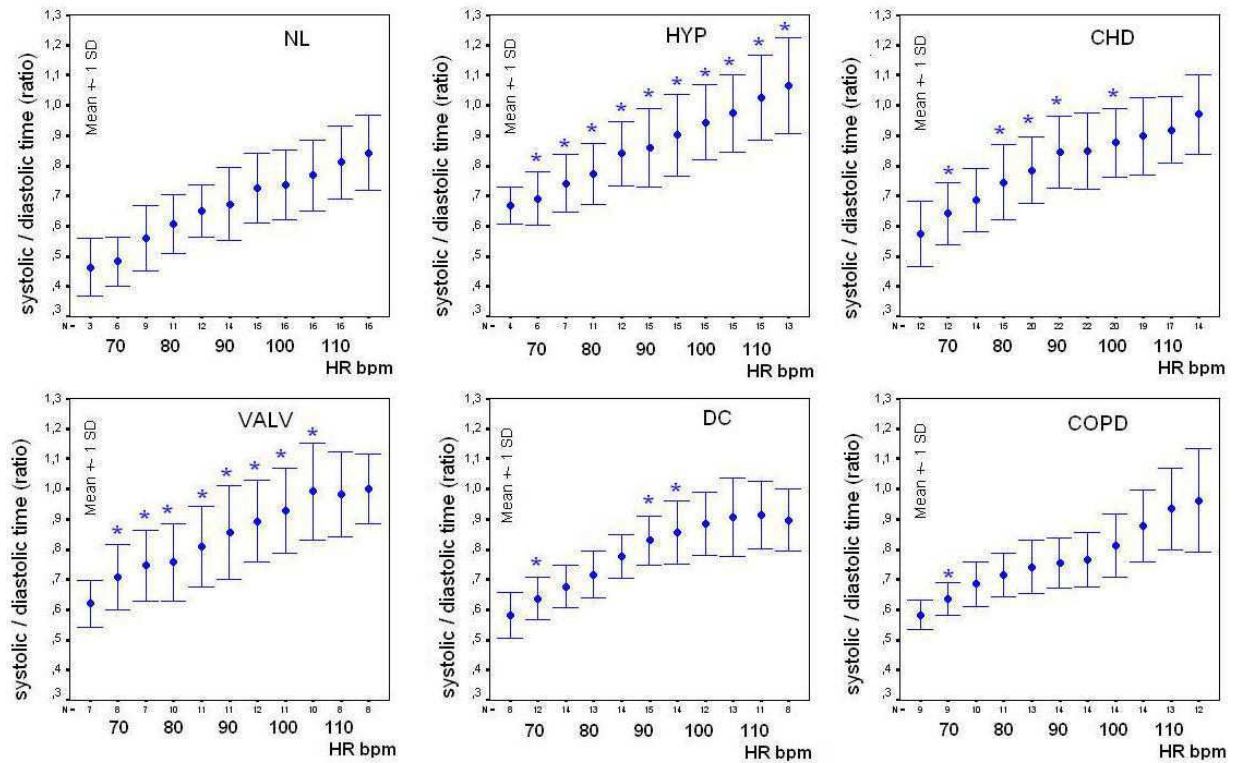


Figure 4: Patients vs. controls. Systolic/diastolic ratio plotted against heart rate in controls (NL, left upper panel) and in patients groups (HYP: systemic hypertension; CHD: coronary heart disease; VALV: valvular heart disease; DC: dilated cardiomyopathy; COPD: chronic obstructive pulmonary disease). At each stage of exercise inter-group comparison was performed and significant differences ($p < 0.05$) are displayed with symbols *.

systolic/diastolic time ratio was lower in the 17 control subjects (0.74 ± 0.12) than in the patients with systemic hypertension (0.94 ± 0.12), coronary (0.88 ± 0.11), valvular (0.93 ± 0.14) or dilated heart (0.86 ± 0.10) disease (all $p < 0.05$ at Post Hoc Scheffé Multiple Comparisons vs. controls). Patients with chronic obstructive pulmonary disease showed a not significantly higher ratio (0.81 ± 0.10) (Fig4).

A typical systolic and diastolic times trend during exercise is shown in Fig 5.

4. Discussion and conclusions

A stable, reproducible, and consistent first heart sound and second heart sound signal was obtained in almost all patients and utilized as time markers to continuously assess cardiologic systole and diastole during exercise. Diastolic time was greater and systolic/diastolic time ratio lower in the 17 control subjects than in the patients. At higher heart rates, the increased systolic/diastolic ratio was accentuated in patients with systemic hypertension, or coronary, dilated or valvular heart disease, reflecting the relatively prolonged systole and shortened diastole in

these patients.

Reversal of the normal systolic/diastolic ratio may compromise cardiac filling and function. Stress-induced "systolic-diastolic mismatch" can be easily quantified by a disproportionate decrease of diastolic time fraction, and is associated to several cardiac diseases, possibly expanding the spectrum of information obtainable during stress.

In conclusion, cardiological systolic and diastolic duration can be monitored in exercise stress test by using an acceleration sensor that measures first and second heart sound vibrations. The same accelerometer sensor and the same arrangement were used, in a previous work, to assess the cardiac force-frequency relation, which can then be assessed simultaneously. Due to the low cost and small dimension of the instrumentation, the approach can be potentially integrated in home monitoring systems.

References

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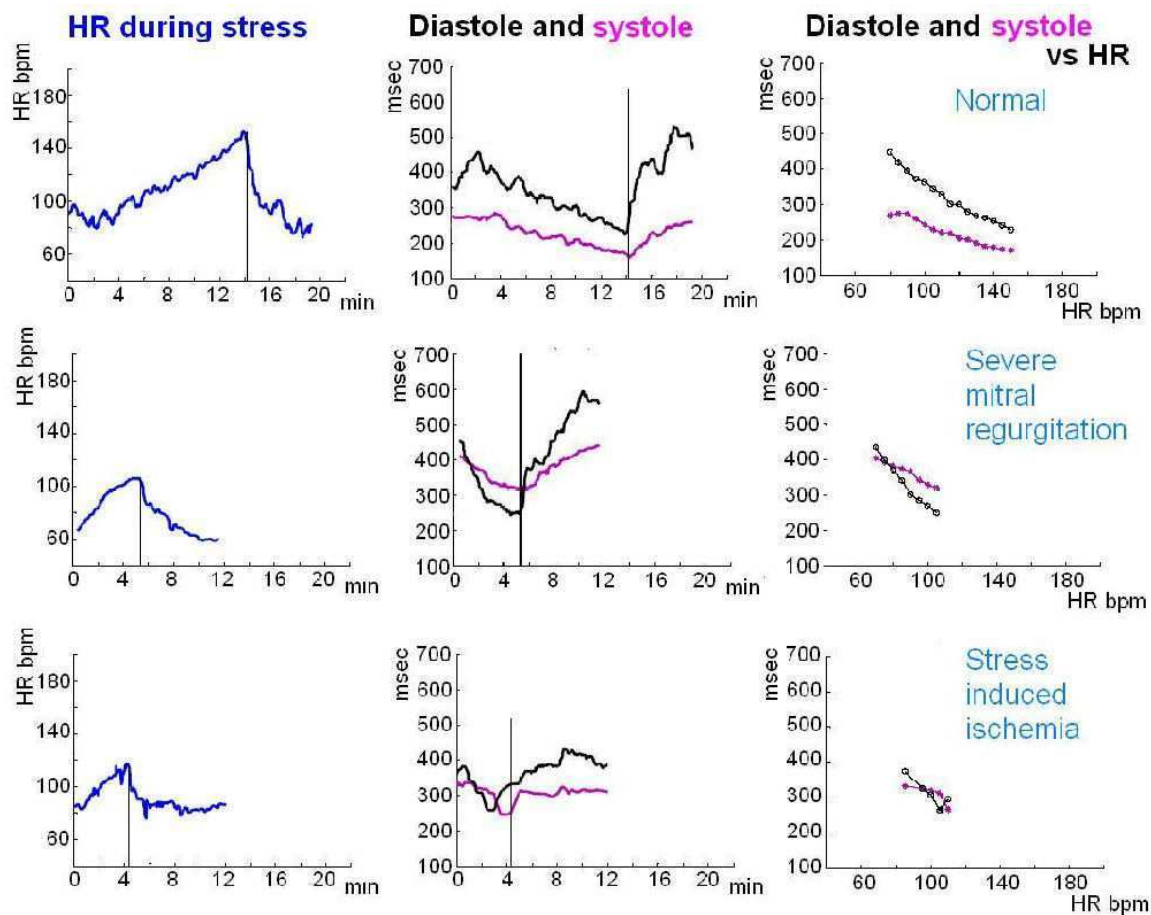


Figure 5: left panels: heart rate during stress; central panels: systolic (pink lines) and diastolic (black lines) times during stress; right panels: systolic and diastolic times plotted vs. heart rate. In a normal subject (upper panels) systolic time was always less than diastolic time. Middle panels show a patient with severe mitral regurgitation and stress induced severe pulmonary hypertension, where the test was stopped at low stress load due to limiting dyspnoea without stress induced ischemia. Prolonged systolic time with systolic/diastolic time reversal occurred during stress. Lower panels show a patient with stress induced ischemia at low stress load: at ischemia systole lengthens with systolic/diastolic time reversal.

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