A Noninvasive Method of Measuring Force-Frequency Relations to Evaluate Cardiac Contractile State of Patients during Exercise for Cardiac Rehabilitation

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Abstract—*Background* **Evaluation of the contractile state of the left ventricle during exercise is important for cardiac rehabilitation. As yet, no noninvasive methods for this purpose have been established. The force-frequency relation (FFR) during exercise has the potential for evaluating the contractile state noninvasively. Color Doppler- and echo tracking-derived carotid arterial wave intensity is a sensitive index of global left ventricular (LV) contractility.**

Objectives **We assessed the feasibility of measuring carotid arterial wave intensity and determining FFR's during exercise totally noninvasively.**

Methods **We enrolled 18 healthy men (age 20.6 ± 2.1 years). Using ultrasonic diagnostic equipment, we measured wave intensity in the carotid artery and heart rate (HR) before and during ergometer exercise. FFR's were constructed by plotting the maximum value of wave intensity (WD_1) against heart rate (HR).**

Results **WD_1 increased linearly with an increase in HR during exercise. The regression line of WD_1 on HR represented the FFR.**

Conclusion **Using WD_1 and gradual exercise test, we obtained FFR's noninvasively. These data should show the potential usefulness of FFR in practicing cardiac rehabilitation.**

***Keywords*—Force-Frequency Relation, Echocardiography, Wave Intensity, Exercise.**

I. INTRODUCTION

When we administer exercise training to a patient for cardiac rehabilitation, we should constantly monitor the patient's condition. In normal subjects, cardiac contractility increases with an increase in heart rate (HR). This phenomenon is called the Force-Frequency Relation (FFR). HR is an important determinant of myocardial performance, but increased HR does not necessarily increase cardiac contractility in patients with heart disease. The relation between cardiac contractility and HR in a diseased heart is different from that in healthy animals and humans(1). Conventionally, FFR's were obtained by measuring the maximum rate of LV pressure rise (Peak dP/dt) with a catheter-tipped micromanometer as an index of cardiac contractility, and using atrial pacing to change heart rate (Peak dP/dt - HR relation) (2). This is an invasive method and cannot be used repeatedly in the clinical setting.

It has been demonstrated that color Doppler- and echo tracking-derived carotid arterial wave intensity is a sensitive index of global left ventricular (LV) contractility (3). During exercise, HR increases with an increase in workload, therefore atrial pacing is not needed for changing HR. It has also been demonstrated that the Peak dP/dt-HR relation is markedly enhanced (the slope is increased) during exercise compared with during pacing in normal hearts, but the enhancement is limited in diseased hearts (2). Therefore, the FFR obtained by exercise may have higher power to discriminate cardiac contractile states than that obtained by pacing.

In this study, we assessed the usefulness of the measurement of wave intensity during exercise in obtaining FFR's, and evaluated the feasibility of an entirely noninvasive method for demonstration of the FFR.

II. METHODS

A. Noninvasive Measurements of Wave Intensity

Wave intensity (WI) is a hemodynamic index, which is defined as

$$WI = (dP/dt)\,(dU/dt), \quad (1)$$

where dP/dt and dU/dt are the derivatives of blood pressure (P) and velocity (U) with respect to time, respectively. The maximum value of WI during a cardiac cycle (W_1) significantly correlates with Peak dP/dt (or max dP/dt) (4).

L.M. Roa Romero (ed.), *XIII Mediterranean Conference on Medical and Biological Engineering and Computing 2013*, IFMBE Proceedings 41,
DOI: 10.1007/978-3-319-00846-2_376,

In our method of obtaining carotid arterial WI, carotid diameter-change waveform was used as a surrogate for carotid pressure waveform (3). Using measured diameter-change waveforms directly, we can also define a wave intensity (WD) as

$$WD = (1/D)(dD/dt)(dU/dt). \quad (2)$$

WD is obtained by measuring U and D without measuring upper arm pressure (Fig.1), which is easier to perform during exercise. The definition of the stiffness parameter (β) gives the relation

$$(1/D)(dD/dt) = (1/\beta P)(dP/dt). \quad (3)$$

Therefore,

$$WD = (1/\beta P)\, WI. \quad (4)$$

Hence, the maximum value of carotid arterial WD during a cardiac cycle (WD_1) correlates with the maximum value of WI (W_1) (Fig.2). Therefore WD_1 correlates with Peak dP/dt as W_1 does. The details of the method of measurements were described elsewhere (3).

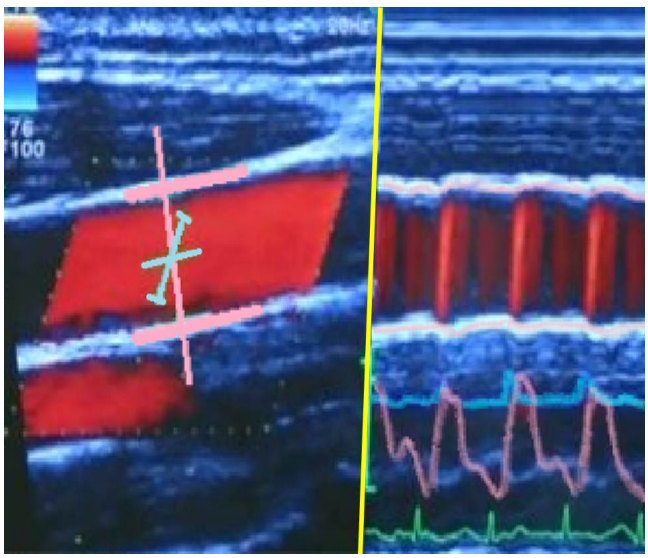

Fig. 1 Simultaneous measurements of diameter change wave form and blood flow velocity. View on the monitor during the measurements. *Left* : Color Doppler / B-mode long-axis view of the common carotid artery. Pink line and blue line indicate the ultrasonic beam for echo-tracking and for blood flow velocity measurement, respectively. By setting the tracking positions displayed as pink bars on the echo-tracking beam to arterial walls, echo-tracking automatically starts. *Right* : The diameter-change waveforms, which is calculated by subtracting the distance to the near wall from that to the far wall, and the velocity waveforms are displayed on the M-mode view.

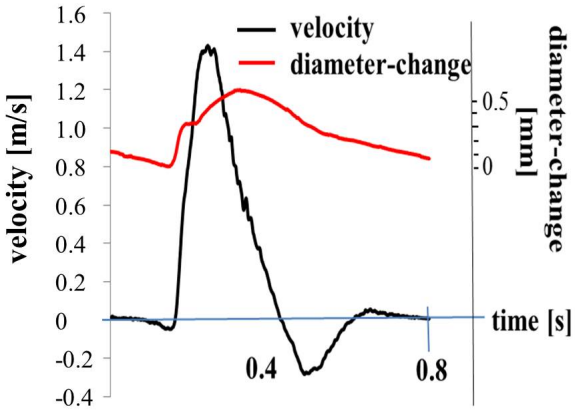

Fig. 2a.: Diameter-change and flow velocity waveforms is displayed.

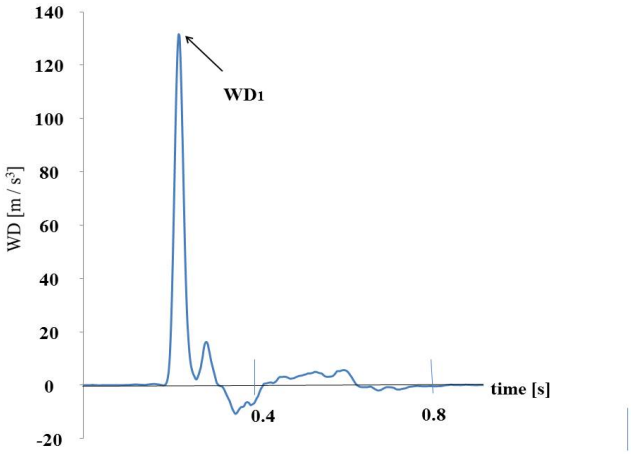

Fig. 2b Wave intensity (WD) is calculated as WD = (1/D)(dD/dt)(dU/dt).

B. Subjects

We studied 18 healthy male volunteers (mean age 20.6 ± 2.1 years, age range 19 – 22 years). We obtained informed consent from all the subjects. Approval from the Ethics Committee of Himeji Dokkyo University was obtained before study initiation.

C. Protocol

First, before the measurements of WD_1 and HR, the subjects lay down in the semi-supine position for 10 minutes on the strength ergometer. The location to be measured was the common carotid artery at about 2cm proximal to the carotid bulb. We used scanning in the long

axis view, and obtained a B-mode image of a longitudinal section of the artery (Fig.1 left). With the B- and M-mode scans displayed simultaneously on a split screen, the echo-tracking system tracked the vessel wall movements to produce displacement waveforms of the anterior and posterior artery walls (Fig.1 right). This gave the diameter-change waveforms.

Next, after the measurements of WD_1 and HR at rest, gradual bicycle exercise was performed starting at an initial workload of 20 W and lasting for 2minutes; thereafter, the workload was increased stepwise by 20 W at 1-minute intervals. Electrocardiogram was continuously monitored. The criteria for the endpoint included increase of heart rate to [(220−age) × 0.8 (bpm)], and the impossibility of continuing exercise. We measured WD_1 and HR during the exercise.

D. Data Analysis

The obtained data are expressed as mean ± standard deviation. The scatter diagram of the points (HR, WD_1) for the data during exercise from each subject was analyzed by the linear regression method, and the regression line was regarded as the FFR.

III. RESULTS

All subjects at exercise were able to pedal the strength ergometer and effectively increased their HR (mean HR at rest vs. mean HR at peak exercise: 75 beats/ min vs. 154 beats/ min, $p < 0.0001$). WD_1 increased linearly with an increase in HR (Fig.3). The goodness-of-fit of the regression line of WD_1 on HR in each subject was very high ($r^2 = 0.67 \sim 0.94$, $p < 0.0001$, respectively). The slope of the WD_1-HR relation (FFR) varied with the individual ranging from 0.31 to 2.2 [m/s^3 (beat / min)] (Fig. 3).

IV. DISCUSSION

The basic property of the FFR to progressively enhance myocardial contractility as heart rate increases (frequency potentiation) has been observed in normal humans. This mechanism is augmented due to β-adrenergic stimulations induced by exercise in normal hearts. However, significant impairment of exercise-induced amplification of the FFR is observed in diseased hearts (2). Therefore, one would expect the diversity of responses of the FFR by exercise ensures increased sensitivity for detection of contractile impairment.

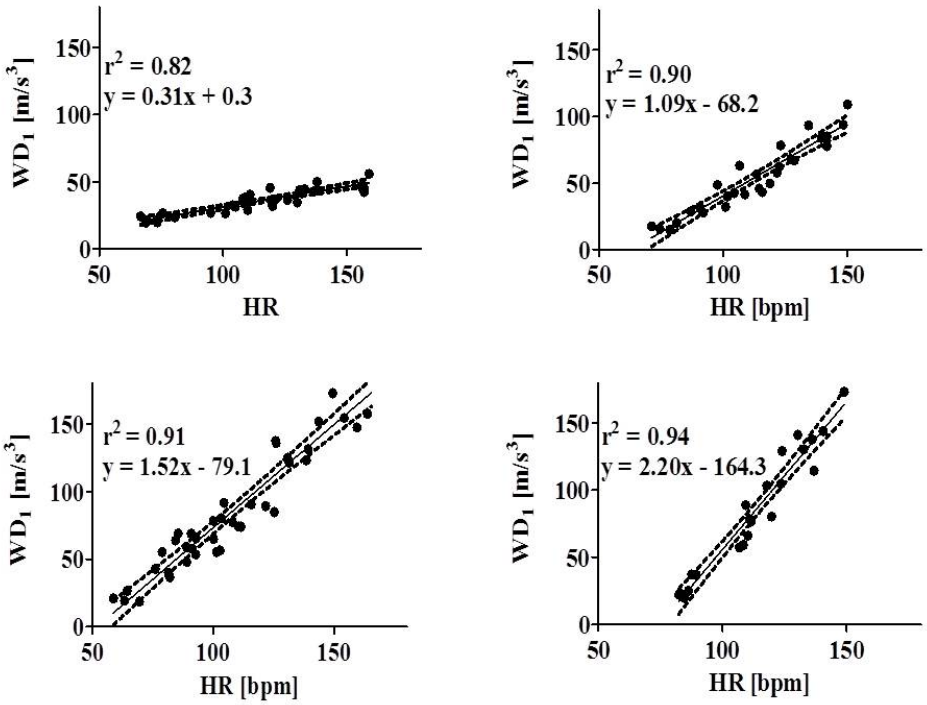

Fig. 3 Representative FFR's in the study.

Equation (4) shows that WD contains β. It is widely known that β increases with age. However, our study subjects only ranged from 19 to 22 in age. Therefore, β was considered to vary only slightly with the individual. In spite of this, the slope of the FFR varied relatively widely. This is considered to show that the FFR during exercise is determined mainly by the cardiac contractile state rather than arterial conditions.

Limitations

Our study subjects only ranged from 19 to 22 in age. We should enroll a greater number of subjects to be divided into age groups in future study. The final goal of our study is to apply the FFR in practicing cardiac rehabilitation. However, we did not enroll patients with heart disease in the present feasibility study.

V. CONCULUSION

Measurements of carotid arterial wave intensity by echocardiography during exercise give the FFR noninvasively. This will be useful for the evaluation of the contractile state of the left ventricle during exercise for cardiac rehabilitation.

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