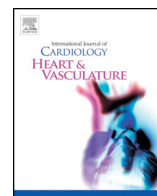




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## Associations of increased arterial stiffness with left ventricular ejection performance and right ventricular systolic pressure in mitral regurgitation before and after surgery: Wave intensity analysis

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### ABSTRACT

**Background:** The effect of increased arterial stiffness on mitral regurgitation (MR) is not clear. Using wave intensity (WI) analysis, which is useful for analyzing ventriculo-arterial interaction, we aimed to elucidate associations of increased arterial stiffness with left ventricular (LV) ejection performance and right ventricular systolic pressure (RVSP) in MR.

**Methods and Results:** We noninvasively measured carotid arterial WI and stiffness parameter ( $\beta$ ) in 98 patients with non-ischemic chronic MR before and after surgery, and 98 age-and-gender matched healthy subjects by ultrasonography. WI is defined as  $WI = (dP/dt)(dU/dt)$  [P: blood pressure, U: velocity, t: time]. The peak value of WI ( $W_1$ ) increases with LV peak  $dP/dt$ . The temporal WI index ( $Q-W_1$ )<sub>st</sub>, which is the standardized interval between the Q wave of the ECG and  $W_1$ , is a surrogate for preejection period. Ejection fraction (EF), left atrial volume index (LAVI), effective regurgitant orifice area (ERO), RVSP, and other echocardiographic data were also obtained.  $W_1$  was enhanced in the MR group before surgery compared with the normal group ( $10.7 \pm 5.7$  vs  $8.5 \pm 3.6 \times 10^3$  mmHg m/s<sup>3</sup>,  $p < 0.05$ ). However, the results of two-way ANOVA showed this enhancement of  $W_1$  was observed only in the subgroup of MR before surgery with lower arterial stiffness ( $\beta < 13$ ,  $p < 0.0001$ ). ERO,  $\beta$  and LAVI were predictor variables before surgery to determine RVSP. EF and ( $Q-W_1$ )<sub>st</sub> before surgery were predictor variables for EF after surgery.

**Conclusions:** In the MR group before surgery, increased arterial stiffness suppresses compensatory enhancement of  $W_1$ , and increases RVSP. Prolonged ( $Q-W_1$ )<sub>st</sub> has the potential for predicting low EF after surgery.

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### 1. Introduction

Aged patients with nonischemic mitral regurgitation (MR) due to the flail leaflet were reported to suffer for excess rate of mortality in comparison with that expected in age-peer general population, but this rate difference between younger MR patients and younger general population did not show statistical significance [1]. The presumed causes are increased left ventricular (LV) myocardial

stiffness, reduced left atrial (LA) function, higher rate of complicated atrial fibrillation (AF), and increased right ventricular systolic pressure (RVSP) in aged patients [1–3]. However, since arterial stiffness increases with age inevitably, the effects of increased arterial stiffness on ventriculo-arterial interaction relevant to the ejection performance in MR should also be taken into consideration.

Wave intensity (WI) is a hemodynamic index obtained from an arterial site, which provides quantitative information about the dynamic behavior of the heart and the vascular system and their interaction [4–6]. The noninvasive measuring system of WI also provides arterial stiffness parameters [6]. We used WI analysis for elucidating the effects of increased arterial stiffness on cardiac performance and RVSP in severe MR, and for predicting ejection fraction (EF) after surgery.

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## 2. Methods

### 2.1. Study population

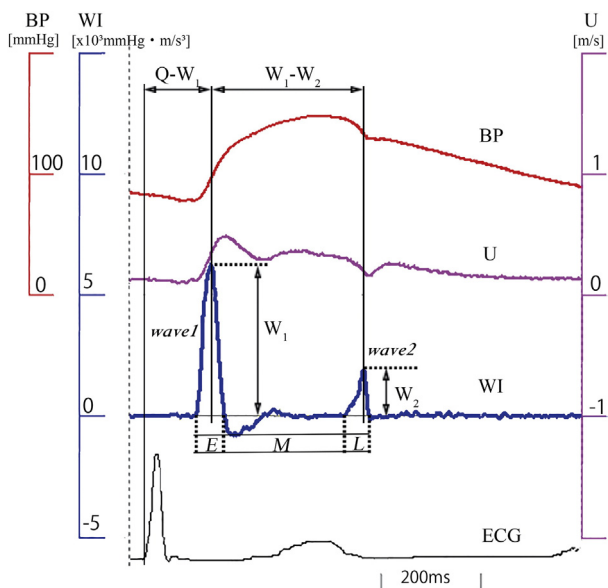
We studied 121 consecutive patients with non-ischemic chronic MR (71 men, age  $54 \pm 15$  years) who underwent surgical treatment for MR. Twenty-three subjects were excluded because of concomitant aortic valve disease ( $n = 7$ ), mitral stenosis ( $n = 4$ ), AF ( $n = 6$ ), ischemic heart disease ( $n = 3$ ), hypertrophic cardiomyopathy ( $n = 1$ ) or aortic disease ( $n = 2$ ), and the remaining 98 subjects were enrolled in the study. The study group included the patients with a history of hypertension ( $n = 37$ ), hyperlipidemia ( $n = 28$ ), diabetes mellitus ( $n = 3$ ) and medication with angiotensin converting enzyme inhibitors or angiotensin II receptor antagonists ( $n = 47$ ), diuretics ( $n = 35$ ), calcium channel blockers ( $n = 17$ ) and  $\beta$ -blockers ( $n = 7$ ). Normal values of WI indices were obtained from 98 age-matched and gender-matched healthy participants (60 men, age  $52 \pm 14$  years). All subjects provided informed consent, and the ethic committee of Sakakibara Heart Institute approved the study protocol.

### 2.2. Noninvasive measurements of WI and arterial stiffness

WI is given by

$$WI = (dP/dt)(dU/dt),$$

where  $dP/dt$  and  $dU/dt$  are the derivatives of pressure ( $P$ ) and velocity ( $U$ ) with respect to time [6]. Carotid arterial WI in a normal subject shows two sharp positive waves during a cardiac cycle, wave 1 and wave 2, which are generated by the left ventricle and divide ejection period into three phases (Fig. 1). Wave 1 occurring in early ejection (indicated by E in Fig. 1) corresponds to the forward travelling compression (pushing) waves, and the height of carotid arterial wave 1 ( $W_1$ ) correlates with LV peak  $dP/dt$  [7]. The interval between the Q wave of the ECG and  $W_1$  ( $Q-W_1$ ) and the interval between  $W_1$  and  $W_2$  ( $W_1-W_2$ ) are temporal indices which are used as surrogates for pre-ejection period and ejection time. Wave 2 is the forward travelling expansion (suction) wave generated by rapid fall in LV pressure [6,8,9].



**Fig. 1.** Representative recordings of pressure (BP), blood flow velocity (U), and calculated wave intensity (WI) in the common carotid artery, and electrocardiogram (ECG) obtained from a normal subject. WI in a normal subject shows two sharp positive peaks during a cardiac cycle, wave 1 and wave 2, and the ejection period is divided into three phases (initial (E), mid (M) and late ejection (L)) by the two waves.

WI was obtained noninvasively with an ultrasonic system (SSD 6500, Hitach-Aloka, Tokyo, Japan) which simultaneously measures arterial diameter-change waveform by echo tracking and blood flow velocity by color Doppler. Arterial diameter-change waveform was calibrated by the upper arm blood pressure measured with a validated automated cuff-type sphygmomanometer (HEM-907, OMRON Healthcare Co., Ltd., Kyoto, Japan) immediately after the waveforms were obtained, and used as surrogates for blood pressure waveforms. By inputting the blood pressure data, WI indices and  $\beta$  were calculated automatically and waveforms and ECG were digitally recorded at an interval of 1 ms. The details and reproducibility of the system were described elsewhere [10]. This system measures the height of the two positive peaks ( $W_1, W_2$ ), the interval between the peaks of the two waves ( $W_1-W_2$ ) and the interval between the R wave of the ECG and  $W_1$  ( $R-W_1$ ). To measure the precise interval from the onset of the QRS complex of ECG to the peak of wave 1 ( $Q-W_1$ ), we used custom software written in our laboratory retrieving stored text file data of WI.

The WI measuring system also calculates the carotid arterial stiffness parameter  $\beta$  [11], which is defined as follows:

$$\beta = \ln(Ps/Pd)/[(Ds-Dd)/Dd],$$

where  $Ps$  and  $Pd$  are systolic and diastolic pressure (mm Hg) and  $Ds$  and  $Dd$  are the maximum and minimum diameters (mm) of the carotid artery in a cardiac cycle, respectively.

### 2.3. Echocardiography

Echocardiographic evaluation was performed in MR subjects using an echo machine (SONOS 7500; Philips Healthcare, MA, USA) according to the recommendations of the American Society of Echocardiography [12]. LV end-diastolic and end-systolic volume index (LVEDVI and LVESVI), LA volume index (LAVI) were determined using the modified Simpson's method. LA volume was measured at the end-systole just before the mitral valve opening. RVSP was obtained by adding the systolic tricuspid pressure gradient calculated by the modified Bernoulli equation and right atrial pressure [13]. Early filling (E) and atrial contraction filling (A) velocities of transmitral flow were measured using pulsed Doppler, and tissue Doppler velocity of the septal mitral annulus in early diastole ( $e'$ ) was also obtained from the apical four-chamber view. MR severity was quantified as averaged effective regurgitant orifice area (ERO) obtained by the Doppler volumetric method and proximal isovelocity surface area (PISA) method [14]. Regurgitant volume (RegV) and Regurgitant fraction (RegF) were calculated as  $RegV = (\text{mitral stroke volume}) - (\text{aortic stroke volume})$ , and  $RegF = RegV/(\text{mitral stroke volume})$ , respectively.

### 2.4. Measurement protocol

Measurements were performed before and early after surgery. After 15 min rest, WI data were acquired from the left common carotid artery at about 2 cm proximal to the carotid bulb. The transducer was held in place by a stereotactic clamp (Point setter, Mitaka Kohki, Co., Ltd., Tokyo, Japan) to avoid the movement of the observer. The measurements were performed three times and averaged data was used for analysis. Echocardiography was performed according to routine practice by skilled sonographers without knowledge of WI data.

### 2.5. Statistical analysis

Results were expressed as mean value  $\pm$  SD (standard deviation). Comparisons among groups were performed by Student's  $t$ -test or ANOVA, followed by Bonferroni or Dunnett test when necessary. To evaluate the relationship between WI indices or echocardiographic indices and  $\beta$ , Pearson's product-moment correlation was used. Each of the MR groups before surgery, after surgery and the normal group

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