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A noninvasive method of measuring wave intensity, a new hemodynamic index: application to the carotid artery in patients with mitral regurgitation before and after surgery

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Summary. Wave intensity (WI) is a new hemodynamic index, which is defined as $(dP/dt)(dU/dt)$ at any site of the circulation, where dP/dt and dU/dt are the time derivatives of blood pressure and velocity, respectively. Arterial WI in normal subjects has two positive sharp peaks. The first peak occurs during early systole when a forward-traveling compression wave is generated by the left ventricle. The magnitude of this peak increases markedly with an increase in cardiac contractility. The second peak, which occurs towards the end of systole, is caused by generation of a forward-traveling expansion wave by the ability of the left ventricle to actively stop aortic blood flow. The interval between the R wave of the ECG and the first peak of WI (R–1st peak interval) and the interval between the first and second peaks (1st–2nd interval) are approximately equal to the preejection period and left ventricular ejection time, respectively. Using a combined Doppler and echo-tracking system, we obtained carotid arterial WI noninvasively. We examined the characteristics of WI in 11 patients with mitral regurgitation (MR) before and after surgery, and 24 normal volunteers. In the MR group before surgery, the second peak was decreased and the (1st–2nd interval)/(R–R interval) ratio was reduced, compared with the normal group (140 ± 130 vs 750 ± 290 mmHg m/s^3, $P < 0.0083$; 20.7% ± 3.4% vs 26.7% ± 2.8%, $P < 0.0083$). There were no significant differences in the first peak between the normal group and the MR group before and after surgery. The second peak in the MR group was increased significantly ($P < 0.016$ vs before surgery) to 1150 ± 830 mmHg m/s^3 in the early period after surgery (stage I), and to 1090 ± 580 mmHg m/s^3 in the late period after surgery (stage II). These values did not differ significantly from that of the normal group. At stage I, the (R–1st peak interval)/(R–R interval) ratio was increased from 13.4% ± 2.7% to 20.6% ± 5.6% ($P < 0.016$ vs before surgery). At stage II, this ratio decreased to 16.2% ± 2.8% ($P < 0.016$ vs stage I), but was still significantly higher than that before surgery. The (1st–2nd interval)/(R–R interval) ratio increased significantly after surgery ($P < 0.016$ vs before surgery) to values (27.0% ± 4.5% at stage I and 28.9% ± 2.6% at stage II) which did not differ significantly from that of the normal group. The recovery of the second peak after surgery suggests that the left ventricle had recovered the ability to actively stop aortic blood flow. Wave intensity is useful for analyzing changes in the working condition of the heart.

Key words: Wave intensity – Ventriculo-arterial interaction – Carotid artery – Mitral regurgitation – Expansion wave

Introduction

Wave intensity is a new hemodynamic index proposed by Parker et al. [1, 2]. It can be defined at any site in the circulatory system, and provides information about the interaction of forward- and backward-traveling waves, and hence, information about the dynamic behavior of the heart and vascular system and their interaction [3–8].

We developed a new method of obtaining wave intensity, noninvasively, using a combined Doppler and echo-tracking system, and applied the method to

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the carotid artery in patients with mitral regurgitation (MR) before and after surgical treatment.

Materials and methods

Definition of time-normalized wave intensity

Wave intensity was originally defined as the product of ΔP and ΔU, where ΔP and ΔU are the changes in blood pressure P and velocity U, respectively, during constant short time intervals [1, 2]. This original wave intensity depends on the sampling interval, Δt, which makes it difficult to compare data taken at different sampling rates. Therefore, we normalized wave intensity by dividing ΔP and ΔU by Δt [3, 5]. Thus, the normalized wave intensity (WI) is given by

$$\mathrm{WI} = (\Delta P/\Delta t)(\Delta U/\Delta t).$$

For small values of Δt, WI can be written as

$$\mathrm{WI} = (\mathrm{d}P/\mathrm{d}t)(\mathrm{d}U/\mathrm{d}t),$$

where $\mathrm{d}P/\mathrm{d}t$ and $\mathrm{d}U/\mathrm{d}t$ are the derivatives of P and U with respect to the time. This time-normalized wave intensity (WI) has the same property as the original wave intensity: if $\mathrm{WI} > 0$, the changes in pressure and velocity caused by the forward wave are greater than those caused by the backward wave, and vice versa (see Appendix 1).

Noninvasive measurements of blood pressure and velocity waveforms

To calculate WI, we need a blood pressure waveform. Many studies have demonstrated that arterial pressure waveforms and diameter-change waveforms are similar [9–11]. Therefore, we measured changes in the diameter of the carotid artery, and calibrated its peak and bottom values by systolic and diastolic blood pressure measured with a cuff-type manometer applied to the upper arm.

We used a combined Doppler and echo-tracking system, QFM 1100 (Hayashi Denki, Kanagawa, Japan), which can measure blood flow velocity and changes in diameter of the carotid artery simultaneously [12]. Four transducers are assembled into the probe of the device. One is for ultrasonic pulses with a frequency of 7.5 MHz. This is utilized for an echo-tracking system with a phase-locked loop, which measures arterial internal diameter and its changes. The actual measuring range of vessel diameter was 2–15 mm.

The other three transducers form a line: one is a transmitter of a continuous wave of 5 MHz and the other two are receivers for a Doppler system to measure blood flow velocity. By using Doppler signals from the two receivers, the absolute value of flow velocity is obtained regardless of the Doppler beam incident angle to blood flow [12]. The actual measuring range of velocity was 5–120 cm/s.

Subjects

We studied 11 patients with MR (7 men and 4 women, age: 49 ± 11 years) and 24 normal volunteers (15 men and 9 women, age: 52 ± 17 years). Surgical therapy was indicated in all patients. Subjects with the complication of mitral stenosis or another valvular disease were not enrolled in this study. The clinical data of the patients are shown in Table 1.

Data acquisition and analysis

We measured carotid arterial blood flow velocity and diameter with the above system in patients with MR and normal subjects in the supine position at rest. In the MR patients, the measurements were performed at

Table 1. Clincial and echocardiographic data

Case	Sex	Age (years)	Height (cm)	Weight (kg)	Dd (cm)	Ds (cm)	FS	Surgical procedure
1	F	59	158	53	5.4	3.2	0.41	Valve replacement
2	M	50	170	57	6.1	4.0	0.34	Valvuloplasty
3	F	58	156	68	5.2	3.2	0.33	Valve replacement
4	F	34	155	45	7.7	4.9	0.36	Valve replacement
5	M	56	171	74	6.2	4.5	0.27	Valvuloplasty
6	F	60	153	54	5.8	3.2	0.45	Valvuloplasty
7	M	51	165	57	6.8	4.6	0.32	Valvuloplasty
8	M	25	181	82	6.3	3.9	0.35	Valvuloplasty
9	M	51	161	62	5.8	3.6	0.38	Valve replacement
10	M	41	178	100	6.4	3.8	0.40	Valvuloplasty
11	M	55	165	59	7.8	4.6	0.41	Valve replacement
Mean		49	165	65	6.3	4.0	0.37	
SD		11	9	16	0.8	0.6	0.05	

Dd, left ventricular diastolic dimension; Ds, left ventricular systolic dimension; FS, fractional shortening

three different times: before surgery, within 1 month after surgery (stage I), and more than 6 months after surgery (stage II).

Simultaneous recordings of blood flow velocity, diameter, and ECG were made on a magnetic tape recorder (PC 116, Sony, Tokyo, Japan). At the same time, blood pressure in the arm was measured with a cuff-type sphygmomanometer. The data were fed into a computer system (FMV desk power, Fujitsu, Tokyo, Japan) via an analog-to-digital converter at a sampling interval of 2 ms. Diameter-change waveforms were interpreted as pressure waveforms after calibration by the measured blood pressure. Data for five consecutive beats were averaged to obtain representative data for one beat. dP/dt and dU/dt were obtained by differentiating digitally the arterial pressure (P) and velocity (U) with respect to the time.

Figure 1 shows representative recordings of pressure (diameter) and blood flow velocity, and calculated WI obtained from the right common carotid artery of a normal subject. WI has two positive peaks. The first peak appears in early ejection, and the second peak in late ejection. We derived two intensive indices from WI: the height of the first and the second peak. We also derived two temporal indices: the interval between the R wave of the ECG and the first peak of WI (R–1st peak interval), and the interval between the first and second peaks (1st–2nd interval). To compare temporal indices obtained at different heart rates, we divided these intervals by the R–R interval of the ECG.

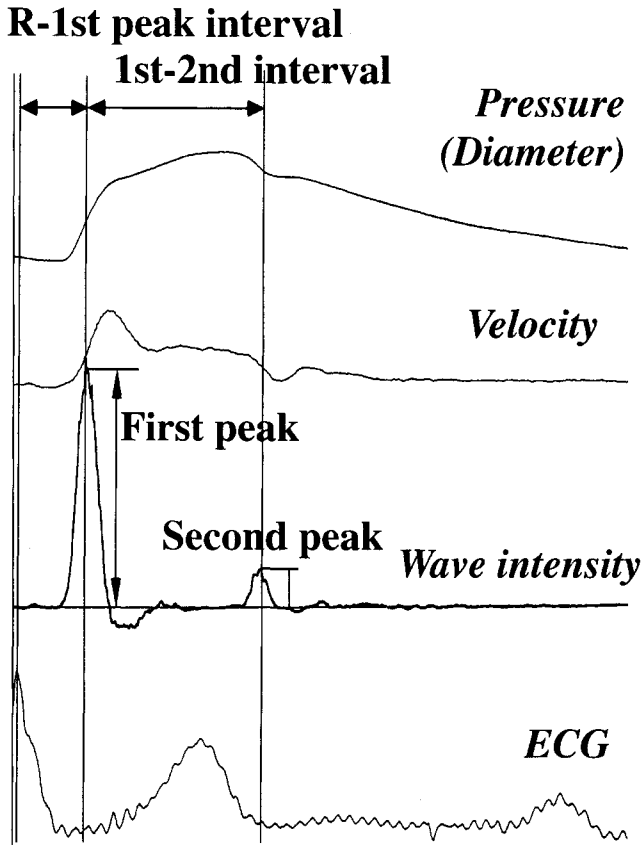

Fig. 1. Representative recordings of pressure, blood flow velocity, and calculated wave intensity (WI) in the right common carotid artery, and electrocardiogram (*ECG*) obtained from a normal subject. The pressure waveform is obtained from the diameter-change waveform, with its peak and bottom values calibrated by the blood pressure in the upper arm measured with a sphygmomanometer. WI has two positive peaks. The first appears in early ejection, and the second in late ejection. We derived two intensive indices: the height of the first peak and the second peak; and two temporal indices: the interval between the R wave of the ECG and the first peak of WI (*R–1st peak interval*), and the interval between the first and second peaks (*1st–2nd interval*)

Statistics

The results are presented as mean ± SD. The data in the normal group and the MR group before surgery, and at stages I and II were compared by analysis of variance (ANOVA), followed by the Bonferroni t-test to isolate differences, when necessary (comparisons were not considered significant unless the corresponding P value was less than $0.05/6 = 0.0083$). Repeated-measures ANOVA was used for comparing the data in the MR group before surgery, and at stages I and II, followed by the Bonferroni t-test (comparisons were not considered significant unless the corresponding P value was less than $0.05/3 = 0.016$).

Results

Characteristics of WI in patients with MR

There were no significant differences in age, heart rate, blood pressure, and maximum blood flow velocity between the group of patients with MR before surgery and the normal group (Table 2).

There was no significant difference in the height of the first peak between the two groups. However, the height of the second peak was significantly reduced in the MR group. In three of the MR patients, the second peak disappeared completely (the 1st–2nd interval data in these patients were not included in the statistical analysis). Concerning the temporal indices derived from WI, there were no significant differences in the R–1st peak interval and the (R–1st peak interval)/(R–R interval) ratio between the two groups, but the 1st–2nd interval and the (1st–2nd interval)/(R–R interval) ratio were significantly shorter in the MR group. Figure 2 shows a comparison of representative carotid arterial WI from the MR group and the normal group.

Table 2. Measurements and indices of wave intensity before and after surgery

	Normal	MR		
		Before	Stage I	Stage II
Age	52 ± 17	49 ± 11		
Men:women	15:9	7:4		
1st peak (mmHg m/s^3)	5500 ± 1950	6180 ± 2810	4600 ± 1280	5940 ± 2610
2nd peak (mmHg m/s^3)	750 ± 290	140 ± 130*	1150 ± 830#	1090 ± 580#
R–1st interval (ms)	118 ± 13	122 ± 17	140 ± 17*	127 ± 22
(R–1st interval)/R–R (%)	12.0 ± 1.7	13.4 ± 2.7	20.6 ± 5.6*#	16.2 ± 2.8*#§
1st–2nd interval (ms)	261 ± 23	201 ± 27*	193 ± 41*	228 ± 20
(1st–2nd interval)/R–R (%)	26.7 ± 2.8	20.7 ± 3.4*	27.0 ± 4.5#	28.9 ± 2.6#§
Heart rate (bpm)	62 ± 7	66 ± 13	87 ± 19*#	77 ± 9*
Systolic pressure (mmHg)	120 ± 24	108 ± 17	102 ± 13	115 ± 20
Diastolic pressure (mmHg)	72 ± 12	68 ± 9	66 ± 7	70 ± 11
Max V (m/s)	0.55 ± 0.18	0.56 ± 0.17	0.57 ± 0.24	0.58 ± 0.17

MR, patients with mitral regurgitation; before, before surgery; stage I, measurements within 4 weeks after surgery; stage II, measurements more than 6 months after surgery; 1st peak, height of the first peak; 2nd peak, height of the second peak; R–1st interval, interval between the R wave of the ECG and the first peak; R–R, R–R interval of the ECG; 1st–2nd interval, interval between the first and second peaks; Max V, maximum velocity in the carotid artery
Values are mean ± SD. * $P < 0.0083$ vs the normal group; # $P < 0.016$ vs before surgery; § $P < 0.016$ vs stage I

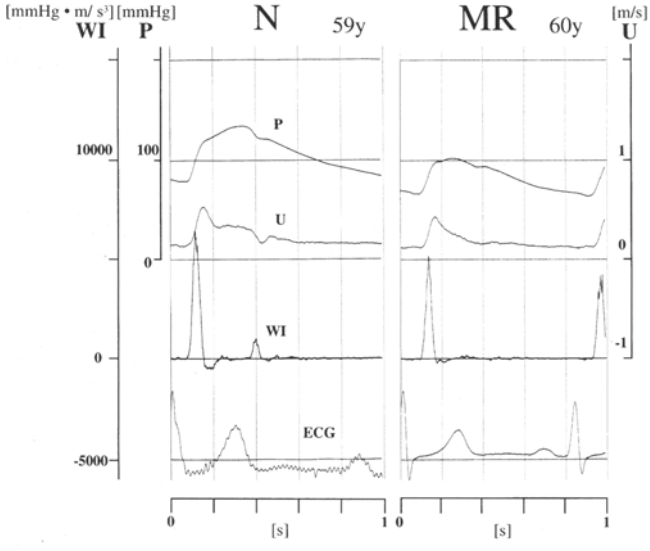

Fig. 2. Recordings of pressure (P), velocity (U), and wave intensity (WI) in the right common carotid artery, and ECGs representative of the normal group (N) and the mitral regurgitation (MR) group (case 6). Compared with the normal group, the second peak is markedly decreased in the MR group

Changes in wave intensity after surgery

Valvuloplasty was performed in six patients and valve replacement with an artificial bileaflet mechanical valve was performed in five patients, all successfully. The measurements at stage II were performed at 19.7 ± 6.7 (mean ± SD) months after surgery. The changes in WI indices and other measurements before and after surgery are shown in Table 2, and representative waveforms of WI before and after surgery are shown in Fig. 3.

There were no significant differences in the first peak between the normal group and the MR group before surgery, and at the two stages after surgery. The second peak was significantly increased at stage I, and remained increased at stage II.

Discussion

Noninvasive measurement of pressure waveform

To obtain pressure waveform noninvasively, we assumed that the carotid arterial pressure–diameter relationship is linear, which may bring about criticism. Sugawara et al. reported the carotid arterial pressure–diameter relationship in eight patients with heart disease [11]. In their study, the pressure was measured with a catheter-tipped micromanometer and the diameter was measured, simultaneously, with an echo-tracking system (QFM-1100). In all eight patients, the carotid arterial pressure–diameter relationship showed slight nonlinearity and hysteresis, especially near the peak pressure. However, the effects were not serious, and we can practically regard the pressure–diameter relationship as being linear. Furthermore, at the time of peak pressure, WI is more or less zero, because dP/dt is very small. Therefore, the nonlinearity and hysteresis do not lead to a noticeable error in the calculation of WI.

Wave intensity as a new index of the working condition of the heart

The physical meaning of WI is simple. If WI is positive at an instant, the effects of the waves traveling from the

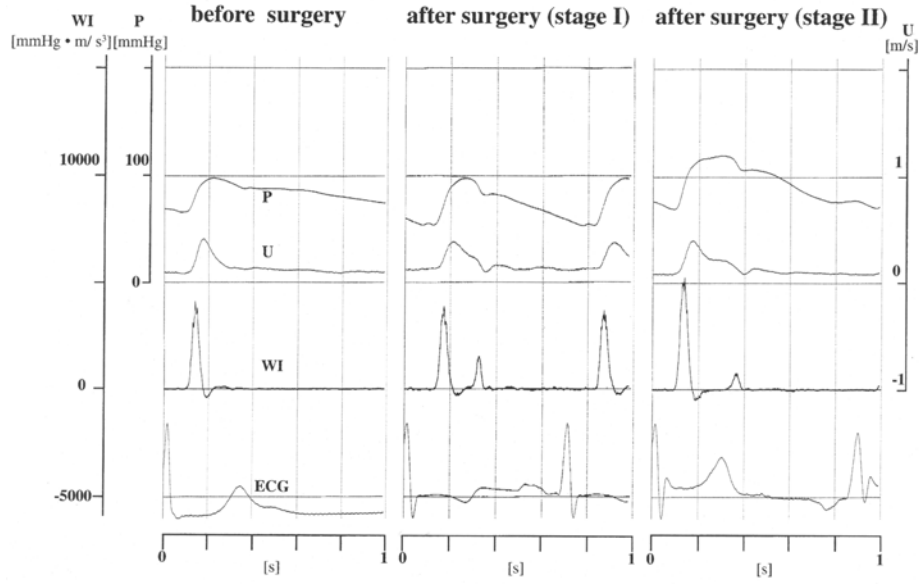

Fig. 3. Recordings of pressure (P), velocity (U), and wave intensity (WI) in the right common carotid artery, and ECGs representative of before and after surgical operation on the mitral valve (case 5). After surgery, the second peak increased markedly, which suggests that the left ventricle now had the ability to generate expansion waves. In the early period after surgery (*stage I*), heart rate was increased from 46 to 85 bpm, and (R–1st peak interval)/(R–R interval) and (1st–2nd interval)/(R–R interval) were increased from 9.4% to 23.0% and from 14.3% to 22.1%, respectively. In the late period after surgery (*stage II*), heart rate was decreased to 68 bpm, (R–1st peak interval)/(R–R interval) decreased to 13.6%, and (1st–2nd interval)/(R–R interval) increased to 25.8%

heart to the periphery on the formation of pressure and velocity waveforms are predominant at that instant. If WI is negative, the effects of the reflected waves from the periphery predominate. It is applicable to any part of the circulation and can be derived easily at any site by simultaneous measurements of pressure and velocity [5].

In a normal subject, WI has two positive peaks (Fig. 1). The first peak appears in early ejection. The second peak appears in late ejection. The nature of these two positive peaks are different. The first peak is associated with acceleration and an increase in aortic pressure; thus, it is a compression wave. The second peak is associated with deceleration and a decrease in pressure, and is therefore an expansion wave. The existence of expansion waves in late ejection was a surprising finding by Parker et al. [1], because it means that the left ventricle actively stops aortic blood flow.

Although the physical meaning of WI is simple, it also has important physiological meanings [3] since it changes with change in the working condition of the heart interacting with the arterial system. Theoretically, the height of the first peak changes with Peak $(dP/dt)^2/\rho c$, and the height of the second peak changes with $\rho c\,(dU/dt)^2$, where Peak dP/dt is the maximum rate of left ventricular pressure increase, dU/dt the maximum aortic deceleration towards the end-ejection, ρ the density of blood, and c the pulse wave velocity in the artery [5] (see also Appendices 1 and 2). Therefore, the first peak reflects cardiac contractility through Peak dP/dt and arterial compliance through c, and the second peak is influenced by arterial compliance through c and the rate of deceleration of aortic blood flow near end ejection through dU/dt. dU/dt towards the end of ejection is related to the momentum of aortic blood flow (inertia force) and myocardial relaxation [13, 14].

The effects of a positive and a negative inotropic agent, which change dP/dt, and those of a peripheral vasodilator and constrictor, which change c, have been investigated in dogs [3]. As expected, the positive inotropic agent caused a dramatic increase in the height of the first peak, and the β-blocking agent reduced it markedly. The vasodilator, which caused a reduction in c, tended to reduce the height of the second peak.

The temporal indices of WI correspond to systolic time intervals: R–1st interval is approximately equal to the preejection period (PEP), and the 1st–2nd interval is approximately equal to left ventricular ejection time (LVET). These temporal indices can be obtained automatically and simultaneously with intensive indices of WI, and reinforce the analysis of the working condition of the heart.

Indices of wave intensity in patients with MR and their changes after repair of MR

In patients with MR, the second peak was reduced significantly. In a normal subject, the second peak appears in late ejection. Expansion wave generation by the normal left ventricle begins when the rate of myocardial shortening falls below the rate at which blood is flowing from the ventricle. At this time, blood still flows out of the ventricle into the aorta under its own momentum, so that the motion of the left ventricular wall no longer matches the movement of the blood out of the left ventricular cavity, which causes a rapid decline in left venticular pressure and generates expansion waves [5, 13–15]. In other words, generation of expansion waves in late ejection is caused by the ability of the left ventricle to stop blood flow by actively terminating myocardial shortening.

However, in MR, the left ventricle continues ejection towards the left atrium even after aortic valve closure, i.e., the cardiac muscle does not stop shortening near end-ejection. Therefore, the left ventricle does not show the ability to stop aortic blood flow actively. We consider that this is the reason why the second peak did not appear or was decreased in patients with MR. After repair of MR, the second peak appeared, which means that the left ventricle stopped ejection actively toward the end of systole. This change in the mode of left ventricular contraction can be observed in the recordings of the M-mode echocardiogram of the left ventricle (Fig. 4). Before surgery, the posterior wall continued thickening beyond the aortic valve closing sound (IIa), i.e., the cardiac muscle continued shortening even after aortic valve closure. After valve replacement, the wall stopped thickening just before IIa, i.e., the cardiac muscle stopped shortening just before aortic valve closure.

The R–1st peak interval and (R–1st peak interval)/(R–R interval) ratio were increased significantly at stage I, which suggests a prolonged PEP. The reason for this is not clear.

The 1st–2nd interval and the (1st–2nd interval)/(R–R interval) ratio were significantly reduced in the MR group before surgery compared with the normal group, which indicates a shortened LVET in the MR group. The shortening of LVET in patients with MR has also been reported in phonocardiographic studies [16, 17].

Heart rate was significantly increased after surgery. This is a phenomenon mentioned in a number of reports which evaluated left ventricular function after mitral valve replacement or valvuloplasty [18–21], although its effects on cardiac function have not been discussed fully.

Effects of the recovery of the second peak

The recovery of the second peak of WI after surgery suggests that the left ventricle had recovered the ability to generate expansion waves. The generation of expansion waves is associated with the rapid decrease in left ventricular pressure near end-ejection, which may be caused by the momentum of flowing blood and which results in rapid late-systolic unloading of the heart [14]. Gillebert et al. [22] showed that late-systolic unloading makes isometric relaxation of the cardiac muscle faster, and makes the subsequent lengthening of the cardiac

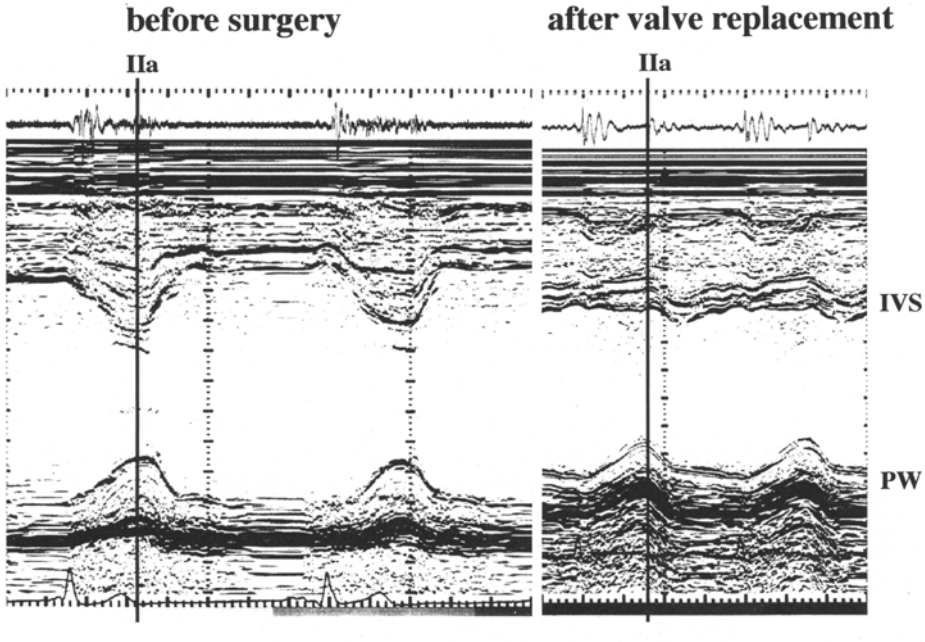

Fig. 4. Recording of M-mode echocardiogram of the left ventricle before and after valve replacement (case 11). Before surgery, the posterior wall (*PW*) continued thickening beyond the aortic valve closing sound (*IIa*), i.e., the cardiac muscle continued shortening even after aortic valve closure. After valve replacement, the wall stopped thickening just before *IIa*, i.e., the cardiac muscle stopped shortening just before aortic valve closure. *IVS*, interventricular septum

muscle more rapid, all of which enhance diastolic function.

Conclusions

In this study, we developed a new method of measuring wave intensity, noninvasively, and applied it to obtain the characteristics of carotid arterial wave intensity in patients with MR and changes in wave intensity before and after the repair of regurgitation. The second peak of wave intensity was significantly smaller in patients with MR. This pattern of wave intensity shows that the left ventricle with MR has lost the ability to actively stop aortic blood flow, since it continues ejection towards the left atrium even after aortic valve closure. After the repair of regurgitation, the second peak recovered, which means that the left ventricle began to clearly exhibit the end of ejection.

Wave intensity has the potential to provide meaningful information about the working condition of the heart interacting with the arterial system.

Appendix 1

The rate of changes in pressure and flow velocity at a fixed point in an artery caused by a forward and a backward (reflected) wave are related by the so-called water-hammer equation, respectively:

$$dP_f/dt = \rho c\, dU_f/dt \quad \text{for a forward wave,} \qquad (1)$$

$$dP_b/dt = -\rho c\, dU_b/dt \quad \text{for a backward wave.} \qquad (2)$$

Here, dP_f/dt and dU_f/dt are the rates of changes in pressure and velocity caused by a forward wave, and dP_b/dt and dU_b/dt are those caused by a backward wave, respectively; ρ is the density of blood, and c is the pulse wave velocity [23].

The measured rate of changes in pressure and velocity, dP/dt and dU/dt, are the sum of the rate of changes caused by a forward and a backward wave:

$$dP/dt = dP_f/dt + dP_b/dt \qquad (3)$$

and

$$dU/dt = dU_f/dt + dU_b/dt. \qquad (4)$$

Using the above four equations, we may write wave intensity (WI) as:

$$\begin{aligned} WI &= (dP/dt)(dU/dt) \\ &= \left[(dP_f/dt)^2 - (dP_b/dt)^2\right]/\rho c \qquad (5) \\ &= \rho c\left[(dU_f/dt)^2 - (dU_b/dt)^2\right]. \qquad (6) \end{aligned}$$

From Eqs. (5) and (6), we can see that if WI > 0, the rates of changes caused by the forward wave, dP_f/dt and dU_f/dt, are greater than those caused by the backward wave, dP_b/dt and dU_b/dt, and if WI < 0, the rates of changes caused by the backward wave are greater than those caused by the forward wave.

During early systole (the first peak of WI) and during late systole (the second peak of WI), WI may be given by $(dP_f/dt)^2/\rho c$ or $\rho c(dU_f/dt)^2$, since forward waves are predominant (see Appendix 2). During early systole, the left ventricle acts predominantly as a pressure generator which controls the maximum rate of rise in left ventricular pressure (the so-called Peak dP/dt). If there is no aortic stenosis, Peak dP/dt is approximately equal to the rate of rise in ascending aortic pressure during early ejection [23]. Therefore, during this period, the left ventricle controls ascending aortic dP/dt ($= dP_f/dt$), so that the first peak of ascending aortic WI, and hence that of carotid arterial WI as well, should be given by $(dP_f/dt)^2/\rho c$. Thus, the first peak of WI changes with Peak dP/dt, which is related to cardiac contractility, and with $1/c$, which is related to arterial compliance.

During late systole, the left ventricle stops blood flow actively, i.e., it acts predominantly as a flow generator which controls dU/dt ($= dU_f/dt$), the rate of deceleration. Therefore, during late systole, WI may be given by $\rho c(dU_f/dt)^2$. Thus the second peak of WI changes with dU/dt and c.

Appendix 2

Since the carotid site is farther from the heart and nearer to the cerebral circulation, there could be larger backward waves there than in the ascending aorta. However, we consider that the characteristics of the first and second peaks of carotid arterial WI are basically the same as those of ascending aortic WI, based on the following reasoning. Reflected waves can be generated only after the arrival of initial forward waves. The first peak of carotid arterial WI appears 30–50 ms after the start of the initial blood flow. Waves can travel only 15–25 cm during this period on the assumption that the pulse wave velocity is about 5 m/s (the pulse wave velocity in the carotid artery may be higher than this). Therefore, we do not consider that the initial waves reflected from the periphery of the cerebral circulation can come back to the carotid artery within the period of the first peak, and have any significant effect on the first peak.

Concerning the second peak, it is true that the backward component of an arterial wave is not small near end-ejection. However, we should be careful about the difference between forward and backward components of a wave and those of WI. In an arterial pressure wave, the backward component near end-ejection is as large

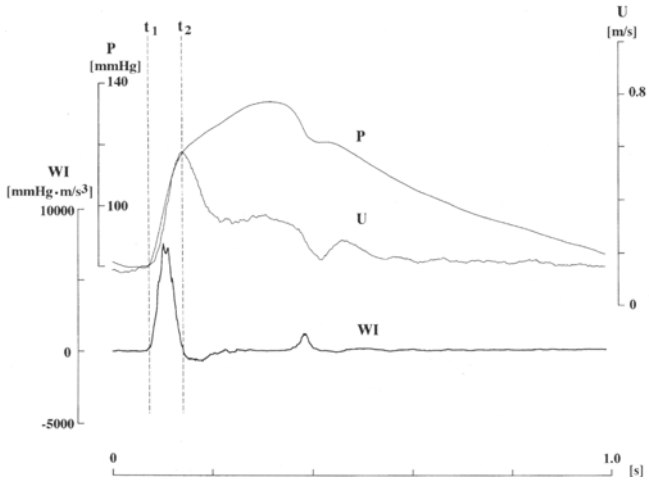

Fig. 5. Superimposition of carotid arterial pressure wave (P) on velocity wave (U) throughout the initial acceleration phase (from t_1 to t_2) which includes the period of the first peak of wave intensity (WI). Similarity between the two waves is very high, which means that the reflected waves have little effect on the formation of pressure and velocity contours during the period of the first peak. The data were obtained from the same subject as in Fig. 1

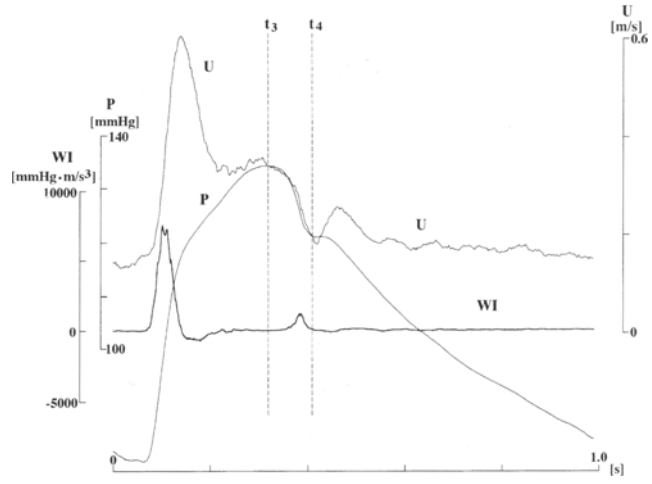

Fig. 6. Superimposition of carotid arterial pressure wave (P) on velocity wave (U) throughout the period from the peak pressure (t_3) to end-ejection (t_4) which includes the period of the second peak of wave intensity (WI). Similarity between the two waves is very high, which means that the reflected waves have little effect on the formation of pressure and velocity contours during the period of the second peak. The data are the same as in Fig. 5

as the forward component. In an arterial velocity wave, the backward component near end-ejection is negative, and its absolute value is as large as the forward component [24]. However, the contours of these backward components near end-ejection are nearly flat, i.e., dP_b/dt and dU_b/dt are nearly zero. Therefore, the backward component of WI, $(dP_b/dt)(dU_b/dt)$, is nearly zero. What this means is that the backward components of pressure and velocity waves do not contribute to the formation of the contours of measured pressure and velocity waves.

To rule out the possibility of the greater effects of the backward waves during the first and second peaks of WI, we checked the similarity of pressure and velocity waves during these periods. If there is no effect of the backward waves (i.e., $dP_b/dt = 0$ and $dU_b/dt = 0$) during the period of concern, Eqs. (1)–(4) yield

$$dP/dt = \rho c\, dU/dt.$$

Since ρc = constant, integration of the above equation gives

$$P(t) - P(0) = \rho c\left[U(t) - U(0)\right],$$

where $P(0)$ and $U(0)$ are the pressure and velocity at the beginning of the period of concern, and $P(t)$ and $U(t)$ are those at any time t during the period of concern. Therefore, the contours of pressure and velocity waves during this period must be similar. The resemblance of pressure and velocity waves decreases with the increase in the effects of the reflected waves [24].

As shown in Figs. 5 an 6, the resemblance of carotid arterial pressure wave to velocity wave during the first and second peaks is very high. Therefore, we can assert that reflected waves have little effect on the formation of pressure and velocity contours during the periods of first and second peaks, i.e., the first and second peaks of carotid arterial WI contain little information on the condition of the periphery which is brought back by reflected waves.

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