

Relationship between Aortic-to-radial Arterial Pressure Gradient after Cardiopulmonary Bypass and Changes in Arterial Elasticity

Masahiro Kanazawa, M.D.,* Haruo Fukuyama, M.D.,† Yoshio Kinefuchi, M.D.,‡ Mamoru Takiguchi, M.D.,§ Toshiyasu Suzuki, M.D.¶

Background: An aortic-to-radial arterial pressure gradient may develop during and after cardiopulmonary bypass (CPB). The mechanisms of this pressure gradient remain controversial. To clarify the cause of the pressure gradient after CPB, the authors investigated the relationship between the pressure gradient and changes in the pulse wave velocity (PWV) before and after CPB.

Methods: The pressure gradient from the aorta to the radial artery and a change in PWV were measured with a wire (0.37 mm in diameter) tipped with a miniature pressure transducer in 12 patients undergoing cardiac surgery. The pressure distributions and waveforms were measured and recorded with electrocardiograph. The PWV was calculated by measuring the propagation time between the R wave of the electrocardiograph and the rising point of the arterial pressure waveform at 10-cm intervals.

Results: After CPB, 7 of 12 patients demonstrated a marked pressure gradient. In these patients, the pressure distribution showed a gradual decrease toward the periphery without a precipitous step-down in pressure at any one specific anatomic location. The PWV decreased as the intraarterial pressure decreased from the aorta to the radial artery, and the relative arterial elasticity decreased linearly toward the periphery.

Conclusions: The results showed that the decrease in PWV implies a decrease in arterial elasticity, and the decrease in the arterial elasticity correlated with the decrease in intraarterial pressure. These findings demonstrated that a radial artery pressure lower than the aortic pressure after CPB may be due to the decrease in arterial elasticity.

THE pressure differences between the aorta and the radial artery are known to sometimes appear after cardiopulmonary bypass (CPB) since Stern *et al.*¹ first reported this phenomenon. Many investigators have reported an aortic- and/or femoral-to-radial artery pressure gradient after CPB. Several of them have suggested a decrease in vascular resistance of the upper extremity or the hand.^{2,3} Maruyama *et al.*⁴ suggested that vasodilators may be responsible for the pressure gradient. In contrast, Baba *et al.*⁵ and Nakayama *et al.*⁶ reported that peripheral vasoconstriction or vasospasm was responsible for the pressure gradient. Although Rich *et al.*⁷ and De Hert *et al.*⁸ demonstrated that appearance of the

pressure gradient was associated with the initiation of CPB, the etiology of the pressure gradient remains controversial. Although off-pump coronary artery bypass grafting has now become a common procedure, it is still considered important to clarify the pressure gradient after CPB. Many previous investigators speculatively discussed the causes of the pressure gradient based on the results in which they measured the pressure gradient between two or three sites (aorta or femoral artery and radial artery) under limited clinical conditions. It is unclear where the pressure gradient takes place in the region ranging from the aorta to the radial artery in their studies. To clarify the cause of the pressure gradient, it is necessary to examine the distribution of blood pressure rather than the blood pressures at the two sites. In this study, we measured the distribution of the blood pressure and distribution of pulse wave velocity (PWV) from the aorta to the radial artery with a micro-pressure transducer. The PWV gradually increased from the aorta to the peripheral artery. The phenomenon wherein the PWV gets faster because of aging is considered to be a sign of arteriosclerosis. Thus, arterial elasticity or stiffness is closely correlated to the PWV. We suspected that changes in arterial elasticity may have played a role in altering the pressure gradient after CPB. It is impossible to measure the actual arterial elasticity (Young's modulus) *in vivo*. However, we can indirectly observe the changes in arterial elasticity by measuring PWV from the aorta to the radial artery. We attempted to determine the relationship between the changes in the arterial wall mechanics, which are closely correlated with the pressure distributions, and the pressure gradient.

Materials and Methods

With approval from the Institutional Review Board of Tokai University School of Medicine (Isehara, Japan), we studied 12 patients who underwent elective cardiac surgery. The summary of the experimental design was explained to patients, and informed consent was received from all patients. All patients were premedicated with morphine (5-10 mg) and scopolamine (0.3 mg) intramuscularly approximately 60 min before the induction of anesthesia. Anesthesia consisted of fentanyl (50-100 $\mu\text{g}/\text{kg}$), diazepam (0.2 mg/kg), and vecuronium bromide for muscle relaxation. Administration of nitroglycerin (0.5-2.0 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and diltiazem (1.0 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was initiated in the patients scheduled for

* Staff Anesthesiologist, † Lecturer, § Professor, Department of Anesthesiology, Tokai University School of Medicine. ‡ Professor, Department of Biomedical Engineering, Tokai University School of High-Technology for Human Welfare.

Received from Tokai University Hospital, Kanagawa, Japan. Submitted for publication March 4, 2002. Accepted for publication February 27, 2003. Support was provided solely from institutional and/or departmental sources.

Address reprint requests to Dr. Kanazawa: Department of Anesthesiology, Tokai University School of Medicine, Bohseidai, Isehara, Kanagawa 259-1193, Japan. Address electronic mail to: masa@webcopy.com. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

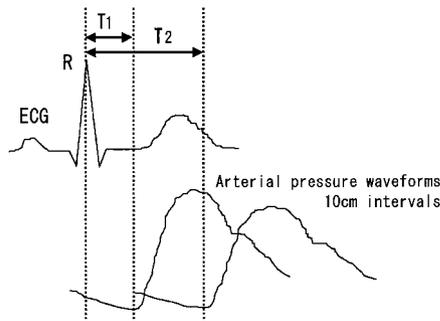


Fig. 1. Pulse wave velocity measurement. A guide wire tipped with a pressure transducer was inserted into the aorta and was then drawn out to the radial artery. Arterial waveforms were recorded with an electrocardiograph (ECG) into a data recorder. The time differences between the R wave of the electrocardiogram and the rising point of the arterial waveforms ($T_2 - T_1$) at the site of 10-cm intervals were measured by an oscilloscope and were converted into the pulse wave velocity.

coronary artery bypass grafting and continued throughout the operation. The CPB flow was regulated between 2.2 and $2.4 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ with nonpulsatile perfusion. Moderate hypothermia (rectal temperature, $28^\circ\text{--}32^\circ\text{C}$) was used. Dopamine and dobutamine ($5\text{--}15 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ each) were administered to maintain cardiac index in all 12 patients after CPB.

A 25-mm-long 22-gauge Teflon catheter (Angiocath; Becton Dickinson Infusion Therapy Systems Inc., Sandy, UT) was placed in the right radial artery prior to anesthesia and was used to monitor the arterial pressure for anesthetic induction. A same sterile 25-mm-long 22-gauge catheter was inserted into the left radial artery, and a 0.37-mm-diameter guide wire tipped with a pressure transducer (Pressure Guide[®]; RADI Medical Systems, Uppsala, Sweden) was advanced through the catheter to a distance of 60 cm, in the aorta, from the insertion point. The guide wire with the transducer was then drawn out gradually, 10 cm each, and the pressure distribution and waveforms from the aorta to the radial artery were measured and recorded with an electrocardiograph. This wire transducer was connected to an interface (PGA-10; RADI Medical Systems). The output signal was then amplified (AP-800P; Nihon Kohden, Tokyo, Japan) and observed on a monitor system (BSM-8800; Nihon Kohden). The transducer was zeroed with the interface prior each measurement. Surgical procedures were suspended during measurement. The measurement was performed twice before the operation and 30 min after CPB with a stable right radial arterial pressure. The findings were stored in a data recorder (A67; Sony Magnescale Inc., Tokyo, Japan). The PWV was calculated by measuring the time between the R wave of the electrocardiogram and the rising point of the arterial pressure wave at intervals of 10 cm, the place of the pressure transducer. An oscilloscope (TDS-420A; Tektronix Inc., Beaverton, OR) was used for all measurements (fig. 1). The pulse wave transmission distance could be

measured as the length of the guide wire in millimeters, and the transmission time was measured by an oscilloscope in $1/1,000 \text{ s}$; as a result, the PWV in this report could be correctly measured.

Because the Moens-Korteweg equation⁹ shows that the arterial elasticity is in proportion to a square of PWV, the relative arterial elasticity was calculated when the aortic elasticity was considered as 1 (Moens-Korteweg equation was explained in the discussion). The volume distensibility (VD) was calculated using the Bramwell-Hill equation¹⁰: $\text{VD} = (3.57/\text{PWV})^2$.

Cardiac output (CO) was measured by the thermodilution method (Vigilance[®]; Baxter Healthcare Corp., Irvine, CA) with a pulmonary artery catheter (744HF75; Swan-Ganz CCombo CCO/SvO₂, Edwards Lifesciences Corp., Irvine, CA) inserted *via* the right internal jugular vein. The cardiac index (CI) was calculated from CO and body surface area (BSA) using the formula CO/BSA . The systemic vascular resistance index (SVRI) was calculated using the standard formula: $\text{SVRI} (\text{dyn} \cdot \text{s} \cdot \text{cm}^{-5} \cdot \text{m}^2) = 80 (\text{MAP} - \text{CVP}/\text{CI})$, where MAP is mean radial artery pressure and CVP is central venous pressure. The hematocrit and rectal temperature were both recorded at the same time that the aortic-to-radial arterial pressure was measured before and after CPB. The data are expressed as mean \pm SD. Intergroup differences for the patient characteristics and intraoperative data (CI, SVRI, hematocrit, rectal temperature) were analyzed with the unpaired *t* test (two-sided test). The paired *t* test was used to analyze intragroup differences. *P* values less than 0.05 were considered to be statistically significant.

Results

All 12 of the adult patients studied were male, and the mean age was 58 yr (range, 38–72 yr). None of the patients had atrial fibrillation or required intraaortic balloon pumping before or after CPB. We considered patients to have a pressure gradient when the systolic arterial pressure was lower in the radial artery than in the aorta, with the differences from 10 mmHg up at 30 min after CPB. As a result, the patients were divided into two groups; seven patients who showed a pressure gradient (group A) and five patients who did not show a gradient (group B). Table 1 shows the characteristics and intraoperative data for the two groups. There were no intergroup differences in patient age, body weight, BSA, or duration of CPB. CI increased significantly in both groups after CPB. However, there was no significant difference between the groups before and after CPB. SVRI and hematocrit decreased significantly in both groups after CPB; there were no significant intergroup differences. Rectal temperature increased after CPB in both groups. There was no difference between the groups before and after CPB.

Table 1. Demographic and Intraoperative Data

	Group A (n = 7)	Group B (n = 5)
Sex (M/F)	7/0	5/0
Age (yr)	59.1 ± 8.5	57.2 ± 14.4
Weight (kg)	65.1 ± 5.9	61.2 ± 6.6
BSA (m ²)	1.74 ± 0.13	1.64 ± 0.13
Operation CABG/valve replacement	5/2	3/2
Duration of CPB (min)	150 ± 65	160 ± 54
CI (l · min ⁻¹ · m ⁻²)		
Before CPB	2.5 ± 0.6	2.6 ± 0.4
After CPB	3.4 ± 0.6*	3.5 ± 0.5*
SVRI (dyn · s · cm ⁻⁵ · m ²)		
Before CPB	2,425 ± 581	2,205 ± 434
After CPB	1,162 ± 205*	1,453 ± 295*
Hematocrit (%)		
Before CPB	34.7 ± 2.0	34.0 ± 1.26
After CPB	26.1 ± 1.9*	25.4 ± 2.6*
Rectal temperature (°C)		
Before CPB	35.9 ± 0.3	35.8 ± 0.3
After CPB	36.4 ± 0.4	36.5 ± 0.2

Values are mean ± SD.

* $P < 0.05$ vs. before cardiopulmonary bypass (CPB).

BSA = body surface area; CABG = coronary artery bypass grafting; CI = cardiac index; Group A = positive-pressure gradient group, group B = negative-pressure gradient group; SVRI = systemic vascular resistance index.

Table 2 shows the systolic aortic-to-radial artery pressure difference (Δ SAP), the mean aortic-to-radial artery pressure difference (Δ MAP), and the diastolic aortic-to-radial artery pressure difference (Δ DAP). The systolic arterial pressure in the radial artery was 9.3 ± 5.3 mmHg higher than that in the aorta before CPB. However, a slight difference was observed between the radial artery and the aorta regarding the mean and diastolic pressures. After CPB, 7 of the 12 patients had a pressure gradient (58%), and a difference of 27 ± 11 mmHg in the systolic pressure existed between the aorta and the radial artery. In the five patients showing no gradient after CPB, the

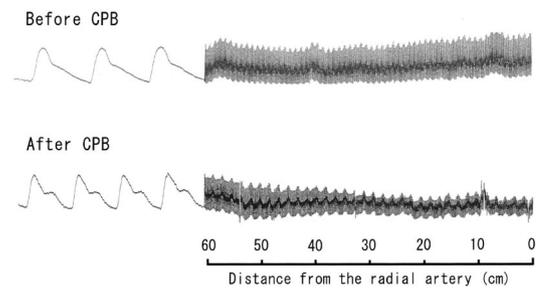


Fig. 2. Arterial pressure distributions (tracings) from the aorta to the radial artery in one patient before (top) and after (bottom) cardiopulmonary bypass (CPB). This patient was a 72-yr-old man who underwent coronary artery bypass grafting. The horizontal scale shows the intraarterial place of the pressure transducer (0 cm = in the radial artery, 60 cm = in the ascending aorta). Before CPB, the systolic intraarterial pressure gradually increased toward the periphery as "peaking" phenomenon, whereas after CPB, the pressure gradient developed and the intraarterial pressure gradually decreased toward the periphery.

pressure differences were nearly equal to pre-CPB results.

Figure 2 (top) shows the pressure distribution from the aorta to the radial artery before CPB, and the systolic pressure rose slowly toward the peripheral artery. Figure 2 (bottom) shows the pressure distribution from the aorta to the radial artery in one patient who had a pressure gradient after CPB. A decrease in the systolic pressure was marked. The radial arterial pressure was lower than both the aortic pressure and the brachial arterial pressure, and no localized pressure depression was observed. The other six patients with a pressure gradient had the same distribution pattern.

Figure 3, A shows the changes in the PWV and VD in 12 patients before CPB. The PWV increased linearly from the aorta to the radial artery, thus resulting in a decrease in the VD toward the radial artery. Figure 3, B shows changes in the PWV and the VD in seven patients who

Table 2. Aortic-to-radial Artery Pressure Gradient before and after Cardiopulmonary Bypass

	Pressure Differences from the Aorta to Each Measurement Point					
	60–50 cm	60–40 cm	60–30 cm	60–20 cm	60–10 cm	60–0 cm
Before CPB (n = 12)						
Δ SAP	-1.0 ± 1.0	-3.2 ± 2.2	-5.1 ± 2.5	-7.4 ± 4.0	-8.9 ± 4.4	-9.3 ± 5.3
Δ MAP	-0.2 ± 0.8	-0.3 ± 1.4	-0.2 ± 2.4	0.2 ± 2.5	0.1 ± 2.7	0.1 ± 3.5
Δ DAP	0.2 ± 0.6	0.2 ± 1.5	-0.1 ± 1.4	0.1 ± 1.9	0.8 ± 1.6	0.8 ± 1.4
After CPB						
Group A (n = 7)						
Δ SAP	0.9 ± 1.1	3.1 ± 2.0	8.4 ± 2.0	15.3 ± 3.0	19.3 ± 2.3	26.7 ± 10.5
Δ MAP	0.4 ± 0.5	2.7 ± 1.5	5.0 ± 2.5	7.7 ± 6.4	10.7 ± 4.0	14.3 ± 6.6
Δ DAP	0.3 ± 0.5	1.4 ± 1.9	2.0 ± 1.6	2.7 ± 2.0	4.0 ± 2.9	5.4 ± 5.6
Group B (n = 5)						
Δ SAP	-0.6 ± 1.9	-2.0 ± 2.5	-3.6 ± 4.2	-4.8 ± 4.9	-6.4 ± 6.7	-9.4 ± 8.6
Δ MAP	-0.4 ± 2.3	-1.4 ± 2.5	-2.4 ± 4.0	-1.6 ± 2.7	-1.6 ± 2.7	-2.0 ± 3.4
Δ DAP	-0.4 ± 2.3	-1.0 ± 2.0	-0.6 ± 2.5	-0.8 ± 2.5	-1.2 ± 1.3	-0.4 ± 1.5

Values are mean ± SD. All pressures shown in mmHg. "60 cm" shows that the pressure transducer is approximately in the aorta, and "0 cm" shows that it is in the radial artery; accordingly, "60–0 cm" means the aortic-to-radial artery pressure difference.

CPB = cardiopulmonary bypass; Δ DAP = difference in diastolic arterial blood pressure; Group A = positive-pressure gradient group; Group B = negative-pressure gradient group; Δ MAP = difference in mean arterial blood pressure; Δ SAP = difference in systolic arterial blood pressure.

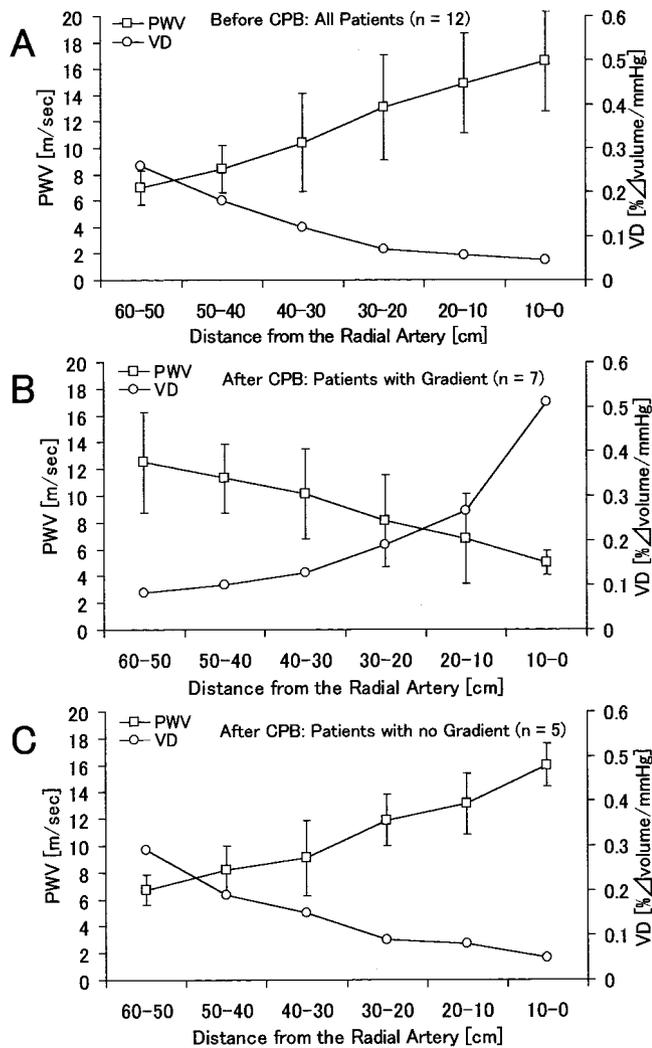


Fig. 3. Changes in the pulse wave velocity and the volume distensibility in all 12 patients before cardiopulmonary bypass (CPB) (A) and in patients (n = 7) showing pressure gradient after CPB (B) and in patients (n = 5) showing no pressure gradient after CPB (C). The pulse wave velocity (PWV; squares) was measured as figure 1. Values are mean ± SD. The volume distensibility (VD; circles) was calculated from the mean values of the pulse wave velocity in each of the measuring points and Bramwell-Hill equation (see text for details). The x axis shows the distance from the insertion point of a 25-mm-long 22-gauge catheter for the transducer wire (e.g., 0 cm = in the radial artery, 60 cm = in the ascending aorta).

had a pressure gradient after CPB. The PWV gradually decreased toward the periphery, thus resulting in an increased VD. In five patients who had no pressure gradient after CPB, the PWV gradually increased toward the periphery (fig. 3, C).

The distribution in the relative arterial elasticity that was calculated from the mean values of the PWV in figure 3, B is illustrated in figure 4, A. Because the PWV was squared and plotted against the elasticity, the relative arterial elasticity in seven patients with the pressure gradient decreased rapidly and linearly toward the periphery. In addition, the differences in the systolic and mean arterial pressure (ΔSAP, ΔMAP) between the aorta

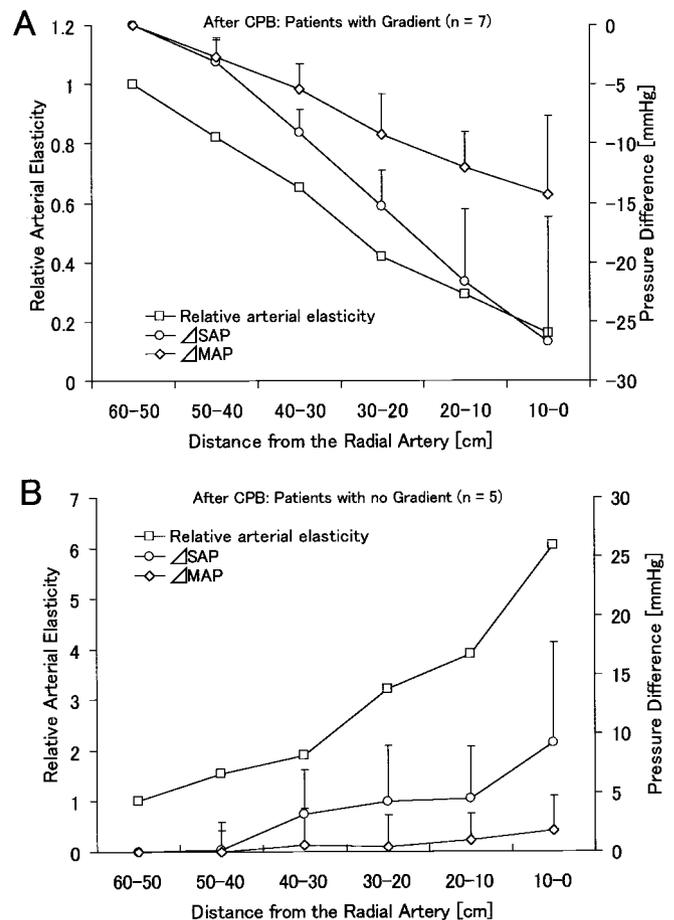


Fig. 4. Changes in the relative arterial elasticity and pressure differences from the aorta to each of the measurement points after cardiopulmonary bypass (CPB) in the positive-pressure gradient group (A) and the negative-pressure gradient group (B). Squares show the changes in arterial elasticity when aortic elasticity is 1. Plotted values were calculated from the mean values of the pulse wave velocity in figure 3, B or C, and Moens-Korteweg equation. Circles show the changes in pressure differences from the aorta to each measurement point (10-cm interval). ΔSAP = difference in systolic arterial blood pressure; ΔMAP = difference in mean arterial blood pressure.

and each of the measurement points are plotted in figure 4, A. There was a remarkable decrease in the pressure between the aorta and the radial artery, and these changes were observed to correspond to the changes in relative arterial elasticity.

In the same manner as that demonstrated in figure 4, A, the relative arterial elasticity and the difference in ΔSAP and ΔMAP in the no-pressure-gradient group are illustrated in figure 4, B. The relative arterial elasticity and ΔSAP increased from a central to a peripheral site.

Discussion

Previous investigators dealt with the region between the aorta and the radial artery as an unknown circuit when assuming the aortic pressure to be input and the radial pressure to be output and described reasons why

the mechanism of the unknown circuit changes. The inside of the unknown circuit and the direct mechanism that leads to the pressure gradient were not clearly explained. To clarify this mechanism, it is essential to investigate what actually occurs in the region from the aorta to the radial artery. We examined a continuous distribution of the intraarterial pressure from the aorta to the radial artery before and after CPB in patients with and without a pressure gradient.

It is generally known that the radial systolic arterial pressure is higher than the aortic systolic pressure in awake humans. We had similar findings in 12 patients anesthetized with fentanyl before undergoing CPB, and the measured pressure distribution indicated that the intraaortic pressure gradually increased from the aorta to the radial artery as a peaking phenomenon. In addition, the PWV gradually increased toward the periphery in all 12 patients before CPB. Physiologically, the central arteries are rich in elastic fibers, and the peripheral arteries, which are rich in smooth muscle cells, tend to have a muscular type artery. As a result, the arteries gradually get "firmer" toward the peripheral sides. The PWV gets faster on the peripheral sides, and this reflects the physiologic characteristics of the arteries.

In seven patients with a pressure gradient after CPB, we found that the intraaortic pressure gradually decreased from the aorta to the radial artery. Furthermore, it was noteworthy that the pressure gradient was not localized and the PWV gradually decreased from the aorta to the radial artery. The PWV in the elastic vessels (C_0) is expressed by Moens-Korteweg's equation as $C_0 = (E h / \rho D)^{1/2}$, where E is the elasticity of the vessel wall (the Young modulus), h is the wall thickness of the elastic vessel, ρ is the density of fluid, and D is the diameter of the vessel. According to this equation, a decrease in C_0 toward the peripheral artery can be explained as follows. The effects of h/D on changes in C_0 are considered to be low because the h/D did not significantly change from the brachial artery to the radial artery.^{11,12} Even if changes in h/D are present, its effects on C_0 must be inconsiderable in the physiologic range. ρ is not affected, either. Changes in C_0 are therefore regulated by the Young modulus, namely, changes in the elasticity of the vessel wall. It is considered that the gradual decrease in the PWV from the aorta to the radial artery in the patients with the pressure gradient demonstrates the gradual decrease in the arterial elasticity from the aorta to the radial artery.

Table 2 demonstrates that a pressure gradient of 10 mmHg or more between the aorta and the radial artery was observed in seven patients after CPB. Assuming that the pressure difference between the above two sites appears as the product of a flow rate and arterial resistance, an increase either in the flow rate or in the arterial resistance is thus needed for the occurrence of a pressure gradient of 10 mmHg or more after CPB. Al-

though there was no pressure gradient in the mean arterial pressure before CPB, it was nevertheless elevated to 14 ± 7 mmHg in 7 of the 12 patients after CPB. Under physiologic circulation, it is unreasonable to assume that an increase in the blood flow (or flow rate) can amplify the pressure gradient from approximately 0 mmHg to approximately 10 mmHg after CPB. Baba *et al.*⁵ reported that the diameter of the radial artery decreased significantly after CPB, but there was no significant increase in the radial arterial flow (velocity) that can explain this pressure gradient. It is possible that such a decreased diameter in the radial artery increases the peripheral arterial resistance. According to the law of Poiseuille, the resistance of the tube is proportional to η/r^4 , where η is the viscosity and r is the radius of the tube. The blood viscosity decreases by hemodilution on CPB. If a pressure gradient of 10 mmHg or more had been present with an associated decrease in the blood viscosity to 70% (0.7η) after the start of CPB, the intraarterial diameter should become one half ($0.5 r$) or less. However, in the same report,⁷ we could not find a reduction of the intraarterial diameter to this degree.

The pulsatile flow passing through a small segment of the blood vessel causes an increase in the intraarterial volume, and then the intraarterial pressure (Δp) increases. Because the stress in the vessel wall is equilibrated to Δp , the following equation should be established¹⁰: $\Delta p = \Delta v \cdot E \cdot h / (2\pi \cdot r^3)$, where Δv is an increase in volume due to a pulse, E is the elasticity of the vessel, h is the wall thickness of the vessel, and r is the radius of the vessel. This equation indicates that changes in the intraarterial pressure provoked by the pulsatile flow depend on the elasticity of the vessel wall. The relationship between the pulsatile flow in elastic vessels and the change in the pressure is explained by the following equation^{13,14}: $\Delta P = \rho \cdot u \cdot C_0$, where ΔP is the change in the pressure, ρ is the blood density, u is the flow rate, and C_0 is the PWV. The changes in intraarterial pressure evidence the changes in E because C_0 is expressed as $(E h / \rho D)^{1/2}$. It is considered that a gradual decrease in the intraarterial pressure, which is regarded as the pressure gradient after CPB, reflects a decrease in the arterial elasticity from the aorta to the radial artery.

However, in the current study, we could not clarify the etiology of a decrease in arterial elasticity. Some investigators^{7,8} have demonstrated that the pressure gradient developed early during CPB. The initiation of CPB is thus associated with hemodilution. There seems no doubt that hemodilution reduces the peripheral resistance with a decrease in blood viscosity. It might be expected that the reduced resistance in the peripheral resistant arteries could decrease the radial arterial pressure. Furthermore, it is possible that a decrease in the intraarterial diameter in the region between the axillary artery and the radial artery, which is due to the decreased radial arterial pressure, results in a decrease in the elasticity of the

vessel walls. Pauca *et al.*^{2,3} demonstrated that a decrease in hand vascular resistance was responsible for the pressure gradient. The decrease in the resistance in the peripheral resistant arteries may become a trigger of the pressure gradient. Their findings also support our theory. Goda *et al.*¹⁵ demonstrated that hemodilution plays an important role in the femoral-to-radial artery pressure gradient after CPB. On the other hand, De Hert *et al.*⁸ found no relationship between the magnitude of the pressure gradient and changes in hematocrit associated with hemodilution.

Baba *et al.*⁵ reported that the radial artery diameter significantly decreased after the initiation of CPB and suggested that radial artery constriction could be responsible for the pressure gradient. Gravlee *et al.*¹⁶ suggested that a decrease in muscular arterial diameter or vasospasm related to catecholamine may contribute to the gradient. We agree with the reduction in the radial artery diameter and consider that such a reduction extends from the axillary artery to the radial artery. However, we believe that the decrease in the arterial diameter does not mean a vasospasm or vasoconstriction due to catecholamine release. The phenomenon seems to be due to hydrostatic changes.

In this study, we found that the intraarterial pressure and the PWV decreased gradually from the aorta to the radial artery in patients with a pressure gradient after CPB. Furthermore, we demonstrated that the decrease in arterial elasticity toward the radial artery may be responsible for the occurrence of a pressure gradient occurring after CPB. However, physical and fluid mechanical experiments are necessary to establish our opinions regarding the relationship between the change in elasticity and internal pressure.

References

1. Stern DH, Gerson JJ, Allen FB, Parker FB: Can we trust the direct radial artery pressure immediately following cardiopulmonary bypass? *ANESTHESIOLOGY* 1985; 62:557-61
2. Pauca AL, Hudspeth AS, Wallenhaupt SL, Tucker WY, Kon ND, Mills SA, Cordell AR: Radial artery-to-aorta pressure difference after discontinuation of cardiopulmonary bypass. *ANESTHESIOLOGY* 1989; 70:935-41
3. Pauca AL, Wallenhaupt SL, Kon ND: Reliability of the radial arterial pressure during anesthesia: Is wrist compression a possible diagnostic test? *Chest* 1994; 105:69-75
4. Maruyama K, Horiguchi R, Hashimoto H, Ohi Y, Okuda M, Kurioka T, Konishi K, Muneyuki M, Kusagawa M: Effect of combined infusion of nitroglycerin and nicardipine on femoral-to-radial artery pressure gradient after cardiopulmonary bypass. *Anesth Analg* 1990; 70:428-32
5. Baba T, Goto T, Yoshitake A, Shibata Y: Radial artery diameter decreases with increased femoral to radial artery pressure gradient during cardiopulmonary bypass. *Anesth Analg* 1997; 85:252-8
6. Nakayama R, Goto T, Kukita I, Sakata R: Sustained effects of plasma norepinephrine levels on femoral-radial pressure gradient after cardiopulmonary bypass. *J Anesth* 1993; 7:8-15
7. Rich GF, Lubanski RE, McLoughlin TM: Differences between aortic and radial artery pressure associated with cardiopulmonary bypass. *ANESTHESIOLOGY* 1992; 77:63-6
8. De Hert SG, Vermeyen KM, Moens MM, Hoffmann VL, Bataillie KJ: Central-to-peripheral arterial pressure gradient during cardiopulmonary bypass: Relation to pre- and intra-operative data and effects of vasoactive agents. *Acta Anaesth Scand* 1994; 38:479-85
9. Hamilton WF, Remington JW, Dow P: The determination of the propagation velocity of the arterial pulse wave. *Am J Physiol* 1945; 144:521-35
10. Bramwell JC, Hill AV: The velocity of the pulse wave in man. *Proc Royal Soc Lond (Biol)* 1922; 93:298-306
11. Learoyd BM, Taylor MG: Alterations with age in the viscoelastic properties of human arterial walls. *Cir Res* 1966; 18:278-92
12. Trager S, Pignataro M, Anderson J, Kleinert JM: Color flow Doppler: Imaging the upper extremity. *J Hand Surg* 1993; 18:621-5
13. Milnor WR: Properties of the vascular wall, Hemodynamics, 2nd edition. Edited by Collins N. Baltimore, Williams & Wilkins, 1982, pp 95-101
14. Binder RC: Unsteady flow-galloping, water hammer, Fluid Mechanics, 5th edition. Edited by Binder RC. Englewood Cliffs, Prentice-Hall, 1973, pp 318-9
15. Goda Y, Takita K, Gando S, Kawahigashi H, Sakamoto H, Ito Y, Kemmotsu O: Hemodilution has an important role in femoral-to-radial artery pressure gradient after cardiopulmonary bypass. *Circ Cont* 1995; 16:223-8
16. Gravlee GP, Brauer SD, O'Rourke MF, Avolio AP: A comparison of brachial, femoral, and aortic intra-arterial pressure before and after cardiopulmonary bypass. *Anesth Intensive Care* 1989; 17:305-11