

Estimation of Aortic-to-Radial Artery Distribution of Arterial Wall Elasticity

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Objects. The purpose of this paper is to clarify the cause of the peaking/steepening phenomenon and pressure gradient after cardiopulmonary bypass by measuring the distribution of intra-arterial pressures and that of pulse wave velocities and estimating the vascular wall elasticity's from the aorta to radial artery. **Methods.** We measured the intra-arterial pressures and pulse wave velocities simultaneously from aorta to radial artery using a guide-wire tipped with a pressure transducer before and after cardio-pulmonary bypass (CPB), and calculated the distribution of the stiffness parameters (called β) of aorta-to-radial artery wall. Thereafter, we converted β 's into the wall elasticity's. **Results.** We found that before CPB the intra-vascular pressures increased gradually from aorta to radial artery, and that corresponding β and wall elasticity increased simultaneously. After CPB, the intra-arterial pressure and β or wall elasticity showed the gradual decrease from aorta to radial artery. These results suggest that aortic-to-radial artery pressure change is attributed to gradual change in β , that is, in the wall elasticity. **Conclusions.** The findings indicate that the gradual rise and fall in β , namely, the increase and decrease in vascular elasticity may account for the peaking, steepening and pressure gradient.

Keywords: cardio-pulmonary bypass, pressure gradient, wall elasticity, stiffness parameter

INTRODUCTION

It is generally recognized as a peaking phenomenon that, in healthy humans, the systolic pressure increases progressively from the aorta to the radial artery. It is also known that the elasticity of the arterial wall gradually increases from the aorta toward the radial artery [1, 3]. On the other hand, after a cardiopulmonary bypass (CPB), this phenomenon has been reported to become reversed, that is, the systolic pressure decreases progressively from the aorta toward the radial artery (aortic-to-radial artery pressure gradient) [2]. We have documented that, with a post-CPB pressure gradient, the elasticity of the arterial wall diminishes progressively from the aorta toward the radial artery, and demonstrated a close relation of the distribution of intravascular pressure and that of vascular wall elasticity in the direction of vascular longitudinal axis. In brief, the pre-CPB gradual increase and post-CPB gradual decrease in intravascular pressure from the aorta toward the radial artery are accompanied with a gradual increase and a gradual decrease in vascular wall elasticity, respectively, hence suggesting that the latter may constitute a primary cause of the former [3].

The elasticity of the arterial wall increases non-linearly with increasing intravascular pressure [4]. The vascular wall is comprised of three layers of tissue, i.e., the intima, media and adventitia. These tunicae differ in elasticity from each other, and mainly the adventitia strongly resists vascular extension as intravascular pressure is elevated. It also has been reported that,

when the extensibility of the vascular wall is expressed in terms of a degree of increase in vascular radius, the intravascular pressure increases exponentially together with increasing vascular radius within the physiological limits [5]. These would suggest that the distribution of vascular wall elasticity in the radial direction also may have bearing upon changes in intravascular pressure.

Thus, measurement or estimation of the vascular wall elasticity of various parts of the blood vessels from the aorta to the radial artery is considered highly important in clarifying the phenomena of peaking and pressure gradient. This paper deals with the method of estimating the vascular wall elasticity of various parts of the blood vessels and with the mechanism operating for the phenomena of peaking and pressure gradient, based on the data obtained by direct and continuous measurements of the distribution of intraluminal pressure and that of pulse wave velocity from the aorta to the radial artery.

THEORETICAL BACKGROUND

As regards the relationship between the stress and strain of the blood vessel, it is generally recognized as described above that the blood vessel (wall) exhibits the characteristic of being gradually less extensible with increasing intravascular pressure. It was demonstrated experimentally by Hayashi, et al., that this relationship can be expressed by a simple exponential function insofar as the changes in intravascular pressure are within the limits of clinical settings [6]. The following

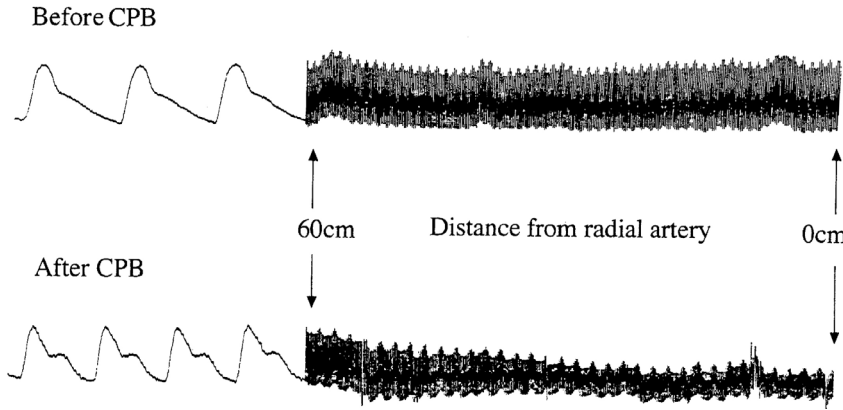


Fig. 1 A typical aortic-to-radial artery intravascular pressure distribution The chart plots blood pressure as the ordinate and aortic-to-radial artery distance as the abscissa. The upper panel represents a pre-CPB tracing and the lower panel represents a post-CPB tracing from the same patient. The chart clearly depicts a reversal of the intravascular pressure distribution between pre- and post-CPB tracings. See the text for details..

equation holds for the relation between the intravascular pressure change and the vascular outer diameter change:

$$\ln(P/P_0) = \beta / \{(R-R_0)/R_0\} = \beta \cdot \Delta R/R_0 \dots\dots\dots (1)$$

where P_0 represents the reference intravascular pressure, e.g., diastolic pressure, and R_0 is the vascular outer diameter. This equation means that the logarithm of intravascular pressure change is proportional to the rate of change in vascular diameter, and the proportionality constant thereof is expressed as β . Hayashi, et al. have designated β a stiffness parameter that depicts the elasticity of vascular wall.

If displacement in the direction of vascular longitudinal axis is disregarded, $\Delta V/V_0 = 2\Delta R/R_0$ can hold, so that Eq. (1) can be shown as:

$$\ln(P/P_0) = (1/2) \cdot \beta \cdot \Delta V/V_0 \dots\dots\dots (2)$$

Then, the pulse wave velocity (PWV) can be expressed as (see Note below):

$$PWV = \{V_0 / (\rho \cdot \Delta V / \Delta p)\}^{1/2} \dots\dots\dots (3)$$

From Eqs. (1) to (3) the following equation is obtained:

$$\beta = (2\rho / \Delta p) \cdot \ln(P/P_0) \cdot (PWV)^2 \dots\dots\dots (4)$$

Where ρ represents the density of blood, and $\Delta p = P - P_0$.

If the internal pressure of any region of the vascular system, e.g., systolic pressure P_s and diastolic pressure P_d , and the PWV in that region can be measured, β for that region can be calculated from Eq. (4).

Also, from β and Eq. (1):

$$\Delta R/R_0 = (1/\beta) \cdot \ln(P/P_0) \dots\dots\dots (5)$$

holds to find the rate of change in vascular radius.

METHODS AND SUBJECTS

Measurement of intravascular pressure and pulse wave velocity distribution

As mentioned above, we have reported the method of determining the aortic-to-radial artery intravascular pressure and PWV and the data obtained thereby [3]. The outline of the report is as follows. With approval from the Institution Review Board of Tokai University School of Medicine and with informed consents from all patients, we studied twelve patients for cardiopulmonary bypass (CPB). A wire tip pressure transducer of 0.37 mm in outer diameter was introduced into the radial artery and advanced about 60 cm to the aorta, and intravascular pressure waveforms were measured at 10-cm intervals while drawing back the wire tip pressure transducer. Simultaneously, the difference

between the R-wave and systolic uprising point on the ECG was determined to calculate the PWV during that interval. The measurements were carried out before and after the start of CPB in the same patient.

Calculation of β

With the method described above, intravascular pressures and PWVs for the consecutive five or six 10-cm segments from the aorta to radial artery were obtained directly.

If the reference intravascular pressure is taken as represented by diastolic pressure and the intravascular pressure as represented by systolic pressure in Eq. (4), then $P_0 = P_d$, $P = P_s$, and $\Delta p = P_s - P_d$. β is calculated for the given segment by substituting these values in Eq. (4).

Relation between intravascular pressure and rate of increase in vascular radius

The values of β found in the foregoing section are substituted in Eq. (5) and the typical representative value of vascular radius from the literature is substituted for R_0 to give the rate of change in vascular radius, $\Delta R/R_0$, corresponding to the change in intravascular pressure from P_d to P_s .

RESULTS

Measurement of intravascular pressure and pulse wave velocity distribution

Figures 1 and 2 depict an example of the changes in aortic-to-radial artery intravascular pressure and PWV changes before and after CPB, as previously reported [3]. The abscissa represents the position in the vascular lumen ranging from the site of catheter insertion in the radial artery at the wrist, being 0 cm, to a maximum of 60 cm toward the aorta. Blood pressures and PWVs are plotted on the ordinate. Prior to the start of CPB, a gradual increase in systolic pressure, i.e., a peaking was evident from the aortic toward the radial artery; whilst after CPB, a pressure gradient characterized conversely by a gradual decrease in systolic pressure was noted. In parallel with these phenomena, there was a progressive rise in PWV from the aorta to the radial artery before CPB and conversely a gradual decline in PWV after CPB.

Calculation of β

For each of the consecutive 10-cm vascular segments, the β value was calculated by substitution of respective measured values in Eq. (4). Pertinent pre- and post-CPB data are presented in Figure 3. As in the

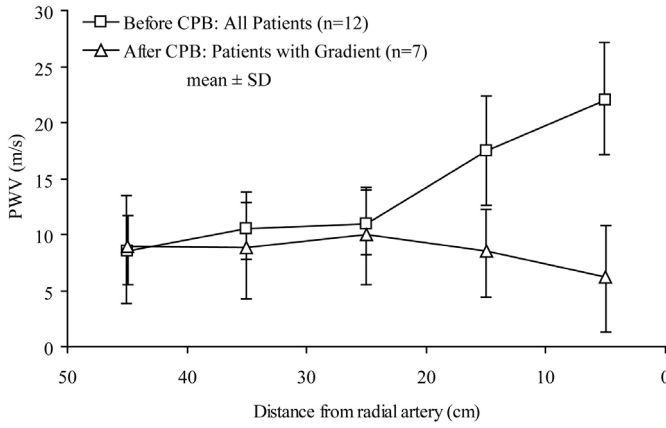


Fig. 2 An aortic-to-radial artery pulse wave velocity distribution The PWV at consecutive 10-cm intervals of vascular length is plotted as ordinate and the aortic-to-radial artery distance as abscissa. £ is a pre-CPB tracing and r represents a post-CPB tracing. The pre-CPB PWV increases progressively with increasing distance from the aorta to the distal side whereas the post-CPB PWV decreases progressively toward the periphery.

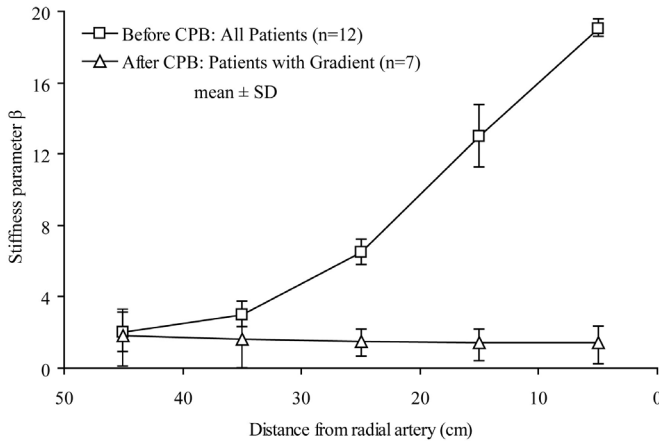


Fig. 3 Distribution of stiffness parameter β The β value at consecutive 10-cm intervals of vascular length is plotted as ordinate and the aortic-to-radial artery distance as abscissa. £ is a pre-CPB tracing and r represents a post-CPB tracing. With increasing distance from the aorta to the distal side, the β showed a rapid increase before CPB, but gently declined after CPB.

section above, the abscissa represents the position in the vascular lumen ranging from the catheter insertion site in the radial artery, being 0 cm, to a maximum of 50 cm toward the aorta. The ordinate indicates the β value. Meanwhile, $\rho = 1.055$.

As seen in this chart, β showed a progressive increase from the aorta to the radial artery before CPB and gently declined toward the radial artery after CPB. As is clear from the definition of β , the increase in β means an increase in the rate of increase in vascular wall elasticity whereas the decrease in β denotes that the rate of increase in elasticity diminishes.

Relation between intravascular pressure and rate of increase in vascular radius

The $\Delta R/R_0$ for each vascular segment was calculated by substituting the β determined in the section above in Eq. (5). Pertinent pre- and post-CPB data are presented in Figure 4, where the abscissa represents the intravascular pressure and the ordinate indicates the $\Delta R/R_0$.

As can be seen from the figure, $\Delta R/R_0$ decreases gradually with increasing intravascular pressure when assessed for the same segment. In other words, this indicates that the greater the intraluminal pressure, the harder the vascular wall becomes. It is evident from the chart that the rate of increase in $\Delta R/R_0$ (slope) as against intravascular pressure decreased gradually from the aorta toward the radial artery before CPB and, conversely, increased after CPB.

DISCUSSION

The aorta is highly extensible and plays a key role in functioning smooth blood circulation in normal adults. The vasculature on the radial artery side, on the other hand, is hard and has an important role in maintaining peripheral blood pressure. Most of those reports documenting these roles are demonstrated by measurements in isolated vascular specimens [8-10]. Similar findings have also been inferred on the basis of data on vascular PWV distribution [8,11-13]. Our previous report documented that the elasticity of the arterial wall progressively decreases from the aorta to the radial artery in association with a post-CPB pressure gradient, and showed a close relation between the intravascular pressure distribution and the vascular wall elasticity distribution in the direction of vascular longitudinal axis from the aorta to the radial artery [3]. In the present study, we calculated the stiffness parameter, β , from the distribution of aortic-to-radial artery intravascular pressure and that of PWV determined *in situ*, with the results showing a reversal of the distributions following CPB. Relations of these results with waveform changes, *viz.*, peaking, steepening and post-CPB pressure gradient, may be considered as follows.

As evident from the definition, β is a parameter that expresses the non-linearity of vascular elasticity in the radial direction. This non-linearity sharpens the systolic moiety of the pulse wave and diminishes the diastolic moiety in contrast. Prior to CPB, β increases gradually from the aorta toward the radial artery as

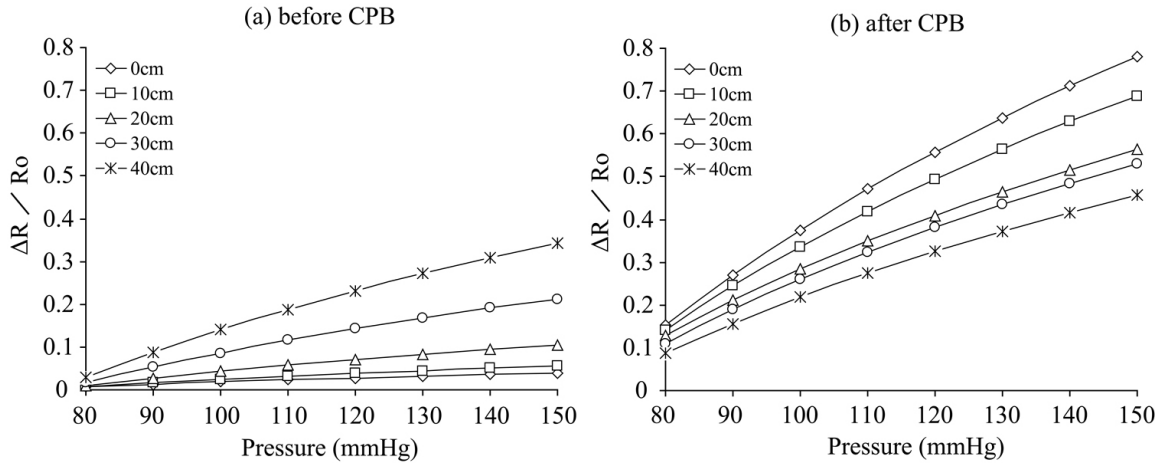


Fig. 4 Blood pressure-dependency of the vascular radius increase rate, $\Delta R/R_0$ The chart plots $\Delta R/R_0$ as the ordinate and blood pressure as the abscissa. The parameter represents various parts of the aortic-to-radial artery (at consecutive 10-cm intervals from the origin of the radial artery being taken as 0 cm). Fig. (a) displays pre-CPB data, indicating that β decreases and the degree of the decrease also diminishes progressively, namely the blood vessel becomes progressively harder, with increasing blood pressure and distance toward the periphery. Fig. (b) exhibits post-CPB data, showing a reversal of the changes in β seen in Fig. (a).

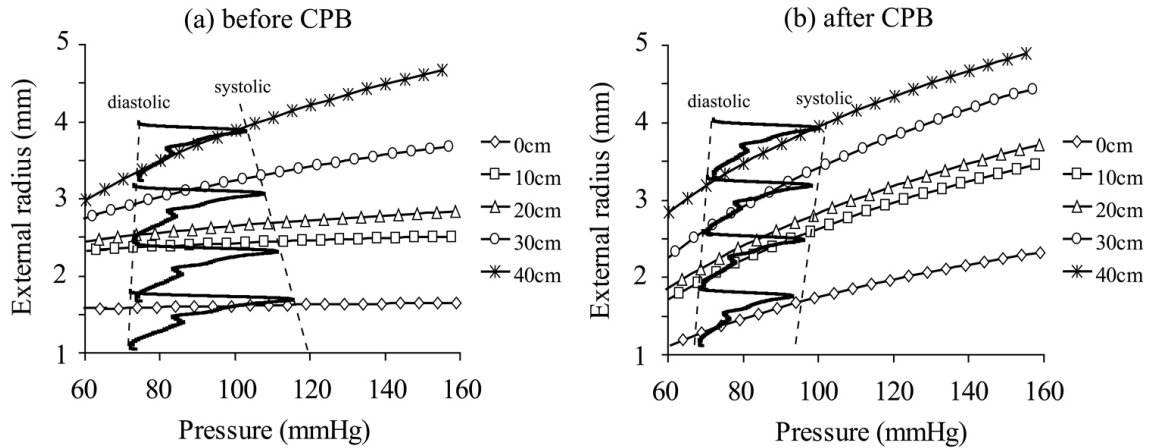


Fig. 5 Blood pressure-dependency of the vascular radius and blood pressure changes The outer diameter of the blood vessel is plotted as ordinate and the blood pressure as abscissa. The chart depicts the vascular outer diameter against intravascular pressure in various parts of the aortic-to-radial artery (at consecutive 10-cm intervals from the origin of the radial artery being taken as 0 cm). The data implies that the greater the gradient to intravascular pressure, the more liable the blood vessel is to expand (softer), and Fig. (a) displays pre-CPB data and Fig. (b) shows post-CPB data. Note that, with increasing distance from the aorta, the radial artery is progressively liable to expand (softer) after CPB. Changes in amplitude observed following the impression of a blood pressure waveform of a certain magnitude from the aortic side are shown with a broken line. See the text for details.

illustrated in Figure 3 (before CPB in the figure), and the above-described sharpening of the systolic component continuously accumulates toward the radial artery side. The pulse wave eventually becomes progressively sharpened to grow in wave height, as seen in Figure 1. After the completion of CPB, on the other hand, β gradually decreases toward the radial artery side as shown in Figure 3 (after CPB in the figure). This implies that the sharpening trend of the systolic component gradually disappears, so that the waveform gradually diminishes toward the radial artery side. Consequently, as seen in Figure 1, the sharpening diminishes progressively and the wave height decreases as the pulse wave advances toward the radial artery side.

When the vascular radius data from the literature

[7] is substituted in Figure 4 or Eq. (5) to depict the relation between vascular radius and intravascular pressure, Figure 5 is obtained. Prior to CPB, the more distal from the aorta, the greater the progressive diminution of the vascular radius increase rate was with increasing intravascular pressure of the radial artery. In other words, the more distal from the aorta, the harder the radial artery wall became. After CPB, in contrast, the more distal from the aorta, the greater the radius increase rate was of the radial artery, i. e., the softer the radial artery wall became.

We think that these findings play an important role in the study of hemodynamics and especially in anesthesia management. Meanwhile, this paper does not refer to the cause of the phenomenon that peaking and steepening vanish and a pressure gradient arises

Table 1 Patient Characteristics After CPB, 7 of the 12 patients had a pressure gradient. BSA=body surface area; CABG=coronary artery bypass grafting; AVR= aortic valve replacement; MVR= mitral valve replacement; CI=cardiac index.

	Patient Characteristics
Number of patients	12
Male/Female	12/0
Age (yrs)	58±10
Height (cm)	167±9
Weight (kg)	62±8
BSA (m ²)	1.70±0.13
Operation	CABG: 8 AVR: 1 MVR: 3
Duration of CPB (min)	160±60
Hematocrit (%)	Before CPB: 34.7±0.8 After CPB: 26.1±1.9*(n=7)
CI (l/min/m ²)	Before CPB: 2.6±0.8 After CPB: 3.4±0.8*

mean ± SD

*P<0.05 versus Before CPB

following CPB. We consider, as previously reported, that hemodilution in CPB may serve as a trigger to this phenomenon [3].

CONCLUSIONS

The pre- and post-CPB stiffness parameter, β , and vascular elasticity distribution were calculated from invasive measurements of the aortic-to-radial artery distribution of intravascular pressure and pulse wave velocity. As a result, β was found to increase gradually from the aorta toward the radial artery prior to CPB and the direction of change in β became reversed, viz. this parameter showed a gradual decrease toward the radial artery, after CPB. The findings indicate that the gradual rise and fall in β , namely, the increase and decrease in vascular elasticity may, account for the peaking, steepening and pressure gradient. This communication does not deal with the mechanism operating for the phenomenon of reversal of the change in vascular elasticity following CPB.

Note) Derivation of the formula for PWV: When pressure Δp is applied inside a hollow viscus with a wall thickness of h and with a radius of R , it balances the stress generated in the wall in the circumferential direction. If the Young's modulus of the wall is designated E , the equation

$$\Delta R = \Delta p \cdot R^2 / (E \cdot h)$$

holds insofar as the outwardly wall-pressing force; f_o , is equal to the inward pressing force, f_i .

Let the change in volume due to an elevation in blood pressure, Δp , be expressed as Δv , therefore:

$$\Delta v = \pi (R + \Delta R)^2 - \pi \cdot R^2 \doteq 2\pi \cdot R \cdot \Delta R = 2\pi \cdot R^3 \cdot \Delta p / (E \cdot h)$$

holds. The PWV is derived to be as

$$PWV = \{E \cdot h / (2 \cdot \rho \cdot R)\}^{1/2}$$

Substitution of Δv in the term $E \cdot h$ on the right side gives:

$$PWV = \{V_o / (\rho \cdot \Delta V / \Delta p)\}^{1/2}$$

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