COMPLIANCE OF HUMAN PULMONARY "VENOUS" SYSTEM ESTIMATED FROM PULMONARY ARTERY WEDGE PRESSURE TRACINGS
— Comparison with Pulmonary Arterial Compliance —

TSUTOMU TANAKA, M.D., MICHIKO ARAKAWA, M.D., TAKAHIKO SUZUKI, M.D.
MASAHIRO GOTOH, M.D., HIROMICHI MIYAMOTO, M.D.
AND SENRI HIRAKAWA, M.D.

To evaluate the reservoir function of the pulmonary vascular bed for the left ventricle, the compliance of the pulmonary "venous" system (Cp'v') (consisting of the pulmonary veins and the left atrium) and that of the pulmonary arterial system (Cpa) were sequentially estimated in each of 31 subjects by using Hirakawa's and Engelberg's methods, respectively.

In control cases (n = 6), Cpa was 6.68 ± 3.52 (mean ± SD) ml/mmHg and Cp'v' was 15.81 ± 6.85 ml/mmHg. In patients with mitral stenosis (MS) of Class I (previous classification of NYHA) (n = 7), Cpa was 4.05 ± 2.71 ml/mmHg and Cp'v' was 13.15 ± 4.51 ml/mmHg. In patients with MS of Class II (n = 13), Cpa was 2.81 ± 1.05 ml/mmHg and Cp'v' was 8.40 ± 2.95 ml/mmHg. In MS of Class III (n = 5), Cpa was 1.54 ± 0.80 ml/mmHg and Cp'v' was 7.10 ± 1.91 ml/mmHg. These results indicate that both systems become less compliant as the cardiac functional capacity deteriorates.

The ratio of Cp'v' to Cpa (Cp'v'/Cpa) was 2.7 ± 1.1 in control cases, 3.9 ± 1.4 in MS of Class I, 3.4 ± 1.6 in MS of Class II and 5.3 ± 2.1 in MS of Class III. When one compares these results with the compliance in the systemic circulation, i.e., 118 ml/mmHg in the veins and 2.5 ml/mmHg in the arteries, giving the ratio of 118/2.5 = 50, it is obvious that the compliance of the pulmonary arterial system shares a sizable portion of the total compliance in the pulmonary vascular bed.

The relationship between Cp'v' and the internal pressure, namely the mean pulmonary artery wedge (PAW) pressure, was expressed with a regression equation of, Cp'v' = 1/(0.003 PAW + 0.080), indicating that Cp'v' is inversely related to the internal pressure.

In 12 of patients with MS, sublingual nitroglycerin shifted the Cp'v' — PAW pressure plots upwards and to the left, roughly along the Cp'v' — PAW regression curve for the entire groups of MS.

THE pulmonary "venous" system, by definition, consists of the pulmonary veins and the left atrium. Although the role of this system has been established to be a reservoir for the left ventricle, little is known about the compliance of this system in humans.

Assuming the same relative distensibility as for the systemic veins, Beneken1 mathematically estimated the compliance of the human pulmonary "venous" system (Cp'v') to be 25 ml/mmHg. Guyton et al.2 reported it to be 10 ml/mmHg without any specific explanation. However, their

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- Pulmonary "venous" system
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- Compliance
- Mitral stenosis

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The Second Department of Internal Medicine, Gifu University School of Medicine, Gifu, Japan
Mailing address: Tsutomu Tanaka, M.D., The Second Department of Internal Medicine, Gifu University School of Medicine, 40 Tsukasa-machi, Gifu 500, Japan

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estimations were based on several assumptions and indirect evidence. Unfortunately their estimated compliances were calculated as a single value, disregarding the interaction of compliance with the level of internal pressure. Therefore, we estimated compliance in the “venous” system in a direct manner taking into consideration the relationship of compliance to internal pressure.

In 1981, Hirakawa et al.3 demonstrated that the $C_p'v'$ could be calculated from the pulmonary artery wedge (PAW) pressure tracings, using mathematical equations based on experimental evidence as well as some reasonable assumptions.
The pulmonary arterial system, by definition, is a “lumped” system, and its compliance (Cpa) can be calculated following Engelberg et al. It is also emphasized that the pulmonary arterial system is extremely distensible; for example, up to 66% of the right ventricular stroke volume may be stored during systole.

In the pulmonary vascular bed, the pulmonary “venous” system as well as the pulmonary arterial system functions as a blood volume reservoir for the left ventricle. The blood volume in the pulmonary vascular bed will be distributed to the pulmonary arterial system and to the pulmonary “venous” system, when their internal pressure is equal, just in proportion to the magnitude of the compliances of those two systems. Therefore, it is worthwhile estimating the compliances in order to evaluate the reservoir function.

We measured \( \Delta P \) and Cpa successively in each of 31 subjects identified as having normal hemodynamics or having mitral stenosis (MS) in order to investigate the capacitative role of compliance with in the entire pulmonary circulation. This study reflects a fundamental approach to the analysis of the blood volume distribution within the pulmonary circulatory system.

**SUBJECTS**

A total of 31 subjects were studied (Tables I and II). They consisted of 11 male and 20 female subjects, ranging from 12 to 62 years of age. The control group of 6 patients (No.1—6 in Table I) included patients with hemodynamically insignificant murmur (n = 4), sinus bradycardia (n = 1, the patient was in normal sinus rhythm at the time of study) and neurocirculatory asthenia (n = 1). Of the 25 patients with MS, there were seven with physical capacity Class I, 13 with Class II and 5 with Class III, using the previous NYHA classification. In the 25 cases with MS, 5 patients had associated aortic regurgitation (1+ or 2+ by Sellers’ classification) and one patient had aortic regurgitation (1+ by Sellers’ classification) and stenosis (the pressure gradient was less than 50 mmHg). Any patient with even minimal degrees of mitral regurgitation was excluded from this study. Twenty-two patients were in normal sinus rhythm and nine were in well-controlled atrial fibrillation.

**METHODS**

A number 7 or 8-French Courmand catheter
### Table II: Hemodynamic Data and Calculated Compliances of the Pulmonary "Venous" System in the Group of Mitral Stenosis

<table>
<thead>
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<th>Case No.</th>
<th>Sex</th>
<th>Age (Yrs)</th>
<th>BW (Kg)</th>
<th>BSA (M²)</th>
<th>Diagnosis</th>
<th>NYHA</th>
<th>Rhythm</th>
<th>P(A-W) mmHg</th>
<th>d mmHg</th>
<th>v mmHg</th>
<th>SV ml/beat</th>
<th>CI L/min</th>
<th>k&quot;</th>
<th>C(p)v (ml/mmHg)</th>
<th>C(p)v/(60Kg BW) ml/mmHg</th>
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</table>

**Mean ± SD**

|          | 52 ± 1.54 | 1.13 ± 0.12 | 13 ± 1.16 | 7.5 ± 1.16 | 3.0 ± 0.18 | 13.5 ± 0.18 | 15.2 ± 0.19 | 8.5 ± 0.19 |

14 M 51 63 MS II NSR 18 16 23 76 2.9 2.25 9.77 9.30 6.26
15 F 35 48 MS II NSR 13 13 19 65 3.3 1.88 8.13 10.16 5.53
16 F 36 42 MS II AF 18 16 23 76 2.2 2.25 8.36 5.14 2.69
17 F 55 42 MS II AF 15 12 21 86 3.1 2.03 8.41 12.73 6.80
18 F 54 56 MS II AF 15 8 19 44 1.9 2.03 3.24 3.47 2.12
19 F 44 55 MS II NSR 14 13 19 64 3.4 1.95 8.32 9.08 5.47
20 F 62 57 MS II NSR 9 9 15 62 3.0 1.58 6.51 6.85 4.12
21 F 41 38 MS II NSR 16 15 21 52 2.9 2.10 7.28 11.49 5.87
22 M 36 52 MS + AR II NSR 8 8 11 58 2.6 1.50 11.60 13.38 7.30
23 F 49 59 MS II AF 15 15 21 53 2.5 2.03 7.16 7.28 4.50
24 F 52 45 MS II AF 18 19 25 65 3.0 2.25 9.75 13.00 7.07
25 F 57 52 MS II NSR 15 15 19 65 2.7 2.03 13.16 15.18 9.01
26 M 37 50 MS II NSR 19 18 24 76 3.4 2.33 11.78 14.14 7.80

**Mean ± SD**

|          | 51 ± 1.47 | 0.7 ± 0.11 | 15 ± 1.19 | 20 ± 1.19 | 60 ± 1.19 | 2.9 ± 0.25 | 2.02 ± 0.25 | 8.40 ± 0.25 | 10.09 ± 0.25 | 5.73 ± 1.98 |

**MS = mitral stenosis; AF = atrial fibrillation; AR = aortic regurgitation; ARS = aortic regurgitation and stenosis**

The rest of the abbreviations is the same as Table I.
was introduced into the pulmonary artery from either the right antecubital vein or the femoral vein. Pulmonary arterial (PA) and pulmonary artery wedge (PAW) pressures were recorded with a strain-gauge transducer (P23Db). The dynamic frequency response characteristics of the pressure measurement system (catheter-transducer system) was adjusted as described by Grossman. In general, the damped natural frequency, thus obtained, was 10–20 Hz, which was appropriate enough to accurately record the PA and PAW pressure profile. The signals of the electrocardiogram and pressure were fed to a polygraph recorder (Model RM-85, Nihon Kohden Kogyo Co., Ltd.) and recorded at a paper speed of 50 mm/sec.

Figure 1 indicates a test to study the dynamic frequency response characteristics of a catheter-transducer system. Figure 2 shows electrocardiogram and PAW pressure tracings. We could clearly indentify and measure the level of d-point (diastasis = pre-a) and v-point (summit of the v wave) in PAW pressure tracings. The mean pressure was obtained electrically. The cardiac output was measured by the thermodilution method during right heart catheterization.

The compliance ($\Delta V/\Delta P$) of the pulmonary "venous" system ($C_p v'$) was calculated following the Hirakawa’s method that estimates $C_p v'$ by employing the parameters of stroke volume, the pressure difference between the d-point (abbreviated as d) and the summit of the following v wave (abbreviated as v) in PAW pressure tracings. In this method, the increment of PAW pressure during “ventricular systole”, from d to v ($\Delta P$), is considered to result mainly from the expansion of the blind-ended pulmonary “venous” system due to the in-flowing bulk of blood ($\Delta V$) from the right ventricle, which is a fraction of the right ventricular stroke volume (SV). Therefore, $\Delta V = k \cdot SV$ and $\Delta P = k' (v - d)$. Thus, the pulsatile compliance is given by an equation, $(\Delta V/\Delta P)$ puls. = $(k/k') SV/(v-d)$. One assumption in this method is that one can measure the ratio of two kinds of compliances, i.e., static (slope) compliance and dynamic (pulsatile) compliance. The ratio $(k''')$ is given by an equation of $k'' = (\Delta V/\Delta P)$ slope/$(\Delta V/\Delta P)$ puls. Finally, $(\Delta V/\Delta P)$ slope = $(k'' \cdot k)/SV/(v-d)$.

A reasonable round figure for $k$ is assumed to be 0.4 based on published records and $k'$ is assumed to be 1.0, because it appears that the pressure difference of $(v-d)$ in the PAW pressure

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### Table IV: Hemodynamic Data and Calculated Compliances of the Pulmonary Arterial System in the Group of Mitral Stenosis

<table>
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<th>Case No.</th>
<th>Sex</th>
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<th>Diagnosis</th>
<th>NYHA</th>
<th>P\textsubscript{A} (mmHg)</th>
<th>P\textsubscript{AW} (mmHg)</th>
<th>Q (ml/sec)</th>
<th>R (mmHg/ml/sec\textsuperscript{-1})</th>
<th>t (sec)</th>
<th>C\textsubscript{pa} (ml/mmHg)</th>
<th>C\textsubscript{pa}((60Kg BW) ml/mmHg)</th>
<th>C\textsubscript{pa}((M² BSA) ml/mmHg/M²)</th>
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</table>

P\textsubscript{A} = mean pulmonary arterial pressure; Q = mean pulmonary blood flow; R = pulmonary arterial resistance; C\textsubscript{pa} = pulmonary arterial compliance
The rest of the abbreviations is the same as Tables I and II.
tracings reflects, faithfully and totally, the pressure difference from d to v in a lumped, single-compartment model of the pulmonary "venous" system. The agreement of (v–d) in the PAW pressure tracings and that in the left atrial pressure tracings was excellent at least in 10 patients with uncomplicated secundum atrial septal defect. The values of $k''$ are obtained from in-vitro studies using pulmonary-vein-left-atrial preparations in dogs. In this regard, Nakano et al. found that $k''$ was an almost linear function of the internal (left atrial) pressure and the relationship between $k''$ and mean left atrial pressure (LAP) in this preparation, could be given by an equation of $k'' = 0.075 \cdot \text{LAP} + 0.90$ over a range of 40 to 90 beats/min of the heart rate and also over a range of 1.4 ml/beat to 2.8 ml/beat of the stroke volume.

Therefore, the final equation used to calculate the slope compliance of the human pulmonary "venous" system is

$$Cp'v' = \frac{\Delta V}{\Delta P}\text{slope} = 0.4(0.075PAW + 0.90) \frac{SV}{(v-d)}$$

Here, PAW pressure was substituted for LA pressure.

When (v–d) was less than 1 mmHg, such data were discarded, because minor inaccuracies in the identification of the pressure of v point or d point could cause large errors to occur in the calculated value of $Cp'v'$.
The compliance ($\Delta V/\Delta P$) of the pulmonary arterial system ($C_{pa}$) was calculated with the modified equation of Engelberg and Dubois. They showed that "lumped" pulmonary arterial compliance could be calculated using the pulmonary arterial pressure profile during diastole and the known left atrial pressure and pulmonary vascular resistance. Namely, the pressure difference between the pulmonary arterial pressure and the left atrial pressure during diastole is the exponential function of time and is related to the time constant. In the present study, the PAW pressure was used instead of the mean left atrial pressure. Therefore, the modified equation is as follows.

$$C = \frac{t}{(R \log_e(\Delta P_0/\Delta P_t))}$$

where

$C_{pa}$ = pulmonary arterial compliance

$P_{aw}$ = pulmonary arterial pressure

$P_{at}$ = left atrial pressure

$P_{di}$ = pulmonary arterial pressure at the mid diastole

$P_{es}$ = pulmonary arterial pressure at the end of systole

$P_{es}$ = pulmonary arterial pressure at the end of systole

$P_{di}$ = pulmonary arterial pressure at the mid diastole

$P_{es}$ = pulmonary arterial pressure at the end of systole

$P_{at}$ = left atrial pressure

$R_{aw}$ = pulmonary vascular resistance

$D$ = pressure gradient between the pulmonary artery and PAW pressure at the dicrotic notch (mmHg)

$D_0$ = pressure gradient between pulmonary artery and PAW pressure at the end of diastole (mmHg)

$t$ = time interval between the points of $D_0$ and $D_t$ (sec)

Figure 3 shows records of the pulmonary arterial pressure and the PAW pressure tracings.

In the present study, 12 of the patients with mitral stenosis were administered sublingual nitroglycerin (0.3 mg) in order to observe the effect of nitroglycerin on the pulmonary venous system. For this purpose, hemodynamic
Compliance of Human Pulmonary “Venous” System

Fig. 6. A ratio of the compliance of the pulmonary “venous” system (Cp′v′) to the pulmonary arterial compliance (Cpa).

In patients with mitral stenosis (MS) of Class III, the ratio of Cp′v′ to Cpa increased significantly compared to those of the other groups, mainly because the extent of reduction in Cpa was greater than that in Cp′v′.

measurements were repeated before and at the end of 5 to 8 minutes after nitroglycerin was given.

RESULTS

1) Compliance of pulmonary “venous” system (Cp′v′)

Table I and Table II indicate the hemodynamic data and the values of Cp′v′ in 31 patients.

In the control group (n = 6), as shown in Table I, the value of Cp′v′ averaged 15.81 ± 6.85 ml/mmHg (mean ± SD).

In the group of mitral stenosis (MS), as shown in Table II, the calculated value of Cp′v′ was 13.15 ± 4.51 ml/mmHg for patients with Class I physical capacity (n = 7). This value was not significantly different from that for the control group. In contrast, patients with MS of Class II (n = 13) and Class III (n = 5) physical capacity showed a much smaller value of Cp′v′ than that shown in patients with MS of Class I. The value of Cp′v′ averaged 8.40 ± 2.95 ml/mmHg and 7.10 ± 1.91 ml/mmHg, respectively. Therefore, the value of Cp′v′ was roughly reduced to a half of that in the control group and the group of MS with Class I physical capacity.

Figure 4 shows a plot of Cp′v′ against the PAW pressure for the entire data of Table I and Table II.

For the group of MS, 25 cases in total, the plots could be fitted to a regression equation:

\[ \text{Cp}′v′ = 1/(0.003 \text{ PAW} + 0.080) \]

as shown in Fig. 4.

2) Compliance of pulmonary arterial system (Cpa)

Tables III and IV show the hemodynamic data together with the values of Cpa for the same populations of the subjects as shown in Tables I and II.

In the control group (n = 6), the value of Cpa averaged 6.68 ± 3.52 ml/mmHg (mean ± SD), as shown in Table III. In the group of MS (Table IV), Cpa was 4.05 ± 2.71 ml/mmHg for patients with Class I physical capacity (n = 7) (0.05 < p < 0.1). The patients with MS of Class II physical capacity (n = 13) showed a relatively small value of Cpa (2.81 ± 1.05 ml/mmHg) (p < 0.001) and those with Class III physical capacity (n = 5) showed a significantly small value of Cpa (1.54 ± 0.80 ml/mmHg) (p < 0.001).

In Fig. 5, Cpa was plotted against the mean pulmonary arterial pressure. The short dashed curve represents the regression line which we calculated from the original data of Reuben,1 obtained mostly in patients with valvular heart disease, based on Engelberg’s theory and equation.4

As is clearly shown in Fig. 5, our plots of Cpa from a total of 31 patients, are in an agreement with the data of Cpa reported by Reuben.

3) Comparison of Cp′v′ with Cpa

Figure 6 shows the ratio of Cp′v′ to Cpa (Cp′v′/Cpa) in 31 patients. Cp′v′/Cpa ratio was 2.7 ± 1.1 (mean ± SD) in the control group, 3.9 ± 1.4 (0.1 < p < 0.2) in Ms of Class I, 3.4 ± 1.6 (0.1 < p < 0.2) in MS of Class II and 5.3 ± 2.1 (0.001 < p < 0.01) in MS of Class III physical capacity.

The ratio in patients with MS of Class III physical capacity was significantly higher than that in the control group. These results suggest that the ratio of Cp′v′ to Cpa (Cp′v′/Cpa) is about 3–5.

4) Effect of nitroglycerin on Cp′v′

Table V shows the hemodynamic data and Cp′v′ obtained before and after the administra-
### TABLE V
HEMODYNAMIC DATA AND CALCULATED COMPLIANCES OF THE PULMONARY "VENOUS" SYSTEM, BEFORE AND AFTER ADMINISTRATION OF NITROGLYCERIN

<table>
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<tr>
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<th>State</th>
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<th>$d$ mmHg</th>
<th>$v$ mmHg</th>
<th>$SV$ ml/beat</th>
<th>$Cl$ L/min</th>
<th>$Cp'v'$ ml/(mmHg)$^{-1}$</th>
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<td>37</td>
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<td>5.14</td>
</tr>
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</table>

Before = before nitroglycerin; after = after nitroglycerin
The rest of the abbreviations is the same as Table I.

$Cp'v'$ (before nitroglycerin) = $7.74 \pm 3.38$ ml/(mmHg)$^{-1}$ (mean ± SD); $Cp'v'$ (after nitroglycerin) = $11.92 \pm 6.62$ ml/(mmHg)$^{-1}$, ($p < 0.01$)

As shown in Fig. 7, along with the nitroglycerin-induced fall in $P_{AW}$ pressure, the $Cp'v'$-$P_{AW}$ pressure plots shifted upwards and to the left, generally along the regression line for the whole group of patients studied before nitroglycerin administration. The characteristic changes were a fall in the pressure and an increase in $Cp'v'$. The possible mechanism for these changes is discussed later in this paper.

**DISCUSSION**

1. Validity of substituting pulmonary artery wedge pressure for left atrial pressure.

In this study, we recorded the pulmonary artery wedge (PAW) pressure that was substituted for the left atrial (LA) pressure. This conventional way was also employed by Hirakawa et al.\(^3\) and Reuben\(^17\) to measure $Cp'v'$ and $Cpa$, respectively.

Numerous reports have been published concerning the agreement between PAW pressure and LA pressure. For instance, Connolly et al.\(^18\) recorded PAW pressure and LA pressure consecutively and concluded that PAW pressure pulse was a reasonably accurate reflection of LA pressure pulse both in magnitude and in contour. This was true mainly in patients with atrial septal defect and those with mitral stenosis (MS) during normal respiration and during assisted respiration at operation. Walston et al.\(^19\) found that at PAW pressure below 25 mmHg, there were no significant differences between the mean PAW pressure and the mean LA pressure in a variety of patients. Lozman et al.\(^20\) found that, at zero and 5 cmH\(_2\)O PEEP (positive end-expiratory pressure), there was a statistically significant correlation between...
the mean LA pressure and the mean PAW pressure, both simultaneously measured. Lappas\textsuperscript{21} concluded that the mean PAW pressure was a reliable index for the clinical estimation of the mean LA pressure in cardiac patients, when measured serially. From this laboratory, Miyamoto et al.\textsuperscript{15} performed rather sophisticated simultaneous measurements of the phasic LA pressure by tip-manometer and the phasic PAW pressure by fluid-filled catheter and showed a good agreement between them.

(2) Compliance of pulmonary "venous" system.

Recently, Hirakawa et al.\textsuperscript{3} described the estimated compliance of the pulmonary "venous" system (Cp'v') in a total of 37 human subjects, by using essentially the same principles and methods used in the present study. In Fig. 8, we plotted the data of Hirakawa et al.\textsuperscript{3} and our data on the same Cp'v'-PAW-plane.

Strong support was given to the present method of calculating Cp'v' by an interesting new observation\textsuperscript{22} The Cp'v' was calculated for 42 patients, a group consisting of "normal" individuals (n = 8), and those with myocardial infarction (n = 21), angina pectoris (n = 4), mitral stenosis (n = 8) and myocardial disease (n = 1). The compliance measurement was performed, in each subject almost simultaneously by two independent methods, one using PAW pressure tracings (the present method) and the other, using the pulmonary blood volume measurement and passive elevation of both legs. A gamma-camera, placed externally over one lung, recorded the radio-activity and its increment upon elevation of both legs. The percentage increment in the ratio-activity over the lung was the basis of calculating the increment in the pulmonary blood volume, after corrections were made for the radio-activity originating in the anterior and posterior chest walls and for the absorption of radio-active beams within the lung and anterior chest wall. The PAW pressure and its increment upon leg-elevation were recorded by a Swan-Ganz catheter. Thus, the compliance calculated from the PAW pressure tracings (Y) related to the compliance calculated from the pulmonary blood volume and passive leg-elevation (X) as follows:

\[ Y = 0.94X + 0.89 \quad (n = 42, r = 0.79, p < 0.01) \]

Shoukas\textsuperscript{23} proved, in his comprehensive study using the isolated perfused lung of dogs, that the compliances of the pulmonary venous vascular bed including LA were 0.1236 and 0.0955 ml/mmHg/Kg for the pulmonary arterial pressures of 15 and 20 mmHg, respectively. Simple calcula-
tion of extrapolating his data to humans weighing 60 Kg shows that the compliance of the pulmonary veins and the left atrium is about 7.4 – 5.7 ml/mmHg. These are nearly a half of our present study (17.17 ± 6.85 ml/mmHg, Table I).

Engelberg et al.4 described, in their study of the isolated rabbit lungs, that the mean static compliance of the pulmonary venous tree (not including the left atrium) was 0.41 (SE ± 0.03) ml/cmH2O. If we would extrapolate their data of rabbit to humans weighing 60 Kg, we could calculate the compliance of the pulmonary venous trees to be 4.2 ml/mmHg.

Liu et al.24 indicated, in their study of the postmortem human left atrium, that the mean initial volume of the left atrium was 27.93 ml at zero pressure, and the mean volumes were 50.20 ml and 61.70 ml at 5 and 10 mmHg, respectively. Therefore, we could calculate the static compliance of the left atrium to be (50.20 – 27.93)/5 = 4.45 or (61.70 – 50.20)/5 = 2.30 ml/mmHg. These were regarded to be about 4 ml/mmHg. Accordingly, if we combine the sum of compliance of the pulmonary veins (4.2) and the left atrium (4.0), the sum equals about 8 ml/mmHg. If we modify the compliance (ΔV/ΔP) of the present studies of patients (Table I) to a standard body weight of 60 Kg, the ΔV/ΔP averages 17 ml/mmHg. In the light of this value, the ratio of the compliance of the left atrium, described just above (4 ml/mmHg, CLA) to the compliance of the pulmonary venous system (17 ml/mmHg, Cp’v”), should be CLA/Cp’v = 0.23. This average value is very close to the ratio of CLA/Cp’v that was determined in humans by right heart catheterization with measurements of the PAH pressure tracings, the stroke volume and the cineangiographic determination of the left atrial volume.25

(3) Compliance of pulmonary arterial system.

A few studies are available that employ the current method of estimating the compliance of the pulmonary arterial system (Cpa) originally invented by Engelberg and DuBois.4 Some problems are intrinsic to their method, such as oversimplification of the pulmonary artery system, an influence of the heart rate, pressure recording systems etc., and are unavoidable. Recently the pulmonary artery input impedance has been commonly used to evaluate the character of the pulmonary artery system. Nevertheless, we chose to employ the present simple method as clinically reliable.

For instance, Engelberg and DuBois4 obtained a mean pulmonary arterial compliance of 0.17 ml/cmH2O (S.E. ± 0.02) in six rabbits, mean weight, 4.4 Kg, which in turn is recalculated to be 1.7 ± 0.3 ml/mmHg (mean ± SE) in humans of body weight of 60 Kg. Reuben17 and Hando et al.26 described, in patients with various hemodynamics from normal to several-fold elevation of the mean pulmonary arterial pressure (up to about 60 mmHg), the mean Cpa to be 0.7 – 2.87 ml/mmHg and 1.64 – 3.93 ml/mmHg, respectively. Apparently, as shown in Fig. 5, Cpa decreased as the pulmonary arterial pressure increased. The regression line which we calculated from the original data of Reuben17 were in a good agreement with our results (Fig. 5).

Independently, Beneken1 mathematically and indirectly estimated the compliance of the pulmonary artery to be 4.3 ml/mmHg based on the past numerical data.

(4) Comparison of Cp’v’ and Cpa.

Based on the reported numerical data of systemic vessels together with several assumptions, Beneken1 calculated the compliance of the systemic arteries, including the aorta (Csa), and the systemic veins (Csv). According to these calculations, usually Csa is 2.5 ml/mmHg and Csv is 118 ml/mmHg. Therefore, the ratio of Csv to Csa (Csv/Csa) is approximately 50. When considering pulmonary vessels, however, the ratio of Cp’v’ to Cpa (Cp’v’/Cpa) is 3 to 5. In addition, Cpa is 1.5 – 6.7 ml/mmHg in the present study. Taking these facts into consideration, it is suggested that the pulmonary arteries share a sizable portion of the total pulmonary vascular compliance and possess the function as the capacitance vessel in the pulmonary circulatory system.

(5) Effect of nitroglycerin on Cp’v’.

Under the effect of nitroglycerin, PAW pressure was reduced and an increment in Cp’v’ occurred. One of the reasonable explanations for this fact comes from the observation of the pulmonary “venus” volume (V, ordinate) and PAW pressure (P, abscissa) in a series of studies using a gamma-camera over the right lung field and 99m-Tc-percetanate. Nitroglycerin clearly causes PAW pressure (P) to decrease, but it decreases V only minimally. What is more important is the fact that nitroglycerin caused the compliance (ΔV/ΔP) to increase significantly.27 With these changes occurring simultaneous-

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ly, it is easily anticipated that the sublingual administration of nitroglycerin increases the compliance (ΔV/ΔP) of this system, associated with a fall of PAW pressure. Therefore, nitroglycerin appears to cause the V-P-plot to shift from a normal V-P curve to a more left-sided, steeper and probably more compliant V-P curve.

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