Journal of Cardiography 11: 591-601, 1981

Pulmonary arterial extensibility in patients with intracardiac left to right shunt

Akihiro SAITO
Ken UEDA
Hiroyuki NAKANO

Summary

The extensibility of the pulmonary artery (PA) were examined using a combined hemodynamic and ultrasonic technique in 14 patients with atrial septal defect (ASD), three with ASD and pulmonary hypertension (PH), 17 with ventricular septal defect (VSD) and 11 with VSD and PH. Seven patients with neither intracardiac shunt nor PH served as controls.

The tension (T) of PA was measured as the product of the pressure and the diameter (D) and was found to be high in the patients with intracardiac shunt. The resting diameter of PA (D_0) was calculated as the intersect with the D axis in the regression line of PA tension-diameter relation. The Lagrangian strain of PA $[(D-D_0)/D_0]$ was obtained using D_0 and correlated with T yielding a linear regression equation. The elastic modulus (E) was obtained as the slope of this equation.

E in the patients with ASD $[(1.0\pm0.5)\times10^5$ dynes/cm] or VSD $[(9.8\pm4.6)\times10^4$ dynes/cm] were slightly higher than that of the control $[(6.7\pm2.9)\times10^4$ dynes/cm], but these differences were not statistically significant. However, E in the patients with ASD and PH $[(2.5\pm1.3)\times10^5$ dynes/cm] or in the patients with VSD and PH $[(2.6\pm0.9)\times10^5$ dynes/cm] were significantly higher than the control. E seemed unlikely to be causally related to the resistance of PA capillary beds, and was suggested to be influenced by the histological changes of the pulmonary trunk.

It was concluded that the extensibility of PA in the patients with ASD or VSD was not altered secondary to the increased blood flow unless PH was associated.

Key words

Pulmonary arterial extensibility Atrial septal defect Ventricular septal defect Pulmonary hypertension Echocardiography

Atrial and ventricular septal defects are often associated with either increased pulmonary flow, increased pulmonary arterial pressure, or both. The evaluation of these hemodynamic changes of the pulmonary artery plays an important role in determining the severity and deciding the indication of the surgical treatment of these disorders.

The pattern of the pulmonary arterial blood flow and pressure must be a function of the

静岡県立こども病院 循環器科
静岡市漆山 860 (〒420)

Division of Pediatric Cardiology, Shizuoka Children's Hospital, Urushiyama 860, Shizuoka 420

Presented at the 21st Meeting of the Japanese Society of Cardiovascular Sound held in Tokyo, October 14–15, 1980
Received for publication January 6, 1981

driving pressure generated by the right ventricle, the physical characteristics of the wall of the pulmonary artery and the impedance of the pulmonary arterial capillary beds. Particulary the mechanical properties of the wall of the pulmonary artery have an important influence on the pulmonary blood flow and pressure[1,2]. Although there are many investigations concerning the histological changes of the pulmonary artery in various congenital heart diseases[3,4], few reports are available relating to the mechanical properties of the pulmonary artery[5~8].

We have therefore undertaken studies to investigate the physical characteristics of the pulmonary artery in vivo using echocardiography and micromanometry in patients with atrial and ventricular septal defects.

Materials and Methods

The patients studied were 52 children who underwent the cardiac catheterization. Patients were divided into 5 groups according to the hemodynamic data. Group 1 contained 14 patients with atrial septal defect (ASD) and normal pulmonary arterial (PA) pressure with the ages ranging from one year and 2 months to 13 years and 7 months (mean age; 5 years and 11 months). The pulmonic to systemic flow ratios (Qp/Qs) ranged from 1.27 to 2.64 (2.01 $\pm$0.38) and the mean PA pressures were less than 25 mmHg and ranged from 14 to 23 mmHg (19$\pm$2 mmHg). Group 2 had 3 patients with ASD and pulmonary hypertension (PH) ranging in age from 7 months to 8 years and four months (7 years and 3 months). Qp/Qs ranged from

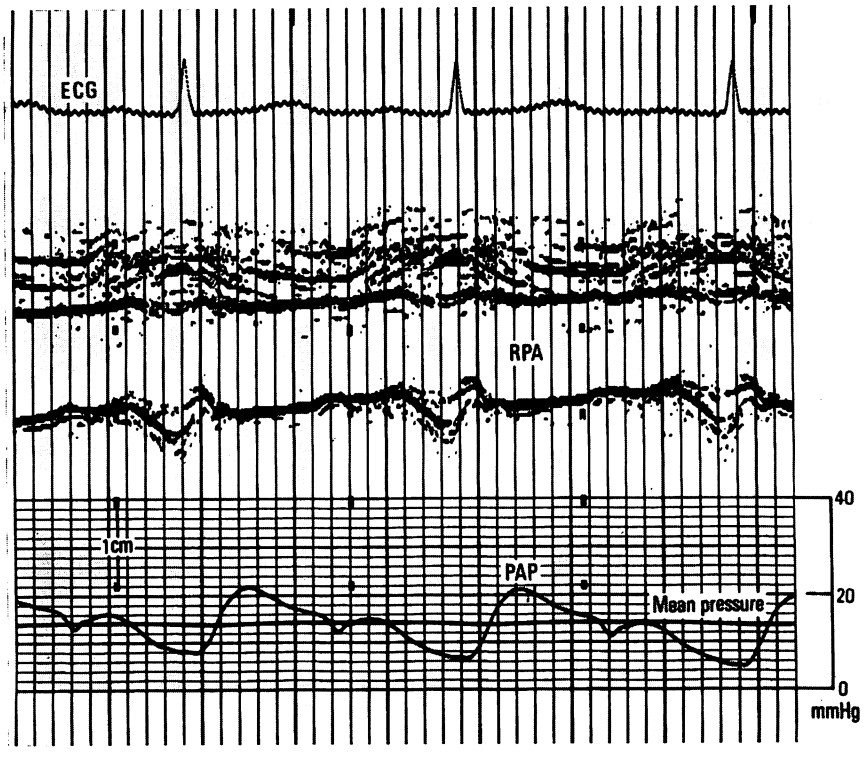

Fig. 1. Representative recordings of pulmonary arterial pressure and the M-mode echocardiogram.

ECG=electrocardiogram; RPA=right pulmonary artery; PAP=pulmonary arterial pressure.

0.99 to 1.69 (1.43±0.38) and the mean PA pressure ranged from 29 to 60 mmHg (49± 17 mmHg). Group 3 contained 17 patients with ventricular septal defect (VSD) and normal PA pressure. The ages of them ranged from one year and a month to 6 years and 3 months (3 years and 9 months), Qp/Qs from 1.10 to 3.30 (1.53±0.59) and the mean PA pressure from 11 to 22 mmHg (19±2 mmHg). Group 4 had 11 patients with VSD and PH ranging in age from 5 months to 5 years and 2 months (2 years and 7 months). Qp/Qs were increased to 2.54± 0.58 ranging from 1.78 to 3.46, and the mean PA pressures were also elevated and ranged from 38 to 77 mmHg (58±11 mmHg). Group 5 (7 patients) consisted of 4 children with mild isolated aortic stenosis (left ventricular-aortic pressure gradient less than 25 mmHg), 2 with the history of Kawasaki's disease without demonstrable cardiovascular abnormalities and one with an abnormal electrocardiogram without other cardiac disorders. All of them had no intracardiac shunt and had normal mean PA pressure ranging from 9 to 17 mmHg (14± 3 mmHg). The ages of them ranged from one year and a month to 14 years and 9 months (6 years and 8 months).

The simultaneous recordings of the M-mode echocardiogram and the PA pressure measured by a micromanometer were performed during the routine diagnostic cardiac catheterizations (**Fig. 1**). M-mode echocardiograms were obtained using a Smith-Klein 20-A ultrasonoscope interfaced with an Electronics for Medicine VR-12 optical recorder and a nonfocused hammer shaped transducer with a frequency rate of 3.5 MHz.

The right pulmonary artery (RPA) was examined using a suprasternal notch approach, and was recorded only in the plane showing the characteristic motion pattern in which the superior wall of the left atrium was separated from the inferior wall of RPA in the phase of the atrial contraction. The diameter of RPA was defined as the internal distance between the superior and the inferior walls of RPA[9].

The PA pressure was recorded using a Millar catheter-tip micromanometer. Since the recording of RPA pressure was not always possible, the main PA pressure was used rather than RPA pressure. To ensure the accuracy of this methodology, the right and main PA pressures were compared in 14 patients in whom the both pressures could be recorded during the cardiac catheterizations. These patients consisted of 5 children with VSD, four children with ASD, three children with tetralogy of Fallot and two children with patent ductus arteriosus.

The diagram of the devices which were used for digitization of the recordings, calculation and display is shown in **Fig. 2**. The diameter of RPA (*D*) and the main PA pressure (*P*) were digitized at 10 msec intervals using a Graf pen sonic digitizer (Science Accessories Corporation) and were fed to a NOVA 01 16K minicomputer. The graphs and the calculations were displayed on a TV monitor and printed by a Casio typuter.

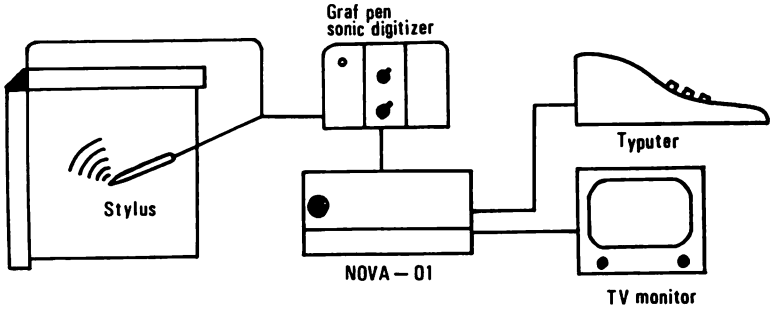

Fig. 2. Diagram of the devices.

The diagram of the data analysis was shown in **Fig. 3**. First, the tension (T) of PA was calculated as the product of D and P using the law of Laplace, and then it was correlated with D. The relation of these variables was almost linear during ejection phase (**Fig. 4**). The linear regression equation was obtained and the slope of this equation (m) and the intersect with the abscissa (D_0) were calculated. D_0 expresses the diameter of PA when the tension of the wall is unloaded, that is, resting dimension. Then the Lagrangian strain, $(D-D_0)/D_0$, was calculated and was correlated with T yielding a linear regression equation. The elastic modulus (E)

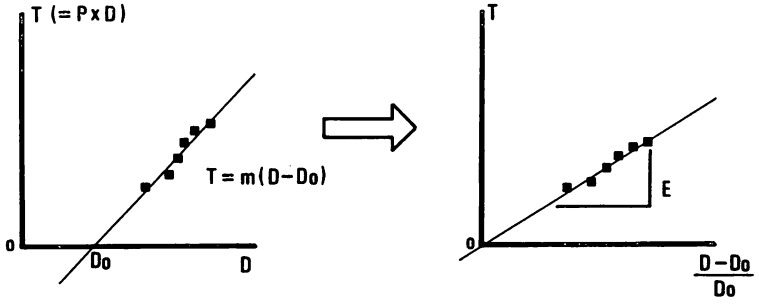

Fig. 3. Diagram of the data analysis.
T=tension; P=pressure; D=diameter; D_0=resting diameter; E=elastic modulus.

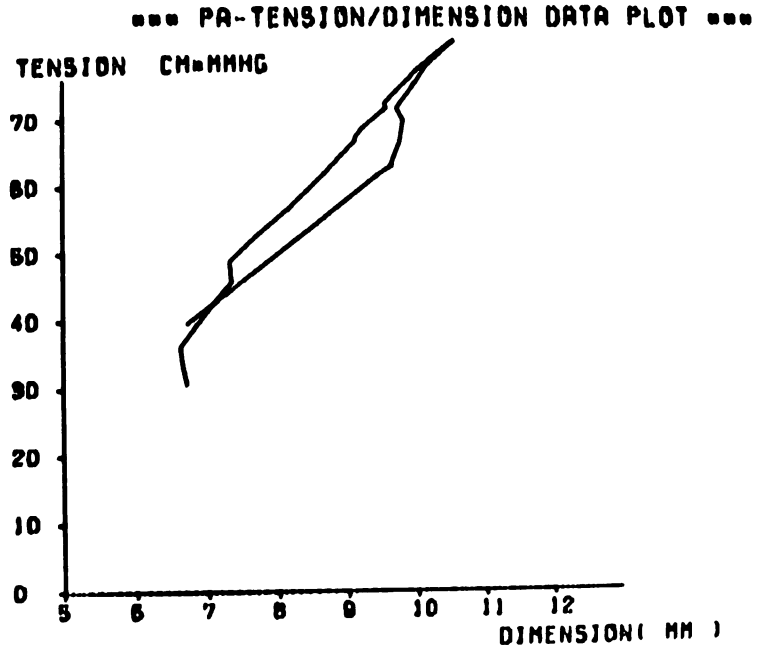

Fig. 4. Representative recordings of the pulmonary arterial tension-dimension relationship.

was obtained as the slope of this equation (**Fig. 3**) which expressed the instantaneous change in tension divided by the corresponding change in strain; $E=T/[(D-D_0)/D_0]$. The wall tension was used in the present calculations rather than the wall stress since the wall thickness of PA could not be measured by this method. The maximum tension of PA throughout the cardiac cycle was also obtained.

Results

The reliability of the method to measure the size of RPA from the M-mode echocardiogram has ascertained in the previous reports.[9] The comparison of RPA diameters determined by both M-mode echocardiography and angiocardiography yielded an excellent correlation coefficient ($r=0.97$) and showed that these two measurements were almost identical (**Fig. 5**).

The right and main PA pressures were compared in both systole and diastole (**Fig. 6**). The regression equations of each correlation demonstrated that the values of these pressures were also almost identical.

The representative tracing of each disorder (ASD, ASD+PH, VSD and VSD+PH) were given in **Fig. 7**.

The resting diameter (D_0) was 0.9 ± 0.2 cm

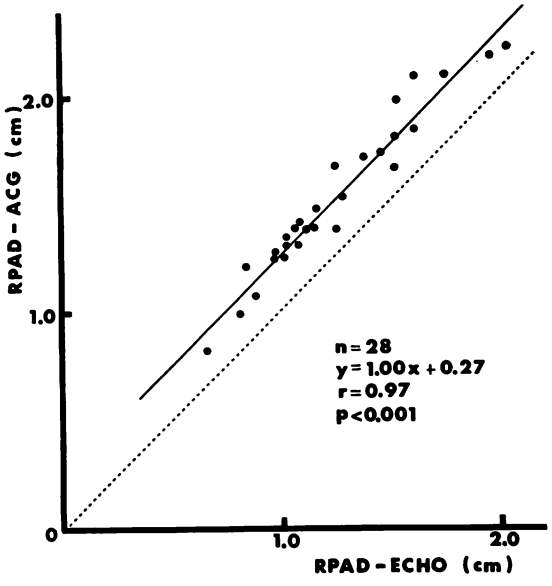

Fig. 5. Correlation between right pulmonary arterial dimensions determined by both echocardiography and angiocardiography.

RPAD-ECHO=right pulmonary arterial dimension determined by echocardiography. RPAD-ACG=right pulmonary arterial dimension determined by angiocardiography.

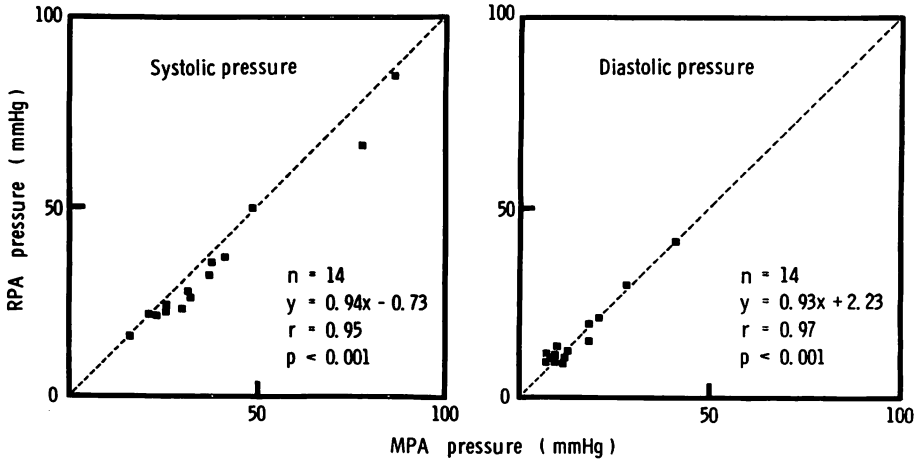

Fig. 6. Correlation between right and main pulmonary arterial pressures.

RPA=right pulmonary artery; MPA=main pulmonary artery.

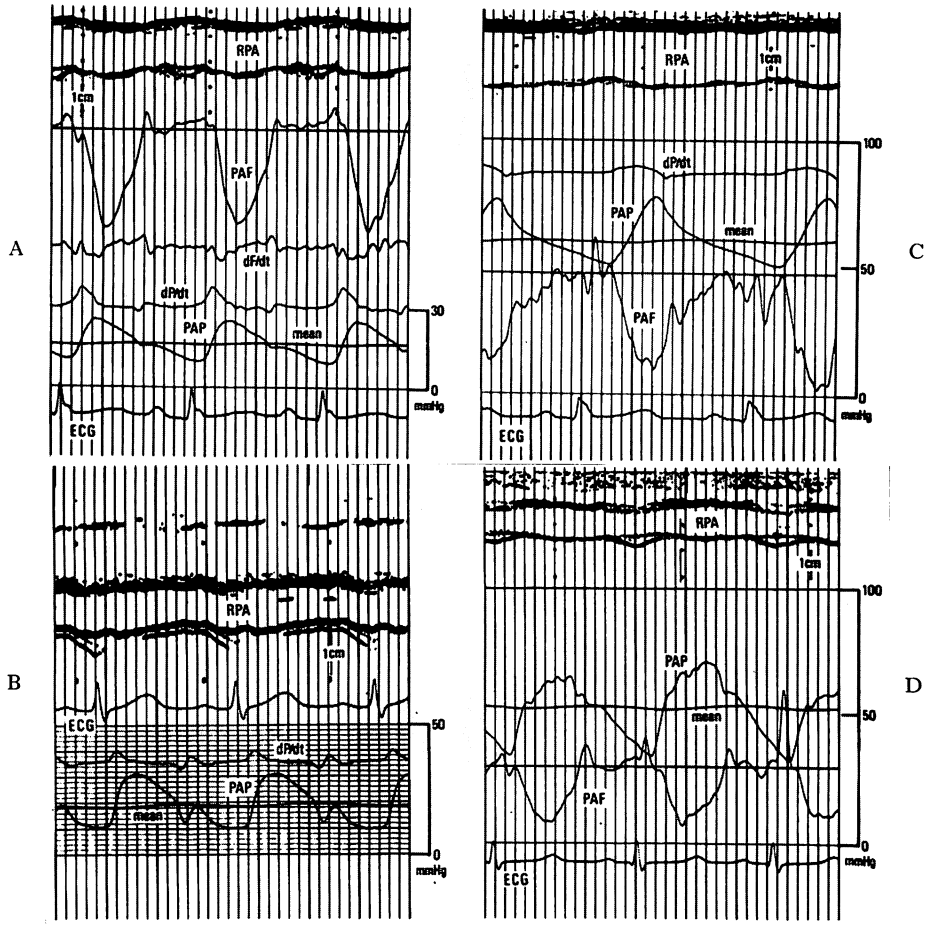

Fig. 7. Representative recordings of each disorder.

A: atrial septal defect. B: atrial septal defect with elevated pulmonary arterial pressure. C: ventricular septal defect. D: ventricular septal defect with elevated pulmonary arterial pressure.

RPA=right pulmonary artery; PAF=pulmonary arterial flow; dF/dt=first time derivative of pulmonary arterial flow; PAP=pulmonary arterial pressure; dP/dt=first time derivative of pulmonary arterial pressure; ECG=electrocardiogram.

in the control group (**Fig. 8**). D_0 was also obtained in the patients with ASD (1.0 ± 0.2 cm), ASD+PH (1.1 ± 0.4 cm), VSD (0.8 ± 0.2 cm) and VSD+PH (0.8 ± 0.2 cm). Although the values of the patients with ASD and ASD+PH were somewhat larger than that of the control group, any of the 4 groups showed no statistically significant difference from the control group.

The slope of the regression line (m) was (7.2 ± 2.3)$\times10^4$ dynes/cm^2 in the control group. This was also calculated for the patients with ASD [(1.1 ± 0.4)$\times10^5$ dynes/cm^2], ASD+PH [(2.2 ± 0.6)$\times10^5$ dynes/cm^2], VSD [(1.2 ± 0.4)$\times10^5$ dynes/cm^2] and VSD+PH [(3.1 ± 0.5)$\times10^5$ dynes/cm^2]. These 4 measurements showed statistically higher values than that of the control group (**Fig. 9**).

The maximum tension of the patients with VSD [(3.7 ± 0.6)$\times10^4$ dynes/cm] was not sig-

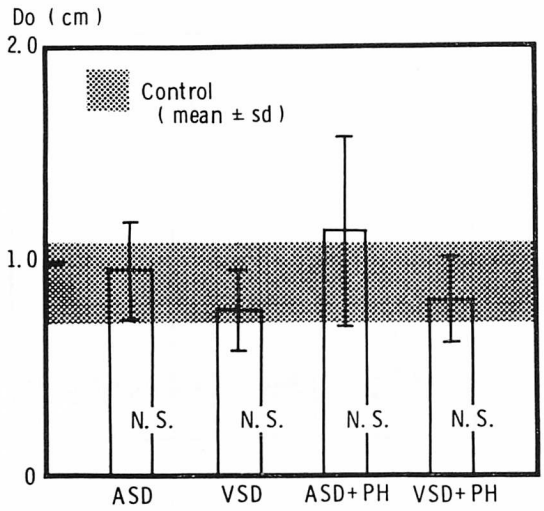

Fig. 8. Resting diameter (D_0) **of each disorder.**
Shaded area indicates the value of control.
ASD=atrial septal defect; VSD=ventricular septal defect; ASD+PH=atrial septal defect with pulmonary hypertension; VSD+PH=ventricular septal defect with pulmonary hypertension.

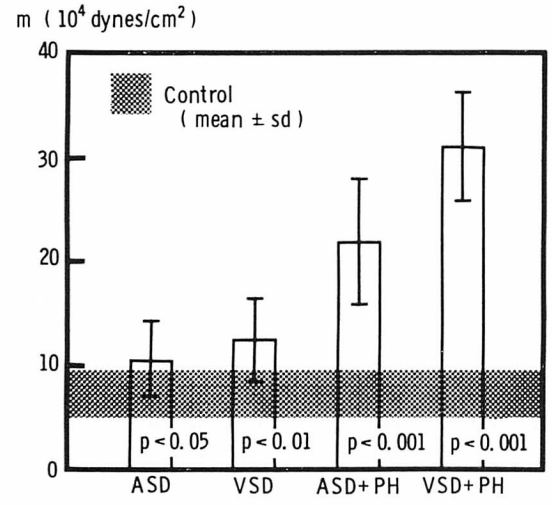

Fig. 9. Slope of regression line (m) **of each disorder.**
Shaded area indicates the value of control.
Abbreviations: See Fig. 8.

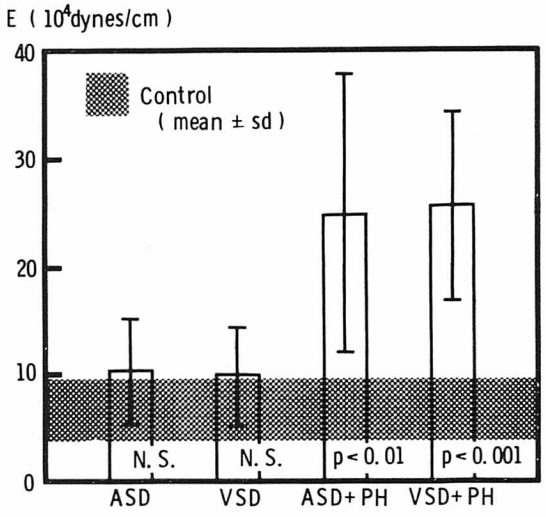

Fig. 10. Maximum tension of each disorder.
Shaded area indicates the value of control.
Abbreviations: See Fig. 8.

nificantly different from that of the control group [$(3.5\pm1.2)\times10^4$ dynes/cm], but the values of other three groups [ASD; $(5.6\pm1.3)\times10^4$ dynes/cm, ASD+PH; $(1.7\pm0.9)\times10^5$ dynes/cm, VSD+PH; $(1.3\pm0.5)\times10^5$ dynes/cm] were higher than the control group (**Fig. 10**).

The regression equation relating the pulmonary arterial tension (T) as a function of the pulmonary arterial diameter (D) is given by the formula

$$T = m(D-D_0)$$
$$= mD-mD_0$$

where m represents the slope of the line, D_0 represents the intersection with the abscissa and $-mD_0$ represents the intersection with the ordinate.

The elastic modulus (E) is calculated from the formula

$$E = T/[(D-D_0)/D_0]$$

Since $T/(D-D_0)=m$ from the foregoing formula, E can also be obtained from the following formula

$$E = mD_0$$

Therefore, E equals the absolute values of the intersection with the ordinate on the re-

gression line relating the PA tension as a function of PA diameter (**Fig. 11**).

The representative regression lines were obtained in each groups by connecting the average values of the intersection with the ordinate and that with the abscissa (**Fig. 11**). As is evident from this graph, the absolute value of the intersection with the ordinate which expresses the elastic modlus is large in the patients with PH, but it does not differ much from control in the patients without PH. This is also demonstrated in **Fig. 12**. The elastic modulus in the patients with ASD [$(1.0\pm0.5)\times10^5$ dynes/cm] or VSD [$(9.8\pm4.6)\times10^4$ dynes/cm] were slightly larger than the control [$(6.7\pm2.9)\times10^4$ dynes/cm], but these differences were not statistically significant. However, the elastic modulus in the patients with ASD+PH [$(2.5\pm1.3)\times10^5$ dynes/cm] or VSD+PH [$(2.6\pm0.9)\times10^5$ dynes/cm] were significantly ($p<0.01$ and $p<0.001$, respectively) larger than the control.

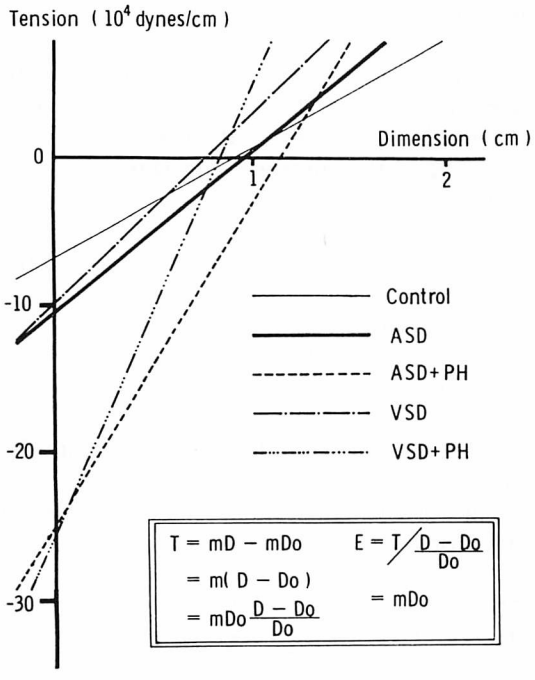

Fig. 11. Representative tension-dimension relations of each disorder.
Abbreviations: See Fig. 8.

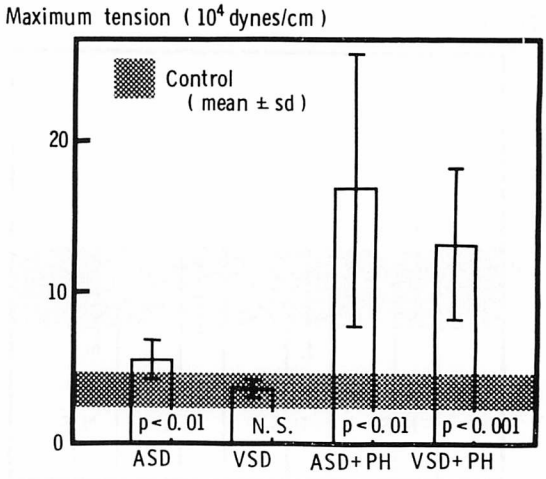

Fig. 12. Elastic modulus (E) **of each disorder.**
Shaded area indicates the value of control.
Abbreviations: See Fig. 8.

The comparison between the PA resistance and the elastic modulus is shown in **Fig. 13**. The values of the resistance were high in the patients with the large elastic modulus and were low in the patients with the small elastic modulus. However, in each group, no statistical correlation was demonstrated between these two variables.

Discussion

Our data showed that the extensibility of the PA walls in the patients with ASD or VSD did not appear to differ significantly from that in the control unless PH was associated. And the elastic modulus seemed unlikely to be causally related to the resistance of the PA capillary beds.

These results agree with the previous reports[5,6]. Harris and his co-workers[5] measured the extensibility of the human PA walls in vivo by hanging weights on strips of the PA walls. The extensibility of the pulmonary trunk in the patients with ASD or VSD with normal PA pressure were within normal limits, but in the patients with elevated PA pressures, the extensibility were less than normal. Jarmakani et al.[6] studied the pressure-radius relationships

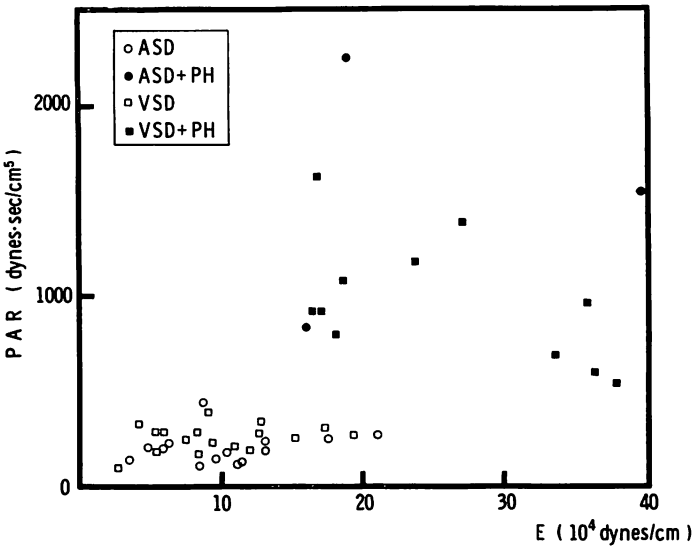

Fig. 13. Correlation between pulmonary arterial resistance and elastic modulus.
PAR=pulmonary arterial resistance; E=elastic modulus.
Other abbreviations: See Fig. 8.

of PA in the patients with congenital heart disease by angiocardiography and showed that the wall of PA was stiffer in the patients with PH but its elastic properties were not altered secondary to increased blood flow.

While on the other hand, Boughner and Roach[7] examined the elastic properties of PA in vivo using angiocardiography in the patients with ASD, and described that PA in those patients were more distensible than the controls. Their result is diametrically opposed to our findings. In their study, PA pressures were not simultaneously recorded with the angiocardiograms, and a fluid filled catheter-transducer system was used for the pressure recordings. It is well known that in this system one can not avoid a damping or an overshoot of the pressure[10]. Actually, a lot of oscillations were found on the pressure tracings in their paper. Moreover, the position of the pulmonary trunk for the measurement of the diameter was not fixed in their work. Since the extensibility of PA must be varied with the position for the measurement, their results might be influenced by the difference in the measured position among the patients. Thus the disagreements in the results between their and our studies may be caused by the difference in the methodology.

Heath and his co-workers[3] reported that in those patients with intracardiac left to right shunts in whom PH had existed from birth, the elastic tissue in the pulmonary trunk preserved its fetal appearance which was similar to that of the aorta, and the tissue of the pulmonary trunk was less extensible than normal. In this study, all patients with PH were also thought to have been associated with PH since birth according to their histories, and showed a decreased extensibility of PA. From these observations, the extensibility of the pulmonary trunk appears to represent the histological changes of this organ.

The technique to evaluate the extensibility of the pulmonary trunk given in this paper is thought to offer several advantages over the previously described manners[5~8]. Firstly, the simultaneous and instantaneous recordings of the pressure and the diameter can be obtained

easily in vivo. Secondary, the influence of the contrast media which may cause the change of the intraluminal pressure, the diameter or the extensibility of the wall of the PA can be avoided. Thirdly, for the evaluation of the extensibility, Lagrangian strain which is dimensionless quantity can be used obtaining the resting diameter from the tension-diameter relationship.

On the other hand, the disadvantages of our method are as follows. The technique can not be applied to the patients who has a pulmonary branch stenosis. And in the process of the analysis, special devices are necessary for the digitization and the calculation.

The present study offered some useful informations about the hemodynamics of the pulmonary trunk in those who have increased pulmonary blood flow, and they are given belows. The increased blood flow augments the wall tension but does not make the pulmonary trunk less extensible by itself. The pulmonary hypertension is associated not only with the increased impedance of the pulmonary arterial capillary beds but also with the changes of the mechanical properties of the pulmonary trunk. In spite of the increased blood flow or the elevated intraluminal pressure, the resting diameters of PA do not differ much from the control.

It was concluded that the technique to evaluate the extensibility of PA from the simultaneous recording of the PA pressure and the echocardiogram was easy to perform and was useful for assessing the effect of the increased pressure or the blood flow on the mechanical properties of PA.

要　　約

左右短絡群心疾患における肺動脈伸展性

斉藤彰博，上田　憲，中野博行

胸骨上窩心エコー図と肺動脈圧との同時記録より，左右短絡群心疾患の肺動脈伸展性を検討した．対象は心房中隔欠損14例，同症の肺高血圧合併例3例，心室中隔欠損17例，同症の肺高血圧合併症例11例で，心内短絡や肺高血圧の合併のない7例を対照とした．

肺動脈張力は肺動脈径と肺動脈圧との積として計算したが，各疾患とも高値を示した．張力と肺動脈径との直線回帰式における肺動脈径軸の切片を，無負荷時の径（D_0）とした．ついで，このD_0を用いて肺動脈ストレイン$(D-D_0)/D_0$を算出し，張力との直線回帰を行い．その傾きを伸展率（E）とした．

Eの値は心房中隔欠損では$(1.0\pm0.5)\times10^5$ dynes/cm，心室中隔欠損では$(9.8\pm4.6)\times10^4$ dynes/cmと，いずれも対照$(6.7\pm2.9)\times10^4$ dynes/cmよりやや高値を示したが，統計的に有意でなかった．これに対し，肺高血圧を合併した心房中隔欠損では$(2.5\pm1.3)\times10^5$ dynes/cm，心室中隔欠損では$(2.6\pm0.9)\times10^5$ dynes/cmと，有意に高値を示した．Eの値は，肺血管抵抗と直接の因果関係を認めず，肺動脈の組織的変化に影響されることが示唆された．

心房中隔欠損や心室中隔欠損においては肺高血圧を合併しないかぎり，肺血流の増大のみでは肺動脈伸展性が犯されないことが示された．

文　献

1) Patel DJ, Schider DP, Mallos AJ: Mechanical properties and dimensions of the major pulmonary arteries. J Appl Physiol **15**: 92, 1960
2) Maloney JE, Bergel DH, Glazier JB, Hughs JMN, West JB: Transmission of pulsatile blood pressure and flow through the isolated lung. Circulat Res **23**: 11, 1968
3) Heath D, DuShne JW, Wood EH, Edwards JE: The structure of the pulmonary trunk at different ages and in cases of pulmonary hypertension and pulmonary stenosis. J Path Bact **77**: 443, 1959
4) Heath D, Edwards JE: The pathology of hypertensive pulmonary vascular disease: A description of six grades of structural changes in the pulmonary arteries with special reference to congenital cardiac septal defects. Circulation **18**: 533, 1958
5) Harris P, Heath D, Apostolopoulos A: Extensibility of the pulmonary trunk in heart disease. Br Heart J **27**: 660, 1965
6) Jarmakani JMM, Graham TP Jr, Benson DW Jr,

Canent RV Jr, Greenfield JC Jr: In vivo pressure-radius relationships of the pulmonary artery in children with congenital heart disease. Circulation **43**: 585, 1971
7) Boughner DR, Roach MR: Etiology of pulmonary artery dilatation and hilar dance in atrial septal defect. Circulat Res **28**: 415, 1971
8) Aziz KU, Nanton MA, Kidd L, Moes CAF, Rowe RD: Variation in the size and distensibility of the pulmonary arteries in d-transposition of the great arteries. Am J Cardiol **38**: 452, 1976
9) Saito A, Ueda K, Nakano H: Methodology and evaluation of the measurement of the right-sided cardiac chambers by M-mode echocardiography. J Cardiography **10**: 967, 1980
10) Yang SS, Bentivoglio LG, Maranhão V, Goldberg H: From cardiac catheterization data to hemodynamic parameters. 2nd ed. F. A. Davis, Philadelphia, 1978