Pulmonary Vascular Volume, Resistance, and Compliance in Man

By William R. Milnor, M.D., Anthony D. Jose, M.R.C.P., and Charles J. McGaff, M.D.

VARIATIONS in the volume of blood in the pulmonary vessels have been assumed to play a role in the physiology of the lesser circulation since the time of William Harvey, but few measurements of the human pulmonary blood volume in vivo are available. This volume is an important variable in the hemodynamics of the pulmonary circulation because of the relationships between volume, pressure, and flow to be expected in an elastic system, which require that all 3 of these parameters be measured in order to interpret changes in any one of them.

In 19 patients with a variety of cardiac and pulmonary abnormalities we have made such measurements in the course of diagnostic catheterization of right and left heart chambers. The dye-dilution method was employed to measure cardiac output and mean transit time from pulmonary artery to left atrium, giving the data needed for calculation of pulmonary blood volume by the Stewart-Hamilton method.

The results provide more direct measurements of pulmonary blood volume than have previously been available, and also give an estimate of relative vascular compliance. One limitation of the method is that it measures the characteristics of the entire pulmonary vascular bed, with no indication of the differences in volume, compliance, and resistance that exist among the arterial, arteriolar, capillary, and venous parts of the bed. In spite of this limitation significant relationships between volume, flow, resistance, and compliance were found.

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Patient Selection and Methods

The patients studied were undergoing diagnostic catheterization of the left heart by the transbronchial or percutaneous dorsal route, and simultaneous right heart catheterization via an antecubital vein. The majority had rheumatic lesions of the mitral or aortic valve; individual diagnoses are listed in Table I. All gave a history of cardiopulmonary symptoms. None showed signs of congestive failure at the time of this study.

Patients were studied after fasting at least 4 hours, and were usually given 10 mg. of morphine sulfate intramuscularly, and in some cases 100 mg. of pentobarbital, 1 hour prior to the procedure. Measurements were made with the patient supine.

A no.-7 cardiac catheter was positioned in the pulmonary artery just beyond the valve by standard methods of intravenous right heart catheterization. The left atrium was catheterized with polyvinyl (I.D. 0.51 mm.) or polyethylene (I.D. 0.58 mm.) tubing passed through the puncture needle. Pressures were recorded by Statham P23-D or P23-A manometers* and a model DR-8 recorder.† Mean pressures were measured at end-expiration by electronic integration. The zero reference level for pressure measurement was a plane parallel to the top of the catheterization table, at a distance above it equal to two thirds the sagittal diameter of the thorax at the sternal angle.

Dilution curves from the brachial or femoral artery following injection of indocyanine green ("Cardiogreen")‡ were recorded by continuous sampling through a cuvette densitometer (Model 103).§ The methods used for injection, sampling, recording, and calibrating have been described in detail elsewhere.⁸

When the catheters were in place and control blood samples were drawn, a dye injection was made into the left atrium and the arterial dilution curve was recorded. As soon as the primary curve was recorded, the sampling syringe was replaced, a new control blood sample was taken, and a pulmonary artery injection was made, with less than 5 minutes intervening between the 2 injections. Since the dye leaves the circulation rapidly, the

*Statham Instruments, Inc., Los Angeles, Calif.
†Electronics for Medicine, Inc., White Plains, N. Y.
‡Hynson-Westcott Company, Baltimore, Md.
§Colson Company, Elyria, Ohio.
## Summary of Results

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<td>3.24</td>
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*Indicates that left heart catheterization was by the percutaneous dorsal route. The transbronchial route was used in all other cases.

MS=mitral stenosis. MI=mitral insufficiency. AS=aortic stenosis. AI=aortic insufficiency. My=myocardial disease, etiology undetermined, probably secondary to coronary atherosclerosis. PPH=primary pulmonary hypertension. PA=pulmonary artery. LA=left atrium. Blood volumes by Stewart-Hamilton method: \( V_{pa-a} \)=pulmonary-left heart arterial volume; \( V_{ih-a} \)=left heart-arterial volume; \( V_{pa-ah} \)=pulmonary blood volume. PVR=pulmonary vascular resistance. C=relative pulmonary vascular compliance (see text). \( C_h \)=pulmonary arterial compliance estimated by the method of Engleberg and duBois.²
control sample for the second-curve baseline was drawn immediately before the second injection. In a few cases the order of the injections was reversed. The total amount of blood withdrawn during the procedure varied between 50 and 100 ml.

Hematocrit levels were measured in Wintrobe tubes centrifuged for 30 minutes at 4,000 r.p.m. at 12-cm. mean radius. No correction was applied for trapped plasma. All blood volumes were calculated from plasma values and the arterial hematocrit values.

Cardiac output and mean transit time (MTT) were calculated from the dilution curves as described by Hamilton, Moore, Kinsman, and Spurling.2

The cardiac output calculated from left atrial injections was consistently higher than that calculated from pulmonary artery injection by a small but significant amount (mean difference 0.390 L min per minute, or 7.5 per cent of the output calculated from the pulmonary artery injection curve). This systematic difference has not been explained. The outputs from pulmonary artery injection were used in the present calculations.

The volume of the sampling system from needle tip to mid-cuvette was 0.35 ml., and the sampling rate was 0.6 ml per second. The distortion of the arterial concentration curve by this system prolongs mean transit time by 0.6 second,7 and this has been subtracted from the observed mean transit times. Since this prolongation affects the curves from both injection sites equally, the difference between their mean transit times, which represents pulmonary mean transit time, is not affected.

Dilution volume, or the volume of blood between sites of dye injection and sampling and all temporally equidistant points, was calculated by multiplying cardiac output per second by the appropriate mean transit time in seconds.2 This volume has been given a variety of names in the past ("Q," "Stewart-Hamilton volume," "needle-to-needle volume," "central volume," etc.), but the term "dilution volume" has the advantages of descriptive accuracy and freedom from conflicting previous usage.

The following symbols are used:

\[ Q = \text{Cardiac output, in milliliters per second} \]
\[ MTT = \text{Mean transit time, in seconds} \]
\[ V = \text{Dilution volume, in milliliters} \]

Subscripts following MTT or V indicate injection and sampling sites in that order:

\[ PA = \text{Pulmonary artery} \]
\[ LA = \text{Left atrium} \]
\[ A = \text{Brachial or femoral artery} \]

Thus:

\[ \text{Pulmonary blood volume} = V_{pa-la} = (V_{pa-a}) - (V_{la-a}) = (Q)(MTT_{pa-a}) - (Q)(MTT_{la-a}) \]

Pulmonary vascular resistance (PVR) was calculated by dividing cardiac output in liters per minute into the pressure gradient from pulmonary artery to left atrium in millimeters of mercury:

\[ PVR = \frac{P_{pa} - P_{la}}{60 Q} \]

The resulting expression gives PVR in resistance units (R.U.), which must be multiplied by 80.0 to convert to dyne sec per cm.5 The use of mean rather than instantaneous pressures and flows introduces an error unavoidable with present techniques, but the work of Engleberg and du Bois3 suggests that the error may not be large.

Mean intravascular pressure in the pulmonary vascular bed \( \bar{P}_{i} \) was estimated by:

\[ \bar{P}_{i} = \frac{P_{pa} + P_{la}}{2} \]

This formula gives a rough estimate of the pressures acting to distend the pulmonary vascular bed, and would approximate mean transmural pressure if extravascular forces (intraalveolar pressure, tension in supporting structures) were negligible. Recent work by Riley, Permutt, Howell, and Proctor8 suggests, however, that the extravascular forces may be of considerable magnitude. Pressure varies over the length of the pulmonary vessels, and \( P_{i} \) clearly underestimates the prearteriolar, and overestimates the postarteriolar pressures. Since our volume measurements include the entire pulmonary vasculature, however, it seems reasonable to use a mean intravascular pressure for the entire bed.

Relative Compliance (C) of the Pulmonary Vascular Bed

Our measurements represent one point on the pressure-volume curve of the lung vessels, and dividing pulmonary blood volume in milliliters by mean intravascular pressure in millimeters of mercury gives a figure \( C \) related to vascular compliance.

\[ C = \frac{(V_{pa-la})}{\bar{P}_{i}} \]

The conventional measurement of compliance by in vitro organ perfusion experiments requires measurement of the increase in volume per unit increase of pressure \( \Delta V/\Delta P \), but this is not readily applicable to man in vivo. The relative compliance, \( C \), described above will be directly related to the true compliance \( \Delta V/\Delta P \), if: (1) vessel lumen diameter depends only on net transmural pressure and the elasticity of the vessel walls, (2) \( P_{i} \) is an accurate measurement of net transmural pressure, (3) vessel length is constant, so that pulmonary blood volume is an index of average lumen diameter. Thickening of the vessel walls with no change in elasticity, for example, would modify the accuracy of assumption (1). The relation between \( C \) and \( \Delta V/\Delta P \), like that between \( V \) and \( \Delta P \), is probably nonlinear. Moreover, compliance changes in opposite directions in different parts

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of the bed might cancel so as to be undetectable. If these limitations are kept in mind, the relative compliance may prove a useful comparative measurement.

To facilitate comparison of patients, all measurements of flow, volume, resistance, and compliance have been related to body surface area in square meters.

Results and Discussion

The results in each patient are listed in table 1. The mean pulmonary blood volume for this group of 19 patients was 365 ml. per M.², with individual values ranging from 126 to 598 ml. per M.² These measurements, like those of Dock, Kraus, Woodward, Dexter, and Haynes,9 who used a similar technic, are much smaller than previous estimates based on more indirect methods, which placed the pulmonary blood volume at 570 ml. per M.² or more.10-12

The mean value for \( V_{pa-a} \) in our patients was 887 ml. per M.², approximately the same as that reported by other investigators for similar patients13-16 (table 2). Borden, Ebert, and Wells17 reported smaller values, but used median transit times in their calculations, which give slightly smaller volumes under these conditions than the correct values obtained with mean transit time.

\( V_{la-a} \), on the other hand, averaged 522 ml. per M.², considerably larger than the radiologic estimate of 246 ml. per M.² given by Lagerlof et al.10 Since our measurements represent a more direct approach than has been available previously, it seems probable that earlier reports have underestimated \( V_{la-a} \) and overestimated pulmonary blood volume.

Although we have as yet no data on normal subjects, the volume between pulmonary artery and peripheral artery in patients with valvular heart disease, without signs of congestive failure, has been shown to be only slightly larger than that in normal subjects,10,13-17,24 so that the normal values for pulmonary blood volume may not differ widely from those reported here.

Three possible sources of error in the present method should be mentioned. First, since the volume measured includes only blood that contributes to dilution of the dye, incomplete mixing in the left atrium would result in the inclusion of some portion of left atrial volume with pulmonary blood volume, and give falsely large estimates of pulmonary blood volume. It seems certain that atrial mixing is incomplete in some cases, but the magnitude of this error is unknown. Second, the portion of the systemic arterial tree included in \( V_{pa-a} \) and \( V_{la-a} \) is undetermined, and may vary in different patients and under different conditions. It seems safe to assume that this factor remains constant during the few minutes required for the 2 curves and is therefore not relevant to the measurement of \( V_{pa-la} \). Third, the pulmonary blood volume must vary continuously throughout the cardiac and respiratory cycles, and the relation of these variations to the mean volume measured by this method has not been evaluated.

Relative Compliance of the Pulmonary Vascular Bed

The absence of any correlation between the volume of blood in the pulmonary vessels and the pressure acting to distend them \( (P_i) \) suggests that there are real differences in pul-

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<td>Milnor et al.</td>
<td>(this report)</td>
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*Calculated from median transit times (see text).
†Estimated radiologically. All other values in this table were determined by indicator-dilution methods.

PA-A=pulmonary-left heart-arterial volume; LA-A=left heart-arterial volume; PBV=pulmonary blood volume.
The absolute pulmonary vascular compliance in our patients (fig. 1).

The relative pulmonary vascular compliance \( \frac{V_{\text{pa-la}}}{P_{\text{i}}} \) averaged 14.7 ml. per M.\(^2\), per mm. Hg when related to body surface area, or 0.408 ml. per Kg., per mm. Hg when related to body weight. If we assume a finite vascular volume at zero transmural pressure, absolute compliance must be smaller than our values. Engleberg and du Bois\(^3\) found an average compliance of 0.214 ml. per Kg., per mm. Hg for the vascular bed of the isolated rabbit lungs, or about one half our average value. Extrapolation from their estimate of human pulmonary arterial compliance,\(^18\) however, gives a value for the whole pulmonary vasculature that is almost equal to our mean relative compliance. If we assume as a first approximation that normal compliance lies somewhere between our relative compliance values and the in vitro determinations of Engleberg and du Bois\(^3\) e.g., 11.0 ml. per M.\(^2\), per mm. Hg, and assume \( V_{\text{pa-la}} = 350 \text{ ml. per } M.^{2}, \), \( \bar{P}_{i} = 12 \text{ mm. Hg} \), and a linear relationship, the pressure-volume curve for the normal human pulmonary vascular bed would follow the line plotted in figure 1. All points below and to the right of this line would then represent decreased compliance.

Relation between Resistance and Compliance

Since resistance to blood flow through the lung could be controlled principally by changes in vessel diameter at a sharply localized level of the vascular bed such as the small arterioles, large changes in pulmonary vascular resistance could occur with little or no change in total pulmonary blood volume, and with no necessary relation between pulmonary vascular resistance and over-all compliance.

Our data nevertheless show a significant inverse correlation between these 2 variables \( (r = -0.584, \ P < 0.01) \). All but one of the patients with a high pulmonary vascular resistance \( (> 4.0 \text{ R.U. per } M.^{2}) \) had relatively low total compliance \( (< 15.0 \text{ ml. per } M.^{2}, \text{ per mm. Hg}) \), (fig. 2), suggesting that a generalized decrease in vascular distensibility either accompanies arteriolar narrowing or is itself responsible for the increased pulmonary vascular resistance in these patients.

This supposition is further supported by the finding that pulmonary arterial compliance \( (C_{a}) \) calculated from the pulmonary arterial pulse wave profile, cardiac output, and left atrial pressure by the method of Engleberg and du Bois\(^3\) varies directly with our values for total pulmonary vascular compliance (fig. 2).

It is not possible to conclude whether the decreased compliance is due to vasoconstriction or to alteration in the structural elements of the vessel walls, both of which have been demonstrated in mitral stenosis.

The possibility that elevated \( P_{ia} \) may invoke decreased over-all compliance independently of arteriolar effects is raised by 2 pa-
PULMONARY BLOOD VOLUME IN MAN

Figure 2
Ordinate. Pulmonary arterial compliance, "C_a", estimated by the method of Englberg and du Bois.8 Abscissa. Relative compliance of the pulmonary vascular bed as a whole "C" (see text). On both axes compliance is expressed in ml. per M.2, per mm Hg. Some patients as listed in table 1, less 3 cases whose "C_a" could not be calculated because adequate tracings of pulmonary arterial pressure pulses were not available.

Relation between Flow and Volume
A direct correlation between cardiac output and pulmonary blood volume has been described by many investigators.14, 15, 19, 20-22 A fall in pulmonary-left heart-arterial volume when cardiac output decreases under the influence of anesthetics, as described by Johnson20 and by Etsten and Li23 is consistent with this relationship, as is Ball, Kopelman, and Witham's finding that the "intrathoracic blood volume" increases with exercise.24 In most studies in vivo14, 15, 20 conclusions were based on measurement of pulmonary artery-to-peripheral artery volume, and the part played by left heart and arterial volume was uncertain.

Johnson,20 Sjostrand,21 and Warren and Weissler,25 using radiologic, plethysmographic, and indicator-dilution techniques, found that pulmonary blood volume and heart volume were both correlated with cardiac output, but that the correlation with stroke volume was even closer than that with minute output.

Our data lead to the same conclusion, and show that the previously reported relationships were not due to variations in left heart volume alone. There was a significant positive correlation between pulmonary blood volume and cardiac index (r = +0.512, p < .01) and a closer correlation between pulmonary blood volume and stroke volume (r = +0.735, p < .001) (fig. 3).

The significance of this correlation can be questioned on the ground that cardiac output enters into the calculation of pulmonary blood volume.14 As pointed out by Snedecor,26 and by Rapaport and his colleagues,15 however, correlation in such cases is not necessarily artificial. Analysis of the relation between MTT and cardiac index leads to the same conclusion, and the improved correlation when heart rate is introduced (in the calculation of stroke volume) suggests that the relationship is a real one.

The reason for this relationship is not clear. It would be expected in a simple elastic system of constant compliance and zero outflow pressure, where flow would be directly pro-
portional to perfusing pressure and volume directly proportional to pressure. This simple
analogy cannot apply to our patients because compliance varied so widely in different sub-
jects, and there was no significant relation between $P_1$ and cardiac index.
A hypothesis consistent with our results is that the volume and distensibility of the pul-
monary vascular bed directly influence the rate and extent of left atrial filling, and thus
contribute to the regulation of left ventricular output. This concept is proposed by
Sjostrand and is closely related to Guyton’s view that “mean circulatory pressure,” which
is determined in part by the volume and distensibility of the vascular system, is an im-
portant factor in regulating cardiac output.

Summary

Pulmonary blood volume was measured in 19 patients, with use of a dye-dilution method
and simultaneous bilateral cardiac catheterization. Pulmonary mean transit time was
measured by subtracting left atrial-to-peripheral artery mean transit time from pulmonary
artery-to-peripheral artery mean transit time.

Average pulmonary blood volume was 365 ml per M.² (range 126 to 598 ml per M.²).
This value is significantly smaller than previous estimates by less direct methods.

The ratio of pulmonary blood volume to mean intravascular pressure was used as an indica-
tion of pulmonary vascular compliance. Patients with high pulmonary vascular resis-
tance had relatively low pulmonary vascular compliance.

A significant correlation between pulmonary blood volume and stroke volume was found,
lending support to the hypothesis that the volume and elasticity of the pulmonary vas-
cular bed contribute to the regulation of cardiac output.

Acknowledgment

We acknowledge gratefully the cooperation of Drs. Henry Bahnsen and Frank Spencer, who performed
the left heart catheterizations, and the technical assistance of Miss Mary Lou Sparger and Miss Mary
Ellen Horner.

MILNOR, JOSE, McGAFF

Summario in Interlingua

Le volumen del sanguine pulmone esseva mesurato in 19 patientes per medio de un metodo a dilution
de colorantes e le effectuation simultanee de catheterismo cardiace bilateral. Valores medie pro le tem-
pore de transit pulmonar esseva obtenite per sub-
traher le valores medie del tempore de transit ab le atri sinistre ad le arteria peripheric ab le valores
medie del tempore de transit ab le arteria pulmonar
ad le arteria peripheric.
Le valor medie del volumen de sanguine pulmone esseva 365 ml per m² de superficie corporee (con un
minimo de 126 e un maximo de 598 ml). Iste valor
es significatamente plus basse que illo previemente
estimate per medio de minus directe methodos.

Le proportion de volumen de sanguine pulmonar
tension intravascular medie esseva usate como indi-
cation del resilientia pulmone-vascular. Patientes con
alte resistantia pulmone-vascular habeva relativamente
tempera resilientia pulmone-vascular.

Esse constatate un correlation significative inter
le volumen del sanguine pulmone e le volumen per
pulso. Isto supporta le hypothese que le volumen e le
elasticitate del vasculatura pulmone contribue ai
regulation del rendimento cardiace.

References

1. Harvey, W.: Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus. Translated

lation. IV. Further analysis of the injection method, and of changes in hemodynamics under


4. Allison, P. R., and Linden, R. J.: The bronchoco-
scopic measurement of left atrial pressure. Cir-
culation 7: 669, 1953.


6. Jose, A. D., McGaff, C. J., and Milnor, W. R.: The diagnostic use of dye dilution curves dur-


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