hemodynamic and metabolic changes also facilitates lactate and coronary blood flow studies.

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**References**


**Pressure-Radius Relationships**

To the Editor:

Although Jarmakani et al. ("In vivo pressure-radius relationships of the pulmonary artery in children with congenital heart disease," Circulation 43: 585, 1971) used methods similar to ours in measuring the pulmonary arteries, they did not demonstrate the increased distensibility of these vessels that we found in a high-flow, normal-pressure situation—atrial septal defect.

The distensibility characteristics of the pulmonary artery are represented by its elastic diagram,2 with the initial low slope and final steep slope. As with all arteries, pulmonary vessels become stiffer the more they are stretched. At systemic pressures arteries operate on the steep slope but the pulmonary arteries are unique since their normally low pressures mean that systole and diastole fall on the initial low slope. This initial portion of the elastic diagram can vary. With age there is an increased slope and increased stiffness, and with poststenotic dilatation it shows decreased slope and decreased stiffness.

Due to the formulae used to analyze their measurements, Jarmakani et al. appear to have missed the altered distensibility in high-flow situations. They first calculated a percent change in radius (ΔR) divided by mean radius (R). With this formula, an abnormal vessel, enlarged due to increased distensibility, will have both an increased R plus an increased ΔR and thence a near "normal" percent change in radius. The PCR can also appear normal with an elevated pressure giving an increased R but, as was seen in three of their groups, accompanied by a widened pulse pressure producing an increased ΔR.

A more meaningful index of vessel distensibility is the percent change in wall strain (ΔR/R0 where R0 = radius at zero wall tension). As part of our paper, I incorrectly mentioned the "percent change about the mean radius" when actually reporting percent change about the mean strain. The differences were quite apparent between normal pulmonary vessels and vessels in atrial septal defect (±4.9% versus ±12.9%, respectively).

Secondly, the elastic properties of a material are correctly represented by the slope of the elastic diagram, the Young’s modulus (E):

\[
\text{stress} = \frac{\text{wall tension/unit thickness}}{\text{strain}} = \frac{\text{radius change/resting radius}}{
\]

Jarmakani et al. used the nonstandard “pressure-strain” elastic modulus (Ep = ΔP × R/ΔR). With this formula a vessel with increased distensibility, with its accompanying increased mean radius and increased ΔR can have a normal Ep when, in fact, the Young’s modulus will be decreased. At high pressures the increased stiffness of the wall will be shown by Ep but may be inaccurate since the elastic diagram is nonlinear. Resting length is an essential part of any elastic modulus.

A quick check on the group differences could be made by calculating ΔT/ΔR, where wall tension (T) = P × R. These values would represent the slope of the elastic diagram between systole and diastole and will be low for low pressures and high for high pressures but again only valid where the curve is essentially linear. Lower than normal values, indicating increased distensibility should be present in the patients with atrial septal defect and low-pressure ventricular septal defects.

The authors’ measurements were made with considerable accuracy and illustrate how much information can be obtained from the in vivo situation. I hope that they can reanalyze them using a calculated R0 rather than R, at least in the region where R0 can be obtained.

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References

The authors reply:
To the Editor:
I would like to thank Dr. Boughner for his comments on our work. There are two main differences in data acquisition and analysis between our methods and those used by Drs. Boughner and Roach:
(1) The radius measurements in our patients were obtained at the middle of the right pulmonary artery (PA) only using the AP projection (approximately perpendicular to the right PA). However, Boughner and Roach obtained their data from the main, the 2nd, or 3rd branch of the PA in different patients, and varied the projection using either "AP, left, or right anterior oblique." It is very important to obtain the systolic and diastolic radius measurements in a relatively cylindrical vessel and at the same point in order to have reproducible and meaningful data. In our experience, this has not been possible in the other branches of the PA. Furthermore, the "jet" in patients with pulmonary stenosis (PS) may produce local dilating effects in the main and left PA. The percentage change in radius in our group was ± 7.5% about the mean and is similar to Patel's data in the dog (±7.8%) and Greenfield's data in humans (±8%), while it was only ±4.9% in Drs. Boughner's and Roach's data. The percent change in their patients with pulmonary stenosis was ±12.1% and with atrial septal defect ±12.9%; both values are higher than our values of ±7% in ASD and ±4.5% in PS. These differences could be due to their use of the main PA rather than the right PA.
(2) The second and most important difference centers on the validity of extrapolating $R_0$ (radius at zero wall tension). Obviously, it would be ideal to measure $R_0$, however, this is not possible in an in vivo preparation, and $R_0$ must be obtained by extrapolation of a nonlinear relationship. Furthermore, as has been discussed at length by Patel et al. (Circ Res 27: 149, 1970), D. H. Bergel (Ph.D. Thesis, University of London, 1960), and Peterson et al. (Circ Res 8: 622, 1960), the stress strain curve is curvilinear and the elastic properties of the vessel must be evaluated only on that segment of the curve where the measurements are made. Thus we have followed the lead of these investigators and chosen to present only data obtained by actual measurement.

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