LETTERS TO THE EDITOR

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Calculating Pulmonary Valve Area

To the Editor:

The article entitled "Comparison of the hemodynamic effects of exercise and isoproterenol infusion in patients with pulmonary valvar stenosis" (Circulation 48: 948, 1974) has emphasized an important point: It is, indeed, well known that the effects of physical exercise and isoproterenol upon cardiac output are not comparable.

However, the authors have utilized a modification of the original Gorlin formula1 for calculation of valve area. The modification2 disregards changes in systolic ejection. Their data suggest that valve areas calculated at rest and during exercise are comparable, but that the valve area calculation during isoproterenol infusion is significantly smaller.

They have discounted the significance of subvalvular pulmonic stenosis by pointing out that "Although in some patients the infundibular area narrowed during systole, it appeared normal during diastole so that fixed infundibular stenosis was not considered to be present." Infundibular stenosis would presumably be related to hypertrophy of right ventricular musculature. It would, therefore, be a dynamic event manifest during systole. It cannot, therefore, be ignored when calculating a gradient across the outflow tract of the right ventricle. It may very well be that the isoproterenol infusion was a greater stimulus to right ventricular contractility in these patients and that the obstruction to outflow across the right ventricular outflow tract was therefore more severe.

I have now reviewed the complete original data kindly sent by Doctors Neal, Lucas, Rao, and Moller. They have accumulated data sufficient to use the Gorlin formula in 16 of their 28 patients. Utilizing these data, and using only the assumption that right ventricular systolic pressure is the equivalent of the pulmonary valve gradient in these patients, calculations yield a valve index of 0.4 cm²/m2 at rest, 0.5 during exercise, and 0.5 during isoproterenol stress. These calculations directly conflict with the results utilizing the formula of Moller and Adams.2

In conclusion then, the data calculations for pulmonary valve area in this study appeared to be greatly dependent upon the assumptions used, and the formula applied. In addition, the contribution of muscular subpulmonic stenosis to the measurement of a right ventricular outflow gradient must be considered more critically. The authors have stated correctly that "It is unlikely that the orifice of the pulmonary valve changes in size during exercise or isoproterenol infusion. The difference between the exercise and isoproterenol states is most likely related to the effect of the pharmacological agent on the infundibulum." They have not, however, discussed the difficulty, with severe stenosis and consequent low pulmonary arterial pressures, of localizing the obstruction to the pulmonary valve or right ventricular outflow tract. The use of an electrode catheter, enabling the recording of a right ventricular electrocar-diographic signal as soon as the catheter is within this chamber has been found to be helpful.

STEVEN WOLFSON, M.D.
Yale University
New Haven, Connecticut 06510

References

1. GORLIN R, GORLIN SG: Hydraulic formula for calculation of the area of the stenotic mitral valve; other cardiac valves, and central circulatory shunts. Am Heart J 41: 1, 1951

The authors reply:

To the Editor:

Our calculations of Pulmonary Valve Area (PVA) were based on a modification of Gorlin's formula,1 which indeed disregards changes in systolic ejection period (SEP). As stated in the results section and summarized in table 1, there is not a significant difference in SEP during the three physiologic states tested: rest, exercise, and isoproterenol infusion. Thus, the observed decrease in PVA during isoproterenol infusion cannot be accounted for by alterations in SEP.

Application of the Gorlin formula in 16 of our 28 patients in whom SEP was measured may well yield results dissimilar to those obtained by applying the formula of Moller and Adams to all 28 patients. The validity of selecting slightly more than half of the patients studied, applying a different formula to measure PVA, rounding results to the nearest tenth vs. one-hundredth, and using these results as a basis of comparison is highly questionable.

Finally, Dr. Wolfson raises a good point with regard to his concern that calculated PVA is a reflection of total gradient during systole, including that which is secondary to infundibular narrowing. The implication is that some of the observed decrease in calculated PVA during isoproterenol infusion may be due to infundibular stenosis. We observed no correlation between the cases with systolic outflow tract narrowing angiographically and those patients who had the greatest decrease in PVA.

WILLIAM A. NEAL, M.D.
RUSSELL V. LUCAS, JR., M.D.
S. RAO, M.D.
JAMES H. MOLLER, M.D.
West Virginia University
Morgantown, West Virginia 26506

Reference

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S Wolfson

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