LETTERS TO THE EDITOR

Comments on "Pulmonary Vascular Impedance Analysis of Adaptation to Chronically Elevated Blood Flow in the Awake Dog"

In a recent report Hopkins and colleagues (1979) determined pulmonary vascular impedance in dogs with chronic arteriovenous fistulae and consequent high pulmonary vascular flow. They reported marked reduction in pulmonary vascular impedance (which was proportional to the increase in pulmonary flow) and interpreted results to suggest decreased elastic moduli of the proximal pulmonary arteries as a result of sustained high blood flow. These findings are important and the clinical implications are obvious. It is possible however to explain these findings, not on the basis of altered elastic moduli, but on a Venturi effect at the point of pressure measurement in the pulmonary artery distal to the cuff-type electromagnetic flow transducer.

There has been controversy as to whether impedance should be determined by relating pulsatile flow to lateral pressure (side-on to flow) or to impact pressure (end-on to flow) in an artery. As advised for determination of vascular resistance (Burton, 1965), M.G. Taylor and the writer have used impact pressure so that the Venturi effect (conversion of pressure energy to kinetic energy at high flow rates) is not overlooked in impedance determination (O'Rourke and Taylor, 1967). The question is of academic interest only in most studies where flow velocities are low, kinetic energy is only a tiny component of total energy, and when there is little difference between impact and lateral pressure waves (O'Rourke, 1968).

Hopkins and colleagues measured pressure side-on to the direction of flow, downstream from the cuff-type electromagnetic flow transducer, through a size 7F catheter which traversed the transducer, in animals with high peak flow velocity. In similar circumstances I have found marked reduction in lateral pressure in animals with high peak flow velocity. In similar circumstances I have found marked reduction in lateral pressure waves when anterior flow was similar to that published by Hopkins et al., showing marked reduction in modulus and more negative phase at low frequencies when the catheter was applied. These findings were attributable to narrowing of the artery by the cuff-type flow transducer (O'Rourke, 1968).

While excluding kinetic energy in impedance determination, Hopkins et al. did attempt to calculate this separately as a component of external heart work. Their calculation of a 12-fold increase may have been an underestimation since this demands very accurate measurement of internal arterial diameter including allowance for the space occupied by the catheter, together with assumption of a flat velocity profile downstream from the flow transducer. Since kinetic energy depends on velocity squared, and velocity depends on diameter squared, small errors in measurement of diameter or slight deviations from a flat profile can cause large errors in calculation of kinetic energy (O'Rourke, 1968).

The subject addressed by Hopkins and colleagues is important, and their conclusions are challenging. Before accepting their suggestion that chronically increased pulmonary flow alters pulmonary artery elasticity, it would be desirable to ensure that similar results could be obtained either with a catheter-tip method for measuring flow (when peak flow velocities would be lower) or with pressure measured end-on to flow beyond a cuff-type flow transducer.

A separate question might be asked of the authors. Findings are attributed to the effects of chronically increased flow, and near normalisation was observed 2 weeks after the arteriovenous shunt was closed. Did they not have the opportunity to check the effects of acute shunt occlusion by compressing the femoral artery proximal to the shunt at the time experimental data were taken?

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Circ Res. 1980;46:731-732
doi: 10.1161/01.RES.46.5.731

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