Aortic-to-Radial Arterial Pressure Gradient after Bypass

To the Editor.—The interesting paper by Pauca et al.\(^1\) correctly identifies the source of the aortic-to-radial pressure gradient following cardiopulmonary bypass as a decrease in “hand vascular resistance.” I would like to propose an hypothesis for this as yet incompletely understood decrease.

Most centers subject cardiac surgical patients to hemodilution during bypass to a hematocrit around 20%. This decreased hematocrit reduces blood viscosity to approximately one half of normal.\(^2,3\) Such viscosity reduction should decrease arteriolar resistance to approximately one half of normal, since resistance is a linear function of viscosity, according to the Poiseuille equation. A decrease in systemic vascular resistance following bypass has indeed been reported in patients with a normal ventricle during coronary surgery;\(^2\) this was compensated for by increased cardiac output.\(^5,6\) This reduction of resistance takes place without vasodilatation, as was shown long ago by Gordon et al.\(^4\) Due to the peculiar physical characteristics of blood, such reduction could be even more marked at the microcirculatory level.\(^8\)

The difference between “peripheral” and “systemic” vascular resistance in the arteries normally is negligible, since the peripheral arteriolar resistance is much larger than the proximal arterial resistance. However, from the physical point of view, a long tube such as the brachial and radial artery does necessarily present a resistance, however small, to blood flow. Thus, systemic vascular resistance should be understood as the sum of two resistances in series (fig. 1), one proximal and one distal. Systemic vascular resistance takes into account both the proximal and distal components and determines the mean aortic pressure; peripheral vascular resistance, equal to the distal component, considers only arterioles and capillaries and determines the mean distal arterial pressure (fig. 1). If the distal resistance decreases markedly and the proximal component increases, as seems to be the case in these patients, a gradient appears between aortic and distal pressures.

Reduced peripheral vascular resistance downstream from the radial cannula tends to decrease the pressure drop across arteries and capillaries, as the pressure difference is a direct function of resistance. A decreased radial arterial pressure ensues. Inasmuch as the arteries are elastic tubes, a reduction in transmural pressure should result in a decrease of the arterial diameter. According to Poiseuille, a smaller arterial diameter would increase the proximal resistance. Therefore, the normally very small decrease in mean pressure between aorta and radial artery becomes more marked. Thus, purely passive hydraulic factors secondary to hemodilution would explain the decrease in peripheral resistance and the increase in “proximal” arterial resistance underlying the aortic-to-radial pressure drop.

It has been suggested that the difference between aortic and radial pressures would invalidate all previous work describing hemodynamics immediately after cardiopulmonary bypass, since this work relied on radial artery rather than aortic pressure measurement. This objection may be justified if, in order to calculate afterload or ventricular work, aortic pressure was extrapolated from radial artery measurements. However, for estimation of peripheral resistance and of perfusion pressure, the correct value is not the aortic but the distal arterial pressure, as this is the pressure that most tissues (except, of course, the heart) effectively “see.”

Jorge Urzua, M.D.
Professor of Anesthesiology and Engineering
Catholic University of Chile
P.O. Box 114-D
Santiago de Chile

REFERENCES