EDITORIAL

Some Hemodynamic Problems in Transposition of the
Great Vessels

SINCE the incidence of transposition of the great vessels is approximately one in 20,000 live births, and since many of the infants affected have short lives, the prevalence of the condition is not such that the practitioner will see many cases. Still, the interest of the physician in these patients should transcend the pragmatic aspects of the minor public-health problem presented by care of the individual patient thus afflicted. The reason for this statement is that locked within the mechanism of survival may be possible clues leading to solution of control of the pulmonary and systemic circulations. Attention of the reader is drawn to two communications pertaining to transposition of the great vessels in this issue of Circulation: one, by Ferencz, describes the anatomic changes in the pulmonary vasculature; the other, by Rahimtoola and associates, presents a method of obtaining pressure and oxygen saturation of the blood in the pulmonary artery.

The extent of the vascular obstructive change reported by Ferencz is remarkable indeed, particularly among members of the very young age group. The change is such that it might well engender concern on the part of the surgeon and physician from the points of view of the optimal time for operation and the degree to which hopes for a low operative mortality rate and successful rehabilitation, can be regarded as realistic.

In many instances of transposition of these vessels a transvenous catheter cannot be manipulated into the pulmonary artery. At such a time adaptation of Radner’s procedure for pulmonary arterial puncture, as described by Rahimtoola and associates, should prove most helpful in measurement of pulmonary arterial pressure and flow which are crucial data in appraisal of the extent of any pulmonary vascular obstruction.

The hemodynamic aberration when the great vessels are transposed and two functioning ventricles are present, particularly when the ventricular septum is intact, has not been freed from wild conjecture. The diagrams that are made for the purpose of demonstrating the pattern of the fetal and neonatal circulations must be regarded as gross approximations of the true situation.

When the two circulations are nearly separate, with the ventricular septum intact (parallel circulations replacing a serial circulation), one hypothesis states that the volume flows are independent. It would be presumed that the “drive” for an increased cardiac output is triggered by the oxygen requirement of tissues; with near-separation of the circuits, the left (pulmonary) ventricular output passively follows that drive, the volume circulated being related to ventricular performance and the total volume within the circuit.

It is trite to say that if life is to continue, some quantity of reduced hemoglobin must enter the pulmonary circulation and that some quantity of oxygenated hemoglobin must return to the systemic circuit. The minimal extent of such transfer is readily calculable. The volume of blood required for the transfer often is conjectural because of lack of knowledge of the locus of the shunts. Dynamic, alternating phasic “bleeding” of one circuit into the other, which would make calculations even more hazardous, is a possibility. Is it possible, as well, that volume equilibrium in some cases can be attained only by approximation of pulmonary and systemic pressures? Could bronchial flow, with its blood at low oxygen pressure, facilitated by acidosis, affect the locus of the resistance vessels in the lung? Whatever bronchial flow may occur has importance beyond the factor of volume alone because of the low oxygen saturation of the blood in that flow, which might be able to take up 10 times the oxygen that the same volume of highly satu-
rated blood in the main trunk of the pulmonary artery could do. Thus, in the presence of an intact ventricular septum, the high oxygen saturation of blood in the pulmonary artery, if used to determine pulmonary flow by the Fick formula, may give an erroneously high value.

Ferencz's report indicates that obstructive vascular changes occur in patients who have an intact ventricular septum. Rahimtoola's measurements of pressure in the pulmonary artery are lower than those in the systemic artery in his group of patients with intact septa. In the institution with which I am associated, the surgical successes achieved in intra-atrial reconstruction for transposition of the great vessels, with closure of a ventricular defect, if one is present, indicate that in many instances the pulmonary vascular obstruction is not severe and fixed, particularly in children more than a year old (Abstr. Circulation 32: II-113, 1965). In all the cases of Rahimtoola and associates the pulmonary arterial blood was sufficiently unsaturated to be a significant recipient of oxygen, and the oxygen saturation in the blood in the pulmonary artery was highest when an intact ventricular septum and pulmonary stenosis were present.

Additional questions that arise are (1) whether the arteriovenous shunts claimed to be present by Recavarren in the preceding editorial could be present and function in cases other than those of high-altitude pulmonary hypertension and (2) whether the changes in the pulmonary vessels in "dead cases" of transposition of the great vessels are in any way representative of the status of the vessels in surviving cases.

It is evident that additional data on pulmonary flow and pressure, the size of the left (pulmonary) ventricle, pulmonary artery and bronchial arteries, and pulmonary-vein-to-systemic-vein anastomoses will be needed in the solution of the present conundrums posed by those cases in which transposition of the great vessels is complicated by the presence of two nearly separated "parallel" circulations.

This editorial comment pertains only to the hemodynamic problems in the afore-mentioned anatomic situations. The two papers referred to in this issue of Circulation have better identified the problems and have made contributions toward their solution, and will be stimuli to further investigations.

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Controversy is harmful not only when it results in neglect of unfavorable evidence, confusion, and misjudgment; it may cause a certain blindness to the significance of facts which are quite evident and which invite further study. It is as if a contestant paid attention only to those features of experience which, at the moment, serve a polemical purpose.—WALTER BRADFORD CANNON: The Way of an Investigator: A Scientist's Experiences in Medical Research. New York, W. W. Norton & Company, Inc., 1945, p. 103.
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