Editorial
Dependent and Obligatory Intracardiac Shunts
In Congenital Heart Disease

This issue of Circulation contains a special article by Dr. Abraham Rudolph, the substance of which is the Conner Lecture, given in 1969 at the Annual Scientific Sessions of the American Heart Association, and entitled “The Changes in the Circulation after Birth: Their Importance in Congenital Heart Disease.” The newer knowledge of how the fetal circulation is altered at birth is ably reviewed, and the fresh leads generated from recent investigations as to why they occur, are discussed. Among the many hemodynamic alterations based on structural change elucidated, are the mechanisms of the closure of the ductus arteriosus and the decreased vascular resistance in the lungs when they are aerated. An underlying theme chosen for discussion is that congenital cardiac defects are not fixed anatomic entities, but rather are a continuing adaptation of form to function and of function to form, which continues after birth, albeit, if the changes were charted on the same time scale as the fetal changes, the curve would be flattened and ultimately a near plateau.

In the hope of increasing the number of students (of all ages) reading Dr. Rudolph’s review, I have chosen to draw attention to the one item wherein new nomenclature is introduced,—namely, a classification of shunts as “dependent” or “obligatory.” The meaning of the latter term is perhaps difficult to grasp and, in concept, is not restricted to shunting between the two (systemic and pulmonary) circulations. For example, an arteriovenous fistula has an obligatory shunt through it, with the tissue flow being maintained at a normal level if the central pump has adequate capacity. The concept could be expanded to the hemodynamic state in mitral insufficiency, wherein an obligatory regurgitant flow is always present with forward flow being maintained at a level related to overall left ventricular function.

Progress in the understanding of complex biologic phenomena is often marked by the introduction of new language. This is mediated not so much by the novelty of the basic idea, as by the increased attention directed to the biologic problem and the spur to attempt interpretation of the semantics. Subspecialty jargon whets the interest of the “in” or “with it” group and if, and when, understood, facilitates and accelerates communication. With this preamble, can one predict the effects of Dr. Rudolph’s classification of intracardiac shunts as dependent or obligatory? As he is an eminent and articulate authority in the field, the first prediction will be that the classification may catch on and the terms may become part of the language of our subspecialty.

As the adjectives dependent and obligatory do not express an exact meaning, anyone using these words to communicate will need to study Dr. Rudolph’s definitions;—otherwise confusion rather than clarification may ensue. It is self-evident that all shunts are dependent upon gradients, as modified by directional kinetics, and some shunts are obligatory for continued life as those between the two circulations in transposition of the great vessels with nearly intact cardiac septa.

If common antonyms were used, one might wonder how confusing the words “obligative” and “facultative” might be, in the sense of the compulsive and the permissive. Such words might send readers scurrying for their dictionaries. As I interpret Dr. Rudolph, he is telling us that some shunts are nicely dependent on the vascular resistance, usually with the pulmonary vascular bed in mind,
while others are immutably associated with a defect across which there will always be an obligatory shunt, or, in other words, abolition of the gradient across such an intracardiac defect would be incompatible with life. Examples of obligatory shunts are a ventricular septal defect opening into the right atrium, an aortic sinus aneurysm rupturing into the right side of the heart, and an aortic "run-off" through a patent ductus with an incompetent pulmonary semilunar valve into the right ventricle. The category of obligatory shunts could be subdivided into those dynamically dependent, e.g., VSD into the right atrium, and those anatomically dependent, e.g., anomalous pulmonary vein to superior vena cava, which illustrate the complexity of the classification.

A body of teaching has held a unitary concept that shunts can be related to pressure gradients, with flows proportioned to the relative pressures at the egress points, that is, the pressure that the pumping chamber "looks at" at the various orifices. In this approach, the clear difference between Dr. Rudolph's categories becomes somewhat smudged, but his stimulating reappraisal of shunt mechanisms does not change such current teaching or conflict with it.

There is the fundamental axiom that, when equivalent pressures related to a large ventricular septal defect are present, the pulmonary vascular resistance determines the left-to-right shunt, and increased resistance is obligatory, as Dr. Rudolph points out. (This axiom conflicts with the Paul Wood classification of pulmonary hypertension which implies that pulmonary flow per se could result in pulmonary hypertension.) As a wit-teaser, an important corollary has been long evident; the pulmonary vasculature may help "set" the systemic pressure as well as the pulmonary pressure.

Bidirectional shunting related to phases on the cardiac cycle should not be ignored when an orifice unguarded by a valve is present. It is this possibility of diastolic shunting that makes the A-V commune defect somewhat less attractive as an example of simple obligatory shunting than the others listed by Dr. Rudolph.

With an obligatory left-to-right shunt, it is clear that pulmonary vascular changes cannot be related to high pressure, and an inferred causal relation to flow would be tenuous; however, with failure of the left ventricular pump and a buildup of its filling pressure, pulmonary venous hypertension may lead to increasing pulmonary arteriolar, reactive and organic, resistance.

What are the inherent values of the classification of dependent and obligatory shunting for the understanding of clinical pulmonary hypertension and for design of experiments in lower animals to elucidate the mechanisms of acquired pulmonary vascular disease? The answer is in doubt. However, this editorial will have served its purpose if scanners or readers of this issue of Circulation, either as clinicians interested in congenital heart disease or as curious followers of Harvey attempting to visualize the circulation of the blood (or both), will study Dr. Rudolph's Conner Lecture.

HOWARD B. BURCHELL

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