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The Heart: Pressure-Propulsion Pump or Organ of Impedance?

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HEMODYNAMIC MONITORING and support of circulation are at the center of acute intervention-based specialties such as anesthesiology and critical care. In spite of the general assumption that the understanding of basic and clinical hemodynamics is relatively complete, clinicians often invoke a number of reasons to explain away the discrepancies between the commonly used mental model of circulation and various pathophysiologic states. A cursory review of the literature on treatment modalities of various hemodynamic states over the past several decades suggests that this mental model has undergone a steady revision. For example, contrary to expectations, the results of a 2012 intra-aortic balloon pump (IABP)-Shock II randomized, open-label multicenter trial found no difference in 30-day mortality (40%) in patients with acute myocardial infarction associated with cardiogenic shock and treated with combined pharmacologic therapy, percutaneous intervention and IABP, or with pharmacologic therapy and percutaneous intervention only.[1] Results of the recently published follow-up study confirmed the original outcomes.[2] On the basis of previously reported meta-analyses and conflicting evidence from data registries, joint American College of Cardiology and American Heart Association, together with the European Society of Cardiology, downgraded the class of recommendation for IABP use from class IB (should be used) to IIbB (may/can be used).[3]

In the wake of these findings, some have questioned the recommendations of potentially harmful adjunct therapies, namely, the use of intra-aortic balloon pumps, in this high-risk group of patients based on "pathophysiologic assumptions and expert opinions" rather than on randomized clinical trials.[4] Moreover, in the editorial to this landmark study, O'Connor and Rogers submitted that "the results of the IABP-SHOCK II trial parallel those from many recent outcome trials that have challenged the understanding of the management of acute and chronic heart failure, including those regarding the use of pulmonary artery catheters and the role of revascularization in ischemic cardiomyopathy."[5]

Similarly, the emerging modalities in pharmacologic therapy of acute and chronic heart failure further question the fundamental understanding of the circulation. Most notable is a shift from the use of potent sympathomimetic amines (epinephrine, isoproterenol, and dopamine) in the 1960s and 1970s,[6,7] to a widespread use of vasodilators (dobutamine and milrinone). On the contrary, the use of inotropes (dobutamine and milrinone) currently is reserved for the treatment of a minority of patients with severe systolic dysfunction who do not tolerate vasodilators due to hypotension.[8] Data from the ADHERE registry showed that fewer than 3% of acute heart failure patients (from a group of 150,000) had a systolic BP of <90 mmHg,[9] and of approximately 14% of those who were treated with inotropes, 19% had higher mortality compared with non-inotrope-treated patients (14%).[10] Practice guidelines of the Heart Failure Society of America (HFSA), the American College of Cardiology Foundation/American Heart Association (ACCF/AHA), as well as the European Society of Cardiology (ESA) therefore recommend the use of vasodilators and deemphasize the use of inotropes in the management of acute heart failure syndromes.[11] It is of note that, from the range of available inotropes, dobutamine and milrinone are chosen for their significant vasodilatory effect. In addition, the use of ß-blockers is recommended universally in all patients with stable mild, moderate, and severe heart failure with ischemic or non-ischemic cardiomyopathy and reduced LV ejection fraction.[12]

The question naturally arises as to whether or not the above-mentioned treatment modalities and recommendations arose from poorly designed trials or whether or not the understanding of pathophysiologic mechanisms involved is in need of "renewed growth and development."[5]

A number of other examples challenge the understanding of the basic tenets of circulation, such as the curious phenomenon of *increase* in cardiac output during aortic cross-clamp by up to 25% in a controlled experimental setting[13] and, in some patients, during aortic surgery.[14] The Fontan repair used for surgical correction of various hypoplastic right and left heart syndromes (HLHS) presents a yet-to-be explained hemodynamic paradox which, in the absence of the right heart complex, the single, often weakened, ventricle supposedly pumps the blood through systemic *and* pulmonary circulations.[15] There are a large amount of conflicting data from exercise physiology in which the concept of a muscle pump has been evoked in order to explain the greatly increased systemic blood flows that exceed theoretical limits of the heart's pumping capacity. Review of literature suggests that increased cardiac outputs can neither be ascribed to the heart (on account of a greatly shortened diastole that precludes adequate filling) nor to contracting muscles.[16]

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From the physiologic perspective, the heart is considered to be a dual pump, driving the blood through pulmonary and systemic circuits arranged in series. In the course of an average life span of 75 years, the heart, weighing around 350 grams, pumps 400 million liters of blood (the amount that fills a lake 1 km long, 40 m wide and 10 m deep)[17] through a system of conduits with the total length of about 100,000 km. Considering the fact that the diameter of the red blood cells frequently *exceeds* the width of the capillary beds, the heart as a pump truly performs a prodigious task.

The idea that the heart is a pump providing the total mechanical energy for blood's propulsion has dominated the field of cardiovascular physiology for well over a century. A detailed discussion of the history of the propulsion pressure circulation model is beyond the scope of this article,[18] but even a cursory look at the leading medical journals in the 1850's showed that there was a lively debate among the proponents of the heart-centered circulation model who supported the view that the heart is the "motor" of the circulation, and those who maintained that the "capillary power," or the force from behind (*vis á tergo*), played a principal role in blood's propulsion.[19] It should be noted parenthetically that the classic concept, *vis á tergo,* goes back to antique medicine when it played only a secondary role to *vis á fronte,* or "force from the front," which referred to suction forces (vacuum) working locally, (eg, ventricular diastolic suction) and at a distance, akin to gravity.[20] By the 1950s, these concepts still were mentioned in physiologic texts for historic interests [21–23] but, largely bereft of their original meaning, slowly acquired a new identity. The force from behind now assumes the dominant role as pressure generated by ventricular contraction, pushing the blood through the capillary beds back to the atria. A portion of this force is stored in vessel walls as elastic energy and is represented in the concept of the mean systemic pressure (Pms). The force from the front, on the other hand, became a generic term for a host of phenomena ranging from ventricular diastolic suction and/or respiratory pump, which facilitate filling of the heart, to a range of factors that *impede* venous return.[22] The latter became the mainstay of Guyton's venous return model of circulation discussed in the following section.

Over time, the pressure-propulsion (PP) model has become deeply engrained in the collective subconscious and, with few exceptions, virtually has remained unchallenged. It is suggested that in the light of rapidly accumulating growth of information obtained with the help of in-vivo experimental and clinical imaging modalities, the number of discrepancies between the observed phenomena and the constraints imposed by the existent circulation model is likely to increase. It is the purpose of this article to present some of the recently collected evidence against the commonly accepted PP model of circulation and to propose the conceptual framework for a new, more complete understanding of the circulatory phenomena.

In the first part of the article a brief historic outline and the salient features of Guyton's venous return (VR) model of circulation are discussed as well as the reason for its incongruence with the left ventricular (LV) model of circulation. Attention then is turned to the heart and to ways in which its mechano-energetic function compares to a standard hydraulic pump. Work on isolated heart preparations demonstrates that the heart is unable to maintain constant pressures or flow in face of the changing loading conditions and suggests that it is a rather inefficient pressure-propulsion pump. It is proposed that the heart functions by interrupting the flow of blood already in motion; that is, as an impedance pump, whose mechanical action can be compared to a hydraulic ram. It is further suggested that in place of the mechanistic PP model, the biologic model of circulation be adopted in which the blood is a self-moving agent driven by the metabolic demands of the tissues. The evidence in support of this model comes from observations of the embryonic circulation, through comparative anatomy and from phenomenology of the mature circulation. It is then shown that the conceptual framework for the PP model is rooted in the principles of a thermodynamically closed system, which, according to current understanding, no longer adequately describes the biologic phenomena in general and, as proposed in this article, the circulatory system in particular. Finally, the phenomenon of autonomous blood movement is discussed in the context of open-systems biology.

WHAT CONTROLS CARDIAC OUTPUT?

In spite of the general assumption that the heart provides the total mechanical energy for blood propulsion, the experimental observations have polarized basic scientists and clinicians into 2 opposing views concerning the control of cardiac output (CO). While proponents of Guyton's VR model contend that the peripheral circulation plays the dominant role in control of CO, adherents of the LV model ascribe this role, by default, to the heart.[24–27] Since the ultimate source for blood propulsion in *both* models can be traced to the hydrodynamic equivalent of Ohm's law (where the power source for the circulating blood clearly originates in the pump, ie, the heart), those seemingly opposing views differ only on the surface but not in essence. It is apparent that this central issue in cardiovascular physiology will not be resolved until the fundamental question ("What makes the blood go around?")[25] is considered not only in the light of the conventional model but also from the observed circulatory phenomena themselves.

GUYTON'S VENOUS RETURN MODEL

Between the 1950s and 1970s, Arthur Guyton and coworkers developed a circulation model that has, in due course, become almost universally accepted. At the core of the model is the idea that venous circulation plays a central role in control of CO. The starting point for the VR model was a number of observations that convinced Guyton and his collaborators that cardiac output largely was unaffected by the activity of the heart.[28] For example, artificial pacing of the heart at rates up to 4 times above baseline in animals[29] and humans[30,31] did not cause an increase in CO. Similarly, experiments on dogs, in which the right heart was replaced by a bypass pump, showed that CO could be maintained at the baseline level only when the pump output matched the autonomous rate of venous return. The increase in pump flows above the baseline would result in collapse of the great veins without change in CO.[32]

Significant to Guyton's model is the division of the circulatory system into 2 parts. The first consists of the heart and lung and the second of the entire systemic circulation.