Peak exercise responses in heart failure: back to basics

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This editorial refers to ‘Exercise haemodynamic variables rather than ventilatory efficiency indexes contribute to risk assessment in chronic heart failure patients treated with carvedilol’1, by U. Corrà et al. on page 3000

Evaluating central haemodynamics became a prominent cardiological feature in the 1950s following Sarnoff and Berglund’s introduction of Starling’s Law into practice via a family of ventricular function curves,1 but it went out of favour after 1981 when Benge2 and Franciosa3 claimed discrepancy between haemodynamics and exercise capacity in heart failure (HF) patients. Dismissing its value became fashionable in subsequent decades with further emphasis of its dissociation with changes in symptoms. In 1991, Mancini and colleagues4 greatly contributed to the widespread use of cardiopulmonary exercise testing (CPX) by showing its prognostic value in patients with severe HF referred for transplantation. Since this landmark study, a large number of studies have confirmed the unsurpassed prognostic value of peak exercise capacity, generally reported as peak oxygen consumption (peak VO2/kg), in HF patients. The simple explanation for this is that, according to the Fick principle, peak exercise VO2 is determined by peak exercise cardiac output and arteriovenous oxygen difference. When Chomsky et al.5 tried to reintroduce the usefulness of haemodynamic evaluation in selecting transplant candidates in 1996, they were rather overwhelmed by counter arguments.6,7 The emergence of such discrepancies is clearly puzzling, especially in view of the fact that central haemodynamics offer by far the best means of looking into cardiac performance and dysfunction.

There are a number of reasons why haemodynamic evaluations went out of favour. Chief amongst these are the choice of unrepresentative haemodynamic variables and the misplaced belief that measurements at rest can be representative of actual cardiac function irrespective of reserve capacity. Such a misconception probably stemmed from the misleadingly title of the 1981 paper by Franciosa,3 which also failed to highlight that in their own study, exercise haemodynamics actually correlated very well with exercise capacity.

A more basic reason why haemodynamics appear to bear little relation to clinical HF status is largely due to a fundamental conceptual problem in defining cardiac function teleologically from the perspective of the metabolizing body tissues in the peripheries. In this framework, the heart is regarded as a black box, the main function of which is to subserve the delivery of oxygen to metabolizing tissues. Since oxygen delivery is dependent on the oxygen-carrying capacity of blood, the flow output from the heart, and regional distribution of flow, then the role of the heart is perceived merely as a supplier of flow, irrespective of how this is achieved. The arterial pressure is then viewed simply from the vascular perspective, as a by-product of how much blood flow enters the vessels and how this interacts with the resistances in the vasculature (pressure = flow × resistance), ignoring the fact that the pressure–flow relationship of the heart itself is entirely different. How good or bad the failing heart is considered is dependent solely on how well it can deliver flow output, commonly referred to as ‘cardiac output’. One problem of this traditional concept is that it is only partially correct in its definition of how the organic pump must perform to fulfil its function, in that only the necessary, but not the sufficient, criterion is met. Such a concept does not take into consideration how the heart performs as a displacement pump and how it functions to maintain the requisite circulation.

A more realistic view is to regard the cardiovascular system as an integral hydraulic pump–pipes system obeying the laws of physics. For this, it is worthwhile going back to basics, by considering the milestone definition of cardiac function attributed to William Harvey, the discoverer of the circulation, who in 1628 stated: ‘...that the movement of the blood is constantly in a circle, and is brought about by the beat of the heart...’.8 Applying Newton’s first Law of Motion to the cardiovascular system, we may infer that blood cannot be constantly in motion in the circulation unless acted upon by the beat of the heart. The entity which is provided by the beat of the heart to allow the continuous motion of blood is the hydraulic energy, without which blood...
in the circulation would come to a standstill, due to the forces opposing flow, the frictional and separational forces. According to the Law of Conservation of Energy, to maintain the circulation, the hydraulic energy lost in the vasculature has to be replenished by the energy imparted by the heart. The function of the heart, expressed in modern physiological terms, is therefore to provide adequate hydraulic energy to maintain the circulation. This definition depicts the notion of energy dissipation in the vasculature, which has to be counterbalanced by the work of the heart. The rate of energy required to move a volume of fluid continuously is the product of pressure and flow rate (power = energy or work per unit time = flow rate \times pressure). Thus the ability of the heart to generate energy and perform external work encompasses not only its ability to generate flow, but also its ability to generate pressure. Pressure generation is an essential part of pump function, unless the impedance to flow in the circulation is zero. During severe exercise, up to 10-fold augmentation of hydraulic energy could be imparted by the heart pump into the heightened circulation. Much greater pressure generation than flow would be required for severe isometric exercises such as weight lifting, whereas greater flow is required for distance running. Elite athletes representing these two extremes of sports display concentric and eccentric left ventricular hypertrophy, respectively. Conceptually, therefore, we can no longer afford to regard the heart’s contribution to arterial pressure as being secondary. It is an intrinsic function of the cardiac pump. Evaluations of cardiac function or dysfunction which ignore this aspect must be deemed incomplete.

It is therefore not surprising to find that Corra and coworkers report that peak systolic blood pressure (SBP) and peak circulatory power (CP) were the most predictive of prognosis in a population of >600 HF patients receiving carvedilol therapy, more so than peak VO₂, or V̇E/V̇CO₂ slopes, based on their analyses of all the standard CPX variables. Their univariate analysis shows that both the peak CP and SBP have the highest prognostic value, as well as the presence of oscillatory ventilation. With multivariate analysis, peak circulatory power disappeared because of collinearity with VO₂ and BP, and exercise SBP emerged as the highest prognostic parameter.

It is now well known that peak VO₂ is influenced by many factors apart from cardiac factors, such as peripheral muscle mass and metabolism, vascular tones, peripheral oxygen extraction, haemoglobin content, and levels of sympathetic and parasympathetic activation. Alterations of these parameters can affect peak VO₂ besides cardiac output response; conversely, a poor cardiac response can be compensated by a good peripheral adaptation such as in athletes suffering from HF. Similarly to the present finding, Osada and co-workers reported that the combination of peak SBP and peak VO₂ significantly enhanced risk stratification in HF patients referred for heart transplantation. Peak cardiac stroke work (SW) and power output (PO) have long been recognized as accurate markers of the pumping capacity of the heart, providing more information than cardiac output. Cardiac power has been shown to be the strongest independent haemodynamic correlate of in-hospital mortality in patients with cardiogenic shock. All studies that have assessed the prognostic value of the peak exercise SW measured by Swan Ganz catheter, or of exercise PO assessed by exercise radionuclide ventriculography have found these measures to have greater prognostic value than peak VO₂. Williams et al. have confirmed that it was possible to assess cardiac PO non-invasively during CPX by measuring exercise cardiac output with a CO₂ rebreathing technique. Its prognostic value was greater than that of peak VO₂. Very recently, Lang and colleagues have confirmed these data by use of an inert gas rebreathing technique. Cohen-Solal et al. have shown that it was possible to approach peak exercise PO non-invasively without any measurement other than that of respiratory gas exchanges, as usually done during CPX: the ‘circulatory power’, defined as the product of peak VO₂ and peak SBP, algebraically equals peak cardiac PO times peak exercise arteriovenous oxygen difference, and has greater prognostic value than peak VO₂, a finding subsequently confirmed by Corra et al. and other studies. The take-home message of the findings of Corra et al., along with those recently reported, is that it is high time to get back to basics and try to identify, among the various exercise parameters, those mirroring the pumping capability and reserve of the heart. Whatever the way of assessing it, either separately assessing exercise BP and VO₂ responses to peak exercise, or indirectly (using CP) or directly assessing cardiac power, the evaluation of the ‘hydraulic’ responses of the heart and the circulatory system should become standard in the assessment of HF patients, especially for prognostic purposes.

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References


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Anomalous connection of the inferior vena cava to the left atrium diagnosed using three-dimensional echocardiography

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Routine transoesophageal echocardiography (TOE) was performed on a 3-year-old boy, following diagnosis of a secundum atrial septal defect (ASD), to assess suitability for device closure. His parents had noted some increasing shortness of breath on exertion over the preceding months, otherwise he remained well. Saturations at rest were normal (97%) and cardiovascular clinical findings were consistent with an ASD.

In addition to a 17 mm secundum ASD, the TOE images demonstrated partial anomalous pulmonary venous connection (with the right upper pulmonary vein draining to the right atrium) and, furthermore, longitudinal views (obtained at 90°) suggested the possibility of abnormal drainage of the inferior vena cava (IVC) (Panel A). Transthoracic 3D echocardiographic images were then obtained with a high-frequency matrix probe (X7-2 probe, IE33 Ultrasound system, Philips, MA, USA). These images clearly demonstrated anomalous connection of the IVC to the inferior aspect of the left atrium (Panel B). Further invasive imaging was not felt necessary, and the anatomy was confirmed during subsequent successful surgical correction.

Anomalous connection the IVC to the left atrium is a rare congenital cardiac abnormality. Associated cardiac defects (most commonly an ASD) are present in just over half the patients and the majority have some degree of systemic cyanosis. Previously reported cases have confirmed the diagnosis with angiography, but advances in non-invasive imaging techniques such as 3D echocardiography can now facilitate accurate diagnosis.

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