Myocardial power delivery is impaired in progressive left ventricular pump failure: a case report

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Abstract

The driving force for the flow of blood is the total energy imparted to it that corresponds to the mechanical, external work performed by the ventricle. In the cardiovascular system, the pumping power of the heart can be determined by cardiac power output, which is the product of cardiac output and mean blood pressure. Similarly to power-to-weight ratio, that is a measurement of the actual performance of any engine, maximal left ventricular power output-to-left ventricular mass is an index of cardiac performance potentially useful in patients with heart failure. In this case report, an example of the usefulness of this parameter is described in a patients with advanced heart failure secondary to dilated cardiomyopathy.

Keywords: heart failure; dilated cardiomyopathy; echo stress

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Case Report

Introduction

In attempting to characterize a system composed of an energy source and pipes conducting this energy, the usual parameters used for this purpose are the power of the energy generator and the resistance of the conducting pipes [1]. In the cardiovascular system, the pumping power of the heart can be determined by cardiac power output, which is the product of cardiac output (CO) and mean blood pressure (BP). By incorporating both flow and pressure in a single entity, cardiac power output represents the amount of energy imparted by the left ventricle to the volume of blood ejected per second [2]. Cardiac power output can be invasively and noninvasively assessed during maximal exercise or pharmacological stress test [3–7].

Power-to-weight ratio is a measure that is widely applied to mechanical engines to compare the performance of vehicles, aircrafts, and other mobile power sources. Similarly to power-to-weight ratio, peak power output-to-left ventricular (LV) mass (peak power-to-mass) is an index of LV performance potentially useful in patients with cardiac diseases. This parameter allows us to assess the relationship between cardiac power measurements and most of the recruitable myocardial reserve available at maximum workload. As a result, peak power-to-mass may be interpreted as a measure of myocardial efficiency, that is a ratio that incorporates the degree of the external work per unit of time and the maximal work possible.

Although the denominator of this equation cannot be measured, it can be argued that in normal ventricles the amount of LV mass is comparative to myocardial power delivery, whereas a disproportion between LV performance and mass is suggestive of the maladaptive features of LV remodeling [1]. To date, peak power-to-mass can be easily assess during exercise or dobutamine stress echocardiography and resulted to be valuable to measure cardiac pumping capacity especially in patients with cardiomyopathies. In the following case report, the clinical significance of peak power-to-mass is described.

Case report

The clinical, biochemical and echocardiographic data of a 64-year-old man with idiopathic dilated cardiomyopathy hospitalized because of symptoms of congestive heart failure (HF) are reported. After stabilization, cardiac right-sided catheterization was carried out using a 7F MPA1 catheter (Cordis, Miami, FL). Mean pulmonary capillary wedge pressure was determined automatically by the monitoring system (Horizon 9000 WS, Mennen Medical Ltd, Israel). LV end-diastolic pressure was recorded using a 6F 145° pigtail catheter (Cordis, Miami, FL). Hemodynamic measurements were acquired before any injection of the contrast medium. LV end-systolic and end-diastolic meridional wall stresses were estimated using invasive measurements of LV pressures. The patient was submitted to a comprehensive transthoracic echocardiography using commercially available Acuson Sequoia C256 ultrasound instrument (Mountain View, CA) with 2nd-harmonic imaging and a 3.5-MHz transducer. Two-dimensional and color-flow Doppler images were obtained in standard parasternal and apical views. The LV mass was determined by using the M-mode method according to the recommendations of the European Society of Echocardiography [2]. A symptom-limited graded bicycle semi-supine exercise was performed at an initial workload of 20 watts lasting for one minute; thereafter the workload was increased stepwise by 10 watts every minute. A 12-lead electrocardiogram and blood pressure determination were performed at baseline and every minute thereafter. At baseline and at peak exercise, Doppler-derived CO at LV outflow tract, heart rate (HR) and arterial systolic blood pressure (BP) and diastolic BP (by cuff sphygmomanometer) were measured. Mean BP was estimated as follows: diastolic BP + 1/3 (systolic BP – diastolic BP). Stroke volume (SV) was calculated as stroke distance × LV outflow tract area and CO as SV × HR as previously described [3]. LV power output was measured as the product of CO and mean BP. In meter-kilogram-second units, the conversion is 10^6 ml/m^3 for SV, and 133 pa (pascal)/mmHg for pressure. Power-to-mass was calculated as LV power output per 100 gram of LV mass: 100 x LV power output divided by LV mass (watt/100 g).

The characteristics of the patient during the hospitalization are shown in Table 1 and Table 2. He was in NYHA class III and his LV ejection fraction (EF) was 21%. The electrocardiogram showed a sinus rhythm and a complete left branch bundle block. B-type natriuretic peptide (BNP) level was 498 pg/ml. The workload reached at the end of the stress test performed the day before discharge was 70 watts. At maximum exercise, mean BP was 107 mmHg, SV was 80 ml and HR was 145 beats per minute; entering these values in the above formulas, we get: BP = 133 × 103 = 13,699 pa, SV = 35 × 10^{-6} m^3 and then we can calculate power as (13,699 × 35 × 145 × 10^{-6})/60 = 1.15 watt. LV mass was 349 g. A simplified formula to calculate power output-to-mass is: 0.222 × CO (l/min) × mean BP (mmHg)/LV mass (g) = (0.222 × 5 × 103)/349 = 0,33 watt/100 g. The patient underwent cardiac resynchronization therapy and was implanted with an automatic cardioveter defibrillator. Then, he was discharged with a therapy that included furosemide, angiotensin converting inhibitors, aldosterone antagonists, beta-blockers and digoxin. Six months later, he

Variable	
Age, years	64
Body surface area	26
Right atrial pressure (mmHg)	10
Pulmonary artery systolic pressure (mmHg)	58
Pulmonary artery diastolic pressure (mmHg)	30
Pulmonary artery mean pressure (mmHg)	30
Pulmonary capillary wedge pressure (mmHg)	33
Left ventricular end-diastolic pressure (mmHg)	25
Cardiac output (l/min)	3.4
Cardiac index (l/min/m^2)	1.7
Left ventricular end-systolic stress (kdyne/cm^2)	53
Left ventricular end-diastolic stress (kdyne/cm^2)	183

Table 1 *Clinical and hemodynamic characteristics of the patient described in the case report.*

	Before	After
Resting variables		
Heart rate (bpm)	81	65
Mean blood pressure (mmHg)	89	60
End-diastolic volume (ml)	205	206
End-systolic volume (ml)	162	161
Stroke volume (ml)	43	45
Stroke volume index (ml/m^2)	23	24
Ejection fraction (%)	21	22
Cardiac output (l/min)	3.5	2.9
Cardiac index (l/min/m^2)	1.8	1.5
Power output (watt)	1.15	0.39
Power output-to-mass (watt/100 g)	0.20	0.11
Exercise		
Workload (watt)	70	60
Heart rate (bpm)	145	85
Mean blood pressure (mmHg)	103	73
End-diastolic volume (ml)	165	181
End-systolic volume (ml)	130	141
Stroke volume (ml)	35	40
Stroke volume index (ml/m^2)	18	21
Ejection fraction (%)	21	22
Cardiac output (l/min)	5.0	3.4
Cardiac index (l/min/m^2)	2.6	1.8
Power output (watt)	0.69	0.55
Power output-to-mass (watt/100 g)	0.33	0.16

Table 2 *Clinical and echo-Doppler-derived hemodynamic characteristics of the patient described in the case report.*

was re-hospitalized for worsening HF. LV EF was not different with respect to the previous hospitalization and no relevant changes were apparent for most of the other echocardiographic and Doppler parameters. Pre-discharge BNP was 1072 pg/ml. The exercise stress test was repeated before discharge. Peak power output-to-mass at exercise stress test was 0.16 watt/100 g. Four months later the patient died due to refractory progressive pump failure.

Discussion

This case report describes a case of dilated cardiomyopathy accompanied by a severe deterioration of cardiac pumping capacity. The patient developed a progressive refractory heart failure, which was clearly evident at the time of the first exercise stress test, where peak cardiac power output showed only a blunted increase during the exercise. Neither optimized medical treatment nor cardiac resynchroniza-

tion therapy were able to retard the progression of the disease.

This report shows the importance of measuring cardiac pumping capacity during exercise stress echocardiography for risk stratification of patients with advanced HF. It is interesting to note that resting LV EF did not change between the first and the second hospitalization. LV EF is the most frequently used index of LV performance, but it may not accurately reflect myocardial contractility and provides little prognostic information in patients with advanced HF. The interpretation of an EF less than 30% in a NYHA class I patient with mild LV dilation may be quite different from that an individual with class III symptoms associated with a severely dilated left ventricle. Both EF declines reflect chamber remodeling despite relative preserved stroke volume. However, in one instance, LV remodeling dominates this decline, with a near normal residual myocardium capable of providing adequate cardiac reserve. In the other, the entire LV myocardium is depressed with little reserve pumping capacity. These two situations may look similar when assessed by conventional measures, such as EF or wall motion score index, yet be very different by alternative methods, such as cardiac power output performed under stress.

Although cardiac power output is a well-established parameter of ventricular function that can be noninvasively acquired during exercise testing, it does not consider alterations in cardiac size and structure that may have an impact on the outcome of patients with HF. The novelty of peak power-to-mass (and peak mass-to-power) is that it encompasses LV mass, that is a major feature of the alterations in ventricular structure that occurs as a part of normal growth or due to a pathologic process, and similarly to EF, that is the ratio between the stroke volume and LV end-diastolic volume, provides integrate information on cardiac function and ventricular remodeling. The amount of LV mass may be equated to the energy stored in the myocardium according to the principle of equivalence of mass and energy as affirmed by Einstein's theory of relativity. An example of this is physiological hypertrophy that is induced by exercise training, whereas the phenotypes that appear in chronically overloaded ventricles are pathological because they are accompanied by maladaptive changes [4,5]. The discrepancy between a severely depressed cardiac power output

albeit an increased LV mass is likely to reveal the presence of maladaptive LV remodeling and this may reflect the inefficiency of the system to comply with the body's metabolic needs. Adverse or maladaptive LV remodeling is a major factor that affects the outcome of patients with advanced HF due to LV systolic dysfunction [6].

The effects of intervening LV hypertrophy on recruitable cardiac power output are important to establish the significance of LV remodeling. When the recruitable power output per unit of LV mass decreases due to progressively decreasing ability of the myocardium to generate force to overcome the load, LV function rapidly deteriorates. Furthermore, the high LV end-diastolic volume and pressure promote subendocardial ischemia that aggravates LV dysfunction and neurohormonal activation, decreases exercise capacity and increases the risk of ventricular arrhythmias. Another factor that may contribute to maladaptive LV remodeling is the inadequate growth of myocardial microvasculature accompanying myocardial hypertrophy [7].

Peak power-to-mass provided incremental prognostic information over resting LV EF as well as other LV parameters recorded under stress. In our experience, the cutoff value for peak cardiac power-to-mass that accurately predicts all-cause mortality or HF hospitalization is 0.58 watt/100 g, but its impact on prognosis is clearer if the patient achieves a peak cardiac power-to-mass less than 1.0 watt/100 g after optimal tailored therapy or myocardial revascularization with interventional cardiac procedures or resynchronization therapy [6].

By coupling peak exercise LV power output and LV mass, peak power-to-mass is useful to identify patients with adverse LV remodeling during stress echocardiography and may provide additional prognostic information either in association with resting echocardiographic studies or cardiopulmonary exercise testing. LV hypertrophy is almost always present in patients with chronic systolic HF accompanied by LV dilatation and low EF, is typically eccentric, and is frequently associated with a normal or lower than normal LV wall thickness [8]. Despite that changes in LV geometry and wall thickness may be temporarily useful in maintaining myocardial pump function, they occurs at significant high cost and are commonly followed by the unfavorable consequences of dilation. LV enlargement increases the force that must be generated to exceed end-diastolic wall stress and to achieve a given level of cavity pressure. •

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