The correlation of cardiac power output to exercise capacity in chronic heart failure


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Summary. Cardiac haemodynamics are deranged in chronic heart failure but fail to predict the exercise capacity of the patient. Cardiac power output is a descriptor of cardiac function derived from preload, blood pressure and cardiac output. Forty-one patients with moderately severe and severe chronic heart failure were exercised on a cycle ergometer to determine the relationship between traditional haemodynamics and cardiac power output and exercise capacity. Resting cardiac power output was no more predictive of exercise capacity than resting stroke-work index or resting cardiac index (r=0.53, 0.61 and 0.51 respectively). Maximum cardiac power output and the ability to increase cardiac power output, however, were correlated with exercise capacity (r=0.79 and 0.80). It is concluded that resting cardiac power output does not predict subsequent exercise capacity but that maximum cardiac power output and the ability to increase cardiac power output on stimulation are good descriptors of functional cardiac reserve.

Key words: Chronic heart failure – Cardiac power output – Exercise capacity – Functional cardiac reserve

Introduction

In patients with chronic heart failure the initial reduction in pumping capacity is compensated by secondary changes in the neurohumeral milieu leading to a redistribution of the reduced cardiac output. However, this in turn reduces blood flow to exercising muscle and results in a reduced exercise capacity. Further reductions in cardiac performance compromise organ function even at rest and hence there are two considerations of overriding importance to the patient. First, what is the prognosis in terms of survival and mortality and second, how much physical activity can they manage? The answer to the first question lies largely in the answer to the second. The greater the degree of functional disability of the patient, as reflected by their symptoms, then the worse the prognosis. Clearly, however, the answer to the second question has important implications not only for the ultimate prognosis but also for the quality of life enjoyed by the patient. In such patients, exercise testing has been used both to assess the severity of their functional disability and to monitor progress during therapy, and it has been shown to be a safe procedure (Weiner 1983; Weber et al. 1984; Tristani et al. 1987).

The diagnosis and evaluation of heart failure is usually based on signs and symptoms of circulatory congestion; however, these symptoms and signs may be manifest in other conditions and may be absent even in the presence of severe cardiac disease (Eichna 1960). Greater information can be obtained from haemodynamic measurements in vivo by using catheters inserted into the right and left heart chambers and connected to pressure transducers to detect the failure to generate pressure during systole and the increased back pressure during diastole. Further, thermodilution techniques using the same catheters can demonstrate the cardiac output (Forrester et al. 1972) and hence disturbances of both flow and pressure can be elucidated. Predicting the exercise capacity from the clinical signs and the degree of haemodynamic disturbance, however, is difficult and whilst it has been shown that symptom score is related to exercise capacity, with the patients most symptomatic able to exercise least (Engler et al. 1982), the connection between other indicators of left ventricular dysfunction and exercise capacity is much more tenuous. Many studies have shown that resting haemodynamic parameters fail to predict subsequent exercise time (Engler et al. 1982; Gelberg et al. 1979; Higginbotham et al. 1983). Ejection fraction is no better as a predictor of functional incapacity (Higginbotham et al. 1983; Benje et al. 1980; Franciosa et al. 1981), with one study showing that 50% of patients with an ejection fraction of less than 30% had normal exercise capacity (Benje et al. 1980). Calculated indices of resting left
ventricular function such as pre-ejection time, velocity of circumferential shortening, stroke volume index and stroke work index have all failed to predict exercise capacity. Surprisingly, changes in exercise capacity induced by therapeutic intervention (Franciosa et al. 1981) or observed during long term follow-up of patients (Engler et al. 1982) have not been correlated with any concomitant changes in ejection fraction or cardiac haemodynamics.

Whilst resting cardiac function may be impaired it is the inability to exercise normally which is the major feature of chronic heart failure and therefore measurements of cardiac function during exercise testing may provide a much better indication of the severity of heart failure than can resting cardiac assessment.

In patients in whom it can be measured, the most accurate method of evaluating maximum achievable exercise capacity is by on-line respiratory gas analysis to determine the maximum oxygen consumption ($V_O^{2}_{max}$) during exercise (Naimark et al. 1964). The equipment required for such analysis is expensive and elaborate (Janicki et al. 1986) and many authors report that it is rare to achieve a true $V_O^{2}_{max}$ during exercise testing (Lipkin et al. 1985; Francis 1986). Buller and Poole-Wilson (1988) have applied a curvilinear fit to the data of VO$_2$ plotted against (carbon dioxide production ($V_C^{O_{2}}$) and used a mathematical extrapolation of the function so determined to project a theoretical $V_O^{2}_{max}$ for each patient, which is then independent of exercise duration. Unfortunately, there is surprisingly little correlation between any of the resting haemodynamic variables and maximum exercise capacity as assessed by anaerobic threshold or $V_O^{2}_{max}$ (Cohn and Francis 1982).

If a relatively simple haemodynamic measure of cardiovascular competence were available then it would have important implications for two reasons. First, if a predictor of cardiovascular reserve existed in the resting patient this would enable the physician to make a much fuller clinical assessment of the patient without resort to exercise testing. Second, if a single more complete descriptor of cardiovascular function existed than is at present afforded by multiple, separate haemodynamic variables then this would enable the physician and scientist alike to follow the progress of individual patients and, in particular, assess the functional rather than merely haemodynamic consequences of therapeutic interventions.

Tan (1987) has shown that maximum achievable cardiac power output (an assessment of the hydraulic power output of the heart) has a highly significant prognostic value for 1-year mortality. Since cardiac power output is calculated from the preload, arterial pressure and cardiac output, and therefore incorporates both the flow and pressure generated by the heart within the cardiovascular system, it represents a potential advance over merely measuring cardiac output or pressures within individual compartments within the cardiovascular system.

This study investigates whether resting cardiac power output can be used to predict maximum cardio-vascular function and reserve as determined by exercise time and also whether the relationship between exercise time and cardiac power output is stronger (or weaker) than the relationships between the more traditional haemodynamic variables and exercise time.

**Methods**

**Patient demographic and cardiovascular details.** Forty-one patients, 34 of whom were men, with chronic, stable heart failure secondary to either ischaemic heart disease (34 patients) or idiopathic congestive cardiomyopathy (7 patients), confirmed by cardiac catheterisation and coronary angiography, were studied (Table 1). Data was collected following catheterisation for diagnostic or therapeutic indications. All patients gave informed consent to the study which had the approval of the Ethical Committee of the East Birmingham Hospital. The majority (32 patients) were in New York Heart Association functional class III, 1 in class II and 8 in class IV (Criteria Committee of the New York Heart Association, 1973). The mean ejection fraction was 25.9 (SD 11.5)%; 37 patients were in sinus rhythm and 4 in atrial fibrillation. All were receiving frusemide (mean dose 134 mg, SD 17) and 29 were also receiving digoxin.

**Resting and exercise haemodynamic data.** On the morning of the study the patients were allowed an early, light breakfast. Between 0900 and 1000 hours a thermodilution Swan-Ganz catheter was inserted percutaneously, via the right subclavian vein and positioned in the proximal pulmonary artery and an arterial catheter inserted in the right brachial or radial artery. The patients rested for at least 30 min following instrumentation. They then transferred to a tilting exercise cycle ergometer and were again rested in the semi-supine position at 45°, for at least 10 min. Baseline resting haemodynamic data were collected in duplicate at 5-min intervals prior to exercise.

Patients exercised commencing at 25 W and increasing by 15-W increments every 3 min until stopped by dyspnoea or exhaustion. During the last min of each exercise stage mean and phasic right atrial (P$_a$), pulmonary artery (P$_p$), pulmonary artery wedge (P$_w$) and systemic pressures (P$_s$) were recorded and cardiac output assessed by thermodilution in duplicate. The electrocardiogram (lead II) was recorded throughout and provided a record of the heart rate and rhythm.

**Formulae.** The following formulae were used:

\[
\text{Pulmonary vascular resistance} = \frac{(P_a - P_w)}{Q_e} \text{ N}\cdot\text{s}\cdot\text{m}^{-5}
\]
\[
\text{Systemic vascular resistance} = \frac{(P_s - P_a)}{Q_e} \text{ N}\cdot\text{s}\cdot\text{m}^{-5}
\]
\[
\text{Stroke work index} = \frac{Vs}{(P_s [systolic]- P_{adw})} \text{ J}
\]
\[
\text{Cardiac index} = \frac{Q_e}{\text{Sur}-\text{face area}} \text{ 1 min}^{-1}\cdot\text{m}^{-2}
\]
\[
\text{Cardiac power output} = \frac{(P_s - P_a) \times Q_e}{450} \text{ W}
\]

where: \(V_s = \text{Stroke volume}\)
\(Q_e = \text{Cardiac output}\)

**Table 1.** Mean age, height and mass of the patients. Values given are the mean (SD)

<table>
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<tr>
<th></th>
<th>Value</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>59.1 (4.3)</td>
<td>42-72</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.72 (0.11)</td>
<td>1.57-1.88</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>74.1 (6.2)</td>
<td>55.3-95.3</td>
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