Exercise Testing In Health and Disease

JOHN R. SUTTON, MD
and NORMAN L. JONES, MD

SUMMARY

This article outlines areas where exercise testing may be helpful to the clinician, and describes how the various tests can be used to quantify simple cardiac and pulmonary responses to exercise. Exercise testing is also discussed as a diagnostic tool in areas such as ischemic heart disease or asthma.

Dr. John Sutton is assistant professor in the Department of Medicine at McMaster University. Dr. Norman Jones is coordinator of the Regional Respiratory Program and associate professor in the Department of Medicine at McMaster University, Hamilton, Ont.

POLLUTION, the energy crisis, and the ever increasing incidence of coronary heart disease have together stimulated a greater community interest in physical activity in everyday life. There has been a rebirth of bicycle riding, both as a means of transport and also for sheer pleasure. Numerous jogging clubs and YMCAs are oversubscribed. The medical profession has been rather slow to respond to this changing community emphasis in both the prescription of exercise as a therapeutic measure, and the use of exercise testing as a diagnostic and assessment tool. The many patients who present to their family physicians complaining of a decreased effort tolerance are all too frequently examined only at rest. Rarely is an attempt made to evaluate the patients under the conditions which provoke their symptoms, i.e. during physical exercise. Furthermore, since the clinical symptoms may not appear until the underlying pathophysiology is quite advanced, it is very difficult to assess the degree of functional impairment simply by questioning the patient. In this article we shall attempt to define those areas in which exercise testing may be helpful to the clinician.

The Exercise Test

In the simplest type of exercise test, heart rate and ventilation are monitored, at increasing levels of power output on a bicycle ergometer or a treadmill. We find it more useful to measure these simple variables over a range of power outputs than to make more complex measurements at one level of activity alone.[1]

In order to interpret the results of such a test it is necessary to know normal expected values for maximal power output, heart rate and ventilation. An increase in cardiac output is the most significant cardiac adaptation to exercise. This is achieved by both an increase in stroke volume and heart rate up to about 30-40 percent of maximum effort. Further increases in cardiac output depend on increases in heart rate. The maximum cardiac output is thus dependent on maximum heart rate. This decreases with age and may be estimated by using the following formula: maximum heart rate = 210 – (0.65 x age).[2] Maximum power output for normal people depends on maximum heart rate and size (see Fig. 1).

Ventilation increases linearly with power output until lactic acidosis occurs, after which the increases in ventilation appear excessive. This is caused by an additional CO_2 stimulus to ventilation produced when lactic acid reacts with the bicarbonate in blood. When such an increase in

Fig. 1

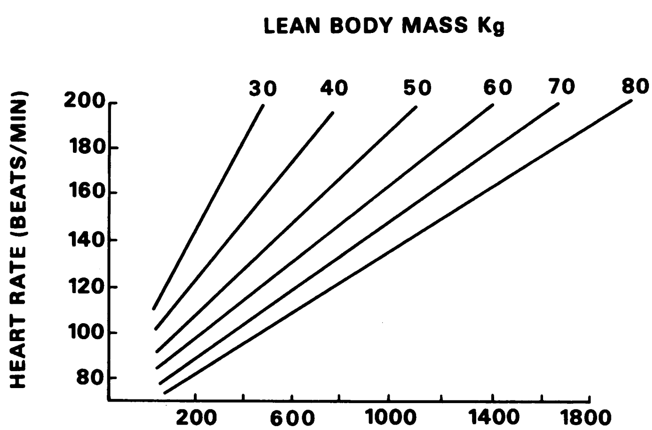

Fig. 1. The relationship of heart rate to power output for different values of lean body mass (fat free body weight).

ventilation is observed, it suggests that oxygen supply to muscle is no longer adequate and further energy requirements must be met by anaerobic metabolism. It further implies that a cardiovascular (oxygen transport) limitation to exercise performance has occurred. This is the usual limitation to exercise tolerance in normal subjects.

An increase in ventilation during exercise is achieved mainly by an increase in tidal volume at lower levels of power output, but thereafter principally by increases in the frequency of breathing. This pattern may be disturbed in certain disease processes, e.g. in a patient with stiff lungs (pulmonary fibrosis) where the overall increases in ventilation during exercise are achieved almost entirely by an increased frequency of breathing.

The maximum possible ventilation for any particular individual may be estimated from the forced expiratory volume in one second (FEV_1) using the following formula: maximum possible ventilation ($\dot{V}Emax$) = $FEV_1 \times 35$.[3] Normal people have a large ventilatory reserve even at maximum exercise. Thus, ventilation is never a limiting factor to exercise tolerance in normal people.

Example 1

A 40 year old male was referred by his family physician for a fitness assessment prior to engaging in a jogging program. He was asymptomatic.

Height 175 cms., Weight 85.5 Kgm. Hemoglobin 15 gm/100 ml. Spirometry – Vital Capacity (VC) 5.1 litres, FEV_1 4.5 litres.

Exercise test: (Before training) see Fig. 2

Maximum power output = 900 kpm/minute
Maximum heart rate = 186 beats/minute
Maximum ventilation = 73 litres/minute

Interpretation: the maximum power output of 900 is low for a person of this age and size (see Fig. 1). The maximum predicted heart rate equals 210 – (0.65 x 40) = 184 beats/minute, which was reached in the test. The maximum possible ventilation equals 4.5 x 35 = 157.5 litres/minute. Hence the ventilation of 73 litres/minute achieved in this test leaves considerable reserve. The increase in ventilation was linear up to a power output of 500 kpm/minute. Thereafter it became excessive, reflecting the onset of lactic acidosis, i.e. a cardiovascular exercise limitation.

Conclusion: this subject had a low maximum power output reasonably typical of middle-aged North American males. The exercise capacity was limited by cardiovascular factors indicated by achieving a maximum heart rate and hyperventilation at the higher work loads, reflecting lactic acidosis. The patient therefore has reduced cardiorespiratory fitness.

When the subject was re-tested after a six month training program, the results were as follows (see Fig. 2).

Exercise test after training:

Maximum power output = 1,400 kpm/minute
Maximum heart rate = 190 beats/minute
Maximum ventilation = 85 litres/minute

Interpretation: the maximum power output of 1,400 kpm/minute is normal for a man of this age and size. From the previous calculations we can see that his maximum predicted heart rate would be 184 beats/minute; now the maximum measured rate was 190 beats/minute. The maximum ventilation recorded after training of 85 litres/minute, although higher than before, still left considerable reserve. Furthermore, increases in ventilation were linear up to a power output of 1,000 kpm/minute and only then became excessive, showing that lactate accumulation did not occur until a higher load.

Conclusion: the training program had a specific effect on the cardiovascular system and resulted in the same level of power output at a lower heart rate, implying also a greater stroke volume. From these results, the subject is shown to have normal myocardial and pulmonary function during exercise without proceeding further with more complex investigations.

Example 2

A 35 year old male presented to his family physician complaining of episodes of chest pain occurring in relation to meals, sometimes to exercise and occasionally also at rest.

No abnormality was found on physical examination, and the resting electrocardiograph was essentially normal (see Fig. 4).

Fig. 2

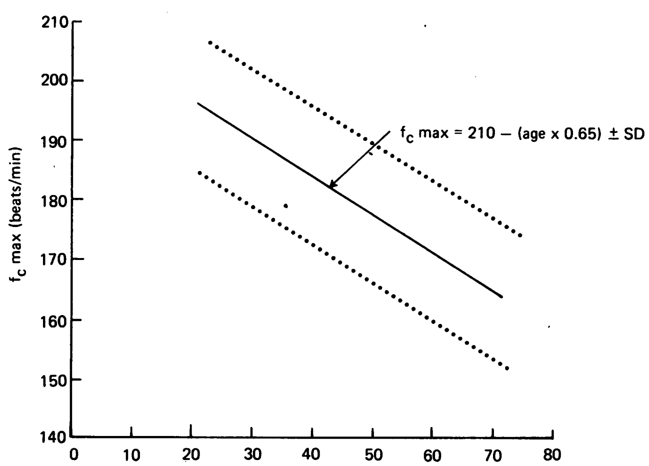

Fig. 2. Relationship of maximum heart rate to age.

Fig. 3

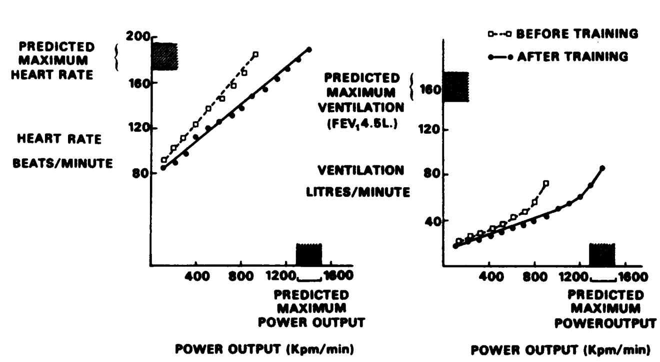

Fig. 3. Heart rate and ventilation during a progressive exercise test before and after a training program in Patient 1.

Height 182 cms. Weight 84 Kgm, Hb 15.0 gm/100 ml
VC 5.2 litres, FEV_1 4.42 litres.

Exercise test:

Maximum power output = 700 kpm/minute
Maximum heart rate = 152 beats/minute
Maximum ventilation = 55 litres/minute

The patient stopped exercise when he developed retrosternal tightness. His pain disappeared within two minutes of stopping the exercise. The ECG monitor showed ST depression at this time. A 12 lead ECG taken immediately after exercise (Fig. 4) shows the classical ST depression diagnostic of myocardial ischemia. These changes returned to normal over the next ten minutes.

Comment: this is a reasonably common clinical problem in which exercise testing can be diagnostic. Sometimes the ECG changes may not become manifest for two to five minutes after the exercise.

The Master Two-Step is probably the most commonly used exercise stress test for diagnosing ischemic heart disease and has been in use for more than 30 years. However, the effort is usually mild and impossible to quantify accurately. Electrocardiographs are usually taken only before and after the test, with no monitoring of the ECG or heart rate during the test itself. This is a potentially dangerous practice, particularly if the patient develops a sudden cardiac arrhythmia during the test. It also yields fewer positive results than the progressive test described above. In addition, by knowing the heart rate at which symptoms or ECG changes occur we can prescribe a safe level of activity and safe heart rate which will produce a training effect. The patients can be taught to count their own heart rate during exercise.

Example 3

A 28 year old female was referred by her family physician with breathlessness of recent onset. She stated that breathlessness was only related to exercise and frequently occurred after the exercise was complete. She rarely had trouble swimming but when she climbed stairs or played basketball she became acutely short of breath.

Height 156 cm, Weight 54 kgm, Hb 14.6 gm/100 ml
VC 3.5 litres, FEV_1 2.8 litres, FEV/VC ratio 89 percent predicted FEV_1 2.7 litres.

Exercise test:

Maximum power output = 600 kpm/minute
Maximum heart rate = 160 beats/minute
Maximum ventilation = 52 litres/minute

She stopped exercise because of general fatigue and became slightly breathless at the higher power outputs. After she stopped exercise she developed a cough. Simple spirometry was repeated immediately following the exercise. The immediate post exercise FEV_1 had fallen to 1.4 litres (Fig. 4). At four minutes post exercise her vital capacity was two litres and her FEV_1 was 0.9 litres. FEV_1/VC ratio = 45 percent.

Comment: this result is found in moderately severe exercise-induced asthma more frequently with jogging and running than with swimming. However, the breathlessness usually occurs after the exercise is completed and the fall in FEV_1 may be maximal within ten minutes. As can be seen from Figure 4 the maximum predicted ventilation on the basis of her resting FEV_1 was about 100 litres/minute. However, when one takes into account her immediate post exercise FEV_1 of 1.4 litres it can be seen that this patient was limited by her maximum breathing capacity at the end of the test.

The test was subsequently repeated following inhalation of a bronchodilator which almost totally eliminated the post exercise airways obstruction. This test was not only diagnostic but had therapeutic implications: the patient was treated with a bronchodilator prior to any severe exertion.

Example 4

A 38 year old female presented with increasing exertional dyspnea. She had a past history of a mitral valvotomy performed for mitral stenosis eight years previously and also had chronic bronchitis.

Following the valvotomy, some degree of mitral incompetence developed and her family doctor wondered if the mitral incompetence was now a significant factor in her exertional dyspnea. He wondered if she might now require a mitral valve replacement.

Fig. 4a

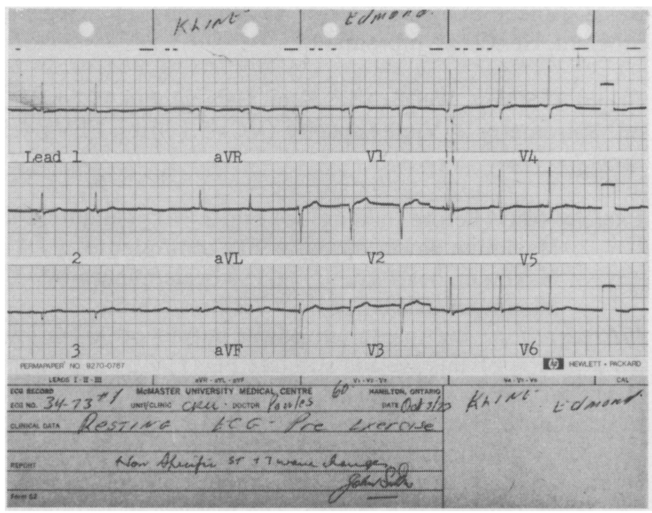

Fig. 4b

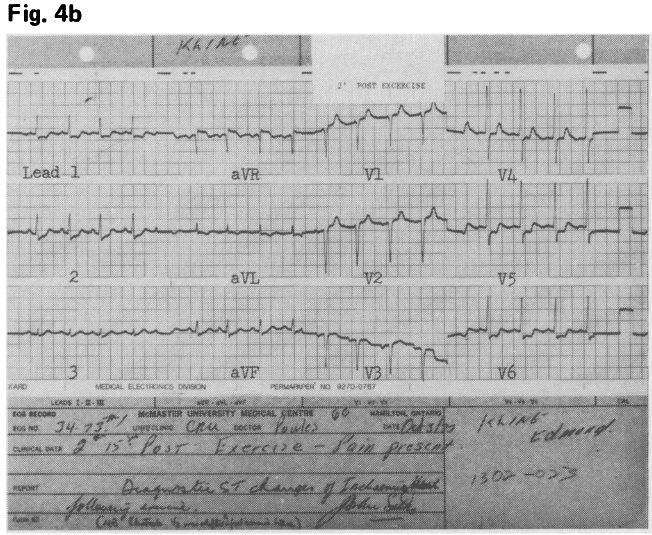

Fig. 4. Electrocardiograph (a) at rest and (b) following a progressive exercise test in Patient 2 showing diagnostic changes of ischemic heart disease.

Height: 155 cm, Weight 51 Kg, Hb 13.5 gm/100 ml
VC 3 litres, FEV_1 1.1 litres, FEV_1/VC 30 percent predicted FEV_1 2.46 litres (moderately severe airway obstruction).

Exercise test:

Maximum power output = 500 kpm/minute
Maximum heart rate = 155 beats/minute
Maximum ventilation = 34 litres/minute

Interpreting these results in the light of the predicted normals for a woman of this age and size we find that the maximum power output was well below that predicted, and the maximum heart rate of 155 beats/minute was well below the predicted maximum for her age of 190 beats/minute (Fig. 5).

Her ventilation followed the normal linear increase and did not show any excessive hyperventilation at the higher workloads. Hence, there was probably no lactic acidosis. However, her predicted maximum ventilation on the basis of the FEV_1 was only 35 litres/minute. From this test then, we conclude that the patient's exercise tolerance was limited by her ventilation and not by her cardiac response.

This patient had two underlying clinical problems, but her main symptom was exertional dyspnea. The exercise test enabled an assessment of the ventilatory as well as the cardiac components to the exertional dyspnea. The results indicated that a mitral valve replacement was not likely to improve her exercise tolerance and this resulted in a change of therapeutic emphasis, with increased attention to her chronic bronchitis.

The four examples given above typify some practical applications of exercise testing in both a diagnostic setting and in assessment of the ventilatory compared with the cardiac components to a reduced effort tolerance. The type of exercise testing discussed can be performed in most laboratories with a minimum of equipment. We use this test as a basic screening procedure, since it is quite simple, fast and non-invasive. If normal, there is no need to follow this with any more complex exercise investigations. On the other hand, if it is abnormal, a more detailed examination of the response pattern during exercise can be performed, including measurement of the dead space/tidal volume relationships, cardiac output and stroke volume. These require steady state exercise tests for their measurement and are slightly more complex maneuvers. However, in the vast majority of clinical settings a simple exercise test will be sufficient. ◀

Fig. 5

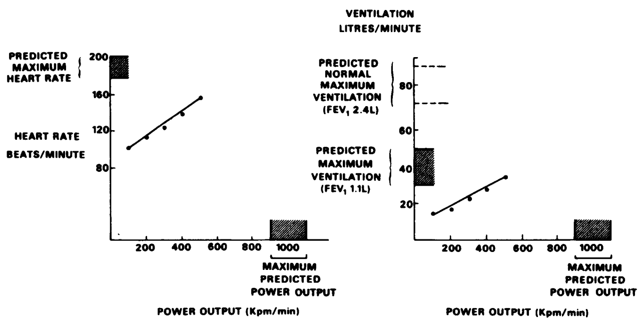

Fig. 5. Ventilation and heart rate response to exercise in Patient 4 — chronic chronic bronchitis and mitral valve disease.

Acknowledgement

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QUOTE

A mature fat man excites pity, like a ship well stocked for its last voyage.

Santiago Ramon y Cajal (1852-1934)
in "Charlas de Cafe"