LEADING ARTICLE

CLINICAL EXERCISE TESTING

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Why perform the exercise test?

A physiological system usually has a generous reserve capacity so that the latter is considerably reduced before symptoms are felt. Thus early cardiorespiratory disease may not be detected unless the limits of cardiorespiratory reserve are determined by exercise testing.

Subjective assessment of severity of symptoms is often an unreliable indicator of severity of disease and varies from patient to patient. In particular, subjects who are claiming compensation for an occupational disorder may exaggerate their symptoms. The exercise test is an objective measurement and allows quantification of a patient's effort status. It can be used to assess a patient's progress or the effect of treatment.

When a dyspnoeic patient has mixed cardiorespiratory disease, it is important but often difficult to decide which direction therapy should be pursued. Exercise testing may reveal predominant cardiac or respiratory limitation and thus be a useful guide to therapy.

The test may be diagnostic of exercise-induced asthma and ischaemic heart disease and is helpful in the investigation of dyspnoea of uncertain cause.

Is it safe?

In a review of 170,000 exercise tests, Rochmis and Blackburn[1] found only 16 deaths (about 1 in 10,000) that were attributable to the procedure. The short answer to the question is therefore "yes", if precautions are undertaken. All patients over the age of 40 years should have an electrocardiogram (ECG) before the test. Contraindications to testing include acute cardiac disease (acute and current ECG changes of myocardial ischaemia, uncontrolled heart failure, unstable angina, acute myocarditis, uncontrolled hypertension and pulmonary oedema), uncontrolled asthma, and an acute febrile illness.[2]

The physician should terminate a test when there are anginal pains, sudden pallor and sweating, cyanosis, malignant ventricular arrythmias, marked ST-T wave changes, falls in systolic blood pressure below pre-exercise values and exercise rises in blood pressure (systolic > 300mmHg or diastolic > 140mmHg).[2] The physician must of course be present by the patient's side throughout the exercise test monitoring the patient closely and ready to attend to any emergency which may arise.

What to measure?

1) *Electrocardiagram*

It is essential to measure the ECG continuously on a monitor where both rate and waveform are displayed. If the heart rate reaches the predicted maximum (Fcmax), for the subject (Fcmax = 210 – 0.65age in years[2]), the patient has put in his maximum effort. The waveform display is necessary to detect ST-T wave changes of ischarmic heart disease.

2) *Ventilation*

The patient/subject breathes through a one-way low resistance low deadspace valve and total ventilation (VE), tidal volume (Vt) and respiratory rate (Fb) are monitored. Normally VE matches metabolic demand during light to moderate exercise level, but above a certain workload, VE becomes excessive for the metabolic demand. This occurs after the anaerobic threshold (AT) and the excessive ventilation results from metabolic acidaemic stimulation to ventilation over and above the metabolic demands. This threshold occurs at about 50-60% of maximum exercise level in normal subjects but is reduced in cardiac disease, anaemia and pulmonary vascular disease.

3) *Gas exchange*

Endtidal CO2 is continuously monitored and an estimate of the dead space to tidal volume ratio (Vd/Vt) is derived from it. In normal subjects, Vd/Vt falls during exercise. A high Vd/Vt ratio which does not fall as expected during exercise may indicate pulmonary disease. The continuous monitoring of CO2 production and O2 consumption allows the derivation of the ventilatory equivalents of CO2 and O2 respectively ($\dot{V}E/\dot{V}ECO2$ and $\dot{V}E/\dot{V}EO2$) and the respiratory exchange ratio (R).*

The study by Duncan and Horvath in this issue of the Journal compared maximal exercise data in young healthy adult males of three ethnic groups, Malays, Indians and Chinese. The authors found no significant changes in the $\dot{V}O2max$, Fcmax, $\dot{V}E$, $\dot{V}E/\dot{V}EO2$ and O2 pulse between the ethnic groups. Also, the normalised $\dot{V}O2max$ and maximal cardiac parameters (corrected for body size) were not very different from those of similar studies on Caucasians.

VO2max depends upon several factors including sex,

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*Footnote: $\dot{V}E/\dot{V}ECO2$ ($\dot{V}E/\dot{V}EO2$) is the amount of ventilation required to excrete a litre of CO2 (or take up a litre of O2). It is thus an indication of the efficiency of ventilation in bringing about gas exchange. Thus an efficient lung will need to ventilate less to exchange a given amount of CO2 or O2, and the ventilatory equivalents for CO2 and O2, $\dot{V}E/\dot{V}ECO2$ and $\dot{V}E/\dot{V}EO2$, will be low. Above the anaerobic threshold, however, the respiratory system shifts its control priority to acid-base balance. And the lactic acidaemia will demand an even higher ventilation to excrete acid i.e. CO2. So the $\dot{V}E/\dot{V}ECO2$ and $\dot{V}E/\dot{V}EO2$ will rise when the AT is exceeded.

The respiratory exchange ratio, or R, is the ratio of the CO2 production ($\dot{V}CO2$) to the O2 consumption ($\dot{V}O2$) i.e. $R = \dot{V}CO2/\dot{V}O2$. Above the AT, more energy is obtained by "burning fuel without oxygen i.e. anaerobically. This pattern of fuel consumption, while producing CO2, does not consume O2. Therefore the R, which is normally 0.85, rises to values above 1.

age, motivation, fitness, haemoglobin level, humidity and temperature of the surroundings and the mode of exercise. A highly motivated trained subject has a higher $\dot{V}O_2max$. A low haemoglobin level and increasing age reduce $\dot{V}O_2max$. A higher ambient temperature and humidity reduce the ability of the body to lose heat. Therefore more of the cardiac output is shunted away from the exercising muscles to the skin for the purpose of heat loss. $\dot{V}O_2max$ is 10% greater during treadmill exercise than in cycle ergometry.

Physiologically speaking,

$\dot{V}O_2max = COmax \times (ArtO_2 - VenO_2)max$ (equation 1)

where COmax = maximum cardiac output

$ArtO_2$ = arterial oxygen content

$VenO_2$ = mixed venous oxygen content

and $COmax = Fcmax \times SV$ (equation 2)

where SV = stroke volume

While Fcmax is not affected by training, SV is.[3] Thus an athlete can achieve a higher CO than an untrained subject largely because of a greater SV (see equation 2). $ArtO_2$ (equation 1) content depends upon the efficiency of the respiratory system in exchanging gas and upon the haemoglobin concentration. $VenO_2$ (equation 1) content depends upon the oxygen extraction by the body. This is dependent upon the body's ability to redistribute blood away from the splanchnic circulation to the exercising muscles. The $VenO_2$ also depends upon local muscle blood flow and the type of muscle fibre. Slow twitch fibres are rich in mitochondria and are able to extract more oxygen than fast twitch white fibres. The proportion of the muscle fibre type is influenced by training. Despite these several variables, $\dot{V}O_2max$ appears to be quite similar between different population groups when the value is normalised for body size.

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