

Commentary

A perspective on tuberculosis & exercise testing

Despite the development of newer anti-tuberculosis drugs, tuberculosis is still a common disease, especially with poverty, crowding, HIV infections, and poor access to medical care. Rest is no longer a necessary part of therapy unless the patient is febrile and toxic. Six month and shorter treatment courses have replaced 18 months and longer periods of drug therapy. We still have a long way to go and multi-drug-resistant tuberculosis, as recently noted, remains a global menace^{1,2}. Yet, for example, in economically-privileged areas of the United States, positive tuberculin tests are rare. In the last decade while testing over 200 adults living in such parts of Los Angeles, I found only one positive tuberculin test, and that was in a European-born man in his 60s. Therefore, to reduce the worldwide threat of tuberculosis, we as physicians must also become better advocates for reducing warfare and starvation and better proponents for improving education, housing, nutrition, and economic opportunities for the destitute and underprivileged.

Current and future status of exercise testing

Sharma and Ahluwalia³ offer us cardiopulmonary exercise testing data from 14 patients early in their course of miliary tuberculosis. On follow-up exercise testing on 7 patients 9-12 months later they found only minimal improvement from their prior low exercise capacity. Not reported are the results of chest radiographs, ECG, or resting pulmonary function tests, *i.e.*, whether or not they have lung restriction or obstruction or loss of alveolar capillaries as a result of their tuberculosis. Presumably they have returned to somewhat normal living, but there is no evidence that they have

participated in a supervised rehabilitative exercise programme. To follow up this preliminary study, I hope that the authors will obtain lung function studies now and offer an intensive, supervised programme of rehabilitative exercise for at least two months, with encouragement to continue, followed later by repeat exercise testing.

In the last three decades, we and others have learned a great deal using exercise testing not only diagnostically, but also to serially evaluate patients with coronary artery disease, heart failure, obstructive and interstitial lung disease, and those who are candidates for consideration of heart and lung transplants⁴. Many studies have demonstrated that physical reconditioning and exercise programmes are important in maintaining and improving recovery. With all of these above diseases, and probably also with tuberculosis and pulmonary vascular disease, patients tend to deteriorate. Not only are the heart and/or lungs damaged by the primary disorder, but the detraining of musculoskeletal system from disuse causes further clinical deterioration of the patient. With detraining, muscles become weaker, fatigue increases, and the anaerobic threshold falls, so that exercise at a level that previously was totally aerobic and comfortable now becomes partly anaerobic and exhausting, resulting in the formation of more lactate ion with production of excess CO₂. Eliminating this excess CO₂ requires extraveilation with resultant dyspnoea.

In the past, tuberculosis and other infectious and interstitial diseases of the lung were thought to primarily affect the mechanics of breathing and gas exchange. But when air spaces are destroyed,

pulmonary capillaries are also destroyed and the resultant pulmonary vasculopathy increases the work of the right heart in pushing blood through a reduced pulmonary capillary bed, the resultant lowering of right ventricular output increases ventilation-perfusion mismatching and dead-space ventilation and usually further increases the ventilatory requirement.

Rehabilitation appears to increase capillarity and mitochondrial density of the peripheral muscles and increase the anaerobic threshold, which results in lesser work for the heart, and a reduced ventilatory requirement. The exact mechanisms and degree of improvement in patients with pulmonary vasculopathy remain to be determined.

Exercise testing is not only helpful diagnostically, but also in determining the specific pathophysiology of individual patients and the effects of therapy in patients with known cardiovascular or pulmonary diseases. For routine testing⁴, we recommend an exercise protocol with resting measures, 3 min of unloaded cycling or slow walking, and an incremental increase in work rate to maximal tolerance lasting from 6 to 14 min, and 2 or 3 min of recovery during which time the patient continues to move his/her legs slowly to avoid hypotension or light-headedness and reduce arrhythmias. During this protocol, while the ECG, blood pressure, gas exchange measures, and oximetry are observed, recorded, and integrated for later tabulation and graphing, the patient is observed for signs of inordinate distress or danger.

Such a study⁴ allows us to relatively quickly and non-invasively evaluate the exercising cardiovascular system with measurements of: (i) peak $\dot{V}O_2$ (maximal exercise capacity), (ii) pattern of change and peak O_2 pulse (the product of stroke volume and the difference in O_2 content between arterial and mixed venous blood), (iii) pattern and values of heart rate and blood pressure response, (iv) peak heart rate and heart rate reserve, (v) pulse-pressure product, (vi) anaerobic threshold (exercise capacity for sustained exercise), and (vii) increase in $\dot{V}O_2$ per Watt

increase in work rate (an index of the ratio of aerobic to anaerobic metabolism). We evaluate the ventilatory system by assessing: (i) breathing pattern (frequency and tidal volumes), (ii) ventilatory limitation from breathing reserve (maximal voluntary ventilation measured for 12 sec and peak ventilation, both in l/min), or encroachment of the tidal volume on the inspiratory capacity, or excess tachypnea, or excessively rising end-tidal PCO_2 values. We evaluate gas exchange by noting: (i) absolute levels and patterns of end-tidal PCO_2 and PO_2 , (ii) the levels and patterns of ventilatory equivalents for CO_2 and O_2 , and (iii) oximetry of the forehead, ear, or finger. When abnormalities of gas exchange are expected, we often obtain a single arterial blood after the anaerobic threshold is reached but well before the patient needs to stop exercise. We expect the use of such non-invasive cardiopulmonary exercise testing will increase as reliable manufactured equipment is now available, patient safety has been acknowledged, pathophysiology is better understood, and physicians realize the benefit of these evaluations.

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