Exercise Capacity

Definition

Exercise capacity is the maximum amount of physical exertion that a patient can sustain. An accurate assessment of exercise capacity requires that maximal exertion is sufficiently prolonged to have a stable (or "steady state") effect on the circulation and that the pattern of patient response is consistent when exertion is repeated.

Techniques

The assessment of exercise capacity is an essential feature of patient evaluation. A complete and accurate history is often the cornerstone of such an assessment. The history should reflect detailed and quantitative information about intensity and duration of maximal effort and specifics about the nature of limiting symptoms. This information will clarify the extent and impact of exertionally related problems and may also have valuable diagnostic and prognostic implications. It is a logical guide to further evaluation and appropriate therapy.

Exercise capacity can be evaluated by interview or by direct laboratory measurement. Both approaches are important, and they are complementary. Information from the patient’s history is immediately available and inexpensive. More significantly, this information defines exercise capacity in the context of the patient’s symptoms, lifestyle, and preferred mode of activity. Historical information is not contaminated by unsuitable forms of exercise or patient anxiety that might occur in an exercise laboratory. In practice, complete historical information about patient activity is very useful when planning exercise laboratory testing.

During the interview patients should be encouraged to be as specific as possible, describing their observations in relation to one or more particular, recent periods of physical activity. Several kinds of information must be elicited. Maximal effort must be defined in terms of intensity and duration of exertion. Quantification of intensity implies estimation of the rate of performing physical work. For example, a flight of stairs may be climbed at a run, a steady walk, or a leisurely pace, possibly interrupted by frequent stops. Thus, exercise performance can be fully characterized by total work output (e.g., 5 flights, 4 blocks) and rate (e.g., brisk walk, normal pace) or duration of effort (e.g., 4 minutes). Incidental information may also be important, particularly the precipitation of exertionally related symptoms. Ambient temperature, recent meals or medications, and emotional state can exert a crucial influence on the circulatory response to exercise and, consequently, alter the likelihood that a given intensity of effort will challenge cardiovascular reserves.

Perhaps the most important item of historical information related to effort is the character and time-course of exercise-induced symptoms, particularly symptoms that cause the patient to stop. Specific symptoms are discussed in detail later in this section. In general, the presence and pattern of effort-limiting dyspnea, chest discomfort, light-headedness, generalized or specific weakness, palpitations, or leg cramping have major, distinctive implications for patient management. Exertional symptoms usually occur in a crescendo fashion once a threshold exercise intensity has been exceeded; these symptoms resolve after a few minutes’ rest. One noteworthy exception is “walk-through” angina. In patients with this unusual condition, angina fades even as exercise persists, probably because of an initially excessive circulatory response to exercise that partially reverses during continuing steady, mild effort. Some symptoms appear only in the immediate postexercise period. Such symptoms likely reflect a greater awareness of somatic sensation when exercise ceases, as well as rapid shifts in hemodynamic and autonomic function in the recovery period. Response of exercise-induced symptoms to appropriate therapy (particularly the prevention of symptoms during effort) may have much clinical utility. A detailed and accurate account of exercise-induced symptoms will often reward the interviewer with remarkably precise indications of relevant pathophysiology and possibilities for effective therapeutic intervention.

Some ingenuity and imagination may be needed to arrive at reliable and meaningful information about exercise performance, especially in ordinarily sedentary patients. Specific, detailed inquiry should be made about physically demanding, common domestic activities (e.g., carrying laundry or groceries, chasing wayward children or pets, yard work, bed making, floor washing), job-related tasks (e.g., lifting, stair climbing, operating machinery), sexual intercourse, travel (e.g., hauling luggage), or sports. The right question can sometimes elicit vivid accounts of experiences during effort that might not otherwise be mentioned. In order to bracket maximal exercise capability, one must define what a patient can do (e.g., 1 flight or 2 blocks at a normal pace) as well what he cannot do (e.g., 2 flights or 4 blocks). Activity that can be sustained for a minute or more is most likely to yield reliable information because the circulation can begin to approach a steady state after this time period. Sustained activity may not be possible, however, in severely symptomatic patients.

Consistency of performance is important when one is defining exercise capacity. Such consistency should be sought by asking about several different kinds of activities. Extent of a patient’s limitation should be equally evident in several different contexts. Certain patients, however, do have fluctuations or inconsistencies in exercise capacity. To some degree, these can be explained by differences in incidental conditioning factors mentioned previously or differences in the character of the exertion (e.g., arm work versus leg work, untrained versus trained muscle groups, isometric versus isotonic effort). When discrepancies cannot be resolved, one may be compelled to define a range of exercise...
Exercise performance reflects a coordinated response of cardiovascular, pulmonary, and neural function along with the action of exercising muscles. Exercise induces graded increases in heart rate, arterial pressure, cardiac output, myocardial contractility, and rate and depth of respiration. Rapid, reversible shifts also occur in the distribution of blood flow and in blood composition. These changes are orchestrated by the central nervous system, which is responding to direct cortical input and to neural and humoral feedback from exercising muscles. Neural control of cardiovascular function is exerted, in part, through characteristic increases in sympathetic stimulation and reciprocal decreases in parasympathetic stimulation. During steady effort, cardiovascular and pulmonary parameters begin to stabilize (after 1 to 2 minutes of rapid change) at levels appropriate for a specific intensity of exercise in a particular patient. These steady-state levels can be used to characterize patient performance. Autonomic and other shifts may trigger pathologic as well as physiologic responses (e.g., exercise-induced arrhythmias or asthma).

Reduced exercise capacity may indicate dysfunction in any portion of the complex exercise response. Thus, a nonspecific symptom such as generalized weakness may reflect endocrine, metabolic, hematologic, neuromuscular, or psychological problems, as well as cardiovascular or pulmonary disorders. Since generalized weakness is so nonspecific, a particular underlying cause must be identified by its associated clinical manifestations.

Exercise capacity can (and, in many instances, should) be measured objectively by testing the patient in an exercise laboratory. Although it has certain limitations, such testing can have great value in selected patients. Maximum exercise performance can be characterized with respect to intensity and duration of effort, and these values and the associated limiting symptom can be compared to estimates obtained from the history. This performance index can be matched with prior performance by the same patient in order to quantify changes occurring in time or with therapy. It can also be matched with performance by other patients in order to assess comparability with other patient groups. Such information can be especially useful for the physician (and the patient) when historical information is unreliable or ambiguous. The latter is not infrequently the case when patients fail to push themselves to meaningful limits. Exercise intensity can be measured as external work (e.g., in watts) or as total body oxygen consumption (ml O2/kg/min).

When exercise is performed according to a generally accepted protocol with workload escalation at fixed intervals, the stage of exercise at termination can provide a summary estimate of exercise capacity. In general, exercise testing, like historical inquiry, seeks to define functional reserve by measuring maximal, symptom-limited exercise capacity. Exercise testing can also utilize a specific physiological end point, an age-related target heart rate, because this value is associated with full deployment of functional reserve.

In addition to objective quantification of exercise capacity and definition of limiting symptoms, laboratory testing can demonstrate important concomitant changes in physiologic parameters such as heart rate, blood pressure, cardiac output, left ventricular ejection fraction, arterial blood gas content, and ventilatory characteristics. It can also reveal (and characterize) electrocardiographic evidence of myocardial ischemia or arrhythmia. The coincidence of exercise-induced symptoms and specific physiologic derangements can go a long way toward identifying an origin, and possibly a remedy, for the patient's problem.
Clinical Significance

A history of limited exercise capacity may be important in characterizing and quantifying a clinically significant problem. A decreased exercise capacity should stimulate a search for a specific cause, usually guided by associated symptoms and signs. Control of exercise-induced symptoms is a major goal of treatment for many cardiovascular and pulmonary disorders. Thus, a complete and accurate assessment of the extent of limitation and its relation to the patient’s pattern of activity is often essential for intelligent selection and evaluation of a treatment program.

Aside from quantifying exercise limitation and its impact on lifestyle, an accurate estimate of exercise capacity may also have value in establishing diagnosis and prognosis. The presence of stable, consistent limitation of exercise implies a static defect in anatomy or physiology. Under these circumstances, measurement of cardiac or pulmonary function at the onset of symptoms will often reveal characteristic changes. In contrast, dyspnea or ischemic-type chest pain occurring unpredictably and not associated with impaired exercise capacity suggests a much more evanescent abnormality such as paroxysmal arrhythmia, bronchospasm, or coronary spasm.

Prognostic implications of reduced exercise capacity can also be very significant. In aortic valve disease (particularly aortic stenosis) heart-related exercise limitation identifies patients at high risk of cardiac death. Regardless of lack of interference with lifestyle or favorable symptomatic response to medical therapy, such patients are generally considered appropriate candidates for operative intervention to prevent cardiac death. A somewhat analogous situation exists in certain subgroups of patients with coronary artery disease. In selected individuals, marked impairment in exercise capacity (e.g., Canadian Cardiovascular Society class III or IV) suggests a relatively high mortality that is likely to improve with a coronary artery bypass procedure, whereas those with better exercise capacity have a lower mortality and lesser likelihood of improved prognosis after operation. Hence, assessment of exercise capacity of patients with aortic valve disease or coronary artery disease offers important clues as to long-term outlook as well as optimal management. It is noteworthy, however, that exercise capacity is not a reliable guide to prognosis for all cardiac patients. In individuals with dilated cardiomypathy, left ventricular ejection fraction is a much more reliable predictor of mortality. Patients with markedly reduced ejection fractions and relatively preserved exercise capacity are at approximately equal risk of early cardiac death as patients with similar ejection fractions and more impaired exercise capacity.

A detailed, searching, and sympathetic discussion with a patient can often yield much useful information about the patient’s exercise capacity. The interviewer must be aware, however, of the limitations of this approach. Patient anxiety, depression, or other potent motivating factors can lead to unconscious denial, distortion, or exaggeration of exercise-related symptoms. In certain patients, symptoms may present themselves in such a way that their character cannot be defined with confidence. Other patients never perform tasks likely to reveal meaningful information about exercise capacity for reasons that are not directly linked to disordered physiology. Thus, the wise clinician will seek corroborative data by objective exercise testing. Under laboratory conditions (and with appropriate patient practice) symptoms appearing vague, inconsistent, or atypical by history are often revealed as distinct, consistent, or typical when reproduced in the course of testing. Correlative changes in physical findings, electrocardiographic findings, and hemodynamic parameters can also clarify the origin and character of otherwise obscure problems. Thus, the exercise laboratory should be viewed as a useful and occasionally indispensable supplement to the interview in the assessment of exercise capacity.

Some patients are unsuited for exercise testing. Such testing is inappropriate when no useful information is likely to emerge, that is, when the clinical situation is fully defined by other findings or when exercise performance is impaired by extraneous factors. Exercise testing, particularly symptom-limited testing, is contraindicated due to safety considerations in patients with symptomatic critical aortic stenosis, unstable angina, very recent myocardial infarction, frequent malignant ventricular arrhythmia, or other conditions that might lead to hemodynamic instability.

References


