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ACUTE CARDIOVASCULAR RESPONSE TO EXERCISE

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IT is difficult to define a generalized cardiovascular response to exercise. The cardiovascular system is one component of an integrated mechanism that functions to compensate for the increased metabolic demands of exercise (Figure 1), and the performance of each component is influenced by the action of the others. Cardiac disease, abnormalities of the oxygen-carrying capacity of the blood (due to anemia, altered hemoglobin etc.) and differences in the degree of peripheral oxygen extraction that occur as a function of physical training all influence the circulatory system. This paper reviews the cardiovascular response to various types of exercise in patients without cardiovascular disease, and discusses the continuing controversy over regulation of the cardiovascular response to exercise.

Static versus dynamic exercise. Exercise has been considered either predominantly static (isometric) or dynamic (isotonic/aerobic). The term static implies a lack of movement of body parts during muscle contraction. Dynamic exercise connotes movement of a large muscle mass in a rhythmic

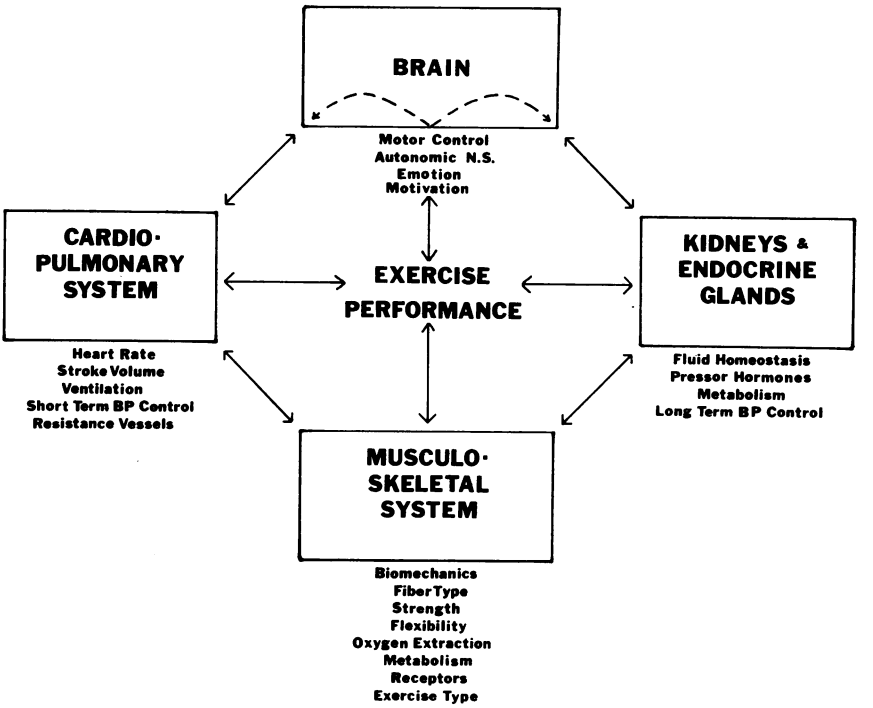

Fig. 1. Factors influencing exercise performance. The function of each system during exercise depends upon the function of the others, and the performance of exercise will influence the activity of each system.

manner. Static exercise usually entails a moderate or greater degree of skeletal muscle contraction, while dynamic exercise involves low intensity of skeletal muscle contraction.

The hemodynamic response to exercise depends in part on whether the exercise is predominantly static or dynamic in nature.[1] Static exercise is characterized by a rise in systolic, diastolic and mean *arterial* blood pressure. In contrast, mean arterial pressure shows only modest increases during dynamic exercise, and the pulse pressure often increases dramatically as a function of an increased systolic pressure and decreased diastolic pressure. This blood pressure response is due to decreased peripheral vascular resistance during dynamic exercise that is lacking in predominantly static exercise. The decrease in total peripheral resistance during dynamic exercise is proportional to the exercising skeletal muscle mass.

It is important to note that most forms of exercise are not exclusively static or dynamic in nature. Blomqvist showed a gradual transition from "static"

to "dynamic" response as a function of increasing active muscle mass.[2] Conversely, exercise which is predominantly dynamic in nature may take on increasing characteristics of static exercise as a result of increased intensity of muscular contraction. For example, high repetition/low resistance work with weights, while predominantly a dynamic exercise, has both static and dynamic components. Lifting progressively heavier weights results in an increasing amount of static exercise and decreasing component of dynamic exercise.

Effect of exercise modality. The circulatory response to exercise also depends on the exercise modality (i.e., running, bicycle riding, etc.) used.[3] This is particularly applicable for arm versus leg exercise. Studies have shown that heart rate and blood pressure rise more steeply in relation to work intensity during arm exercise than leg exercise.[4,5] The explanation for this may include the fact that arm exercise is less mechanically efficient (as judged by the ratio between external work and systemic oxygen uptake) and involves a smaller muscle mass than lower body work. Relative contributions of dynamic and static work and the effect of posture may also contribute to different hemodynamic responses for different types of exercise and exercise modalities.

Influence of the musculoskeletal system. Muscle atrophy,[6] poor flexibility or lack of coordination may significantly affect cardiovascular responses to exercise. If a limb or body segment is weak, muscles must contract at a greater percentage of their maximum to do a set amount of work. This increased intensity of contraction evokes a greater cardiovascular response,[7] and theoretically increases the work of the heart for a given amount of external work. Lack of coordination means that more muscular work must be done to accomplish a given amount of external work, thus increasing demands on the cardiovascular system. Similarly, a patient with reduced flexibility usually requires greater energy expenditure to initiate movement than one with a flexible musculoskeletal system.

These factors do not readily lend themselves to clinical evaluation, and the magnitude of their effect on the cardiovascular system has not been adequately studied. We are currently examining patients with unilateral leg weakness secondary to injury to assess the effect of this variable on their cardiovascular responses to exercise. Such research may indicate whether cardiac rehabilitation of patients with significant leg weakness should include therapy specifically directed toward strengthening the weakened limb.

Changes in cardiac output during exercise. Both static and dynamic ex-

ercise increase cardiac output in normal patients. The increase in cardiac output is a product of changes in heart rate and left ventricular stroke volume during exercise. Dynamic exercise is characterized by a large increase in cardiac output, while static exercise usually results in a more modest increment.

The most important determinant for the increase in cardiac output is the exercise-related change in heart rate.[8] There is a linear increase in heart rate with progressively increasing workloads until a maximal heart rate is attained. Maximal dynamic exercise causes a significantly greater rise in heart rate than maximal static exercise, in large part accounting for the different magnitudes of increase in cardiac output during the two types of exercise.[9] The patient's age[10] and degree of conditioning[11] both affect heart rate response to exercise, the maximal heart rate attained varying inversely with increasing age and training. The type of exercise performed (i.e., arm versus leg) also significantly influences the observed heart rate response to exercise.[5,11,12] The heart rate response for arm exercise is significantly higher than for leg exercise at an equivalent workload.[5]

Changes in left ventricular stroke volume are more variable. In contrast to heart rate, there is no consistent exercise-related increase in stroke volume.[9] Stroke volume is influenced by the net effect of changes in contractility (related to elevated blood catecholamines), afterload (the tension which must be generated for ejection of blood from the left ventricle), heart rate and preload (the end-diastolic stretch of the left ventricle). Each factor in turn is influenced by the degree of exertion and the type of exercise performed, determining whether an increase, decrease or no change in stroke volume occurs.[4,13] For example, left ventricular preload is affected by posture, changes in venous pooling and mechanical venous compression by muscle contraction during exercise. The effect of posture on preload accounts for the fact that stroke volume increases less during supine exercise than when the same exercise is performed in an upright position.

Regulation of the cardiovascular response to exercise

Neural control. Neural regulation of the cardiovascular response to exercise is the subject of controversy. The central control or cortical radiation theory maintains that the hemodynamic response is a function of the connections between the motor cortex and cardiovascular control centers in the brainstem.[14] Support for this idea comes from the observation that a person trying to contract muscles in a paralyzed limb will manifest an increase

in heart rate and blood pressure.[15] The peripheral afferent theory[16] maintains that the brain stem is primarily influenced by input from receptors located within the exercising skeletal muscle. It appears that these receptors are not pain, mechano or joint receptors, and can be stimulated by autocoids such as bradykinin.[17-21]

Mitchell and coworkers demonstrated that an increase in blood pressure and heart rate resulting from motor nerve stimulation to the dog hind limb was abolished by sectioning the dorsal spinal nerves (which carry the afferent impulses), supporting the concept that neural input from the periphery has a significant influence on the cardiovascular response to exercise.[22] In addition, Petrofsky showed that the type of muscle that is stimulated affects this reflex response.[23] He demonstrated that stimulation of slow twitch (type I) skeletal muscle resulted in only small increases in blood pressure, while stimulation of fast twitch (type II) skeletal muscle more significantly increased blood pressure.

Some evidence suggests that peripheral musculature may have a direct neural link to control of the coronary circulation. Mitchell's laboratory showed that skeletal muscle contraction in the dog hind limb can result in alpha coronary vasoconstriction, an effect especially prominent in a beta-blocked preparation.[24]

Hormonal control. Hormonal control may also play an important role in regulating cardiovascular response to exercise. Progressive increase in exercise intensity is associated with a nonlinear increase in plasma lactate content. The degree of exertion at which the curve breaks and the rise in lactate concentration increases has been referred to as the "lactate" or "anaerobic" threshold (Figure 2), but the physiological significance of this concept is unclear.[25]

Recent observations indicate that this point coincides with hormonal changes that may have an important role in regulating the cardiovascular response to exercise. Plasma catecholamine levels during exercise parallel the increase in mixed venous blood lactate content, with a significant rise at the "lactate threshold."[26,27] Our laboratory recently demonstrated that plasma renin levels also show no significant exercise-related increase until exercise beyond the lactate threshold (Figure 2).[28] These studies suggest that the physiological significance of this degree of work is that it represents a "vasopressor threshold" beyond which elevated levels of pressor hormones prevent further decreases in total peripheral resistance from progressive vasodilation within exercising skeletal muscle. The augmented release of

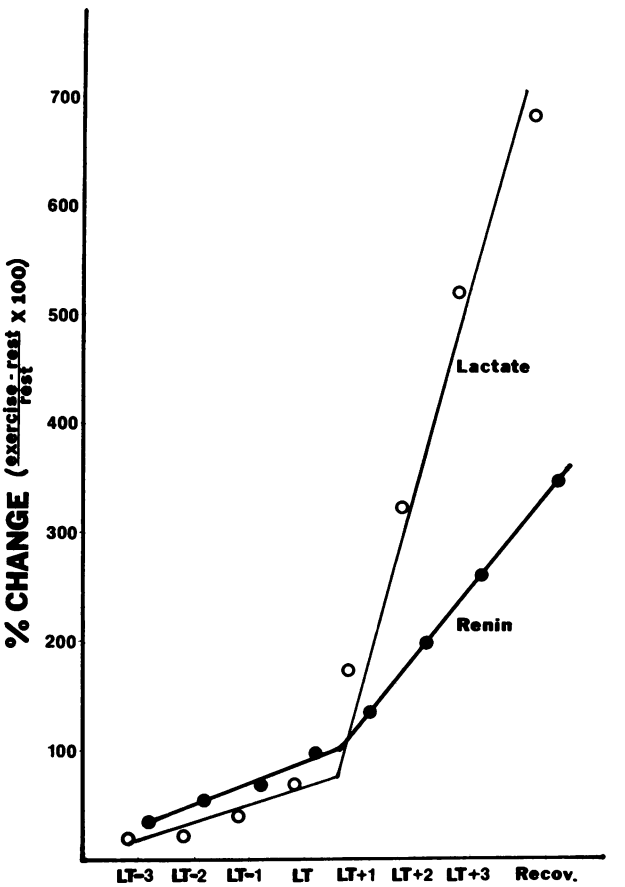

Fig. 2. Increase in plasma lactate and renin during exercise. Both plasma lactate (open circles) and plasma renin (dark circles) show a sudden increase after performance of a certain degree of dynamic exercise. This workload can be termed the "lactate threshold" or the "vasopressor threshold." Plasma catecholamines, not depicted on this graph, also manifest an increase at this point. LT=Lactate threshold; LT-1=1 stage prior to lactate threshold; LT+1=1 stage after lactate threshold; Recov.=recovery period.

pressor hormones may further increase resistance in other nonexercising areas of the body so that mean arterial pressure can be maintained.

SUMMARY

It is not possible to define a generalized acute cardiovascular response to exercise. Cardiovascular performance depends upon the interaction of a number of factors that function to meet the increased metabolic needs of the body during the stress of exercise. In addition, such variables as the type of exercise, degree of training and peripheral muscle factors are all important determinants of cardiovascular response.

Controversy over control of the cardiovascular system during exercise continues, and there is evidence for both central and peripheral neural input. In addition, such humoral factors as exercise-induced changes in catecholamines and pressors may also have an important role in regulating the cardiovascular response to exercise.

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