UNIT 1---- CONTINUED

Long-Term Adaptations to Exercise Training

Cardiovascular and Respiratory Adaptations

Endurance training leads to significant cardiovascular and respiratory changes at rest and during steady-state exercise at both submaximal and maximal rates of work. The magnitude of these adaptations largely depends on the person's initial fitness level; on mode, intensity, duration, and frequency of exercise; and on the length of training (e.g., weeks, months, years).

Long-Term Cardiovascular Adaptations

Cardiac output at rest and during submaximal exercise is essentially unchanged following an endurance training program. At or near maximal rates of work, however, cardiac output is increased substantially, up to 30 percent or more (Saltin and Rowell 1980). There are important differences in the responses of stroke volume and heart rate to training. After training, stroke volume is increased at rest, during submaximal exercise, and during maximal exercise; conversely, posttraining heart rate is decreased at rest and during submaximal exercise and is usually unchanged at maximal rates of work. The increase in stroke volume appears to be the dominant change and explains most of the changes observed in cardiac output.

Several factors contribute to the increase in stroke volume from endurance training. Endurance training increases plasma volume by approximately the same percentage that it increases stroke volume (Green, Jones, Painter 1990). An increased plasma volume increases the volume of blood available to return to the right heart and, subsequently, to the left ventricle. There is also an increase in the end-diastolic volume (the volume of blood in the heart at the end of the diastolic filling period) because of increased amount of blood and increased return of blood to the ventricle during exercise (Seals et al. 1994). This acute increase in the left ventricle's end-diastolic volume stretches its walls, resulting in a more elastic recoil.

Endurance training also results in long-term changes in the structure of the heart that augment stroke volume. Short-term adaptive responses include ventricular dilatation; this increase in the volume of the ventricles allows end-diastolic volume to increase without excessive stress on the ventricular walls. Long-term adaptive responses include hypertrophy of the cardiac muscle fibers (i.e., increases in the size of each fiber). This hypertrophy increases the muscle mass of the ventricles, permitting greater force to be exerted with each beat of the heart. Increases in the thickness of the posterior and septal walls of the left ventricle can lead to a more forceful contraction of the left ventricle, thus emptying more of the blood from the left ventricle (George, Wolfe, Burggraf 1991).

Wilmore JH, Costill DL. Physiology of sport and exercise. Champaign, IL: Human Kinetics, 1994,
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Endurance training increases the number of capillaries in trained skeletal muscle, thereby allowing a greater capacity for blood flow in the active muscle (Terjung 1995). This enhanced capacity for blood flow is associated with a reduction in total peripheral resistance; thus, the left ventricle can exert a more forceful contraction against a lower resistance to flow out of the ventricle (Blomqvist and Saltin 1983).

Arterial blood pressure at rest, blood pressure during submaximal exercise, and peak blood pressure all show a slight decline as a result of endurance training in normotensive individuals (Fagard and Tipton 1994). However, decreases are greater in persons with high blood pressure. After endurance training, resting blood pressure (systolic/diastolic) will decrease on average -3/-3 mmHg in persons with normal blood pressure; in borderline hypertensive persons, the decrease will be -6/-7 mmHg; and in hypertensive persons, the decrease will be -10/-8 mmHg (Fagard and Tipton 1994).

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