Chapter 13

PHYSICAL FITNESS AND PHYSICAL TRAINING FOR MILITARY PERFORMANCE

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INTRODUCTION

Despite the rapid modernization of weapon systems and technological advancements in war-fighting capability, the military profession remains physical in nature. Considerable emphasis is placed on physical training, physical capacity, and ultimately, on physical readiness, regardless of the specific role or occupation that the service member may fill. Irrespective of peacetime or wartime, service members must be prepared to defend themselves and others about them, and to react to emergencies as they may occur.

Like other organizations dealing with emergencies, such as police and firefighters, the military services emphasize physical fitness for purposes of discipline, morale, health maintenance, and physical appearance in addition to the traditional role of meeting the physical demands of the job. Thus, despite the many changes toward automation, mobility, and weapons of mass destruction, the military remains a physical force with an emphasis on high levels of fitness through formal physical training programs. Fitness was highlighted and emphasized to all the military services in 1981 when the Department of Defense (DoD) issued a new directive on physical fitness and weight control. The directive states:

Physical fitness is a vital component of combat readiness and is essential to the general health and well-being of armed forces personnel. Individual members must possess the stamina and strength to perform successfully any potential mission. These qualities, together with weight control, form the basis of the DoD physical fitness program.[1]

An understanding of physical fitness, and physical performance and its development through physical training should be an integral part of the training of military physiatrists. An understanding of normal muscle and exercise function must be present before one can repair, treat, and rehabilitate the injured.

The term "physical fitness" is an often abused term that requires some attention and definition. Ability to perform physically demanding tasks is a function of two groups of factors: (1) factors that determine the capacity for muscular contraction, and (2) factors that relate to the neural control of body movement. The latter, which may be most correctly referred to as *motor fitness,* includes the components of neuromuscular control, such as coordination, speed, agility, and skill, which are achieved primarily through repeated practice. The first group of factors, commonly referred to as *physical fitness*, are those associated with the energy generating capacity for muscular exercise. *Physical fitness* is developed through physical-training-induced physiological adaptations and will be the primary emphasis of this chapter. Thus, we define and use the term physical fitness as the *energy generating capacity to perform physical effort*.

Three distinct, but overlapping, energy generating systems for muscular exercise form the common categories of physical fitness: (1) stored energy located in the muscle cell mitochondria in the form of high energy phosphagens that are associated with *muscle strength*, (2) rapidly produced energy in the form of phosphagens generated by the anaerobic process of glycolysis and associated with *muscular endurance* (or muscular power or anaerobic power), and (3) more slowly produced energy in the form of phosphagens derived from the aerobic metabolism of various substrates and associated with *aerobic power* (or cardiorespiratory endurance). Because each category of fitness involves a distinctly different energy generating metabolic pathway, each also requires different approaches for development through training. This chapter will discuss the capacity for exercise, physical performance, and training adaptations in the context of these three categories of physical fitness or energy generating systems.

STRUCTURE AND FUNCTION OF SKELETAL MUSCLE

Physical activity is the result of skeletal muscle producing force through the process of contraction. This contraction, which produces a pulling action against bone, causes a body segment to move about its joint axis and thereby produces movement. Body movements are actually the result of the coordination between the muscles that are shortening (contracting) and those that are opposing the movement by relaxing (referred to as the antagonist muscles). Most muscular activity consists of the highly complex coordination of a number of muscles or muscle groups.

When muscles contract, they do not always shorten. There are three types of muscular contrac-

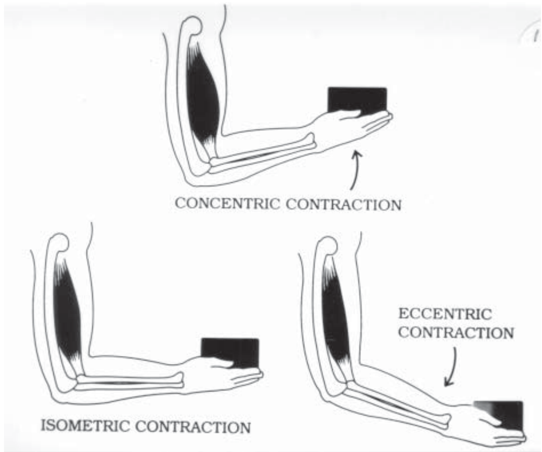

Fig. 13-1. The three types of muscular contractions.

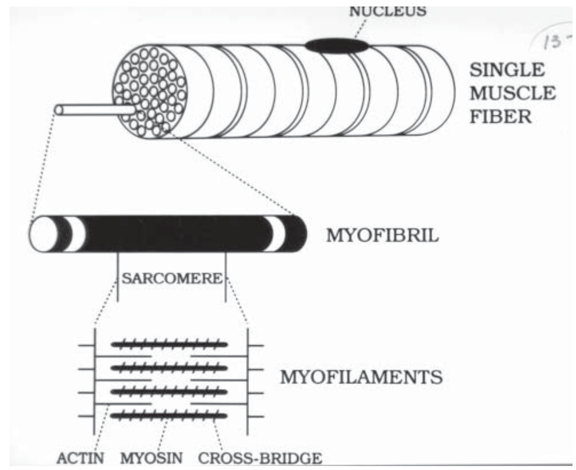

Fig. 13-2. Structure of the skeletal muscle fiber.

tion: (1) shortening (*concentric*) contractions, (2) static (*isometric*) contractions, and (3) lengthening (*eccentric*) contractions. Many movements, such as walking or lifting and lowering a weight, will involve all three types. These are illustrated in Figure 13-1. Only in the first case, concentric contraction, does the muscle actually shorten. In the case of a static contraction, the force generated equals the resistance to movement and, therefore, no movement takes place. In eccentric contractions, the external resistance overcomes the force developed by the muscle and the muscle is actively stretched, such as in the controlled lowering of a weight to the floor.

Muscle Structure and Contractility

Skeletal muscle is composed of muscle tissue, as well as its nerve and vascular supply, and some connective tissue, and is attached to bone at each end by tendons. The muscle tissue itself is composed of muscle cells, commonly referred to as muscle fibers due to their long and slender shape. The structural makeup of the muscle fiber is illustrated in Figure 13-2.

Each muscle fiber is composed of numerous myofibrils lying parallel to each other along the long axis of the fiber. Each myofibril is composed of a series of sequential identical units referred to as sarcomeres. Each sarcomere can independently contract. Simultaneous contraction of all sarcomeres results in tension being generated by the fiber. Each sarcomere, in turn, is composed of two types of protein filaments: a thicker filament of the protein myosin and a thinner filament of the protein actin. The partial overlapping of these filaments causes the appearance of bands on the myofibril.

Muscular contraction is produced by the sliding of the thin filaments past the thicker filaments in a telescopic manner as illustrated in Figure 13-3, thus shortening the sarcomere. Although not precisely known, it is believed that the sliding of the filaments is caused by protruding structures from the myosin molecules called cross-bridges. These cross-bridges have an affinity for actin and contain the enzyme, myosin adenosine triphosphatase (ATPase), which hydrolyzes high energy phosphates to release their energy for the contractile process. In the resting state this affinity, and therefore the interaction between the two filaments, is blocked by two additional proteins (troponin and tropomyosin) found in the thin filaments. This inhibition is released by calcium ions (Ca^{++}). As Ca^{++} binds to troponin and tropomyosin, the actin is freed to bind

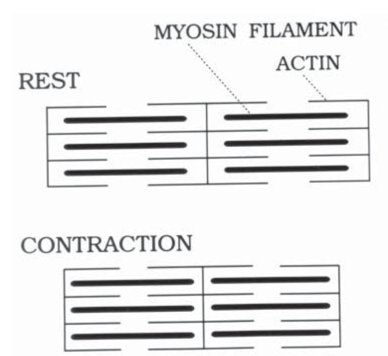

Fig. 13-3. Depiction of the sarcomere shortening process during muscular contraction.

with myosin. The energy then released through the hydrolysis of adenosine triphosphate (ATP) causes the cross-bridges to pull the filaments past each other, possibly in a ratchet-like motion. Muscle contraction is initiated by an increased concentration of Ca^{++} in the myofibril to initiate this chain of events. The reader is referred to Astrand and Rodahl[2] for further discussions of the mechanisms of sarcomere shortening and force development within the muscle fiber.

Fiber Types

Skeletal muscle fibers are not homogenous. Human skeletal muscle is composed of two distinct populations of fibers that differ in terms of chemical makeup and contractile characteristics: type 1, commonly referred to as slow twitch fibers; and type 2, fast twitch fibers. Type 2 fibers may also be divided into two or three subtypes.

Type 1 fibers are functionally characterized by a relatively long buildup time to peak tension (80-100 ms). This build up is the result of the low number of fibers innervated by each neuron (motoneuron). Type 1 fibers have high mitochondrial and capillary densities that result in their high capacity for oxidative phosphorylation. Thus, they are particularly suited for aerobic activities and are relatively resistant to fatigue. Type 2 fibers, in contrast, are characterized by fast rise time to peak tension (40 ms), and having a high fiber-to-motoneuron ratio, reach relatively high peak tensions. These fibers are characterized by high concentrations of the anaerobic glycolytic enzymes, such as ATPase. They are particularly suited for brief, intense periods of activity but are rapidly fatigued.

Type 2 fibers can be further divided into the subtypes 2a and 2b, based on their enzyme content and activity. Type 2a fibers tend to be biochemically intermediate between 2b and 1. The reader is referred to the review by Saltin and Gollnick[3] for more detailed descriptions of fiber characteristics. These fiber types can be distinguished by the strength with which the enzyme myofibrillar ATPase binds to myosin as detected with histochemical staining (Figure 13-4).

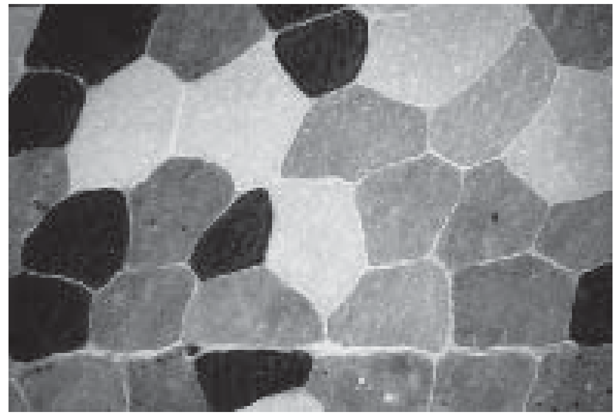

Fig. 13-4. Muscle cross-section showing different fiber types by the density of histochemical staining for ATPase. Darkest fibers are type 2, lightest are type 1, and intermediate density are types 2a and 2b.

Fiber Type Distribution

The relative distribution of type 1 and 2 fibers varies between muscle groups and between individuals. While major muscles of propulsion, such as the vastus lateralis and gastrocnemius, are, on average, approximately evenly divided between the two fiber types, postural muscles, such as the soleus that sustain tension over long periods, have about 70% of type 1. While muscles such as the vastus lateralis are, on average, evenly distributed between the two types, individual differences range from 10% to 95%.[3] For example, high ratios of type 1 are often found in elite endurance athletes, while elite sprinters and weight lifters typically show high type 2 fiber counts. Their athletic success appears to be related in part to their natural endowment with a particular fiber type. Fiber typing has been employed to predict potential success in a particular sport.

The proportion of fiber types in an individual is genetically determined. Fiber type is dictated by its innervation, which is established early in fetal development. A particular motoneuron innervates only one type of fiber. A fiber retains its nominal characteristics throughout life, and, therefore, the relative distribution of types remains unchanged. Intense physical training will cause a fiber's chemical and contractile characteristics to partially shift toward that favored by the type of training, but this shift is temporary and lasts only as long as the training stimulus is continued.[3]

Neural Control of Muscle Activity

Muscle Innervation

Voluntary skeletal muscle function is controlled by the motor unit. The motor unit is composed of the anterior (alpha) efferent motoneuron cell; its

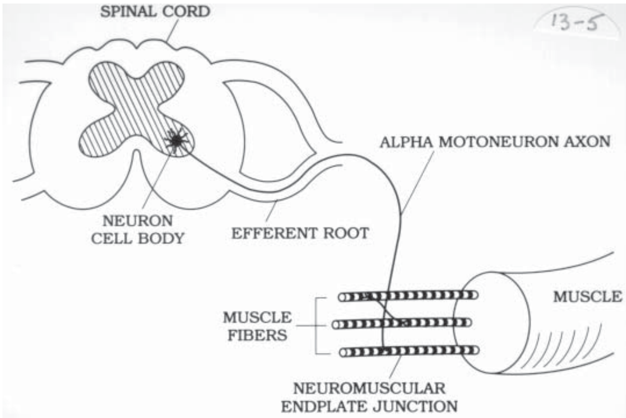

Fig. 13-5. The motor unit.

axon, the motor endplate, and the muscle fibers innervated by it (Figure 13-5). Each neuron innervates multiple fibers, but all of the same type. Muscles involved in fine movements have low neuron-to-fiber ratios, while muscles concerned with gross movements have high ratios. The efferent motoneuron has a corresponding sensory afferent nerve unit consisting of a muscle spindle (sensory receptor) and sensory neuron that synapses with interneurons in the spinal cord, which in turn, synapse with the efferent neuron. This is referred to as the *reflex arc*. The reflex arc provides the feedback and control that many physical movements require.

To produce muscle contraction, the nerve impulse emanates from the brain or spinal cord and travels along the neuron to the muscle across an interface referred to as the motor endplate or neuromuscular junction. Transfer of the electrical signal from the nerve across the junction is accomplished by the chemical (neurotransmitter), acetylcholine. Acetylcholine is stored in, and then released by the nerve signal from, sac-like vesicles at the end of the terminal axons. Acetylcholine released into the interface enters the postsynaptic membrane on the surface of the muscle fiber, causing it to depolarize by allowing the movement of sodium and potassium ions. Depolarization then spreads along the entire fiber to produce the contraction. Repolarization begins when the acetylcholine is destroyed by the enzyme cholinesterase. The end products are used to resynthesize acetylcholine.

Force Development

A single nerve impulse along the alpha motoneuron will produce an action potential that spreads to all of the innervated fibers resulting in a twitch, a rise in muscle tension followed by relaxation. If consecutive action potentials reach the muscle before relaxation is complete, contraction will occur again, but it will start at a higher level and rise each time to a higher tension, called *summation*. At high rates of stimulation, no relaxation occurs and the tension remains constant (tetanus), creating a state of mechanical fusion of the contractions (Figure 13-6).

Initial tension produced during stimulation of a muscle must first overcome the resistance produced by certain components of the muscle, particularly the connective tissue, tendons, and elasticity in the cross-bridges. This resistance is referred to as the *series elastic component* and must be overcome before external force is produced. The series elastic component is comparable to a spring that must be stretched before an action takes place.

Muscle Control

Neural control of muscle contractile force can be brought about in two ways: (1) by adjusting the frequency or the pattern of stimuli and (2) by adding additional motor units. As pointed out earlier, increasing the rate of action potentials along a motor unit will produce increased tension up to tetanus. The more important mechanism, however, is through the addition of more motor units, referred to as *motor recruitment*. High muscle forces can be generated by bringing into play numerous motor units. The production and regulation of tension is a complex function of integrating the rate of action potentials, recruitment of motor units, the type of fibers being recruited, and the synchrony of the firing pattern. For example, in weight lifting there is typically a synchronous pattern (simultaneous firing) of fast twitch motor units firing. Running, on the other hand, is more typically asynchronous, where some units, predominantly slow twitch, are firing while others are recovering.

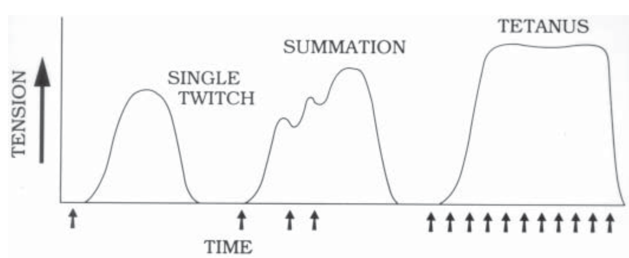

Fig. 13-6. Muscular tension during twitch, summation, and tetanus.

Muscle Fatigue

The term *fatigue* might best be defined, in reference to physical activity, as the loss of muscular power or exercise capacity. If maximal voluntary force is exerted, that maximal force output can only be maintained for a few seconds before the fiber mitochondria are depleted of their phosphagen stores and the tension begins to decrease, thus, "fatigue." If an individual runs at the level of his maximum oxygen consumption (VO_2max), that exercise intensity can only be maintained for 5 to 10 minutes after which he must stop or reduce the intensity level, again referred to as a loss in capacity or fatigue.

The cause and location of fatigue depend on the nature of the muscular activity. In most cases, the contractile mechanism within the muscle is implicated, rather than the neural signal to the muscle. In some cases of very intense effort when all motor units are presumably activated, decreases in action potentials or failure for the action potential to cross the neuromuscular junction have been detected. More common, however, is the situation where the neural impulse to the muscle is maintained, but failure occurs in the fibers. This loss in contractile tension in the fibers is most often the result of either insufficient energy substrate or insufficient blood flow to the muscle tissue. The latter impairs oxygen (O_2) delivery to the cells and the removal of the metabolic end-products, water, and heat. These conditions will interfere with the contractile mechanism of the myofibrils by preventing the repletion of the high energy phosphate stores, interfering with the energy transfer process, or disrupting the depolarization of the myofibrils, or both. In the case of an intense isometric contraction, pressure builds in the tissue through accumulation of fluid so that tissue pressure exceeds arterial pressure and impedes blood flow in and out of the muscle, resulting in anoxia and failure of the fibers to maintain tension. Causes of fatigue in rhythmic and whole body exercise will be discussed in later sections.

Energy Transformation Within the Muscle

As already pointed out, the energy required to cause the filaments to slide together for muscle to shorten and produce tension is derived from high energy phosphate bonds provided by ATP. ATP consists of a molecule of adenosine linked to three phosphate groups. The two linking bonds of the outermost group are referred to as high energy bonds because of their high potential energy. This potential energy is released to myofibril proteins when ATP is hydrolyzed, breaking the outer bond, and leaving adenosine diphosphate (ADP). This energy can be released immediately from ATP in the cells' mitochondria without the presence of O_2. Such stored ATP is the immediate source of energy for the initiation of exercise or a brief exercise bout, such as the lifting of a weight. However, the amount of stored ATP is sufficient for only a few seconds of maximal contractions. For muscle contraction to continue, the ATP must be resynthesized. Energy for this resynthesis comes from another energy-containing phosphate compound, creatine phosphate (CP), which is much more plentiful in the cell.

Hydrolysis of CP provides the energy for the regeneration of ATP from ADP. Energy for this transfer of phosphate bonds (phosphorylation) comes from the oxidation of carbohydrates, fats, and proteins in consumed foods. During this oxidation process, hydrogen atoms are removed from these nutrient substrates. Within the cells' mitochondria, electrons are removed from these hydrogen atoms and passed by electron receptors to molecular O_2 to produce the energy for ATP resynthesis. These electron receptors are the coenzymes, nicotinamide adenine-dinucleotide (NAD^+) and flavin adenine dinucleotide (FAD). These coenzymes are found in the B vitamins, niacin and riboflavin, respectively. They each gain hydrogen and electrons to become NADH and $FADH_2$, respectively. Electrons carried by these molecules are then passed along through a series of five iron-protein electron carriers, the cytochromes. The last cytochrome passes the electrons to molecular O_2. During this electron transfer process, chemical energy is trapped in the formation of the high energy phosphate, ATP. Over 90% of ATP formation takes place during this electron transfer process, referred to as oxidative phosphorylation. The reaction can be represented as follows:

$$\text{NADH} + \text{H}^+ + 3\ \text{ADP} + 3\ \text{P} + \tfrac{1}{2}\ \text{O}_2 \longrightarrow \text{NAD}^+ + \text{H}_2\text{O} + 3\ \text{ATP}$$

It is thus apparent that for the resynthesis of ATP and muscular contraction to proceed beyond a few seconds, the muscle cells must have available to them the nutrient substrates, enzymes, and coenzymes for electron transfer, as well as O_2. The lack of any of these during the energy generating process will result in inability to continue furnishing the fibers with energy except through the more limited and temporary pathway of anaerobiosis. The latter leads to the formation of lactic and pyruvic acid, which must eventually be oxidized or reconverted to glucose or glycogen.

PRINCIPLES OF EXERCISE PHYSIOLOGY

Energy Transfer in Exercise

Dynamic exercise provides the greatest demand for the expenditure of energy by the human body. During such exercise, the ability to produce power through energy transformation represents the most important consideration for physical performance. In events that feature the expression of maximal power, for example, maximal lifting or pushing, the energy output from the exercising muscles may be as much as 120-fold greater than from the muscles at rest, with the energy coming almost entirely from the stored phosphagens. During less intense but sustained exercise, such as long distance running, the energy requirement may be 20- to 30-fold above a resting condition. However, in this situation virtually all of the energy is provided by aerobic metabolic processes, and the power output is in the range of 25% to 30% of maximal potential power. Therefore, the relative contribution of the body's various means for energy transfer can differ markedly, depending on the intensity and duration of exercise, and the power capacity (fitness) of the participant. The stored phosphagens, anaerobic glycolysis, and the aerobic or oxidative system are the energy systems which support the wide range of physical activities and power outputs of which humans are capable.

Immediate Energy: The Phosphagen System

Physical activities of short duration and high intensity, where the power developed by the exercising muscle is near or at maximal level, require an immediate and rapid supply of energy. This energy is provided almost exclusively from the high-energy phosphates, ATP, and CP, stored within specific muscle fibers and activated during the exercise.

Approximately 5 mmol of ATP and 15 mmol of CP are stored within each kilogram of muscle.[4] For a 70-kg person with a muscle mass of 30 kg, this represents between 570 and 690 mmol of high-energy phosphates. If 20 kg of muscle are activated during exercise, then there is sufficient stored phosphate energy to walk briskly for about 1 minute, run a cross-country race for 20 to 30 seconds, or perform sprint activities for about 6 seconds.[5]

All types of physical activity require the utilization of high-energy phosphates, but many, as stated above, rely almost exclusively on those available from muscle stores. For longer duration exercise and for recovery from maximal exertion, additional energy must be generated for the replenishment of ATP.

Short-Term Energy: Anaerobic Glycolysis

For strenuous exercise to continue beyond a brief period of time, high-energy phosphates must be continually resynthesized at a rapid rate. During exercise lasting beyond a few seconds, energy to phosphorylate ADP comes mainly from glucose and stored glycogen during the anaerobic (O_2 lacking) process of glycolysis, with the resulting formation of lactic acid. This allows for the rapid formation of ATP by substrate-level phosphorylation, even though the O_2 supply is inadequate or the energy demands outstrip the capacity for ATP resynthesis aerobically. This anaerobically generated energy for ATP resynthesis can be thought of as reserve fuel that is brought into play during the final seconds of a mile run. In addition, it is of critical importance to supply the rapid energy above that available from stored phosphagens during such events as the 440-yd run or 100-yd swim.

Long-Term Energy: The Aerobic System

Although the energy released in glycolysis is rapid and does not require O_2, relatively little ATP is resynthesized in this manner. Consequently, aerobic reactions provide the important final stage for energy transfer, especially if vigorous exercise exceeds several minutes.

During relatively low intensity, long duration exercise, the amount of O_2 consumed rises rapidly during the first few minutes. By the third or fourth minute, a plateau is reached where the oxygen consumption (VO_2) remains relatively stable for the remainder of the exercise period. This steady rate reflects a balance between the energy required by the exercising muscles and the rate of ATP production through aerobic metabolism. Thus, oxygen-consuming reactions supply the energy for exercise, and any lactic acid produced is either oxidized or reconverted to glucose. Under steady-rate metabolic conditions, lactic acid accumulation is minimal.

The Energy Continuum of Exercise

Figure 13-7 illustrates the relative contribution of the three energy sources during various durations of exercise. The actual contribution that each source makes to total energy provision, however, is difficult to determine. Recently, it has been estimated[6] that the maximal power of the phosphagen

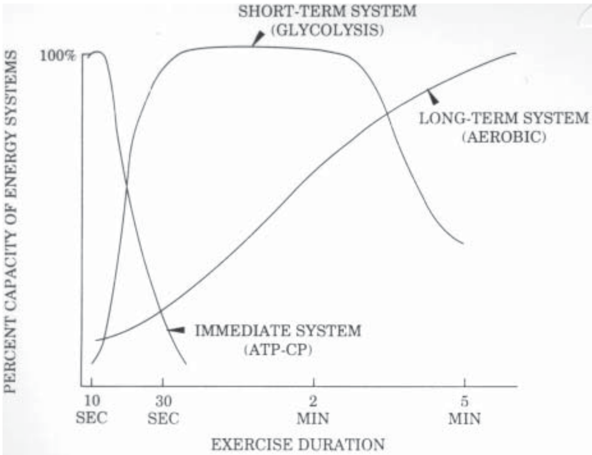

Fig. 13-7. Relative contribution of the three energy sources as a function of exercise duration. Reprinted with permission from McArdle W, Katch F, Katch V. *Exercise Physiology, Energy, Nutrition, and Human Performance.* 3rd ed. Philadelphia, Pa: Lea & Febiger; 1991.

system is reached within 10 seconds, while that of the glycolytic system occurs between 16 to 30 seconds of maximal exercise. Exercise performances of longer duration require an increasing dependence on O_2 to meet the energy requirements of the exercising muscle. As seen in Figure 13-7, approximately 50% of the energy requirement is met by oxidative energy sources by 60 seconds of maximal exercise, while at 120 seconds, this percentage has reached nearly 80%. However, it must be recognized that energy provision for physical exercise is not linked sequentially, and that considerable overlap among the three systems occurs.

Pulmonary Function During Exercise

Dynamic exercise increases the requirement for the utilization of O_2 and the production of carbon dioxide (CO_2) at the cellular level. Thus, the process of pulmonary ventilation must keep pace to allow venous blood to become oxygenated and to maintain arterial CO_2 and hydrogen-ion homeostasis. Indeed, the ventilatory control mechanisms keep arterial CO_2 tension, hydrogen-ion concentration, and O_2 tension remarkably constant despite the marked increase in CO_2 production and O_2 utilization. The exceptions are when exercise is severe enough to produce an elevation in the blood lactic acid concentration, or during exercise at high altitude, or in certain pathophysiologic states.[7]

Regulation of Ventilation During Exercise

The mechanisms involved in the regulation of ventilation are complex and not fully understood. Information is relayed to the medulla through elaborate neural circuits from higher centers in the brain, from the lungs, and from other sensors throughout the body that contribute to the control of ventilation. In addition, the gaseous and chemical states of the blood that reaches the medulla and the chemoreceptors in the aorta and carotid arteries also act to control alveolar ventilation. As a result, relatively constant alveolar gas pressures are maintained even during exhaustive exercise.

Ventilation in Steady-State Exercise. The ventilatory response to constant-intensity exercise is characterized by three phases (Figure 13-8): (1) an abrupt increase in ventilation with the onset of exercise (phase I); (2) a further, more gradual increase to a steady state (phase II); and (3) a steady-state level (phase III). The magnitude of the response in phase I varies with the exercise intensity as well as among individuals. At moderate levels of exercise, this response may account for as much as 50% of the total response (phase III) while at higher exercise intensities, it represents a much smaller fraction of the phase III response.

The increase in ventilation that occurs with moderate exercise is primarily the result of an increase in the depth of respiration. However, as exercise becomes more strenuous, there is an accompanying increase in breathing frequency. At cessation of exercise, there is an abrupt decrease in ventilation, followed by a gradual, exponential decline to preexercise levels.

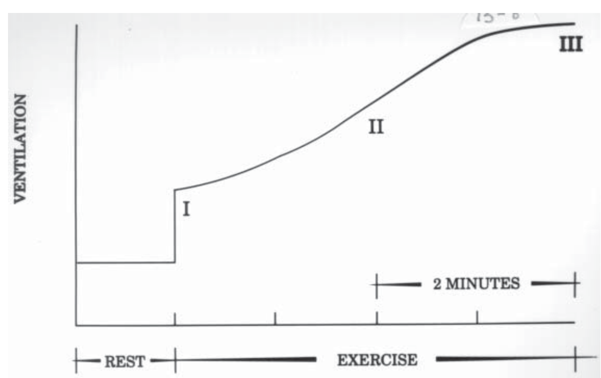

Fig. 13-8. The three phases of the ventilatory response during steady-state exercise.

Control of Ventilation. The rapid response of the ventilatory system at both the onset and cessation of exercise strongly suggests that this portion of exercise hyperventilation is mediated by both cortical and peripheral neurogenic factors. Neural outflow from regions of the motor cortex, as well as cortical activation in anticipation of exercise, stimulates the respiratory neurons in the medulla. In addition, afferent impulses from proprioceptors in muscles, tendons, and joints may influence the ventilatory adjustments to exercise. Although such peripheral receptors have not been identified, experiments involving electrical stimulation of muscles, and voluntary exercise with muscle blood flow occluded, support the existence of such mechanoreceptors in producing a reflex hyperventilation.[8]

The more gradual increase in ventilation during exercise is presumably due to gaseous or chemical humoral factors even though arterial pH, partial pressure of carbon dioxide (PCO_2), and partial pressure of oxygen (PO_2) remain constant during moderate exercise. An increase in body temperature may play a role since it has a direct stimulating effect on the neurons of the respiratory centers in the medulla, and probably exerts some control over ventilation in prolonged exercise. In addition, it may be that the sensitivity of the respiratory center to CO_2 is increased or that the respiratory fluctuations in arterial PCO_2 increase so that, even though the mean arterial PCO_2 does not rise, it is CO_2 that is responsible for the increase in ventilation. Thus, the control of ventilation during moderate exercise is not the result of any single factor but rather the combined, and perhaps simultaneous, result of several humoral and neural stimuli.[9]

Ventilation in Non–Steady-State Exercise. In exercise where the intensity is not constant but increases steadily, the minute ventilation and the rates of VO_2 and CO_2 production increase linearly until a level corresponding to approximately 60% (VO_2 of 2.5 L/min) of the individual's maximal exercise capacity is reached (Figure 13-9). Above this level of exercise intensity, the relationship between minute ventilation and VO_2 becomes curvilinear, increasing disproportionately with the increase in VO_2. As a result, the ventilatory equivalent (ratio of ventilation to VO_2) may increase to 35 or 40 L of air per liter of oxygen consumed. During moderate, steady rate exercise, this ratio is usually maintained at about 25:1.

During moderate, steady-rate exercise, sufficient O_2 is supplied to the exercising muscles. Under these conditions, lactic acid production does not exceed lactic acid uptake and there is no increase in blood concentration of lactic acid. However, as exercise intensity increases, blood lactate begins to increase above baseline levels. The baseline level is termed the *lactate threshold* or point of onset of blood lactate accumulation (OBLA). This normally occurs at an exercise intensity equivalent to 55% to 65% of the VO_2max in healthy, untrained individuals. Almost all of this excess lactic acid is buffered by the sodium bicarbonate system so that hydrogen ion concentration is maintained within acceptable limits. The excess, nonmetabolic CO_2 released by this buffering system stimulates an increase in ventilation and the CO_2 is exhaled into the atmosphere.

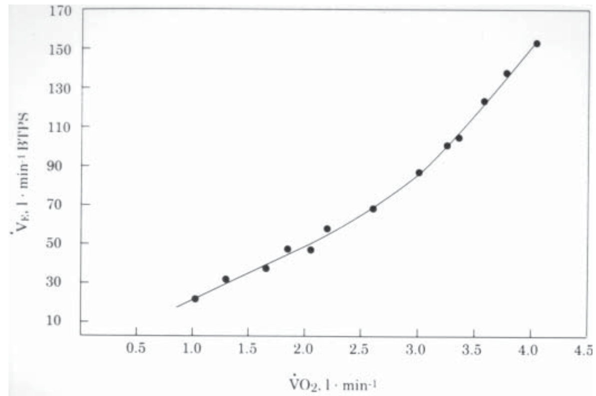

Fig. 13-9. Ventilatory response during non–steady-state exercise.

The exact cause of OBLA is controversial.[10] It has been assumed that OBLA represents an anaerobic state within the muscle. However, it has been shown[10] that muscle lactic acid accumulation is not necessarily linked to muscle anaerobiosis. Lactic acid can accumulate in the presence of adequate muscle oxygenation, implying an imbalance between lactic acid appearance in the blood and its subsequent rate of disappearance. This imbalance may not be only a result of muscle anaerobiosis, but also due to decreased lactic acid clearance, or increased lactic acid production in specific muscle fibers. Thus, caution is urged in interpreting too broadly the specific metabolic significance of OBLA.

Cardiovascular Function During Exercise

In understanding the function of the cardiovascular system during exercise, one must balance the interactions of the heart, blood vessels, and the nervous system. In addition, an integrative approach must be taken to fully appreciate the response by the cardiovascular system to the stimulus of exercise and the adaptations which occur with physical training.

Oxygen Consumption and Cardiac Output

As the intensity of exercise increases, the heart responds by increasing cardiac output (C.O.) in a linear fashion to meet increased metabolic demands. C.O., the product of stroke volume (SV) and heart rate (HR), increases approximately 6 L/min for every 1 L/min increase in VO_2.[11] This relationship is remarkably consistent during all types of exercise. The precise matching of C.O. and VO_2 is brought about by an intricate system of sensory nerves that informs the brain of the body's needs. Indeed, the maximum rate of VO_2max is largely influenced by maximal C.O. Maximum oxygen consumption is defined as:

$$VO_2max = HRmax \bullet SVmax \bullet a\text{-}vO_2diffmax$$

where the latter is the maximal difference between the arterial and mixed venous oxygen content. The a-vO_2diffmax represents the body's ability to extract and utilize O_2 delivered by the blood to the tissues. With an increase in exercise intensity, more arterial blood is directed to the contracting muscles which extract more O_2; therefore, less oxygen is left in venous blood and the a-vO_2diff increases.

Heart Rate

The nerves that control HR originate in the medulla. As a person moves from rest to mild exercise, the increase in HR up to approximately 100 beats/min is predominantly caused by suppression of parasympathetic activity, that is, the normally occurring parasympathetic inhibition is removed. Increases beyond 100 beats/min are brought about primarily by stimulation of the sympathetic nerves.[12]

The medulla, as previously seen for ventilation, receives sensory inputs from many areas in control of HR. One of the most important of these areas is the motor cortex. This part of the brain must progressively recruit more muscle fibers as exercise becomes more intense and as previously recruited fibers fatigue. As more fibers are recruited, the motor cortex simultaneously stimulates the medulla to increase HR (as well as ventilation, blood pressure [BP], etc.). The medulla also receives input from nerves in the contracting muscles that are sensitive to changes in the chemical environment and signal a need for increased blood flow. Thus, while the medulla receives many types of inputs that affect the regulation of HR, inputs from the motor cortex and the muscle are among the most important during exercise.

In exercise where a large muscle mass is involved, for example, running, swimming, cross-country skiing, and where the intensity is sufficient to elicit VO_2max, the medulla activates the sympathetic nerves to the heart to increase its rate to maximum. As a rule, HRmax is approximately 220 minus the individual's age in years. The decrease in HRmax with age is caused by a decreased sympathetic drive from the medulla.[13]

Exercise involving a relatively small muscle mass, such as weight lifting with the arms, is unlikely to raise HR much above 150 beats/min, even with maximal power production.[14] This lower maximal HR is a function of the reduced input to the medulla from both the motor cortex and the exercising muscles, so that fewer muscle fibers are being recruited.

HR may be similar during different types of physical activity, in which the VO_2 varies considerably. For example, performing upper body exercise, such as arm curls at an HR of 130 beats/min requires much less VO_2 than does running at the same HR because a much smaller muscle mass is involved in the arm exercise. Thus, when exercise training is prescribed solely on the basis of HR, important exercise effects on the heart may be overlooked.

Arterial Blood Pressure

Information about BP is relayed to the medulla by sensory nerves from baroreceptors in the aorta and carotid arteries. In turn, appropriate adjustments in C.O. and blood vessel diameter are made to maintain BP at a suitable level. BP is the product of C.O. and total peripheral resistance (TPR), the latter being determined primarily by the extent to which small arterial vessels are constricted.

During exercise, such as running, systolic BP increases moderately as the intensity increases, largely because increased C.O. forces more blood into the arterial system, while TPR decreases slightly.[13] On the other hand, during weight lifting, when a small muscle mass contracts very intensely, especially during an isometric contraction, systolic BP can rise to very high levels.[15] This occurs because intensely contracting muscles compress small arteries in those muscles and drive TPR up at the same time that C.O. is increasing.

Diastolic BP does not change much during exercise involving large muscle groups, even when there is an increase in the intensity of the exercise. However, during weight lifting or during isometric contractions with small muscle mass, progressively more small arteries are compressed and even closed completely as contractions become more intense. Thus, during this type of exercise, diastolic BP rises progressively, and TPR is progressively increased, even during cardiac filling.[16]

Blood Flow Distribution

Increased C.O. during exercise must be appropriately distributed to the various regions of the body. This distribution of blood depends on the degree to which the small arteries in the exercising muscles, resting muscles, and other regions of the body are open, and is controlled to a large extent by the medulla and by chemical changes that occur in the contracting muscle. As exercise intensity increases, local vasodilator substances released by the contracting muscle fibers relax the smooth muscle of the arterial walls, causing the arteries to dilate, and allowing more blood to perfuse the muscle.[16] In theory, in heavy exercise involving many muscle groups, local vasodilators could cause arterial dilation to the extent that blood flow approaching 60 L/min would occur.[17] Since maximal C.O. rarely exceeds 35 L/min, even in elite athletes, such degrees of vasodilation would cause a sharp drop in BP reducing blood flow to the brain and heart. However, neural outflow from the medulla, which leads to vasoconstriction of vessels in active muscle and in other regions of the body, can compensate for any excessive local vasodilation,[13] but in so doing, blood flow to the exercising muscle may be limited, or less then metabolic demands require.

As exercise intensity increases, the medulla directs sympathetic vasoconstrictor stimuli to the arteries that supply the stomach, intestines, liver, and kidneys, as well as inactive muscles, reducing blood flow so that a greater fraction of blood is directed to the exercising muscles. Arterial vessels in the skin are also constricted at the onset of exercise, but this effect is ordinarily reversed as the core temperature rises.

With an increasing heat load, temperature control centers in the hypothalamus instruct the medulla to dilate vessels in the skin so that heat can be dissipated to the environment. However, when exercise intensity exceeds 80% to 90% of VO_2max, blood flow to the skin may again be reduced because the temperature regulation needs of the body are overridden by the requirement to maintain BP.[13] In essence, the muscles and the skin compete for blood flow during heavy exercise, and when confronted with this choice, the medulla determines that the muscles win. As a consequence, the reduced skin blood flow can cause marked increases in body temperature.

Stroke Volume

Stroke volume increases from rest to mild exercise (up to approximately 50% VO_2max) and then remains fairly stable with increasing exercise intensity.[13] During exercise involving a small muscle mass, SV is relatively low due to the high TPR that is present. This rise in TPR causes so much resistance to blood flow that SV is reduced; consequently, the flow of blood back to the right side of the heart is lowered. This reduction in venous return to the heart further compromises the ability of the heart to effectively pump blood.

In contrast, exercise with a large muscle mass elicits a larger SV because the relatively low TPR results in a much larger muscle blood flow. Venous return to the heart is enhanced as well, stimulating baroreceptors in the right side of the heart and in the pulmonary circulation.[17] These low pressure receptors send sensory signals to the medulla that cause it to decrease TPR in the exercising muscles, thereby counteracting the increased C.O. to maintain a stable BP simultaneously with a high muscle blood flow and a large venous return to the heart.

TABLE 13-1

ACUTE RESPONSES TO MAXIMAL EXERCISE IN A YOUNG ADULT MAN

Variable	Rest	Exercise	Ratio
O_2 uptake (L/min)	0.30	4.00	13:1
Minute ventilation (L/min)	8	140	18:1
P_{AO_2}	100	115	1.2:1
P_{aO_2}	95	90	0.95:1
C.O. L/min	6	24	4:1
HR (beats/min)	70	200	2.9:1
Systolic BP (mm Hg)	120	180	1.5:1
Diastolic BP (mm Hg)	80	95	1.2:1
a-vO_2 difference (mL/dL)	5	18	3.6:1

C.O.: cardiac output
HR: heart rate
BP: blood pressure
P_{AO_2}: partial pressure of oxygen, alveolar
P_{aO_2}: partial pressure of oxygen, arterial

Oxygen Transport Variables

Table 13-1 shows commonly found changes in the principal O_2 transport variables that can be measured in healthy volunteers during whole body exercise at intensity levels of VO_2max. These data are round numbers and are not intended to be precisely consistent. There is, of course, considerable variation among individuals in most of these variables. The 13-fold increase in VO_2 from rest to maximal exercise is accomplished by a combination of central and peripheral factors, as previously discussed. The three major responses are in ventilation, C.O., and O_2 extraction by the muscle. Ventilation increases some 20-fold; C.O. and O_2 extraction by about a factor of three to four each. The remaining factors in Table 13-1 change little from rest to maximal exercise.

In summary, all components of the O_2 transport pathway undergo responses to acute exercise. In terms of O_2 supply and thus VO_2max, the changes of significance are in ventilation, blood flow, and muscle tissue O_2 extraction. The resulting VO_2max represents the integrated effects of both central and peripheral factors involved in these three processes.

BODY COMPOSITION AND PHYSICAL FITNESS

Background

The relative composition of the body, particularly in respect to fat and muscle, is often included as one component of physical fitness. Although lying outside our energy pathway definition of physical fitness as presented earlier, the amount and location of both fat and skeletal muscle are strongly associated with physical performance capacity. These components can also be modified through physical training and therefore deserve attention in any discussion of physical fitness, performance, and training.

The term *body composition* refers to the proportional makeup of the body into major components of fat, bone, muscle, and other soft tissues, each of which can be further separated into the chemical constituents. Since body fat, or its excess, is often the focus of interest with respect to physical fitness, the term *fat-free mass* is often used to denote all tissues other than the fat mass. Therefore, in the most basic division, the body is composed of fat and fat-free mass. This simple division is also significant in that the commonly used underwater weighing method to estimate body composition results in two compartments that can be separated by density: fat and fat-free mass. Within the fat-free mass component of healthy individuals, bone, organ, and structural tissues are largely fixed in amount and only skeletal muscle is modifiable. Thus, observed changes in fat-free mass can most often be interpreted as changes in muscle mass.

Body Fat

Body fat can be divided into two categories: (1) essential or obligatory fat, and (2) storage fat. Obligatory fat is lipid, which is required for cell and organ structure, protective cushioning of the organs, nutritional support of specific metabolic activities such as myocardial contractility, and small amounts for cellular functions. Women have additional obligatory fat in the context of sex-specific fat: breast fat and fat associated with the reproductive organs. The amount of the second component, storage fat, is primarily a function of caloric balance, and eth-

TABLE 13-2

REFERENCE BODY COMPOSITION VALUES FOR NONATHLETIC YOUNG ADULTS*

Variable	Women	Men	Ratio (w/m)
Total Fat kg (%)	16.8 (28)	13.5 (18)	1.24:1
Obligatory kg (%)	7.2 (12)	2.3 (3)	3.13:1
Storage kg (%)	9.6 (16)	11.3 (15)	0.85:1
Fat-Free Mass kg (%)	43.2 (72)	61.5 (82)	0.70:1
Skeletal Muscle kg (%)	21.6 (36)	33.8 (45)	0.64:1
Bone kg (%)	7.2 (12)	11.3 (15)	0.64:1
Other kg (%)	14.4 (24)	16.4 (22)	0.88:1

*Values represent a compilation of available data.

nic, genetic, and sex-related patterns. Reference values for obligatory and storage fat in nonathletic populations are found in Table 13-2.

The regional distribution of storage fat varies between genders and among individuals, and is primarily controlled by the sex hormones and their control of lipoprotein lipase enzyme activity and adipocyte sensitivity to insulin.[18,19] Males predominantly store fat in the abdominal region (android, or central-upper fat pattern) while women typically store fat in the hips and buttocks (gynoid, or peripheral-lower fat pattern).[20] The android pattern reflects greater deep or visceral deposited fat, as opposed to greater subcutaneous fat in the gynoid pattern, and is typically associated with greater muscularity.[21]

Excess storage fat, or obesity, is best expressed in terms of the percentage of body weight composed of fat. McArdle and colleagues[22] have suggested that obesity be defined as 5% units over the population norm. When this is applied to young army members, this equates to 20% (5% above the norm of 15%) for men and 30% (5% above the norm of 25%) for women. These values of 20% and 30% are now employed as the upper allowable limits under the U.S. Army's body weight/fat control program[23] for the age group of 17 to 25 years. In 1974, Behnke and Wilmore[24] supported this when they defined obesity in males as exceeding 20%, since data indicate that fat cells are fully saturated at this level of fatness.

The U.S. Navy, on the other hand, has employed the definition of obesity developed by a National Institutes of Health Consensus Development Conference: "weight for height 20% above the midpoint weight listed in the 1983 Metropolitan Life Insurance tables for the medium frame individual."[25] This value corresponds, in Navy personnel, to 26% body fat in men and 36% in women. Table 13-3 presents a compilation of percentage of body fat values reported for various U.S. military populations.[26-31]

Muscle Mass

The average man has 1.5-fold the amount of skeletal muscle as the average woman, primarily as the result of differences in circulating levels of the male steroid hormone, testosterone. Skeletal muscle represents 45% and 36% of body weight, male and female, respectively (see Table 13-2). Testosterone and other anabolic steroids have potent muscle growth actions, which have led to their use by some body builders and strength athletes.

Overweight, by the customary weight-for-height standards, is usually interpreted as over-fat, although it can actually be a case of being overmuscled. Body builders and athletes requiring high levels of strength or muscular power, or both, typically fall in this latter category. To avoid misclassifying overmuscled individuals as being over-fat, body fat assessment has replaced or supplemented simple body weight measurements in the military services and is discussed later in this chapter.

Physical Training

Changes in body composition, that is, reductions in body fat and increases in muscle mass, are common consequences of physical training, although the extent depends on the nature of the training program, the accompanying nutritional state, as well as the genetic makeup of the individual. Aerobic training, plus caloric restriction, are commonly employed to reduce body fat. Strength or strength endurance (resistance) training, and protein supplementation, are commonly employed to build muscle mass.

Body Fat

Many studies have examined the influence of aerobic exercise training to reduce body fat (eg, Zuti and Golding,[32] and Garrow[33]). Because low to moderate intensities of exercise burn fat almost exclusively, aerobic exercise is an effective means of metabolizing excess fat if, at the same time, caloric intake is controlled. Resistance training is a less efficient means of losing body fat, although reductions of 1% to 3% over a 10- to 20-week program have been shown.[34] Spot reduction of fat from a specific area being exercised is not effective.[35]

Muscle Mass

Resistance training is an effective stimulus to muscle growth, leading to increased muscle mass and cross-sectional area of the trained muscles. Increases of 1.5 kg in fat-free mass over a 10 week period are typical.[34] Training programs employing lower intensities but high volumes (frequencies and durations) are more effective in developing muscle and, therefore, are used by body builders. Some studies[34] show that the increase in muscle size is due to fiber hypertrophy, while others implicate fiber hyperplasia. The evidence for fiber hyperplasia in humans is still controversial and the reader is re-

TABLE 13-3

PERCENT BODY FAT VALUES FROM U.S. MILITARY POPULATIONS AS A FUNCTION OF AGE, GENDER, ETHNICITY, OCCUPATIONAL DEMAND RATING, AND TYPE OF ASSIGNMENT

Category	Mean + SD Women	Mean + SD Men
US Army Recruits (new)[1]		
Age (y)		
17-20	27.7 ± 4.2	15.3 ± 4.7
21-25	28.8 ± 4.5	16.1 ± 5.2
26-30	28.3 ± 4.3	18.1 ± 5.2
30-35	31.0 ± 4.8	22.4 ± 4.6
USMA Cadets[2]	26.5 ± 3.2	12.2 ± 3.0
US Army combat and combat support[3]		
Age (y)		
17-20	26.3 ± 5.3	15.4 ± 5.9
21-27	25.4 ± 6.0	16.7 ± 6.7
29-39	29.2 ± 7.0	21.6 ± 7.0
40+		23.1 ± 5.3
Race		
White	28.0 ± 5.5	17.6 ± 5.5
Black	28.0 ± 4.0	14.0 ± 5.7
Hispanic	28.0 ± 5.0	17.4 ± 6.1
Infantry[4]		
Age (y)		
17-20	—	15.8 ± 4.1
21-25	—	17.9 ± 6.1
26-30	—	19.3 ± 5.9
31-35	—	20.0 ± 5.8
Occupational rating		
Heavy	—	17.2 ± 5.0
Moderate	—	19.6 ± 6.7
Light	—	19.9 ± 6.3
Army Artillery[5]	28.6 ± 3.9	19.3 ± 4.9
Army Special Forces[6]	—	16.1 ± 4.5
Army Ranger students[7]	—	14.6 ± 4.1

Data sources: (1) Knapik JJ, Burse RL, Vogel JA. Height, weight, percent body fat, and indices of adiposity for young men and women entering the U.S. Army. *Aviat Space Environ Med.* 1983;54:223-231. (2) Daniels WL, Kowal DM, Vogel JA, Stauffer RM. Physiological effects of a military training program on male and female cadets. *Aviat Space Environ Med.* 1979;50:562-566. (3) Fitzgerald PI, Vogel JA, Daniels WL, Dziados JE, Teves MA, Mello RP, Reich PJ. *The Body Composition Project: A Summary Report and Descriptive Data*. US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 5-87. (4) Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (5) Teves MA, Vogel JA, Carlson DE, Schnakenberg DD. *Body Composition and Muscle Performance Aspects of the 1985 CFFS test.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 12-86. (6) Askew EW, Munro I, Sharp MA, et al. *Nutritional Status and Physical and Mental Performance of Special Operations Soldiers Consuming the Ration, Lightweight or the Meal, Ready-to-Eat Military Field Ration During a 30-day Field Training Exercise.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. 7-87. (7) Unpublished data.

ferred to the review by Saltin and Gollnick[3] for a discussion of the evidence. Growth in muscle tissue may depend on the specific type of training stimulus that is applied. The intensity and volume of training programs can be adjusted to selectively hypertrophy either slow or fast twitch fibers. The fiber hypertrophy is due to an increased size and number of the actin and myosin filaments and additional sarcomeres.[36]

Loss in muscle mass will result from inactivity or severe malnutrition. If the individual is relatively inactive, severe caloric deprivation will result in loss of both body fat and muscle mass.[37] Physical activity during caloric deprivation helps preserve muscle mass so that storage fat is preferentially depleted before muscle mass is used as an energy source.

Body Composition and Physical Capacity

An association between body composition and physical capacity, and performance are readily evident in athletes, as exemplified by the lean, modestly muscled, long distance runner and the heavily muscled, modestly fat Olympic lifter. Whether these relationships, albeit in a less dramatic form, exist in the nonathletic, yet trained population typical of the military services has been the focus of recent studies.[29,38]

Body Fat

Studies by Vogel and colleagues[29] found a correlation coefficient of –0.59 (standard error of the estimate, [SEE] = 4.26) between percent body fat and VO_2max expressed per kg of body weight, for male army recruits beginning basic training and -0.52 (SEE = 5.73) for a diverse sample of soldiers stationed within the continental United States. Similar relationships were found in a larger subsequent study of an army population of 1,117 men and 303 women (Figure 13-10). The correlation coefficient for men was –0.60 (SEE = 5.02) and –0.55 for women (SEE = 3.77). This modest correlation between percentage of body fat and aerobic capacity holds true when aerobic fitness is expressed relative to body weight, but not when capacity is expressed in terms of absolute VO_2max (L/min) as illustrated in Figure 13-11. Absolute aerobic capacity is related to the amount of oxygen-consuming metabolically active muscle, and, therefore, to the fat-free mass, rather than to the metabolically inactive fat tissue. Relative aerobic capacity (VO_2max, mL/min/kg BW) is related to body fat, since increasing fat increases the total body weight (denominator) and thereby lowers the resulting VO_2max value. This corresponds to the physiological situation where the capacity for body propulsion is diminished as body fat adds "dead weight," that is, nonenergy producing mass that must be carried. This is reflected in the association between two-mile-run test scores and body fat content, and is exemplified in long distance runners who typically have the lowest measured body fat content of any athletes, about 4% to 6%.[39]

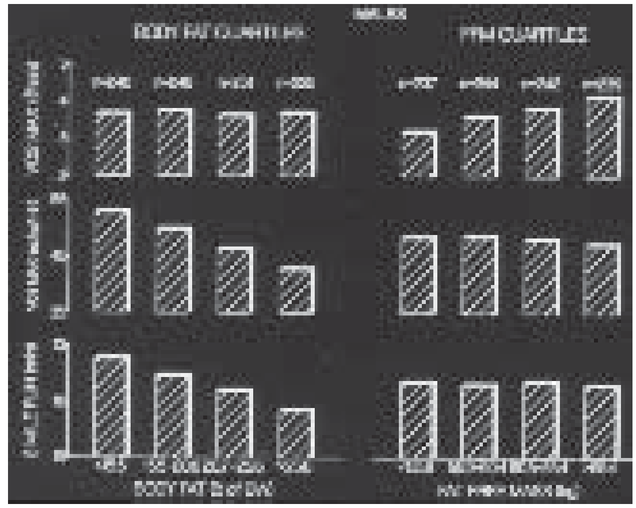

Fig. 13-11. Relation of maximal oxygen uptake to body fat and fat-free mass. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

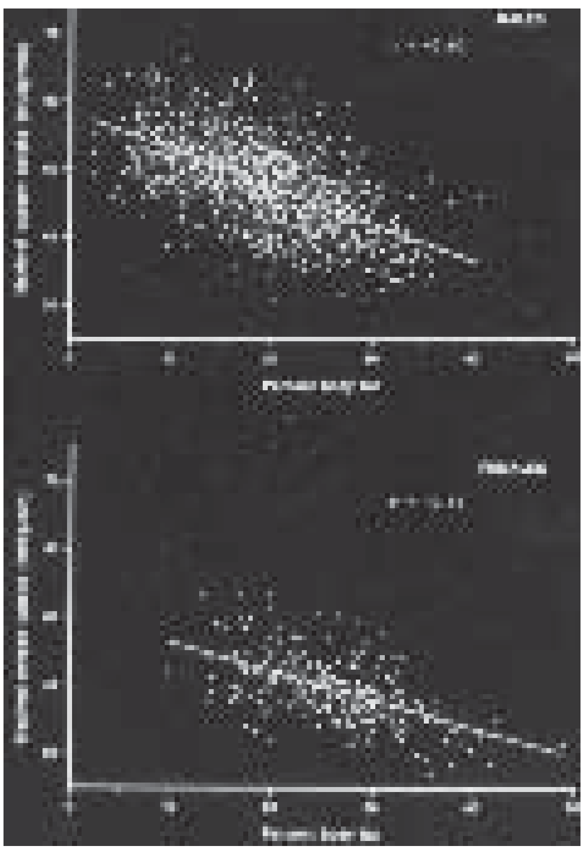

Fig. 13-10. Scatter plot of the relationship between maximal oxygen uptake (per kg body weight) and % body fat in Army men and women. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

Muscle Mass

In contrast to aerobic activities that are influenced by percentage of body fat levels, strength activities are related to muscle mass and not related to body fat (see Figure 13-11). Strength of a particular muscle or muscle group is directly related to its cross-sectional area.[40] This relationship holds true for total body muscle mass (as represented by fat-free mass) with total body strength (as represented by

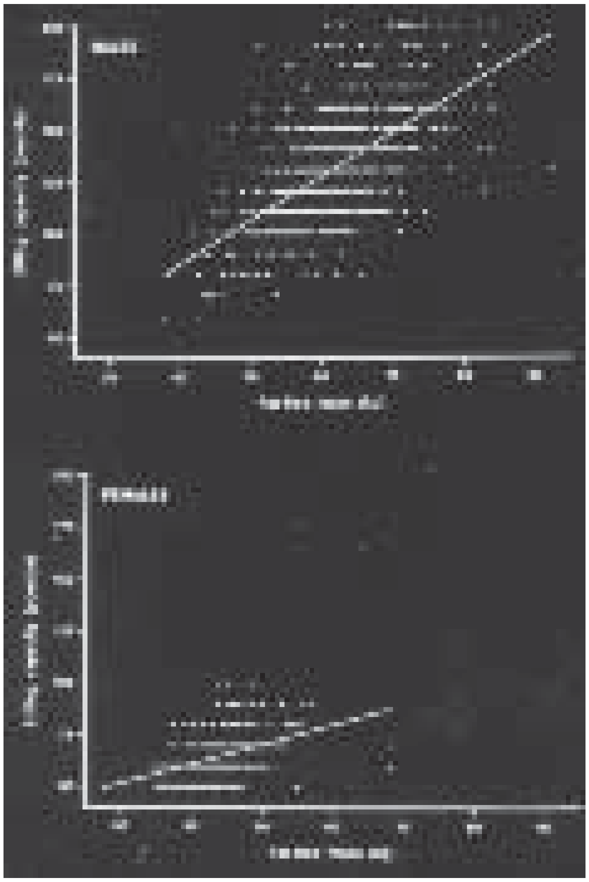

Fig. 13-12. Scatter plot of the relationship between maximal lift strength and fat-free mass. Reprinted with permission from Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy of Sciences. National Academy Press; 1992:97.

maximal absolute lifting strength) as illustrated in Figure 13-12. Corresponding correlation coefficients are 0.50 (SEE = 20.55) for men and 0.38 (SEE = 11.75) for women.

Body Composition Standards

It was not until 1960 that the U.S. Army first established an upper allowable limit for body weight for entrance into the service, and not until 1976 was a similar standard established for retention (on-the-job). Prior to World War II, the emphasis was on a minimal acceptable weight, and it was not until World War II that this focus changed to overweight.[41] In 1980, the DoD physical fitness directive[1] called for a primary body fat standard rather than just a weight-for-height standard. It also set a DoD goal for percentage of body fat to be 20% for men and 26% for women.

In response to this directive, in 1983 the U.S. Army implemented a body fat standard into its weight control program with a revision to Army Regulation (AR) 600-9, The Army Weight Control Program. This revision retained the weight for height as an initial screen, but employed body fat as the ultimate standard. The standards, as revised in October 1991, are shown in Table 13-4.[24,42-45] This revision also established corresponding body fat

TABLE 13-4

BODY FAT STANDARDS OF THE MILITARY SERVICES (values = body fat as a % of body weight)

US Service Branch	Percentage of Body Fat			
	Male		Female	
	Accession	Retention	Accession	Retention
Army[1,2]				
Age (y)				
17-20	24	20	30	30
21-27	26	22	32	32
28-39	28	24	34	34
≥ 40	30	26	36	36
Navy[3]				
All ages	—	22*	—	30*
All ages	—	26†	—	36†
Air Force[4]				
Age (y)				
17-29	—	20	—	28
30-39	—	26	—	34
Marine Corps[5]				
All ages	—	18	—	26

*If this value is exceeded, service member is automatically placed on fat loss program.
†If this value is exceeded, administrative action is taken.
Data sources: (1) US Department of the Army. *Medical Service Standards of Fitness.* Washington DC: Department of the Army; 1991. Army Regulation 40-501. 23. (2) US Department of the Army. *The Army Weight Control Program.* Washington DC: Department of the Army; 1991. Army Regulation 600-9. (3) US Department of the Navy. *Physical Readiness Program.* Washington DC: Office of the Chief of Naval Operations; 1986. CNO Instruction 6110.1c. (4) US Department of the Air Force; *The Air Force Weight and Fitness Program.* Washington DC: Department of the Air Force, 1985. Air Force Regulation 35-11. (5) US Marine Corps. *Weight Control and Military Appearance.* Washington DC: Department of the Navy; 1986. Marine Corps Order 6100.1c.

standards for accession into the army, also shown in Table 13-4. Based on patterns of body fat change observed in army recruits,[46] male recruits are allowed to enter the service at four body fat percentage points above their retention standard while females must enter at, or below, their retention standard.

The original body fat standards established by the U.S. Army in 1983 were derived empirically. Working from a base of 20% for the youngest male grouping, 2% was added for each increasing age group, and an 8% increment was added for females for sex-specific fat. The only objectivity in these decisions was the establishment of the base figure of 20%. This value was based, in part, on the finding by Vogel and coworkers[29] that VO_2max began to decline above a body fat of 20%. Continued study of available data has substantiated that the male standards originally established in 1983 are, in fact, supported by both physical performance and appearance criteria.[41] Figure 13-13 illustrates the correspondence between the body fat standard for one age group and corresponding two-mile-run standard. This correspondence for women did not hold up and led to the liberalization of their body fat standards by 2% from the original settings to those now employed as shown in Table 13-4. Thus, the setting of the army's body fat standards are based on physical performance and appearance criteria in agreement with the rationale for the weight control program as stated in AR 600-9, "to ensure that all personnel a) are able to meet the physical demands of their duties under combat conditions, [and] b) present a trim military appearance at all times."[23 (p3)] The history and the basis for the U.S. Army's body weight/body fat standards has been reviewed by Friedl.[41]

In contrast to the U.S. Army, the U.S. Navy has based its body fat standards on health criteria because a strong relationship between body fatness and the performance of naval shipboard tasks was not found. Shipboard tasks are predominantly strength-demanding tasks and are unrelated to body fatness. The U.S. Navy moved to adopt a definition of obesity as a weight for height 20% above the midpoint weight listed in the 1983 Metropolitan Life Insurance tables for the medium frame individual, developed from a National Institutes of Health consensus development conference in February 1985. This definition translated into a percentage of body fat for the U.S. Navy population of 22% and 33.5% for males and females, respectively. Adding one standard error to this, the navy established its standard at 26% and 36% for males and females, respectively.[25] Exceeding this limit leads to administrative action, while a more restrictive category, 22% and 30%, requires admission to a fat reduction program. The U.S. Air Force has used appearance as its principal criterion,[44] while the U.S. Marine Corps body fat standards are based primarily on health and appearance requirements.[45]

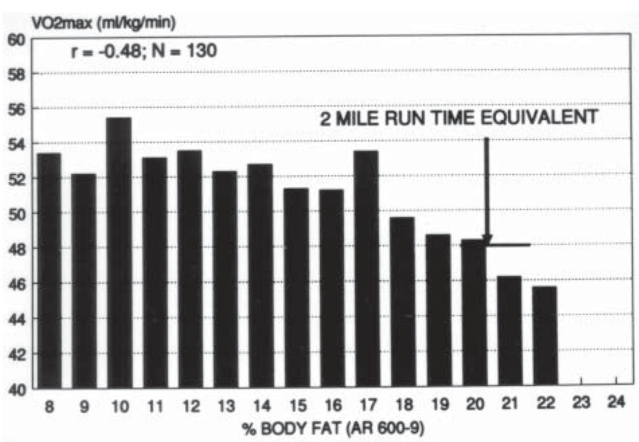

Fig. 13-13. Correspondence between body fat standards (20%) and VO_2max for 17-20 year old male soldiers. Reprinted with permission from: Friedl KE. Body composition and military performance: origins of the army standards. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy of Sciences. National Academy Press; 1992:99.

Body Fat Methodology

Body fat measurement methodology has recently received intense interest and study. The following discussion will be limited to those developments within the military services.

At the time of the DoD directive calling for body fat standards and measurements in the services, most population or "field" measurements for research purposes in the military used the skinfold procedure,[47] based on hydrostatic (underwater) weighing as the criterion method. The hydrostatic procedure employs the known differences in density of fat and nonfat tissue.[48] The skinfold procedure employs a series of skinfold thicknesses, which are summed, and used to predict body fat from empirically derived equations of body density from hydrostatic weighing. Many skinfold prediction equations have been developed, some population specific, and some more generalized.[49] Because most U.S. Army research data on body fat prior to the DoD directive had been collected using the Durnin and Womersley equations,[50] this procedure was adopted for the initial implementation by the army in 1983.

The skinfold procedure suffers from several drawbacks. It can, depending on the equations selected, be population specific since it has been shown that genetic background affects regional fat distribution.[51] The Durnin-Womersley equations were developed from an Anglo-Saxon population and therefore their application to African Americans, Hispanics, and Asians is suspect. Secondly, it is subject to a rather large intermeasurer error, especially outside the research laboratory. Thus the army experienced unsatisfactory variability among body fat measurements throughout many army posts. At the same time, the U.S. Marine Corps had developed simple circumference procedures[52,53] to avoid the problems of measurement variability. Their success inspired the other services to eventually adopt circumference methods for estimating body fat in the "field" for their weight control programs.

The Navy's current circumference procedure and equations were developed by Hodgdon and Beckett[54,55] and are shown in Table 13-5. The army's equations[56] were based on the navy's experience and are similar except for the sites employed for women. The U.S. Air Force has recently adopted the U.S. Navy equations (see Table 13-5). Differences between the equations are the result of different statistical approaches, different limits in what was allowed to enter the regression analysis, and differ-

TABLE 13-5

CIRCUMFERENCE-BASED EQUATIONS EMPLOYED BY THE MILITARY SERVICES TO ESTIMATE PERCENT BODY FAT*

US Service Branch	Gender	R	SEE	Density	Body Fat (%)
Navy and Air Force[1,2]	Male	0.90	3.52	$-0.191 \bullet \log_{10}$ (abdom II − neck) $+ 0.155 \bullet \log_{10}$ (height) + 1.032)	100 • [(3.95/density) – 4.5]
	Female	0.85	3.72	$-0.350 \bullet \log_{10}$ (abdom I + hip + neck) $+ 0.221 \bullet \log_{10}$ (height) = 1.296	100 • [(3.95/density) – 4.5] 100 • [(3.95/density) – 4.5]
Army[3]	Male	0.82	4.02		$76.462 \bullet \log_{10}$ (abdom I – neck) $- 68.678 \bullet \log_{10}$ (height) + 43.742
	Female	0.82	3.60		$105.3 \bullet \log_{10}$ (body weight) – 0.2 • wrist – 0.533 • neck – 1.574 • forearm + 0.173 • hip – 0.515 • height – 35.6
Marine Corps[4]	Male	0.81	3.67		0.74 • abdom II – 1.249 • neck + 40.985
	Female	0.73	4.11		1.051 • bicep – 1.522 • forearm – 0.879 • neck + 0.326 • abdom II + 0.597 • thigh + 0.707

*All measurements except height (cm) and weight (kg) are circumferences measured in cm.
R: Correlation coefficient
SEE: Standard error of the estimate
Data sources: (1) Hodgdon JA, Beckett, MB. *Prediction of Percent Body Fat for US Navy Women from Body Circumferences and Height.* San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-29. (2) Hodgdon JA, Beckett, MB. *Prediction of Percent Body Fat for US Navy Men From Body Circumferences and Height.* San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-11. (3) Vogel JA, Kirkpatrick JW, Fitzgerald PI, Hodgdon JA, Harman EA. *Determination of Anthropometry Based Body Fat Equations for the Army's Weight Control Program.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988: Technical Report No. T17-88. Wright HF, Dotson CO, Davis PO. An Investigation of Assessment Techniques for Body Composition of Women Marines. *US Navy Med*. 1980;71:15-26.(4) Wright HF, Dotson CO, Davis PO. An Investigation ofAssessment Techniques for Body Composition of Women Marines. *US Navy Med*. 1980;71:15-26. Wright HF, Dotson CO, Davis PO. Simple Technique for Measurement of Percent Body Fat in Man. *US Navy Med*. 1981;72:23-27.

ent population characteristics. All are based on underwater weighing as the criterion method.

Attention to body composition methodology in the military services is currently causing a reexamination of hydrostatic weighing as the criterion methological standard.[57] Hydrostatic weighing suffers from the fact that it is based on density assumptions that are from a very limited number of cadaver analyses and may not reflect racial and gender differences in bone and nonfat tissue density. The most likely candidate for a replacement of hydrostatic weighing as the criterion standard, and now under study by both the U.S. Army and the U.S. Navy, is dual photon x-ray absorptiometry (DEXA),[58] which employs the differential attenuation of two different energy levels from an x-ray source to separate fat, bone, and fat-free tissue. DEXA can be employed with hydrostatic weighing and body water measurements to derive a four compartment model (fat, fat-free mass, bone, and water) or can be used alone to separate fat and fat-free mass. Initial research[57] supports its potential to replace body density measurements as the sole criterion method.

PHYSICAL CAPACITY FOR EXERCISE

Aerobic Power

Aerobic power, or cardiorespiratory fitness, can be defined as the capacity of the aerobic metabolic pathways to produce energy for moderate intensity exercise for prolonged periods. A highly developed cardiorespiratory endurance capacity is typified by the marathoner, cross-country runner, or cross-country skier. In the military, this component of physical fitness has an important relationship to the performance of such physically demanding tasks as road marching, prolonged load carriage, and the repetitive lifting and carrying of moderate loads. In addition, maintenance of adequate levels of VO_2max is important for health protection, body weight control, and the building of unit morale and *esprit de corps*.

Measurement

Exercise scientists agree that the best measure of aerobic power is an individual's VO_2max. Indeed, the highest values for VO_2max are generally found in elite athletes who compete in such endurance activities as distance running, swimming, bicycling, and cross-country skiing.[59] These individuals have nearly double the aerobic power of age-related sedentary individuals (Figure 13-14). This is not to say, however, that VO_2max is a complete expression of aerobic power. Many other factors, especially those at the cellular level, such as number of capillaries, enzymes, and fiber type, exert a strong influence on the capacity to sustain high levels of aerobic exercise.[60] However, the measurement of VO_2max provides important information on the overall capac-

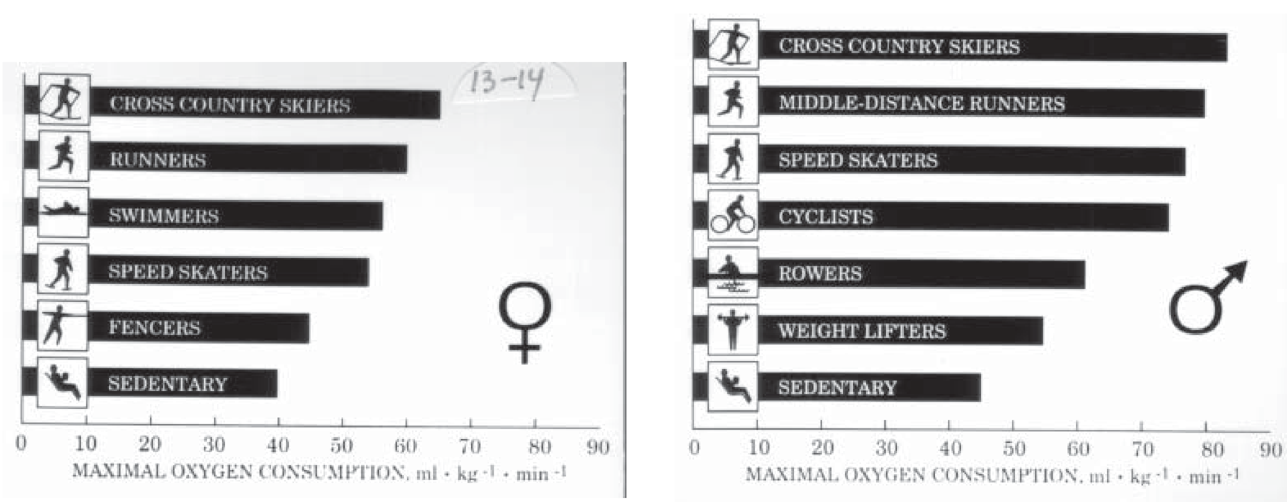

Fig. 13-14. Representative VO_2max values of elite athletes. Reprinted with permission from McArdle WD, Katch FI, Katch VL. *Exercise Physiology. Energy, Nutrition, and Human Performance.* 3rd ed. Philadelphia, Pa: Lea & Febiger; 1991.

ity of the aerobic energy system. The VO_2max attainment also requires integration of the ventilatory, cardiovascular and neuromuscular systems which gives VO_2max significant physiologic meaning.[61]

In the research laboratory, and as long as the exercise is of sufficient intensity and duration to permit the maximum transfer of O_2, VO_2max can be determined by various modes of exercise that activate large muscle groups. Typically, VO_2 is measured directly, by the collection and analysis of expired gases while the subject exercises at increasing intensities on either a motor-driven treadmill or cycle ergometer. Although other types of ergometers can be used, these two are by far the most common. As the intensity of exercise increases, the VO_2 increases in direct proportion. Eventually, the individual will reach his maximum ability to deliver O_2 to the exercising muscle, that is, the VO_2 will plateau despite a continued increase in the intensity of exercise (Figure 13-15).

Numerous tests have been devised and standardized for the measurement of VO_2max. In addition to the different modes of exercise, tests that directly measure VO_2 are usually performed either continuously (with no rest between exercise increments), or discontinuously (with the individual resting several minutes between bouts of exercise). In general, the discontinuous method is recommended, if time is available, as it yields slightly higher values, is more comfortable for the subject, and minimizes complications from such factors as local muscle fatigue.

Since the direct measurement of VO_2max requires expensive equipment, trained technicians, and a maximal effort on the part of the participant, investigators have attempted to develop simple, indirect, submaximal tests that would provide accurate predictions of VO_2max and could be administered to large numbers of individuals in a minimum of time and space. Most studies estimating VO_2max from submaximal exercise tests have used the cycle ergometer. The test developed by Astrand and Ryhming,[62] or modifications of this protocol, has been the most extensively used. The basis for the test is the fact that HR and VO_2 are linearly related over a range of exercise intensities. The underlying assumption is that the more fit individual will achieve a higher rate of exercise for the same HR and thus will be able to achieve a much higher exercise intensity before reaching maximum HR.

Determinants

The capacity to generate energy through aerobic metabolism is dependent on the various components of the O_2 transport system as described earlier in the section, Principles of Exercise Physiology. In considering factors that limit this transport, two schools of thought exist, one favoring a central limitation, and the other favoring a peripheral limitation.[63] "Central" O_2 delivery depends on pulmonary ventilation, arterial oxygen content, and C.O. In healthy individuals the first two components do

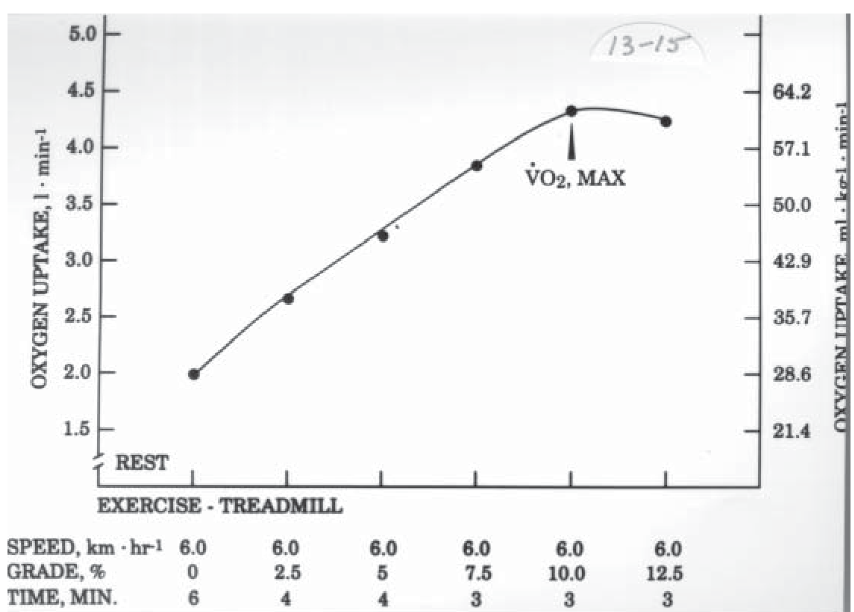

Fig. 13-15. The relation between increased exercise intensity and increased VO_2 and the subsequent determination of VO_2max from the plateau in VO_2 with increased exercise intensity.

not appear to be limiting factors. There is usually considerable reserve in the average person's pulmonary system and, therefore, ventilation does not normally limit exercise performance. In fact, there is no evidence that ventilation reaches a plateau, and it increases exponentially with increasing exercise intensity.

Arterial O_2 content is the product of arterial oxygen saturation and hemoglobin concentration. Neither of these variables changes appreciably from the resting condition up to maximum exercise. Also, in normal, trained, and untrained individuals, there is considerable overlap in hemoglobin and O_2 saturation levels and, therefore, in the magnitude of the arterial O_2 content. This suggests that this component in the O_2 transport system does not limit VO_2max.

Maximum C.O. is determined by the product of maximum SV and maximum HR. Because maximal HR is primarily determined by age and not affected by aerobic fitness level or state of training, the maximum C.O. that can be achieved during exercise is essentially a function of the SV, which becomes, therefore, the prime determinant of the difference in maximum C.O. among individuals. Most studies have also shown a higher SV in maximum exercise as well as submaximum exercise in the trained vs. the untrained individual.

Peripheral factors that may limit the transport of O_2 include local muscle blood flow, extraction of O_2 by the exercising muscle, and the oxidative capacity of the muscle cell. Scientific evidence[13] strongly suggests that muscles are capable of much higher blood flows than they normally receive during maximum exercise, and that O_2 extraction is incomplete even at maximum exercise; both findings imply that the periphery does not limit the maximum transport of O_2. An exception to this may be when limited muscle mass is involved, in which case O_2 extraction may be limiting rather than C.O. The oxidative capacity of the muscle is determined by the number and size of the mitochondria and their concentration of oxidative enzymes. While this could be a rate limiting factor for VO_2max, the evidence[63] suggests that the oxidative capacity of the muscle exceeds the capacity of the circulatory system to transport O_2 to the muscle.

In conclusion, VO_2max depends on the optimal linkage between all components of the O_2 transport system. However, of all the determinants that change with physical training, the cardiac component, that is, C.O., is most adaptable and, within that, it is the SV that is most important.

Anaerobic Power

The term *anaerobic power*, or muscular endurance, refers to the capacity for those types of activities characterized by all-out exercise of brief duration (approximately 5 to 60 seconds), which are fueled mainly by the immediate and short-term (anaerobic) energy systems. In this type of exercise, the energy requirement significantly exceeds the energy generated by oxidation in the respiratory chain. As a result, the anaerobic reactions of glycolysis predominate and are characterized by large quantities of lactic acid accumulating within the muscle cell and ultimately in the blood. Typical military tasks that fall within this category are sprinting, rushing, walking or running with a heavy load, and heavy repetitive lifting.

Measurement

Unlike tests for VO_2max, no specific criteria exist to indicate that a person has reached a maximal anaerobic effort. Indeed, the ability to quantify anaerobic power is difficult due to the considerable overlap that exists in the utilization of the various energy systems during short-term, high-intensity exercise. Efforts to use the measurement of O_2 debt or accumulated lactic acid in the blood have been unsatisfactory in quantifying anaerobic power. Thus, various performance tests have been developed with which to evaluate the capacity for the immediate and short-term means of energy transfer. These tests are generally referred to as power tests, where a maximal force is generated through a distance over a short period of time (approximate range of 5 to 60 seconds).[64]

Maximal sprint runs (time for 50-200 m, or distance covered in a fixed time period) and cycling tests have typically been used to test this energy capacity; although repetitive lifting, shuttle runs, and pushups, situps, or chinups are also tests that have been used. Because of the effects that age, skill, motivation, and body size have on performance, it is difficult to select a suitable criterion test and to develop appropriate norms to evaluate anaerobic power.

Several anaerobic power tests have recently been developed using laboratory ergometers or dynamometers, which provide better precision in quantifying the power output and are more reliable than the performance tests discussed above. One of these is the isokinetic fatigue test, using a Cybex (Lumex Inc.) dynamometer on which the subject performs 50 maximal leg extensions at an angular velocity of

180°/s over a period of 1 minute.[65] Maximal and average torque values are utilized as quantifiable variables. A second test uses a cycle ergometer, where the subject pedals maximally against a very high resistance for 30 seconds.[66] Maximal and average power output values can be calculated.

Determinants

Factors important in an individual's ability to produce high power outputs during short-term exercise are the amount and type of muscle fibers involved in the exercise and the capacity for anaerobic metabolism within those fibers. In tests that measure anaerobic power, there is a strong positive relationship between the amount of power produced and the percentage of fast twitch (type 2) muscle fibers in the exercising muscle. In addition, the greater the muscle mass that is activated during high-intensity exercise, the greater the power output that is achieved.[67] Anaerobic training studies have shown that metabolic changes occurring in the muscle cell include both an increase in the concentration of glycolytic enzymes, and increased levels of anaerobic energy substrates.[68] Thus, the ability to produce power can be attributed primarily to the high anerobic capacity of the type 2 muscle fibers, the percentage of type 2 fibers in the muscle, and the absolute amount of muscle mass available for contraction.

Muscular Strength

The term "muscular strength" is difficult to define in human performance because the term has been employed in various ways by athletes, strength and conditioning specialists, physiologists, physicians, and the lay public. It has been proposed, therefore, that the term "strength" be employed to refer to the maximum force a muscle or muscle group can voluntarily generate at a specified velocity.[69] Physical activities that have a high muscular strength component are those of short duration (less than 5 seconds), where the energy is provided primarily through the splitting of stored phosphagens within the muscle cell, and those that require the generation of high force, for example, the subject must overcome a high resistance. Military tasks that have a large strength component include heavy lifting, pushing, pulling, and throwing.

Measurement

Numerous methods are available to quantify the amount of force that can be generated by a contracting muscle or group of muscles. Which method to use is frequently determined by such factors as availability of equipment, safety concerns of the subject, and the type of muscular contraction to be performed. In general, methods of strength testing are divided into two types: (1) those that measure static or isometric contractions, and (2) those that measure dynamic muscular contractions.

The assessment of static strength requires spring-type or electrical devices that measure the force produced by the muscle and are secured so as to preclude movement. Spring-type devices include dynamometers, spring balances, and the cable tensiometer. Electrical devices primarily include the wire strain gauge connected to a Wheatstone bridge circuit that is permanently mounted to form a load cell.

The cable tensiometer is typically used for measuring muscular force during knee, elbow, and trunk flexion or extension contractions. The tensiometer is lightweight, portable, durable, easy to use, and can be utilized for recording force measurements at various angles in the range-of-motion of a specific joint. Dynamometers, which operate on the principle of compression, have been developed for measuring hand-grip strength, isometric lifting capacity, and upright pull capacity. Load cells are more accurate than the above devices and can be used for assessing the force developed by most any muscle or muscle group.

In contrast to static testing, dynamic strength testing provides for a more functional assessment of strength. Dynamic testing can range from simple tests of the maximum amount of weight lifted in any one movement (eg, elbow flexion or biceps two-arm curl) just one time, to the use of sophisticated laboratory devices that allow the assessment of muscle torque at each point in the range-of-motion. The former, referred to as the one repetition maximum test (1RM), is the maximum amount of weight lifted one time with correct form during the performance of a predetermined weight-lifting exercise, usually using free weights such as barbells. In addition to free weights, there are many types of machines available where movable weights are pushed or pulled through the use of cables or bars. Such machines, however, are more frequently used for training of muscular strength than testing.

The emergence of microprocessor technology has allowed for the development of sophisticated systems that can accurately quantify muscular forces generated during a variety of movements. One such device, the isokinetic dynamometer, allows for the measurement of maximal torque through the full range-of-motion while velocity is held constant.[70]

Determinants

The single most important factor in the ability of muscle to generate force is the total mass of the muscle involved in the contraction. Regardless of gender, human skeletal muscle can generate approximately 3 to 8 kg of force / cm^2 of muscle cross-section. Studies[71] have shown that the greatest force is exerted by individuals with the largest muscle cross-sectional area, and that a linear relationship exists between strength and muscle size. In the body, however, this force-output capacity varies, depending on the arrangement of the bony levers and muscle architecture.

In addition to muscle mass, factors broadly characterized as psychologic or neural can influence the expression of human strength. The initial gains in strength during the first few weeks of a resistance training program without any measurable change in muscle size suggest that such factors are important in muscular force production. These neural factors are related to such processes as increased neural drive to the muscle, increased synchronization of the motor units, increased activation of the contractile apparatus, and inhibition of the protective mechanisms of the muscle (ie, golgi tendon organs), as well as antagonistic muscle.[34] Therefore, the production of maximal force is determined not only by the total number of muscle fibers activated (muscle mass), but also by the coordinated control of motor units.

PHYSICAL CAPACITY OF MILITARY POPULATIONS

A study of military population physical capacity demographics is valuable for several reasons. First, military accessions represent a wide cross-section of the national population and therefore are valuable in assessing national trends and making international comparisons for the young, healthy component of society. Second, population studies within the military can document trends relative to the emphasis and effectiveness of physical training programs, and help establish norms and standards. Third, the data can describe the influence that age, gender, occupation, and unit mission may have on fitness parameters.

The data presented here are largely from the U.S. Army, where most large population studies have been conducted. Where available, both direct laboratory and field tests of fitness are presented.

Aerobic Capacity

Gender

The most prominent factor in the demographics of VO_2max is gender. When factors such as training state and age are taken into account, women have approximately 30% less VO_2max than men. If body weight is taken into account, this difference drops to 25% and, if body fat is taken into account (VO_2max per kg fat-free mass), the difference diminishes to about 12%. The gender difference that remains when body weight and muscle mass are normalized is due to the larger size of the heart, blood and red cell volume, and O_2 transport capacity of the blood of men. These basic physiological differences between genders is recognized by all three U.S. military services in implementing sepa-

TABLE 13-6

EFFECT OF GENDER ON AEROBIC CAPACITY AS ASSESSED BY DIRECTLY MEASURED MAXIMAL OXYGEN UPTAKE (mL/kg BW/min)

	Mean ± SD (n)	
US Army Group	**Women**	**Men**
Army Accessions[1]	37.5 ± 3.7 (212)	51.1 ± 5.1 (210)
Army Trainees[2]	39.2 ± 3.8 (163)	53.6 ± 4.4 (176)
Army Unit Assignees[3]	39.7 ± 4.6 (238)	48.0 ± 6.3 (964)
Army Officer Cadets[4]	49.7 ± 4.2 (26)	60.6 ± 4.7 (29)

Data sources: (1) New recruits prior to basic training, from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (2) Recruits at the completion of 8 weeks of basic initial entry training, from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500. (3) Soldiers assigned to units within the continental U.S., from Fitzgerald PI, Vogel JA, Daniels WL, Dziados JE, Teves MA, Mello RP, Reich PJ. *The body composition project: a summary report and descriptive data.* US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 5-87. (4) First year cadets at the U.S. Military Academy, West Point, from Daniels WL, Kowal DM, Vogel JA, Stauffer RM. Physiological effects of a military training program on male and female cadets. *Aviat Space Environ Med.* 1979;50:562-566.

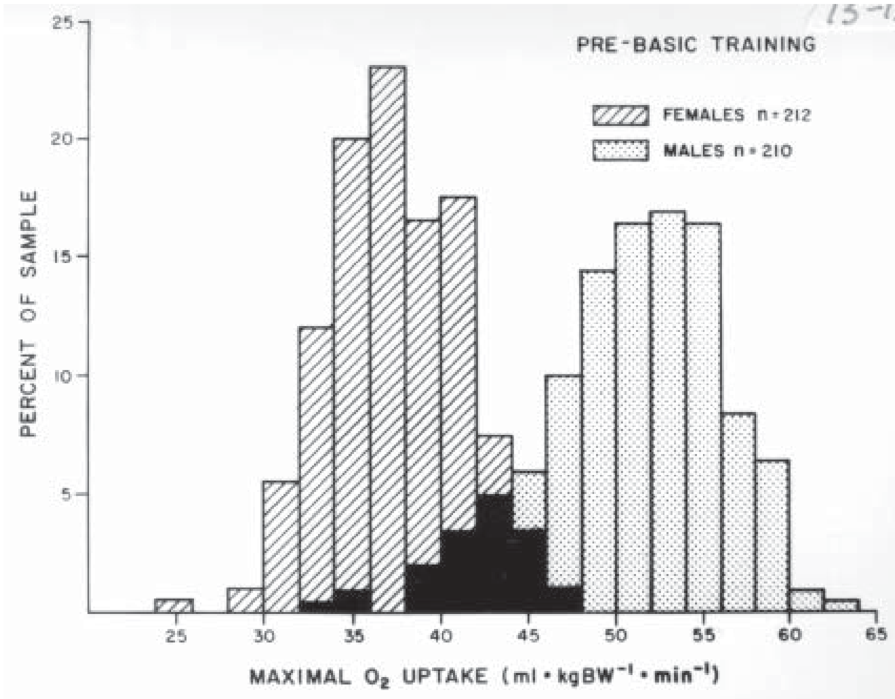

Fig. 13-16. Distribution of Vo_2max in male and female recruits at the beginning of basic training. Reprinted with permission from: Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:497.

rate aerobic fitness standards for the two genders. Available data on gender differences are summarized from several studies in Table 13-6. The minimal overlap between genders in VO_2max (per kg body weight) is illustrated in Figure 13-16. Table 13-7 presents further gender comparisons from estimated aerobic fitness data collected during a large army fitness survey utilizing the two-mile-run event on the physical fitness test.[72]

Age

Age is another generally recognized factor that influences VO_2max. As with gender, the effect of age is, to some degree, unavoidable. Although intense training can reduce or delay the aging effect, its influence can be seen in the majority of the population and, eventually, even in elite athletes.[2] The army takes this into account by adjusting the fitness standards into five age groups. The age effect on aerobic fitness is illustrated in Figure 13-17 and presented in Tables 13-7 and 13-8.

Unit Assignment and Occupation

Other factors that may influence population statistics of aerobic fitness include the type of unit to which the service member is assigned and his occupational specialty. It would be reasonable to ex-

TABLE 13-7

EFFECT OF GENDER AND AGE ON THE US ARMY PHYSICAL FITNESS TEST'S TWO-MILE-RUN FOR TIME FOR THE ESTIMATION OF AEROBIC CAPACITY*

	Minutes:Seconds, Mean (n)	
Age Group (y)	**Women**	**Men**
17-21	18:12 (111)	14:43 (1,157)
22-26	17:43 (228)	14:49 (1,688)
27-31	18:14 (164)	15:09 (1,017)
32-36	19:15 (119)	15:28 (825)
37-41	19:26 (34)	16:01 (382)
42-46	20:08 (18)	16:30 (217)
47-51	21:09 (2)	17:00 (54)

*Standard deviations not available.
Adapted with permission from O'Connor JS, Bahrke MS. 1988 Active army physical fitness survey. *Mil Med.* 1990;12:579-585.

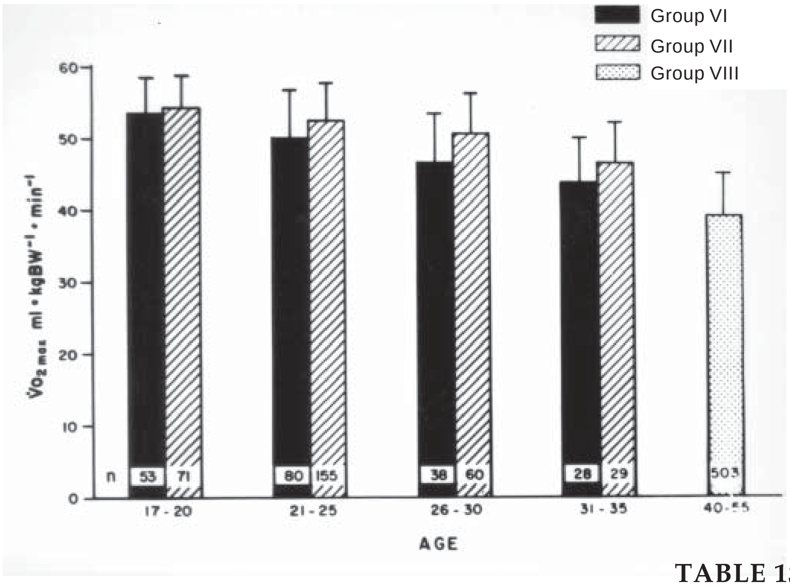

Fig. 13-17. Decline in VO_2max with age in infantry soldiers. Groups representing different training programs: VI—stateside males assigned to units in the Continental United States; VII—males assigned to an overseas combat infantry unit known for its intensive running program; and VIII—males over the age of 40 years. Reprinted with permission from Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:497.

pect that combat units, particularly infantry and infantry occupations, would show among the highest values of aerobic fitness. This may not always be the case because the emphasis placed on physical training by individual and unit training programs can vary greatly and can readily override the occupational effect of the unit or job. Thus, a headquarters or signal unit might show an exceedingly high level of VO_2max because of the commander's emphasis on fitness, regardless of the

TABLE 13-8

EFFECT OF AGE ON AEROBIC CAPACITY, ASSESSED BY DIRECTLY MEASURED MAXIMUM OXYGEN CONSUMPTION (mL/kg BW/min) IN SOLDIERS ASSIGNED TO UNITS WITHIN THE CONTINENTAL UNITED STATES

	Mean ± SD (n)	
Age Group (y)	**Women**	**Men**
17-20	41.2 ± 5.3 (51)	51.9 ± 4.5 (128)
21-27	39.6 ± 3.9 (140)	50.1 ± 5.8 (337)
28-39	38.0 ± 5.4 (46)	45.1 ± 5.7 (276)

Adapted from Fitzgerald PI, Vogel JA, Daniels WL, et al. *The body composition project: a summary report and descriptive data.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1986:Technical Report No. 5-87.

TABLE 13-9

AEROBIC CAPACITY IN A SAMPLE OF US ARMY UNITS*

Unit	n	Mean ± SD
Infantry (Korea)[1]	315	51.9 ± 6.0
Armor (Texas)[2]	62	47.0 ± 5.1
Artillery (Oklahoma)[3]	29	52.1 ± 5.5
Infantry (Washington)[4]	34	53.6 ± 5.6
Infantry (Alaska)[5]	82	57.7 ± 5.2
Infantry (California)[6]	28	58.6 ± 5.7

*Values represent directly measured maximal oxygen uptake during treadmill running (mL / kg BW / min) in male soldiers. Data sources: (1) Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500.73. Knapik J, Daniels W, Murphy M, Fitzgerald P, Drews F, Vogel J. Physiological factors in infantry operations. *Eur J Appl Physiol*. 1990; 60:233-238. (2) Wright JE, Vogel JA, Sampson JB, Knapik JJ, Patton JF, Daniels WL. Effects of travel across time zones (jet-lag) on exercise capacity and performance. *Aviat Space Environ Med.* 1983; 54:132-137. (3) Patton JF, Vogel JA, Damokosh AI, Mello RP, Knapik JJ, Drews FR. *Physical Fitness and Physical Performance During Continuous Field Artillery Operations.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. T 9-87. (4) Knapik J, Daniels W, Murphy M, Fitzgerald P, Drews F, Vogel J. Physiological factors in infantry operations. *Eur J Appl Physiol.* 1990; 60:233-238. (5) Knapik J, Bahrke M, Staab J, Reynolds K, Vogel J, O'Connor J. *Frequency of Loaded Road March Training and Performance on a Loaded Road March.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1990. Technical Report No. T13-90. (6) Mello RP, Damokosh AI, Reynolds KL, Witt CE, Vogel JA. *The Physiological Determinants of Load Bearing Performance at Different March Distances.* Natick, Mass: US Army Research Institute of Environmental Medicine;1988. Technical Report No. T 15-88.

TABLE 13-10

MAXIMAL LIFTING STRENGTH TO 152 CM AS A FUNCTION OF TRAINING STATE USING AN INCREMENTAL DYNAMIC WEIGHT STACK MACHINE (kg)

	Mean ± SD (n)	
Category	**Women**	**Men**
New Accessions*	29.5 ± 5.4 (988)	60.6 ± SD (972)
Post Training†	34.6 ± 5.8 (495)	65.8 ± 10.8 (476)
Unit Assignees‡	30.7 ± 6.8 (302)	61.0 ± 13.6 (1,078)

*Prior to basic initial entry training.
†Upon completion of basic and occupational training.
‡Personnel assigned to various units within the continental U.S.
Adapted from Sharp MA, Vogel JA. Maximal lifting strength in military personnel. In: Kumar S, ed. *Advances in Industrial Ergonomics and Safety IV*. London: Taylor & Francis; 1992.

demands of the occupations or mission. Table 13-9 presents representative values of a variety of units where directly measured VO_2max data have been collected. [29,73-77]

Muscular Strength and Muscular Endurance

Population data on strength and muscular endurance in the military services are relatively scarce. A major impediment in gathering such data is the wide variety of methods used and muscle groups assessed. One exception is the measurement of the "one repetition maximal lift" to a standard height as assessed with an incremental lifting procedure and weight stack machine.[78,79] Such a measurement of "whole body strength" has been used extensively in Armed Forces Military Entrance Processing Stations for occupational screening and qualification. Subsequently, this maximal lift measurement has been collected in a number of army studies and has been reported by Sharp and Vogel.[80] Table 13-10 summarizes this measurement as a function of training state. Figure 13-18 contrasts the gender distributions for this measurement. Measured in this way (whole body length), women average approximately one half the lifting strength of men. This is a slightly larger gender difference than that found in the strength of specific muscle groups. Gender differences are typically greater in the upper body and less in the lower body.[81]

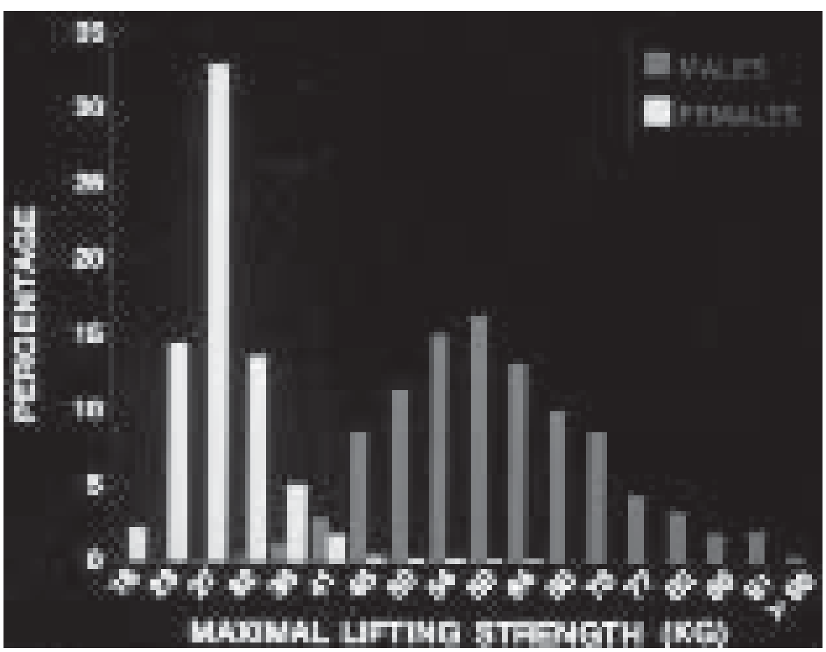

Fig. 13-18. Distribution of maximal lift strength of male and female soldiers. Adapted with permission from Sharp MA, Vogel JA. Maximal lifting strength in military personnel. In: Kumar S, ed. *Advances in Industrial Ergonomics and Safety IV*. London: Taylor & Francis; 1992: 1263.

PRINCIPLES OF PHYSICAL TRAINING

The body's capacity to perform physical activities is a function of its genetically determined body characteristics as well as the extent to which the body and its systems have adapted to a particular type and amount of exercise. While an individual has no control over the genetic component, he can bring about adaptation through an increased level of physical activity. When increased physical activity is formalized into a regular program of scheduled exercises beyond the usual living and occupational activities, it is referred to as *physical training*. Thus, physical training is the activity of imposing increasingly greater loads on the body's systems to bring about adaptive responses that permit the handling of these increased loads with less strain and stress on the individual. Once the desired level of exercise capacity is achieved, physical training must continue to retain the adaptive changes.

The adaptive response appears to have two components; (1) an initial one of depletion or breakdown, which in turn, triggers (2) rebuilding and super repletion, following the pattern of the stress reaction as first proposed in 1957 by Selye.[82] The body's response to physical training follows several well identified principles: overload, progression, specificity, detraining, and overtraining.

Training Principles

Overload

To achieve an adaptation or ability to handle greater amounts of exercise, the body must be challenged with a load greater than that to which it is accustomed. By exercising at a level above "normal" (overload), the body responds physiologically to accommodate this greater load until that load becomes the norm. The overload must be presented progressively and with sufficient intervening recovery time to avoid damage or failure to the systems involved.[83] The added load can be presented by increasing the intensity, duration, or frequency of the training activity. *Intensity* refers to the absolute level of exercise (strength of the stimulus), such as speed of running or the amount of mass lifted. Load can also be modified by adjusting the *duration* of the training bout (minutes that the stimulus is applied) and by the *frequency* (bouts of training per week) of training. Frequently, a combination of intensity, duration, and frequency are used over the course of a formal training program to produce a training overload. Application of these three methods of load adjustment will be discussed at the end of this section.

Progression

It is well established that for training to be most effective, the overload placed on the individual should be given in a regular and gradually increasing fashion. The loading stimulus must be administered frequently enough to retain the adaptation and build on the response. The stimulus then is increased so the new load again becomes an overload. This continues until the desired response is achieved or until no further gains can be elicited. At very high training intensities, large additional loads, usually in terms of duration and frequency, must be added for very small incremental gains. For optimum results, training progression should be individualized, because the starting level and final objective may be quite different among individuals; this, of course, is not usually practical in many military settings. Gains are typically greater at the beginning, more so with the less fit, and then become more gradual with time. Women army recruits typically show large percentage gains in fitness scores during basic initial entry training because of their lower relative level of physical capacity (lower percentage of potential) when they enter the service. Figure 13-19 illustrates the concepts of overload, progression, and recovery during the adaptive process to physical training.

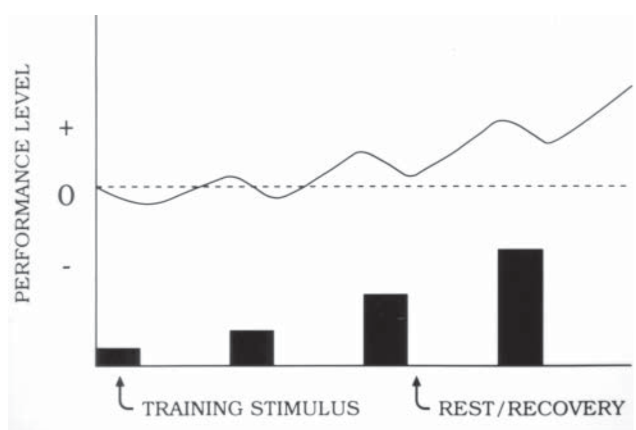

Fig. 13-19. Depiction of the training principles of overload, progression, and recovery.

Specificity

Specificity, in the context of physical training, refers to the concept that adaptive responses to training are specific to the training mode and muscles involved. For example, in general, aerobic training has little effect on muscular strength, while resistance or strength training has minimal effects on VO_2max.[84] Even within the category of aerobic training, run training has the most pronounced effects on running performance and less on other aerobic activities, such as swimming or cycling. This is due to the fact that many training adaptations, as will be discussed later in this section, occur at the cell level of the specific muscles being trained. In resistance training, the responses achieved (strength gained) are generally specific to the speed of contraction, the type of contraction (eg, isometric, isotonic, isokinetic), the muscle group involved, and the energy system utilized (stored vs anaerobic generated energy), although there can be some carry over in strength gained at low isokinetic speeds compared to that measured at higher speeds.

In practice, the principle of specificity should be applied in military physical training programs, in both general physical conditioning and job-specific physical training. For general conditioning, as conducted in basic recruit training, exercises should be selected specific to each of the three categories of fitness. Running is specific to aerobic fitness and will not enhance muscular strength of the upper body. Job-specific physical training should utilize exercises that mimic the anticipated task or that are specific to the muscle groups, intensity, and neuromuscular patterns that occur during the performance of the task; or it should include actual performance of the task.

An example of job-specific physical training would be loaded road marching for the light infantry. While running will provide an adequate base of aerobic fitness, it should not suffice as the only aerobic training for loaded road marching. Knapik and associates[74] found that the addition of road march training twice a month to a well balanced running and resistance training program improved road march performance compared to a program with no road march training. There are many anecdotal reports from military operations in the Falkland Islands and Grenada that load carriage was a major problem in troops who had trained primarily with traditional unloaded running programs.

The movement of artillery projectiles by howitzer crewmen is another task that lends itself to training specificity. Artillery crews should receive a good resistance strength/power program, but it should be supplemented by exercises that simulate the movement patterns employed in the movement of the projectiles.

Detraining

Detraining, or a decrease in the level of exercise capacity, will result when training ceases or when the frequency decreases below a minimum threshold value necessary to maintain the adaptive stimulus for that level of exercise capacity. Thus, once a desired level of exercise capacity is achieved, a level of training must be continued to maintain that level, otherwise it will return to the base level of physical activity; in other words: it is reversible. In the classical bedrest study of Saltin and colleagues,[85] a severe form of detraining, physiological capacity decreased approximately 1% per day. In a more typical form of detraining, Coyle and associates[86] showed that cessation of intense endurance training in track athletes led to a 7% decline in VO_2max over the first 21 days, and after 56 days stabilized 16% lower than initial trained values.

In a review of aerobic detraining research, Neufer[87] suggested that aerobic capacity as measured by VO_2max diminished with detraining, due to a decline in maximal SV and C.O. The latter appears to be related to an initial reduction in blood volume. As the length of detraining continues, O_2 delivery to the tissues will eventually decrease. The specific cause or causes for reduced O_2 delivery are not known.

Less information appears to be available regarding strength and resistance detraining,[88] particularly the time course. Strength detraining leads to muscle atrophy due to diminished fiber cross-sectional area.

Houmard[89] has reviewed the literature concerning detraining on subsequent performance. In well trained endurance athletes, reduction of the weekly volume (frequency and duration) of training by 70% to 80% for up to 3 to 4 weeks will not decrement maximal performance values, as long as training intensity is maintained. Neufer[87] concluded that reductions of 30% to 60% in training volume did not alter aerobic performance as long as the intensity of the exercise session was maintained. This has also been reported for strength training. Furthermore, Coyle and colleagues[86] have shown that athletes who have trained intensely over long periods are more resistant to losses in muscle adaptive changes once training ceases compared to those who have trained only a short period of time. It appears that adaptations to long-term physical training are relatively resistant to change, and major losses can be prevented with modest levels of maintenance training.

Overtraining

Overtraining is a condition characterized by failure to successfully adapt to increased training overload, and is indicated by such symptoms as general fatigue, poor performance, chronic lethargy, and inability to recover from exercise.[90] The probable cause is a too rapid progression in training load, excessive training volume, or inadequate rest and recovery time, or a combination of these. In addition to decrements in exercise capacity, the overtraining syndrome may include a depressed psychological profile, suppressed immune function, and a general disruption of homeostasis.

A state of overtraining is most commonly reported for competitive athletes who are pushing the limits of training tolerance[91]; it is not generally a concern for military unit training. Some possible exceptions include the initial stages of recruit training, elite forces training, and the train-up by individuals attempting to meet a requirement or standard in a short time period, particularly older age service members.

Central to the avoidance of this overtraining syndrome is the gradual application of small increments in loading, combined with adequate rest and recovery. Hard and light intensity workouts, or aerobic and resistance workouts, can be alternated. The recovery period is necessary for homeostasis to be reestablished to the higher level of imposed exercise load. Finally, the signs of overtraining must be heeded. These signs include nervousness or inability to relax, gradual increase in muscle soreness with continued training sessions, unexplained weight loss, inability to complete training sessions, lowered resistance to respiratory infections, loss of appetite, and unexplained drop in physical performance.[88]

The Training Response

Improvement brought about by physical training is the result of adaptive changes that occur in the body that are specific to the type of training. This is a topic of extensive investigation and review.[2,22,34] The primary responses will be reviewed here according to the category of training.

Aerobic Training

Responses to aerobic training occur primarily in the muscle and cardiovascular system. Skeletal muscle responds to aerobic-type training with increased capillarization, vascular conductance, and muscle blood flow during exercise. At the same time, the size and number of muscle cell mitochondria are increased, leading to enhanced levels of oxidative enzymes, thus increasing the oxidative capacity of the muscle. Both this and enhanced muscle blood flow result in greater potential for O_2 delivery and VO_2 by the muscle tissue during exercise.

The primary cardiovascular responses to aerobic training include increased myocardial size and contractility, enhanced blood volume along with a diminished total peripheral vascular resistance. These changes lead to a greater SV, which in turn, permits a greater maximal exercise C.O. The enhanced C.O., combined with the greater O_2 delivery and extraction, results in the greater VO_2max that occurs with aerobic training.

Resistance Training

The most noticeable response to resistance training, as compared to aerobic training, is the enlargement of the muscles being exercised. This enlargement is due to hypertrophy of the individual muscle fibers. This hypertrophy results from an increase in size and number of actin and myosin filaments, and additional sarcomeres within the fibers. Hyperplasia may also occur through the process of fiber splitting, although evidence of this in humans is still debated.

Depending on the nature of the training, the hypertrophy may be selective for fiber type. For example, glycolytic fast twitch fibers show greater enlargement in power lifters who employ high-intensity and low-volume training as opposed to body builders who use lower-intensity and higher-volume training. Increased capillarization of the muscle occurs only in the lower-intensity type of resistance training. Activity levels of the anaerobic enzymes (phosphorylase, phosphofructokinase, hexokinase, lactate dehydrogenase, and pyruvate kinase) change very little with training, and only in the type 2 fibers.

Cardiovascular changes to resistance training are relatively minimal as compared to aerobic training, although some improvement in VO_2max can be achieved. This is particularly true when rest periods are minimized in order to sustain an elevated cardiac stimulus, a prerequisite for improvements in cardiovascular capacity. Cardiac enlargement does occur in resistance training, but for different reasons than in aerobic training. In the latter, the left ventricle enlarges in response to increased demand for SV to supply a greater muscle tissue blood flow demand and a concomitant lower peripheral resistance. Resistance training, on the other hand, causes enlargement because the ventricle must pump against a higher peripheral resistance due to partial occlusion of the vessels during muscle contractions.

In addition to these major changes in the muscle and cardiovascular system, many supportive changes occur with physical training that assist in the adaptation to the higher exercise load. These include changes in the nervous and endocrine systems, connective tissue, and bone.

Neural adaptation plays an important role in resistance training. This includes an increased neural drive to the muscle, increased synchronization of motor units, increased activation of the contractile apparatus, and the release of inhibitory protective mechanisms. Neural mechanisms probably account for the rapid early increase in strength before the slower responding muscle hypertrophy occurs.

The endocrine system also supports the adaptation process in exercise training. The anabolic hormones, testosterone and growth hormone, play a role in the muscle hypertrophy process. Other hormones control the increased substrate availability that is required, that is, insulin, glucagon, thyroxine, and epinephrine.

Finally, structural changes occur in the bone and articular cartilage, which strengthen them to support greater muscle contractile forces. Bone is remodeled when subjected to increased loads with a subsequent increase in bone mineral density and greater mechanical strength. With physical training, articular cartilage increases in thickness, which leads to a greater surface area and compressibility, and both factors will reduce the force per unit area. Training also induces hypertrophy and the strengthening of the tendons and ligaments.[92]

Training Program Components

The effectiveness or gain resulting from a training program depends on the *mode* and extent of the stimulus which is applied. The extent of the training stimulus (or overload) can be further quantified in terms of *intensity* of the workout, *duration* of the exercise period, the *frequency* of the exercise bouts, and the total *length* of the training program.

Mode

The mode of the stimulus refers to the type of physical activity being used, for example, running, cycling, swimming, rowing, and so forth. The mode of activity chosen is important from the standpoint of specificity to the type of gains desired and the effectiveness of the stimulus. Swimming is not an effective training mode for sprinters. Similarly, cycling is not an appropriate training mode for arm and shoulder strength development.

Intensity

Intensity refers to the absolute metabolic or mechanical load imposed by the training program. For aerobic training, intensity is presented typically as a function of velocity, as in running, walking, swimming, skating, and so forth, or grade of incline for walking or running. In stationary cycling, intensity is applied in terms of either mechanical or electrical resistance against pedaling. The intensity of other modes of training, such as climbing, rowing, and bicycling, is more difficult to quantify in absolute terms, and is, therefore, often represented in terms of energy (calories) expended.

In resistance training, intensity is expressed as power output, that is, velocity multiplied by the load or resistance. Thus, intensity of resistance training can be adjusted by changing the speed of the contraction, as well as load displaced or the force exerted against a resistance.

Duration

Duration refers to the time over which the stimulus is applied during a training session and is applicable in both aerobic and resistance training. In resistance training, however, because exercise is typically interspersed with rest periods, it is customary to quantify the "duration" of a resistance training session in terms of the actual number of muscular contractions performed, that is, the number of sets (a period of continuous contractions without rest) and the number of contractions per set performed during a workout.

Frequency

Training frequency refers to the number of training sessions or workouts per week, month, or year.

This is an important descriptive variable since it has been shown that insufficient frequency will not produce gains or maintain an achieved gain, while excessive frequency will lead to overtraining and injuries. The frequency chosen for a particular program will depend on the mode and intensity of training but typically ranges from two to five times per week. This will be further discussed in the subsequent section on training programs.

Length

In physical training, length refers to the time period of a program chosen to meet a desired goal. Typically, programs are 8 to 12 mo long; special applications may be less or much more than this. Training for a particular athletic goal (eg, Olympic competition) may be years in length; training to achieve general fitness and health should be perpetual.

PHYSICAL TRAINING PROGRAMS

Military healthcare providers are consulted about individuals who repeatedly fail periodic physical fitness tests, who are chronically overweight or overfat, who have temporary or permanent medical profiles preventing them from physical training, who are over 40 years of age without medical clearance for testing, and who are new arrivals who may not meet the fitness standards and need individual help. This section is designed to prepare healthcare workers to provide guidance in these situations. Representative examples are given to illustrate the principles and components of training just discussed. The emphasis will be on the individual rather than the unit, although many examples are appropriate to both. Since each fitness component requires separate training activities, each component will be discussed separately, followed by some comments regarding integration into a comprehensive program.

General Considerations

The purpose of any physical training program, whether it be for a unit or an individual, should be to achieve one or more benefits or goals that are identified at the beginning. These goals should not be used for punishment and should not be used simply to pass the periodic physical fitness test. Training programs should have one or more of the following objectives: (*a*) an increase in exercise capacity, which translates into improved physical job performance and physical reserve for emergencies; (*b*) promotion of good health and military appearance (including weight and fat control); and (*c*) promotion of the positive behavioral characteristics of discipline, mental toughness, group cohesion, esprit de corps, and a feeling of self-worth and self-pride. The actual design of individual programs should take into consideration the service member's relative priority among these objectives. Group programs should include all three of these objectives and then vary only in regard to the type and degree of exercise capacity that is to be achieved.

Fitness testing, although not the appropriate end point for a program, is, nevertheless, an important tool in a physical training program. Fitness tests are useful in judging the proper starting point for a program, as well as for measuring its progress. Skilled or experienced individuals can use training activity performance to accomplish the same goal, but prescribed tests are more suitable for the inexperienced or the group training situation.

Finally, several important points of general guidance should be given to the unconditioned individual about to start a training program: (*a*) start slowly, (*b*) progress gradually, and (*c*) incorporate variation. Injuries, discouragement, and loss of interest will occur if the principles of training, particularly those of intensity and progression, are not followed.

Aerobic Training

Mode

An adequate stimulus for the aerobic component of fitness, that is, the various components of the O_2 delivery and utilization system, includes training that involves continuous, rhythmic exercise activities of a large muscle mass. The large muscle mass is needed to create the metabolic demand necessary to bring about the adaptive changes to the heart and circulation. For this reason biceps curls would not be suggested as an aerobic training activity. Appropriate aerobic activities include walking, running, cycling, swimming, rowing, stair climbing, and skiing, or any other rhythmic activity that employs the large muscle groups of the legs, arms, and shoulders. Walking with loads (backpacking) is an excellent aerobic training activity that has the advantage of lower impact forces on the lower extremities than are associated with running.

Frequency

There is ample evidence that a minimum frequency of twice per week, and more preferably three times per week, is necessary to achieve gains and to maintain a desired aerobic fitness goal. Greater gains can be achieved by training up to five times weekly, but this must be weighed against time requirements for other training activities and the potential cost in increased injuries.[93]

Intensity

An adequate stimulus for increasing aerobic capacity is an exercise intensity equivalent to 50% to 85% of VO_2max. More practically, this is equivalent to an intensity level of 60% to 90% of maximal HR reserve (age-adjusted maximal HR minus resting HR). For the beginner or low-fit individual, programs should start at the low end of this range, while those with high initial capacities will need to train at the high end of this range, referred to as the target HR.

Duration

The duration of the aerobic training session is as important as the intensity and frequency. To be effective, the stimulus to the circulation and muscle must be maintained for a minimum of 15 minutes. Training durations of 30 to 60 minutes are optimal, again depending on the starting level and the desired goal.

Summary

Running and loaded road-marching are excellent aerobic activities that take little or no equipment or facilities. Depending on the availability of facilities, other activities can be added or substituted twice a week for 15 to 20 minutes for each session, provided they involve rhythmic activity of large muscle groups at an intensity of at least 60% of maximal HR reserve. Higher intensities, longer training sessions, and more frequent sessions will bring about greater improvement. For more detailed information, the reader is referred to textbooks of exercise physiology and aerobic fitness.[2,23,94,95]

Anaerobic Power Training

Mode

Anaerobic power (muscular endurance) training modes can take several forms: (*a*) traditional resistance exercise with free weights or resistance equipment; (*b*) use of body weight for resistance; or (*c*) adapting traditional aerobic training modes by performing them in a repeated, brief, and relatively intense fashion. The choice between using free weights, weight stack machines, or other resistance machines often depends more on availability than the theoretical advantages of one system or machine over another. The theoretical advantages are more important to the serious competitor and are not of particular concern to the average service member. If no equipment is available, as may be the case in a military unit training environment, an individual can use his own body weight or the weight of a partner for resistance exercise. Many examples of partner-resisted exercises are given in the army's Physical Fitness Training manual.[96] Pushups, pullups, and situps are examples of muscular endurance training using an individual's own body weight.

Common aerobic training modes can be adapted for muscular endurance training by performing them in an anaerobic manner, including sprint running and sprint cycling for the lower body, and sprint rowing for the upper body. Sprint training should employ repeated maximal efforts of 15 to 60 seconds in duration with minimal intervening rest.

Intensity and Duration

Intensity and workout duration are closely related in anaerobic power training. There is a wide range of choices, depending on the specific goal, but the traditional approach employs one-set routines to muscle failure at a relatively low resistance (intensity), for example, 20% to 40% of 1RM. A set refers to a series of movement repetitions without stopping. The 1RM is defined as the maximal amount of weight or resistance that can be moved once. In this case, an exercise training session for a particular muscle group would consist of repeating the movement without rest at 20% to 40% of the 1RM resistance until failure.

Summary

Anaerobic power training programs are quite varied, depending on facilities available, muscle groups chosen, and specific goals. One-set, 20% to 40% of 1RM, is typically used with free weights or other weight and resistance equipment. Other modes of rhythmic exercise can be used if intensity is near maximum and rest periods are minimized. The reader is referred to Fleck and Kraemer,[34] and

O'Shea[97] for further details and examples of muscular endurance training.

Strength Training

Mode

The same training modes mentioned for muscular endurance training can also be used for strength training, but the most effective are free weights or resistance equipment. This is due to the fact that the very high loads (resistance) needed for a strength training stimulus are most readily presented with external weights rather than using body weight, running, or sprint activities. Resistance can be applied in a variety of ways: (*a*) isometrically—constant tension with no range-of-motion activity, (*b*) dynamic constant resistance (also referred to as isotonic)—constant tension throughout the range-of-motion as in typical weight lifting, (*c*) eccentrically—muscle lengthening movement (also called negative resistance), (*d*) variable resistance—changing resistance through the range-of-motion, and (*e*) isokinetic—variable resistance at constant velocity through the range-of-motion. The latter two forms of training require machines that use either cams, lever arms, pulleys, or hydraulics. The choice between these various methods is important only to the serious strength trainer or athlete. The beginner or novice should utilize either dynamic constant resistance or variable resistance, or both, depending on the availability of equipment.

Frequency

The beginner or unconditioned individual should not strength-train more than three times per week. Recovery time is particularly important at the beginning of a strength-training program.

Intensity and Duration

In contrast to muscular endurance training where many repetitions at a relatively low power intensity are employed, strength training optimally requires high intensity with few repetitions between rest. The traditional, time-proven program is performing a training exercise of (*a*) a load of 2 to 6 repetition maximums (the maximum weight or resistance that can be moved in no more than 2 to 6 repetitions), which is equivalent to 85% to 95% of 1RM; (*b*) 3 to 6 sets (one set equals 2 to 6 repetitions); and (*c*) 2 to 3 minutes of rest between sets. Beginners should exercise small muscle groups first and then larger groups, or alternate between upper body and lower body.

This is not to say that low resistance and high repetitions will not produce strength gains as long as the exercise is performed to fatigue. In fact, with an injured individual, to avoid further injuries, it may be preferable to use the less optimal but safer procedure of low resistance and high repetitions.

Summary

The most effective stimulus for strength training utilizes relatively few repetitions between ample rest periods, at near maximal strength intensities. Constant and variable resistance methods are both effective for the beginning and average weight trainer. The reader is referred to Fleck and Kraemer,[34] O'Shea,[97] and Baechle and Groves[98] for additional information.

Training Program Integration

The objective of most military physical training programs is to achieve and maintain fitness in all three categories of exercise capacity: aerobic, strength, and muscular endurance. The next step is to develop a mix of activities during the week to train all three categories. Unit programs may also wish to integrate exercises for flexibility, motor coordination, and sport competition. Table 13-11 gives

TABLE 13-11

EXAMPLE OF AN INTEGRATED TRAINING PROGRAM FOR ALL THREE CATEGORIES OF PHYSICAL FITNESS

Weekday	Category	Activity
Monday	Aerobic	Run 3–6 miles
	Anaerobic	Interval sprint runs (6-8 x 100 m)
Tuesday	Strength	2–3 sets, 8–12 repetitions
	Anaerobic	Pushups, situps, chinups
Wednesday	Aerobic	Run 3–6 miles
	Anaerobic	Circuit weight training, 1 set of low weight, high repetitions
Thursday	Strength	2–3 sets, 8–12 repetitions
Friday	Aerobic	Run 3–6 miles or specialty training, eg, loaded road march

an example program which can be used for unit training or as a guide for individual training. The example is illustrative of how all three categories of training can be integrated during a week's schedule.

Occupational or Mission-Specific Physical Training

The role and importance of training specificity has been pointed out in an earlier section. Running will not completely prepare soldiers for loaded road-marching, and resistance training on a universal gym will not optimally prepare cannoneers to move and load 45-kg projectiles for 155-mm howitzers. For these and other similar physically demanding job tasks, time should be allotted on a weekly or bimonthly basis for additional specificity training.

MEDICAL PROBLEMS ASSOCIATED WITH PHYSICAL TRAINING AND EXERCISE

The benefits and positive gains from physical training are not without costs. These costs are in the form of injury or illness resulting directly or indirectly from the training activity. The importance of this problem, and its cost to the individual in terms of lost time from work or training, as well as the costs of medical care and rehabilitation, have gained attention in the specialties of sports and occupational medicine. The specialties deal with the epidemiology, diagnosis, treatment, and prevention of injuries and illnesses associated with physical activity and occupational work. Concern within the military about the cost of activity related injuries, particularly those associated with physical training, has received considerable attention recently, and active programs in injury epidemiology research are now in progress.[99]

This section will address injuries and illnesses that are associated with physical training and operational (or occupational) activities in the military setting. This will include musculoskeletal injuries, hematological disorders, and cardiovascular disease associated with exercise, and injuries related to exercise in environmental extremes.

Musculoskeletal Injuries

Incidence Rates

Several recent studies[100-102] have documented the incidence of musculoskeletal injuries in army basic initial entry training, and infantry basic and advanced training (one station unit training) and the data are summarized in Tables 13-12 through 13-17. An injury is defined as a complaint leading to a sick call visit for treatment. Table 13-12 presents data for the 8-week army basic recruit training and demonstrates the overall high incidence rates with initial military training. Doubling these monthly rates gives a total percentage incidence of 50% and 27% for women and men, respectively, over the total period of the recruit training. These are similar to the rates reported by Kowal[103] (62% for women, 26% for men) and Bensel and Kish[104] (42% for women, 23% for men). Table 13-12 also shows that the majority of injuries are in the lower extremities. Stress fractures are singled out because of their large cost in limited duty time. The 2-fold higher rate of injuries in women is of particular concern as they move into more physically demanding jobs. Table 13-13 presents incidence rates on the sites and types of injury found during the 12 weeks of basic and advanced (one station unit) infantry training.

One might predict that incidence rates in initial training would be higher than in operational units, but this does not appear to be the case, at least in combat arms units. Recent studies[105] found similar incidence rates in men, about 12% (see Table 13-14).

TABLE 13-12

INCIDENCE OF MUSCULOSKELETAL INJURIES IN EIGHT WEEKS OF US ARMY BASIC COMBAT TRAINING*

Category	Women (n=186)	Men (n=124)	Risk Ratio†
All injuries	50.5%	27.4%	1.8:1
Lower extremity injuries	44.6%	20.9%	2.1:1
Stress fractures	12.3%	2.4%	5.1:1
Time-loss injuries	30.1%	20.2%	1.5:1
Limited duty days (n/100/mo)	129 d	40 d	3.2:1

*Fort Jackson, South Carolina, 1984
†Risk Ratio = female ÷ male injury %
Adapted from Jones BH, Manikowski RM, Harris JM, et al. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report No. T19-88.

TABLE 13-13

CUMULATIVE INCIDENCE OF TRAINING INJURIES DURING ONE INFANTRY UNIT'S 12-WEEK TRAINING SESSION (basic plus advanced, 303 men)

Training Injury	Incidence (%)
Injury Site	
Foot	10.9
Ankle	10.9
Knee	10.2
Calf	8.6
Lower back	5.9
Thigh	1.7
Hip	0.3
Injury Type	
Nonspecific pain syndrome	23.8
Muscle strain	8.6
Ankle sprain	6.3
Other sprain	1.0
Overuse knee injury	5.9
Bone stress fracture	3.0
Bone stress reaction	2.0
Fascitis	2.3
Achilles tendinitis	1.0
Bursitis	0.7
Fracture	0.7
Unknown	1.7

Adapted from Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; Technical Report No. T4-89, 1989.

One notable exception was the lower rate of stress fractures compared to new trainees, apparently a condition primarily found in new recruits and discussed later in this section.

The contrast between these injury rates and those for illness are noteworthy. Table 13-15 makes this contrast for U.S. Army basic trainees and Table 13-16 for infantry trainees and soldiers. While rates were similar in basic trainees, the number of days of limited duty were considerably greater for injuries, particularly for women. In infantry trainees and soldiers, the musculoskeletal injury rate ex-

TABLE 13-14

INCIDENCE OF MUSCULOSKELETAL INJURIES IN MALE ARMY LIGHT INFANTRY SOLDIERS*

Category	6th Infantry Div† (n = 561)	10th Mountain Div‡ (n = 351)
All injuries	11.9	27.6
Lower extremity injuries	10.6	10.8
Stress fractures	0.5	0.8
Limited duty rate (n/100/month)	80	114

*Rate expressed as % of soldiers per month; based on 2-3 month period.
†Fort Richardson, Alaska
‡Fort Drum, New York
Data source: Reynolds K, Pollard J, Cunero J, Knapik J, Jones B. Frequency of training and past injuries as risk factors for injuries in infantry soldiers. *Med Sci Sport Exerc.* 1991;23:S40.

TABLE 13-15

COMPARISON OF INJURY AND ILLNESS* RATES AMONG US ARMY BASIC TRAINEES (sick call visits/100 soldiers/mo)

Category	Injury	Illness	Risk Ratio†
One or more visits			
Women	25.2	24.2	1.0:1
Men	13.7	17.7	0.8:1
Total visits			
Women	39.6	37.2	1.1:1
Men	22.1	26.4	0.8:1
Limited duty days			
Women	129 d	6 d	22.0:1
Men	40 d	8 d	5.0:1

*All medical complaints other than musculskeletal injuries.
†Risk Ratio = injury ÷ illness rate
Data source: Jones B, Manikowski RM, Harris JM, Dziados J, Norton S, Ewart T, Vogel JA. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report No. T19-88.

TABLE 13-16

COMPARISON OF INJURY AND ILLNESS* RATES AMONG ARMY INFANTRY TRAINEES AND SOLDIERS (sick call visits/100 soldiers/mo)

Category	Injury	Illness	Risk Ratio†
One or more visits			
Trainees[1]	14.2	4.5	3.2:1
Soldiers[2]	12.8	10.0	1.3:1
Total visits			
Trainees	34.3	27.0	1.3:1
Soldiers	19.6	12.0	1.6:1
Limited duty days			
Trainees	93 d	18 d	5.2:1
Soldiers	113 d	11 d	11.0:1

*All medical complaints other than musculskeletal injuries.
†Risk Ratio = injury ÷ illness rate
Data source: (1) Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T4-89. (2) Unpublished data.

TABLE 13-17

COMPARISON OF NUMBER OF DAYS OF LIMITED DUTY DUE TO STRESS FRACTURES AND ILLNESS (limited duty days/100 soldiers/mo)

Group	Stress Fracture	Illness	Risk Ratio*
Basic training[1]			
Women	39	6	6.5:1
Men	8	8	1.0:1
Infantry training[2]	19	18	1.0:1
Infantry[3]	23	11	2.1:1

* Risk Ratio = stress fracture ÷ illness number
Data sources: (1) Jones B, Manikowski RM, Harris JM, Dziados J, Norton S, Ewart T, Vogel JA. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; Technical Report No. T19-88, 1988. (2) Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T4-89. (3) Reynolds K, Pollard J, Cunero J, Knapik J, Jones B. Frequency of training and past injuries as risk factors for injuries in infantry soldiers. *Med Sci Sport Exerc.* 1991;23:S40.

ceeded the rate of illness (medical complaints other than musculoskeletal), and again, injuries produced considerably more days of limited duty than did illness. These numbers can also be contrasted for just one injury, stress fractures, as illustrated in Table 13-17. Thus, during peacetime, musculoskeletal injuries are a significantly greater cause of lost duty time than all combined illnesses.

Risk Factors of Injury

The first step toward reducing injury rates is the identification of those factors that place individuals at risk. A list of such potential risk factors follows:[106]

- Extrinsic risk factors
  - Training program parameters
  - Footwear
  - Training surface
- Intrinsic risk factors
  - Level of physical fitness
  - Prior level of physical activity
  - Body fatness
  - Gender
  - Age
  - Prior injury history
  - Flexibility
  - Arch height
  - Other anatomical factors

Gender. It has already been pointed out (see Table 13-12) that femaleness is a risk factor for injuries in the army.[100,102] The reasons for this are not clear, but lower prior physical activity and lower fitness levels, particularly muscle strength, would seem to be implicated. Whether women are more prone to report to sick call for musculoskeletal symptoms has not been studied.

Age. The limited data available on age suggest that there is a bimodal relation in injury rate, with higher rates found in the youngest and oldest age groups, and the highest incidence at the oldest age group.[100]

Training program. Two training parameters have been examined systematically in army basic training populations by Jones and colleagues[102]: (1) running vs marching volume and (2) periods of reduced intensity. In comparing two training companies, they found that musculoskeletal injuries were one-fourth less in the company that ran 60 miles during the 12-week training cycle as compared to the company running the more typical 120 miles. Injury incidence appeared to be closely related to cumulative running mileage. In another study, reduced volume of running during the second, third, or fourth week of basic training did not reduce the incidence of stress fractures or total injuries. This period had been hypothesized as a period of increased risk due to the active cortical bone remodeling that would be taking place.

Prior physical activity and fitness level. Jones and colleagues[100] have shown that low self-assessed physical activity level and running volume prior to entering the service were negatively related to risk of injury. The quartile having the slowest one-mile-run time at the beginning of basic training had an injury incidence 3-fold greater than the quartile with the fastest run time.

Flexibility. Development and maintenance of flexibility is commonly promoted in lay literature as a preventive measure against injuries in runners and other sports. However, Jones and colleagues[100] found that the relationship between injury rate and flexibility (toe touch test) is bimodal, with the highest rates in those highly flexible and those least flexible. Moderate flexibility was associated with the least injuries.

Arch height. Another unexpected finding by Jones[102] was the relationship between arch height and injury rate. They found that the lowest injury rate (all injury types) was in the group with the flattest arches and the highest rate in those with the highest arches.

Footwear. A systematic comparison of various military footwear on injury rate has not been made. Combat boots have been implicated as a cause of stress fractures and other overuse injuries, particularly in new recruits. This has led to the extensive use of soft running shoes in aerobic training. The addition of cushioning material to the combat boot has not proven beneficial.[107]

Other possible risk factors. The research by Jones and colleagues[100,102] has also implicated other factors as being associated with higher rates of musculoskeletal injuries: higher body fat (in men), smoking, smaller stature (in women), and bowed legs.

Bone Stress Fractures

Bone stress fractures (or reactions) deserve special attention in any discussion of military musculoskeletal training injuries because of their high cost in terms of lost training time (the cause of the greatest loss of recruit training time), particularly when it occurs during initial entry training. Stress *fracture* is a misleading term since actual fracture lines are often absent. The term "stress reaction" is more suitable in describing the condition of the bone's response to acute overloading through a remodeling process that forms new periosteal bone.

The epidemiology and etiology of bone stress reactions have recently been reviewed by Jones and colleagues.[108] They reported that the incidence of bone stress reactions in military trainees is from 1% to 3% in men and 10% to 20% in women, depending on the specific criterion used. Considerable recent attention has been given to stress fractures by the Israeli Defense Force who have reported a trainee incidence rate as high as 31%,[109,110] although other reports from Israel report lower rates of about 5%.[111] The differences in rates appear to be due to the use of the more sensitive technique of radioactive bone scans. A 1993 work by Scully and colleagues[112] suggests that a high percentage of recruits show stress-positive bone scans without any symptoms.

Epidemiology studies have shown that new trainees are at a much higher risk than "seasoned" troops in whom stress reactions are seldom observed. It is also apparent that women are at a much higher risk than men, even when other risk factors are taken into account, possibly due to differences in bone density. Other risk factors include age, race, and prior level of physical activity or level of fitness.

Brudvig and associates,[113] and Gardner and colleagues[107] have shown a relationship between increasing age and incidence of stress fractures. This may be confounded with decreasing fitness with age, although lower prior fitness and activity may be an independent factor.[107] African Americans are generally reported to have lower stress fracture rates than Caucasians, which is attributed to higher bone densities in African Americans.[114]

The anatomical location of stress reactions or fractures and the timing of their occurrence probably depends on the nature, progression, and severity of training. During World War II, stress fractures were most commonly reported[113] in feet, while contemporary reports most often list the legs. Currently, the peak incidence of stress reactions in military recruits is during the first 2 to 4 weeks, probably depending, to a large degree, on the amount of running included in the training program.

The etiology of stress reactions and fractures is now thought to be the bone's physiological response to overload. As the bone is subjected to acute increases of physical stress, as in the onset of a running program in basic recruit training, it responds by remodeling its cortex, that is, by moving bone mineral from areas of low strain to areas of high strain. Thus, the lesion is not a mechanical failure of the bone, but an area of porosis.[115] The stimulus for the remodeling appears to be more related to the cyclic nature of the stress rather than its intensity.

Foot Blisters

In military populations, foot blisters are a prevalent, although less serious, type of injury associated with exercise.[104,116] It is a common cause for reporting to sick call during basic training and after loaded road-marching in experienced troops[117]; severe cases can cause several days of limited duty in affected individuals. Blisters occur from increased friction between the sock and the skin and are exacerbated by wetness. Improved sock material and use of antiperspirants[118] are being studied.

Cardiovascular Disease

Because physical training and fitness standards are emphasized, and in some cases required, at all levels of military service, there has been increased concern with exertion-related cardiac morbidity and mortality. The concern about undetected risk for heart disease was originally developed from autopsy reports of young soldiers during World War II,[119] and the U.S. Army's decision in the early 1980s to require fitness testing at all ages resulted in The U.S. Army Surgeon General's Office developing an over-40 cardiovascular risk screening program.[120] This program was designed to identify those individuals who were at significant risk, or had evidence of significant cardiovascular disease, before they underwent biannual physical fitness testing. This cardiovascular risk screening program is now integrated into the periodic physical (medical) examination (every 5 years) of all over-40 aged personnel on active duty in the army.[121] The screen is based on the seven risk factors identified in the Framington Heart Study,[122] consisting of age, gender, smoking habit, BP, resting electrocardiogram, glucose tolerance, and total serum cholesterol. A risk index (from the American Heart Association, Coronary Risk Handbook Publication 70-041-A, 1979) of 5% or more (likelihood of developing cardiovascular disease over a 6-year period) requires further medical evaluation before being cleared for physical fitness testing. The over-40 cardiovascular risk screening program was based on research carried out with U.S. Army populations of active duty personnel.[123-127]

The prevalence of risk factors and disease in military populations continues to be an active area of study.[128-131] Sudden death during exercise has various etiologies, with cardiovascular factors as one of the more prominent causes. Drory and coworkers[132] reported that cardiovascular factors accounted for 50% of sudden exertion-related deaths studied in the Israeli Defense Forces between 1974 and 1986. Exercise and sudden death have also been extensively studied in civilian populations.[133]

Exertional Rhabdomyolysis

Acute rhabdomyolysis is a condition that has historically been related to military recruit physical training.[134,135] This injury syndrome is characterized by myoglobinuria, muscle pain, weakness, and soreness. It occurs with the sudden onset of intense or excessive exercise, a situation that has previously been common in military recruit training. It can be prevented by a graduated program of exercise intensity in those recruits who are unfit or inactive. Severe rhabdomyolysis can have fatal consequences if it progresses to renal failure secondary to marked myoglobinuria and tubular necrosis. In 1988, cases of death and hospitalization due to exertional rhabdomyolysis (brought on by inappropriately sudden and intense physical training) were reported[136] in police trainees in New York City and the state of Massachusetts. This suggests that the risk for this condition for military recruit training deserves continued awareness and surveillance.

Environmentally Caused Injuries

Exercise in extreme environments, whether it be physical training or physical job tasks, may lead to injuries. The risks for injury provided by high and low temperatures, high altitude exposure, and atmospheric pollution will be briefly described. For more detailed discussion on these topics, the reader is referred to Pandolf and colleagues[137] and Vogel and colleagues.[138]

High Ambient Temperature

Heat stress is the most common environmental problem with which the exercising individual is confronted. The body is well adapted to dissipate the metabolic heat produced by exercise, but these processes may be severely compromised when they must work against high external (ambient) heat load or humidity loads, or both.

Internally generated heat is brought to the skin surface by increased C.O. and peripheral blood flow and is dissipated from the skin through radiation, convection, and conduction, and by the production and evaporation of sweat. High ambient temperature and humidity reduce the skin-to-air moisture gradient, thereby reducing the effectiveness of convection and sweat evaporation.

The body responds acutely to heat stress by increasing skin blood flow through regional redistribution of C.O. During chronic exposure to heat, a process of acclimatization occurs. This consists of an expansion of plasma volume, increased sweating, and improved circulation.

The risk of heat injury can be reduced by avoiding ambient conditions to which an individual has not acclimatized. Such conditions are best monitored with a wet-bulb globe temperature (WBGT) index:

$$WBGT = (0.7\ Tnwb) + (0.2\ Tg) + (0.1\ Tdb)$$

where Tnwb is the natural wet-bulb thermometer temperature, Tg is the black-globe thermometer temperature, and Tdb is the ordinary dry-bulb thermometer temperature. Risk index zones based on the WBGT and exercising intensity are readily available from preventive medicine officers, physical training publications, or DA Technical Bulletin Med 507.[139]

Heat injuries include the following conditions:

1. Heat cramps: the benign cramping of muscles due to an imbalance of sodium and potassium resulting from salt loss through heavy sweating during exercise in the heat. Heat cramps are treated by fluid and salt replacement.
2. Heat rash: the benign prickling sensation of the skin during prolonged sweating.
3. Dehydration and salt depletion: conditions not exclusively found with heat stress. Dehydration is indicated by decreased urine production, lethargy, anxiety, and irritability. Severe cases result in irregular gait and altered consciousness. Symptoms of salt depletion are similar to those of dehydration in its mild form. Severe forms can result in vomiting, muscle cramps, and eventually, seizures, coma, and death.
4. Heat exhaustion: potentially serious dehydration and salt depletion, but without tissue damage. Heat exhaustion is recognized by the symptoms of headache, dizziness, pallor, nausea, vomiting, uncoordinated gait, and moderately elevated body temperature. It is treated by removing the injured from the hot environment, replenishing salt and fluids, and if necessary, actively cooling the body.
5. Heat stroke: cases of heat exhaustion that have progressed to the point where thermoregulation fails and body temperature rises above 40ºC. Medically supervised aggressive measures of active cooling and repletion of electrolytes and fluid are required.

Low Ambient Temperature

Injury during exercise in the cold is much less a problem than heat induced problems, because clothing can be added to help retain the metabolic heat that is generated. Risk of injury still remains for exposed or wet skin or extremities. Physical training can continue even in severe cold as long as adequate protection to the body surface is provided. Precaution should be taken not to "overheat" during exercise in the cold, as this will produce excessive sweating and wet skin. The latter will lead to rapid and possibly dangerous cooling once exercise ceases.

Cold injury to the inappropriately clothed or accidentally exposed individual can include

1. Hypothermia: a serious condition in which body core temperature falls excessively to impair normal body function. This has been known to occur on cold days at the end of long races, such as a marathon. As the core temperature falls, blood is shunted to the core, leading to shivering and cold extremities. If the core temperature continues to decline, cardiac, pulmonary, and nervous functions will be impaired, and eventual loss of consciousness occurs. Mild hypothermia can be treated with surface rewarming, while severe cases require extensive medical care and measures to rewarm the body core.
2. Frostbite: a condition that can occur in the skin of exercising individuals when tissue water crystallizes, and causes subsequent tissue dehydration and damage. High wind-chill factors, either from the wind or the speed of exercise (eg, cycling) will greatly increase the risk. "Frost nip," or a whitening of an exposed area of skin without actual damage, is undesirable, because it may make the area susceptible to future frostbite. Frostbite can be serious and may

require amputation. Early signs include numbness and white or yellowish color of the affected surface. Treatment consists of rapid rewarming of the affected area in water at 40ºC to 42ºC, preferably under medical supervision.

High Terrestial Altitude

Military troop deployments, although not common, can take place at altitudes between 2,500 and 4,000 m, which places individuals at risk for high-altitude illness or injury. These altitudes also result in well known decrements in exercise capacity and physical performance due to the reduced PO_2 and accompanying disturbances in acid-base balance. Acclimatization to high altitude does occur and, therefore, troops should, if possible, ascend gradually to allow time for the acclimatization process to develop prior to exercising at the intended altitude.

High terrestrial altitude injury and illness include

1. Acute mountain sickness: a condition common in rapid ascent to an altitude above 2,500 m is characterized by severe headaches, nausea, vomiting, decreased appetite, and sleep disturbances. U.S. Army operational doctrine employs gradual ascent or intermediate stops, as well as prophylactic use of acetazolamide. Most people recover in 2 to 4 days as acclimatization occurs. Severe cases can be treated by descent or supplemental O_2. A portable hyperbaric chamber (Ganow Bag) has also been found useful. Severe cases may lead to cerebral edema, which must be rapidly and aggressively treated.
2. High-altitude pulmonary edema: a condition characterized by fluid leaking into the lung as the result of tissue hypoxia. Symptoms include cough, shortness of breath, and cyanosis of peripheral areas. This is a serious condition, which, if not treated with O_2 and descent, can lead to coma and death.

CONCLUSION

Physical fitness is an important goal of military training; it enables soldiers to optimally perform their physically demanding duties. Understanding the physiology of cardiovascular and muscular conditioning is necessary for the development and implementation of optimal training programs for soldiers.

REFERENCES

1. US Department of Defense. *Subject: Physical Fitness and Weight Control Programs.* Washington DC: Assistant Secretary of Defense (MRA&L), 1981. Directive No. 1308.1.
2. Astrand PO, Rodahl K. *Textbook of Work Physiology.* 3rd ed. New York: McGraw-Hill; 1986.
3. Saltin B, Gollnick PD. Skeletal muscle adaptability: significance for metabolism and performance. In: Peachey LD, Adrian RH, Geiger SR, eds. *Handbook of Physiology: Skeletal Muscle*. Section 10. American Physiological Society. Baltimore: Williams & Wilkins; 1983.
4. Hultman E. Studies on muscle metabolism of glycogen and active phosphate in man with special reference to exercise and diet. *Scand J Clin Lab Invest*. 1967; 94.
5. Bergstrom J. Energy rich phosphagens in dynamic and static work. In: Pernow B, Saltin B, eds. *Muscle Metabolism During Exercise.* New York: Plenum Press; 1971.
6. Serresse O, Lortie G, Bouchard C, Bailey MR. Estimation of the contribution of the various energy systems during maximal work of short duration. *Int J Sports Med.* 1988;9:456-460.
7. Wasserman K. Breathing during exercise. *New Engl J Med.* 1978;298:780-785.
8. Asmussen E, Neilsen M. Experiments on nervous factors controlling respiration and circulation during exercise employing blocking of blood flow. *Acta Physiol Scand.* 1964;60:103-114.

9. Sutton JR, Jones NL. Control of pulmonary ventilation during exercise and mediators in the blood: CO_2 and hydrogen ion. *Med Sci Sports*. 1979;11:198-203.

10. Brooks GA. Anaerobic threshold: review of the concept and directions for future research. *Med Sci Sports Exerc.* 1985;17:22-31.

11. Bevegard BS, Shepard JT. Regulation of the circulation during exercise in man. *Physiol Rev.* 1967;47:178-213.

12. Ekblom BA, Kilbom A, Soltysiak J. Physical training bradycardia and autonomic nervous system. *Scand J Clin Lab Invest*. 1973;32:249-256.

13. Rowell LB. *Human Circulation Regulation During Physical Stress.* New York: Oxford University Press; 1986.

14. Lewis SF, Snell PG, Taylor WF, Harma M, Graham RM, Pettinger WA, Blomqvist CG. Role of muscle mass and mode of contraction in circulatory responses to exercise. *J Appl Physiol.* 1985;58:146-151.

15. MacDougall JD, Tuxen D, Sales DG, Moroz, JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol.* 1985;58:758-790.

16. Andersen P, Saltin, B. Maximal perfusion of skeletal muscle in man. *J Physiol (Lond).* 1985;366:233-249.

17. Johnson JM, Rowell LB, Neiderberger M, Eisman MM. Human splanchnic and forearm vasoconstrictor responses to reductions of right arterial and aortic pressures. *Circ Res.* 1974;34:515-524.

18. Arner P, Engfeldt P, Lithel H. Site differences in the basal metabolism of subcutaneous fat in obese women. *J Clin Endo Metab.* 1981;53:948-952.

19. Evans DJ, Hoffman RG, Kalkhoff RG, Kissebah AH. Relationship of androgenic activity to body fat topography, fat cell morphology and metabolic aberrations in premenopausal women. *J Clin Endo Metab*. 1983;57:304-310.

20. Krotkiewski M, Bjorntorp P, Sjostrom L, Smith U. Impact of obesity on metabolism in men and women: importance of regional adipose tissue distribution. *J Clin Invest.* 1983;72:1150-1162.

21. Vague J. The degree of masculine differentiation of obesities: a factor determining predisposition to diabetes, atherosclerosis, gout, and uric calculous disease. *Am J Clin Nutr.* 1956;4:20-34.

22. McArdle WD, Katch FI, Katch VL. *Exercise Physiology. Energy, Nutrition, and Human Performance.* 3rd ed. Philadelphia, Pa: Lea & Febiger; 1991.

23. US Department of the Army. *The Army Weight Control Program.* Washington, DC: Department of the Army; 1991. Army Regulation 600-9.

24. Behnke AR, Wilmore JH. *Evaluation and Regulation of Body Build and Composition.* Englewood Cliffs, NJ: Prentice-Hall; 1974.

25. Hodgdon JA. *Body Composition in Military Services: Standards and Methods*. San Diego, Calif: Naval Health Research Center; 1990. Report No. 90-21.

26. Knapik JJ, Burse RL, Vogel JA. Height, weight, percent body fat, and indices of adiposity for young men and women entering the U.S. Army. *Aviat Space Environ Med.* 1983;54:223-231.

27. Daniels WL, Kowal DM, Vogel JA, Stauffer RM. Physiological effects of a military training program on male and female cadets. *Aviat Space Environ Med.* 1979;50:562-566.

28. Fitzgerald PI, Vogel JA, Daniels WL, Dziados JE, Teves MA, Mello RP, Reich PJ. *The Body Composition Project: A Summary Report and Descriptive Data*. US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 5-87.

29. Vogel JA, Patton JF, Mello RP, Daniels WL. An analysis of aerobic capacity in a large United States population. *J Appl Physiol.* 1986;60:494-500.

30. Teves MA, Vogel JA, Carlson DE, Schnakenberg DD. *Body Composition and Muscle Performance Aspects of the 1985 CFFS Test.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1986. Technical Report No. 12-86.

31. Askew EW, Munro I, Sharp MA, et al. *Nutritional Status and Physical and Mental Performance of Special Operations Soldiers Consuming the Ration, Lightweight or the Meal, Ready-to-Eat Military Field Ration During a 30-Day Field Training Exercise.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. 7-87.

32. Zuti WB, Golding LA. Comparing diet and exercise as weight reduction tools. *Physician Sportsmed.* 1976;4:49-53.

33. Garrow JS. Effect of exercise on obesity. *Acta Med Scand.* 1978; Suppl. 711:73-76.

34. Fleck SJ, Kraemer WJ. *Designing Resistance Training Programs.* Champaign, Ill: Human Kinetics; 1987.

35. Katch FI, Clarkson PM, Kroll W, McBride T. Effects of sit up exercise training on adipose cell size and adiposity. *Res Quart Exerc Sport.* 1984;55:242-247.

36. MacDougall JD, Sale DG, Moroz JR, Elder GCB, Sutton JR. Mitochondrial volume density in human skeletal muscle following heavy resistance training. *Med Sci Sports*. 1979;11:164-166.

37. Keys A, Brozek J, Henschel A, Mickensen O, Taylor HL. *The Biology of Human Starvation.* Minneapolis: University of Minnesota Press; 1950.

38. Vogel JA, Friedl KE. Army data: body composition and physical capacity. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

39. Pollock ML, Gettmen LR, Jackson A, Ayres J, Ward A, Linnerud AC. Body composition of elite class distance runners. *Ann NY Acad Sci.* 1977;301:361-370.

40. Maughan RJ. Relationship between muscle strength and cross-sectional area. *Sports Med.* 1984;1:263-269.

41. Friedl KE. Body composition and military performance: origins of the Army standards. In: Marriott BM, Grumstrup-Scott J, eds. *Body Composition and Physical Performance.* Washington, DC: National Academy Press; 1992.

42. US Department of the Army. *Medical Service Standards of Fitness*. Washington, DC: Department of the Army; 1991. Army Regulation 40-501.

43. US Department of the Navy. *Subject Physical Readiness Program.* Washington, DC: Office of the Chief of Naval Operations; 1986. Instruction 6110.1c.

44. US Department of the Air Force. *The Air Force Weight and Fitness Program.* Washington, DC: Department of the Air Force, 1985. Regulation 35-11.

45. US Marine Corps. *Subject: Weight Control and Military Appearance.* Washington, DC: Department of the Navy; 1986. Order 6100.1c.

46. Friedl KE, Vogel JA, Bovee MW, Jones BH. *Assessment of Body Weight Standards in Male and Female Army Recruits.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T15-90.

47. Durnin JVGA, Womersley J. Body fat assessed from total density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Brit J Nutr.* 1974;32:77-97.

48. Siri WE. Body composition from fluid spaces and density: analysis of methods. In: Brozek J, Henschel A, eds. *Techniques for Measuring Body Composition*. Washington, DC: National Academy of Sciences; 1961.

49. Lohman TG. Skinfolds and body density and their relation to body fatness: A review. *Human Biology.* 1981;53:181-225.

50. Vogel JA. *A Review of Physical Fitness as it Pertains to the Military Services.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1985. Technical Report No. T14-85.

51. Vogel JA, Friedl KE. Body fat assessment in women - special considerations. *Sports Medicine*. 1992;13:245-269.

52. Wright HF, Dotson CO, Davis PO. An investigation of assessment techniques for body composition of women Marines. *US Navy Med.* 1980;71:15-26.

53. Wright HF, Dotson CO, Davis PO. Simple technique for measurement of percent body fat in man. *US Navy Med.* 1981;72:23-27.

54. Hodgdon JA, Beckett MB. *Prediction of percent body fat for US Navy men from body circumferences and height*. San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-11

55. Hodgdon JA, Beckett, MB. *Prediction of percent body fat for US Navy women from body circumferences and height.* San Diego, Calif: Naval Health Research Center; 1984: Report No. 84-29.

56. Vogel JA, Kirkpatrick JW, Fitzgerald PI, Hodgdon JA, Harman EA. *Determination of Anthropometry Based Body Fat Equations for the Army's Weight Control Program.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988: Technical Report No. T17-88.

57. Friedl KE, DeLuca JP, Marchitelli J, Vogel JA. Reliability of body-fat estimations from a four-compartment model by using density, body water, and bone mineral measurements. *Am J Clin Nutr.* 1992;55:764-770.

58. Heymsfield SB, Lichtman S, Baumgartner RN, Wang J, Kamen Y, Aliprantis A, Pierson RN, Jr. Body composition of humans: comparison of two improved four-compartment models that differ in expense, technical complexity, and radiation exposure. *Am J Clin Nutr.* 1990;52:52-58.

59. Astrand P-O, Saltin B. Maximal oxygen uptake and heart rate in various types of muscular activity. *J Appl Physiol*. 1962;16:977-984.

60. Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol.* 1984;56:831-836.

61. Mitchell J. The physiological meaning of the maximal oxygen intake test. *J Clin Invest*. 1958;37:538-544.

62. Astrand P-O, Ryhming I. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. *J Appl Physiol*. 1954;7:218-221.

63. Sutton JR. Limitations to maximal oxygen uptake. *Sports Medicine*. 1992;13:127-133.

64. Saltin B. Metabolic fundamentals in exercise. *Med Sci Sports.* 1973;5:137-141.

65. Thorstensson A. Muscle strength, fibre types and enzyme activities in man. *Acta Physiol Scand.* 1976; Suppl 443.

66. Bar-Or O, Dotan R, Inbar O. A 30s all-out ergometric test—its reliability and validity for anaerobic capacity. *Israel J Medical Sci.* 1977;13:326-327.

67. Froese EA, Houston ME. Torque-velocity characteristics and muscle fiber type in human vastus lateralis. *J Appl Physiol*. 1985;59:309-314.

68. Gollnick PD, Hermansen L. Biochemical adaptations to exercise: anaerobic metabolism. In: *Exercise and Sport Sciences Reviews*. Vol 1. New York: Academic Press; 1977.

69. Knuttgen HG, Kraemer WJ. Terminology and measurement in exercise performance. *J Appl Sport Sci Res*. 1987;1:1-10.

70. Perrine JJ. Isokinetic exercise. *J Health Phys Ed Rec*. 1968;39:40-44.

71. Ikai M, Fukunaga T. Calculation of muscle strength per unit cross-sectional area of a human muscle by means of ultrasonic measurements. *Int Z Angew Physiol*. 1968;26:26-36.

72. O'Connor JS, Bahrke MS. 1988 Active army physical fitness survey. *Mil Med*. 1990;12:579-585.

73. Knapik J, Daniels W, Murphy M, Fitzgerald P, Drews F, Vogel J. Physiological factors in infantry operations. *Eur J Appl Physiol.* 1990;60:233-238.

74. Knapik J, Bahrke M, Staab J, Reynolds K, Vogel J, O'Connor J. *Frequency of Loaded Road March Training and Performance on a Loaded Road March*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1990. Technical Report No. T13-90.

75. Mello RP, Damokosh AI, Reynolds KL, Witt CE, Vogel JA. *The Physiological Determinants of Load Bearing Performance at Different March Distances.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report No. T 15-88.

76. Wright JE, Vogel JA, Sampson JB, Knapik JJ, Patton JF, Daniels WL. Effects of travel across time zones (jet-lag) on exercise capacity and performance. *Aviat Space Environ Med.* 1983;54:132-137.

77. Patton JF, Vogel JA, Damokosh AI, Mello RP, Knapik JJ, Drews FR. *Physical Fitness and Physical Performance During Continuous Field Artillery Operations*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1987. Technical Report No. T 9-87.

78. McDaniel JW, Skandis RJ, Madole SW. *Weight Lift Capabilities of Air Force Basic Trainees.* US Air Force Aerospace Medical Research Laboratories. Wright Patterson AFB, Oh; 1983. Technical Report No. T1.

79. Teves MA, Wright JE, Vogel JA. *Performance on Selected Candidate Screening Test Procedures Before and After Army Basic and Advanced Individual Training.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1985. Technical Report T 13-85.

80. Sharp MA, Vogel JA. Maximal lifting strength in military personnel. In: Kumar S, ed. *Advances in Industrial Ergonomics and Safety IV*. London: Taylor & Francis; 1992.

81. Laubach LL. Comparative muscular strength of men and women: a review of the literature. *Aviat Space Environ Med.* 1976;47:534-542.

82. Selye H. *The Stress of Life*. London: Longmans Green; 1957.

83. Harre D. *Principles of Sports Training: Introduction to the Theory and Methods of Training*. Berlin: Sportverlag; 1982.

84. Dudley GA, Fleck SJ. Strength and endurance training: Are they mutually exclusive. *Sports Medicine.* 1984;4:79-85.

85. Saltin B, Blomqvist B, Mitchell JH, Johnson RL, Wildenthal K, Chapman CB. Response to submaximal and maximal exercise after bed rest and training. *Circulation*. 1968; Suppl 7:38.

86. Coyle EF, Martin WH, Sinacore DR, Joyner MJ, Hagberg JM, Holloszy JO. Time course of loss of adaptations after stopping prolonged intense endurance training. *J Appl Physiol: Respirat Environ Exer Physiol.* 1984;57:1857-1864.

87. Neufer PD. The effect of detraining and reduced training on the physiological adaptations to aerobic exercise training. *Sports Medicine.* 1989;8:302-321.

88. Fleck SJ, Kraemer WJ. The overtraining syndrome. *Nat Strength Conditioning Assoc J.* 1982;4:50-51.

89. Houmard JA. Impact of reduced training on performance in endurance athletes. *Sports Medicine.* 1991;12:380-393.

90. Fry RW, Morton AR, Keast D. Overtraining in athletes: An update. *Sports Medicine.* 1991;12:32-65.

91. Kuipers H, Keizer HA. Overtraining in elite athletes: a review and directions for the future. *Sports Medicine.* 1988;6:79-92.

92. Booth FW, Gould EW. Effect of training and disuse on connective tissue. In: Wilmore JH, Keogh JF, eds. *Exercise and Sport Sciences Reviews*. Vol 3. New York: Academic Press; 1975.

93. Pollack ML, Gettman LR, Milesis CA, Bah MD, Durstine L, Johnson RB. Effects of frequency and duration of training on attrition and incidence of injury. *Med Sci Sports.* 1977;9:31-36.

94. Sharkey BJ. *Physiology of Fitness*. 3rd ed. Champaign, Ill: Human Kinetics; 1990.

95. Sharkey BJ. *New Dimensions in Aerobic Fitness.* Champaign, Ill: Human Kinetics; 1991.

96. US Department of the Army. *Physical Fitness Training*. Washington, DC: Department of the Army; 1985. Field Manual 21-20.

97. O'Shea JP. *Scientific Principles and Methods of Strength Fitness.* 2nd ed. Reading, Mass: Addison-Wesley; 1976.

98. Baechle TR, Groves BR. *Weight Training - Steps to Success.* Champaign, Ill: Human Kinetics; 1992.

99. Vogel JA. Research initiatives in training-related musculoskeletal injuries. *J US Army Med Dept.* 1991;Jan-Feb:20-22.

100. Jones B, Manikowski RM, Harris JM, Dziados J, Norton S, Ewart T, Vogel JA. *Incidence of and Risk Factors for Injury and Illness Among Male and Female Army Basic Trainees.* Natick, Mass: US Army Research Institute of Environmental Medicine; Technical Report No. T19-88, 1988.

101. Cowan D, Jones B, Tomlinson JP, Robinson J, Polly D, Frykman P, Reynolds K. *The Epidemiology of Physical Training Injuries in US Army Infantry Trainees: Methodology, Population, and Risk Factors.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1989. Technical Report No. T4-89.

102. Jones B, Bovee MW, Harris JM, Cowan D. Intrinsic risk factors for exercise-related injuries among male and female Army trainees. *Am J Sports Med.* 1993;21:705–710.

103. Kowal DM. Nature and causes of injuries in women resulting from an endurance training program. *Am J Sports Med.* 1980;8:265-269.

104. Bensel CK, Kish RN. *Lower Extremity Disorders Among Men and Women in Army Basic Training and Effects of Two Types of Boots*. Natick, Mass: US Army Natick Research & Development Laboratories; 1983 Technical Report No. TR-83/026.

105. Reynolds K, Pollard J, Cunero J, Knapik J, Jones B. Frequency of training and past injuries as risk factors for injuries in infantry soldiers. *Med Sci Sport Exerc.* 1991;23:S40.

106. Jones BH. Overuse injuries of the lower extremities associated with marching, jogging and running: a review. *Mil Med.* 1983;148:783-787.

107. Gardner LI, Dziado JE, Jones BH, Brundage JF, Harris JM, Sullivan R, Grill RP. Prevention of lower extremity stress fractures: a controlled trial of a shock absorbent insole. *Am J Public Health.* 1988;78:1563-1567.

108. Jones BH, Harris JM, Vinh TN, Rubin C. Exercise-induced stress fractures and stress reactions of bone: epidemiology, etiology, and classification. In: Pandolf KB, ed. *Exercise and Sports Sciences Reviews*. Vol 17. Baltimore, Md: Williams & Wilkins; 1989:379–422.

109. Milgrom C, Giladi M, Stein H, Kashton H, Margulies J, Chisin R, Steinberg R, Aharonson Z. Stress fractures in military recruits: a prospective study showing an unusually high incidence. *J Bone Joint Surg.* 1985;67B:732-735.

110. Hallel T, Amit S, Segal D. Fatigue fractures of the tibial and femoral shaft in soldiers. *Clin Orthop Relat Res.* 1976;118:35-43.

111. Giladi M, Ahronson Z, Stein M, Danon YL, Milgrom C. Unusual distribution and onset of stress fractures in soldiers. *Clin Orthop Relat Res*. 1985;192:142-146.

112. Scully TJ, Griffith JC, Jones B, Moreno AJ. Bone scans yield a high incidence of false positive diagnoses of stress fractures. Presented at the 1993 Annual Meeting of the American Association of Orthopedic Surgeons; Feb 18, 1993; San Francisco, Calif.

113. Brudvig TJS, Gudger TD, Obermeyher L. Stress fractures in 295 trainees: A one-year study of incidence as related to age, sex, and race. *Mil Med*. 1983;148:666-667.

114. Trutter M, Broman GE, Peterson RR. Densities of white and negro skeletons. *J Bone Joint Surg*. 1960;42A:50-58.

115. Rubin CT, Lanyon LE. Osteoregulatory nature of mechanical stimuli: Function as a determinant for adaptive remodeling in bone. *J Orthop Res*. 1987;5:300-310.

116. Bensel CK. *The Effects of Tropical and Leather Combat Boots on Lower Extremity Disorders Among US Marine Corps Recruits.* Natick, Mass: US Army Natick Research and Development Command; 1976. Technical Report 76-49-CEML.

117. Knapik J, Reynolds K, Staab J, Vogel JA, Jones B. Injuries associated with strenuous road marching. *Mil Med.* 1992;157:64-67.

118. Darrigrand A, Reynolds K, Jackson R, Hamlet M, Roberts D. Efficacy of antiperspirants on feet. *Mil Med.* 1992;157:256-259.

119. Hrubec Z, Zukel WJ. Epidemiology of coronary heart disease among young army males of WW II. *Am Heart J*. 1974;87:722-730.

120. Zoltick JM, McAllister HA, Bedynek JL. The United States Army cardiovascular screening program. *J Cardiac Rehabil.* 1984;4:530-535.

121. US Department of the Army. *Standards of Medical Fitness*. Washington, DC: Department of the Army; 1988. Army Regulation 40-501.

122. Gordon T, Sorlie P, Kannel W. *Coronary Heart Disease, Atherothrombotic Brain Infarction, Intermittent Claudication—A Multivariate Analysis of Some Study Factors Related to Their Incidence: Framington Study, 16-Year Followup*. Section 27. Washington, DC: U.S. Government Printing Office; 1971.

123. Hollenberg M, et al. *New Engl J Med*. 1985;313:600-606.

124. Patton JF, Vogel JA. Prevalence of coronary heart disease risk factors in a young military population. *Aviat Space Environ Med*. 1980;51:510-514.

125. Patton JF, Vogel JA, Bedynek JL, Alexander D, Albright R. Response of 40 and over aged military personnel to an unsupervised, self-administered aerobic training program. *Aviat Space Environ Med.* 1983;54:138-143.

126. Patton JF, Vogel JA, Bedynek JL, Alexander D, Albright R. Aerobic capacity and coronary risk factors in a middle-aged Army population. *J Cardiopul Rehab.* 1986;6:491-498.

127. Zoltick JM, Patton JF, Vogel JA, Daniels W, Bedynek JL, Davia JE. Cardiovascular screening evaluation to test for coronary artery desease in asymptomatic males over the age of 40. *J Am Coll Cardiol.* 1982;1:638.

128. Denniston JC, Jackson RE, Morgan WP, Ramos MU, Szurek JL, Vogel JA. A survey of cardiopulmonary health and coronary risk factors in a select military population. *Mil Med*. 1977;141:440-444.

129. Uhl GS, Farrell PW. Myocardial infarction in young adults: Risk factors and natural history. *Am Heart J*. 1983;105:548-553.

130. Joswig BC, Hougen T, Vieweg WVR. Heart disease in military recruits: prevalence and outcome. *J Cardiovas Res.* 1981;1:115-120.

131. Hoiberg A. *Longitudinal Study of Cardiovascular Disease in U.S. Navy Pilots.* San Diego, Calif: Naval Health Research Center. Report No. 85-7.

132. Drory Y, Dramer MR, Lev B. Exertional sudden death in soldiers. *Med Sci Sports Exerc.* 1991;23:147-151.

133. Waller BF. Exercise-related sudden death in young (age <30 years) and old (age >30 years) conditioned subjects. *Cardiovasc Clin*. 1985;15:9-73.

134. Demos MA, Gitin EL. Acute exertional rhabdomyolysis. *Arch Intern Med*. 1974;133:233-239.

135. Gitin EL, Demos MA. Acute exertional rhabdomyolysis: a syndrome of increasing importance to the military physician. *Mil Med.* 1974;139:33-36.

136. Centers for Disease Control Morbidity and Mortality Weekly Report. *Exertional rhabdomyolysis and acute renal impairment - New York City and Massachusetts: 1988*. 1990;39:751-756.

137. Pandolf KB, Sawka MN, Gonzalez RR. *Human Performance Physiology and Environmental Medicine at Terrestial Extremes.* Indianapolis: Benchmark Press; 1988.

138. Vogel JA, Rock PB, Jones BH, Havenith G. Environmental considerations in exercise testing and training (Chapter 13). In: Durstine J, King A, Painter P, Roitman J, Zwiren L, Kenney W, eds. *Resource Manual Guidelines for Exercise Testing and Prescription.* 2nd ed. Philadelphia, Pa: Lea & Febiger; 1993:129–147.

139. US Departments of the Army, Navy, and Air Force. *Occupational and Environmental Health. Prevention, Treatment and Control of Heat Injury.* Washington, DC: Departments of the Army, Navy, and Air Force; 1980. TB MED 507, NAVMED P-5052-5, AFP 160-1.