

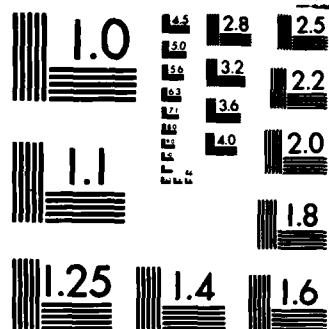
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PHYSIOLOGICAL EFFECTS OF TRAINING

by

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With the evolution of exercise science a vast amount of information concerning the physiological effects of training has been generated. Understanding the basic training responses and adaptations of various modes of conditioning can give the clinician insights into exercise prescription. The purpose of this manuscript is not to present an exhaustive review, but provide the reader with a basic overview of the physiological effects of training.

AEROBIC TRAINING

Aerobic training results in a number of adaptations in humans. The magnitude of this response is dependent upon a number of factors. It depends upon the type, the intensity, the frequency and the duration of the training; as well as the characteristics of the person undergoing training.

In this section, we will discuss the physiological changes associated with the adaptation to training and how the characteristics of the individual and the training itself affect this adaptation. *Keywords: 1473*

In order to determine the response to training, a measure of aerobic fitness is required. The best and most commonly measured laboratory assessment of aerobic fitness is the maximal oxygen uptake ($\dot{V}O_{2\max}$). Astrand¹ defines $\dot{V}O_{2\max}$ as "a measure of (1) the maximal energy output by aerobic processes, and (2) the functional capacity of the circulation..." $\dot{V}O_{2\max}$ is used to assess levels of aerobic fitness and the effects of training on aerobic fitness because it is a reliable and reproducible measure. However, in order to measure $\dot{V}O_{2\max}$ a fair amount of expensive laboratory equipment is required. O_2 consumption can be measured while performing almost any exercise, however, for laboratory purposes the exercise is usually performed on a motor driven treadmill or a cycle ergometer. Figure 1 illustrates a standard treadmill procedure for measuring $\dot{V}O_{2\max}$. The values shown (approx

45 ml/kg.min) represents a normal value for the average male 20-30 years of age. Oxygen consumption is usually reported as an absolute value or relative to body weight. Absolute refers simply to the amount of oxygen used each minute, for example 3.0 L/min, whereas for activities which require an individual to move his body weight (i.e., walking, running), values are best reported relative to body weight. In this case, someone who is walking, using 3.0 L of O_2 /minute, and has a body weight of 60 kg would be using 50 ml/kg.min of oxygen to perform this activity. This is derived in the following manner:

$$\frac{3.0 \text{ L/min}}{60\text{kg}} = \frac{3,000\text{ml/min}}{60\text{kg}} = 50 \text{ ml/kg.min.}$$

Athletes generally have higher values for $\dot{V}O_{2\text{max}}$ than their sedentary counterparts.^{2,3} Also an athlete in an endurance type sport will generally have a higher $\dot{V}O_{2\text{max}}$ than other athletes.^{1,2}

Numerous studies have reported increases in $\dot{V}O_{2\text{max}}$ as a result of aerobic training^{4,5,6,7}. This response to training is affected by several factors. The individuals' initial level of fitness is one of these factors. The effect that initial level of fitness has on the response to training was classically demonstrated by Saltin et al.⁸ They reported that 5 subjects showed a 27 percent decrease in $\dot{V}O_{2\text{max}}$ as a result of 20 days of bed rest. Subjects then went on a 50 day training program. Three of these subjects had been previously sedentary and two were physically active prior to the study. The three sedentary subjects increased their $\dot{V}O_{2\text{max}}$ by 100% over the post bed rest levels. The previously active subjects increased $\dot{V}O_{2\text{max}}$ by 34% above post bed rest levels. When compared to initial levels of fitness, however, the sedentary and active subjects increased by 33% and 4%, respectively, as a result of the 50 day training program.

In another study, Daniels et al⁹ reported increases, no change and decreases in the $\dot{V}O_{2\max}$ values of West Point cadets during a six-week basic training program. The response to training was dependent upon the initial level of aerobic fitness, with those having the lowest initial level of aerobic fitness showing an increase in $\dot{V}O_{2\max}$ and those with the highest initial level of fitness showing a decrease in $\dot{V}O_{2\max}$ as a result of training. This decrease appeared to be due mainly to an increase in lean body weight. It should be pointed out, however, that all three groups improved their performance in the 1.5 mile run as a result of training.

Thus, the initial level of fitness of the individual is an important factor to consider when evaluating the response to aerobic training. Generally, the lower the initial level of fitness the greater the percent increase in $\dot{V}O_{2\max}$ as a result of training. The individual's genetic endowment may also affect this initial level of fitness and the response to training.¹

The intensity, frequency and duration of exercise are other factors that affect the response to training. The American College of Sports Medicine recommends a frequency of 3-5 days per week at an intensity between 50-85% of $\dot{V}O_{2\max}$ for a duration of 15-60 minutes in healthy adults.⁷

Improvement in $\dot{V}O_{2\max}$ in the range of 5% to 25% can be expected as a result of a moderate training program.^{6,9,10} Improvements greater than this have been reported but these are associated with low levels of initial fitness⁸ or very intense and strenuous training programs.¹¹ The levels chosen by the American College of Sport Medicine represent those levels for intensity, duration and frequency that will provide the non-athlete with an adequate training stimulus without unnecessary risk of injury.

Studies have shown that increasing the intensity of exercise raises the drop out rate in the non-athlete runner.³ However, the importance of adequate intensity was shown by Karvonen et al¹². They showed that in two groups of young men training on a treadmill, there was no improvement in the group that ran at a heart rate of less than 135 beats/minute; whereas significant improvement occurred in the group training above 153 beats/minutes.

Miles et al¹³ illustrated the effect of increased duration (15-30-and 45-minutes) of training on aerobic fitness. They found significantly greater improvement in $\dot{V}O_{2\max}$ in the longest duration group (45 minutes) compared to the 15-minute group.

Similar results were reported by Wilmore et al¹⁰ when comparing 12 and 24 minutes of exercise, 3 days per week for 10 weeks. While both groups showed significant increases in $\dot{V}O_{2\max}$, the increase was greater in the 24 minute group.

The increase in $\dot{V}O_{2\max}$ with training also has been shown to be affected by the frequency of the training. In a study which compared two days a week and four days a week of training, Pollock et al¹⁴ found twice as much of an increase in $\dot{V}O_{2\max}$ (35% vs 17%) in the four day a week group. In another report, Pollock³ also reported increased improvement in $\dot{V}O_{2\max}$ as training increased from 1 to 3 to 5 days per week. However, in this same report he also noted a marked increase in the incidence of injury as the frequency increased beyond 3 days per week and when the duration per day increased above 30 minutes.

To summarize thus far, heredity and the initial level of fitness of the individual, the intensity, duration and frequency of the training program affect the response of the individual to a training program. Because of the

increased risk of injury, individuals who are just starting out are advised to begin at the lower intensity, duration and frequency levels given in the guidelines above. It must also be remembered in the discussion to follow that these factors may modify the changes in various organ systems.

Two other factors, age and sex, should also be mentioned when discussing physiological changes associated with aerobic training. $\dot{V}O_{2\max}$ decreases with age in adults. This has been demonstrated by Astrand¹⁵ and more recently by Vogel et al¹⁶. Vogel et al reported a decrease in $\dot{V}O_{2\max}$ at an average yearly rate of 0.5ml/kg.min. in various groups of U.S. military personnel. This agrees with the average yearly decline found by Astrand et al¹⁷ in thirty-five females and thirty-one males studied in 1949 and again in 1970. Their average yearly decline was .438 ml/kg.min and .638 ml/kg.min for females and males, respectively.

Females, on average, have $\dot{V}O_{2\max}$ values that range from 73-85% of the values of males.^{9,16,17,18,19} The physiological response to training is similar in both sexes. The most noted difference is that females often show a larger increase in $\dot{V}O_{2\max}$ as a result of training than their male counterparts.^{9,18,19} However, this is often due to the relatively lower initial level of fitness. The difference in $\dot{V}O_{2\max}$ between the sexes has been related to several factors, all of which probably have some bearing. These include higher percent body fat in women, lower blood hemoglobin in women and larger heart size and blood volumes in men.^{1,16,20}

$\dot{V}O_{2\max}$, therefore, is the laboratory measure most commonly used to assess aerobic fitness and the effect of an aerobic training program. In the remainder of this section, we will discuss the physiological changes that occur as a result of training and which are associated with the increased

aerobic fitness. We will center our discussion around the cardiovascular, cellular and metabolic changes that occur in conjunction with an increase in $\dot{V}O_{2\max}$.

For an in depth discussion of the cardiovascular changes associated with exercise and physical training, the reader is referred to the reviews by Bevegard and Shepherd,²¹ Rowell,²² Clausen,²³ and Scheuer and Tipton.²⁴ $\dot{V}O_{2\max}$ is equivalent to the product of maximal cardiac output and arterial-venous oxygen difference or:

$$\dot{V}O_{2\max} = \text{maximal cardiac output} \times \text{A-V } O_2 \text{ difference.}$$

Since cardiac output is equal to heart rate times stroke volume, oxygen consumption at any level of exercise can be calculated as follows:

$$\dot{V}O_2 \text{ (oxygen consumption)} = (\text{heart rate} \times \text{stroke volume}) \times \text{A-V } O_2 \text{ difference.}$$

Aerobic training could result in a change in $\dot{V}O_{2\max}$ by altering any one of these variables. In reality, changes occur in all three. Heart rate is generally reduced at all submaximal exercise levels including rest as a result of training. Wilmore² states that..."resting heart rate will be reduced by 1 beat/minute for each week of participation in a moderate exercise program for previously sedentary individuals." Although this statement applies only to short-term programs, the resting heart rate is normally reduced as a result of aerobic training in all age groups.¹⁰ Studies with rats indicate that after training the decreased heart rate may be due to increased parasympathetic

activity.²⁴ In addition to lower resting heart rates, training results in lower heart rates at similar exercise loads.^{23,24} For example, Pollock et al²⁵ reported a decrease of 10 to 21 beats per minute in the heart rate of middle aged men on a standard treadmill run (6.0mph, 2.5%grade) as a result of 20 weeks of running. Yoshida et al²⁶ reported similar findings after 8 weeks of training on a cycle ergometer. Maximal heart rate is either unchanged or slightly reduced as a result of aerobic training. This result is common in both sexes and across age groups.^{9,10,11,19,26,27} While the decrease in heart rate at rest is due to increased parasympathetic drive, the decrease during submaximal and maximal exercise is believed to be due to decreased sympathetic drive.^{23,28} Cardiac output does not change substantially from the pre to post-training state at the same absolute workload.^{1,20,28} Therefore in order to maintain cardiac output in conjunction with a decreased heart rate, stroke volume has to increase. Stroke volume increases in the transition from rest to exercise in all healthy subjects up to a certain heart rate. Beyond a certain heart rate (approx. 110 beats/min)²⁸ the stroke volume falls because the end-diastolic volume becomes smaller. This is due to the decreased filling time available. Trained individuals have higher stroke volumes at rest, submaximal and maximal exercise.^{1,26} Therefore, the same cardiac output is maintained at a reduced heart rate making the heart more efficient. At maximal workloads, cardiac output after training is increased significantly because stroke volume is increased and heart rate is either unchanged or only slightly decreased compared to untrained. Rowell²² reported that increased stroke volume accounted for approximately half of the 15% increase in $\dot{V}O_{2\max}$ that he saw in sedentary subjects after 3 months of training. Keul et al²⁸ recently described how moderate and intensive endurance training affect stroke

volume. Basically, they found that moderate training resulted in a reduction in adrenaline and noradrenaline (sympathetic drive) at the same absolute work load. This resulted in a decreased heart rate and arterial pressure. This allowed for more intensive filling of the heart and a larger end diastolic volume, as well as, a decreased afterload on the heart which allowed for a smaller end systolic volume. The overall effect is an increased stroke volume without any change in heart size. More intensive training results in an increase in heart size. Again the increase in stroke volume is partially due to increased end diastolic volume. Thus, the pumping ability of the heart, i.e. increased stroke volume, is improved with training; but there is only presumptive evidence of increased contractility of cardiac muscle.²⁴

As indicated above, Rowell²² reported that approximately half of the increase in $\dot{V}O_{2\max}$ could be accounted for by increased stroke volume. The remainder of the increase was due to an increase in the A-V O_2 difference. Adams et al²⁹ report similar results. This increase could be due to two factors. First of all, the cardiovascular system may have become more efficient in supplying the exercising tissue with O_2 or secondly, the tissue may be better adapted to the use of O_2 with which it is provided.

Brooks²⁰ reports that after training blood flow to active muscle is either unchanged or slightly reduced. Clausen²³ also presents evidence that during submaximal exercise, blood flow to active muscle is reduced. This, as well as other experimental findings to be discussed, strongly support the contention that trained muscles extract more oxygen from the blood. However, during maximal exercise, the blood flow to exercising muscle is also increased in the trained state.^{20,23}

Scheuer and Tipton²⁴ suggested several possible mechanisms that might be responsible for the increased A-V O₂ difference with training. These included changes in "...muscle blood flow, number of capillaries, the fiber type of muscles being recruited, alterations in concentration and activity of aerobic enzymes in cells, plus changes in the number and function of the mitochondria."

Research, to date, documents a number of changes in muscle cells as a result of training. These biochemical changes increase the ability of cells to perform aerobic metabolism. Thus, while there is no direct evidence to conclusively link these cellular adaptations to the increase in A-V O₂ difference, data strongly suggests that this is the case.

The remainder of this section will be devoted to the cellular and metabolic changes associated with aerobic training. Reviews by Holloszy,³⁰ Holloszy and Booth³¹ and Howald³² have discussed the biochemical, morphological and functional changes associated with aerobic training. Aerobic training results in a number of changes in muscle tissue itself. These changes include increases in the density of capillaries supplying muscle fibers, increases in mitochondrial densities, changes in the substrates utilized for aerobic metabolism, increases in enzyme activities supporting oxidative reactions and an increase in myoglobin content of muscle. Some studies also indicate that training can result in changes in muscle fiber type distribution patterns.^{33,34} All of these changes assist in the maintenance of cellular homeostasis during prolonged exercise.

The number of capillaries per unit of fiber has been reported to increase with physical training and to be higher in endurance trained athletes.^{35,36} Appell³⁷ presents evidence to indicate that the number of capillaries does not

change but that adaptations occur in capillaries as a result of training that cause them to travel a more "tortuous" route and this augments their cross-sectional area.

The increase in the mitochondrial densities as a result of aerobic training increases the oxidative capacity of trained muscle. Davies et al³⁸ reported a 100% increase in the mitochondrial content of muscle and in the tissue oxidative capacity as a result of 10 weeks of training in rats. They reported a strong relationship between the muscle oxidative capacity and endurance performance. Holloszy and Booth³¹ summarized the mitochondrial enzyme level increases that occur as a result of endurance training. These enzymes are involved in the activation and oxidation of fatty acids, in the Krebs cycle and in the electron-transport chain. Holloszy³⁰ listed the increases in the specific enzymes that occur as a result of aerobic training. Training increased the levels of activity of enzymes (palmityl CoA synthetase, carnitine palmityl transferase and palmityl CoA dehydrogenase) involved in the activation, transport and catabolism of long chain fatty acids. Citrate synthase, DPN-specific isocitrate dehydrogenase and succinate dehydrogenase; all enzymes involved in the Krebs cycle, showed a twofold increase in their level of activity as a result of endurance training. Davies et al³⁸ reported a 108% increase in the mitochondrial content of succinate dehydrogenase.

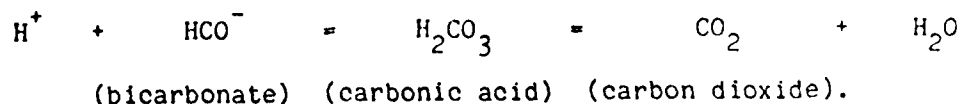
Increases in the activity of components of the electron transport chain have also been shown to occur with endurance training. Several authors^{20,30,31,38} have indicated that as a result of endurance training, the number and the content of muscle mitochondria are increased. The increase in activity levels, however, is an increase in total muscle activity, rather than an increase in the specific activity of the individual enzymes. Brooks and

Fahey²⁰ present data which shows that while mitochondrial content is increased, the specific activity of the individual components of the electron transport chain is unaffected by endurance training.

According to Holloszy³⁰ "...when skeletal muscle adapts to endurance exercise, it becomes more like cardiac muscle in that its content of mitochondria and its capacity to generate ATP from oxidation of pyruvate and fatty acids increases."

The net result of this increase in the oxidative capacity of skeletal muscle is that submaximal exercise in the trained state causes less of an alteration in cell homeostasis. The increased capacity for fatty acid oxidation results in a slower utilization of glycogen by working muscles in the trained state. This helps prevent the depletion of glycogen which has been demonstrated to be one of the factors limiting prolonged exercise.^{39,40} The glycogen sparing effect associated with training has been demonstrated by Saltin and Karlsson.⁴¹ They found that 6-8 weeks of endurance training resulted in a slower depletion of muscle glycogen for the same workload in five subjects. They also found that at the same relative per cent of $\dot{V}O_2$ max, well-trained subjects were using glycogen at a similar rate despite the fact that they were working at a higher absolute oxygen uptake. This was associated with a lower blood lactate level. One of the metabolic effects of aerobic training is a change in the workload, both absolute and per cent of $\dot{V}O_2$ max, at which lactate begins to accumulate in the peripheral blood.⁴² Gleser and Vogel⁴³ found that endurance time varied inversely with level of blood lactate. Blood lactate levels have been shown to be a useful measure for predicting the ability of individuals in endurance performance.

Costill et al⁴⁴ and Costill⁴⁵ noted that trained distance runners were capable of running at speeds much closer to maximum than untrained individuals before showing elevations in blood lactate. Since then several investigators^{46,47} have shown high correlations between some measure of blood lactate accumulation and running performance. Other investigators^{48,49} have used the concept of "anaerobic threshold" and found strong correlations with running performance. The concept of "anaerobic threshold" was introduced by Wasserman and McIlroy.⁵⁰ According to their theory, at low exercise intensities, lactate is not produced by exercising muscle and blood lactate levels are the same as at rest. At some exercise intensity, blood lactate concentration begins to increase and as ~~work~~^{exercise} intensifies, blood lactate progressively rises until the maximum effort is achieved. Furthermore, the theory states that these changes in blood lactate can be determined by changes in ventilatory parameters. This is based on the assumption that the rise in blood lactate concentration results in an exponential increase in ventilation as the ventilatory control mechanisms attempt to buffer lactic acid by blowing off "excess" CO₂. Lactic acid formed in muscle almost completely dissociates to hydrogen ion(H⁺) and lactate. The H⁺ produces CO₂ thru the following reaction:



They felt that the increase in blood lactate was linked to the onset of local muscle hypoxia at some workload. The word "anaerobic" was used to indicate that the supply of O₂ was not sufficient to meet all of the muscle energy

demands by aerobic metabolism. Recently, considerable controversy has occurred over the theory that the muscle is hypoxic and also whether ventilatory parameters are a valid indicator of lactate accumulation. The concept of anaerobic threshold was recently reviewed by a supporter⁵¹ and an opponent⁵² of the theory. Brooks⁵² does not think that the accumulation of lactate is caused by muscle tissue becoming anaerobic. He presents evidence to demonstrate that there is sufficient O_2 present to carry on aerobic metabolism. He believes lactate accumulation is not due to a sudden increase in lactate production but rather it "...is the result of (1) those processes which produce lactate and contribute to its appearance in the blood and (2) those processes which catabolize lactate after its removal from the blood." Brooks reports that, in the rat, the major effect of training on lactate metabolism is an increase in lactate clearance. This has not been demonstrated in humans.

Regardless of the outcome of this controversy, lactate accumulation in blood has become a useful measure for predicting endurance performance. However, the cause and the mechanisms responsible for the accumulation and how these mechanisms are affected by training remains to be defined.

Other metabolic effects of training center around the enhancement of mobilization and utilization of free fatty acid and the glycogen sparing effect discussed earlier. Since an in depth discussion of these change is beyond the scope of this paper, readers are referred to the following reviews.⁵³⁻⁵⁵ Basically, catecholamines, insulin and glucagon are involved in regulating lipolysis, hepatic glycogenolysis and gluconeogenesis. Galbo⁵³ reports that in trained and untrained subjects the hormonal response depends on the relative rather than the absolute workload.

Other factors which can be favorably affected by aerobic training but which will not be discussed here include body composition,⁵⁶ cardiovascular risk factors⁵⁷ and psychological well-being.⁵⁸

In summary, aerobic training results in a number of adaptations which improve not only maximal performance but also the ability to do prolonged exercise. The amount of improvement depends upon the characteristics of the individual and the training that is undergone. One of the most commonly measured changes is an increase in the maximal oxygen uptake. This is associated with an increase in the maximum cardiac output. Generally, at submaximal workloads, heart rate is decreased and stroke volume increased when compared to the untrained state. Metabolic changes include increased respiratory capacity in skeletal muscle, increased mitochondrial density and enzyme activity and lower blood lactate levels at a given workload. There is also a shift towards the use of fatty acids for fuel which results in a glycogen sparing effect in muscle. As a result of these adaptations, the cardiovascular system is generally considered to be more efficient and exercise causes less of a disturbance in the homeostasis of skeletal muscle.

RESISTANCE TRAINING

Resistance training is a popular form of conditioning in rehabilitation, fitness and athletics. The term "strength training" has been coined from the obvious objective of most resistance exercise programs. For the purpose of this manuscript the term "strength" will refer to the muscle's ability to produce force.⁵⁹

The physiological alterations consequent to resistance training are not just strength improvement. Furthermore, all resistance training programs can not be classified as being the same. Depending on the choices made for

program variables such as the choice of exercise, order of exercise, number of sets, load used and duration of rest allowed, resistance training programs can be quite diverse.^{127,130} The extent of physiological variation in response to changes in these program variables remains speculative but probably runs along a continuum.⁶⁵ The vast number of programs possible with simple manipulations probably helps explain why there are so many possible resistance training programs.

Due to variations in pre-training status of subjects, program variables and the duration of the training period, it has been difficult to make more than general observations on the apparent effects of specific resistance training programs. In the attempt to examine the physiological effects of chronic resistance training programs, cross-sectional studies of competitive lifters (olympic lifters, powerlifters and bodybuilders) have been undertaken. Certain limitations are inherent with this type of research including subjective groupings of various caliber of lifters for statistical analysis, lack of a universal definition for "elite" lifters by researchers and possible augmentation of exercise stimulus with the use of anabolic steroids. Still it appears to be our best model to examine long term training effects.

The classic response which has been observed in most studies examining resistance training is the increase in muscular strength.^{63,64,65,126,129,144} Most of the research over the past four decades had focused on the program variables of the number of sets, and the load used and its relationship to strength gains.

It is pretty well established that multiple sets of an exercise is superior to single set systems for development of maximal strength.^{62,-66} Most research indicates the use of 3 to 6 sets of a particular load.⁶⁵ It

would appear that diminishing returns occurs after 6 to 8 sets.^{135,136,137} Yet it is important to remember that these observations are for relatively novice subjects who may not have been able to tolerate the greater volume (sets x repetitions x load) of exercise stress. For experienced lifters variation in volume of training may be essential for optimal gains in strength.^{131,134}

Data from research concerning the optimal load for maximal strength gains generally suggests that gains occur with the use of a 5 or 6 repetition maximum (RM) loading.^{65,135-142} Again this observation is inferred from subject populations who are essentially novice weight trainers after a short term program. A load continuum exists.^{63,65,127,124} Generally, heavy loads (1RM to 6RM) results in the greatest strengths gains. Use of moderate (7RM to 12RM) to light (greater than 12RM) produces strength gains but the magnitude of the increase is as one moves to the lighter loads on the continuum. Data from Anderson and Kearney⁷⁰ supported this concept. In their study it was demonstrated that greatest strength gains occur with the heaviest loads and diminished with lighter loads. Atha⁶⁵ suggests that minimal returns on strength occur at about 20 to 25 RM loads.

Training is typically performed with 24 to 48 hours rest between workouts.^{162,163} The major concern is that adequate recovery is allowed between workouts. In athletes with a great deal of training background it is not unusual for workouts to be performed six days a week. A recent study by Hunter⁸⁹ demonstrated that four straight days of training in a row to be superior to alternate day workouts. This may be related to the recovery phenomenon and does require further study.

Hakkinen⁶⁸ has demonstrated the importance of pre-training status as a determinant of the magnitude of strength increase. It was shown that nonathletes accustomed to resistance training made increases in strength to nearly twice the degree of weight trained athletes in half the time. This again suggests that with an individual of low functional capacity, gains are made easily and as one gets closer to their genetic potential, more work is needed to make even small improvements.

As training periods extend to longer periods of time (i.e. over a year) the type of workouts used may need to be varied.¹³⁴ This concept has been called "cycling" or "periodization" of training.¹³¹ By changing the workout intensity (load) and volume (sets x repetitions x load) over the course of a training period, the muscle(s) apparently responds with greater maximal strength improvement. When compared to conventional resistance training programs, periodization of training even in a short term program appears to be superior for development of maximal strength.¹²⁸

The various types of strength training contraction styles (i.e. isometric, dynamic concentric/eccentric, isokinetic) have been extensively reviewed by Fleck and Schutt.⁶⁴ Each contraction style may be appropriate for different situational demands. For example, isometric programs may be appropriate in rehabilitation where pain or injury limit the range of joint motion. Dynamic programs may be more appropriate for athletes who need to train the entire joint motion maximally.^{147,148} For more definitive reviews on contraction styles and terminology the reader is referred to the following references^{59,63,65}. The effects of different contraction styles of training is related to the inherent differences between them. One must be aware of the advantages and disadvantages of each style of training. Clinical decisions

need to be made for each individual case based on the need, safety, range of motion of the strength gains and effectiveness of the exercise stimulus.

Thus, development of an optimal program for improvement of strength is dependent on the initial fitness level of the subject,⁸⁷ high intensity overload of the muscle⁶³ and variation in the exercise stimulus to maintain effectiveness.¹³¹

Cellular Changes

Strength training causes an increase in the growth of muscle.^{91,102,103} This process called hypertrophy has typically been attributed to increases in the cross-sectional area of muscle fibers, the increase in the size and number of actin and myosin filaments and addition of sarcomeres to existing muscle fibers.^{69,85,98,104,146} A lot of controversy has surrounded the concept of hyperplasia (the increase in the number of fibers) which could account for the increased size of muscle observed following different strength training programs. Initial evidence for hyperplasia was presented in animal studies.^{84,88} Over the past five years examination of bodybuilders revealed that the cross-sectional size of muscle fibers in the triceps brachii were not any different than untrained controls.^{99,118} It appeared that since the arms of these athletes were significantly larger than controls, the adaptation may be the result of an increase in the number of fibers. A recent study of the biceps brachii of bodybuilders contradicted previous evidence in the triceps brachii and thus the issue remains equivocal.¹⁷⁶

Different strength training workouts seem to result in a significantly different response when examining the capillary density of muscle. Powerlifters and olympic style lifters typically perform workouts which are high intensity and allow for longer rest periods (2 to 5 min) between sets

contrasted with bodybuilders who use moderate to high intensity loads, allow very little rest between sets and probably perform more exercises. The data from the bodybuilders suggests that this type of training program may induce capillary growth whereas powerlifting and olympic lifting styles of training may reduce it.^{115,119} The physiological reason for this adaptation and the exact stimulus requires more definitive study. How this may relate to aerobic function or cardiovascular fitness remains speculative.

Strength training appears to decrease the volume density of mitochondria. In a study by MacDougall et al.¹⁰⁰ stereological analysis indicated that the mitochondrial volume density in the triceps brachii decreased following a six month high intensity resistance training program. The 26% reduction in mitochondrial volume density seemed to indicate that heavy resistance training programs may not be suitable for those individuals concerned with aerobic performance. Yet the limited data on compatibility of aerobic metabolism and resistance training does not seem to support this fear as no decreases in aerobic capacity have been reported.²⁰ More study is needed to further understand the magnitude of this response in other muscles and when using other resistance programs with lower intensity.

Nervous System

The effects of resistance training on the nervous system response is an important factor because of the functional relationship between the nerve and the muscle.¹²⁰ Using an integrated electromyogram (EMG) methodologies, Moritani and DeVries demonstrated that significant strength gains can take place in the absence of any significant tissue hypertrophy in the initial stages (2 to 4 weeks) of a training program.¹⁰⁸ The neural component appears to be the primary mechanism underlying initial strength gains. Yet the

hypertrophy component still made the largest contribution to the increased strength observed in young adult males following training.¹⁰⁸ This was contrasted with older males whose increased strength was explained by a greater involvement of the neural component throughout the training period.¹⁰⁹ It would appear that strength gains are mediated by a different neural response to training as one grows older. It has been proposed that females may utilize similar neural mechanisms to achieve strength gains.²⁰ The effects of resistance training in females is not significantly different from males except for initial upper body strength levels and obvious hormonal differences which may be responsible for the lack of excessive hypertrophy observed in some males.⁶⁰

Data suggests that the nervous system plays a very large role in the possible mediation of strength gains.^{77,92} This may include an improved recruitment pattern, increased synchronization, longer tonic activity and less inhibition.^{73,93,106,113} The efficacy of these mechanisms in different muscles and the responses to different styles of resistance training remains unknown. Still it is clear that resistance training significantly alters neural function.

Bioenergetics and Enzyme Activities

Limited data suggests that strength training increases enzyme activities involved with splitting of energy rich phosphates, anaerobic glycolysis and glycogenolysis along with oxidation of carbohydrates.^{1,20,32} This is associated with increases that have been demonstrated for high energy phosphates and glycogen following training. The main energy source for high intensity resistance training exercise is primarily anaerobic.¹ Thus, it would appear that most adaptive changes are associated with the ability to

support anaerobic functions.^{76,86,114} This is an attractive hypothesis but one should be aware that it has been demonstrated that aerobic enzyme markers can increase following anaerobic training. Howald³² maintains that "the metabolic adaptations occurring in the different fiber populations are strongly related to their recruitment pattern during exercise." With recent advances in single fiber analysis of enzyme activities, it has been shown that most vary over more than a ten fold range implicating a spectrum of metabolic profiles with significant overlap between fibers.³² Thus, contractile properties do not always determine metabolic capacities. A great deal of study is still needed to determine the responses to different resistance training programs.

Cardiovascular Fitness

The ability to increase aerobic fitness using a resistance training program has been of interest to both scientist and practitioner.¹¹⁷ Circuit weight training programs utilizing light loads and low rest periods between sets and exercises were the primary programs used to examine this possibility.^{71,90,123} Gettman and Pollock⁶⁷ examining the various studies concerning aerobic changes with circuit weight training found only moderate increases (5 to 8%) in maximal oxygen consumption to occur following this style of training. A recent study by Hurly et al.⁹⁰ demonstrated no significant improvement in aerobic capacity was realized when a high intensity single set circuit was used. The potential lack of aerobic stimulus for this type of express circuit training was further supported in a subsequent study examining the heart rate and oxygen consumption demands.¹⁵⁸ The responses were well below recommended minimal aerobic training intensity levels. Only moderate gains are realized in aerobic function using resistance training and

are highly dependent on the fitness level of the subject.^{81,82,83} It is unlikely that endurance trained individuals would enhance aerobic fitness by using resistance training.¹⁶⁶

Preventive medicine as it relates to diet and weight control habits along with exercise have gained quite a lot of attention over the past ten years and have been linked to cardiovascular health. Most studies have demonstrated that resistance training can make effective changes in the body composition of an individual. The typical response is an increase in lean body mass and a reduction of the fat content.^{20,61} The use of more strenuous workout schedules may even produce more drastic changes as evidenced by the low body fat percentages (8 and 13%) observed in male and female competitive bodybuilders.^{80,95,111,116,125}

Elevation of certain blood lipids have been linked to cardiovascular disease. Aerobic exercise appears to reduce those blood lipid levels and thus reduce potential cardiovascular risk.⁵⁶ Conversely cross-sectional studies of competitive lifters have failed to demonstrate a similar response for chronic resistance training exercise as blood lipid levels were no different than nonathletes.⁷⁸ This seemed to implicate a less effective role for modifying blood lipids. Hurly, et al.^{159,168} demonstrated that training programs used by body builders is associated with a more favorable lipid profile than the training programs used by powerlifters. This might be related to the potential higher metabolic cost of bodybuilding workouts compared to power lifting workouts. In addition, it was shown that the use of anabolic steroids may increase the risk of coronary disease do to the adverse effects on lipid profiles. Goldberg, et al.¹⁶⁰ did recently demonstrate that weight training could have a favorable result on the blood lipid levels of previously

sedentary men and women (mean age 33 and 27 respectively). Significant reductions were observed in the absolute values of low-density lipoprotein (LDL) cholesterol and triglycerides, along with the ratios of total cholesterol-high-density lipoprotein (HDL) cholesterol and LDL cholesterol-HDL cholesterol. Thus, resistance training may be effective in reducing blood lipid levels, but not with concurrent use of anabolic steroids.

Reduction of resting blood pressure is a common response to aerobic training presumably associated with improvements in cardiovascular fitness and health.^{28,57} Conversely, resistance training has never been viewed as positively affecting resting blood pressure due to the large pressor response typically elicited with lifting activities. Furthermore much less is known about resting and exercise blood pressure responses to resistance exercise. MacDougall, et al.¹⁰¹ has reported the extreme elevations in arterial blood pressure response (255/190 to 320/250) associated with the performance of a single arm curl and a double leg press. In a recent report by Fleck and Dean⁷⁹ extreme increases in resistance exercise arterial blood pressure were observed but not to the same magnitude as the previous study. This disparity may be due to the elimination of the Valsalva maneuver usually associated with weight lifting exercise. Furthermore the level of the competitive bodybuilder used in each study may have been different.

The relationship of the exercise response to resting blood pressure has not been fully examined. Resting blood pressure responses of competitive lifters are still not reported as being abnormal or hypertensive.^{79,97,101} Short term resistance training programs have not resulted in increases in resting blood pressure response. Yet, in a report by Hunter and McCarthy¹⁵² it was demonstrated that not all individuals can tolerate intense anaerobic

training. Resting systolic blood pressures significantly increased over the course of an intense cycling and resistance training program. This response may have been related to an overtraining syndrome and lack of any periodization of training. The responses of individual subjects were variable and again pointed to the need for individual monitoring and prescription.

Hormonal Response

At the present time very little is known about the hormonal response to different resistance training programs. Most of the data has been generated from partial workouts or a single exercise response. The physiological role of hormones is typically connected with metabolic responses and growth.^{53,54,172}

It has been suggested that testosterone may be related to both strength and indirectly involved in the augmentation of exercise induced-hypertrophy. Few studies have examined the responses of testosterone in men and women to resistance training exercise. Weiss et al.¹⁷⁵ described testosterone responses in men and women following a four station exercise circuit (latissimus pull down, supine bench press, arm curl and leg press) with a 2 minute rest period between sets and exercises. Subjects exercised at 80% of their 1RM. Men significantly increased testosterone levels following the circuit and women did not. It was concluded that men have greater absolute testosterone responses to resistance training. These results may have implications for differences observed in muscle with hypertrophy associated with chronic resistance training in men and women. Other studies have demonstrated similar findings but have been unable to link resting testosterone levels to strength.^{167,171} Skjerfve¹⁷¹ did not report any increases in testosterone following a 30 minute weight lifting workout in

highly trained lifters but this study failed to report intensity levels and if subjects were using anabolic steroids. These data also suggest that intensity used plays an important role in testosterone's response to lifting exercise.

Growth hormone appears to be sensitive to intensity and frequency of lifting.^{169,173} It is involved with both the metabolic and growth process.¹⁷⁰ Data from Vanhelder et al.¹⁷³ suggests that when using a single leg press exercise load and frequency of exercise are determining factors involved with the response of growth hormone levels. It is noted that muscle lactate and oxygen deficit may also play regulatory roles in the response of growth hormone. A considerable amount of research needs to be done to gain greater understanding of the interface between the hormonal responses and resistance training adaptations.

ANAEROBIC TRAINING - ----

In addition to resistance training, use of other conditioning programs such as sprint running or cycling are also anaerobic in nature. This type of training may have more of a direct relationship to the motor activity of a some sports. The requirement for various degrees of anaerobic endurance (moderate to high intensity) in some sports is great.^{20,161} Depending upon the percent of the maximal power output used in training concurrent effects on aerobic metabolism are possible. This is evidenced by the associated cellular changes and increases in maximum oxygen consumption observed with interval sprint and cycling training which are similar to the aerobic training changes. This may be due to the number and length of exercise intervals.^{132,133} One must again remember that all effects are on a continuum so that it is difficult to determine exact thresholds for these changes. Also, the genetic predisposition of the individual plays a large role in the magnitude of the training response.^{1,32}

Interval sprint or cycle training typically increases maximum oxygen consumption. Fox, et al.¹⁵¹ has demonstrated that low power and high power output interval training in males elicits similar changes in maximum oxygen consumption, but that production of lactate during heavy submaximal exercise is reduced to a greater extent with a low power program. Similar responses have been observed with females for increases in maximal oxygen consumption¹⁵⁷ but are seemingly independent of distance and high-intensity of the interval training program.¹⁵⁰

In a study by Saltin¹⁷⁴ it is appears that the magnitude of response for maximal oxygen consumption and aerobic enzyme activity is greater for endurance training than sprint training. The sprint leg demonstrated more FT cross-sectional diameter increases.

Interval and sprint-type activities appear to significantly alter enzyme activities following training.¹⁵⁵ Roberts, et al.¹⁵⁶ demonstrated increases in enzymes (phosphofructokinase, glyceraldehyde phosphate dehydrogenase, lactate dehydrogenase, malate dehydrogenase) following sixteen training sessions consisting of 200-meter runs at 90% maxiaml speed. No increases were observed for succinate dehydrogenase. The improvement in anaerobic performance was linked to increases in key anaerobic enzyme activity. Green, et al.¹⁶⁵ also demonstrated that changes in enzyme profiles or fiber distributions do not occur in response to a short term stimulus period (2 consecutive days) of supramaxiaml exercise.

Changes in the energy substrates stored in the muscle is similar to resistance training and again depends on the aerobic component of the training program.³² High energy phosphates and glycogen increase. It is unlikely that triglycerides stores are significantly increased in those programs with high anaerobic components.

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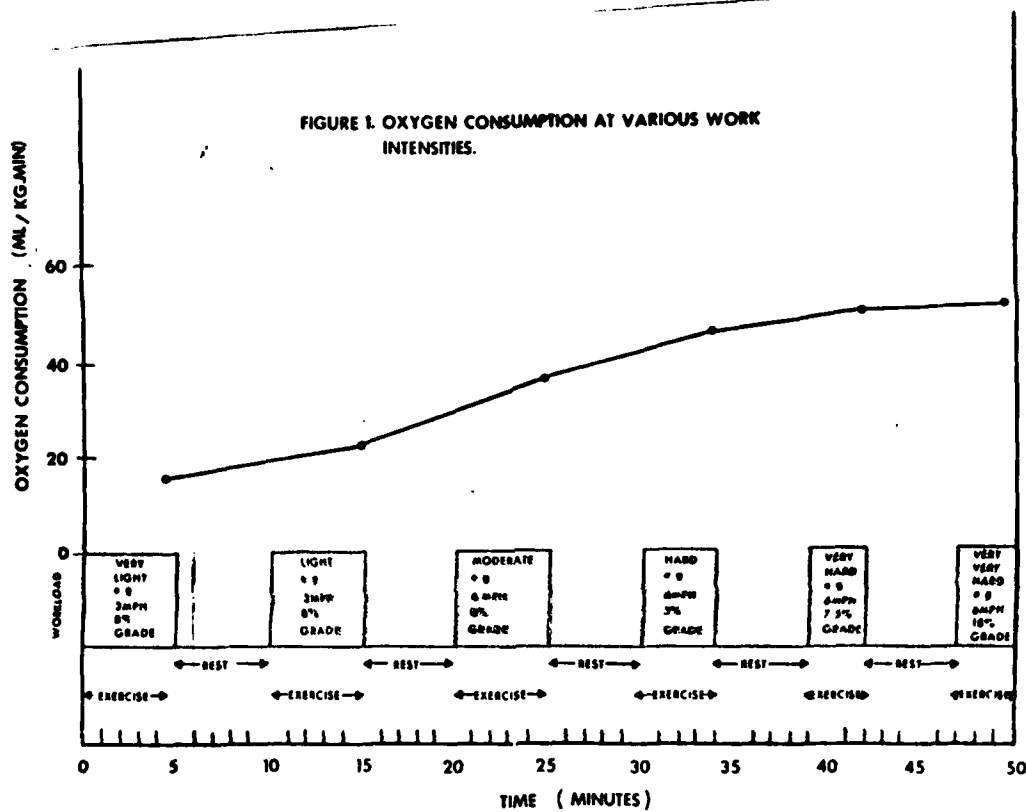
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