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## Exercise Detraining: Applicability to Microgravity

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## Reversibility of Adaptations Induced by Training

Physical training exposes the various systems of the body to potent physiologic stimuli. These stimuli induce specific adaptations that enhance an individual's tolerance for the type of exercise encountered in training. The level of adaptation and the magnitude of improvement in exercise tolerance is proportional to the potency of the physical training stimuli. Likewise, our bodies are stimulated by gravity, which promotes adaptations of both the cardiovascular and skeletal muscles. Exposure to microgravity removes normal stimuli to these systems and the body adapts to these reduced demands. In many respects the cessation of physical training in athletes and the transition from normal gravity to microgravity represent similar paradigms.

Inherent to these situations is the concept of the reversibility of the adaptations induced by training or by exposure to normal gravity. The *reversibility concept* holds that when physical training is stopped (i.e., detraining) or reduced, or a person goes from normal gravity to microgravity, the bodily systems readjust in accordance with the diminished physiologic stimuli. The focus of this chapter is on the time course of loss of the adaptations to endurance training as well as on the possibility that certain adaptations persist, to some extent, when training is stopped. Because endurance exercise training generally improves cardiovascular function and promotes metabolic adaptations within the exercising skeletal musculature, the reversibility of these specific adaptations is considered. These observations have some applicability to the transition from normal to microgravity.

## Cardiovascular Detraining

### Maximal Oxygen Uptake

Endurance training induces increases in maximal oxygen uptake (i.e., $\dot{V}O_{2max}$), cardiac output, and stroke volume [1, 2]. When sedentary men participate in a 7-week, low-intensity training program (20 min/day$^{-1}$, 3 days/week$^{-1}$), $\dot{V}O_{2max}$ levels increased by 6 percent, with a return of $\dot{V}O_{2max}$ values to pretraining levels with 8 weeks of detraining [3]. Moderate endurance training increases $\dot{V}O_{2max}$ by 10 to 20 percent, yet again $\dot{V}O_{2max}$ may decline to pretraining levels when training is stopped [4-6]. Values of $\dot{V}O_{2max}$ decline rapidly during the first month inactivity, whereas a slower decline to untrained levels occurs during the second and third months of detraining [4-6]. Therefore, the available evidence suggests that the increases in $\dot{V}O_{2max}$ produced by endurance training involving exercise of low to moderate intensities and durations are totally reversed after several months of detraining.

Investigators have not yet examined and then exposed untrained individuals to several years of intense endurance training and subsequent inactivity to determine if extreme training results in a persistent elevation of $\dot{V}O_{2max}$ above untrained levels. Our present knowledge is limited to findings of studies involving already trained endurance athletes who agreed to cease training so that reversibility of their physiologic adaptations should be studied periodically [7]. Figure 1 is a

display of the time course of the decline in $\dot{V}O_{2max}$ and its components of cardiac output, and arteriovenous $O_2$ difference when people become sedentary after training intensely for approximately 10 years.
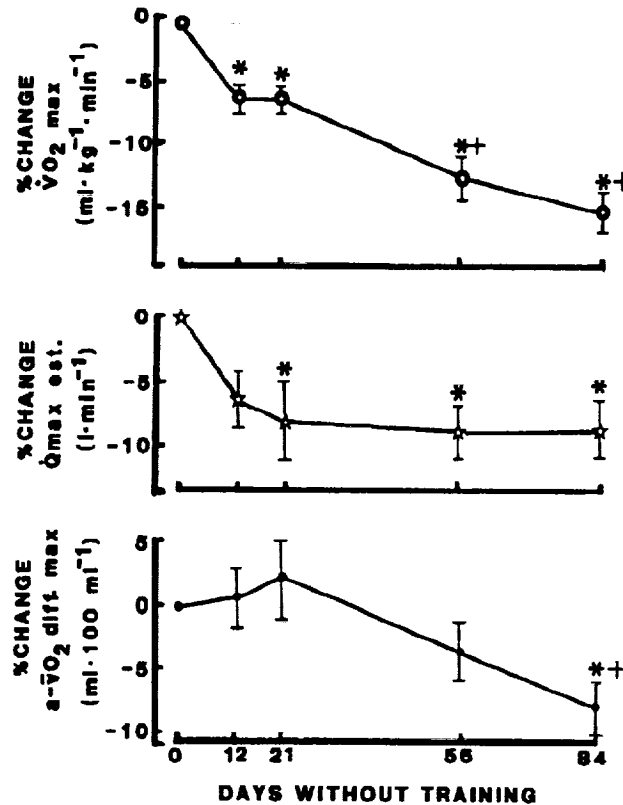


*Significantly lower than trained; $p<0.05$. +Significantly lower than day 21; $p<0.05$.

**Figure 1.** *Effects of detraining upon the percent changes in maximal oxygen uptake (i.e., $\dot{V}O_{2max}$), maximal cardiac output estimated from submaximal stroke volume and maximal heart rate (i.e., Qmax est.) and maximal arterial-venous oxygen difference (i.e., a-$\dot{V}O_{2diff\cdot max}$).*

The $\dot{V}O_{2max}$ value was relatively high in trained subjects (i.e., 62 mL/min$^{-1}$ at 0 days without training) and it declined a total of 16 percent after 84 days of detraining. A rapid decline of 7 percent occurred in the first 12 to 21 days with a further decline of 9 percent during the period from 21 to 84 days [7]. The rapid, early decline in $\dot{V}O_{2max}$ was related to a reduction in maximal stroke volume measured during exercise in the upright position. Most of the decline in stroke volume occurred during the first 12 days of inactivity. Adaptive increases in maximal heart rate compensated somewhat for this loss of stroke volume. The decline in $\dot{V}O_{2max}$ during the 21 to 84-day period was associated with a decline in maximal arteriovenous $O_2$ difference.

The 84-day period of detraining resulted in a stabilization of $\dot{V}O_{2max}$ and maximal stroke volume. Thus, the subjects appeared to have detrained for a sufficient length of time to display a complete readjustment of cardiovascular response in accordance with their sedentary lifestyle. Note that maximal stroke volume during upright exercise in the detraining subjects was virtually the same as that observed in people who had never engaged in endurance training. The idea that this finding does not necessarily imply a loss of heart function is subsequently discussed. Although maximal cardiac output and stroke volume declined to untrained levels, $\dot{V}O_{2max}$ levels in the detrained subjects remained 17 percent above that of untrained individuals, primarily because of an elevation of maximal arteriovenous $O_2$ difference. The persistent elevation of $\dot{V}O_{2max}$ values in the detrained subjects, the result of an augmented ability of the exercising musculature to extract oxygen, may be related to the observation that these subjects displayed no loss of the increased capillary density derived from the training and only a partial loss of the increase in muscle mitochondria.

## Stroke Volume and Heart Size

Prolonged and intense endurance training is thought to promote an increase in heart mass, and researchers believe detraining results in a decline in heart mass [1, 8, 9]. What is not clear, however, is whether the training-induced increases in ventricular volume and myocardial wall thickness regress totally with inactivity. Athletes who become sedentary have enlarged hearts and an elevated $\dot{V}O_{2max}$ level in contrast to people who have never trained [10].

One of the most striking effects of detraining in endurance-trained individuals is the rapid decline in stroke volume. To gain information regarding the cause of this large and rapid decline, Martin *et al.* [11] measured stroke volume during exercise in trained subjects in both the upright and supine positions and again after 21 and 56 days of inactivity (Figure 2).

A

B

○──○ Supine
●──● Upright

DAYS WITHOUT TRAINING
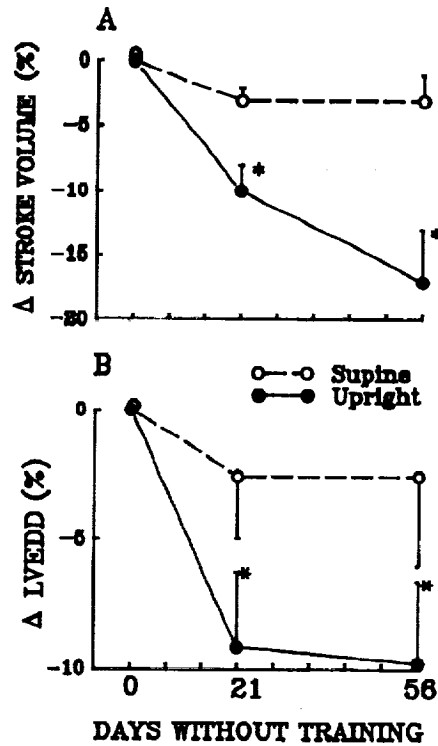
*Response in the upright position is significantly lower than the responses in the supine position ($p<0.05$).

**Figure 2.** *Percentage decline in exercise stroke volume (A) and left ventricular end diastolic diameter (LVEDD) during exercise in the upright and supine positions.*

Simultaneous measurements of the diameter of the left ventricle were obtained echocardio-graphically. The large decline in stroke volume during upright cycling was associated with parallel reductions in the diameter of the left ventricle at end-diastole (LVEDD). When the subjects were evaluated during exercise in the supine position, a condition that usually augments ventricular filling because of the drainage of blood from the elevated legs, reduction in LVEDD was minimal. As a result, stroke volume during exercise in the supine position was maintained within a few percent of trained levels during the 56-day detraining period.

## Role of Blood Volume

Along the same lines, results of recent studies indicate that the rapid reduction with detraining of stroke volume during exercise in the upright position is related to a decline in blood volume [12]. Intense exercise training usually results in an increase in blood volume by approximately 500 mL through the expansion of plasma volume [13, 14]. This adaptation is gained after only a few bouts of exercise, and it is quickly reversed when training ceases [13, 14]. The decline in stroke volume and the increase in heart rate during submaximal exercise, which normally accompanies several weeks of detraining, can be essentially reversed, and values return to near trained levels when the blood volume of detrained men expands to a level similar to that when the subjects were trained [12].

The observation that stroke volume during exercise is maintained at near trained levels when blood volume is high suggests that the ability of the heart to fill with blood is not significantly altered by detraining. If ventricular mass does indeed decline, a thinning of the ventricular walls and not a reduction in LVEDD is probably involved [11]. Thus, the reduction in intrinsic cardiovascular function is apparently minimal after several weeks of inactivity in men who had been training intensely for several years [12]. The large reduction in stroke volume during exercise in the upright position is largely a result of reduced blood volume and not of a deterioration of heart function [12].

## Detraining and Muscle Metabolism

### Enzymes of Energy Metabolism

Endurance exercise training induces enzymatic adaptations in the exercising musculature that result in slower rates of glycogen utilization and lactate production and improved endurance during submaximal exercise [15]. One of the more important alterations is an increase in the activity of mitochondrial enzymes, which results in an increased ability to metabolize fuels in the presence of oxygen. Moderate endurance training (2 to 4 months duration) increases mitochondrial enzyme activity by 20 to 40 percent from untrained levels [5, 16]. When moderate training ceases, however, and the stimuli for adaptation are removed, the increases in mitochondrial activity are quickly and totally reversed. Mitochondrial activity returns to pretraining levels within 28 to 56 days after the cessation of training [5, 16].

The pattern of change in enzyme activity observed when individuals who trained intensely for 10 years stopped training for 84 days is curvilinear [17]. Mitochondrial enzyme activity in trained subjects (i.e., citrate synthase, succinate dehydrogenase, malate dehydrogenase, and ß hydroxyacyl-CoA dehydrogenase) is initially two-fold higher than those in untrained persons, declines progressively during the first 56 days of detraining, and stabilizes at levels that are 50 percent higher than the values obtained from sedentary control subjects. The half time of decline is approximately 12 days (i.e., declines on half the distance between trained and detrained in 12 days). Therefore, prolonged and intense training, in contrast to training programs that last only a few months, appears to result in only a partial loss of mitochondrial enzyme activity and thus a persistent elevation of activity above untrained levels. This elevation occurred almost entirely because of a persistent 80 percent elevation above untrained levels in the mitochondrial enzyme activity in fast-twitch muscle fibers [18].

### Muscle Capillarization

Endurance training promotes increased capillarization of the exercising musculature, which theoretically both prolongs the transit time of blood flow through the muscle and reduces diffusion distances, thus improving the availability of oxygen and nutrients to the muscle while also allowing for better removal of metabolic waste products. Moderate endurance training of several months duration increases muscle capillarization by 20 to 30 percent above pretraining levels [5, 19]. Results of preliminary studies indicate that certain indices of muscle capillarization remain somewhat higher than pretraining levels 8 weeks after the cessation of moderate training [5].

More prolonged and intense training increases muscle capillary density by 40 to 50 percent from untrained levels [7, 19]. No indication exists that increases in muscle capillary density in highly trained people are reversed during 3 months of detraining [7].

## Muscular Adaptations that Persist with Detraining

The detraining responses in the skeletal musculature of highly trained people who regularly engaged in intense exercise for several years apparently differ from those in individuals who have trained for only a few months. No loss of the increase in muscle capillarization occurs with the cessation of prolonged intense training, although such a loss does occur when moderate training is stopped. The cessation of moderate training results in a complete reversal of the training-induced increases in mitochondrial enzyme activity, whereas only a partial decline and therefore a persistent elevation of mitochondrial activity above untrained levels occurs with the cessation of exercise after prolonged intense endurance training [3, 5, 16, 17].

## Exercise Responses of Detrained Subjects

Currently, scant evidence is available to imply that the cardiovascular or skeletal musculature adaptations derived from mild and moderate endurance training are maintained above pretraining levels with cessation of training for more than approximately 8 weeks. Therefore, a person should be stressed to the same degree during exercise of a given intensity whether untrained or after a prolonged detraining period. This hypothesis has yet to be fully evaluated, however, and one factor to consider is the possibility that people may perceive exercise to be more comfortable when they are in the detrained state having already experienced physical training, as compared to the untrained condition.

In agreement with the findings that individuals who exercise intensely on a regular basis for several years remain superior in the detrained state with respect to their muscle metabolism and intrinsic heart function (i.e., stroke volume when ventricular filling is high) compared with untrained people, it appears that these detrained people can exercise more intensely before becoming inordinately stressed. One indication of this ability is the observation that detrained persons not only possess a $\dot{V}O_{2max}$ level that is well above untrained values, but also they maintain the ability to exercise at a high percentage of $\dot{V}O_{2max}$ before lactic acid begins to accumulate in the blood [17].

## Summary

Our hypothesis is that the alterations in physiological function upon transition from normal gravity to microgravity may share similar trends to the process of "detraining." When physical training ceases (i.e., detraining), the bodily systems readjust in accordance with the diminished physiologic stimuli, and many training-induced adaptations are reversed to varying extents. The available evidence to date suggests that the increases in $\dot{V}O_{2max}$ produced by endurance training of low to moderate intensities and durations are totally reversed after several months of detraining. When people detrain after several years of intense training, they display large reductions (i.e., 5 to 15 percent) in stroke volume and $\dot{V}O_{2max}$ during the first 12 to 21 days of inactivity. These declines do not indicate a deterioration of heart function, but instead are largely a result of reduced blood volume and the ability to return venous blood to the heart. The $\dot{V}O_{2max}$ of endurance athletes continues to decline during the 21 to 56 days of detraining because of reductions in maximal arteriovenous $O_2$ difference. These reductions are associated with a loss of mitochondrial enzyme activity within the trained musculature, which declines with a half-time of approximately 12 days. Endurance athletes, however, do not regress to levels displayed by individuals who never participated in exercise training. Levels of mitochondrial enzyme activity remain 50 percent higher

than those of sedentary subjects, skeletal muscle capillarization is maintained at high levels, and $\dot{V}O_{2max}$ and the maximal arteriovenous $O_2$ difference stabilize at a point that is 12 to 17 percent higher than untrained levels after 84 days of detraining.

## References

[1] BLOMQVIST, C. G. AND B. SALTIN. Cardiovascular adaptations to physical training. *Ann. Rev. Physiol.* 45:169-189, 1983.

[2] ROWELL, L. B. Human cardiovascular adjustments to exercise and thermal stress. *Physiol. Rev.* 54:75-159, 1974.

[3] ORLANDER, J., K. H. KIESSLING, J. KARLSSON, AND B. EKBLOM. Low intensity training, inactivity and resumed training in sedentary men. *Acta Physiol. Scand.* 101:351-362, 1977.

[4] FOX, E. L., ET AL. Frequency and duration of interval training programs and changes in aerobic power. *J. Appl. Physiol.* 38:481-484, 1975.

[5] KLAUSEN, K., L. B. ANDERSEN, AND I. PELLE. Adaptive changes in work capacity, skeletal muscle capillarization and enzyme levels during training and detraining. *Acta Physiol. Scand.* 113:916, 1981.

[6] DRINKWATER, B. L. AND S. M. HORVATH. Detraining effects on young women. *Med. Sci. Sports Exerc.* 4:91-95, 1972.

[7] COYLE, E. F., ET AL. Time course of loss adaptations after stopping prolonged intense endurance training. *J. Appl. Physiol.* 57:1857-1864, 1984.

[8] EHSANI, A. A., J. M. HAGBERG, AND R. C. HICKSON. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *Am. J. Cardiol.* 42:52-56, 1978.

[9] HICKSON, R. C., G. T. HAMMONS, AND J. O. HOLLOSZY. Development and regression of exercise-induced cardiac hypertrophy in rats. *Am. J. Physiol.* 236:H268-272, 1979.

[10] SALTIN, B. AND G. G. GRIMBY. Physiological analysis of middle-aged and old former athletes: comparison with still active athletes of the same ages. *Circulation* 38:1104-1115, 1968.

[11] MARTIN, W. H., E. F. COYLE, S. A. BLOOMFIELD, AND A. A. EHSANI. Effects of physical deconditioning after intense training on left ventricular dimensions and stroke volume. *J. Am. Coll.Cardiol.* 7:982989, 1986.

[12] COYLE, E. F., M. K. HEMMERT, AND A. R. COGGAN. Effects of detraining on cardiovascular responses to exercise: role of blood volume. *J. Appl. Physiol.* 60:95-99, 1986.

[13] CONVERTINO, V. A., ET AL. Exercise training-induced hypervolemia: role of plasma albumin, renin, and vasopressin. *J. Appl. Physiol.* 48:665-669, 1980.

[14] GREEN, H. J., ET AL. Alterations in blood volume following short-term supramaximal exercise. *J. Appl. Physiol.* 56:145-149, 1984.

[15] HOLLOSZY, J. O. AND E. F. COYLE. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J. Appl. Physiol.* 56:831-838, 1984.

[16] HENRIKSSON, J. AND J. S. REITMAN. Time course of changes in human skeletal muscle succinate dehydrogenase and cytochrome oxidase activities and maximal oxygen uptake with physical activity and inactivity. *Acta Physiol. Scand.* 99:91-97, 1977.

[17] COYLE, E. F., ET AL. Effects of detraining on responses to submaximal exercise. *J. Appl. Physiol.* 59:853-859, 1985.

[18] CHI. M. M-Y., ET AL. Effects of detraining on enzymes of energy metabolism in individual human muscle fibers. *Am. J. Physiol.* 244:C276-287, 1983.

[19] INGJER, F. Capillary supply and mitochondrial content of different skeletal muscle fiber types in untrained and endurance-trained men: a histochemical and ultrastructural study. *Eur. J. Appl. Physiol.* 40:197-209, 1979.

[20] BLOMQVIST, C. G. AND H. L. STONE. Cardiovascular adjustments to gravitational stress. In: *The Handbook of Physiology. The Cardiovascular System,* edited by J. T. Shepherd, F. M. Abboud, and S. R. Geiger. Bethesda, MD: American Physiology Society, 1982.