



## CHAPTER 21

# DECONDITIONING AND RETENTION OF ADAPTATIONS INDUCED BY ENDURANCE TRAINING

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The human organism possesses an amazing ability to respond to the stimulus of regular exercise. After several weeks of training, specific systems (e.g.; cardiovascular, muscular, nervous) that are stressed display physiological adaptations which improve tolerance for the specific 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 training stimuli.

Although training promotes a variety of physiological adaptations, long periods of inactivity (i.e., detraining) are associated with reversal of many of these adaptations. The "reversibility concept" holds that, when physical training is stopped or reduced, systems readjust in accordance with diminished physiologic stimuli. The focus of this chapter is on the time course of loss of 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 exercising skeletal musculature, the reversibility of these specific adaptations is considered.

### CARDIOVASCULAR DETRAINING

#### Maximal Oxygen Uptake

Endurance training induces increased in maximal oxygen uptake ( $\dot{V}O_2\text{max}$ ), cardiac output, and stroke volume (1, 2). When sedentary people participate in a 6–10 week, low-intensity training program,  $\dot{V}O_2\text{max}$  increases by 6–10% and may remain at this level for 2–3 weeks after cessation of low intensity training (3, 4). However, prolonged detraining (8–10 weeks) results in a return of  $\dot{V}O_2\text{max}$  to pretraining level (5). Moderate endurance training increases  $\dot{V}O_2\text{max}$  by 10–20%, yet  $\dot{V}O_2\text{max}$  may decline to pre-training levels when training is stopped (6–9).  $\dot{V}O_2\text{max}$  declines rapidly during the first month of inactivity, whereas a slower decline to un-

trained levels occurs during the second and third months of detraining (6–9). Therefore, the available evidence suggests that increases in  $\dot{V}O_2\text{max}$  produced by endurance training resulting from exercise of low to moderate intensity and duration are totally reversed after several months of detraining and a sedentary lifestyle.

Studies involving already-trained endurance athletes who cease training have allowed study of the reversibility of physiological adaptation and whether long-term, intense endurance training results in a persistent maintenance of  $\dot{V}O_2\text{max}$  (10). Figure 21.1 illustrates the time course of decline in  $\dot{V}O_2\text{max}$ , maximal stroke volume, heart rate and arteriovenous oxygen ( $a-vO_2$ ) difference induced from detraining after intense training for approximately 10 years.

$\dot{V}O_2\text{max}$  value was relatively high initially in these trained subjects (62 ml/kg/min) and it declined 16% after 84 days of detraining. A rapid decline of (7%) occurred in the first 12–21 days with a subsequent decline (9%) during days 21–84. The rapid, early decline in  $\dot{V}O_2\text{max}$  was associated with a reduction in maximal stroke volume and cardiac output, despite increased heart rate (Fig. 21.1). Most of the decline in stroke volume occurred during the first 12 days of inactivity. Adaptive increases in maximal heart rate partially compensated for loss of stroke volume. The decline in  $\dot{V}O_2\text{max}$  during the 21–84 days was associated with a decline in maximal  $a-vO_2$  difference.

The 84-day period of detraining resulted in stabilization of  $\dot{V}O_2\text{max}$  and maximal stroke volume. Thus, subjects appeared to have detrained for a sufficient length of time to display a complete readjustment of cardiovascular response in accordance with a sedentary lifestyle. Maximal stroke volume during upright exercise in detrained subjects is the same as that observed in those who had never engaged in endurance training (Table 21.1). This finding does not necessarily imply decreased cardiovascular function. Although maximal cardiac output and stroke volume declined to untrained levels,

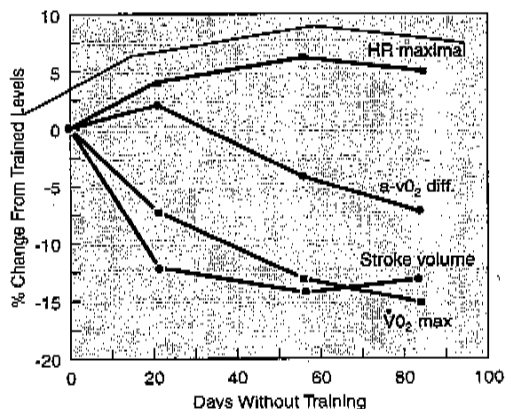


Figure 21.1. Effects of detraining upon percent change in stroke volume during exercise, maximal O<sub>2</sub> uptake (VO<sub>2</sub>max), maximal heart rate (HR), and maximal arteriovenous O<sub>2</sub> difference (a-vO<sub>2</sub> diff.). (Modified from Coyle EF, et al. Time course of loss adaptations after stopping prolonged intense endurance training. *J Appl Physiol* 57:1857-1864, 1984.)

Table 21.1. Responses of Highly Trained Individuals After 3 Months of Detraining Compared to Sedentary Controls

	SEDENTARY CONTROL	% OF SEDENTARY CONTROL	
		TRAINED	DETRAINED 3 MONTHS
VO <sub>2</sub> max (ml/kg/min)	43.3	143%*	117%†
Stroke Volume (ml)	128	120%*	101%†
a-vO <sub>2</sub> diff at max (ml/100 ml)	12.6	122%*	116%†
Citrate Synthase Activity (mol/kg/hr)			
Whole Muscle of Both Fiber Types	4.1	243%*	149%*†
Type I Fibers	4.8	140%*	108%†
Type II Fibers	2.6	246%*	180%†
Capillary Density (cap/mm <sup>2</sup> )	318	146%*	150%*

Trained responses are expressed as a percentage of sedentary control values. \* Higher ( $p < 0.05$ ) than sedentary control. † Detrained lower than trained:  $p < 0.05$ . (Modified from Coyle EF, et al. Time course of loss adaptations after stopping prolonged intense endurance training. *J Appl Physiol* 57:1857-1864, 1984; Coyle EF, Martin WM, Bloomfield SA, et al. Effects of detraining on responses to submaximal exercise. *J Appl Physiol* 59:853-859, 1985; Costill DL, et al. Metabolic characteristics of skeletal muscle during detraining from competitive swimming. *Med Sci Sports Exerc* 17:339-343, 1985.)

VO<sub>2</sub>max in detrained subjects remained 17% higher than untrained individuals, primarily because of an elevation of maximal a-vO<sub>2</sub> difference. The persistent elevation of VO<sub>2</sub>max in detrained subjects as a result of augmented ability of exercising musculature to extract oxygen may be related to maintenance of increased capillary density

and only partial loss of increased muscle mitochondria derived from training.

### Stroke Volume and Heart Size

Prolonged and intense endurance training promotes increased heart mass, whereas detraining results in decreased heart mass (1, 11, 12). Whether training-induced increases in ventricular volume and myocardial wall thickness regress totally with inactivity is not known. Athletes who become sedentary have enlarged hearts and elevated VO<sub>2</sub>max in contrast to people who have never trained (13).

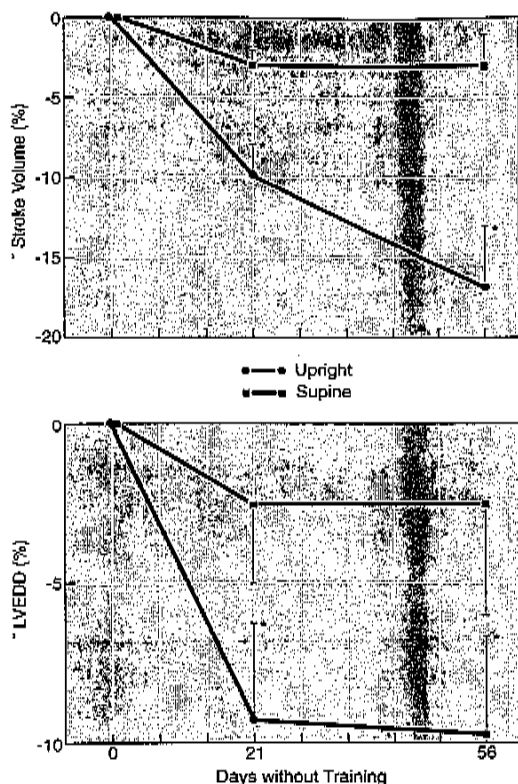
The rapid decline in stroke volume is a striking effect of detraining in endurance-trained individuals. Martin et al. measured stroke volume during exercise with echocardiography in trained subjects, in both upright and supine position, and after 21 and 56 days of inactivity (Fig. 21.2) (14). The decline in stroke volume during upright cycling was associated with parallel reductions in diameter of the left ventricle at end-diastole (LVEDD). When 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 (Fig. 21.2). These observations indicate that ventricular filling is an important factor for stroke volume during exercise and, when it declines, perhaps due to reductions in blood volume, stroke volume also declines. Furthermore, it is also possible that training-induced increases in stroke volume may be partially due to increases in cardiac filling as a result of increases in blood volume.

Endurance-training in rats promoted significant increases in heart mass that were, essentially, completely reversed after 3-7 weeks of detraining (12, 15). Endurance-trained athletes also experience decreased heart mass after 3-8 weeks of detraining associated with a reduced posterior and septal wall thickness of the left ventricle (11, 14). However, it appears that these reductions do not lower stroke volume during submaximal exercise when ventricular filling is high (supine exercise) (14).

Cullinane et al. found that 10 days of detraining did not alter VO<sub>2</sub>max or echocardiographically determined left ventricular mass (16). Furthermore, Pavlik et al. reported that 60 days of detraining resulted in no reduction in left ventricular end-diastolic diameter at rest (17).

### Blood Volume

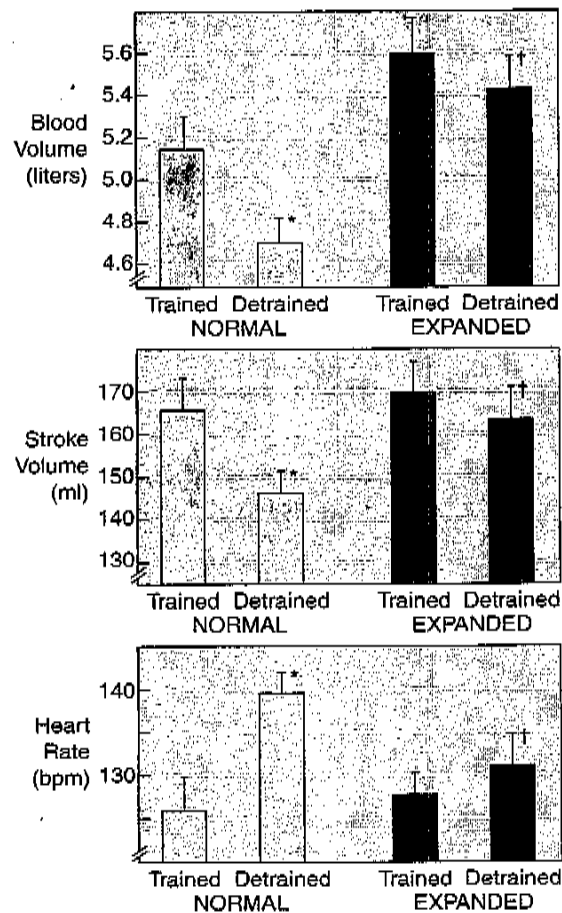
It appears that rapid, detraining-induced reduction of stroke volume during exercise in the upright position is related to decreased blood volume (Fig. 21.3) (18). Intense exercise training usually results in increased blood volume (approximately 500 ml) through expansion of plasma volume (19, 20). This adaptation is gained after



**Figure 21.2.** Percentage decline in exercise stroke volume (A) and left ventricular end diastolic diameter (LVEDD) (B) during exercise in upright and supine postures when trained and after 21 and 56 days of inactivity. \* = responses in upright position are significantly ( $p < 0.05$ ) lower than in supine position and lower than when trained. (With permission from Martin WH, Coyle EF, Bloomfield SA, et al. Effects of physical deconditioning after intense training on left ventricular dimensions and stroke volume. *J Am Coll Cardiol* 7:982-989, 1986.)

only a few bouts of exercise and is quickly reversed when training ceases (18-20). The decline in stroke volume and increased heart rate during submaximal exercise, normally accompanied by several weeks of detraining, can be reversed and returns to near trained levels when blood volume expands to levels similar to trained subjects (Fig. 21.3) (18).

Since stroke volume during exercise is maintained at near trained levels when blood volume is adequate, the filling capacity left ventricle may not be significantly altered by detraining. If ventricular mass declines, myocardial thinning rather than reduction in LVEDD may be responsible (14). Thus, reduction in intrinsic cardiovascular function, at least during submaximal exercise is apparently minimal after several weeks of inactivity in previously intensely trained men (18). The large reduction in stroke volume during exercise in the upright position



**Figure 21.3.** Responses to upright exercise with normal and expanded blood volume when trained and detrained. Significantly different from trained normal (\* =  $p < 0.05$ ). Detrained with expanded blood volume significantly different from detrained with normal blood volume ( $\dagger = p < 0.05$ ). (With permission from Coyle EF, Hemmert MK, Coggan AR. Effects of detraining on cardiovascular responses to exercise. Role of blood volume. *J Appl Physiol* 60:95-99, 1986.)

is largely a result of reduced blood volume and not deterioration of heart function (18).

### Heart Rate During Maximal and Submaximal Exercise

Maximal heart rate increases markedly with detraining, indicating some cardiovascular compensation which may offset a large reduction in blood volume and stroke volume. Coyle et al. observed increased maximal heart rate after 3 and 12 weeks of inactivity (Fig. 21.1) (10). These results generally agree with the findings of others (16, 21). Heart rate also increases markedly during exercise at any given submaximal intensity during detraining.

ing. For example, 12 days of inactivity have been shown to increase heart rate from 158 to 170 beats per minute, and eventually to 184 beats per minute after 84 days of detraining (22).

## DETRAINING AND MUSCLE METABOLISM

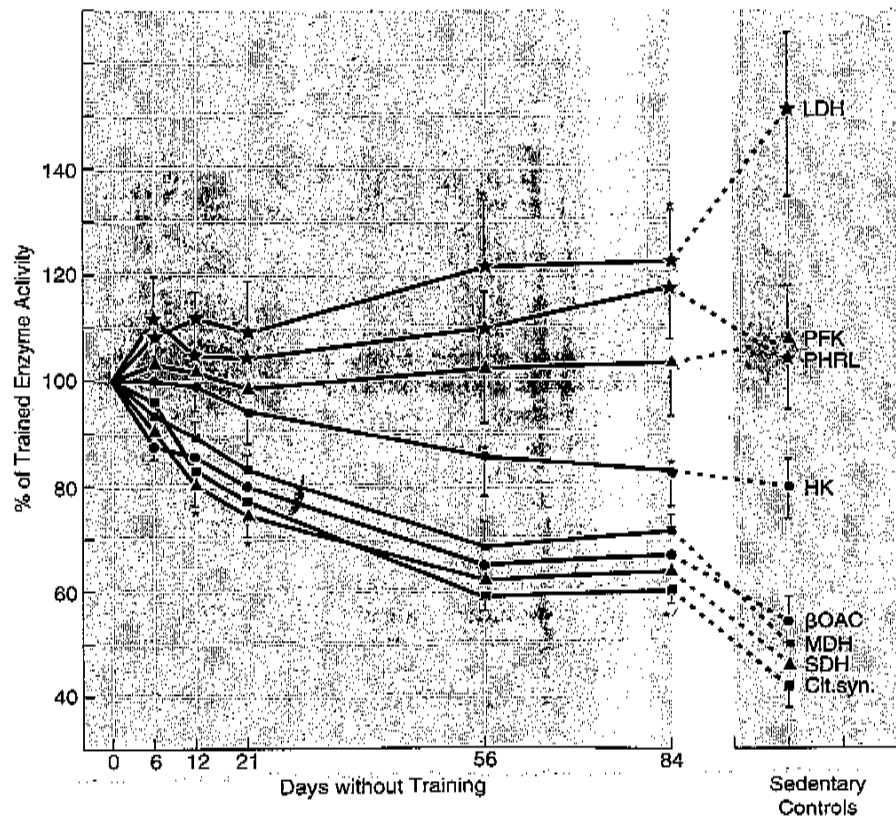
### Enzymes of Energy Metabolism

Endurance exercise training induces enzymatic adaptations in exercising musculature resulting in slower rates of glycogen use and lactate production and improved endurance during submaximal exercise (23). Increased activity of mitochondrial enzymes, resulting in increased ability to metabolize fuels in the presence of oxygen is another important adaptation of endurance training. Moderate endurance training (2–4 months) increases mitochondrial enzyme activity by 20–40% (8, 24). When moderate training ceases and the stimuli for adaptation are removed, increases in mitochondrial activity are quickly and totally reversed. Mitochondrial activity returns to pretraining levels within 28–56 days after cessation of training (8, 10, 25).

Figure 21.4 illustrates a pattern of change in enzyme activity in individuals who trained intensely for 10 years

and ceased for 84 days (26). Mitochondrial enzyme activity in trained subjects (citrate synthase, succinate dehydrogenase, malate dehydrogenase, and  $\beta$ -hydroxyacyl-CoA dehydrogenase), which is initially twofold higher than in untrained persons, declines progressively during the first 56 days of detraining and then stabilizes at levels that are 50% higher than sedentary control subjects. Others have also reported similar declines in mitochondrial activity with detraining (25, 27). Figure 21.4 indicates that the half-life of decline is approximately 12 days (50% decline in 12 days). Therefore, prolonged and intense training, in contrast to training lasting only a few months, appears to result in a partial loss of mitochondrial enzyme activity, thus a persistent elevation of activity above untrained persons (detrained citrate synthase activity in trained individuals is 149% of sedentary controls, see Table 21.1). This elevation occurs almost entirely because of a persistent 80% elevation in the mitochondrial enzyme activity in fast twitch muscle fibers (26). The mechanism for this persistent elevation in mitochondrial activity of fast twitch fibers from detrained endurance athletes is unclear. One possibility is that fast twitch fibers were more readily recruited to contract during daily activities of a sedentary lifestyle in detrained

Figure 21.4. Enzyme activity during detraining compared to sedentary controls. Values expressed as percentage of trained values. Cit. syn., citrate synthase; SDH, succinate dehydrogenase; MDH, malate dehydrogenase;  $\beta$ OAC,  $\beta$ -hydroxyacyl-CoA dehydrogenase; HK, hexokinase; LDH, total lactate dehydrogenase; PHRL, phosphorylase; PFK, phosphofructokinase. \* = significantly different from trained ( $p < 0.05$ ); † = significantly different from 21 days ( $p < 0.01$ ); ‡ = control significantly different from 84 days ( $p < 0.05$ ); †‡ = control significantly different from 84 days ( $p < 0.001$ ). (With permission from Coyle EF, Martin WH, Bloomfield SA, et al. Effects of detraining on responses to submaximal exercise. *J Appl Physiol* 59:853–859, 1985.)



persons. Other explanations including neuromuscular factors or even that these individuals are atypical and predisposed to higher fast twitch mitochondrial activity are also possibilities, although less likely (10, 26).

### Muscle Capillarization

Endurance training promotes increased capillarization of exercising musculature, which theoretically both prolongs transit time of blood flow through muscle and reduces diffusion distance, thus improving availability of oxygen and nutrients to muscle as well as improving removal of metabolic waste. Moderate endurance training lasting several months increases muscle capillarization by 20–30% (8, 28). However, it appears that 8 weeks of detraining can fully or partially reverse increases in capillarization (8, 29). More prolonged and intense training increases muscle capillary density by 40–50% from untrained levels and this high degree of capillarization may be maintained for up to 3 months of detraining (Table 21.1) (10, 28).

### MUSCULAR ADAPTATIONS PERSISTING WITH DETRAINING

Detrained responses in skeletal muscle of highly trained athletes regularly engaged in intense exercise over several years seems to differ from those who have trained a short duration. With cessation of prolonged, intense training, no loss of increased muscle capillarization occurs for at least 3 months although such a loss does occur when moderate training is stopped. Cessation of moderate training results in complete reversal of training-induced increases in mitochondrial enzyme activity, whereas only a partial decline (therefore, a persistent elevation above untrained levels) occurs with cessation of exercise after prolonged, intense endurance training (10, 22, 26).

It is likely that the relatively high  $\dot{V}O_2\text{max}$  observed in detrained athletes is partially due to genetics, hence untrained values are higher than normal, predisposing endurance activities. However, it also seems likely that some persistent muscular adaptation contributes to a relatively high a- $\dot{V}O_2$  difference and maximal oxygen uptake (Table 21.1). It has also been observed that detrained subjects display an ability to exercise at a relatively high percentage of  $\dot{V}O_2\text{max}$  before becoming fatigued or experiencing increased blood lactate (22). The ability to exercise at a higher percentage of  $\dot{V}O_2\text{max}$ , in the detrained state, reflects maintenance of muscular adaptations (high capillary density and mitochondria in fast twitch muscle fibers).

### REDUCED TRAINING RATHER THAN DETRAINING

Detraining implies total cessation of exercise, therefore removal of stimuli for adaptation. Detraining pro-

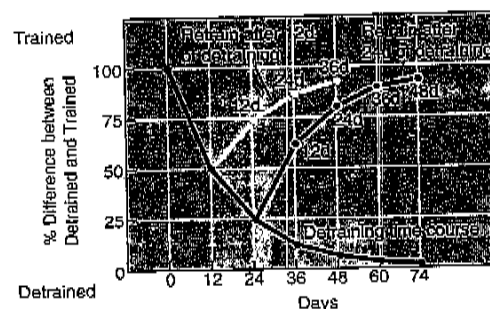


Figure 21.5. Theoretical time course of decline in mitochondrial enzyme activity and endurance performance ability with detraining, as well as rate of increase when training is resumed after 12 days or 24 days of detraining.

duces more marked effects than reduced training, which may maintain cardiovascular and metabolic adaptations more effectively. Indeed, Hickson has demonstrated that  $\dot{V}O_2\text{max}$  and heart size are maintained at trained levels when training frequency is reduced from 6 to 2 days/week, provided that intensity is sufficiently high (85–100%  $\dot{V}O_2\text{max}$ ) (30).

### DETRAINING AND RETRAINING OF MUSCLE MITOCHONDRIA

While it seems logical that detrained subjects, who display only partial loss of adaptations, should be able to retrain to former levels more rapidly, this has never been directly studied in a controlled setting. Detrained former athletes seem to maintain morphological adaptations (heart size and muscle capillarization), which require years to develop, for at least 3 months. Detraining primarily causes reductions in blood volume and mitochondrial enzyme activity and it appears that blood volume is restored with several weeks of retraining (19, 20). Therefore, the limiting factor determining the time course of retraining seems likely to be the rate of increase in mitochondrial enzyme activity. As previously stated, the half-life of decline in mitochondrial enzyme activity is approximately 12 days (Fig. 21.5); therefore, 12 days of detraining requires at least 36 days of re-training to restore previous levels of mitochondrial activity. It appears that a longer period of retraining is required to restore the mitochondrial adaptations lost from a given period of detraining (3:1). Reduced training will attenuate this loss, therefore off season training should be encouraged.

### ► SUMMARY

When physical training ceases (detraining), systems readjust in accordance with diminished physiological

stimuli and many training-induced adaptations are reversed. Available evidence suggests that increases in  $\dot{V}O_2\text{max}$  produced by endurance training of low to moderate intensities and durations are totally reversed after several months of detraining. After several years of intense training, detraining is associated with significant reductions (5–15%) in stroke volume and  $\dot{V}O_2\text{max}$  during the first 12–21 days. These changes do not indicate deterioration of myocardial function, but instead largely result from reduced blood volume and venous return.  $\dot{V}O_2\text{max}$  of endurance athletes continues to decrease during 21–56 days of detraining because of reductions in maximal a- $\dot{V}O_2$  difference. These reductions are associated with a loss of mitochondrial enzyme activity within trained skeletal muscle, which decreases with a half-life of approximately 12 days. Endurance athletes, however, do not regress to levels displayed by individuals who have never trained. Prolonged, intense endurance training is associated with persistent elevation of mitochondrial enzyme activity, skeletal muscle capillarization, maximal a- $\dot{V}O_2$  difference, and  $\dot{V}O_2\text{max}$ .

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