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# Catecholamines, Growth Hormone, Cortisol, Insulin, and Sex Hormones in Anaerobic and Aerobic Exercise*

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**Summary.** Seventeen male physical education students performed three types of treadmill exercise: (1) progressive exercise to exhaustion, (2) prolonged exercise of 50 min duration at the anaerobic threshold of 4 mmol · $l^{-1}$ blood lactate (AE), (3) a single bout of short-term high-intensity exercise at 156% of maximal exercise capacity in the progressive test, leading to exhaustion within 1.5 min (ANE).

Immediately before and after ANE and before, during, and after AE adrenalin, noradrenalin, growth hormone, cortisol, insulin, testosterone, and oestradiol were determined in venous blood, and glucose and lactate were determined in arterialized blood from the earlobe. Adrenalin and noradrenalin increased 15 fold during ANE and 3–4 fold and 6–9 fold respectively during AE. The adrenalin/noradrenalin ratio was 1 : 3 during ANE and 1 : 10 during AE. Cortisol increased by 35% in ANE (12% of which appeared in the postexercise period) and 54% in AE. Insulin increased during ANE but decreased during AE. Testosterone and oestradiol increased by 14% and 16% during ANE and by 22% and 28% during AE. The results point to a markedly higher emotional stress and higher sympatho-adrenal activity in anaerobic exercise. Growth hormone and cortisol appear to be the more affected by intense prolonged exercise. Taking plasma volume changes and changes of metabolic clearance rates into consideration, neither of the exercise tests appeared to affect secretion of testosterone and oestradiol.

**Key words:** Catecholamines – Hormones – Anaerobic exercise – Prolonged exercise

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## Introduction

Intensity and duration are major determinants of the metabolic response to muscular exercise. With exercise up to 2-min duration, the bulk of energy is delivered by glycolysis, as indicated by maximal lactate acidosis after high-intensity short-term exercise (Hermansen and Osnes 1972; Kindermann and Keul 1977). With continuous exercise exceeding 2-min duration, aerobic energy liberation prevails (Kindermann and Keul 1977). As energy metabolism is influenced by various hormones, the hormonal response to predominantly anaerobic or aerobic exercise must be different, in parallel with differences in substrate liberation and turnover.

A number of reports on hormonal responses to prolonged exercise are available and have been reviewed by Galbo et al. (1977). In contrast, reports on the effects of short-term anaerobic exercise on the endocrine system are scarce (Kuoppasalmi et al. 1976; Kuoppasalmi et al. 1980), and comparisons between aerobic and anaerobic exercise are almost completely missing (Kuoppasalmi et al. 1980). It is the purpose of the present study to examine the behaviour of selected hormones regulating energy metabolism during a single bout of standardized anaerobic running exercise, and to compare these effects with those during prolonged aerobic running exercise.

## Material and Methods

Seventeen healthy male physical education students (age 22.7 ± 2.2 years, height 178.8 ± 4.9 cm; weight 70.4 ± 5.5 kg) were examined. Within 1 week every subject performed three types of exercise tests on a motor-driven treadmill. Initially a progressive exercise test was performed. The treadmill was set at a constant slope of 5%, the initial speed was 6 km · $h^{-1}$. Every 3 min it was increased by 2 km · $h^{-1}$ till volitional exhaustion. After each 3 min the treadmill was stopped for 30 s for blood sampling from the earlobe. In addition, capillary blood was taken immediately prior to and then repeatedly till 12 min after the cessation of exercise. Heart rate was determined from the ECG during the last 10 s of each work step. Oxygen uptake was measured continuously with an open system.

At least 3 days after the progressive test a prolonged exercise test (AE) was performed, consisting of steady-state exercise at the anaerobic threshold of 4 mmol · $l^{-1}$ blood lactate for 50 min (Kindermann et al. 1979; Mader et al. 1976). After each 4.5 min the treadmill was stopped for 30 s to allow blood sampling from the earlobe. Prior to the start of the exercise, after 25 min, and immediately after cessation and 6 min later, venous blood was taken from a forearm vein through an indwelling catheter. For assessment of plasma catecholamine levels, additional venous samples were taken after 15 and 35 min of exercise. Heart rate was determined from the ECG during the last 10 s of each 4.5 min bout. Perceived exertion was assessed by means of the Borg scale (Borg and Noble 1974).

One day after AE a short-term anaerobic exercise test (ANE) was carried out. The subjects had to perform an all-out run at a treadmill slope of 5% and a speed of 20 km · $h^{-1}$ (= 156% of the maximum speed in the progressive test). The mean time to exhaustion was 1.5 ± 0.25 min. Capillary blood was taken from the earlobe immediately prior to and immediately after cessation of the run, and then repeatedly till 12 min into the postexercise period. Venous blood was taken immediately before and after the run, and at 6 min in the postexercise period. In both exercise tests catecholamines were not determined in the postexercise period.

The subjects were requested to abstain from food at least 3 h before each test. All tests were performed between 2 and 3 p.m.

Glucose and lactate were determined enzymatically in the arterialized capillary blood. In addition, ph was determined in the capillary blood samples taken in ANE (Corning BGA 168). Adrenalin and noradrenalin were determined by radioenzymeassay in venous plasma (Da Prada and Zürcher 1976), growth hormone (Andler et al. 1976), cortisol (Rolleri et al. 1976), insulin (Biro and Weinges 1973), testosterone (Nieschlag 1975) and oestradiol (Kuss and Goebel 1972) were determined by radioimmunoassay. All hormone assays carried out in duplicate. Changes of plasma volume were estimated from hemoglobin and hematocrit (Dill and Costill 1974).

Data are expressed as means ± standard deviation, differences of means were tested for statistical significance by *t*-test for paired observations.

## Results

Table 1 shows the results of the progressive treadmill test. The treadmill speed at the anaerobic threshold equaled 69% of maximum speed corresponding to 75% of maximal oxygen uptake.

ANE elicited a marked lactate acidosis. Lactate increased to a mean maximum of 16.25 mmol · $l^{-1}$ 6 min after cessation of exercise, pH decreased to a minimum of 7.11. Glucose increased to a maximum of 6.04 mmol · $l^{-1}$ in the 8th min post exercise (Fig. 1). In AE the blood levels of lactate and glucose remained constant till the end of exercise, heart rate increased continuously up to 184.5 beats · $min^{-1}$ (Fig. 1).

In ANE adrenalin and noradrenalin (Fig. 2) increased 15 fold in relation to the respective pre-exercise values. By contrast, in AE the increase was 6–9 fold for noradrenalin and 3–4 fold for adrenalin ($p < 0.001$ for all increases). In AE adrenalin and noradrenalin increased continuously between minutes 15 and 50 ($p < 0.001$). Adrenalin and noradrenalin correlated in ANE ($r = 0.81$; $p < 0.001$) as well as in AE ($r = 0.55$; $p < 0.05$), however the slope of the regression lines was different (Fig. 3):

in ANE: [adrenalin] = 0.146 + 0.303 [noradrenalin]

in AE: [adrenalin] = 1.100 + 0.066 [noradrenalin]

The adrenalin/noradrenalin ratio was about 1 : 3 in ANE and 1 : 10 in AE.

**Table 1.** Results of the progressive treadmill test ($\bar{x} \pm SD$); left: maximal values; right: parameters at the anaerobic threshold at 4 mmol · $l^{-1}$ blood lactate

| Maximal values | | | | |
|---|---|---|---|---|
| $\dot{V}O_2$ (ml · $min^{-1}$) | $\dot{V}O_2$/kg (ml · $min^{-1}$) | Speed (km · $h^{-1}$) | Lactate (mmol · $l^{-1}$) | HR ($min^{-1}$) |
| 3880 ± 432 | 55.1 ± 6.9 | 14.0 ± 1.8 | 12.7 ± 2.7 | 193.2 ± 7.2 |
| 4 mmol · $l^{-1}$ lactate | | | | |
| $\dot{V}O_2$ (ml · $min^{-1}$) | $\dot{V}O_2$/kg (ml · $min^{-1}$) | Speed (km · $h^{-1}$) | HR ($min^{-1}$) | |
| 2929 ± 421 | 41.6 ± 6.7 | 9.7 ± 1.6 | 175.6 ± 7.0 | |

[a] 5% slope

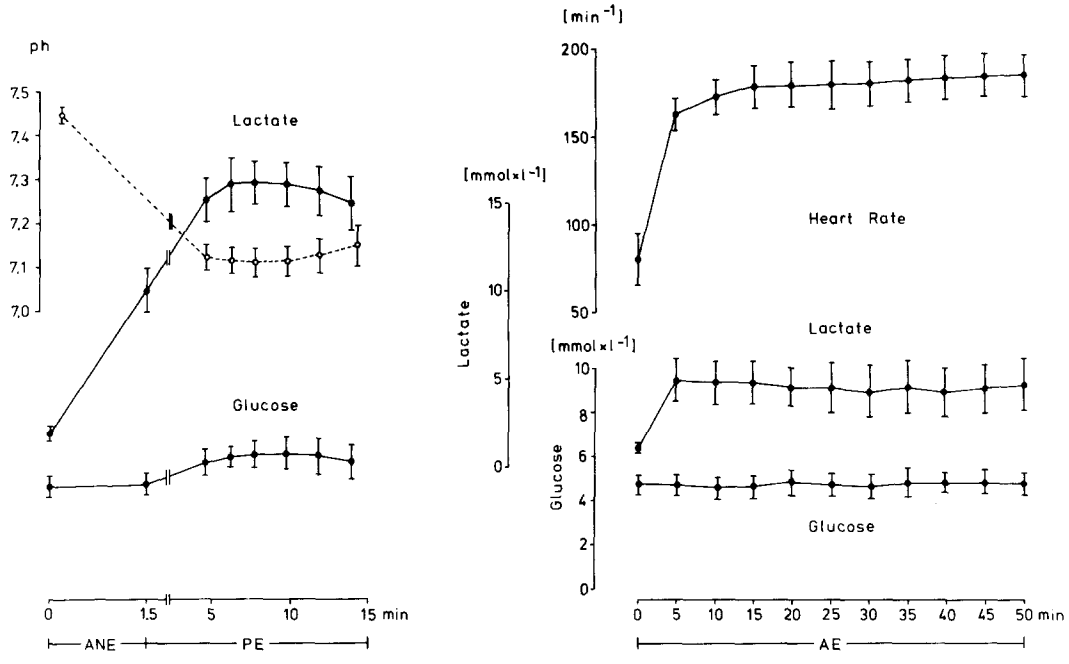


**Fig. 1.** left: ph, lactate, and glucose in ANE; right: heart rate, lactate, and glucose in AE ($\bar{x} \pm SD$)

The growth hormone level doubled in ANE, the increase occurred in the postexercise period and was not statistically significant (Fig. 4). In AE growth hormone increased 14 fold ($p < 0.001$), but only a minor increase occurred in the postexercise period. The difference in growth hormone levels between ANE and AE was statistically significant ($p < 0.002$).

The increase of cortisol in ANE totaled 35% ($p < 0.001$), 12% of which appeared in the postexercise period. In AE the increase was 54% ($p < 0.001$). Statistical significance of the difference in cortisol levels after ANE and AE was not achieved (Fig. 4).

The insulin responses to ANE and AE contrasted diametrically. During ANE insulin increased by 137% ($p < 0.001$), during AE it decreased to 20% of the pre-exercise value by the end of exercise ($p < 0.001$). The increase of insulin concurred with an increasing blood glucose level ($p < 0.001$), either of these exhibited the steepest increase in the postexercise period (Fig. 5). In AE the blood glucose level was unchanged.

Testosterone and oestradiol increased by 14% and 16% respectively in ANE ($p < 0.001$) and 22% and 28% in AE ($p < 0.001$ and $p < 0.01$). ANE and AE did not differ significantly in the increase of these hormones (Fig. 6).

In contrast to ANE, growth hormone and cortisol did not display any additional changes in AE at 6 min post exercise. At this time point the insulin level did not differ significantly from the pre-exercise value. As in ANE, in AE

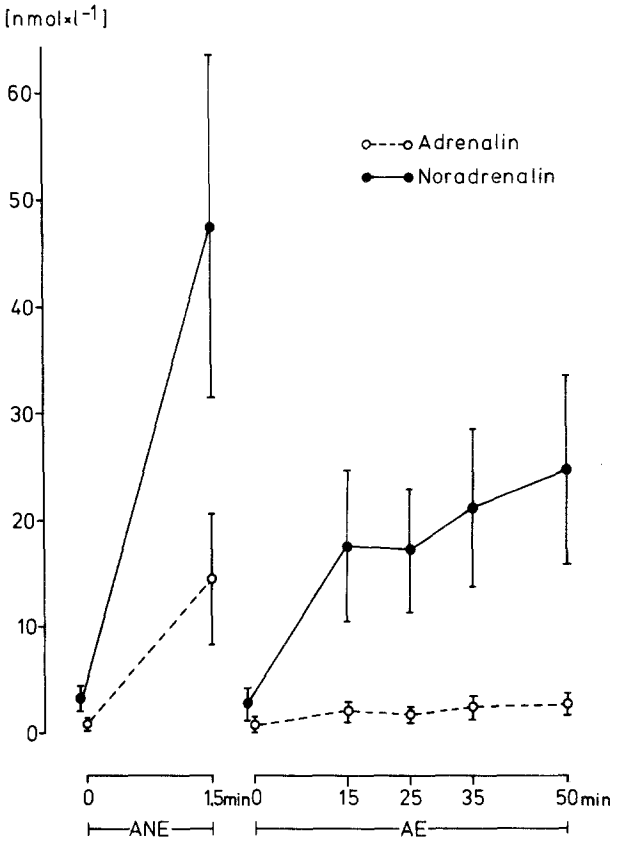


**Fig. 2.** Adrenalin and noradrenalin in ANE and AE ($\bar{x} \pm SD$)

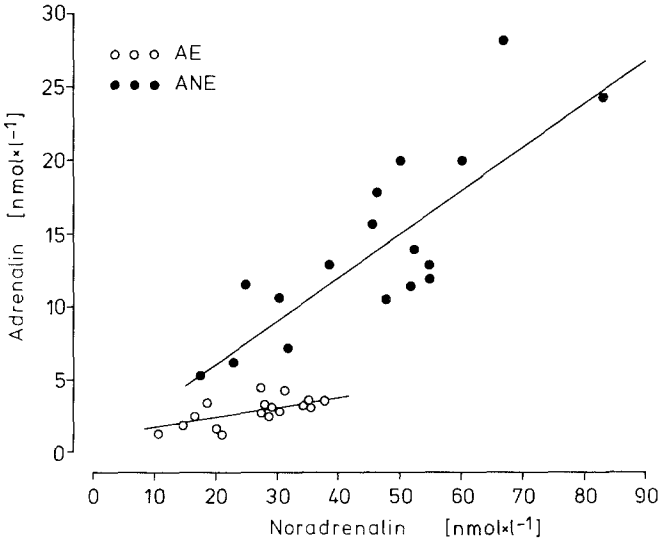


**Fig. 3.** Regression lines between adrenalin and noradrenalin in AE and ANE

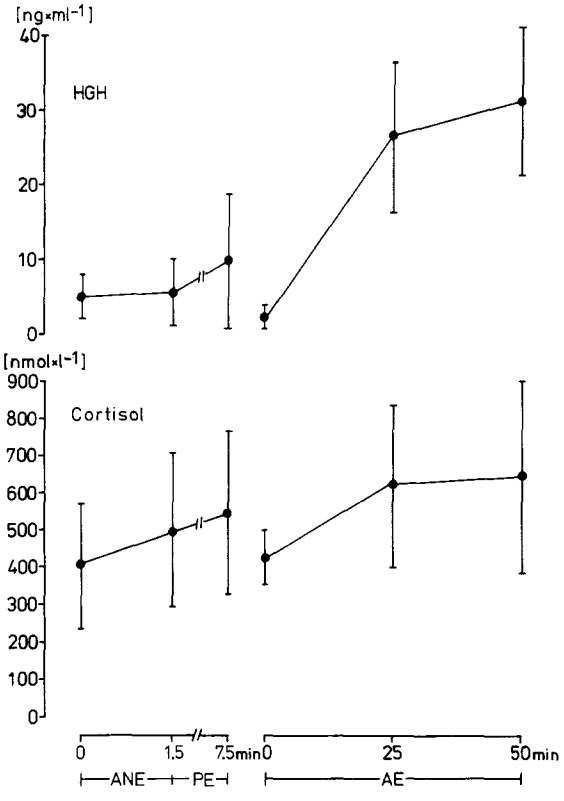


**Fig. 4.** HGH and cortisol in ANE and AE ($\bar{x}$ + SD)

the sex hormones did not show any changes between the end of exercise and 6 min post exercise.

Plasma volume decreased by 9.3% in ANE and 11.5% in AE. The mean Borg scale rating in AE increased from 11.3 at 15 min to 14.9 at 50 min.

## Discussion

The differences in arterial lactate point to the differences in energy liberation during ANE and AE. During AE the arterial lactate attains a constant level, suggesting that energy demand is met primarily by aerobic metabolism, whereas only a minor percentage is supplied anaerobically (Costill 1970; Farell et al. 1979; Kindermann et al. 1979; Mader et al. 1976). The imposed work load, which equals 75% of maximal oxygen uptake, just exceeds the exercise intensity at which lactacid energy release is brought into action (Costill 1970; Farell et al. 1979). In contrast, ANE imposes a supramaximal load demanding predominantly anaerobic energy liberation with marked lactate acidosis. With a mean time to exhaustion of 1.5 min anaerobic energy liberation amounts to approximately 60% of total energy turnover (Åstrand and Rodahl 1977).

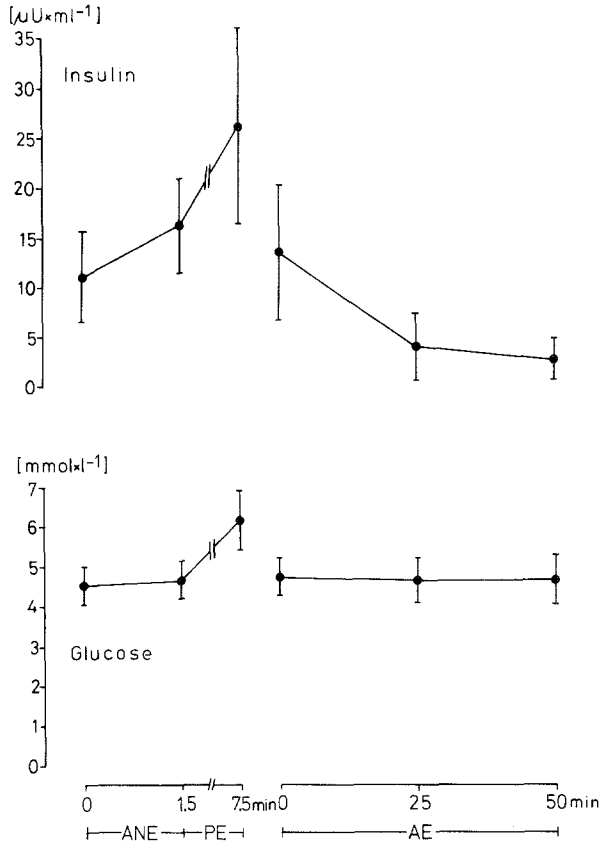


**Fig. 5.** Insulin and glucose in ANE and AE ($\bar{x}$ + SD)

Adrenalin and noradrenalin regulate important cardiocirculatory and metabolic functions. Their blood levels reflect sympatho-adrenal activity (Christensen et al. 1979; Euler and Hellner 1952; Hartley et al. 1972; Lehmann and Keul 1979; Lehmann et al. 1980; Pierce et al. 1976). The markedly higher catecholamine levels in ANE as compared with AE confirm former evidence (Galbo et al. 1977; Lehmann and Keul 1979; Lehmann et al. 1980). The steeper increment in the regression line between adrenalin and noradrenalin in ANE demonstrates that in proportion to noradrenalin, adrenalin increases the most during work of this nature. Since the adrenalin level is supposed to represent primarily emotional stress whereas the noradrenalin level reflects physical stress (Euler and Hellner 1952; Pierce et al. 1976), the emotional stress during ANE appears to exceed that during AE considerably. The high sympatho-adrenal activity appears to be necessary to accomplish the high glycolytic turnover during ANE.

The continuous increase in catecholamines in the course of AE conforms with the findings of other investigators (Christensen et al. 1979; Galbo et al. 1979). Among others, it has to be attributed to circulatory needs resulting from

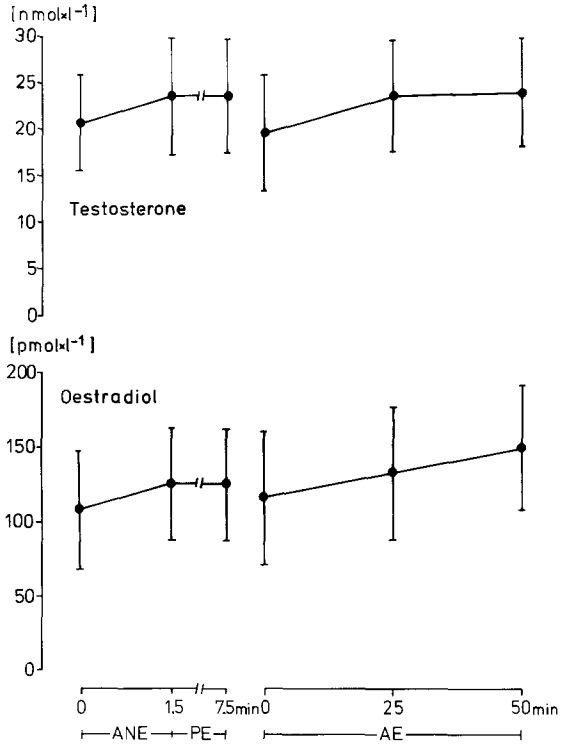


**Fig. 6.** Testosterone and oestradiol in ANE and AE ($\bar{x} \pm SD$)

thermoregulation. The cutaneous vasodilatation with increasing body temperature leads to decreasing venous and right-ventricular filling pressures and subsequently decreasing stroke volume (Rowell 1974). The compensatory increase of heart rate and splanchnic vascular resistance due to increasing noradrenalin are determined to counteract these deteriorative effects and maintain an appropriate cardiac output. The continuous increase of perceived exertion, as indicated by the Borg scale ratings, is similar to the increase of adrenalin level.

Increase of growth hormone during extended exercise is a frequently reported finding (Galbo et al. 1977; Hartley et al. 1972; Karagiorgos et al. 1979; Kindermann and Keul 1977; Sutton et al. 1969; Sutton et al. 1976). A stimulative effect of lactate on growth hormone secretion during exercise has been postulated (Sutton et al. 1969), however, more recent evidence defeats this hypothesis (Karagiorgos et al. 1979; Sutton et al. 1976). In this study the growth hormone increase was markedly less in ANE than in AE depite a four times higher lactate in ANE. Consequently, a single bout of short-term high-intensity exercise seems to elicit only minor increases of growth hormone. Apparently, a high energetic turnover rate with predominantly glycolytic energy release can be accomplished without substantial growth hormone secretion. However, as the

highest value was measured in the last sample drawn it cannot be ruled out that growth hormone continued to rise beyond this time point and the actual maximum evaded detection. During repeated bouts of anaerobic running exercise, growth hormone has been found to increase markedly higher (Kindermann and Keul 1977; Kuoppasalmi et al. 1976). Consequently, an appropriate combination of duration and intensity of exercise seems to be necessary for elevating the growth hormone level in blood. Whether suppression of growth hormone by increasing blood glucose (Fig. 1) becomes operative in this condition is unclear (Galbo et al. 1977).

The significant increase of cortisol by 35% during ANE has to be attributed to increased adreno-cortical secretion. It clearly exceeds the changes of plasma volume and hepatic clearance in magnitude (Rowell 1974). The half-life period of cortisol in blood greatly exceeds the duration of ANE including the 6 min postexercise period (Few 1974). As with growth hormone, the highest cortisol level was found in the last sample at 6 min post exercise, leaving uncertain whether it represented the actual maximum. Cortisol increases of similar magnitude during short-term running tests have been found by other investigators (Kuoppasalmi et al. 1980). During aerobic exercise the cortisol increase has been shown to be related to exercise intensity and duration (Galbo et al. 1977; Hartley et al. 1972; Kuoppasalmi et al. 1976; Scheele et al. 1979). The cortisol responses in this study leave the impression that prolonged exercise at an intensity of 75% of maximal oxygen uptake elicits a higher increase than a single bout of short-term high-intensity exercise.

The insulin level during muscular exercise depends primarily on the catecholamine and glucose levels (Galbo et al. 1977; Galbo et al. 1979). During aerobic exercise with stable or decreasing blood glucose, adrenalin and noradrenalin depress the insulin level (Galbo et al. 1977; Galbo et al. 1979; Hickson et al. 1979; Scheele et al. 1979). Increasing blood glucose overrules the suppressive effect of the catecholamines and leads to increasing insulin levels. Accordingly, the different behaviour of insulin in ANE and AE is due to the different glucose responses. Consequently, duration and intensity of exercise are major determinants of insulin level.

The available evidence on the responses of testosterone and oestradiol to exercise are inconsistent. In connection with aerobic exercise, unchanged testosterone levels have been reported (Kuoppasalmi et al. 1980; Wilkerson et al. 1980). Other reports suggest increased testosterone secretion during exercise of up to 1 h duration (Galbo et al. 1977). Likewise, oestradiol has been demonstrated to increase during prolonged exercise (Schmitt et al. 1981). The evidence on sex hormone responses to anaerobic exercise is limited (Kuoppasalmi et al. 1976; Kuoppasalmi et al. 1980). In this study the percent increases of testosterone and oestradiol exceed only slightly the percent changes in plasma volume. Consequently, secretion of these hormones appears to be essentially unaffected by a single bout of anaerobic exercise. In addition to hemoconcentration, decreased metabolic clearance due to reduced hepatic blood flow has to be considered (Rowell 1974). According to previous evidence the increase in testosterone by 20% during AE in this study could be explained entirely by decreased clearance (Sutton et al. 1976). The significance of the finding that

testosterone secretion is suppressed by cortisol remains to be elucidated (Doerr and Pirke 1976).

In conclusion, this study suggests that a single bout of short-term high-intensity exercise goes along with higher emotional stress and elicits a markedly stronger sympatho-adrenal response than prolonged exercise of 50 min duration. It is conceivable that the risk of over-training during inappropriately tuned anaerobic training is related to these factors. The responses of growth hormone and cortisol to anaerobic exercise appear to be smaller in magnitude than to aerobic exercise, testosterone and oestradiol secretion appear to be essentially unaffected by both types of exercises. The differences in the hormonal responses reflect the differences in substrate supply and turnover.

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