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Sympathoadrenal Response to Resistance Exercise in Men, Women and Pubescent Boys

With Special Reference to Interaction with Other Hormones and Neuromuscular Performance

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Academic Dissertation

Neuromuscular Research Center,
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University of Jyväskylä

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ABSTRACT

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The present study was designed to compare the sympathoadrenal responses (i.e. the increase in plasma catecholamines, noradrenaline and adrenaline) to different acute resistance exercise sessions between adult men, women and pubescent boys. In addition, the behaviour of the plasma catecholamine response in the early phases of a resistance training period was examined. Correlations of the catecholamine response with those of testosterone, cortisol and growth hormone, and with the neuromuscular performance of the subjects were also looked for. In general no differences in the sympathoadrenal activity during the resistance exercises could be found between men and boys. However, as expected the results indicated that in very exhaustive high-intensity resistance exercise the plasma noradrenaline level increase may be lower in pubescent boys than in adult men, suggesting reduced maximal sympathetic nervous activity in the former. The results may also suggest higher plasma adrenaline and cortisol increase in certain resistance exercises in boys than in men, indicating higher stress response in the former. The activation of the sympathetic nervous system and the release of adrenaline from the adrenal medulla in response to resistance exercise seemed to be similar in men and women. The plasma noradrenaline response remained unaltered in the early phase of a resistance-training period. Although the postexercise plasma concentration of adrenaline tended to decline after the first exercise session changes in the peak plasma adrenaline increase from preexercise may also not be expected. No obvious interrelationship between the plasma catecholamine responses and the other hormone responses, or the neuromuscular performance of the subjects was observed. Nevertheless, it is obvious that the plasma catecholamine response is high when the exercise intensity, sets, and recovery periods are organised so that high blood lactate response may also be expected.

Key words: Noradrenaline, adrenaline, testosterone, growth hormone, cortisol, resistance exercise, age, gender
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This thesis is based on the following papers, which will be referred to by their Roman numerals. In addition some data not presented in these papers are also included.


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ABBREVIATIONS AND DEFINITIONS

1-RM: one repetition maximum
A: adrenaline
aEMG<sub>av</sub>: average electromyograph signal amplitude during exercise
aEMG<sub>avg</sub>: average electromyograph signal amplitude in MVC or in 1-RM
B-La: blood lactate
CA: catecholamines
CK: creatine kinase
COR: cortisol
DOMS: delayed onset muscle soreness
ΔPV%: percentage plasma volume change
ΔMVC: before-after change in maximal voluntary contraction force
EMG: electromyography
f<sub>H</sub>: heart rate
GH: growth hormone
GLU: glucose
Hb: haemoglobin
Hct: haematocrit
HPLC: high-performance liquid chromatography
iEMG<sub>tot</sub>: sum of integrated electromyographic activity of examined muscles during exercise
LBM: lean body mass
MVC: isometric maximal voluntary contraction force
NA: noradrenaline
P<sub>mean</sub>: mean power
P<sub>peak</sub>: peak power
REP<sub>max</sub>: maximal number of repetitions
SH: serum hormones
SHBG: sex hormone binding globulin
SNS: sympathetic nervous system
TES: testosterone
TES<sub>free</sub>: free testosterone
TES<sub>tot</sub>: total testosterone
VO<sub>2</sub>: oxygen consumption
VO<sub>2</sub>max: maximal oxygen uptake
W<sub>tot</sub>: total work done
1 INTRODUCTION

The autonomic nervous system is divided into sympathetic and parasympathetic divisions. In general, the SNS is activated under stress to evoke "fight or flight" responses, the concept popularised by Walter Cannon in 1929, which prepare the individual for emergencies. The parasympathetic system produces the "feed or breed" responses. The activation of the SNS and the secretion of A from the adrenal medulla is called the sympathoadrenal activity. Sympathoadrenal activity plays a key role in hormonal, metabolic and circulatory adaptations to exercise. By measuring circulating CA concentrations, NA and A, the functional state of this system has been assessed under a variety of different exercise stimuli, and it is well known that in submaximal dynamic exercise the plasma NA and A responses are dependent both on the duration and intensity of the exercise (Galbo 1983, Kjaer 1989). Less information is available on the factors influencing the magnitude of the plasma CA response to high-intensity exercises. Nevertheless, it seems obvious that the plasma A response in heavy anaerobic exercise is higher than in aerobic endurance exercise (Kindermann et al. 1982, Schwarz & Kindermann 1990). Relatively few studies have examined plasma CA responses to resistance exercise. However, exercise protocol consisting of various resistance exercises with short rest periods (Kraemer et al. 1987), and even one set of maximal number of squats with 70 % of 1RM (Fry et al. 1994) have been shown to produce CA concentrations similar to heavy anaerobic sprint and cycle exercises.

The literature lacks studies on the plasma CA responses to resistance exercise in women and adolescent, despite their increasing participation in this type of physical activity. However, in other types of physical exercise gender differences and differences between adults and adolescent in the plasma CA responses have been observed. In adolescent boys the maximum plasma NA concentration after an exhaustive incremental treadmill exercise has been found to be significantly lower than in men, suggesting reduced sympathetic nervous activity in the former (Lehmann et al. 1981). Also studies where children alone have been investigated have suggested differences in the exercise-induced plasma CA responses between children and adults (Lehman et al. 1980,
Delamarche et al. 1992). There are also some data suggesting gender differences in the plasma A response to maximal exercise. Lower plasma A concentration have been observed in physically active women than in men after repetitive treadmill sprints (Brooks et al. 1990). The plasma A response has also been found to be lower in female sprinters than in male sprinters after a 30s Wingate test (Gratas-Delamarche et al. 1994). Nevertheless, the possible gender differences, and the differences between adults and adolescents, in the plasma CA responses to dynamic resistance exercise are still unknown. In addition, also the majority of the other hormonal research connected to resistance exercise training has been done with adult male subjects, and without simultaneous evaluation of the SNS response.

The general purpose of the present study was to examine the plasma CA responses to different acute resistance exercise sessions, and especially possible gender differences and differences between adults and adolescents. In addition, the behaviour of the plasma catecholamine response in the early phases of a resistance training period was examined. Correlations of the catecholamine response with those of testosterone, cortisol and growth hormone, and with the neuromuscular performance of the subjects were also looked for.
2 REVIEW OF LITERATURE

2.1 Sympathoadrenal response and physical exercise

2.1.1 General aspects

The SNS, like the parasympathetic system, is based on a two-neuron motor pathway (Fig. 1). The cell bodies of the preganglionic neurons are located in the lateral gray horn of the spinal cord. The axons leave the cord via the ventral roots and enter the sympathetic trunk, which is made up of a series of ganglia and axon fibers on each side of the vertebral column. Here they synapse with the postganglionic neurons, which go out and innervate the glands and other structures. The principal neurotransmitter in the preganglionic neurons is acetylcholine and in the postganglionic neurons NA. Some of the preganglionic axons pass to the adrenal gland, the medullary cells of which, the chromaffin cells, are postganglionic neurons that are specialised to secrete the hormone A (Galbo 1983, Gilbey & Spyer 1993, Green 1990). NA is also formed in the adrenal medulla. However, the ratio of NA:A in the adrenal medulla varies between 1:8 and 1:10, while it is about 6:1 in the postganglionic nerve endings (Weicker 1986). The chromaffin cells also secrete enkephalin-containing polypeptides (e.g. proenkephalin peptide F) (Livett et al. 1981, Viveros et al. 1979). The secretory products released from the adrenal medulla leave the adrenal gland through a single large central vein which anastomoses with either the inferior vena cava or renal vein (Coupland 1975). Both central and local factors in the working muscles influence the activation of the SNS during physical exercise (Kjaer 1989, Kjaer et al. 1987, Vissing 1989).

In the postganglionic neurons manufacture of NA begins with the uptake of the amino acid tyrosine into the mitochondria of the cell where it is oxidised to dihydroxyphenylalanine (dopa) (Fig. 2). Dopa is decarboxylated to form dopamine which then enters the storage vesicles. Dopamine acts as a neurotransmitter in some central SNS neurones, but in peripheral
noradrenergic neurones and in adrenal medulla dopamine is converted to NA (Green 1990, Weicker 1986). Some of the NA released from nerve terminals always enters the bloodstream where in high concentrations (> 10 nmol\(^{-1}\)) it may be considered to be acting as a hormone (Clutter et al. 1980, Green 1990). In the adrenal medulla NA leaves the storage vesicles, is methylated to A and then enters the storage vesicle again. A is about ten times more potent as a hormone than NA with levels as low as 0.3 nmol\(^{-1}\) producing physiological effects (Clutter et al. 1980, Green 1990).

![Figure 1: Organisation of the autonomic nervous system (redrawn with modifications from Galbo 1983 and Green 1990)](image)

Both NA and A exert their physiological effects via binding to cell surface receptors, which are collectively known as adrenergic receptors. They have been traditionally divided into \(\alpha\) and \(\beta\) types (Ahlquist 1948), and further into \(\beta_1\), \(\beta_2\) (Lands et al. 1967) and \(\alpha_1\), \(\alpha_2\) subclasses (Berthelsen & Pettinger 1977) based on their affinity for pharmacological agents, but to date several other
subtypes are known to exist (Liggett & Raymond 1993). A and NA are equally potent with regard to the $\alpha_1$- and $\beta_1$-receptors whereas A is more potent than NA with regard to the $\beta_2$-receptors. The $\beta$- and $\alpha_2$-adrenoceptors mediate their effects to the target cell via adenylylate cyclase enzyme and cyclic AMP as a second messenger. The effects of the $\alpha_1$-receptors are mediated by a calcium ($\text{Ca}^{2+}$)-calmodulin complex as a second messenger within the cell. In general, noradrenergic nerves are primarily concerned with cardiovascular responses, and circulating A is primarily concerned with $\beta$-adrenoceptor mediated effects on metabolism. When receptor stimulation is increased, the number of adrenoceptors is reduced. Conversely, when receptor stimulation is reduced, the adrenoceptor density is increased (Green 1990).

During acute physical stress the activation of the SNS and the secretion of A from the adrenal medulla, the *sympathoadrenal* activity, results in well-described circulatory and metabolic effects (Galbo 1983, 1986). Furthermore, the sympathoadrenal response obviously has an important role in the exercise-induced immunomodulation (Pedersen & Hoffman-Goetz 2000).

The influence of the sympathoadrenal response on exercise performance has been studied by using $\beta$-adreceptor blockers. Reductions in prolonged endurance performance observed after $\beta$-blockade may be explained by combined effects of reduced mobilisation of free fatty acids, impaired glycogenolysis and hypoglycaemia. However, in heavy endurance exercise where the energy demand approaches VO$_{2\text{max}}$ the decline in performance is primarily a result of a decreased cardiac output (Tesch 1985). In short-term (≤ 1 min) anaerobic cycling and treadmill exercise decrements in performance have been found after acute (Kaiser et al. 1981, Karlsson et al. 1983, Rusko et al. 1980, Schnabel et al. 1983), but not after chronic $\beta$-blockade (Derman et al. 1993). Both reduced rate of glycogenolysis and reduced O$_2$ supply are potential explanations for the performance decline in this type of exercise (Tesch 1985). Declined glycogenolytic energy supply could also explain a decrease in

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**FIGURE 2** Schematic illustration of the CA release to the circulation from sympathetic nerve endings and adrenal medulla
sustained isometric contraction performance (Tesch et al. 1984, Tesch 1985), which, however, has not been observed in other studies (Grimby & Smith 1978, Rusko et al. 1980). Maximal force production does not seem to be affected by β-blockade (Grimby & Smith 1978, Rusko et al. 1980, Yorko et al. 1990).

2.1.2 Assessment of sympathoadrenal response by plasma catecholamines

Most of the NA released from the nerve terminals is taken back up again by the nerve (Fig. 2) (Melmon 1981), but the neuronal removal of A is not as efficient as that of NA (Eisenhofer et al. 1990). Some of the circulating CA are also degraded by one of two enzymes, catechol-o-methyl transferase (COMT) and monoamine oxidase (MAO) (Green 1990). The circulating free CA are also conjugated to sulphates, which may be seen as one form of deactivation (Cleroux et al. 1985). Plasma half-lives of CA sulphates are about 3-4 h (Kuchel & Buu 1984), but those of the free plasma CA only about 2-3 min (Hagberg et al. 1979, Peronnet et al. 1988). Only a few percentage of circulating CA is eventually excreted in the urine (Green 1990).

Measurement of the free plasma NA and A concentrations is the most common and practical method used in evaluating the sympathoadrenal response during physical exercise (Galbo 1983, Green 1990). Although both A clearance (Kjaer et al. 1985) and NA clearance (Leuenberger et al. 1993) may decrease during exercise, the important point is that the small changes in clearance cannot explain the dramatic increases in NA and A usually seen during exercise. It is therefore clear that the changes in free plasma CA concentration do reflect the secretion of the CA. During exercise the main source of the circulating NA are the sympathetic nerve endings in the exercising muscles while A comes mainly from the adrenal medulla (Kjaer et al. 1993, Savard et al. 1987). The CA increase is also proportional to the fraction of the total muscle mass recruited during exercise (Jensen-Urstad et al. 1994, Savard et al. 1989). It is, however, obvious that conventional blood sampling from antecubital vein may introduce some problems with regard to the use of the circulating CA concentrations in the evaluation of the sympathoadrenal response to exercise (Hjemdahl 1993). In this method the measured plasma NA concentration is known to be disproportionately influenced by forearm sympathetic nerve activity, and is thus not a generalisable marker for overall sympathetic activity. The A secretion, on the other hand, may be underestimated due to forearm tissue extraction of A (Hjemdahl 1993). One possibility to get closer to the arterial CA concentrations is the use of arterialised back hand venous blood by local heating (Kjaer et al. 1985, Kjaer 1989). Radioenzymatic and especially high performance liquid chromatography (HPLC) assays are sensitive enough to detect the small amounts (in nmolL⁻¹) of free plasma CA in plasma (Green 1990, Weicker 1986).

Direct neural activity recordings from superficial sympathetic nerves in inactive muscle, tissue turnover of NA, urinary production of CA and different pharmacological and surgical methods have also been used in the assessment of sympathoadrenal activity (Green 1990). It has also been suggested that measurement of NA sulphate level after exercise could be an appropriate
estimate of the overall activation of the SNS both during prolonged exercise (Ratge et al. 1986, Sagnol et al. 1990) and during repeated bouts of short-term exercise (Strobel et al. 1999).

**2.2 Plasma catecholamine response to acute exercise and training**

It is well known that in submaximal dynamic exercise the plasma NA and A responses are dependent both on the duration and intensity of the exercise (Fig. 3). Less information is available on the factors influencing the magnitude of their response to short-term maximal exercises. Nevertheless, it seems that especially the plasma A response to heavy anaerobic exercise is higher than in aerobic endurance exercise (Kindermann et al. 1982, Schwarz & Kindermann 1990). It is also obvious that the anaerobic contribution to energy supply is closely related to the magnitude of plasma CA response in heavy short-term exercise. In different maximal treadmill and ergometer sprint exercises the plasma CA response has been shown to correlate to the postexercise blood lactate concentration (Brooks et al. 1990, Cheetham et al. 1986), to the plasma lactate increment (Strobel et al. 1999), to the postexercise muscle lactate concentration (Cheetham et al. 1986), to the estimated energy production from anaerobic sources (Cheetham et al. 1986, Nevill et al. 1989), to the postexercise blood pH (Cheetham et al. 1986), to the accumulated oxygen deficit (Strobel et al. 1999) and to the peak power output (Nevill et al. 1989). It has also been shown that in maximal repetitive anaerobic exercise the plasma A level may peak already during the exercise rather than after it (Brooks et al. 1990).

**FIGURE 3** Arterial plasma CA concentrations in relation to exercise intensity and duration (redrawn with modifications from Kjaer 1989)

At a given absolute oxygen uptake the A response is known to be lower in endurance trained individuals than in untrained subjects (Kjaer 1989, Kjaer et al. 1985). This effect of training on the plasma A response occurs in the first few
weeks of training during which VO$_2$max increases markedly (Winder et al. 1978). However, at identical relative exercise intensities, as well as at maximal intensity exercise, the plasma A concentration is higher in endurance trained individuals than in sedentary subjects, while the plasma NA levels are similar (Kjaer et al. 1985, Kjaer 1989). It has been speculated that the higher maximal exercise A response in endurance trained as compared to untrained individuals might be due to simultaneous reductions in peptide F and/or other proenkephalin polypeptide secretion rates (Kraemer et al. 1985). In animal studies also increased volume of the adrenal gland, as well as the adrenal content of A, has been demonstrated after endurance training (Stallknecht et al. 1990). Since A has several advantageous effects in relation to exercise performance, an increased capacity to secrete A may be considered beneficial in physical exercise. It has actually been suggested that exercise training may lead to the development of a sports adrenal medulla, an increased capacity of the adrenal medulla to secrete A (Kjaer & Galbo 1988). However, it is obvious that several years of training is required to advance this adaptation (Kjaer 1998). Recently, in line of this type of training adaptation the A response to supramaximal exercise was also demonstrated to be higher in sprinters than in untrained individuals (Zouhal et al. 1998). Furthermore, both the plasma A and NA responses to short-term supramaximal treadmill exercise were found to be higher in anaerobically trained athletes than in aerobically trained athletes (Strobel et al. 1999).

2.2.1 Special features of resistance exercise

Plasma CA concentration response to acute resistance exercise in adult men has been measured in numerous studies (e.g. Bush et al. 1999, Fry et al. 1994, Guezennec et al. 1986, Horstmann et al. 1994, Kraemer et al. 1987, Kraemer et al. 1999, Niemann et al. 1994, Stock et al. 1995). The variation in the magnitude of the plasma CA increase between these studies is large. An exercise protocol consisting of various resistance exercises with 10 RM sets and with short rest periods of 10 - 60 s (Kraemer et al. 1987), and even one set of maximal number of squats with 70 % of 1RM (Fry et al. 1994) has been shown to produce very high venous plasma CA concentrations similar to heavy anaerobic sprint and cycle exercises. On the other hand, only moderate venous plasma concentration increases of CA have been observed after a leg squat exercise performed until exhaustion with sets of 10 repetitions, with a cadence of one repetition every 6 seconds and with 3 min rest between the sets (Niemann et al. 1994), as well as after a leg press exercise with maximal number of repetitions with 80 % of 1RM (Kraemer et al. 1999). In addition to variations in the exercise stresses and blood sampling times, postural factors may have contributed to these different plasma CA responses, since the response is known to be higher in upright than in supine position (Galbo 1986). However, in general the plasma CA response to resistance exercise has been suggested to be primarily related to the force of muscular contraction, the amount of muscle tissue stimulated and the amount of rest between sets and repetitions (Kraemer 1988). It was also recently suggested that total work done is a key factor in influencing the
sympathoadrenal response to heavy resistance exercise stress, as small
differences in power output and force production did not affect the plasma CA
response to resistance exercise when total work was kept constant (Bush et al.
1999). In addition, changes in blood flow in exercising muscles may also
influence the plasma CA response to resistance exercise. The plasma NA
response has been shown to be higher in dynamic than in isometric knee
extension exercise although the active muscle mass and time to exhaustion was
similar in the two modes of exercise. However, no differences were observed in
the plasma A responses between the two types of exercise (Lewis et al. 1985).
The reason for a lower NA:A ratio during static compared to dynamic
contractions may be a lower blood flow in the exercising muscles in the former
than in the latter (Kjaer & Secher 1992).

Regular strength training of several months has been reported to reduce
plasma A response both to sub maximal (i.e. 6 sets of 8 reps at 70 % load of 1
RM) and maximal (one set to exhaustion at the same workload) bench pressing,
while causing no alterations in those of NA (Guezennec et al. 1986).
Exceptionally low plasma A levels were also found in weight-lifters after
exhaustive incremental cycling, as compared to endurance-trained subjects and
control subjects (Lehmann & Keul 1986). These authors also suggested that the
low A responses in the weight lifters might be associated with a reduced
exercise induced strain due to the increased muscle power. Thus, these data
might suggest that the adrenal medullary adaptation to resistance training may
be different from that to endurance and sprint exercise training. However, on
the contrary to this possibility, both the plasma NA and A levels after an
exhausting leg press exercise were recently found to be higher in powerlifters
than in untrained men (Kraemer et al. 1999). The plasma CA response has also
been reported to be similar in well-trained weightlifters and untrained men
(McMillan et al. 1993).

Very little is known about the plasma CA responses to repeated resistance
exercise sessions in previously untrained subjects. However, it is known that
during the very first resistance training sessions strength gain is fast and
maximal force increases mainly due to enhanced motor unit activation, as
indicated by increased electromyographic activity (Häkkinen 1994, Komi 1986,
Sale 1988). Unaccustomed exercise, especially if it contains eccentric muscle
contractions, is also known to result in disruption of contractile tissue (Friden &
Lieber 1992) and acute inflammatory response which in turn is associated with
the development of DOMS (MacIntyre et al. 1996, Smith 1991). Both an increase
in maximal force (Lehmann & Keul 1986) and muscle damage could potentially
influence the plasma CA response in the early phases of a resistance training
period. The secretion of the CA is known to be stimulated by cytokines released
from the injured tissue (Madden & Felten 1995), and it has been shown earlier
that already one training session may provide a protective effect for further
muscle damage, even if the second exercise is performed before full recovery
and restoration of muscle function (Ebbeling & Clarkson 1990).
2.2.2 Gender differences in the plasma catecholamine response to exercise

In prolonged endurance exercise, with identical relative intensity (% VO$_2$max) in men and women, no gender difference in the plasma CA response has usually been observed (Favier et al. 1983, Friedmann & Kindermann 1989), although a lower CA response in women than in men has also been reported (McMurray et al. 1987). In addition, in one study the NA levels were similar in men and women throughout a 90 min treadmill exercise, but the A level was significantly lower in women than in men at the end of the exercise, as well as also 15 min after the exercise (Tarnopolsky et al. 1990). After incremental exercise until exhaustion the plasma levels of CA in women have been reported to be both lower (McMurray et al. 1987) and higher than in men (Lehmann et al. 1986). All these contradictory results may be explained by differences in exercise duration and, at least in part, by different training status of the subjects in these studies. It has been shown in animal studies that the adrenomedullary adaptation to endurance training is more pronounced in males than in females (Stallknecht et al. 1990). In addition, it has been shown that the plasma A increase may be higher in women in the follicular phase rather than in the luteal phase of the menstrual cycle (Sutton et al. 1980). However, in another study the A response tended to be higher in the luteal than in the follicular phase, although not significantly so (Lavoie et al. 1987).

Only a few studies have compared the plasma CA responses to short-term anaerobic exercises between men and women. However, lower plasma A levels have been observed in physically active women than in men after maximal repetitive treadmill sprints, although the NA levels were identical (Brooks et al. 1990). The plasma A response was also found to be lower in trained female sprinters than in male sprinters after a 30s Wingate test, while again no gender differences were observed in the NA levels (Gratas-Delamarche et al. 1994). Following a 5-min isometric exercise the plasma A levels were similar in men and women, although after the 1st min significantly higher A levels could be observed in men (Sanchez et al. 1980).

2.2.3 Influence of age on the plasma catecholamine response to exercise

In general older adult individuals show a higher plasma NA response to any relative intensity of work than younger ones (Fleg et al. 1985, Lehmann & Keul 1986a, Mazzeo et al. 1997, Zouhal et al. 1999). It has been suggested that this exaggerated plasma NA increase results from enhanced SNS activity in an attempt to compensate for reduced target organ responsiveness to adrenergic stimulation (Lehmann & Keul 1986a, Mazzeo & Grantham 1989). However, it is obvious that a reduction in NA clearance also explains, in part, the higher NA response in the older individuals (Mazzeo et al. 1997). The exercise induced A response at identical relative intensity has either shown no age dependence (Mazzeo et al. 1997), or it has been higher (Fleg et al. 1985) or lower in older individuals than in the younger ones (Lehmann & Keul 1986a).

In one study the plasma CA response was compared between adult men and adolescent boys (12.8 ± 0.8 years) in an exhausting incremental treadmill
exercise (Lehmann et al. 1981). The plasma CA levels were apparently very similar in the two groups of subjects at identical submaximal relative exercise loads (% VO₂max). However, the plasma NA level after exhaustion was significantly lower in boys (30%) than in men, while no differences in the A levels were observed between the groups. These authors suggested a reduced maximal sympathetic nervous activity in the adolescent as compared to the adults (Lehmann et al. 1981). Studies where children alone have been investigated have also suggested differences in the exercise-induced plasma CA responses between children and adults. Lehmann and coworkers (1980) suggested that despite a lower NA response the plasma A response to maximal short-term exercise (300 m run) is higher in adolescent boys (12.6 ± 1 years) than in adult men. It has also been suggested that during a 60 min cycle exercise (60% VO₂max) the A response in relation to the NA response is higher in prepubertal boys (8.5 - 11 years) than in adults (Delamarche et al. 1992). In this study the plasma NA level was observed to peak already after 15 min of exercise and to decline then slowly during the rest of the exercise, a pattern clearly different from that of observed in the adults. However, the plasma A level increased throughout the exercise. It was suggested that the early peak in NA was related to a small blood glucose decline in the beginning of the exercise, i.e. to prevention of hypoglycaemia (Delamarche et al. 1992). The same authors also later confirmed this pattern in boys of same age, but not in girls who also tended to have lower NA levels during the exercise than the boys. It was suggested that a higher maturation level in the girls than in boys might have accounted for the gender difference (Delamarche et al. 1994). However, after a Harward step test the plasma NA response was found to be higher in 14 year old girls than in boys of same age (Gerra et al. 1994).

2.2.4 Plasma catecholamine response in relation to serum testosterone, cortisol and growth hormone

In general, the exercise-induced increases in TES have been attributed to three basic mechanisms: decreased plasma volume (Kinderman et al. 1982, Metivier et al. 1980), reduced clearance rate resulting from decreased hepatic blood flow, especially in long duration submaximal exercises (Cadoux-Hudson et al. 1985, Kinderman et al. 1982) and increased gonadal secretion (Cumming et al. 1986, Metivier et al. 1980). Sympathetic stimulation, together with plasma NA and A, may increase TES secretion in males (Elk-Nes 1969, Jezova & Vigas 1981), and positive correlations between submaximal endurance exercise-induced CA and TES responses have indeed been reported (Fahrner & Hackney 1998, Jezova & Vigas 1981, Jezowa et al. 1985). However, an inhibitory role for CA on the TES production has also been suggested (Levin et al. 1967, Wheeler et al. 1994).

Sympathoadrenal activity may stimulate COR release from the adrenal cortex (Al-Damluji 1993, Berne & Levy 1993, Chouros 1995, Kuoppasalmi et al. 1981). However, there are also data showing that physiologic elevations of circulating CA have no effect on the pituitary-controlled COR release, obviously due to their inability to cross the blood/brain-barrier (Oberbeck et al. 1996). The GH secretion from the anterior pituitary gland may also be
stimulated by sympathoadrenal activity (Al-Damluji 1993). Indeed, high sympathetic activity and plasma CA levels and elevated GH response to exercise have been linked by several investigators (e.g. Chwalbinska-Moneta et al. 1996, Kjaer et al. 1987, Kozlowski et al. 1983, Weltman et al. 1997). However, the precise relationship between peripheral plasma CA levels and the activity of central adrenergic neurons is unknown (Pritzlaff et al. 1999).

In adult men TES, COR and GH, which obviously play an important role in the muscular adaptation to exercise (Florini 1985, Kraemer 1988), have been repeatedly demonstrated to show increased serum concentrations in response to resistance exercise (e.g. Häkkinen & Pakarinen 1993, Häkkinen et al. 1988a, Kraemer et al. 1991). However, in women normally no resistance exercise-induced increase in serum TES level (Fahey et al. 1976, Kraemer et al. 1991, Kraemer et al. 1993, Weiss et al. 1983), or only a slight increase in some individuals is observed (Cumming et al. 1987, Häkkinen & Pakarinen 1995). Also the GH and COR concentration responses may be lower as compared to men (Häkkinen & Pakarinen 1995).

In weight trained adolescent boys (around 17 years of age) a resistance exercise induced increase in serum TES level has been shown in few studies (Fry et al. 1993, Kraemer et al. 1992). In another study no TES increase in response to resistance exercise could be observed in relatively untrained boys (16 ± 0.8 years) (Fahey et al. 1976). In addition to differences in exercise protocols also the weight training background may account for these differences between these studies. A resistance exercise-induced increase in serum TES level was only found in elite junior weight lifters with more than two years of training experience (Kraemer et al. 1992). Also the serum COR and GH levels may increase in response to resistance exercise in adolescent boys (Fry et al. 1993, Kraemer et al. 1992), but no comparisons on the magnitude of these hormonal responses to resistance between adolescent and adults have been reported.

The parallelism of the plasma CA response to resistance exercise and that of serum TES, COR or GH have not been studied neither in adults nor in adolescents.
3 PURPOSE OF THE STUDY

The sympathoadrenal responses to different types of aerobic and anaerobic exercises are well documented. However, relatively few studies have examined the sympathoadrenal responses to resistance exercise. Furthermore, the literature almost completely lacks studies on the sympathoadrenal responses to resistance exercise in women and adolescent, despite their increasing participation in this type of physical activity. Therefore the possible gender differences, and the differences between adults and adolescents, in the sympathoadrenal responses to resistance exercise are still unknown. In addition, the majority of the other hormonal research connected to resistance exercise training has been done with adult male subjects, and without simultaneous evaluation of the sympathoadrenal response. The general purpose of the present study was to examine the sympathoadrenal responses to different acute resistance exercise sessions by using plasma CA measurements. Especially possible gender differences and differences between adults and adolescents were looked for. The hypotheses and aims of the present study were as follows:

1) Based on the existing literature both age and sex may influence the sympathoadrenal response to physical exercise. It was hypothesised in the present study that the plasma A increase in response to exhausting resistance exercise in women, and that of the plasma NA in pubescent boys might be lower than in adult men. Therefore the first aim of the project was to investigate the possible differences in the plasma CA concentration responses to different acute resistance exercise sessions:

   A. between adult men and women (II, IV)
   B. between adult men and pubescent boys (I, IV),

2) Exercise training decreases the sympathoadrenal response to constant exercise stimulus. It was hypothesised that a decrease in the plasma A
response may already be found in the early phases of a resistance-training period where strength gain is fast due to neural adaptation (III). It was further hypothesised that the plasma CA response may be higher in unaccustomed resistance exercise leading to muscle damage than in identical exercise performed soon after the first exercise (V). The second aim of the present investigations was therefore to examine the plasma CA concentration responses in the early phases of a resistance-training period, where strength gain is fast due to neural adaptation, and muscle damage/soreness is common (III, V).

3) Several hormonal responses may be potentially influenced by sympathoadrenergic regulation, and differences between men and women, as well as between men and adolescent boys may exist in the magnitude of these responses. The sympathoadrenal response is also known to be influenced both by different peripheral factors as well as by central command, and it may have effects on performance by several different ways. It was hypothesised that correlations between the plasma CA and other hormones, as well as between CA and neuromuscular performance would exist. Therefore, the third aim of the study was to examine whether the sympathoadrenal response to resistance is interrelated:

   A. with the responses of serum TES, GH and COR (I, IV, V)
   B. with the performance of the subjects, with the activity level of exercising muscles or with the indicators of muscle fatigue (I, II, III, IV, V).
4 RESEARCH METHODS

4.1 Subjects

A total of 30 men, 14 women and 13 adolescent boys volunteered to participate in these studies. Table 1 summarises the physical characteristics of the subjects in each study. In Experiment 1 the subjects were Finnish national level track and field athletes (sprinters, jumper and decathletes), but in all the other experiments relatively untrained or physically active men, women and boys. The subjects were fully informed about the possible risks associated with the experiments and their written consent was obtained. In addition, written consent was obtained from the parents of the adolescent subjects. These studies were approved by the University Ethics Committee.

<table>
<thead>
<tr>
<th>Exp.</th>
<th>Men (n = 7)</th>
<th>Boys (n = 7)</th>
<th>Mean age (years)</th>
<th>Mean stature (cm)</th>
<th>Body mass (kg)</th>
<th>Body fat (%)</th>
<th>Original paper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exp. 1</td>
<td>25</td>
<td>15</td>
<td>25</td>
<td>184</td>
<td>77.4</td>
<td>9.4</td>
<td>I</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>1</td>
<td></td>
<td>9</td>
<td>4.5</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>77.4</td>
<td>4.5</td>
<td>0.9</td>
<td></td>
</tr>
<tr>
<td>Exp. 2</td>
<td>29</td>
<td>15</td>
<td>29</td>
<td>178</td>
<td>64.8</td>
<td>9.1</td>
<td>II</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1</td>
<td></td>
<td>5</td>
<td>7.8</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>64.8</td>
<td>7.8</td>
<td>1.9</td>
<td></td>
</tr>
<tr>
<td>Exp. 3</td>
<td>27</td>
<td>27</td>
<td>27</td>
<td>171</td>
<td>64.2</td>
<td>25.3</td>
<td>III</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>4</td>
<td></td>
<td>4</td>
<td>5.5</td>
<td>4.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>64.2</td>
<td>5.5</td>
<td>4.4</td>
<td></td>
</tr>
<tr>
<td>Exp. 4</td>
<td>26</td>
<td>27</td>
<td>26</td>
<td>181</td>
<td>74.4</td>
<td>12.8</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>3</td>
<td></td>
<td>6</td>
<td>10.1</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>74.4</td>
<td>10.1</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>Exp. 5</td>
<td>28</td>
<td>14</td>
<td>28</td>
<td>168</td>
<td>65.7</td>
<td>25.1</td>
<td>V</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0</td>
<td></td>
<td>5</td>
<td>7.7</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>65.7</td>
<td>7.7</td>
<td>4.0</td>
<td></td>
</tr>
</tbody>
</table>

TABLE 1 Physical characteristics of the subjects.
4.2 Experimental design, testing procedures and analysis

4.2.1 Approach to the problems and experimental design

Five separate experiments were performed in this study. The first experiment consisted of a comparison of the plasma CA and serum TES, COR and GH responses to four different half-squatting exercises in adult and adolescent male athletes (I). Although all the subjects were familiar with half-squatting, due to the age of the younger subjects the exercise load was kept relatively low (< 50 % of 1-RM). However, the exercise units were planned to induce different metabolic demands by altering the recovery periods between the sets, the squatting frequency, exercise load and repetition number. The W_set was kept equal in every exercise. Correlations of the plasma CA to other hormones and different indicators of neuromuscular performance were calculated individually for each exercise session and subject group, but also by combining the two groups of subjects and the four exercise sessions.

In all the rest of the experiments dynamic bilateral knee extension with a variable-resistance knee extension machine (David 200, David International, Vantaa, Finland) was used as an exercise model. This exercise machine has been shown to cause exhaustion with significantly less repetitions at a given load level than a normal constant-resistance knee-extension device (Häkkinen et al. 1988). The subjects were all relatively unfamiliar with the exercise machine.

In the second experiment the plasma CA responses to four exercise tests at different load levels, performed until exhaustion, were compared between men and women (II). Correlations of the plasma CA to different indicators of neuromuscular performance were calculated also by combining the two groups of subjects.

The third experiment investigated the plasma CA responses during five successive identical exercise sessions in men (III). Exercise load and recovery period were chosen so that 12 repetitions could be performed in each set of repetitions. This length of a set was considered appropriate, as it is probably close to that commonly used in recreational resistance training and also long enough to induce a marked metabolic response. Before the exercises the MVC and 1-RM were measured, and correlations between plasma CA response changes during the training period and those of the MVC, 1-RM and other indicators of neuromuscular performance were calculated.

The fourth experiment compared the plasma CA responses to an exhausting exercise between men, women and boys together with the serum TES, GH and COR responses (IV). Due to the age of the younger subjects the exercise load was kept relatively low (40 % of 1-RM), but the last two sets in the exercise were continued until exhaustion. Correlations of the plasma CA to other hormones and to different indicators of neuromuscular performance were calculated also by combining the subjects groups.
Finally, the fifth experiment examined the effects of an unaccustomed exercise session on the plasma CA, serum TES, GH and COR responses in the second exercise (V). Assuming that the exercise loading would not have to be significantly declined in the second exercise, and thus being able to keep the exercise stimulus (total work performed and exercise duration) relatively unchanged, the same exercise protocol was used as in the previous experiment (IV) to examine the hormonal responses to a resistance exercise performed soon after an unaccustomed exercise bout. The second exercise was timed to the second recovery day, where the DOMS was expected to peak. If decreases in the CA (and/or other stress hormone) response were found, this might further strengthen the role of a muscle damage protective effect of the first exercise in explaining the decline, as it might be speculated that exercise stress may even increase when performed with sore muscles. Correlations of the plasma CA to other hormones were also calculated by combining the two exercise sessions.

The exercise protocols, different types of indicators of neuromuscular performance and other measurements used in the present experiments are presented in detail in the following paragraphs. All the present experiments were started with a routine warm-up of about 20 min, consisting of jogging (I, II) or ergometer cycling (III, IV, V), stretching and familiarisation to the exercise machine with moderate weights (I-V).

4.2.2 Half-squatting exercises (I)

In experiment 1 the subjects performed four different half-squatting exercise units (Table 2). The exercise units were planned so that the W_{tot} was equal in every exercise. All the exercise units were done in a squatting apparatus in which the barbell was mounted with ball bearings on two vertical bars. This apparatus allowed maximal squatting frequency with no difficulties in keeping the balance. The range of the movement was controlled with an adjustable flexible metal bar touching the hamstring muscles at the lowest position of the squat (knee angle 90\degree). The exercise units E1, E2 and E3 were performed with maximal squatting frequency. The frequency in exercise unit E4 was 50 % of the frequency of the fastest set in E1, and it was controlled with a metronome. During the recovery periods between the sets the subjects were encouraged to have a few walking steps, but they were also allowed to sit if they preferred. Exercise unit E1 or E2 was always done first and the other units followed with at least four days intervals. However, E3 and E4 were done in two consecutive days. The sessions were always started at same time of the day.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>The exercise sessions in experiment 1.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sets</td>
</tr>
<tr>
<td>E1</td>
<td>10</td>
</tr>
<tr>
<td>E2</td>
<td>10</td>
</tr>
<tr>
<td>E3</td>
<td>2</td>
</tr>
<tr>
<td>E4</td>
<td>5</td>
</tr>
</tbody>
</table>
4.2.3 Knee extension exercises (II, III, IV, V)

The experiments 2, 3, 4 and 5 were all performed with a variable-resistance knee extension machine (David 200, David International, Vanta a, Finland). During each movement cycle, the leg extensor muscles worked concentrically during the extension phase and eccentrically during the lowering of the weight stack. The duration of each phase was indicated to the subject by an auditory feedback given by a metronome. The imposed cycle pace was set at 0.5 Hz in all these experiments. The particular machine used was specially modified so that the lever arm could be locked and the MVC measurements could also be performed.

In experiment 2 the subjects performed four testing sessions separated by at least three days of minimal physical activity in between. Care was taken that the tests were not performed with sore muscles. Each test consisted of one REPmax set either at 80%, 60%, 40% or 20% of the 1-RM of each subject (E80, E60, E40 and E20, respectively) (Table 3). The number of repetitions was counted loudly during the exercises and the subjects were also encouraged verbally to maximal performance. The first testing session started with the determination of the concentric 1-RM, and continued with E80. The other tests, E60, E40 and E20 were performed in a randomised order on the 3 other testing sessions. The sessions were always started at same time of the day.

<table>
<thead>
<tr>
<th>Sets</th>
<th>Repetitions</th>
<th>Load</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>E80</td>
<td>1 maximal</td>
<td>80% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E60</td>
<td>1 maximal</td>
<td>60% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E40</td>
<td>1 maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E20</td>
<td>1 maximal</td>
<td>20% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
</tbody>
</table>

Experiment 3 consisted of six testing sessions. The first testing session included a familiarisation of the subjects to the measurements of MVC and 1-RM. Then the resistance training protocol included five identical resistance exercise sessions separated by two days of rest in between and starting every time at the same time of the day (E1, E2, E3, E4, and E5, respectively) (Table 4).

<table>
<thead>
<tr>
<th>Sets</th>
<th>Repetitions</th>
<th>Load</th>
<th>Frequency</th>
<th>Rest between sets</th>
</tr>
</thead>
<tbody>
<tr>
<td>E1</td>
<td>6 12</td>
<td>50% of 1-RM</td>
<td>0.5 Hz</td>
<td>2 min</td>
</tr>
<tr>
<td>E2</td>
<td>6 12</td>
<td>50% of 1-RM</td>
<td>0.5 Hz</td>
<td>2 min</td>
</tr>
<tr>
<td>E3</td>
<td>6 12</td>
<td>50% of 1-RM</td>
<td>0.5 Hz</td>
<td>2 min</td>
</tr>
<tr>
<td>E4</td>
<td>6 12</td>
<td>50% of 1-RM</td>
<td>0.5 Hz</td>
<td>2 min</td>
</tr>
<tr>
<td>E5</td>
<td>6 12</td>
<td>50% of 1-RM</td>
<td>0.5 Hz</td>
<td>2 min</td>
</tr>
</tbody>
</table>

Experiment 4 consisted of seven sets of bilateral knee extensions. In the first, submaximal, part of the exercise (Esubmax) the subjects performed five sets of ten
repetitions at 40 % load of the 1-RM, measured before the exercise. In the second, maximal, part of the exercise three min later two sets of maximal number of repetitions at 40 % load were performed (E_{max1} and E_{max2}, respectively) (Table 5).

**TABLE 5** The exercise session in experiment 4.

<table>
<thead>
<tr>
<th>Sets</th>
<th>Repetitions</th>
<th>Load</th>
<th>Frequency</th>
<th>Rest between sets</th>
</tr>
</thead>
<tbody>
<tr>
<td>E_{submax}</td>
<td>5</td>
<td>10</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E_{max1}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E_{max2}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
</tbody>
</table>

Experiment 5 utilised the same exercise protocol as experiment 4. However, the subjects performed the exercise twice (E1 and E2, respectively), and the E2 was timed to 48 h after the E1 where DOMS was supposed to peak (Table 6).

**TABLE 6** The exercise sessions in experiment 5.

<table>
<thead>
<tr>
<th>Sets</th>
<th>Repetitions</th>
<th>Load</th>
<th>Frequency</th>
<th>Rest between sets</th>
</tr>
</thead>
<tbody>
<tr>
<td>E1</td>
<td>E_{submax}</td>
<td>5</td>
<td>10</td>
<td>40% of 1-RM</td>
</tr>
<tr>
<td>E_{max1}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E_{max2}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>E2</td>
<td>E_{submax}</td>
<td>5</td>
<td>10</td>
<td>40% of 1-RM</td>
</tr>
<tr>
<td>(48 h) E_{max1}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
<tr>
<td>later) E_{max2}</td>
<td>1</td>
<td>maximal</td>
<td>40% of 1-RM</td>
<td>0.5 Hz</td>
</tr>
</tbody>
</table>

4.2.4 Determinations of 1-RM and exercise loads (I, II, III, IV, V)

One day before the beginning of the experiment 1 the 1-RM in half squat was measured for the determination of the exercise loads. The 1-RM measurement was performed in the same apparatus that was then used in the actual exercises. The subject started the trial by bending the knees with a loaded barbell on his shoulders, went down to 90° knee angle, and returned to the standing position. The range of the movement was controlled with an individually adjustable flexible metal bar touching the hamstring muscles at the lowest position of the squat. The 1-RM determination (in kg) could be performed with 4-6 trials in every subject, and the exercise loads in percentage of the 1-RM were calculated and set as accurately as possible (to the nearest 0.125 kg).

In experiments 2, 3, 4 and 5 the lowest possible increase in load was 5 kg in the 1-RM measurements. The range of movement in the knee joint was 90° (from 90° to full extension 180°) and it was controlled with light signals at both ends of the desired range (II, III), or only at the desired extension angle (IV, V). The 1-RM determination could be performed in all cases with 4-6 trials, and the exercise loads in percentage of the 1-RM were calculated and set as accurately as possible (to the nearest 5 kg). In experiment 2 the first testing session started with the determination of the 1-RM. In experiment 3 the 1-RM was measured
two days before the exercise session and in experiments 4 and 5 the exercise sessions were started with the measurements of the 1-RM.

4.2.5 Calculations of work and power (I, II, III, IV, V)

In experiment 1 the displacements of the barbell were videotaped together with a stationary cm-scale for the determination of the \( W_{\text{tot}} \) in each exercise unit (work was calculated as the product of the force and the vertical distance moved). The \( P_{\text{mean}} \) was then calculated by dividing the \( W_{\text{tot}} \) by the total working time measured from the videotape recordings. In addition, power was also calculated individually for each successive six-repetition period in every exercise unit, and the highest value was taken to represent the \( P_{\text{peak}} \).

In experiments 2 and 3 the range of movement in the knee joint (90°) was controlled with light signals at both ends of the desired range, and in experiment 2 the displacement of the weight stack was measured individually for each subject. The \( W_{\text{tot}} \) and \( P_{\text{mean}} \) were then calculated similarly as in experiment 1. In experiments 4 and 5 the actual displacement of the weight stack during each extension and flexion was calculated by using the lever arm angle data for the determination of the \( W_{\text{tot}} \) during the different parts of the exercise.

4.2.6 Measurements of electromyography (EMG) (II, III, IV, V)

Surface EMG activity of the knee extensor muscles, vastus lateralis (II, III, IV, V), vastus medialis (II, III, IV, V) and rectus femoris (III, IV, V), was recorded either unilaterally (II, IV, V) or bilaterally (III) during the measurements. In all cases bipolar surface EMG recording was employed, and the electrodes (Beckman miniature sized skin electrodes, electrode diameter 4 mm, interelectrode distance 20 mm) were placed longitudinally on the muscle belly. The positions of the electrodes were kept identical for the different sessions by using small ink dots on the skin (II, III, V). EMG signals were monitored throughout the exercise sessions and were simultaneously stored on the computer by using an EMG-measurement program (Myosystem 1000, Noraxon, Finland) (II), or telemetrically (Biomes 2000, Glonner Electronic GmbH, Munchen, Germany) (III, IV, V) on the computer together with the force and knee angle data. The bandwidth for the EMG recording was 3-360 Hz, and the sampling frequency for all signals was either 500 Hz (II) or 1 kHz (III, IV, V).

The EMG signals recorded in 1-RM (II), in MVC (III, IV, V) and during the actual exercise contractions (II, III, IV, V) were first integrated (iEMG) and then time-normalized (aEMG). These analyses were performed by using two different EMG-analysis programs (Myosystem 1000, Noraxon, Oulu, Finland, II and Mega Electronics ME3000P, Kuopio, Finland, III, IV and V). In each experiment every muscle was analysed individually, and the mean value of the aEMG of the analysed muscles was taken for further analysis.

The test-retest reproducibility for most of the parameters in EMG patterns during stretch-shortening-cycle (SSC) muscle actions, indicated by reliability
coefficients, has been reported to be higher than 0.90 (Gollhofer et al. 1990). Under isometric conditions reproducibility is even higher.

4.2.6.1 Muscle activation level during the exercises (II, III, IV, V)

In experiments 2, 3, 4 and 5 the level of muscle activation was evaluated by the mean aEMG_{exc} normalised for the aEMG_{max}. This calculated value indicates the mean muscle activation level in percentage of the aEMG_{max} during each exercise. In addition, in experiment 2 the amount of muscular activity was evaluated by determining the iEMG_{tot} of the agonist muscles.

In order to examine potential adjustments of the nervous system to the progressive contractile fatigue during each test in experiment 2, the relative change in the aEMG normalised by the load level, \[ \Delta \text{aEMG:load} = (\text{aEMG:load}_{\text{end}} - \text{aEMG:load}_{\text{beg}})/\text{aEMG:load}_{\text{beg}} \times 100 \], was calculated by using 2, 3, 4, and 5 repetitions from the beginning and from the end in the E80, E60, E40 and E20 tests, respectively. In experiments 3, 4 and 5 the relative change in the aEMG_{exc} (ΔaEMG_{exc}) from first set to the last set, during each exercise, was also calculated. Increase in EMG signal amplitude during fatiguing exercise at submaximal loading levels may be explained by a recruitment of new motor units, and/or an increase in firing frequency of the already active units (Komi 1983).

4.2.7 Indicators of muscle fatigue (I, III, IV, V)

In experiments 3, 4 and 5 the lever arm of the exercise machine was locked at 100° knee angle for the MVC measurements before and after each exercise. A strain gauge that was attached to the lever arm was used in the measurement of the MVC, which was calculated in N taking into account the individually adjusted length of the arm. The best MVC of the two to three trials, both before and after the exercise, was chosen for further analysis, and the ΔMVC was calculated to evaluate the exercise induced fatigue. The aEMG_{max} values measured during the MVC measurements were used to evaluate the nature of fatigue (i.e. whether it was associated with a decrease in muscle activation).

In experiment 1 fatigue was evaluated by calculating a fatigue index (percentage decline in the performance during each exercise): \[ \left( \frac{P_{\text{peak}} - P_{\text{end}}}{P_{\text{peak}}} \right) \times 100 \], where \( P_{\text{peak}} \) is the highest six repetition period mean power and \( P_{\text{end}} \) is the mean power in the last six repetition period.

4.2.8 Blood sampling and analyses

4.2.8.1 Methods of blood sampling (I, II, III, IV, V)

In experiments 1, 2 and 3 conventional blood sampling with a heparinised needle from antecubital vein was employed. In all of these experiments one sample was taken before and one after the exercises. In experiment 1 the samples were taken in a lying position and the postexercise samples were taken
3 min after the exercise. In experiments 2 and 3 the samples were drawn in sitting position and the postexercise samples were also taken immediately after the exercises. During experiments 4 and 5 arterialised venous samples were obtained from the vein of the back hand with a winged infusion set. The fingers and knuckles were placed in 40°C water 5 min before the preexercise sample and in addition to the postexercise sample, blood was sampled also after submaximal part of the exercise and after the first REP<sub>max</sub> set. The preexercise samples were always taken after the warm-up and 10 min rest. No food intake was allowed during the last 3 hours before any of the measurements.

The morning samples for SH (I, IV, V) and for CK activity (III, V) were taken from antecubital vein in standardised fasting conditions (i.e. an overnight fast). The samples for B-La (I, II, III, IV, V) and GLU (IV, V) were taken from fingertip. For Hb and Hcr either fingertip blood samples (IV, V) or venous blood samples were used (III). Experimental testing of the female subjects was either timed to the early follicular phase of the menstrual cycle (days 3-5, IV), or the cycle phase was not controlled (II).

### 4.2.8.2 Plasma CA analyses (I, II, III, IV, V)

The samples for the plasma CA determination were centrifuged at 3°C with an anti-oxidant solution containing ethyleneglycoltetra-acetic acid (EGTA) and reduced glutathione. The plasma was removed and stored at -80°C.

In experiments 1 and 2 the proteins in an 0.8 ml plasma sample were precipitated by addition of 80 μl of 20 % HClO<sub>4</sub>, which contained 5 mmol/l ethylenediaminetetra-acetic acid (EDTA). The tube was mixed in a vortex mixer and centrifuged at 4°C for 5 min at 16500 rpm. The resulting supernatant (0.6 ml) was transferred to an Eppendorf tube which contained 20 mg of activated alumina. The internal standard, 150 μl of dihydroxybenzylamine (DHBA) 10<sup>-7</sup> mol/l was added and the pH of the mixture was adjusted to 8.6 by addition of 0.3 ml TRIS buffer (2 mol/l). This was vortexed vigorously for 30 s and then centrifuged for 2 min. The supernatant was removed by suction and the alumina pellet washed with 1.0 ml of ice cold distilled water, vortexed, centrifuged and supernatant removed. This washing was repeated twice.

Finally the CA were eluted from the alumina by addition of 125-μl 2 % CH<sub>3</sub>COOH, vortexed, centrifuged and 75 μl transferred to a microtube for insertion in the automatic sampler of the high-performance liquid chromatography apparatus (HPLC). The HPLC set up consisted of a pump (LKB) delivering buffer at a rate of 1.3 ml/min. The buffer itself consisted of 0.1 mol/l sodium acetate; 0.1 mol/l citric acid; 150 mg/l sodium octylsulphate and 10 % methanol. The analytical column was C18 reversed phase column (25 x 0.6 cm) from Beckman (Ultrasphere ODS). Detection of the agents eluting from the column was coulometric (ESA 5011 cell) set at +0.35 V. This system measures the oxidation of catecholamines as they pass through the electrode. The ratio of the peak height of the amine of interest to the peak height of the internal standard was divided by equivalent ratio from known standards to obtain the concentration (in nanomoles per litre). The lower detection limit of the assay for
both NA and A was 0.2 nmol/l and the intra-assay coefficient of variation was 3-7%.

In experiment 3 the CA from 500 μl plasma were extracted into 30 mg Al₂O₃ in 3 ml of tris-HCl buffer (pH 8.65) in 5 ml conical test tubes. 3,4-dihydroxybenzylamine hydrobromide (Sigma, St. Louis, MO, USA) was used as an internal standard to correct for absolute recovery variations in CA. After washing four times with 2 ml H₂O, the CA were eluted into 100 μl 0.2 M HClO₄ solution. The CA in the eluates (50 μl) were measured by high pressure liquid chromatography with electrochemical detector (Esa Coulchem Multi-Electrode, model 5100 A). For the analysis of NA and A ESA CA HR-80 column (C-18 reversed phase column, 80 x 4.0 mm, 3 μm) and methanol-phosphate buffer, pH 2.2 (ESA Cat-A-Phase reagent) as mobile phase were used. Flow rate was 0.6 ml/min⁻¹.

In experiments 4 and 5 the CA from 1000 μl plasma were later extracted into 30 mg Al₂O₃ in 3 ml of tris-HCl buffer (pH 8.65) in 5 ml conical test tubes. 3,4-dihydroxybenzylamine hydrobromide (Sigma, St. Louis, MO, USA) was used as an internal standard to correct for absolute recovery variations in CA. After washing four times with 2 ml H₂O, the CA were eluted into 100 μl 0.2 M HClO₄ solution. The CA in the eluates (50 μl) were measured by high pressure liquid chromatography (HPLC) with multichannel electrochemical detector (Esa CoulArray, model 5600). For the analysis of NA and A Inertsil ODS-3 column (4.0 x 150 mm, 3 μm, GL Sciences Inc., Tokyo, Japan) and citric acid-monochloracetic acid- acetonitrile buffer (pH 3.4) as mobile phase were used. Flow rate was 0.6 ml/min⁻¹.

In experiments 3, 4 and 5 known CA standards were treated in the same way as samples for calibration purposes, and the peak height ratios (relative to the peak height of the internal standard) of unknown CA were compared to those of synthetic standards (D-2500 Chromato-Integrator, Merck Hitachi, Japan, III; CoulArray Software 1.003, IV and V). The detection limit of CA standards in the described methods was 0.2 nmol/l and the inter-assay coefficient of variation was 5-6%.

4.2.8.3 Analysis of serum hormones (I, IV, V)

Serum samples for the hormonal analysis were kept frozen at -25°C until assayed. In experiment 1 the concentrations of serum TES<sub>tot</sub> and COR were measured by RIA (Farmos Diagnostica, Turku, Finland), as well as those of GH (Pharmacia Diagnostics, Uppsala, Sweden). Serum TES<sub>tot</sub> concentrations were measured using RIA kits from Diagnostics Products (Los Angeles, CA). The sensitivity of the TES<sub>tot</sub> assay was 0.3 nmol/l and the coefficient of intra-assay variation was 6.2%. The respective values for COR were 0.05 μmol/l and 4.0%, for GH 0.2 μg/l and 4.4% and for TES<sub>exo</sub> 0.52 pmol/l and the intra-assay variation was 4.4%. In addition, the concentration of serum SHBG was determined by RIA using reagent kits from Farmos Diagnostica (Oulunsalo, Finland). The sensitivity of the assay was 0.5 nmol/l and the intra-assay coefficient of variation was 2.2%.
In experiments 4 and 5 the concentrations of serum TES<sub>tot</sub> were measured by the Chiron Diagnostics ACS:180 automated chemiluminescence system using ACS:180 analyzer. The sensitivity of the assay was 0.4 nmol/L and the coefficient of intra-assay variation was 6.7%. Serum GH and SHBG concentrations were analyzed by two-site fluoroimmunometric methods using AutoDELFIA analyzer (Wallac Oy, Turku, Finland). The sensitivity and the intra-assay coefficient of variation of the GH assay was 0.01 μg/L and 2.7% and those of the SHBG assay 1.2 nmol/L and 2.0%. Serum COR concentrations were measured by radioimmunoassay kits from Orion Diagnostica (Espoo, Finland). The sensitivity of the assay was 0.05 μmol/L and the intra-assay variation was 4.0%. The concentrations of TES<sub>tot</sub> were measured by radioimmunoassay kits from Diagnostic Products Corp. (Los Angeles, CA). The sensitivity of the assay was 0.52 pmol/L and the intra-assay variation was 3.8%.

4.2.8.4 Other blood analyses (I, II, III, IV, V)

The samples for the B-La were analysed enzymatically in each experiment (Boehringer Mannheim, Germany). The determination of blood GLU (IV, V) was performed photometrically (HemoCue, Angelholm, Sweden), as well as also that of serum CK activity (Boehringer Mannheim, Germany) (III, V). The Hcr and Hb (HemoCue, Angelholm, Sweden) values were used to calculate the ΔPV% by the method of Dill and Costill (1974) (III, IV, V). The exercise-induced increases of TES<sub>tot</sub>, COR and GH concentrations were also evaluated after correction for ΔPV% (IV, V), as these hormones bind to proteins in the circulation and concentrate when plasma volume decreases.

4.2.9 Other measurements (I, II, III, IV, V)

Heart rate (f<sub>H</sub>) was measured in every experiment. In experiments 1 and 2 a PE-3000 Sport Tester (Polar Electro, Kempele, Finland), and in experiments 3, 4 and 5 a Polar Vantage NV heart rate monitor (Polar Electro, Kempele, Finland) was used.

In experiments 3, 4 and 5 the subjects were given a questionnaire for the evaluation of DOMS during the following recovery days after the exercise session (scale 0 = no soreness, 5 = intolerable soreness).

Body fat content was determined in every experiment from skinfold thickness. In experiments 1, 2 and 3 the method of Durnin & Rahaman (1967) and in experiment 4 and 5 the method of Durnin & Womersley (1974) was employed. The LBM was always determined by subtracting the mass of the body fat from body mass.

4.2.10 Statistical methods

All statistical analyses were done by the SPSS™ programme (ver. 6.1.3, SPSS Inc, USA). Means and standard deviations (SD) were first calculated. In experiment 1 the before-after comparisons and group comparisons were performed by
means of nonparametric ranking tests, Wilcoxon’s for paired data, and Mann-
Whitney’s for unpaired data. Friedman’s test was used to evaluate whether
significant differences occurred between the exercise sessions. The extent of
association between variables was evaluated according to Spearman. In
experiments 2 and 3 analysis of variance (ANOVA) with repeated measures
and Student’s t-test for unpaired and paired data were used. In experiment 4
multivariate ANOVA with repeated measures was employed, and the
Newman-Keuls post hoc test was used in the event of a significant (P < 0.05) F
ratio. The before-after comparisons were performed by using paired t-test. In
experiment 5 two way ANOVA with repeated measures and Student’s t-test for
paired data were employed. Before these analyses in experiments 2, 3, 4 and 5
the normality of the data distribution was tested by using the Kolmogorov-
Smirnov Test. Pearson’s product-moment correlation coefficients were
calculated to evaluate the bivariate relationships between variables (II, III, IV,
V). In every experiment the correlations between the plasma CA and other
blood parameters were calculated both by using the postexercise concentrations
and the exercise induced changes from preexercise (Δvalues). The accepted
level of significance was P < 0.05. Statistical power (at P < 0.05) was calculated
for the different hormone concentration comparisons between two independent
groups and for paired data (I, II, III, V) (Altman 1992), as well as for multiple
comparisons between three groups (IV) (Glantz 1992). In the results the
statistical power of the most important findings in the light of the hypotheses
and purpose of the study are presented.

4.2.11 Summary of the experimental design

Table 7 summarises the experimental design and measurements of the present
study.

TABLE 7 Experimental design and measurements of the study

<table>
<thead>
<tr>
<th></th>
<th>Experiment 1</th>
<th>Experiment 2</th>
<th>Experiment 3</th>
<th>Experiment 4</th>
<th>Experiment 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>SH</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>B-La</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>GLU</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>ΔPV%</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>DOMS</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>f_s</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>W_tot</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>P1_mus</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2_mus</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EMG</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Fatigue</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
</tbody>
</table>
5 RESULTS

The main findings from the present experiments are presented below. For more details the original papers (I-V) should be consulted. Some unpublished results are also included.

5.1 Neuromuscular performance

In experiment 1 the men had a higher 1-RM than the boys [177.5 (SD 20.0) vs. 127.5 ± 32.5 kg, P < 0.05]. However, the $P_{\text{mean}}$ and $P_{\text{peak}}$ values normalised for body weight were higher in men than in boys only in one exercise unit (E3), where also the fatigue index and B-La levels were higher than in the other exercises in both groups. The performances and B-La levels of the two subject groups in the four half-squatting exercise sessions are summarised in table S.

The performance results of the male and female subjects in the four resistance exercise tests are shown in table 9. No gender differences in the REP$_{\text{max}}$ in any of the tests were observed. However, due to the higher 1-RM [145 (SD 25) vs. 95 (SD 15) kg, P < 0.01)] the $W_{\text{tot}}$ and $P_{\text{mean}}$ were higher in men than in women in every test. Gender differences remained in the $W_{\text{tot}}$ after normalization for body mass in E60 and E40 (P < 0.05) and in $P_{\text{mean}}$ in every test (P < 0.05), but disappeared when normalized for LBM. As expected, the iEMG$_{\text{tot}}$ followed the same pattern as $W_{\text{tot}}$, while the aEMG$_{\text{exc}}$ was found to be highest in E80 with the highest $P_{\text{mean}}$ and lowest in E20 with the lowest $P_{\text{mean}}$. The $\Delta$aEMG:load ratio was significantly higher in E20 than in the other three exercise tests in both groups.
### TABLE 8  Performance and postexercise B-La levels of the men and boys in the four half-squatting exercise sessions (E1-E4).

<table>
<thead>
<tr>
<th></th>
<th>E1</th>
<th>E2</th>
<th>E3</th>
<th>E4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>$W_{tot}$ Boys</td>
<td>27.4</td>
<td>6.4</td>
<td>25.1</td>
<td>4.7</td>
</tr>
<tr>
<td>(kJ) Men</td>
<td>40.4</td>
<td>3.0$^b$</td>
<td>40.0</td>
<td>3.1$^b$</td>
</tr>
<tr>
<td>$P_{mean}$ Boys</td>
<td>7.1</td>
<td>1.4</td>
<td>6.9</td>
<td>1.7</td>
</tr>
<tr>
<td>(W kg$^{-1}$) Men</td>
<td>8.2</td>
<td>0.9</td>
<td>8.4</td>
<td>0.9</td>
</tr>
<tr>
<td>$P_{peak}$ Boys</td>
<td>7.2</td>
<td>1.5</td>
<td>7.1</td>
<td>1.6</td>
</tr>
<tr>
<td>(W kg$^{-1}$) Men</td>
<td>8.4</td>
<td>0.8</td>
<td>8.6</td>
<td>0.9</td>
</tr>
<tr>
<td>Fatigue Boys</td>
<td>7.4</td>
<td>2.9</td>
<td>4.7</td>
<td>4.2</td>
</tr>
<tr>
<td>index (%) Men</td>
<td>8.2</td>
<td>0.9</td>
<td>8.4</td>
<td>0.9</td>
</tr>
<tr>
<td>B-La Boys</td>
<td>3.1</td>
<td>0.4</td>
<td>3.5</td>
<td>1.6</td>
</tr>
<tr>
<td>(mmol l$^{-1}$) Men</td>
<td>3.6</td>
<td>1.2</td>
<td>3.8</td>
<td>1.6</td>
</tr>
</tbody>
</table>

$^a$ P < 0.05, $^b$ P < 0.01 Differences between the groups. $^{1,2,4}$ Difference from corresponding value of the corresponding exercise session P < 0.05, (n = 7 in both groups).

### TABLE 9  Performance, muscle activity and postexercise B-La levels of the male and female subjects in the four knee-extension tests.

<table>
<thead>
<tr>
<th></th>
<th>E80</th>
<th>E60</th>
<th>E40</th>
<th>E20</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>REP$\text{max}$ Women</td>
<td>7.4</td>
<td>0.9</td>
<td>14.5</td>
<td>3.7</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>6.8</td>
<td>1.0</td>
<td>13.9</td>
</tr>
<tr>
<td>$W_{tot}$ Women</td>
<td>3.8</td>
<td>0.8</td>
<td>5.4</td>
<td>0.9</td>
</tr>
<tr>
<td>(kJ) Men</td>
<td>5.4</td>
<td>1.3$^e$</td>
<td>8.2</td>
<td>1.9$^e$</td>
</tr>
<tr>
<td>$P_{mean}$ Women</td>
<td>4.9</td>
<td>0.9</td>
<td>3.7</td>
<td>0.6</td>
</tr>
<tr>
<td>(W kg$^{-1}$LBM) Men</td>
<td>5.6</td>
<td>0.9</td>
<td>4.1</td>
<td>0.5</td>
</tr>
<tr>
<td>iEMG$\text{tot}$ Women</td>
<td>3.04</td>
<td>0.90</td>
<td>4.07</td>
<td>1.40</td>
</tr>
<tr>
<td>(mVs) Men</td>
<td>4.21</td>
<td>1.61</td>
<td>6.69</td>
<td>2.67$^e$</td>
</tr>
<tr>
<td>aEMG$\text{max}$ Women</td>
<td>119</td>
<td>22</td>
<td>88</td>
<td>23</td>
</tr>
<tr>
<td>(%) Men</td>
<td>91</td>
<td>16$^e$</td>
<td>71</td>
<td>23</td>
</tr>
<tr>
<td>$\Delta$EMG$\text{load}$ Women</td>
<td>27</td>
<td>21</td>
<td>49</td>
<td>33</td>
</tr>
<tr>
<td>(%) Men</td>
<td>12</td>
<td>21</td>
<td>33</td>
<td>22$^{1,2}$</td>
</tr>
<tr>
<td>B-La Women</td>
<td>4.0</td>
<td>1.1</td>
<td>5.1</td>
<td>1.1$^e$</td>
</tr>
<tr>
<td>(mmol l$^{-1}$) Men</td>
<td>4.0</td>
<td>1.2</td>
<td>5.8</td>
<td>1.5$^e$</td>
</tr>
</tbody>
</table>

$^a$ P < 0.05, $^b$ P < 0.01, $^c$ P < 0.01 Differences between the groups. $^{1,2,4}$ Difference from corresponding value of the corresponding exercise session P < 0.05. In both groups REP$\text{max}$, $W_{tot}$, and $P_{mean}$ (P < 0.001) as well as iEMG$\text{tot}$ and aEMG (P < 0.05) were significantly different in every test from all the other tests, (women, n = 8 and men, n = 9).
In the knee-extension exercise (exp. 4) the adult male subjects had a higher 1-RM and exercise load than the women and boys. However, no differences between the groups were observed in the number of repetitions performed, in the W_{tot} relative to LBM, in aEMG_{max}, or in ΔaEMG (table 10). No differences between the groups were also observed in the MVC decrease after the exercise, but the postexercise B-La was higher in men than in the other two groups. Muscle soreness peaked two days after the exercise both in men [2.3 (SD 1.6)] and in women [2.3 (SD 1.0)], while the boys reported the highest soreness ratings already at the testing day [1.5 (SD 1.2)], and almost no soreness two days later [0.3 (SD 0.8)].

**TABLE 10 Performance, muscle activity, fatigue and postexercise B-La levels in boys, men and women in exhaustive knee-extension exercise session.**

<table>
<thead>
<tr>
<th></th>
<th>Boys</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1-RM (kg)</strong></td>
<td>87 ± 23</td>
<td>154 ± 29</td>
<td>103 ± 9</td>
</tr>
<tr>
<td><strong>Repetitions</strong></td>
<td>E_{submax} 5×10</td>
<td>5×10</td>
<td>5×10</td>
</tr>
<tr>
<td></td>
<td>E_{max1} 23 ± 9</td>
<td>21 ± 6</td>
<td>24 ± 3</td>
</tr>
<tr>
<td></td>
<td>E_{max2} 18 ± 3</td>
<td>17 ± 4</td>
<td>20 ± 4</td>
</tr>
<tr>
<td><strong>W_{tot} (J·kg⁻¹·LBM)</strong></td>
<td>E_{submax} 271 ± 50</td>
<td>331 ± 40</td>
<td>322 ± 32</td>
</tr>
<tr>
<td></td>
<td>E_{max1} 117 ± 31</td>
<td>133 ± 33</td>
<td>147 ± 29</td>
</tr>
<tr>
<td></td>
<td>E_{max2} 93 ± 12</td>
<td>102 ± 17</td>
<td>115 ± 25</td>
</tr>
<tr>
<td><strong>aEMG_{max} (%)</strong></td>
<td>E_{submax} 60 ± 12</td>
<td>51 ± 10</td>
<td>58 ± 8</td>
</tr>
<tr>
<td></td>
<td>E_{max1} 75 ± 14</td>
<td>64 ± 8</td>
<td>73 ± 11</td>
</tr>
<tr>
<td></td>
<td>E_{max2} 89 ± 13</td>
<td>72 ± 11</td>
<td>78 ± 15</td>
</tr>
<tr>
<td><strong>ΔaEMG_{max} (%)</strong></td>
<td>53 ± 8</td>
<td>47 ± 15</td>
<td>44 ± 19</td>
</tr>
<tr>
<td><strong>MVC_{pre} (N)</strong></td>
<td>1346 ± 275</td>
<td>1916 ± 363</td>
<td>1455 ± 202</td>
</tr>
<tr>
<td><strong>aEMG_{max} (mV)</strong></td>
<td>0.36 ± 0.07</td>
<td>0.36 ± 0.09</td>
<td>0.28 ± 0.07</td>
</tr>
<tr>
<td><strong>ΔMVC (N)</strong></td>
<td>-14.9 ± 9.4</td>
<td>-25.2 ± 9.0</td>
<td>-25.0 ± 11.6</td>
</tr>
<tr>
<td><strong>B-La (mM·l⁻¹)</strong></td>
<td>8.4 ± 1.4</td>
<td>10.6 ± 1.2</td>
<td>8.4 ± 1.6</td>
</tr>
</tbody>
</table>

* P < 0.05 Significantly different from men (n = 6 in each group)

The performance of the adult male subjects in the five successive knee-extension exercises (exp. 3) is summarised in table 11. The 1-RM and consequently also the exercise load and W_{tot} were higher, but the aEMG_{max}:load ratio and VO_{2 tot}:load lower in E5 than in E1. The preexercise MVC and aEMG_{max} were also higher in E5 than in E1 (9 and 19 %, respectively, P < 0.05). Decrease in the MVC immediately after the exercise was similar in every exercise session (33.9 – 36.1 %, P < 0.01). The E1 induced DOMS which peaked one day after the exercise [2.0 (SD 1.5)] that had not totally disappeared in E2 three days after the E1 [0.6 (SD 1.1)], but the serum CK activity was not different between E1 and E2 [330 (SD 323) U·l⁻¹ vs. 412 (SD 273) U·l⁻¹, ns].
TABLE 11 Performance, muscle activity, fatigue and postexercise B-La levels of the adult male subjects in the five successive resistance exercise sessions.

<table>
<thead>
<tr>
<th></th>
<th>E1</th>
<th>E2</th>
<th>E3</th>
<th>E4</th>
<th>E5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-RM (kg)</td>
<td>166 ± 30</td>
<td>170 ± 35</td>
<td>175 ± 29</td>
<td>178 ± 29</td>
<td>178 ± 30</td>
</tr>
<tr>
<td>W&lt;sub&gt;tot&lt;/sub&gt; (kJ)</td>
<td>38.2 ± 7.6</td>
<td>38.3 ± 8.3</td>
<td>40.0 ± 8.4</td>
<td>41.0 ± 7.6</td>
<td>42.5 ± 7.8</td>
</tr>
<tr>
<td>aEMG&lt;sub&gt;exerc&lt;/sub&gt;</td>
<td>69.2 ± 10.7</td>
<td>68.9 ± 6.7</td>
<td>69.9 ± 9.6</td>
<td>66.4 ± 8.8</td>
<td>63.1 ± 9.4</td>
</tr>
<tr>
<td></td>
<td>(% aEMG&lt;sub&gt;max&lt;/sub&gt;)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aEMG&lt;sub&gt;exerc&lt;/sub&gt; 1-load</td>
<td>3.52 ± 1.63</td>
<td>3.56 ± 1.59</td>
<td>3.60 ± 1.69</td>
<td>3.60 ± 1.81</td>
<td>3.36 ± 1.66</td>
</tr>
<tr>
<td>ΔaEMG&lt;sub&gt;exerc&lt;/sub&gt; (%)</td>
<td>21 ± 17</td>
<td>22 ± 12</td>
<td>20 ± 5</td>
<td>16 ± 9</td>
<td>13 ± 12</td>
</tr>
<tr>
<td>MVC&lt;sub&gt;PRE&lt;/sub&gt; (N)</td>
<td>1672 ± 229</td>
<td>169 ± 302</td>
<td>170 ± 279</td>
<td>1750 ± 274</td>
<td>1822 ± 302</td>
</tr>
<tr>
<td>aEMG&lt;sub&gt;max&lt;/sub&gt; (mV)</td>
<td>0.37 ± 0.06</td>
<td>0.38 ± 0.08</td>
<td>0.40 ± 0.12</td>
<td>0.43 ± 0.13</td>
<td>0.44 ± 0.12</td>
</tr>
<tr>
<td>ΔMVC (N)</td>
<td>-35.3 ± 13.4</td>
<td>-8.5</td>
<td>-7.8</td>
<td>-35.5 ± 9.7</td>
<td>-33.9 ± 11.3</td>
</tr>
<tr>
<td>B-La (mmol&lt;sup&gt;1&lt;/sup&gt;)</td>
<td>7.9 ± 0.8</td>
<td>9.4 ± 1.1</td>
<td>8.0 ± 1.5</td>
<td>7.3 ± 1.4</td>
<td>7.8 ± 1.5</td>
</tr>
</tbody>
</table>

1234 Different from corresponding value of the corresponding exercise unit P < 0.05, (n = 8).

TABLE 12 Performance, muscle activity, fatigue and postexercise B-La levels in men in two successive exhaustive knee-extension exercise sessions.

<table>
<thead>
<tr>
<th></th>
<th>E1</th>
<th>E2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-RM (kg)</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Repetitions</td>
<td>E₁</td>
<td>E₂</td>
</tr>
<tr>
<td>W&lt;sub&gt;tot&lt;/sub&gt; (kJ)</td>
<td>E₁</td>
<td>E₂</td>
</tr>
<tr>
<td>aEMG&lt;sub&gt;exerc&lt;/sub&gt;</td>
<td>E₁</td>
<td>E₂</td>
</tr>
<tr>
<td>(% aEMG&lt;sub&gt;max&lt;/sub&gt;)</td>
<td>E₁</td>
<td>E₂</td>
</tr>
<tr>
<td>ΔaEMG&lt;sub&gt;exerc&lt;/sub&gt; (%)</td>
<td>47 ± 15</td>
<td>37 ± 17</td>
</tr>
<tr>
<td>MVC&lt;sub&gt;PRE&lt;/sub&gt; (N)</td>
<td>1916 ± 363</td>
<td>1862 ± 257</td>
</tr>
<tr>
<td>aEMG&lt;sub&gt;max&lt;/sub&gt; (mV)</td>
<td>0.36 ± 0.09</td>
<td>0.36 ± 0.11</td>
</tr>
<tr>
<td>ΔMVC (N)</td>
<td>-25.2 ± 9.0</td>
<td>-20.9 ± 3.1</td>
</tr>
<tr>
<td>B-La (mmol&lt;sup&gt;1&lt;/sup&gt;)</td>
<td>10.6 ± 1.2</td>
<td>10.2 ± 1.9</td>
</tr>
</tbody>
</table>

Significantly different from E₁ P < 0.05 and E₂ P < 0.01, (n = 6).
In experiment 5 no differences between the two exercises were observed in the performance of the subjects (1-RM, MVC, REP<sub>max</sub>, W<sub>max</sub>), in fatigue (ΔMVC) or in postexercise B-La levels, but the aEMG<sub>conc</sub> was lower in E2 than in E1 (Table 12). Although the postexercise B-La levels were similar in the two exercise sessions, the blood GLU level 8 min postexercise decreased from preexercise in E2 (P < 0.05), and was also slightly but significantly lower than in E1 [4.9 (SD 0.3) vs. 4.4 (SD 0.2) mmol/l, P < 0.05]. The DOMS peaked just before the E2 [2.3 (SD 1.6)] and declined thereafter, but the CK activity increase did not reach the level of statistical significance [E1: 240 (SD 203) U/l vs. E2: 333 (SD 163) U/l, ns].

5.2 Plasma catecholamine responses in men and women

In experiment 2 no gender differences in the NA or A levels, or in the NA:A ratios, after the four REP<sub>max</sub> exercise tests could be observed (Fig. 4). No differences between women and men were also observed in the ΔNA [E80: 1.1 (SD 1.7) vs. 1.6 (SD 1.7) mmol/l, E60: 3.6 (SD 2.0) vs. 2.8 (SD 1.9) mmol/l, E40: 4.6 (3.2) vs. 3.6 (SD 3.8) mmol/l, E20: 6.8 (SD 4.9) vs. 8.1 (SD 6.6) mmol/l] or ΔA from pre- to postexercise [E80: 1.5 (SD 2.0) vs. 2.4 (SD 3.7) mmol/l, E60: 3.5 (SD 3.4) vs. 2.9 (SD 1.7) mmol/l, E40: 5.1 (SD 4.1) vs. 3.2 (SD 3.8) mmol/l, E20: 3.2 (SD 2.2) vs. 6.1 (SD 5.8) mmol/l]. In experiment 4 no gender differences were observed in the plasma NA concentrations either before or after the different parts of the exercise, but the A levels after the REP<sub>max</sub> sets were lower in women than in men. The NA:A ratios were also higher in women than in men throughout the exercise (Fig. 5). However, the ΔA from preexercise to the last two sets was similar in men [2.4 (SD 0.7) and 2.1 (SD 0.9) mmol/l] and women [2.1 (SD 0.7) and 1.7 (SD 0.6) mmol/l]. Also the ΔA from preexercise to the individual peak plasma level was similar in men [2.5 (SD 0.8) mmol/l] and in women [2.1 (SD 0.6) mmol/l].

5.3 Plasma catecholamine responses in men and boys

After the most exhausting half-squatting exercise session (E3) in experiment 1 the plasma NA level was about 50% lower in boys than in men, while the NA levels in the other three exercises, and the A levels in every exercise were similar in these two groups of subjects (Fig. 6). Also the ΔNA from pre- to postexercise was lower in boys than in men in exercise session E3 [14.3 (SD 7.2)] vs. 29.9 (SD 10.6) mmol/l, P < 0.01, statistical power 0.65], as well as in the exercise session E1 [1.7 (SD 2.1)] vs. 4.5 (SD 2.3) mmol/l, P < 0.01, statistical power 0.55]. In the knee extension exercise (exp. 4) the differences in the plasma NA or A concentrations between men and boys did not reach the level of statistical significance either before or after the different parts of the exercise (Fig. 5). However, the ΔA from preexercise to the last two sets in boys [5.0 (SD
2.6) and 3.8 (SD 2.2) nmol\(\text{L}^{-1}\) was significantly higher than in men [2.4 (SD 0.7) and 2.1 (SD 0.9) nmol\(\text{L}^{-1}\)] \(P < 0.05\), statistical powers 0.35 and 0.30, respectively). Also the plasma A increase from preexercise to the individual peak level, observed in most of the subjects already after the sixth set, was twice as high in boys [5.0 (SD 2.6) nmol\(\text{L}^{-1}\)] as in men [2.5 (SD 0.8) nmol\(\text{L}^{-1}\)] \(P < 0.05\), statistical power 0.35).

![FIGURE 4](image1.png)

**FIGURE 4** Mean and SD plasma NA and A concentrations and NA:A ratio in men (n = 9) and women (n = 8) before (PRE) and after (POST) the four knee-extension tests performed with 80 \%, 60 \%, 40 \%, and 20 \% loads of 1-RM (E80, E60, E40 and E20, respectively). Significantly different from pre-exercise \(^a P < 0.05\), \(^b P < 0.01\) and \(^c P < 0.001\). Significantly different from the corresponding value of the corresponding exercise test \(P < 0.05\).

![FIGURE 5](image2.png)

**FIGURE 5** Mean and SD plasma NA and A concentrations and NA:A ratio in boys (n = 6), men (n = 6) and women (n = 6) before (PRE) and after the submaximal (E\(_{\text{submax}}\)) and maximal (E\(_{\text{max1}}\) and E\(_{\text{max2}}\)) parts of the knee-extension exercise. Significantly different from pre-exercise \(^a P < 0.05\), \(^b P < 0.01\) and \(^c P < 0.001\). Significantly different from previous value \(^d P < 0.05\) and \(^e P < 0.01\). Significant difference between the groups \(^* P < 0.05\) (statistical powers 0.20-0.75).
5.4 Plasma catecholamine responses to repeated exercises

The plasma NA concentrations in men before and after the five knee-extension exercises performed with two days of rest in between remained unchanged (Fig. 7), but the postexercise plasma A level was already significantly lower in the second exercise than in the first one (62%, \( P < 0.05 \), statistical power 0.80) (Exp. 3). Also the plasma A from pre- to postexercise was clearly lower (75%) in the second exercise than in the first one [0.3 (SD 0.5) nmol\(^{-1}\) vs. 1.1 (SD 1.0) nmol\(^{-1}\), \( P < 0.05 \), statistical power 0.73]. Both the postexercise A level and the postexercise NA:A ratio tended to return to the same level in the last exercise.
session as in the first one, although the exercise induced increase in plasma A concentration reached the level of statistical significance only in the first exercise session.

In experiment 5 the plasma A concentration increased significantly both in the first and second exercise session, but only after the REPmax sets. However, the postexercise plasma A level was lower in the second exercise session than in the first one (23 %, \( P < 0.05 \), power 0.73). Again no differences in the plasma NA levels between the first and the second exercise could be observed (Fig. 8). Also no differences between the two exercises were observed in the plasma \( \Delta A \) from preexercise to the peak A level [2.5 (SD 0.8) vs. 2.7 (SD 0.5) nmol\(^{-1}\), ns].

![Graph of plasma NA and A concentrations and NA:A ratios before and after the five resistance exercises (E1-E5).](image1)

![Graph of plasma NA and A concentrations and NA:A ratios in two successive resistance exercises (n = 6).](image2)

**FIGURE 7** Mean and SD plasma NA and A concentrations and NA:A ratios before and after the five resistance exercises (E1-E5). \( * \), \( \text{b} \), \( \text{c} \), \( \text{f} \) Significantly different from pre-exercise \( P < 0.05 \), \( \text{b} \) \( P < 0.01 \) and \( P < 0.001 \), respectively, \( \text{l} \), \( \text{a} \), \( \text{e} \), \( \text{s} \) significantly different from the corresponding value of the corresponding exercise unit \( P < 0.05 \).

**FIGURE 8** Mean and SD plasma NA and A concentrations and NA:A ratios in two successive resistance exercises (n = 6). Significantly different from pre-exercise \( P < 0.05 \), \( \text{b} \) \( P < 0.01 \) and \( \text{c} \) \( P < 0.01 \). Significant difference between the exercises * \( P < 0.05 \).
5.5 Other hormonal responses and their relationships with plasma catecholamines

The concentrations of serum TES\textsubscript{tot}, TES\textsubscript{free}, and SHBG in the half-squatting exercises in men and boys (exp. 1) are presented in Fig. 9. The postexercise TES\textsubscript{tot} and TES\textsubscript{free} levels were significantly lower in boys than in men in every exercise session, but exercise-induced increases could be observed in both groups.

FIGURE 9 Mean and SD serum TES\textsubscript{tot}, TES\textsubscript{free}, and SHBG concentrations in the morning (REST), before (PRE) and after (POST) four half-squatting exercise units in young (Boys, n = 7) and adult male athletes (Men, n = 7). a Significantly different from preexercise P < 0.05. b, c Significant difference between the groups P < 0.05 and P < 0.01, respectively (statistical powers 0.23-0.92). Significantly different from the corresponding value of the corresponding exercise unit P < 0.05. The TES\textsubscript{free} and SHBG are previously unpublished data.
In experiment 1 also the $\Delta TES_{\text{tot}}$ from preexercise was lower in boys than in men in exercise sessions E1, E2 and E3 [-0.5 (SD 2.1) vs. 5.1 (SD 3.7) nmol/l, $P < 0.01$, statistical power 0.77; 3.1 (SD 0.8) vs. 5.7 (SD 2.8) nmol/l, $P < 0.05$, power 0.47; 4.5 (SD 2.2) vs. 10.6 (SD 3.7) nmol/l, $P < 0.01$, power 0.76, respectively], as well as also the $\Delta TES_{\text{free}}$ in exercise session E3 [4.0 (SD 4.8) vs. 27.2 (SD 16.1) pmol/l, $P < 0.01$, statistical power 0.55]. No correlation between the plasma CA and serum $TES_{\text{tot}}$ and $TES_{\text{free}}$ concentration responses were observed in either group, even if the four exercises were combined. Only by combining the two groups in E3 a weak positive correlation between the $\Delta NA$ and $\Delta TES_{\text{tot}}$ could be observed (Fig. 10).

\[
\begin{array}{c}
\text{FIGURE 10} \quad \text{Relationship between the plasma $\Delta NA$ and serum $\Delta TES_{\text{tot}}$ after the half-squatting exercise session E3 when boys (grey dots) and men (black dots) are combined.}
\end{array}
\]

In the knee-extension exercise (exp. 4) the postexercise $TES_{\text{tot}}$ and $TES_{\text{free}}$ levels were also lower in boys than in men, but no increase in $TES$ level was observed in boys (Fig 11). In this experiment (exp. 4), after correction for the $\Delta PV\%$, which was significant in both groups but larger in men than in boys both after the submaximal part of the exercise [-11.9 (SD 3.8) % vs. -3.7 (SD 3.2) %, $P < 0.01$] as well as at the end of the exercise [-14.6 (SD 5.2) % vs. -7.1 (SD 3.1) %, $P < 0.05$], no increase in $TES_{\text{tot}}$ levels could be observed in either group of subjects. No correlation between the plasma CA and serum $TES_{\text{tot}}$ and $TES_{\text{free}}$ concentration responses were also observed in either group, or even if the two groups were combined.

In women the $TES_{\text{tot}}$ and $TES_{\text{free}}$ levels were significantly lower than in men in the knee-extension exercise session (exp. 4) at rest and before, as well as after the submaximal and maximal parts of the exercise, and no increases were observed in response to the exercise (Fig. 11). After the correction for the $\Delta PV\%$, which tended to be larger in men than in women [after $E_{\text{submax}}$: -11.9 (SD 3.8) % vs. -4.1 (SD 4.5) %, $P < 0.01$ and after $E_{\text{max}}$: -14.6 (SD 5.2) % vs. -7.1 (SD 3.1) %, ns], no gender differences in $TES_{\text{tot}}$ changes from pre- to postexercise were observed. No correlations between plasma CA and serum $TES_{\text{tot}}$ and $TES_{\text{free}}$ responses were observed in women.
FIGURE 11 Mean and SD serum TES$_{tot}$, TES$_{free}$ and SHBG concentrations in boys (n = 6), men (n = 6) and women (n = 6) in the morning (REST), before (PRE) and after the submaximal (E$_{submax}$) and maximal (E$_{max}$ and E$_{max2}$) parts of the knee-extension exercise. Significantly different from pre-exercise $^a$ P < 0.05 and $^b$ P < 0.01. Significantly different from previous value $^c$ P < 0.05. Significant difference between the groups $^*P$ < 0.05 (statistical powers 0.20-0.82).

The postexercise COR or GH levels in half-squatting did not show any differences between men and boys (exp. 1), although the COR concentration increase in boys reached the level of statistical significance in every exercise session, and in men only in the most exhausting exercise (E3) (Fig. 12). No differences between the groups were also observed in the increase of these hormones from preexercise. Also in the knee-extension exercise (exp. 4) no differences in the postexercise serum COR or GH levels were observed between the groups, but the COR concentration increased significantly in response to the exercise only in boys (Fig. 13). The COR concentration increase in boys remained significant after the correction for ΔPV%. 

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FIGURE 12 Mean and SD serum COR and GH concentrations in the morning (REST), before (PRE) and after (POST) four half-squatting exercise units in young (Boys, n = 7) and adult male athletes (Men, n = 7). Significantly different from preexercise P < 0.05. Significant difference between the groups P < 0.05 and P < 0.01, respectively (statistical powers 0.66-0.76 for COR and 0.25 for GH). Previously unpublished data.

FIGURE 13 Mean and SD serum COR and GH concentrations in boys (n = 6), men (n = 6) and women (n = 6) in the morning (REST), before (PRE) and after the submaximal (E_{submax}) and maximal (E_{max1} and E_{max2}) parts of the knee-extension exercise. Significantly different from pre-exercise P < 0.05. Significantly different from previous value P < 0.05 and P < 0.01.
No gender differences in the GH or COR levels could be observed at any point of the knee-extension exercise session (Fig. 13). No correlations between plasma CA and COR or GH responses were also observed even if the two groups were combined. In boys plasma A and serum COR correlated positively in the half-squatting exercise session E3 (Fig. 14), and plasma NA and serum GH in the knee-extension exercise (Fig. 15).

**FIGURE 14** Relationship between the concentration of plasma A and serum COR after the half-squatting exercise session E3 in boys.

**FIGURE 15** Relationship between the concentration of plasma NA and serum GH after the knee-extension exercise in boys.

**FIGURE 16** Mean and SD serum COR and GH concentrations in the morning (REST), before (PRE) and after the submaximal (E_submax) and maximal (E_max1 and E_max2) parts of the two knee-extension exercises (E1 and E2). Significantly different from pre-exercise *P < 0.05. Significantly different from previous value †P < 0.05. Significant difference between the two exercises * P < 0.05 and ** P < 0.01 (statistical powers 0.75-0.95).
In experiment 5 the serum GH and COR concentrations were significantly lower in the second exercise than in the first one, both after the submaximal and maximal parts of the exercise (Fig. 16). However, the serum TES$_{tot}$, TES$_{free}$ and SHBG concentrations were similar in these two exercises performed with one day of rest in between (Fig. 17). After the correction for the $\Delta$PV% no differences in any of the hormonal responses were observed between the two exercise sessions in experiment 5. No correlations between the plasma CA and other hormonal responses were also observed in either session, or even if the two exercise sessions were combined.

**FIGURE 17** Mean and SD serum TES$_{tot}$, TES$_{free}$ and SHBG concentrations in the morning (REST), before (PRE) and after the submaximal (E$_{submax}$) and maximal (E$_{max}$) parts of the two knee-extension exercises (E1 and E2). Significantly different from pre-exercise $^a$ $P < 0.05$, $^b$ $P < 0.01$ and $^c$ $P < 0.001$. Significantly different from previous value $^d$ $P < 0.05$ and $^e$ $P < 0.01$. 
5.6 Plasma catecholamine responses in relation to neuromuscular performance

Although the fatigue index, B-La levels and the plasma CA responses all tended to be higher in the half-squatting exercise unit E3 than in the other exercises the only correlations found were those in boys between the postexercise concentrations of B-La and plasma NA (r = .86, P < 0.05) and A (r = .81, P < 0.05) in E3 (exp. 1). The performance of the subjects (1-RM, W_{tot}, P_{max}, P_{peak}) and fatigue (fatigue index) did not correlate with the plasma CA concentration responses in any of the exercise units in either group, or even if the groups and/or exercise sessions were combined.

In the four knee-extension tests the performance of the subjects (1-RM, W_{tot}, P_{mean}, iEMG_{tot}, aEMG_{exce}, and B-La responses did not correlate with the plasma CA concentration responses in any of the tests in either group, or even if the groups were combined (exp. 2). However, the plasma ΔNA correlated both in men and in women with the ΔaEMG:load ratio during the longest exercise test (E20, Fig. 18).

In experiments 3, 4 and 5 no correlations between the indicators of muscle activity, muscle fatigue or B-La response and the plasma CA concentration response were observed. However, those subjects in experiment 3 who had the highest strength gain (highest increase in preexercise MVC) during the training period tended also to have an increase in the postexercise plasma A concentration (Fig. 19).

![Figure 18](image1.png)  ![Figure 19](image2.png)

**FIGURE 18** Relationship between the ΔaEMG:load and the ΔNA in men (filled circles, n = 9) and women (open circles, n = 8) in the knee-extension exercise E20.

**FIGURE 19** Relationship between the change in the preexercise MVC (ΔMVC_{pre}) during five knee-extension exercise sessions and the corresponding change in the postexercise plasma adrenaline concentration (ΔA_{post}).
6 DISCUSSION

6.1 Differences in the plasma catecholamine responses between men and women

Previous studies that have examined gender differences in the plasma CA response to physical exercise have shown contradictory results. No difference in endurance exercise has been found in some studies (Favier et al. 1983, Friedmann & Kindermann 1989), but the plasma CA response in women has also been reported to be somewhat lower (McMurray et al. 1987, Tarnopolsky et al. 1990) or higher than in men (Lehmann et al. 1986). In short-term anaerobic exercise the plasma A response has been reported to be lower in women than in men, while no differences were observed in the NA response (Brooks et al. 1990, Gratas-Delamarche et al. 1994). In addition to differences in exercise duration and intensity these contradictory result may be accounted for by differences in menstrual cycle phase of the female subjects and, at least in part, by different training status of the male and female subjects. Different training status in men and women may also not be excluded in the present study, although the subjects were selected carefully to match with their physical activity level.

In the present study the plasma NA responses to the four knee-extension exercise tests were found to be equally high in women and men. Also, on the contrary to the hypothesis, the plasma A response was not lower in women than in men in these short-term anaerobic exercise bouts (Fig. 4). However, in the knee-extension exercise consisting of multiple sets of repetitions the plasma A concentration was slightly but significantly (with statistical power of only 0.20) lower in women than in men after the last two (REP₉₉) sets (Fig. 5). As the plasma NA concentrations were similar in this exercise in the two groups, the NA:A ratios were higher in men throughout the exercise (statistical powers 0.65-0.75). Earlier lower plasma A concentration in women than in men after maximal short-term exercise has also been observed in two studies. Brooks and co-workers (1990) found lower plasma A concentration in physically active
women than in men after maximal repetitive treadmill sprints, although the NA concentrations were identical (Brooks et al. 1990). The plasma A concentration was also found to be lower in trained female sprinters than in male sprinters after a 30s Wingate test, while again no gender differences were observed in the NA concentrations (Gratas-Delamarche et al. 1994). The present results from the knee-extension exercise (exp. 4) therefore tend to agree with these data. However, as the ΔA from preexercise to the end of the exercise, or to the individual peak concentration, was not different between men and women, the results may not suggest a lower A secretion in response to this type of exercise in women than in men. In experiment 2 the plasma A response in E40 and E60 tended actually to be even somewhat higher in women than in men, although not significantly so (Fig. 5).

The fact that the experimental testing of the female subjects in experiment 4 was timed to the early follicular phase of the menstrual cycle may have a role in diminishing the gender difference with regard to the plasma A response. It has been shown that the exercise-induced plasma A increase may be higher in women in the follicular phase rather than in the luteal phase of the menstrual cycle (Sutton et al. 1980). Nevertheless, the results from the experiment 2, where the menstrual cycle phase was not controlled, may also not suggest any systematic gender difference in the plasma CA response to the resistance exercises used in the present study. It is also of interest that in the above mentioned studies, where lower A response to maximal short-term exercise in women than in men has been observed, the power output referred to LBM was also significantly lower in the former than in the latter (Brooks et al. 1990, Gratas-Delamarche et al. 1994). However, in the present experiments no gender differences were observed either in $P_{\text{mean}}$ or $W_{\text{tot}}$ relative to LBM (Tables 9 and 10), which may well be one reason for the lack of gender difference in the A response to these exercises.

6.2 Differences in the plasma catecholamine responses between men and boys

As expected, lower NA response in boys than in men could be observed in the present experiments. The postexercise plasma NA concentration after the most exhausting half-squatting exercise session (E3, Fig. 6), as well as also the ΔNA from pre- to postexercise, was about 50% lower in boys than in men (statistical powers 0.65 and 0.55). Although the $P_{\text{mean}}$ relative to body weight was lower in the boys than in men in half-squatting exercise session E3, differences in the relative exercise intensity between men and boys were probably minor, as the fatigue index and B-La response were not significantly different in these two groups (Table 8). Furthermore, the longer experience from strength training in men than in boys, may also not necessarily explain the difference in the NA response between the two groups. Regular strength training is known to reduce the exercise-induced plasma A response, while causing no alterations in those
of NA (Guezenne et al. 1986, Lehmann & Keul 1986). However, on the contrary to these results, both the plasma NA and A concentrations after an exhausting leg press exercise have also been found to be higher in trained powerlifters than in untrained men (Kraemer et al. 1999). Therefore, it is obvious that at least part of the observed difference in the plasma NA response may be related to the age difference.

In general older adult individuals show a higher plasma NA response to any relative intensity of work than younger ones (Fleg et al. 1985, Lehmann & Keul 1986, Mazzeo et al. 1997, Zouhal et al. 1999). This higher plasma NA increase may result from enhanced SNS activity (Lehmann et al. 1986, Mazzeo & Grantham 1989) and/or from a reduction in NA clearance (Mazzeo et al. 1997). Thus, the higher plasma NA response in men than in boys might reflect increased NA secretion, but it may also be explained by a declined NA removal from circulation. If there was a difference in the NA removal between the two groups in experiment 1, its contribution to the observed difference in the plasma NA response between the boys and men was possibly substantial, as the postexercise samples were taken 3 min after the cessation of the exercise.

Earlier the plasma CA response has been compared between adult men and adolescent boys (12.8 ± 0.8 years) in an exhausting incremental treadmill exercise (Lehmann et al. 1981). The plasma CA concentrations were apparently very similar in the two groups of subjects at identical submaximal relative exercise loads (% VO₂max). However, the plasma NA concentration after exhaustion was significantly lower (30%) in adolescent boys (12.8 ± 0.8 years) than in men, while no differences in the A concentrations were observed between the groups. These authors suggested a reduced maximal sympathetic nervous activity in the adolescent as compared to the adults (Lehmann et al. 1981). The data from the experiment 1 in the present study are in good agreement with these results. No differences in the postexercise plasma CA concentrations were observed between the groups in the lighter exercise sessions (Fig. 6), but after the exhausting E3 the NA response was lower in boys than in men. Also after the submaximal part of the knee-extension exercise the NA concentrations were similar in men and boys (Fig. 5). However, no differences between the groups in the NA concentrations were also observed after the exhausting REPₘₐₓ sets in that exercise. Nevertheless, it is possible that due to the smaller active muscle mass, lower repetition rate and postural difference the knee-extension exercise did not activate the SNS as strongly as the half-squatting exercise E3.

Studies where children alone have been investigated have also suggested differences in the exercise-induced plasma CA responses between children and adults. Lehmann and coworkers (1980) suggested that despite a lower NA response the plasma A response to maximal short-term exercise (300 m run) is higher in adolescent boys (12.6 ± 1 years) than in adult men. It has also been suggested that during a 60 min cycle exercise (60 % VO₂max) the A response in relation to the NA response is higher in prepubertal boys (8.5 - 11 years) than in adults (Delamarche et al. 1992). In the present study, in spite of the similar plasma NA responses there were some differences in the plasma A concentration responses between men and boys in the knee-extension exercise
(Fig. 5). The plasma A concentration did not increase significantly in either group in the submaximal part of the exercise. However, the plasma ΔA from preexercise after the last two sets was significantly higher in boys than in men, and also the peak plasma A increase from preexercise was about twice as high in boys as in men (statistical powers 0.30-0.35). Although the variation in A concentrations in boys was large the results might suggest a higher responsiveness of the adrenal medulla to same sympathetic input in the boys than in the adults in response to this particular exercise. However, no differences between men and boys were also observed in the plasma A responses in any of the half-squatting exercises (Fig. 6). In two exercises (E2 and E4) the postexercise plasma A concentration tended to be higher in boys than in men, although not significantly so. Nevertheless, as blood was sampled in experiment 1 only before and 3 min after the exercises the peak plasma A concentrations were probably missed. It could clearly be seen in experiment 4 that the peak plasma A concentrations may be found already during the exercise consisting of multiple sets rather than after it.

Although different blood sampling methods were used in the experiments 1 and 4 (conventional vs. arterialised venous), it is difficult to see how these different methods could explain the somewhat controversial results in these two studies. Therefore, it may be suggested that in general no differences in sympathoadrenal response to resistance exercise may be expected between men and boys. However, the results may also indicate that in very exhaustive high-intensity resistance exercise, which strongly activates the SNS, the plasma NA concentration increase may be lower in pubescent boys than in adult men, suggesting reduced maximal sympathetic nervous activity in the former. The results may also suggest a higher plasma A response to certain resistance exercises in pubescent boys than in adult men.

6.3 Plasma catecholamine responses to repeated resistance exercise sessions in men

The limited data on the effects of resistance training on the plasma CA response to exercise is controversial. Guezennec et al. (1986) have reported that regular strength training of four months reduces resistance exercise induced plasma A responses, while causing no alterations in the responses of NA. Lehmann and Keul (1986) have also reported exceptionally low NA related A responses in weight lifters as compared to endurance-trained subjects and control subjects after exhaustive incremental cycling. The plasma CA response has also been reported to be similar in well-trained weightlifters and untrained men (McMillan et al. 1993), and recently both the plasma NA and A concentrations after an exhausting leg press exercise were found to be higher in powerlifters than in untrained men (Kraemer et al. 1999).

In the present study the plasma CA responses in the early phases of a resistance-training program were examined. As expected a decline in the
plasma A response was observed, but it was clearly not related to the development of the neuromuscular performance. In experiment 3 a decreased A response was observed after only one resistance exercise session, while no change in the NA response during the five exercise sessions was found (Fig. 7). Experiment 5 confirmed this pattern. The NA responses were similar in the two successive exercises, but the postexercise A concentration was again significantly lower in E2 than in E1 (Fig 8). The statistical power of this result was also relatively high in both experiments (0.73-0.83). However, as the individual peak A concentrations, observed in most of the subjects already after the sixth set, were not different between the two exercises, the results may not suggest a decrease in the responsiveness of the adrenal medulla to sympathetic input in the second exercise (Exp. 5). As blood was sampled only before and after the exercises in experiment 3, the results do not also necessarily mean that there was a decline in the peak plasma A concentration in the second exercise. The peak concentrations may have occurred already during the exercise rather than after it, as was the case in most of the subjects in experiment 5. In addition, as conventional blood sampling from antecubital vein was employed in experiment 3, there is a theoretical possibility that changes in the forearm extraction of A could also explain, at least in part, the different plasma A responses observed between the first and second exercise. However, since the extraction of A mimics that of NA quite closely (Hjemdahl 1993), and the NA responses were similar in the first and second exercise in experiment 3, decreased A secretion from the adrenal medulla cannot be totally ruled out.

The mechanism for the possible reduction in A secretion is unclear. According to Frankenhaeuser et al. (1976) plasma A is a sensitive indicator of mental stress, while the threshold for plasma NA response is higher. However, it is difficult to consider that lower emotional stress could explain the lower A response in the second exercise than in the first one in the experiment 3. On the contrary, it could even be expected that the mental stress would have been higher in the second exercise than in the first one taking into account that the exercising muscles still tended to be somewhat sore in the second exercise. Nevertheless, as the secretion of the CA is known to be stimulated by cytokines released from the injured tissue (Madden & Felten 1995), it might be suggested that the stimulus for CA release is higher in an acute muscle damaging exercise than in an exercise where the process of inflammation in the exercising muscles is already operative. It has been shown earlier that already one training session may provide a protective effect for further muscle damage, even if the second exercise is performed before full recovery and restoration of muscle function (Ebbeling & Clarkson 1990). Furthermore, it is also possible that there is a switch in the primary secretory product of the adrenal medulla during the process of inflammation. It has been recently shown that the secretion of proenkephalin peptide F may increase dramatically during the recovery as late as 4 h after heavy resistance exercise (Bush et al. 1999). These authors also suggested that there is a potential role for the peptide F to enhance the immune system and therefore the recovery process of damaged muscle. However, no data is available on the peptide F concentrations during later recovery following a heavy resistance exercise.
In general, the results of the present study may not suggest any significant alterations in the plasma CA response to resistance exercise in the early phases of a resistance training period. The postexercise A concentration, however, may be lower already in the second exercise session than in the first one, but no difference in the peak plasma A concentration may necessarily not be seen.

6.4 Other hormonal responses and their correlation with plasma catecholamines

The postexercise serum TES concentrations tended to be lower in boys than in men in the present experiments (Fig. 9 and 11). The serum testosterone concentration may be potentially influenced by sympathetic stimulation in male subjects (Eik-Nes 1969, Jezova and Vigas 1981), but no correlations could be observed in the present experiments between the plasma CA and serum TES concentrations either in boys or in men. Only by combining the two groups a weak (but statistically artificial) correlation between ΔNA and ΔTES

\[ \text{f1}\] could be observed in the half-squatting exercise session E3 (Fig. 10). It is, however, obvious that also this correlation might have disappeared if the ΔTES

\[ \text{f1}\] could have been corrected for the ΔPV%.

Although the ΔPV% was not determined in the half-squatting exercises the increase in the SHBG concentration during the exercises varied in men between 8 and 22 %, and in boys between 7 and 14 %, which may reflect quite well the magnitude of the plasma volume shift (Kargotich et al. 1997). Thus the increases in TES

\[ \text{f1}\] (men 15-36 %, boys 13-16 %) may be explained to large extent by the changes in plasma volume, and it is therefore not surprising that no correlation with plasma CA could be observed. However, the increases in TES

\[ \text{f1}\] concentrations (men 19-30 %, boys 11-14 %) cannot be explained by haemoconcentration, as TES

\[ \text{f1}\] is not bound to proteins and may move freely from the vascular space together with fluid. Nevertheless, no correlation between the plasma CA and serum TES

\[ \text{f1}\] responses could be found in either group of subjects, even if the two groups were combined. In experiments 4 and 5 the ΔPV% was of anticipated magnitude (Collins et al. 1986, 1989), and it could totally explain the TES

\[ \text{f1}\] concentration increases. However, no correlations between the plasma CA and serum TES

\[ \text{f1}\] could also be observed.

The lack of correlation between the plasma CA and serum TES responses do not exclude a role for the sympathetic stimulation in increasing the serum TES secretion from the testicles during resistance exercise. On the other hand, if such correlation could have been found in the present experiments, it would also not mean that the relationship is causal. The present results simply indicate that no obvious parallelism between CA and TES responses to these resistance exercises existed.

The postexercise serum COR concentrations were not significantly different between the boys adults in the present experiments (Fig. 12 and 13). However, the COR increase was significant in boys after every half-squatting
exercise session, while it reached the level of statistical significance in men only after the most exhausting exercise session (E3). In the knee-extension exercise the increase in COR concentration after the exercise was significant only in boys, and remained significant also after correction for the ΔPV%. The higher COR response, relative to the preexercise concentration, in boys than in the adult men may reflect stronger stress response in the former, and would be also well in line with the higher A response already discussed above. Although the sympathoadrenal activity may stimulate COR release from the adrenal cortex (Al-Damluji 1993, Berne & Levy 1993, Chouros 1995, Kuoppasalmi et al. 1981), no obvious parallelism between the CA and COR responses in the present study were found. However, after the most exhausting half-squatting exercise session (E3) a positive correlation between A and COR concentrations was observed in boys (Fig. 14). Nevertheless, this correlation does not necessarily indicate a causal relationship between the plasma A and COR release. There is indeed data showing that physiologic elevations of circulating CA have no effect on the pituitary-controlled COR release, obviously due to their inability to cross the blood/brain-barrier (Oberbeck et al. 1996).

Earlier the COR response to resistance exercise has been shown to be dependent on the magnitude of the exercise stress, and significant increases in adult men (Guezennec et al. 1986; Häkkinen and Pakarinen 1993; Kraemer et al. 1987) and women (Kraemer et al. 1993) as well as in boys (Fry et al. 1993, Kraemer et al. 1992) have been demonstrated. The present results may, however, suggest that a lower exercise stimulus is sufficient to increase the COR concentrations in boys than in adult men. It is also of interest, that both A and COR, that seemed to increase somewhat stronger in the boys than in the adult men in the knee-extension exercise, have effects that may decrease the inflammatory muscle tissue damage (Smith 1997). In line with this, muscle soreness, usually associated with the muscle damage (Smith 1991), was clearly lower and occurred earlier in boys than in the adult subjects in experiment 4. However, it has also been shown previously that muscle soreness ratings in adolescent boys after exhausting resistance exercise may be lower than in adult men (Soares et al. 1996).

The GH secretion from the anterior pituitary gland may be stimulated by the activation of the α2-adrenoceptors in the brain both by increasing the secretion of the growth hormone releasing hormone and by inhibiting the secretion of somatostatin (Al-Damluji 1993). Previously the serum GH concentration has been shown to increase in response to resistance exercise both in adult men and women (e.g. Häkkinen & Pakarinen 1995, Kraemer et al. 1991), as well as in adolescent boys (Fry et al. 1993, Kraemer et al. 1992). In the present experiments, with identical relative loading in each group, the serum GH concentrations before, during and after the exercises, as well as the GH responses corrected for the ΔPV% were not different between the groups. There was, however, a trend for higher postexercise serum GH concentrations in boys than in adults (Fig. 12 and 13). In line with this, it has been recently shown that the exercise-induced GH response may be higher in children than in adults in endurance exercise (Cappa et al. 2000). No obvious parallelism between the CA and GH responses in the present study could be observed. However, in the
knee extension exercise a correlation between the NA and GH response was found (Fig. 15). Nevertheless, this interrelationship does probably not reflect any causal relationship between the sympathoadrenal activity and GH release, as the ΔPV% could totally explain the GH concentration increase also in boys. However, the GH concentration may have still elevated after the cessation of the exercise. Earlier high sympathoadrenal response and elevated GH response to exercise have been linked by several investigators (e.g. Chwalbinska-Moneta et al. 1996, Kjaer et al. 1987, Kozlowski et al. 1983, Weltman et al. 1997).

It is of interest that in experiment 5 both the serum COR and GH concentrations clearly were lower in the second exercise than in the first one (Fig. 16, statistical powers 0.75-0.95). The trend for the declined GH and COR concentrations already in the preexercise samples in the second exercise is difficult to explain. However, the process of muscle damage might be associated with these lower concentrations. As the resting concentrations of these hormones in the morning were similar before the two exercises and the preexercise samples were drawn 12 min after the cessation of the measurements of 1-RM and MVC it is possible that the influence of these measurements was decreased, although the possible mechanism is unclear. It has been suggested that the COR concentration at rest may be even increased when the leg muscles are sore due to the stress associated with normal ambulatory movements (Gleeson et al. 1995). Furthermore, it is also difficult to say whether the observed decline had any physiological influence. In the case of GH it must be kept in mind that multiple variants of GH exist in the circulation (Baumann 1991), and that different variants may respond differently to exercise stimulus (McCall et al. 1997). In the present experiments only the immunoassayable form of GH could be measured. Nevertheless, it is obvious that the stimulus for the increase of these hormone concentrations in the second exercise was not decreased by a possible muscle damage protective effect of the first exercise, which is known to exist even if the second exercise is performed before full recovery and restoration of muscle function (Ebbeling & Clarkson 1990). In experiment 5 the exercise-induced increase in GH concentration from preexercise was similar in E1 and E2 and could totally be explained by the plasma volume shift, and no increase in COR concentration was observed in either exercise. However, it has been shown earlier that the GH concentration response may be higher in resistance exercise with muscle damage than without it (Kim 1998).

The reason for the slight but significant blood GLU concentration decrease in E2 is unclear. Nevertheless, it is of interest that both COR and GH are involved in the blood GLU regulation during exercise (McArdle et al. 1996). No decrease in the blood GLU concentration during resistance exercise has been observed earlier (e.g. Kraemer et al. 1991, Tesch et al. 1986).
6.5 Correlation of the plasma catecholamine responses with the neuromuscular performance

In general the present results showed that the plasma CA increase tends to be highest in resistance exercises which strongly elevate the B-La concentration. The results from the experiment 1 also suggest that the glycolytic contribution to energy supply is more closely related to the magnitude of plasma CA response in resistance exercise than the $P_{\text{mean}}$ (Fig. 6, Table 8). The CA increase was clearly highest in half-squatting exercise session E3, although the $P_{\text{mean}}$ tended to be lower in that exercise than in the E1 and E2. Therefore, the organisation of the sets, and recovery periods so that high B-La concentrations may be expected obviously also leads to high plasma CA response. The results from the $\text{REP}_{\text{max}}$ sets are also in line with this view (Fig. 4, Table 9).

Earlier in different maximal treadmill and ergometer sprint exercises the plasma CA response has been shown to correlate to the postexercise blood lactate concentration (Brooks et al. 1990, Cheetham et al. 1986), to the plasma lactate increment (Strobel et al. 1999), to the postexercise muscle lactate concentration (Cheetham et al. 1986), to the estimated energy production from anaerobic sources (Cheetham et al. 1986, Nevill et al. 1989), to the postexercise blood pH (Cheetham et al. 1986), to the accumulated oxygen deficit (Strobel et al. 1999) and to the peak power output (Nevill et al. 1989). Although the fatigue index, B-La concentrations and the plasma CA responses all tended to be higher in the half-squatting exercise unit E3 than in the other exercises the only correlations found were those in boys between the postexercise concentrations of B-La and plasma NA ($r = .86, P < 0.05$) and A ($r = .81, P < 0.05$) in E3. These correlations may well reflect an influence of local metabolic changes in exercising muscles on the SNS activation, and/or a stimulatory effect of plasma CA on muscle glycolysis (Kjaer 1989). No other correlations could be observed even if the groups and/or exercise sessions were combined.

The lack of correlations between the neuromuscular performance and the plasma CA responses was evident also in the other experiments in the present study. However, the plasma $\Delta$NA from preexercise correlated both in men and in women with the $\Delta$EMG:$\text{load}$ ratio during the longest knee-extension test (Exp. 2, Fig. 18). It has been shown that during dynamic exercise the contracting skeletal muscles may contribute to a larger extent than resting skeletal skeletal muscles to increasing the concentration of plasma NA (Savard et al. 1987). It has also been shown that increase in the activity of the motor centers in the brain may directly increase sympathoadrenal secretion. By using tubocurarine, which weakens skeletal muscle and increases the motor command necessary to produce a certain work output, increased plasma CA responses at a given work load have been reported (Kjaer et al. 1987). Thus the significant positive correlations, observed in the present study, between the $\Delta$EMG:$\text{load}$ ratio and $\Delta$NA in E20 might reflect, at least in part, the effect of an increased motor command on the sympathetic nervous activity. However, no such correlations were observed in any other experiments in the present study.
Lehmann and Keul (1986) suggested that the low plasma A responses in weight-lifters after exhaustive incremental cycling as compared to endurance-trained subjects and control subjects might be associated with a reduced exercise induced strain due to the increased muscle power. On the contrary to this the results of present study indicated that during the short-term resistance training period those subjects who had the highest strength gain tended actually to have an increase in the postexercise plasma A concentration (Fig. 19). However, as the mean postexercise plasma A concentration was not different between the first and last exercise (Fig. 7) this correlation may not be seen to reflect any positive influence of A on maximal force production.

6.6 Perspective

The results of this study clearly indicated that differences in plasma CA response to resistance exercise between adult men and adolescent boys may exist. In order to more precisely describe these differences, further studies should utilise exercise arrangements that activate the SNS very heavily. The relationship between the resistance exercise-induced muscle damage and the response of the plasma CA and other hormones that have anti-inflammatory effects should also be studied in children and adults. A project is in progress to examine these questions.
The main findings and conclusions of the present study can be summarised as follows:

1) The results of the present study did not indicate any differences between adult men and women in the plasma NA response to resistance exercise. Also, on the contrary to the hypothesis of this study, the plasma A response was not lower in women than in men. The results may thus suggest that the activation of the SNS and the release of A from the adrenal medulla in response to resistance exercise is similar in men and women.

2) In general no differences in sympathoadrenal activity during resistance exercises could be found between men and boys. However, as expected the results indicated that in very exhaustive high-intensity resistance exercise the plasma NA concentration increase may be lower in pubescent boys than in adult men, suggesting reduced maximal sympathetic nervous activity in the former. The results also suggested higher plasma A response to one of the resistance exercises in pubescent boys than in adult men.

3) The plasma NA response remained unaltered in the early phase of a resistance-training period. Although the postexercise plasma concentrations of A tended to be lower already in the second exercise than in the first one, no changes in the peak plasma A increase from preexercise may perhaps also be expected during the first training sessions of a resistance training program.

4) The present results did not suggest any correlation between the plasma CA responses and serum TES secretion during resistance exercises. Also the correlations of plasma CA responses with serum COR and GH responses were weak. The lack of correlations, however, do not
necessarily mean that the sympathoadrenal system would not influence the secretion of these hormones during resistance exercise.

5) No consistent correlations between the plasma CA responses and the neuromuscular performance of the subjects were observed. However, it is obvious that the plasma catecholamine response is high when the exercise intensity, sets, and recovery periods are organised so that high blood lactate response may also be expected.
Sympaattisen hermoston aktivoituminen on merkittävässä roolissa verenkier-ron, aineenvaihdunnan ja hormonitoiminnan adaptointumisessa fyysiseen stressiin. Sympaattisen hermoston aktivoitumisessa vapautuu verenkiertoon postgang- lionaarisiasta hermopäätteistä huomattavia määriä välittäjäaine noradrenaliinia, joka toimii näin ollen myös hormonina. Ko. hermosto sätelee pitkästi myös hermottamansa lisämunuaisiytimen adrenaliinin eritystä. Mittaamalla katekolamiinejä, noradrenaliinia ja adrenaliinia, pitoisuutta veriplasmassa voidaan varsin hyvin arvioida ns. sympatoadrenaliinisen aktiivisuuden voimakkuutta stressitilanteessa.


Lyhyen voimaharjoitteljakson alkuvaiheessa plasman noradrenaliini-vaste sääliytyi muuttumattomana, mutta harjoituksen jälkeinen adrenaliinipitoisuus pyrki olemaan jo toisessa harjoituksessa matalampi kuin ensimmäisessä. Ero saattaa kuitenkin selittäyä adrenaliinipitoisuuden huipun ajoittumisella aikaisemmassa.


Tässä tutkimuksessa ei yleisesti tarkastella merkittäviä systemaattisia eroja plasman katekolamiinivasteissa voimaharjoituskuormituksen tutkittujen ryhmien välillä havaittu. Havaittujen pienet erojen merkitsivät miesten ja puberteetti-ikäisten poikien välillä olisi jatkotutkimuksissa syytyä selvittää.
REFERENCES


