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β-Endorphin, adrenocorticotropic hormone, cortisol and catecholamines during aerobic and anaerobic exercise

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Summary. Twelve non-specifically trained volunteers (aged 26.5 years, SD 3.6) performed exhausting incremental graded exercise (ST) and 1-min anaerobic cycle ergometer exercise (AnT) at 2-h intervals for the purpose of investigating β-endorphin (β-E) behaviour dependent on exercise intensity and anaerobic metabolism. In order to determine [β-E], adrenocorticotropic hormone [ACTH], cortisol [C], adrenaline [A] and noradrenaline [NA] concentrations, venous blood samples were collected prior and subsequent to exercise until the 20th min of the recovery period, as well as in ST before and after exceeding the individual anaerobic threshold ($TH_{an,i}$). Before, during and after ST, lactate concentration, heart rate and perceived degree of exertion were also determined; after AnT maximum lactate concentration was measured. Both types of exercise led to significant increases in [β-E], [ACTH], [A] and [NA], with levels of [β-E] and [ACTH] approximately twice as high after ST as after AnT. The [C] increased significantly only after ST. During ST significant changes in [β-E] and [ACTH] were measured only after exceeding $TH_{an,i}$. At all measuring times before and after ST and AnT both hormones correlated positively. In AnT the increases of [β-E] and [A] demonstrated a correlation ($r=0.65$; $P<0.05$). Both in AnT and ST there was a relationship between the maximum concentrations of β-E and lactate ($r=0.63$ and 0.71; each $P<0.05$). We therefore conclude that physical exercise with increasing or mostly anaerobic components leads to an increase in [β-E], the extent correlating with the degree of lactate concentration. The similar behaviour of [β-E] and [A] after AnT might suggest mutual influences of endogenous opioids and catecholamines.

Key words: β-Endorphin – Stress hormones – Cycle exercise – Anaerobic threshold

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Introduction

After the discovery of endogenous opioids (Hughes et al. 1975), which demonstrated that β-endorphin (β-E) particularly revealed potent morphine-like activity (Li 1977), in several subsequent studies the dependence of exercise-induced β-E-increases upon exercise intensity was investigated (Farrell et al. 1982; De Meirleir et al. 1986; Donevan and Andrew 1987; Goldfarb et al. 1987). The findings were in part contradictory, especially with reference to the minimum intensity necessary for β-E secretion. The conflicting findings might be due partly to methodology. Since the exercise intensities were set as percentages of the maximum oxygen uptake ($\dot{V}O_{2max}$), different procedures in the ergometric tests and different peaks of exertion make comparisons difficult.

The aim of the present study was to investigate the behaviour of β-E as dependent upon energy yield during incremental graded cycle exercise. We hypothesized that an increase in β-E concentration [β-E] does not occur until the individual anaerobic threshold ($TH_{an,i}$) has been exceeded, this being a measurement of the overproportionate increase in lactate concentration. In addition to performance or oxygen uptake ($\dot{V}O_2$), therefore, lactate concentration was also measured as an intensity-related metabolic parameter. Moreover, there are indications that the extent of anaerobic energy yield correlates with the behaviour of β-E (De Meirleir et al. 1986; Mougin et al. 1987). This presumed correlation was also investigated during supramaximal exercise with primarily anaerobic energy yield. Both tests were performed as a double load with a 2-h interval in order to examine the restitution of [β-E]. In addition, concentrations of adrenocorticotropic hormone [ACTH], cortisol [C], adrenaline [A] and noradrenaline [NA] were determined in order to obtain information about possible hormonal interactions, since there was evidence of contradictory findings in this connection (Berkenbosch et al. 1981; Dearman and Francis 1983; Farrell et al. 1983).

Table 1. Anthropometric characteristics and parameters of physical performance capacity [maximal values and values corresponding to individual anaerobic threshold ($TH_{an,i}$)] in incremental graded exercise

	Age (years)	Height (cm)	Mass (kg)	$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Maximal exercise intensity ($W \cdot kg^{-1}$)	$\dot{V}O_2$, $TH_{an,i}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	Exercise intensity, $TH_{an,i}$ ($W \cdot kg^{-1}$)
Mean	26.5	181.2	73.5	53.8	4.29	37.4	2.88
SD	2.9	6.5	6.0	6.0	0.53	6.5	0.59

$\dot{V}O_{2max}$, Maximal oxygen uptake; $\dot{V}O_2$, oxygen uptake

Methods

Subjects. Twelve healthy male, non-specifically trained volunteers took part in the study, performing basically endurance-oriented exercise lasting 3–5 h, weekly. Part of the training programme consisted in cycling; a few of the subjects had already taken part in short triathlon events. Anthropometric and physiological data are presented in Table 1. A routine physical examination, ECG at rest, blood pressure at rest and routine laboratory status revealed no pathological findings. All of the exercises were conducted in the morning in a sitting position on an electrically-braked cycle ergometer 2 h after a standard breakfast.

Incremental graded exercise. First, all of the subjects underwent incremental graded exercise (step test = ST; start at 50 W, after every 3 min an increase of 50 W until volitional exhaustion) to accustom the subjects to test conditions. They also performed a second ST after an interval of several days, in order to determine individual anaerobic threshold ($TH_{an,i}$) (Stegmann et al. 1981). Also after an interval of several more days, a third ST was performed to measure hormone levels. At 30 min prior to this test, a catheter was inserted into a cubital vein; [β-E], [ACTH], [C], [A] and [NA] were determinated immediately before the onset of exercise, at the end of the step just prior to the $TH_{an,i}$, at the end of the step directly after $TH_{an,i}$, upon completing exercise and in the 5th and 10th min of the recovery period. At rest, at the end of each level of exercise and repeatedly in the post-exercise period, capillary samples were drawn from the earlobe, to measure lactate concentrations. At the same time, heart rate and the rate of perceived exertion were also ascertained.

Anaerobic test. The 1-min anaerobic test (AnT) was performed at 2-h intervals. The test objective – maximal power output within 60 s – was agreed with the subjects and practised for a few days beforehand. During AnT, intermediate times were called every 10 s. In contrast to ST, the ergometer in AnT calculated performance according to the specific number of revolutions. The individual setting at 120 pedal rpm was 1.5 times the maximal performance achieved in ST. Electronic assessments of the performance in AnT were made at 0.5-s intervals, depending upon the given number of revolutions and upon the performance set for 120 rpm. Venous blood samples to determine the above-mentioned hormones were collected prior to, directly after (a 2-ml syringe was fixed on the venous catheter, blood was drawn simultaneously with breaking-off exercise), and 5 and 10 min after exercise. One subject did not undergo a hormonal analysis for [β-E] and [ACTH]. Lactate concentration was measured before, directly after and repeatedly during the recovery phase until 20 min after the cessation of exercise.

Lactate concentrations were determined enzymatically in whole blood (Hohorst 1962). The concentrations of β-E, ACTH and C were ascertained radio-immunologically in serum ([β-E], [C]) or plasma ([ACTH]) (Rolleri et al. 1976; Vuolteenaho et al. 1981; Hodgkinson et al. 1984). The [A] and [NA] were measured radio-enzymatically in plasma (Da Prada and Zürcher 1976). All hormone assays were performed in duplicate. Intra-assay variation of [β-E] amounted to 4%, inter-assay 8%. The cross-reactivity to β-lipotropin was 16%. Heart rate was determined from an ECG record. The $\dot{V}O_2$ during ST was measured at 0.5-min intervals using an open spirometric system. The rate of perceived exertion was determined by means of the Borg scale (Borg 1973).

The $\dot{V}O_{2max}$ and maximal exercise intensity served as parameters of the maximal physical performance capacity. The endurance capacity was ascertained by $\dot{V}O_2$ and exercise intensity at $TH_{an,i}$. In ST, the parameter for the anaerobic capacity was considered to be the product of lactate increase and performance relative to body mass.

All data are expressed as means and SD. For statistical analysis one-way variance analysis with repeated measures and the Scheffé test were used. In the comparison between ST and AnT the Wilcoxon test was applied. Correlations were calculated by means of linear correlation analysis. The level of statistical significance was set at $P<0.05$.

Results

Incremental graded exercise

Changes in lactate concentration, heart rate and perceived exertion are presented in Fig. 1. The highest val-

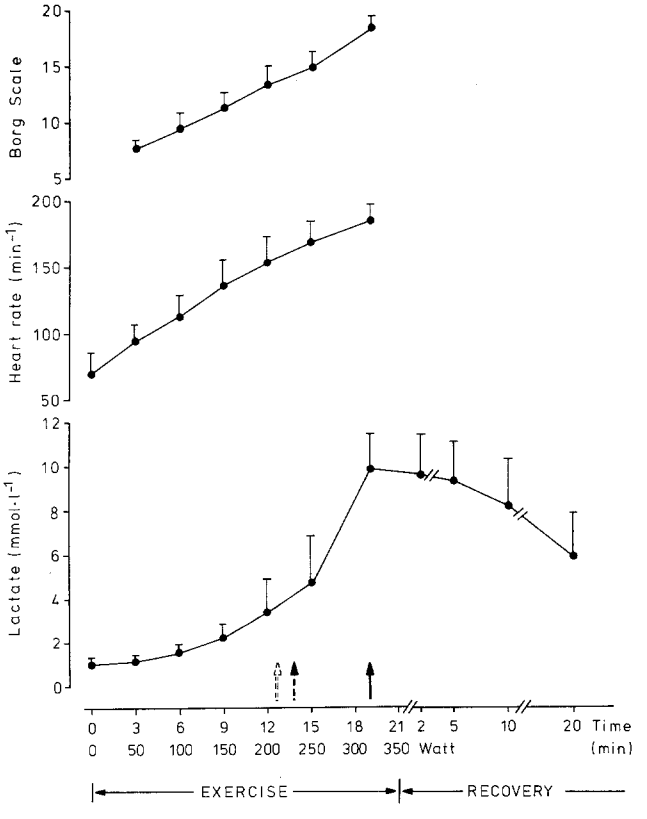

Fig. 1. Changes of lactate concentration, heart rate and rate of perceived exertion (Borg scale) during incremental graded exercise (means and SD). The *arrows* over the abscissa denote (from left to right) the individual anaerobic threshold, the anaerobic threshold of 4 $mmol \cdot l^{-1}$ lactate and the discontinuation of exercise

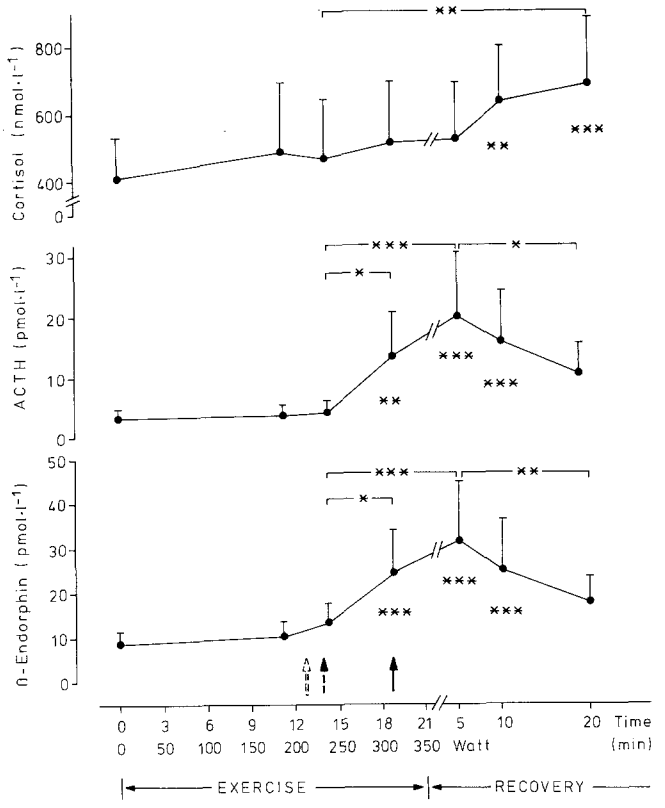

Fig. 2. Concentrations of β-endorphin, ACTH and cortisol before, during and after incremental graded exercise (means and SD). For *arrows* see legend to Fig. 1. Asterisks *below* the graph denote significant changes compared to the respective initial values (* = $P<0.05$; ** = $P<0.01$; *** = $P<0.001$)

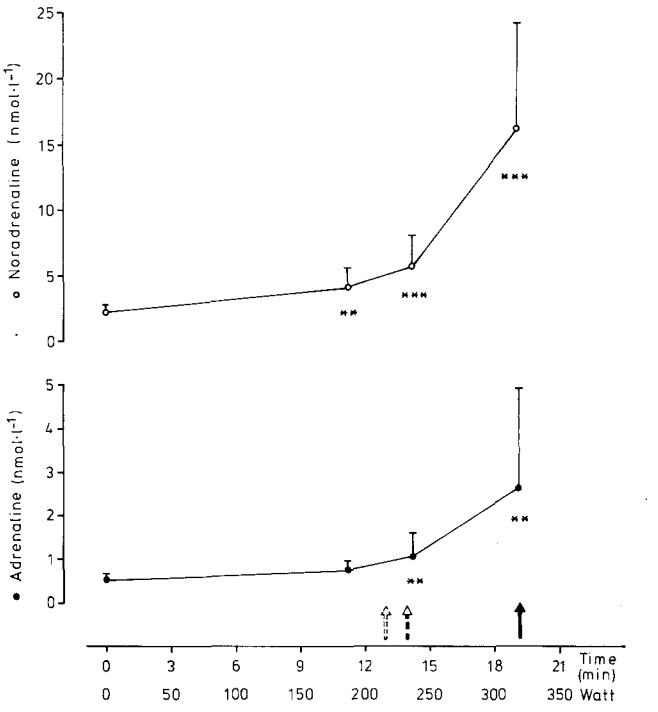

Fig. 3. Plasma levels of adrenaline and noradrenaline before and during incremental graded exercise (means and SD). For symbols see legend to Fig. 2

ues achieved were 10.06 mmol·l^{-1}, SD 1.66 lactate, a heart rate of 187 min^{-1}, SD 9 and a perceived rate of exertion of 18.8, SD 0.9 out of 20 on the Borg Scale.

Directly after exercise and in the 5th and 10th min after exercise, [β-E] (Fig. 2) was significantly higher than at rest ($P<0.001$). The highest concentration of β-E was reached in the 5th min after exercise and was 3.6 times higher than the pre-exercise level. In the 20th min of the recovery period there was no significant difference to the starting value. During exercise, there was a significant difference for [β-E] between the exercise level following the $TH_{an,i}$ (238 W = 75% $\dot{V}O_{2max}$) and the cessation of exercise. The [ACTH] (Fig. 2) demonstrated analogous behaviour to [β-E]. In comparison to [β-E], however, the exercise-induced increase in [ACTH] was higher and in the 5th min after exercise was 6 times the value at rest. The [C] (Fig. 2) was signi-

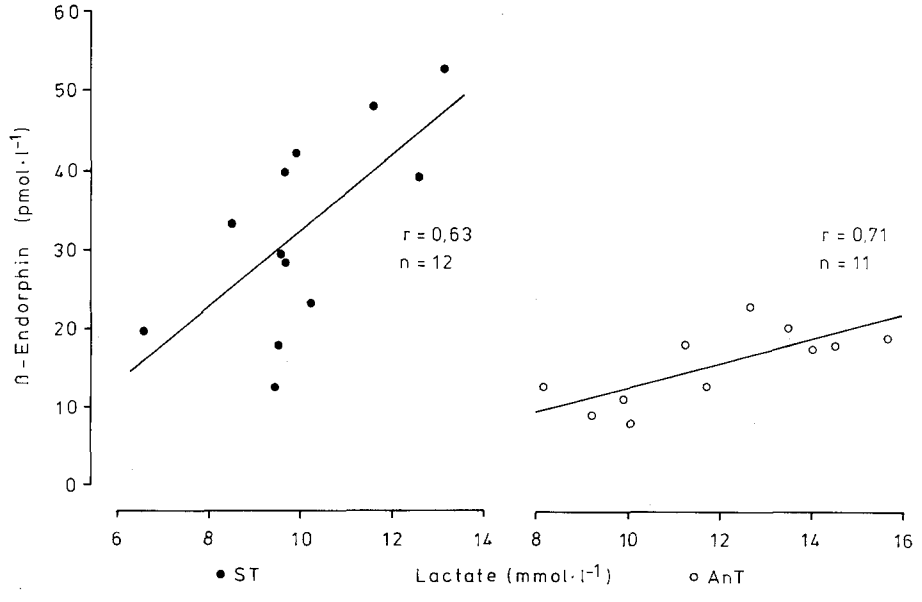

Fig. 4. Correlation between maximal concentration of β-endorphin and lactate in incremental graded exercise (*ST*, step test, *left panel*) and in the 1-min anaerobic test (*AnT*, right panel)

ficantly higher (56%, $P<0.01$ and 69%, $P<0.001$) in the 10th and 20th min after exercise than at rest. There were no significant differences noticed during exercise.

At the end of exercise, [A] and [NA] (Fig. 3) had increased to 5 and 7 times the pre-exercise concentration ($P<0.01$ and $P<0.001$). During exercise [NA] was significantly higher than at rest for every measurement taken, whereas [A] did not increase significantly until after $TH_{an,i}$ had been exceeded.

Hormonal interrelations were detected between [β-E] and [ACTH] both prior to exercise ($r=0.75$; $P<0.01$) and at every measurement stage after exercise (e.g. 5th min after exercise: $r=0.86$; $P<0.01$). In addition, slightly positive correlations were found between the concentrations of β-E and C at the end of exercise and between the exercise-induced increases in the two hormones ($r=0.69$ and 0.63). In contrast, there were no statistical relationships between [C] and [ACTH].

Both the maximal concentrations and the exercise-induced increases of [β-E] correlated with the maximal lactate concentrations ($r=0.63$ and 0.62; $P<0.05$: Fig. 4, left panel). The extent of the increase in [β-E] was independent of the physiological parameters $\dot{V}O_{2max}$, maximal performance capacity and physical performance corresponding to $TH_{an,i}$.

Anaerobic test

In the 1-min AnT, the performance relative to body mass was 6.6 $W \cdot kg^{-1}$, SD 0.8. The maximal lactate concentration, generally reached between the 6th and 8th min of exercise, was 12.00 $mmol \cdot l^{-1}$, SD 2.02 corresponding to an increase of 10.57 $mmol \cdot l^{-1}$, SD 1.75.

The [β-E] (Fig. 5) was 64% and 60% above the starting value in the 5th and 10th min after exercise, respectively ($P<0.01$). At the same points in time there was a definite increase in [ACTH] by 289% and 276%, respectively ($P<0.001$). In the 20th min after exercise there was also a significant increase in [ACTH] by 167% compared to the value prior to exercise ($P<0.05$). The [C] remained unchanged until the 20th min after exercise. Directly after AnT, [A] and [NA] increased to 7.1 and 7.4 times that of the starting value (each time $P<0.001$) and in the 5th min after exercise still showed a tendency to be higher than at rest.

The values of [β-E] in the 5th, 10th and 20th min after exercise were positively correlated with the corresponding ACTH concentrations (e.g. 10th min: $r=0.92$; $P<0.001$, Fig. 6). Correlations were also detected between the maximal concentrations of β-E and A ($r=0.61$; $P<0.05$). The values of [ACTH] and [C] prior to AnT were just significant in their negative correlation ($r=-0.65$; $P<0.05$). The maximal concentrations of β-E ($r=0.71$; $P<0.05$: Fig. 4, right panel), A ($r=0.84$; $P<0.001$) and NA ($r=0.67$; $P<0.05$) were correlated with the maximal lactate concentrations. No correlations were revealed between the performance achieved, or between the product of performance and increase in lactate concentration and the increase in [β-E].

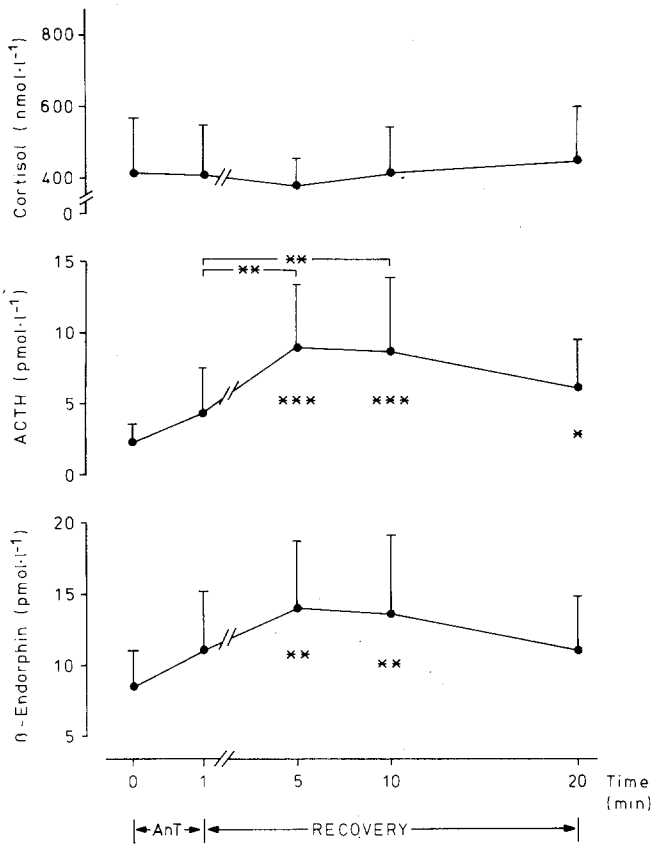

Fig. 5. Concentrations of β-endorphin, ACTH and cortisol before and after the 1-min anaerobic test (*AnT*; means and SD). For symbols see legend to Fig. 2

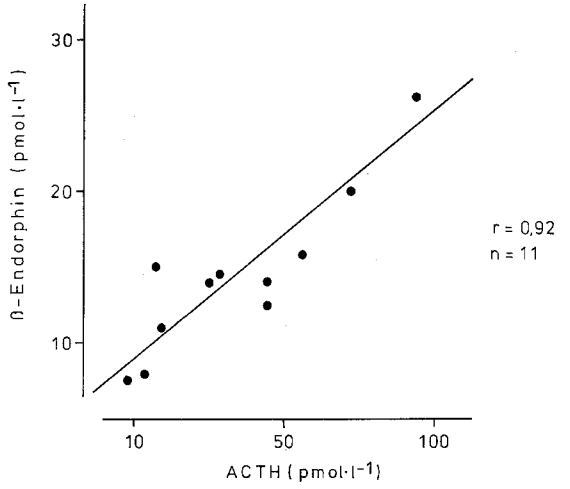

Fig. 6. Correlation between β-endorphin and ACTH in the 10th min after the 1-min anaerobic test

The comparison of the results of ST and AnT (Fig. 7) demonstrates that with identical pre-exercise values of [β-E], there was an almost double maximal level in ST. Prior to AnT, [ACTH] was one-third lower than prior to ST ($P<0.05$). The maximal values of [ACTH] in ST were slightly more than double those in AnT. After

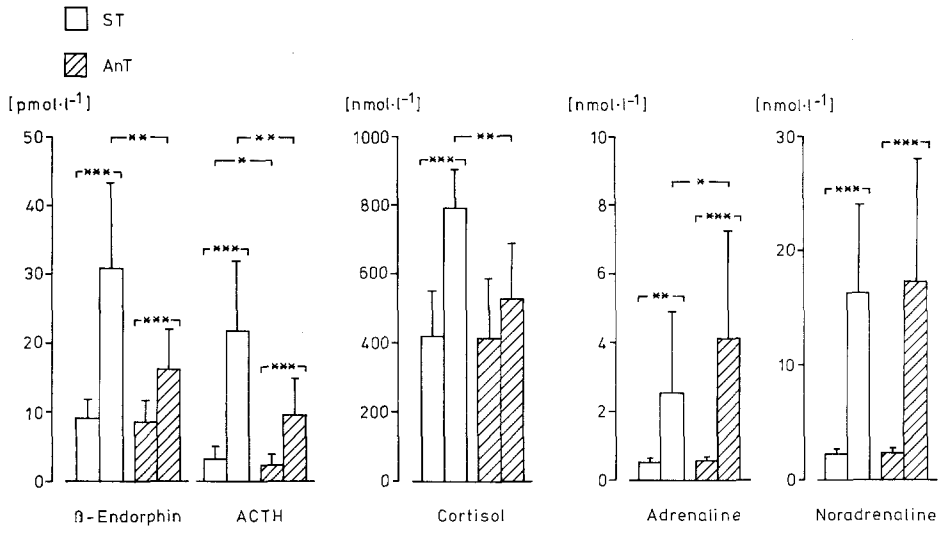

Fig. 7. Behaviour of the hormones in incremental graded exercise (*ST*, step test) and the 1-min anaerobic test (*AnT*). Comparison of the concentrations at rest (first column of each pair) and of the maximum concentrations (second column of each pair; means and SD, $*=P<0.05$; $**=P<0.01$; $***=P<0.001$)

AnT, [A] was significantly higher than in ST, whereas [NA] showed a similar increase.

Discussion

In substantiating previous investigations (De Meirleir et al. 1986; Donevan and Andrew 1987; Brooks et al. 1988), both incremental graded exercise and short-term anaerobic exercise caused an increase in the peripheral β-endorphin concentration. It is debatable whether the exercise-induced increase in [β-E] is related to the intensity of the exercise. There are studies which suggest such a correlation (Donevan and Andrew 1987; McMurray et al. 1987; Rahkila et al. 1987) and others which refute it (Farrell et al. 1982; Goldfarb et al. 1987). In addition, the former showed no consistent findings as to the intensity at which [β-E] increases.

The comparison of the individual studies is hindered by methodological influences, since both the intensities (defined as the percentages of $\dot{V}O_{2\,max}$) and the duration of exercise at each step varied. In two studies – under laboratory conditions (De Meirleir et al. 1986) and in a field test (Kraemer et al. 1989) – lactate concentration was also determined as a metabolic parameter under the assumption that the behaviour of [β-E] was primarily dependent upon anaerobic metabolic demand. This is confirmed by the present study. The [β-E] in ST did not increase until the $TH_{an,i}$ or the anaerobic threshold of 4 mmol·l^{-1} lactate was exceeded. More precisely, [β-E] increased significantly between the end of the exercise intensity in the $TH_{an,i}$ range and the cessation of exercise. It should be taken into consideration, however, that blood samples were drawn in only brief breaks after $TH_{an,i}$ was exceeded and there was already a tendency at that point for [β-E] to increase. Thus, the point of overproportionate increase in lactate concentration and [β-E] increase were probably identical. A connection between the increase in [β-E] and anaerobic metabolism was also indicated by the positive correlations between [β-E] and maximal lactate concentration both in ST and AnT. In the post-exercise period [β-E] and lactate concentration demonstrated synchronous behaviour and reached their highest points at approximately the same time. These current findings suggest that in incremental graded exercise with a combination of aerobic and anaerobic energy supply or short-term maximal exercise, the behaviour of [β-E] depends on the degree of metabolic demand; a connection with parameters of physical performance capacity was not detectable.

The physiological link between lactate concentration and [β-E] remains unclear. In earlier studies describing correlations between [ACTH] and lactate concentration (Farrell et al. 1983; Buono et al. 1986), it was assumed that substances resulting from anaerobic metabolism stimulated the hypothalamic-pituitary axis via chemoreceptors in muscle. In AnT, which focused on the anaerobic lactacidic energy supply, both the exercise-induced increases of [β-E] and the increase in [A] and [NA] were correlated to the maximal lactate concentrations. The same results have been found earlier only for A (Brooks et al. 1988). Sympatho-adrenal activity is necessary for a high glycolitic flow rate, whereas the role of β-E remains unclear. Results of animal experiments suggest a stimulating effect of β-E upon glycogenolysis and gluconeogenesis (Allan et al. 1983; Matsumura et al. 1984).

Apart from the influence of the intensity of exercise, duration of exercise also influenced the changes of [β-E]. Endurance exercise, performed at lactate steady-state at the intensity of $TH_{an,i}$, did not lead to an increase in [β-E] until approximately 60 min of exercise had been exceeded (Schwarz and Kindermann 1989). The behaviour of β-E apparently depended both on intensity, defined as an increase in anaerobic metabolism, and on the duration of exercise. In keeping with the findings of an earlier investigation (Brooks et al. 1988), this accounted for the slighter increase in [β-E] in the shorter AnT as compared to ST. The similar value at the start of exercise in AnT and ST and the increase of [β-E] in AnT suggest that intensive double exercise in-

tensities in a few hours do not tax the responsiveness of β-E.

An increase in [β-E] during exercise might also suggest a simultaneous increase in other hormones. β-E and ACTH both originate from the same precursor molecule (Mains et al. 1977). Both hormones are secreted concomitantly (Guillemin et al. 1977; Fraioli et al. 1980). In the present study, a correlation was also noticed between [β-E] and [C], although this was not statistically apparent for [ACTH] and [C]. An ACTH-triggered increase in [C] cannot be expected until after a certain time-lage due to the induction of the adrenocortical enzymes. Since β-E has a relatively long half-life of 20 min – as opposed to only 3 min for ACTH (Krieger 1982), this can result in the statistical correlation between [C] and [β-E] which is secreted equimolar with ACTH, without necessarily any direct relationship between [β-E] and [C]. Comparable earlier research findings (Mougin et al. 1987) showing positive correlations between [C] and [β-E] might also, therefore, have to be interpreted more carefully. After ST, the negative feed-back of increased [C] affecting [ACTH] (Sutton et al. 1974) is reflected in the lowered ACTH concentration before AnT, correlating negatively with [C].

It is not yet clear whether a correlation exists between the catecholamines and [β-E]. In animal experiments β-adrenergic stimulation by means of isoproterenol infusion led to dose-related increases in [β-E] (Berkenbosch et al. 1981). On the other hand, after the blocking of the opiate receptors with naloxone, an increase in [A] was observed with higher doses, as compared to lower doses (Estilo and Cottrell 1982; Manelli et al. 1984). Since opiate receptors were identified in suprarenal medulla (Chavkin et al. 1979), endogenous opioids might have an influence on [A] release. In the present study in AnT, in which lactate acidosis was an essential stress component, a relationship between the behaviour of β-E and A was noticed. In ST no correlation between [β-E] and [A] was demonstrated; thus, it might be necessary to discuss different factors influencing these hormones, such as duration of exercise and concomitant metabolic demand or subjective perception of exertion. There are no conclusive findings which clearly reveal whether the sympatho-adrenal system and endogenous opioids are mutually influential.

The present study allows a number of conclusions. Physical exercise with increasing or mostly anaerobic components led to a simultaneous increase in [β-E] and [ACTH] occurring when the anaerobic threshold had been exceeded or at the point of an overproportionate increase in lactate concentration. The extent of the increase in β-E concentration correlated with the level of lactate concentration indicating the degree of metabolic demand to be the decisive factor for the release of β-E. The similar behaviour of [β-E] and [A] during supramaximal exercise might substantiate the hypothesis that endogenous opioids and catecholamines are mutually influential.

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References

Allan EH, Green IC, Titheradge MA (1983) The stimulation of glycogenolysis and gluconeogenesis in isolated hepatocytes by opioid peptides. Biochem J 216:507–510

Berkenbosch F, Vermes I, Binnekade R (1981) Beta-adrenergic stimulation induces an increase of the plasma levels of immunoreactive alpha-MSH, beta-endorphin, ACTH and of corticosterone. Life Sci 29:2249–2256

Borg GAV (1973) Perceived exertion: a note on "history" and methods. Med Sci Sports 5:90–93

Brooks S, Burrin J, Cheetham ME, Hall GM, Yeo T, Williams C (1988) The responses of the catecholamines and β-endorphin to brief maximal exercise in man. Eur J Appl Physiol 57:230–234

Buono MJ, Yeager JE, Hodgdon JA (1986) Plasma adrenocorticotropin and cortisol responses to brief high-intensity exercise in humans. J Appl Physiol 61:1337–1339

Chavkin C, Cox BM, Goldstein A (1979) Stereo-specific opiate binding in the bovine adrenal medulla. Mol Pharmacol 15:751–753

Da Prada M, Zürcher G (1976) Simultaneous radioenzymatic determination of plasma and tissue adrenaline, noradrenaline, and dopamine within the femtomole range. Life Sci 19:1161–1174

Dearman J, Francis KT (1983) Plasma levels of catecholamines, cortisol, and β-endorphins in male athletes after running 26.2, 6, and 2 miles. J Sport Med 23:30–38

De Meirleir K, Naaktgeboren N, Steirteghem V, Gorus F, Albrecht J, Block P (1986) Beta-endorphin and ACTH levels in peripheral blood during and after aerobic and anaerobic exercise. Eur J Appl Physiol 55:5–8

Donevan RH, Andrew GM (1987) Plasma β-endorphin immunoreactivity during graded cycle ergometry. Med Sci Sports Exerc 19:229–233

Estilo AE, Cottrell JE (1982) Hemodynamic and catecholamine changes after administration of naloxone. Anesth Analg 61:349–353

Farrell PA, Gates WK, Morgan WP, Maksud MG (1982) Increases in plasma β-endorphin/β-lipotropin immunoreactivity after treadmill running in humans. J Appl Physiol 52:1245–1249

Farrell PA, Garthwaite TL, Gustafson AB (1983) Plasma adrenocorticotropin and cortisol responses to submaximal and exhaustive exercise. J Appl Physiol 55:1441–1444

Fraioli F, Moretti C, Paolucci D, Alicicco E, Crescenzi F, Fortunio G (1980) Physical exercise stimulates marked concomitant release of β-endorphin and ACTH in peripheral blood in man. Experientia 36:987–989

Goldfarb AH, Hatfield BD, Sforzo GA, Flynn MG (1987) Serum β-endorphin levels during a graded exercise test to exhaustion. Med Sci Sports Exerc 19:78–82

Guillemin R, Vargo T, Rossier J, Minick S, Ling N, Rivier C, Yair W, Bloom F (1977) β-Endorphin and adrenocorticotropin are secreted concomitantly by the pituitary gland. Science 197:1362–1369

Hodgkinson SC, Allolio B, Landon J, Lowry PhJ (1984) Development of a non-extracted 'two-site' immunoradiometric assay for corticotropin utilizing extreme amino- and carboxy-terminally directed antibodies. Biochem J 218:703–711

Hohorst HJ (1962) L-(+)-Lactat, Bestimmung mit Lactatdehydrogenase und DPN. In: Bergmeyer HU (ed) Methoden der enzymatischen Analyse. Verlag Chemie, Weinheim

Hughes J, Smith TW, Kosterlitz HW, Fothergill LA, Morgan MA, Morris HR (1975) Identification of two related pentapeptides from the brain with potent opiate against activity. Nature 258:577

Kraemer WJ, Fleck SJ, Callister R, Shealy M, Dudley GA, Maresh CM, Marchitelli L, Cruthirds C, Murray T, Falkel JE (1989) Training responses of plasma beta-endorphin, adrenocorticotropin, and cortisol. Med Sci Sports Exerc 21:146–153

Krieger DT (1982) Endorphins and enkephalins. Dis Mon 28:1–53

Li CH (1977) β-Endorphin: a pituitary peptide with potent morphine-like reactivity. Arch Biochem 183:592–604

Mains RE, Eipper BA, Ling N (1977) Common precursor to corticotropins and endorphins. Proc Natl Acad Sci USA 74:3014–3018

Mannelli M, Maggi M, De Feo ML, Cuomo S, Delitala G, Giusti G, Serio M (1984) Effects of naloxone on catecholamine plasma levels in adult men. A dose-response study. Acta Endocrinol 106:357–361

Matsumura M, Fukushima T, Saito H, Saito S (1984) In vivo and in vitro effects of β-endorphin on glucose metabolism in the rat. Horm Metab Res 16:27–31

McMurray RG, Forsythe WA, Mar MH, Hardy CJ (1987) Exercise intensity-related responses of β-endorphin and catecholamines. Med Sci Sports Exerc 19:570–574

Mougin C, Baulay A, Henriet MT, Haton D, Jacquier MC, Turnill D, Berthelay S, Gaillard RC (1987) Assessment of plasma opioid peptides, β-endorphin and met-enkephalin at the end of an international nordic ski race. Eur J Appl Physiol 56:281–286

Rahkila P, Hakala E, Salminen K, Laatikainen T (1987) Response of plasma endorphins to running exercises in male and female endurance athletes. Med Sci Sports Exerc 19:451–455

Rolleri E, Zannino M, Orlandini S, Malvano R (1976) Direct radioimmunoassay of plasma cortisol. Clin Chim Acta 66:319–330

Schwarz L, Kindermann W (1989) β-endorphin, catecholamines, and cortisol during exhaustive endurance exercise. Int J Sports Med 10:324–328

Stegmann H, Kindermann W, Schnabel A (1981) Lactate kinetics and individual anaerobic threshold. Int J Sports Med 2:160–165

Sutton J, Coleman MT, Casey JH (1974) Adrenocortical contribution to serum androgens during physical exercise. Med Sci Sports 6:72

Vuolteenaho O, Leppäluoto J, Vakkuri O, Karppinen J, Höyhtyä M, Ling N (1981) Development and validation of a radioimmunoassay for beta-endorphin-related peptides. Acta Physiol Scand 112:313–321