Training – overtraining: performance, and hormone levels, after a defined increase in training volume *versus* intensity in experienced middle- and long-distance runners

M. Lehmann MD, U. Gastmann MD, K. G. Petersen MD*, N. Bachl MD, A. Seidel cand. med., A. N. Khalaf MD*, S. Fischer Dipl. oec. troph. and J. Keul MD

Department of Sports and Performance Medicine and *Department of Endocrinology, University Clinic Freiburg, Freiburg, Germany

Performance and hormones were determined in eight middle- and nine long-distance runners after an increase in training volume (ITV, February 1989) or intensity (ITI, February 1990). Seven runners participated in both studies. The objective was to cause an overtraining syndrome. The mean training volume of 85.9 km week^{-1} increased within 3 weeks to 176.6 km week^{-1} during ITV and 96–98% of training volume was performed as long-distance runs at mean(s.d.) 67(8)% of maximum capacity. Speed endurance, high-speed and interval runs averaging 9 km week^{-1} increased within 3 weeks to 22.7 km during ITI, and the total volume increased from 61.6 to 84.7 km. A plateau in endurance performance and decrease in maximum performance occurred during ITV, probably due to overtraining, with performance incompetence over months. Nocturnal catecholamine excretion decreased markedly (47–53%), contrary to exercise-related plasma catecholamine responses, which increased. Resting and exercise-related cortisol and aldosterone levels decreased. Improvement in endurance and maximum performance occurred during ITI indicating a failure to cause an overtraining syndrome in ITI. Decrease in noctural catecholamine excretion was clearly lower (9–26%), exercise-related catecholamine responses showed a significant decrease, cortisol and aldosterone levels remained almost constant, exercise-related prolactin levels decreased slightly. There were no differences in insulin, C-peptide, free testosterone, somatotropic hormone (STH), follicle stimulating hormone (FSH), luteinizing hormone (LH), thyroid stimulating hormone (TSH), tri-iodothyronine (T_3) and thyroxine (T_4). The decrease in nocturnal catecholamine excretion during ITV might indicate a decrease in intrinsic sympathetic activity in exhausted sportsmen. But it remains open whether this reflected a central nervous system incompetence.

Keywords: Training, catecholamines, hormones

The overtraining syndrome describes a chronic imbalance between training and recovery in athletes with accumulation of fatigue, different physical and psychological symptoms and performance incompetence over weeks and months[1–3]. It has to be differentiated from short-term overtraining (overreaching) and local muscular overstrain[1,3]. From a clinical and descriptive standpoint[1], the overtraining syndrome can be divided into hypothetical parasympathetic and sympathetic types which describe the clinical pattern not the pathophysiological basis. To examine the hypothesis of a neuroendocrine, autonomic nervous system, or hormonal impairment[1–3], we performed two prospective experimental training studies with the objective of producing an experimental overtraining syndrome in experienced middle- and long-distance runners.

Subjects and methods

Anthropometric data are summarized in *Table 1*. The middle- and long-distance runners were acquainted with laboratory examination procedures. Eight athletes participated in the ITV study in February 1989, nine in the ITI study in February 1990, and seven in both studies. All athletes were informed about procedure and objective. They granted informed consent in writing and received DM1000 for participation in each study. Ethics Commission approval was obtained.

Diet

The athletes maintained their customary diet without alteration. No dietary concentrates, mineral beverages, vitamins or any medication were permitted (*Table 2*).

Protocol

Training volume and content were documented during the prephase of 1 week (*Table 3*). A 3-week training phase followed. During the ITV study, training was performed exclusively (96–98%) as long-distance runs at mean(s.d.) 67(8)% of maximum capacity.

Training volume increased by one-third per week, to about double the baseline values (*Table 3*). In the

Address for correspondence: Professor Dr M. Lehmann, University Clinic Freiburg, Department of Sports and Performance Medicine, Hugstetter Strasse 55, D-7800 Freiburg, Germany

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Table 1. Anthropometric data, oxygen uptake capacity ($\dot{V}O_{2max}$), and running experience of the athletes participating in the increase in volume study (ITV) and the increase in intensity study (ITI) at baseline (1) and final examination (2)

	n*	Age (years)	Height (cm)	Weight (kg)		$\dot{V}O_{2max}$ (ml kg^{-1} min^{-1})		Running experience (years)
				1	2	1	2	
ITV	8	33(7)	179(4)	70(7)	70(6)	66.8(5.6)	64.6(4.3)†	5.6(1.3)
ITI	9	34(7)	180(4)	73(4)	72(4)‡	65.8(5.2)	67.6(5.7)†	6.4(1.5)

Values are mean(s.d.)
* Seven athletes participated in both studies; †$P < 0.10$; ‡$P < 0.05$; Dixon and Mood sign test

Table 2. Caloric supply

	Increase in volume study	Increase in intensity study
Total caloric supply per day (kcal)	4110(942)	3515(781)
Carbohydrates (%)	49(6)	45(5)
Proteins (%)	14(3)	13(2)
Lipids (%)	33(6)	38(5)
Alcoholics(%)	4(3)	3(3)

Values are mean(s.d.)

ITI study, speed endurance, high-speed and interval runs increased from 9 km (first week) to a mean of 22.7 km (fourth week). The overall volume increased from 61.6 km week^{-1} to 84.7 km week^{-1}, and the long-distance runs (52.7–62.0 km) were performed at a mean(s.d.) of 68(7)% of maximum performance. Intensity training took place on Mondays (six to 10 × 400-m interval runs at a mean(s.d.) of 70(4) s per 400-m run), Wednesdays (six to 10 × 1000-m high-speed runs at a mean(s.d.) of 86(6)% of maximum performance), and Fridays (8000–10 000-m speed-endurance runs at a mean(s.d.) of 77(7)% of maximum capacity). No training took place on Sunday in either study. Speed endurance, high-speed and interval runs were performed in a stadium and were monitored continuously. Long-distance runs were performed on known cross-country courses and randomly controlled. The last 2 weeks of the first study were also controlled continuously.

Incremental treadmill tests took place on days 0 and 28. After the subjects had rested for 30 min in a supine position, venous blood was drawn before ergometry and immediately after exhaustion for determination of cortisol (RIA Cortisol Bridge, Serono, Freiburg, Germany), aldosterone (RIA Aldosterone, AIA, Serono, Freiburg, Germany), free testosterone (RIA Free Testosterone, Biermann, Bad Nauheim, Germany), insulin and C-peptide (RIA-Gnost Insulin and hC-Peptide, Behringwerke, Marburg), human growth hormone (hGH) (RIA hGH 100, Pharmacia Diagnostics, Uppsala, Sweden), prolactin (Prolactin AIA Clone, Serono, Freiburg, Germany), follicle stimulating hormone (FSH) and luteinizing hormone (LH) (FSH/LH AIA Clone Immunoradiometric Assay, Serono, Freiburg, Germany), thyroid stimulating hormone (TSH) (hTSH RIA-Gnost, Behringwerke, Marburg, Germany), triiodothyronine (T_3)/thyroxine (T_4) (free T_3/T_4 RIA, Becton, Dickinson, New York, USA). The analyses were performed by the same experienced technical assistant. All samples taken from one subject during one study were processed in one assay. The tests were performed on a motor-driven treadmill (Jäger, Würzburg, Germany; 1.5% slope) starting with a speed of 10 km h^{-1}, which increased at 3-min intervals and a 30-s pause by 2 km h^{-1} to subjective exhaustion. Heart rate (from the electrocardiogram) and lactate levels (earlobe capillary blood, enzymatic[4] were determined before exercise, immediately in each pause, and after termination of exercise. The catecholamine levels (earlobe capillary blood) were determined radioenzymatically[5,6] before exercise, at

Table 3. Training data

	Week 1	Week 2	Week 3	Week 4	Intensity as percentage of maximum performance*
Increase in volume study					
Training volume (km)*	85.9(14.2)	115.1(17.5)	143.1(20.0)	174.6(26.7)	
LDR (%)	92.7	96		98	67(8)
SDR/SR/IR (%)	7.3	4		2	
Increase in intensity study					
Training volume (km)*	61.6(21.4)	62.9(6.0)	75.5(8.0)	84.7(9.0)	
LDR (%)	85.2	76.3	75.4	73.2	68(7)
SDR (%)	8.3	10.8	11.3	11.3	77(7)
SR (%)	5.9	9.0	9.2	10.7	86(6)
IR (%)	0.5	3.8	4.2	4.7	70†(4)

* Values are mean(s.d.)
† Measured in s per 400-m run
LDR, long-distance runs; SDR, speed-distance runs; SR, speed runs; IR, interval runs

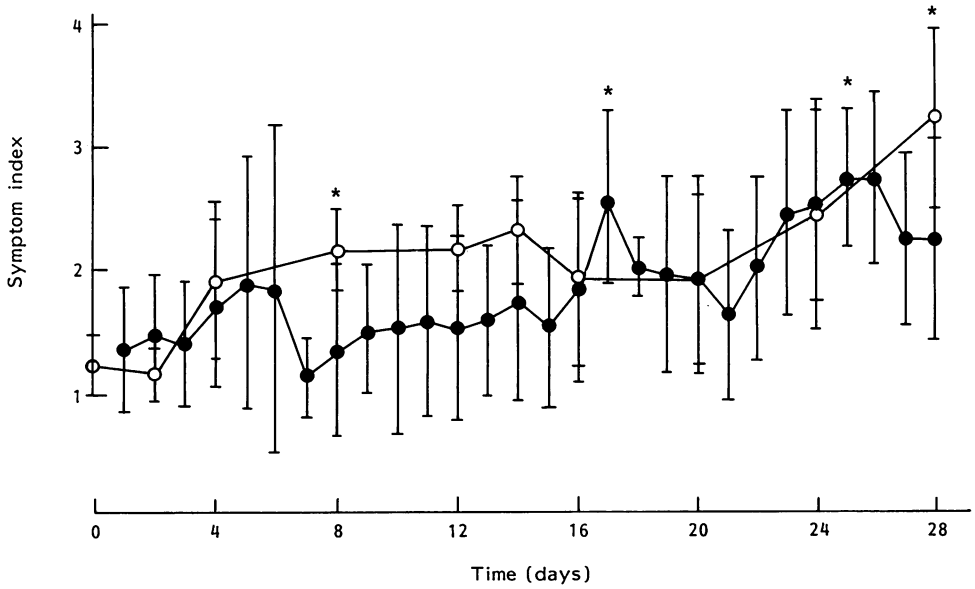

Figure 1. Symptom index during the increase in volume study (○) and the increase in intensity study (●). *The symptom index was significantly increased at day 8 compared with day 0 and at day 28 compared with days 0 and 8 during the increase in volume study ($P < 0.05$); a significant increase was observed at days 17, 25 and 26 compared with baseline values during the increase in intensity study ($P < 0.05$)

the same submaximum level ($v = 16\,h^{-1}$) and immediately after termination. The running velocity at the 4 mmol lactate level was taken as an index of the endurance performance capacity and the total distance during incremental running as an index of maximum performance capacity.

The subjects kept a diary of heart rate on waking, body weight, training, diet (special recording sheets on 11 days of each study), and any kind of medical symptom. In addition, they rated their well-being daily on a four-point scale: 1, no symptoms; 2, mild; 3, moderate; 4, severe symptoms (*Figure 1*).

During the prephase of 1 week and the prospective training phase of 3 weeks, and for an additional week thereafter, the athletes collected total nocturnal urine on two nights week^{-1} in collection flasks prepared with hydrochloric acid (having an approximate pH value of 2) for determination of nocturnal catecholamine excretion[5], which we took as a parameter of intrinsic sympathetic activity. Collection was made during a night after intensive training and a night after a day of relaxation, and the mean was taken. The 24-h urine was collected on another day each week to determine cortisol elimination.

Assessment and statistics

Means and standard deviations were calculated for performance-related data. The statistical examination within a group was made using the sign test of Dixon and Mood[7], which is particularly critical for small random samples – if the data of two subjects (in a random sample of eight or nine subjects) differed in sign from 6–7 subjects (difference between baseline and final values), this only meant a probability of error of $P < 0.10$ (not significant); if one subject differs, $P < 0.05$ (significant); concordance of all eight or nine subjects resulted in $P < 0.01$. The Wilcoxon, Mann–Whitney U test[7] was used between the groups. Median values, 50% confidence range and overall range of data were determined for hormonal parameters and catecholamines. Statistical comparisons were performed using the sign test and the U test.

Results

Anthropometric data are listed in *Table 1* and performance behaviour is shown in *Figure 2*. Slight weight loss was observed during the ITI study. The running velocity at the 4 mmol lactate level did not change in the ITV study and improved in the ITI study. The total running distance in the incremental test of six of the eight athletes decreased during the ITV study and increased in seven of nine athletes during the ITI study. Heart rate is given in *Figure 3* and symptom index in *Figure 1*. The morning resting heart rate remained constant in both studies – it decreased before exercise, at identical exercise levels, and during maximum exercise in the ITV study. Symptoms such as exhaustion, fatigue, burn-out and muscle stiffness, were significantly increased during the ITV study compared with the ITI study.

Plasma catecholamines are listed in *Table 4*. The lower dopamine levels in the ITI compared with the ITV group most likely reflect inter-assay variability. No significant dopamine changes were observed in the two studies. Noradrenaline and adrenaline levels were in the same range in both studies, with the exception of differences in the adrenaline maxima. At the same exercise levels in the ITV study, there was an increase in noradrenaline and adrenaline re-

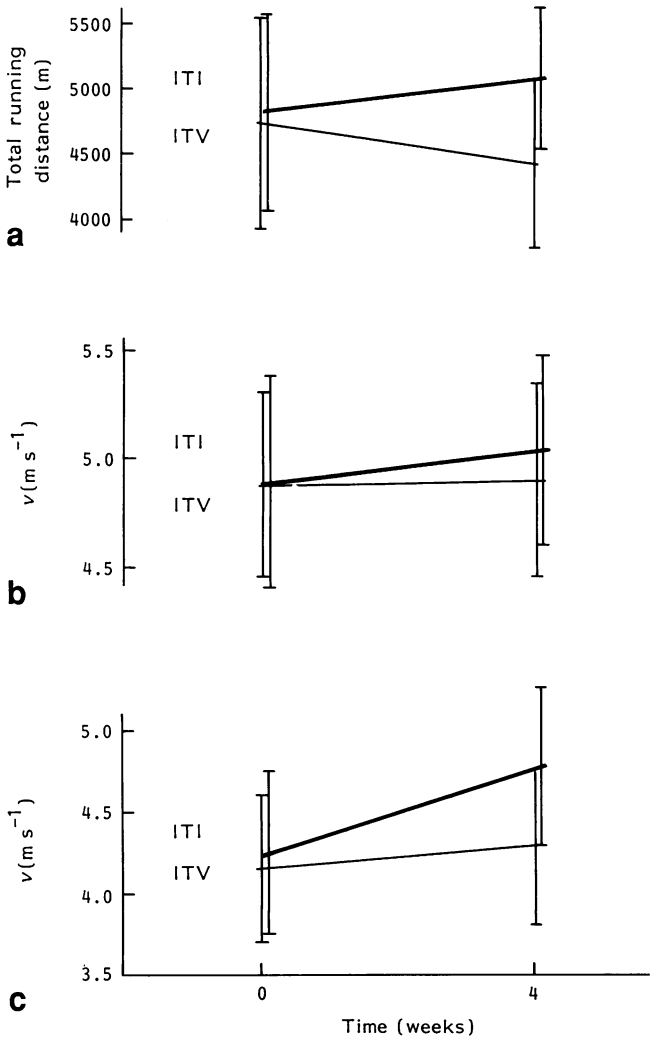

Figure 2. Running speed at **b**, 2 and **c**, 4 mmol lactate level increased significantly during the increase in intensity study (ITI) but not during the increase in volume study (ITV). The total running distance decreased in six of eight athletes during ITV and increased in seven of nine athletes during ITI. **a** ITI, $P < 0.10$; ITV, $P < 0.10$; **b** ITI, $P < 0.01$; ITV not significant; **c** ITI, $P < 0.01$; ITV, $P < 0.05$

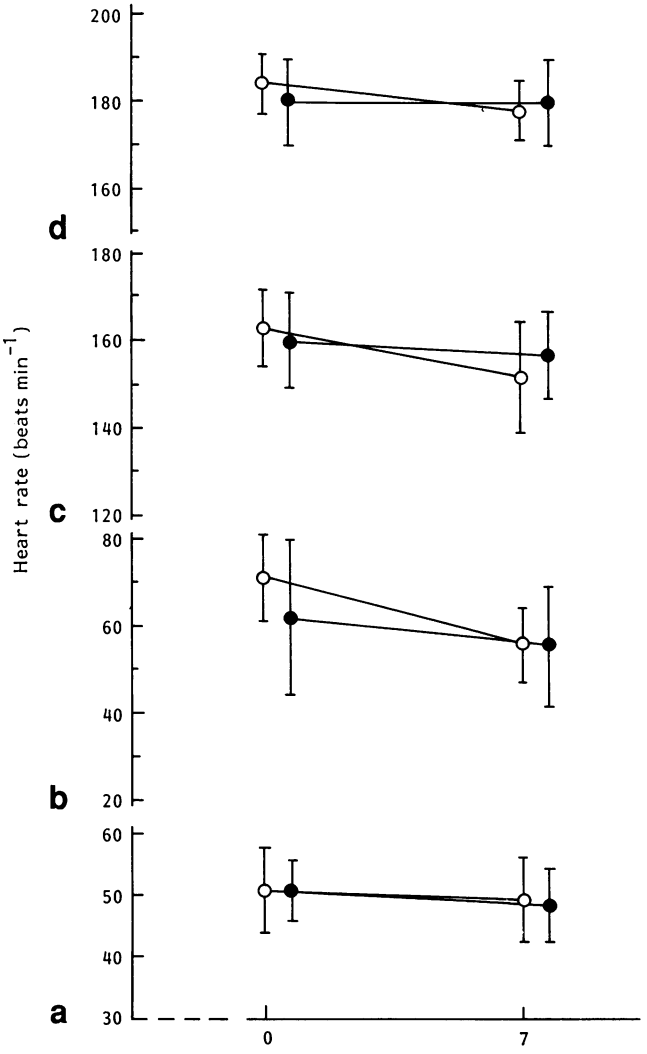

Figure 3. Heart rate **a** on waking remained constant during the increase in volume (○) and intensity (●) studies. Heart rate **b**, before ergometric exercise, **c** at identical exercise load (v = 16 km h^{-1}) and **d**, during maximum treadmill running decreased significantly during the increase in volume study ($P < 0.05$) and remained constant during the increase in intensity study

sponses, with a decrease in the ITI study. The levels before exercise showed the same trend.

Nocturnal urinary catecholamine elimination is given in *Table 5*. Catecholamine elimination remained comparatively constant in both studies. Immediately after completion of the ITV study, decreases of 47% (dopamine), 53% (noradrenaline) and 48% (adrenaline) were observed. The decreases following the ITI study were only between 9 and 26%.

Hormonal parameters are displayed in *Tables 6* to *8*. The serum levels of cortisol and aldosterone decreased slightly at rest and following maximum exercise in the ITV study, and remained constant following ITI. The exercise-induced prolactin response was also slightly lower in the ITI study. There were no essential differences in dependency on ITV or ITI in the other hormonal parameters (free testosterone, insulin C-peptide, STH, FSH, LH, TSH, T_3 and T_4) at the same time of measurement. The resting values of cortisol and aldosterone were already different between the two studies ($P <$ 0.05–0.01), which we consider evidence of the inter-assay variability. The maximum levels also differed between the two studies ($P < 0.05$) for which study-specific effects and the differing baseline values may be responsible. The 24-h cortisol excretion remained constant over 5 weeks in both studies.

Discussion

The plateau in endurance performance and the decrease in maximum performance (six of eight athletes) with competition incompetence for several months during the ITV study is probably attributable to an exhaustion or overtraining syndrome caused by an imbalance between training and recovery[1,3]. A

Table 4. Behaviour of free plasma catecholamines

	Increase in volume study		*P*	*Increase in intensity study*	
	1	*2*		*1*	*2*
Dopamine (nmol l^{-1})					
Before exercise					
Median	2.140	1.216	†/‡	0.483	0.377
50% confidence interval	0.449–1.898	0.778–1.469		0.178–0.533	0.241–0.774
Range	0.043–3.659	0.702–2.048		0.045–1.151	0.032–1.606
At v = 16 km h^{-1}					
Median	1.222	1.059	‡/†	0.644	0.566
50% confidence interval	1.059–1.632	0.858–1.632		0.423–0.871	0.416–0.748
Range	0.728–4.095	0.215–2.594		0.299–1.281	0.039–0.806
Maximum exercise					
Median	2.691	2.594	–/†	1.560	1.333
50% confidence interval	1.943–2.886	2.275–2.919		1.203–2.730	0.962–1.892
Range	0.143–4.108	0.708–3.367		0.839–3.062	0.280–2.717
Noradrenaline (nmol l^{-1})					
Before exercise					
Median	3.068	3.623*	–	2.950	2.301
50% confidence interval	2.714–3.451	3.062–4.189		2.301–3.481	2.006–2.773
Range	2.000–5.800	2.508–8.078		1.974–3.776	1.770–4.602
At v = 16 km h^{-1}					
Median	8.779	10.284†	–	8.614	7.257†
50% confidence interval	6.112–10.555	8.413–13.263		7.316–11.918	6.077–9.617
Range	3.664–12.664	5.994–18.626		5.251–22.892	4.307–18.526
Maximum exercise					
Median	26.816	28.267	†/–	32.270	27.317
50% confidence interval	15.322–37.205	26.515–36.899		27.317–43.542	20.470–39.530
Range	11.670–44.693	13.841–52.934		20.945–56.935	18.762–51.566
Adrenaline (nmol l^{-1})					
Before exercise					
Median	0.977	0.813	–	0.993	0.578†
50% confidence interval	0.601–1.108	0.650–0.961		0.633–1.113	0.437–0.841
Range	0.235–1.119	0.448–1.327		0.338–2.020	0.317–1.092
At v = 16 km h^{-1}					
Median	1.218	1.316*	–	1.239	0.884†
50% confidence interval	0.890–1.322	1.141–1.376		1.010–1.567	0.688–1.611
Range	0.688–2.113	1.016–1.649		0.497–3.243	0.622–2.490
Maximum exercise					
Median	2.413	2.588	†/–	5.242	4.532
50% confidence interval	2.200–3.058	1.867–3.964		2.730–10.183	1.835–7.043
Range	1.370–5.143	1.349–4.646		1.392–11.062	1.387–10.953

1, baseline examination; 2, final examination; v running velocity (treadmill running)
Statistical examination between both groups 1:1/2:2 was conducted using the Wilcoxon Mann–Whitney U test: *$P < 0.10$; †$P < 0.05$; ‡$P < 0.01$

training cycle of 4 weeks should be long enough to bring improvement in endurance and maximum performance capacity in experienced athletes, since, 1 year later in the same athletes, at the same time of year, ITI resulted in improved performance within a 4-week period (*Figure 2*) – the examined athletes appeared to demonstrate a 'genetic reserve'. Severe glycogen deficiency is also less likely to cause lack of improvement in performance following ITV because pronounced glycogen deficiency is usually accompanied by lower lactate concentrations[8], elevated plasma adrenaline levels and tachycardia at the same absolute exercise level[9]. Moreover, a decrease in the insulin level in endurance exercise can be expected in the presence of glycogen deficiency. In the present ITV study, the lactate level remained constant, the adrenaline level showed only a discrete insignificant increase at the same exercise level, the heart rate decreased slightly (*Figure 3*), and the insulin level showed no significant change (*Tables 6* and *7*). Assessment of the dietary data also shows no convincing evidence of a severe glycogen deficiency (*Table 2*).

The significant decrease in the plasma noradrenaline response at the same exercise level in the ITI study is typical of effective endurance training[10–12], contrary to the ITV study, in which there is an increase in the noradrenaline response (*Table 4*). Since the plasma half-life of the catecholamines in healthy individuals is not notably dependent on physical training[13,14], the altered plasma catecholamine levels probably represent a primary expression of altered release. The training status of the skeletal muscle is considered important for the adaptation of sympathetic activity[15] and afferent conduction pathways have been described[16]. However, it is unclear how their receptors in the musculature are stimulated. There are at least two different mechanisms, since reduction

Table 5. Urinary excretion of free catecholamines (pmol min^{-1}) during the night*

	Week 1	*Week 2*	*Week 3*	*Week 4*	*Week 5*
Increase in volume study					
Dopamine					
Median	1422	1283	1497	1475	764‡
50% confidence interval	1127–2106	786–1885	1384–1852	1046–1963	513–1003
Range	857–3519	552–6025	955–2606	546–3152	273–2800
Noradrenaline					
Median	178	164†	166	160	85§
50% confidence interval	125–348	97–182	108–237	150–187	78–96
Range	98–551	91–483	94–626	80–327	64–126
Adrenaline					
Median	50	47	47	50	26§
50% confidence interval	37–86	24–62	25–75	43–65	24–27
Range	23–213	16–218	17–149	9–142	13–42
Increase in intensity study					
Dopamine					
Median	1296	1205	1176	884	968†
50% confidence interval	1191–1487	945–1268	1106–1249	765–953	663–1044
Range	533–1946	515–1949	604–1553	571–1378	449–2320
Noradrenaline					
Median	126	146†	126	117	103‡
50% confidence interval	98–137	139–157	102–137	90–129	93–118
Range	70–180	67–187	60–150	79–138	48–151
Adrenaline					
Median	33	35	38	24†	30
50% confidence interval	27–41	31–50	32–44	23–28	26–36
Range	20–50	19–59	20–58	22–57	14–64

* There are no significant differences in urinary catecholamine excretion between both examinations, except for noradrenaline excretion in week 1 (Wilcoxon Mann-Whitney U test, $P < 0.05$); noradrenaline, adrenaline and dopamine excretions decreased significantly during ITV, dopamine and noradrenaline during ITI (Dixon and Mood sign test: †$P < 0.10$; ‡$P < 0.05$; §$P < 0.01$)

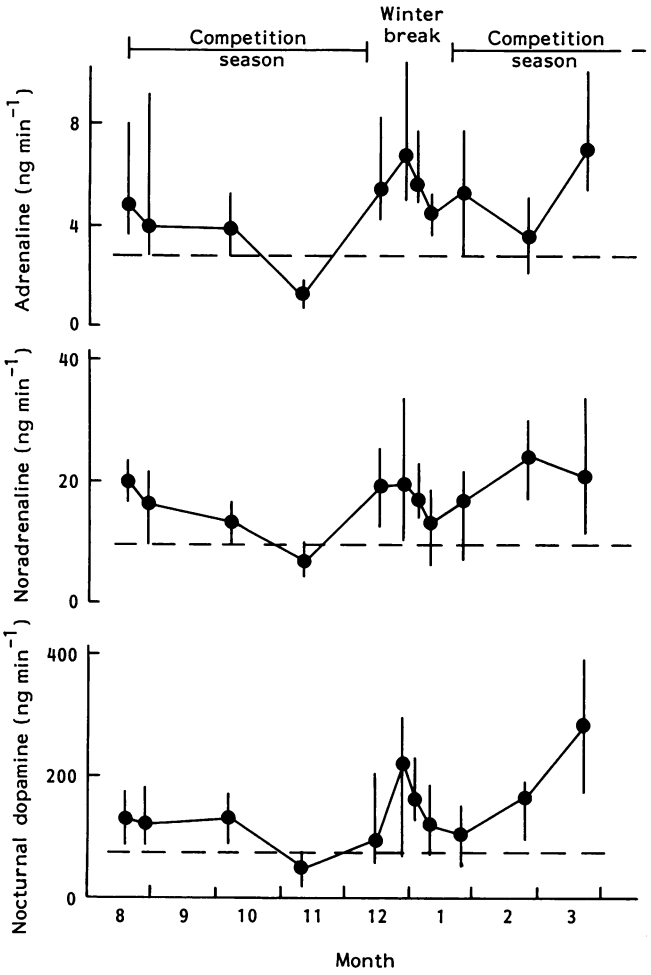

Figure 4. A significant decrease in excretion of nocturnal dopamine, noradrenaline and adrenaline was observed in 16 soccer players from August to November 1990 ($P < 0.001$). The running speed at 4 mmol lactate level was simultaneously decreased in November compared with August ($P < 0.05$) – the team was in places two and three in August–September and in places six and seven in November–December. Decrease in nocturnal catecholamine excretion (more than 50% for noradrenaline and adrenaline), in running speed at 4 mmol lactate level, and in the league rank may point to an exhaustion syndrome. During the recovery period (winter break) a reincrease in nocturnal catecholamine excretion was observed, running speed at 4 mmol lactate level remained constant and the team was in the second league rank after February 1991[28]

in catecholamine levels is not only observed when the aerobic work capacity of the muscles is improved[10–12], but also when muscular strength is increased[17,18].

The higher catecholamine level at the identical exercise rates, coupled with a lower heart rate on day 28 of the ITV study may indicate a reduced sensitivity to catecholamines in exhaustion, since the constancy of heart rate in the morning on waking contradicts a decisive increase in vagotonia. A marked decrease in sensitivity to catecholamines is thus found in experiments with long-term stimulation for hours with isoproterenol[19]. We therefore hypothesize that excessive training volume coupled with a neglect of rest and relaxation leads to a loss of sensitivity to catecholamines as an expression of 'peripheral exhaustion'. This hypothesis is still to be confirmed.

We view the more marked decrease in basal catecholamine elimination in the ITV study as an indicator of a decrease in intrinsic sympathetic activity, caused by hypothalamic dysfunction[20] as an equivalent of 'central fatigue' in exhausted athletes.

Table 6. Behaviour of hormones at baseline, interim and final examination (a) before ergometry and (b) after maximum ergometric exercise during the increase in volume study

	Baseline		*Interim*		*Final*	
	a	*b*	*a*	*b*	*a*	*b*
Cortisol (nmol l^{-1})						
Median	425	425	391	438	336*	378†
50% confidence interval	314–485	408–499	369–452	400–579	267–372	317–430
Range	201–701	284–814	322–452	257–742	168–444	267–574
Aldosterone (nmol l^{-1})						
Median	207	1559	238	1473	160*	1052*
50% confidence interval	144–407	1102–1758	182–326	1121–1941	113–263	936–1163
Range	63–581	509–2808	127–526	642–2792	36–354	517–2265
Testosterone (pmol l^{-1})						
Median	69	83	65	76	62	83
50% confidence interval	44–72	62–96	62–69	62–86	58–69	79–89
Range	41–96	51–131	44–76	44–100	44–93	55–124
Insulin (μU ml^{-1})						
Median	7	14	6	10	8	8
50% confidence interval	5–9	7–21	5–8	5–12	5–9	8–10
Range	2–13	5–40	2–17	4–36	2–14	5–18
C-Peptide (ng ml^{-1})						
Median	1.9	2.3	1.9	1.8	2.3	2.0
50% confidence interval	1.7–2.2	1.8–2.8	1.5–2.2	1.6–2.4	2.0–2.4	1.6–2.3
Range	1.4–2.4	1.4–3.6	1.0–3.0	1.4–3.4	1.5–3.2	1.4–2.4
GH (ng ml^{-1})						
Median	2.6	5.0	1.6	3.6	2.3	7.1
50% confidence interval	0.6–3.0	2.9–5.1	0.5–2.3	3.3–10.0	0.2–2.5	3.2–13.2
Range	0.1–4.0	1.8–32	0.1–9.5	2.8–19	0.1–5.1	0.2–22
Prolactin (μU ml^{-1})						
Median	117	414	119	350	115	384
50% confidence interval	92–141	344–610	89–126	309–405	97–131	262–340
Range	67–182	166–666	52–142	87–560	64–174	138–661
FSH (μU ml^{-1})						
Median	3.7	4.4	3.3	3.9	3.5	4.0
50% confidence interval	3.2–4.8	3.8–5.4	3.1–4.8	3.4–5.4	3.0–5.2	3.3–5.8
Range	1.8–6.9	2.1–8.1	2.2–7.5	2.4–7.9	1.8–6.5	2.2–6.9
LH (μU ml^{-1})						
Median	2.7	3.1	2.2	2.8	3.1	2.8
50% confidence interval	2.5–2.8	2.5–4.5	1.5–2.9	2.2–3.6	1.7–4.1	1.9–4.0
Range	1.6–4.2	1.6–5.7	1.4–5.6	1.1–4.8	1.4–4.6	1.4–5.9
TSH (μU ml^{-1})						
Median	0.97	1.3	0.87	1.02	0.87	1.25
50% confidence interval	0.9–1.1	1.0–1.9	0.7–0.9	0.9–1.3	0.7–1.1	1.0–1.5
Range	0.1–2.0	0.8–2.8	0.5–1.9	0.7–2.2	0.6–1.8	0.9–2.6
Triiodothyronine (nmol l^{-1})						
Median	1.68	—	1.68	—	1.59	—
50% confidence interval	1.53–1.83	—	1.53–1.82	—	1.37–1.68	—
Range	1.37–1.85	—	1.22–3.14	—	1.20–1.69	—
Thyroxine (nmol l^{-1})						
Median	94	—	100	—	96	—
50% confidence interval	90–105	—	97–101	—	83–100	—
Range	75–142	—	68–129	—	69–106	—

*$P < 0.10$; †$P < 0.05$; Dixon and Mood sign test

Table 7. Behaviour of hormones at baseline, interim and final examination (a) before ergometry and (b) after maximum ergometric exercise during the increase in intensity study

	Baseline		*Interim*		*Final*	
	a	*b*	*a*	*b*	*a*	*b*
Cortisol (nmol l^{-1})						
Median	262	364	215	295	320	331
50% confidence interval	237–300	292–419	168–270	218–320	259–361	300–389
Range	182–358	212–513	93–413	146–339	176–554	190–538
Aldosterone (nmol l^{-1})						
Median	113	711	191	972	240*	731
50% confidence interval	102–229	554–1094	181–282	578–933	188–301	722–908
Range	58–379	487–1811	88–725	493–2606	66–598	529–1102
Testosterone (pmol l^{-1})						
Median	69	89	69	89	65	93
50% confidence interval	55–72	83–103	62–79	83–100	62–83	86–103
Range	41–110	41–93	44–100	51–141	44–121	83–159
Insulin (μU ml^{-1})						
Median	9	13	7	12	10	11
50% confidence interval	6–12	9–19	5–10	9–17	8–18	9–18
Range	4–24	7–26	4–26	8–28	5–22	5–21
C-Peptide (ng ml^{-1})						
Median	2.2	2.8	2.2	1.7	2.2	2.1
50% confidence interval	1.3–2.6	1.7–2.6	1.1–2.4	1.6–2.4	1.8–2.8	1.9–2.2
Range	1.0–3.1	1.3–2.9	0.9–2.9	1.3–2.9	1.4–3.8	1.7–4.1
GH (ng ml^{-1})						
Median	1.9	9.0	1.0	3.3	1.3	11.5
50% confidence interval	0.4–3.5	2.0–12.0	0.3–2.0	2.8–7.4	0.6–3.8	2.9–18.1
Range	0.2–4.8	1.3–29.1	0.2–32.0	1.0–27.3	0.1–13	1.0–25
Prolactin (μU ml^{-1})						
Median	93	353	81	289	113	204*
50% confidence interval	78–135	243–423	68–100	149–338	109–116	171–368
Range	40–150	72–579	35–114	82–489	37–188	46–421
FSH (μU ml^{-1})						
Median	5.5	6.2	5.9	6.2	6.6	6.7
50% confidence interval	4.8–6.3	5.6–6.6	4.0–6.2	3.9–6.8	3.9–6.8	5.2–7.2
Range	2.0–7.5	2.2–8.0	1.9–6.7	2.1–7.2	1.5–7.2	2.0–7.9
LH (μU ml^{-1})						
Median	2.5	2.6	3.1	2.9	3.4	3.2
50% confidence interval	2.4–3.7	2.3–3.1	2.8–3.3	2.0–3.0	1.8–3.7	2.9–3.8
Range	2.0–4.0	2.1–4.8	2.1–5.3	2.0–3.8	1.5–4.9	1.8–4.0
TSH (μU ml^{-1})						
Median	0.74	1.06	0.80	1.17	0.80	0.92*
50% confidence interval	0.7–0.9	0.9–1.2	0.7–1.0	0.8–1.3	0.5–1.0	0.7–1.2
Range	0.6–1.0	0.7–1.8	0.5–1.4	0.7–1.6	0.3–1.4	0.8–1.8
Triiodothyronine (nmol l^{-1})						
Median	1.53		1.53		1.37	
50% confidence interval	1.37–1.68		1.36–1.68		1.22–1.52	
Range	1.22–1.69		1.22–1.69		1.07–1.53	
Thyroxine (nmol l^{-1})						
Median	100		96		105	
50% confidence interval	86–109		92–109		88–117	
Range	79–114		88–120		84–129	

$P < 0.10$; Dixon and Mood sign test

Table 8. 24-h urinary cortisol excretion (nmol $24\,h^{-1}$)

	Week 1	*Week 2*	*Week 3*	*Week 4*	*Week 5*
Increase in volume study					
Median	289	206*	299	281	278
50% confidence interval	245–344	184–267	264–314	184–352	267–314
Increase in intensity study					
Median	265	271	247	278	291
50% confidence interval	231–339	201–292	199–289	186–347	257–331

*$P < 0.05$; Wilcoxon Mann–Whitney *U* test

The maximum plasma catecholamine response is, however, still unchanged or even elevated (*Table 4*). A reduction in the maximum catecholamine response is assumed if overexertion continues for more than 4 weeks. In a further prospective study we recently confirmed our hypothesis, that exhaustion is accompanied by a profound decrease and recovery by an increase in the decreased basal catecholamine excretion (*Figure 4*). The clear decrease in basal catecholamine excretion and the behaviour of cortisol and testosterone in the present study do not confirm specifically the hypothesis of increased catabolism in overtrained athletes such as is assumed on the basis of the cortisol–testosterone ratio[2,21].

The different behaviour of cortisol and aldosterone levels between the two studies is probably not important in diagnosing performance since these changes are too small to be reliably differentiated from inter-assay variance in cross-sectional studies. Also to be considered are the different baseline values of the two studies, which are also most likely due to inter-assay variability. The decrease in resting and exercise cortisol concentration following ITV (*Table 6*) does not indicate decreased cortisol metabolism, since the 24-h excretion of cortisol remains constant (*Table 8*). The observed decrease in basal cortisol level (ITV) contradicts the findings of Barron *et al.*[20] and Adlerkreuz *et al.*[21]. These authors described increased 'catabolic' cortisol levels in 'overtrained athletes'. Their studies were, however, performed only on four athletes[20] or for 1 week only [21]. Additional prospective experimental data were therefore necessary.

With the exception of the slight exercise-induced decrease in prolactin during the ITI study, no performance–differential diagnostic statements could be made for the other hormonal parameters examined. This is especially true for free testosterone. In intensive training phases, a decrease in basal free testosterone of about 10% can be expected[2]. This, however, is within the range of variation of the assay method. In the present study, the median value decreased by 10% following ITV but this change was not significant. A decrease of up to 40% of the total testosterone level following exhaustive endurance exercise has been described by various authors[22–27], but not as yet for free testosterone. On the morning after 15-km and 25-km runs, the basal levels of total testosterone were nearly back to baseline values[25] but after a marathon race, the level remained reduced for 2–3 days[25]. However, it is unclear whether these results can be transferred to high-performance athletes.

Overall, it must be stated that only a possible and marginal differential diagnostic relevance was found in the parameters measured in these studies for plasma catecholamines, cortisol and aldosterone. In practice, these are probably only useful in longitudinal studies. However, the differences in basal catecholamine excretion appear somewhat clearer. Nevertheless even these results strictly apply only to the current study. To generalize further would require additional experimental studies and confirmation by other workers.

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Courses in Sports Medicine

We apologise for the misleading title to **Courses in Sports Medicine** in the September issue which gave the impression that the courses were run by BASM which is not the case.

1. British Association of Sport and Medicine

One week/weekend introductory and advanced courses in sports medicine for medical practitioners and physiotherapists.

Contact: Ms Nancy Laurenson, BASM Education Officer, London Sports Medicine Institute, c/o Medical College of St. Bartholomew's Hospital, Charterhouse Square, London EC1M 6BQ, UK.
Tel: 071-253 3244 and 071-251 0583;
Fax: 071-251 0774

2. London Sports Medicine Institute

Three-year part-time course in sports medicine for general practitioners.

Contact: Academic Secretary, London Sports Medicine Institute, c/o Medical College of St. Bartholomew's Hospital, Charterhouse Square, London EC1M 6BQ, UK. Tel: 071-251 0583; Fax: 071-251 0774

3. The London Hospital Medical College

One-year full-time diploma course in sports medicine for medical practitioners.

Contact: Mrs Dot Blake, The Diploma Course in Sports Medicine, Department of Sports Medicine, London Hospital Medical College, 1st Floor Fielden House, Stepney Way, London E1 1BB, UK.
Tel: 071-377 7389

4. University of Nottingham Medical School

Two-year part-time MSc in sports medicine for medical practitioners and chartered physiotherapists with a first degree or equivalent.

Contact: Professor E. Idris Williams, Department of General Practice, The Medical School, Queens Medical Centre, Nottingham NG7 2UH, UK.
Tel: (0602) 709396; Fax: (0602) 709389

5. The University of Bath

A modular course in sports medicine by distance learning for medical practitioners

Contact: Mrs Sally Jeffries, Distance Learning Unit, Centre for Continuing Education, University of Bath, Claverton Down, Bath BA2 7AY, UK.
Tel: (0225) 826342; Fax: (0225) 826849

6. Association of Chartered Physiotherapists in Sports Medicine

Six-month practical course leading to certificate in sports physiotherapy and a two-year part-time academic course leading to a diploma in sports physiotherapy.

Contact: Dr Ian Roberts, Assistant Director, Crewe and Alsager College of Higher Education, Hassall Road, Alsager, Cheshire ST7 2HL, UK. Tel (0270) 882500

7. Diploma in Academic and Practical Physiotherapy for Sports

One-year part-time course in sports medicine/physiotherapy for chartered physiotherapists.

Contact: Joanne Marshall, Department of Sports Medicine, London Hospital Medical College, 1st Floor Fielden House, Stepney Way, London E1 1BB, UK.
Tel: 071-247 7636

8. Edinburgh Post-Graduate Board for Medicine

One-week introductory course in sports medicine for doctors and physiotherapists.

Contact: Dr Elizabeth McSwan, Moray House College of Education, Cramond Campus, Cramond Road North, Edinburgh EH4 6JD, UK.
Tel: 031 3126001

9. Diploma in Podiatric Sports Medicine

Two-year part-time course in sports podiatry.

Contact: Dr Ian Roberts, Assistant Director, Crewe and Alsager College of Higher Education, Hassall Road, Alsager, Cheshire ST7 2HL, UK.
Tel: (0270) 882500