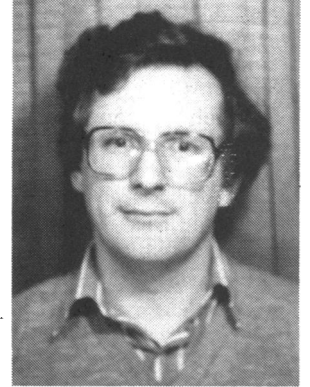


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ENDOCRINE RESPONSES TO MARATHON RUNNING

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ABSTRACT

We have examined the hormone status of ten healthy adult males who completed the Glasgow marathon. Serum thyroxine, free thyroxine, triiodothyronine and thyroid stimulating hormone were unaffected by the exertion. Serum cortisol as well as other steroid hormones, androstenedione, dehydroepiandrosterone sulphate and oestradiol showed distinct rises ($p < 0.01$) but serum testosterone fell significantly ($p < 0.01$). There was no change in sex hormone binding globulin capacity but a significant fall in LH levels ($p < 0.05$) accompanied the fall in serum testosterone. The mechanism of the fall in serum testosterone in this situation requires clarification but it is unlikely to have adverse consequences.

Key words: Hormone, Testosterone, Exertion, Marathon.

INTRODUCTION

Marathon running is becoming increasingly popular and whilst it is generally thought that exercise is a healthy pastime, the consequences of extreme physical exertion remain to be established.

Physical effort is known to alter blood levels of many hormones, in particular those increased by stressful situations such as growth hormone, prolactin, catecholamines and adrenocortical steroids. The effect of exercise on the pituitary-testicular-axis is controversial. Serum testosterone has been found to rise, remain unchanged or fall after different forms of exertion (Sutton et al, 1973; Kindermann et al, 1982; Dessypris et al, 1976). Other forms of physical or mental stress (Kreuz et al, 1972) have also been found to lead to a fall in testosterone levels. Findings with regards to the pituitary-thyroid axis have also been inconsistent with one group finding a small increase in thyroxine and accompanying fall in triiodothyronine after exercise (O'Connell et al, 1979) while another (Refsum and Strömme, 1979) found an increase in thyroxine, triiodothyronine and thyroid stimulating hormone.

Because of the increasing interest in Marathon running we decided to attempt to clarify this confusing situation by measuring various endocrine parameters before and after a

marathon. [Part of this data has previously been presented to the 1984 meeting of the British Endocrine Societies (C. Semple et al, 1984) but not previously published except as an abstract in Proceedings.]

MATERIALS AND METHODS

Ten healthy adult males (age range, 23-49, mean 33.5 years) gave informed consent to this study. Five were medical or paramedical personnel, five were volunteers from a local business. Ethical committee permission had been obtained. All had been in regular training for at least three months prior to the Glasgow marathon. Due to difficulties in sampling just before the race and also to eliminate the effects of diurnal hormonal variation, pre-race samples were withdrawn during the week before the race at a time considered to be the likely finishing time for the race. Fluids were freely available en route and weather conditions were cool. Repeat blood sampling was performed within 30 minutes of completing the race (range 6-30, mean 20 minutes).

Each pair of samples was assayed for the following:—

Thyroxine, triiodothyronine, free thyroxine, thyroid stimulating hormone, testosterone, sex hormone binding globulin, follicle stimulating hormone (FSH), luteinising hormone (LH), prolactin, cortisol, oestradiol, androstenedione, dehydroepiandrosterone sulphate and microhaematocrit. Blood was centrifuged and serum stored at -20°C prior to assay. Each pair of samples was assayed in the same hormone system to reduce inter-assay variation. Statistical analyses were performed using the Wilcoxon Rank Test for paired data.

Serum thyroxine and triiodothyronine were estimated by radioimmunoassay (RIA) using methods in which the primary antisera are coupled to microfine cellulose. Free thyroxine was measured with an "Amerlex" (Amersham International) kit,

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which uses an analogue binding principle. Serum thyroid stimulating hormone was determined with a two site immunoradiometric assay based on two different polyclonal sheep anti-human thyroid stimulating hormone sera. All these assays performed with intra-assay and inter-assay coefficients of variation of < 8% and < 10% respectively.

Testosterone, oestradiol and androstenedione were estimated by RIA in ether extracts of serum. Dehydroepiandrosterone sulphate was measured directly in diluted serum. Details of all these methods have been described previously (Grant and Beastall, 1983). The assays all perform with intra-assay and inter-assay coefficients of variation of < 8% and < 12% respectively. Serum cortisol was measured by a standard fluorimetric assay (Mattingly, 1962).

Serum FSH, LH and prolactin were quantitated with conventional double antibody RIA's. The FSH and LH assays employ reagents recommended by the National External Quality Assessment Scheme and are standardised against MRC 78/549 and MRC 68/40, respectively. The serum prolactin assay has been described in detail elsewhere (Cowden et al, 1979). These assays perform with intra-assay and inter-assay coefficients of variation of 8% and 12% respectively. The sex hormone binding globulin capacity of serum was determined by a method based on saturation with H_3 -dihydrotestosterone (Rosner, 1972). The microhaematocrit was measured using the Hawksley method after centrifuging samples at 13,000G for five minutes.

RESULTS

All 10 runners completed the race in reasonable physical condition (mean duration 232, range 182-258 minutes). There was no significant difference between the microhaematocrit (mean \pm SD) before ($46.6 \pm 2.5\%$) and after the race ($45.9 \pm 1.14\%$) suggesting no significant change in plasma volumes. All basal hormonal values were within the normal range with the exception of a raised prolactin level in one individual.

Serum thyroxine, triiodothyronine, free thyroxine, and thyroid stimulating hormone were not altered by Marathon running (Table I).

TABLE I
Thyroid function tests (Mean \pm SD)

(Normal range)	Pre	Post
Thyroxine (55-144 nmol/l)	111.3 \pm 14.4	118.4 \pm 22.3
Triiodothyronine (0.9-2.8 nmol/l)	2.50 \pm 0.13	2.58 \pm 0.35
Free thyroxine (9-25 pmol/l)	17.8 \pm 2.9	18.0 \pm 2.9
Thyroid stimulating hormone (0.5-0.5 mU/l)	2.49 \pm 0.55	2.59 \pm 0.97

All the adrenocortical steroids which were measured (Fig. 1) increased significantly ($p < 0.01$). Serum cortisol rose from 338 ± 147 (range 160-580) to 1640 ± 884 nmol/l, (range 650-3800), androstenedione from 3.64 ± 0.98 (range 1.9-5.1) to 7.11 ± 2.03 nmol/l (range 3.7-10.0), dehydroepiandrosterone sulphate from 6.70 ± 3.20 (3.1-12.0) to 9.83 ± 5.27 μ mol/l (4.7-17.0) and oestradiol from 192 ± 74 (100-300) to 222 ± 52 pmol/l (150-280). In contrast serum testosterone (Fig. 2) decreased from 18.5 ± 5.0 (10.0-25.0) to 14.9 ± 3.6 nmol/l (8.8-21.0) ($p < 0.01$). This fall was not accompanied by any change in sex hormone binding globulin capacity (30.5 ± 8.7 (15-46) v 30.0 ± 7.1 (17-40) nmol/l). LH however fell from 4.07 ± 1.91 (1.5-7.1) to 2.53 ± 1.19 (1.0-5.1) U/l ($p < 0.05$) but FSH showed no significant change (3.72 ± 1.44 (2.0-6.6) v 3.39 ± 1.19 (2.1-5.2) U/l). Serum prolactin rose from 139.8 ± 100.8 (90-420) to 333.5 ± 283.6 (95-1100) mU/l ($p < 0.01$).

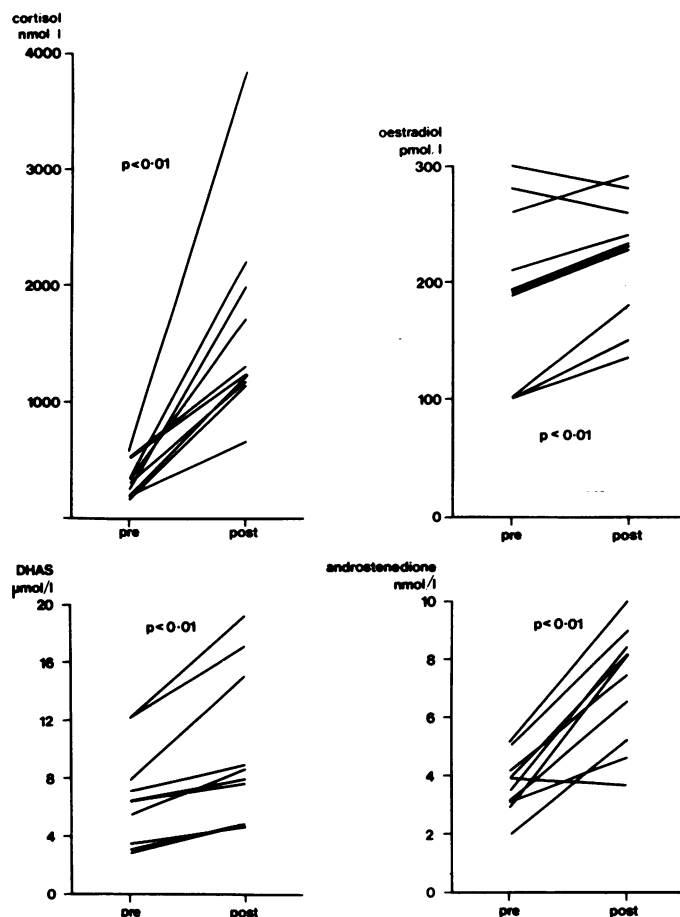


Fig. 1: Serum levels of cortisol (330-770 nmol/l), oestradiol (undetectable-300 pmol/l), dehydroepiandrosterone sulphate (2.9 μ mol/l) and androstenedione (2-11 nmol/l) before and after the marathon (normal ranges in brackets).

DISCUSSION

Our failure to find any change in thyroid hormone levels following exercise conflicts with the findings of O'Connell et al (1979) who found a small increase in thyroxine and a decrease in triiodothyronine following moderate bicycle exercise in four males. Refsum and Strømme (1979) found increases in thyroxine, triiodothyronine and thyroid stimulating hormone after cross-country skiing. It would appear unlikely that changes in thyroid hormone status play any significant role in the metabolic response to physical effort lasting several hours.

Our finding of a reduction in LH and testosterone levels suggests hypothalamic-pituitary impairment following marathon running. This finding is in agreement with the study of Hale et al (1983) who found a reduction in LH levels in females following a marathon run. Our study was limited to the observation of single pre- and post-race levels. In view of the known fluctuation in LH levels and in particular the pulsatility of LH release, serial sampling throughout the race would have been a more physiological way of studying hypothalamic-pituitary function. However, our volunteers would not have agreed to serial sampling and indeed such a study would not be practical within the context of a competitive marathon run.

Serum testosterone has been found to fall in a variety of stressful situations. Myocardial infarction (Wang et al, 1978a) and surgical procedures under general anaesthesia (Wang et al, 1978b) induce a fall accompanied by an increase in LH levels in keeping with a direct effect on the testes. In contrast patients with an exacerbation of chronic obstructive airways disease have low testosterone levels with low LH levels as evidence of impaired hypothalamic-pituitary function (P. Semple

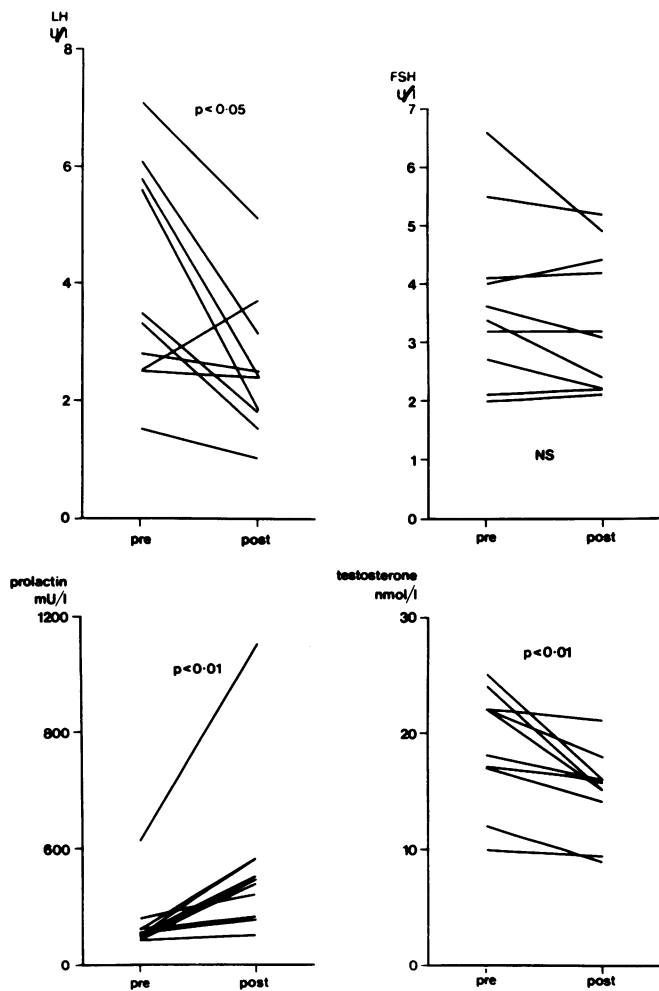


Fig. 2: Serum levels of LH (undetectable-8.6 U/l) FSH (undetectable-6.4 U/l), prolactin (60-360 mU/l) and testosterone (11-36 mU/l) before and after the marathon.

et al, 1981). Oestradiol in the male is derived partly from peripheral conversion from adrenal androgens. The rise in this hormone after exercise may reflect enhanced interconversion from an increased pool of adrenal androgens.

Elevated glucocorticoid levels are known to exert a suppressive effect on the hypothalamic-pituitary-testicular axis (Sakakura et al, 1975). However it has been suggested that glucocorticoids might also exert a direct inhibitory effect on the testes thus accounting for the findings after surgery and myocardial infarction (Doerr and Pirke, 1976). Cumming et al (1983) found that the stress following insulin induced hypoglycaemia was accompanied by a fall in testosterone without any change in LH or prolactin. Moreover an intravenous injection of hydrocortisone also caused a fall in serum testosterone with no change in LH levels. Thus glucocorticoids can exert a direct inhibitory effect on the testes. However, in the first of these experiments the stress attributable to hypoglycaemia only caused a twofold increase in serum cortisol, prolactin levels being unchanged. In contrast after this Marathon cortisol levels rose five fold and prolactin levels doubled. It seems probable that this reflects increased secretion due to the extreme nature of the stress which occurs during a marathon. An alternative explanation might be that elimination of these hormones is reduced during exercise. However, in the case of cortisol, elimination has been shown to be increased during exercise (Few, 1974). Such high cortisol levels might be sufficient to suppress LH release as well as having a direct inhibitory effect on the testes. No doubt there are other possible causes of pituitary-testicular axis suppression during prolonged vigorous exercise. Thus endogenous opioid peptides

which increase with exercise (Farrell et al, 1982) are known to exert an inhibitory effect on LH release (Morley et al, 1980); such a mechanism might well have contributed to the fall in LH levels seen in this study. We feel that it is unlikely that the modest hyperprolactinaemia seen in this study would have an antagonodotrophic effect.

Although we have observed a fall in LH and testosterone levels following marathon running it is difficult to envisage any important physiological consequences. Although all subjects showed a fall in serum testosterone, changes were relatively modest. Only two subjects whose pre-race levels were around the lower limit of normal had subnormal post-race levels (8.8, 10.0, lower limit of normal 11.0 nmol/l). It has been proposed that such a reduction might have biological implications for the reproductive adaptation to stress (Cumming et al, 1983). However, male sexual activity correlates rather poorly with serum testosterone and lack of libido following a marathon could be more logically attributed to exhaustion than to a small fall in serum testosterone.

ACKNOWLEDGEMENTS

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BOOK REVIEW

Title: KORONARERKRANKUNGEN (In German)
Editor: H. Roskamm
Publisher: Springer-Verlag, Berlin
 Price \$188.20 DM 480 1386 pages

The volume "heart diseases" is part of the handbook of internal medicine and probably the most revised part of the three volumes "heart and circulation". Since the last edition 25 years have passed, during which time diagnostic techniques and conservative and surgical therapy of coronary diseases have fortunately made great advances, which are discussed in the 46 chapters. After the introductory chapters about physiology and pathophysiology of coronary blood flow and pathological anatomy of coronary insufficiency the epidemiology of the risk factors (including the psychosocial aspects) are described, followed by the discussion of the functional diagnostic methods (e.g. the different ECG-possibilities, X-ray methods, computer-tomography, echocardiography, methods using radio isotopes and different heart catheterisation methods including coronary angiography and ventriculography). This part not only describes technical details, but also informs about the methodological value of diagnosis and prognosis of coronary heart disease.

The surgical methods (aortocoronary bypass, aneurysmectomy, intra-aortal pump and heart transplantation) are also presented. Problems of re-establishment of coronary patients into their jobs are described in the last chapter. Every chapter finishes with some hundred references from international literature.

The interest group therefore includes all physicians working on cardiology in hospitals in consultant practice and sports physicians who may be interested in cardiac rehabilitation. For them the topics "early mobilisation after myocardial infarction and coronary operation", "movement therapy" and "prevention and therapy of cardiac incidents" are of particular importance. The heart patients who take part in rehabilitation programmes are divided into two groups: (a) those who are able to perform more than 75 watts exercise at a higher level with an increasing adaptation effect to most organs including their oxygen supply. (b) the less efficient performing group who exercise predominantly neuromuscular co-ordination in order to economise movements.

An important topic of movement therapy is the prevention or rather the positive influence on cardiovascular risk factors. The reader will find partly contradictory epidemiological and experimental investigations of the past 20 years critically evaluated, and trends are formulated (e.g. for the hyperlipoproteinaemia or moderate hypertension). Other topics still under discussion are clearly analysed in regard to the different points of view: e.g. the slowly ascending ST-segment in the exercise ECG or the therapy of cardiac patients with anticoagulants. There is, however, no discussion about the potentiality of two risk factors with women: oral contraceptives and smoking, which increase the risk of myocardial infarction by a factor 10 and 40 respectively.

The handbook character of the book in question is guaranteed on one side by the complete reversion of the topic "coronary diseases" by a team of more than 50 well-known specialists and on the other side by the number of references. But nevertheless this book is no reference book. The physician who in practice is confronted with cardiological questions will successfully find an answer in this book which is easily understandable and divided into small chapters and he probably wants to read more – as the reviewer did – as would be necessary to answer the question.

E. D. Lübs, Professor of Cardiology, University of Kassel, Germany

BOOK REVIEW

Title: MANUAL OF STRUCTURAL KINESIOLOGY
Author: Clem W. Thompson
Publishers: Times Mirror/Mosby College Publishing, 1985, 10th Edition
 Price: £19.50 146 pages Index Softback ISBN 0-8016-4952-8

The first edition of this book was published nearly forty years ago so its relevance and popularity in the field of basic kinesiology are well established. One joint of the body is considered at a time, along with an illustration of each of its accompanying muscles. The muscles are then grouped together as prime movers and typical functional activities delineated. This treatment prevents students becoming so engrossed in learning individual muscles that they lose sight of the total muscular system. Only about a hundred of the largest and most important prime movers are considered so the appeal is towards the physical educator and coach rather than the anatomist and physician.

Photographs, colour plates and useful Student Objectives provide a more attractive format than previous editions with additional details of multigym and isokinetic machine extending the modern range of applied training methods.

It is surprising that the "rotator cuff" muscles do not find a place in the Index and the author has still to find a cogent method of illustrating the serratus anterior muscle, admittedly a difficult problem. It is also hard to imagine a woman performing an unmodified pull-up with so little apparent strain and using an incorrect grip. Apart from these minor cavils, this is an excellent edition, well up to former standards, if somewhat overpriced.

D. H. Williams