Endocrine Changes in Relaxation Procedures

by

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To

My Father and Mother

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SUMMARY

Four studies were designed to examine possible endocrine bases for the effects of relaxation procedures on stress:-

(1) Long-term plasma cortisol, urinary free cortisol (UFC), total urinary catecholamines (UCA), serum thyroxine (T_4) and serum triiodothyronine (T_3) changes were investigated on subjects who underwent seven months' training in Transcendental Meditation (TM), progressive relaxation, autohypnosis-relaxation and yoga-meditation. Untrained controls and experienced meditators (> 1 year's TM training) were studied concurrently.

(2) Short-term plasma cortisol, serum T_4 and serum T_3 changes in relaxation procedures were studied, using both relaxation trainees (novices) from the long-term study, as well as untrained controls and experienced meditators.

(3) In the third and fourth studies, TM was used as a model relaxation procedure.

A second short-term study was conducted to investigate both serum cortisol and UFC, UCA, serum human growth hormone (hGH), prolactin, total T_4, free T_4 index, total T_3 and reverse T_3 changes during TM. Quiet rest in re-studied experienced meditators, as well as ordinary relaxation of untrained subjects, were used as controls.

(4) The fourth study was performed during a week-end TM residence course, on subjects practising intensive meditation.

No long-term endocrine changes related to relaxation training were apparent for any group.

Significant TM-induced reductions of both circulating and urinary cortisol levels were observed in trained subjects, in contrast to a lack of significant cortisol change in relaxation of both relaxation trainees and untrained controls. Similarly, a significant reduction in UFC excretion of experienced meditators was found during the TM residence course, whereas no significant change occurred in novice subjects.

hGH and prolactin also fell significantly during TM of experienced meditators, in contrast to a lack of significant change during both ordinary relaxation of untrained controls and quiet rest of experienced meditators. The hGH reduction commenced before the onset of TM and appeared to be a response in anticipation of meditation (phase 1), which was sustained during the meditation period (phase 2). hGH levels subsequently returned to pre-meditation values (phase 3).

The triphasic hGH response pattern in TM is the inverse of that found in anticipated acute stress, and the TM-induced hGH and prolactin changes are distinct from those occurring during the sleep-wake cycle.

A significant reduction of serum T_3 was observed in TM, however, it was not clear whether the change was mediated by alterations in thyroidal secretion or by peripheral mono-deiodination of T_4. No significant changes in either packed cell volume or total serum protein were found.

Lower pre-meditation serum cortisol and daily UFC levels were observed in experienced meditators than in matched non-meditator controls, and appeared to be the result of:- 1) TM-induced and ordinary rest-induced cortisol reductions and 2) decreased cortisol stress responsiveness. Both 1) and 2) are apparently cumulative with increased meditation experience.

The lack of significant UCA changes during both meditation and the TM residence course, along with the higher daily catecholamine excretion of meditators than matched non-meditator controls, challenges the notion of a generalised decrease of sympatho-adrenomedully function in relaxation procedures. The TM-induced reduction of cortisol, hGH and prolactin, three hormones which increase with stress, might be mediated by alterations in hypothalamo-anterior pituitary function.

DECLARATION

This is to certify that the material contained in this thesis has not been accepted for the award of any other degree or diploma and is the work of the author except where otherwise acknowledged.

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ABBREVIATIONS

AHR	autohypnosis-relaxation
ANS	8-anilino-1-naphthalene sulphonic acid-sodium salt
BSA	bovine serum albumin
CBG	cortisol binding globulin
DAS	donkey anti-sheep-goat
DIT	diiodothyronine
EDTA	di-sodium ethylenediaminetetra-acetic acid
EEG	electroencephalographic
FFA	free fatty acids
FTI	free thyroxine index
GARGG	goat anti-rabbit gamma globulin
hGH	human growth hormone
HSA	human serum albumin
IFW	ion-free water
LH	luteinizing hormone
MIT	monoiodothyronine
NA	noradrenaline
NAp	not applicable
NAv	not available
NS	not significant
NSS	normal sheep serum
PBS	phosphate buffered saline
PCV	packed cell volume
PR	progressive relaxation.
rT_3	reverse triiodothyronine
TETRAC	tetraiodothyroacetic acid
T_4	thyroxine
THFS	thyroid hormone free serum

THS	thyroid stimulating hormone
TM	Transcendental Meditation
TRIAC	triiodothyroacetic acid
T_3	triiodothyronine
T_3SU	T_3 sephadex uptake
UCA	urinary catecholamines
UFC	urinary free cortisol
VMA	vanillyl-mandelic acid
YM	yoga-meditation

DEFINITIONS OF RELAXATION EXPERIENCE OR TRAINING

Untrained control or non-meditator (control):-	no specific relaxation or meditation training
Novice or in-training subject or meditator; relaxation or meditation trainee:-	< 1 year's practice
Experienced or trained subject or meditator:-	> 1 year's regular (10-14 TM/wk) practice
Long-term meditator:-	1-5 years' regular TM practice
Advanced meditator:-	> 5 years' regular TM practice; often with advanced technique(s) (See p 168)

CHAPTER I : INTRODUCTION

A. Psychophysiological Definition of Stress

Psychophysiological stress is best defined in terms of both stimulus and response (Patkai, 1974). According to Selye (1975; p 27) "stress is the non-specific response of the organism to any demand made upon it". Although adrenocortical activation (Selye, 1946) was central to Selye's definition, the "non-specific response" of stress involves an integrated hypothalamic response (Smelik, 1970), and may also be measured by activation of cerebrocortical and autonomic functions, emotional and behavioural arousal, as well as metabolic and endocrine hyperactivity (Patkai, 1974). However, affective arousal, as characterised by adrenocortical and sympatho-adrenomedullary activation (Greene et al, 1970; Brown and Heninger, 1975; Pinter et al, 1975), appears to be the most non-specific aspect of the stress response.

An important aspect of stress is its generality; that is, it is concerned with changes which arise in response to a variety of stimuli (Selye, 1956; Oken, 1967). Stressors are multidimensional stimuli consisting of both physical and psychological components (Mason, 1971). The physical component is greater in the more severe stressors such as physical trauma, surgical operation, acute infection, and extreme physical exercise. Stimuli which communicate potential threat to psychophysiological homeostasis constitute purely psychogenic stressors because they induce a stress response in anticipation of damage (Wolff, 1953; pp 10, 14 and 15; Patkai, 1974). Psychogenic stressors include military combat (Bourne, 1969), scholasitc examinations (Bliss et al, 1956; Bogdonoff et al, 1960; Baseer and Rab, 1975; Kujalova et al, 1976), interviews (Hetzel et al, 1955; Kurokawa et al, 1977), public speaking (Somerville et al, 1971), motor racing (Frost et al, 1951), aeroplane flights (Hale et al, 1971; Carruthers et al, 1976), violent or amusing

films (Wadeson et al, 1963; Levi, 1965), game playing (Patkai, 1971), and everyday domestic or work situations (Rahe et al, 1974; Levi, 1964). Venepuncture or cannulation, as with other invasive or surgical procedures, involves a combination of both psychic and physical components. In such stressors, it is difficult to isolate the effects of individual constituents. Stressors are characterised by the production of intense emotional arousal which may be either pleasant or unpleasant in quality (Levi, 1965; Patkai, 1970). Conditioning factors (Selye, 1975) act as intermediaries between stressor and response, and are responsible for individual variation in both degree (Oken, 1967) and patterning (Lacey, 1967) of response.

B. Endocrine and Metabolic Adaptation to Stress

Although differential endocrine changes often occur in stress (Curtis et al, 1960; Mason et al, 1961; Okada et al, 1972; Natelson et al, 1974), the overall adaptive response involves increased secretion of catabolic hormones[1] with subsequent mobilisation of metabolic energy reserves (Johnston, 1975; Freeman, 1975). The stress response functions as a survival mechanism in life-threatening situations. The adaptive response to stress has been termed the emergency reaction (Cannon, 1914) (popularly called the fight or flight response) and the alarm reaction (Selye, 1936), and involves elevated catecholamine and cortisol levels, respectively (Mason, 1968a; 1968b). Increased human growth hormone (hGH) and/or prolactin secretion is also often associated with stress (Greenwood and Landon, 1966; Schalch and Reichlin, 1968; Noel et al, 1971, 1972; Kurokawa et al, 1977). Thyroid changes in stress are more controversial (Mason, 1968c) and often difficult to interpret because of peripheral effects (Chan et al, 1978). Nevertheless, increased thyroid function during psychogenic stress has been observed in several studies (Hetzel

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1. Anabolic hormones, such as testosterone, decrease in stress (Rose, 1969; Kreuz et al, 1972; Nakashima et al, 1975).

et al, 1952; Tingley et al, 1958; Levi, 1972; p 130; Mason, 1968c). A diagrammatic representation of these and other hormonal-metabolic changes in stress are given in figure 1.

In the absence of threat to homeostatis, hormone levels are maintained within set limits which are known as the normal basal resting range. Such hormone values are referred to as "non-stress" levels in the following text. Elevation of a hormone's concentration above the upper limit of its basal resting range constitutes stress, with respect to that particular hormone (Ruff and Korchin, 1967).

C. The Aetiology of Stress-related Diseases

It is popularly theorized that the present-day inappropriateness of phylogenetically ancient paleocortical-hypothalamic responses is the fundamental cause of stress-related diseases (Hamburg, 1962; Folkow and Neil, 1971; p 344; Benson et al, 1978a). Often social situations require supression of the somatomotor component of the stress response in order to maintain harmoneous social relationships. Meanwhile the underlying neuroendocrine preparation for action still continues. According to this theory, the consequent dissociation of somatomotor and neuroendocrine components is responsible for the development of stress-related diseases (Hamburg, 1962; Folkow and Neil, 1971; p 348). Furthermore, it has been suggested that repeated psychogenic activation of limbic function, in response to the many challenges of our rapidly changing technological society, might permanently increase levels of arousal. The hypothesised consequence is the disturbance of protective homeostatic mechanisms, thereby predisposing the organism to the harmful effects of stress (distress[2]) (Levi, 1972; pp 12 and 13; Charvat et al, 1964; Folkow and Neil, 1971; p 348; Hinkle, 1974; Benson et al, 1975; Patel, 1976). However, evidence in support of these theories is either anecdotal

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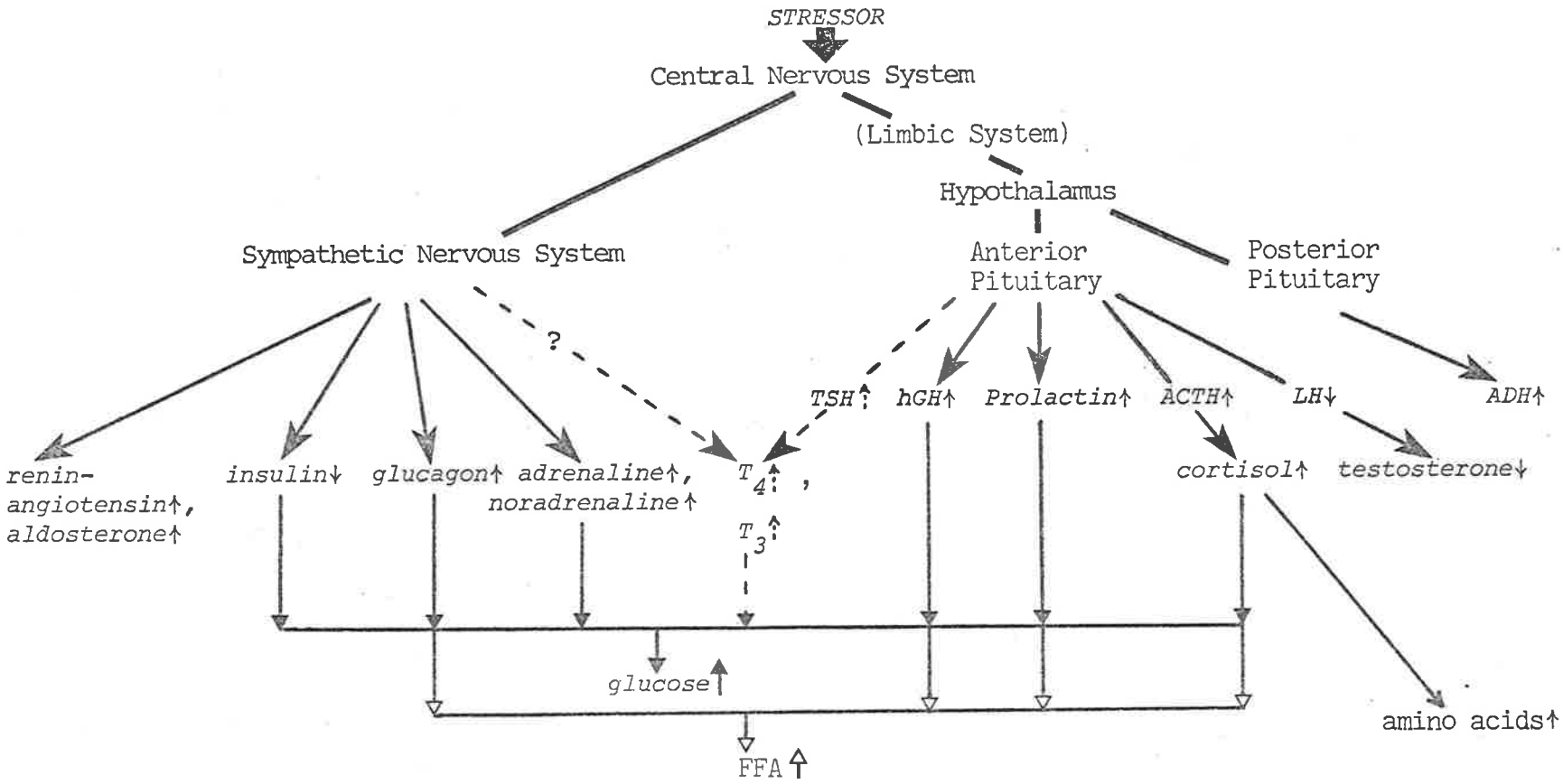
2. Selye (1975) has adopted the term distress to describe the harmful effects of stress which, presumably, arise when the demands on the organism exceed its capacity for homeostasis.

FIGURE 1. DIAGRAMMATIC REPRESENTATION OF HORMONAL-METABOLIC RESPONSES IN STRESS



As well as the references given in the text (pp 2 and 3), this information was derived from the following sources:- Ganong, 1975; pp 159, 160, 298, 263, 343 and 287; Harper, 1975; pp 492 and 493; and Zilva and Pannall, 1979; p 183. (TSH: thyroid stimulating hormone; LH: luteinizing hormone; FFA: free fatty acids).

(Gutmann and Benson, 1971; Backus and Dudley, 1974), or extrapolated from studies involving repeated arousal of rats by direct electrical stimulation of the hypothalamus (Folkow and Rubinstein, 1966). Therefore, although psychogenic stress can synergistically interact with other conditioning factors, the extent of its role in the aetiology of stress-related diseases is conjectural (Backus and Dudley, 1974; Friedman and Iwai, 1977; Esler, 1977). In direct contrast, psychogenic therapeutic intervention in stress-related disorders has been well documented.

D. Application of Relaxation Procedures

With the ever increasing incidence of stress-related diseases in our society, the need for methods of counteracting the harmful effects of stress has become more apparent. In recent years passive[3] self-regulation strategies, such as psychogenic relaxation training (Lazarus, 1974), have gained popularity because of their success in the treatment of stress-related disorders and in the management of stress reactivity. Relaxation procedures have been shown to be beneficial in the treatment of essential hypertension (Luthe and Schultz, 1969, Vol 2, pp 69-76; Jacob et al, 1977; Frumpkin et al, 1978; Benson et al, 1978a; Frankel et al, 1978; Black, 1979), headache (Luthe and Schultz, 1969, Vol 2, pp 83-89; Beaty and Haynes, 1979; Hutchings and Reinking, 1976; Benson et al, 1974b) and insomnia (Woolfolk et al, 1976; Freedman and Papsdorf, 1976), and are of special importance where conventional methods have proved ineffectual[4], or where deleterious side-effects arise from pharmacological management. Furthermore, relaxation procedures may also be useful for prevention

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3. Methods for self-regulation of stress are either active or passive, involving externally or internally orientated behaviour, respectively (Lazarus, 1974).

4. Relaxation procedures may also be used as a therapeutic adjunct to conventional treatment of stress-related diseases, in particular essential hypertension (Patel, 1975; Blackwell et al, 1976; Black, 1979).

of stress-related disorders, especially in predisposed persons (Stoyva, 1976; Davidson and Schwartz, 1976; Patel and Carruthers, 1977; Benson, 1977), and in generally eliminating distress.

(a) Glossary of relaxation procedures

Amongst the most commonly used relaxation procedures are Transcendental Meditation (TM); hypnosis-relaxation; autogenic training; progressive relaxation[5]; and various yoga-meditation techniques, including Buddhist meditation exercises such as Zen, and hatha yoga[5] (commonly called yoga).

Transcendental Meditation (Maharishi Maheshi Yogi, 1966; 1969): A meditation technique involving systematic and passive replacement of thoughts by a mantram (or mantra) until mental activity ceases (is transcended). Mantra are easily repeatable, sonorous and flowing Sanskrit words.

Hypnosis-relaxation: Hypnosis is a psychogenically induced artificial state characterised by increased suggestibility (Gorton, 1949; p 317). Hypnosis-relaxation requires specific induction procedure(s) in which the dominant suggestions are aimed at producing deep generalised relaxation.

Autogenic training (Luthe and Schultz, 1969): A system of medical therapy based on six standard exercises:- Exercises 1 and 2 involve cultivation of sensations of heaviness and warmth, respectively. The feelings are initiated in the limbs and subsequently spread throughout the body. Exercise 3 deals with cardiac regulation and exercise 4 involves passive concentration on the natural breath. In exercise 5 a sensation of warmth is cultivated specifically in the upper abdomen. Exercise 6 engenders a feeling of coolness in the forehead.

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5. These procedures involve a somatic component as well as a cognitive component (Davidson and Schwartz, 1976).

Progressive relaxation: A method of systematically producing widespread muscle relaxation. Progressive (step by step) relaxation of each major muscle group is achieved by passively concentrating on muscle tension. Muscle contraction followed by relaxation is often used to cultivate an awareness of muscle tension (Jacobson, 1938).

Hatha-yoga: One of India's traditional systems of yoga (spiritual union) which encorporates the use of yogāsanas:- body postures especially designed for gentle and controlled stretching and massage of the skeletomusculature. Meditation techniques involving passive concentration on body sensations and/or on the natural breath are often an integral part of hatha-yoga (Iyengar, 1975; Goswami, 1971).

(b) Influences of relaxation procedures on responses to stress

Reduced cortisol stress reactivity has been associated with the practice of both autogenic training (Alnaes, 1966) and TM (Michaels et al, 1979). Decreased responsiveness of heart rate and skin conductance to cold-stress has been induced by hypnosis (Barber, 1971; p 233), and subjects in an autogenic state of relaxation showed fewer galvanic skin responses to electrical shock (Luthe, 1970; p 91). Diminished neuroticism and increased confidence have also been reported in cadet parachutists who were given autogenic training (Reshetnikov, 1978), and relaxation techniques were recently reported to be effective in reducing patient discomfort during dental treatment (Corah et al, 1979a; 1979b). Furthermore, TM practitioners were found to maintain more frontalis muscle relaxation than matched non-meditator controls, during performance of a mental task (Sultan, 1975). More rapid recovery from stress has been found in subjects with specific relaxation training (Orme-Johnson, 1973; Goleman and Schwartz, 1976; McDonagh and Egenes, 1977; Miskiman, 1977a).

E. Physiological Characterisation and Definition of Relaxation Procedures

The term "relaxation response" was proposed by Benson et al (1974a) to describe the common physiological response underlying various relaxation procedures. Decreased metabolic rate, as assessed by reduced oxygen consumption, was used as a fundamental criterion of the relaxation response (Benson et al, 1974a; Beary et al, 1974; Benson et al, 1978b). According to Jacobson (1938; p 29), the removal of residual muscular tension distinguished the effects of progressive relaxation from changes seen in ordinary relaxation, and decreased oxygen consumption in relaxation is probably secondary to diminished muscle tone (De Vries et al, 1976; Fenwick et al, 1977). However, these changes are not peculiar to specific relaxation procedures because similar changes were observed during ordinary relaxation of untrained subjects (Fenwick et al, 1977). Furthermore, complete muscular relaxation may occur in anxiety states (Davidson and Schwartz, 1976), and reduced anxiety is typically associated with relaxation procedures (Paul, 1969; Shapiro and Giber, 1978; Zuroff and Schwarz, 1978; Benson et al, 1978c).

Heart rate and galvanic skin resistance are also commonly used physiological measures of relaxation induction, however, decreases in both heart rate (Travis et al, 1976; Michaels et al, 1979) and skin conductance (Schwartz, 1973; Morse et al, 1977) have also been found during ordinary relaxation of untrained controls, as well as in specific relaxation procedures. Electroencephalographic (EEG) changes have failed to be very useful as specific measures of relaxation procedures because of the close similarities with EEG characteristics of slow wave sleep phases 1 and 2 (Luthe, 1970; p 92; Younger et al, 1975; Pagano et al, 1976; Elson et al, 1977; Fenwick et al, 1977).

Therefore, it is difficult to isolate a single physiological measure which characterises the relaxation response, and a multi-dimensional approach, aimed at identifying response patterning, is

probably more appropriate[6] (Schwartz et al, 1978; Davidson and Schwartz, 1976). However, further clarification is still required to distinguish the relaxation response from both sleep and ordinary (non-specific) relaxation (Fenwick et al, 1977). Hence, relaxation procedures are probably best defined in terms of their designated function; that being, systematic induction of generalised psychophysiological relaxation, involving cerebrocortical deactivation (EEG synchronisation[7]), reduced autonomic-metabolic activity (decreased heart-rate, skin conductance, respiratory rate, oxygen consumption, carbon dioxide elimination, and muscle tone), and diminished affective arousal (Benson et al, 1974a; Davidson and Schwartz, 1976; West, 1979a), while simultaneously sustaining (Wallace et al, 1971; Williams and West, 1975; Luthe, 1970; p 91), or even increasing, alertness (Hernández Péon, 1977; Banquet et al, 1974a; Levine et al, 1977).

During the last decade, TM has been widely used as a model relaxation procedure in laboratory investigations, because volunteer subjects with standardised training are readily available. More particularly, sufficiently large samples of adept practitioners with long-term training are available for study. Several recent studies, which directly compare physiological changes in various relaxation procedures, highlight the existence of a common underlying physiological basis (Tebēcis, 1975; Morse et al, 1977; Zaichkowsky and Kamen, 1978).

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6. Indeed, biofeedback pre-training for reduced heart rate and frontalis muscle tension had no influence over performance during theta EEG training (Lutzenberger et al, 1976), and the appearance of theta activity has been associated with advanced stages of hypnosis-relaxation (Tebēcis et al, 1975), autogenic training (Luthe, 1970; p 92) and meditation (Kasamatsu and Hirai, 1966; Banquet, 1972; Hebert and Lehmann, 1977).

7. Patterns of EEG synchronisation in relaxation procedures involve predominance of alpha activity, with theta bursts during deep relaxation of advanced practitioners (see foot-note 6 above). Generally the alpha-theta activity does not descend into deep sleep (Younger et al, 1975; Fenwick et al, 1977; Luthe, 1970; p 92; Elson et al, 1977).

Although Davidson and Schwartz (1976) have hypothesised relaxation procedure-specific changes, there is very limited supportive evidence from studies of resting[8] relaxation techniques (Paul, 1969). Nevertheless, certain procedures may be more effective for inducing relaxation in specific physiological systems, as appears to be the case for muscle relaxation (Morse et al, 1977) and state anxiety reduction (Davies, 1977; Zuroff and Schwartz, 1978).

F. Working Hypothesis:- The Endocrine Antithesis of Stress in Relaxation Procedures

The aim of the following studies was to investigate possible endocrine bases for the effects of relaxation procedures on stress. The null hypothesis was that there are no endocrine bases for the effects of relaxation procedures on stress. A working hypothesis, that the endocrine changes in relaxation procedures are opposite to those which occur in stress, was pursued.

G. Previous Endocrine and Other Biochemical Studies of Relaxation Procedures

(a) Transcendental Meditation

The original studies of TM revealed slight but statistically significant reductions in arterial pH and base excess during TM of experienced meditators (Wallace, 1970a; Wallace et al, 1971). More recently, a significant fall in salivary pH was found immediately after TM of a novice subject re-studied during 10 meditation sessions (McCuaig, 1974). A significant reduction in blood lactate during TM of experienced meditators was originally found by Wallace et al (1971). Lactate levels remained low after meditation. These observations were later

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8. Davidson and Schwartz (1976) include physical exercises, such as jogging, in their definition of relaxation procedures. However, by the definition given earlier (p 8), such methods are not relaxation procedures because they increase autonomic-metabolic activity.

substantiated by other workers[9] (Rama Rao et al, 1977; Nandagopal et al, 1976; Jevning and Wilson, 1976; Jevning et al, 1978c). However, Michaels et al (1976; 1979) reported no significant change in blood lactate during TM of experienced meditators, nor did Jevning and Wilson (1976) find significant arterial pH changes in TM. Both Wallace et al (1971) and McCuaig (1974) concluded that TM produces a mild state of metabolic acidosis. A significant decrease in rate of blood lactate generation during TM, along with no significant arterial pH, pCO_2, pO_2, glucose or packed cell volume changes, led Jevning and Wilson (1976) to infer that TM reduces red cell metabolism. A lack of significant changes in arterial pO_2 and pCO_2 was also found in previous studies of TM (Wallace, 1970a; Wallace et al, 1971). McCuaig (1974) also reported significant differential increases in salivary electrolytes (sodium, magnesium, calcium, phosphorous and potassium) and elevation of both acid soluble and insoluble salivary protein immediately following TM.

Jevning et al (1977a; 1978a) reported a significant reduction in plasma cortisol during TM of experienced meditators in contrast to a lack of significant change during ordinary relaxation of non-meditator controls. Following TM, plasma cortisol levels remained low and appeared to be gradually returning to pre-meditation values. A similar trend was observed in novice meditators, but, the change was not statistically significant (Jevning et al, 1978a). Originally, Jevning et al (1977a) reported significantly lower basal plasma cortisol levels in experienced meditators than in non-meditator controls, however, their later study revealed no significant difference (Jevning et al, 1978a). Conflicting findings were reported by Michaels et al (1979) who found significant plasma cortisol reductions in both TM and ordinary relaxation of non-meditator controls. The decreased cortisol levels followed the expected

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9. Reduced blood lactate in meditation is probably due to decreased anaerobic respiration, and correlates with decreased oxygen consumption, while there is no change in respiratory quotient, during TM (Wallace, 1970a; Wallace et al, 1971).

morning circadian decline (Krieger et al, 1971). A significant increase in plasma cortisol was also observed 20 minutes after cannulation of non-meditator controls in contrast to no significant change in experienced meditators (Michaels et al, 1979). Avorn and Benson (1974) found no significant difference between the plasma cortisol values of meditators and matched non-meditator controls, although, day-to-day plasma cortisol variation was significantly lower in the meditators.

Significant elevations in plasma prolactin have also been observed following TM of both experienced meditators and controls re-studied after 3 to 4 months' TM training (Jevning et al, 1977a; 1978b). However, no significant changes in either plasma testosterone (Jevning et al, 1978a) or plasma growth hormone (Jevning et al, 1978b) were found in TM of either experienced or novice meditators.

Michaels et al (1976) found no significant changes in either plasma adrenaline or noradrenaline during TM, and Bujatti and Riederer (1976) observed no changes in vanillyl-mandelic acid (VMA) following meditation. In contrast, Lang et al (1979) recently reported a significant increase in total plasma catecholamines (predominantly noradrenaline) following TM of highly trained (advanced) meditators, whereas no significant TM-induced plasma catecholamine changes were observed in less experienced (long-term) meditators. Following both sitting-reading and TM treatments, physical exercise produced plasma catecholamine increases or decreases for long-term and advanced meditators, respectively. Furthermore, after exercise long-term meditators had significantly higher adrenaline excretion than advanced meditators. In both long-term and advanced meditators plasma catecholamines rose following supine-rest.

Lang et al (1979) also found significantly higher catecholamine and VMA excretion in advanced meditators than long-term meditators, whereas Bujatti and Riederer (1976) found significantly

lower basal VMA excretion in meditators than in non-meditator controls. A significant TM-induced increase in 5-hydroxyindoleacetic acid excretion in contrast to no significant change for non-meditator controls, along with a lack of significant homovanillic acid change in TM, were also observed by Bujatti and Riederer (1976).

Recently, Michaels et al (1979) reported a slight but significant increase in plasma renin activity during TM, in contrast to a lack of significant change during ordinary relaxation of non-meditator controls. However, Pollack et al (1977) found no significant change in plasma renin activity during six months' training of hypertensive patients. No significant plasma aldosterone changes were observed during either TM or ordinary relaxation (Michaels et al, 1979).

Other biochemical studies of TM have revealed:- a significant increase in plasma phenylalamine during TM of experienced meditators, in contrast to no significant change in both novice meditators and untrained controls; no significant changes of any other amino acids during TM (Jevning et al, 1977b); and no significant changes in blood sugar, blood urea or serum cholesterol following TM (Rama Rao et al, 1977).

In summary, the subject of endocrine changes in TM is controversial and there are conflicting reports about TM-induced changes in cortisol, catecholamines and plasma renin activity. One group of workers has observed a significent post-meditation prolactin rise and no significant hGH or testosterone changes in TM. Although not undisputed, blood lactate reduction appears to be the most consistently reported biochemical change in TM.

(b) Hypnosis

Hypnosis typically involves a wide variety of different induction procedures, and the nature of the dominant suggestion(s) appears to determine the response (Barber, 1971; Kroger, 1977; p 27).

Endocrine and other biochemical studies of hypnosis clearly exemplify the phenomenon of suggestion-specific responses in hypnosis:- Heterohypnotic induction of anxiety and other states of affective arousal have been found to produce increased catecholamine (Pinter et al, 1967) and hGH (Kurokawa et al, 1977) secretion, elevated blood free fatty acid (Fishman et al, 1962b; Pinter et al, 1967) and plasma hydrocortisone levels (Persky et al, 1959; Levitt and Persky, 1960), while no significant blood glucose changes were observed in either diabetic or normal healthy subjects (Weller et al, 1961). Conflicting findings were reported by Levitt et al (1960) who observed no significant increases in plasma hydrocortisone during heterohypnotic anxiety induction. However, Barber (1971) suggested that the discrepant findings were due to relatively high pre-treatment levels in the study by Levitt et al (1960); the degree of plasma hydrocortisone elevation with affective arousal being negatively related to the pre-treatment levels. Significant increases of plasma renin activity and angiotensin II concentration have recently been observed in hypnotic suggestion of running, however, no significant aldosterone changes were found (Kosunen et al, 1977).

In contrast to heterohypnotic anxiety, heterohypnotic induction of relaxation produced significant reductions in both plasma hydrocortisone (Sachar et al, 1965; 1966) and blood glucose of diabetics (Gigon et al, 1926; Stein, 1949). Neutral heterohypnosis was also found to be associated with diminished plasma cortisol levels (Alnaes, 1966; Alnaes and Skaug, 1966). During neutral heterohypnosis, experienced subjects showed more marked cortisol elevations in response to auditory and cutaneous disturbances than did novice subjects, while novice trainees responded more than untrained controls (Alnaes, 1966). A positive correlation between ease of hypnotic induction and decreased cortisol levels was also apparent (Alnaes and Skaug, 1966). A significant reduction in arterial pO_2 has been observed during heterohypnotic induction of drowsiness, lethargy and sleep (Lovett Doust, 1953).

(c) Autogenic training

The practice of standard autogenic training exercises, in particular passive concentration on heaviness and warmth, produced a significant plasma cortisol fall for long-term trainees in contrast to a lack of change in untrained controls (Alnaes, 1966; Alnaes and Skaug, 1966; Okamura and Goto, 1967[10]). The reduction in plasma cortisol was more marked in experienced trainees than novice subjects, and appeared to be proportional to autogenic training experience (Alnaes and Skaug, 1966; Alnaes, 1966). Furthermore, experienced autogenic trained subjects were less cortisol-stress responsive to auditory and cutaneous disturbances while in the autogenic state (Alnaes, 1966), and had generally higher pre-treatment plasma cortisol levels (Luthe, 1970; p 77) than both heterohypnosis and untrained subjects. A progressive return of plasma cortisol levels to pre-treatment levels occurred following autogenic exercises (Alnaes, 1966) and a positive correlation between depth of relaxation and degree of cortisol reduction was apparent (Alnaes and Skaug, 1966). A fall in plasma cortisol levels in women who practised autogenic exercises during labour and delivery has also been reported (Okamura and Goto, 1967).

Decreased serum cholesterol levels have also been observed following autogenic exercises (heaviness and warmth) in novice subjects (Aya, 1967), and variable blood sugar changes were found depending on the standard exercise used (Marchand, 1961). Luthe and Schultz (1969, Vol 2) reviewed the long-term normalising influence of autogenic training on hypoglycemia and glycosuria in patients with diabetes mellitus (pp 107-113), hyper- and hypothyroidism (pp 113-117) and hypercholesterolemia (pp 118-121).

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10. For a review, in English, of these studies, see Luthe, 1970; pp 76 to 78, inclusive.

(d) Progressive relaxation

Using a modification of Jacobson's (1938) progressive relaxation procedure, Davidson et al (1979) found a significant reduction in plasma noradrenaline during deep muscle relaxation of patients with organic heart disease. No significant changes in either plasma adrenaline or dopamine were reported during relaxation in these novice trainees.

(e) Yoga-meditation

Increased 17-hydroxycorticosteroid, 17-ketosteroid, testosterone and VMA excretion, along with elevated serum protein levels, have been observed following 6 months' systematic hatha-yoga training (Udupa and Singh, 1972; Gode et al, 1974). Significant decreases in both plasma acetylcholine and serum cholinesterase (Udupa et al, 1973), along with reduced blood sugar and serum cholesterol levels (Udupa and Singh, 1972) were also found.

Significant reduction of both plasma dopamine-beta-hydroxylase and renin activity of hypertensive patients occurred after six months' training in a Buddhist meditation procedure (Stone and De Leo, 1976). Ghista et al (1976) found decreased glucose, lactate and pyruvate levels following Ananda Marga meditation in a single subject.

(f) Summary

The most characteristic endocrine change in relaxation procedures appears to be a significant short-term cortisol reduction in TM, heterohypnotic relaxation, and autogenic training. However, decreased cortisol levels have also been associated with viewing bland natural scenery films (Handlon et al, 1962; Wadeson et al, 1963) and abnormally low 17-hydroxycorticosteroid excretion has been reported

during euphoric regression transference in psychoanalysis[11] (Menzer-Benaron, 1963). Therefore, a cortisol fall may not be peculiar to specific relaxation procedure-induced states.

In contrast to cortisol, changes in catecholamines, and other hormones related to sympathetic nervous activity, are less consistent. Reports of changes in other endocrine functions are scanty and there do not appear to be any previous studies of thyroid hormone changes in relaxation procedures.

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11. In this context, it is interesting to note that regression has been proposed as a mechanism involved in both hypnosis and meditation (Meares, 1962; Maupin, 1965; Shafii, 1973; Meares, 1977).

CHAPTER II : METHODS

Preface to Methods:- Selection and Preparation of Subjects

The following four studies were conducted on healthy, day-active, young-adult, Caucasian volunteers:- A long-term study (part 1), two short-term studies (parts 2 and 3), and a TM residence course study (part 4).

TM subjects had received standardised instruction in meditation (Maharish Mahesh Yogi, 1966; 1969) and were routinely practising TM twice daily, morning and evening, for between 20 and 30 minutes per session. Experienced meditators were defined arbitrarily as having more than a year's regular TM practice (10 to 14 TM/wk). The lack of sufficient availability of subjects who were both experienced, and of standardised training in other relaxation procedures, restricted the study of other relaxation groups to novice[12] subjects only. In the second short-term study and the TM residence course study TM was used as a model relaxation procedure.

As preparation for each of the following four studies, subjects attended a preliminary meeting at which, in accordance with the Declaration of Helsinki, prepared by the World Medical Association (1976), the nature and purpose of the research was explained. Informed consent for voluntary participation in the experimentation was obtained from all subjects. The correct procedure for collection of a twenty-four hour urine sample was outlined. Standardised instructions regarding experimental requirements were given to all subjects (Bieman, 1976).

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12. "Novice" in contrast to "experienced" subjects were defined arbitrarily as having less than one year's regular practice with a specific relaxation procedure.

PART 1 :- LONG-TERM STUDY

A. Subjects

54 relaxation trainees, 12 controls, and 10 experienced Transcendental Meditators were studied. Both males and females were included in all groups. Prior to the study neither the relaxation trainees nor the controls had received any specific relaxation training. The controls remained untrained throughout the study. The relaxation trainees and untrained controls were predominantly students. The experienced TM group consisted mainly of school teachers and tertiary students.

B. Experimental Design

(a) Outline

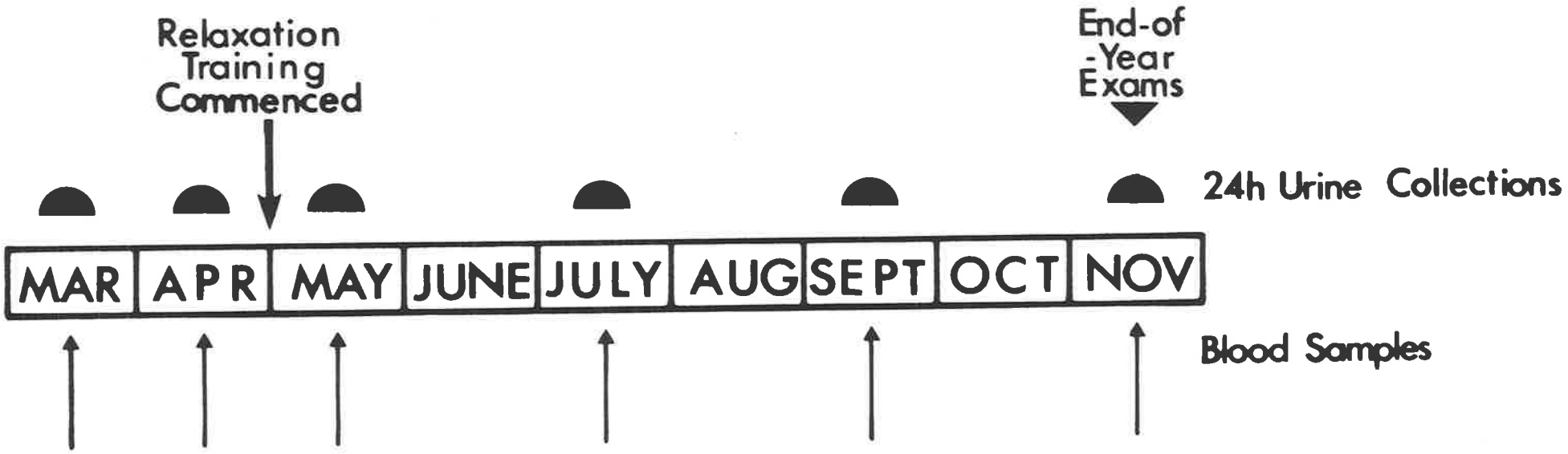
The study was conducted from April to November of the 1975 academic year. As shown in figure 2, a time-series experimental design (Glass et al, 1975) with a pre-training control period was used. Twenty-four hour urine collections and venous blood samples were collected during the week before commencement of relaxation training and at about two monthly intervals over the seven month training period. The control group and the experienced TM group were studied concurrently with the training groups.

(b) Treatments

(i) Relaxation procedures

Prospective relaxation trainees chose instruction in one of four relaxation procedures:- Transcendental Meditation (TM), progressive relaxation (PR), autohypnosis-relaxation (AHR) or yoga-meditation (YM). Jacobson's (1938) progressive relaxation procedure, modified by

FIGURE 2. Experimental Design of Long-term Study



exclusion of preliminary muscle tensing[13], and Erickson's (1961) indirect hand-levitation and progressive relaxation hypnotic induction methods were used. The yoga-meditation procedure combined hatha yoga with passive concentration on body sensations and the natural breath. The TM, PR and AHR procedures involved preliminary active cognitive induction, whereas the YM procedure employed active somatic induction (Davidson and Schwartz, 1976). In all procedures, passive concentration was used to deepen relaxation (Benson et al, 1974a; Davidson and Schwartz, 1976).

(ii) Specific relaxation training

Details of the principles and performance instructions of the relaxation procedures used are given in Appendix II pp 154-161. Only details of the relaxation training programmes are given here.

Transcendental Meditation:- 10 TM subjects[14] were trained[15]. Instruction in TM followed a standardised well established format involving introductory and preparatory lectures followed by four training sessions, each of between one half and one hour's duration. During the first session personal instruction in TM was given by a trained teacher. The three other sessions were conducted in a group setting. The aim of the three follow-up sessions was to check for correct use of the TM procedure and to assist students with any difficulties in their practice.

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13. Exclusion of preliminary muscle contraction in progressive relaxation results in it being a purely cognitive relaxation procedure, as is the case with both TM and AHR (Davidson and Schwartz, 1976).

14. An attrition rate of about thirty percent was anticipated to occur during relaxation training (Kanellakos and Lucas, 1974; p 45).

15. The TM subjects were trained at the North Adelaide TM Centre (176 Archer Street) through the Students' International Meditation Society. The TM teachers were Mr. Ned Roberts and Mr. Alexander Wealleans.

Progressive relaxation and autohypnosis-relaxation:- 14 progressive relaxation[14] and 15 autohypnosis relaxation[14] subjects were trained. For both the progressive relaxation and autohypnosis-relaxation groups, training consisted of four half-hour sessions. The instruction programme was designed to be directly comparable with that for TM[16], having one initial private instruction session followed by three group sessions. Emphasis was placed on cultivating the ability for self-induced relaxation without the aid of a teacher or therapist[17]. Further follow-up supervision in the above procedures was given if and when required, by appointment with the teacher concerned.

Yoga-meditation:- 15 yoga-meditation[14] subjects were trained. The yoga-meditation group[18] met once or twice a week during the study. A more extensive training period was required for the yoga-meditation group because more learning was involved (see Appendix II, pp 159-161).

(c) Urine samples

Urine volumes were recorded and the pH of two 20 ml aliquots was immediately adjusted to 3-6 and 1-2.5, and stored at $4^{o}C$ or $-20^{o}C$ until assayed for cortisol and catecholamines, respectively.

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16. Despite arrangement of a special concession fee for TM instruction, only 10 TM students were available for study and consequently fewer subjects were included in the TM group than the other relaxation groups.

17. The progressive-relaxation and autohypnosis-relaxation groups were independently trained at the University of Adelaide Student Counselling Service by counsellor Mr. Norman Greet.

18. The yoga-meditation trainees met at the University of Adelaide. The group was trained by an advanced practitioner, Mr. Robert Hill, who had received intensive training in India under Swami Karunananda.

(d) Blood samples

Venous blood specimens were collected from an antecubital vein at times which were standardised for each subject in order to avoid influence of circadian variation in plasma cortisol levels (Perkoff et al, 1959; De Lacerda et al, 1973). Blood samples for cortisol assay were collected into di-sodium ethylenediaminetetra-acetic acid (EDTA) tubes, centrifuged at 2000 g for 10 minutes, and the plasma was stored at -20°C until assayed. Approximately one hour after collection, blood samples for serum thyroid hormone assays were centrifuged for 10 minutes at 2000 g and the serum was stored at -20°C until assayed.

C. Independent Variables

(a) Standardisation of relaxation training

(i) Possible influence of positive conditioning:- motivation and expectation characteristics

Positive conditioning is an integral part of the TM training programme (Morse et al, 1977). Therefore, in order to standardise the expectation and motivation characteristics (Shapiro and Giber, 1978) of all groups, subjects in the progressive relaxation, autohypnosis-relaxation, and yoga-meditation groups were also enthusiastically conditioned about the beneficial effects of the regular practice of their respective relaxation procedure.

(ii) Possible influence of training intensity

Regular practitioners of relaxation procedures, as is the case for more experienced subjects (Kanellakos and Lucas, 1974; p 47 and p 89; Luthe, 1970; pp 91 and 92), show more marked psycho-physiological changes than erratic practitioners (Luthe, 1970; pp 91 and 92; West, 1976b). The usual length of individual relaxation

sessions might also influence the extent of any changes (Corby, 1979). Therefore, training subjects were encouraged to routinely practise their respective relaxation procedures twice daily, morning and evening, for between 20 and 30 minutes each session. Furthermore, during the study all subjects were encouraged to continue their practice regularly. The experienced TM subjects continued their routine twice-daily practice.

(b) Drug usage

Because of the known and suspected influences of various non-prescribed drugs on the endocrine system (Gold and Ganong, 1972; Frankenhaeuser, 1971; Levi, 1972; p 31), the use of non-prescribed drugs was restricted immediately prior to and during sample collection. During the 48 hours prior to blood sample collection, all subjects were asked to abstain from alcohol, marijuana and other non-prescribed drugs, excepting tea, coffee and tobacco. Abstinence from the above mentioned non-prescribed drugs applied also to the 24 hour period of urine sample collection and to the day before commencement of collection. No restrictions were placed on the use of tobacco and caffeinated beverages because it was considered to be a necessary compromise between experimental control and continued subject participation and cooperation. It can also be viewed as a compromise between experimental control of everyday life stimuli (independent variables) and the study of a realistic life situation (Levi, 1972; p 39). No subjects were on medication during the week prior to blood or urine sample collection.

(c) Subject characterisation

Data on age and, where relevant, meditation experience, were collected from all subjects. A record of any changes in relaxation

practice, such as discontinuation of training, was kept on all subjects.

D. Dependent Variables

(a) Hormone assays[19]

Plasma and urinary free cortisol were measured using a modification of Murphy's (1967) competitive protein-binding assay. Total urinary catecholamines (UCA) were measured using a semi-automated trihydroxyindole method (Head et al, 1977). Radioimmunoassays with charcoal separation were used to measure total serum triiodothyronine (T_3) (Queen Elizabeth Hospital method) and total serum thyroxine (T_4) (Chopra, 1972). Details of the assays used are given in Appendix I.

E. Statistical Analysis

Parametric statistical testing was carried out using a Wang 600 desk-top computer (Wang Program Library, Volume 133:- Analysis of Variance and Regression Analysis). Within-subject analysis was generally used because it has the advantage of eliminating normally large inter-individual variation in basal hormone concentrations (Fox et al, 1961; Johansson and Post, 1974; Hammond et al, 1976), as well as the need to consider group differences in subject characteristics. It is more valid to make inter-group comparisons on UFC than plasma cortisol values because of the rapid episodic fluctuations in circulating cortisol concentrations (Hellman et al, 1970). Inter-group urinary hormone comparisons were made on males only because of the inherent sex differences in endocrine function (Markiewicz et al, 1973; Johansson and Post, 1974; Frankenhaeuser et al, 1976) and because the majority of subjects were males. Subjects who stopped their relaxation training

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19. The plasma cortisol assays were performed in the Department of Obstetrics and Gynaecology at the Queen Elizabeth Hospital. All other assays were performed in the Department of Clinical Chemistry at the Queen Elizabeth Hospital.

were excluded from analysis.

PART 2 :- SHORT-TERM STUDY (1)

A. Subjects

Novice subjects from the long-term study, who had six months' continuous relaxation training with regular practice, were studied. Four TM, four YM, two PR and 2 AHR subjects were investigated. Seven experienced TM subjects and six untrained controls from the long-term study were examined under comparable experimental conditions.

B. Experimental Design

(a) Outline

Figure 3 outlines the experimental design. Subjects arrived at the laboratory at 1400 hours and subsequently blood samples and cardiovascular measurements were taken before and after a half-hour Sunday afternoon relaxation session[20]. The experimental procedures and experimenters were familiar to all subjects.

(b) Treatments

During the 30 minute treatment period the controls sat with eyes shut and relaxed. The other subjects practised their respective relaxation procedure. Standardised instructions to start and stop relaxing were given to all subjects. Silence was kept during the relaxation session and a group of 4 to 6 subjects was studied on each occasion.

(c) Urine samples

To investigate possible basal inter-group endocrine differences, 24 hour urine samples were collected over the same

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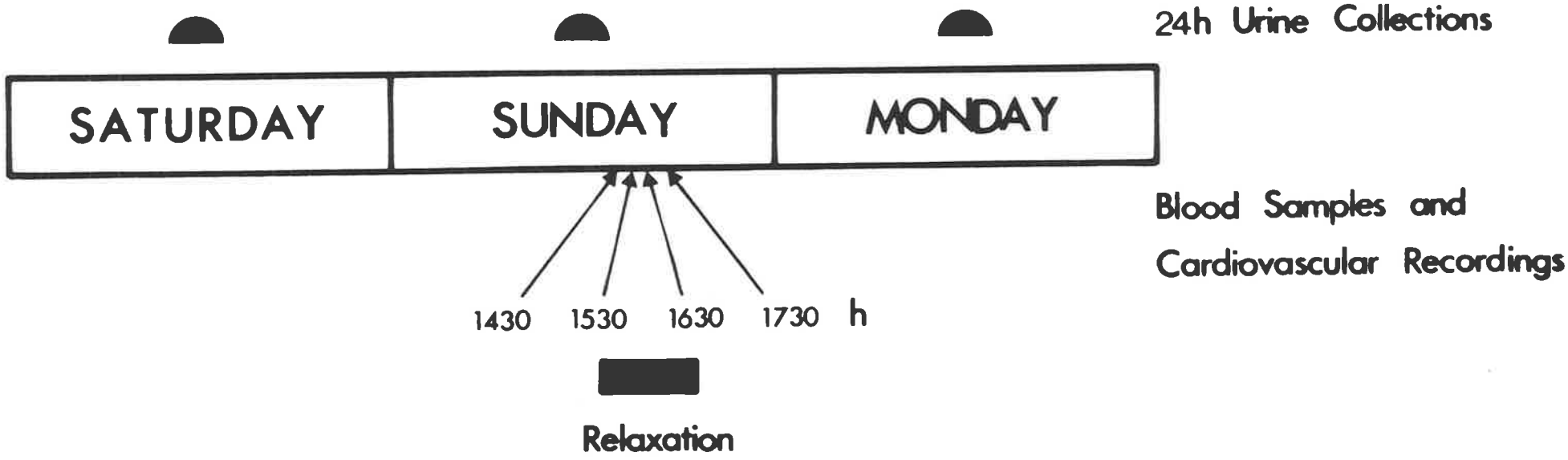
20. The study was conducted at the Department of Anatomy and Histology, the University of Adelaide.

FIGURE 3. Experimental Design of Short-term Study (1)



week-end as the laboratory experiment and on the following Monday. Urine volumes were recorded and, as described for the long-term study (p 20), all urine specimens were treated with preservative and stored until assayed.

(d) Blood sampling

Four serial venous blood samples were collected by venepuncture from an antecubital vein at approximately one hourly intervals. Blood specimens were centrifuged and stored using the same procedures as in the long-term study (p 21). Afternoon was chosen for the experimental observations because of the relative stability of plasma cortisol levels at this stage in the circadian rhythm (Krieger et al, 1971).

(e) Cardiovascular recordings

Blood pressure and heart rate were measured following each blood sampling[21] to avoid the possible influence of cuff-induced venous constriction on protein-bound hormone concentrations (Judd et al, 1975; Cashmore et al, 1976). Before and after the relaxation session, all subjects sat in comfortable armchairs in a pleasant room, talking amongst themselves and reading relaxing material of their own choice[22]. All subjects remained seated during the three hour experimental period in order to exclude the influence of postural changes on protein-bound hormone concentrations (Papacostas et al, 1960; Judd et al, 1975) and on sympathetic nervous function (Sundin, 1958; Sever et al, 1977).

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21. A tourniquet was not used for blood sampling.

22. No pornographic reading material was allowed because of possible sympatho-adrenomedullary or adrenocortical activation in response to visual stimulation (Levi, 1972; Zuckerman, 1971; Brown and Heninger, 1975; Wiedeking et al, 1977).

C. Independent Variables

(a) Drug usage

The following drug restrictions were made to minimise possible drug-induced hormone changes (Gold and Ganong, 1972; Frankenhaeuser, 1971; Levi, 1972; p 31). No subjects were on medication during the week prior to the experiment. Standardised restrictions were placed on tea, coffee, tobacco and alcohol consumption during the week before the study in order to minimise their usage. On the day of the blood sampling experiment, all subjects abstained from tea, coffee and alcohol (Poisner, 1973; Jenkins and Connolly, 1968) and where applicable reduced tobacco smoking to the degree that it was not uncomfortable (Myrsten et al, 1977). No smoking was allowed during the afternoon laboratory experiment.

(b) Food

On the Sunday of the blood sampling experiment lunch-time was standardised at 1200-1300 hours in order to standardise the influence of meal-related cortisol changes (Montagu, 1968). Moreover, no food or beverages were allowed between lunch and the end of the laboratory study in order to exclude food-related cortisol changes (Montagu, 1968; Krieger et al, 1971). Water was available on request.

(c) Physical and mental activity

On the morning before the laboratory study, all subjects were requested to avoid both strenuous physical and mental activity, as well as emotional disturbances, because of possible influences on endocrine function (Frankenhaeuser, 1971; Hartley et al, 1972; Raymond et al, 1972; Rahe et al, 1974). However, in all other respects the morning activity before the laboratory study was as usual for each subject.

(d) Efficacy of treatments

Following the relaxation session, all subjects gave written reports of their relaxation experiences. Using a modification of the rating scheme devised by Maupin (1965), three independent judges blindly rated the reports for depth of relaxation. (See Appendix III, pp 162 and 163, for details of relaxation rating schemes). Heart rate was measured because decreased values are generally associated with psychogenic relaxation (Benson et al, 1974a; Davidson and Schwartz, 1976).

(e) Subject characterisation

Data on age, occupation, and, where relevant, meditation experience, were collected from all subjects.

D. Dependent Variables

(a) Hormone assays

All blood samples from one subject were run in the same assay to eliminate inter-assay variation. Plasma cortisol, serum T_4, serum T_3, UFC and UCA were measured by the methods described in the long-term study (p 23).

(b) Cardiovascular measurements

Blood pressure was measured by the indirect auscultatory method using a sphygmomanometer (Berne and Levy, 1972; pp 98 and 99). Heart rate was measured by palpation of the radial pulse. All cardiovascular measurements were made by the same experimenter to avoid experimenter variation. Mean arterial blood pressure was estimated using the standard formula:- Mean arterial blood pressure ≈ diastolic blood pressure + $\{\frac{\text{systolic blood pressure - diastolic blood pressure}}{3}\}$ (Berne and Levy, 1972; p 89).

E. Statistical Analysis

The materials and methods used for statistical analysis in the previous study were also employed here (p 23).

PART 3 :- SHORT-TERM STUDY (2)

A. Subjects

(a) Selection

21 experienced male meditators and 15 male non-meditator controls were studied. Both vegetarians and non-vegetarians were included in each group.

(i) Meditators

Experienced meditators only were studied because the most outstanding physiological changes during TM generally occur in regular long-term practitioners[23] (Kanellakos and Lukas, 1974; West, 1979a).

(ii) Non-meditators

Control subjects were interested in meditation, however, they had no prior experience with meditation or specific relaxation procedures. Three of the controls subsequently started TM. By using control subjects who were interested in meditation, any psycho-physiological predisposition towards evoking particular endocrine or other physiological changes, which might peculiarly bias the meditation group, was excluded (Smith, 1975). Since meditators as a group are characterised by low usage of both prescribed and non-prescribed drugs (Benson and Wallace, 1977; Shafii et al, 1974; 1975; Lazar et al, 1977) and dietary changes, such as reduced meat consumption (personal observation, A. Bevan), care was taken to ensure that control subjects had similar characteristics. Hence, the control subjects were recruited from advertisements in a vegetarian restaurant and whole-foods store,

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23. To optimise the quality of the experimental meditation sessions, all subjects had their meditation checked by a qualified TM teacher during the week prior to the laboratory study, and were instructed to complete their Saturday morning meditation by 0900 hours.

and approximately matched the meditators for the above independent variables[24] (See pp 36-38 for further methodological considerations).

(b) Preparation

At the preliminary meeting (see p 17), trial intravenous cannulations and blood sampling were performed on all subjects in order to familiarise them with the experimental proceedings (Sabshin et al, 1957). The aim of the trial intravenous catheterisation was to accustom subjects to the experience. Thereby any "first experience" effects of cannulation were eliminated (Fishman et al, 1962a), and the amount of apprehension and anxiety experienced on the day of the actual experimentation was minimised (Davis et al, 1962; Mason, 1968a).

B. Experimental Design

(a) Outline

As shown in the outline of the experimental design given in figure 4a, the study was conducted over two consecutive week-ends[25].

(b) Treatments

(i) Non-meditators as controls:- meditation or ordinary relaxation

16 experienced meditators (group 1) and 15 non-meditator controls (group 2) were studied before, during, and after a half-hour

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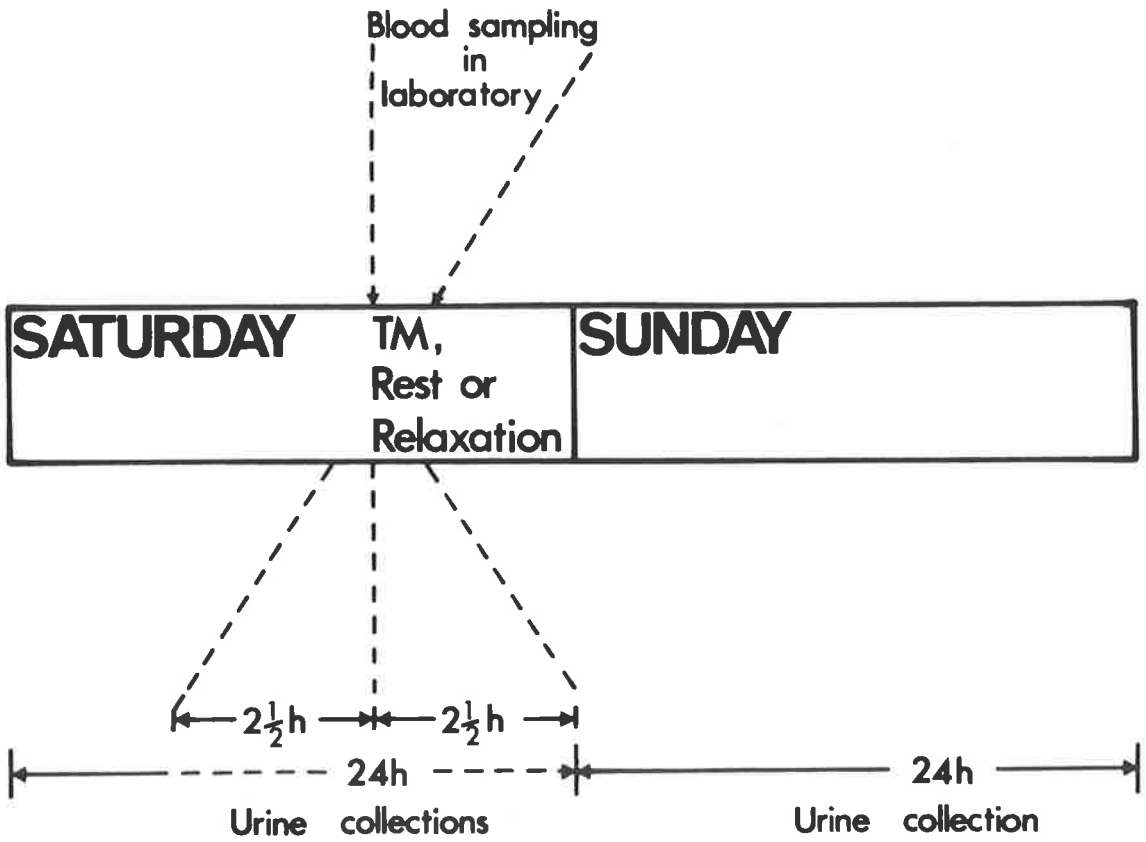
24. These considerations were particularly important when inter-group comparisons were made between meditators and non-meditators (see p 40).

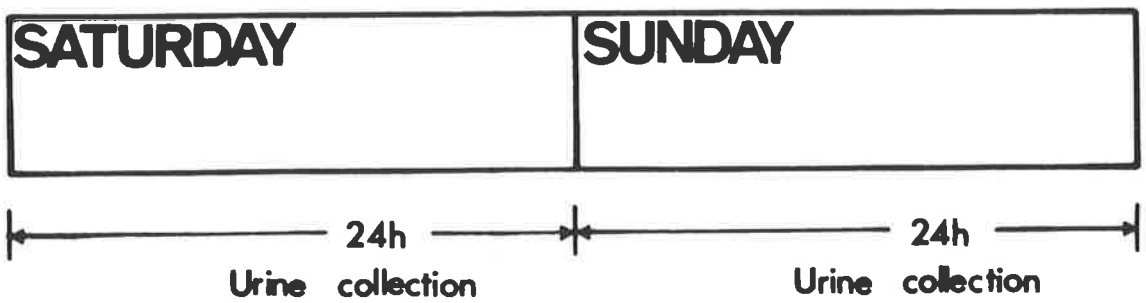
25. The Saturday afternoon laboratory study was conducted at the Queen Elizabeth Hospital.

FIGURE 4a. Outline of Experimental Design Used in Short-term Study (2)

1) EXPERIMENTAL WEEK-END:- Saturday Afternoon Laboratory Study



2) CONTROL WEEK-END:- Ordinary Everyday Life Situation



TM or ordinary relaxation[26] session. Standardised instructions to start and to stop meditating or relaxing were given to all subjects. During the 30 minute treatment period the controls sat with eyes shut and relaxed. The meditators practised their usual TM. Silence was kept during the period of meditation or relaxation.

(ii) Meditators as controls:- meditation or quiet rest

Meditators have been reported to show distinctive physiological responses to stressful stimuli (Orme-Johnson, 1973; Goleman and Schwartz, 1976) and to be less anxious than non-meditators (Nidich et al, 1977; Hjelle, 1974). Furthermore, the demand and expectation characteristics of trained subjects are likely to be very different to those of untrained controls (Shapiro and Giber, 1978), because meditators undergo positive conditioning during their training, and generally have a positive attitude towards their meditation practice (Smith, 1975). Therefore, to give due consideration to the above mentioned independent variables, as well as the large inter-individual variation in basal hormone levels (Fox et al, 1961; Johansson and Post, 1974; Hammond et al, 1976), meditators, rather than non-meditators, were also used as controls.

Theoretically the best control condition is an eyes-closed rest period, free from a TM-induced state. However, this is not feasible because electro-physiological changes which are characteristic of TM have been reported in experienced meditators during eyes-closed, non-meditation rest periods (Williams and West, 1975; Kanellakos and Lucas, 1974). Hence five subjects from the meditation group (group 1A) were re-studied under the same experimental conditions, excepting that instead

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26. "Ordinary" relaxation is used here to mean non-specific relaxation; that is no specific relaxation or meditation procedure was used.

of meditating, they read and talked quietly (group 1B[27]). An "active" treatment was chosen in preference to a silent, eyes-open "passive" treatment, to avoid endocrine changes due to understimulation (Frankenhaeuser et al, 1971), and because it was considered more relaxing, particularly under experimental blood sampling conditions involving a group setting (Greene et al, 1970; Kollar et al, 1966).

Restudying meditators created a "second experience effect" which might significantly influence endocrine changes (Mason, 1968a). Therefore, a comparable group of five, previously unstudied, experienced meditators were examined under the same non-meditation, quiet rest conditions as for group 1B (group 3).

Table 1 summarises the experimental conditions of each group. On any one occasion, two groups of 2 or 3 subjects were studied in separate rooms.

TABLE 1. Summary of Experimental Conditions used in Short-term Study (2).

GROUP	SUBJECTS	1st or 2nd OCCASION STUDIED	TREATMENT	NUMBER
1	Meditators	1st	Transcendental Meditation	16
2	Non-meditators	1st	Ordinary Relaxation	15
1A	Meditators	1st	Transcendental Meditation	5
1B	Meditators	2nd	Reading and Talking	5
3	Meditators	1st	Reading and Talking	5

(c) Urine sampling

(i) Two and a half hour urine collections

Consecutive 2½ hour urine samples were collected before and during the Saturday afternoon laboratory investigations (See

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27. Group 1B subjects were selected from group 1 subjects (ie group 1A) who did not require cannula re-insertion during the afternoon. Cannulation on the second occasion was performed at exactly the same time as on the first occasion.

figure 4a). All subjects voided at 1130 hours, prior to arriving at the laboratory. The exact time of collection was recorded. Subjects voided again at 1400 hours, before the blood sampling commenced, and on completion of blood sampling at 1630 hours. The exact time of the 1400 hour and 1630 hour urine collections were noted.

(ii) Twenty-four hour urine collections

To investigate possible basal endocrine changes, 24 hour urine samples were collected on the Saturday of the laboratory experiment[28] and on the following Sunday. On the weekend after the blood sampling experiment, 16 of the meditators and 12 of the non-meditators collected two "control" 24 hour urine specimens. There were two reasons for the further standardised urine collection over an ordinary week-end:- 1) to assess the influence of experimental stress, in particular venepuncture stress, on urinary cortisol and catecholamine excretion in meditators compared with non-meditators; and 2) to provide further information about the long-term endocrine changes associated with TM.

Both 2½ hour and 24 hour urine volumes were recorded and, as previously described (p 20), all urine specimens were treated with preservative and stored until assayed.

(d) Blood sampling

As shown in figures 4a and 4b, a time-series experimental design (multiple unit, single treatment), with a pre- and post-treatment period (Glass et al, 1975), was used to study short-term

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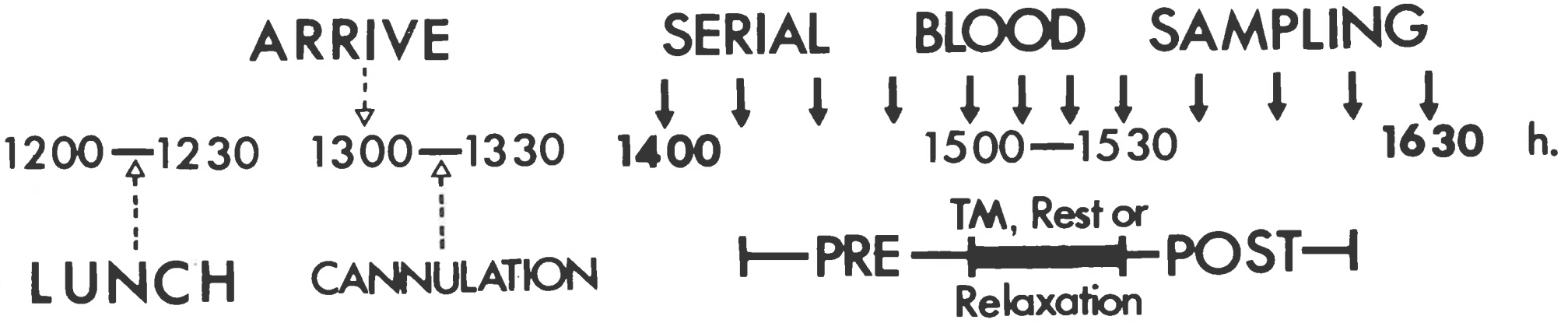
28. The 24 hour collection on the Saturday of the laboratory study consisted of the two 2½ hour urine saves plus a save from the remaining 19 hours. Urine samples from each of the three parts were assayed, and the results summated to give the total 24 hour excretion.

FIGURE 4b. Experimental Design of Saturday Afternoon Study



endocrine changes. An intravenous catheter placement unit was inserted into an antecubital vein about an hour before the first blood sampling[29], in order to minimise possible venepuncture stress-induced hormone elevations (Coppen and Mezey, 1960; Helge et al, 1969; Copinshi et al, 1967; Adler et al, 1975). Twelve serial blood samples were drawn at 10 to 15 minute intervals, before, during and after a 30 minute mid-afternoon meditation, ordinary relaxation, or quiet rest period. Frequent blood samples were taken to avoid observation of misleading changes due to episodic hormone secretion, which produces rapid fluctuations in serum hormone concentrations (Hellman et al, 1970; Parker et al, 1973; Alford et al, 1973; O'Connor et al, 1974). Mid-afternoon (1415-1630 hours) was chosen for the experimental observations because of the relative stability of cortisol levels at this stage in the circadian rhythm (Krieger et al, 1971).

As shown in figure 5, all subjects were seated in comfortable armchairs in a pleasant room with a constant room temperature of 22^{o}C. Before and after the meditation, relaxation or quiet rest period, all subjects talked and read relaxing material of their own choice[30]. All subjects remained seated during the 150 minute experimental period in order to exclude the influence of postural changes on protein-bound hormone concentrations (Papacostas et al, 1960; Judd et al, 1975[31]) and on sympatheto-adrenomedullary function (Sundin, 1958; Sever et al, 1977).

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29. The exact times of cannulation and blood sampling were recorded for each subject.

30. See foot-note 22, p 26.

31. Sphygmomanometers were loosley attached to the upper arms of all subjects during the study. However, venous compression was applied very infrequently and only when difficulty in sampling arose.

FIGURE 5. Photograph of Two Non-meditators During Ordinary Relaxation of Laboratory Study.

The first and last samples were used for multiple blood biochemistry analysis only. Initially blood samples for packed cell volume (PCV) and lactate assay[32] were taken using a 5 ml graduated syringe. For lactate assay, 1 ml whole blood samples were collected into 2 ml of 0.6 M perchloric acid and mixed thoroughly. Specimens were stored at $4^{o}C$ until subsequent centrifugation at 2000 g for 15 minutes at $4^{o}C$. The supernatants were stored at $4^{o}C$ until analysis within 7 days. Samples for PCV assay were collected into EDTA tubes, and mixed gently until assayed during the afternoon.

Approximately one hour after collection, blood samples for the remaining assays were centrifuged at 2000 g for 10 minutes. The serum for multiple blood analysis and glucose assay was stored at $4^{o}C$ until assayed within 48 hours. Serum aliquots for each hormone assay and for protein analysis were stored at $-20^{o}C$ until assayed.

C. Independent Variables

(a) Drug Usage

The drug restrictions used in the first short-term study (p 27) also applied to the experimental week-end of short-term study (2). For the control week-end subjects were requested to keep their drug usage as well as diet and sleep-activity patterns, consistent with the week-end of the laboratory study.

(b) Diet

On the Saturday of the blood sampling experiment lunch-time was standardised at 1200 to 1230 hours in order to standardise the influence of meal-related endocrine changes (Sukkar et al, 1967; Montagu, 1968; Catt, 1970). No food or beverages were allowed

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32. Blood lactate was assayed on six of the meditators and twelve of the non-meditator controls.

between lunch and the end of the laboratory study in order to exclude endocrine changes related to carbohydrate, protein (Glick et al, 1965), glucose (Montagu, 1968) or caffeine (Poisner, 1973) ingestion. Water was readily available on request. In consideration of possible dietary protein influences over cortisol levels (Galvão-Teles et al, 1976; Smith et al, 1975), dietary protein intake and vegetarian status were assessed on each subject[33] using diet records[34] and a diet questionnaire (Appendix III, pp 166 and 167), respectively.

(c) Physical and mental activity

The restrictions on physical and mental activity used in the first short-term study (p 27) also applied to short-term study (2).

(d) Efficacy of treatments

Blood lactate was measured because reduced levels have been associated with a TM-induced state (Wallace et al, 1971; Nandagopal et al, 1976; Jevning and Wilson, 1976; Jevning et al, 1978c), although Michaels et al (1976; 1979) have recently reported contrary findings which challenge the notion of a specific TM-induced lactate reduction. Questionnaires were administered to all subjects following meditation or relaxation to assess the efficacy of meditation or relaxation and the amount of subjectively experienced experimental stress. The Meditation-Relaxation Questionnaire was developed from the method of Maupin (1965), and rating schemes were devised to determine meditation-relaxation depth and degree of experimental discomfort-involvement. (See Appendix III, pp 163-165 for details).

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33. This is important because, as previously mentioned (p 30), there is a tendency towards vegetarianism amongst meditators.

34. From the diet records dietary protein intake was estimated using food composition tables (Thomas and Corden, 1977; Watt, 1975).

(e) Subject characterisation

Data on age, height, weight, diet, and non-prescribed drug usage were collected from all subjects, and used especially to characterise the groups of matched meditators and non-meditator controls (see p 40). Body surface area was calculated using the equation:

$$A = (W^{0.425}) \times (H^{0.725}) \times 71.84,$$

where A = area in m^2; W = weight in kg; and H = height in cm (Cannon, 1974). In further consideration of possible influences of independent variables on endocrine functions, information about occupation (Miller et al, 1970; Takahashi, 1972), physical training (Hartley et al, 1972; Markiewicz et al, 1973; Tandon et al, 1974), sleeping habits[35] (Köbberling and Von Zur Mühlen, 1974; Sassin, 1977), and meditation experience (Kasamatsu and Hirai, 1966; Corby et al, 1978) was collected from all subjects by general questionnaire (Appendix III, pp 168-170).

D. Dependent Variables

(a) Hormone Assays and Calculations[36]

All blood samples from one subject were run in the same assay to eliminate inter-assay variation. Serum cortisol was measured using a modification of Murphy's (1967) competitive protein-binding assay with charcoal separation. Serum growth hormone, prolactin, total T_4, total T_3 and total reverse triiodothyronine (rT_3) were measured by double antibody radioimmunoassays. hGH was assayed using the CEA, IRE, SORIN kit (HGHK/B76/R) and all other serum hormones were assayed by routine Queen Elizabeth Hospital[37] methods. T_3 sephadex

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35. To avoid possible sleep-deprivation induced endocrine changes (Sassin et al, 1969; Sassin, 1977), all subjects were instructed to have adequate sleep during the week prior to the study.

36. All serum hormone assays were performed in the Department of Clinical Chemistry at the Queen Elizabeth Hospital.

37. Department of Clinical Chemistry.

uptake (T_3SU) was determined (Liewendahl and Helenius, 1975) and free thyroxine index (FTI) was calculated from the product of T_4 and T_3SU. UFC and UCA[38] were measured by the same assays used in the long-term study (p 23). Details of the hormone assays used are given in Appendix I.

(b) Other Biochemical or Haematological Methods

The following routine assays were performed:- blood lactate enzymatic method (Calbiochem. Rapid Lactate Stat-Pak Kit), glucose oxygen rate method (Beckman analyser), total serum protein[39] (refractometry), packed cell volume (PCV)[40] (Coulter Counter model S), urinary urea diacetylmonoxene method and urinary creatine Jaffe method (Technicon series II autoanalyser). Osmolality was derived from the formula: $2.[Na^+] + 2.[K^+] + [Urea] + [Glucose]$ (Zilva and Pannall, 1979; p 36). Creatine clearance was calculated by standard formula (Creatinine Clearance = $\frac{[\text{Urinary Creatinine}] \times \text{Urine Flow}}{[\text{Serum Creatinine}]}$; Cannon, 1974).

E. Statistical Analysis

Statistical analysis was carried out using a Wang 600 desk-top computer (Wang 600 Series Program Library volume 133: Analysis of Variance and Regression Analysis, 1972) and a Control Data Corporation 1700 series computer. Parametric testing was used except for comparing self-report measures (Levi, 1972; pp 45-47).

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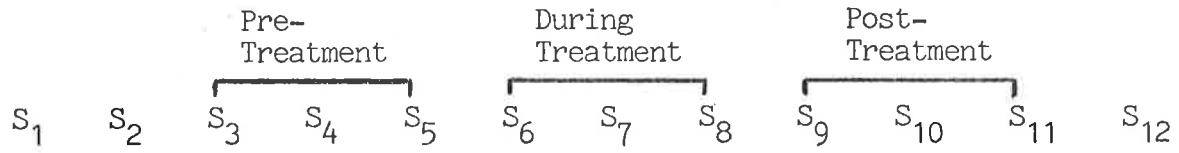
38. The urinary free cortisol assays were performed in the Department of Clinical Chemistry at the Queen Elizabeth Hospital. The urinary catecholamine assays were carried out in the Department of Pharmacology at the University of Adelaide.

39. Total serum protein was assayed on samples 2, 5, 8 and 11, only.

40. PCV was assayed in the Department of Haematology, the Queen Elizabeth Hospital.

The blood samples constituting the pre-, during and post-treatment experimental periods used in statistical analysis are shown in figure 6.

FIGURE 6. Schematic diagram showing experimental periods and associated blood samples (S_1 to S_{12}) used in statistical analysis of short-term study (2).



Sample 2 was excluded because of possible residual catheterisation stress-induced changes (Copinschi et al, 1967) and in order to equalise the number of samples per experimental period. In accordance with the working hypothesis that reductions in stress-related hormones occur during psychogenic relaxation, the following paired t-tests were used:-

pre- versus during treatment : 1 tailed (fall or no change)[41]

during versus post- treatment : 2 tailed (fall, no change or rise)

pre- versus post- treatment : 1 tailed (fall or no change)

Within-subject comparisons were generally used because they have the advantage of eliminating both the relatively large inter-individual variation in basal hormone concentrations (Fox et al, 1961; Johansson and Post, 1974; Hammond et al, 1976), as well as the need to consider group differences in subject characteristics. For the purpose of making inter-group comparisons, twelve meditators were matched with twelve non-meditator controls, according to their age, and according to the time lapsed between cannulation and first blood sampling for hormone assay (Δ (C-S)). Samples S_2 to S_5, inclusive (see figure 6), which were taken during the hour resting period before treatment, were used in the statistical analysis (unpaired t-tests and 1 way analysis of variance).

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41. With respect to urinary levels, this testing also applies to pre- versus during laboratory study.

PART 4 :- TM RESIDENCE COURSE STUDY

A. Subjects

Four experienced male meditators, five experienced female meditators, five novice male meditators and three novice female meditators were studied.

B. Experimental Design

(a) Outline

As shown in figure 7, the study was conducted over a week-end TM residence course[42] and on the following Monday, which was an ordinary work-day.

(b) Treatment:- intensive meditation in retreat

During the week-end retreat, subjects followed a programme of intensive meditation (4 or 5, 20 minute sessions per day[43]) under supervision of trained TM teachers.

(c) Sampling:- Twenty-four hour urine collection

24 hour urine samples were collected by all subjects during the residence course week-end and on the following Monday. Urine volumes were recorded, and as described in the long-term study (p 20), all urine specimens were treated with preservative and stored until assayed.

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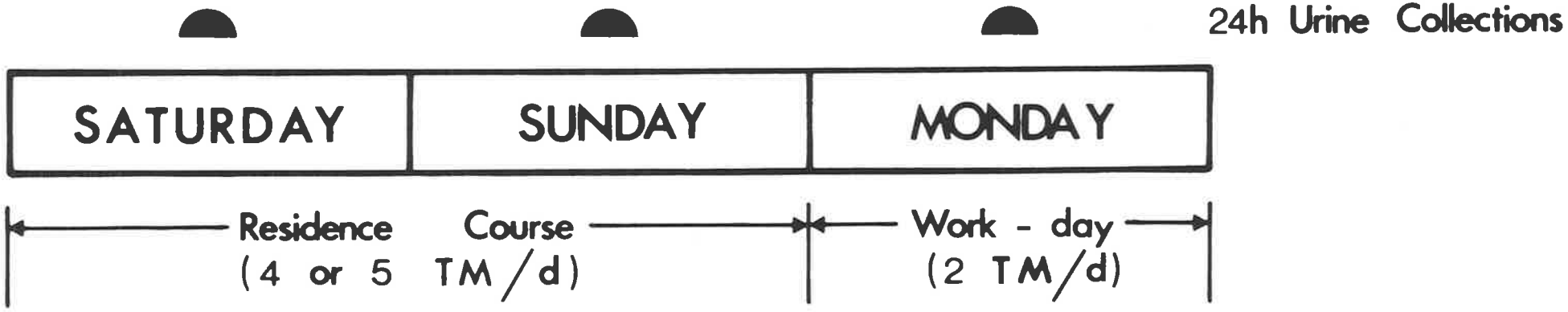
42. The residence course was conducted at Retreat House, Belair, S.A., under the supervision of TM teachers, Mrs. Athena Sikand and Mr. Alexander Wealleans.

43. As mentioned earlier, TM is usually practised for 20 minutes, twice a day.

FIGURE 7. Experimental Design of TM Residence Course Study



C. Independent Variables

(a) Drug usage

In order to minimise possible drug-induced endocrine changes (Gold and Ganong, 1972; Frankenhaeuser, 1971; Levi, 1972; p 31), subjects were asked to abstain from the use of all non-prescribed drugs, except tea, coffee and tobacco, for the duration of the study. Subjects were requested to moderate their usage of tea, coffee and tobacco. No subjects were on medication during the study.

(b) Subject characterisation

Data on age and meditation experience were collected from all subjects.

D. Dependent Variables

UFC and UCA were assayed using the same methods as in the long-term study (p 23).

E. Statistical Analysis

The novice meditation group was used as an active control. Within-group statistical analysis was performed using a Wang 600 desk-top computer.

TABLE 2. SUMMARY OF METHODS

NAME OF STUDY	EXPERIMENTAL DESIGN AND CONDITIONS									
	LENGTH OF STUDY	RELAXATION PROCEDURE (GROUP)	RELAXATION EXPERIENCE	FAMILIARITY WITH EXPERIMENT	TREATMENTS	TIME OF LABORATORY OBSERVATIONS	NUMBER OF SERIAL BLOOD SAMPLES	TIME OF 2½h URINE COLLECTIONS	NUMBER OF 24h URINE COLLECTIONS
LONG-TERM	8 months	Control	-		--		≥3 ≥3		≥2
		TM	+		--				
		TM	++		--				
		PR	+		--				
		AHR	+		--				
		YM	+		--				
SHORT-TERM(1)	210 minutes	Control	-	-	+	1430-1730h	4 4		3
		TM	+	-	++				
		TM	++	-	++				
		PR	+	-	++				
		AHR	+	-	++				
		YM	+	-	++				
SHORT-TERM(2)	210 minutes	Control (group 2)	-	-	+	1400-1630h	10 10 10 10,3	Pre- and During Lab Study	2
		TM (groups1, 1A)	++	-	++				
		TM (group 1B)	++	+	-				
		TM (group 3)	++	-	-				
TM RESIDENCE COURSE	3 days	TM	+	-	+++				3
		TM	++	-	+++				

LEGEND:- RELAXATION EXPERIENCE OR TRAINING

- \- no training
- \+ in training or novice (< 1 yr)
- ++ trained or experienced (> 1 yr)

FAMILIARITY WITH EXPERIMENT

- \- unfamiliar; 1st experience
- \+ 2nd experience

DEPENDENT VARIABLES				STATISTICAL ANALYSIS		
HORMONE ASSAYS	OTHER BIOCHEMICAL AND HAEMATOLOGICAL TESTS	CARDIOVASCULAR MEASUREMENTS	SUBJECTIVE MEASURES	NUMBER OF SUBJECTS	WITHIN-SUBJECT COMPARISONS	BETWEEN-GROUP COMPARISONS
plasma cortisol				13	✓	
serum T_4, T_3					✓	
UFC, UCA					✓	✓
				7		
				8		
				9		
				6		
plasma cortisol				6	✓	✓
serum T_4, T_3					✓	
UFC, UCA					✓	✓
		Heart Rate			✓	✓
		Blood Pressure			✓	✓
			Relaxation Depth			
				4		
				8		
				4		
				4		
serum cortisol, blood lactate				14	✓	✓
hGH, prolactin,					✓	✓
T_4, FTI, T_3, rT_3,					✓	✓
UFC, UCA					✓	✓
	PCV, serum protein				✓	
UFC, UCA	serum osmolality				✓	
	urine flow, creatinine clearance				✓	
			Relaxation Depth			✓
				16(5)		
				5		
				5		
UFC, UCA				8	✓	
				9		

EXPERIMENTAL TREATMENT

-	reading and talking quietly
--	ordinary, everyday life situation
+	non-specific or ordinary relaxation
++	specific relaxation
+++	intensive meditation in retreat

CHAPTER III : RESULTS

Preface to Results

Data on each individual from the long-term study, short-term study (1), short-term study (2) and TM residence course study are contained in Appendix IV, sections A, B, C and D, respectively.

Where analysis of variance (ANOVA) was used, results are given in the standard notation $F(v_1, v_2)$. Results of t-tests are presented using the standard form t(df), while results from linear regression analysis are given as r(df) (Lehmann, 1959; p 273, p 172, pp 192 and 206, and p 181 for one-way ANOVA, Student's t-test, paired t-test, and linear regression analysis, respectively). A negative t value denotes that the mean of the first sample is lower than the mean of the second sample being compared.

Levels of statistical significance were adjusted for multiple comparisons, using the formula: $p_n = 1-(1 - p_o)^n$, where n = number of multiple comparisons; p_o = original probability; and p_n = adjusted probability (Cox and Hinkley, 1974; pp 77-78). The criterion for statistical significance was set at $p < 0.05$, and NS, *, **, and *** are used to indicate that $p > 0.05$, $p < 0.05$, $p < 0.01$, and $p < 0.005$, respectively.

Hatched lines parallel to the ordinate are used in figures to represent the upper limit of the normal basal resting range for the variable concerned.

Whenever means are referred to in the text, standard errors of the means (SEM's) have also been calculated. The term overall is used where an inter-group statistical comparison(s) has been made using several estimations from each subject. Not available, and not applicable, are abbreviated NAv and NAp, respectively.

Results of the long-term study, short-term study (1) and the TM residence course study were originally calculated in Imperial units. Any slight discrepancies between statistics given here and statistics derived from the raw data in Appendix IV are attributable to conversion of results to *Système International d'Unités* following statistical analysis.

PART 1 :- LONG-TERM STUDY

A. Group Characteristics

Table 3 gives the number of subjects in each group who continued giving samples and, where applicable, continued relaxation training for the duration of the long-term study. Due to a high attrition from relaxation training and infrequency of sample collection[44] in some subjects, sample sizes were reduced considerably. The PR and AHR groups were combined for purposes of statistical analysis because results on only two AHR subjects were available. Similarly, a combined UFC and UCA analysis was made on all in-training subjects because of small sample sizes for individual groups.

The least attrition from relaxation training occurred for the YM group in which seventy percent of subjects who commenced training were still practising[45] YM after seven months. Relaxation training attrition rates of fourty percent, sixty percent, and sixty-five percent occurred during the seven months' training for the TM, PR and AHR groups, respectively[46].

The mean ages of subjects in each group were very similar (Table 3). The average TM experience of the trained meditation group

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44. Subjects were included in within-group statistical analysis if they had given three or more samples during the study. The limit was reduced to two samples per subject for inter-group urinary hormone comparisons.

45. Practising was defined arbitrarily as using the relaxation procedure more than four times a week for at least fifteen minutes per session. It should be noted that this is much less than the usual fourteen sessions per week of trained meditation subjects.

46. Any discrepancies between the number of subjects used in the results section and the attrition rates given here, are due to exclusion from statistical analysis of some subjects whose sample collection was infrequent; even though they were still practising their respective relaxation procedure after seven months' training.

was 3.4 (± 0.6) years with a range of 1.0 to 7.2 years.

TABLE 3. Group Characteristics for the Long-term Study

TRAINING	RELAXATION PROCEDURE	NUMBER	AGE (yr.)
Trained	Transcendental Meditation (TM)	8	23.7 ± 1.1
Untrained	None	14	20.0 ± 0.5
In-training	Transcendental Meditation	7	22.8 ± 1.7
In-training	Progressive Relaxation (PR)	7	22.5 ± 2.4
In-training	Autohypnosis Relaxation (AHR)	2	
In-training	Yoga Meditation (YM)	6	20.1 ± 0.7

B. Temporal Hormone Changes

Results from one-way ANOVA on each group, and on each dependent variable, are presented in table 4:-

TABLE 4. Statistics from One-way ANOVA on Results of the Long-term Study

TRAINING	RELAXATION PROCEDURE(S)	PLASMA CORTISOL	SERUM T_4	SERUM T_3	UFC	UCA
Trained	TM	11.32(3,17)***	2.03(3,16)	3.37(3,18)*	2.08(4,24)	0.21(4,24)
Untrained	NAp	5.35(5,52)***	3.68(4,39)*	2.14(4,41)	0.99(3,25)	2.73(3,22)
In-training	TM	5.22(4,20)**	1.37(4,23)	0.14(4.23)	NAv	NAv
In-training	PR & AHR	0.88(5,30)	4.46(2,14)*	0.56(3,19)	NAv	NAv
In-training	YM	2.74(3,16)	NAv	NAv	NAv	NAv
In-training	Combined	NAp	NAp	NAp	0.46(4,36)	0.04(2,23)

Mean hormone levels of groups showing significant F values are given in tables 5, 6 and 7. In order to provide the best interpretation of any alterations in thyroid function, when either T_3 or T_4 showed significant changes, means were also calculated for the thyroid hormone in which no significant change was found.

T_4 levels of both untrained controls and PR trainees showed a general decline during the study (Table 5), with a significant rise during the end-of-year exams in November ($t = -2.69$; $df = 10$ [47]; $p < 0.05$; 2-tailed paired t-test). In contrast, T_3 values of both untrained controls and PR trainees fell during the exams (Table 6).

TABLE 5. Mean ± SEM Serum T_4 Levels (n mol/l) During the Long-term Study

TRAINING	RELAXATION PROCEDURE	MAR	APR	MAY	JULY	SEPT	NOV
Untrained	NAp	NAv	97 ± 5	99 ± 4	85 ± 2	83 ± 6	104 ± 4
In-training	PR	NAv	118 ± 13	104 ± 13	101 ± 6	77 ± 4	111 ± 17
Trained	TM	NAv	101 ± 4	108 ± 10	85 ± 5	88 ± 4	NAv

TABLE 6. Mean ± SEM Serum T_3 Levels (n mol/l) During the Long-term Study

TRAINING	RELAXATION PROCEDURE	MAR	APR	MAY	JULY	SEPT	NOV
Trained	TM	NAv	2.9±0.2	2.7±0.2	2.2±0.2	2.1±0.2	NAv
Untrained	NAp	NAv	2.5±0.1	2.3±0.1	2.4±0.1	2.6±0.2	2.1±0.2
In-training	PR	NAv	2.6±0.1	NAv	2.5±0.3	2.5±0.2	2.1±0.3

A progressive fall in plasma cortisol was observed during the study for both trained TM subjects and TM trainees (Table 7). A similar trend was found for the untrained control group which also showed a significant rise in May ($t = -10.74$; $df = 6$; $p < 0.005$; 2-tailed paired t-test), with a subsequent fall in July ($t = 4.87$; $df = 8$; $p < 0.005$; 2-tailed paired t-test). T_3 levels of the trained meditators followed the same trend as plasma cortisol, levels tending to progressively fall during the study (Tables 6 and 7).

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47. A combined analysis on untrained controls and PR subjects was made because only three PR subjects were available for comparison.

TABLE 7. Mean ± SEM Plasma Cortisol Levels (n mol/l) During the Long-Term Study

TRAINING	RELAXATION PROCEDURE	MAR	APR	MAY	JULY	SEPT	NOV
Trained	TM	NAv	375±34	NAv	258±19	182±31	159±28
Untrained	NAp	232±26	169±32	319±27	211±23	221±27	136±13
In-training	TM	NAv	304±31	283±47	190±41	190±18	128±20

No significant temporal changes in UFC or UCA were observed for any group. However, the UFC and UCA levels of one TM trainee (subject 90), whose initial pre-training levels were well above the upper limit of the normal reference ranges[48] (280 n mol/d and 550 n mol/d, for UFC and UCA respectively), fell markedly from the onset of relaxation training to values within the normal ranges. As shown in figure 8, UCA levels of subject 90 continued to fall during TM training. The changes in UFC during training followed a similar pattern as UCA, however, they occurred less rapidly, lagging behind the UCA changes by several months. After seven months' TM training, both the UFC and UCA levels appeared to have stabilised well within the normal reference ranges (Figure 8). The UFC and UCA levels of the other TM trainees were well within the normal reference ranges before training commenced. The levels of these subjects showed minor fluctuations during the study (For individual data, see Appendix IV, Section A, pp 175 and 176).

C. Overall Inter-group Urinary Hormone Comparisons

Overall UFC and UCA levels (Mean ± SEM) of males during the training period (May, July and September) are given in table 8:-

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48. Subject 90 had no overt signs of Cushing's disease or hypertension, nor did he have a history of psychiatric illness.

FIGURE 8. Urinary Hormone Levels of Subject 90 Before and During TM Training

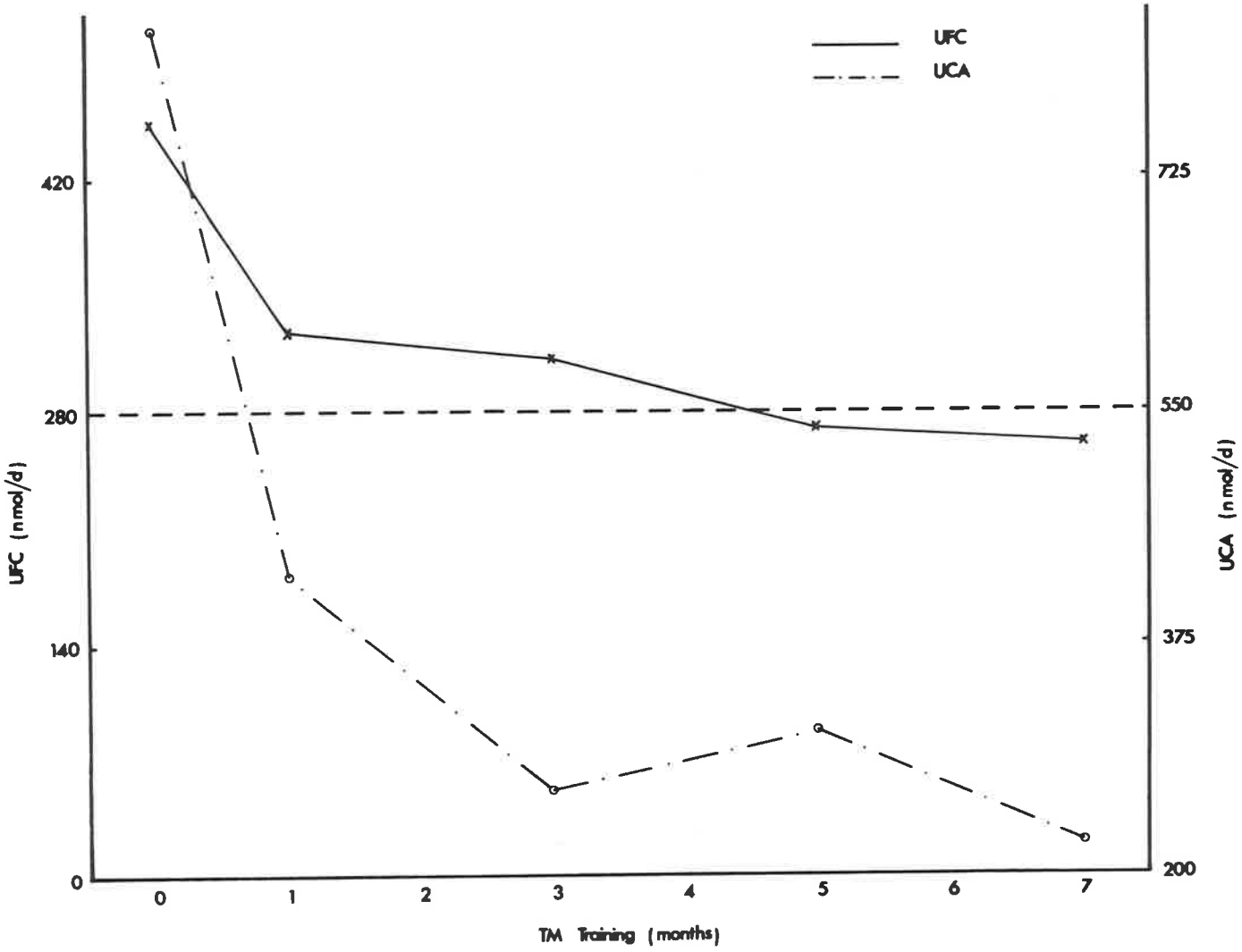

TABLE 8. Overall UFC and UCA Levels (Mean ± SEM) of the Long-term Study

TRAINING	UFC (n mol/d)	UCA (n mol/d)
Trained	151 ± 9	272 ± 17
In-training	181 ± 16	338 ± 30
Untrained	191 ± 13	361 ± 25

Results of Student's t-tests between the urinary hormone levels of the three groups are presented in table 9. Overall UFC levels were significantly lower in the trained meditators than in the untrained controls. UFC levels of the trained meditators were also lower than the levels of relaxation trainees, however, the difference was not statistically significant. There was no significant difference between UFC levels of relaxation trainees and untrained controls. No significant inter-group differences in UCA levels were found. The UFC and UCA results are summarised in figure 9.

TABLE 9. Statistics from Overall Inter-group UFC and UCA Comparisons for the Long-term Study

	UFC (1-tailed t-tests)			UCA (2-tailed t-tests[49])		
TRAINING	Trained	In-training	Untrained	Trained	In-training	Untrained
Trained	-	-1.73(38)	-2.47(37)*	-	-2.00(36)	-1.27(36)
In-training	-	-	-0.51(35)	-	-	-0.58(32)
Untrained	-	-	-	-	-	-

(p value was corrected for 3 multiple comparisons)

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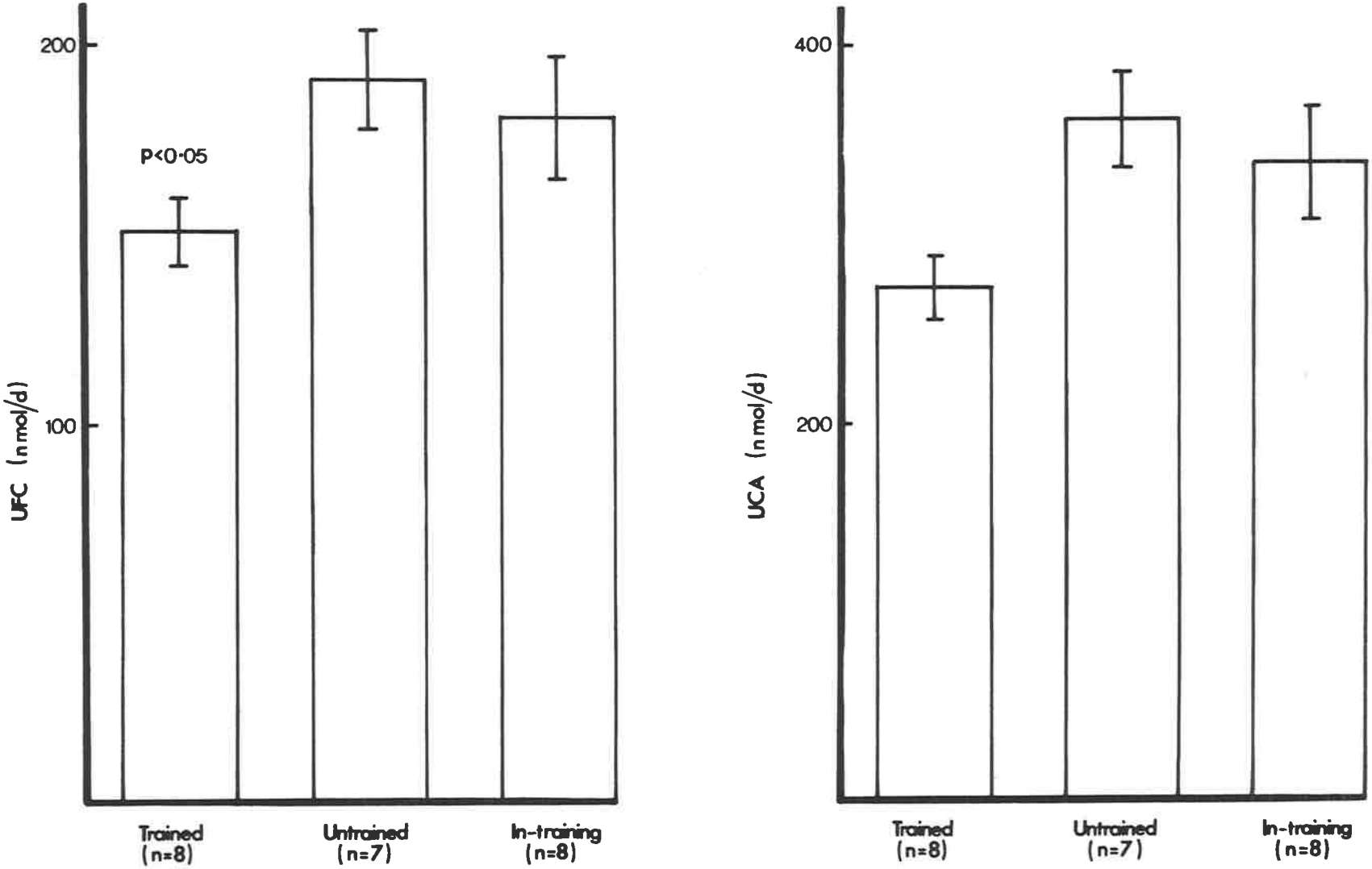
49. Two-tailed tests were used for UCA analysis in order to be consistent with later UCA findings (Short-term Study (2)), where significantly higher UCA levels were found in meditators than in matched non-meditator controls.

FIGURE 9. Overall Urinary Hormone Levels (Mean±SEM) of Long-term Study



PART 2 :- SHORT-TERM STUDY (1)

A. Group Characteristics

The mean ages of subjects in all groups were similar (Table 10). The average TM experience of the trained meditator group was 3.6 (± 0.7) years, with a range of 1.0 to 7.2 years.

TABLE 10. Group Characteristics for Short-term Study (1)

TRAINING	RELAXATION PROCEDURE	NUMBER	AGE (yr.)
Trained	TM	8	22.8 ± 0.7
Untrained	NAp	6	20.1 ± 1.1
In-training	TM	4	25.5 ± 2.0
In-training	PR and AHR	4	19.2 ± 0.4
In-training	YM	4	20.8 ± 1.3
In-training	TM, PR and AHR	8	22.8 ± 1.5

For the purpose of statistical analysis, the PR and AHR groups were combined because of very small sample sizes for these groups. For similar reasons a combined analysis was also performed on novice TM, PR and AHR subjects[50], thus making the sample size suitably comparable with both the trained and untrained groups. A larger sample size also reduces the possibility of a type 2 statistical error (Croxton and Cowden, 1965; p 639). The in-training group referred to in figures 10 and 11 represents a combined novice TM, PR and AHR group.

B. Cardiovascular Changes

Table 11 gives pre- and post- relaxation heart rate, and mean arterial blood pressure (BP) levels (mean ± SEM) for each group:-

50. Relaxation training was very similar for these groups (see pp 19 and 20 of Methods and Appendix II, pp 154-159).

TABLE 11. Heart Rate and Mean Arterial Blood Pressure Levels (Mean ± SEM) Before and After Relaxation

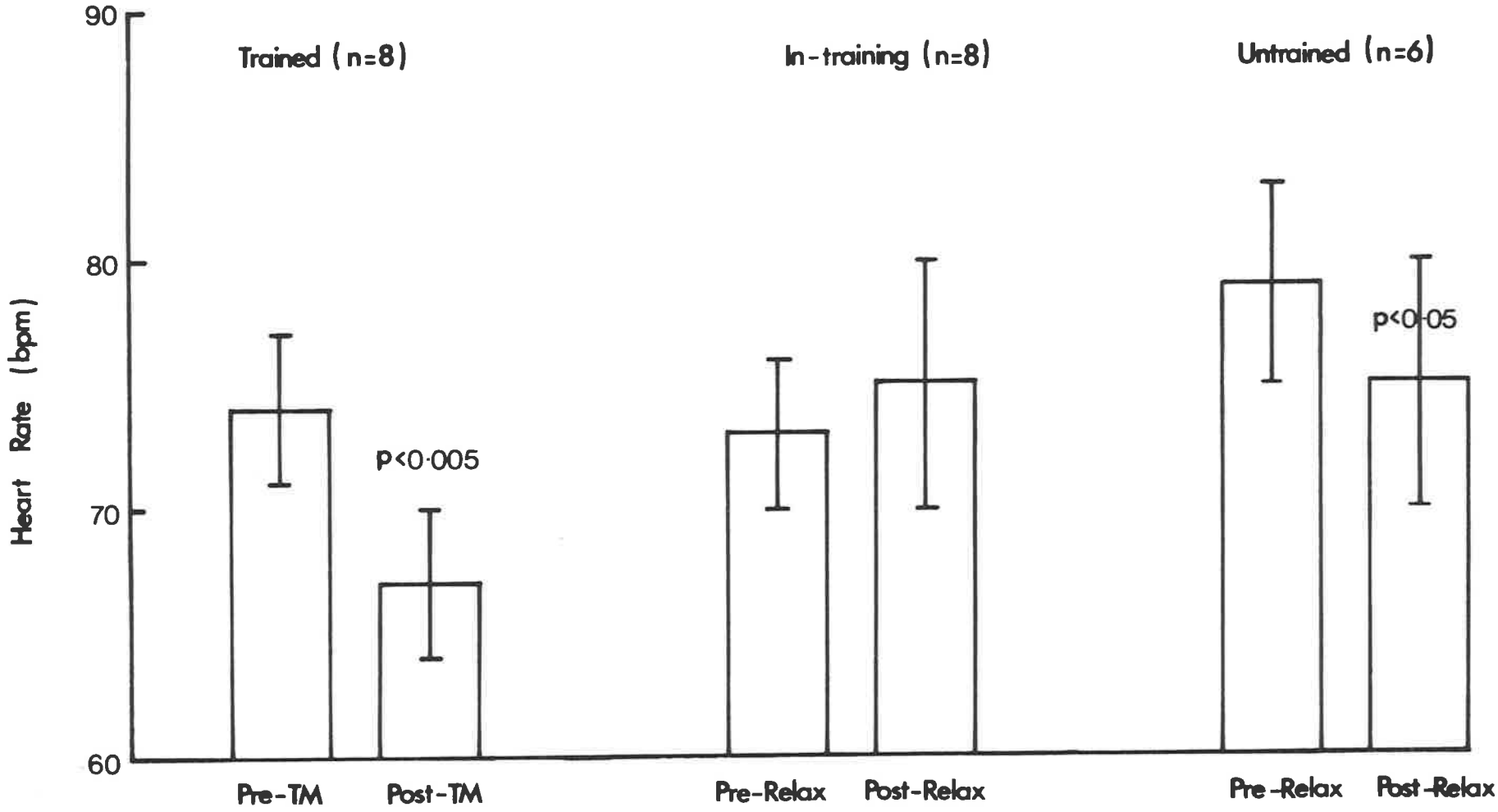
TRAINING	RELAXATION PROCEDURE	HEART RATE (bpm)		MEAN ARTERIAL BP (mm Hg)	
		Pre-	Post-	Pre-	Post-
Trained	TM	74 ± 3	67 ± 3	97 ± 1	97 ± 1
Untrained	NAp	79 ± 4	75 ± 5	99 ± 4	101 ± 3
In-training	TM	70 ± 2	66 ± 3	95 ± 3	100 ± 3
In-training	PR and AHR	77 ± 5	85 ± 8	94 ± 4	95 ± 4
In-training	YM	73 ± 5	75 ± 7	98 ± 4	94 ± 2
In-training	TM, PR and AHR	73 ± 3	75 ± 5	94 ± 2	97 ± 2

Results of within-group paired t-test comparisons on heart rate and blood pressure levels are presented in Table 12. Heart rate changes in relaxation are summarised in figure 10. A significant 9 percent reduction in heart rate occurred following meditation for the experienced TM group. A slight, but statistically significant, 5 percent fall in heart rate was also observed in the control group of untrained subjects. No significant changes in heart rate were found in individual or combined groups of relaxation trainees. No significant within-group changes in mean arterial blood pressure were observed, except for a fall following yoga-meditation (Table 12).

TABLE 12. Statistics from Within-group Comparisons on Heart Rate and Blood Pressure Levels.

TRAINING	RELAXATION PROCEDURE	HEART RATE	MEAN ARTERIAL BP
Trained	TM	4.70(7)***	0.47(7)
Untrained	NAp	2.63(5)*	-1.43(5)
In-training	TM	0.88(3)	-0.98(3)
In-training	PR and AHR	-1.76(3)	-1.86(3)
In-training	YM	-0.80(3)	2.72(3)*
In-training	TM, PR and AHR	-0.57(7)	-1.45(7)

FIGURE 10. Heart Rate (Mean ± SEM) Before and After Relaxation



Results of inter-group[51] Student's t-test comparisons on pre-relaxation heart rate and blood pressure are given in table 13. No significant inter-group differences in either heart rate or blood pressure were found.

TABLE 13. Statistics from Between-group Comparisons on Heart Rate and Blood Pressure Levels

TRAINING	HEART RATE			MEAN ARTERIAL BP		
	Trained	In-training	Untrained	Trained	In-training	Untrained
Trained	-	0.51(11)	1.26(11)	-	1.08(11)	0.52(11)
In-training	-	-	1.46(10)	-	-	1.10(10)
Untrained	-	-	-	-	-	-

C. Relaxation Depth Results

The medians and ranges of relaxation depth ratings for all groups are shown in table 14. Surprisingly, the median relaxation depth scores were lower in both the experienced and novice TM groups than in all other groups including the untrained controls. The yoga-meditation group had the highest median self-reported depth of relaxation.

TABLE 14. Relaxation Depth Ratings from Short-term Study (1)

TRAINING	RELAXATION PROCEDURE	NUMBER	RELAXATION DEPTH	
			Median	Range
Trained	TM	8	3	1 to 5
Untrained	NAp	6	4	1 to 6
In-training	TM	4	3	1 to 5
In-training	PR and AHR	4	4	1 to 5
In=training	YM	4	4.5	3 to 6
In-training	TM, PR and AHR	8	3.5	1 to 5

51. Inter-group comparisons were made on males only because of the intrinsic sex differences in cardiovascular function (Hamilton et al, 1954), and because the majority of subjects were males.

D. Blood Hormone Changes

Pre- and post- relaxation hormone levels (mean ± SEM) for each group are presented in table 15:-

TABLE 15. Mean ± SEM Hormone Levels (n mol/l) Before and After Relaxation

TRAINING	RELAXATION PROCEDURE	PLASMA CORTISOL		SERUM T_4		SERUM T_3	
		Pre-	Post-	Pre-	Post-	Pre-	Post-
Trained	TM	148±22	98±18	92±6	91± 4	2.3±0.1	2.5±0.2
Untrained	NAp	113±17	106±15	96±1	97± 2	2.4±0.1	2.5±0.2
In-training	TM	183±56	176±55	80±2	90± 4	2.5±0.3	2.7±0.2
In-training	PR and AHR	198±13	172±26	89±6	89± 9	2.0±0.1	2.4±0.2
In-training	YM	179±50	121±28	108±4	110±11	2.3±0.3	2.3±0.1
In-training	TM,PR and AHR	190±27	174±28	85±3	90± 4	2.2±0.2	2.5±0.1

Results of within-group paired t-test comparisons on serum hormone levels are given in table 16. Irrespective of type of relaxation training, thyroid hormones generally rose during the study. However, statistically significant T_4 increases were observed only for the TM trainees, while statistically significant T_3 rises occurred only for PR and AHR trainees (Table 16).

TABLE 16. Statistics from Within-Group Comparisons on Serum Hormone Levels of Short-term Study (1)

TRAINING	RELAXATION PROCEDURE	PLASMA CORTISOL	SERUM T_4	SERUM T_3
Trained	TM	7.56(7)***	0.36(7)	-1.75(6)
Untrained	NAp	0.36(5)	-0.48(5)	-0.54(5)
In-training	TM	0.32(3)	-3.26(3)*	-0.79(2)
In-training	PR and AHR	1.18(3)	0.00(3)	-3.06(3)*
In-training	YM	2.05(2)	-0.28(2)	-0.38(2)
In-training	TM, PR and AHR	1.12(7)	-1.60(7)	-2.59(6)*

Plasma cortisol findings are summarised in figure 11. A significant 34 percent reduction in plasma cortisol was observed following TM for the trained meditators, in contrast to no significant change for relaxation trainees or untrainecd controls (Table 16). The pre-treatment plasma cortisol levels of untrained controls were lower than pre-treatment values of both trained and in-training groups (Table 15), however, the differences were not statistically significant ($t = 0.97$; $df = 11$; $p > 0.05$ and $t = 2.02$; $df = 10$; $p > 0.05$, respectively; 2-tailed Student's t-tests).

E. Summary of Short-term Endocrine and Cardiovascular Changes

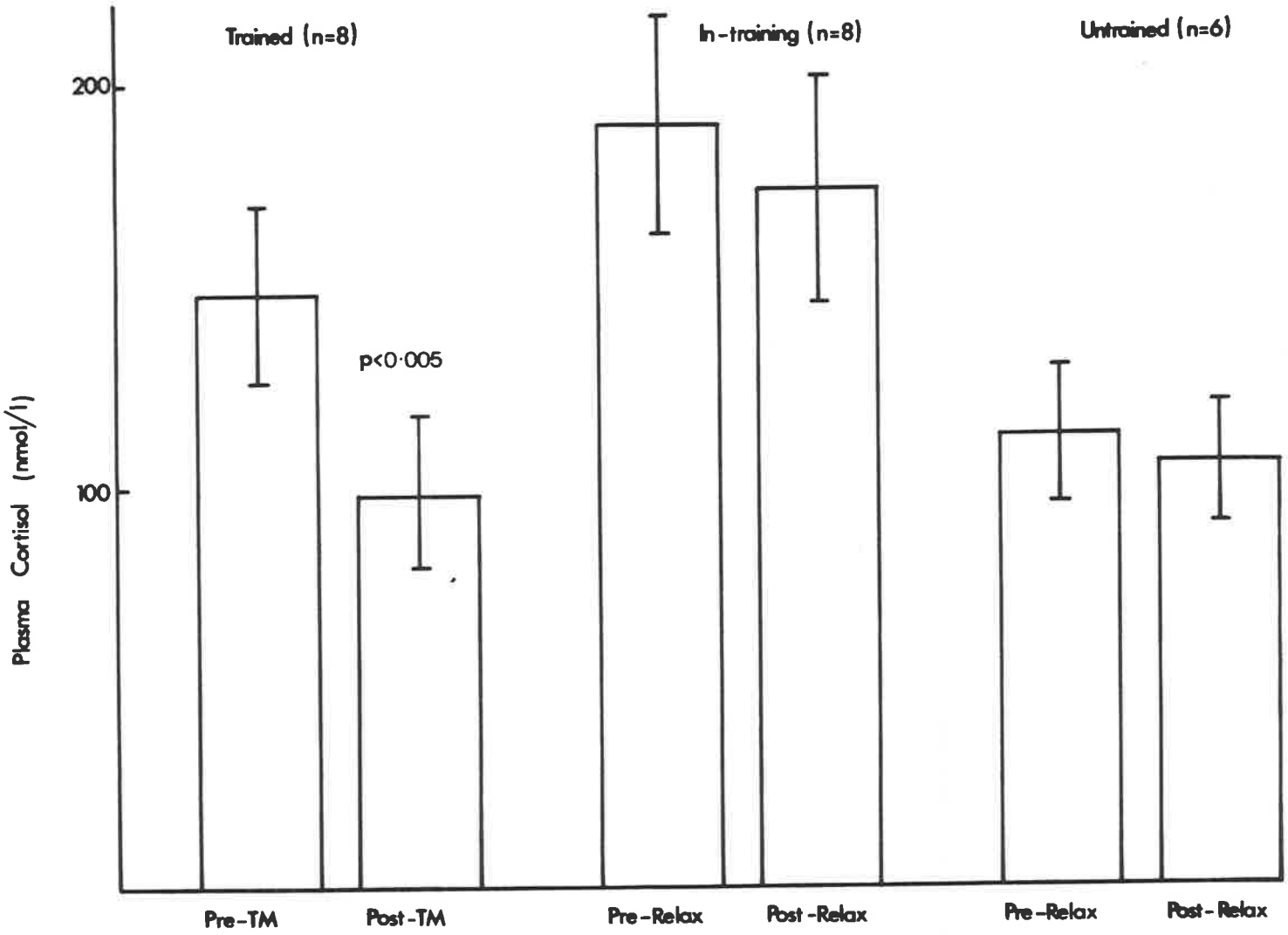
Table 17 summarises the endocrine and cardiovascular changes observed in relaxation procedures. The most outstanding findings were:- 1) a significant reduction in plasma cortisol following TM of experienced meditators, in contrast to a lack of significant change for both relaxation trainees and untrained controls; and 2) a marked heart rate fall in TM of experienced subjects, in contrast to a slight decrease following ordinary relaxation of untrained controls and no change for relaxation trainees.

TABLE 17. Summary of Endocrine and Cardiovascular Changes Following Relaxation

MEASUREMENT	TRAINED	IN-TRAINING		UNTRAINED
	TM	TM	PR + AHR	YM	-
plasma cortisol	↓↓↓	NS	NS	NS	NS
serum T_4	NS	↑	NS	NS	NS
serum T_3	NS	NS	↑	NS	NS
mean arterial BP	NS	NS	NS	↓	NS
heart rate	↓↓↓	NS	NS	NS	↓

↑ ; ↓ significant rise or fall, respectively; $p < 0.05$
↓↓↓ significant fall; $p < 0.005$

FIGURE 11. Plasma Cortisol Levels (Mean±SEM) Before and After Relaxation



F. Urinary Hormone Results

Group mean overall UFC and UCA levels of males from the week-end of the laboratory study and the following Monday are shown in table 18:-

TABLE 18. Overall UFC and UCA Levels (Mean ± SEM) of Short-term Study (1)

TRAINING	RELAXATION PROCEDURE	NUMBER	UFC (n mol/d)	UCA (n mol/d)
Trained	TM	7	53 ± 3	312 ± 24
In-training	TM	4	75 ± 8	286 ± 20
In-training	PR, AHR and YM	5	74 ± 9	262 ± 21
Untrained	NAp	6	74 ± 6	338 ± 22

Results from Student's t-test inter-group comparisons are given in tables 19 and 20 for UFC and UCA, respectively:-

TABLE 19. Statistics from Between-group Comparisons on UFC Results of Short-term Study (1)

GROUP	Experienced Meditators	Novice Meditators	Other Trainees	Untrained Controls
Experienced Meditators	-	2.92(30)*	2.56(33)	3.30(36)*
Novice Meditators	-	-	-0.09(25)	-0.06(28)
Other Trainees	-	-	-	-0.05(31)
Untrained Controls	-	-	-	-

(p values were corrected for 6 multiple comparisons)

TABLE 20. Statistics from Between-group Comparisons on UCA Results of Short-term Study (1)

GROUP	Experienced Meditators	Novice Meditators	Other Trainees	Untrained Controls
Experienced Meditators	-	0.75(30)	1.48(33)	-0.78(36)
Novice Meditators	-	-	-0.79(25)	-1.64(28)
Other Trainees	-	-	-	-2.43(31)
Untrained Controls	-	-	-	-

(p values were corrected for 6 multiple comparisons)

Overall UFC and UCA results of the short-term study (1) are summarised in figure 12. As in the long-term study, UFC levels were significantly lower in the experienced meditators than in the untrained controls. UFC levels of the trained meditators were also lower than the other relaxation trainees, however, the difference was not statistically significant. No significant differences in UFC levels of novice meditators, other trainees, or untrained controls were found (Table 19). UCA levels of relaxation trainees (both novice meditators and other trainees) were slightly lower than UCA levels of both trained and untrained subjects, however, the difference was not statistically significant (Table 20).

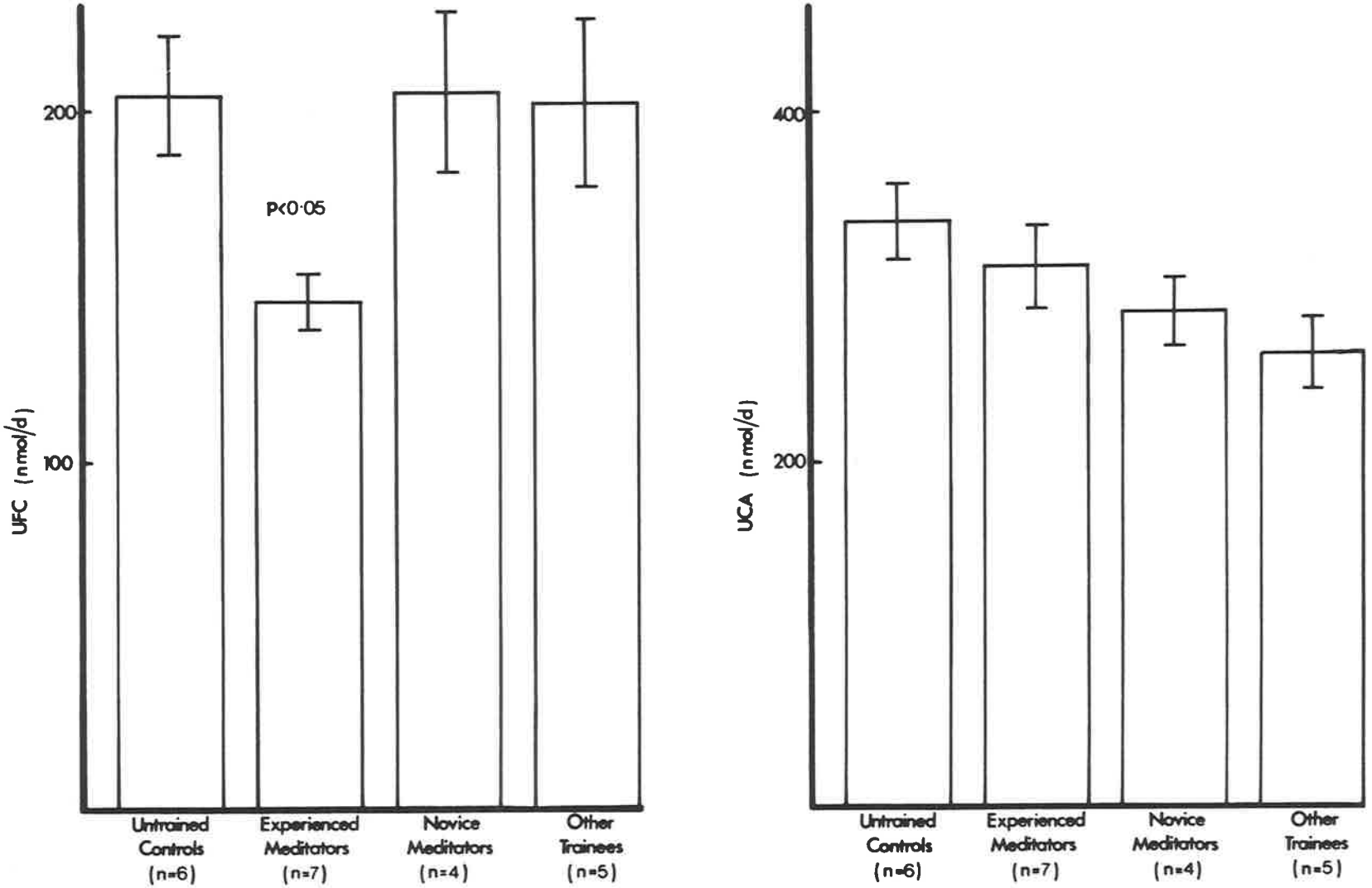
Results of paired t-test comparisons on UFC levels over the week-end of the laboratory study and on the following Monday are given in table 21. No significant changes in UFC levels over the week-end and following Monday were observed in any group.

TABLE 21. Statistics from Within-group Comparisons Between Saturday, Sunday, and Monday UFC Levels of Short-term Study (1)

	Sat cf Sun	Sun cf Mon	Sat cf Mon
Experienced Meditators	0.95(7)	1.94(6)	0.86(6)
Novice Meditators	1.29(3)	0.27(3)	1.69(3)
Other Trainees	1.62(6)	2.26(6)	0.17(6)
Untrained Controls	0.78(5)	1.38(5)	0.30(5)

(p values were corrected for 3 multiple comparisons)

FIGURE 12. Overall Urinary Hormone Levels (Mean±SEM) of Short-term Study (1)



PART 3 :- SHORT-TERM STUDY (2)

A. Group Characteristics

The mean ages of subjects in each treatment group were very alike (Table 22). The average TM experience of subjects in groups 1 and 1A were very similar, with ranges of 1.1 to 9 years and 1.1 to 6 years, respectively. However, the mean TM experience of group 3 was 0.8[52] to 5.1 years.

TABLE 22. Group Characteristics for Short-term Study (2)

GROUP	SUBJECTS	TREATMENT	NUMBER	AGE(yr)	TM EXPERIENCE(yr)
1	Meditators	TM	16	27 ± 2	4.0 ± 0.6
2	Non-meditators	Ordinary relaxation	15	26 ± 1	None
1A	Meditators	TM	5	24 ± 2	4.3 ± 0.7
1B	Meditators	Quiet rest	5		
3	Meditators	Quiet rest	5	27 ± 2	2.4 ± 0.7 [52]

B. Statistical Analysis Using Non-meditators as Controls

(a) Blood lactate and serum glucose

Mean pre-, during and post- meditation or ordinary relaxation blood lactate and serum glucose levels are given in table 23:-

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52. One volunteer with nine months' TM experience was included in group 3 because only four previously unstudied subjects with more than one year's TM experience (ie experienced meditators; see p 17) were available for study.

TABLE 23. Blood Lactate and Serum Glucose Levels (Mean ± SEM) Before, During and After TM or Ordinary Relaxation

TEST	EXPERIMENTAL PERIOD	TREATMENT	
		TM	Ordinary Relaxation
Blood Lactate (m mol/l)	Pre-	0.97 ± 0.12	1.48 ± 0.14
	During	0.83 ± 0.14	1.48 ± 0.15
	Post-	0.77 ± 0.09	1.52 ± 0.16
Serum Glucose (n mol/l)	Pre-	4.2 ± 0.2	4.8 ± 0.1
	During	4.4 ± 0.1	4.6 ± 0.1
	Post-	4.2 ± 0.1	4.4 ± 0.1

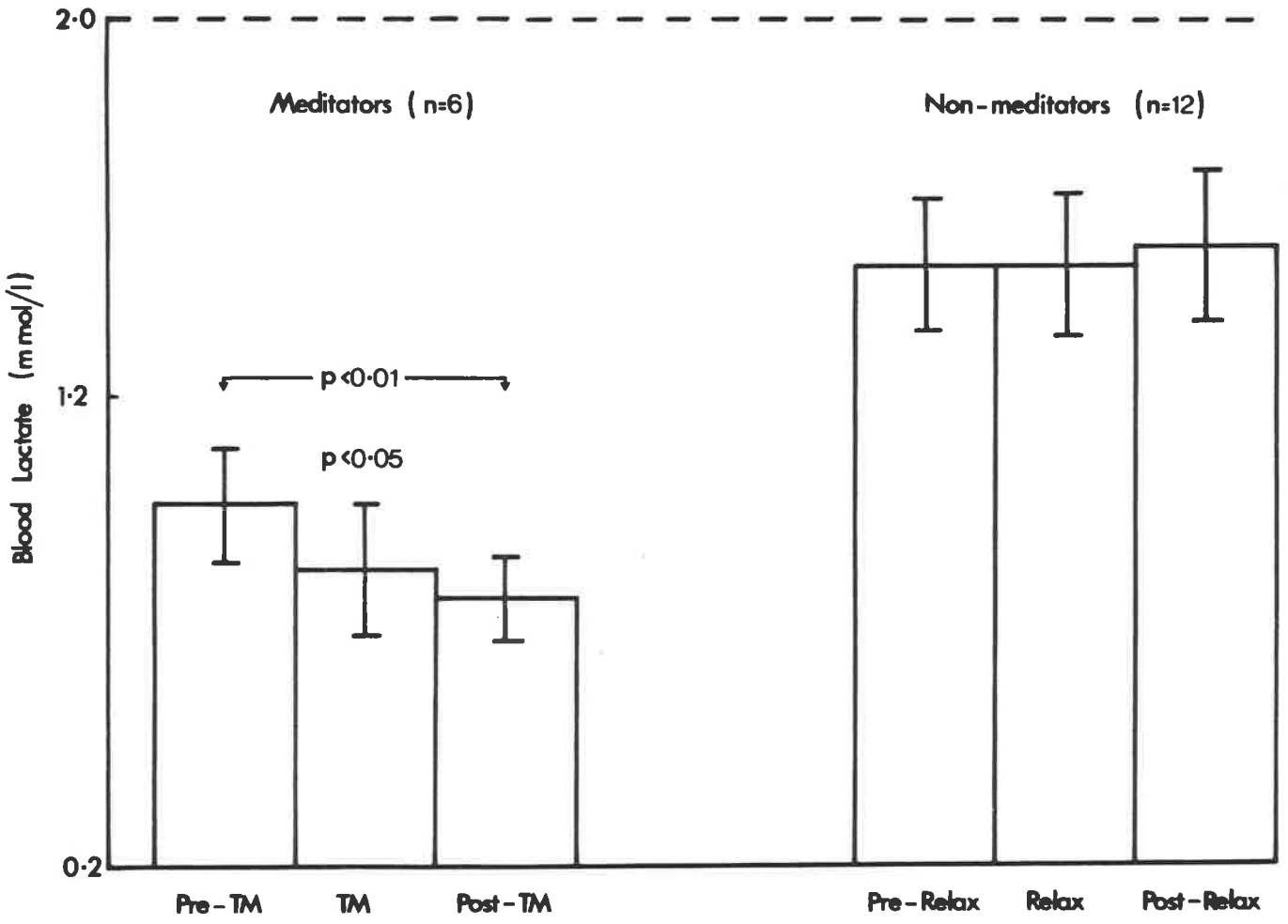
Results of within-group paired t-test comparisons between blood lactate levels and serum glucose levels from pre-, during and post-treatment periods are presented in table 24. Blood lactate fell significantly during TM in contrast to no significant change during ordinary relaxation of non-meditator controls. Lactate levels continued to fall during the post-meditation period (Figure 13). No statistically significant changes in serum glucose were observed in TM or ordinary relaxation, however, a slight rise occurred during TM and levels of the non-meditator controls progressively fell throughout the study (Table 24).

TABLE 24. Statistics from Within-group Comparisons on Blood Lactate and Serum Glucose Levels

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
Blood lactate	4.07(5)*	1.12(5)	6.44(5)**
Serum glucose	-1.94(15)	2.24(15)	0.10(15)
	PRE-RELAX cf RELAX	RELAX cf POST-RELAX	PRE-RELAX cf POST-RELAX
Blood lactate	0.06(11)	0.28(11)	0.28(11)
Serum glucose	1.61(13)	2.63(13)	3.16(13)

(p values were corrected for 3 multiple comparisons)

FIGURE 13. Blood Lactate Levels (Mean±SEM) Before, During and After TM or Ordinary Relaxation



(b) Subjective Changes

The median (range) meditation or ordinary relaxation depth ratings were 15 (10 to 18) and 9.5 (7 to 15), respectively. The depth of relaxation during meditation was significantly greater than the depth of relaxation during ordinary relaxation in non-meditator controls ($U = 20$; $n_1 = 16$, $n_2 = 14$; $p < 0.005$; Mann-Whitney U test; Siegal, 1956; pp 116-126). Meditation depth was higher during the laboratory meditation than during the control Saturday afternoon home meditation, when the median depth was 13.5. The difference bordered on statistical significance at $p < 0.05$ ($T = 14.5$; $N = 12$; Wilcoxon matched-pairs signed-ranks test; Siegal, 1956; pp 75-83). However, home meditation depth ratings were still higher than laboratory relaxation depth scores of non-meditator controls ($U = 42.5$; $n_1 = 14$, $n_2 = 14$; $p < 0.05$; Mann-Whitney U test). There was no significant difference between the self-reported experimental stress of the meditators and non-meditator controls ($U = 106.5$; $n_1 = 16$, $n_2 = 14$; $p > 0.05$; Mann-Whitney U test).

(c) Blood Tests

(i) Hormones

Mean pre-, during and post-meditation or ordinary relaxation levels of each blood hormone test are shown in table 25:-

TABLE 25. Hormone Test Levels (Mean ± SEM) Before, During and After TM or Ordinary Relaxation

TEST	EXPERIMENTAL PERIOD	TREATMENT	
		TM (n=16)	Ordinary Relaxation (n=14[53])
Serum Cortisol (n mol/l)	Pre-	210 ± 26	292 ± 37
	During	169 ± 23	266 ± 46
	Post-	174 ± 24	227 ± 36
Serum Total T_4 (n mol/l)	Pre-	115 ± 5	105 ± 3
	During	111 ± 5	105 ± 3
	Post-	114 ± 5	106 ± 3
FTI (Units)	Pre-	111 ± 4	106 ± 3
	During	108 ± 4	105 ± 3
	Post-	111 ± 4	104 ± 3
Serum Total T_3 (n mol/l)	Pre-	1.5 ± 0.1	1.5 ± 0.1
	During	1.4 ± 0.1	1.5 ± 0.1
	Post-	1.4 ± 0.1	1.6 ± 0.1
Serum Total rT_3 (n mol/l)	Pre-	0.27±0.02	0.23±0.01
	During	0.28±0.02	0.25±0.01
	Post-	0.30±0.02	0.29±0.02
Serum hGH (m U/l)	Pre-	5.6 ± 1.6	7.4 ± 2.0
	During	2.8 ± 0.9	5.9 ± 1.9
	Post-	3.2 ± 0.6	4.5 ± 1.3
Serum Prolactin (m U/l)	Pre-	111 ± 12	147 ± 18
	During	101 ± 12	140 ± 17
	Post-	98 ± 12	126 ± 13

Results of paired t-test comparsions between pre-, during and post- TM or ordinary relaxation serum hormone levels are given in tables 26 and 27, respectively. Serum cortisol, T_4, T_3, hGH, prolactin, and FTI fell significantly during TM in contrast to no significant change during ordinary relaxation of non-meditator controls. Serum cortisol, hGH and prolactin findings are summarised in figures 14, 15 and 16, respectively. No significant rT_3 changes were observed.

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53. Blood clotting in the cannula of subject 18 led to re-cannulation attempts which were, however, thwarted by venospasm. Consequently, subject 18 was excluded from the blood sampling part of the study.

TABLE 26. Statistics from Within-TM Group Comparisons on Serum Hormone Test Levels

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
cortisol	2.96(15)*	-0.44(15)	1.66(15)
T_4	2.88(14)*	-1.58(14)	0.53(14)
FTI	3.50(14)**	-1.36(14)	-0.10(14)
T_3	2.66(14)*	-1.38(14)	1.42(14)
rT_3	-0.71(14)	-1.66(14)	-1.58(14)
hGH	3.28(15)*	-0.36(15)	1.44(15)
prolactin	3.20(15)*	0.73(15)	2.78(15)*

(p values were corrected for 3 multiple comparisons)

TABLE 27. Statistics from Within-non-meditator Group Comparisons on Serum Hormone Test Levels

TEST	PRE-RELAX cf RELAX	RELAX cf POST-RELAX	PRE-RELAX cf POST-RELAX
cortisol	1.03(13)	2.31(13)	2.74*(13)
T_4	0.55(13)	-1.28(13)	-0.73(13)
FTI	0.69(13)	0.28(13)	0.70(13)
T_3	0.70(13)	-2.04(13)	-1.38(13)
rT_3	-1.98(13)	-3.59(13)*	-4.20(13)**
hGH	0.84(13)	0.75(13)	1.66(13)
prolactin	0.44(13)	1.32(13)	1.15(13)

(p values were corrected for 3 multiple comparisons)

Percentage changes in TM or ordinary relaxation for each serum hormone test are contained in table 28:-

FIGURE 14. Serum Cortisol Levels (Mean±SEM) Before, During and After TM or Ordinary Relaxation.

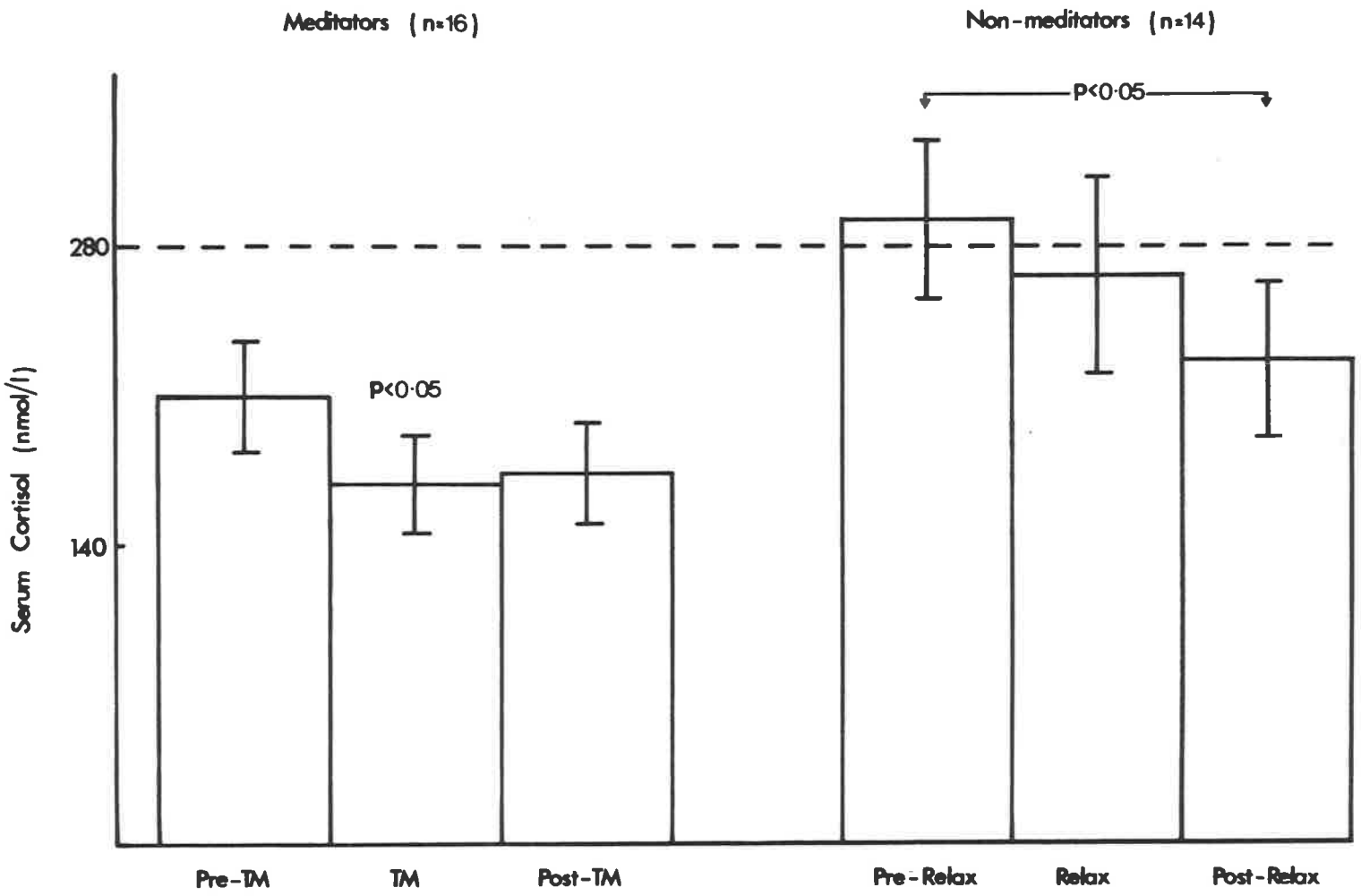


FIGURE 15. Serum hGH Levels (Mean±SEM) Before, During and After TM or Ordinary Relaxation

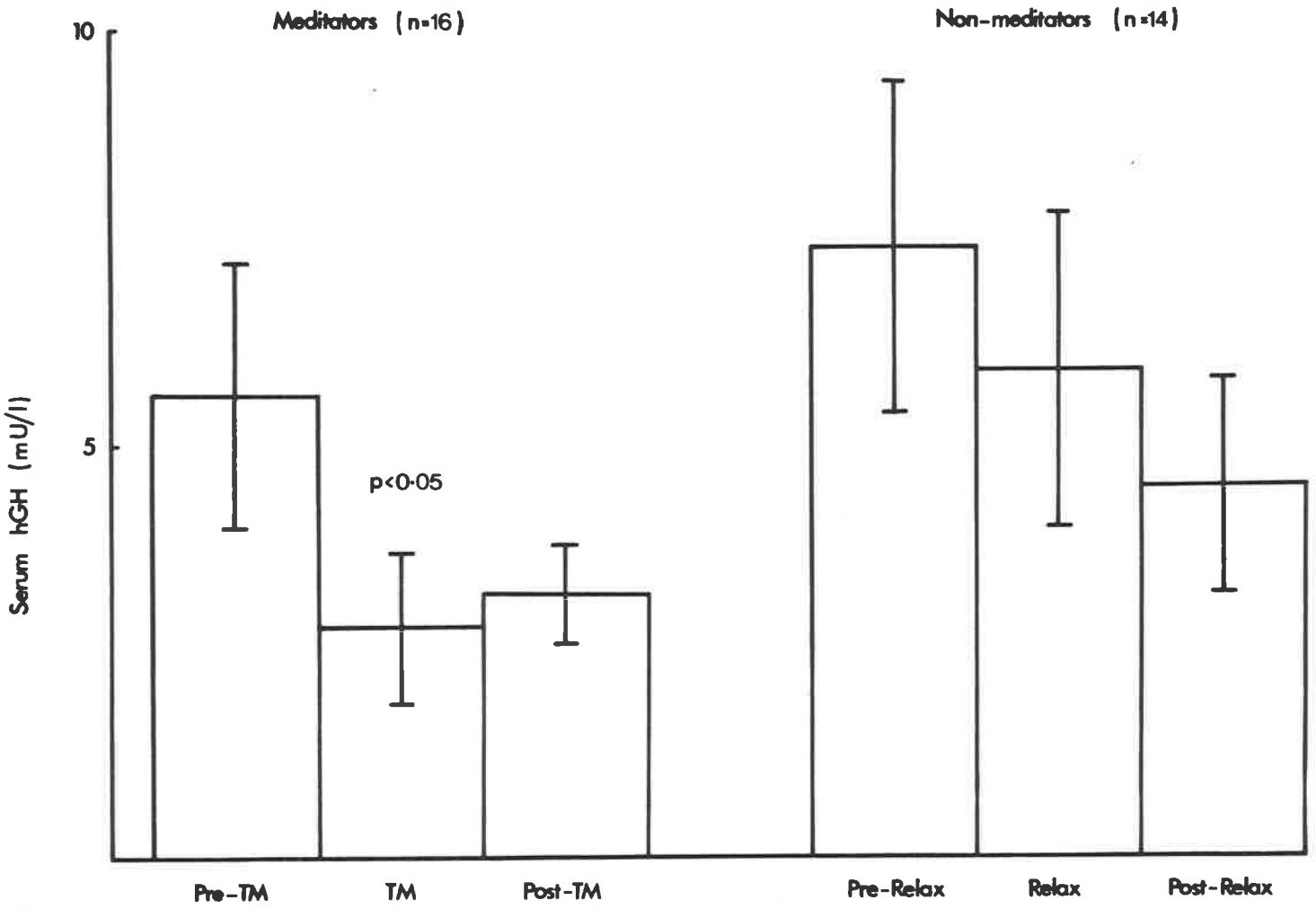


FIGURE 16. Serum Prolactin Levels (Mean±SEM) Before, During and After TM or Ordinary Relaxation

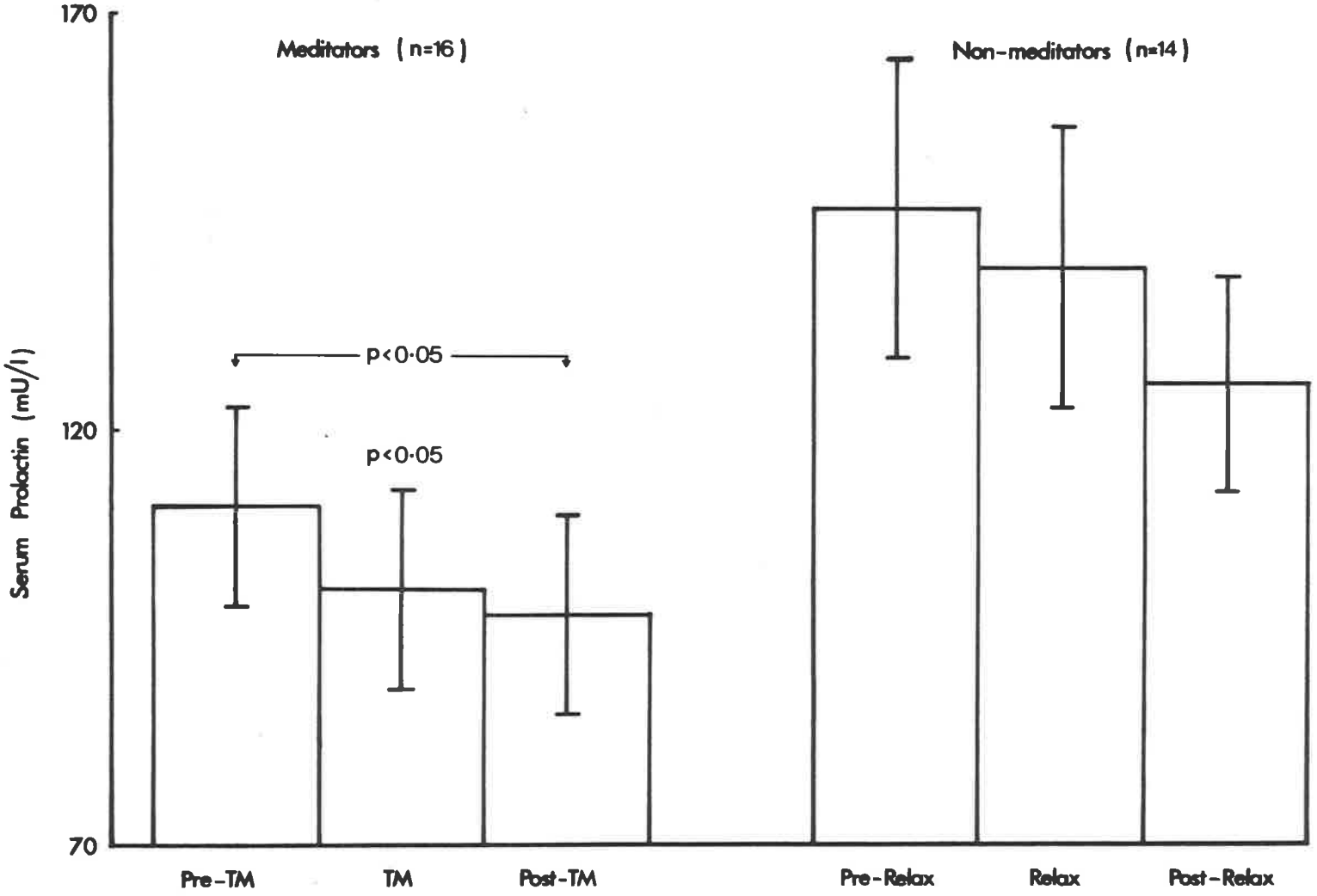


TABLE 28. Percentage Changes of Serum Hormone Tests in TM or Ordinary Relaxation

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-RELAX cf RELAX	RELAX cf POST-RELAX
cortisol	-20%	+3%	-9%	-15%
T_4	-3%	+3%	0%	+1%
FTI	-3%	+3%	-1%	-1%
T_3	-7%	0%	0%	+7%
rT_3	+4%	+7%	+9%	+16%
hGH	-50%	+14%	-20%	-24%
prolactin	-9%	-3%	-5%	-10%

Mean serum cortisol, hGH, and prolactin levels on all samples (1415 to 1615 hours, inclusive) are given in tables 29, 30 and 31, respectively. Levels of both meditators and non-meditators generally declined during the pre-treatment experimental period. However, as previously noted in tables 26 and 27, statistically significant hormone reductions during treatment were observed only in the TM group. Levels of all three hormones fell abruptly at the onset of meditation (1510 hours) and decreased levels were sustained into the post-treatment period (Tables 29 to 31, inclusive). The most marked hormone change in TM was observed for hGH which fell by 50 percent during meditation. hGH rose slightly following TM, and levels appeared to gradually return to pre-meditation values. The 20 percent reduction in serum cortisol during TM was sustained throughout the post-treatment period, and there was only a slight return of cortisol towards pre-meditation values. The continued reduction of serum cortisol throughout the experimental period for non-meditator controls produced a statistically significant decrease on a pre- to post- relaxation comparison (Table 27). The relatively small, 9 percent, fall in prolactin during TM continued into the post-meditation period when levels dropped by a further 3 percent.

TABLE 29. Mean ± SEM Serum Cortisol Levels (n mol/l) on All Samples

GROUP	SAMPLE TIME									
	1415	1430	1445	1500	1510	1520	1530	1545	1600	1615h
1	249±30	233±29	200±25	189±25	156±14	167±24	145±12	169±26	180±25	172±23
2	346±42	317±39	267±41	276±43	250±47	267±46	263±53	253±43	232±38	222±35

TABLE 30. Mean ± SEM Serum hGH Levels (m U/l) on All Samples

GROUP	SAMPLE TIME									
	1415	1430	1445	1500	1510	1520	1530	1545	1600	1615h
1	6.0±1.4	6.4±1.7	5.3±1.7	4.5±1.5	3.4±1.2	2.7±0.9	2.4±0.8	2.5±0.7	3.0±0.6	4.0±0.9
2	9.9±3.7	8.6±2.8	7.4±2.3	6.2±1.8	6.0±2.2	6.0±2.2	5.9±1.7	4.8±1.2	4.7±1.4	4.4±1.6

TABLE 31. Mean ± SEM Serum Prolactin Levels (m U/l) on All Samples

GROUP	SAMPLE TIME									
	1415	1430	1445	1500	1510	1520	1530	1545	1600	1615h
1	123±14	120±11	103±13	107±13	91±10	108±11	88±10	102±15	96±12	95±11
2	164±24	146±21	138±16	149±21	134±20	149±17	146±21	139±14	128±14	115±14

Although statistically significant reductions in serum T_4, FTI and serum T_3 also occurred during TM in contrast to no significant change during ordinary relaxation of non-meditator controls (Tables 26 and 27), percentage changes of thyroid hormones during meditation were small (Table 28). A 7 percent fall in T_3 during TM was sustained following meditation, while a slight reciprocal rise in rT_3 occurred during TM and was continued into the post-meditation period. In contrast, a statistically significant increase in rT_3, along with a slight T_3 rise, was found following ordinary relaxation of non-meditator controls (Tables 27 and 28).

(ii) Packed cell volume and total serum protein

PCV and total serum protein results from within-group comparisons between pre-, during and post- meditation or ordinary relaxation periods are presented in table 32. No significant changes in PCV were observed during meditation or ordinary relaxation. However, a slight but statistically significant 2 percent rise in total serum protein was observed following ordinary relaxation whereas no significant change was found in TM. Nevertheless, the 2 percent serum protein increase for non-meditator controls represented an absolute change of only one g/l.

TABLE 32. Statistics from Within-group Comparisons on PCV and Total Serum Protein

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
PCV	-0.57(15)	-1.15(15)	-1.64(15)
protein	0.19(14)	-1.52(14)	-0.91(15)
	PRE-RELAX cf RELAX	RELAX cf POST-RELAX	PRE-RELAX cf POST-RELAX
PCV	1.10(13)	-2.00(13)	-1.27(13)
protein	0.20(13)	-3.46(13)*	-4.22(13)***

(p values were corrected for 3 multiple comparisons)

(iii) Non-stress serum cortisol and serum hGH

Despite the preliminary meeting and early cannulation, the initial pre-relaxation serum cortisol levels of the non-meditator controls were above the upper limit of the basal reference range (see figure 14). Pre-meditation or relaxation serum cortisol levels were significantly higher in the controls than in the meditators ($t = 1.86$; $p < 0.05$; $df = 28$; Student's t-test). The pre-relaxation serum hGH levels of the controls also tended to extend above the upper limit of the basal reference range (13 m U/l[54]) (see figure 15). However, the pre-meditation serum hGH levels were not significantly different than the pre-rest levels of the non-meditator controls ($t = 0.76$, $p > 0.05$; $df = 28$; Student's t-test). Serum prolactin levels of both meditators and non-meditators were mostly within the normal reference range (70 to 300 m U/l).

In order to compare endocrine changes during meditation and ordinary relaxation without interference from temporal return of serum cortisol and hGH levels to non-stress values, statistical analysis was repeated on all non-stress levels (Tables 33 and 34).

TABLE 33. Non-stress Serum Cortisol and Non-stress Serum hGH Levels (Mean ± SEM) Before, During and After TM or Ordinary Relaxation

TEST	EXPERIMENTAL PERIOD	TREATMENT	
cortisol		TM (n=13)	Ordinary Relaxation (n=6)
(n mol/l)	Pre-	167 ± 13	164 ± 19
	During	137 ± 10	133 ± 11
	Post-	148 ± 14	130 ± 13
hGH		TM (n=13)	Ordinary Relaxation (n=8)
(m U/l)	Pre-	2.9 ± 0.7	2.7 ± 1.3
	During	1.6 ± 0.3	3.0 ± 1.2
	Post-	3.2 ± 0.6	2.4 ± 1.2

54. A 13 mU/l upper limit to the adult, basal resting range of serum hGH is quoted by CEA-IRE-SORIN for the hGH assay used here. However, slightly lower upper limits of 10 m U/l are given elsewhere (Catt, 1970; Martin, 1973)

TABLE 34. Statistics from Within-group Comparisons (Paired t-tests) on Non-stress Serum Cortisol and Non-stress Serum hGH Levels

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
cortisol	2.74(12)*	0.97(12)	0.97(12)
hGH	3.19(12)*	2.34(12)	0.28(12)
	PRE-RELAX cf RELAX	RELAX cf POST-RELAX	PRE-RELAX cf POST-RELAX
cortisol	2.26(5)	0.15(5)	1.22(5)
hGH	0.65(7)	1.41(7)	0.42(7)

(p values were corrected for 3 multiple comparisons)

As shown in figure 17, non-stress serum cortisol levels fell during both TM and ordinary relaxation of non-meditators, however, as before (p 62) the change was significant only during TM (Table 34). Serum cortisol levels rose slightly during the post-meditation period.

Non-stress serum hGH changes are presented histographically in figure 18. hGH fell significantly during TM (-45 percent) in contrast to no significant change during ordinary relaxation (+ 11 percent) of non-meditators (Table 34). Although not statistically significant, a distinctive return of hGH levels to pre-meditation values was observed following meditation.

(d) Urinary tests

(i) Temporal changes

Group mean UFC and UCA levels before and during the laboratory study are shown in table 35:-

TABLE 35. Mean ± SEM UFC and UCA Levels (p mol/min) Before and During the Laboratory Study

GROUP	UFC		UCA	
	Pre-Laboratory	Laboratory	Pre-Laboratory	Laboratory
1:- Meditators	212 ± 26	174 ± 22	288 ± 37	320 ± 42
2:- Non-meditators	219 ± 20	237 ± 32	269 ± 32	305 ± 58

FIGURE 17.
Non-stress Serum Cortisol Levels (Mean ± SEM) Before, During and After TM or Ordinary Relaxation

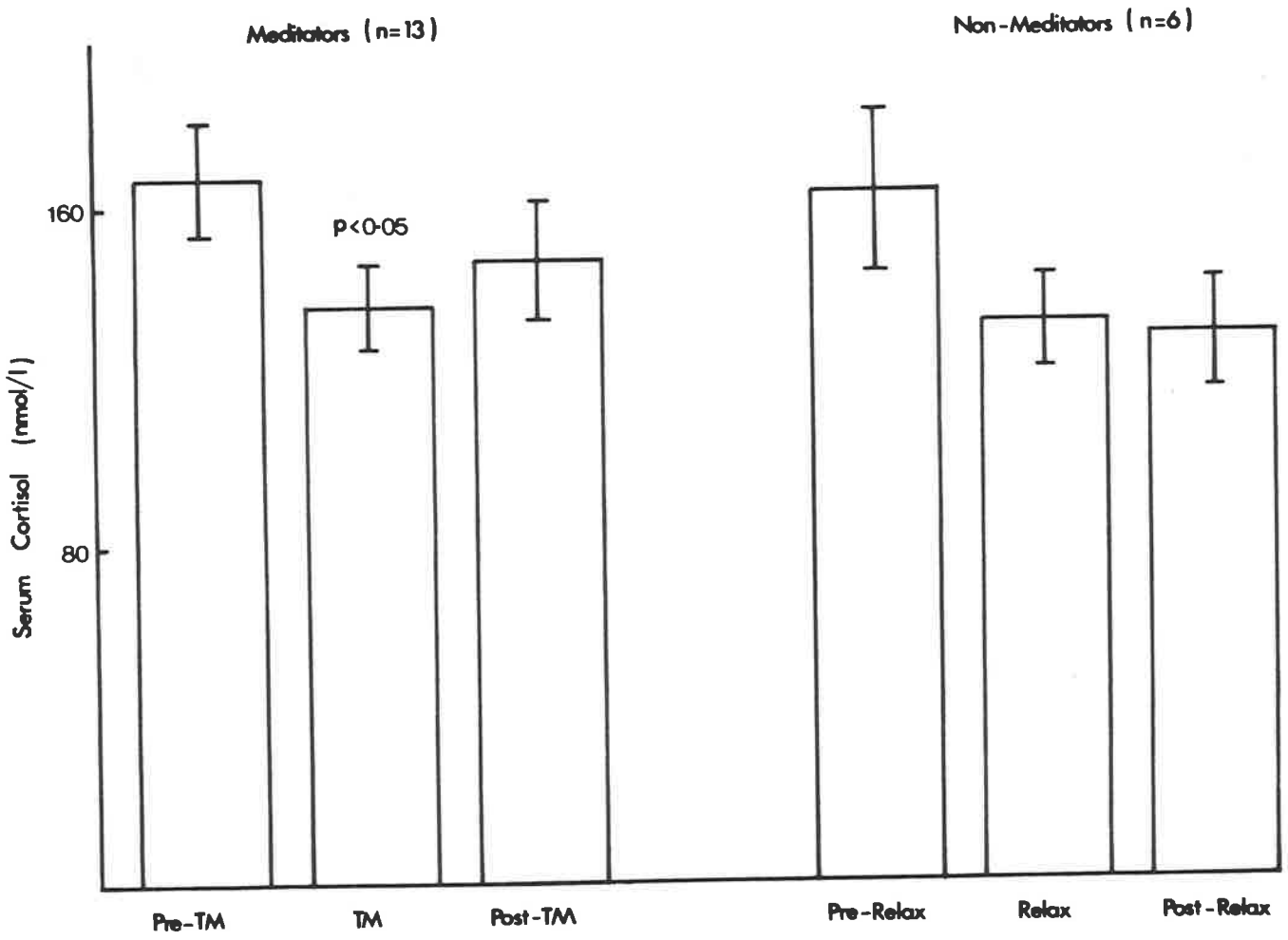
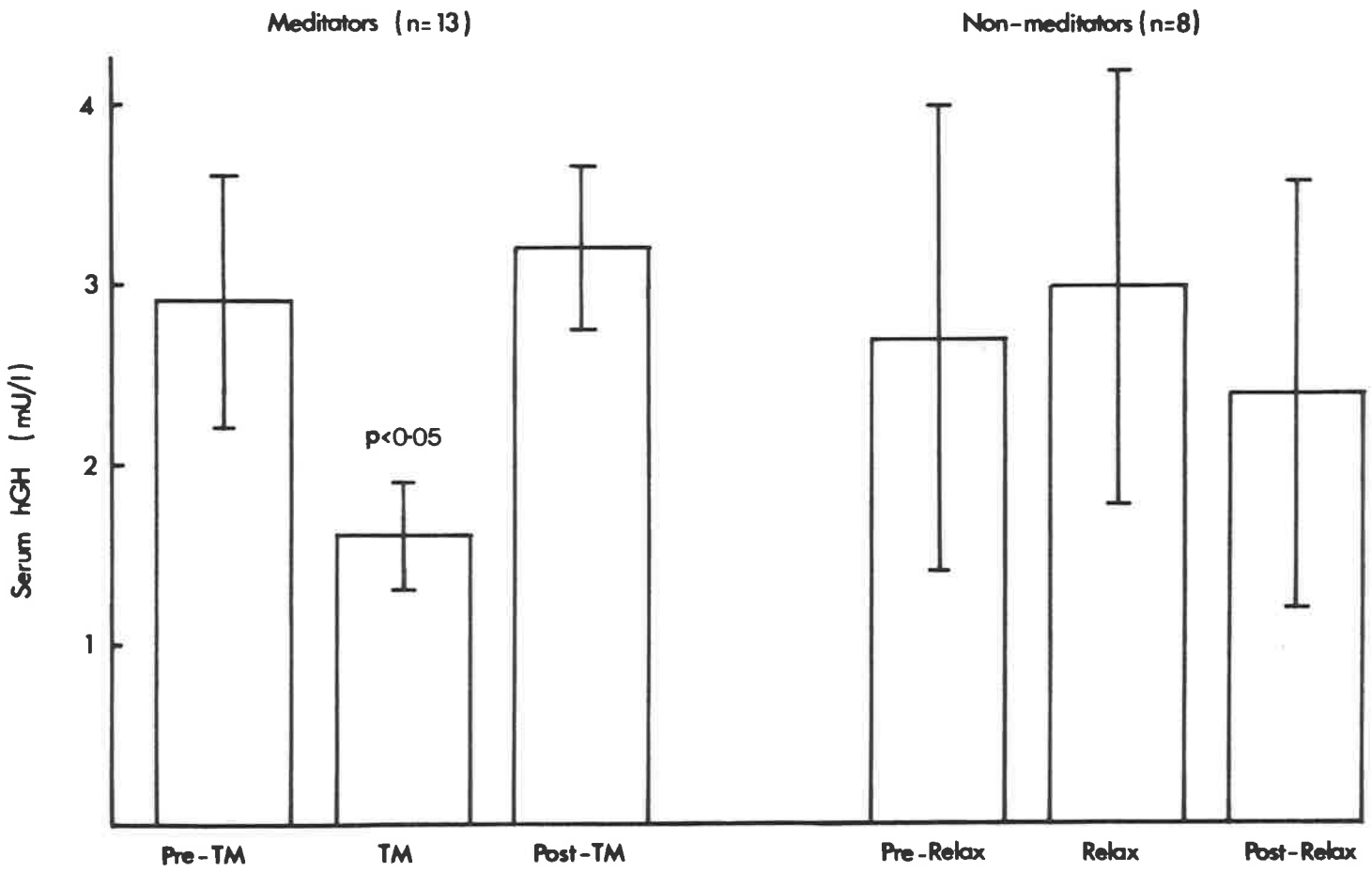


FIGURE 18.

Non-stress Serum hGH Levels (Mean±SEM) Before, During and After TM or Ordinary Relaxation



Results of within-group paired t-test comparisons between the pre-laboratory and laboratory levels of UFC, UCA, urine flow, creatinine clearance and derived serum osmolality are given in table 36. A significant decrease in UFC excretion occurred during laboratory investigation of TM in contrast to no significant change in UFC excretion of non-meditators studied under ordinary relaxation conditions. No significant changes in UCA, urine flow, creatinine clearance or osmolality were found in either group 1 or group 2. No significant inter-group differences in pre-laboratory UFC or UCA levels were observed ($t = 0.20$ and $t = 0.38$, respectively; $df = 28$; Student's t-tests). The UFC findings are summarised in figure 19.

TABLE 36. Statistics from Within-group Comparisons on UFC, UCA, Urine Flow, Creatinine Clearance and Serum Osmolality

GROUP	UFC	UCA	Urine Flow	Creatinine Clearance	Serum Osmolality
1:- Meditators	2.28(15)*	-1.08(15)	-1.29(15)	-1.00(15)	0.85(15)
2:- Non-meditators	-0.75(13)	-0.60(13)	-0.49(13)	-1.30(12)	1.01(12)

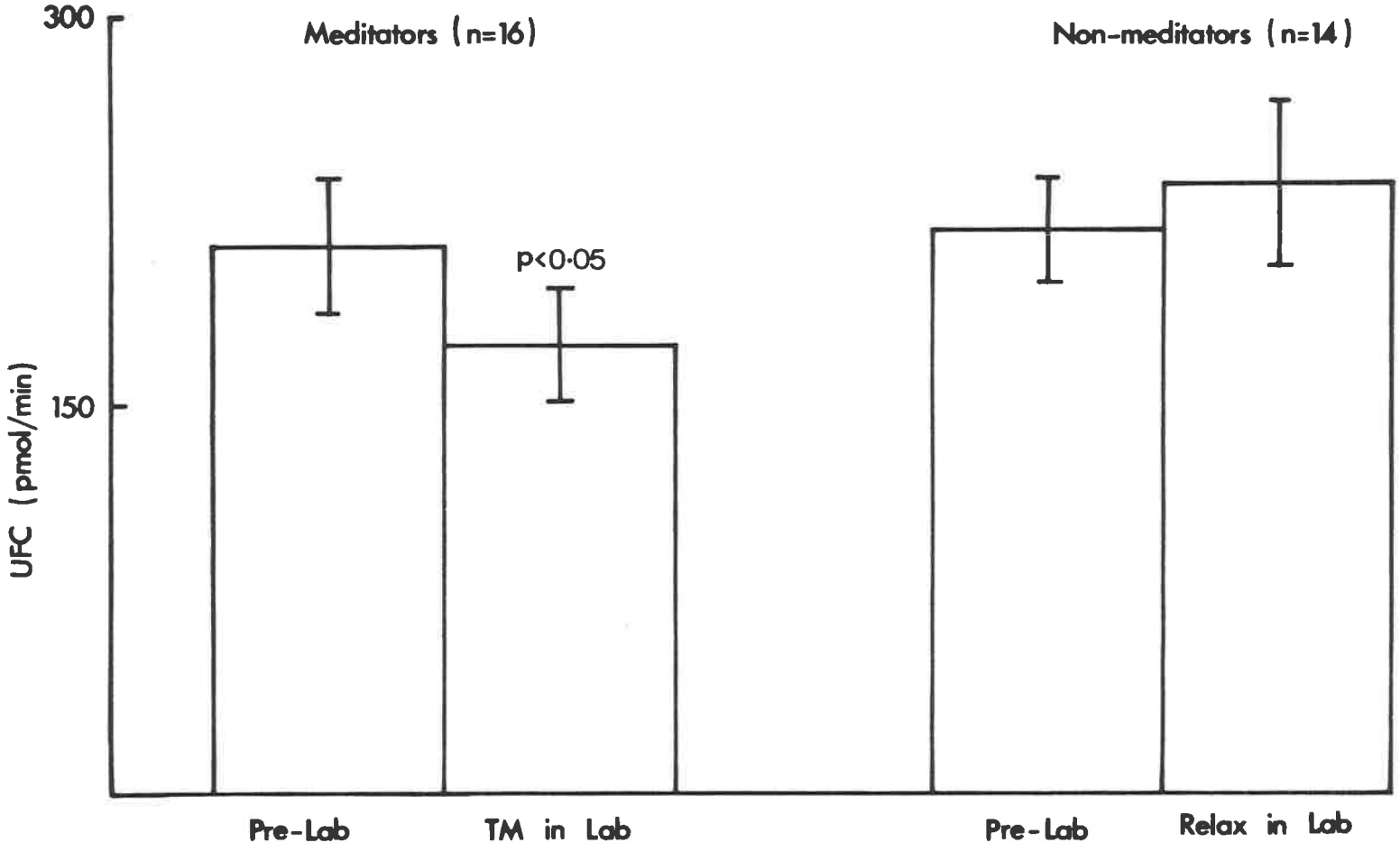
Mean UFC and UCA levels on the laboratory Saturday and control Saturday for both the meditation and non-meditation groups are given in table 37:-

TABLE 37. Mean ± SEM UFC and UCA Levels (n mol/d) on the Laboratory Saturday and Control Saturday

	LABORATORY SATURDAY		CONTROL SATURDAY	
GROUP	UFC	UCA	UFC	UCA
1:- Meditators	201 ± 16	443 ± 44	168 ± 14	413 ± 56
2:- Non-meditators	259 ± 27	324 ± 29	208 ± 28	386 ± 38

FIGURE 19.

Urinary Free Cortisol Levels (Mean±SEM) Before and During Laboratory Study of TM or Ordinary Relaxation



The UFC results are presented in figure 20. UFC levels of the meditators were significantly higher on the Saturday of the laboratory study than on the Saturday of the control week-end ($t = 2.38$; $df = 12$; $p < 0.05$; paired t-test). In contrast, no significant difference was found between the laboratory Saturday and control Saturday UFC levels of the non-meditator controls ($t = 1.27$; $df = 12$; $p > 0.05$; paired t-test), although non-meditator UFC levels on the laboratory Saturday extended above the upper limit of the basal resting range. Surprisingly, the UCA levels of the non-meditators were significantly higher on the control Saturday than on the laboratory Saturday ($t = -2.35$; $df = 11$; $p < 0.05$; paired t-test). No significant difference was observed between the laboratory Saturday and the control Saturday UCA excretion of the meditators ($t = 0.36$; $df = 12$; $p > 0.05$; paired t-test).

(ii) Overall inter-group comparisons

For the purpose of comparing 24 hour UFC and UCA excretion of meditators and non-meditator controls, an overall analysis was performed on all first experience meditators (i.e. groups 1 and 3) and all non-meditator controls. Mean overall UFC and UCA levels from the week-end of the laboratory study are shown in table 38:-

TABLE 38. Overall Mean ± SEM UFC and UCA Levels (n mol/d) of Short-term Study (2)

SUBJECTS	NUMBER	UFC	UCA
Meditators	21	183 ± 9	438 ± 30
Non-meditators	15	225 ± 15	334 ± 22

The results are represented histographically in figure 21. UFC levels of the meditators were significantly lower than the non-meditator controls ($t = 2.59$; $df = 70$; $p < 0.01$, Student's t-test),

FIGURE 20.
Urinary Free Cortisol Levels (Mean±SEM) of Laboratory Saturday and Control Saturday

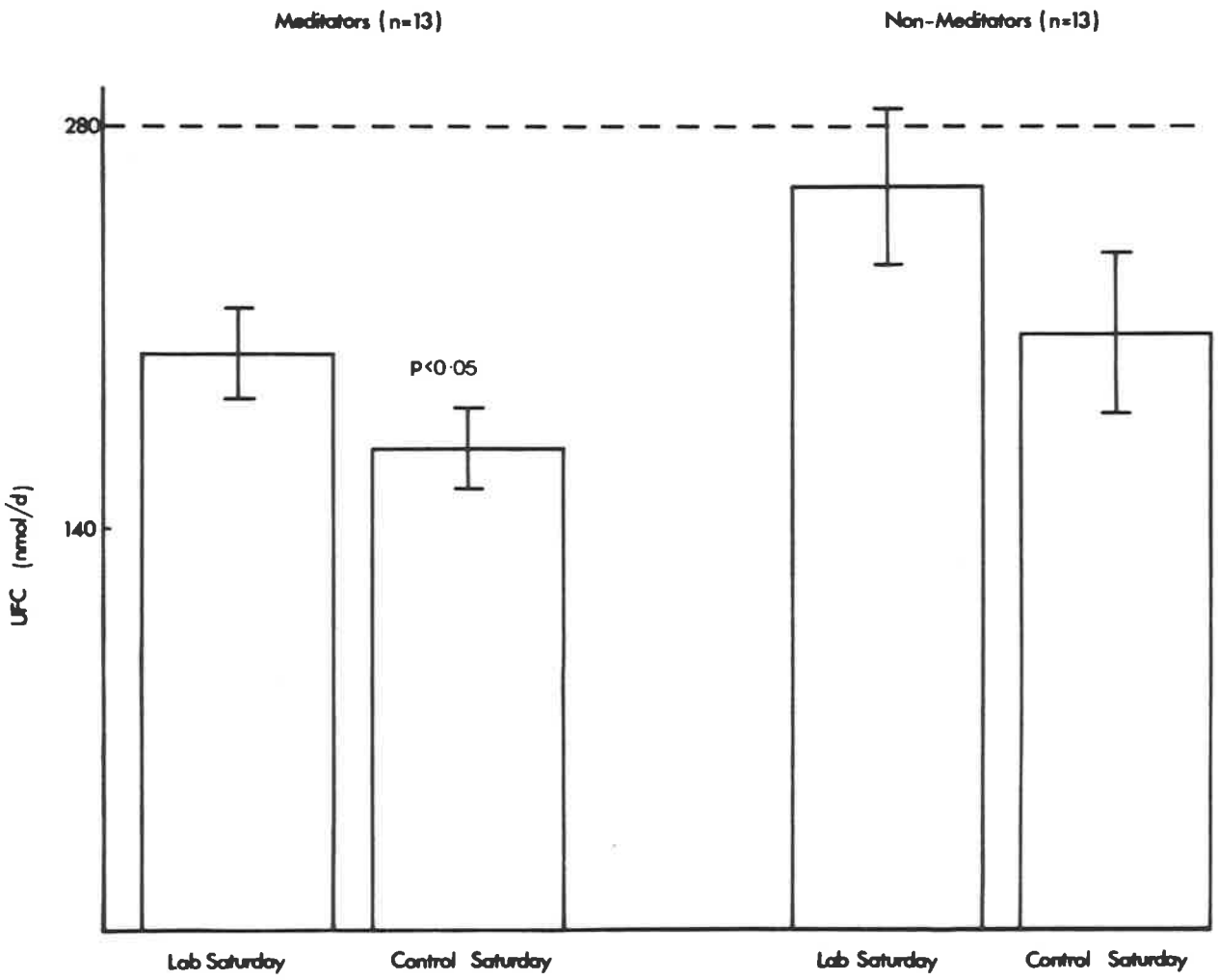
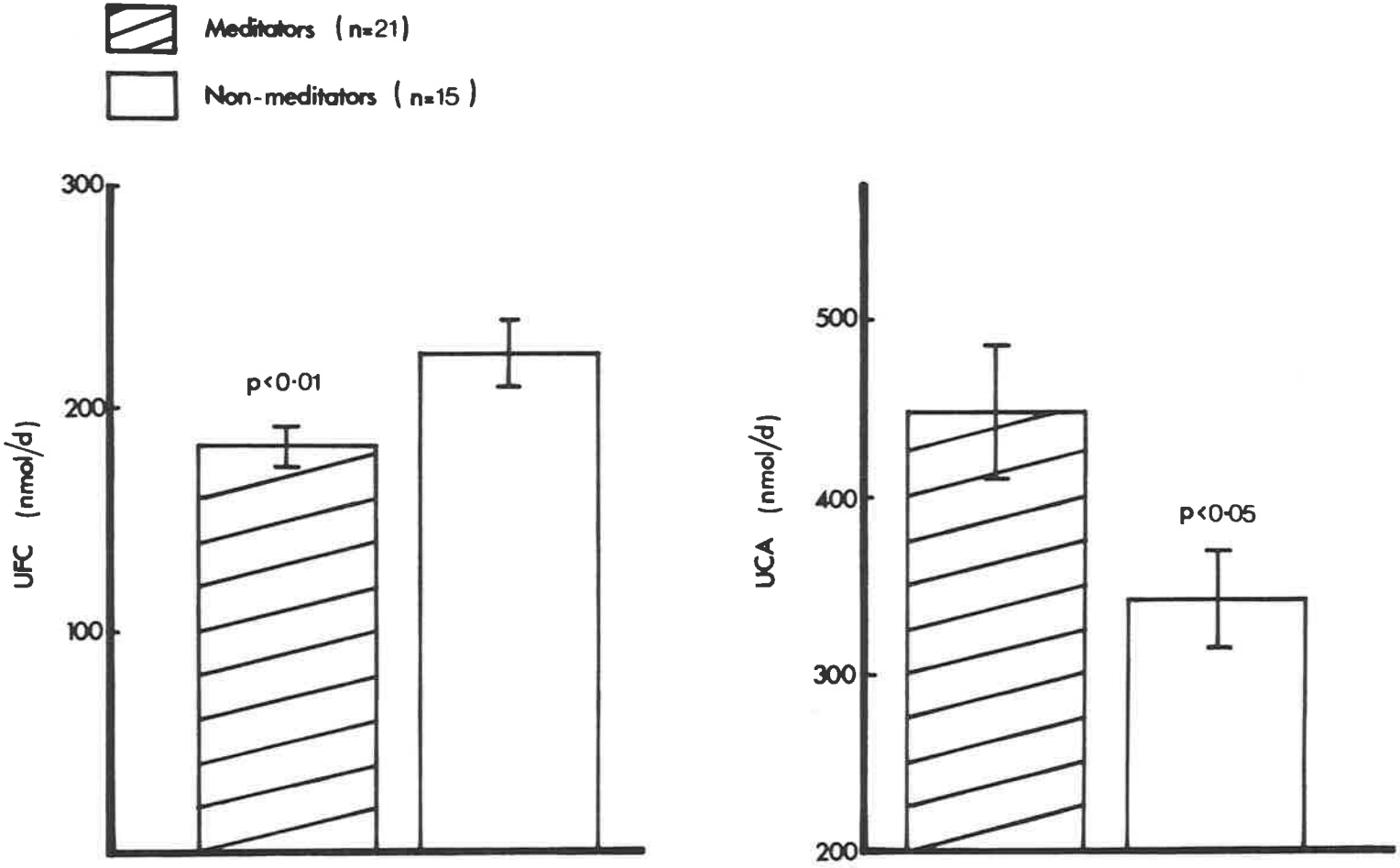


FIGURE 21.
Overall Urinary Hormone Levels (Mean±SEM) on Week-end of Laboratory Study



whereas the UCA levels of the meditation group were significantly higher than the UCA levels of the non-meditators ($t = -2.58$; $p < 0.05$; $df = 70$; Sutdent's t-test).

(e) Summary of cortisol findings

The most outstanding differences between the cortisol levels of meditators and non-meditator controls were:- 1) the lack of significant difference between the pre-laboratory UFC levels; 2) the higher pre-treatment serum cortisol levels of the non-meditator controls; 3) the significant reduction of both serum cortisol and UFC during TM, in contrast to no significant change during ordinary relaxation, for both stress and non-stress cortisol levels; 4) the significantly lower 24 hour UFC excretion of meditators than of non-meditator controls; and 5) the significantly higher UFC excretion on the laboratory Saturday than on the control Saturday in the meditation group only.

C. Statistical Analysis Using Meditators as Controls

(a) Blood tests

(i) Hormones

Mean pre-, during and post- meditation (group 1A) or quiet rest (groups 1B and 3) levels of each hormone test are shown in table 39:-

TABLE 39. Serum Hormone Test Levels (Mean ± SEM) Before, During and After TM or Quiet Rest

TEST	EXPERIMENTAL PERIOD	GROUP 1A (TM; 1st experience)	GROUP 1B (Quiet rest; 2nd experience)	GROUP 3 (Quiet rest; 1st experience)
serum cortisol (n mol/l)	pre-	152 ± 24	154 ± 16	212 ± 38
	during	132 ± 14	141 ± 15	178 ± 34
	post-	130 ± 21	134 ± 28	142 ± 22
serum T_4 (n mol/l)	pre-	117 ± 7	122 ± 7	115 ± 12
	during	114 ± 7	125 ± 6	113 ± 14
	post-	117 ± 6	122 ± 5	115 ± 13
FTI (Units)	pre-	111 ± 6	123 ± 7	118 ± 13
	during	111 ± 6	126 ± 6	117 ± 14
	post-	113 ± 4	124 ± 5	116 ± 13
serum T_3 (n mol/l)	pre-	1.5 ± 0.1	1.4 ± 0.2	1.2 ± 0.1
	during	1.5 ± 0.2	1.5 ± 0.2	1.2 ± 0.2
	post-	1.4 ± 0.1	1.5 ± 0.2	1.2 ± 0.2
serum rT_3 (n mol/l)	pre-	0.25 ± 0.03	0.31 ± 0.02	0.31 ± 0.03
	during	0.28 ± 0.04	0.33 ± 0.02	0.33 ± 0.04
	post-	0.29 ± 0.02	0.35 ± 0.03	0.35 ± 0.03
serum hGH[55] (m U/l)	pre-	1.6 ± 0.2	4.6 ± 1.8	6.8 ± 3.2
	during	0.9 ± 0.1	3.9 ± 1.3	4.0 ± 2.1
	post-	2.4 ± 0.6	2.3 ± 0.9	3.6 ± 1.0
serum prolactin (m U/l)	pre-	89 ± 15	99 ± 30	136 ± 38
	during	76 ± 13	84 ± 19	117 ± 25
	post-	70 ± 12	82 ± 16	115 ± 18

Results from within-group paired t-test comparisons between pre-, during and post-meditation or quiet rest periods are given in tables 40, 41 and 42 for groups 1A, 1B and 3 respectively. Significant hormone changes occurred only for hGH and prolactin which fell during meditation (Table 40). For serum hGH there was a significant 44% reduction during TM (group 1A). Levels subsequently rebounded to 50% above pre-meditation values. No significant changes in hGH were observed under quiet rest conditions for either group 1B or group 3 (Tables 41 and 42). The results are summarised in figures 22 and 23.

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55. Subject 17(40) was excluded from serum hGH analysis because of a marked hGH stress response due to re-cannulation during experiment 1B.

TABLE 40. Statistics from Within-group 1A Comparisons on Serum Hormone Test Levels

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
cortisol	1.00(4)	0.18(4)	0.65(4)
T_4	0.85(4)	-1.16(4)	0.10(4)
FTI	0.10(4)	-0.27(4)	-0.31(4)
T_3	0.55(4)	0.47(4)	0.98(4)
rT_3	-1.24(4)	-0.26(4)	-1.85(4)
hGH	6.48(3)*	2.24(3)	1.19(3)
Prolactin	9.00(4)**	1.25(4)	4.03(4)*

(p values were corrected for 3 multiple comparisons)

TABLE 41. Statistics from Within-group 1B Comparisons on Serum Hormone Test Levels

TEST	PRE-REST cf REST	REST cf POST-REST	PRE-REST cf POST-REST
cortisol	1.00(4)	0.47(4)	1.20(4)
T_4	-0.91(4)	1.27(4)	-0.02(4)
FTI	-0.90(4)	1.15(4)	-0.05(4)
T_3	-1.16(4)	0.91(4)	-1.39(4)
rT_3	-1.42(4)	-1.30(4)	-1.57(4)
hGH	0.34(3)	2.47(3)	1.06(3)
prolactin	1.19(4)	0.15(4)	0.76(4)

(p values were corrected for 3 multiple comparisons)

FIGURE 22. Serum hGH Levels (Mean ± SEM) Before, During and After TM or Quiet Rest

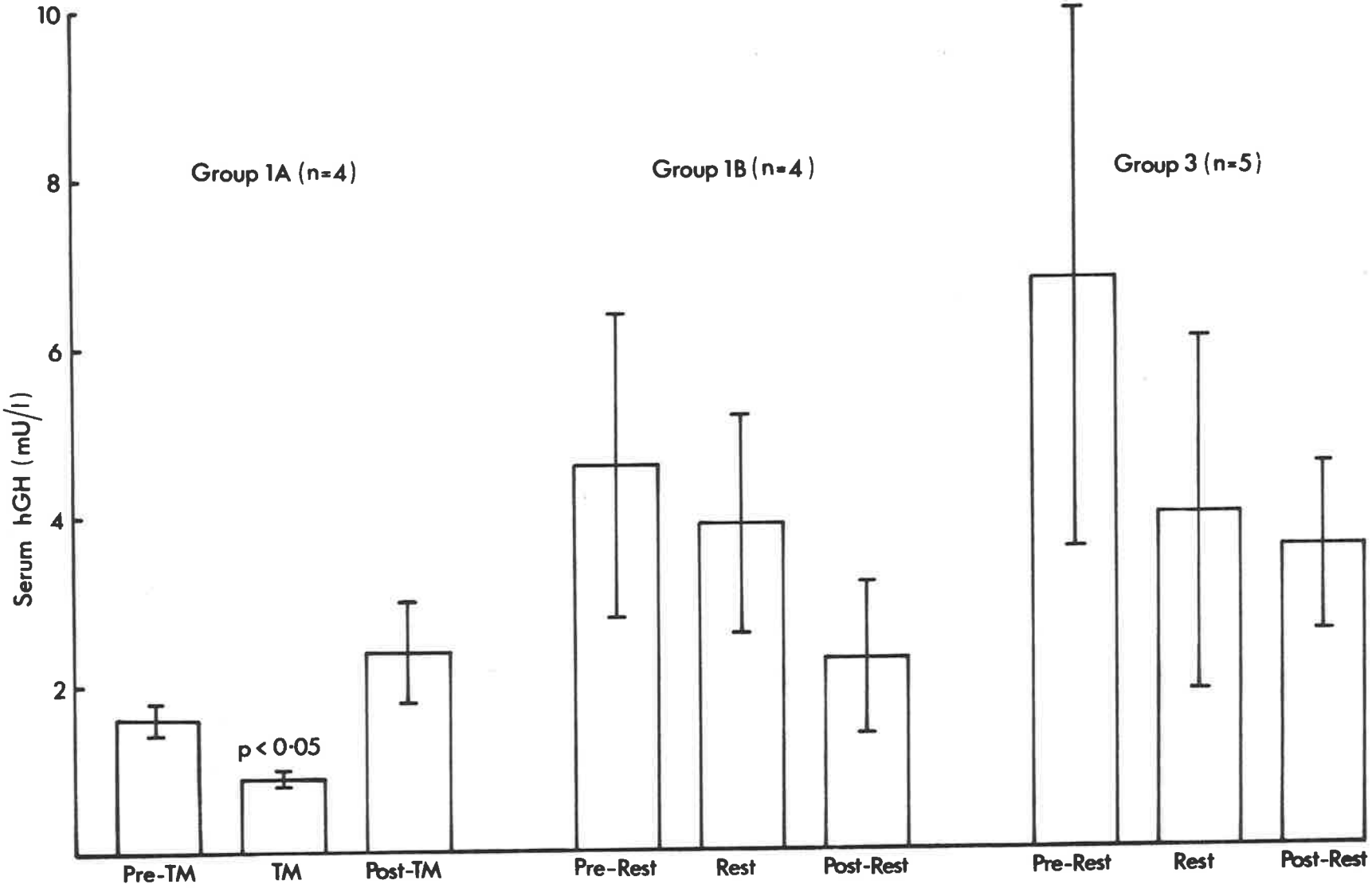


FIGURE 23.

Serum Prolactin Levels (Mean ± SEM) Before, During and After TM or Quiet Rest

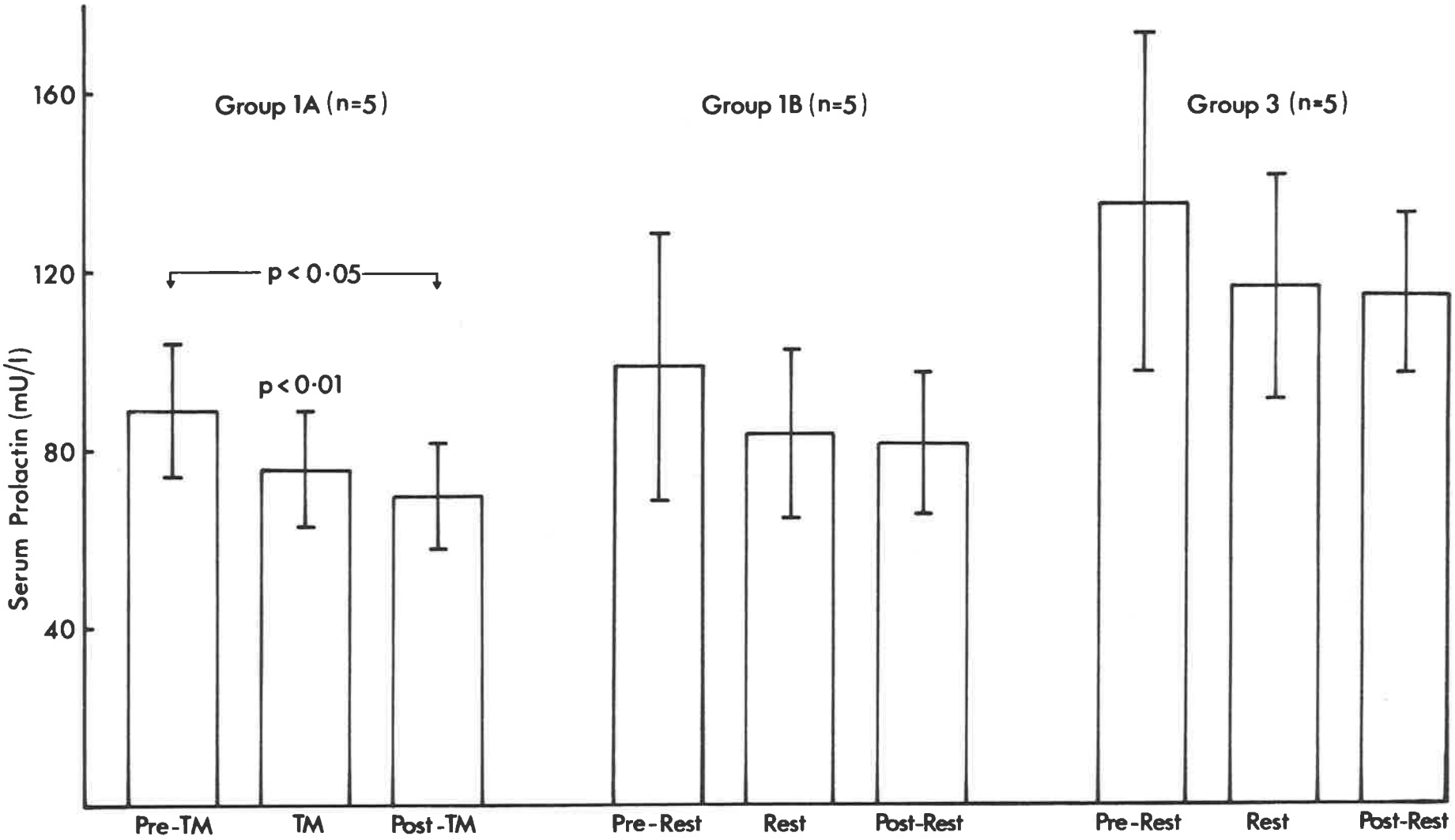


TABLE 42. Statistics from Within-group 3 Comparisons on Serum Hormone Test Levels

TEST	PRE-REST cf REST	REST cf POST-REST	PRE-REST cf POST-REST
cortisol	1.51(4)	2.54(4)	2.36(4)
T_4	0.77(4)	-0.59(4)	0.11(4)
FTI	0.37(4)	0.14(4)	0.46(4)
T_3	0.51(4)	0.35(4)	0.43(4)
rT_3	-1.81(4)	-0.43(4)	-1.45(4)
hGH	1.17(4)	0.27(4)	0.94(4)
prolactin	0.86(4)	0.25(4)	0.98(4)

(p values were corrected for 3 multiple comparisons)

Mean serum hGH and prolactin concentrations for each of the 10 sample times of groups 1A, 1B and 3 are shown in tables 43 and 44, respectively.

Figure 24 presents a detailed histographic representation of serum hGH changes in meditation. Group 1A levels are represented by blank bars while group 1B levels are represented by hatched bars. In group 1A, hGH fell before the onset of meditation and appeared to be a response in anticipation of meditation, which was sustained throughout the meditation period. Levels gradually rose again following meditation. In contrast, no significant changes in serum hGH occurred in the same subjects when re-studied during quiet rest (group 1B).

Serum prolactin levels also fell significantly during TM, however, no distinctive prolactin reduction was observed before the onset of meditation (Table 44). As previously observed for group 1, prolactin levels continued to fall following meditation. In contrast, the reductions in serum prolactin under quiet rest conditions, for both group 1B and group 3 were not statistically significant (Tables 41 and 42).

TABLE 43. Mean ± SEM Serum hGH Levels (m U/l) on All Samples

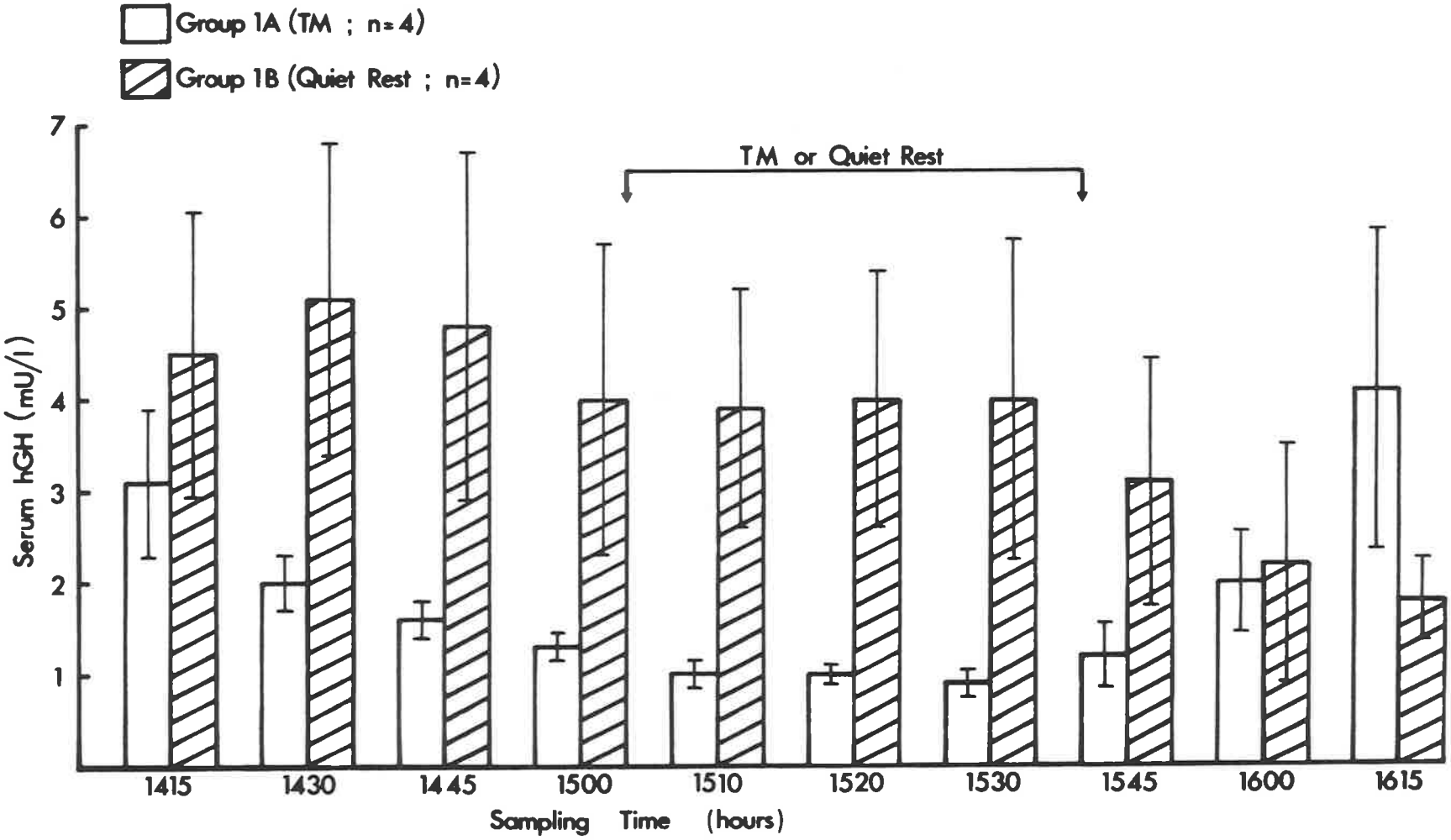
GROUP	SAMPLE TIME									
	1415	1430	1445	1500	1510	1520	1530	1545	1600	1615h
1A	3.1±0.8	2.0±0.3	1.6±0.2	1.3±0.2	1.0±0.1	1.0±0.1	0.9±0.1	1.2±0.3	2.0±0.5	4.1±1.7
1B	4.5±1.5	5.1±1.7	4.8±1.9	4.0±1.7	3.9±1.3	4.0±1.4	4.0±1.7	3.1±1.4	2.2±0.8	1.8±0.4
3	11.8±6.8	7.8±4.7	6.6±3.2	6.0±3.4	4.7±2.6	4.1±2.3	3.3±1.4	3.1±0.8	3.6±1.3	3.8±1.3

TABLE 44. Mean ± SEM Serum Prolactin Levels (m U/l) on All Samples

GROUP	SAMPLE TIME									
	1415	1430	1445	1500	1510	1520	1530	1545	1600	1615h
1A	101±18	110±11	82±14	77±20	76±23	88± 8	62±12	68±15	66±16	74± 9
1B	100±32	103±33	107±34	86±23	95±19	78±20	78±18	80±19	84±16	82±13
3	177±67	154±45	127±38	126±33	112±32	117±26	124±18	115±17	125±23	104±15

FIGURE 24.

Serum hGH Levels (Mean ± SEM) Before, During and After TM or Quiet Rest



As shown in figure 25, serum cortisol fell during both TM (group 1A) and quiet rest in meditators (groups 1B and 3), however, the changes were not statistically significant. No significant changes in serum T_4, FTI, serum T_3 or serum rT_3 were observed during meditation or quiet rest (Tables 40, 41 and 42).

Pre-treatment serum hGH, prolactin and cortisol levels of group 3 were higher than the pre-treatment values of both group 1A and group 1B, however, the differences were not statistically significant ($t = 0.54$, $df = 7$; $t = 0.76$, $df = 8$; $t = 1.40$, $df = 8$; respectively; Student's t-tests between pre-quiet rest levels of groups 1B and 3).

Results of inter-group paired t-test comparisons between each of the three experimental periods of groups 1A and 1B are given in table 45. Pre-meditation serum hGH levels were significantly lower than pre-rest levels of the same subjects. Similarly, serum hGH levels during meditation were significantly lower than levels during quiet rest. No significant differences in serum cortisol and serum prolactin levels of groups 1B and 3 were observed. Direct comparisons between levels during TM and quiet rest (group 1B) are shown in figure 26.

TABLE 45. Statistics from Between-group Comparisons on Serum Cortisol Serum hGH and Serum Prolactin Levels

TEST	PRE-TM (1A) cf PRE-REST (1B)	TM (1A) cf QUIET REST (1B)	POST-TM (1A) cf POST-REST (1B)
cortisol	0.16(14)	0.79(14)	0.31(14)
hGH	3.33(11)*	3.65(11)**	-0.70(11)
prolactin	0.60(14)	0.72(14)	1.47(14)

(p values were corrected for 3 multiple comparisons)

FIGURE 25.

Serum Cortisol Levels (Mean ± SEM) Before, During and After TM or Quiet Rest

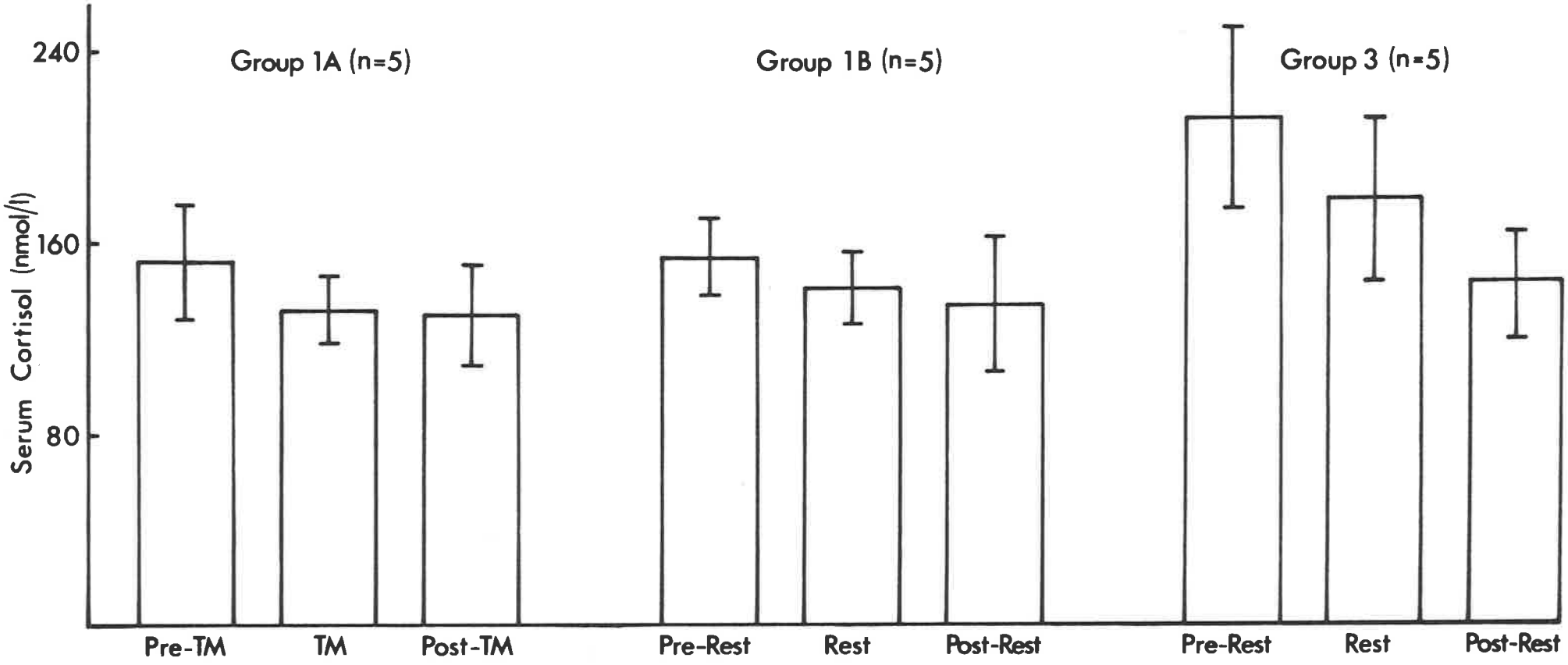
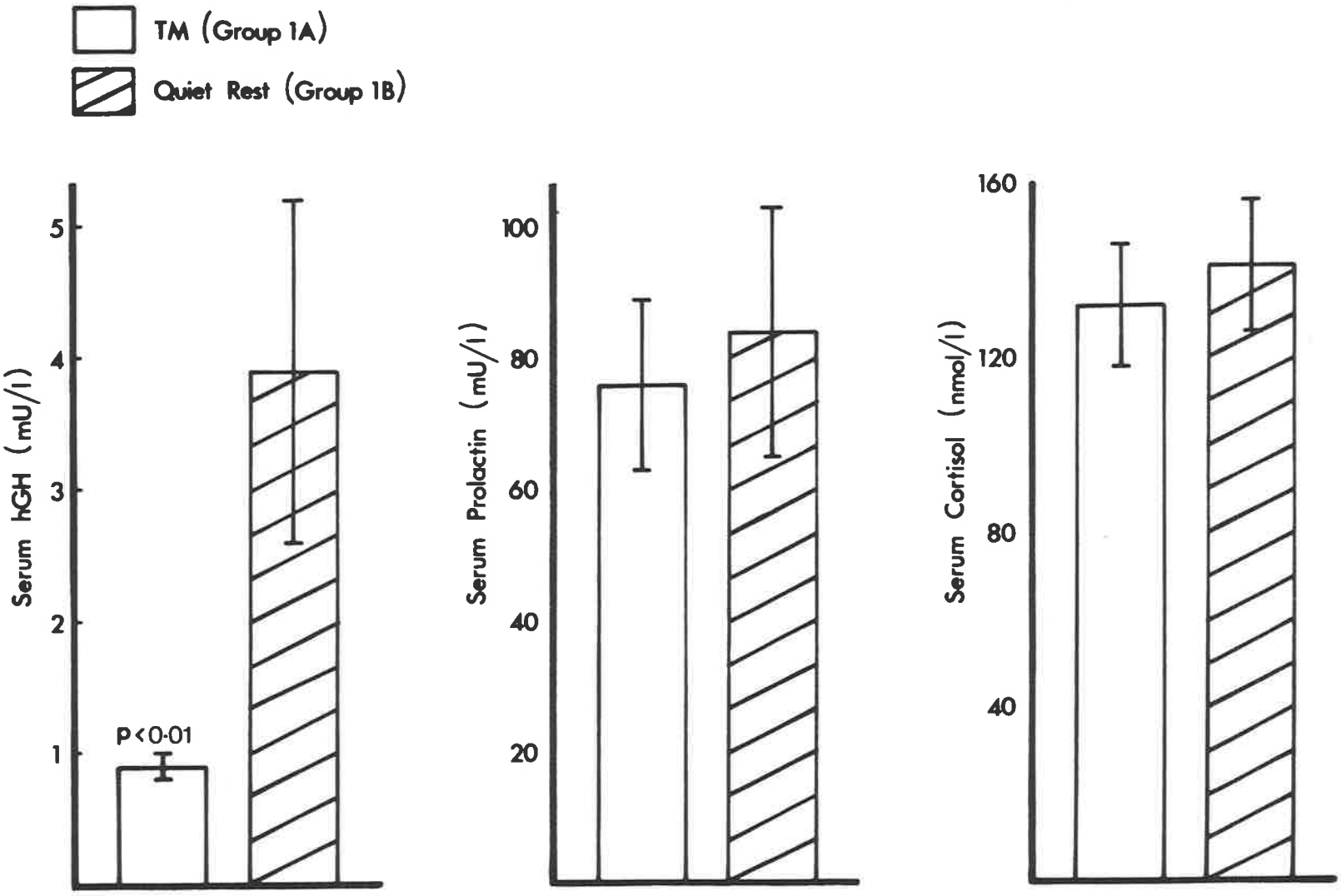


FIGURE 26. Serum Hormone Levels (Mean±SEM) During TM or Quiet Rest



(ii) Packed cell volume and total serum protein

PCV and total serum protein results from within-group paired t-test comparisons between pre-, during and post- meditation or quiet rest periods are given in tables 46, 47 and 48 for groups 1A, 1B and 3, respectively. No significant changes in PCV or total serum protein were observed during TM or quiet rest of either group 1B or group 3.

TABLE 46. Statistics from Within-group 1A Comparisons on PCV and Total Serum Protein Levels

TEST	PRE-TM cf TM	TM cf POST-TM	PRE-TM cf POST-TM
PCV	1.34(4)	0.18(4)	1.22(4)
Serum protein	0.78(4)	-3.16(4)	-1.18(4)

(p values were corrected for 3 multiple comparisons)

TABLE 47. Statistics from Within-group 1B Comparisons on PCV and Total Serum Protein Levels

TEST	PRE-REST cf REST	REST cf POST-REST	PRE-REST cf POST-REST
PCV	0.49(4)	0.40(4)	0.07(4)
Serum protein	0.00(4)	0.59(4)	0.78(4)

TABLE 48. Statistics from Within-group 3 Comparisons on PCV and Total Serum Protein Levels

TEST	PRE-REST cf REST	REST cf POST-REST	PRE-REST cf POST-REST
PCV	1.00(4)	1.80(4)	1.97(4)
Serum protein	-2.14(4)	1.83(4)	0.30(4)

(p values were corrected for 3 multiple comparisons)

(b) Urine tests

Group mean UFC and UCA levels before and during the laboratory study are shown in table 49:-

TABLE 49. Mean ± SEM UFC and UCA Levels (p mol/min) Before and During the Laboratory Study

GROUP	UFC		UCA	
	Pre-laboratory	Laboratory	Pre-laboratory	Laboratory
1A:- TM; 1st experience	239 ± 72	194 ± 70	305 ± 72	284 ± 69
1B:- Quiet Rest; 2nd experience	122 ± 33	138 ± 22	227 ± 17	317 ± 44
3:- Quiet Rest; 1st experience	157 ± 21	177 ± 50	343 ± 76	305 ± 79

Results of within-group paired t-test comparisons between the pre-laboratory and laboratory levels of all tests are given in table 50. No significant changes in UFC or UCA were observed during the laboratory study of TM or quiet rest. However, as shown in figure 27, pre-laboratory UFC levels of group 1A were significantly higher than pre-laboratory UFC levels of group 1B ($t = 2.91$; $df = 4$; $p < 0.05$; paired t-test). Pre-laboratory UCA levels were similarly higher for group 1A than group 1B, however, the difference was not statistically significant ($t = 0.96$; $df = 4$; $p > 0.05$; paired t-test). Pre-laboratory urine flow was also significantly higher on the first occasion (group 1A) than on the second occasion (group 1B) ($t = 2.69$; $df = 4$; $p < 0.05$; paired t-test). Pre-laboratory creatinine clearance levels were also higher for group 1A than group 1B, however, the difference was not statistically significant ($t = 2.13$; $df = 4$; $p > 0.05$; paired t-test). No significant difference was found between pre-laboratory UFC or UCA levels of groups 1B and 3 ($t = 0.92$; $df = 8$; $t = 1.49$; $df = 8$, respectively; Student's t-tests).

Serum osmolality fell significantly during laboratory quiet rest conditions for both groups 1B and 3. A significant reduction in urine flow was also observed in group 1B. No significant changes in any other tests were found for groups 1A, 1B or 3 (Table 50).

FIGURE 27 Urinary Hormone Levels (Mean±SEM) Before Laboratory Study of TM or Quiet Rest

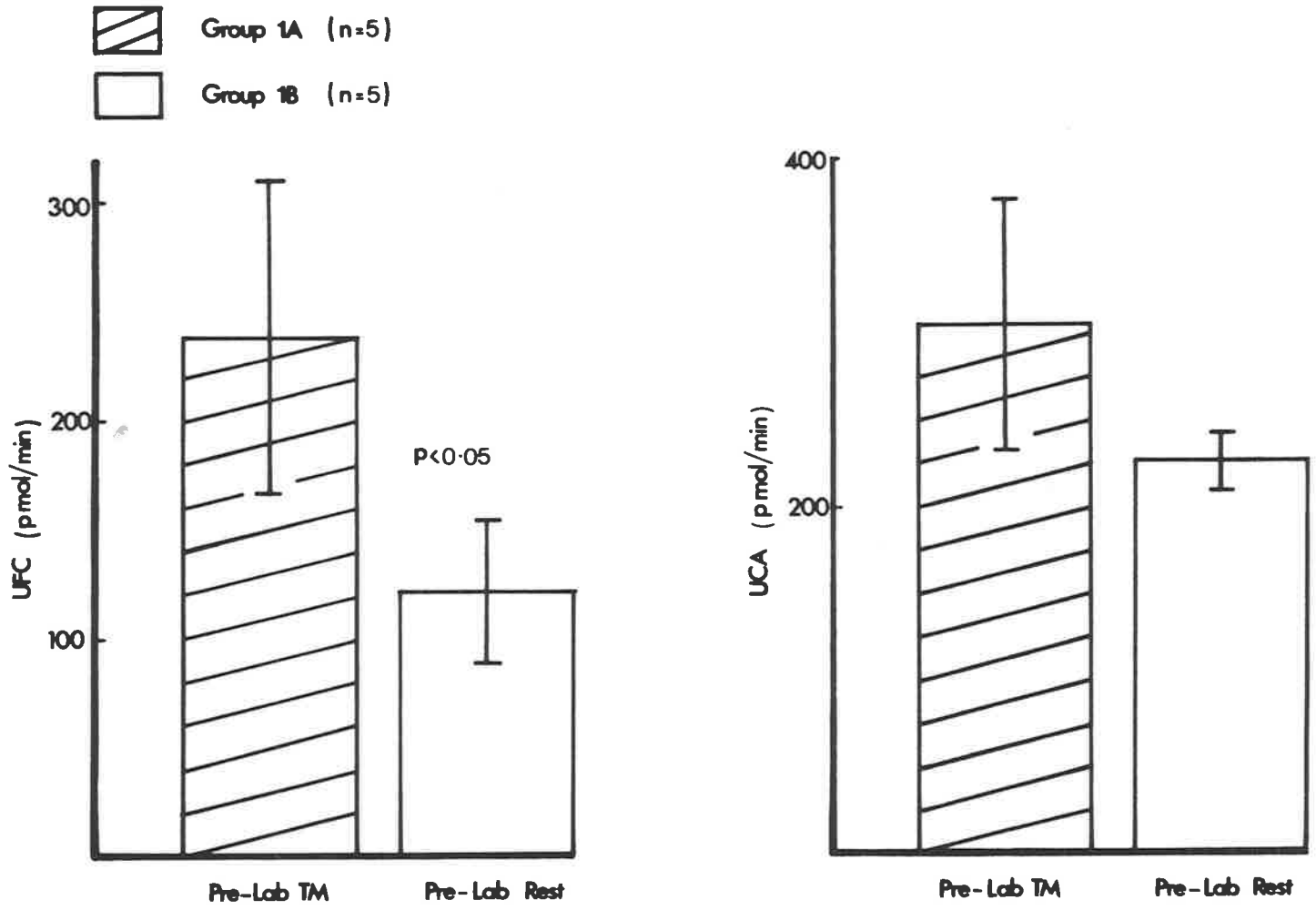


TABLE 50. Statistics from Within-group Comparisons on UFC, UCA, Urine Flow, Creatinine Clearance and Serum Osmolality

GROUP	UFC	UCA	Urine Flow	Creatinine Clearance	Serum Osmolality
1A:- TM; 1st experience	1.66(4)	1.11(4)	-1.22(4)	-1.00(4)	1.06(4)
1B:- Quiet Rest; 2nd experience	-0.63(4)	-1.93(4)	-2.27(4)*	-2.04(4)	3.81(4)**
3:- Quiet Rest; 1st experience	-0.50(4)	1.49(4)	-1.33(4)	-0.60(4)	3.16(4)*

On the day of the laboratory study UCA excretion was significantly higher in group 1A than group 1B ($t = 5.14$; $df = 4$; $p < 0.05$; paired t-test). Although UFC excretion was also higher in group 1A than 1B, the difference was not statistically significant ($t = 0.70$; $df = 4$; $p > 0.05$; paired t-test).

D. Summary of Short-term Endocrine, Other Biochemical and Haematological Changes in TM

Statistically significant reductions of blood lactate, serum cortisol, UFC, serum hGH and serum prolactin were generally[56, 57] (p 80) observed during TM, in contrast to a lack of significant change during quiet rest of experienced meditators and ordinary relaxation of untrained controls. No significant changes in UCA, PCV or total serum protein were found during any of the three treatments (Table 51).

TABLE 51. Summary of Endocrine, Other Biochemical and Haematological Changes During TM or Quiet Rest of Experienced Meditators, and Ordinary Relaxation of Untrained Controls

TEST	MEDITATORS		NON-MEDITATORS
	TM (n=16)	Quiet Rest (n=5)	Ordinary Relaxation (n=14)
blood lactate	↓[56]	-	NS
serum cortisol	↓[57]	NS	NS
UFC	↓[57]	NS	NS
UCA	NS	NS	NS
serum hGH	↓	NS	NS
serum prolactin	↓	NS	NS
serum T_4	↓[57]	NS	NS
FTI	↓[57]	NS	NS
serum T_3	↓	NS	NS
serum rT_3	NS	NS	NS
PCV	NS	NS	NS
serum protein	NS	NS	NS

↓ significant fall; $p < 0.05$

E. Statistical Analysis for Matched Meditators and Non-meditator Controls

(a) Group characteristics

Meditators and non-meditator controls were closely matched for time between cannulation and first blood sample collection (Δ (C-S)), and for age (Table 52). Approximate matching was achieved for all the other parameters. The mean meditation experience of the TM group was 2.6 (± 0.5) years with a range of 0.8 to 5.1 years. The occupations of subjects in each group were similar (For specific details, see Appendix IV, pp 185 and 186).

56. Not applicable to group 1A

57. NS for group 1A (n = 5)

TABLE 52. Group Characteristics (Mean ± SEM or Median) of Matched Meditators and Non-meditator Controls

CHARACTERISTIC	MEDITATORS (n=12)	NON-MEDITATORS (n=12)
Δ(C-S) (min)	45.9 ± 3.0	45.9 ± 3.3
Age (yr)	25.8 ± 1.1	24.8 ± 1.1
Body Surface Area (m^2)	1.86 ± 0.02	1.86 ± 0.03
Dietary Protein (g)[58]	67.9 ± 8.8	76.2 ± 8.2
Diet Rating (units)[58]	3	2
Urea Excretion (m mol/d)	295 ± 24	344 ± 34
Urine Volume (ml)	1142 ± 107	1365 ± 154
Previous Night's Sleep (h)	7.9 ± 0.3	8.1 ± 0.3
Usual Sleep (h)	7.8 ± 0.3	7.9 ± 0.3
Serum Protein (g/l)	68.5 ± 0.4	69.4 ± 0.7
Caffeinated Beverages (cups/d)	0	0
Tobacco (cigarettes/d)	0	0
Physical Training (min/wk)	214 ± 53	233 ± 82

(b) Overall inter-group comparisons

Overall group means of pre-treatment serum hormone levels, FTI, blood lactate, serum glucose values (samples 2 to 5 inclusive), and urinary hormone excretion on the same day are shown in table 53, along with the results of inter-group Student's t-test comparisons:-

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58. The eating pattern of all subjects was stable during the month prior to the study.

TABLE 53. Means ± SEMs and Statistics from Between-group Comparisons on Basal Serum Hormone Levels, FTI, Lactate, Glucose and Daily Urinary Hormone Values

TEST	MEDITATORS	NON-MEDITATORS	t(df)
cortisol (n mol/l)	197 ± 11	310 ± 21	4.82(93)***
hGH (m U/l)	5.8 ± 1.1	8.5 ± 1.5	1.45(93)
prolactin (m U/l)	125 ± 10	148 ± 11	1.54(92)
lactate (m mol/l)	1.2 ± 0.1	1.5 ± 0.1	2.70(45)***
glucose (m mol/l)	4.1 ± 0.1	4.8 ± 0.1	4.99(93)***
T_4 (n mol/l)	121 ± 3	105 ± 2	-4.41(90)***
FTI (units)	118 ± 3	106 ± 2	-3.49(90)***
T_3 (n mol/l)	1.530 ± 0.046	1.457 ± 0.048	-1.09(93)
rT_3 (n mol/l)	0.28 ± 0.01	0.23 ± 0.01	-2.92(87)***
UFC (n mol/d)	199 ± 16	262 ± 30	1.86(22)*
UCA (n mol/d)	462 ± 41	328 ± 19	-2.95(22)**

Serum cortisol, UFC, serum glucose and blood lactate levels were significantly lower, while total serum T_4, FTI, total serum rT_3 and UCA levels were significantly higher for the meditators than for the matched non-meditator controls (Figures 28, 29 and 30). There were no significant differences between the serum hGH, prolactin or total T_3 levels of the two groups (Table 53).

(c) Inter-group comparisons on temporal afternoon variation of blood test levels

Inter-group comparisons on temporal afternoon variation (one-way ANOVA on samples 2 to 5, inclusive) in blood test levels revealed significantly lower variation of serum cortisol, hGH and blood lactate, along with significantly higher variation of both FTI and serum rT_3, for meditators than for matched non-meditator controls (Table 54). No

FIGURE 28.
Basal Serum Cortisol, Serum Glucose and Blood Lactate Levels (Mean ± SEM)

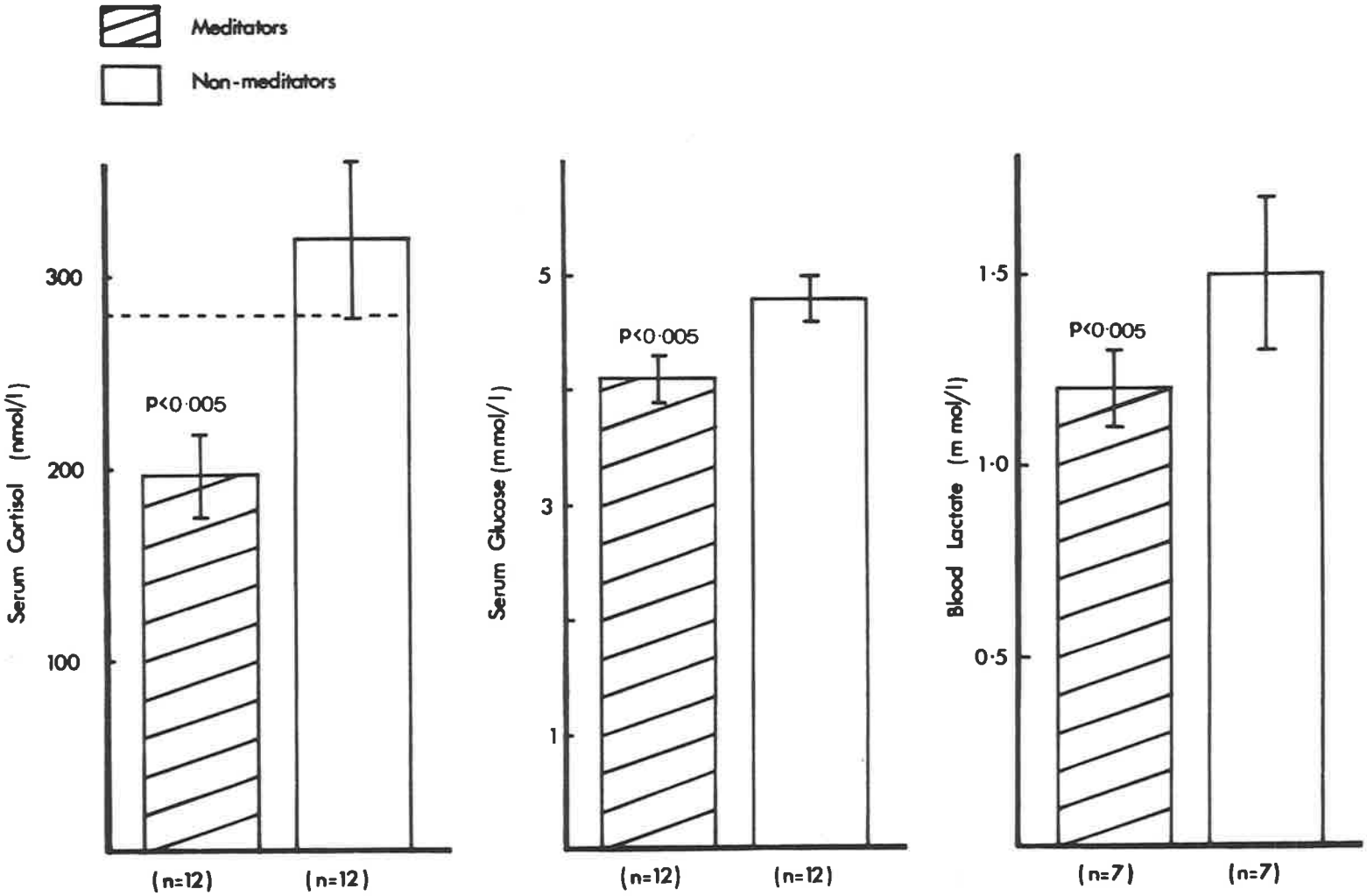


FIGURE 29.

Basal Free Thyroxine Index, Serum Triiodothyronine and Serum Reverse Triiodothyronine Levels (Mean±SEM)

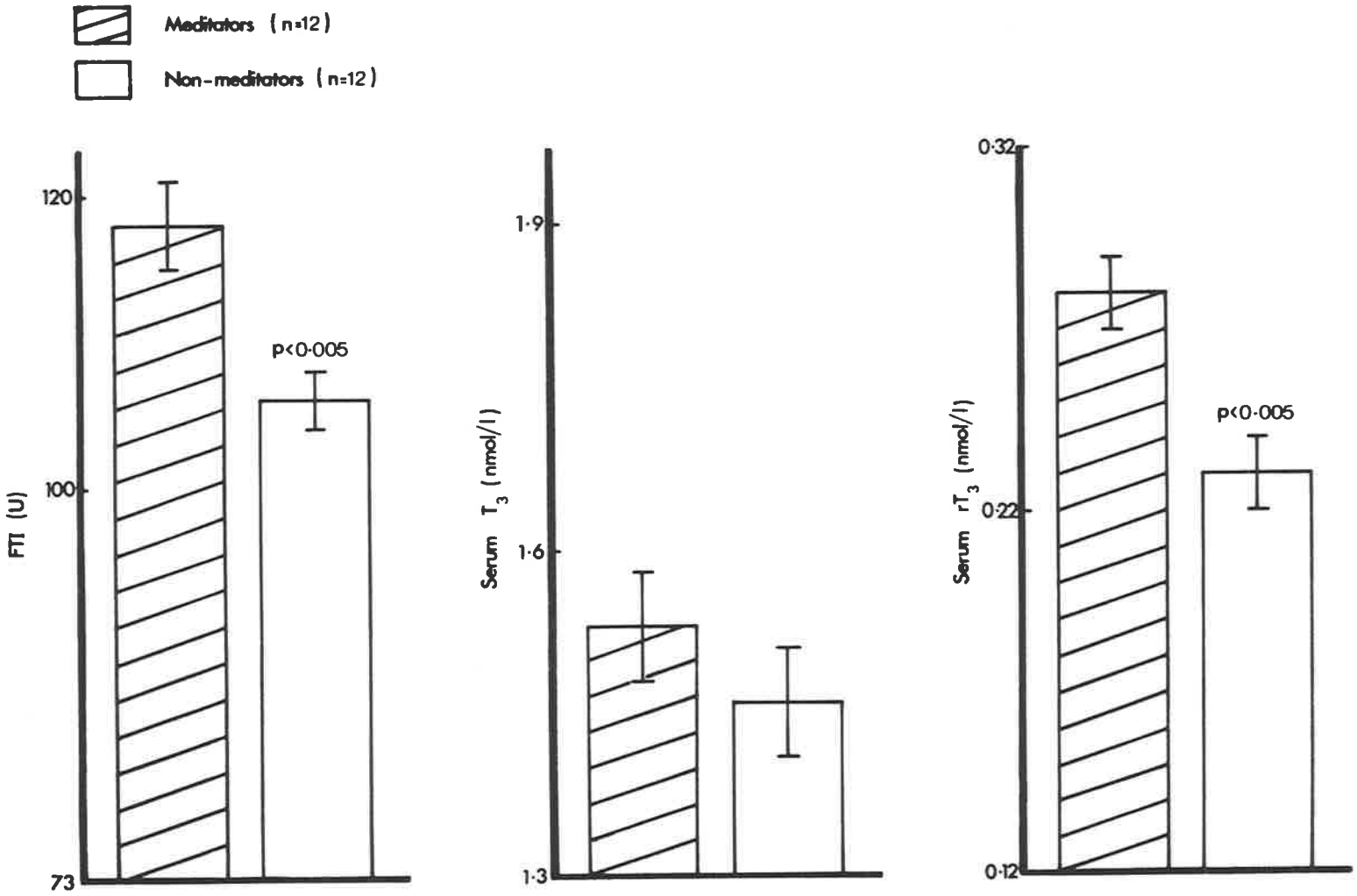
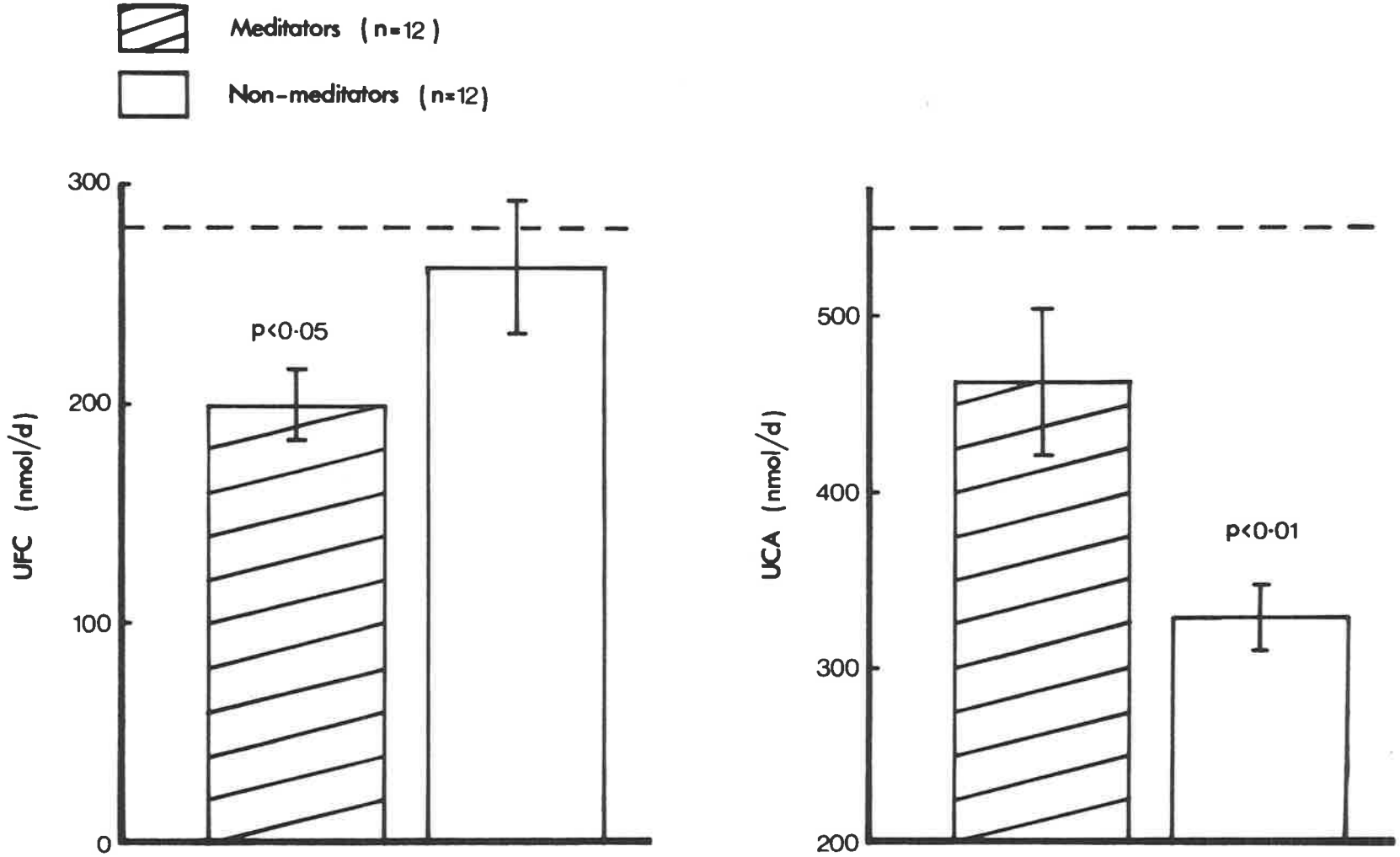


FIGURE 30. Urinary Hormone Levels (Mean±SEM) on Day of Laboratory Study



significant difference was found between meditators and controls in temporal afternoon variation of all other hormone tests.

TABLE 54. F Values from Inter-group Comparisons on Temporal Afternoon Variation of Serum Hormone, FTI, Glucose and Lactate Levels (v_1 = 11; v_2 = 11)

cortisol	hGH	prolactin	lactate	glucose	T_4	FTI	T_3	rT_3
3.13*	4.70**	1.16	3.85*	2.04	1.24	4.84**	1.23	2.84*

(d) Summary of basal endocrine changes associated with TM

The most outstanding differences between the basal hormone levels of experienced meditators and matched non-meditator controls were:- 1) significantly lower serum cortisol and daily UFC concentration in meditators than in non-meditators: 2) significantly lower afternoon variation in serum cortisol and hGH levels of meditators than of non-meditators; 3) significantly higher thyroid hormone test values (T_4, FTI and rT_3) and afternoon variation (FTI and rT_3) in meditators than in non-meditators; and 4) significantly higher daily UCA excretion in meditators than in non-meditators (Table 55).

TABLE 55. Summary of Basal Endocrine Differences Between Experienced Meditators and Matched Non-meditator Controls

HORMONE TEST	CONCENTRATION Meditators cf Non-meditators	TEMPORAL VARIATION Meditators cf Non-meditators
serum cortisol	<<<	<
serum hGH	NS	<<
serum prolactin	NS	NS
serum T_4	>>>	NS
FTI	>>>	>>
serum T_3	NS	NS
serum rT_3	>>>	>
daily UFC	<	NAp
daily UCA	>>	NAp

< ; > $p < 0.05$
<< ; >> $p < 0.01$
<<< ; >>> $p < 0.005$

F. TM Experience, TM Depth, Experimental Stress and Inter-hormone Relationships

(a) TM experience

Linear regression analysis on TM experience and experimental Saturday and Sunday UFC levels of all experienced meditators[59] (groups 1 and 3) revealed a significant negative correlation on Sunday but not on Saturday (Table 56). The results are presented diagramatically in figure 31. Similar results were obtained on the control week-end. No significant correlations were found between TM experience and UCA

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59. A significant negative correlation was found between TM experience and age ($r = 0.60$; $df = 19$; $p < 0.01$). Therefore, because of the possible influence of age-related endocrine changes (Finkelstein et al, 1972; Juselius and Kenny, 1974; Ziegler et al, 1976; Sever et al, 1977), subjects 14 and 15, the two oldest and most experienced meditators were excluded from the analysis. The correlation was thereby removed ($r = -0.03$; $df = 17$; $p > 0.05$). The possible age-related effect did not apply to within-subject comparisons.

FIGURE 31. Correlation of Week-end Urinary Free Cortisol Levels and TM Experience

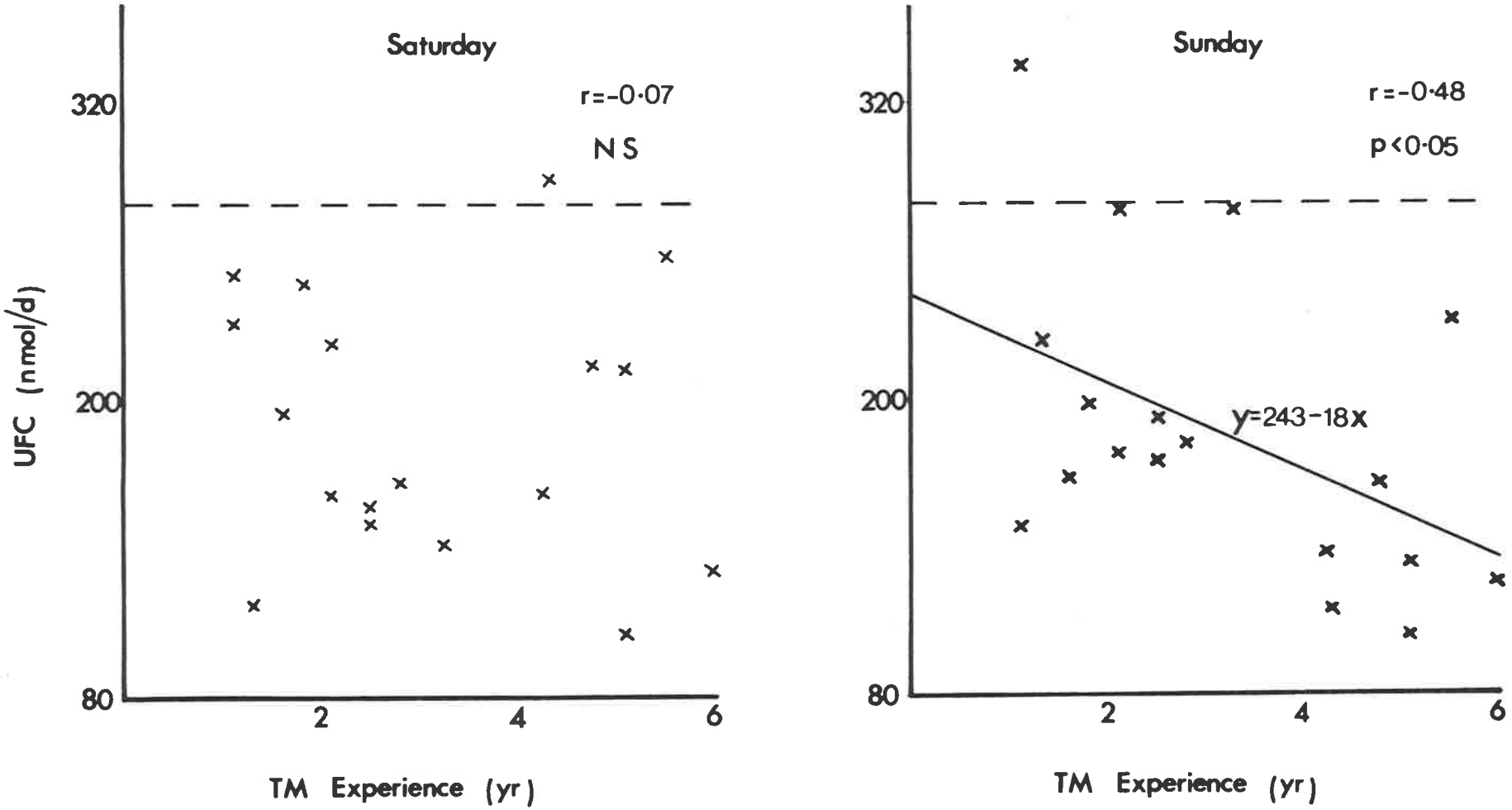
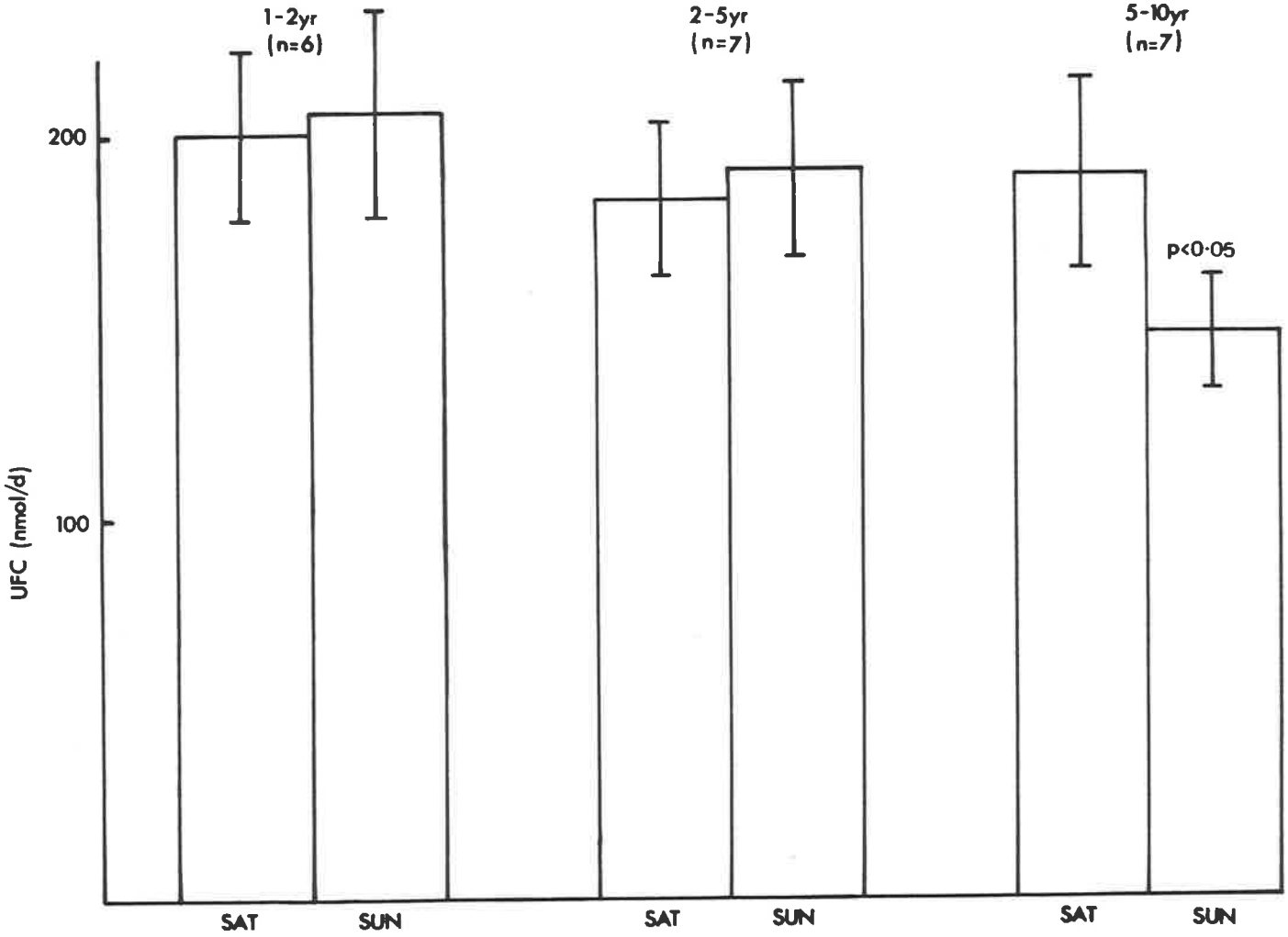


FIGURE 32.

Urinary Free Cortisol Levels (Mean±SEM) Over Week-end of Laboratory Study For Groups of Increasing Meditation Experience :-



levels of either the experimental or control week-ends (Table 56).

TABLE 56. Statistics from Linear Regression Analysis on TM Experience and Daily Urinary Hormone Levels

HORMONE	EXPERIMENTAL WEEK-END		CONTROL WEEK-END	
	SATURDAY	SUNDAY	SATURDAY	SUNDAY
UFC	-0.07(16)	-0.48(16)*	-0.06(12)	-0.40(12)
UCA	0.04(16)	0.09(16)	-0.49(12)	-0.23(12)

Table 57 gives the mean UFC levels, along with results of paired t-tests over the experimental week-end, for the three TM experience categories:- 1 to 2 years, 2 to 5 years, and 5 to 10 years regular meditation practice. As shown in figure 32, a significant (22%) fall in UFC from Saturday to Sunday occurred only in the advanced meditators (5 to 10 years' TM experience).

TABLE 57. UFC Levels (Mean ± SEM) and Statistics from Within-group Comparisons on Increasing TM Experience Categories

TM EXPERIENCE (yr)		UFC (n mol/d)		
CATEGORY RANGE	MEAN ± SEM	SATURDAY	SUNDAY	t(df)
1 to 2	1.5 ± 0.2	200 ± 22	206 ± 27	0.20(5)
2 to 5	3.1 ± 0.3	198 ± 21	193 ± 23	0.22(6)
5 to 10	6.4 ± 0.7	191 ± 25	149 ± 15	2.29(6)*

A large negative linear correlation was also observed between TM experience and both pre- and during- meditation serum cortisol and prolactin levels, however, a significant correlation during meditation occurred only for serum cortisol (Table 58). The trend towards significant serum cortisol-TM experience correlation as subjects entered meditation is shown in figure 33.

FIGURE 33. Correlation of Serum Cortisol and TM Experience

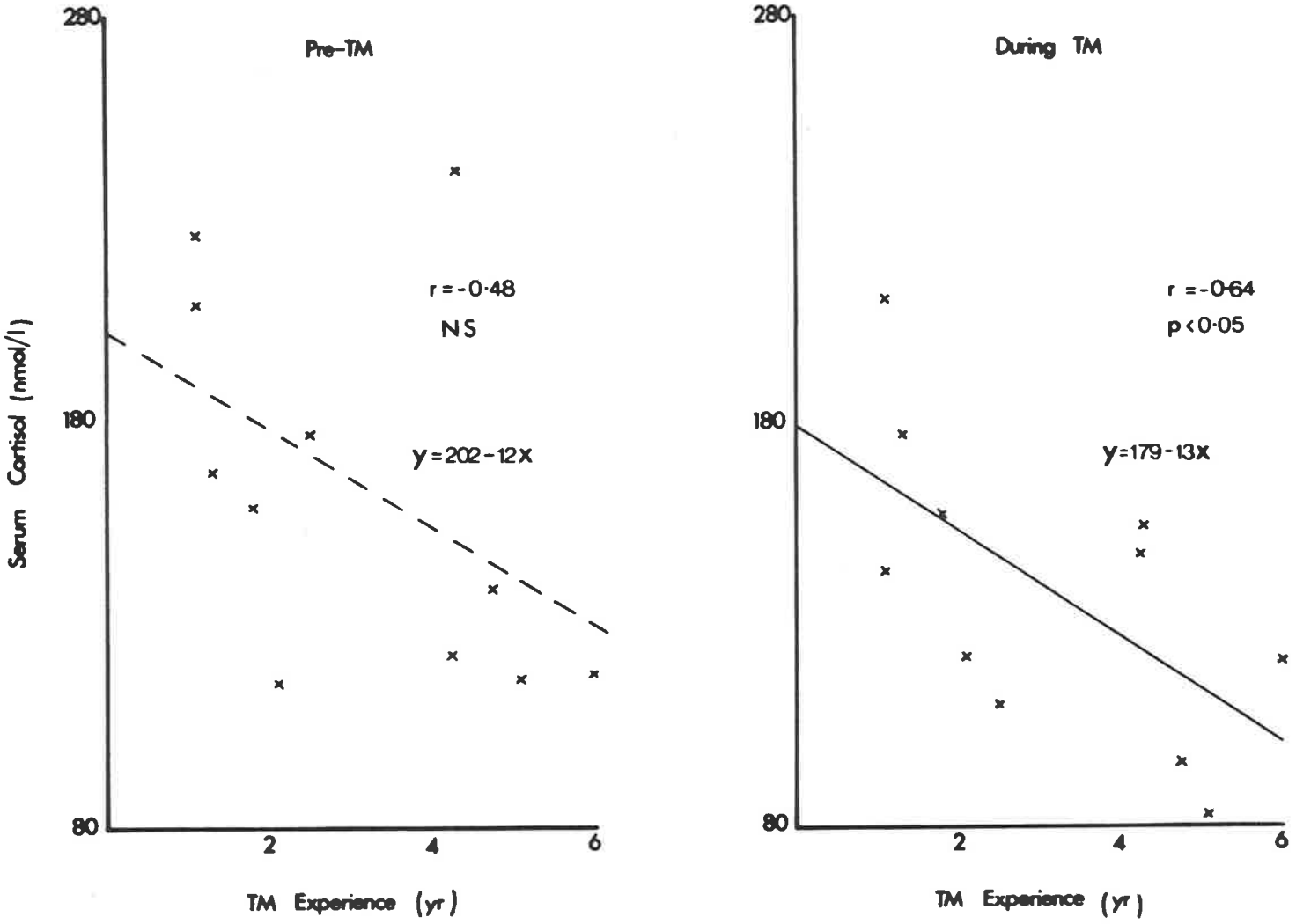


TABLE 58. Statistics from Linear Regression Analysis on TM Experience and both Pre-TM and TM-Induced Serum Hormone Levels[60]

HORMONE	PRE-TM	TM
serum cortisol	-0.48(9)	-0.64(9)*
serum hGH	0.08(9)	-0.06(9)
serum prolactin	-0.49(9)	-0.44(9)

Mean non-stress serum cortisol levels[61] before and during TM for the three TM experience categories previously given, along with results of within-group paired t-test comparisons, are presented in table 59. The findings are represented histographically in figure 34. The percentage reduction in serum cortisol during TM progressively increased from 10% in low-experience meditators (1 to 2 yr), through to 18% in moderately experienced subjects (2 to 5 yr), and finally reached significance (22% decrease) in the advanced group (5 to 10 yr).

TABLE 59. Mean ± SEM Serum Cortisol Levels (n mol/l) and Statistics from Within-group Comparisons on Increasing TM Experience Categories

TM EXPERIENCE (yr)	PRE-TM	TM	t (df)
1 - 2 yr	191 ± 16	172 ± 15	0.87(3)
2 - 5 yr	165 ± 29	135 ± 9	1.12(3)
5 - 10 yr	128 ± 6	100 ± 8	2.67(3)*

(b) TM depth and experimental stress

Linear regression analysis on ratings of TM depth and serum

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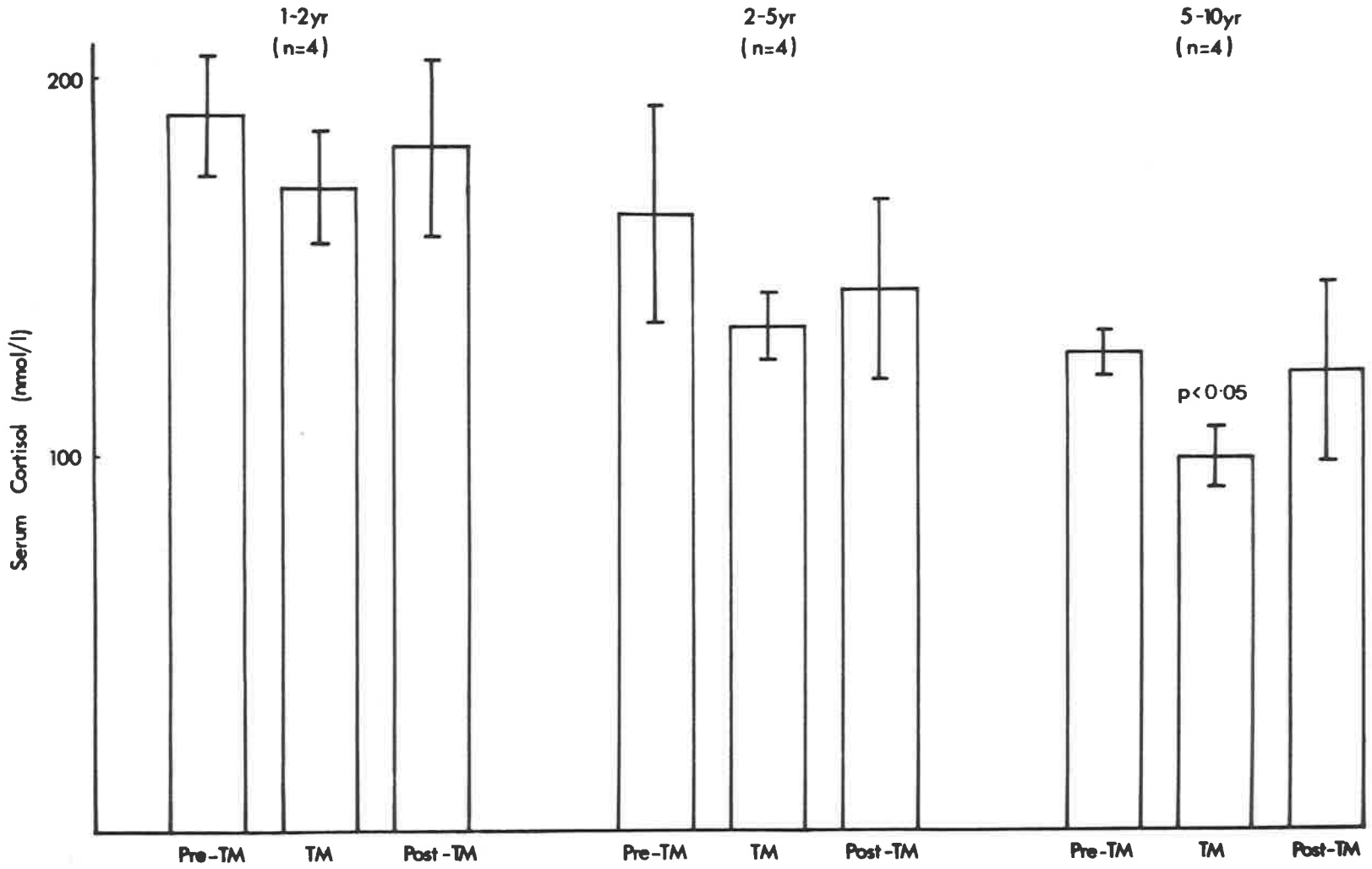
60. Subjects 14 and 15 were excluded because of a significant age-TM correlation (see foot-note 59, p 84). Subjects 1, 6 and 13 were also excluded here because of possible endocrine changes in response to re-cannulation during the experiment.

61. Subject 1 was also excluded here because of possible cortisol changes in response to re-cannulation.

FIGURE 34.

Serum Cortisol Levels (Mean± SEM) Before, During and After TM For Groups of Increasing Meditation Experience



cortisol, hGH and prolactin levels during meditation revealed a significant negative correlation for serum hGH only (Table 60). No significant TM experience - meditation depth correlation was observed ($r = 0.32$; $df = 14$; $p > 0.05$).

Linear regression analyses on self-reported experimental stress ratings (discomfort-involvement ratings) and TM-induced serum hormone levels revealed large positive correlations for both cortisol and prolactin. In contrast, the correlation between self-reported experimental stress and TM-induced serum hGH levels was small (Table 60).

TABLE 60. Statistics from Linear Regression Analysis Between TM Depth or Experimental Stress and Serum Hormone Levels During Meditation

	cortisol	hGH	prolactin
TM depth	0.19(14)	-0.71(14)**	-0.09(14)
Experimental stress	0.48(14)	0.11(14)	0.71(14)**

The above correlations are qualitatively similar to those observed between TM experience and serum hormone levels. However, Spearman rank correlation tests (Siegel, 1956; pp. 202-213) failed to reveal a significant relationship between TM experience and self-reported experimental stress ($T = -1.75$; $df = 14$; $p > 0.05$), nor between TM experience and depth of laboratory meditation ($T = 1.42$; $df = 14$; $p > 0.05$). A significant correlation was found, however, between depth of meditation and discomfort-involvement ratings ($T = -2.72$; $df = 14$; $p < 0.05$[62]; Spearman rank correlation test).

(c) Serum hormone inter-relationships

High positive post-meditation inter-hormone correlations were found for serum prolactin, hGH and cortisol. However, a significant

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62. p value was corrected for 3 multiple comparisons.

correlation was found only between prolactin and cortisol levels during TM, while the pre-meditation prolactin-cortisol coefficient of correlation was also high (Table 61).

TABLE 61. Statistics from Linear Regression Analysis Between Serum Cortisol, hGH and Prolactin Levels Before, During and After TM

	PRE-TM		TM		POST-TM	
	prolactin	cortisol	prolactin	cortisol	prolactin	cortisol
hGH	0.11(14)	0.03(14)	0.02(14)	-0.12(14)	0.46(14)	0.44(14)
prolactin	-	0.42(14)	-	0.66(14)*	-	0.55(14)

(p values were corrected for 3 multiple comparisons)

PART 4 :- TM RESIDENCE COURSE STUDY

A. Group Characteristics

The mean ages of subjects in both novice and experienced TM groups were similar (Table 62), however, there was a marked inter-group difference in meditation experience. The ranges of TM experience were 0.1 to 0.6 years and 1.1 to 4.3 years for the novice and experienced meditators, respectively.

TABLE 62. Group Characteristics (Mean ± SEM) for the TM Residence Course Study

GROUP	NUMBER	AGE (yr)	TM EXPERIENCE (yr)
Novice Meditators	8	23.4 ± 1.5	0.4 ± 0.1
Experienced Meditators	9	29.1 ± 3.0	2.5 ± 0.4

B. Temporal Hormone Changes

The mean UFC and UCA levels for the novice and experienced TM groups on the Saturday and Sunday of the TM residence course and on the following Monday are presented in table 63:-

TABLE 63. UFC and UCA Levels (Mean ± SEM) During the TM Residence Course Study

GROUP	UFC (n mol/d)			UCA (n mol/d)		
	SAT	SUN	MON	SAT	SUN	MON
Experienced Meditators	178±13	128±15	131±11	226±47	198±17	245±38
Novice Meditators	196±17	190±31	176±15	372±92	301±66	331±109

Results of within-group paired t-test comparisons for UFC and UCA are given in tables 64 and 65, respectively. A significant 28 percent fall in UFC levels of the experienced meditators occurred

from Saturday to Sunday of the TM residence course[63]. UFC levels remained low on the following Monday. In contrast, no significant changes in UFC levels of the novice group were observed (Table 64). The UFC findings are represented histographically in figure 35. No significant changes in UFC levels were observed for either the experienced TM group or novice TM group (Table 65).

TABLE 64. Statistics from Within-group Comparisons between UFC Levels of the TM Residence Course Study.

GROUP	SAT cf SUN	SUN cf MON	SAT cf MON
Experienced Meditators	6.40(8)***	-0.32(4)	2.86(4)
Novice Meditators	0.27(7)	0.18(5)	0.49(5)

(p values were corrected for 3 multiple comparisons)

TABLE 65. Statistics from Within-group Comparisons between UCA Levels of the TM Residence Course Study.

GROUP	SAT cf SUN	SUN cf MON	SAT cf MON
Experienced Meditators	0.83(8)	-1.72(4)	-0.68(4)
Novice Meditators	0.94(7)	-0.77(5)	0.91(5)

(p values were corrected for 3 multiple comparisons)

C. Summary of Endocrine Changes During TM Residence Course and Ordinary Week-end

In summary of the urinary hormone changes observed for meditators during a week-end TM residence course and ordinary week-end (Part III, Section F; p 85), significant UFC reductions were found in both long-term meditators (1 year < TM experience < 5 years) studied during a residence course and in advanced meditators (> 5 years' TM

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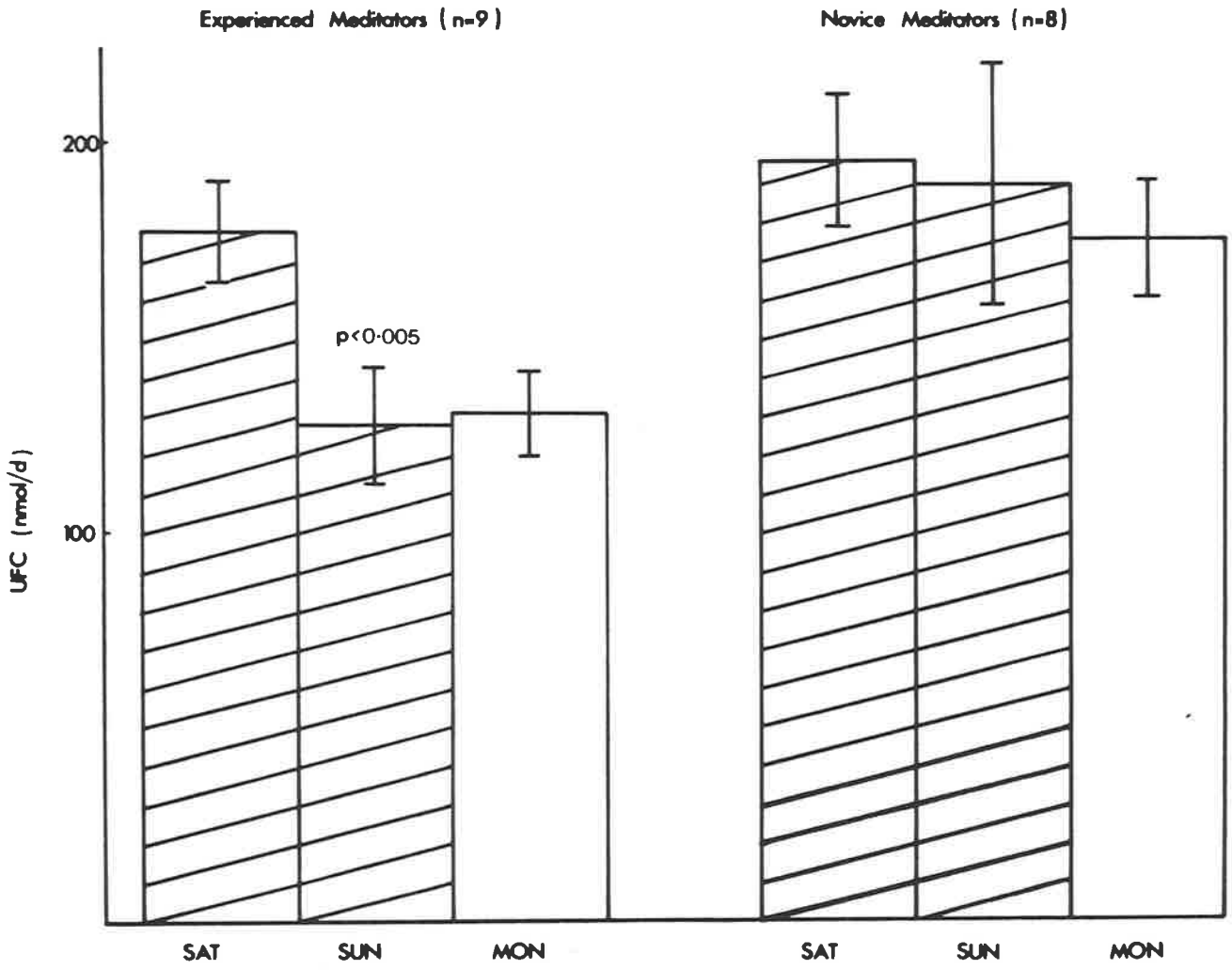
63. No significant change in urine volume was found ($t = 0.11$; $df = 8$; $p > 0.05$; paired t-test).

FIGURE 35.

Urinary Free Cortisol Levels (Mean±SEM) Over Week-end of TM Residence Course:- 



experience) investigated during an ordinary week-end. These decreases contrast with the lack of significant UFC changes observed in 1) novice meditators during the TM residence course, and 2) both novice and long-term meditators studied over an ordinary week-end. No significant UCA changes were found either during the TM residence course or ordinary week-end for any of the meditation experience categories (Table 66).

TABLE 66. Summary of Endocrine Changes which Occurred from Saturday to Sunday of TM Residence Course or Ordinary Week-end

	RESIDENCE COURSE		ORDINARY WEEK-END		
HORMONE	Novice Meditators	Long-term Meditators	Novice Meditators	Long-term Meditators	Advanced Meditators
UFC	NS	↓↓↓	NS	NS	↓
UCA	NS	NS	NS	NS	NS

↓ significant fall ; $p < 0.05$
↓↓↓ significant fall ; $p < 0.005$

CHAPTER IV : DISCUSSION

A. Inherent Methodological Problems of the Long-term Study

There are relatively few long-term studies of relaxation procedures probably because of the inherent methodological problems associated with them. The most successful long-term investigations have been performed on patients whose participation in the study was an integral part of their treatment for a particular stress-related disorder (See, for example, Borkovec and Krogh Sides, 1979).

The main methodological problems of this long-term study arose from reliance upon voluntary participation of subjects. High drop-out rates from relaxation training along with infrequency of sample collection in some subjects were inevitable, however, they were greater than anticipated when designing the study (see foot-note 14, p 19). Similar problems with high attrition during relaxation training were reported by Otis (1974).

The lesser drop-out in the yoga-meditation group might be associated with the continued follow-up training, which was an integral part of the yoga-meditation training programme, rather than some special feature of the yoga-meditation procedure. Although follow-up training was encouraged in the other procedures, it was not a structural part of the training programme. Follow-up training, with its associated positive reinforcement (Morse et al, 1977), therefore, appears to be important for continued relaxation practice.

B. Endocrine Changes During Relaxation Training of the Long-term Study

(a) Plasma cortisol

The significant long-term reduction in plasma cortisol levels observed in both untrained controls and trained meditators, as well as TM trainees, demonstrates that the change was not specific to TM training. The lack of significant UFC changes in all groups indicates

that the observed reduction in plasma cortisol was not due to seasonal variation (Watanabe and Yoshida, 1956; Halberg et al, 1965b). The progressive reduction in plasma cortisol during the study probably represents an habituation to venepuncture stress, other stresses related to blood sampling, and novelty of testing (Davis et al, 1962; Markiewicz et al, 1973; Mason et al, 1973; Mikulaj et al, 1976). It is difficult to place any specific interpretation on the significant plasma cortisol rise observed during May in the untrained controls.

(b) Thyroid hormones

The progressive falls in T_4 for the untrained controls and progressive relaxation trainees and the decline in T_3 levels of the trained meditators might also be associated with adaptation to the stress of blood sampling (Falconer and Jacks, 1972; Falconer, 1976; Mikulaj et al, 1976). The lack of consistent significant reductions in all groups probably reflects individual variation in both previous exposure to venepuncture and response to venepuncture stress (Davis et al, 1962; Copinschi et al, 1967; Mason 1968c).

The significant T_4 rise observed during academic examinations concurs with the reports by Tingley et al (1958) and Mason (1968c) of significant protein bound iodine (PBI) elevations in exam stress. However, Volpé et al (1960) found no significant change in serum PBI during examinations of either medical students or graduates, although the highest PBI levels were found during annual exams of medical students. The simultaneous fall in T_3 during exams might be associated with a reciprocal increase of rT_3 (Chopra et al, 1975; Burr et al, 1975).

The absence of plasma cortisol, UFC and UCA rises during academic exams conflicts with other reports of significant adrenocortical (Bliss et al, 1956; Melick, 1960; Hodges et al, 1962; Bloch and Brackenbridge, 1972; Baseer and Rab, 1975) and adrenomedullary

(Bogdonoff et al, 1960; Bolshakova, 1976) activation. However, the above studies involved a short-term experimental design and looked at changes immediately following the examination. In most cases, the volunteer subjects studied here refused to give blood samples on the day of exams, and consequently, the long-term experimental design might not have been suitable for detecting changes in adrenal hormones which have much shorter biological half-lives (and are, therefore, more short-lived) than thyroid hormones. In fact, a relatively longer duration of elevated thyroid function than both adrenocortical and adrenomedullary activity was observed by Mason et al (1961), in association with psychogenic stress.

These preliminary findings suggest that alterations in thyroid function might provide a useful measure for monitoring long-term physiological changes associated with exam stress, and further detailed investigations, which take into consideration possible changes in peripheral monodeiodination of T_4 (Chopra et al, 1975; Burr et al, 1975) and/or compartmental shift (Chan et al, 1978), are warranted.

(c) Urinary hormones

Normalisation effects on physiological functions during relaxation training, similar to those observed here for the UFC and UCA levels of subject 90, have been previously reported by other workers. Luthe and Schultz (1969 Vol 2) extensively reviewed the subject of normalising influences on endocrine and metabolic disorders during autogenic training (see p 14). Normalising influences on blood pressure of patients with essential hypertension have been associated with various forms of relaxation training (Luthe and Schultz, 1969, Vol 2, pp 69-76; Jacob et al, 1977; Frumpkin et al, 1978; Benson et al, 1978a; Frankel et al, 1978; Black, 1979) and normalisation of body weight has been reported in the TM program (Weldon and Aron, 1977).

C. Assessment of Subjective Changes in Relaxation Procedures

(a) Short-term study (1)

The lack of difference between relaxation-depth ratings of trained, untrained, and in-training subjects, except the yoga-meditation group, might reflect the semantic problems associated with rating the subjective reports. The failure of this method in differentiating between ordinary relaxation and all of the specific relaxation states, except yoga-meditation, may be because it was initially designed for rating Buddhist meditation (Maupin, 1965). Consequently its suitability for rating relaxation procedures might be restricted to procedures such as yoga-meditation, which have a Buddhist origin[64]. The inadequacy of this method for subjectively defining and characterising non-Buddhist meditation, or other specific relaxation procedures, is highlighted by the marked inter-group differences in plasma cortisol changes (see p 98).

(b) Short-term study (2)

The success of the meditation-relaxation questionnaire in discriminating between TM and ordinary relaxation indicates the usefulness of this method of assessing subjective changes in TM. The surprisingly higher laboratory meditation ratings than home (Saturday evening) meditation scores, might be due to a group-dynamics effect of the laboratory session. Indeed, meditators commonly report better group meditations than private meditations (personal observation, A. Bevan). Therefore, although successful laboratory meditation is definitely implicated by the high meditation depth ratings, the endocrine changes observed during the laboratory investigation of TM might only be representative of meditation in a group setting with concomitant novelty. In fact group dynamics, as well as novelty, have been found

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64. However, the yoga-meditation procedure used here was not entirely based upon Buddhist methods.

to significantly influence endocrine function (Fishman et al, 1962a).

Further development of meditation-relaxation questionnaires, involving a sliding scale of possible responses and computerised analysis, would be useful for more specific characterisation of subjective changes in meditation and other specific relaxation states. Signalling of subjective changes by button-pressing has been successfully used in electrophysiological identification of specific TM-induced states (Banquet, 1973; Farrow, 1977), and might also be applied to more detailed endocrine characterisation of TM, particularly where reductions in hormones with short half-lives are concerned[65].

D. Cardiovascular Changes in Relaxation Procedures

The lack of significant changes in mean arterial blood pressure following relaxation for both relaxation trainees and trained subjects agrees with previous reports on normotensives (Wallace, 1970a; Wallace et al, 1971; Michaels et al, 1979). The significant reduction in blood pressure following yoga-meditation is difficult to interpret because of the effects of postural changes (Ward et al, 1966) involved in performance of the yoga-meditation procedure[66] (see Appendix II, pp 160 and 161). Cardiovascular studies of various yoga-meditation procedures have revealed increases (Wegner and Bagchi, 1961), decreases (Gopal et al, 1973), and no change (Karambelkar et al, 1968) in blood pressure, and differing blood pressure changes probably occur in response to the

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65. The button-pressing method is inherently less useful in endocrine studies than electrophysiological investigations. In endocrine studies, it is most amenable to trophic hormone measurement because of the relatively large time-lag between neural response and the effect on target hormones.

66. Many of the yoga postures were performed in a supine or semi-supine position (See Appendix II, pp 160 and 161).

various procedures used (Woolfolk, 1975). Furthermore, cardiovascular changes specific to individual yoga postures have been found (Gopal et al, 1974).

Disregarding the possibility of methodological interferences, it is worth noting that the yoga-meditation group was also characterised by markedly higher relaxation depth scores than any other group. Therefore, the significant reduction in blood pressure might be associated with induction of a deeper state of relaxation by the yoga-meditation procedure than by the other procedures studied.

The significant nine percent fall in heart rate observed following TM in experienced subjects is consistent with other reports of reduced heart rate during TM (Wallace, 1970b; Wallace et al, 1971; Jevning et al, 1978c), and demonstrates successful laboratory meditation. However, these other workers did not find significantly lower post-meditation levels. The slight, but statistically significant, five percent reduction in heart rate following ordinary relaxation of untrained controls, in contrast to no significant change in relaxation trainees, might be due to selection bias. All controls but one were medical students, whereas the relaxation trainees were predominantly arts students who were less familiar with the medical school laboratory setting and experimental proceedings. Consequently the relaxation trainees were probably less inclined to relax during the laboratory investigations (Moss, 1974). The slightly lower pre-treatment plasma cortisol levels of the untrained controls than both trained and in-training subjects might also reflect intrinsic group differences in both predisposition to the stressor effects and novelty of the laboratory study. For a similar reason the report of significant heart rate fall during ordinary relaxation for non-meditator controls, in contrast to no change during TM (Michaels et al, 1979), was probably due to use of controls who were volunteers from the research unit which conducted the

study (Michaels et al, 1976). The common practice of using laboratory personnel as controls has been criticised for similar reasons (Levi, 1972; p 36), however, restriction to the use of volunteer subjects in this study presented an inherent, but unforeseen, methodological problem.

Only one of the trained meditators was a medical student, all others being as unfamiliar with the laboratory environment and experimental proceedings as the relaxation trainees. Therefore, the observed reduction in heart rate for the trained group is independent of such sample bias and the best comparison is with the relaxation trainees.

E. Blood Lactate Changes in TM

The significant reduction in venous blood lactate during and after TM, in contrast to a lack of change during ordinary relaxation of non-meditator controls, is in agreement with other reports of a significant TM-induced arterial blood lactate reduction (Wallace et al, 1971; Jevning and Wilson, 1976; Nandagopal et al, 1976; Rama Rao et al, 1977; Jevning et al, 1978c). The congruency of changes in both blood lactate during TM, and the higher relaxation depth scores in TM than ordinary relaxation, provides complementary evidence for the usefulness of reduced lactate levels as a biochemical reference for effective meditation (Jevning et al, 1978c).

F. Endocrine Changes in Relaxation Procedures: Short-term Study (1)

(a) Plasma cortisol

The significant fall in plasma cortisol following meditation for the trained group, in contrast to a lack of significant change for the relaxation trainees and untrained controls, agrees with findings from studies of autogenic training (Alnaes, 1966; Alnaes and Skaug, 1966) and a recent report on TM (Jevning et al, 1978a). The lack of

significant changes in both heart rate and plasma cortisol in the relaxation trainees confirms that the most marked physiological changes occur in experienced subjects (Kanellakos and Lucas, 1974, p 47 and p 89; Luthe, 1970, p 91 and p 92). The observed similarity between relaxation ratings and heart rate changes of the untrained controls and trained meditators, along with observation of significant TM-induced plasma cortisol reduction, in contrast to no significant change in ordinary relaxation of untrained controls, demonstrates the importance of using response patterning to identify and characterise relaxation states[67].

(b) Thyroid hormones

The significant increase of T_4 concentration following TM in meditation trainees, and the significant increase of T_3 levels following the relaxation session of progressive relaxation and auto-hypnosis-relaxation trainees, are difficult to interpret. The lack of consistent intra-group increases in concentration of all protein-bound hormones studied, demonstrates that the changes were not due to a haemoconcentration effect. The increase in thyroid hormone levels might be the result of similar stress-induced changes to those occurring in other endocrine systems in response to repeated venepuncture (Copinschi et al, 1967; Frankenhaeuser et al, 1976). However, the lack of consistency in direction of T_3, T_4 and cortisol changes makes this possibility unlikely.

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67. The importance of using response patterning in the characterisation of physiological stress reactions was emphasised by Lacey (1967) and more recently has been extrapolated to changes induced by instrumental biofeedback (Schwartz, 1975).

G. Basal Urinary Free Cortisol Levels of Relaxation Trainees and Trained Meditators

The short-term plasma cortisol reduction in TM is probably at least partially responsible for the lower UFC excretion of trained subjects which was observed in both the long-term study and the short-term studies. The lower UFC levels in these subjects might reflect the more marked TM-induced changes of trained meditators, rather than endocrine changes outside of a meditation-induced state. However, the possibility remains that the significantly lower overall UFC levels in trained meditators than in untrained controls, might be associated with reduced basal adrenocortical secretion and/or decreased cortisol stress responsiveness (see p 103 for further discussion). The lack of significant difference between UFC levels of trained meditators and trainees, as well as between trainees and untrained controls, suggests that the changes in adrenocortical activity might be cumulative with increased relaxation training.

Reduced basal adrenocortical secretion would involve one or a combination of the following changes:- fewer secretory episodes, shorter secretory episodes, reduced amount of cortisol per episode, and more rapid clearance possibly caused by a reduced half-life. Decreased cortisol stress responsiveness would involve a diminished initial response with consequent reduction to overall response; and/or more rapid adaptation with a less sustained response; or a combination of increased initial response and more rapid adaptation with a less sustained response (For further discussion see pp 103 and 104).

H. Endocrine Changes in TM: Short-term Study (2)

(a) Serum cortisol and urinary free cortisol

Reduced cortisol levels have previously been reported in both heterohypnotic relaxation (Sachar et al, 1965; 1966) and autogenic training (Alnaes and Skaug, 1966; Alnaes, 1966). However, studies of

cortisol changes in TM have produced conflicting findings. Jevning et al (1977a; 1978a) found a significant plasma cortisol reduction in TM, whereas Michaels et al (1979) reported no significant difference between the effects of meditation and ordinary relaxation on plasma cortisol levels. There are several outstanding methodological shortcomings of the study by Michaels' group, which might explain the discrepant findings:- The study by Michaels et al (1979) was conducted in the morning when initially high cortisol levels follow a morning circadian decline, whereas Jevning's group made their observations during the afternoon, a period of relative cortisol stability (Krieger et al, 1971). Michaels' team studied only 9 meditators compared with Jevning et al's 15, therefore, a type 2 statistical error (Croxton and Cowden, 1965; p 639) and/or TM experience effect (see p 124) are likely explanations. Furthermore, Michaels et al made no allowance for habituation to cannulation stress, blood sampling commencing immediately after cannula insertion. Consequently, possible differences in cannulation stress responsiveness between meditators and non-meditators were not accounted for (Davis et al, 1962; Greene et al, 1970). However, the possibility remains that the discrepancy between results of afternoon and morning studies might be associated with circadian differences in the effects of TM on cortisol secretion. Furthermore, long-term regular meditation practice, with its accompanying short-term cortisol reductions, might have an effect upon cortisol's circadian rhythm. Meditators often report marked differences in the quality of their morning and evening meditation sessions (personal observation, A. Bevan), and possible differences in physiological response await specific investigation. Although, by the law of initial values, reductions are less likely to be observed when values are already low, relatively low initial, pre-meditation cortisol levels, such as those occuring during the afternoon, might be a prerequisite for significant reduction in

meditation. In fact, advanced meditators (more than 5 years' experience), who had the lowest pre-meditation cortisol levels, were the only group to show significant cortisol reductions in TM. A similar observation was made by Morse et al (1977) who found greater skin resistance increases during meditation or hypnosis when the pre-treatment state was relaxation rather than alertness. The possibility of an effect of pre-treatment state on endocrine changes requires further investigation.

The marked 34 percent fall in plasma cortisol in the preliminary short-term study, compared with the lesser 17 percent reduction after TM in the follow-up study, is probably due to the longer period of observation used in the first instance. The fact that post-meditation observation for the preliminary study extended into late afternoon, a usual circadian phase of cortisol reduction (Weitzman et al, 1971), probably also contributed to the difference in degree of cortisol change. Cortisol's half-life of about 70 minutes (Weitzman et al, 1971; Gallagher et al, 1973) means that, with a treatment period of a half-hour, the most marked effects would probably be observed following the meditation period.

The lack of difference between the pre-laboratory UFC excretion of meditators and non-meditator controls suggests that the adrenocortical activation of the non-meditators occurred during the laboratory experimentation rather than in apprehension of it[68]. Furthermore, the similarity of pre-laboratory UFC excretion, and the marked inter-group differences in cortisol responsiveness to both

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68. It is important to note that because of the high protein binding of circulating cortisol, urinary levels provide a less sensitive determination of changes in adrenocortical function. UFC estimation is, however, a very useful method for determining integrated levels, and has the advantage of involving a non-stressful collection procedure (assuming exclusion of novelty of collection effects; Fishman et al, 1972a).

experimental stress and relaxation, suggests that the lower 24 hour UFC excretion of meditators is due to changes in cortisol stress responsiveness, and short-term TM-induced changes, rather than differences in basal secretion.

The extension of both the non-meditators' pre-treatment serum cortisol levels and laboratory Saturday UFC excretion above the upper limits of the normal reference ranges indicates a marked cortisol stress response to the experimental proceedings. The similarity of self-reported experimental stress for meditators and non-meditator controls, along with lower serum cortisol levels of meditators, suggests a more rapid adaptation to the experimental stress, rather than general dampening of the cortisol stress response, as proposed by Michaels et al (1979). Furthermore, the significantly higher UFC excretion of the meditators on the laboratory Saturday than control Saturday, in contrast to a lack of significant difference for the non-meditator controls, suggests a greater, rather than lesser, initial adrenocortical stress response. The study by Michaels et al (1979) was not designed specifically to look at adrenocortical stress responsiveness, and the 20 minutes between blood samples might have been too long to accurately depict the response pattern (Gallagher et al, 1973). Other methodological short-comings of this study were discussed earlier (pp 101 and 102).

The significantly higher UFC excretion of the meditators on the laboratory Saturday than on the control Saturday, and the significantly higher first experience than second experience pre-laboratory UFC levels of meditators, implies that the low basal cortisol values are not due to a deficit in adrenocortical reserve (Friedman et al, 1972).

Other studies of stress reactivity in meditators have used non-endocrine measures. Orme-Johnson (1973) reported similar initial galvanic skin responses to noise stress, with more rapid adaptation to stress, in experienced meditators than non-meditator controls. Short-

term effects of TM on physiological stress responsiveness to a stressful film were studied by Goleman and Schwartz (1976). Initial heart rate and galvanic skin responses of experienced meditators were greater, and recovery following the stressful stimuli faster, in the experienced meditators than in non-meditator controls. Since concurrent cortisol and heart rate increases appear to occur in response to stress (Raab, 1968), it seems likely that TM would also increase, rather than decrease, the initial adrenocortical stress response. A pattern of stress response involving a marked initial response with rapid recovery appears to be indicative of a healthy adaptive response (Stern et al, 1965; Lazarus and Averill, 1972). In this context it is interesting to note that following work stress more rapid recovery of adrenaline to pre-stress levels occurs in persons with higher basal adrenaline excretion (Johansson and Frankenhaeuser, 1973). Therefore, the observation of higher basal catecholamine levels provides corroborative evidence for such adaptive endocrine responses in meditators. Furthermore, the similar temporal adaptation to repeated venepuncture stress, observed for both meditators and non-meditator controls in the long-term study, provides further evidence against a reduced initial cortisol stress response for meditators. Nevertheless, further studies designed specifically to investigate the cortisol stress responsiveness of meditators are still required in order to clarify the picture.

(b) Growth hormone

The significant reduction of hGH during TM in contrast to no significant change during ordinary relaxation in non-meditators or quiet rest in meditators, along with the significant negative correlation between TM depth and hGH levels during meditation, indicate that decreased hGH levels are specific to a TM-induced state. These findings conflict with those of Jevning et al (1978b) who reported no significant hGH

changes in TM. There are several outstanding methodological differences between the two studies. Jevning et al (1978b) used arterial rather than venous catheterisation and sampling[69], less frequent blood sampling (20 minute in contrast to 10 minute sampling intervals), and fasting rather than fed subjects. Furthermore, Jevning's team appeared to conduct their experiments on single subjects rather than on groups of subjects (see p 95 for consideration of possible effects of group dynamics).

Arterial catheterisation is inherently more stressful than venous catheterisation in terms of hGH stress responsiveness (Copinschi et al, 1967), however, the lengthy two and a half hours between initial catheterisation and first blood sampling should have been adequate for any stress-related elevations to return to basal levels (Copinschi et al, 1967). Indeed, the pre-treatment values reported by Jevning et al (1978b) were very similar to the pre-treament levels found here.

Frequency of blood sampling is an important methodological difference because of the marked and rapid episodic fluctuations in hGH concentrations (Quabbe et al, 1966; Glick et al, 1965). Consequently, results from less frequent sampling are more likely to be biased by such intrinsic variations. Moreover, the frequency of major hGH release episodes appears to be diminished during fed wakefulness, and a circhoral[70] pattern in wakeful hGH can be induced by fasting (Goldsmith and Glick, 1970; Parker et al, 1972; Parker and Rossman, 1973). Therefore, a combination of both fasting and relatively infrequent sampling might explain the discrepant hGH findings.

The use of fasting subjects has the advantage of ensuring observation of basal hGH levels because of meal-related hGH elevations

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69. Arterial catheterisation was presumably used to enable simultaneous invasive cardiovascular measurements (Jevning et al, 1978c).

70. This term, which is derived from circa (about) and hora (hour), refers to oscillations with periods of approximately one hour (Diershke et al, 1970).

(Sukkar et al, 1967; Catt, 1970). However, fasting might interfere with the efficacy of meditation and standardisation of meal times should make any effects of feeding consistent between groups. Fenwick et al (1977) reported significant reductions of metabolic rate for non-fasted subjects in contrast to no significant change for fasted subjects. The difference between the two groups was explained in terms of the law of initial values, pre-treatment oxygen consumption of the unfasted subjects being higher than the pre-treatment levels of oxygen consumption observed in the fasted state. The initial hGH levels reported by Jevning et al (1978b) were, however, slightly higher, rather than lower, than those of group 1A in which a marked 44 percent reduction in hGH was observed during meditation. The rebound of hGH levels following meditation in both group 1A and non-stressed meditators provides further evidence for a specific TM-induced reduction. Assuming a 25 minute half-life for circulating hGH[71] (Glick et al, 1964; Hunter and Greenwood, 1964), a 44 percent hGH reduction in 30 minutes represents complete inhibition of hGH secretion during the meditation period. If complete quiescence of hGH secretion occurred for 30 minutes, serum hGH levels would fall to $C_o^{\ln 0.5 \times \frac{30}{25}}$[72] $= C_o \times 0.5$; that is a 40 percent decrease for group 1A[73].

The following observations support the interpretation of hGH reduction in anticipation of meditation:- 1) The reduction in hGH before the onset of meditation which was sustained throughout the

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71. 100 percent clearance with a single passage through the liver and first order kinetics are also assumed.

72. Equation derived from standard kinetics equations:-
$C = C_o . e^{-Kel.t}$ and $t_{\frac{1}{2}} = \frac{\log_e 2}{Kel}$
(Avery, 1976; pp 4 and 5, respectively).

73. All four pre-meditation levels (ie samples 2, 3, 4 and 5) were used in this calculation because of the observed hGH reduction in anticipation of meditation.

meditation period (group 1A); 2) The significantly lower pre-meditation (group 1A) than pre-quiet rest (group 1B) serum hGH levels; 3) Rebound of hGH following meditation (group 1A); and 4) The lack of difference between post-treatment hGH levels of groups 1A and 1B. There do not appear to be any other reports of physiological changes in anticipation of meditation, although physiological changes characteristic of a TM-induced state have been found during eyes-closed rest in meditators (Wallace et al, 1971; Banquet, 1973; Kanellakos and Lucas, 1974; Williams and West, 1975). The fall of hGH in anticipation of meditation provides evidence for the notion of TM being a conditioned physiological state[74].

Such physiological preparation for meditation is parallelled by the changes which occur shortly before habitual sleep onset (Snyder, 1971; p 526). Conditioned endocrine changes have been found for anti-diuretic hormone (ADH) (Hofer et al, 1963) and conditioned changes in exocrine gland excretion have also been reported (Miller, 1969). Furthermore, expectation was found to have a significant influence over responsiveness to adrenaline administration (Penick and Fisher, 1965). All subjects in group 1A expected to meditate on the first occasion studied, whereas when re-studied (group 1B), they know beforehand that they would simply be resting instead of meditating. Subjects in group 3 were uncertain as to whether they would be meditating or not, until the pre-treaement period. Therefore, the initially high cortisol, hGH and prolactin levels for group 3 might be associated with an uncertainty effect. Indeed, Wadeson et al (1963) found elevated plasma 17-hydroxy-corticosteroid levels during an ambiguous pre-treatment period while subjects waited to view an unknown film.

The apparently triphasic hGH response pattern in meditation,

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74. Conditioning of a relaxation response with regular long-term practice is the common experience of meditators, particularly if they are accustomed to meditating at the same time each day (personal observation, A. Bevan).

involving an initial reduction in anticipation of meditation (phase 1), sustained reduction during meditation (phase 2), and a return to basal levels afterwards (phase 3), is the inverse of that seen in anticipated acute stress (Goleman and Schwartz, 1976). In order to examine purely TM-induced hGH changes, that is phase 2 changes, single blind investigations, using subjects with no foreknowledge of treatment type (TM or quiet rest), are indicated. Double-blind studies would be advantageous for also excluding possible influences of experimenter-subject interaction (Greene et al, 1970; Kurokawa et al, 1977). However, a blind experimental design is likely to introduce an uncertainty effect with concomitant hormone elevations. The subsequent temporal return of hormone levels to basal values would make accurate observation of treatment-specific changes impossible. Consequently, the only way to gain a true picture of TM-induced changes is to replicate studies, using both subjects with and subjects without foreknowledge of the type of treatment to be used. In order to exclude possible influences of novelty and ordering effects, subjects should be randomly assigned for study on say four different occasions, using treatment order alternation.

(c) Prolactin

The slight but statistically significant reduction in serum prolactin during TM, in contrast to no significant change during quiet rest in meditators or ordinary relaxation in non-meditators, suggests that the decrease observed in prolactin levels is also specific to the TM-induced state. These findings conflict with the report by Jevning et al (1978b) in which no significant change during meditation, and a significant rise following meditation, were observed in both novice and experienced meditators. The methodological differences and possible reasons for these discrepant findings are probably similar to those

discussed earlier for hGH (p 105). A further methodological difference, which seems unimportant from first impressions, is the 40 minute meditation treatment used by Jevning et al (1978b), in contrast to the 30 minute treatment used here. It is feasible that the longer 40 minute treatment period would induce more sleep than a shorter 30 minute treatment. Prolactin rises occur a minimum of 60 minutes after sleep onset[75] (Sassin et al, 1972) and with more than half an hour of continuous sleep (Parker et al, 1973). Although similar amounts of EEG recorded sleep occurred in both meditators and non-meditator controls (Jevning et al, 1978b), regular meditation also reduces time to sleep onset (Miskiman, 1977b; 1977c). Therefore, the post-meditation prolactin rise observed by Jevning et al (1978b) might have been triggered by early sleep onset during meditation. Indeed, the prolactin response pattern reported by Jevning et al (1978b) is remarkably similar to that observed by Parker et al (1973) in day-time napping.

(d) Thyroid hormones

The extremely long half-life of circulating T_4 means that short-term T_4 reductions are very unlikely. Assuming a T_4 half-life of 6.5 days[76] (Ingbar and Woeber, 1974; p 110), complete cessation of T_4 secretion for the whole, 120 minute, experimental period would result in only a one percent decrease[77]. Therefore, it is difficult to make conclusions about possible short-term reduction in thyroidal secretion during TM or other transient states of relaxation. Reciprocal changes in rT_3 as compared with T_3 during TM, along with reductions in T_4

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75. There is no apparent synchronisation of sleep-induced prolactin and hGH rises (Parker et al, 1973).

76. First order kinetics and 100 percent clearance with a single passage through the liver are also assumed.

77. Calculation based on pre-treatment levels of meditator and non-meditator groups. The standard kinetics equations given in foot-note 72 (p 106) were used.

and FTI, suggest that the observed thyroid changes were mediated by changes in peripheral mono-deiodination of T_4 (Chopra et al, 1975; Burr et al, 1975). However, the lack of reciprocity of T_3 and rT_3 changes, both following meditation and during ordinary relaxation, are difficult to explain. The absence of T_4 changes following ordinary relaxation for the non-meditator controls implies that the significant rT_3 increase and slight T_3 rise were not due to a haemoconcentration effect. The differing thyroidal responses to laboratory investigation of trained meditators and non-meditator controls, suggests distinctive thyroid activity for meditators; further evidence for which is suggested by basal differences in thyroid hormone tests (see p 119).

(e) Total urinary catecholamines

The lack of significant changes in total urinary catecholamines during TM is in agreement with the report by Michaels et al (1976) on plasma catecholamines, although adrenaline results from this study were inconclusive because of limited assay sensitivity. More recently, Lang et al (1979) found a significant increase in urinary catecholamines following TM of advanced meditators in contrast to no significant change for less experienced long-term TM practitioners.

The findings of neither significant UCA changes during the TM residence course nor blood pressure changes following TM provide corroborative evidence for a lack of TM-induced effects on sympatho-adrenomedullary activity. Moreover, the observations of both a significant TM-induced heart rate reduction and a lack of significant catecholamine change suggest that there may be a reduction of sympathetic nervous function in TM which is independent of changes in adrenomedullary activity. Dissociation of heart rate and catecholamine changes has previously been reported in psychogenic stress (Carruthers

et al, 1973). Reduced sympathetic nervous activity in meditation was originally proposed by Gellhorn and Kiely (1972) as part of their "trophotropic - ergotropic" hypothesis. Benson et al (1974a) extrapolated the hypothesis to relaxation procedures in general. However, as described earlier (pp 11-16), the subject of sympatho-adrenomedullary changes in TM and other relaxation procedures is controversial, and there is relatively little supportive evidence of reduced sympatho-adrenomedullary activity from studies of healthy, normotensive subjects.

Although noradrenaline changes provide a sensitive index of sympathetic nervous function (Lake et al, 1976), urinary catecholamine determination might not be sensitive enough for detecting rapid changes in catecholamine levels, and the possibility of different catecholamine changes remains[78]. Replication studies on healthy subjects, using suitably sensitive plasma catecholamine assays, are required in order to dispel any controversy about changes of sympatho-adrenomedullary activity in relaxation procedures.

(f) General discussion

(i) Influence of independent variables

Experimental stress-induced cortisol and hGH elevations posed a major methodological problem, especially because of the marked difference in cortisol-stress responsiveness between meditators and non-meditator controls. The significant reduction of serum cortisol levels of the unselected non-meditator control group during the afternoon study probably represents temporal decline from the initially

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78. Indeed, there is evidence which also demonstrates the need for considering both catecholamine precursors and metabolites in order to elucidate the characteristic sympatho-adrenomedullary function of various psychogenically-induced states (Bolshakova, 1976).

high pre-treatment stressed levels. The lack of significant change in non-stress cortisol levels of control subjects provides confirmatory evidence that the significant reduction observed in the unselected subjects was artefactual to the experimental conditions, rather than an effect of ordinary relaxation.

The lack of significant changes in both PCV and total serum proteins during meditation and also quiet rest in meditators demonstrates that the observed hormone reductions in TM were not due to haemodilution effects. Although a statistically significant increase in total serum protein was observed following ordinary relaxation in non-meditators, it amounted to only a 3 percent change, and was not considered of physiological significance.

Jevning et al (1978c) have reported reduced liver and kidney blood flow in TM which might decrease hormone clearance rates during meditation. Such an effect would give greater confidence to the observation of significant hormone reductions in meditation. The possibility of changes in hormone half-lives during TM remains to be investigated.

The absence of significant serum hormone changes of meditators under quiet rest conditions on both the first and second occasions studied, demonstrates that the observed hormone reductions in TM were not due to a first experience effect. However, the possibility of an effect of treatment order on endocrine changes remains to be investigated.

It is important to note that because of the time-lag between hormone secretion and appearance in the urine (Migeon et al, 1956), the pre-laboratory urinary levels would not reflect cannulation-induced hormone changes. Pre-laboratory levels would, however, show any apprehension or anticipation effects on cortisol and catecholamines (Davis et al, 1962; Frankenhaeuser and Rissler, 1970). Therefore, the

significantly higher pre-laboratory UFC levels, along with slightly higher UCA excretion, on the first occasion (group 1A) than on second occasion (group 1B) studied, were probably due to a first experience pre-experimental stress apprehension effect (Fishman et al, 1962a; Davis et al, 1962; Frankenhaeuser and Rissler, 1970). The marked first experience UFC effect was unexpected and makes differences between UFC changes in TM (group 1A) and quiet rest (group 1B) difficult to interpret. The significantly higher 24 hour UCA and slightly higher daily UFC excretion on the first occasion (group 1A) than on the second occasion (group 1B) provide corroborative evidence for a marked novelty-apprehension effect. Hence, a complete mock study, rather than just a preliminary meeting with trial cannulation, is apparently required in order to avoid such effects.

Increased urine flow and creatinine clearance have previously been reported in various forms of psychogenic stress, including apprehension (Levi, 1972; p 67 and p 82; Frankenhaeuser and Rissler, 1970). The significant reduction in serum osmolality observed during laboratory quiet rest for both first and second experience groups, in contrast to no significant change for first experience subjects during laboratory TM, suggests that TM might influence ADH secretion. However, meditation does not appear to be counteractive to experimental stress-induced ADH changes because stress increases ADH levels (Johnston, 1975), and increased rather than decreased osmolality would therefore be expected under non-meditation control conditions. Interpretation of these findings is confounded by experimental stress-induced changes and also possible interactions between catecholamines and renal function (Levi, 1972; p 33).

The lack of physical activity during the laboratory study, along with other possible differences in amount and severity of physical exercise on the experimental and control week-ends, probably explains

the higher catecholamine excretion of non-meditators on the control Saturday than on the experimental Saturday (Kotchen et al, 1971; Frankenhaeuser et al, 1976; Nilsonn et al, 1975). The contrasting lack of difference in control and experimental Saturday UCA levels of the meditators is probably related to group differences in physical exercise on each of the two week-ends. Catecholamines are relatively more sensitive to changes in physical activity (Hartley et al, 1972; Raymond et al, 1972; Nilsson et al, 1975), therefore, the lack of difference between experimental and control Saturday UFC levels of non-meditators is not necessarily incongruous with significant UCA differences. However, the combination of laboratory stress-induced cortisol and catecholamine increases on the experimental Saturday and exercise-induced hormone increases on the control Saturday makes these results difficult to interpret.

(ii) Are the observed hormone changes unique to TM?

There do not appear to be any other reports of hGH or prolactin reductions in psychogenic relaxation. Although hGH and prolactin reductions appear to be specific to the TM-induced state, it is not clear whether they are peculiar to TM or the result of regular induction of a state of deep relaxation. Further studies of hGH and prolactin in other forms of psychogenic relaxation are required in order to determine the uniqueness of the changes observed in TM.

The decreased hGH and prolactin levels during the TM-wake cycle contrast distinctly with those seen in the sleep-wake cycle. A marked episode of hGH release occurs with the onset of slow wave sleep phases 3 and 4 (Takahashi et al, 1968; Parker et al, 1969) while prolactin peaks appear to occur with each successive period of slow wave sleep (Parker et al, 1974). Similar hGH and prolactin changes have been observed during shortened sleep periods and napping (Takahashi et

al, 1968; Alford et al, 1973; Parker et al, 1973). However, hGH and prolactin rises occur after a minimum of 30 and 60 minutes, respectively (Weitzman et al, 1974; Sassin et al, 1972), which are equal to or greater than the experimental meditation period used here. Therefore, further studies of meditators, comparing endocrine changes during napping with changes in meditation, are required in order to distinguish between hGH changes in TM and brief periods of sleep. Although there are no nyctohemeral or phase relationships between cortisol and sleep (Weitzman et al, 1970; Weitzman, 1972), slight reductions in cortisol and a period of relative quiescence in cortisol secretory activity occur around sleep onset and are sustained until the early morning circadian peak (Krieger et al, 1971; Weitzman et al, 1971; De Lacerda et al, 1973).

Controversy over the uniqueness of TM as a discrete major state of consciousness, distinct from the waking, sleeping and dreaming states (Wallace, 1970a; Wallace et al, 1971), arose with findings of a high proportion of slow wave sleep during meditation[79] (Younger et al, 1975; Pagano et al, 1976; Fenwich et al, 1977). The hypnagogic state hypothesis was proposed to explain both the observed physiological changes and the benefits associated with regular meditation practice (Fenwick et al, 1977). Decreased hGH and prolactin levels in meditation might be a useful physiological index for dintinguishing the TM-induced state from sleep, although similar reductions might also occur in a prolonged hypnagogic state which does not descend into sleep phases 3 and 4[80] (Elson et al, 1977). This possibility could be investigated by

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79. Advances in coherence spectral array EEG analysis (Levine, 1976) have been recently applied to detailed studies of both intra- and inter-hemispheric phase coherence during TM and TM-sidhis (Levine et al, 1976; Orme-Johnson et al, 1977a) (See p 168). The TM program is characterised by increased phase coherence in contrast to decreased phase coherence in sleep (Kenellakos, 1978; Orme-Johnson et al, 1979).

80. Long periods of drowsiness in sleep phases 1 and 2 are not sufficient for hGH release (Sassin, 1972).

using the techniques of hypnagogic induction described by Bertini et al (1972).

The significant reductions of cortisol, hGH and prolactin, along with lack of change in catecholamines, observed in TM, would be expected to result in decreased serum glucose levels (Zilva and Pannall, 1979; p 182; Harper, 1975; p 495). However, no significant glucose changes occurred. Increased glucagon and/or adrenaline levels might explain this apparent anomaly (see Figure 1). Other studies of glucose changes in relaxation procedures in healthy subjects have produced conflicting findings (see pp 9-15).

I. Endocrine Changes During TM Residence Course

The significant decrease in UFC excretion of experienced meditators, in contrast to no significant change in novice meditators, over the week-end of a TM residence course, confirms that TM-induced cortisol reductions are peculiar to experienced meditators. The lack of significant UFC changes in experienced meditators studied during the first short-term study suggests that the week-end cortisol reduction was specific to the TM residence course. However, the fall in UFC might simply be the accumulated effect of repeated[81] TM-induced short-term reductions, which, as described earlier (p 98), are significant only for experienced meditators. Indeed, the overall pattern of UFC changes over the TM residence course is very similar to that observed for serum cortisol in TM. Significant cortisol changes over a week-end do not appear to have been previously reported, although lower urinary 17-hydroxycorticosteroid levels on Saturdays and Sundays than on week-days have been found in both humans and monkeys (Mason and Brady, 1965).

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81. It is important to note that subjects meditated 4 or 5 times per day during the residence course, whereas 2 meditations per day are usual.

Halberg et al (1965a) also reported lower human 17-ketosteroid excretion on week-ends than week-days, which was presumed to at least partially reflect diminished cortisol excretion. No significant differences between Saturday and Sunday hormone excretion were observed, however, and a sharp rise in hormone excretion occurred on Mondays in both studies (Mason and Brady, 1965; Halberg et al, 1965a).

The lack of significant changes in catecholamines, which are relatively more sensitive than cortisol to changes in physical activity (Hartley et al, 1972; Raymond et al, 1972; Nilsson et al, 1975), along with the persistence of the cortisol reduction into the following work-day Monday, demonstrates that the cortisol changes were not simply due to reduced physical activity.

Although less marked, the significant UFC reduction of advanced meditators (>5 years' experience), over the week-end of short-term study (2), demonstrates that cortisol reductions can also occur on a non-residence course week-end. However, the mean TM experience of the experienced meditators in the residence course study was at the lower end of the 2 to 5 year range, a TM experience category which showed neither significant UFC changes over the week-end of short-term study (2), nor significant serum cortisol changes during TM. Therefore, the observed cortisol reduction over the TM residence course was not simply due to effects of ordinary rest on experienced meditators, and the TM residence course appears to have accentuated cortisol reductions in moderately experienced subjects; in terms of cortisol changes, giving them the status of highly trained (advanced) meditators. Although the effect of the TM residence course in reducing cortisol excretion persisted into the following Monday, further studies involving continued measurements of changes during the following working week are necessary in order to determine how long-lasting the residence course-induced changes are.

J. Basal[82], Waking Endocrine Response Patterning of Meditators

The close similarity between independent variables of meditators and non-meditators excluded the possibility that any extraneous influences biased the basal endocrine findings. The lack of difference between the amount of sleep reported by meditators and non-meditators implies that the observed inter-group hormone differences were not due to a dissimilarity in quantity of sleep[83]. However, the possibility of differences in quality of sleep-related endocrine changes remains to be investigated (see also p 120 and p 131).

The significantly lower basal blood lactate and glucose levels of meditators than of non-meditator controls are possibly secondary to the significantly lower cortisol concentrations (Ganong, 1975; p 214), and may be associated with a basal reduction in anaerobic respiration. In this context, it is interesting to note that a greater increase in haemoglobin concentration was found in athletes who were practising TM than in non-meditator controls (Reddy et al, 1977). The lack of difference in pre-treatment plasma cortisol levels of trained meditators and untrained controls, in the first short-term study, is probably connected with the following methodological differences:- 1) The use of control subjects in the first short-term study who, as discussed earlier (p 97), were probably less affected by the experimental conditions; 2) Better matching of meditators and non-meditator controls in short-term study (2), and 3) The use of larger sample sizes[84] in the second of the short-term studies.

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82. It is important to note that the term "basal" may be a misnomer with respect to blood levels of stress-related hormones because of the stressful nature of the invasive sample collection (see also p 133).

83. Alterations in amount of nocturnal sleep have been reported during TM training (Banquet et al, 1977; Orme-Johnson et al, 1979).

84. Larger sample sizes would exclude a possible type 2 statistical error (Croxton and Cowden, 1965; p 639).

Although Jevning et al (1977) also found significantly lower basal plasma cortisol levels in experienced meditators than in non-meditator controls, other studies have not shown significant differences (Avorn and Benson, 1974; Jevning et al, 1978a). Studies by other workers of lactate levels in meditators have not revealed significantly lower basal values (Michaels et al, 1976; 1979; Jevning et al, 1978c). The levels found by Jevning et al (1978c) were, however, lower in meditators than non-meditators. Lower basal lactate levels and lactate variation in meditators are possibly secondary to the similar differences observed for serum cortisol (Ganong, 1975; p 264). There do not appear to be any previous reports of lower basal glucose levels in meditators than in non-meditator controls.

The significantly higher T_4, FTI and rT_3, with no significant difference in T_3, of meditators compared with non-meditators, suggests an increased thyroid secretion with selective increase in peripheral deiodination of T_4 to rT_3. The elevated thyroidal secretion might be mediated by the significantly higher catecholamine levels (Melander et al, 1975; 1976). The basal thyroid hormone differences between the two groups would produce little difference in metabolic rate because of the relatively small contribution of T_4, and lack of rT_3 contribution, to the calorigenic actions of thyroid hormones (Ganong, 1975; p 239). Furthermore, a selective increase in deiodination of T_4 to rT_3 might denote an adaptive mechanism whereby the relatively potent T_3 remains unchanged. Selectively increased deiodination of T_4 to rT_3, in preference to T_3, has been proposed as an adaptive mechanism in response to stress (Chopra et al, 1975). The significantly higher variance of FTI and rT_3 in meditators than in non-meditator controls is difficult to interpret, however, it is suggestive of greater lability of thyroidal secretion and peripheral deiodination of T_4 in meditators.

The higher total 24 hour urinary catecholamine levels observed in meditators than non-meditators are somewhat surprising, particularly in view of the popular theory of reduced sympathetic nervous function in relaxation procedures[85] (see p 111). However, maintenance of some activity in the ergotropic system, which is concerned with sympathetic nervous function (Gellhorn and Kiely, 1972), is demonstrated by failure to habituate to alpha-blocking during TM (Wallace, 1970b; Wallace et al, 1971). Indeed, the TM-induced state was originally characterised as a wakeful hypometrabolic state (Wallace, 1970a, Wallace et al, 1971) and TM appears to be a state of sustained attention (Williams and West, 1975). Furthermore EEG changes associated with increased wakefulness, have been found in TM (Banquet et al, 1974; Levine et al, 1977), while EEG evidence of heightened alertness in meditators during eyes-closed non-TM rest has been observed (Williams and West, 1975). There is also a report of increased amounts of light sleep (phases 1 and 2), along with decreased deep sleep (rapid eye movement and phase 4), in meditators (Banquet et al, 1977). The alerting influence of circulating catecholamines, acting via the reticular activating system (Rothballer, 1959), might at least partially be responsible for the maintenance and increases of alertness associated with TM. Schwartz (1975) proposed that meditation states might be characterised by dampened limbic function concomitant with increased cortical activity. The EEG synchronisation (that is, absence of EEG arousal), which is generally observed during TM, might be due to a simultaneous reduction of hypothalamo-cortical discharges (Gellhorn and Kiely, 1972).

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85. The possibility remains that the higher total catecholamine excretion of meditators is associated with some unidentified biogenic amine rather than noradrenaline and/or adrenaline.

The lack of consistency between the preliminary finding of no significant difference between catecholamine levels of meditators and non-meditators and the significant difference observed in later studies, is probably due to the larger sample sizes (see foot-note 84, p 118) and better matching of meditators and controls[86].

Johansson and Frankenhaueser (1973) observed that subjects with comparatively high basal adrenaline excretion performed better on a choice-reaction task and had lower neuroticism scores. More recently, a significant positive correlation was found between adrenaline excretion rate and performance efficiency (O'Hanlon and Beatty, 1976). Furthermore, significant negative correlation between noradrenaline excretion and covert anxiety (Markiewicz et al, 1973), as well as significant positive correlation between adrenaline excretion and ego strength (Roessler et al, 1967) have been found.

Elevated basal cortisol levels have been associated with anxiety in both psychiatric patients and healthy subjects (Fiorica and Muehl, 1962; Persky et al, 1968). Furthermore, increased cortisol levels are generally associated with states of non-specific affective arousal (Brown and Heninger, 1975; Pinter et al, 1975) and the maintenance of low levels appears to be indicative of effective ego defense mechanisms (Friedman et al, 1963; Wolff et al, 1964a, 1964b; Mason et al, 1965; Katz et al, 1970; Knight et al, 1979).

EEG correlates of development in ego strength (Van Der Berg and Mulder, 1977), improved academic performance (Collier, 1977; Heaton and Orme Johnson, 1977), increased intelligence (Tjoa, 1977a, 1977b), enhanced reaction time (Shaw and Kolb, 1977; Appelle and Oswald, 1977;

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86. Although the lack of significant inter-group differences in pre-relaxation blood pressure levels from the first short-term study may be suspected for similar reasons, adrenomedullary activation may occur independent of sympathetic nervous (and also adreno-cortical) activation (Von Euler et al, 1959).

Orme-Johnson et al, 1977b), and decreased neuroticism and anxiety, both of normal healthy subjects (Hjelle, 1974; Tjoa, 1977a, 1977b; Davies, 1977; Ross, 1977) and psychiatric patients (Glueck and Stroebel, 1975) have been associated with the TM program. Therefore, it seems likely that the increased basal catecholamine excretion and decreased basal cortisol levels of meditators are related to the above mentioned positive personality and behavioural changes of meditators. The unusual combination of low physiological activation accompanied by heightened subjective alertness in meditators is parallelled by the simultaneous occurrence of low basal cortisol along with high basal catecholamine levels[87]. Dissociation of catecholamine and cortisol elevations in stress have been reported (Von Euler et al, 1959; Friedman et al, 1960), however, there do not appear to be any other reports of naturally occurring healthy states in which opposite catecholamine and cortisol changes are found.

Orme-Johnson (1973) concluded that fewer spontaneous galvanic skin responses in meditators than non-meditator controls demonstrated greater stability of autonomic nervous function. Decreased temporal afternoon variation, and presumably greater stability, of serum cortisol and hGH might be similarly indicative of more stable hypothalamo-anterior pituitary function in meditators. The observation of comparatively low afternoon serum cortisol variation concurs with the report by Avorn and Benson (1974) of lower plasma cortisol day-to-day variation in meditators than non-meditator controls. Greater serum cortisol and hGH stability would involve changes in duration and/or number of secretory episodes and further studies, using smaller sample

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87. Feedback effects of circulating cortisol, T_4 and catecholamines on the central nervous system (Woodbury and Vernadakis, 1972) might be responsible for some of the perceptual and behavioural changes associated with TM (see also p 126).

intervals (Gallagher et al, 1973), are required to investigate these possibilities. The contrasting lack of inter-group differences in serum prolactin variation might reflect intrinsically fewer or smaller prolactin release episodes, or inherently less prolactin stress responsiveness than for hGH and cortisol. Comparatively less stress responsiveness for prolactin is also suggested by the relative lack of elevation of pre-treatment prolactin levels above the upper limit of the basal resting range.

Cortisol elevation concomitant with a lack of change in both hGH and prolactin have been found during affective arousal (Brown et al, 1975; Pinter et al, 1975). Furthermore, there does not appear to be a significant correlation of prolactin levels with anxiety (Mathew et al, 1979) whereas elevated cortisol levels have been associated with anxiety (Fiorica and Muehl, 1962; Persky et al, 1968). Therefore, the meditators' pre-treatment serum cortisol, hGH and prolactin levels observed here are probably associated with lower overall experimental stress-induced affective arousal.

K. State and Trait Endocrine Characteristics of TM

The slight reduction in serum cortisol during both TM (group 1A) and quiet rest (groups 1B and 3) in trained meditators suggests that decreased cortisol levels are not specific to a TM-induced state but occur also during quiet rest in meditators. Significant reductions in plasma cortisol have also been observed in subjects viewing bland films (Handlon et al, 1962; Wadeson et al, 1963), and it appears that focus on some internal or external non-affective stimulus might be the essential principle underlying psychogenic induction of decreased cortisol levels.

The significant reduction of UFC over an ordinary week-end[88]

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88. TM twice a day, as usual.

in advanced meditators[89] provides corroborative evidence for the non-specificity of the cortisol reductions. Reduced cortisol levels have been associated with easements in everyday life situations (Handlon, 1962), and it appears that advanced meditators also have a greater ability to relax outside the TM-induced state.

Significant negative correlation of both basal UFC and serum cortisol with TM experience, and the relationship of TM experience with both serum cortisol changes in meditation and week-end changes in UFC, suggest that both reduced cortisol-stress responsiveness and TM-induced cortisol changes are cumulative with increased TM experience. The mean TM experience of group 1A lies within the range 2 to 5 years, an experience category for which no significant cortisol changes were observed during meditation or over an ordinary week-end. Therefore, a TM experience effect explains the lack of significant serum cortisol reduction in group 1A and might also explain the absence of significant change during quiet rest.

A graded effect of meditation training on physiological changes was described by Kasamatsu and Hirai (1966) for the EEG changes observed in Zen monks. Recently, significant differences between catecholamine response patterns of advanced meditators and long-term meditators have been reported (Lang et al, 1979; see p 11), however, a significant relationship between TM experience and UCA excretion was

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89. The similar pattern of correlation in daily UFC and meditation experience from the Saturday to Sunday of both the experimental and control week-ends implies that the TM experience-related UFC changes observed over the experimental week-end were neither due to chance correlations, nor to more experienced meditators being relatively more cortisol stress reactive on the Saturday of the laboratory study. {Although contrary evidence has been found (see following discussion), experimental Saturday UFC excretion which was relatively high for advanced meditators could result in a significant Saturday to Sunday UFC reduction}.

not found here. Apart from the common discrimination between psycho-physiological changes in trainees (novices) and experienced subjects (Luthe, 1970; p 91 and 92; Kanellakos and Lucas, 1974; p 47 and p 89), meditation experience effects have been given surprisingly little attention by other workers. This may generally be accounted for by the observed lack of correlation between TM experience and meditation depth.

The negative correlation of TM experience and both basal serum cortisol levels and UFC excretion, implicates cortisol reduction as a trait characteristic of TM. The strengthening of the correlation during meditation suggests a state reinforcement of a trait characteristic. In this context, it is interesting to note that both trait and state anxiety have been reported to decrease with TM (Hjelle, 1974; Blackwell et al, 1976; Nidich et al, 1977; Davies 1977), and elevated cortisol levels have been associated with both state and trait anxiety (Fiorica and Muehl, 1962; Persky et al, 1968). The lack of significant correlation between meditation depth and serum cortisol levels, in contrast to significant correlation with hGH, might be due to relatively marked cortisol sensitivity to experimental disturbances. Further support for this suggestion is provided by the comparatively large positive correlation between experimental disturbance-involvement ratings and serum cortisol concentrations[90] during meditation. In fact, adreno-cortical activation, concomitant with no change in hGH, has been demonstrated in association with affective arousal (Brown and Heninger, 1975; Pinter et al, 1975). In view of the lack of association between stress-induced cortisol elevations and prolactin levels (see p 123), the observation of a significant positive correlation between TM-prolactin levels and both experimental stress ratings and TM-cortisol values is

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90. A significant positive correlation between plasma 17-hydroxy-corticosteroid levels and discomfort-involvement ratings has previously been reported in cardiac and pulmonary surgery patients (Price et al, 1957).

difficult to interpret.

The significant negative correlation of TM experience with TM-cortisol levels suggests that reduction in affective arousal during meditation is proportional to the amount of TM training. Moreover, the significant negative correlation of TM depth and experimental disburbance-involvement, along with the significant positive correlation of experimental disturbance and TM-cortisol levels, implies that successful meditation (as determined by depth ratings) is associated with a temporary state of analgesia, a state of unresponsiveness to extraneous disturbances in which affective arousal is absent[91].

TM experience effects on basal cortisol levels might reflect the alteration of perceptual and cognitive mechanisms which is associated with TM (Wandhoefer and Plattig, 1973; Kobal et al, 1975; Bennett and Trinder, 1977). Indeed, cortisol administration in man decreases selective attention to significant stimuli (Kopell et al, 1970), and thereby interferes with optimal integration of sensory information (Carpenter and Bunney, 1971). Furthermore an individual's perception and cognitive appraisal of a potentially stressful situation determines whether a stress response ensues (Hinkle, 1974; Lazarus, 1974).

The following observations provide evidence for serum hGH reduction being a state-specific characteristic of TM:- 1) Significant reduction of serum hGH in TM with subsequent rise, in contrast to no significant changes associated with ordinary relaxation in non-meditators or quiet rest in meditators; 2) No significant difference between basal serum hGH levels of meditators and non-meditators; 3) A significant negative correlation between serum hGH and depth of meditation; and 4) A lack of significant correlation between serum hGH and TM experience. Furthermore, lack of drop in cortisol and

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91. Alnaes (1966) reported a similar analgesic effect of autogenic training which was also associated with low cortisol levels.

prolactin levels to pre-meditation values, distinguishes hGH changes from other hormone changes in TM.

Although pre-meditation correlation of both prolactin and cortisol levels with TM experience was similar, significant prolactin correlation did not develop during meditation. The significant positive correlation between prolactin levels and experimental stress during meditation, along with lack of significant prolactin correlation with TM depth, demonstrates that prolactin is relatively more influenced by extraeous conditions and less susceptive to TM-induced reductions. Furthermore, prolactin levels fell only slightly during TM and levels also tended to fall during quiet rest in meditators. Therefore, although prolactin reduction is distinctly enhanced by TM, it cannot be clearly defined as either a state or trait characteristic.

Lack of changes in catecholamines during TM, along with higher basal excretion in meditators, suggests that increased catecholamine levels might be a trait-specific characteristic of TM. However, catecholamine changes are less well defined as a trait characteristic than cortisol, because of lack of correlation with TM experience. Although the finding of higher UCA levels in advanced meditators than in long-term meditators (Lang et al, 1979) was not confirmed here, it does provide corroborative evidence for an association between TM training and elevated catecholamine excretion.

L. Possible Neuroendocrine Mechanisms Underlying TM

The significant reduction of cortisol, growth hormone and prolactin, along with the lack of change in catecholamines during meditation, suggests that TM is associated with decreased anterior-hypothalamo-pituitary function and no change in posterior-hypothalamo-pituitary activity (Whybrow and Silberfarb, 1974). The lack of short-term changes in catecholamines during TM demonstrates that the

mechanisms underlying TM are not, as proposed by Benson et al (1974a), the simple opposite to the fight or flight stress response.

Since serotonin appears to have a stimulatory effect on GH, prolactin and ACTH secretion (Symthe, 1977), reduced hypothalamic serotoninergic activity might be responsible for the combined decrease of all three hormones in TM. Furthermore, because of the relatively large and TM-specific hGH reduction and the likely major serotoninergic control of hGH release (Smythe, 1977; Collu , 1977; p 54), a principal involvement of a serotoninergic mechanism is implicated in TM. This is consistent with the proposed involvement of serotoninergic activity in Hess's (1954; pp 26 and 60) trophotropic system (Kiely, 1974), reduced activity of which is popularly viewed as a neurophysiological mechanism underlying meditation (Gellhorn and Kiely, 1972; Benson et al, 1974a; Davidson, 1976).

The relatively small prolactin and cortisol, compared with hGH reductions in TM, implicates the involvement of other neuroendocrine mediators apart from serotonin. Moreover, the similar cortisol and prolactin response patterns in TM, along with the significant positive correlation between cortisol and prolactin levels during meditation, suggests similar neuroendocrine mechanisms for these two hormones. However, different neuroendocrine control mechanisms have been established for prolactin and cortisol secretion:- Noradrenaline appears to have an inhibitory effect on ACTH secretion (Ganong, 1972; Collu, 1977; p 55), whereas it seems to have a stimulatory effect on prolactin secretion (Smythe, 1977). Apparently, dopamine has an inhibitory influence over prolactin secretion, in contrast to a lack of effect on ACTH (Smythe, 1977). Moreover, the lower basal cortisol levels of meditators and the TM experience-cortisol relationships, in contrast to lack of both basal and meditation experience-related hGH and prolactin changes, suggests involvement of a separate neuroendocrine mechanism underlying

the cortisol changes. A combination of a slight short-term decrease in dopaminergic activity in TM, along with a long-term increase in basal noradrenergic activity which was both cumulative with increasing meditation experience and TM-reinforced, might provide a possible neuroendocrine basis to the prolactin and cortisol changes, respectively. Both noradrenaline and dopamine are intrinsic mediators of ergotropic system function (Kiely, 1974) and the proposed decrease in dopaminergic activity, along with a reciprocal increase in noradrenergic activity, implies a lack of change in ergotropic function in TM. Such a neurophysiological mechanism is consistent with the finding of no significant change in catecholamines during meditation.

Although some insights into possible neuroendocrine mechanisms underlying meditation have been gained, it appears that several different mechanisms might be acting simultaneously, and pharmacological studies using specific antagonists to serotonin and other neurotransmitter substances are required in order to further elucidate the neuroendocrine activities underlying TM. However, it is important to note that because "... a given neurotransmitter could produce totally divergent responses depending, for example, upon where it is released" (Cryer and Daughaday, 1977; p 253), it is inherently difficult to establish exact mechanisms in humans.

Studies of TM-induced endocrine changes in naturally occurring states of high prolactin secretion and high circulating sex steroids, during pregnancy and different phases of the menstrual cycle, respectively, might also provide further insights into underlying neuroendocrine mechanisms. Apart from a preliminary and rather inconclusive study of the effects of the endorphin blocker naloxone on TM of novice meditators, the subject of neuroendocrine mechanisms in meditation remains relatively unexplored. In view of the analgesic properties of endorphins (Miller and Cuatrecasas, 1978), it is

interesting to recall the earlier suggestion of an analgesic effect of meditation (p 126). In fact, research into possible endorphin changes in meditation or other specific states of deep relaxation might be a useful approach to providing an understanding of the neuroendocrine mechanisms underlying psychogenic relaxation.

M. Concluding Remarks with Directives for Future Research

The TM-induced reduction of cortisol, hGH and prolactin, three hormones associated with stress, appears to denote the removal of stress. Although the working hypothesis (p 9) may therefore be accepted in the particular case of TM, the use of highly trained subjects in further endocrine studies of other specific relaxation and meditation procedures is required in order to determine the uniqueness of the hGH and prolactin changes during TM. Furthermore, the use of other control treatments apart from reading and talking quietly should be considered. Listening to quiet music or viewing a bland film, for example, might produce more marked neuroendocrine relaxation, although no significant difference was found between galvanic skin response changes during reading quietly and listening to classical music (West, 1977).

These studies were performed mainly on young-adult male subjects and the possibility of age and/or sex differences in endocrinological effects of relaxation procedures remains to be investigated[92]. Studies during habitual nocturnal sleep are advantageous for investigation of basal endocrine changes because short-term influences of physical activity, eating and anxiety are

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92. In this context, it is worth noting that there is, for example, an apparent waning of sleep-related hGH release with aging (Carlson et al, 1972; Sassin, 1977), and sex difference in endocrine stress responses have been found (Frankenhaeuser et al, 1976; Weitzman, 1972; p 225; Quabbe et al, 1966; Frantz and Rabkin, 1965).

minimised (Friedman et al, 1975; Kalucy et al, 1976). Furthermore, studies of habitual sleep might also show differences between meditators and non-meditators in sleep-related secretion of such hormones as hGH and prolactin, as well as differences in diurnal rhythm of hormones like cortisol and catecholamines.

Although the control non-meditators used in the second short-term study were generally interested in TM, the possible effects of psychophysiological predisposition towards evoking endocrine changes requires further investigation. Furthermore, the experienced subjects, and particularly the advanced meditators in whom the most marked changes were observed, might constitute a select group whose predisposition towards continuing training (Otis, 1974) is responsible for their ability for psychogenic induction of endocrine changes such as those observed in these studies. If an influence of predisposition does exist, then doubt would be cast upon the universality of the ability to induce such changes and, therefore perhaps also, the general usefulness of relaxation procedures for controlling stress reactivity and treating stress-related diseases. Nevertheless, regardless of any predisposition effects, the unusual ability for voluntary psychogenic control over neuroendocrine function remains outstanding. Indeed, persons with such capabilities may even be at a selective advantage, particularly in a society where both the need for adaptation and adjustment to change is ever increasing, and phylogenetically ancient stress responses are often inappropriate (Hamburg, 1962)[93].

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93. In order that voluntary control over stress-related hormones might be of selective advantage, there must be significant fertility-reducing influences of stress on persons of reproductive age. Although unconfirmed by experimental evidence, fertility-reducing influences of emotional and mental stressors on human ovarian and endometrial functions have been suggested (Priedkalns, 1975). Furthermore, the reducing effect of psychogenic stress on androgen levels of males (see foot-note 1, p 2) might be associated with decreased sperm count and/or sperm motility. However, significantly reduced testosterone levels are apparently only associated with extreme oligospermia and sperm hypomotility (Bain, 1978), and there do not appear to be any reports of significant effects of stress on non-endocrine human male reproductive function.

Theoretically, a within-subject long-term experimental design is the best for studies on the effects of relaxation procedures, because not only does it exclude inter-individual variation, it also accounts for the possible influence of predisposition. Such an approach is suitable for measurement of both long-term basal changes and short-term changes during relaxation procedures. Jevning et al (1978a; 1978b) have applied it to short-term studies of endocrine changes during meditation, by re-studying untrained subjects after six months' regular TM practice. However, the inherent methodological problem of drop-out [94] and the possibility of predisposition towards continued training to a level of advanced proficiency, remain unaccounted for. Furthermore, other independent changes, which are difficult to control in human experimentation, are often concomitant with regular long-term practice. Changes in diet, drug-usage, sleep habits and quality of sleep[95], for example, might have a significant influence on endocrine function. Consequently, the confidence with which conclusions about the specific effects of relaxation procedures can be made, is essentially restricted.

Conditioning during training and associated effects upon motivation and expectation characteristics (Morse et al, 1977; Shapiro and Giber, 1978) are also difficult to control. Conditioning, along with other aspects of training in relaxation procedures, result in relaxation training by package (For example the TM Program; see p 168), rather than simply by instruction in a relaxation procedure on its own. The differentiation of active and essential components of relaxation procedures, as recently advocated in several review articles (Woolfolk, 1975; Davidson, 1976; Shapiro and Giber, 1978), might prove useful in discerning procedures which are especially appropriate for the treatment

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94. The drop-out problem might, at least partially, be overcome by paying subjects.

95. As previously mentioned (pp 30, 109 and 120, and foot-note 83, p 118) all of these independent variables have been reported to change during TM training.

of specific stress-related disorders[96] (Shapiro and Giber, 1978), and perhaps also, for specially selecting procedures which are inherently more suitable to particular individuals[97] (Schwartz, 1975). However, any attempts at isolating and studying individual components automatically change or even destroy the integrity, and perhaps functionality, of the procedure concerned. Meditation techniques like TM, which have an ancient origin in Eastern religious traditions, and which have been moulded by thousands of years of cultural evolution[98], are especially good examples. Aspects of such procedures, involving belief and contitioning, may indeed be essential to their efficacy.

Major advances to psychoendocrine research have resulted from improvements in methods of measuring blood biochemistry, particularly development of radio-ligand assays. However, there has been relatively little progress in the development of blood sampling procedures. Stress-induction by venepuncture, both in apprehension of and immediately after sampling, together with the possibility of continuing stressor effects associated with the presence of an indwelling catheter, means that true basal levels of any stress-related hormones are difficult to measure. Consequently, the further development and refinement of non-invasive methods for biochemical assay, such as application of an external light source to a lip or an ear lobe and examination of the absorption spectrum, are likely to be of great advantage in psychoendocrine research. Instantaneous monitoring of temporal endocrine changes would become possible and studies involving simultaneous endocrine, electrophysiological

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96. For example, combining muscle biofeedback with psychogenic relaxation training appears to be the most successful non-pharmacological mode of treating tension headache (Hutchings and Reinking, 1976).

97. Sittenfield et al (1976) have found evidence for the need to adapt EEG biofeedback training to the physiological characteristics of the individual.

98. TM is based upon Vedic teachings (Maharishi Mahesh Yogi, 1969).

and subjective measures would be made relatively easy. In fact, such intensive multivariate investigations are likely to provide the most useful approach to characterisation of relaxation procedures, particularly where paradoxical states of cerebrocortical-autonomic dissociation are concerned (Davidson, 1976; Corby et al, 1978).

APPENDIX I : HORMONE ASSAYS

A. Serum Cortisol and Urinary Free Cortisol (UFC) Competitive Protein - Binding Assay

REAGENTS:

(1) Phosphate buffer, 0.01 M, pH 7.4.

(2) Borate buffer, 0.1 M, pH 7.4.

(3) Bovine serum albumin (BSA), 0.1% in distilled water.

(4) Dichloromethane, Merck GR.

(5) Ethanol, Merck GR.

(6) Corticosteroid-binding globulin (CBG). Obtain plasma from women using oestrogen-containing contraceptives.

(7) Tracer, 1,2-^{3}H-cortisol, specific activity 40,000 µCi/m mol (Radiochemical Centre, Amersham). Dilute in ethanol to 5 µCi/ml. Store at -25^{o}C.

(8) CBG - tracer solution. 50 µl tracer/100 ml phosphate buffer, followed by 6 ml CBG plasma (6).

(9) Sephadex columns, 10 x 50 mm re-usable plastic columns of sephadex G25 in 0.01 M phosphate buffer.

(10) Charcoal solution, 1 g Norit A activated charcoal/100 ml ion-free water. Store at 4^{o}C.

(11) Scintillation cocktail, 6 g 2,5-diphenyloxazole (PPO), 1 l toluene (AR), 500 ml triton x 10 (AR).

(12) Standards, 1 m mol/l Sigma hydrocortisone (cortisol) in ethanol. Store at -25^{o}C.

METHOD:

(1) Serum - 1 ml sample plus 1 ml distilled water.
Urine - 2 ml sample.

(2) Extract with 10 ml dichloromethane by shaking 20 min. Remove aqueous layer by aspiration. Transfer 1.0 ml dichloromethane extract in duplicate to 13 cc 100 mm test-tubes.

(3) Standards - Prepare in duplicate 0, 10, 20, 40 and 60 n mol/l standards in 13 x 100 mm test-tubes.

(4) Evaporate standards and samples to dryness at 45°C under a gentle stream of air.

(5) Add 1.0 ml CBG-tracer solution. Incubate, 45°C, 5 min.

(6) Incubate, 4°C, 1 h.

(7) Two free/bound separation techniques were used; the latter providing greater technical ease.

(a) Gel filtration - Pre-wash columns with 25 ml distilled water. Add:- 4.0 ml 0.1 M borate buffer, drain; 0.2 ml 0.1% BSA, drain; 0.3 ml sample, drain; 0.5 ml 0.1 M borate buffer, drain; 1.0 ml 0.1 M borate buffer. Collect eluate in a scintillation counting vial.

(b) Charcoal - Add 50 µl charcoal, vortex, and allow to equilibrate 4°C, 20 min. Centrifuge 1000 g, 15 min, 4°C. Transfer 0.5 ml supernatant to a scintillation counting vial.

(8) Add 5.0 ml scintillation cocktail to all vials and count over 10 min.

(9) Calculate results as in section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of CBG with other steroids is summarised below:-

Compound	Cross-reactivity (%)
Cortisol	100
11-deoxycortisol	64
Deoxycorticosterone	55
Progesterone	37
Tetrahydrocortisol	11
Cortiscosterone	60
Cortisone	26
17-OH progesterone	79
Dehydroepiandrosterone	7.6
Spironlactone	0.86
Prednisone	5.8
Prednisolone	42.5
Fluorocortisone	1.5
Dexamethasone	1.6
Digoxin	0.9

(2) Precision:

	Within-assay	Between-assay
n	13	13
$\bar{x}$ (n mol/l)	333	333
SD (n mol/l)	23	30
CV (%)	7	9

(3) Normal Ranges:

a. *UFC* < 280 n mol/d

b. *Serum and plasma cortisol* < 280 n mol/l in day-active resting subjects during the afternoon.

B. Semi-automated Method for Measuring Total Urinary Catecholamines

I. Sample Preparation

REAGENTS AND MATERIALS:

(1) Ion free water (IFW), 4°C.

(2) Acetate buffer, 0.2 M.

(3) Acetate-di-sodium ethylenediaminetetra-acetic acid (EDTA) buffer, 10 g/l EDTA disodium AR/l 0.2 M acetate buffer, 4°C.

(4) Acetic acid, 0.5 M.

(5) Standards

a. *Stock* - 1028.5 µg Noradrenaline (NA) Hydrochloride/ 100 ml 0.1 M HCl (Radiochemical Centre, Sydney)

b. *Working* - Dilute stock in 0.5 M acetic acid to give: 125, 250, 500 and 1000 p mol/ml.

(6) Oxford Catecholamine Columns, Catalog No 331, each containing 2 g activated alumina.

METHOD:

(1) Add IFW to Oxford column until an air-bubble-free slurry has formed.

(2) Add 15 ml IFW, drain.

(3) Add 15 ml 0.2 M acetate-EDTA buffer, drain.

(4) Add 15 ml IFW, drain.

(5) Prepare samples. Include a column blank.

(i) 15 ml urine; add 1.0 ml 0.2 M acetate-EDTA.

(ii) pH to 8.4.

(iii) Add 50 µl 2.5 µCi/ml ^{3}H-NA to all samples.

(6) Add samples to columns.

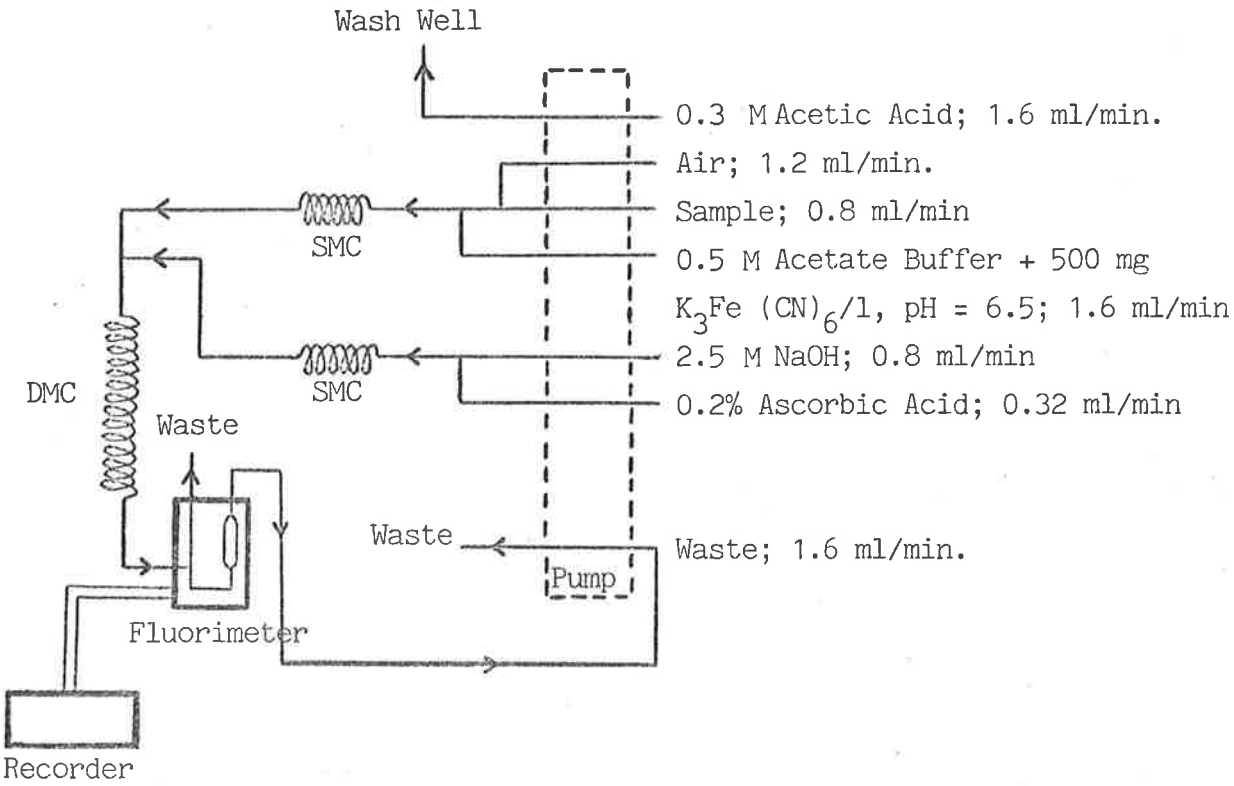
(7) Add 15 ml 0.2 M acetate-EDTA, drain.

(8) Add 15 ml IFW, drain.

(9) Add 10 ml 0.5 M acetic acid and retain eluate. Assay immediately or store eluates frozen. A 0.5 ml aliquot is added to scintillation cocktail and counted for radioactivity to determine recovery of each sample.

II. Automated Assay of Column Eluates

(1) Eluates and working standards were assayed using a Technicon Phase II Autoanalyser with Technicon Fluorimeter. A flow diagram showing manifold, concentration and flow rates of reagents is given below. Excitation and emission wavelengths were 410/510 nm. SMC and DMC refer to single and double mixing coils, respectively.



(2) Column blank is deducted from sample fluorescence, corrected for recovery[99], and total catecholamines expressed as noradrenaline standard.

ASSAY CHARACTERISTICS:

(1) Precision:

	Within-assay	Between-assay
n	15	12
$\bar{x}$	401	478
SD	53	72
CV	13%	15%

(2) Recovery: ^{3}H-NA - 70%

NA Standard - 85%

(3) Normal Range: < 550 n mol/d

C. Total Serum Thyroxine (T_4) Radioimmunoassay Using Charcoal Separation

REAGENTS:

(1) Barbital buffer, 0.1 M, pH 8.6.

(2) Barbital-BSA buffer, 0.1% Sigma bovine albumin in barbital buffer.

(3) 8-anilino-1-naphthalene sulphonic acid - sodium salt (ANS) solution (K and K Laboratories Inc, California). Prepare fresh 60 mg dissolved in 10 ml barbital-BSA buffer.

(4) Thyroid hormone free serum (THFS), 20%. Mix 100 ml pooled, normal, non-haemolysed serum with 10 g activated charcoal at 4°C overnight. Centrifuge 2500 g, ½ h. Centrifuge the supernatant for 90 min, 25,000 g, 4°C. Dilute 1:4 with HSA-phosphate buffer. Store at -70°C in 1 ml aliquots.

(5) T_4 antiserum. Antibodies were prepared in Australian rabbits against human thyroglobulin and used at a final dilution of 1:1000 (Chopra, 1971).

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99. Only the samples from short-term study (2) were corrected for recovery, because it was not the convention when the other studies were conducted.

(6) Tracer, ^{125}I-T_4, specific activity 200 µCi/ml (Amersham Co). Diluted with barbital-BSA buffer to 2 µCi/25 ml on day of use.

(7) Dextran coated charcoal, 150 mg dextran T_{70} (Pharmacia, Uppsala, Sweden) in 200 ml 1% BDH activated charcoal/0.1 m barbital buffer, pH 8.6.

(8) Standards, Sigma free-acid T_4.

a. *Stock* - 10 mg/100 ml barbital-buffer. Store frozen in 1 ml aliquots.

b. *Working* - Dilute stock in barbital-BSA to give:

0, 5, 10, 20, 40, 60, 100, 150, 200, 300, 400 n mol/l.

METHOD:

(1) Dilute test sera and T_4 free serum 1:4 in barbital-BSA buffer.

(2) Set up duplicate tubes as in table:

REAGENT	TOTAL	BLANK	ZERO	STANDARD	UNKNOWN
Barbital-BSA Buffer (ml)	0.9	0.6	0.4	0.3	0.4
ANS Solution (ml)	-	0.1	0.1	0.1	0.1
T_4 Free Serum (ml)	-	0.2	0.2	0.2	-
Standard (ml)	-	-	-	0.1	-
Unknown (ml)	-	-	-	-	0.2
Antiserum (ml)	-	-	0.2	0.2	0.2
Tracer (ml)	0.1	0.1	0.1	0.1	0.1

(3) Mix, incubate $4^{\circ}C$, 2 h.

(4) Add 500 µl dextran coated charcoal at $4^{\circ}C$ to each tube. Mix. The addition of charcoal and mixing should not take longer than 3 min.

(5) Incubate, room temperature, 5 min.

(6) Centrifuge 1500 g, room temperature, 10 min.

(7) Decant supernatant into counting tubes. Count to 10 K.

(8) Calculate results as in section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of T_4 antiserum with T_4 metabolities is summarised below:-

Compound	Cross-reactivity (%)
Thyroxine	100
Triiodothyronine	0
Monoiodothyronine (MIT)	0
Diiodothyronine (DIT)	0
Triiodothyroacetic acid (TRIAC)	6.5
Tetraiodothyroacetic acid (TETRAC)	21.7

(2) Precision: Approximately the same as for double antibody T_4 radioimmunoassay (see p 145).

(3) Normal Range: 71 - 150 n mol/l.

D. Total Serum Triiodothyronine (T_3) Radioimmunoassay Using Charcoal Separation

REAGENTS:

(1) Phosphate buffer, 0.1 M, pH 7.6.

(2) Human Serum albumin (HSA) - phosphate buffer, 0.25% HSA in 0.1 M phosphate buffer.

(3) Barbital buffer, 0.1 M, pH 8.6.

(4) BSA-barbital buffer, 0.1% BSA in barbital buffer.

(5) NaCl-HSA, 0.9% NaCl with 0.25% HSA.

(6) Tris-HCl-HSA-merthiolate, pH 9.2. Make up fresh 25 ml of 0.2 M Tris (Tris Hydroxymethyl) aminomethane, Trizma base, Sigma. Add 1.5 ml 0.2 M HCl. Mix, check pH 9.2. Add 1 ml 25% HSA. Mix gently. Add 0.1 g merthiolate (Thimerosal, Ethylmercurithiosalicylate). Make up to a final volume of 100 ml.

(7) THFS (see p 139).

(8) T_3 antiserum. Antibodies were prepared in Australian rabbits against T_3 conjugated to BSA (see p 153) and used at a final dilution of 1:5000 (Oliver et al, 1968).

(9) Tracer, ^{125}I-T_3 (Amersham Co). Diluted on day of use with Barbital-BSA buffer to give a working solution of 500 pg/ml.

(10) Dextran coated charcoal, 150 mg Dextran T_{70} (Pharmacia, Uppsola, Sweden) in 200 ml of 1% BDH activated charcoal/0.1 M phosphate buffer, pH 7.6.

(11) Standards - Sigma free-acid T_3

a. *Stock* - Dissolve 10 mg T_3 in a mixture of 1 ml NaOH and 2 ml propylene glycol/mg T_3. Dilute to 100 ml with barbital buffer. Store frozen in 1 ml aliquots.

b. *Working* - Dilute stock in NaCl-HSA to give:

0, 0.2, 0.5, 0.8, 1.3, 2.0, 3.0, 4.0, 8.0, 15.0 and 25.0 n mol/l.

METHOD:

(1) Dilute test sera and T_3 free serum 1:5 with Tris-HCl-HSA-Merthiolate buffer.

(2) Set up duplicate tubes (standards are set up in triplicate) as in table:-

REAGENT	TOTAL	BLANK	ZERO	STANDARD	UNKNOWN
HSA-Phosphate Buffer (ml)	0.9	0.7	0.5	0.3	0.5
T_3 Free Serum (ml)	-	0.2	0.2	0.2	-
Standard (ml)	-	-	-	0.2	-
Unknown (ml)	-	-	-	-	0.2
Antiserum (ml)	-	-	0.2	0.2	0.2
Tracer (ml)	0.1	0.1	0.1	0.1	0.1

(3) Mix, incubate $4^{o}C$, 24 h.

Steps (4) to (8) as for T_4 assay (Section C, p 140).

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of T_3 antiserum with T_3 metabolites is summarised below:-

Compound	Cross-reactivity (%)
T_3	100
T_4	0.32
TRIAC	26.6
TETRAC	0.37

(2) Precision: Approximately the same as for double antibody T_3 radioimmunoassay (see p 146).

(3) Normal Range: 0.8 - 2.1 n mol/l.

E. Double Antibody Total Thyroxine Radioimmunoassay

REAGENTS:

(1) Barbital buffer, 0.025 M, pH 8.6.

(2) BSA, CSL, 30%.

(3) Diluent buffer:- BSA-barbital buffer, 0.1% BSA in barbital buffer. Prepared daily.

(4) ANS, Sigma.

(5) THFS (see p 139).

(6) T_4 antiserum. Antibodies were prepared in Sheep number 357, Queen Elizabeth Hospital, and used at a final dilution of 1:7000 (see Section K, p 153 for Immunization Protocol).

(4) Tracer, ^{125}I-T_4 specific activity approximately 4 µCi/µg (Amersham Co). Diluted on day of use to give working solution of 5 ng/ml.

(8) Second antibody, donkey anti-sheep-goat (DAS), Wellcome RD39. Used at final dilution of 1:110.

(9) Non-immune sheep serum, normal sheep serum (NSS) used at a final dilution of 1:2000.

(10) Standards - Sigma free-acid T_4.

a. *Stock* - 31.075 mg T_4 is dissolved in a mixture of 3 ml absolute ethanol and 3 drops 2 N NaOH. Dilute to 100 ml with ethanol.

b. *Working* - Dilute stock with THFS to give:

0, 5, 10, 20, 40, 60, 100, 150, 200, 300, 400 n mol/l.

METHOD:

(1) Prepare Reagent I by mixing 40 mg ANS with 1.3 ml DAS, 5.3 ml tracer solution and 111 ml diluent buffer (sufficient for 100 tubes).

(2) Prepare Reagent II by adding 20 µl T_4 antibody and 70 µl NSS to 14 ml diluent buffer (sufficient for 100 tubes).

(3) To duplicate tubes add 0.9 ml Reagent I, 0.025 ml serum or standard, and 0.1 ml Reagent II.

(4) Mix, incubate 37°C, 2 h.

(5) Centrifuge 30 min, 4°C, 1000 g.

(6) Aspirate supernatant.

(7) Count precipitate to 10 K.

(8) Calculate results as in Section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of T_4 antiserum with T_4 metabolites is summarised below:-

Compound	Cross-reactivity (%)
T_4	100
T_3	2.8
MIT	0
DIT	0
TRIAC	1.3
TETRAC	8.3

(2) Precision:

	Within-assay	Between-assay
n	14	16
$\bar{x}$ (n mol/l)	116.8	116.2
SD (n mol/l)	4.1	7.35
CV (%)	3.5	6.3

(3) Normal Range: 71 - 144 n mol/l.

F. Double Antibody Total Triiodothyronine Radioimmunoassay

REAGENTS:

(1) Barbital buffer, 0.1 M, pH 8.6.

(2) BSA, CSL, 30%.

(3) Diluent buffer:- BSA-barbital buffer, 0.17 M BSA in barbital buffer. Prepared daily.

(4) ANS, Sigma.

(5) THFS (see p 139).

(6) T_3 antiserum. Antibodies were prepared in sheep number 204, Queen Elizabeth Hospital, and used at a final dilution of 1:70,000 (See Section K, p 153 for Immunization Protocol).

(7) Tracer, ^{125}I-T_3, specific activity approximately 500 µCi/µg (Abbott Laboratories). Diluted on day of use to give working solution of 300 pg/ml.

(8) Second antibody, DAS, Wellcome RD30. Used at final dilution of 1:150.

(9) NSS. Used at a final dilution of 1:5000.

(10) Standards, Sigma free-acid T_3

a. *Stock* - 1.628 mg T_3 is dissolved with 3 drops 2 N NaOH in 100 ml absolute ethanol.

b. *Working* - Dilute stock with THFS to give:

0, 0.2, 0.5, 0.8, 1.3, 2.0, 3.0, 4.0, 8.0, 15.0, 25.0 n mol/l.

METHOD:

(1) Prepare Reagent I by mixing 9.0 mg ANS with 1 ml DAS, 15 ml tracer solution and 104 ml diluent buffer (sufficient for 100 tubes).

(2) Prepare Reagent II by adding 10 µl T_3 antibody and 140 µl NSS to 70 ml diluent buffer. Store at 4°C until used.

(3) To duplicate tubes add 0.8 ml Reagent I, 0.1 ml serum or standard, and 0.1 ml Reagent II.

(4) Mix, incubate 4°C, 16 h.

(5) Centrifuge 30 min, 4°C, 1000 g.

(6) Aspirate supernatant.

(7) Count precipitate to 10 K.

(8) Calculate results as in Section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of T_3 antiserum with T_3 metabolites is summarised below:-

Compound	Cross-reactivity (%)
T_3	100
T_4	0.004
MIT	-
DIT	0.006
TRIAC	17.4
TETRAC	0.04

(2) Precision:

Within-assay and between-assay

n	approximately 14
$\bar{x}$ (n mol/l)	approximately 1.60
SD (n mol/l)	approximately 0.10
CV (%)	approximately 8

(3) Normal Range: 1.3 - 2.1 n mol/l.

G. Total Reverse Triiodothyronine (rT_3) Radioimmunoassay

REAGENTS:

(1) Phosphate buffer, 0.05 M, pH 7.4.

(2) HSA - Phosphate buffer, 0.25% HSA in phosphate buffer.

(3) Disodium EDTA - HSA - Phosphate buffer, 0.05 M disodium EDTA in HSA - Phosphate buffer.

(4) ANS, Sigma.

(5) THFS (see p 139).

(6) rT_3 antiserum. Antibodies were prepared in Australian rabbits against BSA - dl - rT_3 and used at a final dilution of 1:2000 (See Section K, p 153 for Immunization Protocol).

(7) Tracer, ^{125}I-rT_3. rT_3 label was prepared from diiodothyronine (Henning, Berling) using the chloramine T/sodium metabisulphite method (Greenwood et al, 1963), separating free from bound ^{125}I with sephadex LH-20 columns. The label, which is immunogenically stable for at least 6 weeks, is diluted on the day of use to give a working solution of 1.25×10^6 cpm.

(8) Goat Anti-rabbit Gamma Globulin (GARGG), Calbiochem Catalog No 539844 (125 U per vial).

(9) Non-immune rabbit serum, normal rabbit serum.

(10) rT_3 standards, Henning, Berlin

a. *Stock I* - 150 g rT_3 in 1 l of 0.025 M NaOH.

Stock II - Dilute Stock I with NaOH to give 1.5 g/l.

b. *Working* - Dilute Stock II with 20% THFS to give 0.02, 0.04, 0.08, 0.16, 0.32, 0.64, 1.28 and 2.56 n mol/l.

(11) T_4 standards. 100 n mol/l T_4 stock diluted with 20% THFS to give the following: 7.81, 15.63, 31.25, 62.5, 125, 250, and 500 n mol/l.

METHOD:

(1) Prepare tracer solution by dissolving 31.25 mg NAS with 250 µl normal rabbit serum and 8 µl rT_3 ^{125}I in a final volume of 50 ml HSA-phosphate buffer.

(2) Set up duplicate tubes as in table:-

REAGENT	TOTAL	BLANK	ZERO	STANDARD	UNKNOWN
HSA-Phosphate Buffer (ml)	-	0.1	-	-	0.4
Tracer (ml)	0.4	0.4	0.4	0.4	0.4
Standards (rT_3 or T_4[100]) (ml)	-	0.5	0.5	0.5	-
Unknown (ml)	-	-	-	-	0.1
Antiserum (ml)	-	-	0.1	0.1	0.1

(3) Mix, incubate 4°C, 24 h.

(4) Reconstitute a vial of GARGG with 12.5 ml EDTA-HSA-phosphate buffer and add 0.1 ml to each tube.

(5) Mix, incubate 4°C, 1 h.

(6) Centrifuge 30 min, 4°C, 3000 g.

(7) Carefully aspirate supernatant.

(8) Count to 10 K.

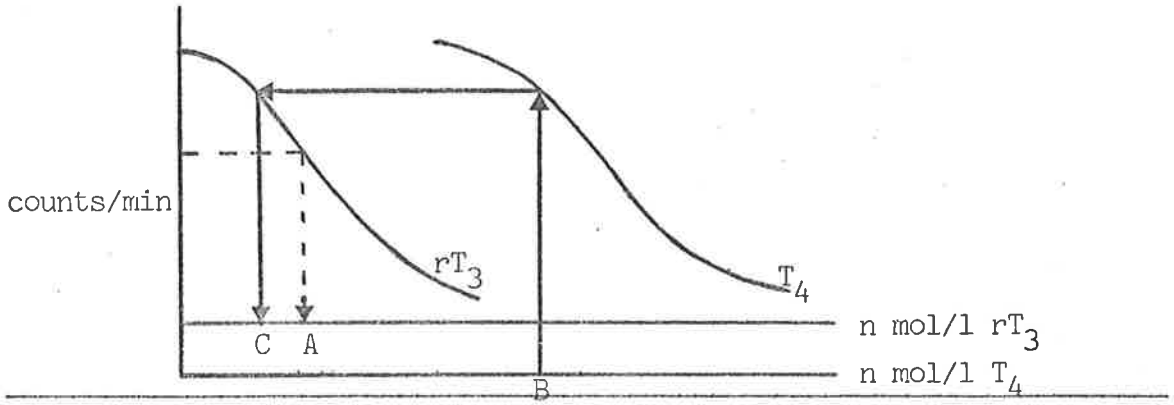
(9) Calculate results as in Section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

A correction for cross-reaction of rT_3 antiserum with T_4 is made using the equation:-

corrected rT_3 = (A - C) n mol/l, for a sample of known T_4 concentration B, and uncorrected rT_3 of A n mol/l. C is derived from extrapolation of B from T_4 standard curve onto rT_3 standard curve - see diagram below (semilog paper).



100. See rT_3 assay specificity on this page.

(2) Precision:

	Within-assay	Between-assay
n	12	20
$\bar{x}$ (n mol/l)	0.30	0.30
SD (n mol/l)	0.02	0.03
CV (%)	6.3	11.8

(3) Normal Range: 0.12 - 0.38 n mol/l.

H. T_3 Sephadex Uptake Test

REAGENTS

(1) Phosphate buffer, 67 ml, pH 7.5.

(2) Sephadex G25, medium or fine grain.

(3) ^{125}I-T_3, specific activity 40-50 mCi/mg (Abbott Laboratories).

METHOD:

(1) Prepare sephadex suspension by mixing 15 g sephadex and 5 µCi ^{125}I-T_3 in 100 ml phosphate buffer for 3-5 min.

(2) Dispense 5.2 ml suspension per counting tube.

(3) Add 50 µl test serum.

(4) Vortex and rotate tubes for 30 min.

(5) Allow particles to settle for at least 2 min.

(6) Count 1 ml supernatant.

(7) Calculate results as % of normal reference using the equation:-

Test % sephadex uptake =

$$\frac{\text{Hyland Unassayed (reference) Counts}}{\text{Test Counts}}$$

ASSAY CHARACTERISTICS:

(1) Precision:

	Within-assay and between-assay
n	approximately 12
$\bar{x}$	approximately 106
SD	approximately 2
CV (%)	approximately 3

(2) Normal Range: 89 - 111.

I. Human Growth Hormone (hGH) Radioimmunoassay

REAGENTS:

(1) Borate buffer, 0.13 M, pH 8.4, 0.5% BSA (provided in kit).

(2) HSA (provided in kit).

(3) hGH antiserum, raised in guinea pigs (provided in kit).

(4) Precipitating antiserum (provided in kit).

(5) Tracer, ^{125}I-hGH (provided in kit).

(6) Standards, Australian reference hGH preparation.

a. *Stock* - 200 ng/ml.

b. *Working* - Dilute stock with borate buffer to give 1.25, 2.5, 5.0, 10.0, 20.0 mU/l.

METHOD:

(1) Reconstitute borate buffer, hGH antiserum and HSA.

(2) Set up duplicate tubes as in table:-

REAGENT	TOTAL	BLANK	ZERO	STANDARD	UNKNOWN
Borate Buffer (ml)	-	0.2	0.1	-	-
Standard (ml)	-	-	-	0.1	-
Unknown (ml)	-	-	-	-	0.1
Antiserum (ml)	-	-	0.1	0.1	0.1
HSA (ml)	-	0.1	0.1	0.1	0.1

(3) Mix, incubate 37^{o}C, 6 h.

(4) Reconstitute ^{125}I-hGH and add 0.1 ml to each tube.

(5) Mix, incubate 37^{o}C, 18 h.

(6) Reconstitute precipitating antiserum and add 0.1 ml to all tubes except totals.

(7) Mix, incubate 37^{o}C, 1 h.

(8) Centrifuge 30 min, 4^{o}C, 4000 g.

(9) Decant carefully the supernatant.

(10) Add 0.5 ml borate buffer to each tube.

(11) Centrifuge 30 min, 4^{o}C, 4000 g.

(12) Decant carefully the supernatant.

(13) Count to 50 K.

(14) Calculate results as in Section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of hGH antiserum with other human peptide hormones is summarised below:-

Compound	Cross-reactivity (%)
hGH	100
Prolactin	1.0
Human placental lactogen	0.4
Follicle stimulating hormone	0.4
Luteinizing hormone	0.3
Human chorionic gonadotrophin	0.3
Thyroid stimulating hormone	0.1

(2) Precision:

	Within-assay	Between-assay
n	9	9
$\bar{x}$ (mU/l)	3.8	3.7
SD (mU/l)	0.4	0.5
CV (%)	11	14

(3) Normal Range: < 13 mU/l in day-active resting males.

J. Prolactin Radioimmunoassay

REAGENTS:

(1) Phosphate Buffered Saline (PBS) - Azide Buffer, 0.01 M, pH 7.4.

(2) Diluent Buffer, BSA-PBS-Azide buffer, 1% BSA in PBS-Azide buffer.

(3) Prolactin antiserum. Antibodies were prepared in rabbits against human prolactin (Calbiochem Catalog No 869041).

(4) Tracer, ^{125}I-Prolactin (CIS) is diluted to give a working solution of 10 µCi/ml.

(5) GARGG, Calbiochem Catalog No 539844 (125 U per vial).

(6) Standards, Calbiochem human prolactin.

a. *Stock* - 200 ng/ml.

b. *Working* - Dilute stock in distilled water to give:

50, 100, 200, 400, 800, 1600, 3200 mU/l.

METHOD:

(1) Prepare tracer solution by adding 200 µl normal rabbit serum and 100 µl working tracer to 9.7 ml diluent buffer.

(2) Set up duplicate tubes as in table:-

REAGENT	TOTAL	BLANK	ZERO	STANDARD	UNKNOWN
Diluent Buffer (ml)	-	0.5	0.45	0.4	0.4
Tracer Solution (ml)	0.1	0.1	0.1	0.1	0.1
Standard (ml)	-	-	-	0.5	-
Unknown (ml)	-	-	-	-	0.5
Antiserum (ml)	-	-	0.1	0.1	0.1

(3) Mix, incubate at room temperature, 72 h.

(4) Reconstitute a vial of GARGG with 12.5 ml of diluent buffer and add 0.1 ml to all tubes except totals.

(5) Mix, incubate at room temperature, 24 h.

(6) Add 2 ml PBS-Azide buffer and spin at 3000 g for 30 min, 4°C.

(7) Aspirate leaving 100-200 µl in tube.

(8) Repeat (6) and (7) above.

(9) Count to 50 K.

(10) Calculate results as in Section L, p 153.

ASSAY CHARACTERISTICS:

(1) Specificity:

The cross-reactivity of prolactin antiserum with other human peptide hormones is summarised below:-

Compound	Cross-reactivity (%)
Prolactin	100
Thyroid stimulating hormone	< 0.3
Luteinizing hormone	< 0.1
Follicle stimulating hormone	< 1.0
hGH	< 0.3

(2) Precision:

	Within-assay	Between-assay
n	9	8
$\bar{x}$ (mU/l)	150	142
SD (mU/l)	14	16
CV (%)	9	11

(3) Normal Range: 80 - 300 mU/l in day-active males.

K. Immunization Protocol for Production of Thyroid Hormone Antibodies in Sheep

(1) Prepare T_4 (T_3 or rT_3) - BSA conjugate by dialysing a suspension of BSA, carbodiimide (Sigma), and a tracer amount of T_4 (T_3 or rT_3) against distilled water, $4^{o}C$, 2-3 d {Ideal T_4 (T_3 or rT_3) - BSA substitution is about 25:1}.

(2) Emulsify 0.9% saline solution of 5 mg T_4 - BSA conjugate in 2 ml Freud's Complete Adjuvant.

(3) Inject intradermally at 6 - 8 sites on either side of sheep's spine.

(4) Follow 3 months later with a 2 ml booster injection of 1 mg conjugate in 0.7 ml saline emulsified with 1.3 ml Freud's Complete Adjuvant.

L. Calculation of Results from Competitive Protein - Binding Assays and Radioimmunoassays.

Results were calculated on a Wang 600 desk-top computer using the programme of Burger et al (1972). The programme fits the standard counts, using a least squares method, to the model:-

$$Y = \frac{A}{C + X^{E}} \pm \varepsilon$$

where Y = counts per 10 minutes; X = concentration of standard; A, C and E are constants; and ε is an estimate of the random error.

APPENDIX II : RELAXATION PROCEDURES

A. Basic Components

There are five essential components common to all relaxation procedures:- (1) eyes closed, (2) a comfortable posture, (3) a quiet environment, (4) a mental device and (5) a passive attitude (Benson et al, 1974a). Other components are belief, learning and habit formation.

(1) Eyes Closed

Closed eyes eliminate external visual distractive influences, thereby facilitating concentration on the mental device (component 4). Furthermore closing the eyes initiates relaxation of the occular muscles and is a preliminary step towards generalised reduction of muscle tonus (see component 2).

(2) Comfortable Posture

A comfortable posture should be adopted in order to minimise muscular activity. Proprioceptive stimuli are thereby reduced. Induction of relaxation further decreases muscle tonus (see p 7). Certain procedures, such as progressive relaxation, place special emphasis on muscle relaxation (Jacobson, 1938). An upright posture is recommended for most procedures because it reduces any tendancy towards falling asleep. Certain yoga sitting postures (eg siddhāsana: accomplished posture, and padmāsana: lotus posture) are designed especially for this purpose (Ornstein, 1972; p 108).

(3) Quiet Environment

A quiet environment prevents distraction from outside noises[101]. The desired overall effect of closing the eyes (component 1), adopting a comfortable posture (component 2), and a quiet environment (component 3)

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101. A quiet environment seems less important in more adept, experienced practitioners. Transcendental Meditation students are taught that noise is no barrier to meditation and meditators are known to practise while travelling on trains and in other public places.

is minimisation of exteroceptive stimuli, thereby facilitating concentration on the mental device (component 4)[102].

(4) Mental Device

The mental device is some constant stimulus which provides a focus for concentration. The most characteristic feature of a particular relaxation procedure is its mental device(s). The mental device(s) used is specific to each relaxation procedure. These devices may operate through any sensory modality. The auditory, visual and tactile modalities are the most commonly used. The sensory modality is generally internalised on a mental or ideational level. A prominent feature of most relaxation procedures is a tendancy towards sensory detatchment from the external environment[103]. Figure 36 contains details of the mental foci and associated sensory modalities used for each of the relaxation procedures which were investigated. Repetition is a very common principle involved in the application of mental devices to relaxation procedures. Following the breath, silently repeating a mantram, and repetition of suggestions are forms of constant stimulus repetition used in relaxation procedures. The mental foci in figure 36 which involve repetition are followed by an R in parentheses.

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102. Traditional avoidance of the post-prandial period for meditation practices, and the abstinence from the stimulants tea, coffee and tobacco, further minimise interfering stimulation and enhance the efficacy of meditation by facilitating attainment of a state of deep relaxation.

103. In this respect deep relaxation departs from the normal waking state of consciousness (see also pp 163 and 164).

FIGURE 36. The Mental Foci and Associated Sensory Modalities of the Relaxation Procedures which were Studied

RELAXATION PROCEDURE	METHOD	MENTAL FOCUS	SENSORY MODALITY
Transcendental Meditation	Maharishi Mahesh Yogi (1966;1969)	mantram (R)	auditory
Autohypnosis-relaxation	modification of Erickson's(1961) arm-levitation method	verbal suggest-ion(R)	auditory
		breath(R)	tactile,auditory
		eyelids,arm, face	tactile
Progressive relaxation[104]	modification of Jacobson's(1938) method	muscle tension	tactile,visual
		breath(R)	tactile,auditory
		verbal suggest-ion(R)	auditory
Yoga-meditation[104]			
(a) hatha-yogāsanas	traditional hatha-yoga (Iyengar,1975; Goswami,1959)	muscle tension	tactile
(b) prānāyāma		breath(R)	tactile,auditory
		nostrils,upper lip	visual

(5) Passive Attitude

A passive attitude requires that all distracting thoughts be regarded as insignificant. When awareness lapses from the mental focus, attention is gently returned to it. The functional counterpart of components 1 and 2 is passive concentration. "Passive" concentration is distinguishable from "active" concentration in that it involves no effort (Luthe and Schultz, 1969, Vol 1; p 14).

(6) Belief, Learning and Habit Formation

A certain amount of belief in the value of the relaxation procedure is required in order that the necessary passive attitude might be adopted. Learning and habit formation probably also play an important role in cultivation of the ability to readily induce relaxation. A cumulative conditioning of the ability to deeply relax probably results

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104. There is considerable overlap between the mental foci and sensory modalities of the progressive relaxation and yoga-meditation procedures.

from repeated association of a particular mental set (components 4 and 5) and setting (components 1, 2 and 3) with the activation of certain neural mechanisms; that is those of the relaxation response (Benson et al, 1974a) (see discussion on p 107 and foot-note 74).

B. Performance Instructions

(a) Transcendental Meditation

1) Sit quietly and comfortably with eyes closed.
2) Observe your thoughts[105] for several minutes.
3) Effortlessly replace the thoughts by silent repetition of the mantram (mantra).
4) Whenever you become aware of thoughts, passively return to repetition of the mantram. Don't force the mantram; don't resist thoughts.
5) To terminate meditation, stop repeating the mantram, wait several minutes, and then slowly open the eyes.

(b) Autohypnosis-relaxation

1) Focus on a spot on the ceiling with the eyes slightly strained but the neck relaxed.
2) Suggest to yourself that your eyelids are getting heavier and heavier, blinking more and more, closing and staying firmly closed. Keep giving yourself the suggestion even after they have closed.
3) Keep suggesting to yourself that, as your eyelids are closing, you are becoming more and more relaxed.
4) Focus on your breathing and suggest that the relaxation is spreading through all your body with each continuing breath.

---

105. Thoughts comprise all such cognitive activities as day-dreaming, dreaming, imagining, remembering, guessing, planning, understanding, and especially problem solving.

5) Focus on the right arm, suggesting it become lighter and lighter. This may take quite a lot of repeated suggestions, even up to 10 minutes, before levitation occurs[106].

6) Suggest to yourself that, as your arm becomes lighter, you are becoming even more relaxed.

7) Suggest that, as the weight flows out of your arm and it becomes lighter, it is floating towards your face and as it does so you become even more relaxed.

8) Suggest to yourself that when your hand touches your face, it will become heavy and fall, and as it does so you will become twice as deeply relaxed as you are now.

9) In this very relaxed state suggest to yourself that you will find it easier at every subsequent session to attain this state of trance and to go even more deeply into a trance state. Suggest to yourself that it will be easier and easier to return to this very relaxed state under even adverse conditions. Suggest to yourself that after a period of relaxation you will have an increased sense of well-being.

10) Suggest to yourself that when you count to 7 you will open your eyes on 7 and feel wide awake and alert, completely refreshed, with no feeling of drowsiness but bright, alert, cheerful and confident. Count to 7 and on 7 open your eyes.

(c) Progressive Relaxation

1) Close the eyes. Focus on the back of the neck. Visualise the shape of the neck and the impression that it makes in the chair.

2) Become aware of any physical sensations of either neck muscle tension or the actual physical contact of the neck on the chair.

---

106. Support for the right arm is important for easy application of the arm-levitation technique.

3) Focus on these sensations and allow the muscles in this area to become relaxed[107].

4) Following relaxation of the neck muscles take the attention to the face.

5) Visualise the face and focus on any muscular tension. Allow relaxation to spread to these muscles.

6) In a similar manner progress over the ventral part of the body, relaxing the shoulders, back, thighs, calves, feet, and toes in turn.

7) Then progress from the toes over the dorsal part of the body, relaxing the legs, abdomen and chest in turn.

8) On reaching the chest, focus on the natural breath. Breathe regularly and gently without forcing. With each exhalation your whole body will become more and more relaxed as tension flows out in phase with the flow of air. Feel the chest rise and fall with each inhalation and exhalation.

9) To terminate relaxation take several deep breaths and open the eyes. You will feel wide awake and ready to engage in activity.

(d) Yoga-meditation

The following programme of yoga-meditation was used by the yoga-meditation group during the Long-term Study and is representative of the programme used in Short-term Study (1). As well as the meditation periods indicated below, 30 to 60 seconds was spent in meditation between each yogāsana. The meditation involved passive concentration on bodily sensations and the natural breath (prānāyāma).

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107. Some muscle groups will take longer to relax than others simply because they are initially more tense. Other muscle groups will require little or no relaxation.

HATHA-YOGASANA		DESCRIPTION OR COMMENTS	DURATION OF PERFORMANCE
SANSKRIT NAME	COMMON NAME		
1) Savāsana	Corpse Posture		3 min
2) Utthita Dvipādāsana	Risen Leg Posture		45 sec
3) Utthita Tolāngulāsana	Risen Balance Posture	A simplified version was performed with legs straight	15 sec
4) Bhālaspristajāna Merudandāsana	Knee-touching Spine Posture	A simplified version was performed with hands on knees	2 min
5) Halāsana	Plough Posture		1 min
6) Savāsana	Corpe Posture		1 min
7) Bhujaṅgāsana	Serpent Posture		1 min
8) Dhanurāsana	Bow Posture		15 sec
9) Catuspādāsana	Quadruped Posture		30 sec
10) Sukhāsana or Padmāsana	Pleasant Posture or Lotus Posture	sitting and meditating	5 min
11) Parvātāsana	Mountain Posture	A simplified version was performed while sitting	2 min
12)	Neck Flexion - extension and Lateral Neck Flexion	Extend neck backwards and forwards; to left, then right	15 sec each way
13) Vāchā	speech	silent accentuation of the vowels A,E,I,O,U, in this order	3-4 times
14) Vakrāsana or Ardha Matsyendrāsana	Spine Twist Posture or Modified Spine Twist Posture		4 min each side
15) Sukhāsana or Padmāsana	Pleasant Posture or Lotus Posture	sitting and meditating	2 min
16) Jānuśirāsana	Head-knee Posture		2 min
17) Sukhāsana or Padmāsana	Pleasant Posture or Lotus Posture	sitting and meditating	5 min

HATHA-YOGASANA		DESCRIPTION OR COMMENTS	DURATION OF PERFORMANCE
SANSKRIT NAME	COMMON NAME		
18) Vrksāsana	Tree Posture		2 min
19) Pristhāsana	Back Posture	A simplified version was performed with hands to knees	15 sec
20) Pādahastāsana	Foot-hand Posture	A simplified version was performed with hands to floor	1 min
21) Bhūnamanāsana	Forward Head-bend Posture		1 min
22) Sukhāsana or Padmāsana	Pleasant Posture or Lotus Posture	sitting and meditating	1 min
23)	Massage	While sitting, massage back of neck, head and face	2 min
24) Sukhāsana or Padmāsana	Pleasant Posture or Lotus Posture	sitting and meditating	5 min

APPENDIX III : QUESTIONNAIRES

A. Assessment of Self-reported Subjective Changes

(a) Short-term Study (1)

The following patterns of response to relaxation have been adapted from the patterns of response to meditation which were first identified by Maupin (1965), and later used by Simmons (1973) and Roberts (1975) with practitioners of TM. Here, they have been slightly modified for the purpose of comparing various relaxation procedures.

(i) Instructions for scoring response to relaxation

The patterns outlined are guidelines by which you can rate the subject's reports. Subjects may experience several of these patterns in one relaxation session. In rating each report assign the rating of the highest level that the subject described in that report. For example, if a report described type 2, 3 and 4, assign to it a rating of 4.

(ii) Types of relaxation

Type 1. The subject reports difficulty in concentration and his remarks indicate that little else has occurred. He may report restlessness, boredom, unpleasant feelings or preoccupation with things happening in his surroundings.

Type 2. The identifying characteristic of these sessions is normal or ordinary experiences. The subject may report everyday thoughts and images, relaxation or sleepiness.

Type 3. The main feature of these sessions is "regressive" uncontrolled mental activity. Characteristically thoughts and images are dissociated and fragmentary and may involve "regressive material" (eg memories, " flashes" of inspiration). The subject does not follow or expand thoughts as in Type 2, but rather is passively aware of them coming and going. The subject may also report relaxation and at this stage the technique is easy and unconscious.

Type 4. In these sessions there is little mental activity. Relaxation, which is now very deep, seems to take the form of pleasant body sensation, which would be strange in ordinary states of waking consciousness. Sensations such as "floating" or "waves" are often reported. These sensations seem to reflect a growing detachment from normal waking consciousness. The subject is often aware of "deeper" levels of consciousness which is commonly associated with a feeling of pleasantness and calmness.

Type 5. The subject is unaware of his body and his surroundings. There is a feeling of detachment and non-striving. The technique is very easy. Mental activity is low. This is a state of consciousness different from normal waking states and different from sleep; calm, pleasant, and alert.

Type 6. This is the deepest level of consciousness where the subject reports no thoughts and no images, just blankness. There is a total detachment from body and surroundings. Subjects commonly express this state as "transcending" or "blankness", and altered states of consciousness. Alert or pleasant feeling may be reported. Following such a relaxation session beneficial after-effects are often reported.

(b) Short-term Study (2)

(i) Design and rating schemes of meditation - relaxation questionnaire

Part I:- Experimental stress (discomfort-involvement) was scored out of 5 using the rating scheme given in the following prototypal questionnaire.

Part II:- Depth of meditation or relaxation was scored out of 20 using the rating scheme given in the following prototypal questionnaire. Questions 1 to 4, inclusive, denote the amount of internal focus (Ornstein, 1972; p 107) as opposed to the amount of external intrusion (Van Nuys, 1971). Questions 5 to 10, inclusive, denote the amount of

general relaxation (Maupin, 1965). Questions 11 to 19, inclusive, pertain to hypnagogic state effects (Vogel et al, 1966; Oswald, 1966, pp 44 and 45; Foulkes and Vogel, 1965; Kleitman, 1963, pp 74 and 75; Davis et al, 1937) and are characteristic of alterations to the ordinary waking state of consciousness (Diekman, 1963; Maupin, 1965; Benson et al, 1974a; Davidson, 1976). Question 20 concerns muscle relaxation (Kleitman, 1963; pp 12 and 13).

(ii) Meditation-relaxation questionnaire

Code No.....

Please be honest in filling out this questionnaire. Please be sure to give an answer to every question.

Part I

The following questions apply to any stage during the 30 minute meditation or relaxation period. Please circle yes or no where applicable.

1. Were you aware of the presence of the cannula in your arm?	Yes	No
	1	0
2. Was the presence of the cannula disturbing?	Yes	No
	1	0
3. Where you aware of the blood sampling?	Yes	No
	1	0
4. Was the blood sampling disturbing?	Yes	No
	1	0
5. Did you experience any other physical discomfort?	Yes	No
	1	0

Part II

1. Were you aware of thoughts?	Yes	No
	1	0
2. Were you aware of images?	Yes	No
	1	0
3. Did noises distract you?	Yes	No
	0	1
4. Were noises disturbing?	Yes	No
	0	1
5. Did you feel rested?	Yes	No
	1	0

6. Do you feel rested now?	Yes	No
	1	0
7. Did you feel calm?	Yes	No
	1	0
8. Do you feel calm now?	Yes	No
	1	0
9. Do you feel tranquil?	Yes	No
	1	0
10. Do you feel tranquil now?	Yes	No
	1	0
11. Did time pass quickly?	Yes	No
	1	0
12. Did you lose awareness of your body?	Yes	No
	1	0
13. Did you lose awareness of the surroundings?	Yes	No
	1	0
14. Did you experience a drifting or floating sensation?	Yes	No
	1	0
15. Did you experience a falling sensation?	Yes	No
	1	0
16. Did you see inner light or experience a glowing sensation?	Yes	No
	1	0
17. Did you experience sensations like waves or vibrations?	Yes	No
	1	0
18. Did you experience any spontaneous bodily jerks?	Yes	No
	1	0
19. Did you experience a feeling of heaviness?	Yes	No
	1	0
20. Did your neck drop forward or to the side?	Yes	No
	1	0

B. Assessment of Vegetarian Status

(a) Rating Scheme

Diets were rated from the following questionnaire using the scheme shown in the table:-

DIET CATEGORY	PROTEIN SOURCE				DESCRIPTION
	Meat	Fish	Eggs	Milk and Cheese	
1	✓	✓	✓	✓	eats meat regularly
2		✓	✓	✓	eats fish regularly
3			✓	✓	oro-lacto vegetarian
4				✓	lacto-vegetarian
5					vegan

(b) Diet Questionnaire

Code No.....

Fill in the table provided on the next page with the approximate frequency with which you ate the following foods over the past one month. Please be honest and as accurate as possible in filling out this questionnaire.

A MEAT - including all meat products eg meat pie, fritz, salami, hamburgers etc.

B FISH - all types fresh, tinned, shellfish etc.

C CHEESE AND MILK PRODUCTS - do not count dash of milk in beverages, include cottage cheese, cream cheese, cheese pie, cheese cake, yoghurt, custard milk puddings, milk on cereal, milk drinks etc.

D EGGS - include egg custard, egg flan etc.

E NUTS - include nut rissoles, nutmeat etc.

F PULSES - include all dried peas, beans, lentiles, baked beans, bean salad, chick peas but not green beans.

G CEREALS - include breakfast cereals, bread, cake, biscuits, rice, pasta etc.

For example, for meat consumption:-

	everyday	more than 4 times per week	less than 3 times per week	occasionally	never
breakfast					A
morning snack					A
lunch				A	
afternoon snack					A
dinner			A		
evening snack				A	

N.B.: Letter A should appear once in each row of the table. There is one row for each meal: that is, a total of 6 rows.

	EVERDAY	MORE THAN 4 TIMES PER WEEK	3 OR LESS TIMES PER WEEK	OCCASIONALLY	NEVER
BREAKFAST					
MORNING SNACK					
LUNCH					
AFTERNOON SNACK					
DINNER					
EVENING SNACK					

N.B. Each letter, A to G, inclusive, should appear once in each row of of the table.

(c) Additional Questions

Has your eating pattern changed over the past one month?

.....

In what way has your eating pattern changed?

.....

.....

C. Subject Characterisation

(a) Meditation Experience

As well as basic TM instruction and regular, twice daily practice, the TM Program provides advanced meditation techniques and TM residence courses[108]. Therefore, questions 2, 4 and 5 from part II of the following questionnaire were collectively used to determine levels of meditation experience and thereby assign subjects to the three meditation experience categories described on p 85.

(b) Physical Training

An overall estimation of the amount of physical training (minutes per week) was made from part II of the following questionnaire using the formula:-

Answers to questions $[(c \times d) + \sum_{n=1}^{p} i\,(g + h) + \sum_{n=1}^{q} (l \times m)]$,

where p and q equal the number of sports and other professional or recreational physical exercises, respectively, practised at least once a week for more than one month. Special questions were included to determine the amount of physical activity derived from hatha-yoga because hatha-yoga is commonly practised by meditators.

(c) General Questionnaire

Code No.....

Please be honest in filling out this questionnaire. Please be sure to give an answer to every question.

Part I

1. What is your occupation? .....
2. How long have you been practising TM? ..... yr .... mo
3. How many times per week do you usually meditate? .....

---

108. Recently The TM-Sidhis (Orme-Johnson and Farrow, 1976) were added to the Program.

4. How many advanced TM techniques do you have? .....

5. On average, how many hours sleep per day do you usually have? .....

Part II

a. Do you practise hatha yoga? Yes No

b. How long have you been practising hatha yoga? .....yr ....mo

c. How many times per week do you usually practise hatha yoga? .....

d. About how long (in minutes) do you usually spend practising hatha yoga at each session? .....

e. Do you play sport? Yes No

f. How long have you been playing each sport?

Type of sport: ..... yr mo

.....

.....

g. How many times per week do you usually play each sport? times/wk

Type of sport: .....

.....

.....

h. How many times per week do you usually train for each sport? times/wk

Type of sport: .....

.....

.....

i. About how long (in minutes) do you usually spend playing or training for each sport each time? minutes

Type of sport: .....

.....

.....

j. Do you have other type(s) of professional or recreational physical exercise? Yes No

k. How long have you been practising each type of physical exercise? (years and months) .....yr....mo

Type of exercise: .....

.....

.....

l. How many times per week do you usually practice each type of exercise: times/wk

Type of exercise: .....

.....

.....

m. About how long (in minutes) do you usually spend practising each type of exercise? minutes

Type of exercise: .....

.....

.....

APPENDIX IV : RAW DATA

A. Long-term Study

(a) Subject Characteristics

Subject Code No	Relaxation Training	Relaxation Procedure	Sex	Age (yr)	TM Experience (yr)
1-80	Trained	TM	M	28.6	3.1
1-81	Trained	TM	M	20.0	1.0
1-84	Trained	TM	M	24.9	4.0
1-85	Trained	TM	M	22.4	3.0
1-88	Trained	TM	M	25.3	7.2
1-91	Trained	TM	M	26.3	3.3
1-92	Trained	TM	M	21.6	3.2
1-95	Trained	TM	M	20.3	2.0
1-5	Untrained	None	M	19.4	NAp
1-6	Untrained	None	M	18.3	NAp
1-8	Untrained	None	M	19.2	NAp
1-14	Untrained	None	M	25.3	NAp
1-18	Untrained	None	M	19.0	NAp
1-19	Untrained	None	M	19.6	NAp
1-23	Untrained	None	M	19.3	NAp
1-38	Untrained	None	M	22.2	NAp
1-58	Untrained	None	M	18.8	NAp
1-13	Untrained	None	F	19.2	NAp
1-15	Untrained	None	F	19.3	NAp
1-27	Untrained	None	F	21.5	NAp
1-31	Untrained	None	F	20.7	NAp
1-37	Untrained	None	F	18.7	NAp
1-67	In-training	TM	M	28.4	< 1
1-72	In-training	TM	M	19.0	< 1
1-83	In-training	TM	M	20.7	< 1
1-89	In-training	TM	M	29.3	< 1
1-90	In-training	TM	M	23.6	< 1
1-30	In-training	TM	F	18.3	< 1
1-52	In-training	TM	F	20.2	< 1
1-16	In-training	PR	M	20.8	NAp
1-49	In-training	PR	M	19.8	NAp
1-54	In-training	PR	M	19.9	NAp
1-56	In-training	PR	M	18.7	NAp
1-7	In-training	PR	F	18.0	NAp
1-20	In-training	PR	F	28.6	NAp
1-57	In-training	PR	F	18.4	NAp
1-21	In-training	AHR	F	39.8	NAp
1-47	In-training	AHR	F	18.1	NAp
1-66	In-training	YM	M	18.9	NAp
1-68	In-training	YM	M	21.2	NAp
1-69	In-training	YM	M	19.4	NAp
1-70	In-training	YM	M	20.8	NAp
1-77	In-training	YM	M	22.4	NAp
1-79	In-training	YM	M	18.0	NAp

(b) Serum Thyroxine Results (n mol/l)

Subject Code No	Relaxation Training	Relaxation Procedure	Apr	May	July	Sept	Nov
1-80	Trained	TM	90	138	94	98	NAv
1-81	Trained	TM	93	129	NAv	89	85*
1-84	Trained	TM	107	104	84	NAv	65
1-85	Trained	TM	119	98	NAv	83	NAv
1-88	Trained	TM	103	114	NAv	90	NAv
1-95	Trained	TM	93	65	77	79	NAv
1-6	Untrained	none	116	107	NAv	68	110*
1-8	Untrained	none	108	NAv	85	106	93*
1-18	Untrained	none	94	97	NAv	101	116*
1-19	Untrained	none	117	99	NAv	68	90*
1-23	Untrained	none	108	NAv	88	68	107*
1-38	Untrained	none	106	85	81	NAv	NAv
1-13	Untrained	none	65	94	80	107	97*
1-15	Untrained	none	77	99	NAv	75	103*
1-27	Untrained	none	81	86	90	67	NAv
1-31	Untrained	none	108	121	90	98	119*
1-37	Untrained	none	85	NAv	81	71	NAv
1-67	In-training	TM	94	77	83	NAv	101*
1-72	In-training	TM	103	97	NAv	92	72*
1-83	In-training	TM	119	104	NAv	97	NAv
1-89	In-training	TM	97	110	NAv	79	79*
1-90	In-training	TM	70	88	79	80	79*
1-30	In-training	TM	85	81	84	NAv	104*
1-52	In-training	TM	74	86	45	NAv	62*
1-16	In-training	PR	168	NAv	NAv	68	124*
1-49	In-training	PR	141	NAv	95	72	NAv
1-54	In-training	PR	98	108	90	NAv	NAv
1-56	In-training	PR	95	NAv	117	76	130*
1-7	In-training	PR	75	NAv	NAv	97	77*
1-20	In-training	PR	155	124	99	68	NAv
1-57	In-training	PR	95	79	NAv	80	NAv

* Results from samples collected during end-of-year exams.

(c) Serum Triiodothyronine Results (n mol/l)

Subject Code No	Relaxation Training	Relaxation Procedure	Apr	May	July	Sept	Nov
1-80	Trained	TM	2.8	2.6	2.2	1.9	NAv
1-81	Trained	TM	2.9	3.1	1.8	2.4	NAv
1-84	Trained	TM	3.7	3.1	2.0	NAv	NAv
1-85	Trained	TM	2.4	2.1	1.8	1.9	NAv
1-88	Trained	TM	2.1	2.9	3.0	2.8	NAv
1-95	Trained	TM	3.2	NAv	2.4	1.6	NAv
1-6	Untrained	none	3.1	2.6	2.6	2.4	1.8*
1-8	Untrained	none	2.4	NAv	2.6	3.0	1.6*
1-18	Untrained	none	2.8	1.9	2.5	2.1	3.2*
1-19	Untrained	none	2.9	NAv	2.4	2.0	2.3*
1-23	Untrained	none	NAv	2.4	2.2	3.5	2.2*
1-38	Untrained	none	2.1	2.4	2.3	NAv	NAv
1-13	Untrained	none	2.5	2.4	2.0	3.2	1.7*
1-15	Untrained	none	1.9	2.0	2.9	3.2	1.7*
1-27	Untrained	none	2.4	2.4	2.7	1.9	NAv
1-31	Untrained	none	2.4	NAv	2.1	2.2	2.1*
1-37	Untrained	none	2.8	NAv	2.1	2.4	NAv
1-67	In-training	TM	3.0	2.5	3.3	NAv	2.7*
1-72	In-training	TM	2.5	NAv	2.6	3.4	2.4*
1-83	In-training	TM	3.1	1.9	3.3	NAv	NAv
1-89	In-training	TM	2.0	2.6	2.6	3.2	2.5*
1-90	In-training	TM	2.8	NAv	2.4	2.1	3.1*
1-30	In-training	TM	2.8	3.1	2.2	NAv	2.4*
1-52	In-training	TM	2.4	2.7	NAv	2.3	3.1*
1-16	In-training	PR	2.6	NAv	2.7	3.0	1.5*
1-49	In-training	PR	2.9	NAv	3.8	3.0	NAv
1-54	In-training	PR	2.4	NAv	2.3	NAv	NAv
1-56	In-training	PR	2.2	NAv	1.9	1.7	2.8*
1-7	In-training	PR	2.7	NAv	NAv	2.4	2.0*
1-20	In-training	PR	2.8	NAv	1.9	3.0	2.2*
1-21	In-training	AHR	2.4	NAv	2.2	1.8	NAv

* Results from samples collected during end-of-year exams.

(d) Plasma Cortisol Results (n mol/l)

Subject Code No	Relaxation Training	Relaxation Procedure	Mar	Apr	May	July	Sept	Nov
1-80	Trained	TM	NAv	420	NAv	271	247	NAv
1-81	Trained	TM	NAv	283	NAv	227	219	225*
1-84	Trained	TM	NAv	345	NAv	262	NAv	116
1-85	Trained	TM	NAv	387	NAv	341	132	106*
1-88	Trained	TM	NAv	511	NAv	234	228	NAv
1-95	Trained	TM	NAv	304	NAv	213	86	187
1-6	Untrained	none	291	77	257	204	95	78*
1-8	Untrained	none	250	105	298	242	296	124*
1-18	Untrained	none	141	148	NAv	104	112	174*
1-19	Untrained	none	155	155	338	172	292	136*
1-23	Untrained	none	155	NAv	320	118	268	132*
1-38	Untrained	none	372	347	496	342	NAv	NAv
1-13	Untrained	none	197	186	NAv	275	211	118*
1-15	Untrained	none	292	NAv	250	204	349	196*
1-27	Untrained	none	219	104	295	139	150	NAv
1-31	Untrained	none	349	300	384	317	248	128*
1-37	Untrained	none	128	94	234	198	186	NAv
1-67	In-training	TM	NAv	286	236	NAv	215	77*
1-72	In-training	TM	NAv	221	214	144	192	85*
1-89	In-training	TM	NAv	330	NAv	197	126	NAv
1-90	In-training	TM	NAv	250	405	301	184	178*
1-30	In-training	TM	NAv	297	174	117	NAv	157*
1-52	In-training	TM	NAv	438	387	NAv	232	142
1-16	In-training	PR	257	163	185	255	159	223*
1-49	In-training	PR	290	352	207	141	155	NAv
1-54	In-training	PR	343	306	108	110	NAv	NAv
1-56	In-training	PR	294	193	291	206	933	135*
1-7	In-training	PR	178	NAv	84	NAv	407	161*
1-20	In-training	PR	519	174	149	76	240	193*
1-21	In-training	AHR	142	NAv	103	163	186	252*
1-66	In-training	YM	NAv	340	261	106	166	126*
1-68	In-training	YM	NAv	236	108	207	220	218*
1-69	In-training	YM	NAv	290	255	208	NAv	NAv
1-70	In-training	YM	NAv	411	378	273	NAv	NAv
1-77	In-training	YM	NAv	254	127	NAv	NAv	125*
1-79	In-training	YM	NAv	432	509	100	NAv	NAv

*Results from samples collected during end-of-year exams.

(e) Urinary Free Cortisol Results (n mol/d)

Subject Code No	Relaxation Training	Relaxation Procedure	Apr	May	July	Sept	Nov
1-80	Trained	TM	281	161	195	248	NAv
1-81	Trained	TM	122	75	101	181	88*
1-84	Trained	TM	228	145	115	137	142
1-85	Trained	TM	NAv	200	161	164	135*
1-88	Trained	TM	NAv	NAv	199	81	NAv
1-91	Trained	TM	116	127	182	124	128
1-92	Trained	TM	241	144	138	NAv	NAv
1-95	Trained	TM	193	NAv	127	159	NAv
1-5	Untrained	none	NAv	185	235	206	NAv
1-6	Untrained	none	NAv	NAv	272	190	208*
1-14	Untrained	none	NAv	151	179	NAv	NAv
1-18	Untrained	none	NAv	243	106	170	189*
1-19	Untrained	none	NAv	132	324	236	266*
1-23	Untrained	none	NAv	130	158	177	202*
1-58	Untrained	none	NAv	195	156	NAv	NAv
1-13	Untrained	none	NAv	147	115	136	196*
1-15	Untrained	none	NAv	106	137	156	134*
1-27	Untrained	none	NAv	135	205	186	NAv
1-67	In-training	TM	96	76	185	113	146*
1-89	In-training	TM	NAv	135	163	182	NAv
1-90	In-training	TM	451	328	312	270	262*
1-30	In-training	TM	139	114	126	171	145*
1-16	In-training	PR	NAv	139	228	NAv	NAv
1-49	In-training	PR	NAv	162	94	NAv	NAv
1-20	In-training	PR	159	NAv	145	192	195*
1-21	In-training	AHR	206	NAv	150	174	214*
1-47	In-training	AHR	172	76	117	77	NAv
1-68	In-training	YM	120	199	197	NAv	185*
1-70	In-training	YM	240	NAv	187	119	NAv
1-77	In-training	YM	127	149	201	NAv	213*
1-79	In-training	YM	192	NAv	NAv	135	199*

* Results from samples collected during end-of-year exams.

(f) Urinary Catecholamine Results (n mol/d)

Subject Code No	Relaxation Training	Relaxation Procedure	Apr	May	July	Sept	Nov
1-80	Trained	TM	584	426	295	203	NAv
1-81	Trained	TM	197	235	257	262	213*
1-84	Trained	TM	66	276	360	341	371
1-85	Trained	TM	NAv	205	137	180	109*
1-88	Trained	TM	NAv	NAv	456	186	NAv
1-91	Trained	TM	210	341	292	268	259
1-92	Trained	TM	150	276	240	NAv	NAv
1-95	Trained	TM	300	NAv	229	243	NAv
1-5	Untrained	none	NAv	374	380	NAv	NAv
1-6	Untrained	none	NAv	NAv	527	298	265*
1-14	Untrained	none	NAv	410	636	NAv	NAv
1-18	Untrained	none	NAv	407	262	292	251*
1-19	Untrained	none	NAv	303	333	238	404*
1-23	Untrained	none	NAv	311	442	336	377*
1-58	Untrained	none	NAv	281	306	NAv	NAv
1-13	Untrained	none	NAv	426	259	197	325*
1-15	Untrained	none	NAv	344	210	172	216*
1-27	Untrained	none	NAv	404	257	117	NAv
1-67	In-training	TM	NAv	322	308	401	470*
1-89	In-training	TM	NAv	729	NAv	145	NAv
1-90	In-training	TM	835	426	265	308	227*
1-30	In-training	TM	404	719	156	459	366*
1-16	In-training	PR	NAv	NAv	390	NAv	NAv
1-49	In-training	PR	NAv	300	349	NAv	NAv
1-20	In-training	PR	NAv	NAv	374	300	270*
1-21	In-training	AHR	NAv	NAv	554	513	360*
1-47	In-training	AHR	NAv	126	265	109	NAv
1-68	In-training	YM	573	NAv	341	NAv	268*
1-70	In-training	YM	NAv	NAv	385	191	NAv
1-77	In-training	YM	NAv	358	289	NAv	344*
1-79	In-training	YM	NAv	NAv	NAv	243	172

* Results from samples collected during end-of-year exams.

B. Short-term Study (1)

(a) Subject Characteristics

Subject Code	Relaxation Training	Relaxation Procedure	Sex	Age (yr)	TM Experience (yr)	Average Relaxation Depth Rating
1-81	Trained	TM	M	20.3	1.1	4
1-85	Trained	TM	M	22.8	3.2	2
1-88	Trained	TM	M	25.7	7.3	3
1-92	Trained	TM	M	21.9	3.3	5
1-95	Trained	TM	M	20.6	2.2	4
1-97	Trained	TM	M	24.8	4.4	4
1-98	Trained	TM	F	22.4	2.6	2
1-99	Trained	TM	M	24.3	5.1	3
1-6	Untrained	None	M	18.6	NAp	5
1-8	Untrained	None	M	19.5	NAp	4
1-14	Untrained	None	M	25.7	NAp	4
1-18	Untrained	None	M	19.3	NAp	6
1-19	Untrained	None	M	19.9	NAp	3
1-23	Untrained	None	M	19.7	NAp	1
1-67	In-training	TM	M	28.8	< 1	1
1-83	In-training	TM	M	21.0	< 1	3
1-89	In-training	TM	M	29.6	< 1	4
1-90	In-training	TM	M	23.9	< 1	4
1-39	In-training	PR	F	19.3	NAp	5
1-49	In-training	PR	M	20.3	NAp	3
1-43	In-training	AHR	M	20.3	NAp	4
1-44	In-training	AHR	F	18.8	NAp	2
1-75	In-training	YM	F	19.5	NAp	6
1-77	In-training	YM	M	22.8	NAp	5
1-79	In-training	YM	M	18.4	NAp	4
1-101	In-training	YM	M	23.9	NAp	5

(b) Cardiovascular and Blood Hormone Results at 1430 Hours

Subject Code No	Relaxation Training	Relaxation Procedure	Heart Rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Plasma Cortisol (n mol/l)	Serum T_4 (n mol/l)	Serum T_3 (n mol/l)
1-81	Trained	TM	64	115	90	66	83	1.8
1-85	Trained	TM	80	115	70	113	67	1.3
1-88	Trained	TM	80	135	90	138	84	2.2
1-92	Trained	TM	64	120	90	227	70	2.7
1-95	Trained	TM	68	125	85	227	93	2.2
1-97	Trained	TM	78	120	80	54	63	2.5
1-98	Trained	TM	82	130	90	188	93	2.1
1-99*	Trained	TM	74	120	70	148	98	1.4
1-6	Untrained	none	64	120	82	84	99	1.7
1-8	Untrained	none	84	125	80	85	98	2.7
1-14	Untrained	none	98	140	90	172	111	2.1
1-18	Untrained	none	76	130	90	89	95	2.0
1-19	Untrained	none	72	95	70	88	94	2.2
1-23	Untrained	none	80	125	85	76	97	3.3
1-67*	In-training	TM	70	130	90	172	90	2.2
1-83	In-training	TM	80	120	85	127	74	2.0
1-89	In-training	TM	68	95	70	211	89	3.5
1-90	In-training	TM	74	120	85	94	57	1.8
1-39	In-training	PR	82	130	90	184	99	1.6
1-49	In-training	PR	62	115	75	191	94	2.1
1-43	In-training	AHR	84	110	75	232	88	1.7
1-44	In-training	AHR	87	102	80	102	89	2.2
1-75†	In-training	YM	70	125	85	485	164	1.3
1-77	In-training	YM	60	130	90	71	123	2.8
1-79	In-training	YM	74	108	78	250	111	2.4
1-101	In-training	YM	84	130	85	226	106	1.7

* Subjects 99 and 67 were excluded from T_3 statistical analysis because only two T_3 results were available on each of them.

† Subject 75 was excluded from blood hormone statistical analysis because only the two pre-relaxation blood test results were available on her.

(c) Cardiovascular and Blood Hormone Results at 1530 Hours

Subject Code No	Relaxation Training	Relaxation Procedure	Heart Rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Plasma Cortisol (n mol/l)	Serum T_4 (n mol/l)	Serum T_3 (n mol/l)
1-81	Trained	TM	64	120	80	61	108	1.9
1-85	Trained	TM	72	120	90	127	74	2.7
1-88	Trained	TM	82	130	85	116	166	2.7
1-92	Trained	TM	70	120	90	218	86	2.0
1-95	Trained	TM	74	120	85	146	103	2.1
1-97	Trained	TM	80	115	90	83	94	2.7
1-98	Trained	TM	88	130	85	169	106	3.2
1-99*	Trained	TM	68	120	90	287	86	NAv
1-6	Untrained	none	68	125	80	186	85	2.9
1-8	Untrained	none	80	120	90	68	97	1.9
1-14	Untrained	none	98	155	100	156	77	1.9
1-18	Untrained	none	70	130	90	68	99	3.4
1-19	Untrained	none	76	110	85	211	95	2.7
1-23	Untrained	none	80	115	90	75	106	2.1
1-67*	In-training	TM	64	120	90	136	77	NAv
1-83	In-training	TM	72	120	80	174	81	3.0
1-89	In-training	TM	64	110	80	475	76	2.6
1-90	In-training	TM	66	120	90	70	93	2.4
1-39	In-training	PR	80	135	90	140	102	2.0
1-49	In-training	PR	60	115	85	234	97	2.3
1-43	In-training	AHR	84	115	85	205	63	2.4
1-44	In-training	AHR	74	100	80	295	84	1.8
1-75†	In-training	YM	80	120	85	484	104	1.8
1-77	In-training	YM	60	130	90	94	103	2.7
1-79	In-training	YM	72	105	80	250	111	2.6
1-101	In-training	YM	84	130	95	182	95	1.9

* Subjects 99 and 67 were excluded from T_3 statistical analysis because only two T_3 results were available on each of them.

† Subject 75 was excluded from blood hormone statistical analysis because only the two pre-relaxation blood test results were available on her.

(d) Cardiovascular and Blood Hormone Results at 1630 Hours

Subject Code No	Relaxation Training	Relaxation Procedure	Heart Rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Plasma Cortisol (n mol/l)	Serum T_4 (n mol/l)	Serum T_3 (n mol/l)
1-81	Trained	TM	62	115	80	40	62	2.1
1-85	Trained	TM	64	110	80	77	66	1.7
1-88	Trained	TM	72	130	80	64	128	2.7
1-92	Trained	TM	64	110	80	184	104	1.9
1-95	Trained	TM	64	120	90	153	83	2.9
1-97	Trained	TM	80	125	90	50	76	2.8
1-98	Trained	TM	80	125	90	152	58	2.5
1-99*	Trained	TM	50	125	80	190	95	NAv
1-6	Untrained	none	64	115	85	99	108	2.9
1-8	Untrained	none	76	130	90	70	98	2.3
1-14	Untrained	none	96	145	98	99	83	2.2
1-18	Untrained	none	72	120	90	143	104	2.3
1-19	Untrained	none	62	115	85	135	104	2.0
1-23	Untrained	none	78	125	90	82	90	3.4
1-67*	In-training	TM	63	115	80	241	99	NAv
1-83	In-training	TM	68	160	90	92	83	3.2
1-89	In-training	TM	78	110	80	370	99	3.1
1-90	In-training	TM	62	120	90	102	84	2.4
1-39	In-training	PR	98	130	100	98	89	1.8
1-49	In-training	PR	60	105	80	213	95	2.1
1-43	In-training	AHR	80	110	75	155	85	2.7
1-44	In-training	AHR	96	105	80	258	77	2.4
1-75†	In-training	YM	80	115	80	NAv	NAv	NAv
1-77	In-training	YM	54	125	90	100	126	2.5
1-79	In-training	YM	80	105	75	190	111	1.7
1-101	In-training	YM	90	115	90	136	95	1.7

* Subjects 99 and 67 were excluded from T_3 statistical analysis because only two T_3 results were available on each of them.

† Subject 75 was excluded from blood hormone statistical analysis because only the two pre-relaxation blood test results were available on her.

(e) Cardiovascular and Blood Hormone Results at 1730 Hours

Subject Code No	Relaxation Training	Relaxation Procedure	Heart Rate (bpm)	Systolic BP (mm Hg)	Diastolic BP (mm Hg)	Plasma Cortisol (n mol/l)	Serum T_3 (n mol/l)	Serum T_4 (n mol/l)
1-81	Trained	TM	60	120	90	41	103	1.7
1-85	Trained	TM	68	115	80	61	88	2.4
1-88	Trained	TM	68	130	90	43	98	2.2
1-92	Trained	TM	60	110	80	124	77	2.4
1-95	Trained	TM	66	115	95	95	126	2.1
1-97	Trained	TM	74	115	85	31	102	2.9
1-98	Trained	TM	80	115	90	128	111	3.9
1-99*	Trained	TM	64	120	90	130	74	1.6
1-6	Untrained	none	60	120	80	252	94	2.7
1-8	Untrained	none	76	130	95	74	98	2.6
1-14	Untrained	none	92	140	98	59	92	2.1
1-18	Untrained	none	76	120	90	82	94	2.1
1-19	Untrained	none	64	100	85	84	93	2.5
1-23	Untrained	none	78	130	90	94	108	2.7
1-67*	In-training	TM	60	125	85	162	84	2.0
1-83	In-training	TM	64	120	90	102	83	2.7
1-89	In-training	TM	72	125	90	272	101	2.5
1-90	In-training	TM	60	130	85	65	86	2.2
1-39	In-training	PR	100	135	90	129	132	1.9
1-49	In-training	PR	68	120	80	122	99	3.1
1-43	In-training	AHR	84	132	85	130	65	2.7
1-44	In-training	AHR	92	100	80	171	74	2.4
1-75†	In-training	YM	76	120	80	NAv	NAv	NAv
1-77	In-training	YM	56	120	75	59	126	2.6
1-79	In-training	YM	80	115	80	160	116	2.8
1-101	In-training	YM	84	120	90	80	85	2.4

* Subjects 99 and 67 were excluded from T_3 statistical analysis because only two T_3 results were available on each of them.

† Subject 75 was excluded from blood hormone statistical analysis because only the two pre-relaxation blood test results were available on her.

(f) Urinary Hormone Results (n mol/d)

Subject Code No	Relaxation Training	Relaxation Procedure	Sat UFC	Sun UFC	Mon UFC	Sat UCA	Sun UCA	Mon UCA
1-81	Trained	TM	83	130	213	142	175	126
1-85	Trained	TM	139	117	173	205	213	355
1-88	Trained	TM	130	115	167	289	360	401
1-92	Trained	TM	215	185	NAv	374	375	NAv
1-95	Trained	TM	192	136	123	568	355	371
1-97	Trained	TM	116	104	117	347	259	450
1-98	Trained	TM	102	105	106	328	292	205
1-99	Trained	TM	153	157	153	330	303	240
1-6	Untrained	none	159	189	201	339	289	287
1-8	Untrained	none	175	221	268	292	265	257
1-14	Untrained	none	195	123	128	606	371	268
1-18	Untrained	none	171	230	184	235	240	344
1-19	Untrained	none	439	195	255	396	407	472
1-23	Untrained	none	186	149	215	401	257	360
1-67	In-training	TM	168	116	132	360	330	339
1-83	In-training	TM	201	184	116	251	232	210
1-89	In-training	TM	221	247	185	243	265	268
1-90	In-training	TM	348	192	364	442	224	265
1-39	In-training	PR	NAv	153	NAv	NAv	191	NAv
1-49	In-training	PR	145	192	169	177	235	293
1-43	In-training	AHR	160	149	97	287	147	197
1-44	In-training	AHR	121	184	98	235	128	243
1-75	In-training	YM	163	141	163	431	96	218
1-77	In-training	YM	150	150	156	330	349	352
1-79	In-training	YM	141	421	232	126	169	281
1-101	In-training	YM	286	374	228	369	303	322

C. Short-term Study (2)

(a) Subject Characteristics

Subject Code No	Group*	Age (yr)	TM(yr)	MEDITATION EXPERIENCE			SUBJECTIVE REPORT RATINGS		
				Number of Advanced Techniques	Number of Residence Courses	Category	Experimental Week-end Meditation or Relaxation Depth	Experimental Week-end Stress	Control Week-end Meditation Depth
2-1	1	27	5.5	1	about 18	A	16	2	13
2-2(37)	1A(1B)	22	1.8	none	10	C†	14	4	16
2-3	1	22	1.1	none	4	C	15	2	15
2-4(38)	1A(1B)	27	6.0	1	4	A	15	3	14
2-5	1	24	2.5	none	4	B	10	3	8
2-6	1	24	2.8	1	7	B	15	5	8
2-7	1	27	1.3	none	about 7	C	14	3	NAv
2-8(36)	1A(1B)	20	4.25	none	4	B	15	3	11
2-10(39)	1A(1B)	23	4.3	1	6	B	17	2	17
2-12	1	24	4.75	1	**	A†	13	3	18
2-13	1	27	3.25	none	8	B	16	2	15
2-14	1	48	9.3	2	8	A	16	1	13
2-15	1	35	9.0	2	3	A	18	2	14
2-16	1	30	2.1	none	about 20	B†	16	2	13
2-17(40)	1A(1B)	28	5.1	1	about 13	A†	12	2	NAv
2-35	1	22	1.1	1	4	C	15	3	11

* See Table 1, p 33.

** Six months' teacher training and advanced teacher training in residence.

† In order to approximately equalise the number of subjects in each meditation experience category, subjects with TM experience (yr) at the border of a meditation experience category (ie, arbitrarily, ± 0.3 yr) were categorised according to number of advanced techniques and number of residence courses attended.

Subject Code No	Group*	Age (yr)	TM(yr)	MEDITATION EXPERIENCE			SUBJECTIVE REPORT RATINGS		
				Number of Advanced Techniques	Number of Residence Courses	Category	Experimental Week-end Meditation or Relaxation Depth	Experimental Week-end Stress	Control Week-end Meditation Depth
2-9	2	27	NAp	NAp	NAp	NAp	10	2	NAp
2-11	2	23	NAp	NAp	NAp	NAp	7	1	NAp
2-18	2	29	NAp	NAp	NAp	NAp	NAv	NAv	NAp
2-19	2	26	NAp	NAp	NAp	NAp	9	3	NAp
2-20	2	23	NAp	NAp	NAp	NAp	15	1	NAp
2-21	2	21	NAp	NAp	NAp	NAp	8	3	NAp
2-27	2	20	NAp	NAp	NAp	NAp	11	3	NAp
2-29	2	32	NAp	NAp	NAp	NAp	13	1	NAp
2-31	2	30	NAp	NAp	NAp	NAp	15	3	NAp
2-32	2	31	NAp	NAp	NAp	NAp	7	3	NAp
2-33	2	27	NAp	NAp	NAp	NAp	10	2	NAp
2-34	2	25	NAp	NAp	NAp	NAp	10	3	NAp
2-42	2	25	NAp	NAp	NAp	NAp	9	4	NAp
2-43	2	24	NAp	NAp	NAp	NAp	9	5	NAp
2-44	2	21	NAp	NAp	NAp	NAp	8	3	NAp
2-28	3	24	5.1	1	3	A†	NAp	NAv	NAv
2-30	3	31	1.6	none	about 12	C	NAp	NAv	NAv
2-41	3	24	2.5	none	about 12	B	NAp	NAv	NAv
2-45	3	32	2.1	none	2	C†	NAp	NAv	NAv
2-46	3	24	0.8	none	none	C	NAp	NAv	NAv

* See Table 1, p 33.

† In order to approximately equalise the number of subjects in each meditation experience category, subjects with TM experience (yr) at the border of a meditation experience category (ie, arbitrarily, ± 0.3 yr) were categorised according to number of advanced techniques and number of residence courses attended.

(b) Matched Subject Characteristics

Subject Code No (matched pairs)	Δ(C-S) (min)	Age (yr)	Body Surface Area(m^2)	Dietary Protein (g)	Diet Rating (units)	Urea Excretion (m mol/d)	Urine Volume (ml)	Previous Night's Sleep (h)	Usual Sleep (h)	Caffeinated Beverages (cups/d)	Tobacco (cigarettes/d)	Physical Training (min/wk)	Occupation
2-45	35	32	1.80	37.2	3	323	730	6	8	0	6	340	brake service proprietor
2-9	38	27	1.99	82.3	1	371	1750	9.5	NAv	0	0	0	university student
2-10	58	23	1.91	103.3	4	219	1555	8	8	0	0	0	assistant film editor
2-11	56	23	2.08	103.8	1	508	1165	9	6	1	0	150	university student
2-17	49	28	1.82	55.7	2	353	790	8.5	7.5	2	0	500	school teacher
2-19	50	26	1.81	107.5	1	597	1155	8.5	7	0	0	420	technician
2-28	52	24	1.80	68.4	1	248	1122	8.4	7	0	0	0	telecom technician
2-20	55	23	NAv	48.6	4	197	1300	8.5	8.5	0	0	480	gardener
2-3	49	22	1.77	67.3	3	255	1245	7.3	10	0	0	200	student teacher
2-21	44	21	1.89	131.3	1	385	1220	7.3	8	0	0	960	university student

Subject Code No (matched pairs)	Δ(C-S) (min)	Age (yr)	Body Surface Area(m^2)	Dietary Protein (g)	Diet Rating (units)	Urea Extretion (m mol/d)	Urine Volume (ml)	Previous Night's Sleep (h)	Usual Sleep (h)	Caffeinated Beverages (cups/d)	Tobacco (cigarettes/d)	Physical Training (min/wk)	Occupation
2-2	52	22	1.81	55.3	4	205	1510	7.7	7	0	0	125	university student
2-27	56	20	1.89	53.4	3	307	890	7.5	7	1	0	120	university student
2-16	49	30	1.87	26.8	1	346	1540	8	7	0	15	0	school teacher
2-29	51	32	1.85	57.4	3	284	1550	7.6	7	0	0	120	clerk
2-7	47	27	1.88	35.7	4	130	430	8.5	8	0	4	450	school teacher
2-32	47	31	1.85	58.8	3	352	721	8.5	9	0	0	0	builder's labourer
2-12	31	24	1.90	58.6	4	331	1180	8.3	8	0	0	150	TM teacher
2-34	33	25	1.75	77.6	3	285	1166	9.2	7.5	0	2	210	research worker
2-46	35	24	1.75	73.7	2	397	906	9.5	9	0	0	90	unemployed
2-42	39	25	1.75	75.1	1	197	1164	6.6	9.5	0	0	335	unemployed
2-30	63	31	2.01	123.2	2	339	1575	7.1	6.5	2	0	435	research officer
2-43	61	24	1.86	88.4	1	274	2810	7.4	9.5	0	0	0	cleaner
2-35	31	22	1.94	109.9	3	390	1125	7.8	8	1	0	280	farm assistant
2-44	21	21	1.70	30.6	4	367	1490	7.5	7.5	0	0	0	nurseryman

(c) Results of Blood Tests at 1415 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.7	165.	168.	.45	42.0	70.	19.2	4.8	NAv	235.7	131.
2-2	1	1.4	NAv	NAv	.21	41.0	67.	1.0	5.4	NAv	161.5	136.
2-3	1	1.7	118.	110.	.26	39.0	70.	7.2	4.6	NAv	305.4	NAv
2-4	1	1.1	108.	103.	.31	40.0	65.	3.2	4.7	NAv	134.8	55.
2-5	1	1.4	128.	121.	.31	44.0	70.	14.8	3.8	NAv	256.5	117.
2-6	1	NAv	NAv	NAv	NAv	38.0	62.	12.4	4.9	NAv	439.0	287.
2-7	1	2.1	175.	152.	.24	43.5	72.	3.2	3.8	NAv	184.6	132.
2-8	1	1.4	118.	132.	.20	42.0	69.	3.0	3.7	NAv	165.7	99.
2-10	1	1.9	143.	120.	NAv	42.0	69.	5.0	4.6	NAv	308.5	146.
2-12	1	1.7	109.	94.	.18	42.0	72.	3.8	3.0	NAv	153.9	160.
2-13	1	.9	77.	79.	.42	45.0	73.	10.2	3.5	1.2	453.9	121.
2-14	1	1.2	103.	100.	.33	42.5	66.	.2	5.5	.8	487.1	70.
2-15	1	1.5	102.	107.	.29	38.5	79.	1.0	4.9	.7	163.2	74.
2-16	1	1.9	109.	117.	.31	41.0	68.	1.0	3.2	.8	137.1	91.
2-17	1	1.5	116.	103.	.30	37.5	69.	9.6	3.8	1.1	172.7	67.
2-35	1	1.4	93.	101.	.23	43.0	65.	1.0	5.2	1.2	228.0	162.
2-9	2	1.7	105.	102.	.16	47.4	78.	1.2	4.8	NAv	435.1	169.
2-11	2	1.5	118.	129.	.24	43.9	74.	.6	5.4	NAv	168.2	141.
2-19	2	.9	81.	84.	.20	42.6	65.	7.4	4.8	1.0	444.7	133.
2-20	2	1.7	107.	109.	.22	43.0	72.	28.0	4.2	1.7	161.9	166.
2-21	2	2.0	118.	107.	.35	41.3	66.	26.0	4.8	1.1	454.0	106.
2-27	2	1.0	88.	102.	.28	42.7	70.	43.0	5.8	1.0	498.2	271.
2-29	2	1.2	103.	102.	.25	45.0	72.	.8	4.1	1.8	142.3	114.
2-31	2	1.4	116.	114.	NAv	42.8	65.	.8	4.3	1.8	106.1	122.
2-32	2	1.9	126.	116.	.26	42.6	66.	1.0	4.8	1.5	292.9	104.
2-33	2	1.7	117.	109.	.26	41.2	71.	3.4	4.8	1.6	392.3	175.
2-34	2	1.5	95.	98.	.27	40.4	67.	2.0	5.1	.9	426.7	72.
2-42	2	1.2	86.	92.	.28	41.2	64.	22.0	6.6	2.4	646.1	425.
2-43	2	2.0	104.	100.	.17	43.4	70.	1.6	4.8	1.4	344.9	122.
2-44	2	1.4	134.	132.	.19	44.8	72.	.6	3.6	1.8	325.9	181.
2-36	1B	1.7	128.	132.	.32	46.6	68.	1.2	3.7	1.4	196.0	53.
2-37	1B	.9	122.	123.	.28	41.0	62.	7.0	5.4	1.4	192.3	101.
2-38	1B	2.0	116.	116.	.27	NAv	68.	7.2	4.4	.8	156.7	48.
2-39	1B	1.7	146.	149.	.33	42.1	70.	2.6	4.6	1.2	190.0	223.
2-40	1B	1.0	125.	125.	.34	NAv	67.	1.0	4.2	1.3	138.1	73.
2-28	3	1.3	149.	153.	.30	44.8	65.	37.0	5.3	1.7	442.0	409.
2-30	3	1.3	116.	130.	.34	41.2	67.	.8	4.5	.8	154.1	122.
2-41	3	.7	89.	93.	.21	44.2	63.	6.6	4.2	1.3	224.3	241.
2-45	3	NAv	96.	99.	.48	43.1	70.	13.6	3.1	1.2	233.0	69.
2-46	3	1.6	101.	97.	.28	40.9	70.	.8	3.6	1.6	253.4	45.

* See Table 1, p 33.

(d) Results of Blood Tests at 1430 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.4	123.	121.	.43	40.0	NAv	10.2	4.6	NAv	279.9	114.
2-2	1	1.2	113.	108.	.24	41.0	NAv	1.4	5.3	NAv	189.4	147.
2-3	1	1.7	132.	136.	.17	38.0	NAv	6.8	4.6	NAv	270.8	107.
2-4	1	1.2	98.	91.	.33	39.0	NAv	2.0	4.1	NAv	125.5	98.
2-5	1	1.7	131.	121.	.32	44.0	NAv	20.0	3.6	NAv	207.8	109.
2-6	1	.0	NAv	NAv	.00	38.0	NAv	8.2	3.6	NAv	437.7	245.
2-7	1	1.9	154.	149.	.25	41.0	NAv	3.2	4.4	NAv	187.5	114.
2-8	1	1.5	126.	139.	.19	42.0	NAv	1.8	4.0	NAv	138.6	92.
2-10	1	1.8	126.	105.	.23	42.0	NAv	2.8	4.9	NAv	288.2	123.
2-12	1	1.6	114.	103.	.20	43.0	NAv	5.2	3.0	NAv	139.8	151.
2-13	1	.9	75.	79.	.43	44.5	NAv	18.0	3.4	1.1	400.9	134.
2-14	1	1.3	100.	103.	.33	40.0	NAv	.4	5.8	.9	466.6	71.
2-15	1	1.6	107.	107.	.28	39.5	NAv	1.0	4.4	.7	132.1	91.
2-16	1	1.8	104.	110.	.35	41.0	NAv	.8	3.4	.7	121.5	69.
2-17	1	1.5	102.	97.	.30	37.5	NAv	19.2	4.8	1.0	136.0	89.
2-35	1	1.3	101.	106.	.22	42.0	NAv	1.2	5.1	1.6	198.6	168.
2-9	2	1.6	89.	92.	.15	47.5	NAv	1.0	4.8	NAv	402.9	156.
2-11	2	1.5	110.	124.	.24	43.4	NAv	.6	5.2	NAv	180.80	140.
2-19	2	.8	93.	93.	.17	43.9	NAv	9.0	5.2	.8	459.1	73.
2-20	2	1.5	108.	108.	.25	42.7	NAv	23.2	4.3	2.0	170.8	141.
2-21	2	1.7	109.	108.	.32	42.2	NAv	23.0	5.0	1.4	413.1	138.
2-27	2	.9	92.	113.	.27	43.0	NAv	33.0	5.3	1.0	293.7	227.
2-29	2	1.2	113.	110.	.21	45.6	NAv	.6	4.6	1.8	131.6	81.
2-31	2	1.4	117.	110.	.28	44.9	NAv	.8	4.4	1.7	104.4	88.
2-32	2	1.8	123.	114.	.24	42.0	NAv	2.6	4.5	1.6	273.3	90.
2-33	2	1.8	112.	105.	.27	41.4	NAv	5.4	NAv	1.7	312.1	196.
2-34	2	1.1	90.	93.	.26	39.4	NAv	5.0	4.5	.9	413.8	54.
2-42	2	1.3	85.	91.	.16	41.4	NAv	14.0	6.3	2.0	651.3	357.
2-43	2	2.0	108.	107.	.16	44.2	NAv	1.0	4.5	1.2	352.1	134.
2-44	2	1.3	113.	108.	.17	44.8	NAv	.6	5.4	1.8	273.8	170.
2-36	1B	1.6	123.	128.	.37	46.2	NAv	1.0	3.7	1.7	177.9	73.
2-37	1B	1.0	120.	122.	.23	40.4	NAv	9.2	5.7	1.3	131.4	82.
2-38	1B	2.0	100.	97.	.34	41.3	NAv	4.8	4.5	.9	178.4	50.
2-39	1B	1.8	147.	147.	.35	42.4	NAv	5.4	5.2	1.1	190.0	233.
2-40	1B	1.1	128.	131.	.29	39.7	NAv	1.8	4.5	1.3	118.9	75.
2-28	3	1.5	164.	164.	.27	NAv	NAv	26.0	4.5	1.5	339.3	276.
2-30	3	1.2	118.	124.	.35	NAv	NAv	.6	4.7	1.2	119.3	240.
2-41	3	.9	81.	83.	.21	44.2	NAv	4.8	4.1	1.3	190.5	133.
2-45	3	.9	98.	99.	.47	43.4	NAv	6.8	2.8	1.1	205.90	71.
2-46	3	1.5	108.	103.	.26	40.3	NAv	.6	3.3	1.3	224.7	48.

* See Table 1, p 33.

(e) Results of Blood Tests at 1445 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.3	138.	128.	.41	41.0	NAv	7.6	4.6	NAv	242.7	83.
2-2	1	1.0	142.	129.	.25	41.0	NAv	1.6	5.3	NAv	134.7	122.
2-3	1	1.5	120.	120.	.16	39.0	NAv	4.6	4.1	NAv	235.7	118.
2-4	1	1.2	114.	96.	.33	41.0	NAv	1.6	3.9	NAv	111.0	51.
2-5	1	1.5	109.	102.	.31	45.0	NAv	24.0	3.5	NAv	176.4	116.
2-6	1	NAv	NAv	NAv	NAv	39.0	NAv	4.8	3.6	NAv	429.0	230.
2-7	1	1.9	148.	123.	.20	43.0	NAv	5.0	4.6	NAv	154.7	109.
2-8	1	1.6	108.	112.	.18	42.0	NAv	1.0	4.2	NAv	123.7	78.
2-10	1	1.9	136.	125.	NAv	43.0	NAv	2.0	4.7	NAv	239.4	105.
2-12	1	1.6	111.	100.	.18	42.0	NAv	5.6	3.8	NAv	124.5	143.
2-13	1	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv
2-14	1	1.0	104.	104.	.27	39.5	NAv	.4	5.6	.9	412.2	23.
2-15	1	2.3	94.	91.	.22	40.5	NAv	1.0	4.4	.8	164.9	100.
2-16	1	1.8	106.	112.	.31	41.0	NAv	1.0	2.7	.7	119.5	60.
2-17	1	1.8	NAv	NAv	.35	38.0	NAv	18.4	4.0	1.0	119.2	53.
2-35	1	1.5	97.	102.	.18	42.0	NAv	1.0	4.7	1.4	205.2	158.
2-9	2	1.4	93.	96.	NAv	46.5	NAv	.8	4.3	NAv	353.3	129.
2-11	2	1.3	111.	120.	.23	43.3	NAv	.6	4.9	NAv	167.3	141.
2-19	2	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv
2-20	2	1.7	95.	97.	.24	42.6	NAv	17.6	4.0	1.7	204.7	125.
2-21	2	1.4	114.	117.	.31	41.0	NAv	15.6	5.1	1.4	370.1	127.
2-27	2	.9	97.	113.	.30	42.9	NAv	23.0	4.4	1.1	413.6	207.
2-29	2	1.3	93.	90.	.26	46.5	NAv	.6	4.5	1.7	115.7	161.
2-31	2	1.5	121.	115.	.24	45.0	NAv	.8	4.4	1.5	108.3	104.
2-32	2	1.8	124.	117.	.24	42.4	NAv	9.8	4.8	1.5	163.7	92.
2-33	2	NAv	NAv	NAV	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv
2-34	2	1.3	92.	96.	.27	39.1	NAv	9.2	5.0	.6	199.5	50.
2-42	2	1.5	98.	107.	.21	41.5	NAv	9.0	5.6	1.8	576.2	259.
2-43	2	1.8	111.	108.	.16	42.9	NAv	.8	4.4	1.2	313.6	99.
2-44	2	1.3	114.	111.	.18	41.7	NAv	.6	5.4	2.0	221.3	165.
2-36	1B	1.4	119.	121.	.31	NAv	NAv	1.0	3.4	2.0	199.9	63.
2-37	1B	.8	120.	120.	.28	40.6	NAv	9.6	5.3	1.2	152.2	90.
2-38	1B	1.9	104.	103.	.32	40.6	NAv	2.6	4.4	.8	173.5	54.
2-39	1B	1.7	145.	148.	.34	NAv	NAv	5.8	4.7	1.3	170.0	240.
2-40	1B	.7	112.	116.	.28	NAv	NAv	5.0	4.8	1.1	89.8	90.
2-28	3	1.5	148.	155.	.32	NAv	NAv	12.8	3.7	1.6	362.5	234.
2-30	3	1.3	119.	126.	.37	41.7	NAv	.6	4.5	1.1	115.4	176.
2-41	3	.9	83.	85.	.24	44.9	NAv	3.2	4.1	1.3	206.6	141.
2-45	3	1.0	105.	108.	.34	43.0	NAv	15.6	2.7	1.2	173.3	42.
2-46	3	1.7	118.	114.	.26	39.6	NAv	.8	3.4	1.5	231.5	43.

* See Table 1, p 33.

(f) Results of Blood Tests at 1500 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.4	137.	144.	.25	40.0	71.	5.4	4.9	NAv	204.2	91.
2-2	1	1.3	NAv	NAv	.14	41.0	68.	1.6	4.4	NAv	153.7	129.
2-3	1	1.7	123.	114.	.25	39.0	69.	3.0	4.1	NAv	171.2	106.
2-4	1	1.3	100.	94.	.34	40.0	67.	1.4	4.0	NAv	116.9	21.
2-5	1	1.5	120.	106.	.31	44.0	68.	25.0	3.7	NAv	145.6	134.
2-6	1	NAv	NAv	NAv	NAv	39.0	64.	4.6	3.5	NAv	462.5	228.
2-7	1	1.8	163.	140.	.22	43.0	69.	4.6	3.5	NAv	160.6	130.
2-8	1	1.5	118.	117.	.20	43.0	67.	.8	4.5	NAv	105.7	86.
2-10	1	2.1	149.	131.	.24	42.0	70.	1.4	4.7	NAv	199.7	106.
2-12	1	1.3	113.	90.	NAv	41.0	69.	5.2	3.3	NAv	151.2	163.
2-13	1	.8	82.	82.	.48	44.5	73.	8.6	3.9	1.1	265.5	124.
2-14	1	1.3	120.	122.	.29	40.0	64.	.2	5.1	.7	346.4	46.
2-15	1	1.5	99.	94.	.24	40.0	77.	1.0	4.7	.7	121.2	96.
2-16	1	2.1	108.	122.	.36	41.5	70.	1.0	4.4	.6	106.3	70.
2-17	1	1.3	98.	104.	.27	37.0	67.	6.6	4.0	1.1	97.0	41.
2-35	1	1.3	101.	105.	.18	42.0	67.	1.2	4.6	1.4	223.1	137.
2-9	2	1.4	75.	71.	NAv	46.5	75.	.8	4.5	NAv	289.2	130.
2-11	2	1.4	95.	105.	.25	43.3	70.	.6	4.7	NAv	141.8	153.
2-19	2	2.4	110.	99.	.18	41.9	71.	.4	4.1	.8	216.7	158.
2-20	2	1.6	106.	109.	.26	43.0	72.	8.0	4.1	1.4	165.4	106.
2-21	2	1.4	113.	118.	.28	41.9	67.	7.2	5.4	1.2	326.2	130.
2-27	2	1.1	104.	119.	.31	43.2	68.	12.4	4.4	1.0	383.3	243.
2-29	2	1.4	111.	108.	.21	45.4	72.	.6	4.9	1.6	115.0	112.
2-31	2	1.6	121.	116.	.24	44.2	68.	1.0	3.8	1.7	98.4	91.
2-32	2	1.5	133.	126.	.24	41.6	65.	9.2	4.5	1.2	161.7	85
2-33	2	1.6	96.	91.	.25	41.9	70.	20.0	5.0	3.1	628.1	349.
2-34	2	1.4	101.	106.	.27	39.2	67.	18.8	4.8	.8	266.8	57.
2-42	2	1.5	94.	99.	.25	NAv	65.	6.0	4.3	1.4	551.0	201.
2-43	2	1.7	103.	98.	NAv	NAv	70.	.6	4.4	1.1	329.2	NAv
2-44	2	1.3	132.	125.	.21	43.3	68.	.8	5.3	2.4	186.5	125.
2-36	1B	1.5	118.	119.	.32	46.2	67.	1.6	2.8	1.7	191.0	53.
2-37	1B	1.4	120.	124.	.27	40.8	62.	9.0	5.0	1.0	131.4	79.
2-38	1B	1.9	104.	103.	.36	40.7	68.	2.0	4.5	1.0	154.8	52.
2-39	1B	1.9	144.	145.	.38	NAv	70.	3.4	4.5	1.3	160.0	177.
2-40	1B	.8	122.	128.	.28	NAv	65.	15.6	4.7	1.3	95.8	68.
2-28	3	1.3	157.	165.	.34	45.5	65.	7.8	3.9	1.4	286.3	191.
2-30	3	1.3	130.	140.	.37	41.6	67.	.4	4.7	1.0	110.6	206.
2-41	3	.8	91.	93.	.23	44.7	64.	2.4	4.0	1.1	185.8	134.
2-45	3	.7	97.	103.	.40	42.2	68.	18.4	2.9	1.2	194.5	47.
2-46	3	1.5	105.	102.	.26	NAv	70.	.8	3.8	1.1	170.4	52.

* See Table 1, p 33.

(g) Results of Blood Tests at 1510 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.5	133.	126.	.46	42.0	NAv	4.0	4.9	NAv	175.0	84.
2-2	1	1.3	102.	102.	.18	39.0	NAv	.8	4.2	NAv	195.7	154.
2-3	1	1.2	NAv	NAv	.22	36.0	NAv	1.8	4.5	NAv	129.2	97.
2-4	1	1.0	102.	84.	.45	39.0	NAv	1.0	3.9	NAv	134.2	37.
2-5	1	1.5	114.	107.	.29	NAv	NAv	19.0	4.1	NAv	115.8	83.
2-6	1	NAv	NAv	NAv	NAv	39.0	NAv	NAv	NAv	NAv	NAv	NAv
2-7	1	1.5	163.	150.	.16	43.0	NAv	3.4	4.6	NAv	141.7	103.
2-8	1	1.4	119.	121.	NAv	43.0	NAv	.8	4.4	NAv	156.9	76.
2-10	1	2.1	143.	130.	.26	42.0	NAv	1.4	4.9	NAv	196.4	88.
2-12	1	1.4	106.	98.	NAv	41.0	NAv	3.4	4.5	NAv	92.2	158.
2-13	1	.6	74.	80.	.36	45.5	NAv	6.2	4.8	1.2	202.8	97.
2-14	1	1.3	105.	113.	.32	41.0	NAv	.2	5.7	.6	282.6	54.
2-15	1	1.5	88.	86.	.25	39.5	NAv	1.0	4.8	.5	102.4	93.
2-16	1	1.6	114.	117.	.33	41.5	NAv	.8	4.2	.4	107.9	58.
2-17	1	1.5	92.	98.	.27	37.5	NAv	5.4	3.9	1.0	95.6	27.
2-35	1	1.5	105.	107.	.19	42.0	NAv	1.2	4.7	1.2	209.9	156.
2-9	2	1.4	85.	87.	NAv	45.7	NAv	.8	4.9	NAv	275.9	132.
2-11	2	1.2	99.	114.	.25	42.0	NAv	.8	4.7	NAv	127.3	156.
2-19	2	2.0	100.	89.	.23	43.1	NAv	.4	4.1	1.1	160.6	122.
2-20	2	1.5	103.	106.	.29	42.8	NAv	5.6	4.4	1.5	158.0	110.
2-21	2	1.5	111.	114.	.31	42.6	NAv	5.6	5.5	1.4	316.3	112.
2-27	2	1.0	96.	108.	.29	42.8	NAv	9.0	4.4	.7	377.3	152.
2-29	2	1.4	99.	97.	.26	45.3	NAv	1.0	.2	.6	113.7	135.
2-31	2	1.6	124.	117.	.24	44.0	NAv	.8	3.8	1.6	105.2	104.
2-32	2	1.5	139.	133.	.27	41.9	NAv	7.2	4.5	1.3	115.2	96.
2-33	2	.8	104.	97.	.24	42.0	NAv	27.0	5.0	2.9	691.2	351.
2-34	2	1.3	96.	100.	.29	NAv	NAv	17.2	4.7	.8	233.9	48.
2-42	2	NAv	NAv	NAv	NAv	43.0	NAv	NAv	NAv	1.7	NAv	NAv
2-43	2	2.1	110.	108.	.15	44.9	NAv	1.2	4.9	1.1	419.5	80.
2-44	2	1.3	111.	110.	.19	44.0	NAv	.8	4.4	2.2	159.0	139.
2-36	1B	1.7	126.	131.	.30	46.6	NAv	3.8	2.8	1.7	190.0	82.
2-37	1B	1.0	107.	111.	.29	40.8	NAv	7.4	4.7	1.3	106.2	83.
2-38	1B	1.8	108.	107.	.40	NAv	NAv	1.4	4.5	.9	131.4	53.
2-39	1B	2.1	157.	157.	.34	NAv	NAv	2.8	4.4	1.1	180 0	167.
2-40	1B	1.1	118.	120.	.28	39.1	NAv	22.0	4.7	.9	163.8	89.
2-28	3	1.6	156.	160.	.33	45.2	NAv	5.0	4.0	1.4	311.8	219.
2-30	3	1.3	119.	128.	.40	41.8	NAv	.6	4.7	1.1	93.1	113.
2-41	3	.8	86.	89.	.26	44.7	NAv	2.2	4.1	1.0	148.5	121.
2-45	3	.8	99.	105.	.42	42.2	NAv	14.8	2.8	1.1	225.1	24.
2-46	3	1.7	96.	101.	.25	40.7	NAv	.8	4.1	1.3	170.8	83.

* See Table 1, p 33.

(h) Results of Blood Tests at 1520 Hours

Subject Code No	Group *	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.4	137.	NAv	.42	42.0	NAv	3.2	4.3	NAv	167.7	88.
2-2	1	1.2	118.	139.	.25	38.0	NAv	.8	4.4	NAv	143.1	110.
2-3	1	1.8	107.	114.	.29	39.0	NAv	1.6	4.3	NAv	149.7	115.
2-4	1	1.2	110.	101.	.40	41.0	NAv	1.0	4.3	NAv	115.6	78.
2-5	1	1.3	119.	123.	.35	45.0	NAv	15.0	4.0	NAv	112.8	93.
2-6	1	NAv	NAv	NAv	NAv	39.0	NAv	2.8	4.4	NAv	467.5	226.
2-7	1	2.1	138.	120.	.18	43.0	NAv	2.6	4.2	NAv	182.4	119.
2-8	1	1.4	116.	124.	.21	43.0	NAv	.8	4.4	NAv	127.3	92.
2-10	1	1.9	139.	120.	.31	43.0	NAv	1.2	5.0	NAv	126.6	97.
2-12	1	1.5	100.	88.	.18	42.0	NAv	2.6	3.2	NAv	101.9	162.
2-13	1	.2	68.	69.	NAv	44.5	NAv	4.6	4.8	1.0	186.3	114.
2-14	1	1.0	104.	102.	.24	40.5	NAv	.4	5.5	.6	260.4	76.
2-15	1	1.2	95.	94.	.24	39.5	NAv	1.0	4.6	.6	106.9	65.
2-16	1	1.6	NAv	NAv	.31	42.0	NAv	.8	3.9	.7	114.9	54.
2-17	1	1.6	108.	110.	.25	37.0	NAv	4.0	4.1	.9	77.9	64.
2-35	1	1.4	95.	99.	.19	42.0	NAv	1.4	4.9	1.3	233.3	168.
2-9	2	1.6	94.	86.	.17	46.1	NAv	.8	4.9	NAv	240.9	232.
2-11	2	1.4	100.	108.	.26	43.0	NAv	.8	4.7	NAv	177.7	173.
2-19	2	2.2	104.	91.	.23	43.7	NAv	.4	4.2	1.1	154.1	133.
2-20	2	1.7	108.	109.	.22	42.6	NAv	4.0	4.5	1.4	159.3	127.
2-21	2	1.5	113.	120.	.32	41.9	NAv	4.0	5.4	1.4	333.5	97.
2-27	2	.8	101.	113.	.34	43.1	NAv	7.8	4.3	.8	320.4	163.
2-29	2	1.3	99.	96.	.21	45.5	NAv	2.4	4.1	1.7	106.3	153.
2-31	2	1.2	121.	117.	.25	44.4	NAv	.8	3.8	1.9	101.1	100.
2-32	2	1.4	117.	109.	.30	41.3	NAv	5.0	4.5	1.1	145.2	79.
2-33	2	1.8	NAv	NAv	NAv	41.3	NAv	30.0	5.2	2.5	687.5	299.
2-34	2	1.5	89.	90.	.32	38.5	NAv	15.6	4.8	.9	230.2	79.
2-42	2	1.3	101.	109.	.29	42.6	NAv	8.8	4.6	2.1	491.0	146.
2-43	2	1.9	99.	98.	.15	42.8	NAv	3.2	4.8	1.5	440.7	92.
2-44	2	1.2	117.	116.	.15	43.5	NAv	.8	4.2	1.7	149.9	216.
2-36	1B	1.7	117.	120.	.32	NAv	NAv	7.0	2.7	1.5	179.3	78.
2-37	1B	1.0	125.	129.	.26	40.5	NAv	5.8	4.5	1.2	106.0	63.
2-38	1B	2.0	116.	114.	.38	40.5	NAv	1.2	4.5	1.0	145.5	39.
2-39	1B	1.8	139.	136.	.36	NAv	NAv	2.0	4.6	1.2	130.0	153.
2-40	1B	.7	117.	118.	.29	NAv	NAv	32.0	4.7	.9	132.7	58.
2-28	3	1.6	169.	171.	.32	44.7	NAv	3.4	3.9	1.7	279.4	191.
2-30	3	1.3	111.	121.	.47	41.6	NAv	1.0	4.7	1.1	124.9	117.
2-41	3	1.1	79.	83.	.25	44.3	NAv	2.0	4.1	1.0	84.3	157.
2-45	3	.7	102.	107.	.39	42.6	NAv	13.2	2.7	1.2	245.2	51.
2-46	3	1.5	98.	97.	.25	40.0	NAv	.8	4.4	1.2	154.8	67.

* See Table 1, p 33.

(i) Results of Blood Tests at 1530 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.2	126.	122.	.46	NAv	71.	2.6	4.7	NAv	141.6	83.
2-2	1	1.2	116.	129.	.24	40.0	68.	.6	4.2	NAv	131.9	85.
2-3	1	1.5	129.	128.	.29	39.0	70.	1.2	4.3	NAv	150.4	84.
2-4	1	1.1	106.	89.	.37	NAv	65.	1.2	4.2	NAv	111.5	26.
2-5	1	1.5	98.	94.	.34	44.0	68.	14.0	4.5	NAv	117.1	98.
2-6	1	NAv	NAv	NAv	NAv	39.0	NAv	NAv	NAv	NAv	NAv	NAv
2-7	1	1.8	157.	132.	.19	43.0	69.	2.2	4.5	NAv	205.5	119.
2-8	1	1.4	111.	121.	.21	43.0	66.	.8	4.4	NAv	158.0	50.
2-10	1	2.1	134.	103.	.34	43.0	71.	1.0	5.2	NAv	138.2	94.
2-12	1	1.4	106.	88.	.20	42.0	73.	1.8	3.1	NAv	94.6	170.
2-13	1	.6	63.	66.	.38	44.5	72.	3.2	4.6	1.0	203.3	106.
2-14	1	1.0	99.	105.	.33	40.0	63.	.2	5.2	.6	233.8	48.
2-15	1	1.4	90.	88.	.28	39.5	76.	1.0	4.5	.4	89.0	78.
2-16	1	1.6	109.	109.	.29	42.0	70.	1.0	3.7	.5	143.3	70.
2-17	1	1.4	100.	94.	.27	37.5	67.	3.0	4.1	1.0	75.5	55.
2-35	1	1.5	92.	94.	.16	42.0	66.	1.6	5.0	1.5	186.7	161.
2-9	2	1.8	89.	89.	.16	47.2	75.	.8	5.0	NAv	194.4	282.
2-11	2	1.3	108.	97.	.27	43.2	72.	.8	4.6	NAv	193.3	165.
2-19	2	2.3	89.	77.	.23	41.5	69.	1.8	3.8	.9	153.0	129.
2-20	2	1.7	93.	95.	.26	42.4	73.	3.2	4.1	1.6	131.2	103.
2-21	2	1.4	113.	128.	.26	41.8	65.	3.2	5.3	1.4	359.2	89.
2-27	2	1.0	101.	113.	.34	43.3	69.	7.4	4.6	.8	250.3	148.
2-29	2	1.2	93.	93.	.26	44.5	72.	4.6	4.0	1.6	95.8	134.
2-31	2	NAv	NAv	NAv	NAv	44.7	68.	NAv	NAv	2.2	NAv	NAv
2-32	2	1.7	111.	105.	.28	41.6	65.	3.2	4.5	1.1	117.3	79.
2-33	2	1.5	120.	119.	.21	41.5	70.	19.2	4.9	2.1	707.3	280.
2-34	2	.9	96.	99.	.32	39.4	67.	13.2	4.9	1.0	326.6	61.
2-42	2	1.7	91.	97.	.29	40.4	66.	12.4	4.6	2.1	486.1	86.
2-43	2	NAv	NAv	NAv	NAv	43.2	68.	NAv	NAv	.7	NAv	NAv
2-44	2	1.2	119.	117.	.17	43.8	70.	.6	4.9	1.9	135.8	193.
2-36	1B	1.9	103.	106.	.35	NAv	67.	8.8	3.2	1.6	203.2	76.
2-37	1B	1.1	122.	126.	.29	41.0	61.	4.0	4.2	1.1	83.8	61.
2-38	1B	1.7	126.	127.	.45	NAv	69.	1.2	4.4	.9	114.8	50.
2-39	1B	2.1	153.	154.	.41	43.5	70.	1.8	4.4	1.3	150.0	147.
2-40	1B	1.0	136.	139.	.29	NAv	65.	37.0	4.4	1.2	99.0	54.
2-28	3	1.4	164.	170.	.33	44.5	66.	2.2	3.6	1.4	248.0	163.
2-30	3	1.2	116.	123.	.40	40.8	68.	2.0	4.6	.9	97.0	130.
2-41	3	.7	85.	88.	.33	44.5	64.	2.0	4.0	1.0	115.5	161.
2-45	3	.7	98.	100.	.46	43.2	70.	9.0	2.7	1.0	233.0	70.
2-46	3	1.3	114.	110.	.22	40.3	70.	1.4	4.2	1.0	133.7	94.

* See Table 1, p 33.

(j) Results of Blood Tests at 1545 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.5	NAv	NAv	.54	42.0	NAv	2.2	4.4	NAv	129.5	73.
2-2	1	1.2	119.	113.	.22	42.0	NAv	.4	4.6	NAv	108.4	96.
2-3	1	1.5	130.	129.	.30	40.0	NAv	1.2	4.0	NAv	134.6	75.
2-4	1	1.1	110.	131.	.39	40.0	NAv	2.0	4.3	NAv	110.2	47.
2-5	1	1.5	108.	99.	.37	46.0	NAv	9.0	4.1	NAv	99.4	153.
2-6	1	NAv	NAv	NAv	NAv	39.0	NAv	8.6	4.6	NAv	498.8	255.
2-7	1	1.9	151.	136.	.22	43.5	NAv	2.2	3.1	NAv	234.3	140.
2-8	1	1.5	128.	116.	.22	43.0	NAv	1.0	4.0	NAv	153.2	55.
2-10	1	2.0	140.	113.	.33	42.0	NAv	1.2	4.9	NAv	119.7	109.
2-12	1	1.6	113.	99.	.27	41.0	NAv	1.8	2.4	NAv	108.7	151.
2-13	1	.4	68.	79.	.35	45.0	NAv	2.0	3.8	.8	196.3	79.
2-14	1	1.4	100.	114.	.23	41.0	NAv	.6	4.9	.7	246.8	42.
2-15	1	1.6	97.	103.	.29	42.5	NAv	.8	4.4	.4	107.9	103.
2-16	1	1.5	116.	125.	.33	42.0	NAv	1.0	3.9	.6	193.5	53.
2-17	1	1.4	99.	99.	.29	37.5	NAv	1.6	4.1	.9	70.6	33.
2-35	1	1.4	93.	98.	.18	42.0	NAv	4.2	4.8	1.2	186.5	164.
2-9	2	1.7	105.	99.	.22	46.3	NAv	1.0	4.6	NAv	178.0	205.
2-11	2	1.4	102.	103.	.30	44.8	NAv	.8	4.7	NAv	204.0	169.
2-19	2	2.5	106.	90.	.35	42.7	NAv	9.4	4.1	.8	122.1	170.
2-20	2	1.7	105.	109.	.30	41.6	NAv	2.0	4.1	1.4	111.0	130.
2-21	2	1.6	124.	126.	.30	42.3	NAv	2.0	4.9	1.4	291.3	86.
2-27	2	1.0	102.	119.	.47	43.5	NAv	9.4	4.4	.7	237.8	142.
2-29	2	1.4	105.	104.	.26	45.1	NAv	3.6	4.0	1.5	NAv	190.
2-31	2	1.4	125.	116.	.25	45.6	NAv	1.2	3.3	2.2	118.1	113.
2-32	2	1.6	115.	109.	.32	41.6	NAv	2.0	4.5	1.2	105.0	61.
2-33	2	1.4	104.	98.	.27	43.1	NAv	8.4	5.1	2.0	571.7	213.
2-34	2	1.4	92.	93.	.32	39.7	NAv	13.2	4.5	.8	419.5	84.
2-42	2	1.3	93.	97.	.26	NAv	NAv	12.0	4.6	2.0	456.2	80.
2-43	2	1.9	108.	104.	NAv	42.3	NAv	1.2	4.4	1.2	357.7	NAv
2-44	2	1.2	114.	112.	.20	43.0	NAv	.6	4.7	2.2	115.6	170.
2-36	1B	1.5	109.	114.	.37	45.9	NAv	7.0	3.7	1.8	214.8	100.
2-37	1B	1.1	126.	130.	.32	40.6	NAv	2.8	3.8	1.2	85.9	43.
2-38	1B	2.2	117.	117.	.46	41.3	NAv	1.0	4.5	1.0	116.6	51.
2-39	1B	2.2	146.	146.	.36	NAv	NAv	1.4	4.5	1.1	170.0	146.
2-40	1B	.9	129.	133.	.27	38.9	NAv	24.0	4.4	.9	85.9	60.
2-28	3	1.6	167.	169.	.34	NAv	NAv	1.6	3.6	1.1	214.9	158.
2-30	3	1.3	122.	128.	.34	41.4	NAv	3.8	4.5	1.1	90.0	130.
2-41	3	.9	85.	89.	.37	43.4	NAv	1.6	3.8	1.3	101.7	134.
2-45	3	.6	101.	106.	.46	43.0	NAv	5.8	2.6	1.3	209.5	71.
2-46	3	1.3	115.	113.	.26	39.4	NAv	2.8	3.7	1.3	117.9	80.

* See Table 1, p 33.

(k) Results of Blood Tests at 1600 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.1	140.	126.	.48	41.0	NAv	2.0	4.6	NAv	125.2	76.
2-2	1	1.2	112.	101.	.23	40.0	NAv	1.6	4.3	NAv	216.8	94.
2-3	1	1.8	138.	138.	.28	39.0	NAv	1.6	4.2	NAv	122.1	104.
2-4	1	NAv	104.	99.	.30	39.0	NAv	3.6	3.8	NAv	166.3	31.
2-5	1	1.6	102.	105.	.38	45.0	NAv	6.2	4.1	NAv	107.2	153.
2-6	1	NAv	NAv	NAv	NAv	37.0	NAv	5.8	4.8	NAv	496.5	198.
2-7	1	1.8	135.	117.	.18	44.0	NAv	1.8	3.3	NAv	206.2	104.
2-8	1	1.4	124.	122.	.22	42.0	NAv	1.6	4.3	NAv	212.0	57.
2-10	1	2.0	137.	127.	.33	42.0	NAv	1.2	4.8	NAv	103.5	112.
2-12	1	1.6	115.	99.	.25	41.5	NAv	1.6	3.2	NAv	96.9	143.
2-13	1	.9	83.	90.	.34	45.0	NAv	1.2	3.9	1.0	154.0	84.
2-14	1	1.3	95.	93.	.23	41.5	NAv	5.6	4.9	.6	193.7	26.
2-15	1	1.4	93.	90.	.28	41.0	NAv	1.0	4.5	.7	220.6	98.
2-16	1	1.5	118.	123.	.33	42.0	NAv	3.2	3.8	.5	184.6	62.
2-17	1	1.4	104.	105.	.32	37.0	NAv	1.2	4.3	.8	68.6	38.
2-35	1	1.5	91.	94.	.20	42.0	NAv	9.2	4.8	1.1	208.0	153.
2-9	2	1.7	99.	95.	.18	47.8	NAv	1.0	4.3	NAv	168.7	163.
2-11	2	1.5	96.	87.	.30	43.8	NAv	.6	4.8	NAv	145.2	179.
2-19	2	2.5	93.	80.	.29	41.5	NAv	16.0	4.0	.9	147.5	139.
2-20	2	1.6	109.	110.	.31	43.3	NAv	1.4	4.2	1.4	118.3	85.
2-21	2	1.4	115.	116.	.29	42.7	NAv	1.6	4.7	1.5	233.6	92.
2-27	2	.9	94.	105.	.45	43.5	NAv	12.0	4.4	.9	180.2	144.
2-29	2	1.4	110.	112.	.25	45.8	NAv	2.0	4.3	1.4	206.8	220.
2-31	2	NAv	NAv	NAv	NAv	44.2	NAv	NAv	NAv	1.5	NAv	NAv
2-32	2	1.7	120.	115.	.29	41.4	NAv	1.6	4.5	1.1	92.3	83.
2-33	2	1.7	105.	103.	.37	41.8	NAv	4.2	4.9	2.7	534.9	191.
2-34	2	1.5	95.	96.	.34	NAv	NAv	8.8	4.6	.8	289.1	65.
2-42	2	1.4	99.	NAv	.31	43.1	NAv	10.0	4.7	1.8	464.9	88.
2-43	2	2.3	113.	108.	.16	NAv	NAv	.8	4.9	NAv	312.7	74.
2-44	2	1.2	123.	123.	.27	43.9	NAv	.8	3.5	1.4	118.1	142.
2-36	1B	1.6	108.	111.	.34	45.2	NAv	4.4	2.8	1.9	218.6	128.
2-37	1B	1.0	121.	126.	.31	40.8	NAv	2.2	4.2	1.1	113.4	60.
2-38	1B	1.8	110.	111.	.46	40.9	NAv	1.0	4.5	.8	105.7	62.
2-39	1B	1.7	145.	145.	.41	NAv	NAv	1.2	4.4	1.1	150.0	120.
2-40	1B	.8	116.	119.	.28	NAv	NAv	17.6	4.4	1.0	66.8	52.
2-28	3	1.6	154.	159.	.36	44.3	NAv	1.2	3.6	1.2	204.4	194.
2-30	3	1.4	116.	118.	.36	41.6	NAv	2.4	4.6	1.0	111.3	153.
2-41	3	.9	93.	98.	.36	44.4	NAv	1.4	3.9	1.0	96.7	130.
2-45	3	.5	88.	91.	.42	42.8	NAv	5.0	2.5	1.2	198.6	83.
2-46	3	1.7	114.	108.	.24	41.0	NAv	8.2	3.9	1.0	141.9	65.

* See Table 1, p 33.

(1) Results of Blood Tests at 1615 Hours

Subject Code No	Group*	T_3(n mol/l)	T_4(n mol/l)	FTI(Units)	rT_3(n mol/l)	PCV (%)	Protein(g/l)	hGH(m U/l)	Glucose (m mol/l)	Lactate (m mol/l)	Cortisol (n mol/l)	Prolactin (m U/l)
2-1	1	1.2	128.	136.	.51	43.0	72.	1.6	4.5	NAv	99.9	61.
2-2	1	1.4	98.	98.	.28	39.0	69.	9.0	4.5	NAv	140.9	91.
2-3	1	1.7	158.	149.	.35	38.0	69.	3.2	4.3	NAv	137.0	75.
2-4	1	1.3	110.	105.	.31	39.0	66.	3.8	3.9	NAv	141.0	41.
2-5	1	1.3	116.	111.	.40	45.0	70.	4.2	4.0	NAv	100.1	146.
2-6	1	NAv	NAv	NAv	NAv	38.0	65.	3.0	4.7	NAv	444.8	175.
2-7	1	1.7	163.	137.	.20	43.5	69.	1.6	3.2	NAv	214.0	121.
2-8	1	1.3	118.	132.	.21	43.0	68.	2.0	4.3	NAv	186.4	87.
2-10	1	1.8	134.	119.	NAv	42.5	72.	1.4	4.8	NAv	89.9	78.
2-12	1	2.2	113.	94.	.25	41.5	75.	1.6	4.0	NAv	111.4	151.
2-13	1	.8	62.	72.	.38	45.0	72.	.8	4.3	.9	164.7	78.
2-14	1	1.0	98.	103.	.23	41.0	63.	8.8	4.9	.6	173.3	40.
2-15	1	1.4	90.	89.	.35	41.0	77.	1.0	4.6	.7	211.7	95.
2-16	1	1.6	NAv	NAv	NAv	41.5	68.	7.8	4.0	.6	191.4	58.
2-17	1	1.3	114.	111.	.32	37.0	67.	.8	4.5	.6	57.3	73.
2-35	1	1.4	97.	100.	.17	42.0	65.	12.8	4.7	1.2	290.9	151.
2-9	2	1.6	91.	96.	.20	47.7	78.	.6	4.2	NAv	134.7	139.
2-11	2	1.4	102.	94.	.30	43.6	73.	.6	4.8	NAv	163.1	144.
2-19	2	2.2	104.	90.	.27	42.4	71.	17.2	4.1	.8	131.4	211.
2-20	2	1.8	96.	96.	.30	43.3	74.	1.0	4.0	1.2	96.5	67.
2-21	2	1.6	116.	121.	.30	42.8	67.	1.2	4.8	1.6	220.1	64.
2-27	2	.9	102.	108.	.50	43.9	70.	14.4	4.4	1.1	245.5	149.
2-29	2	1.4	99.	99.	.26	45.7	74.	1.6	4.4	1.3	212.4	160.
2-31	2	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv	NAv
2-32	2	1.6	114.	106.	.36	NAv	66.	1.4	4.4	1.2	106.1	74.
2-33	2	1.7	102.	104.	.30	40.4	72.	3.2	5.0	2.1	494.6	166.
2-34	2	1.2	88.	91.	.28	NAv	67.	5.6	4.5	.9	230.8	43.
2-42	2	1.4	104.	NAv	.32	NAv	67.	8.8	4.7	2.6	443.7	76.
2-43	2	2.0	104.	99.	.15	44.3	71.	.8	4.8	3.6	304.8	71.
2-44	2	1.4	127.	124.	.23	42.0	68.	.8	3.3	1.2	99.2	136.
2-36	1B	1.8	112.	113.	.32	46.3	68.	2.8	2.8	1.6	259.2	99.
2-37	1B	1.0	111.	113.	.30	40.3	61.	2.0	4.5	.9	76.5	69.
2-38	1B	1.8	112.	112.	.42	43.5	66.	.8	4.5	1.0	117.1	61.
2-39	1B	1.7	130.	129.	.40	42.5	70.	1.4	4.4	.9	140.0	124.
2-40	1B	1.0	135.	136.	.22	NAv	65.	9.2	4.4	1.0	75.9	56.
2-28	3	1.6	161.	158.	.40	45.2	67.	1.0	4.2	.9	164.1	141.
2-30	3	1.3	127.	132.	.33	41.0	67.	2.8	4.6	1.2	112.8	117.
2-41	3	.9	90.	94.	.31	44.5	62.	1.4	3.8	1.2	95.1	125.
2-45	3	NAv	86.	89.	.44	42.3	68.	8.0	2.5	1.1	179.8	84.
2-46	3	1.3	99.	95.	NAv	40.0	69.	6.0	3.9	.9	86.8	55.

* See Table 1, p 33.

(m) Results of Urinary Hormone Tests, Urine Flow, Creatinine Clearance and Serum Osmolality Before the Laboratory Study

Subject Code No	Group*	UFC (p mol/min)	UCA (p mol/min)	Urine Flow (ml/min)	Creatinine Clearance (ml/min)	Serum Osmolality (mosmol/l)
2-1	1	301	363	1.14	163	300.2
2-2	1	270	209	2.11	73	302.8
2-3	1	293	223	2.11	114	298.7
2-4	1	122	561	0.72	161	301.3
2-5	1	198	59	1.74	66	297.6
2-6	1	164	343	1.02	141	298.4
2-7	1	159	449	0.65	95	303.4
2-8	1	160	138	1.07	88	304.0
2-10	1	507	325	1.47	106	297.7
2-12	1	151	385	0.76	87	302.5
2-13	1	306	539	1.14	171	295.5
2-14	1	87	129	0.52	91	301.2
2-15	1	197	177	1.83	180	293.4
2-16	1	132	300	0.74	133	297.4
2-17	1	135	291	0.83	108	304.0
2-35	1	217	121	0.97	144	297.0
2-9	2	333	466	0.85	117	295.9
2-11	2	230	478	0.86	130	300.8
2-18	2	205	355	0.56	NAv	NAv
2-19	2	NAv	NAv	NAv	NAv	294.9
2-20	2	153	196	0.94	143	290.0
2-21	2	331	413	1.20	187	296.8
2-27	2	278	229	0.67	89	290.1
2-29	2	246	145	2.28	106	288.8
2-31	2	112	154	0.57	83	300.4
2-32	2	137	187	0.55	143	299.6
2-33	2	213	223	0.82	127	306.7
2-34	2	111	215	0.79	127	295.0
2-42	2	207	213	0.77	95	301.6
2-43	2	316	347	0.88	106	302.9
2-44	2	197	151	1.89	160	298.9
2-36	1B	89	239	0.61	54	306.6
2-37	1B	116	230	0.48	73	305.5
2-38	1B	72	195	0.48	85	301.0
2-39	1B	249	185	0.88	79	302.6
2-40	1B	84	284	0.42	102	304.7
2-28	3	211	284	0.98	151	289.4
2-30	3	155	236	1.08	142	291.7
2-41	3	184	644	1.07	185	297.1
2-45	3	87	249	0.36	170	290.8
2-46	3	150	303	0.75	139	302.3

* See Table 1 , p 33.

(n) Results of Urinary Hormone Tests, Urine Flow, Creatinine Clearance and Serum Osmolality During the Laboratory Study

Subject Code No	Group*	UFC (p mol/min)	UCA (p mol/min)	Urine Flow (ml/min)	Creatinine Clearance (ml/min)	Serum Osmolality (mosmol/l)
2-1	1	150	295	0.72	99	297.0
2-2	1	319	242	3.11	198	295.0
2-3	1	160	211	1.26	206	297.7
2-4	1	99	537	0.70	129	298.4
2-5	1	173	181	2.38	121	295.8
2-6	1	178	295	0.41	147	295.5
2-7	1	171	458	0.96	125	298.2
2-8	1	95	119	0.87	82	302.2
2-10	1	403	243	4.09	125	294.5
2-12	1	99	491	1.13	97	297.0
2-13	1	242	697	1.27	164	300.8
2-14	1	194	492	0.99	167	305.6
2-15	1	117	93	2.74	106	297.6
2-16	1	116	251	0.73	135	303.0
2-17	1	55	281	0.70	136	308.6
2-35	1	216	229	0.93	102	293.6
2-9	2	302	179	2.00	127	291.0
2-11	2	172	249	1.51	119	295.1
2-18	2	214	435	0.63	NAv	NAv
2-19	2	NAv	NAv	NAv	NAv	298.6
2-20	2	108	241	0.92	106	295.9
2-21	2	314	323	1.01	148	297.8
2-27	2	399	377	0.78	162	290.1
2-29	2	114	339	0.93	148	293.3
2-31	2	97	131	0.53	120	NAv
2-32	2	98	217	0.66	135	299.7
2-33	2	382	278	0.94	135	300.0
2-34	2	182	164	0.47	90	295.2
2-42	2	346	156	0.56	177	298.8
2-43	2	411	987	1.43	193	298.7
2-44	2	174	187	2.31	162	291.6
2-36	1B	189	308	1.38	138	304.7
2-37	1B	130	285	1.60	100	301.8
2-38	1B	106	470	0.83	150	298.4
2-39	1B	188	197	3.69	148	300.3
2-40	1B	79	327	0.66	70	296.8
2-28	3	354	252	1.38	177	288.6
2-30	3	222	113	3.75	188	288.9
2-41	3	115	593	0.95	125	293.9
2-45	3	86	283	0.65	165	288.1
2-46	3	107	285	0.88	195	295.8

* See Table 1 , p 33.

(o) Urinary Hormone Results (n mol/d)

SUBJECT CODE NO	GROUP*	EXPERIMENTAL WEEK-END				CONTROL WEEK-END			
		Saturday		Sunday		Saturday		Sunday	
		UFC	UCA	UFC	UCA	UFC	UCA	UFC	UCA
2-1	1	259	294	232	459	172	321	111	255
2-2	1	247	297	196	205	NAv	NAv	NAv	NAv
2-3	1	230	504	146	493	137	456	89	386
2-4	1	131	523	124	799	NAv	NAv	NAv	NAv
2-5	1	150	159	189	129	149	914	86	101
2-6	1	166	579	180	359	158	430	122	567
2-7	1	117	367	221	700	79	237	NAv	NAv
2-8	1	162	492	137	200	193	353	106	216
2-10	1	290	335	114	329	217	230	146	408
2-12	1	214	666	165	300	117	145	165	488
2-13	1	141	616	275	998	89	290	64	335
2-14	1	144	376	145	408	200	649	138	485
2-15	1	271	257	143	340	194	382	240	255
2-16	1	222	629	274	637	242	531	205	597
2-17	1	105	657	104	455	NAv	NAv	NAv	NAv
2-35	1	251	487	333	267	232	432	233	299
2-9	2	302	355	230	342	NAv	NAv	159	215
2-11	2	200	278	247	294	190	434	NAv	NAv
2-18	2	294	484	239	617	165	494	323	383
2-19	2	306	368	133	253	520	376	202	473
2-20	2	159	444	209	533	134	635	208	321
2-21	2	295	259	222	284	203	325	192	537
2-27	2	321	433	249	276	200	400	148	249
2-29	2	210	307	206	173	238	NAv	139	244
2-31	2	160	141	122	213	223	271	187	475
2-32	2	172	241	154	307	175	218	219	264
2-33	2	223	239	149	225	192	195	176	273
2-34	2	185	342	129	255	205	328	137	135
2-42	2	329	341	274	564	148	549	198	435
2-43	2	508	323	140	578	112	407	NAv	NAv
2-44	2	158	247	239	315	NAv	NAv	NAv	NAv
2-36	1B	202	301	95	239	110	501	108	249
2-37	1B	176	209	121	473	175	339	174	NAv
2-38	1B	119	372	106	262	NAv	NAv	NAv	NAv
2-39	1B	279	237	174	323	NAv	NAv	NAv	NAv
2-40	1B	97	405	106	262	87	298	95	162
2-28	3	213	252	133	242	163	211	103	174
2-30	3	194	534	166	168	151	654	195	305
2-41	3	157	535	173	802	NAv	NAv	187	450
2-45	3	161	349	175	288	291	962	320	1418
2-46	3	148	470	94	433	105	633	87	183

* See Table 1 , p 33.

D. TM Residence Course Study

(a) Subject Characteristics

Subject Code No	Sex	Age	TM Experience (yr)
1-87	F	22.8	3.7
1-91	M	26.8	3.7
1-104	M	40.6	1.8
1-105	F	28.6	4.3
1-106	M	21.7	2.1
1-107	F	25.1	1.3
1-111	M	48.3	1.1
1-113	F	22.8	1.2
1-114	F	25.5	3.2
1-103	M	28.0	0.6
1-108	M	19.3	0.3
1-109	F	21.4	0.4
1-110	F	20.5	0.5
1-115	M	28.9	0.6
1-116	F	28.8	0.5
1-117	M	18.7	0.5
1-118	M	21.9	0.1

(b) Urinary Hormone and Urinary Volume Results

Subject Code No	Meditation Experience	UFC (n mol/d) Sat	Sun	Mon	UCA (n mol/d) Sat	Sun	Mon	Urine Vol (ml/d) Sat	Sun	Mon
1-87	experienced	130	62	139	169	164	137	556	943	696
1-91	experienced	211	139	138	109	168	363	1905	1805	1025
1-104	experienced	204	181	NAv	459	314	NAv	1070	670	NAv
1-105	experienced	116	73	NAv	246	177	NAv	535	770	NAv
1-106	experienced	219	197	163	429	238	276	975	1275	525
1-107	experienced	175	105	104	128	139	197	990	710	485
1-111	experienced	218	139	NAv	283	207	NAv	480	775	NAv
1-113	experienced	142	121	112	156	202	251	1170	1050	700
1-114	experienced	186	131	NAv	55	175	NAv	1050	585	NAv
1-103	novice	203	203	130	251	142	41	625	745	515
1-108	novice	186	157	170	287	298	311	690	775	810
1-109	novice	117	144	144	96	109	254	1320	1735	865
1-110	novice	226	81	190	726	194	183	1870	380	925
1-115	novice	287	363	NAv	224	431	NAv	2340	1960	NAv
1-116	novice	168	124	NAv	205	180	NAv	545	500	NAv
1-117	novice	175	195	228	369	388	371	1300	750	660
1-118	novice	204	250	197	816	663	824	1185	1285	685

APPENDIX V: PUBLICATIONS

Bevan, A.J.W., Young, P.M., Wellby, M.L., Nenadovic, P. and Dickins, J.A. Endocrine Changes in Relaxation Procedures. *The Endocrine Society of Australia Proceedings 19*:59, 176.

Both the sympatho-adrenomedullary and adrenocortical systems are activated in response to stress. Thyroid function is also probably susceptible to stress(1). Benson(2) has proposed the term "relaxation response" to describe a common integrated physiological reaction underlying the practice of relaxation techniques. According to Benson the relaxation response is the counterpart of Cannon's "fight or flight" stress response.

The following 3 studies were performed on normal subjects to examine the possible endocrine effects of various relaxation procedures.

1) Chronic endocrine effects were investigated on subjects supervised in Transcendental Meditation (n=7), progressive relaxation, autohypnosis (n=10) and yoga-meditation (n=7) before training and at regular intervals over 8 months during training. A control group (n=12) which had no specific relaxation instruction and a group of experienced (> 1 year of practice) Transcendental Meditation (TM) subjects (n=8) were studied concurrently.

2) Acute endocrine effects were studied in all groups by serial blood sampling before and after a half-hour Sunday mid-afternoon relaxation session. Urine samples were collected over the same weekend and the following Monday.

3) The third study was conducted during a TM residence course on subjects practising intensive meditation. Both experienced (n=9) and novice (n=7) TM subjects were investigated.

Plasma and urinary free cortisol (UFC) were measured by competitive protein binding, urinary catecholamines (UCA) by fluorimetry and serum thyroxine (T_4) and triiodothyronine (T_3) by radioimmunoassay.

Results: 1) During the 8 months no temporal endocrine trends relating to the relaxation technique practices were apparent for all groups. However, overall UFC levels of experienced TM subjects were lower than those of novice TM subjects ($p < 0.05$). A combined analysis of both TM groups revealed a negative correlation between TM experience and UFC. No significant differences were found between UFC levels of novice meditators and controls. No significant group differences in UCA occurred.

2) Short-term pre-relaxation versus post-relaxation comparisons revealed a decrease in plasma cortisol following meditation in the experienced TM group ($p < 0.0005$). No significant plasma cortisol changes were found for all other groups. T_4 levels fell immediately following meditation in the experienced TM group ($p < 0.05$), however, no significant changes in T_3 were observed.

Analysis on UFC and UCA revealed a general fall-off from Saturday to Sunday with a rise on the following Monday although these trends were not significant. Overall UFC and UCA levels confirmed the results of the first study.

3) The UFC levels of experienced TM subjects fell significantly from the Saturday to the Sunday of the TM residence course ($p < 0.0005$). Levels remained lower on the following Monday ($p < 0.0025$). Novice meditators showed a similar pattern. However, these results were not statistically significant. No significant changes in UCA occurred over the weekend.

Conclusions: The most obvious observations were the highly significant decreases in plasma and urinary free cortisol during TM, the effect being cumulative with increased meditation experience. However, there were no significant effects on catecholamine excretion. The changes in thyroid function are difficult to interpret without further observations but effects on peripheral metabolism of T_4 and T_3 have to be considered.

The results suggest that TM produces a marked acute and chronic reduction in trophotropic anterior hypothalamic activity but little effect on ergotropic posterior hypothalemic function. Although the endocrinological effects of TM appear to denote the removal of stress, the mechanisms underlying the practice are not a simple counterpart of Cannon's fight or flight response. Therefore Benson's concept of the relaxation response(2) is probably not applicable to TM.

(1) Hetzel, B.S., D.S. De La Haba and L.E. Hinkle, Jr. Trans. Am. Goitre Assoc. 1952, p. 242.

(2) Benson, H., J.F. Beary and M.P. Carol. Psychiatry 1974, 37:37.

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Transcendental Meditation (TM) is widely practised as a relaxation procedure for its stress reducing effects and has been reported to be characterized by a hypometabolic physiological state (Wallace et al, 1971). There are no observations of significant endocrine changes associated with TM except for decreased cortisol and increased prolactin levels.

The purpose of the study was to examine short-term changes in some serum hormone levels associated with stress. Fifteen experienced male meditators were examined. Frequent serial blood samples were taken via indwelling venous catheter before, during, and after a 30 minute mid-afternoon meditation. Nine non-meditator male controls were observed under comparable experimental conditions before, during, and after 30 minutes of ordinary relaxation.

Serum growth hormone (GH), triiodothyronine (T_3), reverse triiodothyronine (rT_3), and thyroxine (T_4) were measured by radioimmunoassay. Serum cortisol was assayed by competitive protein binding.

Pre-meditation or pre-relaxation concentrations of GH and cortisol were higher in the control group than the meditators. Falls in serum GH, cortisol and T_3 occurred during meditation ($p < 0.05$, d.f. > 9, paired t-tests). Changes observed in non-meditator controls were inconsistent and less significant. Levels remained low following meditation and tended to gradually return towards pre-meditation values. No significant changes in T_4, free T_4 index, rT_3, haemoglobin, packed cell volume and total serum proteins were found.

The three hormones GH, cortisol and T_3, which increase with stress, fall significantly during TM in contrast with ordinary relaxation. These changes might be mediated by alterations of hypothalamo - anterior pituitary function in TM.

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Transcendental Meditation (TM) is widely practised as a relaxation procedure for its stress-reducing effects and has been found successful in the treatment of stress-related disorders and in the management of stress reactivity. Significant reductions of cortisol and growth hormone (hGH) in experienced meditators in contrast with non-meditator controls have been reported previously (1, 2). However, some of these differences might be attributable to individual variation or to differential experimental stress effects.

Hence, this study was designed to reinvestigate possible endocrine bases for the effects of TM on stress, giving due consideration to the above mentioned independent variables.

All subjects were day-active, healthy, Caucasian, young-adult, male volunteers. Five experienced meditators were studied before, during and after a 30 minute mid-afternoon meditation (group 1A). The same five meditators were restudied under the same experimental conditions except that instead of meditating, they read and talked quietly amongst themselves throughout the period of observation (group 1B).

Restudying meditators created a "second experience" effect which might significantly influence endocrine changes. Therefore, a comparable group of five previously unstudied meditators were examined under the same non-meditation conditions as for group 1B (group 2).

Frequent serial blood samples were taken via indwelling venous catheter during the afternoon. Serum hGH, prolactin, thyroxine (T_4) triiodothyronine (T_3) and reverse triiodothyronine (rT_3) were measured by specific double antibody radioimmunoassay. Serum cortisol was assayed by competitive protein binding using charcoal separation. Sephadex uptake of T_3 was used to derive free T_4 index values (FTI).

A significant 38% reduction in serum GH occurred during TM (group 1A) ($p < 0.0025$; paired t-test). The GH fall commenced before the onset of meditation and appeared to be a response in anticipation of meditation. Serum hGH concentrations after TM rebounded to 50% above premeditation values.

Group 1A also showed slight decreases in prolactin and cortisol during meditation, which were not statistically significant. There were no statistically significant changes in T_4, FTI, T_3, rT_3 nor in haemoglobin, packed cell volume or total serum protein during the experimental period.

No statistically significant changes in any test were observed during the experimental period for both group 1B and group 2.

It is concluded that (1) a significant short-term reduction of hGH occurred during TM in contrast with a lack of change in the same subjects during a comparable non-meditation experimental period, (2) the

absence of hGH changes during this comparable non-meditation experimental period was not due to a second experience effect.

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