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PREFACE

The present publication summarizes the background, methods and the main results of following reports:

(I) Sarviharju, P. J., M. E. Huikko, P. I. Jouppila and N. T. Kärki: Effect of endurance training on the urinary excretion of noradrenaline and adrenaline during ground and flying activity

Aerospace Medicine, 42 (12), 1297—1302, 1971

(II) Sarviharju, P. J.: Kestävyysharjoittelun vaikutukset eräisiin biokemiallis-fysiologisiin muuttujiin tietyissä psykofyysisissä kuormitustilanteissa (Effect of endurance training on some biochemical and physiological variables during psychophysical loadings. An English abstract.)

Public Health Publication no. 13, University of Jyväskylä, 1972

(III) Sarviharju, P. J.: Effect of physical exercise on the urinary excretion of catecholamines and 17-hydroxycorticosteroids in young healthy men

J. Sports Med. and Phys. Fitness, 1972. In press.

(IV) Sarviharju, P. J. and V. Vihko: Plasma FFA during psychophysical loading and endurance training

J. Sports Med. and Phys. Fitness, 1972. In press.

(V) Sarviharju, P. J. and E. Mattila: Urinary excretion of catecholamines during psychomotor choice reaction test before and after endurance conditioning

Public Health Publication no. 14, University of Jyväskylä, 1972

(VI) Sarviharju, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning

Public Health Publication no. 15, University of Jyväskylä, 1972

In the text these will be referred to by Roman numerals (I)—(VI). A part of my design and a minor part of the results of my work (II) in a modified form have been previously reported by Kirjonen and Nieminen (1970), Kirjonen and Rusko with Sarviharju (1971) and Kirjonen (1971). The purpose of the present work is to survey the six papers mentioned as well as propose aspects for the construction of a theoretical framework. The studies included in this paper were carried out at the Department of Biology of Physical Activity (I—VI), the Department of Public Health (II—VI), University of Jyväskylä, the Department of Pharmacology, University of Oulu (I) and the Department of Anatomy, University of Turku (II). Furthermore, help was provided by the Headquarters of the Finnish Air Force (I—II), Air Force Flying School (I—II), the Signal School of the FAF (I—II), and, in particular, all the military aviation courses and their instructors (I—II) as well as the numerous students of the University of Jyväskylä (III—VI) and many others inspiringly participating in my project.

The first work was initiated in 1965 and its progress was followed intensively by Professor Esko Karvinen, M.D., Ph.D., Head of the Department of Biology of Physical Activity, University of Jyväskylä and by Professor Niilo Kärki, M.D., Head of the Department of Pharmacology, University of Oulu. I am particularly grateful for their encouragement and advice. Following the study (II) in all phases of the work, I have received much enthusiastic and valuable advise, criticism and support from Professor Jeddi Hasan, M.D., Head of the Department of Public Health, University of Jyväskylä.

My thanks are due to Professor Leo Hirvonen, M.D., Head of the Department of Physiology, University of Oulu and Professor Esko Iisalo, M.D., Head of the Department of Pharmacology, University of Turku, who have revised the manuscript and offered valuable criticism. My thanks are also due to Professor Raimo Konttinen, Ph.D., University of Jyväskylä, for his advice concerning the statistics. I wish to express my thanks to Veikko Vihko, M.Sc., for his great contributions in chemistry and chemical determinations (II—VI); Colonel Kalervo Mustonen, Colonel Reino Nykänen, Heads of the Air Force Flying School, Licutenant-Colonel Juhani Leinonen, Head of the Signal Flying School of the Air

Force, Professor Mikko Niemi, M.D., Head of the Department of Anatomy, University of Turku and their personnel for contributing both directly and indirectly to the final realization of the project.

The various phases of this work have profited greatly from contributions by Miss Satu Hannula, Miss Liisa Kemppinen, Miss Anja Kiiskinen, Mr. Pentti Korhonen, Mr. Olavi Manninen, Mr. Erkki Mattila, Miss Leena Multamäki, Miss Kirsti Vaarna (chemical determinations), Mr. Aarno Sorsa (recording and test apparatus), Mr. Erkki Jääskeläinen, Mr. Alvar Koppinen, Mr. Erkki Lehtinen, Mr. Keijo Lipén, Miss Annikki Poutiainen and Mr. Jyrki Toiviainen (programming), Miss Patricia Baker, Mr. Andrew Chesterman, Mrs. Liisa Havola-Pitkänen, Mr. Glyn Hughes, Mr. Anthony P. May, Miss Malle Vörk (English revision), Mrs. Pirkko Audejev, Mrs. Raija Hasan, Mrs. Anneli Kaarniemi, Mrs. Onerva Kotilainen, Mrs. Leila Kärkkäinen, Mrs. Maire Laine, Mrs. Sisko Lemettinen, Miss Ritva Makkonen, Miss Aulikki Penttinen, Miss Pirkko Puttonen, Mrs. Kaija Raulo, Mrs. Inga Soininen, Mrs. Helena Suominen, Miss Sirpa Taipale, Mrs. Liisa Vihko, Miss Pirkko Änkilä (technical assistance and typing) and many others.

All studies, excluding study I, were supported financially by grants from the Finnish Research Council for Physical Education and Sports (Ministry of Education). In 1970 I was awarded a grant for young scholars by the University of Jyväskylä and in 1971 a grant from the Foundation of Eine and Artturi Nyyssönen. I thank the University of Jyväskylä for the acceptance of my summary for its series of Studies in Sport, Physical Education and Health.

Jyväskylä, October 1972

Pekka J. Sarviharju

INTRODUCTION

The present task was to study the stress response of man in relation to his physical fitness. As an introduction to this problem the background of this area will first be reviewed with regard to the concept of stress and the suggestions about the overall significance of the study of stress and physical activity.

View of milieu interactions and stress

Due to the wealth of current data available, it seems obvious that experiences, emotions, mental and physical aberrations, i.e. various kinds of mental and physical loadings in numerous qualitative and quantitative combinations, are all accompanied by great changes in the internal milieu of the human body. The resultant state of an organism is often termed stress.

In the stress regulating functions, it has been suggested that the nervous and endocrine systems are interrelated, interact and co-operate both structurally and functionally to sustain the homeostasis of the organism.

Much of the data available suggests that the stimuli from the environments affect the central nervous system in such a way that they cause a change in the output of the neuroendocrine axis, involving especially the hypothalamus, the hypophysis, the target glands and tissues as reviewed, for example, by Bajusz (1969). There are two functional areas connected with this complex of various systems which are often measured in experimental endocrinology by relatively accurate biochemical techniques. These functions, very often measured in only one point of the subcomplex, are (1) the neurohormonal adrenergic and (2) the adrenocortical secretory reactions known to be elicited by experimental and reflectory stimulations of the hypothalamic centers, which activate (1) the adrenergic system via sympathetic pathways to liberate catecholamines, and (2) the secretion of adrenocortical 17-hydroxy-

corticosteroids via bloodborne ACTH, as suggested by many authors and reviewed, for example, by Raab (1968). Exposures to many kinds of environmental stimuli are widely known to be reflected, at least quantitatively, in both of these hormone discharges. Consequently, the catecholamines and 17-hydroxycorticosteroids were selected as parameters in this project to indicate the psychophysical strain on an organism.

Many authors suggest that the urinary noradrenaline and adrenaline are particularly well suited to quantitative stress evaluaions on human subjects and they play an important role in current trends of experimental endocrinology (cf. Euler, 1964; Schildkraut and Kety, 1967; Frankenhaeuser, 1971). Similarly the steroid excretion was accepted as a valid measureable parameter of an organism's response to stress (e.g. Bourne, 1969).

The interpretation of the results makes it necessary to consider the fact that undoubtedly these endocrine functions do not account for all the important aspects of modulation processes of the internal state and may not even be primary in genesis. Recent advances in several fields of experimental endocrinology have yielded information suggesting that endocrine regulation is very likely to be organized on a broad basis and that multiple endocrine systems, in addition to those involving the adrenal gland, may participate in endocrine responses.

Such terms as »physical stress», »emotional stress», or »psychological stress» are used to describe a variety of states which may have a lot or a little in common. A dissecton of the meaning of these terms could proceed along anyone of several lines. The definitions of stress seem quite contradictory; in engineering one refers to stress as any external force directed at some physical object. This force will result in strain, the temporary or permanent alteration of the object. The analogy in psychology and physiology refers to an external stimulus or agent as »stress» and the resulting effect as »strain». This analogy is generally implicit in the use of the term. Thus »stress» can have the meaning of some unusual condition (Grinker and Spiegel, 1945) or a condition disturbing normal functioning (Arnold, 1960). The definition is often similar in physiology with the exception of the classical terms used by Selye (1950), who defined stress as the »state manifested by a

specific syndrome which consists of all the nonspecifically induced changes within a biologic system» or as the »rate of wear and tear in the body». In this usage the stimulating conditions are called stressors.

Lazarus (1971) points out one of the fundamental features of stress as a concept that refers to the relations between an organism and the environment rather than to just the organism or just the environment. He emphasizes that the stimulus is just as important an element as is the reaction and is to be regarded as part of the definition. Lazarus (1971), states that the subtopic of stress, psychological stress, is to be distinguished from the physiological one in that in the former the reaction depend on conscious or unconscious personal interpretation or appraisal of the significance of a harmful, threatening or challenging event, while in the latter it is the condition of the tissues which directly determines noxiousness. The stress reaction is psychologically determined by mediating cognitive processes consisting of direct actions or reappraisal (Lazarus 1966, 1971; Lazarus et al., 1971). Among the many approaches to the stress concept one of the broadest and, therefore, most profitable seems to be the view of man as a problem-solving phenomenon proposed by Howard and Scott (1965). They feel that problems as caused by external or internal stimuli on the one hand and on the other by symbolic or nonsymbolic stimuli.

A general approach to studying stress is isolation, at least in part of a particular kind of stressor and studying its effects. The aim herein was, under quite obvious restrictions, to select certain acute external stressors in order to achieve quantitatively and qualitatively different combinations of psychophysical loading. The term loading refers herein to the external stimuli and stress on change in the internal state.

The purpose of the present study

The evaluation of the interactions of the regulatory mechanisms under stress continues to be a problem of special interest due to two lincs of thought. The first is the point of view of public health. The reversibility and/or irreversibility of the consequences

due to intense acute and especially chronic interactions during various kinds of every-day life exposures would then be problematic with regard to human well-being. It has been suggested that these burdens are especially pathogenic in predisposed individuals (e.g. Wolff, 1953; Tanner, 1960; Raab, 1966). Therefore, for example, the assumed and reviewed implications of neurogenic and hormonal responses could be relevant in human myocardial pathology in view of the continuous positive data about the potential metabolic cardiotoxicity of the two combined functions, namely (a) adrenergic catecholamines which potentially necrotize and deplete the potassium in the myocardium and (b) the simultaneous overaction of glucocorticoids which deplete potassium equally (Selye, 1958; Raab et al., 1961; Bajusz, 1963; Raab, 1968). Those implications are suggested, besides animal experimentation, by the statistical evidence of a high death rate caused by degenerative heart disease in occupationally strained (Russek, 1962) and emotionally excitable (Friedman et al., 1960) persons.

The other argument possibly relevant to human well-being is the role of physical and mental performance; the indistinct role of hormonal individuality and capacity in the psychophysical performance in acute and chronic situations of everyday work which possibly becomes accentuated under the often exceptionally overwhelming loadings of warfare. Some evidence suggests different arousal functions of noradrenaline and adrenaline, while noradrenaline may be related to mechanisms concerned with focusing the attention upon specific features of a complex stimulus situation (Patkai, 1967) and correlating positively with improvement in performance in mental performances during stress (Frankenhaeuser and Patkai, 1964).

One of the most common exposures having obviously potent acute manifestations is physical work, e.g. sport activities, the importance of which, when regularly exercised, lies in the maintaining of a good physical fitness. The interest in this area, however, has mainly been concentrated on the direct measuring of the respiratory and cardiovascular functions. A problem will be the actual situation where we are short of systematic data about the effects of regular physical activity on the regulatory functions

in an organism, and where and how to measure these functions of a complex nature.

In view of the interdisciplinary nature of the research of physical activity, the topic discussed should receive much attention due to the implications of prolonged exercise and lack of exercise. It is suggested that this kind of intensive physical training is generally followed by an increase in the parasymphathetic tone and/or decrease in the sympathetic one; these states are normally characterized as cardiac vagal tone causing a progressive bradycardia, high efficiency of the heart muscle (e.g. Knehr et al., 1942; Reindell et al., 1954; Mellerowicz, 1956; Jokl, 1959), coronary and pulmonary vasoconstriction, vasodilatation elsewhere, increased peristaltics and glandular function in the stomach and intestines and increased tone in the skeletal muscles (e.g. Nöcker, 1971). On the other hand a marked cardiac acceleration in healthy subjects has been observed in many studies after a prolonged voluntary bedrest (e.g. Miller et al., 1964).

On the level of performance, regular intensive physical training will result in an increased ability to continue prolonged physical activity. The term endurance may be the definition of this ability (Davis et al., 1965). In the present study the terms 'endurance training' and 'conditioning' will be used as synonyms.

Arguing from the presented background it seemed quite valuable with regard to the exercise physiology and the general physiology theories to elucidate by repeated experiments, and confirm each time with quite similar design considerations, the following:

(a) the importance of certain psychophysical stimulations in the activation of the sympathetic adrenomedullary and pituitary adrenocortical hormone secretion and certain changes in blood parameters

(b) the effect of regular progressive physical activity on selected endocrine and blood parameters

The main aim of this study which guided the design was the effect training had on the selected parameters. The investigator tried experimentally, under control, to disturb the prevailing psychophysical balance of the human organism during relative rest, and to study those endocrine and other selected biochemical reac-

tions in relation to physical conditioning, which in itself could be seen as a chronic imbalancement of various regulatory mechanisms of an organism. Thus, an attempt was made to experimentally operate in a very complex area, where a great amount of literature has been devoted, on the one hand, to the endocrine disorganization effects of loading and on the other hand, to a huge extent to various cardiac, circulatory, pulmonary, metabolic, physical performance manifestations of physical conditioning, and where we have only little systematic evidence of the relationship in question and its possible importance for vital functions. This study has been very painstaking due to the great lability and individuality of endocrine reactions and consequent difficulty in the standardization of measurements.

Except for some essential evidence presented about the main parameters in each separate report, the details on the backround of the topic will be discussed. These will concern the nature, biogenesis, action and metabolism of studied hormones in sympatho-adrenomedullary and pituitary adrenocortical systems and the system functions during exposure to psychophysical loading and physical conditioning.

Sympatho-adrenomedullary system

Nature, biogenesis and metabolism of catecholamines

Following the many discoveries on the adrenomedullary hormone »adrenaline» along with the first formulation of the concept of chemical neurotransmission during the first years and the early part of this century, new data made it necessary to reassess adrenomedullary function. This occurred after the demonstration of the natural occurrence of noradrenaline in the body by Euler (1946) and Holtz (1947) and after the establishment of its role both as an adrenomedullary hormone and transmitter substance of adrenergic nerves as reviewed by Euler (1955).

Further histochemical studies indicated that the medullary catecholamines are contained in the cytoplasmic granules of the chromaffin cells, two types of cells secreting predominantly noradrenaline or adrenaline (Hillarp and Hökfelt, 1953; Eränkö, 1955). These cells are in close connection with the preganglionic fibers of the sympathetic nervous system and the secretion appears to be controlled completely by stimulation through the splanchnics, the section of which will prevent any secretion (Cannon an Rosenblueth 1937). About twenty per cent of the catecholamine content of the adrenal medulla in man is calculated to be noradrenaline secreted as an endocrine hormone (Starling and Lovatt Evans,

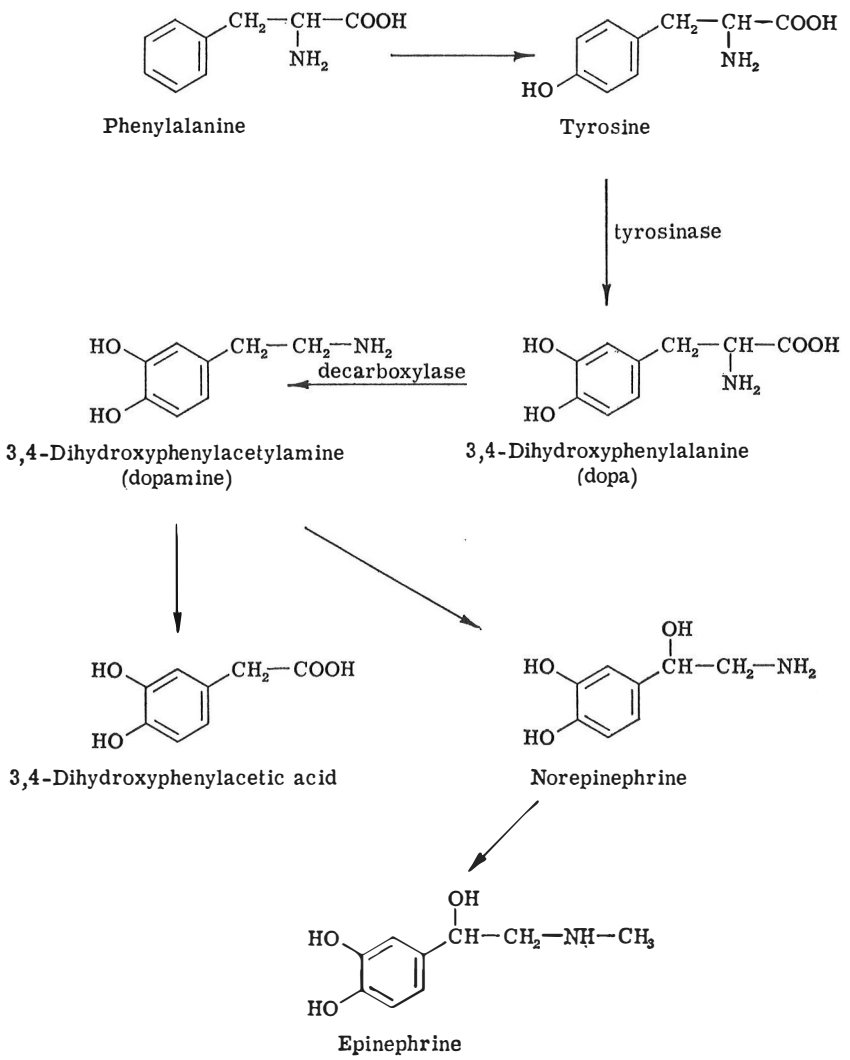

Fig. 1. The biosynthetic pathway for the formation of noradrenaline and adrenaline. From Blascko (1959).

1968). Small quantities of catecholamines may be produced by chromaffin cells in other organs than the adrenal medulla.

Due to prolific evidence since Euler's (1946) discoveries among others and reviewed, for example, by Iversen (1967 a), the current

view seems to be that the more important function of noradrenaline is its role as the only transmitter substance in adrenergic nerves in most mammals and that the traces of adrenaline found in many peripheral tissues are not connected with adrenergic neurotransmission (Euler, 1959).

The use of the fluorescence histochemical method of Falck (1962) has confirmed that the noradrenaline present in peripheral mammalian tissues is located almost exclusively in the sympathetic nerve terminals (Falk and Torp, 1962). Besides the cholinergic synapses between preganglionic and postganglionic neurones in the sympathetic and parasympathetic systems and between nerve and muscle cells in the parasympathetic and peripheral nervous systems, it is also thought that there are adrenergic and cholinergic synapses between certain neurones of the central nervous system and it is even suggested that dopaminergic synapses occur in the mammalian brain as reviewed, for example, by Iversen (1967 b).

Both the noradrenaline and adrenaline are derivatives of β-phenylethylamine, e.g. derivatives of specific amino acids obtained from the nitrogen pool of the body and their biosynthesis according to Blaschko (1959) and it is presented in Figure 1.

The hormones are rapidly removed *in vivo* through absorption by the tissues and degradation of the molecules (Figure 2) and only about 0.5—6 per cent of the infused catecholamines are excreted in their free form to the urine (cf. Euler and Luft, 1951; Pekkarinen and Pitkänen, 1955; Elmadjian et al., 1958).

Many fluorimetric methods are available for the quantitative estimation of catecholamines in blood as well as in urine (e.g. Crout, 1961; Euler and Lishajko, 1961; for review see Iversen, 1967). The urinary excretion has been shown to reflect the catecholamine release in quite a consistent way (Patkai and Frankenhaeuser, 1964). The reliability of the technique, as handled by a skilled technician, has been presented to lie around .85 to .95 (Frankenhaeuser, 1971).

Action of catecholamines

Adrenaline and noradrenaline have differences in their physiological actions as reviewed, for example, by Kärki (1956), Zarrow

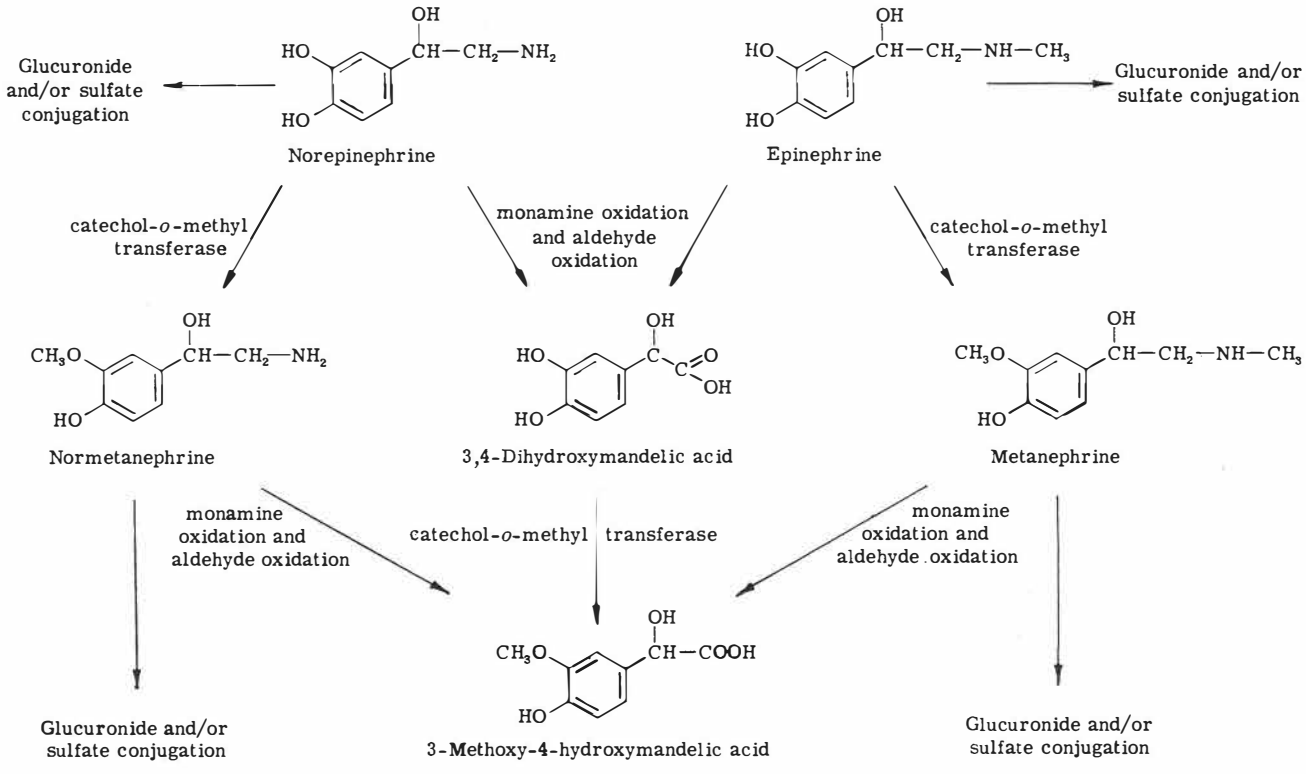

Fig. 2. Metabolic pathways of noradrenaline and adrenaline. From Gitlow et al (1960).

et al., (1964), Euler (1966) and Axelsson (1971). Some of those actions are mentioned herein without reference to the source.

Adrenaline increases both the contractibility and the irritability of the heart, excites the atrioventricular pacemaker in the heart block, and shortens the atrioventricular conduction time on prolonged stimulation, increases the cardiac output and systolic blood pressure with no effect on the diastolic one, elevates pulse pressure with only a moderate increase in the mean pressure, usually decreases peripheral resistance and in large doses induces peripheral vasocontriction.

Noradrenaline is out of action or causes a slight fall in the cardiac output, increases markedly peripheral resistance with a rise in both the diastolic, systolic and mean blood pressure with no effect on pulse pressure. Both these catecholamines increase pulmonary pressure, vasodilate coronary vessels with a resulting increase in the coronary blood flow, relax the smooth muscle of the brochi, constrict skin and the splenic vessels and contract the spleen.

Adrenaline increases the blood flow through the skeletal muscle and liver. It decreases the flow slightly in the kidney and brain as does noradrenaline. The net peripheral vascular effect seems to be vasodilatation due to adrenaline and vasoconstriction due to noradrenaline with a limited vasodilative action. Adrenaline also has a marked decrease in the motility and tone of the gastric and intestinal musculature, contracts the sphinchters and inhibits the secretion.

With dosages insufficient to elevate the blood pressure, adrenaline has a marked hyperglycemic effect as a result of three actions: it increases the rate of glycogenolysis in the liver and thus the output of glucose into the circulation; it also takes part in the glycolytic process of the muscle, possibly indirectly, by causing a release of ACTH from the hypophysis, which results in an increased release of adrenal corticoids extremely potent in stimulating glycogenesis. In contrast to adrenaline, noradrenaline has a very small effect on the oxygen consumption in man (e.g. Goldenberg, 1951).

Exogenous noradrenaline increases the FFA concentration in blood (e.g. Havel and Goldfien, 1959) and it has been suggested

that the increased sympathetic activity is responsible for the lipid mobilization possibly via the release of noradrenaline at the sympathetic nerve endings within adipose tissue (Havel et al., 1963). However, it has been suggested that noradrenaline is of minor importance in the mobilization of fats *in vivo* (Carlsten et al., 1965).

System during loading and conditioning

Cannon in 1915 was the first author to elucidate the principal functions of the adrenal medulla, when he introduced his emergency function theory based upon the view that the many physiological or metabolic consequences of adrenaline release are all directly serviceable in making the organism more efficient in the struggle which fear or rage or pain may involve (Cannon, 1963).

At present a wealth of data demonstrates that the sympathomedullary secretion is increased and followed by an increase in the urinary excretion of noradrenaline and adrenaline due to various stimuli of varying dominance of physical and psychic origin as reviewed in the authors previous papers and by many others, for example, Euler (1964), Levi (1967) and Mason (1968 b). A large inter- and intraindividual variation seems to characterize those responses as well as many other complicating factors requiring accurate standardization in experiments (Levi, 1967).

Physical work of even a moderate degree will stimulate the sympathoadrenal responses and induce an increased excretion of catecholamines into blood and urine as suggested in many studies (e.g. Euler and Hellner, 1952; Kärki, 1956; Gray and Beetham, 1957) even during the first half of this century (cf. Takahashi, 1961). It has been demonstrated since then in studies reviewed by Euler (1969) and confirmed by many others both in the laboratory (e.g. Vendsalu, 1960; Banister, 1966, Lindmar et al., 1968; Conard et al., 1969; Becker and Kreuzer, 1969; De Schaepdrijver and Hebbelinck, 1969; Frankenhaeuser et al., 1969; Howley et al., 1970; Häggendahl et al., 1970; Raven et al., 1970; Kotchen et al., 1971) and under natural experimental conditions (e.g. Ehringer and Spreitzer, 1967; Vuori and Pekkarinen, 1969; Metze and Linke, 1971). On the

other hand we do not know as much about the role of good or poor physical condition in the endocrine excretions in a state of muscular strain. Adrenal medullary hypertrophy has been produced by an intensive physical training program among rats as caused by an increase of both the adrenaline- and noradrenaline-storing parenchyma (Eränkö et al., 1962) and also without any effects on the adrenal weight or its adrenaline content in guinea pigs (Östman and Sjöstrand, 1971). In recent studies when there was no cardiac hypertrophy, a significant reduction in heart catecholamines was noticed after a prolonged physical training (DeSchryver et al., 1967; DeSchryver et al., 1969) but when it resulted in cardiac hypertrophy, the total noradrenaline and its concentration in the heart increased significantly (Östman and Sjöstrand, 1971). Schahab et al., (1972) also report an increase of noradrenaline concentration in the heart muscle of rats after training. This discrepancy in results and the functional significance of the phenomenon are still obscure.

The previous suggestion that a well trained individual should be more strained physically than an untrained one in order to produce an equal increase in the excretion of catecholamines (Euler and Hellner, 1952) is consistent with some evidence concerning the decreasing effect of intensive physical training on the urinary excretion of VMA (Adam et al., 1968). It also parallels the finding in a cross-sectional study that in a submaximal area of physical exercise the ergotropic sympathetic system was obviously less activated in athletic groups than in non-athletic ones, as the athletes excreted less catecholamines during the same submaximal exercise (Metze et al., 1971). In a cross-sectional study an increased excretion of VMA has been found in top sportsmen after the training season (Ostermeyer et al., 1971) and no significant difference during rest between athletes and non-athletes (Letunov et al., 1965). An over-all concept can be visualized by which an certain kind of physical training will increase the parasympathetic tone or decrease the sympathetic one during rest (e.g. Raab et al., 1960; Nöcker, 1971).

Pituitary adrenocortical system

Nature, biogenesis and metabolism of steroids

An organism reacts to the changes in the internal and external environment not only via the hypothalamo-sympathico-adrenomedullary pathway but also via the hypothalamo-hypophyseo-adrenocortical pathway. Since the formulation of Selye's (1950) classical concept of stress, referred to later in this paper, an enormous amount of literature reveals the crucial role of adrenocortical steroids in the maintenance of homeostasis and it has been reviewed by many authors, for example, Ramey and Goldstein (1957), Smelik (1959) and Mason (1968 a).

The three various zones of the adrenal cortex have been pointed out as the sites of origin of the secretions of steroids, which include the sex hormones (progesterone, estrogens and androgens) in addition to the adrenocorticoids, corticosterone or compound B, 17-hydroxycorticosterone or cortisol or hydrocortisone or compound F, 17-hydroxy-11-dehydrocorticosterone or cortisone or compound E and others as reviewed, for example, by Russel, (1960) and Zarrow et al., (1964) (Figure 3).

In man the principal steroid quantitatively is 17-hydroxyform; cholesterol and acetate are the main possible precursors for this and also for the other steroids secreted in minute amounts and rapidly removed from the blood (Russell, 1960). The major site of degradation is in the liver, where the steroids may be esterified to the glucuronide or sulfate or degraded to completely inactive forms (Zarrow et al., 1964).

A very large number of different steroids can be isolated from urine, most of them excreted either as glucuronides or as sulfates and requiring hydrolysis before either a chemical or a biologic estimation (Russell, 1960).

Action of steroids

The adrenocorticoids are classified into two categories which have various physiological functions. The glucocorticoids influence the carbohydrate metabolism providing the amino acid conversion

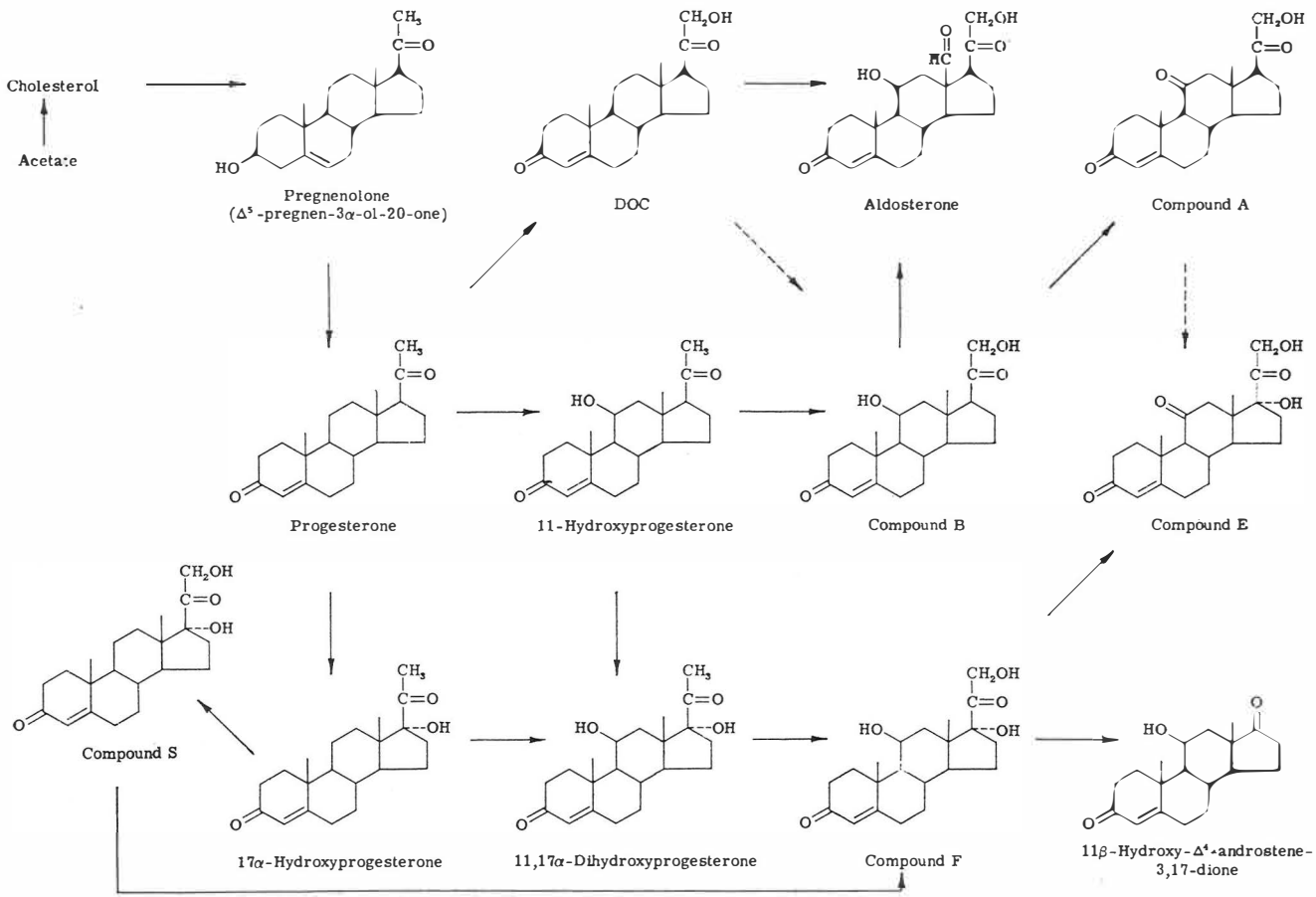

Fig. 3. Biosynthetic pathways for adrenal steroids. From Zarrow et al. (1964).

to carbohydrate, which during administration of glucocorticoids can induce a negative nitrogen balance and a reduced growth rate due to the catabolism of muscle protein (Grollman, 1941). This amino acid conversion coupled with the interference in utilization of carbohydrates by glucocorticoids can produce hyperglycemia symptoms known as the steroid diabetes in a normal animal (e.g. Greenman and Zarrow, 1961). Further, the glucocorticoids inhibit the conversion of carbohydrate to fat and stimulate fat mobilization from the subcutaneous tissues, which result in increased concentration of fat in both the serum and the liver (Zarrow et al., 1964). Some evidence suggests that aldosterone is the main electrolyte regulating the mineralocorticoid hormone of the adrenal gland (e.g. Simpson and Tait, 1955).

The vital role of the adrenal cortex is characterized by the fact that severe muscular work is known to present a fatal stressor to an adrenalectomized animal and generally leads to death within a few days or a few weeks, which is documented by many authors and reviewed, for example, by Ramey and Goldstein (1957); Russell (1960) and Zarrow et al., (1964).

System during loading and conditioning

Following the elucidation of the control of the adrenotropic hormone ACTH from the adrenohypophysis over the secretion of the adrenal cortex, Sayers and Sayers (1948) proposed a humoral feedback theory for the regulation of the ACTH secretion. They demonstrated a quantitative relationship between the degree of inhibition of ACTH secretion and the amount of administered cortical hormones and between the intensity of stress and the required amount of corticoids for inhibition of the ACTH release. The mechanism by which ACTH accelerates the synthesis and release of glucocorticoids is currently suggested to act possibly by providing cofactors which stimulate a dehydrogenase enzyme in corticoid biosynthesis (Cohen, 1961).

Besides the concept of servomechanism there are also supposed to exist other mechanisms to control the ACTH synthesis and release. It has been suggested that an important one is the

hypothalamic corticotrophin releasing factor (e.g. Vernikos-Danellis, 1964). Saba et al. (1963) suggest direct control of the hypothalamus to be possible. Supposedly, the secretion of ACTH is biphasic under loading, when there is a fast neural component and a slower metabolic or hormonal feedback component (Brodish, 1964).

After many early findings, Selye (1950) developed his impressive theory of the General Adaptation Syndrome (GAS) placing the main emphasis on the pituitary adrenocortical system. The GAS was stated to be the sum of all nonspecific systemic reactions of the body, which follow the continued exposure to stress elicited by stimuli such as cold, heat, toxins or radiations, burns, trauma, haemorrhage, overdosage of hormones, muscular work, anoxia and nervous and emotional excitement. The GAS theory states that the first reaction, the Alarm Reaction (A—R) involves the activation of the adrenal cortex with an increased excretion of corticoids in an effort of the organism to acquire adaptation, which is reached in the second stage, the Stage of Resistance (S—R), during which, under the influence of corticoids, survival is maintained until the third phase, the Stage of Exhaustion (S—E), when all the adaptation energy is consumed and death ensues. Several studies have been undertaken in order to evaluate this theory with regard to the development of diseases. One concept presently supported by many authors seems to be the idea of the permissive action of the corticoids instead of the causative role. This concept contains the idea that the cortical hormones are thought to support the normality of tissue and organ responses to other nonadrenal stimuli (e.g. Ingle, 1956; Engel and Fredericks, 1957).

With regard to the role of the GAS theory in interpreting stress reactions, there are obvious limitations in view of the present concept of the many interdependent endocrine systems functioning simultaneously under stress.

Until about the early 1950's most studies dealing with the pituitary-adrenal cortical activity in human subjects involved the measurement of urinary 17-ketosteroids, blood eosinophiles and lymphocytes, urinary electrolyte excretion and a variety of certain urinary metabolites and all of them lacked in specificity such things as indices of the activity concerned (Mason, 1968 a).

Not until the application of chromatographic methods for plasma and urinary 17-hydroxycorticosteroid measurement was a high degree of specificity achieved. Since those days, an essential activation of the adrenal cortex has been demonstrated by numerous authors following after various kinds of mental and/or fairly severe physical strains such as trauma, physical and surgical procedures directed to a human being or an animal as reviewed recently by Mason (1968 a). He also reviews a lot of data suggesting that the most intensive reactions are found both in humans and in animals in first experience situations coupled with some kind of unfamiliarity and threat. Some evidence suggests that the excretion can correlate positively with the strength of an unpleasant affect unrelated to this quality (Bliss et al., 1956; Board et al., 1956; Price et al., 1957; Board et al., 1957; Persky et al., 1958).

The picture about the effect of exercise, especially on the adrenocortical activity, is not yet clear. The level of 17-hydroxy-corticosteroids has increased during exercise in blood (Kägi, 1955; Pace et al., 1956; Crabbe, 1958; Nazar, 1965; Vuori and Pekkarinen, 1969) and in urine (Venning and Kazmin, 1946; Hatch et al., 1956; Hill et al., 1956; Pace et al., 1956; Alimpic et al., 1967; Vuori and Pekkarinen, 1969), has decreased in plasma (Cornil, 1965), and also after the primary increase during prolonged exercise (Kägi, 1955), did not alter in plasma (Wegman, 1968; Moncloa et al., 1970) or in urine (Thorn et al., 1953; Hellman et al., 1956). The urinary excretion of KGS has not altered, either, during exercise (Connell et al., 1958). Moncloa et al. (1965, 1970) have demonstrated a transient increased plasma concentration and urinary excretion of cortisol under hypoxia during exercise in man and a fall in cortisol concentration during exercise under hypoxia at high altitudes.

Consequently the effect of exercise on the adrenocortical activity as measured with the excretion values would be in some way related to the intensity and/or duration of exercise. The increased activity during the early phase of exercise would then obviously agree with the concept of the important role of the adrenal cortex participating in adaptation processes. Possibly the decrease in the activity of the adrenal cortex later during the exercise refers to an increase in either the consumption or diffusion of adreno-

cortical hormones as suggested previously (Kägi et al., 1955; Hill et al., 1956; Connel et al., 1958; Cornil et al., 1965).

Systematic experimental evidence of the relationship of physical condition and the activity of adrenal cortex with its reflections on the levels of hormones in blood and urine are few. Some data suggest a significant hypertrophy of the adrenal glands in animals with a corresponding increase in the hormone levels at the beginning of training (Frenkl and Csalay, 1962). Since the onset of the research they found the weight of the adrenals increased continuously when the plasma hormone level decreased below the normal concentrations. Hatch et al. (1956) found a significant negative correlation between the Harvard step-test scores and the urinary excretion rate of 17-hydroxycorticosteroids. They suggest that the plasma flow and filtration rate would be nearer to normal in subjects who could increase the minute volume more effectively, for example, in good physical condition.

Summary from review of literature

The sympathoadrenal and adrenocortical systems have been for many decades a subject of very intensive research. Advanced biochemical methods of analysis have been developed and some of them are succesful in practice. For all the wealth of data, we are still short of knowledge to formulate a distinct theoretical frame for the neurohormonal regulating systems in an organism during stress situations. A large variety of psychophysical loadings will be reflected in the increased excretory pattern of the two hormonal systems studied in the present work. However the role of good or poor physical fitness in the regulation of endocrine functions in the state of physical and mental loading is less known.

Theoretically, the vast changes in many regulatory and energy production activities due to intensive physical training also suggest changes in hormonal regulatory functions which would perhaps be measureable in practice in the changes of excretory pattern in spite of the well-known and large variation both inter- and intraindividually. Herein lies the impetus to do this study.

From another aspect, we know much about the intense regulatory effects also of the endogenous hormone outbursts during loading. Positive research data about training effects would therefore emphasize the necessity to know more about the possible irreversible consequences of the chronic pattern of this kind of intensive acute regulatory activity in health and performance.

Problems

The following problems have been studied:

1. The effect of selected moderate loadings with a suggested mental or physical prevalence on the following biochemical correlates of loading in man:
   - 1.1 Urinary excretion of noradrenaline (NA) and adrenaline (A)
   - 1.2 Urinary excretion of 17-hydroxycorticosteroids (17-OHCS)
   - 1.3 Hematocrite value, concentration of free fatty acids (FFA), potassium (K^+), sodium (Na^+), lactic acid in plasma and of hemoglobin and glucose in blood

2. The effect of regular, progressive endurance conditioning on these correlates

Material, methods and procedure

Subjects

The studies were conducted on four occasions. The first study is reported in paper (I), the second in (II), the third in (III), the fourth in (IV—VI). In all, 87 volunteer male subjects participated in these studies. Descriptive data of the subjects are presented in TABLE 1.

TABLE 1. DESCRIPTIVE DATA OF THE PRESENT STUDIES. THE STUDIES, CONTD.

Study no.[1]	n: Control subjects	n: Test subjects	Age (years)	Remarks	Type of progressive endurance conditioning	Conditioning time	Loadings	Loading no.[2]
I	9	9	20—24	Air Force cadets receiving similar military instruction	Interval runnig	12 weeks; 3 times per week; 30—60 min. each time	a) A relative rest (forenoon-afternoon)	1.
							b) Waiting for a flight as a pilot	6.1
							c) Flying as a pilot	6.2
II	10	20	19—22	Air Force conscripts participating in the same course of combat control. The test subjects were matched into two groups having a different training program	Pedalling on a bicycle ergometer	12 weeks; 4 times per week; 30 min. each time	a) Loading No. 1.	
							b) Bicycle ergometer work 6 min. 1200 kpm/min.	3.
							c) Bicycle ergometer 20+10 min. 600+900 kpm/min., respectively	4.4
							d) 4 hours military marching	5.
							e) Choise reaction test 54 minutes	6.3
III	—	15	19—30	All served as their own controls; University students	No conditioning		a) A relative rest (forenoon)	2.
							b) Loading No. 3.	
							c) Bicycle ergometer work 24 min. 750 kpm/min.	4.1
							d) A combination of loadings No. 3. and 4.1	
IV	12	12	20—30	The groups were combined from two separate groups exposed to the same test treatment during different seasons; University students	Pedalling on a bicycle ergometer	9 weeks; 5 times per week; 30—60 min. each time	a) Loading No. 2.	
							b) Loading No. 3.	
							c) Bicycle ergometer work 20 min. 900 kpm/min.	4.2
							d) Bicycle ergometer work 19+1 min. 900+1500 kpm/min. respectively	4.3
							e) A combination of loadings No. 3., 4.2 and 4.3	4.6
							f) Choice reaction test 45 minutes	6.3
V	6	6	20—25	The group consisted of one of the subgroups in study (IV)	»	»	a) Loading No. 2.	
							b) Loading No. 6.3	
VI	11	11	20—30	The same subjects as in (IV)	»	»	The same loadings as	

Psychophysical loading

Referring to the measured variables, the following situations of loading were used in the studies involved (TABLE I):

1. A relative rest (forenoon—afternoon)	I, II
2. A relative rest (forenoon)	III—VI
3. Bicycle ergometer work with anaerobic prevalence, 6 minutes 1200 kpm/min	II—IV, VI
4. Bicycle ergometer work with aerobic prevalence	II—IV, VI
4.1 24 minutes 750 kpm/min	III
4.2 20 minutes 900 kpm/min	IV, VI
4.3 19 + 1 minutes 900 + 1500 kpm/min respectively	IV, VI
4.4 20 + 10 minutes 600 + 900 kpm/min respectively	II
4.5 A combination of 3. and 4.	III
4.6 A combination of 3., 4.2 and 4.3	IV, VI
5. Prolonged work of four hours' military marching	II
6. Special mental loadings	
6.1 Waiting for a strenuous flight (6.2) as a pilot	I
6.2 Piloting during low-level attack and stunt flying totalling 70 minutes	I
6.3 Psychomotor loading in choice reaction test competing (CRT) during:	
54 minutes	II
45 minutes	IV—V

The work loading of 6 minutes at 1200 kpm/min. served as a standardized test for the physical fitness of subjects, according to Åstrand and Ryhming (1954). The work loads and the time of loading were combined, firstly, for the purpose of loading by different ways aerobically and anaerobically and, secondly, according to the average performance capacity of the subjects and, thirdly, because of numerous technical reasons which had to be considered in order to complete the experiment successfully and to offer the subjects an opportunity to participate. More detailed information is included in each separate report.

Endurance conditioning

Two different ways of endurance conditioning were used (TABLE I): interval running in (I), and pedalling on a bicycle ergometer in (II) and (IV—VI).

The control group was denied participation in any physical exercise (I), or some of the subjects were allowed to maintain their previous physical activity and were controlled by an inquiry (II) and (IV—VI). In all cases the training for the test group was progressive and controlled by the investigators.

In (I) the test group trained for 12 weeks, three times a week by running in two alternative programmes of interval training, timed so as to produce a working pulse rate of about 170 per minute. In (II) there were two test groups training on a bicycle ergometer for 12 weeks, four times a week and half an hour each time in two different programmes, in the former in order to maintain the working heart rate at approximately 140 per minute and in the later at approximately 160 per minute. In (IV—VI) the test group trained for nine weeks on a bicycle ergometer five times a week and the exercise time was increased gradually from half an hour in the beginning of training to one hour. The load was chosen in order to maintain a working heart rate of approximately 160—170 per minute. To control the proper load for each subject, and to measure the training effect, the heart rate was recorded by an electrocardiograph during rest and at various phases of work. The predicted $\dot{V}_{O_2max.}$ was determined by the method of Åstrand and Ryhming (1954) by using a work intensity of six minutes at 1200 kpm/min.

Biochemical methods

Free catecholamines in the urine were determined by the fluorimetric methods of Crout (1961) in (I), Pekkarinen (1968) in (II), and Euler & Lishajko (1961) in (III), (V) and (VI). Thus, catecholamines were adsorbed onto alumina in pH 8.3—8.5, eluated with acetic acid in (I), (III), (V) and (VI) or with a solution of 30.0 1-N HCl + 270.0 10 % NaH_3PO_4 in (II), oxidized with iodine in

pH 6.5 and 3.5 in (I) or with potassium ferricyanide in (II), (III), (V) and (VI) in pH 6.2 to adrenochromes and transformed into fluorescent trihydroxyindoles with alkaline and ascorbic acid. The intensity of fluorescence was measured with an Aminco Bowman in (I) or a Perkin-Elmer MPF-2A fluorescence spectrophotometer with an activation wave length of 400 and 436 mμ and emission wave length of 530 mμ in (II), 395, 505 and both 410 and 520 mμ in (I) or 396, 440 mμ and 510 mμ respectively in (III), (V) and (VI).

The total amount of 17-hydroxycorticosteroids in the urine was determined by the method described by Appleby et al. (1955) and modified by Pesonen (1965) (in II), or by the method of Few (1961) and modified by Thomas (1965) in (II) and (VI). Consequently, the corticoids were reduced first with sodiumborohydride and oxidized with sodiumvismutate in (II) or with sodiummetaperiodate in (III) and (VI) to 17-ketosteroids. After hydrolysis the steroids were extracted into dichloretane in (II) and (VI) and washed with natriumhydroxide in (II) or with natriumditionite in (III) and (VI) and distilled water. The dichloretane was evaporated and from the residuum the reaction of Zimmerman was taken. The colour intensity was measured with a Beckman DB-spectrophotometer with wave lenghts of 470 and 520 mμ in (II) or 440, 520 and 600 mμ in (III) and (VI). The compound of comparison was dehydroisoepiandrosterone and the Allen correction coefficient was used.

From the blood, the hemoglobin and hematocrite were determined and the rest of the samples immediately centrifugated for 10 minutes at 3000 rpm. The plasma was separated for the determination of free fatty acids (IV), K^+ and Na^+ (II, VI) and lactic acid (II, III, VI). The precipitation of proteins was taken from 0.1 ml of whole blood and the glucose concentration determined (II, III, VI) by the method of Hultman and modified by Hyvärinen and Nikkilä (1962).

Plasma lactic acid was determined by the entzymatic method with reagents and instructions of CF Boehringer-Soehne GmbH (1967). The intensity was measured with a Beckman DB-spectrophotometer with a wave length of 366 mμ for lactic acid, 630 mμ for glucose and 540 mμ for hemoglobin.

The concentration of plasma-K^+ and -Na^+ was determined from proper dilutions of plasma with an EEL-flame photometer and by using Merck's Titrisol solutions as standards.

Free fatty acids were determined essentially by the titrimetric method described by Trout et al. (1960) with minor modifications (IV). The solvent volumes were modified for the plasma volume used (0.4 ml) and instead of heptane, hexane was employed in the extraction mixture and sodium methylate in the titration (O'Brien and Ibbott, 1964).

More details of the present use of these methods and the collection procedures of the blood and urine samples are reported in each separate paper.

Standardization

Much attention was paid to keeping the conditions as similar as possible in every measurement and to equalizing effects of uncontrolled factors in the control and test groups. The standardization procedures were applied to the various factors known to be relevant such as the diurnal and seasonal variation, food and fluid, drugs, tobacco, alcohol, caffein-containing beverages, the treatment of urine samples and others (cf. Levi 1967). The details are depicted in the respective reports.

Statistical analysis

For measuring the effects of various loadings the two-way analysis of variance (McNemar, 1959; Winer, 1962) was used in (II). The significance of the mean pairs were then tested by the method of Tukey (Winer, 1962). Except for a part of (IV) the loading effect in the other studies were tested by the Student t-test for correlating means. In (IV), to obtain the effect of the choice reaction test, the McNemar test for the significance of changes (Siegel, 1956) was used. The effect of training was tested by the covariance analysis for repeated measurements (McNemar, 1959; von Wright, 1959; Winer, 1962) the pretraining values acting as covariates.

In (IV), for the choice reaction test values this was done by the randomization test for matched pairs (Siegel, 1956). More details, e.g. the log-transformation and missing data procedures (II, IV—VI) in the analyses are reported in the respective papers. Some correlations have been calculated by Pearsons' r (IV, VI).

Loading

Blood parameters

Six and thirty minutes of muscular work in (II) as well as all the work phases of muscular work in (III) and (VI) (TABLE II)* increased the concentration of *plasma lactic acid* when compared to the situation of relative rest ($p < .05$—.001). The concentration of *blood glucose* was increased during the control afternoon, after the choice reaction test and four hour marching in (II) ($p < .05$). In (III), it was significantly decreased after the last 24 minutes of muscular work ($p < .01$) (TABLE III). In (VI) no significant differences were noticed during the phases of work and rest (TABLE II). *Hemoglobin concentration* increased after the first six minute phase of muscular work at 1200 kpm/min. in (VI) ($p <$ .05) and decreased again after the second phase, 20 minutes at 900 kpm/min. ($p < .01$) (TABLE IV). *Hematocrite* behaved similarly, but increased significantly again after the last anaerobic phase of muscular work ($p < .02$—.001) (TABLE IV).

The concentration of K^+ *in the plasma* increased after all phases of muscular work in (VI) (TABLE V), except the first phase, as compared to relative rest before the work ($p < .05$—.001). This was not the case during any of the loadings in (II). The only significant difference in Na^+ *concentrations* between the loadings and the control measurements was found in (II) between six minutes of work and the respective control situation. In (VI) *the ratio of* Na^+ *and* K^+ concentrations was lower than in the control situation during the muscular work ($p < .05$—.001) (TABLE VI) and in (II) during the choice reaction test (CRT) ($p < .05$).

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* all TABLE notations refer to the table numbers in the present report.

TABLE II. LACTIC ACID AND BLOOD GLUCOSE DURING RELATIVE REST AND AFTER PHYSICAL WORK DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

			(A) During relative rest				(B) 5 minutes after work I; 6 minutes 1200 kpm/min			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Lactic acid mg %	Control group	$\bar{X}$	4.7	4.5	4.5	4.1	18.6	17.0	18.1	15.7
		S.E.	0.58	0.38	0.68	0.35	2.14	2.42	2.97	2.52
				[2] F = 0.316				[2] F = 6.863 $p < .025$		
	Trained group	$\bar{X}$	4.7	4.0	4.4	5.3	20.3	14.6	11.5	9.4
		S.E.	0.24	0.34	0.54	0.48	4.07	2.17	2.01	1.54
	Total	$\bar{X}$	4.7	[3] (A) — (B) $p < .001$			19.5	[3] (B) — (C) $p < .001$		
Glucose mg %	Control group	$\bar{X}$	71.4	73.8	72.4	73.0	71.1	73.2	70.8	71.7
		S.E.	1.84	1.20	1.69	1.25	2.34	1.71	1.43	2.66
				[2] F = 0.055 NS				[2] F = 0.488 NS		
	Trained group	$\bar{X}$	72.5	73.2	73.0	74.7	72.2	70.0	71.3	72.0
		S.E.	2.62	1.63	1.90	1.58	1.32	1.39	1.26	1.67
	Total	$\bar{X}$	72.0	[3] (A) — (B) NS			71.6	[3] (B) — (C) NS		

			(C) 5 minutes after work II; 20 minutes 900 kpm/min				(D) 5 minutes after work III; 19 + 1 minutes 900 + 1500 kpm/min, respectively			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Lactic acid mg %	Control group	$\bar{X}$	6.8	6.7	6.5	6.9	13.8	12.2	12.9	11.1
		S.E.	0.78	0.99	1.08	1.16	1.81	2.04	2.09	1.32
				[2] F = 2.498 NS				[2] F = 7.487 p = .025		
	Trained group	$\bar{X}$	10.0	6.7	5.5	6.1	16.3	12.1	9.3	9.7
		S.E.	1.97	0.85	0.70	0.58	3.19	1.54	1.31	1.19
	Total	$\bar{X}$	8.4	[3] (A) — (C) p < .001			15.0	[3] (A) — (D) p < .001 (C) — (D) p < .001		
Glucose mg %	Control group	$\bar{X}$	70.2	72.9	72.5	72.7	71.1	71.5	71.4	70.5
		S.E.	2.66	1.41	1.85	2.21	2.49	1.86	1.58	2.79
				[2] F = 0.819 NS				[2] F = 2.348 NS		
	Trained group	$\bar{X}$	68.3	72.0	74.3	73.8	68.3	70.3	74.5	73.7
		S.E.	0.98	1.55	1.71	2.06	1.31	1.71	1.94	1.94
	Total	$\bar{X}$	69.2	[3] (A) — (C) NS			69.7	[3] (A) — (D) NS (C) — (D) NS		

[1] M_0 = before training; M_3 — M_9 = 3, 6, 9 weeks after the beginning of training.
[2] analysis of covariance for the between group difference; M_0 as covariate, M_3 — M_9 as criterion.
[3] t-test for the total means in M_0.

TABLE III. BLOOD GLUCOSE AND PLASMA LACTIC ACID IN RELATIVE REST AND IN RECOVERY AFTER SUBMAXIMAL WORK ON A BICYCLE ERGOMETER

(From the report SARVIHARJU, P. J.: Effect of physical exercise on the urinary excretion of catecholamines and 17-hydroxycorticosteroids in young healthy men, J. Sports Med. and Phys. Fitness, 1972. In press.)

Variable		Before run on a bicycle ergometer (1) n = 15	p	5 minutes after the first run: 6 minutes 1200 kpm/minute. (2) n = 14	p	5 minutes after the second run: 24 minutes 750 kpm/minute. (3) n = 15	Difference between (1) and (3) p
Lactic acid	(mg % ± S.D.)	5.1 ± 1.17	< 0.001	35.4 ± 15.79	< 0.001	11.9 ± 7.26	< 0.01
Glucose	(mg % ± S.D.)	77.1 ± 6.49	NS	78.7 ± 11.25	< 0.02	71.0 ± 6.63	< 0.01

Free fatty acids in the plasma in (IV) increased ($p < .01$—.001) after the phases of muscular work on a bicycle ergometer except after the first six minutes of work at 1200 kpm/min. (TABLE VII). No significant difference was found between the FFA concentrations before and after the choice reaction test (TABLE VIII).

Urinary catecholamines and 17-hydroxycorticosteroids

The urinary excretion of *adrenaline* increased significantly during most of the measurements that, according to the proposition, contained varying amounts of mental or anticipatory strain, such as the situations while waiting for a flight as a pilot ($p < .05$) before the endurance conditioning in (I), during a flight in (I) (TABLE IX), and during a competition in a choice reaction test ($p < .001$) in (V) both before and after the conditioning (TABLE X). Even during the submaximal muscular work of the total 30 minutes in (III) (TABLE XIII) and the total 46 minutes in (VI) (TABLE XIV) on a bicycle ergometer the adrenaline excretion increased significantly ($p < .001$), but neither during the duration of six minutes and thirty minutes of work in (II) nor during the four hours' military marching in (II). No significant difference was found between the forenoon and afternoon experimental conditions during the control days in (I) (TABLE IX) and in (II).

Except for the control afternoon in (II) and the waiting for a flight in (I) (TABLE XI), the urinary excretion of *noradrenaline* was significantly greater than under the respective control circumstances in every loading situation ($p < .05$—.001). The difference between the control forenoon and the afternoon was significant only before the training in (I), where the excretion was smaller in the afternoon ($p < .05$) (TABLE XI).

The ratio of noradrenaline to adrenaline decreased during the waiting for a flight before the training in (I) (TABLE XII) ($p < .05$—.001), but not after it, as well as during the flight in (I) (TABLE XII) and the choice reaction test competition in (V) (TABLE X) both before and after the training ($p < .05$—.01). It increased ($p < .05$) during all of the muscular work except for the total 46 minutes of work in (VI) (TABLE XIV), when it even

TABLE IV. HEMOGLOBIN AND HEMATOCRITE DURING RELATIVE REST AND AFTER PHYSICAL WORK DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

			(A) During relative rest				(B) 5 minutes after work I; 6 minutes 1200 kpm/min			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Hemoglobin g/100 ml	Control group	$\bar{X}$	15.3	15.5	15.4	15.8	15.7	15.8	15.9	16.1
		S.E.	0.36	0.22	0.26	0.25	0.32	0.23	0.32	0.26
				[2] F = 1.643 NS				[2] F = 6.018 $p < .025$		
	Trained group	$\bar{X}$	15.5	15.4	15.4	15.4	15.8	15.6	15.4	15.3
		S.E.	0.29	0.23	0.19	0.20	0.25	0.21	0.21	0.21
	Total	$\bar{X}$	15.4	[3] (A) — (B) $p < .05$			15.7	[3] (B) — (C) $p < .01$		
Hematocrite %	Control group	$\bar{X}$	45.2	45.5	45.2	45.9	46.3	46.6	46.2	46.5
		S.E.	1.14	0.72	0.93	0.76	1.08	0.82	1.01	0.81
				[2] F = 0.017 NS				[2] F = 3.710 NS		
	Trained group	$\bar{X}$	45.1	45.6	45.6	45.1	46.1	45.3	45.7	44.7
		S.E.	0.56	0.60	0.28	0.51	0.57	0.49	0.43	0.54
	Total	$\bar{X}$	45.2	[3] (A) — (B) $p < .01$			46.2	[3] (B) — (C) $p < .001$		

			(C) 5 minutes after work II; 20 minutes 900 kpm/min				(D) 5 minutes after work III; 19 + 1 minutes 900 + 1500 kpm/min, respectively			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Hemoglobin g/100 ml	Control group	$\bar{X}$	15.3	15.5	15.7	15.8	15.5	15.6	15.7	15.9
		S.E.	0.32	0.28	0.37	0.24	0.33	0.28	0.35	0.24
				[2] F = 4.381 $p < .05$				[2] F = 2.991 NS		
	Trained group	$\bar{X}$	15.6	15.4	15.5	15.3	15.6	15.5	15.5	15.2
		S.E.	0.19	0.18	0.22	0.22	0.24	0.17	0.24	0.22
	Total	$\bar{X}$	15.4	[3] (A) — (C) NS			15.6	[3] (A) — (D) NS (C) — (D) NS		
Hematocrite %	Control group	$\bar{X}$	44.7	44.9	45.1	45.6	45.6	45.4	45.8	45.6
		S.E.	1.04	0.83	0.95	0.79	1.11	0.91	0.77	0.79
				[2] F = 2.480 NS				[2] F = 0.299 NS		
	Trained group	$\bar{X}$	45.0	44.8	44.9	44.3	45.3	45.1	45.7	44.8
		S.E.	0.42	0.44	0.39	0.56	0.46	0.36	0.49	0.36
	Total	$\bar{X}$	44.9	[3] (A) — (C) NS			45.5	[3] (A) — (D) NS (C) — (D) $p < .02$		

[1] M_0 = before training; M_3 — M_9 = 3, 6, 9 weeks after the beginning of training.
[2] analysis of covariance for the between group difference; M_0 as covariate, M_3 — M_9 as criterion.
[3] t-test for the total means in M_0.

TABLE V. PLASMA $-K^+$ AND $-Na^+$ DURING RELATIVE REST AND AFTER PHYSICAL WORK DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

			(A) During relative rest				(B) 5 minutes after work I; 6 minutes 1200 kpm/min			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Plasma K^+ mEq/l	Control group	$\overline{X}$	4.5	4.7	4.6	4.6	4.6	4.8	4.6	4.7
		S.E.	0.07	0.11	0.08	0.06	0.08	0.09	0.07	0.06
				[2] F = 2.057 NS				[2] F = 1.892 NS		
	Trained group	$\overline{X}$	4.5	4.7	4.7	4.7	4.5	4.8	4.7	4.6
		S.E.	0.07	0.14	0.07	0.08	0.10	0.12	0.08	0.07
	Total	$\overline{X}$	4.5	[3] (A) — (B) NS			4.6	[3] (B) — (C) $p < .001$		
Plasma Na^+ mEq/l	Control group	$\overline{X}$	147.2	153.7	148.2	148.0	148.6	154.2	148.0	148.7
		S.E.	0.91	2.10	0.56	1.37	0.91	1.70	1.01	1.62
				[2] F = 0.129 NS				[2] F = 0.231 NS		
	Trained group	$\overline{X}$	148.0	153.1	147.9	148.5	148.7	154.6	148.1	148.9
		S.E.	0.92	2.62	0.95	1.30	0.93	2.09	1.90	1.65
	Total	$\overline{X}$	147.6	[3] (A) — (B) NS			148.7	[3] (B) — (C) NS		

			(C) 5 minutes after work II; 20 minutes 900 kpm/min				(D) 5 minutes after work III; 19 + 1 minutes 900 + 1500 kpm/min, respectively			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Plasma K^+ mEq/l	Control group	$\bar{X}$	4.8	4.9	4.7	4.7	4.7	4.8	4.7	4.7
		S.E.	0.05	0.08	0.05	0.07	0.09	0.10	0.07	0.09
				[2] F = 0.395 NS				[2] F = 0.734 NS		
	Trained group	$\bar{X}$	4.8	5.0	4.8	4.8	4.6	4.8	4.8	4.7
		S.E.	0.08	0.13	0.07	0.06	0.09	0.12	0.10	0.07
	Total	$\bar{X}$	4.8	[3] (A) — (C) $p < .001$			4.7	[3] (A) — (D) $p < .05$ (C) — (D) NS		
Plasma Na^+ mEq/l	Control group	$\bar{X}$	147.6	154.2	148.3	148.7	147.2	152.7	148.0	150.5
		S.E.	0.90	2.16	0.79	1.79	0.69	1.81	1.08	1.55
				[2] F = 0.164 NS				[2] F = 0.520 NS		
	Trained group	$\bar{X}$	149.8	153.9	147.3	148.7	148.3	152.8	146.2	149.2
		S.E.	0.60	2.23	1.15	1.38	0.91	2.18	0.94	1.55
	Total	$\bar{X}$	148.7	[3] (A) — (C) NS			147.8	[3] (A) — (D) NS (C) — (D) NS		

[1] M_0 = before training; M_3 — M_9 = 3, 6, 9 weeks after the beginning of training.
[2] analysis of covariance for the between group difference; M_0 as covariate, M_3 — M_9 as criterion.
[3] t-test for the total means in M_0.

TABLE VI. THE RATIO OF PLASMA — Na^+ AND — K^+ (Na^+/K^+) DURING RELATIVE REST AND AFTER PHYSICAL WORK DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

			(A) During relative rest				(B) 5 minutes after work I; 6 minutes 1200 kpm/min			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Na^+/K^+ mEq/l / mEq/l	Control group	$\bar{X}$	32.5	32.8	32.4	32.2	32.2	32.4	32.2	32.0
		S.E.	0.52	0.50	0.57	0.47	0.58	0.43	0.49	0.48
				[2] F = 2.222 NS				[2] F = 1.277 NS		
	Trained group	$\bar{X}$	33.0	32.6	31.5	31.9	33.1	32.2	31.3	32.2
		S.E.	0.44	1.11	0.46	0.55	0.84	1.00	0.68	0.51
	Total	$\bar{X}$	32.7	[3] (A) — (B) NS			32.7	[3] (B) — (C) $p < .001$		

			(C) 5 minutes after work II; 20 minutes 900 kpm/min				(D) 5 minutes after work III; 19 + 1 minutes 900 + 1500 kpm/min, respectively			
			[1] M_0	M_3	M_6	M_9	[1] M_0	M_3	M_6	M_9
Na^+/K^+ mEq/l / mEq/l	Control group	$\bar{X}$	31.0	31.2	31.3	31.4	31.1	31.6	31.8	32.1
		S.E.	0.29	0.47	0.25	0.59	0.54	0.59	0.53	0.61
				[2] F = 1.924 NS				[2] F = 1.598 NS		
	Trained group	$\bar{X}$	31.3	31.0	30.6	31.3	32.3	31.7	30.8	31.8
		S.E.	0.50	0.80	0.41	0.45	0.67	0.92	0.62	0.51
	Total	$\bar{X}$	31.2	[3] (A) — (C) $p < .001$			31.7	[3] (A) — (D) $p < .05$ (C) — (D) NS		

[1] M_0 = before training; M_3 — M_9 = 3, 6, 9 weeks after the beginning of training.
[2] analysis of covariance for the between group difference; M_0 as covariate, M_3 — M_9 as criterion.
[3] t-test for the total means in M_0.

TABLE VII. PLASMA FFA [μEq/l ± S.D.] AT RELATIVE REST AND DURING RECOVERY FROM SUBMAXIMAL WORK ON A BICYCLE ERGOMETER BEFORE AND AFTER A 9-WEEK PERIOD OF ENDURANCE TRAINING. M_0 = BEFORE TRAINING; M_3—M_9 = 3, 6 AND 9 WEEKS AFTER THE COMMENCEMENT OF TRAINING.

(From the report SARVIHARJU, P. J., and V. VIHKO: Plasma FFA during psychophysical loading and endurance training, J. Sports Med. and Phys. Fitness, 1972. In press.)

Subjects	Measurement during training period	At relative rest	5 minutes after ergometer work I; 6 minutes 1200 kpm/min.	5 minutes after ergometer work II; 20 minutes 900 kpm/min.	5 minutes after ergometer work III; 19 + 1 minutes 900 + 1500 kpm/min. respectively
Control group (12)	M_0	662 ± 190	696 ± 201	908 ± 310	1090 ± 290
	M_3	554 ± 196	666 ± 163	853 ± 318	992 ± 302
	M_6	462 ± 144	602 ± 194	764 ± 198	984 ± 193
	M_9	561 ± 178	619 ± 169	840 ± 209	1021 ± 252
	Analysis of covariance M_0 as covariate and M_9 as criterion: Over-all F-test for between-subject effects F = 1.175 (NS).				
Trained group (12)	M_0	696 ± 151	763 ± 218	865 ± 324	1030 ± 203
	M_3	482 ± 196	691 ± 207	785 ± 302	954 ± 321
	M_6	491 ± 173	627 ± 212	789 ± 269	931 ± 247
	M_9	593 ± 200	754 ± 235	771 ± 377	965 ± 336
Total means (M_0)		679 ± 167 1	732 ± 206 2	887 ± 316 3	1060 ± 257 4
Significance of difference between total means (M_0); Student t		1—2 $P > 0.1$	2—3 $P < 0.01$	3—4 $P < 0.02$	1—4 $P < 0.001$

TABLE VIII. PLASMA FFA [μEq/l ± S.D.] BEFORE AND AFTER A 45 MINUTE CHOICE REACTION TEST IN SITUATION FOR COMPETING. M_0 = BEFORE TRAINING; M_9 = 9 WEEKS AFTER THE COMMENCEMENT OF TRAINING.
(From the report SARVIHARJU, P. J., and V. VIHKO: Plasma FFA during psychophysical loading and endurance training, J. Sports Med. and Phys. Fitness, 1972. In press.)

Subjects	Measurement during training period	Before the choice reaction test		5 minutes after the choice reaction test
Control (6) group	M_0	817 ± 299		874 ± 519
	M_9	601 ± 199		619 ± 110
		No significant difference between the changes in the groups [1]		
Trained (6) group	M_0	804 ± 121		911 ± 243
	M_9	653 ± 325		670 ± 152
Total mean	M_0	811 ± 207	$\chi^2 = 0.08$ NS[2]	896 ± 332

[1] the randomization test for matched pairs; $\alpha = .05$
[2] the Mc Nemar test for the significance of changes
Both [1] and [2] in Siegel, S.: Nonparametric Statistics for the Behavioral Sciences, Mc Graw-Hill Book Company, Inc., 1956, p. 88 and 65, respectively.

decreased ($p < .01$), and the 30 minutes work in (III) (TABLE XIII) with no significant change.

No significant diurnal difference in the ratio of noradrenaline to adrenaline (NA/A) was found during the daytime used in the present studies.

The urinary excretion of *17-hydroxycorticosteroids* (17-OHCS) increased significantly after the muscular work of the total 30 minutes in (III) as compared to the situation before it ($p < .02$) (TABLE XIII). No other significant differences were found in this parameter.

The *urine output* during the work phase as compared to the pre-work phase significantly decreased in (VI) before the conditioning period ($p < .01$) (TABLE XIV). Thereafter, there seemed to be a slight tendency of a relative increased output during the exercise in the trained group.

TABLE IX. EFFECT OF FLIGHT STRESS AND ENDURANCE TRAINING ON THE URINARY EXCRETION OF ADRENALINE (NG/MIN ± S.E.)

(From the report SARVIHARJU et al.: Effect of endurance training on the urinary excretion of noradrenaline and adrenaline during ground and flying activity, Aerospace Med., 42 (12): 1297—1302, 1971).

Subjects	Control Day Forenoon		Control Day Afternoon		Waiting For the Flight		In Flight	
	Before Training	After Training	Before Training	After Training	Before Training	After Training	Before Training	After Training
Control (9) Group	3.2 ± 0.80	10.5 ± 1.32	7.6 ± 0.69	10.9 ± 1.04	12.3 ± 0.88	11.6 ± 0.69	24.5 ± 2.71	19.5 ± 2.15
Trained (9) Group	7.6 ± 0.64	7.1 ± 0.64	6.6 ± 0.39	10.9 ± 1.18	10.8 ± 3.42	6.9 ± 1.18	17.8 ± 2.62	16.2 ± 1.81
All (18)	7.9 ± 0.51	8.8 ± 0.88	7.2 ± 0.39	10.9 ± 0.78	11.5 ± 2.38	9.3 ± 0.90	21.2 ± 2.06	17.1 ± 1.47

Statistical Analysis	Measurement	Effect of loading situations on excretion; Statistical significance between the means; Student t-test for the correlating means: Afternoon [1]	Waiting For Flight [2]	In Flight [3]	Effect of training on excretion; Statistical significance of the difference between group means after training; One-way analysis of covariance: Forenoon [4]	Afternoon [4]	Waiting For Flight [4]	In Flight [4]
	Before Training							
	Control	$t = 0.88$ NS	$t = 5.12$ $p < 0.001$	$t = 6.14$ $p < 0.001$				
	Trained	$t = 2.25$ NS	$t = 0.91$ NS	$t = 6.63$ $p < 0.001$	$F = 5.56$	$F = 0.21$	$F = 9.42$	$F = 0.10$
	All	$t = 1.85$ NS	$t = 2.13$ $p < 0.05$	$t = 6.98$ $p < 0.001$				
	After Training				$p < 0.05$	NS	$p < 0.01$	NS
	Control	$t = 0.29$ NS	$t = 0.66$ NS	$t = 4.19$ $p < 0.01$				
	Trained	$t = 2.86$ $p < 0.05$	$t = 0.04$ NS	$t = 2.31$ $p < 0.05$				
	All	$t = 1.68$ NS	$t = 0.44$ NS	$t = 4.48$ $p < 0.01$				

1 = Difference between the forenoon and the afternoon on the control day
2 = Difference between the forenoon of the control day and the forenoon before the flight
3 = Difference between the afternoon of the control day and the flight afternoon
4 = Using the original scores

TABLE X. URINARY EXCRETION OF CATECHOLAMINES (ng/min) DURING RELATIVE REST AND CHOICE REACTION TEST OF 45 MINUTES BEFORE AND AFTER A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING.

(From the report SARVIHARJU, P. J., and E. MATTILA: Urinary excretion of catecholamines during psychomotor choice reaction test before and after endurance conditioning, Public Health Publication no. 14, 1972, University of Jyväskylä, Finland)

Subjects	Before training				After training [2]			
	During rest	During CRT	p [1]	Ratio of CRT/Rest	During rest	During CRT	p [1]	Ratio of CRT/Rest
Trained (6)								
Adrenaline	3.3 ± 1.2	17.4 ± 7.4	< .01	5.3	3.0 ± 1.1	13.5 ± 4.9	< .01	4.5
Noradrenaline	24.0 ± 6.7	35.6 ± 5.7	< .01	1.5	19.1 ± 4.2	31.1 ± 9.3	< .01	1.6
NA/A	8.7 ± 4.7	2.3 ± 0.7	< .05	0.3	8.2 ± 6.4	2.4 ± 0.7	< .05	0.3
Control (6)								
Adrenaline	5.2 ± 3.3	12.5 ± 4.5	< .01	2.4	2.9 ± 1.1	13.0 ± 7.2	< .01	1.5
Noradrenaline	14.8 ± 2.3	23.6 ± 7.3	< .01	1.6	16.2 ± 7.1	25.0 ± 15.0	NS	1.5
NA/A	3.5 ± 1.5	2.3 ± 1.9	NS	0.7	5.9 ± 2.6	2.3 ± 1.5	< .002	0.4
All (12)								
Adrenaline	4.3 ± 2.5	15.0 ± 6.2	< .001	3.5	3.0 ± 1.2	13.2 ± 7.5	< .001	4.4
Noradrenaline	19.4 ± 6.8	29.6 ± 15.7	< .001	1.5	17.7 ± 5.4	28.1 ± 13.5	< .001	1.6
NA/A	6.1 ± 4.3	2.3 ± 1.3	< .05	0.4	7.1 ± 4.8	2.4 ± 1.0	< .01	0.3

[1] t-test for the correlating means between rest and CRT

[2] no significant differences between the groups after training; analysis of covariance with log-transformations the pretraining values as covariate and the post-training values as criterion

TABLE XI. EFFECT OF FLIGHT STRESS AND ENDURANCE TRAINING ON THE URINARY EXCRETION OF NORADRENALINE (NG/MIN ± S.E.)

(From the report SARVIHARJU et al.: Effect of endurance training on the urinary excretion of noradrenaline and adrenaline during ground and flying activity, Aerospace Med., 42 (12): 1297—1302, 1971.)

Subjects	Control Day Forenoon		Control Day Afternoon		Waiting For the Flight		In Flight	
	Before Training	After Training	Before Training	After Training	Before Training	After Training	Before Training	After Training
Control (9) Group	31.8 ± 4.52	24.9 ± 4.29	28.0 ± 3.17	21.7 ± 2.17	29.1 ± 4.64	24.7 ± 1.11	42.7 ± 3.64	37.9 ± 4.81
Trained (9) Group	30.9 ± 3.09	16.6 ± 2.65	25.7 ± 2.68	25.2 ± 1.50	29.3 ± 3.01	21.6 ± 2.50	35.6 ± 2.12	32.6 ± 1.47
All (18)	31.3 ± 2.74	20.7 ± 2.79	26.8 ± 2.00	23.5 ± 1.66	29.2 ± 2.76	23.2 ± 1.44	39.2 ± 2.28	35.2 ± 2.60

Statistical Analysis	Measurement	Effect of loading situations on excretion; Statistical significance between the means; Student t-test for the correlating means: Afternoon [1]	Waiting For Flight [2]	In Flight [3]	Effect of training on excretion; Statistical significance of the difference between group means after training; One-way analysis of covariance: Forenoon [4]	Afternoon [4]	Waiting For Flight [4]	In Flight [4]
	Before Training							
	Controls	$t = 1.27$ NS	$t = 0.76$ NS	$t = 4.08$ $p < 0.01$	$F = 2.66$	$F = 25.94$	$F = 1.24$	$F = 2.02$
	Trained	$t = 1.84$ NS	$t = 0.63$ NS	$t = 2.86$ $p < 0.05$				
	All	$t = 2.25$ $p < 0.05$	$t = 1.01$ NS	$t = 4.50$ $p < 0.001$				
	After Training				NS	$p < 0.001$	NS	NS
	Controls	$t = 1.02$ NS	$t = 0.06$ NS	$t = 4.21$ $p < 0.01$				
	Trained	$t = 3.43$ $p < 0.01$	$t = 1.34$ NS	$t = 4.59$ $p < 0.01$				
	All	$t = 1.13$ NS	$t = 0.92$ NS	$t = 5.16$ $p < 0.001$				

[1] = Difference between the forenoon and the afternoon on the control day
[2] = Difference between the forenoon of the control day and the forenoon before the flight
[3] = Difference between the afternoon of the control day and the flight afternoon
[4] = Using the original scores

TABLE XII. EFFECT OF FLIGHT STRESS AND ENDURANCE TRAINING ON THE RATIO OF URINARY EXRETION OF NORADRENALINE TO ADRENALINE (NA/A $\frac{\text{NG/MIN}}{\text{NG/MIN}}$ ± S.E.)

(From the report SARVIHARJU et al.: Effect of endurance training on the urinary excretion of noradrenaline and adrenaline during ground and flying activity, Aerospace Med., 42 (12): 1297—1302, 1971.)

Subjects	Control Day Forenoon		Control Day Afternoon		Waiting For the Flight		In Flight	
	Before Training	After Training	Before Training	After Training	Before Training	After Training	Before Training	After Training
Control (9) Group	3.9 ± 0.33	2.3 ± 0.18	3.8 ± 0.33	2.1 ± 0.19	2.4 ± 0.31	2.2 ± 0.16	1.8 ± 0.17	2.0 ± 0.20
Trained (9) Group	4.2 ± 0.47	2.4 ± 0.39	4.0 ± 0.40	2.4 ± 0.19	3.5 ± 0.36	3.4 ± 0.38	2.4 ± 0.42	2.2 ± 0.25
All (18)	4.0 ± 0.30	2.4 ± 0.21	3.9 ± 0.24	2.3 ± 0.15	3.0 ± 0.28	2.8 ± 0.24	2.1 ± 0.23	2.1 ± 0.16

Statistical Analysis	Measurement	Effect of loading situation on excretion; Statistical significance between the means; Student t-test for the correlating means: Afternoon [1]	Waiting For Flight [2]	In Flight [3]	Effect of training on excretion; Statistical significance of the difference between group means after training; One-way analysis of covariance: Fore-noon [4]	After-noon [4]	Waiting For Flight [4]	In Flight [4]
	Before Training							
	Controls	$t = 0.26$ NS	$t = 3.52$ $p < 0.01$	$t = 7.41$ $p < 0.001$	$F = 0.001$	$F = 2.08$	$F = 6.67$	$F = 2.84$
	Trained	$t = 0.59$ NS	$t = 1.08$ NS	$t = 3.81$ $p < 0.01$				
	All	$t = 0.65$ NS	$t = 2.84$ $p < 0.02$	$t = 6.70$ $p < 0.001$				
	After Training				NS	NS	$p < 0.025$	NS
	Controls	$t = 1.81$ NS	$t = 0.03$ NS	$t = 0.68$ NS				
	Trained	$t = 0.113$ NS	$t = 2.22$ NS	$t = 0.78$ NS				
	All	$t = 0.32$ NS	$t = 1.74$ NS	$t = 1.04$ NS				

[1] = Difference between the forenoon and the afternoon on the control day
[2] = Difference between the forenoon of the control day and the forenoon before the flight
[3] = Difference between the afternoon of the control day and the flight afternoon
[4] = Using the original scores

TABLE XIII. URINARY EXCRETION OF CATECHOLAMINES AND 17-HYDROXYCORTICOSTEROIDS IN HEALTHY YOUNG MEN BEFORE AND AFTER TWO-PHASE WORK ON A BICYCLE ERGOMETER

(From the report SARVIHARJU, P. J.: Effect of physical exercise on the urinary excretion of catecholamines and 17-hydroxycorticosteroids in young healthy men, J. Sports Med. and Phys. Fitness, 1972. In press.)

Variable	n	Before the 1st run [1]	After the 2nd run [2]	t	p
Noradrenaline (NA) ng/minute ± S.D.	12	23.1 ± 12.34	42.0 ± 17.05	6.45	< 0.001
Adrenaline (A) ng/minute ± S.D.	12	8.5 ± 5.71	15.8 ± 6.22	5.03	< 0.001
NA/A ± S.D.	12	3.2 ± 1.61	2.7 ± 0.80	1.34	NS
17-OHCS µg/minute ± S.D.	12	8.9 ± 2.93	10.7 ± 3.18	3.00	< 0.02

[1] = 6 minutes 1200 kpm/min. [2] = 24 minutes 750 kpm/min.

Endurance conditioning

Performance and blood parameters

In the test group the training induced a decrease ($p < .05—.001$) in the *steady state heart rate* during the submaximal 6 minute work on ergometer at 1200 kpm/min. in all the experiments including endurance conditioning (I—II and IV—VI), in (IV) and (VI) even during other physical loadings of a more aerobic kind as well as during the recovery phases (an example in TABLE XV). Some kind of adaptation was observed also in the control group in six minutes' work in (V) and (VI) (TABLE XV), where the heart rate decreased after the training ($p < .05$). In studies about the conditioning effect, except in (II) and (V), the *predicted maximal capacity of O_2 uptake* was calculated and it was found to increase in the trained group ($p < .01—.001$) (TABLE XV). No significant difference was found in the heart rate during relative rest before the muscular work (IV and VI) (TABLE XV). The training was followed also by a decrease ($p < .025$) in the concentration of *plasma lactic acid* in (VI) after the two physical work phases having the most increasing effect on this concentration (TABLE II). The

training had no significant effect on the *choice reaction test scores* in (II) and (V), but a pronounced improvement was found in the choice reaction test performance in both groups.

After training the only significant difference in *blood glucose* between the trained and the untrained was found in (II), where this concentration stayed at a higher level in the trained groups during the four hour marching ($p < .01$).

After training in (VI), the *hemoglobin concentration* was lower in the trained group after the first two phases of ergometer work ($p < .05$—$.025$), but not after the third phase nor during the relative rest (TABLE IV).

No significant differences between the groups were found in the respective situations in *hematocrite* values (VI) (TABLE IV) nor in the concentration of *plasma free fatty acids* after any of the phases of muscular work or after the choice reaction test competition (IV) (TABLES VII and VIII). The plasma free fatty acids and the urinary excretion of catecholamines did not correlate significantly (IV).

After training, the concentration of *plasma* K^+ was greater in the trained group than in the control group after the physical work on a bicycle ergometer in (II) ($p < .01$). The respective ratios of Na^+ and K^+ were lower in the trained group in (II) ($p < .01$). The *Na^+ concentrations* in the plasma in a trained group were lower during the control forenoon ($p < .001$) and after the ergometer works ($p < .05$) while being higher during the control afternoon ($p < .001$) and after the marching and choice reaction test competition ($p < .05$) with no significant differences in Na^+/K^+ or K^+. After the training in (VI) no significant differences were found either in Na^+ or K^+ or in their ratio in any of the measurements (TABLES V and VI).

Urinary catecholamines and 17-hydroxycorticosteroids

After the conditioning procedure the urinary excretion of *adrenaline* was significantly lower in the trained group during the control forenoon ($p < .05$) and during the forenoon while waiting

for a flight ($p < .01$) in (I) (TABLE IX) and in the afternoon ($p < .001$) in (II). The excretion of *noradrenaline* was greater in the trained group during the control afternoon ($p < .001$) in (I) (TABLE XI) and during the control forenoon ($p < .05$) in (II). In (VI), no significant differences were found either in catecholamine excretion or in urine output (TABLE XIV). The catecholamine excretion did not correlate significantly with the *urine output* (VI). After training, no significant differences during rest or work between the groups were found in the urinary excretion of *17-hydroxycorticosteroids* (II, VI) (TABLE XIV).

TABLE XIV. URINE OUTPUT AND URINARY EXCRETION OF CATECHOLAMINES AND 17-OHCS DURING RELATIVE REST AND WORK ON A BICYCLE ERGOMETER DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines, 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

Variable	Groups n = 11	During relative rest: Measurement during training period (M) Index numbers are weeks after the outset				Test for loading effect [1] p
		M_0	M_3	M_6	M_9	
Urine output ml/min	Control	1.03 ± 0.16	0.87 ± 0.16	1.02 ± 0.17	0.78 ± 0.13	< .01
	Trained	1.03 ± 0.23	0.88 ± 0.11	1.35 ± 0.16	0.81 ± 0.09	
Adrenaline (A) ng/min	Control	5.3 ± 0.90	5.0 ± 0.76	4.7 ± 0.71	5.1 ± 1.31	< .001
	Trained	4.2 ± 0.68	4.8 ± 0.30	4.3 ± 0.74	4.6 ± 0.82	
Noradrenaline (NA) ng/min	Control	18.6 ± 1.76	15.3 ± 1.82	18.8 ± 2.28	19.5 ± 2.17	< .001
	Trained	23.6 ± 2.65	17.3 ± 1.80	22.1 ± 2.98	19.3 ± 1.71	
NA + A ng/min	Control	24.0 ± 2.36	20.3 ± 1.93	23.6 ± 2.99	24.7 ± 3.08	< .001
	Trained	27.9 ± 3.05	22.1 ± 1.98	26.4 ± 3.43	23.8 ± 2.08	
NA/A	Control	4.5 ± 0.87	4.4 ± 1.02	4.5 ± 0.60	5.2 ± 0.75	< .01
	Trained	6.3 ± 0.93	3.6 ± 0.29	6.1 ± 0.84	6.2 ± 1.49	
△ A ng/min	Control	△-NUMBERS TO RIGHT INDICATE THE CHANGE FROM REST TO WORK				
	Trained					
△ NA ng/min	Control					
	Trained					
△ NA + A ng/min	Control					
	Trained					
17-OHCS µg/min	Control	8.3 ± 0.67	7.9 ± 1.06	7.9 ± 0.55	8.5 ± 0.93	NS
	Trained	8.7 ± 0.60	8.2 ± 1.01	7.5 ± 0.42	7.4 ± 0.59	

Variable	Groups n = 11	During work of 46 minutes: Measurement during training period (M) Index numbers are weeks after the outset				Test for training effect [2]	
		M_0	M_3	M_6	M_9	During rest	During work
Urine output	Control	0.98 ± 0.24	0.77 ± 0.13	0.73 ± 0.13	0.74 ± 0.09	F = 0.785	F = 1.491
ml/min	Trained	0.73 ± 0.19	1.11 ± 0.26	1.27 ± 0.19	1.08 ± 0.29		
Adrenaline (A)	Control	16.9 ± 1.94	13.0 ± 1.31	15.1 ± 2.25	14.1 ± 2.03	F = 0.042	F = 0.068
ng/min	Trained	15.7 ± 2.20	15.6 ± 2.08	14.6 ± 1.82	15.0 ± 2.99		
Noradrenaline (NA)	Control	42.0 ± 3.04	36.2 ± 3.21	40.2 ± 5.28	41.8 ± 4.17	F = 0.134	F = 0.373
ng/min	Trained	50.2 ± 4.94	34.5 ± 2.71	43.0 ± 4.43	41.1 ± 4.51		
NA + A	Control	58.9 ± 3.69	49.2 ± 3.83	55.3 ± 6.42	56.0 ± 5.49	F = 0.653	F = 0.170
ng/min	Trained	65.9 ± 6.45	50.0 ± 3.42	57.5 ± 5.91	56.1 ± 7.15		
NA/A	Control	2.8 ± 0.37	3.1 ± 0.38	3.0 ± 0.35	3.3 ± 0.38	F = 0.423	F = 1.320
	Trained	3.9 ± 0.67	2.7 ± 0.32	3.2 ± 0.34	3.3 ± 0.37		
Δ A	Control	10.7 ± 1.23	8.4 ± 0.85	10.8 ± 1.69	8.7 ± 1.08	—	F = 0.052
ng/min	Trained	10.8 ± 2.02	8.2 ± 1.22	10.3 ± 1.33	10.7 ± 2.42		
Δ NA	Control	23.5 ± 2.62	20.7 ± 2.74	22.0 ± 4.01	20.9 ± 1.89	—	F = 0.250
ng/min	Trained	24.7 ± 3.96	16.1 ± 1.86	21.2 ± 2.21	21.9 ± 3.99		
Δ NA + A	Control	34.1 ± 2.50	29.1 ± 3.09	31.8 ± 4.61	30.6 ± 2.45	—	F = 0.031
ng/min	Trained	35.5 ± 5.24	25.9 ± 2.40	31.5 ± 3.13	32.7 ± 6.03		
17-OHCS	Control	8.0 ± 0.95	8.1 ± 1.05	7.3 ± 0.78	7.9 ± 0.55	F = 0.161	F = 0.031
μg/min	Trained	8.1 ± 1.07	7.5 ± 1.58	8.3 ± 0.59	6.8 ± 0.99		

[1] t-test for the total means between rest and work in 0-situation
[2] analysis of covariance for the between group difference M_0 as covariate and M_4—M_8 as criterion

TABLE XV. PREDICTED MAXIMAL CAPACITY OF O_2-UPTAKE AND STEADY STATE HEART RATE PER MINUTE DURING RELATIVE REST, SUBMAXIMAL PHYSICAL WORK AND RECOVERY DURING A NINE-WEEK PERIOD OF ENDURANCE CONDITIONING

(From the report SARVIHARJU, P. J.: Urinary excretion of catecholamines 17-hydroxycorticosteroids and concentration of certain blood parameters during progressive endurance conditioning, Public Health Publication no. 15, 1972, University of Jyväskylä, Finland)

Subjects	Measurement during training period [1]	During relative rest	During ergometer work I; 6 minutes 1200 kpm/min.	During recovery from work I	During ergometer work II; 20 minutes 900 kpm/min.	During recovery from work II	During ergometer work III; 19 + 1 minutes 900 + 1500 kpm/min. respect	Predicted $\dot{V}_{O_2}$ max. ml/min/kg of weight [3]
Control group (11)	M_0	[2] 64 ± 2.9	159 ± 5.0	73 ± 4.1	141 ± 5.3	70 ± 5.2	169 ± 4.9	57 ± 2.3
	M_3	60 ± 2.9	157 ± 4.5	73 ± 4.3	142 ± 5.3	74 ± 5.4	167 ± 4.8	59 ± 2.3
	M_6	63 ± 3.8	156 ± 4.4	72 ± 4.4	143 ± 5.6	71 ± 4.4	168 ± 5.1	59 ± 1.9
	M_9	60 ± 3.5	154 ± 4.4	71 ± 4.2	141 ± 5.4	73 ± 4.3	165 ± 5.1	60 ± 1.6
		[4] NS	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
Trained group (11)	M_0	60 ± 4.6	158 ± 5.5	74 ± 4.6	149 ± 6.0	78 ± 5.8	171 ± 5.4	59 ± 3.0
	M_3	60 ± 4.3	148 ± 5.5	64 ± 4.9	134 ± 4.9	65 ± 3.1	158 ± 4.6	66 ± 4.0
	M_6	57 ± 3.6	145 ± 4.4	64 ± 3.6	129 ± 4.4	64 ± 4.1	156 ± 4.6	69 ± 3.0
	M_9	56 ± 3.5	139 ± 4.6	60 ± 4.2	123 ± 4.6	64 ± 5.0	150 ± 4.6	74 ± 3.0

[1] M_0 = before training; M_3—M_9 = 3, 6 and 9 weeks after the beginning of training
[2] numbers are Mean ± S.E.
[3] with the indirect method of estimation of Åstrand and Ryhming (1954)
[4] analysis of covariance for between group difference; M_0 as covariate, M_3—M_9 as criterion

DISCUSSION

Loading

Blood parameters

The ergometer loadings used were selected in order to represent roughly the various amounts of the aerobic and anaerobic energy production during work. Except for the military marching in (II), all of the loadings also employed a slight to moderate anaerobic energy production, as can be seen in the concentrations of *plasma lactic acid.* All the loadings were submaximal in relation to the maximum of approximately 150 mg per cent as stated by Margaria (1963). Similar evidence is shown by the steady state heart rates which were far below the supposed maximum of above 200 per minute in young men (Karpovich, 1959).

On the other hand, the present results with respect to lactic acid in (II), (III) (TABLE III) and (VI) (TABLE II) do not agree very well with the evidence of Margaria (1963), who states that the maximum is reached about 5—8 minutes after stopping work. In (III) and (VI) (TABLES III and II) the average concentration of lactic acid five minutes after the moderate anaerobic work of six minutes 1200 kpm/min. was only about one half to one quarter of the respective values only one minute after a very similar kind of six minute work in (II). The reason for this is uncertain.

Obviously the loadings of six minutes at 1200 kpm/min., 20 minutes at 900 kpm/min. and 19 + 1 minutes at 900 + 1500 kpm/min., respectively, used in a sequential way in (VI), were not sufficient to produce a significant decrease in the *blood glucose* in spite of the fast before and during the experiment (TABLE II). However, this kind of decrease was demonstrated in a loading combination of six minutes at 1200 kpm/min. and 24 minutes at 750 kpm/min. in (III) (TABLE III). Due to the food intake during the experiments in (II) between the forenoon and afternoon and before the choice reaction test competition, the effect of loading

was very confused in this respect. These results in (VI) (TABLE II) are consistent with the results of some authors (e.g. Rougier and Babin, 1966; Young et al., 1966; Ahlborg, 1967; Keul and Haralambie, 1972), who have demonstrated only a minor tendency for the blood glucose to decrease at the beginning of prolonged physical work in postabsorptive subjects. The present result was possibly due to the glucose mobilization during the muscular work neutralizing the tendency to decrease because of the increased uptake of peripheral tissues. The relative contributions of different substrates for the energy metabolism during exercise are not yet well understood. However, the glucose uptake to the exercising muscle seems to be dependent on both the duration and the intensity of the work performed (e.g. Wahren et al., 1971). Acceleration of the hepatic glucose release is only seen when the glycogen reserves in the muscle disappear (e.g. Keul and Haralambie, 1972). It has been suggested that a decreased insulin level increases this hepatic output and possibly limits glucose oxidation in the working muscle, in this way maintaining normal blood sugar in the face of an increased glucose mobilization and oxidation (e.g. Paul, 1971). A minor part of up to about 20 per cent of the glucose release is explained by hepatic gluconeogenesis from lactate (Keul and Haralambie, 1971; Wahren et al., 1971). This over-all hepatic output of glucose may also be a result of direct adrenergic stimuli as suggested from evidence for the necessary innervation and its relation to the activation of hepatic phosphorylase (Shimazu and Fukuda, 1965).

Continuous lipolysis during the muscular work in (IV) (TABLE VII) is probable. The working time of six minutes did not suffice to induce a significant increase in the *free fatty acids concentration during the recovery after work,* which otherwise, it is suggested, has a decreasing effect on the plasma free fatty acids during work. This would be consistent with the evidence presented, for example, by Carlson et al. (1965), who demonstrates that the plasma FFA decrease at the beginning of excercise and increase rapidly after the cessation of exercise. Possibly the present slight decrease was only covered by the respective increase after the exercise.

The fate of triglycerides and glycogen seem to be closely connected with each others. The use of stored glycogen, first mainly

in white fibers (Stubbs and Blanchaer, 1965; Kugelberg and Edström, 1968) occurs at the beginning of all levels of exercises (Hultman, 1967; Pernow and Wahren, 1967) and its subsequent use, mainly in red fibers (Kugelberg and Edström, 1968), seems to depend upon the extent to which other fluids, particularly FFA from the blood, can supply energy needs (cf. Havel, 1971). The evidence for FFA is that the uptake into the legs during a bicycle exercise increases from about 25 per cent of total outflow transport at rest to 55 per cent for light work (Havel et al., 1967 a) and to 75 per cent during heavy work (Havel et al., 1967 b). The FFA inflow into the blood from adipose tissue increases rapidly with exercise (Havel et al., 1963). It is suggested that this is mainly a result of the adrenergic stimulation of the adenyl cyclase system in adipose tissue, leading to the activation of its hormone-sensitive lipase (cf. Havel, 1971). Evidence for this is provided by the rapidity of fat mobilization and the latters's sensitivity to infused catecholamines, especially beta-mimetics (e.g. Havel, 1965; Rosell, 1966; Rosell and Ballard, 1971) and inhibition of this process by beta-adrenergic blocking agents (e.g. Havel and Goldfien, 1959). However, some data indicate regional differences of physiological importance in the ability of the adrenergic neuro-humoral system to elevate the lipolytic rate, the sympathetic nerves to adipose tissue playing a greater role than circulating catecholamines (Rosell and Ballard, 1971). Furthermore, there is some evidence for the minor importance of noradrenaline in the mobilization of fats (Carlsten et al., 1965).

The present results regarding the *effect of mental loading on the plasma FFA* (TABLE VIII) do not agree with Bogdonoff and Nichols (1965), who demonstrated that the examinations caused a rise in the plasma FFA level. The present mental loading, reflected in a clear increase in sympathoadrenal activity, resulted in an increased urinary excretion of catecholamines (TABLE X). If we accept that the role of catecholamines partly induces fat mobilization, the present lack of a consequent change in the plasma FFA level remains unclear.

The present demonstration of the increased *hemoglobin* concentration and/or *hematocrite after exercise* (TABLE IV) agrees well with the results reported by many authors as reviewed and

confirmed by many authors (e.g. Holmgren, 1956; Hollman and Kastner, 1969). This increase was first suggested as resulting from the expulsion of blood from the depots (e.g. Barcroft and Stevens, 1927). At present this is generally interpreted as showing a decrease in the blood volume, probably induced by a passage of the plasma filtrate from the circulating blood to the interstitial space (e.g. Kaltreider and Meneely, 1940; Ebert and Stead, 1941). In the present work this shift seem to bee roughly related to the intensity of the exercise, the hemoglobin concentration and hematocrite fluctuating according to the varying anaerobic pattern of the loading phase (TABLE IV). However, during prolonged muscular work a relative hydremia would obviously appear, due to the need of decreased blood viscosity during the tendency of exsiccosis in order to continue intensive work succesfully (Israel and Weber, 1972) These authors demonstrated an increased hemoglobin concentration and an increased amount of erythrocytes only after the termination of very intensive prolonged exercise.

In spite of the five minute latency before the collection of blood samples after the exercise an increase in *plasma* K^+ was found, when the subjects were recovering from the second phase of pedalling in (VI) (TABLE V). This is consistent with some previous evidence regarding the increased concentration of K^+ in plasma due to muscular loading of even short duration (e.g. DeLanne et al., 1959; Kilburn, 1966; Metivier, 1969). Some authors suggest that the increase of K^+ is related to the amount of work done during repeated muscular work (Grob et al., 1957). They suggest that the organic phosphate compounds exist as K^+ salts in cells and that the release on K^+ during exercise is produced by their phosphorylation and glycolysis. It has been suggested that the increase in permeability known to result from depolarization of the muscle membrane (Fatt, 1954) results from the hypoxic state of the muscles due to the excretion of catecholamines (Highman et al., 1959). According to the postulation of Kilburn (1966), exercise produces acidosis in the muscle cells, some K^+ is exchanged for H-ions and both are released from striated muscles. He states that this is the finding most consistent with the increased H-ion concentration of arterial blood during moderate systemic exercise and in blood draining from the exercising forearm. However, it

was suggested that in view of the small fraction of change in muscle K^+ required to produce a large increase in extracellular K^+, some other mechanisms may explain the liberation of K^+ into venous blood during exercise. Possibly the increased permeability and the increase produced in the electrolyte concentration in plasma could be interpreted as a mechanism of protection necessary to maintain the blood osmolality and plasma volume, as stated by Metivier (1969).

No effect of exercise on K^+ concentration was found in (II), where six minutes of pedalling work was possibly even more anaerobic than in (VI). Because there was no reason to believe in any latency in the increase of K^+ in plasma, the possible explanation for the inconsistency in (II) would be the relatively great lability of this electrolyte in plasma which may also be due to various factors other than exercise or any other kind of loading as well as an inaccuracy in the present use of the method of determination. The permanence of the plasma Na^+ in (VI) (TABLE V) is similar to the previous data, which indicates that this concentration is quite stable (e.g. Schönholzer, 1959; DeLanne et al., 1959; Aurell et al., 1967).

Urinary catecholamines and 17-hydroxycorticosteroids

The average catecholamine excretions show a relatively great variation obviously due at least to group, methodological, situational, diurnal, day to day and seasonal variations. However, the present range of this variation agrees quite well with the variation which generally appears in studies and reviews on the topic (cf. Euler, 1964; Euler, 1969; Frankenhaeuser et al., 1969).

The present studies (I—VI) have shown indisputable confirmation of the large body of previous evidence, which states that in connection with mental and/or physical loading the urinary excretion of both *noradrenaline and adrenaline* may increase (TABLES IX—XII). Assuming that the rate of this excretion accurately reflects the total secretion into the blood as generally suggested (e.g. Euler, 1964), both the adrenal medulla and the sympathetic nerve endings are then activated. *During mental loading* the

change in the adrenomedullary secretion, *adrenaline,* will obviously be more sensitive as shown by an increased urinary excretion of adrenaline during waiting situations, as exemplified by passenger flight (Euler and Lundberg, 1954) or orbital flight (e.g. Jackson et al., 1961). The present evidence of adrenaline excretion during flight waiting (I) is parallel to these observations (TABLE IX). Some data reveal a consistent relationship between the amount of this adrenaline release on the one hand and the degree of mental loading and upleasantness on the other (Frankenhaeuser et al., 1965, 1967) well in agreement with other data reviewed by Mason (1968).

However, the views advanced by Funkenstein (1956), suggesting a simple relation between adrenaline and anxiety on one hand, and noradrenaline and aggression on the other, are not supported by the empirical data from catecholamine determinations (cf. (Frankenhaeuser, 1971). According to Schachter and Singer (1962), a physiological state does not in itself provide a sufficient measure to account for a psychological occurence.

Obviously an adaptation tendency may occur, when repeated exposures to a mental loading exist. In the present study this was found in (I) when the adrenaline excretion even during flight-waiting increased significantly before the training period but not after it (TABLE IX). The change of the respective *ratio of noradrenaline to adrenaline* could be considered parallel in significance (TABLE XII). These observations are consistent with the findings Hale et al. (1965) concerning an adaptation tendency due to increased flying experience, as suggested. This kind of adaptation tendency was not found in the response to the choice reaction test competition in (V) (TABLE X). Consequently this reflects the attempts to conceptualize those phenomena of emotion whose quality is argued as being explainable by the motoric impulse generated by the stimulus, whose value to an individual is appraised as positive or negative, helpful or harmful (Arnold, 1960). One possible way to interpret these emotional reactions would be to suggest the processes of coping activities or impulses, which Lazarus (1966, 1967, 1971) states to be generated by the stimuli from an environment and followed by the mobilization of energy and to have certain characteristics ordinarily attributed to emotional states,

and finally to result in physiological changes. Here we should be at the main point of convergence between physiological and psychological stress analysis, when the coping reactions or the consequent mobilizations disturb the physiological equilibria. In this theoretical frame, in my opinion, an adaptation process in the present mental loadings could be regarded primarily as a change in the coping processes modifying the pattern of physiological response, i.e. the sympatho-adrenal activity.

As to *adrenaline* excretion during the choice reaction test competition (TABLE X) and *noradrenaline excretion* during both the flight and choice reaction test competition (TABLE XI and X) no significant adaptation was found. This obviously indicates here to a certain kind of stimulus and response specificity propounded previously (Lacey et al., 1953; Lacey and Lacey, 1958). As to adrenaline excretion, the present evidence agrees with the suggestion made by Graham (1962) giving the broader view that in psychosomatic medicine »each attitude has its own specific physiological concomitants».

As to the excretion of noradrenaline and adrenaline during loading, the stability of the former as compared with the consistent change of the latter could be suggested as an expression of change, even in the response stereotypy, which is demonstrated as being quite consistent in individuals under different conditions of threat or physiological strain and from one occasion to another (Lacey and Lacey 1958). Consequently present evidence indicates the possibility of acquiring a specific response pattern by conditioning. In stress research it would also imply the necessity, except for strictly qualifying the individual pattern of response related to the loading concerned in the measurement, even to control the effect of these changes in stimulus and response specificity.

The increase in *noradrenaline* excretion *during flight* (TABLE XI) could possibly be partly induced by the mental load similar to the movie situation reported by Levi (1963). Obviously most of the excretion was a result of physical strain produced by gravitation, which is known to have an increasing effect on the noradrenaline excretion as demonstrated by the human centrifugation tests referred to in (I). The reason for the increase of noradrenaline during the choice reaction test competition is less clear (TABLE X).

Probably this was due to some static muscular tension in addition to slight indispensable muscle work during the test.

The present results indicate significant increase in the *noradrenaline* excretion *after* all types of *exercise* (TABLES XIII—XIV). This increase has been considered a part of the cardiovascular-response system regulating reactions in muscular work, while the mechanism responsible for the increased adrenaline output is not clear (Euler 1967). Frankenhaeuser et al. (1965, 1967) state a consistent relationship between the amount of adrenaline release and the degree of mental stress or unpleasantness and consider the adrenaline increase likely to be at least partly associated with the subjective emotional reaction accompanying heavy physical work rather than that elicited by the work itself (Frankenhaeuser et al., 1969). This was partly confirmed by the present results by the significant increase in *adrenaline* excretion *after* the thirty minute *muscular work* combination from slightly to moderately anaerobic in (III) (TABLE XIII). The loading of thirty minutes in (II) could be classified as a slightly less strenuous complex of the load and time. There are no definite ways of analyzing all the relevant factors concerned. However, the results of nonsignificant change in the excretion of adrenaline after the latter-mentioned work (II) and especially after the very strenuous four-hour military marching in (II) could be considered inconsistent with some previous findings as reported by De Schaepdryver and Hebbelinck (1969). With regard to the urinary excretion of catecholamines and their metabolites, these authors failed to differentiate between the two types of ergometer work during which there was a pronounced difference in lacticacidemia and went on to find a significant increase in the adrenaline excretion even after a mean aerobic ergometric task of 15 minutes duration. They suggested that the stimulating effect of muscular work on the sympatho-adrenomedullary system tended to be enhanced in an anaerobic type of work, where noradrenaline and adrenaline reached maximal values sooner than in the aerobic type of exercise.

The present effect of group differences between the separate studies remain uncertain. Consequently, however, it seems probable that it will be possible to load a person physically during an aerobic kind of work without any significant increase in the urinary

excretion of adrenaline. This could possibly be done even anaerobically, if the working time could be short enough. The role of coping processes and for example the role of perceived exertion (Borg 1962) on the one hand and that of the metabolic consequences during these circumstances on the other would be worth investigating in the future. Concerning the importance to the organism, those increased activities of the sympatho-adreno-medullary system producing generalized vasoconstriction in non-active regions, positive chronotropic and inotropic effects on the heart and metabolic consequences, would be, at least in extreme conditions, necessary for the potent regulatory processes preparing and maintaining the ability of an organism to cope efficiently with the muscular work conditions.

However during different intensities of exercise the relative importance of the adrenals, the blood catecholamines and the adrenergic nervous system, looked upon as a functional unit, is still obscure (Häggendahl, 1971). Some authors suggest that in general the level of circulating catecholamines is probably of less importance for most circulatory and metabolic effects than amines released directly from the nerves (Häggendahl, 1971; Rosell and Ballard, 1971). However, in some tissues receptors have been suggested as being present and very sensitive to small fluctuations in the circulating catecholamine levels (Häggendahl, 1971).

The examination of the urinary excretion of *17-hydroxycorticosteroids* was restricted to the measurements before and after the muscular work conditions in (II), (III), and (VI) (TABLES XIII—XIV). At first, the results are quite contradictory because of the significantly increased excretion of 17-hydroxycorticosteroids after the thirty minutes' ergometric exercise in (III). Otherwise the results could possibly be seen to agree with the previous evidence cited in this review of literature. Consequently, the first increase in the concentration of steroids in plasma after the outset of muscular work would be followed by a decrease due to the increased peripheral utilization of diffusion of the adrenal cortical hormone (Kägi, 1955; Hill et al., 1956; Cornil et al., 1965). This increased utilization would possibly be met by a corresponding increased secretion from the adrenal cortex. Therefore the present observations in (II) and (VI) could reflect the phases of this utiliza-

tion process already in operation sufficiently to be reflected in urinary 17-hydroxycorticosteroids, but not to have yet reached any significantly decreased level either. This would be due to the fact that the work time was not followed by any prolonged hypoxia, which according to the demonstrated amount will produce a transient increase in plasma concentration and urinary excretion of cortisol (Moncloa et al., 1965, 1970). The significantly increased excretion of 17-hydroxycorticosteroids after the thirty minute exercise in total in (III) could possibly be explained as being induced by the first experience history (TABLE XIII). This would be consistent with the previous data indicating that novelty is an especially potent stimulus to the pituitary-adrenal cortical response in man (Sabshin et al., 1957; Sloane et al., 1958; Davis et al., 1962) and suggesting that this impact of a novel situation may be lessened by reassurance and briefing aimed at reducing feelings of ambiguity and uncertainty (Wadeson et al., 1963; Mason, 1968).

To summarize the loading effects on the urinary excretions of both catecholamines and 17-hydroxycorticosteroids one may well suggest a consistency with the idea that the difference in steroid response to physical exercise and emotional loading on the one hand (Connell et al., 1958) and the affective states of differing quality on the other (Curtiss et al., 1960) does not agree with the nonspecificity in Selye's sense. This is especially true if one relates them to the balance between hormones rather than to some single hormone alone.

Endurance conditioning

Performance and blood parameters

A part from the inconsistency in (II), where the decrease in the *steady state heart rate during work* was most prominent in the group exposed to the least demanding training program, intensive progressive endurance conditioning resulted in a considerable *improvement in the physical fitness* of the test groups in every experiment (e.g. TABLE XV). This inconsistency in (II) was

probably induced by the better initial condition of the test group exposed to the heaviest training program as well as possibly by the existing optimal rate of endurance training without producing some kind of irritation state in the central or vegetative nervous system resulting in a higher level of heart rate. The latter possibility would be consistent with some previous observations of the state of over-training known to result in various characteristics such as hypertension, general nervous tenseness as well as a decrease in the efficiency of the metabolism (e.g. Prokopp, 1963). Prokopp (1963) suggests the overtraining to be an adaptation disturbance, the cause of which is to be sought in a relative suprarenal cortex hormone insufficiency, which can be treated favourably and quickly by giving suprarenal cortex hormone. The present results do not elucidate any significant difference in the 17-hydroxycorticosteroid response (TABLE XIV). Certainly the cortex insufficiency is not probable in this instance as it is during a course of very intensive training for top athletes. With reference to the symptoms Prokopp's concept of over-training seems to be equivalent to the concept of sympathicotonic or basedowoidian over-training, which is described as a state of insufficient change in the organism from work to recovery (Israel and Weber, 1972). Consequently the explanation of the irritation state could lie somewhere in the co-operation of the central nervous system — vegetative nervous system — endocrine functions, which Israel and Weber (1972) suggest controls the over-training process.

Generally one can consider that the training, used in the present studies was relatively strenuous, but not very close to maximal as represented by the training programmes of endurance athletes. To state the general importance of physical activity for the health of an individual, one probably can not find an argument for increasing the present intensity of conditioning procedures. However, the possibility of demonstrating a certain kind of absolute effect is then omitted.

The *heart rate during relative rest* is obviously not sensitive enough to show the effectiveness of training of the degree and duration, used here, although the means of the heart rates show a decreasing tendency as seen in (IV) and (VI) (TABLE XV). Also in the control group in (IV) and (VI) the submaximal heart rate

during a standardized ergometer test was found to decrease significantly after a six-week period, i.e. since the second measurement. One possible reason for this cannot be increased physical activity if the crude approximation of the physical activity of the control subjects is valid on the basis of their exercise diaries. It was supposed that some kind of adaptation to or »learning» of the experimental loading procedure has a central role in the understanding of this curious »training effect». This kind of improvement in the physiological state at a consistent level of performance can indicate, it is suggested, either the physiological adjustment to training or the psychological adjustment to habituation (Glaser and Griffin 1962). Direct measurements of oxygen consumption during loading might eliminate the effects of adaptation to the situation better than the measurement of the heart rate alone.

The fact that only a slight training effect was produced in (II) is obvious also from the finding of a significant effectiveness of training on the *plasma lactic acid* in (VI) (TABLE II) alone. Several investigations have shown this kind a reduced blood lactate concentration in the more conditioned subjects at a same submaximal (e.g. Crescitelli and Taylor, 1944; Saltin et al., 1969) and relative work load (e.g. Ekblom, 1969; Edwards et al., 1971). Training is also known to result in a increased capacity for accumulating lactic acid due to a greater utilization of anaerobic energy reserves as suggested, for example, by Knehr et al. (1942).

Much evidence has been presented showing that these phenomena indicate mainly the major biochemical adaptations occurring in skeletel muscles in response to endurance training. During exercise the functional steady state is attained at the point where the rates of mitochondrial electron transport and oxidative phosphorylation increase sufficiently to balance the rate of ATP hydrolysis (cf. Holloszy et al., 1971). This functional steady state in which oxygen consumption balances ATP hydrolysis during submaximal exercise is attained at lower concentrations of P_i and ADP in the working muscle cells of trained individuals as compared to sedentary individuals due to approximately twice as many mitochondrial cristae per gram in trained muscle as in untrained muscle (Holloszy et al., 1971). As a result, pyruvate and DPNH should be formed at a slower rate at a given submaximal work load, accounting for a

lower lactate production in trained muscles than in untrained ones (Holloszy et al., 1971). However, similar glycogen depletion after training at the same relative work load suggests that lower lactate values are not due to a lower lactate production by glycolysis (Saltin and Karlsson, 1971 b). These investigators have found evidence for the less pronounced splitting of the phosphagens at the same absolute work load in the most trained stage and they explain the lactate reduction to be possibly due largely to the activation of relatively more red fibers and some of the formed lactate to be possibly utilized immediately in other parts of the muscles, the latter hypothesis being supported by the data, namely an increased number of mitochondria after endurance training (Kiessling et al., 1971) and by Jorfeldt's data (1970).

The present finding of a greater *glucose concentration in the blood* during prolonged physical work in (II) is in agreement with the known effect of training to increase the stores of glycogen in human muscle (e.g. Short et al., 1969) and enhancing the capacity to synthesize intracellular glycogen (e.g. Morgan et al., 1971). Consequently there could be a decreased demand for the blood glucose utilization during prolonged exercise. This possibility seems also to be supported by previous evidence showing that the relative amounts of carbohydrate and fat utilized at different work loads depend on the level of physical fitness, with less glycogen (e.g. Saltin and Karlsson, 1971 a) and more fatty acid oxidation serving as a source of energy in trained individuals than in untrained ones (e.g. Havel et al., 1963, 1964; Issekutz et al., 1965; Leusink, 1972; cf. Keul and Haralambie, 1972). In addition to these biochemical adaptations of muscle, some training induced adaptations in the lipolytic system also appear and are reflected during exercise in a greater release of FFA from adipose tissue in trained than in untrained subjects (e.g. Havel et al., 1964; Issekutz et al., 1965; cf. Keul and Haralambie, 1972). During exercise, this fatty acids mobilization has a sparing effect on glycogen depots (Issekutz and Paul, 1968) and possibly an effect on blood glucose concentration during prolonged exercise. During the more short-term ergometric work in (IV, VI), no significant differences in blood glucose on in *free fatty acids* were found between the trained and untrained groups (TABLES VII and II).

After the training the trained group does not seem to respond to exercise with such a prominent increase in the *hemoglobin* concentration *and hematocrite* as the control group (TABLE IV). There seems to be a logical explanation to this change due to the endurance conditioning. It has been suggested that the increase in the hemoglobin concentration is a compensatory function for the reduced oxygen saturation of the arterial blood necessary to increase the oxygen transport capacity (Kjellberg et al., 1949; Holmgren, 1956). Consequently the increased circulatory capacity of the trained subjects will result in a less pronounced need to mobilize that compensatory mechanism during an exercise of the same level of intensity.

In (II) the observations of *plasma* K^+ *and* Na^+ tended to show a possibility that the trained subjects would react at least to a short and intensive physical loading with more increased concentrations of plasma K^+ and with a lowered *ratio of* Na^+ *to* K^+ and Na^+ than would the untrained subjects. These results were not confirmed in (VI) (TABLES V and VI). Some previous evidence suggest that physical training will increase the internal K^+ in skeletal muscle cells during rest and the capability to empty these stores when exposed to exercise and thereafter to result in an increased exchange of K^+ and Na^+ in the cells (Nöcker, 1964). Consequently it would be more probable during the same level of exercise to find a lower concentration of K^+ in plasma in the trained subjects than in the untrained ones due to the possibly greater efficiency in muscle work. The over-all greater lability and the result of no significant differences between the trained and untrained subjects in (VI) does not support the previous idea of some kind of greater sensitivity in the trained subjects to emptying the K^+ stores during exercise.

Urinary catecholamines and 17-hydroxycorticosteroids

Besides the methodological errors other uncontrolled factors might probably have had an effect especially on the level of hormone excretions. Such hormone diurnal and seasonal rhythmicity is known to be relevant with reference to catecholamines

(e.g. Hale et al., 1966) and 17-hydroxycorticosteroids (e.g. Bartter et al., 1966). The rhythmicity does not necessarily follow a 24-hour periodicity as suggested by Orth et al., (1967). This makes it possible that the present aim to control the diurnal fluctuation by performing the measurements at the same time of the days through the whole period of experiment will not suffice to eliminate the error due to rhythmicity. Lack of sufficient data makes it impossible to evaluate this effect. Relatively minor evidence was found to support the hypothesis of a decreased sympatho-adrenal activity due to endurance conditioning. The present evidence refers to a decreased *adrenaline excretion* in situations of relative rest, for example, in the forenoons of the control days in (I) (TABLE IX) and in the afternoon in (II), but not in actual stress situations such as during flights (TABLE IX), choice reaction test competition (TABLE X), during exercise (TABLE XIV) or not even in the afternoon of control days (I) (TABLE IX). This would suggest that the effect probably manifest itself at full rest, for example, in sleep, and yet does not extend even to a low level of stress such as in light daily work. Training of much greater intensity and continuity than the kind used in the present studies might be needed to extend the effect to cover the reactions of catecholamine excretion during some physically and/or mentally more loading conditions.

The present data concerning *noradrenaline excretion* are even contradictory to the hypothesis, because a more increased excretion of noradrenaline was found in the trained subjects in the afternoon of a control day in (I) (TABLE XI) and in the respective forenoon in (II) with no significant difference between the groups during physical or mental loadings (TABLES XIV and XI). Because of the well known effect and a relative specificity of physical loading on this excretion, it could have been expected in particular that endurance conditioning would induce a decrease in the loading of the sympathetic nervous system at the same level of physical work. Because of the inconsistency in the results of the first studies and of the lack of significant differences in catecholamine excretions in (VI) (TABLE XIV) the suggestions presented will remain quite hypothetical.

There are some points of view to be considered as factors possibly masking the effect of training on the endogenous excretion

of catecholamines reflected in the urinary excretion. Wurtman (1965) suggests that the fraction of the active material of catecholamines metabolized or rebound before its excretion does not necessarily remain constant, when there is a change in its rate of release. Further there is an inconsistency in previous studies as a whole, as to the relationship between the urine output and the excretion of catecholamines. Insignificant correlation between these functions was found to be consistent with previous data (e.g. Bloom et al., 1963; Levi, 1963; Graham et al., 1967) and not with some others (e.g. De Schaepdryver and Leroy, 1961; Dawson and Bone, 1963; Hathway et al., 1969).

Evidence has also been presented stating that the noradrenaline excreted in the urine is solely derived from the catecholamines in circulating blood, that its clearance is not affected by changes in urine pH or flow and that a partial reabsorbance of about 40 per cent from the glomerular filtrate exists (Overy et al., 1967). However, this mechanism of reabsorbance was not identified with certainty since the possibility of metabolic degradation by means of catechol-o-methyl transferase or by conjugation was not exluded. The investigator feels that anyway this evidence suggests the possibility of intervening mechanisms which could change the sensitivity of the direct measurement of urinary noradrenaline as a general acceptable indicator of sympathetic activity. The change would be in rebound, degradation and reabsorbtion mechanisms due to chronic exposure to intensive physical activity.

Besides for the known antidiuretic effect of acute exercise many authors suggest even an emotional antidiuresis in man and animals; this is reviewed and demonstrated in dogs with remarkable and stable individual differences suggesting the participation of genetic factors in the determination of the psychogenic renal reactions (Corson and Corson, 1971).

During the conditioning in the present study (VI) we see a slight tendency to a relative increased *urine output* during the exercise period as compared to the pre-exercise period in the trained group, instead of the previous effect of the exercise when it decreased the output (TABLE XIV). Omitting the possible influence of the latency time before the end of urine collection these results, if accurate, would agree with the suggestion of

Hatch et al., (1956) that the plasma flow and filtration rate are closer to normal in subjects, who could increase the minute volume more effectively as do those in good physical condition. Despite data concerned with the positive correlation of infused catecholamines and their urinary excretion under normal conditions (e.g. Elmadjian et al., 1958), we cannot exclude the possibility of producing in relatively more decreased urinary excretion of catecholamines in the nontrained subjects than in trained ones in a case of possibly more increased excretion into the blood in nontrained subjects under the same physical work load.

After the conditioning no significant differences between the groups were found in the urinary *excretion of 17-hydrocycorticorticosteroids.* The over-all variations and renal functions discussed in connection with catecholamines in this text may be valid even in this case. One can suggest the excretion of 17-hydroxycorticosteroids to be a resultant of many factors such as the secretion, peripheral utilization and excretory functions. Since we do not have explainable systematic data about these function profiles during muscular work of varying degrees of intensity and duration, the present method of collection seems to be crude and holds quite insufficient promise to be valid in the present kind of use in the evaluation of the 17-hydroxycorticosteroids response in exercise.

SUMMARY AND CONCLUSIONS

Experimental studies on healthy young men undertaken with similar designs were performed to throw light on the effects of psychophysical stimuli and various types of regular moderate progressive endurance conditioning on the following biochemical correlates of psychophysical loading: Urinary excretion of noradrenaline, adrenaline and 17-hydroxycorticosteroids, and hematocrite value, concentration of blood hemoglobin and glucose, plasma lactic acid, K^+ and Na^+. The loadings used for this purposes were the following: A relative rest situation (forenoon and afternoon of control days), seven bicycle ergometric tasks of various combination of intensity and time, four hours' military marching, a highly-motivated competition in 54 and 45 minute psychomotor choice reaction tests, waiting for a strenuous flight as a pilot and subsequently piloting during low-level attack and stunt flying totalling 70 minutes.

The progressive conditioning programs, from nine to twelve weeks, running or bicycle ergometric work, from half an hour to one hour at a time, resulted in a significant improvement of physical performance, as measured by a decreased heart rate during a standardized task and an increased predicted capacity of maximal oxygen uptake. At its height even a significant decrease in plasma lactic acid concentration was found in the trained group after anaerobic muscular work.

The present approach when related to certain previous data holds sufficient promise to support the following suggestions:

1. During acute muscular work consisting of varying anaerobic and aerobic periods the concentration of blood hemoglobin and the hematocrite value fluctuated and was significantly higher during more intensive anaerobic phases of work. During endurance conditioning this tendency to fluctuation seemed to decrease slightly in the trained group. The groups differed significantly in hemoglobin concentration, the untrained group

showing greater mean value than the trained group after the initial phases of exercise. However, the consistent and similar tendency in hematocrite values was not found to be statistically significant.

2. Plasma potassium concentration increased significantly during muscular work and the level was higher for at least some minutes after prolonged work. The sodium concentration did not change significantly. The change in the potassium concentration resulted in a respective significant change in the ratio of sodium to potassium in plasma. No significant consistent changes due to training were found.
3. A significantly increased concentration of plasma free fatty acids was found during recovery only after a total of 26 minute two-phase muscular work, which agrees with the findings of previous authors, namely that an increased lipolysis occurs even during early phases of exercise. The first six-minute task was not yet followed by any significant change in free fatty acids concentration during recovery. Any respective change in this parameter did not even occur after an emotional loading which resulted in a significant increase of the urinary excretion of both noradrenaline and adrenaline. This is consistent with the previous suggestions that the lipolyzing effect of endogenous catecholamines is of minor importance.
4. During muscular work of four hours' duration the concentration of blood glucose remained at a higher level in individuals having a greater capacity for physical performance. This possibly refers to greater glycogen stores known to be a result of proper training.
5. Emotional loading during flight-waiting was reflected in the increased urinary excretion of adrenaline. More advanced change was found with the respective increase also in the noradrenaline excretion during some exposures possibly due to, for example, contributory static or dynamic muscular work of even minor groups of muscles during the choice reaction test competition or due to this and g-loads during flight. The evidence agrees well with the previous suggestions.
6. A smaller increase in adrenaline excretion after training with reference to flight but not to the choice reaction test competi-

tion suggest that an adaptation tendency occurs differentially during various exposures to emotional loading.

7. Relatively moderate intensities of muscular work during 30—46 minutes resulted in a significant increase in the urinary excretion of both noradrenaline and adrenaline possibly due to both the physically and mentally loading characteristics of this kind of exposure. The bicycle ergometer work seemed to represent such a kind of loading, which may under moderate intensities have an even more pronounced effect on increasing the excretion of adrenaline than on that of noradrenaline. Generally the present evidence for the effect of exercise on catecholamine excretion into the urine agrees well with the present suggestions and confirms the previous data of many authors.
8. After various combinations of exercise quality, intensity and duration the urinary excretion of 17-hydroxycorticosteroids was measured. As compared to the preceding relative rest, only in one study was a significantly increased average excretion found. This was after a relative intermediate intensity and duration of ergometric work. The presented change pattern in the urinary excretion of 17-hydroxycorticosteroids during exerxise is supposed to parallel suggestions that this pattern can be generally related to the emotional characteristics of the situation, for example, due to the novelty of the experience. A suggestion with reference to present and previous findings was made that the ratio of the increased secretion and the peripheral utilization of adrenal steroids is possibly relevant enough to make necessary the measurement of the 17-hydroxycorticosteroid response by a profile, i.e. to have several measurements during an acute loading period. This would necessitate making the determinations from the blood.
9. After conditioning programs only in two studies was the average urinary excretion of adrenaline significantly less in trained subjects than in untrained ones. This was the result during certain relative rest situations. Some of the respective urinary excretions of noradrenaline were found to be significantly more in the trained subjects but the results were inconsistent. Other significant differences between the groups, induced possibly by the training, were found neither in the urinary excretion of

catecholamines nor in the urinary excretion of 17-hydroxycorticosteroids. Consequently due to the inconsistency of results, no indisputable evidence was found for the suggested changes in the hormonal regulatory functions reflecting changes of excretory pattern in urine. Logically, of course, a non-effectiveness, at least of the intensities of conditioning procedure used could be the truth. However, the inter- and intraindividual variations in hormone excretions obviously due to several endogenic and exogenic reasons were huge. This kind of fact renders it difficult to prove statistically even the possible moderate changes during a prolonged period of time. From another viewpoint an improved technique of determination of hormones from blood would possibly be the recommended way in future. This is emphasized by the fact that the changes in renal functions related to the hormone excretions due to chronic alterations of prolonged physical activity are not entirely known.

Generally as to the functions of the adrenal gland and sympathetic nervous system during psychophysical loading it seems to be theoretically valid to suggest that the acute changes and the adaptation mechanisms correlate with the state of training on the one hand and the physical performance on the other. However, very detailed design and advanced technique will be needed in the present kind of research in future.

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