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A STUDY OF THE

PHYSIOLOGICAL RESPONSES OF HUMAN BEINGS

IN ADAPTATION TO STRESS, **IN** DIFFERENT SIMULATED LABORATORY

ENVIRONMENTAL CONDITIONS.

By

MICHEL SALIM MROUE

A thesis submitted in fulfilment of the requirements

for the award of

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Supervisor: Dr. **E.J.** Hamley Department of Human Sciences

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ABSTRAC

A study in the search for the physiological responses in human beings in adaptation to stress. in different simulated laboratory environmental conditions with no protective clothing mainly for a short period of exposure.

Different groups of subjects were exposed to one of the following environmental conditions:

A - (at rest) $(1\frac{1}{2}$ hours) - 10°C above and below ambient temperature.

From the analyses of urine, sweat and blood, it was found that incomplete adaptation has occurred. during which the physiological processes responded to the environmental changes in a conservative way to counteract the effects of serious stress in order to *maintain* body homeostasis. Urine and blood-analyses indicated certain similarities *in* processes and levels of the parameters under study and those clinically observed changes due to clinical disorders.

Parameters studied were:

Urine: Nat, K^+ , Cl^- , urea and creatinine.

Blood: Haemoglobin; P.C.V.; Red and white blood cell count. Heart rate; blood pressure. Skin and oral temperature.

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1. INTRODUCTION

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1 INTRODUCTION

What is environmental stress?

To what extent, and within what precise limits, can the human body react physiologically to adapt to environmental stimuli7

To begin with, the word stress is misleading. To avoid confusion I would like to define the word stress as used throughout this thesis as a condition or state caused by an external stimulus, which upsets the human body from its normal balance of body functions.

To maintain homeostasis, (i.e. the constancy of the internal environment), adjustments of the physiological mechanisms and activities are made in response to any influence (from the external environment) on the body that tends to disturb its steady state and encourages survival in this external environment.

The compensatory responses of homeotherms to environmental stress caused by temperature changes are through acclimation and acclimatization. Acclimation consists of physiologically well-marked changes which occur in reaching:equilibrium:withJcontrolledcenvironmental conditions; while acclimatization refers to the adaptive changes which occur under more complex situations in nature.

The body homeostasis, response to environmental stress is similar to the body response to a disease, where feedback control reactions tend to maintain internal constancy; at some limiting gradients their control reactions fail and the internal state changes abruptly, resulting in severe illness.

Although the body temperature of acclimated man is held in a narrow range, a lowered perspiration threshold, increased production of dilute sweat and improved vascular responses are the general effects of exposure to heat; similarly the ability to maintain high rates of heat production in cold climates is the most significant effect of acclimation to a cold environment.

Thus man's ability to survive and work in a hot or cold climate depends on physiological mechanisms and. behavioural responses by which he can alter the effect of new environmental conditions so as to heat or cool the body to the zone of comfort. Thus the body acquires acclimation to the new external environment through these compensatory changes, and as a result the organism seeks to regain its normal physiological constants through the process of acclimatization.

To meet the challenge of climatic changes of heat and cold, this research was designed to study the physiological responses of the human body in adaptation to stress in different simulated environmental conditions and for different durations.

Many studies have been made based on long-term exposure to different environmental conditions in search fer acclimatization, which is the process of adaption to an environment other than that to which the subject was accustomed. and which, when completed, enables the subject to retain his capacity to live and work in the new environment without showing symptoms of distress.

This study was carried out to examine mainly the effects of short-term exposure to different environmental conditions, and to see whether acclimation had occurred during the exposure.

In our daily life we are exposed to great cbanges in temperature conditions between indoors and outdoors. This situation is similar to a short-term exposure. How well do we adapt to such changes? Can we accommodate properly and to what extent do the physiological systems respond in maintaining the operating equilibrium.

Keeping in mind the limitations to which this search was confined, the environmental conditions ranged as follows:

A selection of parameters were chosen guided by the work conducted previously on similar sorts of experiments.

They were as follows:

(i) Blood

 (a) - Haemoglobin

(b) - Haematocrit

(c) - Red and white cells count

(ii) Urine analysis

(a) - Electrolytes Na^+ , K^+ and Cl^-

 (b) - Urea

(c) - Creatinine

- (iii) Oral temperature
	- (iv) Skin temperature
		- (v) Heart rate

(vi) (vii) Blood pressure Sweat rate $(a) - Na⁺$ $(b) - K^{+}$

, .

Not all of these parameters were measured in any one experiment but different combinations were planned in designing the environmental condition and duration of each experiment to meet the availability of the necessary equipment and the willingness of the subject to provide samples for the 'parameters under study.

In an attempt to test the validity of the main findings, the work was extended, to include 24 hours exposure under one environmental condition and to give us an insight to the level of acclimation acquired and to what extent the physiological system responded.

2. LITERATURE REVIEW

2. LITERATURE REVIEW

It was obvious that the effect of environment on man had been receiving a great amount of attention, and that any progress in this field of research required a modern fine control of climatic variables, to enable the investigators to study and predict the. capacities and tolerance limits of people living in extreme weather conditions. Thus the idea of an environmental chamber with which climatic variables could be controlled was developed.

Experiments in climatic chambers were conducted in such a way that subjects were exposed to certain durations at certain environmental conditions to meet the requirements and the aims of the experiment under test. The physiological responses were measured in terms of heart rates, sweat rates, body temperature. The physiological responses due to the effect of the environment were measured by examining the differences *in* response between the initial. preexposure state and the final end-exposure state.

Acclimatization in man to both heat and cold showed a variety of responses which indicated that fundamental physiological, biochemical and endocrlnological changes took place which were not apparent prior to acclimatization.

Thus the purpose of this review is to present the physiological changes which occured during exposure to different environmental conditions and to give us an insight of what to expect by applying short-term exposure, to combine these theoretical expectations with the results obtained, to search for a deeper . understanding of the mechanisms involved, and to open up the possibilities of useful diagnostic and therapeutic aids to overcome thermal stress.

Acclimatization and acclimation are adaptive changes that occur, *in* the former, under natural conditions when multiple factors are involved, where as the latter consists of changes that occur under laberatory conditions where only one factor is varied. (Prosser, 1970). Acclimation is the equilibrated steady state,

Repeated exposure to the same work and temperature would result in a progressive improvement in the performance of the task and acclimatization would occur rapidly. (Lichna, , 1950, Robinson, et al. 1943, Wyndham, 1967).

In man acclimatization to cold environment is characterized by a decrease in shivering which is accompanied by a decrease in total oxygen consumption. (Davis 1961, 1967, 1963; Joy et al, 1962).

Water exchange through the kidney, skin and respiratory tract may range from $1-2$ liters per day in individuals in a cool environment to about 10-12 liters/day in individuals working in hot environments. (Robinson and Wiegman, 1974).

Even when allowance is made for sweat loss, the urine flow at a given water load is halved when temperature is increased from 32 to 49^oC. (Kenney and Miller, 1949).

, The effect of kidney hemodynamics is one of the classic experiments where a decrease in renal blood flow from 695 ml/min at 21° C to 426 ml/min at 50 $^{\circ}$ in five resting male subjects was shown (Radigan and Robinson, 1949). Also they reported that glomerular filiration rate dropped in the hot environment. Even moderate exercise in the heat was reported to cause a further decrease in glomerular filtration rate. (Smith et al., 1952).

Dehydration causes a marked renal conservation of water. Thus the decrease in urine flow during dehydration can be attributed to the decrease in glomerular filtration rate and to an increase in antidiuretic hormone (ADH) secretion. (I'toh, 1960.)

Salt outputs by the kidney and sweat glands were found to be reduced by increased mineral corticoid activity in response to salt deficiency. (Robinson et.al, 1955).

Salt depletion, induced by heavy and sustained sweating, is followed by decreased urinary output of water, sodium, potassium and chloride. (Ladell, 1964).

During heat exposure there was a decline in the urinary Na⁺ and CI^- concentration but an increase in K^+ concentration (Kanter, 1955). The decrease in renal NaCl was attributed to the decrease in glomerular filtration rate, supported by adrenocorticoid activity, while K^+ increase was due to rapid cellular dehydration with a large release of K^+ from cells. (Itoh, 1960).

Nat/K⁺ratios decreased during acclimatization to heat, due to an increase in ,aldosterone activity. (Macfarlane 1956).

An increase in the excretion of urinary creatinine in subjects exposed to heat, was due to increased net loss of muscle creatine . Thus enhanced creatinine output was due to an increase in urinary nitrogen losses. (Streeten et al., 1960).

Sweating is one of the important factors that occur during acclimatization to heat stress. It provides a means of heat loss by surface evaporation of water. Sweating is the most effective

means of maintaining thermol regulation and hence evaporative cooling is found to increase as the environmental temperature increases.

Insensible perspiration, which is water reaching the skin surface by diffusion, . constitutes a small proportion of body sweat. It increases with skin temperature, but decreases with an increase *in* water vapour pressure at the skin surface (Hale et·al 1958). However, because it is a diffusate it is virtually solute free. (Ladell 1964). Sensible perspiration represents the true secretion of ecrine glands of the skin. Its composition has been reviewed by Robinson and Robinson (1954), Weiner and Hellman (1960}, and It'oh (1960).

When the environmental temperature exceeds 29°C, evaporation from skin is increased by increased sweat secretion (Robinson and Wiegman, 1974), providing the evaporative cooling mechanism for the body. In hot humid climates sweat largely drips from the skin limiting the evaporative cooling effect and resulting in aLsmaller fall in body temperature, whereas in hot dry climates, the same increase *in* sweat production brings about a larger reduction in body temperature because the water is evaporated. (Fox, 1965).

Since sweating is the major source of heat loss, an increase in sweat rate would be a beneficial adaptation. The fact that sweat rate increased·· due to acclimatization has been confirmed by several workers (Moss, 1923; Adolf and Dill, 1938; Robinson et al. , 1941; Wyndham, 1967; Ladell, 1957 and Eichna et.al.,1954).

The important constituents of sweat, from the point of view of physiological responses to heat, are sodium, potassium and chloride ions. An increase in skin or body temperature produces an increase in the salt content of sweat, as does an increase in sweat rate (Robinson, 1968).

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The chloride concentration in sweat was found to be affected by skin temperature, rectal temperature, sweat rate, and individual variations (Johnson, Pitts, and Consolazio, 1944). They found an increase in chloride concentration with an increase in rectal and skin temperatures. Lowering the skin temperature reduced the chloride concentration. They showed that sweat sodium concentration behaved like chloride concentration, but not potassium.

A decrease in sweat sodium concentration, during acclimatization of men working in the heat, was a feature of the acclimatization ϵ mechanism and beneficial to the maintenance of body fluid volume and composition. (Dill et al., 1933). These results were confirmed by Bass et al., 1955, where they attributed the decrease in sodium and chloride concentrations largely to the decrease in rectal and skin temperatures. They found that a more dilute sweat was produced during the acclimatization process and concluded that the sweat glands excreted more water relative to solutes as acclimatization progressed.

It was suggested that the decrease in sweat sodium concentration was due to the secretion of aldosterone in men exposed to heat (Kenney, 1963).

Some workers reported that the decrease in sweat rates was due· to the sweat gland mechanism being fatigued. (Gerking and Robinson, 1946).

Ladell, 1951; Thaysen and Schwartz, 1955; Weiner and Hellman, 1960; Peter and Wyndham, 1966 also attributed the decline in sweat output to glandular fatigue.

Some 'investigators attributed the decline in sweat rates to a central nervous failure of the sweat mechanism (Wolkin et al., 1944; Kuno, 1956). Others suggested that the mechanism involves excessive skin hydration around sweat gland orifices, resulting in the occlusion of the sweat gland ducts and causing the reduction in sweat during heat exposure. (Randall and Peiss, 1957; Collins and Weiner, 1962).

On exposure to heat the onset of sweating was achieved when the rectal temperature of, about 38° C was reached which took up to 20° C depending on the magnitude of heat stress and rate of work (Ladell, 1951). With hard work, sweating is not dependent upon body temperature (Robinson, 1959).

It is not clear whether the mechanism responsible for the decrease in sweat rate is an adaptive device on the part of the organism or a funetion of salt intake. but from the literature the general trend is directed towards peripheral changes rather than central nervous system fatigue.

Maintenance of a constant body temperature is a neat balance between heat production and heat loss. It demands a sensitive thermostat in the brain with a capacity not only to use the heat formed as a by-product of metabolism but also to increase the output of metabolic energy as required. Core temperature. measured at different sites such as the oesophagus,

tympanic membrane, ear canal, mouth and rectum, differ from one another, but behave in the same way when work rate or environment is changed, although the time relations and magnitudes of the changes may vary from site to site. (Piizonen, 1970).

The normal deep body temperature in man at rest is 35.8 - 37.7° C. This level is kept fairly constant by maintaining a balance between heat gain and heat loss. Investigators showed that over a wide range of environments the core temperature is independent of the environmental stress and it was kept at a level within the normal range (Nielson, 1938; Winslow and Gagge, 1941; Robinson, 1949; Wyndham et.al., 1952 a, b; 1954; Astrand, 1952; Nielson and Nielson, 1962; Lind, 1962; 1963, a, b; B1ackley, 1965; Saltin.and Hermansen, 1966; Webb and Annis, 1967; 1968; StolwYk, Saltin and Gagge, 19€8). This was explained by the complicated physiological mechanisms of the body to keep the core temperature, the vital part which plays the main role in . survival, constant. When this temperature increased or decreased to the extremes through illness or accident, the thermoregulatory mechanisms fail leading ultimately to death.

Skin temperature responds rapidly and sensitively when an individual is exposed to an environment warmer or colder than a neutral environment. Skin temperature has two characteristics *(a)* - rapid change in skin temperature when exposed to a different environment (Adolph, 1946); (b) - the final skin temperature during cold exposure is a function of air movement and ambient temperature and during heat exposure it is, in addition, a function of ambient humidity. Thus, skin temperature is lower when ambient

temperature is lower and skin temperature is higher during exposure to high temperatures and high humidities. (Goldman, Green and Iampietro, 1965; Iampietro, 1961; Iampietro, Bass and Buskork, 1958; Iampietro and Buskork, 1960; Iampietro, Chiles Higgins and Gibbons,1969; Iampietro, Fiorica, Higgins, Mager. and Goldman, 1966; Iampietro, Goldman, Buskirk and Bass, 1959; Iampietro, 1971).

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On exposure to heat, skin temperature increased over the whole body and stabilized at a certain level, which is above the normal level, whereas upon exposure to cold the skin temperature dropped.

The adjustments of the cardio vascular system in response to heat, plays an important role in adaptation to heat stress. Several aspects of cardio vascular responses have been reviewed by Eisato, (1956), Thaiser, (1965), and Wyndham, (1973).

It was accepted that cardiac output increased on exposure , " to heat stress, and adjustment of the cardio vascular system to such exposure was confirmed by many investigators (Wyndham et al., 1954; Eichna et al., 1945; Kerslake, 1972).

A decrease in pulse rate was one of the most classical findings in : repeated heat exposure. (Bean and Eichna, 1943; 'Robinson et al 1943; Eichna et.al., 1945; Harvath and Shelley, 1946; Eichna, 1950; Bass et al., 1955). This adjustment in cardioyascular response to heat was reported by many investigators (Wyndham et,al., 1954; Eichna et.al.; Kerslake, 1972) and confirmed by many investigators. (Scott, Bazett, and Mackie,1939; Bean and Eichna, 1943; Taylor, Henschel and Keys, 1943).

The increase in cardiac output and the decrease in diastolic pressure were the result of reduction in peripheral resistance by cutaneous vasodilation for purpose of thermal regulation during heat exposure. During heat there is initially an increase in plasma volume (Senay, 1965), whereas during·cold exposure cutaneous vascoconstruction, for the purpose of reducing heat loss, causes an increase in blood pressure.

There is no doubt of the important role which the endocrine system plays in heat acclimatization, through the complex readjustments required by the body.

, Aldosterone levels in urine are reported to be higher in summer than those in winter, due to the intermittent sweating during summer which enhances aldosterone production (Yoshimura, 1960).

Deprivation of salt brought an increase in aldosterone excretion (Biddle et al., 1955), and salt loading reduced it. (Thorn et al., 1957).

The aldosterone participates in the regulation of renal and sweat gland losses of salt during exposure to heat. The amount of aldosterone increases with an increase in sodium loss in sweat (Collins and Weiner, 1968). A fall of sodium levels in man increases the amount of renin and angiotensin in plasma (Brown et al., 1964), which in turn stimulates aldosterone secretion. Thus, lowered plasma sodium, raised potassium concentration, and the action of angiotensin 11 stimulates aldosterone excretion. (Blair - West et al., 1963). The aldosterone excretion

explains the mechanisms of sodium conservation taking place during heat, i.e. reduced sodium concentration in urine and sweat. (Streeten et al., 1960).

ADH or vasopressin (antidiuretic hormone) is secreted in urine and its production is stimulated by a fall in blood volume or an increase in osmotic pressure during exposure to heat. (Itoh, 1960). Vassopressin acts by activating adenyl-cyclase which increases renal cyclic AMP, resulting in more energy supplies to kidney cells and increasing permeability of distal tubule and collecting duct to water and sodium.

3. EXPERIMENTAL DESIGN AND PROTOCOL

Experimental Design and Protocol,

3.1 Experimental Design

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In searching for a physiological indication in human beings as a response of adaptation to stress in different simulated laboratory environmental conditions, the following initial considerations governed the selection of experimental conditions:

- (a) Safety and availability of subjects.
- (b) The necessity of a medical doctor to collect large volumes of blood for different analysis, restricted the search, only for the study of packed cells volume and haemoglobin in some of the experiments.
- (c) Random selections of temperatures and humidities, which are similar to many envionmental conditions on our planet.
- (d) Correlation between the effects of two environmental conditions on the same subject.
- (e) Short-term effects of different environmental conditions on working men.
- (f) Short-term effects of different environmental conditions on resting subjects.
- (g) Correlation between the effects of short-term exposures to two humidities on the same subjects.

3.2 Experiments

Restricting the research investigation to the above-mentioned factors the experiments were designed and conducted as follows:

Experiment A

A group of subjects was exposed to temperature of 10° C difference from ambient temperature. for a period of $1-1\frac{1}{2}$ hours. Heart rate was recorded, sodium, potassium, and creatinine levels in urine were measured. Heart rate was recorded and samples of urine were collected at the beginning, mid-point, and end of exposure

Experiment B

This experiment consists of three parts:-

- i. Different groups of subjects were exposed to three environmental conditions (250 C, 67% Rh), (300 C, 50% Rh) and (350 C, 38% Rh).
- ii. A group of subjects Was. exposed to two humidities (80% Rh and 38% Rh) at a comfortable air temperature
- iii.A group of subjects was exposed to two temperatures 30°C \mathbf{A}^{\dagger} and 350 C at low humidity.

In these three parts heart rate was recorded and sodium, potassium levels were measured. Sweat rate was recorded in some of the conditions. Heart rate was recorded and samples of urine were collected thirty minutes befcre, forty minutes interval during, and thirty minutes after exposure. The period of exposure was 2 hours.

Experiment C

A group of subjects was exposed to one environmental condition 400 C, 25% Rh. Heart rate and packed cells volumes were recorded. The period of exposure was $1\frac{1}{2}$ hours.

Experiment D

This experiment consists of three parts:-

- i. A group of subjects was exposed to two environmental conditions 100 C, 50% Rh and *400* C, 25% Rh, for'a short-term exposure. In this part urine samples were collected at the following times:
	- a. pre-exposure sample
	- b. end-exposure sample
	- c. post-exposure sample.

Heart rate was recorded and sodium,potassium, chloride, urea and creatinine levels of urine were measured.

ii. A group of subjects was exposed to two environmental conditions 100 C, 50% Rh and *400* C, 25% Rh for a short-term exposure. In this part urine samples were collected at the following times:

a. early morning sample

b. pre-exposure sample

c. mid-exposure sample

d. end-exposure sample

e. post-exposure sample.

Red and white blood cell count, haemoglobin packed cells volumes, heart rate were recorded and sodium, potassium, chloride, urea, creatinine levels of urine were measured.

iii. A group of children was exposed to two environmental conditions 100 C, 50% Rh and *400* C, 25% Rh, for a short-term **exposure.**

urine samples were collected at the following times:

a. pre-exposure sample

b. end-exposure sample

c. post-exposure sample.

Heart rate was recorded and sodium, potassium. urea, creatinine levels of urine were measured.

The period of each exposure in both conditions was 2 hours.

Experiment E

A group of subjects was exposed to one long-term environmental condition 40^o c, 25% Rh and up to 24 hours. Sodium, potassium, chloride, urea, creatinine levels in urine were measured, in addition to the heart rate, blook pressure, surface skin temperature and packed cells volumes. Urine samples were collected at the beginning, then every four hours throughout, until four hours after the end of exposure.

Experiment F

A control experiment was conducted for resting and working conditions at room temperature. Sodium, potassium, chloride, urea, creatinine levels in urine were measured. The period of exposure was 2 hours.

The subjects reported to the laboratory wearing swimwearand were weighed and tested. The pre-exposure control sample was collected and heart rate was recorded. The subjects were asked to record the last time they emptied their bladders before coming for the experiment and then samples were collected as it was mentioned previously in the plan of each experiment.

In all experiments with the exception of Experiment B part ⁱ no work was done. The subjects were kept in the chamber for 1-24 hours depending on which experiment they were involved in. They were provided with a comfortable chair and nothing to do except what they wanted like reading and writing. In Experiment B part i subjects were set a light task, which· involved the threading of a brush. (inserting nylon fibres into a metal holder).

At the end of the exposure, the sUbjects were removed from the chamber, thoroughly dried and reweighed. In all experiments with the exception of Experiment A and E a post exposure recording and samples were collected in accordance to the plan given before

For those subjects involved in the experiments where two different environmental conditions were used, they were asked to have a normal day after the first exposure and before the second different exposure.

All experiments with the exception of Experiment B part i were performed with subjects who had fasted overnight. No liquid was consumed on the morning of an experiment. The subjects were well acquainted with the methodology and aims of the experiments, and informal consent was obtained from each.

4. INSTRUMENTATION AND PROCEDURE

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4.1 It is important for a modern clinical biochemist to apply and adapt automation in most of the clinical biochemistry tests, for the significance has been made in using such instruments in terms of accuracy, precision and analysis time. But the most important and vital part for a biochemist is to master handling these tests by simple basic concepts that are usually time consuming and elaborate procedures, which should be carried out with high efficiency.

4.2 Environmental Chamber

The chamber is a heat and sound insulated unit having a dimension of 4.72m x 2.92m x 3.10m (Figs.l & 2), and facilities for manipulating the environmental conditions like temperature and relative humidities. The range of selection is for temperature 10° C - 40°C \pm 1% C, and for relative humidity 20% - 95% \pm 2.5%. (Figs 384) The chamber has ~avities in the walls, ceiling and floor. Within these cavities are copper pipes that carry glycol solution (basically anti-freeze) which. can be cooled or heated for heating or cooling the surface of the inner walls, ceiling and floor. The air is recirculated by a variable speed fan, which displaces 1500 cubic feet per minute. This air can be heated or cooled and humidified or dehumidified depending on the requirements within the range given previously. fan speed can be controlled manually from zerc to full output. It draws the recirculating air from the room through a filter and The mixes a pre-determined amount of fresh air into the system. After passing through the fan the air can be humidified by steam injection. The steam being generated by two independently controlled immersion heaters. If dry air is required the dryer unit (silicair dryer

 ${\tt Fig.1~ -~ Environmental~ chamber.}~~({External~view}).$

Fig. 2 - Environmental chamber (Internal view).

Drier and humidifier (Environmental chamber). Fig. 3 \blacksquare

unit), which is composed of two silica gel banks; one of these in use will dry the air down to 20% Rh by extracting the moisture. Whilst one bank is being used the other is being dried out by passing heat through to the outside atmosphere. They automatically switch over as the one in use becomes saturated .

The liquid service unit comprises : four circulator type pumps one for each of the four surfaces. (Fig. 5) Each circuit is controlled separately by a motorized mixing valve. This valve with its sensors and electronic control system allows the mixing of hot or cold liquid into the recirculating liquid system and maintains close temperature control .

The interior walls are covered by thin aluminium tiles. The floor is made up of a system of wooden joists through which the pipework runs and is insulated from the concrete base on which it stands. R.h, temperature and amount of fresh air can be controlled mannually or automatically. (Fig. 6)

For operating instruction see Appendix. 10.3

Main automatic and manual controls (Environmental chamber).

4.3 The Spectrophotometer

Classical spectrophotometer is by far the most useful instrumental methods of measurement in the clinical laboratory, and it will doubtless remain so for a long time to come. Basically we are ma king use of the properties of atoms and molecules to absorb or emit electromagnetic energy in one of the many areas of the total electromagnetic spectrum .

The spectrophotometer is an instrument used in photo-electric colorimetry, in which the instruments used are not colorimeters but absorptiometers, since it is the amount of light absorbed which is measured .

The Unicam S.P. 600 spectrophotometer (Fig. 7). It has two vacuum photocells,a red for above 620nm and a blue for below this. For operation instructions see Appendix 10.3.

Essential parts:

- 1. A power supply to furnish appropriately regulated electrical energy for the operation of the instrument.
- 2. A source of radiant energy, usually containing a large group of wavelengths e.g. a tungsten lamp.
- 3. A monochromator, which is a device for selecting the required range of radiant energy from the system, e.g. a filter prism, or diffraction grating .
- 4. A sample container to hold the material being measured e.g. a cuyette.
- 5. A detector system to detect the transmitted radiant energy and convert it into electrical energy so that it may be measured e.g. a photocell.

Fig. $7 -$ Spectrophotometer (Unicam Sp 600)

6. A readout device to present the electrical current in a manner that allows it to be measured in useful units e.g. a meter reading transmittance or absorbance .

4 . 4 Flame photometer .

The average clinical laboratory places great reliance on flame photometry for the measurement of sodium and potassium. (Fig. 8).

Flame emission photometry is based on the principle that atoms of many metallic elements given sufficient energy in one form or another will emit this energy at particular wavelengths that are characteristic for the element. Energy in flame photometry is supplied as heat or thermal energy.

A particular amount or quantum of thermal energy is absorbed by an orbital electron, thus allowing it to occupy a higher, more energetic but less stable orbit. Being unstable, the electron returns almost instantaneously to its previous base or ground state and emits this quantum of absorbed energy in the form of a photon of a particular wavelength. In the average flame, only about one per cent of the atoms present undergo this transformation and emission of energy.

The very small proportion of atoms that emit coupled with the relatively low intensity of emission of most elements rather severely limits the biological applications of this technique: sodium and potassium are the only elements that have sufficiently high intensities to be measured with reasonable ease. Calcium has a fairly low intensity emission, but if very sensitive instruments are used, it can be measured reasonably well.

The flame photometer is used for measuring the concentrations of sodium and potassium in an unknown solution. These ions are estimated by reference to solutions of known concentration - the standard solutions. Suitable dilutions of standard solutions must be made in order to calibrate the instrument over the range of concentrations to be measured, the sensitivity of the method is: $Na - 2 p.p.m.; K - 3 p.p.m.$

For methods and operating instructions see Appendix 10.2 and 10.3

4 . 5 Autoanalyzer

The main device used for the determination of $Na⁺$, $K⁺$ Cl⁻, Urea and creatinine was a complete, serial, and continuous flow autoanalyzer labelled as Technicon S.M.A. (Sequential Multiple Analyzer) 6/60. (Fig. 9). The basic autoanalyzer consists of six modular units: 1. A sampler for picking up specimens to be analyzed. 2. A proportioning pump which maintains the flow of liquid in the system.

3. A dialyzer which produces a dialysate for analysis.

4. A heating bath for colour reaction development.

5. A spectrophotometer (colorimeter).

6. A recorder.

The S.M.A. 6/60 works on the principle of flame photometers. For methods and operating procedure see Appendix 10.2 and 10.3.

Fig. 9 - Technicon S.M.A. 6/60 (Autoanalyzer).

4.6 Measurement of Environmental Conditions.

Environmental condition measurements were made during each exposure to ensure the chamber maintained the required conditions. The fcllowing equipment was utilized :

a. The Dry and Wet Bulb Temperatures.

The whirling hygrometer (Fig. 10) was used to measure wet and dry bulb temperatures. The hygrometer was whirled several times until a stable temperature was achieved. From the difference of these readings, the percentage relative humidity was found from psychrometric tables. Appendix 10.5.

 (v')
b. The AGlobe Temperature.

The globe temperature is a measure of radiant temperature. The globe temperature was read from a globe thermometer which was correctly set up in an open area of the room (Fig. 10).

c. Air Movement.

This was measured by using a Kata thermometer of range 125-130° F. The time taken for the Kata thermometer to cool together with the Kata thermometer factor and the dry bulb temperature were used to estimate the air velocity from the nomogram. Appendix 10.5.

d. Light Laboratories

W. B. G.T. Index

Meter MIN₃ MK₅

It was a compact Min3lab (Fig. 11) which was used to give the **basic environmental mc:asurements mentioned in a , b ,** c .

For operating instructions see Appendix 10.3.

Fig.11 - $Min 3 lab$, with various probes.

4.7 Light Laboratories Thermometer

Apparatus used to record skin temperatures from subjects is shown in Fig. 12 . The dial scales are marked in divisions of 0.2 $^{\circ}$ C and can be read easily to 0.1° C, estimation to 0.05° C being possible. The black knob on the dial units, on rotation by hand, switch to the thermistor, which is indicated by the number in the vertical rectangle which lights up in the white base. The thermistors and the measurement units into which their leads plug are supplied in matched sets as the light electric thermometer. The thermistor used was calibrated and it showed that thermistor 6 required deduction of 1.9^oC over the range of its readings during the experiment. (Burry 1976)

4.8 In addition to the main instruments used in this research the following apparatus was used.

- a. Capillary haematocrit tube
- b. Special centrifuge for capillary tubes (Fig.13)
- c. Haematocrit reader (Fig.13)
- d. Microscope
- e. Red cell and white cell counting chamber
- f. Blood lancet
- g. Stethoscope and sphygmomanometer
- h. Electro-cardiograph (Fig. 14)
- i. Pipettes, flasks, beakers, tubes
- j. Hicroburette
- k. Scale for weighing persons upto 150 kg. (Herbert and Son)
- 1. Medical thermometer.

4 . 9 Sample Collection

Although instrumentation has a major role in terms of accuracy, precision and analysis time, sample collection plays an important

Fig. 12 - Electric thermometer (Light laboratories) with probes.

 $Fig.13$ -Haematocrit centrifuge and reader.

Fig. 14- Heart rate reader (ECG).

role in the final results, which are accurate, if handling the specimen, which is the first step in all clinical biochemical analyses. is correctly carried out.

1. Urine Samples.

Urine samples were collected from all subjects in accordance to the plan of each experiment. The collection of samples was timed because timed specimens are necessary to measure the rate of excretion of a substance. Urine samples were kept at -10°C overnight for analysis the following day. The frozen state was used for keeping . the urine samples because microbial growth is completely inhibited at this state. Concentrated HCl was used as a preservative for the urine samples. For the purpose of analysis portions of urine samples were diluted 1/100 and 1/200 for the use of the flame photometer whereas the same sample was diluted 1/10 and 1/20 for the use of the autoanalyzer. The addition of HCl negates the value of the chloride analysi in experiment E. For methods applied in measuring Na^+ , K^+ , Cl^- , urea, creatinine see Appendix 10.2.

2. Sweat .

Sweat was collected by attaching a pre-weighed ashless filter paper (Whatman 541) of known area to both subject's sub-scapulas. The area of the sub-scapula to be used was first cleaned with pieces of ashless filter paper moistened with de-ionised water. On removal, they were quickly transferred into a pre-weighed dish and re-weighed. Ten ml of de-ionised water was then added to disolve the paper into a fine pulp. The liquid from the resulting pulp was then filtered through a Gooch crucible and the filtrate was stored .

Blank determinations for sweat , sodium and potassium were done in the same manner as the test-sample. Blank readings were deducted for

each ion from the sweat values.

For methods of measuring sweat rate and ionic concentration of sweat see Appendix 10.2.

3. Blood Sample.

An extremely old technique for obtaining blood is the puncture wound of the capillaries and the finger puncture is the most used.

The fingertip is cleansed with a sterile gauze pad or sponge. moistened with 70% alcohol, leaving no moisture. The blood will form full rounded drops. By means of a loose wrist motion the finger is punctured with a sterile disposable lancet. The puncture is made by firmly grasping the finger and making a quick deliberate stab to a depth of about 3 mm into the finger. It is better to induce a good puncture rather than make the patient apprehensive by having to do a repeat. A deep puncture is no more painful than a superficial one.

The first drop of blood containing tissue juices and foreign matter,' is always. wiped off. The diluted and contaminated drop of blood would give unreliable results.

Free-flowing blood should be used for examination, although gentle pressure may be applied if needed to enhance the flow of blood. Never exert heavy pressure, because that may cause the flow of tissue juices in the blood.

After the required amount of blood has been taken, an antiseptic pad, 70% alcohol, is placed on the puncture. The patient may be instructed to apply pressure to the wound until the bleeding has ceased. (See Fig. 15 $)$)

For methods and for blood analysis see Appendix 10.2 .

4.10 Many measurements in clinical laboratories are facilitated by the use of instruments, some relatively simple, and others quite complicated. Upon the great request for rapid analysis and detection of ··early clinical abnormalities more and more instruments are being developed and automation in one form or another is increasing quickly. From here the problem emerges that a clinical biochemist should not depend totally on machines without complete understanding of the basics on which these machines were built.

Having this in mind when the plan for this research work was designed, it was purposely, made in such a way that all tests should be carried out by the most up to date, sophisticated instruments, when available in addition to the routine manual clinical biochemistry tests. The use of the fundamental manual procedures together with commonsense and practice helps the clinical biochemist to master and understand the functioning of the more highly complicated machines in his field of work and above *all* solving any problems arising from the use of instruments and the meaningful interpretation of the results.

Accuracy and precision.

Accuracy is the deviation of an estimate from the true value whereas precision is the exactness in the repeated determination of a test. A quality control includes more than tabulating the results from a series of standards and controls. Factors to be taken into consideration include methodology and instrumentation and proficiency

of personnel. Belk and Sunderman (1947); Wooten'and King (1953), reported the need of quality control in clinical diagnostic laboratories, due·to the results they obtained from surveys in which wide variation in blood analysis was obtained by different laboratories.

In assessing the allowable variation in results, factors such as diet, economic level, geographic location, social life and climate in addition to the methodology and instrumentation applied are of great importance in determining what is generally acceptable as normal values.

To ensure the reliability of the results obtained several points have been considered:

- **1.** Each calculation was checked twice.
- 2. Each instrument was recalibrated against known standards and its calibration curves rechecked before use with a known value against the originals.
- 3. Accuracy of the weighing scales in the climatic chamber was tested by a standard mass on each occasion.

The result showed that the scales used were not influenced by the heat and humidity applied during exposure. The makers confirmed that they would not expect any changes due to the range of conditions used in these experiments.

4. The result obtained by the flame photometer was calibrated periodically by using split

¥olume test samples through an autoanalyzer. The differences in readings for these samples were found to be insignificant.

- 5. Preliminary experience in blood pressure measurement was obtained by teaching films at Nottingham Medical School and by practice on human beings where the readings obtained were compared to the readings obtained by two clinically experienced people. However in the absence of automatic sphygmomanometer cuffs and arterial needle pressure gauges no better calibration of blood pressure was possible nor was found to be necessary.
- 6. Errors in packed cell volumes were reduced by using capillary tube haematocrits and reading with magnifying lens against a black grid of high contrast and good illumination.
- 7. Errors in blood cell counts were reduced by careful control of dilutant volume on both the white and red cell pipettes. In all cases this was ensured by allowing sufficient for'the technique and discarding any sampling in.which the volumes appeared to be in doubt or;where the capillarity of the pipettes was not sufficiently good to allow easy fiow of the blood sample. By practice it was found that consistent readings were obtained when time was taken to repeat a blood sample with a new pipette whenever there

was doubt of the measurements. The counting chamber samples were repeated often enough to gjve three consistent readings.

57

8. Urine samples were repeated twice on different ali/quots and when the measurements differed a further pair of alifquots were taken. This technique though time consuming gave high consistency in the measurements and whenever possible urine samples were set aside and reassessed next day.

, .

 $5.$ **RESULTS**

5.1 Statistics deal with techniques for collecting, analyzing, and drawing conclusions from data. However a proper statistical interpretation should take into consideration the variability present in all instruments and methods.

In order to obtain a meaningful interpretation of the data obtained, the mean and standard deviation were applied to all of the results obtained. T-test, the correlation coefficient (Computer programme g 202 Department of Human Sciences), and the Cusum Technique (Woodwood, Rh and Goldsmith, PP), were calculated and used where applicable.

It is worth mentioning that the pre-exposure sample represents the control values of parameters before the subject was exposed to the environmental condition under study.

5.2 Subjective Report.

As a part of the experiment where the subjects were exposed to two environmental conditions hot and cold, some of the subjects selected at random, were asked to express the extent to which they 'felt' the factors mentioned in Tables S₁ and S₂ and the overall impressions are dealt with in the 'discussions'.

Table S₁

Key to Table S_1 :

mild
strong
very strong \ddagger $+$ $+++$

0 nil
X Indifferent.

 $5.2.2.$

COLD

Table S_2

 $\ddot{}$ $++$

Key to Table S_2 :

mild
strong

 nil

 $\mathbf 0$

5.3 Experiment A. (10° above and 10° below ambient temperature at rest).

5.3.1 Urine, Na^+ , K^+ , Na^+/K^+ and creatinine values showed an increase during the two different exposures. (Fig. 16) (Tables 3,6). The values obtained for the t-test comparing the results of the two exposures showed a significant difference for Na⁺, K⁺ and Na⁺/K⁺ at 5% level (t \leq 1.9), whereas creatinine values showed insignificant difference $(t \leq 2.8)$.

5.3.2 The mean heart rate (beats/minute) showed at 100C above ambient temperature, that there was an initial increase followed by a decrease in the level. (Table 1) (Fig. 17). At 10ºC below ambient, however, the level went down throughout the experiment (Table 5) (Fig. 17). Statistically there was a significant difference between the values obtained in the two experiments, $(t \leq 1.9)$ for the 4 first readings, whereas the last three readings showed no significant difference (t \leq 4.09).

5.4 Experiment B

5.4.1.1 Urine Na+ and K+ increased initially but showed little decrease in values towards the end of exposure. (Fig. 18) (Tables 10, 17, 25). Urine Nat/K^+ increased initially during exposure I and II followed by a decrease, while during exposure HI it showed some fluctuation in value. (Fig. 18) (Tables 10,17',25)

Fig. 16 Exp. A₋₁₈₂ I 10⁰ above ambien Temp.[\mathbb{I} - > below +

Tab, 3 & 6

Fig. 17 Exp. A₋₁₈₂ I-18C above ambient Temp. $\left\{\frac{1}{160}\right\}$ below $\overline{\nu}$

 $Ta\underline{b}$

Tab. 10,17 & 25
correlation coefficient r showed relationship between Na^t and $K[†]$ values at the first 3 stages of experiment I, where $r = 0.802$, 0.728, 0.648. During the last two stages of the experiment $r \leq 0.39$.

During exposure II value of r for the correlation between Na^t and K^t was as follows:

 $r = 0.60$, 0.57, 0.72 and 0.61 while r value for the last stage of the experiment was $= 0.23$.

During exposure III $r = 0.71$, 0.52, 0.54 at the stages of pre-exposure, exposure and post exposure for Na⁺ and K⁺ respectively, while r for the two first samples during exposure was equal to 0.32, and 0.21.

5 .4 .1 .2 Chloride values followed the same trend in increasing during the first 80 minutes of the exposure and decreasing thereafter. (Tables 18,26).

 $-5.4.1.3$ Heart rates for subjects under experiment part I decreased throughout the exposure, whereas in part 11 the heart rates increased for the first 40 minutes and decreased thereafter during the exposure (Fig. 19) (Tables 9, 16, 24).

5.4.1.4 Eardrum temperature increased initially during experiments I and II and decreased in experiment Ill. Forehead and chest temperature decreased during the three exposures. (Fig. 19) (Tables 9, 16, 24).

5.4.1.5 Sweat Na⁺ and K+ decreased noticeably during the second half of the exposures. Sweat rate remained constant

during exposure I (Plot I Fig. 25) while it decreased during exposures II, II (Figs. 24, 25) (Tables 12, 19, 27). $Na⁺/K⁺$ ratic was increased during the three exposures (Fig. 25) (Tables 12, 19, 27). Comparing exposure I to II statistically, sweat Na^+ and K^+ were significantly different at 5% level $(t \leq 2.3)$. Sweat rate showed a significant difference during the first hour of exposure $(t \le 2.35)$.

Sweat Na^t and K^t values of exposures I and III indicated a significant difference between the two conditions (·at 5% level values of $t \leq 2.2$). There is a significant difference $(t \leq 1.5)$ in sweat Na⁺ and K⁺ and sweat rate between exposure II and Ill.

5.4.2.1 Urine Nat, K^+ , Nat/K⁺ ratio and creatinine increased . in the first forty minutes of exposure I (Fig. 20). Nat decreased after 40 minutes while K⁺ increased, and creatinine remained more or less constant (Fig. 20) (Tables 33, 35, 39, 41).

Cnloride showed the same trend as N a⁺ in increasing during the first 40 minutes followed by a decrease thereafter. (Tables 34, 40). Statistically there is a significant difference among all 5 variables between the two exposures (at 5% level $t \leq 1.9$).

5.4.2.2 Heart rate decreased throughout the experiment in both exposures. (Tables 32, 38) (Fig. 21). T-test, where $t \leq 1.7$, indicates a significant difference in heart rate between the two exposures.

5.4.2.3 Sweat rate and Nat and K^+ in sweat and Nat^+/K^+ values were higher during the humid conditions than those of the dry conditions. (Tables 36, 42). The difference was statistically significant at 5% level ($t \leq 1.84$).

Experiment B(iii) 30° C I 50% Rh 5.4.3 (at rest) II 35° C 38% Rh

5.4.3.1 During exposure I Na⁺ and Na⁺/K⁺ values increased initially while K⁺ value decreased and creatinine value showed little variability (Table 46) (Fig. 22). During exposure 11 Nat , K^+ , and Nat^+/K^+ increased initially while creatinine remained fairly constant. (Tables 52, 53) (Fig. 22). Chloride concentration showed little variability during either exposures (Tables 47,52). Statistically there was a significant difference at 5% level $(t \leq 1.89)$, between the two conditions.

5.4.3.2 Results of part I and 1I showed an increase in heart rate about 80 minutes after the beginning of exposure followed by a decrease in the values thereafter (Fig. 23) (Tables $45,51$).

5.4.3.3 Na^t and K^+ in sweat and sweat rate decreased in both conditions. Na^t/K^t value remained fairly constant during condition I but increased during condition II (Figs. 24,25) (Tables 49, 54). The values obtained during the two exposures were significantly different (t ≤ 1.80).

5.5 Experiment -C- (at rest) 400C, 25% Rh.

Heart rate increased 45 minutes after the beginning of the exposure followed by a decrease, (Table 57) (Fig. 26). Packed cells volume showed a little increase by the end of the exposure (Table 57).

 71

50%Rh $38 <$

Tab. 46, 48, 52 & 53

 $\overline{6}$

 $\overline{7}2$

Fig. 25 $Exp.B.$ (iii) I 25°C 67% Rh II 30 % 5_o \overline{z} III.35 = 38 $\overline{\mathbf{z}}$

5.6 Experiment D. (at rest)

5.6.1.1 Urine Na^+ , K⁺ and Na^+/ K^+ showed an increase by the end of exposure. (Fig. 27) (Tables 61,66). Creatinine (Fig. 27) and creatinine/Kg(bodyweight) were fairly constant in both parts (Tables 62, 67). Urea and chloride followed the same pattern in increasing during exposure (Tables 62, 67 and Tables 61, 66 respectively). Correlation coefficient r in experiment 1 indicates that there is a correlation between Na⁺ and K+ and Na⁺ and Cl⁻ and K⁺ and Cl⁻.

Exposure II

t value ≤ 1.34 indicates that there is a significant difference in values of creatinine between exposure 1 and 11 at 5% level. There is a significant difference at 5% level in K^+ and Nat/K^+ during exposure $t \le 2.20$ at 5% level.

5.6.1.2 Minute urine volume showed little increase during exposure 1 and a noticeable increase in exposure 11 (Fig. 28) (Tables 60,65).

5.6.1.3 Heart rate increased noticeably in exposure I (Fig. 28) followed by a decrease at the end of the experiment. (Table 60) \backslash In part 11 heart rate decreased throughout the experiment. (Table 65) (Fig. 28).

5.6.2.1 Urine sodium decreased during exposure I while K+ and $Na⁺/K⁺$ increased and creatinine remained fairly constant (Fig. 29) (Table' 72). Chloride velues increased initially followed by a decrease at mid-exposure. (Table 42). Urea values remained fairly constant (Table 71). Creatinine $\frac{1}{5}$ (body weight) remained constant (Table 73).

For exposure I

Urine Nat , K^+ and Nat/K^+ increased in value in part II during the exposure (Fig. 31) (Table 79). Creatinine values were fairly constant (Fig. 29) (Table 80). Urea and Chloride values increased during exposure (Tables 78, 79). Creatinine/Kg (body weight) was fairly constant (Table 80). The values of r .

(correlation coefficient)for experiment 11 'are as follows:

There was a significant difference in Na⁺, K⁺, Na⁺/K⁺ and creatinine between the two exposures at 5% level where $t \leq 2.2$.

5.6.2.2 Minute urine volume showed a decrease toward the end of exposure I while it increased during exposure 11. (Fig. 30) (Table 71, 78).

5.6.2.3 Heart rate increased 60 minutes after the beginning of exposure I followed by a decrease thereafter. In part 11, however, there was a continuous decrease in heart rate values. (Fig. 30) (Tables 70, 77).

".5.6.2.4 Blood analysis in part I showed fairly constant haemoglobin values, a decrease in P.C.V., a decrease in red blood count, and an increase in white blood count. (Table 74).

In part 11, blood analysis showed a little increase in haemoglobin, an increase in P.C.V., an increase in red blood cell count and an increase in white blood count. (Table 81).

5.6.3.1 Urine Na⁺, K⁺ and Na⁺/K⁺ decreased during exposure I, while creatinine remained constant. (Fig. 31) (Tables 87,88).

Urea and creatinine/body weight decreased in value during exposure I (Table 88). r for $\text{Nat-}K^+$ at stage three of experiment $I = -0.619.$

During exposure II urine Na^t and Na^t/K^t increased while K^t and creatinine decreased. (Fig. 31) (Tables 87.88). Urea and creatinine/Kg bodyweight)decreased in value during exposure II (Table 88). Correlation coefficient r for exposure I1 is equal to -0.60 for Na^t-K⁺ at stage one of the experiment; $r = -0.59$ at stage two of the experiment.

Significant difference in Na⁺, K^+ , Na⁺/K+ and creatinine values indicated by the value of t obtained $t \leq 2.0$ at 5% level.

5.6.3.2 Minute urine volume was constant during exposure I (Fig. 32) (Table 86), while it increased during exposure II (Fig. 32) (Table 86).

5.6.3.3 Heart rate increased during experiment I and decreased \cdot during experiment II. (Fig. 32) (Table 85).

There was a significant difference in heart rate between the two exposures indicated by value of $t \le 2.3$ at 5% level.

5.7

Experiment E (at rest) 40° C 25% Rh

24 hours exposure.

5.7.1 Urine Na^t and creatinine decreased in value for the first 8 hours of exposure (Fig. 33), while K⁺ continued to decrease in value and Nat/K^+ continued to increase for the first 18 hours and then decreased after. (Fig. 33) (Tables 100, 101). Addition of HCl negates the values obtained for chloride which

initially decreased, but showed an increase' towards the end of the exposure (Table 101). Urea values were fluctuating during the exposure (Table 102), while creatinine/Kg (body weight) showed a decrease with a narrow range of fluctuation during' the exposure. (Table 103).

Correlation coefficient r shows the following values for r:

Application of the cusum techniques for Nat . $K⁺$ and creatinine values showed that the main change in the value of the parameters occured during the early hours of the exposure. (Figs. 34. 35, 36, 37. 38, 39, 40, *41, 42).*

5.7.2 Minute urine volume showed a little decrease in value during exposure (Fig. 43) (Table 92).

5.7.3 Heart rate during experiment E showed some fluctuation by increasing at the early stage of the experiment followed by a decrease and an increase in the value tcwards the end of the experiment. (Fig. 43) (Table 98). This same pattern was followed by blood pressure. (Table 98).

5.7.4 P.C.V. decreased at the early stages of the experiment, I followed by a fairly constant level towards the end of the exposure (Table 99).

 $67'$

89 '

5.7.4 Skin temperature showed a decrease in value after· it increased initially in the early hours of the exposure. (Fig. 44) (Tables 94, 95). Oral temperature was. fluctuating with a slight decrease in value towards the end of the experiment (Fig. 44) (Table 96).

 $98.$

5.8 Experiment -F- Ambient temperature and humidity.

> 5.8.1 This control experiment showed a slight variability in Na^t. K^t. Na^t/K^t.Cl⁻, urea, creatinine and creatinine/body weight)values obtained from resting subjects I and subject set a light task 11 at ambient temperature. (Fig. 45) (Tables 106, 107).

5.9 It is worth mentioning that in dealing with the results the interpretation was founded on the mean values obtained from each experiment. It was noticed that a significant standard deviation was obtained in most of the experiments and this is mainly due to the different reactions of the subjects to such experiments. Unfortunately the subjects taking part can never be considered as a random sample, because it depends on the willingness of the subjects to participate. To overcome diurnal variation, subjects who had to attend two exposures, came at the same time of the day to take part in the experiment in both conditions. In the statistical interpretation t values were taken at the 5% level while r for correlation coefficient was considered for those values \mathbb{Z} + 0.5 (Snedecor and Cochran 1957).

Tab. 94,95 & 96

.
Fig. 45 Exp. F Ambient Temp At Rest $\overline{1}$ II At Work

Tab. 106&107

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 $\overline{5}$

 \overline{L}
DISCUSSION $6.$

 $\hat{\mathcal{A}}_1$

6.1 Introduction

The aim of this investigation was to study the physiological response of human beings in their adaptation to different environmental conditions created in a climatic chamber.

The experiments were designed and applied in such a way to give a clear idea about the variability of the psychological responses in addition to the biochemical and physiological responses to environmental stress.

In an attempt to reach a better understanding of the results obtained, the discussion is divided into:

- a Subjective report
- b General discussion of the biochemical and physiological responses

c - General comments

d The similarities as a result of the various. analyses obtained, between processes of physiological adaptation operating and these clinically observed changes due to known clinical disorders.

e - Accuracy and precision.

6.2 Subjective report

, "

The subjective report reflects the direct psychological and physiological responses of the people involved (5.2). This is of great importance because it will help to understand the tolerance of human beings to similar conditions.

:

In general there was a fear of the so called "Hot chamber" which was reflected on the raised pulse rate before entering the chamber among subjects (Tables 2, 9, *16,* 24, 32, 38, 45, *51,* 57, 60, 70, 85, 97). During exposure all subjects found it very difficult to concentrate on whatever they were planning to do e.g. academic work, playing games, or writing letters, with a general atmosphere of quietness, sleepiness (observed perfectly with children who slept during exposure) and the main question which was frequently asked by all subjects "what is the time now?". This discomfort in the hot chamber was mainly due to the dehydration observed,by a feeling of dizziness by some subjects, and the decrease in minute urine volume (Figs. 28, 32, 43), and salt loss observed in sweat and urine $(5.3.1, 5.4.1.1, 5.4.1.2,)$ *5.4.1.5, 5.4.2.1,* 5.4.2.3, 5.4.3.1, 5.4.3.3, 5.6.1.1, *5.6.1.2, 5.6.2.1,* 5.6.2.2, 5.6.3.1, 5.6.3.2, *5.7.1,* 5.7.2, 5.7.3).

No experimental data to support the feelings of apprehension was obtained, because it was not practicable to achieve or observe at the same time while conducting the biochemical and physiological tests , .. under study.

Adaptation was observed by the comfort some of the subjects started to feel towards the end of exposure by the false feelings these subjects gathered about the drop in temperature and humidity of the chamber.

During cold exposure there was a fear of the cold, not because the temperature was low, but because of the idea that the subjects had to stay in such a cold environment wearing only swimwear. This was sthown by the increase in heart rate during the pre-exposure stage (Tables 5, 65, 77, 85).

Heat conservation can be achieved during cold exposure by cutaneous vasoconstriction and by a layer of subcutaneous fat which provides excellent insulation. During the cold exposure the subjects .sat 'folded up' with bent knees in an attempt to decrease the exposed surface area of the body to conserve heat. Their pale bodies were an indication of cutaneous vasoconstriction in an attempt to reduce the amount of heat transferred by the blood from the body core to the body surface.

'Pins and needles' feelings in the subjects' legs and particularly the feet were due to cold, vasodilation occurs on exposure of these parts to moderate cold 5-10°C when the body is generally warm.

No recognized psychological tests were carried out for various reasons such as the many numbers of the other parameters to be studied, the inability of stopping the interference of the technician for technical reasons during·the exposure, the fact that on certain occasions more than one subject had to take part at the same.time and the unwillingness of SOme subjects to get involved in such tests.

6.3 General discussion

"The organism is only a living machine constructed in such a way that on the one hand there is full communication between the external environment and the milieu interieur . . ." (Claude Bernard 1865).

Thus complex physiological processes involving the different vital organs of the human body work cooperatively to maintain the homeostasis of the internal systems.

To simplify dealing with the data the discussion will deal with the hot and cold environmental conditions separately.

6.3.1 Hot environment

The general trend during short-term exposure was an initial rise *in* urinary sodium and chloride levels which declined *in* such a way that the more severe the heat stress the lower the level (5.3.1, 5.4.1, 5.4.12, 5.4.2.1, 5.4.3.1, 5.6.1.1, 5.6.2.1, 5.6.3.1,5.7.1). This was *in* agreement with the literature (Kanter, 1955; Itoh, 1960). The delay in the decline of the levels of these parameters (Tables 39, 46, 52) indicated that the Subjects took longer to acclimatize. The reason behind the initial rise in the levels of Na⁺, Cl⁻ was that the subjects were not acclimatized to the environmental conditions during the first period of the exposure. Such delays in the conservative acclimatization response to the environmental changes was attributed to age, sex, physical fitness, race and habitat, all of which played an important role in defining the limits at which such adaptive mechanisms were triggered, and which vary in different subjects.

There was a decline in urinary minute volume upon exposure to heat (5.6.1.2, 5.6.2.2, 5.6.3.2, 5.7.2) (Figs. 28, 3D, 32 and 43), due to water conservation mechanisms which were stimulated and water was reabsorbed at the level of the distal tubule via the action of anti-diuretic hormone. A similar result was obtained by Itoh, 1960; Collin and Weiner, 1968.

Salt and water balance are affected greatly in conditions of heat stress, which might lead to distinctive changes in the distribution of the electrolytes between the cells and the extracellular fluid, changes in osmotic pressure, acid-base balance, kidney function, and water balance. On the other hand The human body possesses remarkable adaptability in such a way that the physiological mechanisms involved are so flexible that they operate efficiently within a wide range of environmental changes in order to counteract the effects of serious stress. The mechanisms causing these physiological responses to conserve water and salt could be suggested as follows:

Heat Stress $\sum_{i=1}^n$ HYPOTHALAMUS

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\adrenocorticotrophic hormone releasing factor

ANTERIOR PITUITARY $a.c.t.h.$

ADRENALCORTEX ~ldosterone

KIDNEY TUBULES

 Λ increase Na⁺ retention POSTERIOR PITUITARY ~a.d.h. (antidiuretic hormone) KIDNEY TUBULES

 $\sum_{\texttt{High plasma sodium}}$

although the mechanism has been suggested for Na⁺ reabsorption, sodium cannot be reabsorbed without accompanying cat-ion, Cl⁻, and water (Wright, 1971).

Many workers have suggested that salt deficit increases aldosterone output (Collins and Weiner, 1968; Collins, Hellman, Jones and Lunnon, 1955). This is supported by evidence that aldosterone contributes to the renal conservation of Na+ and Cl⁻ and that aldosterone secretion increases during heat. exposure (Collin and Weiner, 196B; Itoh, 1960, Hellman et al., 1956).

Potassium exhibited a similar trend to Na⁺ which is attributed to a certain adjustment probably occuring to maintain the sodium/potassium ratio (Figs. 16, IB, 20, 22, 27, 29, 31, 33). Aldosterone in retaining Na+ results in an effective secretion of potassium (Bass et al., 1955), however this contradicts the level of K+ obtained in this research which might be attributed to the stimulation of growth hormone through stress and excitement, . and this caused retention of potassium.

In the present studies urine Na^+/ K^+ decreased during heat exposures (Figs. 16, 18, 20, 22, 27, 29, 31, 33) suggesting that aldosterone was secreted and indicating acclimatization to hot ." conditions. This confirms the suggestion in the literature that aldosterone lowers Na+/K+ during heat acclimatization (Macfarlane, 1956; Robinson et al., Collins, Hellman, Jones and Lunnon, 1955).

Creatinine acts as a good reliable indicator of renal function and the relatively constant levels obtained (Figs. 16, 20, 22, 27, 29, 31, 33) in this study reflect the neutralizing effect produced by reduced glomerular filtration rate by the kidney.This due tothefact that there was insufficient stress to cause interference with kidney function or to increase creatinine production by the muscles on exposure to heat stress (Streeton et al., 1960).

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The elevated urea values would be due to excessive protein broken down as a result of stress. $(5.6.1, 5.6.2.1,$ 5.6.3.1, 5.7.1).

.. , Results obtained in this study indicated that sweat rate, and Na^+ , K^+ levels in sweat declined during the second hour of exposure $(5.4.3.2)$ and (Figs. $24,25$). This was similar to the findings of Peter and Wyndham, 1966; Wyndham, 1954; Bass et al., 1955; Allan and Wilson, 1971. The sweat rate and Na⁺ K⁺ values are higher in exposures where work was achieved (Figs. 24, 25). The decrease was attributed to sweat gland fatigue (Peter and Wyndham, 1966). The results obtained (5.4.1.5, 5.4.2.3, 5.4.3.3). confirmed the indication of conservation mechanisms operating to prevent salt depletion. This is an adaptive mechanism to reduce salt loss which has been confirmed by several workers (Moss, 1923; Adolf and Dill, 1938; Robinson et al., 1941; Wyndham, 1967; Ladell, 1957; Eichna et al., 1954 and Dill et al., 1933). It is likely that?renin-angiotensin mechanism of mineral corticoid is involved when salt losses are large causing a low Na⁺ level thus enhancing aldosterone secretion in men exposed to heat (Brown et al., 1964). It was suggested that the decrease in sweat sodium concentration was due to the secretion of aldosterone (Kenney, 1963; Collins and Weiner, 1968).

Thus the kidney and the sweat gland are capable of making remarkable adaptive changes in their salt output to changes in the salt balance of the body (Figs. 16, 18, 20, 22, 24, 25, 27, 29, 31, 33). In response to gradual salt depletion by sweating, the kidney may quickly begin to reduce salt output and complete

lOT

adaptation rapidly while the sweat glands reduce the salt concentration of sweat more slowly. These conservational responses are brought about by the increased adrenal corticol activity, in which kidney and sweat glands reduce salt output in response to salt deficiency in the blood.

Changes in blood and circulation during exposure to heating is another physiological adaptive mechanism. to maintain homeothermy. The reduction in packed cells volume indicated an increase in plasma volume. This is also reflected with the haemoglobin values and red blood cells count (5.6.2.3). The results showed that a fundamental change has taken place between the two components of blood i.e. cells and plasma. Eitherthe cells decreased in number. a hypothesis which is not tenable because one must postulate where the cells could go, or the plasma volume has increased, which is a more probable answer for the results obtained. The findings in this study indicated a change in the blood composition towards haemodilution. An increase in white blood count could be attributed to the stimulation of the mobilization of body defenses against elevation in temperature (Table 74) and may be a part of the same mechanism. by which most conditions of stress cause an increase in white cell counts.

Heart rate increased initially on exposure to the chamber and then decreased throughout the exposure periods. (Figs. 17, 19, 21. 23. 26, 28, 30, 32, 43). The initial increase could be, attributed to a) the initial fear among the subjects of the experiment, b) an adrenalin response to the stress condition

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causing vasodilation, reduction of peripheral arterial resistance and increase in blood flow to the skin.

Increase in heart rate was due to a general rise in temperature since impulses are under the same influence of agents that raise body temperature. When a general rise in $\overline{}$ the temperature of the body occurs the rate of passage of the cardiac impulses along the conductive system are increased which increases the heart rate. The observed decrease in the heart rates to a relatively constant level during the exposure, indicates an improvement in the cardiovascular responses to heat stress and is in agreement with many workers (Eichna et al., 1945; Bass et al., 1955; Wyndham et al., 1964).

The cutaneous vasodilation decreases the peripheral resistance and lowers the diastolic pressure, thus lowering blood pressure (Table 98) and heart rate (Figs. 17, 19, 21, 23, 26, 28, 30, 32, 43). All of these cardiovascular adjustments . 'were to help the body to meet the stressful conditions with the least side effects throughout the acclimation process. These cardiovascular adjustments were also confirmed in the literature. (Scott, Bagett, and Mackie, 1939; Bean and Eichna, 1943; Taylor, Henshel and Key, 1943).

The deep body temperature is maintained at an approximately constant level by the balance between heat gained and heat lost by the body (Table 96 and Nielson; 1938; Webb and Annis, 1967; 1968; Xstrand, 1952; Wyndham et al., 1952a, b; 1954; Robinson, 1949), (Fig. 44).

The skin is the one physiologicaL factor which responds immediately to all environments, and therefore is a useful indicator of the effects of thermal environment on man's ability to perform in and tolerate the changes in environmental conditions. The results obtained (Figs. 19, 44) (5.4.1.4, 5.7.4), indicate· initial increase followed by a decrease caused by cooling effect due to evaporation. Thus *in* dry heat the evaporation of sweat maintained the stabilized level of skin temperature despite an environmental temperature which was higher. The environment and core temperature interact at the skin in such a way as to maintain homeostasis of the central body temperature. Temperature regulation seems to.be governed by thermoresponsiveness in the anterior hypothalamus which controls the heat loss by means of adaptive mechanisms such as vasodilation of the skin and the sweating mechanisms. In the passive phase this is by simple heat loss from the skin to the cooler environment. In the active phase this is by sweat mechanisms requiring the hypothalamus to operate as a mineralcorticoid regulator over skin and kidney in the control of sweat and urine electrolytes.

6.3.2 Cold Environment

It is very interesting to see that the trend in Na^+ , K^+ and Cl⁻ values tends to rise in accordance with urine minute volume as would be expected as a result of cold induced diuresis. (5.3.1, 5.6.1.1, 5.6.2.1, 5.6.3). This confirmed the fact that cold induced diuresis suppressed aldosterone secretion and thus sodium levels should be increased throughout the exposure. This appears to contradict the observations obtained on certain

individuals in this study (Tables 66 , 79 , 87). The answer for this contradiction can be explained by the fact that growth hormone which was stimulated by the cold exposure enhanced retention of Na⁺, K⁺ and Cl⁻. The idea of endocrinological adjustment is supported by Davis et al., 1955, 1960.

The initial slight increase in the urinary creatinine (Figs. 16, 27, 29) is due to an increase in muscular activity by shivering to increase body temperature. This finding of the fact that shivering and its consequent thermogenesis, would increase with decreasing ambient temperature has been reported by many workers (Seift, 1932; Adolf and Molnar, 1946: Hemingway, 1953; Horvath, Spurr, Hutt, and Hamilton, 1956; Iampietro, Vaughan, Goldman, Kreider, Masucci and David, 1960).

Increased metabolism may also be stimulated by the production of thyroxine (Goldstein - Golaire: et al., 1970), in order to increase heat production during acclimatization process (Poe and Davis, 1962). Adrenalin may also be increased which will increase heat production by increasing muscular activity, together with an increase in noradrenalin level. This has been reported by Smith, 1962.

The initial increased values of urea could be attributed to protein broken down as a result of stress (5.6.1.1, 5.6.2.1).

Blood analyses showed that P.C.V. increased, haemoglobin remained almost constant and red blood cell count increased (Table 81). All of these values suggest an effective decrease in plasma volume. This decrease in volume would be the result of

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diuresis as well as shift in plasma volume. The increase in the white cell count suggests a general defense mechanism against stressful conditions (Table *81).*

Application of cold to the body surface is followed by peripheral ·.venous constriction and decreased peripheral blood flow. Heat loss is reduced by cutaneous vascoconstriction. This helps by decreasing the amount of heat transferred by the blood from the internal body core tc the body surface. The cutaneous vasoconstriction will raise blood pressure during exposure to cold (Samson Wright 1961; Wakin et al., 1952). The results showed heart rate was decreased (Figs. *17,* 28, 30, 32) due to the peripheral vasoconstriction reducing the blood flow to skin and thus reducing the need fer the heart pumping action. Increased secretion of adrenalin and noradrenalin may also occur during exposure to cold and would contribute further to the rise in blood pressure, and would mask the decrease in pulse rate and thus will account for the different heart rate levels observed in some individuals. These findings of heart rate changes during cold exposure are similar to what is reported in the literature. (Murphy, 1959; Hookard, Stormont, 1941; Prec, Rosenman, Brawn, Rodbard and Katz. *1949;* Sabiston, Theilen and Gregg. *1955).*

6.4 General Comment

The physiological processes which occurred during the two exposures of heat and cold indicate on the one hand the processes involved and on the other the ability of these processes to regulate and to express their adaptive mechanisms in maintaining body constancy according to environmental needs. Thus the physiological responses to heat are:

a - Temperature increase

- b Increase in cellular metabolic exchange which means an increase in the rate of general metabolism
- c Vasodilation
- d A general change in blood flow.

The physiological responses to cold are:

- a Physical regulation mainly to reduce the heat loss from the body surface (a conservation of body heat).
- b Metabolic regulation, in which the endocrine and autonomic system are involved to alter heat production in the body.

Acclimatization is a necessary factor in man's ability to withstand environmental strain, and to maintain body homeostasis. Among those regulatory mechanisms involved is the need to maintain relative constancy of the deep body temperature upon which normal metabolic function depends. This relatively constant body temperature is maintained by self-activated reactions keeping an equilibrium between heat production and heat loss; this operates as the body's thermostat.

The above example is one of many concerned with regulating mechanisms which act to maintain the constancy of the internal environment. The physiological response to any change in environmental conditions may produce changes in total heat production, skin temperature, heart rate, and sweat secretion. These involve the endocrine system in its role in the physiological adjustments of the homeostatic state of the body.

Adaptation to hot and cold environmental conditions might be attributed to three factors: (i) genetic, (ii) physiological and (iii) cultural.

Genetically determined variations are transmitted in the genotype. These genetic features are expressed in the general morphology of the human body within the limitations of its sex. All of the factors must be considered advantageous as they have appeared by natural selection in a specific type of environment.

The physiological features allow the flexibility by which the body can adapt remarkably rapidly to ambient changes in the thermal environment.

The cultural features are concerned with behaviour and have been evolved to allow survival more successfully in the extremes of environmental conditions where neither the genetic nor physiological features of the body have evolved. The development of such behavioural characteristics as clothing, housing and formalized habits of life have allowed human migration to new environments with conditions that are frequently incompatible with life. Obviously an important part of the cultural features is the ingenuity and intelligence of man.

Results obtained in this study indicate an incomplete adaptability to rapid changes in the thermal environment. Acclimation is a slow process and when incomplete can be detected by physiological responses to changes in environmental conditions. These tend to show an initial rise in most physiological parameters followed by a decline to approximately normal pre-exposure levels. In some occasions the continuing exposure may result in a continuing small change in the physiological parameter and frequently indicates that the body has established a new level of acclimation. A remarkable feature of the results was the variation among subjects as each adapted to changes in the environmental condition. It seems that acclimation or complete adaptation cannot be achieved until after prolonged exposure or 'repeated exposures to the new environmental conditions. Perhaps this is a similar process to immunization and acclimation, like immunization, requires sufficient time and exposure for the various adaptive processes of the body to reorganize and acclimatize to the new conditions. In both cases physiological parameters show ,'abnormal values during the transition period and may be incomplete if the exposure is extreme or too short. However it also seems that acclimation is more similar to 'temporary' immunity since it readjusts quite soon when the body is withdrawn from the conditions which caused it. This is seen in most forms of environmental acclimation and the time to readjust varies with the particular condition studied. In man thermal acclimation is rapid and within the experimental conditions of this study, it may well be only a few hours but in more extreme changes such as a trip to the Sahara from winter time England it may take a few days. However in both cases the initial adaptive responses appear to begin in only 45 minutes!!

This suggests that one of the physiological.parameters might be used as an index of acclimatization. Kerslake (1972) has said "A good Index is a map, not an aerial photograph, and its scale and content should be chosen in relation to its application. Its precision should match that of the data with which it will be used". Under the conditions of this study, creatinine is a reliable index of heat stress due to the fact that the creatinine levels reflect these processes best.

However a 'Comfort index' would be most valuable in this type of research. Some observations during the adaptation phases reflected decline in the parameters that continued below normal pre-exposure levels. Also several aspects of 'comfort' were mentioned by the subjects during exposure. Obviously a personality index which includes behaviour, might give some insight to this and allow some fcrm of correlation between exposure, sex, age and personal characteristics. However personality alone is insufficient as we are concerned with the feeling of 'well-being' or comfort in the new environment. \mathcal{A}^{\dagger} This might then be related to the stages in adaptation and differentiate ,between claustrophobic effects and other forms of stress experienced during the exposure period. Frequently in people who complained of claustrophobia there was a decline in these complaints towards the end of the test period and this was more rapid when the victims were able to know and see the time left for the test. This is only one aspect of comfort and was quite variable between subjects. Few people made no comment or appeared to be unaffected by the experiments; most people expressed an opinion best described as a description of comfort or its lack. The components of a comfort index are too varied to quantify in this study but a table of 'comments' is given in the

results $(5, 2.1, 5.2.2)$. Certainly the biggest part of a comfort index must relate to psychophysiological factors. These must include irritability, fatigue, the ability to concentrate, and social effects such as cooperation. Some of these factors may be measured' by work productivity and quality control tests as used in industry and commerce by setting suitable work tasks as a part of the exposure period. Unfortunately no such tests were included in this study as the experimental chamber was poorly designed for this, purpose. In an attempt to conduct a pilot experiment of this in the task of assembling brushes, the results indicate that exposure to 100C above or below acclimation causes a decrease in work rate, but the tests were too few to give significant results.

The need for a heat index has been discussed earlier, this study highlights the need also for a cold index. As in the hot exposure urinary creatinine was also found to reflect the effects of adaptation to cold. A good cold index should be one which includes a subjective thermal sensation scale as a direct measure of the state of comfort or discomfort eg.

It is interesting to notice that the 50C or scale 1 conditions are identical in effect with normal diurnal changes.

Such an index should include in addition the speed of·air movement. I suggest that a mathematical approach for such a cold index should involve a correlation between creatinine level. mean skin temperature and dry bulb temperature.

6.5 Clinical diagnosis

It is very striking that certain similarities exist between the processes of physiological adaptation and clinically observed changes due to known clinical disorders.

The immediate clinical consequences of water and sodium disturbance are changes in extracellular osmolality. If sodium balance is disturbed without a corresponding change in water. a change in plasma osmolality will occur. This change in the subjects in this study was coincident with reported feelings of headache and minor disorientation in the response to the request for comments about "comfort". Headache, and confusion are also common symptoms in the early stages of two clinical conditions caused by sodium and water imbalance these are: hyponatraemia, causing cellular overhydration and hypernatraemia causing cellular dehydration of cerebral cells leads to the symptoms of headache and confusion which in worse stages can lead to coma.

Aldosterone production increases due to low plasma sodium levels. This is an attempt to conserve plasma sodium. The clinical effects of shock. heart failure and liver cirrhosis are due to a decrease in sodium excretion which lead to an increase in aldosterone production. Clinically a high level of urinary sodium is associated with dehydration as can be seen in addison disease, and the digestive disturbance of vomitting and diarrhea.

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Decreased values of urinary potassium are frequently associated clinically with Addison's disease, acute infections, pneumonia, uremia and acute bronchial asthma. Increased values of potassium are associated with severe diarrhea, diabetes and malignant growth. It is probable that the subjects in these studies were not exposed to potassium variation for sufficient time for these symptoms to occur at an observable level except for respiratory discomfort and slight tendencies to cough and to clear the throat coincidental with reading of low urinary potassium. However this was more easily associated with dry atmosphere conditions rather than potassium depletion.

Decreased values of Chloride are associated with prostatic obstruction. nephritis. cardiac conditions, hyperventilation. and increased values of chloride are associated with Addison's disease. fevers, heat cramps, diarrhea, vomitting and profuse sweating. Certainly in this study the symptoms of hyperventilation and more than predictable sweating occurred in the two changes in urinary chloride. However the other clinical symptoms, particularly heat cramps were not observed in these experiments due to the short periods of exposure (even after 24 hours). The non-electrolyte observations must be considered in the same way. The variations observed while significant were always within the durations of the experiments/recovered to pre-test levels rapidly. The clinical conditions associated with diagnostic observations are invariably associated with long duration unrelieved changes in the materials measured and thus have a clinical significance.

Although changes in blood composition occurred, the range of changes observed were still within the normal range for healthy people

given in Documenta $\left|$ Geigy. The most striking feature: was the quick response of the white cells, which provide defense mechanisms for the body. The total white cell count increased in response to the initial rise in body temperature in'adaptation to the environmental changes. This rise gradually decreased with continued exposure but remained elevated about the pre-test level until the return to pre-test environmental conditions. Unfortunately it was not possible to persuade these subjects to give sufficient blood samples to study this in greater detail. It may be that this white cell response can be related more closely to exposure as the increased count appears to be transient with the exposure. Reference to the results in hot and cold shows that there was a more marked change in cold exposure than in hot. Further use of these results would require differential counts to determine which leucocytes are most responsive to the environmental changes and to see to what extent this is similar to such clinical cases as shock or whether this is a simple nondescript stress response.

The rise in the temperature which results in vasodilation is initiated by the body temperature regulating centres. These act like a thermostat. This rise in temperature is similar to the situation with bacterial and viral infections in which profound vascoconstriction occurs followed by shiverings. Following this the, temperature rises to a new level which is the state of fever. Once the new temperature reaches the skin, vessels will dilate and the human being will appear flushed. When the infection is terminated the body temperature returns to normal causing a marked sweating to enable heat to be lost quickly.

An important feature which is worth mentioning because of its clinical importance is that in the hot exposure (40°C) after the the
initial sweat period,{body retained water especially during the twenty four hours exposure. Retaining this fluid resulted in the observed constant level of packed cells volume. This retention of water caused haemodilution of the blood. When this diluted blood perfused the cranral circulation, diffusion of water into cerebral fluid occurred causing cerebral oedema, resulting in the characteristic symptoms of headache and mental confusion/disorientation.

While the purposes of this study have been to observe normal responses to laboratory experiments in normal people, the results can be seen from the foregoing discussion to have similar causes to many clinical symptoms. Perhaps the major mechanisms involved are directly related in the transient conditions of these experiments and the unrelieved continued conditions in clinical disorders. Thus it may be possible to predict physiological abnormalities which may occur to normal people over-exposed to environmental conditions to which they cannot acclimate and suggest that an inability to acclimate has a direct parallel with clinical malfunction and may need similar therapeutic treatment for recovery.

7. CONCLUSION

7. The effects of heat and cold on the physioldgical systems has been shown in this study. As seen in the results the physiological processes involved counteract the effects of stress due to environmental changes through adaptation to keep the body homeostasis.

1. Incomplete adaptation was observed during the short~term exposures. Freaturesof adaptation observed were:

- I Hot
	- *(i)* Conservation of salt indicated by reduction of urinary Na^t and Cl⁻ concentration. Similar reduction of urinary K^t was achieved. Reduction in sweat Nat and K^+ was observed.
	- (ii) Decrease in urinary minute volume.
	- (iii) Decrease in $\text{Na}^{+}/\text{K}^{+}$ ratio.
		- (iv) Gradual decrease in heart rate.
		- tv) Decrease in sweat rate during exposure.
		- (vi) Decrease in skin temperature.
	- (vii) Decrease in P,C,V , and red blood cell count together with an increase in white blood cell count.
- II Cold
	- (i) Initial increase in Na^+ , K^+ and Cl⁻ levels due to cold induced diuresis followed by a decrease due to conservative mechanisms operating.
	- (ii) Increase in urinary minute volume.
	- (iii) Decrease in heart rate.
	- (iv) Increase in haemoglobin, P.C.V., red blood cell and white blood cell counts.

2. Na⁺, K^+ , Na⁺/ K^+ were a direct reflection of the environmental conditions. The higher the temperature and humidity the higher the values of these parameters.

3. Sweat Na^t, K^t and sweat rate were higher at high temperature and humidity. The values of these parameters were higher with subjects at work than those at rest.

4. Heart rate levels increased with temperature. Subjects at work showed higher heart rates than those at rest.

5. 24 hours exposure showed fluctuation similar to diurnal variation,, with more retention of Nat, K^+ , Cl⁻ and water.

6. After initial increases in skin temperature, little variability was shown during the last 6-8 hours of the 24 hours exposure.

7. Creatinine values showed little variability during all exposures.

,·8. Urea values were fluctuating during the exposures with an initial increase at the beginning of each exposure.

9. There were similarities between the changes noticed in this study and changes associated with clinical disorders.

8. RECOMMENDATION FOR FURTHER WORK

8. If further work is to be conducted in this field of study, . I suggest the following points:

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1. Further direct study is needed of the mechanisms of sweat gland function and of the effect of hormones of the adrenal cortex. on both sweat gland and kidney, to understand the differences in the handling of salt by these two organs. Such experiments must take· into consideration both the amount of salt consumed by subjects during exposure and their habitual use of salt as a flavouring material.

2. More investigation should be carried out in order to find the effect of short or long exposure to heat and cold on 17-Ketosteroids and 17-hydroxycorticosteroids in an attempt to obtain an index of stress. Such studies should be carried out many times on the same subjects using a rather constant diet and taking into consideration sex, age, race, nutrition or body size and behaviour characteristics of the subjects.

3. Further to this study and to previous studies the role and involvement of the endocrines in adaptation to heat and cold is very close. Such involvement leads one to postulate that since heat and cold affect endocrine activity then a change in hormone level due to a change in environmental condition will lead to a change in,metabolic pathway generally and the activity of the enzyme activated by the hormone under study in particular. Thus an intensive enzymological study should be carried out in search of the effect of changes in environmental conditions on enzymes.

Eventually one must aim at a general "autonomic" description of the process of acclimatization. It is obvious that acclimation,

when complete, involves a dynamic equilibrium between the environment and the organism in a way that biochemical analyses of hormones and enzyme systems cannot describe completely. Many environmental . factors operate through psychological responses which act via the autonomic system and only much later may be associated with hormonal changes. At the present time there are too few measurements of general autonomic response available and future research must be devoted to developing more methods.

9. REFERENCE S

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10. APPENDIX

10.1 EXPERIMENTAL DATA

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Experiment-A-

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Experiment A-I

Table 1: Environmental conditions for

Experiment A-I conducted at 100 C above

ambient temperature.

Experiment A-I

Table 2: Heart Rate, means and standard deviation for the whole group and for Females (F), Males (M), separately, for experiment conducted at 100 C above ambient temperature. Experiment A-I.

Experiment A-1

Table 3: Na^+ , K^+ , Na^+/K^+ , and creatinine values (mg/min) in urine of 12 subjects for experiment conducted at 10º C above ambient . temperature, in addition to the mean, standard deviation for the whole group and for Females (F), Males (M) separately. Experiment A-1.

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Experiment A-2.

Table 4 : Environmental conditions for

Experiment A-2 conducted at 100 below

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ambient temperature.

Experiment A-2

Table 5: Heart rate means and standard deviation for the whole group and for Females (F), Males (M) separately, for experiment conducted at 10° C below ambient temperature. Experiment A-2.

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Experiment A-2

Table 6: Na⁺, K⁺, Na⁺/K⁺ and creatinine values (mg/min) in urine of 12 subjects for experiments conducted at 10° C below ambient temperature in addition to mean, standard deviation for the whole group, and for Females (F), Males (M) separtely. Experiment A-2.

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Experiment-B-

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Experiment B-(i) Part I

 25° C Dry Bulb 20.5° c Wet Bulb **Rh %** 67

Table 7: Environmental conditions for Experiment B-(i) Part I. (at work)

Experiment-B-(i) Part I

Table 8: Age, height. weight, and surface area, in addition to the mean and standard deviation for subjects under Experiment -B-(i) Part I (at work).

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Table 9: Heart rate, eardrum temperature, forehead temperature and chest temperature, mean and standard deviation for experiment conducted at 250 C 68% Rh. Experiment B-(il Part **I.** (at work)

1 - pre-exposure

2,3,4 - during exposure at 40 minute intervals

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5 - post exposure.

Table 10: Na⁺, K⁺, Na⁺/K⁺ values in urine (mg/min) and their means, standard deviations for experiment conducted at 250 C 68% Rh. Experiment B-(i) Part I (at work).

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Experiment *B-(i)* Part I

Table 11: Chloride in urine mg/min (at work), means and standard deviation. Experiment B-(i) Part I

- **1.** 40 minutes after the beginning of the exposure
- 2. 80 minutes after the beginning of the exposure
- 3. 120 minutes after the beginning of the exposure.
Experiment $B-(i)$ - Part I

Table 12: Sweat rate and Nat_2 , K^+ concentration in sweat. (at work) Mean and standard deviation. Experiment B-(i) - Part I.

- 1. mid-exposure
- 2. end-exposure

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Experiment B-(i) - Part I

Table 13: Na+/K+ in sweat (mg/min) (at work) Mean and standard deviation. Experiment $B-(i)$ - Part I.

- 1. mid-exposure.
- 2. end-exposure.

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Experiment $B-(i)$ Part II

 $.30^{\circ}$ c Dry Bulb $.22.2^{\circ}c$ Wet Bulb **%·Rh** 50%

Table 14; Environmental conditions for Experiment B-(i) - Part **11**

Experiment B-(i) Part II

Table 15: Age, height, weight, and surface area in addition to the mean and standard deviation for subjects under Experiment B-(i) Part II (at work).

Table 16: Heart rate, eardrum temperature, forehead temperature, and chest temperature, mean and standard deviation for experiment conducted at 300 C 50% Rh. Experiment B-(i) Part II (at work).

> $\mathbf{1}$ pre-exposure

 $2,3,4$ during exposure at 40 minute intervals.

 5 post-exposure

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Table 17: Na^t, K^t, Na^t/K^t values (mg/min) in urine, and their means standard deviation for experiment conducted at 300C 50% Rh. Experiment B-(i) Part 11 (at work).

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Experiment B-(i) Part **11**

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Table 1&: Chloride in urine (mg/min), mean and standard deviation Experiment B-(i) Part II (at work).

1. 40 minutes after the beginning of the exposure.

2. 80 minutes after the beginning of exposure.

3. 120 minutes after the beginning of exposure.

Experiment B-(i) Part 11

Table 19: Sweat rate and Na⁺, K⁺ concentration in sweat (at work). Mean and standard deviation. Experiment B-(i) Part II.

1. mid-exposure.

2. end-exposure.

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Experiment B-(i) Part 11

Table 20: Na^+/ K^+ in sweat (mg/min) (at work). Mean and standard deviation. Experiment B-(i) Part 11

1. mid-exposure.

2. End-exposure.

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Experiment B-(i) Part II

Table 21: Heart rate, mean and standard deviation. Experiment B-(i) Part II at work.

1 . pre-exposure

2,3,4,- during exposure at 40 minute, intervals 5 ' post-exposure.

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Experiment B-(i) Part III

 30° C Dry Bulb Wet Bulb $23,300$ Rh % 38

Table 22: Environmental conditions for Experiment B~(i) Part **Ill.**

Experiment B-(i) Part III

Table 23: Age, height, weight and surface area in addition the mean and standard deviation for subjects under Experiment B-(i) Part III (at work)

Experiment B-(i) Part III

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Table 24: Heart rate, eardrum temperature, forehead temperature, chest temperature, and mean, standard deviation for experiment conducted at 35° C 38% Rh. Experiment B-(i) Part Ill. (at work)

pre-exposure

2,3,4 during exposure at 40 minute: intervals $\overline{}$

post-exposure.

Table 25·

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Table 25: Na+, K+, Na+/K+ values in urine and their means, standard deviation for experiment conducted at 350 C 38% Rh Experiment B-(i) Part III (at work).

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Experiment B-(i) Part III

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Table 26: Chloride in urine (mg/min). Mean and standard deviation. Experiment B-(i) Part III (at work).

1. 40 minutes after the beginning of the exposure.

2. 80 minutes after the beginning of the exposure.

3. 120 minutes after the beginning of the exposure.

Experiment B-(i) Part III

Table 27: Sweat rate and Na⁺, K⁺ concentration in sweat (at work) Mean and standard deviation. Experiment B-(i) Part III

1 - mid-exposure.

2 - end-exposure.

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Experiment B-(i) Part III

Table 28: $Na⁺/K⁺$ in sweat (mg/min) (at work). Mean and standard deviation. Experiment B-(i) Part III

1. mid-exposure.

2. end-exposure.

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Experiment B-(i) Part III

Table 29: Heart rate, mean and standard deviation. Experiment B-(i) Part III (at work).

> 1 - pre-exposure 2,3,4-during exposure at 40 minute intervals 5 - post-exposure.

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Experiment *B-(ii)* Part I

Table 30: Environmental conditions for Experiment B-(ii) Part I (at rest).

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Experiment *B-(ii)* Part I and Part 11

Table 31: Age, height, weight and surface area, in addition the mean and standard deviation for subjects under Experiment B-(ii) Part I and Part II (at rest). Table 31:

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Experiment B-(ii) Part I

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Table 32: Heart rate, mean and standard deviation. Experiment B-(ii) Part I (at rest).

1,2,3, - during exposure at 40 minute intervals.

Experiment B-(ii) Part ^I

Table 33: Na⁺, K⁺ and Na⁺/K⁺ values in urine. Mean and standard deviation Experiment B- (ii) Part I (at rest).

1 - pre-exposure

2,3,4,- during exposure at 40 minute intervals

5 - post-exposure.

Table 34·

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Experiment B-(ii) Part I

Table 34: Chloride in urine. Mean and standard deviation. Experiment B-(ii) Part I (at rest).

Experiment *B(ii)* Part I

Table 35: Creatinine in urine. Mean and standard deviation. Experiment B-(ii) Part I (at rest).

Experiment B-(ii) Part I

Table 36: Sweat rate, and Na⁺, K⁺, Na⁺/K⁺ values in sweat. Mean and standard deviation. Experiment B-(ii) Part I (at rest).

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Experiment B-(ii) Part II

Table 37: Environmental conditions for \cdot Experiment B-(ii) Part **11** (at rest).

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Experiment B-(ii) Part 11

Table 38: Heart rate, mean and standard deviation. Experiment B-(ii) Part II (at rest).

Experiment B-(ii) Part II

Table 39: Na^t, K^t and Na^t/K^t values in urine. Mean and standard deviation. Experiment B-(ii) Part II.

1 - pre-exposure

2,3,4,- during exposure at 40 minute intervals.

5 - post-exposure.

Experiment *B-(ii)* Part 11

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Table 40: Chloride in urine. Mean and standard deviation. Experiment B-(ii) Part II (at rest).

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Experiment *B-(ii)* Part II

.Tab1e 41: Creatinine in urine. Mean and standard deviation. Experiment B-(ii) Part II (at rest).

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Experiment B-(ii) Part II

Table 42 : Sweat rate, Na⁺, K⁺ and Na⁺/K⁺ values in sweat. Mean and standard deviation. Experiment B-(ii) Part **II** (at rest).

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Experiment B-(iii)Part I

Table 43: Environmental conditions for Experiment B-(iii) Part **I.** (at rest)

Experiment B-(iii) - Part I and Part 11

Table 44: Age, height, weight and surface area in addition to the mean, standard deviation for subjects under Experiment B-(iii) Part I and Part II (at rest).

Experiment B-(iii) Part I

Table 45: Heart rate, mean and standard deviation. Experiment B-(iii) Part I (at rest).

1, 2,3, during exposure at 40 minute intervals.

Experiment *B-(iii)* Part I

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Table 46: Na+, K+ and Na+/K+ values in urine. Mean and standard deviation. Experiment B-(iii) Part I (at rest).

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 $1 -$ pre-exposure 2,3,4,- during_exposure at 40 minute intervals. 5 - post-exposure.

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Experiment B-(iii) Part I

.. Table 47: Chloride in urine, mean and standard deviation. Experiment B-(iii) Part I (at rest).

Experiment B-(iii) Part I

Table 48: Creatinine in urine, mean and standard deviation. Experiment B-(iii) Part I (at rest).

Experiment B-(iii) Part I

Table 49: Sweat rate, \mathtt{Nat} , \mathtt{K}^+ and $\mathtt{Nat}/\mathtt{K}^+$ values in sweat. Mean and standard deviation. Experiment B-(iii) Part I (at rest).

1 - mid-exposure.

2 - end-exposure.

Experiment **B-(iii)** Part **II**

Table 50: Environmental conditions for Experiment B-(iii) Part II (at rest).

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Experiment B-(iii) Part II

Table 51: Heart rate, mean and standard deviation. Experiment $B-(iii)$ Part II at rest.

> $1,2,3$, - during exposure at 40 minute intervals.

Experiment B-(iii) Part II

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Table 52: Nat , K^+ , and Nat/K^+ values in urine. Mean and standard deviation. Experiment *B-(iii)* Part 11 (at rest).

- 1 pre-exposure
- $2,3,4, -$ during exposure at 40 minute intervals
- 5 post -exposure.

Table 52a

Experiment B-(iii) Part **¹¹**

Table 52a: Chloride in urine. Mean and standard deviation. Experiment B-(iii) Part **11** (at rest).

- 1 pre-exposure
- 2,3,4, during exposure at 40 minute intervals
- 5 post-exposure.

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Experiment B-(iii) Part 11

Table 53: Creatinine in urine. Mean and standard deviation. Experiment B-(iii) Part II (at rest).

Experiment B-(iii) Part II

Table 54: Sweat rate, Na+, K+ and Na+/K+ values in sweat. Mean and standard deviation. Experiment B-(iii) Part II (at rest)

> $1 - mid-exposure$
 $2 - end-exposure$ end-exposure.

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Experiment **-C-**

Experiment c-

Table 55: Environmental conditions for Experiment-C-.

Table 56:

Experiment C

Tab1e56: Age, height, weight, surface area. Mean and standard deviation for subjects under experiment-C.

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Table 57 :

Experiment -c-.

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Table 57: Heart rate, PCV values for subjects under Experiment -C-.

Experiment **-D-**

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. Table 58

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Experiment D-(i) Part I

Table 58: Environmental conditions for Experiment D-(i) Part I.

Experiment D-(i) Part I

Table 59: Age, height, weight, surface area, mean and standard. deviation of subjects under experiment D-(i) Part I.

> 1 2 pre-exposure. end-exposure.

Experiment D-(i) Part I

Table 60: Heart rate and urine minute volume values, mean and standard deviation for subjects under Experiment *D-(i)* Part I.

Table 61: Na⁺, K⁺, Na+/K+, Cl⁻ values in urine, mean and standard deviation for subjects under Experiment D-(i) Part I.

> 1 - pre-exposure; 2 - end-exposure; 3 - post-exposure. (control)

Experiment D-(i) Part I

Table 62: Urea, creatinine and creatinine/bodyweight values in urine, mean and standard deviation for subjects under Experiment D-(i) Part I.

 1 - pre-exposure (control); 2 - end-exposure; 3 - post-exposure.

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Experiment *D-(i)* Part **II**

Table 63: Environmental conditions for E>:periment D-(i) Part **II.**

Experiment *D-(i)* Part 11

Table 64: Height, weight, surface area and mean, standard deviation for SUbjects under Experiment *D-(i)* Part 11.

> 1 - pre-exposure. 2 - end-exposure.

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Experiment D-(i) Part II

Table 65: Heart rate, urine minute volume, mean and standard deviation for subjects under Experiment D-(i) Part 11.

Experiment D-(i) Part II

Table 66: Na⁺, K⁺; Na⁺/K⁺, Cl⁻ values, mean and standard deviation for subjects under Experiment D-(i) Part II.

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- 1 pre-exposure (control)
- $2 -$ end-exposure
- 3 post-exposure.

Experiment D-(i) Part II

Table 67: Urea, creatinine, creatinine/body weight, values in urine, mean, and standard deviation for subjects under Experiment D-(i) Part II.

1 -pre-exppsure; 2 - end-exposure; 3 ~ post-exposure. (Control)

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Experiment D-(ii) Part I

Table 68: Environmental conditions for Experiment D-(ii) Part I.

Experiment D-(ii) Part I

Table 69: Age, height, weight, surface area, mean and standard deviation for subjects under Experiment D-(ii) Part I.

1 - pre-exposure.
2 - end-exposure.

end-exposure.

Experiment O-(ii) Part I

Table 70: Heart rate, mean and standard deviation for subjects under Experiment O-(ii) Part I.

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Experiment D-(ii) Part I

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Table 71: Urine volume, minutes urine volume, and urea values in urine for subjects under Experiment D-(ii) Part I.

1 - early morning sample

- 2 pre-exposure sample
- 3 mid-exposure sample
- 4 end-exposure sample
- 5 post-exposure sample.

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Experiment D-(ii) Part I

Table 72: Na $^{\tt f}$, K $^{\tt f}$, Na $^{\tt f}/$ K $^{\tt f}$, and Cl $^-$ values in urine, mean and standard deviation for subjects under Experiment D-(ii) Part 1.

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1 - early morning sample; 2 - pre-exposure sample; 3 - mid-exposure sample; 4 - end-exposure sample; 5 - post-exposure sample.

Experiment D-(ii) Part I

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Table 73: Creatinine and creatinine/Kg(body weight) values in urine, and mean and standard deviation for subjects under Experiment D-(ii) - Part I.

> 1 - early morning sample; 2 - pre-exposure sample; 3 - mid-exposure sample; $4 - end-exposure 5 - post-exposure sample.$

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Table 74: Haemoglobin, naematocrit values, red and white blood cells count, mean and standard deviation for subjects under Experiment D-(ii) Part I.

> 1 - pre-exposure. 2 - mid-exposure. 3 - end-exposure.

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Experiment D-(ii) Part 11

Table 75: Environmental conditions for Experiment D-(ii) Part **11 •**

Experiment D-(ii) Part II

Table 76: Age, height, weight, surface area, mean and standard deviationfor.'subjects under Experiment D-(ii) Part II.

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Experiment D-(ii) Part II

Table 77: Heart rate, mean and standard deviation for subjects under Experiment D-(ii) - Part 11.

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Experiment D-(ii) Part II

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Table 78: Urine volume, minute urine volume, urea values in urine, mean and standard deviation for subjects under Experiment *D-(ii)* Part 11.

1 - early morning sample

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- 2 pre-exposure sample 3 - mid-exposure sample
- 4 end-exposure sample
- 5 post-exposure. sample.

Experiment D-(ii) Part 11

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 $^{\circ}$ Table 79: Na⁺, K⁺, Na⁺/K⁺ and Cl⁻ values in urine, mean and standard deviation for subjects under Experiment D-(ii) Part II.

- 1 early morning sample
- 2 pre-exposure sample
- 3 mid-exposure sample
- 4 end-exposure sample
- 5 post-exposure sample.

Experiment D-(ii) Part II

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Table 80: Creatinine and Creatinine/Kg (Body weight) values in urine and mean, standard deviation for subjects under Experiment D-(ii) Part II.

- 1 early morning sample
2 pre-exposure sample
- 2 pre-exposure sample
3 mid-exposure sample
- $3 \text{mid-exposure sample}$
 $4 \text{end-exposure sample}$
- 4 end-exposure sample
5 post-exposure.sample
- post-exposure.sample.

Experiment D-(ii) Part 11

Table Bl: Haemoglobin, haematocrit values, red and white blood cell count, mean and standard deviation for subjects under Experiment D-(ii) Part II.

- ·1 pre-exposure 2 - end-exposure.
- 3 post-exposure.

Experiment *D-(iii)* Part I

Table 82: Environmental conditions for Experiment *D-(iii)* Part I.

Table 83:

Experiment D-(iii) Part II

Table 83: Environmental conditions for Experiment D-(iii) Part II.

Experiment O-(iii) Part I and Part II

Table 84: Age, height, weight, mean and standard· deviation for subjects under experiment O-(iii) Part I and II.

Table 85:

Experiment D-(iii) Part I and II

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Table 85: Heart rate, mean and standard deviation for subjects under Experiment D-(iii) Part I and **I1.**

1 - pre-exposure

2 - end-exposure

3 - post-exposure.

$\frac{6}{10}$ Table 86

Experiment D-(iii) Part I and II

Table 86: Urine volume, minute urine volume, mean and standard deviation'fer subjects under Experiment D-(iii) Part I and II.

1 - pre-exposure

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2-- end-exposure

J - post-exposure.

Experiment D-(iii) Part I and II

Table 87: Na⁺, K⁺,Na⁺/K⁺ values in urine, mean and standard deviation for subjects under Experiment D-(iii) Part I and II

1 - pre-exposure

2 - end-exposure

3 - post-exposure.

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Table 88: Urea, creatinine, creatinine/Body weight values in urine, mean and standard deviation for subjects under Experiment D-(iii) Part I and 11.

> 1 - pre-exposure 2 -.end-exposure 3 - post-exposure.

Experiment **-E-**

Experiment **-E-**

Table 89: Envircnmental conditions for. Experiment-E~

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Experiment -E-

Table 90: Age, height, weight, surface area, mean and standard deviation for subjects under Experiment -E-.

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Experiment -E-



Table 91: Total water intake, and urine output in 24 hours for subjects under Experiment -E-

- 1 Total urine output in 24 hours.
- 2 Volume of urine output 'I hours after the end of exposure.

#### Table 92:

#### Experiment **-E-**



Table 92: Body weight, during 24 hours exposure, mean and standard deviation for subjects under Experiment-E<del>.</del>

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Table 93: Urine volume (ml), minute urine volume (ml/min), mean and standard deviation for subjects under Experiment-E-for 24 hours exposure.

A - urine volume at that time of the day

B - minute urine volume at that time of the day.

# Experiment **-E-**



*Time* 



Table 94: Forehead and chest temperature (OC) mean and standard deviation for subjects under Experiment -E- for 24 hours exposure.

 $F$  Forehead temperature at that time of the day. C Chest temperature at that time of the.day.

Experiment **-E-**





Time  $\cdot$ 

Table 95: Hand and foot temperature (<sup>O</sup>C), mean and standard deviation for subjects under Experiment E for 24 hours exposure.

> H Hand temperature at that time of the day<br>F Foot temperature at that time of the day Foot temperature at that time of the day

#### Experiment\_E\_



Table 96: Oral temperature (°C) mean and standard deviation for subjects under Experiment-E-for 24 hours exposure.

#### Table 97.

Experiment-E-



Table 97 : Heart rate (Beats/min), mean and standard deviation for subjects under Experiment-E-for 24 hours exposure.

Experiment -E-



Table 98: Blood pressure (mm, Hg), mean and for 24 hours exposure. standard deviation for subjects under Experiment -E-

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## Experiment E



table 99: Haematocrit values (%), mean and standard deviation for subjects under Experiment -E- for 24 hours exposure.

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Table 100: Na<sup>t</sup>, K<sup>t</sup> values (mg/min) in urine, Na<sup>t</sup>/K<sup>t</sup>, mean and standard deviation for subjects under Experiment -E- for 24 hours exposure.

Experiment -E-



Table 101: Chloride values (mg/min) in urine, mean and standard deviation for subJects under Experiment -E- for 24 hours exposure.

Experiment -E-

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Table 102: Urea values *(mg/min)* in urine, mean and standard deviation for subjects under Experiment -E- for 24 hours exposure.



Table 103: Creatinine values in urine (mg/min), and creatinine/body weight (mg/min/Kg), mean and standard deviation for subjects under Experiment -E- for 24 hours exposure.

A Creatinine value at that time of the day<br>B Creatinine/Body weight value at that time

Creatinine/Body weight value at that time of the day.

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## Experiment -E-



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Table 104: The total 24 hour sample values for Na⁺, K⁺, Cl⁻, Na⁺/K⁺, urea, creatinine, creatinine/body weight, and minute urine volume for subjects under Experiment -E- for 24 hours exposure.

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Experiment **-F-**

Table 105: Age, height, weight, surface area, mean and standard deviation for sUbjects under Experiment-F-Part I and **II.**

Experiment F - Part I and II

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Table 106: Na⁺, K⁺, Na⁺/K⁺, Cl⁻ values in urine, mean and standard deviation for subjects under Experiment -E-

- l pre-exposure
- 2 mid-exposure
- 3 end-exposure
- I resting condition
- **1I -** working condition.

Experiment **-F-**

Table 107: Urea, creatinine values in urine, creatinine/body weight, mean and standard deviation for subjects under Experiment **-F-**

- 1 pre-exposure
- 2 mid-exposure
- 3 end-exposure
- I resting condition
- I1 working condition.

10.2 METHOD

A Sodium, Potassium (Flame photometer)

(Based on the method used by the Department of Human Biology, Loughborough University of Technology).

Preparation of the Standards

- a. Molar solutions of NaCl (58.46 *g/L*) and KCl (74.56 g/l) were made up and stored in polythene bottles.
- b. Stock solutions were made up

c. A Standarization series was prepared as follows:

All solutions were made up to 1 litre with deipnized **water.**

Calibration

The galvonometer scale is linear, and readings are proportional to the current generated in the photocell. The relationship of flame intensity to solution concentration is not necessarily linear. Thus, calibration curves were constructed using the above standards. The instrument was calibrated to read 80 with the highest standard concentration prepared. The range chosen 0-80. Sample solutions that did not lie within this range were further diluted. The lowest convenient range was used, since interference effects are less marked at low concentrations.

Urine Samples

Urine samples were diluted 1:100 for Na and 1:200 for K. When any of the samples were too concentrated and the scale reading fell outside the range, the Urine sample was further diluted as required and the dilution was taken into account when calculating the ion concentration in milligrams per minute.

Calculation

- 1. Calibrate photometer with the standards prepared, using appropriate filter. Plot calibration curves for each ion. (There is a different curve for Na and K).
- 2. The ,flame photometer readings of the samples were converted to mM/L from the calibration curve for Na and K.
- 3. Multiply by dilution to give correct *mM/R.* for each sample.
- 4. Ion concentration $\text{Im}M/\ell$ x atomic weight Na⁺ KT *(gm)*

 \equiv mq/l

- 5. Urine Volume (m*L*) % Time (minutes) = m*L*/min
- 6. $~\frac{mg}{L}$ 1000 ^x*mR./m.i.n* x *mg/m.i.n* concentrations.

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B Chloride Test (Titrimetric method)

This test is a modified version of the shales and shales method 1941 by SCIAVO.

Chloride 100 mEq/t

Procedure:

a. Urine was not diluted.

b. All reagents were brought up to room temperature.

c. The sample and standard were made up as follows:

Mixed well and titrated adding with a microburette (calibrated in 0.01 *m!)* the titrating reagent drop by drop. Mixed until a pale persistent violet colour was obtained: the amount of reagent added was recorded in *m!6.* 283

Calculations

 $m\ell$ of reagent for the sample
 $m\ell$ of reagent for the standard x 100 = Cl mE q/ ℓ

Cl mEq/² Urine minute volume $(m\ell/min)$ *x atomic weight* of Cl 1000

= Cl concentration in urine *mg/m.i.11*

C Creatinine Test

The estimation of creatinine has been done by a method by I.S.V.T. SCLAVO, which is a modification of the method developed by Henry 1968.

Principle

Reaction of picric acid with urine creatinine in an alkaline solution lead to the formation of a red-orange complex photometrically measurable.

Reagents

- 1. Deproteinizing reagent.
- 2. Chromogen prepared just before use in the experiment by mixing equal volumes of the sodium hydroxide and picric acid provided. Stability of chromogen lasts for 8 hours if kept at room temperature and away from direct sunlight.
- 3. Standard was ready to use.

Procedure

1. The reagents were brought up to room temperature.

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- 2. The urine samples were diluted 0.1:5 with distilled water.
- 3. The sample, standard and the blank were prepared as follows:

4. The blank and the standard test tubes were ready.

- 5. The sample test tube was mixed well and allowed to stand for 5 minutes. The test tube was then centrifuged at 3000-6000 r.p.m. for 10 minutes.
- 6. The clear supernatant was then transferred to a clean test tube. From the clear supernatant 1.5 *ml* was transferred to another tube.
- 7. Chromogen was now prepared and 1.5 *ml* was added to each "of the sample, standard and blank. The tubes were all mixed well and incubated at room temperature (22º C to 28° C) for 30 minutes avoiding direct sunlight.
- 8. A spectrophotometer was used to read the optical density of the samples and standard at 525 nm against the blank.

Calculation

O.D. sample O.D. standard x 100 = creatinine in *mg/100 ml*

creatinine (mg/100 ml x urine minute volume (ml/min) 100

= creatinine in *mg* per min.

The estimation of urea in urine was based on berthelot urease method and modified by I.S.V.T. SCALVO.

Principle

Urease catalyses hydrolysis of urea to ammonium carbonate. The ammonium ions produced react with phenol and hypochlorite in the presence of nitroprusside to form a blue indophenol. The colour developed is proportional to the concentration of urea.

Reagents:

1. Standard

2. UI'ease

3. Reagent F (phenol and Nitroprusside)

4. Reagent I (Hypochlorite and Sodium hydroxide) Stability of reagents F and I: 3 months (in a dark bottle).

Procedure: - Urine was diluted 1:100 with distilled water.

- Reagents were brought up to room temperature. - Sample, standard and blank tubes were prepared as follows:

Mixed well, and incubated for 10 minutes at 37⁰ in a water bath

Mixed well after addition of each reagent and incubated for 10 minutes at 37° C in a water bath.

spectrophometer was used to read the optical density of all tubes at 580 *nm* against the blank.

Calculation

0.0. sample 0.0. standard $x = 3$ = urea $q/d\ell$ of urine

urea (mg/m*l*) x urine minutes volume *ml/min*

= urea *m9/m.i.11*

E Estimation of sodium,potassium, chloride, Urea and Creatinine using Auto analyzer SMA 6/60

Reagents

Urea \cdots Ferric Chloride and orthophosphoric acid in 20% sUlphuric acid Diacetyl menoxime and thioseme carbazide in water + brij 35.

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Creatinine : saturated picric acid solution. 0.5 .N sodium hydroxide *1.8%* w/v sodium chloride + brij 35.

Methods:

- 1. Sodium and Potassium: The sample is diluted in lithium sUlphate. The sodium, potassium and lithium ions dialyse into a water recipient stream which, after a time delay, is sprayed by an atomiser into the flame of a flame photometer. In the flame, the ions emit a wavelength of light characteristic of the element concerned, whose intensity is proportional to the concentration of that element. A photocell detects the light output and the outputs of the sodium and potassium photocells are balanced against the lithium photocell in order to eliminate fluctuations caused by variations in flame.
- 2. Chloride: The sample is diluted in water and dialysed into dilute nitric acid. Colour reagent which contains mercuric thiocyanate and ferric nitrate is added to this .' stream. The chloride ions displace thiocyanate ions forming mercuric chloride and the thiocyanate ions react with the ferric ions to produce the red/brown coloured ferric thiocyanate which has an absorbance maximum at 480 **nm.**
- 3. Urea: The sample is diluted in water and the urea dialyses into the colour reagent recipient stream. Acid is added and a condensation reaction takes place between the urea and the diacetul menoxime in the colour reagent. The condensation product is pink coloured with a maximum absorbance at 520 nm.

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4. Creatinine: The plasma is diluted with saline and dialysed into water. A solution of 0.5 *N* sodium hydroxide is added, followed by saturated picric acid. Creatinine forms a red colour with alkaline picrate with a maximum absorbance at 505 *nm.*

Calculation

- a. Readings of samples from auto analyzer in m moles/litre.
- b. Multiply by dilution to give correct m moles/litre for each sample.
- c. m moles/litre x atomic weight = mg/l litre.
- d. Urine vol $%$ time = $m\ell/m\ell n$.
- e. *mg/l* 1000 x m ℓ /min $mq/m\zeta n$

Calibration of the S.M.A. 6/60

The chloride linearity was set daily using aqueous standards. Every 10 samples used a serum standard (wellcome Autoset M) for all channels, while wellcome Autoset Hand "Armitrol" were used as control sera at positions 5 and 9. If the values fell outside the pre-determined limits, then that batch of 10 samples would be repeated.

F

Blood count a. white cell count

b. red cell count

(Based on methold used by the Department of Human Biology)

Collection of sample

Immerse the hand in waterbath at 48° C or in a bucket of

of "warm" water to arterialise the blood. Quickly dry a finger a puncture the finger-tip skin using a sterile disposable lancet. Do not squeeze the finger but let a drop of blood form by natural blood pressure. Reject the first drop then collect the next drop by capillarity or minimum suction into a clean haemogytometer "red pebble" pipette.

a. White cell count (the number of white cells contained $\frac{1}{2}$ $\frac{m}{3}$ of blood):

. Diluting fluid: 2% glacial acetic, to lyse red cells, tinged with methylene blue, to stain the white cells.

Method: Dilute 0.02 *mt* of blood in 0.38 *mt* diluting fluid to make a 1 in 20 dilution. Mix for 2 minutes and fill counting -chamber by. means of a pasteur pipette. Count white cells in white cell counting areas.

Calculation: If N cells are counted in 0.1 $mm³$ then the bucocyte count per mm^3 = $N \times 10 \times 20$ (dilution) $=$ N x 200 cells/mm³

Range: 6 x 10^3 to 12 x 10^3 cells/mm³ depending on state of health and exposure to common infeotious conditions.

b. Red cell count: (the number of red blood cells contained in 1 mm^3 of blood)

Diluting fluid: Hayems fluid has been used which was:-

Mercuric chloride -0.59 g Sodium sulphate -5.0 g Sodium chloride -1.0 g Distilled water to 200 *mt*

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This fluid is isotonic, of high specific gravity to prevent the cells from settling too quickly and to prevent "rouleau formation" and the formation of bacterial growth, which might cause cell wall damage.

Method: Dilute 0.2 *mt* blood in 4 *mt* diluting fluid to make a 1 in 200 dilution. Mix for 2 minutes and fill counting chamber. Count red cells in 5 squares of red cell counting areas.

Calculation: If *N* cells are counted in 80 small square 0.1 mm in depth (0.02 mm^3 in volume), then red-cell count in millions per *mm³ •*

> = $N \times 1/_{0.02}$ x 200 (dilution) *=* N x 10,000

Range: 4.3 x 10% to 5.9 x 10⁶ cells/mm³ depending on nutrition iron state, generally activity level, sex and point in menstrual cycle.

G Measurement of haemoglobin using a photoelectric colorimeter (Based on method used by the Department of Human Biology

Collection of sample: Same procedure as in blood count (F) **'was used.**

The cyanmethaemoglobin method

The basis of the method is to dilute blood in a solution containing potassium . cyanide and potassium ferricyanide (Drabkin's Reagent). Haemoglobin, methaemoglobin and

carboxyhaemoglobin (but not sulphaemoglobin) are all rapidly converted to cyanmethaemoglobin. The absorbance of the solution is then measured in a photoelectric colorimeter at a wavelength of 540 nm .

Reagents:

This is based on Drabkin's cyanide-ferricyanide solution. Modification as recommended by van Kampen and Zijlstra.

The pH of the solution should be adjusted to 7.0-7.4. and if stored at room temperature in a black painted polythene bottle the solution keeps for several months.

Method:

0.02 $m\ell$ of blood is added to 4 $m\ell$ of reagent. The tube containing the solution is stoppered with a rubber bung and inverted several times. After being allowed to stand at room temperature for a sufficient period of time to ensure the completion of the reaction, the solution of cyanmethaemoglobin is ready to be compared with the standard in a photoelectric cOlorimeter. at 540 *nm* against a reagent blank. An ampoule of cyanmethaemoglobin standard (brought to room temperature if previously stored in a refrigerator) is opened and the optical density of the solution is

measured in the same photoelectric colorimeter against the blank. The standard should be discarded at the end of the day, and during the day must be kept in the dark.

Calculation

Haemoglobin (g/l *OOml)* =

O.D. blood sample O.D. standard ^xconc. st *(mg/l00 ml)* x dilution factor 1000

H Packed Cell Volume P.C.V.

(Based on method used by Department of Human Biology)

Method:

For the standard method capillary tubes are available which are 75 mm in length and have an internal· diameter of about lmm. They can be obtained plain or coated inside with a layer of heparin. The latter type is suitable for the direct collection of capillary blood. A plain tube is used for anticoagulated venous blood. The blood is allowed to enter the tube by capillarity, leaving at least 15 mm unfilled. The tube is then sealed by heating the dry end of the tube rapidly in a fine flame, e.g. the pilot light of a bunsen burner, combined with rotation, or by the use of a plastic seal. After centrifugation for 5 minutes the PCV can be measured using a reading device.

Normal range for PCV (haematocrit) value:

.1 SWEAT RATE

(Based on the method used by the Department of Human Btology)

- 1. Two pieces of filter paper (total surface area 47.49 cm^2) placed in a petri dish was weighed on an analytical balance (with the lid of the petri dish). Care was taken that the petri dish was absolutely clean.
- 2. At all times the filter paper was handled with a clean forcep.
- 3. The petri dish was labelled with black marker pen. Paper labels were not used.
- 4. When in the chamber, the filter paper was removed with a forcep and placed over the right and left scapulae, of the subject.
- 5. Each filter paper was covered with a square plastic sheet (washed in distilled water and blotted dry) and fixed in place with adhesive strapping.
- 6. After a certain time period, the wet filter papers were removed with a forcep and transferred into their original petri dish. The lid was placed on. The petri dish was immediately weighed.
- 7. Same procedure was followed when fresh filter paper was placed on the scapulae of the subjects. The weighed filter papers in the petri dish was prepared in advance for each time period.
- S. The difference in weight of the petri dish with the filter paper gave the amount of sweat collected in grams.

Calculation

Petri dish + filter paper + sweat - petri dish + filter paper = sweat in gms.

Sweat in $gms./1000/47.49/time =$ sweat rate in mg/cm²/time.

J THE DETERMINATION OF THE IONIC CONCENTRATION OF SWEAT

- 1. After the weight of the sweat cOllected was determined, the sweat filter papers were transferred to a container (small beaker) containing 10 mls of distilled water.
- 2. The filter papers were macerated to a fine pulp in the **container.**
- 3. The liquid from the resulting pulp was filtered through a Gooch crucible and the filtrate collected in a test tube.
- 4. The filtrate was analysed for Na and K by flame photometry (procedure already described).
- 5. Before analysis, suitable dilutions of the filtrate was prepared: 1:5 for both Na and K.
- **6;' A blank solution was prepared by macerating the same weight** of filter paper in 10 mls. of distilled water, filtered and analysed in the same manner.
- 7. The blank scale readings for each ion was deducted from the sweat analysis readings for each ion.

Calculations.

- 1. The flame photometer readings (corrected) were converted to mM/l from the calibration curves.
- 2. Correction factor was found:

x - weight of sweat collected in grams.

 \overline{X}

Correction factor (CF) = $\underline{10 + X}$

3. Determination of Na and K concentrations in mg/min. Corrected FP reading (mM/1) x Dilution (5) x CF x weight of Na (23g) or K (39.1 g) x sweat rate in mg/min/ 1000 = Ion concentration in mg/min.

10.3 OPERATING INSTRUCTIONS

PYE UNICAM

UNICAM SP 600 SERIES 2 SPECTROPHOTOMETER

Procedure:

- **1.** Turn main switch to zero.
- 2. Allow 10 minutes for the instrument to become stabilized. 3. - Select an appropriate photocell:

335-400 nm Blue photocell with filter *in* position 2 *400-625 nm* Blue photocell with filter *in* position 1 *625-1000 nm* Red photocell with filter *in* position 1

- 4. Bring meter needle to zero with the zero control.
- 5. Set wavelength control to required wavelength.
- 6. Turn main switch to 100% with blank or solvent *in* the light beam. Bring the meter needle to zero with the slit control.
- 7. Switch to test. Move sample into light beam and bring meter needle to zero with the transmittance control.
- 8. Read off the transmittance or absorbance of the sample from the dial.
- 9. Repeat the above sequence from operation (6) whilst sample *is* being measured.
- 10. In between sets of readings turn the main switch back to zero and check, then to 100% and check as *in* (6)

B Flame photometer

- 1. Turn the sensitivity control fully anti-clockwise.
- 2. Select and insert the appropriate optical filter for the ion' under test.
- 3. Switch the instrument ON, ensuring that the galvanometer is unclamped.
- 4. Move aside the mica window and insert a lighted taper above the burners.
- 5. Turn on the gas supply to light the flame, withdraw the taper and close the window.
- 6. Switch on the compressor. Adjust the air control to give a reading of 10 p.s.i. on the pressure gauge. The flame will tend to be noisy when starting from cold and the instrument should be left for a few minutes to stabilize.
- 7. Slide a beaker of distilled water into the sample recess.
- 8. Adjust the gas control to produce a flame with one large central blue cone.
- 9. With the distilled water sample still in position, slowly close the gas control until ,ten separate blue flame cones just form. This provides a reference level for all subsequent settings; the control settings may be left undisturbed by turning the gas and air supplies off after use at their sources and not at the instrument. Thus little, if any readjustments should be necessary for subsequent operation. Do not adjust the gas supply to alter the sensitivity of the instrument.
- 10. Set the galvanometer to zero by means of the "zero" control against a reagent "blank" solution.

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- 11. Replace the reagent blank by a standard solution of the concentration corresponding to a reading of 80 on the calibration curve *in* use and adjust the sensitivity control to give approximately full-scale deflection.
- 12. Reset the zero against the blank solution.
- 13. These two settings may need to be rechecked back and forth until both reed correctly.
- 14. Check the zero reading with the reagent blank.
- 15. Present in turn each standard solution prepared and note the readings. For the calibration curve plot meter readings on the ordinate and concentrations on the abscissa.
- N.B.Use correct filter for each standard, also wash instrument through with the distilled water before presenting a different standard.

When the instrument has been calibrated for all the test ions you may proceed with your experiment.

W.B.G.T. Index Meter Min 3 MK5

- a. On the top panel will be found the following controls:
	- 1. Toggle switch marked (Instrument)
	- 2. $" " " " " " (Fan)$
	- 3. Rotary pointed knob marked.
	- 4. press switch marked (Battery)

5. Jack socket marked

c

b. Switch on the instrument batteries by means of the toggle switch marked "Instrument ON".

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- c. With the pointed knob set to "probe and test", check the batteries by pressing the "battery test" switch.
- d. The meter pointed should fall inside the red segment on the right of the meter scale.*

* If it fails batteries should be changed.

- e. To measure Wet Bulb Temperatures,turn pointed knob to position **"W",** read off temperature on top scale.
- f. To measure Dry Bulb Temperatures, turn pointed knob to position "D", read off temperature from top scale
- g. To measure Radiation Globe Temperatures, turn pointed knob to position "G" and read temperature on top scale.
- h. To read combined WBGT Index, turn pointed knob to WBGT and read from the second scale coloured red.

D Auto Analyzer SMA 6/60

- 1. Switch instrument power to "ON".
- 2. Switch on pumps and turn the wash valves to reagents.
- 3. Connect sodium and potassium lines to the side arm of the flame photometer de-bubbler.
- 4. When reagents have been pumping for at least 10 minutes, set colorimeter energy levels as follows:

(i) Switch function switch of programmer module to "Energy-R".

(ii) Set meter to read 50 (mid-scale) with colorimeter reference aperture.

(iii) Switch function switch to "Energy-S" and set each channel to 50 on the meter with the appropriate colorimeter aperture.

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(iv) Switch function switch tc "Record".

- 5. Set base lines for each channel with "Baseline" knob on programmer.
- 6. Disconnect top line of flame photometer de-bubbler. Turn "Air ATOMISER" anti-clockwise until flame starts to make a noise. Turn the knob clockwise until the air pressure gauge reads 19 p.s.i. and then adjust the knob until the size of the air and liquid segments in the top line of the de-bubbler is equal. Re-connect the top line of the de-bubbler.
- 7. Set chloride expansion as follows:

(i) Place three high chloride expansion standards alternately with three low standards on sample turntable. (ii) Switch on sampler when silver segment of timing cam on programmer is opposite the marker.

(iii) When the first high standard comes through, set the chloride to 160 mark on chart recorder with the "calibration" knob.

(iv) 'Set first low standard to 70 on the chart recorder with Cl EXP knob on programmer sub-panel.

(v) Switch to manual advance and follow the chloride peak. When chloride reaches maximum pen deflection on the second high standard switch EXP/NORM switch to EXP.

(vi) If pen position alters, re-set with EXP/CAL knob to original position.

(vii) Re-set advance to AUTO and Re-set next low chloride as before, if necessary.

(viii) The third high and low chloride standards are for checking and re-setting as necessary.

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- 8. Set Technilogger to required number m 1 (i.e. if starting at no. 21 set to 20) and leave on standby.
- 9. Sample three wellcome II's and set to appropriate values with "calibration" knob on programmer. Switch on teletype.
- 10. On last WII switch logger.from Standby to Operate.
- 11. When first test comes through ensure that the teletype prints out correctly and in the right position.

PHASING

- 1. Place WII unassayed serum on sampler turntable as follows: WII, WII, water wash, WII, WII, water wash, etc.
- 2. Start sampling as normal.
- 3. Switch to "manual" and advance to "creat".
- 4. Allow the creatinine peak to draw on the chart recorder and adjust the knurled outer wheel of the timing wheel until the "creat" segment of the wheel coincides with the last third of the creatinine plateau

- 5. Advance to the channel shown on timing wheel and switch to **"Auto".**
- 6. Watch the $TCO₂$ peak and assess whether the coil needs to be bigger or smaller.

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N.B. If the slope is on the frontof the peak the coil. size should be decreased and vice versa. Do not forget that the peak *is* moved to suit the timing.

the scan takes place over

- 7. Alter the TCO_2 coil as necessary. This channel is phased first because the phasing coil is at the beginning of the manifold so that any alteration takes time to come out at the colorimeter.
- 8. Alter the other channels in a similar way as required.

E Environmental Chamber

- 1. Switch onmaihs water supply.
- 2. Turn on main isolater switch.
- 3. Select 'Billman Switch' and or 'silican'liquid services'.
- 4. Switch on Isolater on 'Billman' Unit.
- 5. Press start button on fan dial up to 90-95%.
- 6. Adjust fresh air dial to the amount of fresh air, required.
- 7. Set up the conditions required: -10^0 -40 $^{\circ}$ C $+1\$ C and for Rh 20%-95% ± 2.5%. Any of the conditions required could be controlled automatically or manually by adjusting the knobs to the proper position.
- 8. Take reading outside and inside chamber.
- N.B. Few adjustments using the controls could be necessary at a time depending on the general environmental condition.

10,4 INSTRUCTIONS AND FORMS OF SUBJECT'S DATA

Instructions to Volunteers

A research investigation at the Department of Human Sciences at Loughborough University of Technology, to find whether there is a physiological indication in human beings as a response of adaptation to stress in different simulated laboratory environmental conditions.

- 1. If you accept to volunteer as a subject for this research work you should be aware of the following:
	- a. It is your right to know about the experimental procedures.
	- b. You are able to withdraw from the experiment at any time.
	- c. The experimentor will respect the confidentiality of any personal information the subject may give.
	- d. You should be prompt and follow the timetable assigned to you.

2. Your participation in the research starts on

AT

3. Record the last time you empty your bladder am/pm .. time date

4. Empty your bladder when you get up in the morning

......... am/pm ... time date

keep/discard this urine.

5. Don't empty your bladder until you start the experiment

at **a.m.**

Please be sure that:

- Avoid alcoholic intake the night before the experiment. $a.$
- Don't take any stimulant or drugs. $b.$
- other: $\ddot{\rm c}$.

Thank you M.S. MROUE

Confidential Subject code no: Date:

1. Surname: Mr./Mrs./Miss

2. First names:

3. Date of Birth:

4. Nationality

5. Are you in good health?

6. Are you at present under medical treatment of any kind?

7. Please add here other things which you may think $\frac{arc}{n}$. important and may influence the experiment.

M.S. MROUE

Research student.

Research work by M.S. MROUE

Personal note concerning the subject:

Contract

M.S. MROUE LOUGHBOROUGH UNIVERSITY OF TECHNOLOGY 3 FALKNER COURT LOUGHBOROUGH LEICS. LE11 3TU

 $\hat{\mathcal{A}}$

Date of Experiment

Col c

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10.5 EXPERIMENTAL CHARTS

Conversion of Temperature Units (Fahrenhelt/Centigrade) *

Messuremer

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Ol

 $-14^{\circ}C \rightarrow 4.8^{\circ}F$: 38.3°C = 100.9°F; 89°C = 192.2°F
550°C = 1022°F Examples: Fahrenheit - Centinrade Centigrade - Fahrenheit - 59 to +79°F (1°F inscrede) -59 to 0° C (1°C integrals) 5 T T 7 'n ī Ŧ ö Ā ÷ x ī $\frac{1}{3222}$ $\frac{1}{2}$ $\frac{47.0}{100}$
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70 -10.03
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24 , 陆航航航视 弹引放转机 列特列列列 10:11:20:10 12:20:50 12:20:10 12:20:10 12:20:10 12:20:10 12:20:10 12:20:10 12:20:10 12:20: $\begin{array}{l} 31.72 \\ 32.28 \\ 12.29 \\ 33.39 \\ 13.39 \\ 13.39 \\ 14.59 \\ 15.39 \\ 15.39 \\ 15.39 \\ 15.39 \\ 15.46 \\ 15.46 \\ 15.46 \\ 15.46 \\ 15.47 \\ 15.47 \\ 15.47 \\ 15.47 \\ 15.47 \\ 15.47 \\ 15.48 \\ 15.49 \\ 15.41 \\ 15.42 \\ 15.43 \\ 15.43 \\ 16.47 \\ 17.48 \\ 18.49 \\ 19$ 32.22
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106, 7
108, 3
110, 3
112, 1
113, 7
113, 7 ----
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108 $\frac{105}{107}$
 $\frac{107}{105}$ 7 7 ï ō ī Ŧ ī T $\begin{array}{r} 3 & 4 & 7 \\ 46.167 & 47.22 \\ 31.67 & 52.72 \\ 31.67 & 52.76 \\ 32.21 & 53.76 \\ 43.19 & 43.19 \\ 43.19 & 44.87 \\ 50.19 & 44.47 \\ 71.89 & 14.47 \\ 73.60 & 55.56 \\ 85.60 & 55.56 \\ 95.61 & 91.11 \\ 95.61 & 91.22 \\ 101.7 & 102.2 & 102.8 \\ 101.7 & 102.2 &$ 17.78

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64.64.11

75.56

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75.56

84.22

77.78

11.4.4

120.0

11.4.4

120.0 $\begin{array}{c}\n 43.83 \\
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114,4
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119,9
119,9 $\begin{array}{c} \begin{array}{c} 111.5 \\ 113.5 \end{array} \end{array}$ $\frac{1}{1}$ $\begin{array}{l} 61.11 \\ 64.67 \\ 72.22 \\ 77.75 \\ 83.89 \\ 94.44 \\ 94.44 \\ 101.0 \\ 105.6 \\ 111.7 \\ 114.7 \\ \end{array}$ $\begin{array}{c} \n 73.48 \\
 02.78 \\
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 105.0 \\
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 16.1\n \end{array}$ 50 to 199°C (1°C intervals) 5 7 7 ï \bullet ÷, 131.0
149.0
167.0
165.0
203.0 13223234232323 118.5
123.9
129.4
135.0
146.1
146.1 $\begin{array}{c} 125.6 \\ 131.1 \\ 136.7 \\ 142.2 \\ 147.8 \end{array}$ ---
254
260
270 $\begin{array}{c} 122.2 \\ 127.8 \\ 133.3 \\ 133.5 \\ 138.9 \\ 144.4 \end{array}$ $\begin{array}{c} 1.73.8 \\ 1.23.5 \\ 1.33.9 \end{array}$ $\begin{array}{c} \n 123.3 \\
 124.9 \\
 134.4\n \end{array}$ $\begin{array}{c} 124.4 \\ 130.0 \\ 135.6 \\ 141.1 \\ 146.7 \end{array}$ $\begin{array}{c} 1125.0 \\ 130.6 \\ 136.1 \\ 141.7 \\ 147.2 \end{array}$ $\begin{array}{c} 126 \\ 131 \\ 132. \\ \end{array}$ $\begin{array}{r} 213.6 \\ 231.8 \\ 231.8 \\ 249.8 \\ 267.8 \\ 265.8 \end{array}$ 215.6
233.6
251.6
269.6
287.6 $\begin{array}{c} 217.6 \\ 235.4 \\ 253.4 \\ 253.4 \\ 271.4 \end{array}$ oe.od $\frac{38.3}{43.9}$ 244
142
280 10) to 1870*F (16*F is 150
150
160
170
190 303.
321.
339.
357. $\overline{10}$ $\overline{\mathbf{v}}$ 40 \bullet 70 w m $\begin{array}{|l|l|l|l|l|} \hline 20 & 85.6 & 11.1 & 11.$ $\frac{1364}{210.0}$ $\begin{array}{|c|c|c|c|}\n\hline\n176.7 & 182.2 & 237.8 & 237.8 & 237.8 & 237.8 & 237.8 & 237.8 & 237.8 & 237.8 & 249.4 & 249.4 & 454.6 & 469.0 & 512.6 & 231.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.6 & 249.$ $\frac{1}{204}$ $\frac{193.3}{248.9}$ $\frac{1}{2}$ 1237.8

1293.9

1348.4

1440.0

515.6

515.6

516.7

521.7

521.7

73.8

73.9 $\begin{array}{c} 265.6 \\ 265.6 \\ 321.1 \\ 376.7 \end{array}$ 304.4
360 0
415.6
471.1
526.7 228.9
354.4
410.0
445.4
521.1
576.7
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632.2
637.4
743.3
798.9 310.0 100 to 990° C (10° Cincernis) $^{432.2}_{437.1}$ 10 30 40 90 \overline{a} 80 70 543.3
 598.3
 654.4
710.0 $\frac{3656}{621.1}$ $\begin{array}{r} \n 391.8 \\
 591.8 \\
 643.3 \\
 693.9 \\
 154.4 \\
 120.0 \\
 136.6 \\
 865.6 \\
 921.1 \\
 276.7\n\end{array}$ $\begin{array}{r} \n 212 \\
 392 \\
 512 \\
 712 \\
 112 \\
 1292 \\
 1472\n \end{array}$ $702\n
\n462\n
\n462\n
\n1022\n
\n1202\n
\n1202\n
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\n1244\n
\$ $\begin{array}{c}\n 230 \\
 410 \\
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 1100\n\end{array}$ 1235858 342828141329 2664162645 ||335 休憩||337 休憩| 3565716671675671256 748.9
BOL4
BOL4
BOL0
DIS.6
PIL1 760.0
815.6
571.3
926.7 $\begin{array}{l} \n 765.6 \\
 765.6 \\
 876.7 \\
 932.2 \\
 987.6\n \end{array}$ $\begin{array}{|c|c|c|c|}\n\hline\n1771.1 & 1776.7 \\
316.7 & 832.2 \\
1832.2 & 1887.6 \\
1937.9 & 1943.3 \\
\hline\n\end{array}$ 762.2 787.B $\begin{array}{c} 0.37 \\ 0.37 \\ 10.3 \\ 344.9 \end{array}$

224

 312

Units.

TABLE OF RELATIVE HUMIDITY

(For use with sling or Assmann type hygronolees)

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 314

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Nomogram for Calculating the Body Surface Area of Adults!

 $\label{eq:2} \frac{1}{2\sqrt{2\pi}}\sum_{i=1}^{N} \frac{1}{2\pi i} \int_{0}^{2\pi} \frac{1}{2\pi i} \left[\frac{1}{2\pi i} \frac{1}{2\pi i} \right] \frac{1}{2\pi i} \frac{1$