SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA

2004

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA

2004

Dr. T.Ramakrishna
Emeritus Professor of Physiology and formerly Head,
Department of Life Sciences,
University of Calicut, and
Visiting professor, Dept. of Biotechnology,
Bangalore University, Bangalore.

CERTIFICATE

This is to certify that the thesis entitled "**Some aspects of the physiology of temperature-induced stress in human volunteers with particular reference to cardiovascular system**" was conducted by Mr. Baburaj.T.P., in the department of Life Sciences and Defence Institute of Physiology and Allied Sciences under my guidance and supervision. It is hereby certified that the thesis submitted is a bonafide record of research done by the candidate in partial fulfilment of the requirements for Ph.D. Degree and that the thesis has not previously formed the basis for the award to the candidate of any degree, diploma, asssociateship, fellowship, or other similar title of any other university or society.

Calicut University | Dr.T.Ramakrishna
March, 2004.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

DECLARATION

This thesis entitled "**Some aspects of the physiology of temperature-induced stress in human volunteers with particular reference to cardiovascular system**" is being submitted by me to the University of Calicut in partial fulfilment of the requirements for the award of degree of Doctor of philosophy in the faculty of Science. The thesis is entirely the result of my work carried out in the department of Life Sciences and Defence Institute of Physiology and Allied Sciences under the guidance and the supervision of Dr. T.Ramakrishna, Emeritus Professor of Physiology and formerly head, Department of Life Sciences, University of Calicut. This thesis or any part thereof has not been submitted for any degree, diploma or associateship.

Calicut University Baburaj. T.P.

March, 2004.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

I dedicate this to my teachers

Prof. T. Ramakrishna

Prof. S. Vatsala

Prof. R P R. Nair

Mr. Eipunni

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

ACKNOWLEDGEMENTS

I am extremely fortunate to have worked under the supervision of Dr T.Ramakrishna., Emeritus Professor of Physiology and formerly head, Department of Life Sciences, University of Calicut. I extend my sincere and respectful gratitude for the same for his inspiration, guidance and invaluable suggestions and criticisms, with out which I would not have achieved this.

I would like to thank Dr. P.K Banerjee, The Director, Defence Institute of Physiology and Allied Sciences, for all his help rendered to complete this thesis.

I express my immense gratitude to DR. W. Selvamurthy, CCR&D, DRDO, Ministry of Defence, for his immense support and guidance, inspiration and for facilitating my expedition to Antarctica continent.

I express my sincere gratitude to Lt. Col.G. Himashree and Dr.Elavazhagan for their help and inspiration. I express my heartfelt gratitude to Mr. Gulab Singh and Mr. Bajaj for their association in experiments and all the help they rendered for the completion of thesis. I am highly indebted to Dr. Y.K Sharma for his support in statistical analysis of the data and moral support. I express my gratitude to all my colleagues in DIPAS who helped me in different capacities for the successful completion of this thesis.

I acknowledge the help of Dr. S. Vatsala with all my gratitude for the immense help in completing my thesis successfully.

I would like to thank Dr. Zuhara Prof. Microbiology, and head of Department of Life Sciences, University of Calicut, Kerala for her timely help for the completion of my thesis work. I am extremely grateful to Dr. V. K. Sasidharan. Professor of Biochemistry and formerly head of the Department of Life Sciences, University of Calicut, for his immense support for my thesis work. I extend my gratitude to teaching and non-teaching staff of Life Science Department.

My Family and my friends are my strength, I thank them.

Baburaj. T.P.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

CONTENTS.

DISCUSSION

Part- I

Study on cold stress

Part- II

Study on heat stress

SUMMARY AND CONCLUSIONS

REFERENCES

APPENDICES

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

D

ABBREVIATIONS

ALC	=	Air Lock Chamber.
ARM	=	Arm temperature
AVAs	=	arteriovenus anasthamosis
BRAH	=	Brahmi
BSA	=	Body surface area
CIVC	=	vasoconstriction
CIVD	=	vasodilatation
Clo	=	Unit for clothing insulation
DB	=	Dry Bulb
DOR	=	Dorsal temperature
ELECT	=	Electral
ERR V	=	Error Variance
EXE	=	Exercise
FH	=	Fore Head temperature
FING	=	Finger temperature
FOOT	=	Foot temperature
HCC	=	Human Climatic Chamber
HR	=	Heart rate
MBT	=	Mean Body Temperature
MC	=	Main Chamber
MI	=	Medical Inspection Room
NAN	=	Nanari
OT	=	Oral Temperature
RES	=	Rest
RH	=	Relative humidity
ROOH	=	Rooh Afza
S-LAT	=	South Latitude
SVT	=	supraventricular tachyarrhythmias
TOE	=	Big toe temperature
TS	=	Mean Skin Temperature
WBGT	=	Wet bulb globe temperature

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

Abstract

The study, "Some aspects of the physiology of temperature induced stress in human volunteers with particular reference to cardiovascular system" was conducted in two parts. Part-I, study on cold stress was carried out on six expedition members of Indian Antarctica Scientific (XVIII summer) expedition during December 1998to April 1999. Physiological variables like cardiac function, core temperature, mean skin and mean body temperature as well as peripheral temperature were studied during down and up sail and the stay in Antarctica. Suppression of T-Wave was observed when sojourners reached 70-S-latitude (Maitri, Indian permanent station at Schumacher oasis in Antarctica) due to the impact of cold. During the stay, elevation of ST-segment as well as appearance of J-wave (Osborne wave) were found in ECG of volunteers who were exposed to sub-zero temperature of Antarctica. It was an early repolarisation syndrome due to the cold and is reversible. The cold impact on heart was recovered during up sail from 40°-S-Latitude onwards. In the case of body temperature variables, significant fall in core, mean skin and mean body temperature was observed in 70-S-latitude, which improved during up sail form 40°-S-Latitude onwards and a full recovery observed towards the end of voyage. Digits temperature reached the lower level of tolerable limit. Part-II of the study was conducted on 5 volunteers in human climatic chamber simulated for DB 40°C, RH 60%, 34°WBGT. With out intake of fluid, exposure of 2% pre-hypohydrated volunteers showed significant increase in physiological variables viz. heart rate, core, mean skin and mean body temperature in progressive fashion during 60 min standard work heat test. ECG showed elevation of T-wave. The same volunteers during 1% replenishment fluid intake, showed much less rise in physiological variables such as body temperature and heart rate and a reduction in T-wave elevation.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

INTRODUCTION

INTRODUCTION

Part - I

Study on cold stress:

Humans, like all mammals, are homeotherms. Heat is both required and produced at the cellular level. The environment acts as either a heating or a cooling force on the body (David Jensen. 1924). The body must be able to generate heat, retain heat, and discharge heat depending on the body activity and ambient external temperature. Body temperature is a measure of the metabolism, i.e the general level of chemical activity within the body (Changnon et al 2000).

The hypothalamus is the major centre of the brain for regulating body temperature. It is sensitive to temperature changes in the blood of as little as 0.5 degrees Celsius and also reacts to nerve impulses received from nerve endings in the skin (Wansder 1981).

The optimum temperature for chemical reactions to take place in the body is 36.6°C (97.88°F). Above 40.5°C (105° F) many body enzymes become denatured and chemical reactions cannot take place leading to death. Below 36.6°C (97.88° F), chemical reactions slow down with various complications, which can lead to death (Burton 1935). Core temperature below 32 °C is lethal (Pozos et al 1982).

Hypothermia: Some general observations

It is defined as "a decrease in the core body temperature to a level at which normal muscular and cerebral functions are impaired" (Bittel et al.1987 and Mills et al. 1997). Some of the available studies had defined hypothermia more substantially (Peter et al 1999).

Hypothermia is lowering of the body's normal temperature. Significantly, hypothermia begins at body temperatures below 95 degrees Fahrenheit (35 degrees Celsius) (Mills et al 1997), and severe hypothermia occurs at temperatures below 90°F/32°C. All body functions are slowed in severe hypothermia, including heart rate, breathing rate, metabolism and mental activity (Petty Kevin et. al 1998). A victim of severe hypothermia may display a variety of different signs and symptoms. Symptoms like, Pulse (slow to none); Breathing (slow to none); Mental status (slurred speech, unresponsiveness to pain or verbal stimulus, staggering walk or unconsciousness); Cold skin; and Low rectal temperature. Severely hypothermic condition may have other problems that are not easily detected by rescuers, but which may affect the patient's survival (Pozos et. al. 1982).

According to the Backpackers's field manual (1998), the impact of hypothermia extends deep into tissue and cellular level viz. Changes in blood chemistry; Changes in oxygen and carbon dioxide content of the blood; Irregular heart beats; Dehydration; Differences in temperature between deep body tissues and superficial body tissues.

The book 'Medicine for Mountaineering' (1998) describes, Hypothermia as the decrease in the core body temperature to a level at which normal muscular and cerebral functions are getting impaired. Earlier studies reported that the conditions leading to hypothermia (Paul et al 1983) are cold temperatures, Improper clothing and equipment, Wetness, Fatigue, exhaustion, Dehydration, Poor food intake, carelessness or unawareness of hypothermia.

Mild Hypothermia:

It is reflected in the core temperature between 37.2° and 36.11 °C [99° - 97°F] Normal, shivering can begin. If the temperature falls down further to 36.11° - 35°C (97° - 95°F), there starts cold sensation, goose bumps, and inability to perform complex tasks with hands. Shivering can be mild to severe and the hands may become numb (Mills 1956 and Bittel et al 1998).

Moderate Hypothermia:

In mild hypothermia core temperature may be in the range of 35° to 33.89°C (95° - 93°F). This causes intense shivering, muscle incoordination becomes apparent, with slow and laboured movements, stumbling pace, and mild confusion. This low temperature perpetually leads to dazed consciousness, Loss of fine motor coordination - particularly in hands due to restricted peripheral blood flow, slurred speech, violent shivering, irrational behaviour like paradoxical undressing, person starts to take off clothing, unaware that s/he is cold as well as "I don't care attitude" called flattened affect (Petty 1998). If the temperature goes down further to a range of i.e.33.89° and 32.22°C (93°-90°F) there is a persistence of violent shivering, difficulty in speaking, sluggish thinking and amnesia (Peter et. al. 1999, and Gisbercht et al. 1994, 1997, 1998)

Severe hypothermia:

In severe hypothermia, core temperature may be in the range of 32.22° to 30°C (90° - 86°F) leading to cessation of shivering, puffy skin, poor muscular coordination, inability to walk, confusion, incoherent/ irrational behavior Danzl et al. 1995). Further decrease in temperature to 30° to 27.78°C (86° - 82°F), leads to muscle rigidity, stupor, loss of awareness of others, decrease in pulse and respiration rate and possible heart fibrillation (James et al.1992). The moment body temperature reaches 27.78°-25.57°C (82°-78°F) person becomes unconscious, heartbeat and respiration become erratic, pulse may not be palpable. The pupils turn out to be fixed and dilated (Danzl 1998). Person falls to the ground, can not walk, curls up into a foetal position to conserve heat. Muscle rigidity develops because of reduction in peripheral blood flow and accumulation of lactic acid and CO_2 in the muscles. At the level of 25.57° to 23.89°C (78° - 75°F) core temperature, the victim may have pulmonary oedema. Cardiac and respiratory failure and eventually death may follow. Death may occur before this temperature is reached (Paul et al. 1983).

Cold Injuries:

Tissue temperature in cold weather is regulated by two factors, the external temperature and the internal heat flow (Hardy 1972). All cold injuries described below are intimately connected with the degree of peripheral circulation. As peripheral circulation is reduced to prevent heat loss to the core these conditions are more likely to occur (Budd et al. 1973).

Factors influencing cold injuries:

Teichner (1957), Van Someran et al. (1982) and Yoshimura (1952) conducted studies on cold injuries. They gave the possible reasons for the cold injury mostly during the sojourns in polar expedition (Edlich 1986). Pathological tissue freezing is another worst situation the Antarctica sojourners often come across (Pozos et al 1982 and Loussie 1983).

Pathophysiology of Tissue Freezing:

As tissue begins to freeze, ice crystals are formed within the cells. As intracellular fluids freeze, extracellular fluid enters the cell and there is an increase in the levels of extracellular salts due to the water transfer (Pozos et al. 1982). Cells may rupture due to the increased water and/or from tearing by the ice crystals. As the ice melts there is an influx of salts into the tissue there by damaging the cell membranes. Cell destruction results in tissue death and loss of tissue.

Distal areas of the body and areas with a high surface to volume ratio are the most susceptible e.g. ears, nose, fingers and toes. Surface frostbite generally involves destruction of skin layers resulting in blistering and minor tissue loss. Blisters are formed from the cellular fluid released when cells rupture. Deep frostbite can involve muscle and bone (Edlich et al. 1986). As defined by Edlich et al. (1986), Mackworth et al. (1953), Miller (1974), and Yoshimura et al. (1950) different degrees of cold injuries are as follows.

Cold Response:

a) Circulation is reduced to the area to prevent heat loss, b) The area may be pale, cold and c) It may have sensation or be numb (Danzl 1998 and The Backpacker's field manual 1998).

Frostnip

a) Freezing of top layers of skin tissue is involved b) It is generally reversible. White, waxy skin may appear, top layer feels hard, rubbery but deeper tissue is still soft, c) Numbness and d) Most typically seen on cheeks, earlobes, fingers, and toes (Tanaka 1971).

Frostbite

Superficial frostbite includes all layers of skin and it appears as white and "wooden", Deep frostbite can include freezing of muscle and/or bone, it is very difficult to rewarm the appendage without some damage (Edlich et al. 1986).

Trench Foot - Immersion Foot

It is caused by prolonged exposure of the feet to cool, wet conditions. This can occur at temperatures as high as 60°F (15°C) if the feet are constantly wet (Le Blance et al 1960 and 1975).

A similar phenomenon can occur when hands are kept wet for long periods of time such as kayaking with wet gloves or porgies. The damage to the circulatory system is known as Reynaud's Phenomenon. (Edlich et al. 1986)

Chilblains

This is caused by repeated exposure of bare skin to temperatures below 60°F (15.56°C). Redness and itching of the affected area particularly found on cheeks and ears, fingers and toes are the symptoms. The cold exposure causes damage to the peripheral capillary beds which is not permanent.

Snow-blindness

Snow-blindness is the Sunburn of the eyes. Snow blindness can even occur during a snowstorm if the cloud cover is thin. The symptoms which occur 8-12 hours after exposure are as follows; eyes feel dry and irritated, then feel as if they are full of sand. It is extremely painful, exposure to light hurts the eyes, eyelids may swell, and excessive tearing may be seen (Weinberg et al. 1990).

Cardiovascular system:

The cardiovascular system is initially stimulated by cold, resulting in peripheral vasoconstriction, tachycardia, and elevated blood pressure (BP). As hypothermia progresses, the myocardium is depressed, causing hypotension and progressive sinus bradycardia. Severe hypothermia can lower BP and heart rate to barely detectable levels, which in rare cases leads to an erroneous pronouncement of death. Various cardiac arrhythmias may be precipitated by cold temperatures, including atrial fibrillation and flutter, premature ventricular beats, and atrio-ventricular rhythm. Cardiac arrest, from either ventricular fibrillation or asystole, is increasingly likely as body temperature falls below 30°C (86° F).

Hypothermia causes a number of disorders to cardiac function. Most noted cardiac malfunctions are related to electrocardiogram (ECG)(Kolb 1977). Electrocardiographic changes in hypothermia include a) Sinus bradycardia, b) Junctional rhythm, c) Atrial fibrillation with slow ventricular response, d) prolonged PR, QRS, and QT intervals, e) Osborne wave (J-wave) and f) T-wave inversions (Gussak et al.1995, 1999 and Kalla et al. 2000).

When death occurs, cardiac arrest or ventricular fibrillation is usually the cause. The body temperature at which each cardiac event appears varies, but at temperatures less than 29.4°C (< 85° F), risk of death is high, particularly in patients with latent heart disease.

Resuscitation during warming should be aggressive and prolonged in patients who are profoundly hypothermic; unexpected recoveries have been reported in such cases.

Some clinical studies (Antzelevitch et. Al. 1998) were carried out by Yan et al. [1996] and Southwick et al. (1980) who reported that the initial response to hypothermia is tachycardia with vasoconstriction. As hypothermia continues, then the patient becomes bradycardic, with a pulse decrease of 50% by the time core body temperature reaches 28°C. This bradycardia results from decreased spontaneous depolarization of the Purkinje cells, which is unresponsive to atropine (Kalla et al 2000).

J- wave (Osborne wave)

Janssens et al (1998) reported that one-third of hypothermic patients will exhibit a J wave (Osborne wave), manifested by a slow positive deflection at the end of the QRS segment. It should be noted however that J waves can be seen in other conditions such as myocardial ischemia and cerebral injuries and are not diagnostic of hypothermia.

Stroke caregivers should be familiar with the ECG J-wave for two reasons. First, in the clinical setting of severe accidental hypothermia with impairment of consciousness, the J wave indicates a risk of life-threatening dysrhythmias.

COLD STRESS IN ANTARCTICA

Untamed icy wilderness of Antarctica is a physiological challenge to sojourners in the light of above mentioned effects of cold stress. Comfort is achieved in Antarctica by balancing heat-loss and heat gain. One of the ways it can be achieved is by selecting proper clothing, which in turn helps to balance, the heat gained from exercise and sunlight against the heat loss to the cold wind, sky and terrain. But, if the required clothing-adjustments is inadequate or delayed, man becomes too hot or too cold for comfort (Budd et al 1966, Wyndham et al. 1969). Frequent stimulation by cold and warm would affect the underlying vascular system. Most outdoor occupations in Antarctica require greater expenditure of energy than their counterparts in tropical or temperate Zone. This is the result of the nature of terrain underfoot, wind resistance, and bulky heavy clothing (Bolte et al. 1998).

Studies on the tropical population in terms of physiological coping to Antarctica's sub-zero temperature are sporadic. To understand the heat exchange process of tropical men in Antarctica with respect to special clothing in subzero climate is of immense interest. By nature, tropical populations are exposed to warm climate because of the equatorial proximity, and are adapted to either hot humid (coastal area climate) or hot dry conditions (arid or semi arid climate). In Indian subcontinent this is true except Himalayan regions. Tropical population possesses a well-tuned homeostasis with the prevailing warm climate; it will obviously induce physiological problems with sub-zero climatic situations of the polar regions. A pilot study had been conducted in Antarctica by Nair (1988) during Dakshin Gongotri expedition. His study showed that there was an inadequacy in personal protective clothing to provide protection against the sub-zero climate. His study did not include cardiac function of sojourners exposed to cold.

Aim and objective:

From the available reports, it is difficult to derive a proper assessment regarding the cardiac function due to cold stress and the features of adaptation. Therefore the present study is proposed with a view to understand the physiology of tropical populations in terms of cold stress on body temperature due to outdoor sub-zero climate with particular reference to cardio vascular system.

Part - II

Study on heat stress:

Heat stress is a very strong limiting factor for human performance. This is the major problem encountered by athletes and soldiers as well as civilian population (Adolph 1947). Outdoor operations in hot weather, including surface mining, roofing, constructions, Farming operations, Iron, steel and nonferrous foundries, etc are posing problems of high temperatures and humidity.

Workers who are suddenly exposed to working in a hot environment face additional and generally unavoidable hazards to their safety and health (Barcroft et al 1943).

Hyperthermia: some general observations

Heat stroke:

The most serious health problem for workers in hot environments, is caused by the failure of the body's internal mechanism to regulate its core temperature. Sweating stops and the body can no longer rid itself of excess heat (Beller et al 1975) Signs include (1) mental confusion, delirium, loss of consciousness; convulsions or coma; (2) a body temperature of 106 degrees F or higher; and (3) hot dry skin which may be red, mottled, or bluish (Bouchama et al. 1991, 1997 and Al Mashhadani 1994).

Heat exhaustion:

It results from loss of fluid through sweating when a worker has failed to drink enough fluids or take in enough salt or both. The worker with heat exhaustion still sweats but experiences extreme weakness or fatigue, giddiness, nausea, or headache. The skin is clammy and moist, the complexion pale or flushed, and the body temperature normal or slightly higher (Chao et al 1981)

Heat cramps:

Painful spasms of the muscles, are caused when workers drink large quantities of water but fail to replace their bodies' salt loss. Tired muscles, those used for performing the work - are usually the ones most susceptible to cramps. Cramps may occur during or after working hours (Cade et al 1972).

Fainting: (heat syncope)

This may be a problem for the worker unacclimatized to a hot environment who simply stands still in the heat. Victims usually recover quickly after a brief period of lying down (Clark et al. 1985).

Heat rash:

It is also known as prickly heat, may occur in hot and humid environments where sweat is not easily removed from the surface of the skin by evaporation. When extensive or complicated by infection, heat rash can be so uncomfortable that it inhibits sleep and impedes a worker's performance or even results in temporary total disability (Clark et al. 1985).

Heat balance in the body:

The human body, being warm blooded, maintains a fairly constant internal temperature, even though it is being exposed to varying environmental temperatures. To keep internal body temperatures within safe limits, the body must get rid of its excess heat, primarily through varying the rate and amount of blood circulation through the skin and the release of fluid onto the skin by the sweat glands (Clark et al 1985). These autonomic responses usually occur when the temperature of the blood exceeds 30.6°C (97.88°F) and are kept in balance and controlled by the brain (Eshel et al 2002). In this process of lowering internal body temperature, the heart begins to pump more blood, blood vessels expand to accommodate the increased flow, and the microscopic blood vessels (capillaries) which thread through the upper layers of the skin begin to fill with blood (Armstrong et al. 1997). The blood circulates closer to the surface of the skin, and the excess heat is lost to the cooler environment. If heat loss from increased blood circulation through the skin is not adequate, the brain continues to sense overheating and signals the sweat glands in the skin to shed large quantities of sweat (Harrison 1985).

The human beings are the most prolific sweaters in the entire animal kingdom. Sweating is accomplished through specialized eccrine sweat glands. These glands are found in the dermis and epidermis, distributed all over the body, except for the margins of the limbs, sex organs, and ear drums. They average between 150 and 340 glands per square centimeter of skin for a total between 2,000,000 and 5,000,000. The sweat glands are innervated by the sympathetic nervous system. This sweating process is effective refrigeration (heat dissipitatory process) system of the body to protect from heat illness (Kalksten 2000).

Cardiovascular system:

Work under a heat stress condition sets up a competition for cardiac output, particularly as the blood vessels in the skin dilate to their maximum and less blood is returned to the central circulation. Gradually, less blood is available in the venous return to fully fill the heart between beats, causing the stroke volume to decreases and therefore heart rate must increase to maintain the same cardiac out put (Kellerman et al 1996).

If the intensity of work results in a heart rate in excess of these values, the intensity of work should be reduced. Thus heat added to the demands of work rapidly results in problems even in the healthy, young workforce (Knochel et al 1990, 1994). These problems are amplified if circulation of blood volume is reduced as a result of inadequate water-intake to replace sweat loss.

The crisis point, i.e heat exhaustion and collapse, is manifestation of the inadequate blood supply to the brain; this occurs when cardiac out put becomes inadequate, because of insufficient return of blood from the periphery to fill the heart for each beat, because of inadequate time between beats to fill the heart (Lithhead et al 1982).

ECG:

Studies on ECG under heat stress are very few. Supraventricular tachyarrhythmias (SVT) had been reported in hyperthermia cases (Massett et al 1996).

Ventricular fibrillation:

This is a chaotic ventricular rhythm that rapidly results in death. It is often precipitated by a critically timed extra systole, that occurs during the relative refractory period of the myocardial fibres. Conventional wisdom has it that this results in chaotic, un-coordinated wavelets of depolarisation moving through the ventricular mass (Zahger et al 1982).

Ventricular flutter:

Ventricular 'flutter' is a bizarre sine-wave like rhythm, and usually degenerates into ventricular fibrillation. Elevation of T-wave is the prominent symptom observed during heat exposure (Schafler et al 2002).

Cardiovascular adaptation:

The normal cardiovascular adaptation to severe heat stress is an increase in cardiac output by up to 20 liters per minute and a shift of heated blood from the core circulation to the peripheral circulation. An inability to increase cardiac output because of salt and water depletion, cardiovascular disease, or a medication that interferes with cardiac function can impair heat tolerance and result in increased susceptibility to heat stroke (Rowell et al 1974).

Drinking Water:

In the course of a day's work in the heat, a worker may produce as much as 2 to 3 gallons of sweat. Because so many heat disorders involve excessive dehydration of the body, it is essential that water intake during the workday be about equal to the amount of sweat produced. Most workers exposed to hot conditions drink less fluids than needed because of an insufficient thirst drive. Therefore, accrual demand of water intake is 5 to 7 ounces of fluids every 15 to 20 minutes to replenish the necessary fluids in the body (Nelsen et al. 1947).

Dehydration: (Hypohydration)

Nadel et al (1980, 1990) reported that as the sweat rate increases, body water-loss increases, and the need for replacement fluids becomes crucial. Without adequate fluid replacement during exercise, the body's ability to dissipate heat is compromised. Hydration status prior to exercise is equally as important in avoiding dehydration. If properly hydrated at the start, the effectiveness of fluid replacement while exercising is increased. As dehydration occurs, the body experiences a decrease in the plasma volume of the blood. As plasma volume decreases, the body's ability to lose heat is compromised (Noakes 1993).

A critical deficit of 1 percent of body weight elevates core temperature during exercise. As the magnitude of water deficit increases, there is a concomitant graded elevation of core temperature during exercise under heat stress. The magnitude of elevation of core temperature ranges from 0.10 to 0.23 *f*C (Cardiac frequency) for every percent body weight loss, and this elevation is greater during exercise in hot than in temperate climates. As little as a 2% loss of body mass from fluid loss will impair exercise performance (Ohara et al 1996).

Hypohydration not only elevates core temperature response, but it also negates the core temperature advantages conferred by high-aerobic fitness and heat acclimation. Therefore, heat-acclimated persons (who have increased sweating rates) who do not drink adequately may more rapidly experience the adverse effects of hypohydration than their non-acclimated counterparts (Regan et al 1996).

In hot and dry environments there is loss of body water resulting in a reduction of blood volume and impaired thermoregulatory function (Harrison 1985). Replacement of fluids lost during exercise in hot climates is important for work performance. However, recommendations concerning replacement of body fluids are often complicated by paucity of knowledge regarding the ideal timing, volume and composition of the drinks to be used (Gisolti et al. 1992).

Lyons and colleagues (1990) reported that glycerol and water hyperhydration had some effects in improving a person's ability for thermoregulation during exercise-heat stress. They found that the rectal temperature-rise was attenuated as well as sweating rate was elevated above control levels. Burgess (1991) and Alan (1999) had reported similar core temperature and sweating rates in fluid replenishment study with glycerol and water euhydration before exercise in warm climate. These studies did not confirm the thermoregulatory benefits of these fluids during exercise under heat stress (Goldfinch et al. 1988, Goldfinch et al. 1998)

Mugham et al (1994) conducted a study on whole body water and electrolyte balance. Coyle et al. (1986) and McCoell (1994) have shown that ingesting carbohydrate drinks during prolonged exercise can delay fatigue. The aim of the study was to test the hypothesis that fluid intake maintained at regular intervals during exercise in heat, delays fatigue (as measured by perceived exertion) and decreases the need for rehydration after exercise. But the study gives less insight into ergogenic support during endurance in heat. Cade et al. (1972) carried out a study to determine the need for fluid replacement during and after exercise in heat. The results of his studies show large differences in the rating of perceived exertion at 2 and 3 hrs of exercise between the groups exercising with no fluid intake and gap-up fluid intake (Mudambo 1997).

From the available reports it can be seen that, 1)during exercise-heat stress, thermoregulatory responses were identical regardless of whether volunteers were euhydrated, water hyperhydrated or glycerol hyperhydrated. 2) Glycerol hyperhydration provided hydrational advantage over water hyperhydration during exercise heat stress because both hyperhydration approaches increased total body water (TBW) by similar amounts. 3) Pre-hyperhydration delayed the development of body water deficits, if fluids were not replaced during exercise-heat stress (Dokkum et al 1996). These studies concluded that hyperhydration provides no meaningful advantages over the maintenance of euhydration during compensable exercise heat stress. In a nutshell, understanding of heat dissipatory mechanism of man and suggesting remedial measures to handle the problems of heat in tropical population is of immense interest. The Indian sub continent has a wide range of seasonal variations of hot humid and hot dry climate. Though the tropical population have enhanced heat tolerance capacity, heat dissipatory process is not satisfactory during extreme heat. Topographically nearly half of the Indian subcontinent is arid or semiarid. "Thar desert" is one of the most populated deserts in the world. In hot summer, increased aridity causes the atmospheric temperature to rise as high as 50°C and the surface temperature of sand reaches as high as 70°C.

Heat-physiological study in India with respect to hypohydration is imperative. The existing ethnic wisdom on replenishment studies requires the consumption of herbal fluids as cooling agents during the summer. Some of these herbal formulations are presumed to be effective cooling agents. Furthermore these herbal drinks are believed to be harmless compared to artificial formulation. No scientific study however has been reported so far regarding the efficacy of herbal replenishment fluids except a pilot study conducted by Pichan et al. (1996).

Aim and objective:

To understand the impact of heat stress on tropical men with special reference to cardiac function and to identify ergogenic replenishment drinks, which would be of use for civilian and military population who work in extreme thermal stress conditions.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

MATERIALS AND METHODS

MATERIALS AND METHODS:

Part-I

Study on cold stress

Antarctica is the coldest continent on earth. It has the coldest average annul temperature and the lowest temperature ever recorded was -89°C at Vostok Mountains in Antarctica.

Low temperature-induced stress study was carried out on team members of XVIII Indian Antarctica Scientific Expedition (IASC). This Scientific expedition was a package of journey, such as sail off from Goa in the month of December, stay back in the continent during Antarctica summer and sail back (up-sail at the beginning of winter and return to Goa by April.

Voyage:

The expedition started from 11 degree latitude on the northern hemisphere, i.e. Goa harbor of Indian sub-continent. The voyage crossed equatorial line and proceeded further down till south to 70° latitude of southern hemisphere (Fig.1).

During the initial days of voyage from Goa, some of the sojourners showed sea sickness but it was cured soon. The initial data collection was carried out while the ship was approaching zero degree Latitude and data collection completed when ship reached the equatorial line. The sailing of the ship was comfortable till 40-degree latitude. This is the region, where the temperature started falling down. When the ship reached

40° latitude, temperature started declining well below 10°C. Apart from the lowering of temperature, the ocean became furious and tempestuous waves did splash over monkey deck. The southern ocean is prone to have strong whirling winds and it creates low-pressure systems or depressions. The ship was troubled with "Pitching and rolling". This is the three-dimensional vigorous tilting movement of ship as if it was a horrifying cradle. The chances to capsize existed between 40 and 50-degree latitude but averted. This situation disturbed physiological data collection in this region during down sail (40 Degree Latitude). At 50 degree Latitude, the sojourn encountered the iceberg along with the decline of ambient temperature (fig. 2). At 60-degree latitude, freezing is rapid in May and June, and ocean ice edge advances northwards by as much as four km a day. There after the ship came across pack ice, the remnant of previous year's seasonal ice (fig. 3). The next kind of ocean ice encountered by the ship was "fast ice", i.e. Fast-ice was being spread over kilometers, with a few meters thickness over the ocean surface and it appeared as if it was white solid carpet. Here the ambient temperature was observed to be of zero degree centigrade in summer, but in winter it will be well below 0°C. In this region, ship waited for days till the fast ice was cleared (By breaking into peaces due to ocean movement and wind) before proceeding to Antarctica.

The whole voyage took almost one month, starting on 14^{th} December, and reaching the Antarctica territory by 11^{th} January. Ship waited a couple of days for the fast ice to clear, before reaching Antarctica polynia (fig 4).

Polynia was ice-free area of ocean before reaching Antarctica shelf-ice. The mooring of the ship was done at 70°-S-Lattitude and 5° longitude. This place is called Indian bay (fig 5). The ship was moored on 15th January and the explorers started moving to Indian permanent station 'Maitri' by 17th of January. There is climatological paradox as northern hemisphere goes through winter when the southern hemisphere experiences summer. The summer of Antarctica was characterized by sub-zero temperature.

Wind, cold, snow, ice and cloud make Antarctica's one of the world's most treacherous climates (fig. 6). Snow and wind produce blizzards, which can bring all human activity to a halt, often for several days at a stretch. The entire atmosphere seems to be full of whirling snow, which winds over 60 km/h and visibility often reduced to less than 10 meters. Whiteout was almost as dangerous. This was caused by a combination of snow and cloud. On a uniformly white-snow surface, it was impossible to judge height or distance, so that walkers stumble and aircraft and even birds can crash into the snow.

Wind chill.

Cold and wind combined were worse than either of the two by itself, because of the wind chill effect. The discomfort that one encounters in the cold depends on the rate at which our bodies lose heat to the air around us. This in turn depends on how cold that air actually is, and how rapidly it is moving, taking our body heat away. The stronger the wind, the more quickly we lose heat, and the colder we feel. For example, in a 10 km/hr wind at

-10°C, we lose heat quickly as we would at -70°C in still air. The corresponding wind chill index is 1200.

The physiological data collection was conducted during up sail also. During up sail physiological data collection was conducted at different latitudes viz. 40, 20, and Zero degree latitude. During up sail, the region at 40-degree latitude was relatively stable and ship did not suffer much pitching and rolling.

"Maitri" is the permanent station of India on Antarctica continent (fig.7). This is located at Schumacher Oasis, an ice-free terrain having a few kilometers width and 10 km length. This oasis is bounded by permanent polar ice (Glacier) at the southern side and shelf ice at the northern ocean side.

Volunteers

The volunteers of the study were briefed about the experiment and protocol before the commencement of experiment. They were told about possible hypothermic consequences and were motivated positively for the successful completion of the experiment. Six volunteers from the sojourners participated in the complete package of experiment, with the written consent, obtained prior to the commencement of the journey.

Five healthy, non-smoking male volunteers and a female volunteer with the following characteristics (Table-1) participated in all sessions of the study.

Body surface area (BSA) was calculated by using the formula of DuBois (1916).

TABLE-1:

Physical characteristics of volunteers who participated in the study on cold stress

Volunteer	AGE Years	WEIGHT kg	HEIGHT cm	B S A. Sq. m
YKM [M]	38	53.00	161	1.50
RKD [M]	32	80.20	175	1.95
AR [M]	39	64.10	159	1.65
PK [M]	45	55.40	163	1.60
RTK [M]	42	52.30	169	1.58
KK [F]	43	57.24	160	1.70
Mean	**39.8**	**60.40**	**164.5**	**1.66**
SD ±	**4.62**	**10.59**	**6.25**	**0.15**

Protocol

The protocol of the study was designed as per the expedition package and it was divided into six phases:

Phase I: basal readings were collected of the volunteers from tropical zone before the ship crossed zero degree latitude during the down sail to Antarctica.

Phase II: readings were obtained during the stay in Antarctica (70-S-Latitude).

Phase III: Data collection was done during up-sail in the following manner.

a) Data collection while crossing 40° latitude.

b) Data collection while crossing 20° zone.

c) Data collection at equatorial zone. [0° latitude]

Physiological variables recorded.

A) Core temperature (oral temperature),

B) Mean skin temperature (Hardy - Dubois method 1916): Weightage given is as follows:

1) 0.07 head,
2) 0.14 arm,
3) 0.05 hand,
4) 0.18 chest,
5) 0.17 back,
6) 0.19 thigh,
7) 0.13 leg,
8) 0.07 foot.

Body temperatures were recorded by YSI -400 telethermometer and leads.

C) Heart rate. (BPL defibrillator with lead system II)

D) Sweat loss. [Human weighing balance ± 20gm]

In phase-I, 20 volunteers participated. Volunteers were provided light breakfast, after which they took rest for 30 min, before reporting at Medical Inspection (MI) room of the ship. The MI room of the ship was maintained at thermo-neutral temperature (25°C and 50% relative humidity). Volunteers were asked to empty their bladder before the commencement of experiment. Afterwards, volunteers were instrumented with probes, for temperature-recording. Mean skin temperature was recorded as per Dubois method, from 8 sites. Temperature of digits (forehead, finger-tip and big toe) was also recorded. The Core temperature was sublingually (oral) recorded (fig.8).

ECG-surface electrodes were placed over torso according to Lead II system. Duration of exposure in MI room (thermo-neutral condition 25°C ± 1) was 20 minutes. Zero and 20 min. physiological variables were recorded. Male volunteers wore shorts and female volunteers wore loose thin vests during exposure. Prior and immediately after the exposure, nude body weight was recorded to compute the sweat rate.

In phase-II 6 volunteers participated as shown in table-I. Data collection in phase-II was done towards the last lap of their stay (after 40 days of their arrival) in Antarctica. This session of the study was conducted in the premises of "Maitri" station at Schumacher Oasis, at 70°-S-latitude and 5° longitude. The field laboratory was established in Aravali summer hut (Fig.9), 500 meters away from 'Maitri station'. Temperature in Aravali summer hut was observed to be sub-zero through out the period of polar summer.

Volunteers reported at Maitri station around 9 am after their breakfast. A rest of 20 min. in the thermoneutral MI (Medical Inspection) room of Maitri station was allowed prior to the experiment. At the end of rest period they evacuated their bladder and moved to field lab. Subsequently, Initial nude body weight was recorded and the volunteers were instrumented with YSI thermister probes as well as ECG surface electrodes (as mentioned in previous phase). Thereafter, they had worn the polar-clothing ensemble for experimental exposure. Over the Inner layer (polar underwear) designed and produced by DMSRDE (Defense Materials and Store Research and Development Establishment) Kanpur (details were given in appendix -III) and outer layer of clothing (over the out fits) they were instrumented with YSI surface electrodes according to Ramanathan formula (1961). The experimental set-up of phase-II was designed for 60-minutes outdoor exposure at the prevailing climate (wind chill index of 1000 $\pm$ 200) of Schumacher Oasis of Antarctica Continent. During the period of experiment, volunteers were instructed to sit on a chair against the wind and physiological variables were recorded at every 10 minutes interval (Fig.10). Data were recorded with respect to heart rate, core temperature; mean skin temperature, temperature from digits (finger tip & big toe) as well as over the clothing layers. Immediately after the completion of out door exposure once again nude body weight was taken to compute the sweat rate. Thereafter, volunteers were called back to MI room of Maitri (DB 25°C $\pm$ 1 and RH 60 %) where ECG was recorded with 12 lead system.

All exposures were scheduled at the same time (morning sessions) of the day to avoid circadian clashes. Care was taken to exclude people having cardio-respiratory problem as well as high blood pressure. The experimental sessions of female volunteer was avoided during the menstrual period. During experiment precautions were taken to protect the volunteers against hyperthermia.

After experiment, volunteers were provided by questionnaire of "Bedford comfort vote" (Revolier 1987) to collect subjective feeling regarding cold stress [appendix-II].

Data collection during up-sail.

Phase -III was meant for data collection during up sail. The mode of data collection during up-sail was carried out in the same manner as the down sail. The data were collected at 40°, 20°, and 0° Latitude.

Part II

Study on heat stress:

Heat stress studies were conducted in human climatic chamber [HCC], located in Defense Institute of Physiology and Allied Sciences [DIPAS], in Delhi.

Human climatic chamber [HCC]

General description:

The human climatic chamber is a walk-in simulator to study the effects on human beings of various environmental stresses such as hot dry, hot humid climatic condition along with the air velocity (fig. 11).

Technical data:

HCC consists of Main chamber and Airlock chamber.

Main chamber [MC]

Temperature range 5°C to 60°C

Relative humidity 10% to 95% RH.

Radiation panel to provide mean radiation temperature of maximum at 150°C

One fresh air change per hour.

Dimension: L 6Mtrs x W 3.2 Mtrs x H 3 Mtrs

8 persons can be accommodated

Air lock chamber [ALC]

Temperature range 5°C to 60°C

Relative humidity 10% to95% RH.

Radiation panel to provide mean radiation temperature of maximum at 150°C

Dimension: L 1.2Mtrs x W 3.2 Mtrs x H 3 Mtrs

Dew point of the refrigeration system : +10°C.

Physiological data-recording facility:

YSI telethermometer to record temperature

BPL Cardiac monitor

Human weighing balance.

Psychrometer (to measure wet bulb and dry bulb)

Bicycle ergometr. [Monark 824E and Vinky make]

BioPack Data Acquisition system (MP-100) to record physiological variables like body temperatures and ECG.

Volunteers.

The study was conducted on tropical volunteers residing in Delhi. The volunteers were screened to ensure that they were clinically normal and physiologically fit for the experiment. The mean physical standards of the volunteers are given in table-2. They were briefed about the experiment and the precautions to be taken before the commencement of actual experiment. Written consent was obtained from all volunteers.

Preparation of volunteers for the experiment.

Heat-physiological studies were conducted in the laboratory on volunteers who had undergone initial acclimation. This was meant to bring about a homogeneous physiological base line among all volunteers prior to participating in the experiment as well as to avert causalities of the heat exposure.

Acclimation:

Acclimation is specific to the exposure-temperature of the protocol. Here, temperature had been simulated for hot humid, DB 40°C and RH 60% (34°C WBGT). Acclimation was scheduled as noted below. Duration of exposure was 110 minutes. Initial 50-minute light exercise in simulated HCC was followed by 10 min. rest. After the rest the second half of light exercise was continued for 50 min. Water was provided during the 10 min rest. Eight days of consecutive exposure during morning session was intended to acclimatize volunteers. A day's rest was given to volunteers before actual experiment.

Protocol of the experiment (heat exposure).

The entire experiment was carried out in three sessions.

Session-I	Pre-hypohydration,
Session-II	Replenishment-fluid treatment
Session - III	Standard work heat test.

TABLE-2:

Physical characteristics of volunteers who participated in the study on heat stress

Volunteer	AGE Years	WEIGHT kg	HEIGHT cm	B S A. Sq. m
R T [M]	31	68.44	162	1.7
R K [M]	21	67.9	172	1.8
Y B [M]	22	63.08	164	1.68
N B [M]	22	70.52	166	1.78
RJN [M]	24	59.58	169	1.68
Mean	**24**	**65.90**	**166.6**	**1.728**
STDV ±	**4.06**	**4.46**	**3.97**	**0.057**

Session - 1 (Pre-hypohydration).

Pre-exercise session was gone through in simulated HCC (hot humid, 34°C WBGT) till the body weight reached 2% body weight deficit. The approximate duration of this session was two hours. During this period of exposure the volunteers were weighed intermittently to assess the body weight deficit. At the end of this session volunteers were evacuated to thermoneutral room from the simulated HCC (hot humid, 34°C WBGT).

Session -II. (Replenishment fluid-treatment):

In this session volunteers were instructed to take rest in thermoneutral room. The rest period would terminate while the body attains physiological baseline in terms of heart rate, core temperature and BP. During this time partial replenishment with experimental fluids was carried out. 1% residual-hypohydration was ensured during partial replenishment regimen.

Replenishment fluids used for the study:

1. **Rooh-afza formulation.**

Each dose of 50 ml. (70 gms. approx) contains;

1. Inverted sugar base.............. 40.ml
2. Pineapple juice.............. 4.0 ml

Distilled extract of :-

1. Coriandrum sativum (dhania)
2. Daucus carota (Gajar)
3. Portulace olerancea (Khurfa)
4. Citrullus vulgaris (Tarbooz)
5. Spinacia oleracea (Palak)
6. Luffa clindrica (Hara Ghia)
7. Circhorium intybus (Kasni)
8. Vitis vinifera (Manaqqa)
9. Santalum album(Sandle sufed) [items1 to 9 of 2.25 ml]

10.	Vetiveria Zizanides (Khas Hind)	
11.	Parmelia periate (Chharrhila)	
12.	Nymphaea alba (Gul Nilofar)	
13.	Onosma bracteratum (Barge gaozaban)	
14.	Menta arvensis (pudina)	
15.	Distillate of Keora…………	1.75 ml
16.	Orange juice……….	1.0 ml
17.	Distillate of citrus medica… .	0.4 ml
18.	Distillate of rose damascena…	0.3 ml
19.	Permitted food colour…	qs

Dilution was made to 1:9 with mineral water (Bisleri).

2. Nanari preparation:

1. Hemidesmus indicus	50 gm
2. Cuminum cyminum	30gm
3. Coriandrum sativum	20 gm
4. Vetiveria zizanioides	10 gm
5. Nelumbo nucifera	5 gm

The above contents were dried and powdered and treated with tender coconut water. The mixture was exposed to direct sun for 4 days and dried. 5 gm of this powder was dissolved in one litre of mineral water (Bisleri) and boiled for 10 minutes. 15 gm of cane sugar was added just before the use.

3. Brahmi Preparation

1. Bacoppa monera	25 gm
2. Centella asiatca	25 gm
3. Cuminum cyminum	30gm
4. Coriandrum sativum	20 gm
5. Vetiveria zizanioides	10 gm
6. Nelumbo nucifera	5 gm

The above contents were dried and powdered and treated with tender coconut water. The mixture was exposed to direct sun for 4 days and dried. 5 gm of this powder was dissolved in one litre of mineral water (Bisleri) and boiled for 10 minutes. 15 gm of cane sugar was added just before the use.

4. Electral.

35 gm.of Electral in 1 litre of water supplies electrolytes in the following concentration.

(Electrolyte m mols/litre)

Sodium.......	51
Potasium	20
Chloride	10

Dextrose 150 m mols/litre

Dilution was made in mineral water (Bisleri)

5. Water

Mineral water (bottled Bisleri water).

Session - III (Standard work heat test). Volunteers were directed to evacuate their bladder and their nude body weight was recorded. Thereafter YSI telemetric surface probes and ECG electrodes were instrumented at the stipulated location. Thereafter volunteers were sent to the HCC simulated for 34°C WBGT. Duration of this exposure was 60 minutes. Initial 20 minutes rest was allowed as micro-acclimatization, which was followed by 40 min sub-maximal endurance test. This sub-maximal endurance was carried out on a bicycle ergometer set for 60W. During standard work heat test, physiological variables such as heart rate, core temperature, and meanskin temperature were recorded at every 10 min. interval (fig 12). Immediately after the completion of the experiment, volunteers were transferred to ALC and physiological probes removed. Sweat was wiped with a towel. Nude-body weight was recorded and sweat loss was computed.

Physiological variables recorded.

1. Heart rate
2. Core temperature (oral) [sublingual temperature]
3. Mean skin temperature according to Ramanathan (1961)
4. Sweat rate. Sweat loss was calculated form the nude body weight difference between pre and post experiment. From this sweat loss sweat rate was computed against the surface area (BSA).

Statistical validation

The study was designed as per Latin squire system. Statistical analysis of the multiple comparisons of various physiological responses with in the groups had been made by the method of two-way classification of ANOVA using the criteria of least significant difference. Statistical significance of various responses at two different locations of the same group has been done by the paired t-test; $P \leq 0.05$ was considered as significant.

Sea route to Antarctica

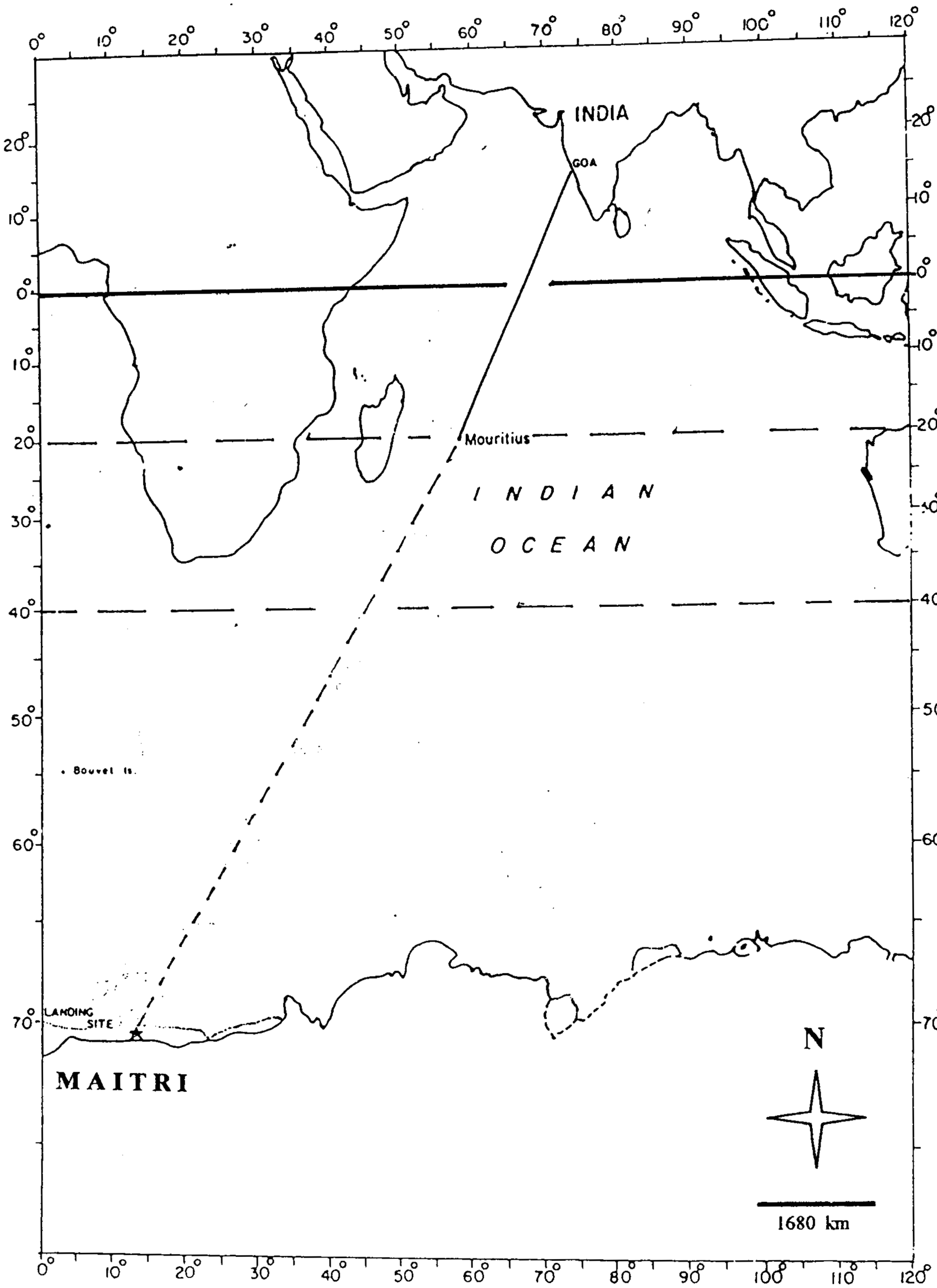


Fig.2

Sighting of iceberg at 54°-S-Latitude

Fig.3

Sighting of pack ice around 65°-S-Latitude.

Fig.4

Polynia, the ice free area in ocean before reaching shelf ice, in summer.
In winter this area will be covered by seasonal ice (fast-ice)

Fig.5
Anchoring of ship at shelf ice Zone at 70°- S- Latitude

Fig.7

"MAITRI" Indian permanent station in Antarctica continent at Schumacher Oasis (70°-S-Latitude and 5° -Longitude).

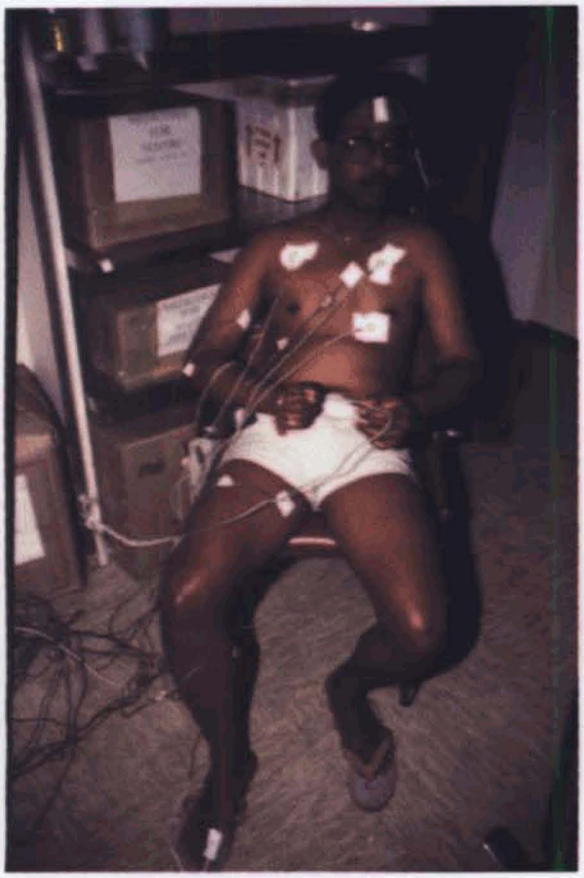


Fig. 8

Volunteer with probes to record ECG and body temperature on the ship, during voyage

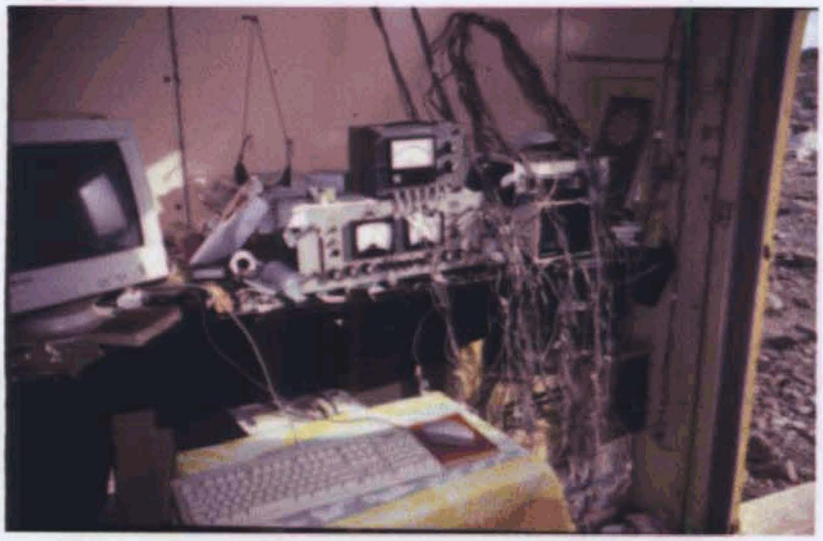


Fig.9

Field Laboratory in Antarctica [Distance from Maitri-500 meters]

Fig.10

Volunteer exposed to out side temperature in Antarctica for recording ECG and body temperature [distance from Maitri, 500 mtr]

Fig.11

Human Climatic Chamber
For studies on heat stress

fig.12

Volunteer in HCC during standard work heat test
for replenishment fluid experiment

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

RESULTS

RESULTS

Part - I

Study on cold stress:

Cardiac function:

Heart rate (HR) (beats per minute) of the volunteers during the down-sail and up-sail is shown in fig 13. The basal heart rate significantly dropped ($p < 0.05$) at 70-S-degree latitude. This HR was found to increase in the course of up-sail at equatorial line, but the recovery was not statistically significant.

During the stay in Antarctica (70°-S-lat), the 60-minute out door exposure showed significantly lower heart rate in comparison with the base line recording. From zero minute exposure, heart rate showed increase at the end of 20-minute exposure. HR increased slightly after 40-minute exposure as well as after 60-minute exposure. However, when compared to initial HR of 75 ± 4.89 none of these increases is statistically significant (fig. 14).

Analysis of ECG:

In lead-II system-recordings, during out door exposure the amplitude of T-wave showed reduction. This t-wave suppression is significant in comparison with the tropical recording made at the initial stage of voyage (fig. 15 and fig.16). The 12 leads-ECG recording was carried out in Maitri station immediately after the out door exposure. The ECG was analysed for the time intervals between waves. The intervals of P, PQ, QRS and QT waves (table-3a) were longer in comparison to normal ECG. The ECG wave's axis was found with in range of normal limits (table-3b). The presence of J-wave in ECG was observed in volunteers who were exposed to out door environment (fig.17 and 17a).

Body temperature during down sail:

Fig. 18 shows the body temperature (°C) of volunteers during the voyage in different phases. At 70°-S-Latitude volunteers were observed to have significantly reduced core temperature. Mean skin temperature also showed significant reduction at the 70°-S-Latitude. Mean body temperature (MBT) also showed decline. The difference in these temperatures (OT, MST and MBT) measured at 0°-latitude and at 70°-S-latitude was statistically significant ($p \leq 0.05$).

Body temperature during up sail:

The reduction in core temperature was found nearly recovered in progressive manner on the way up to zero degree latitude. The recovery is statistically significant. The reduction of mean skin temperature observed at 70°-S-latitude, had a better (statistically significant $p \leq 0.05$) recovery at the end of voyage towards 0°-latitude. The reduced MBT at 70°-S-latitude was also seen improved progressively at zero degrees latitude. A significant recovery of MBT was observed at the end of up-sail (fig.18).

Body temperature during stay in Antarctica:

Sojourners stayed in Antarctica for duration of 55 days. During the last lap of stay (i.e. after 45 days),

TABLE – 3a

Analysis of ECG Profile: Effect of hypothermia

Profile intervals (ms)

	P	PQ	QRS	QT
Mean (ms)	(100) 114	(120) 146	(90) 107	(370) 377
SD	±12.56	±15.40	±12.54	±13.56

Figures in the parentheses are normal values (Weatherall et al 1987)

TABLE – 3b

Axis of profile in degree: effect of hypothermia.

	P	QRS	T
Mean (degree)	+44	+63	+37
SD	±9.77	±12.75	±35.32

Normal electrical axis of the heart is reported to be in the range of -30 and +90 (Weatherall et al 1987) body temperature of volunteers was recorded for duration of 60 minutes while they were staying out-door under sub-zero temperature. During the exposure, OT varied between 35.63 and 35.70°C. This temperature was remarkably low in comparison with 36.67°C basal reading. The reduction in OT was statically significant. The OT was brought down by 60-minute exposure. However the difference is not statistically significant in comparison with initial reading (Zero minute reading) (fig.19).

Mean body temperature [MBT] observed a downward trend, towards the end of exposure. MBT had come down to 31.30°C. This reduction in MBT was significantly low when compared to basal reading of 34.73°C ±0.26. MBT measured during the duration of out-door exposure (60 minute) was not significantly different in comparison with zero minute reading (fig.19).

Mean skin temperature seemed to be more affected by the out-door exposure to sub-zero temperature with reference to basal reading. This significantly reduced mean skin temperature had shown a progressive reduction through out the span of 60-minute exposure (fig.19). But this reduction was not statistically significant with reference to its Zero minute reading.

Peripheral temperature during down-sail:

Extremity temperatures like toes, fingers and foot were significantly ($p \leq 0.05$) low at the 70°-S-latitude when compared to the same measured at zero-degree-latitude of down sail. Fore head temperature was found affected by the down voyage and was lowest at 70°-S-latitude. Arm appeared less affected in comparison with other peripheral temperatures, even though significant decrease in arm was observed at 70°-S-latitude (fig.20).

Peripheral temperature during up-sail:

During up-sail a progressive improvement of peripheral temperatures was observed in general (fig.21). The reduction of extremity temperature viz. toes, fingers, and foot were recovered towards the end of the voyage, at equatorial region. Improvement of these extremity temperatures is statistically significant ($p \leq 0.05$). The lowest temperature of fore head observed in 70°-S-latitude was nearly recovered at 40°-S-latitude and the same was observed for the rest of up-sail. The observed reduction of arm temperature found in 70°-S-latitude was recovered during the course of up sail. The reduction of dorsal temperature in down sail was recovered during the course of up sail. But, The recovery of dorsal temperature was not completed in comparison with the basal reading (fig-20).

Peripheral temperature during stay in Antarctica:

During 60-minute exposure, forehead showed a significant increase in 20 and 40 minute and non-significant increase at 60-minute exposure. Dorsal temperature continued to decrease but is statistically not significant during the exposure. Foot temperature showed significant decrease at 40 and 60 minute exposure. Toe temperature did not show appreciable changes in comparison with initial reading. Finger recorded the lowest among the peripheral temperatures (Fig. 21). It showed a progressive decrease during 60 minute exposure, but the decrease is statistically not significant.

Sweat Rate during out door exposure:

The mean sweat rate recorded during the 60 minute exposure was 47.91 $gm/m^2/hr$ (SD ± 13.95). This was remarkably low sweat rate for a span of 60 minutes (fig.22) in comparison with the invisible perspiration in tropical area (200 $gm/m^2/hr$).

Bedford "comfort vote":

To assess the subjective feeling during out door exposure to sub-zero temperature in Antarctica, Bedford comfort voting technique was administered. The volunteers were asked to fill the questionnaire as shown in appendix-I of 'Bedford Comfort Voting' after the cold-exposure to 60 minutes duration. The volunteers recorded their subjective feeling, as "just cool". Neither sweating nor shivering was reported. Remarkably no cold spot was recorded. The hourly comfort vote recorded for trunk was 'cold'. In some cases, face had reached the level of numbness and pain was felt in the feet.

Part - II

Study on heat stress:

Without replenishment fluids:

Heart rate:

The heart rate recorded with out intake of fluid is shown in fig. 23. The basal heart rate of 76 per minute showed progressive increase during standard work heat test. There was a significant increase in HR observed at the end of 20 min. rest in HCC. The significant increase in heart rate occurred through out the 40-minute period thereafter. Heat stress in terms of heart rate was observed in the higher HR at 160 per minute, which is the upper limit of tolerance (Fig-23).

ECG:

The amplitude of QRS complex was found reduced towards the end of exposure in comparison with initial recording. On the other hand amplitude of t-wave was found to be higher towards the end of 60-minute exposure (fig.24).

Body temperature during normal exposure:

Core temperature recorded as oral temperature had shown a significant increase in volunteers during standard work heat test. The progressive increase of oral temperature continued till the end of the exposure. Mean skin temperature had remarkable rise form the basal reading during standard work heat test. The rise in mean skin temperature was observed through out the duration of exposure. A significant increase ($p \leq 0.05$) was shown in mean body temperature right from the end of 20-minute rest in HCC. It has progressively increased for the remaining 40-minutes of sub-maximal endurance (fig.25).

Sweat rate:

Sweat rate with out fluid intake in standard work heat test was observed as 418 ±85.46. It is considerably high as far as heat exposure is concerned (fig.26).

Study on the effect of replenishment fluids:

Heart rate:

Statistically no significant difference was seen after the intake of fluids, so far as the HR was concerned. However, HR was lower after the intake of electral and brahmi (fig.27). Profiles of ECG with fluid and with out fluid intake showed noticeable differences (fig.28). In the case of exposure with fluid-intake, elevation of amplitude of T-wave was observed exponential with the duration of exposure. Elevation of T-wave (fig.29) was higher in 'with out fluid exposure' in comparison to exposure 'with fluids'.

Body temperature:

The rise in oral temperature during standard work heat test did not have any statistical significance among fluids. However, A measurable lowest core temperature had been observed in the case of brahmi, nanari and electral (fig.30) at the end of the exposure. Statistically, no significant differences were observed among the replenishment fluids regarding the mean skin temperature.

But, rise in MST was lower after in take of brahmi and nanari as replenishment fluids (fig.31).

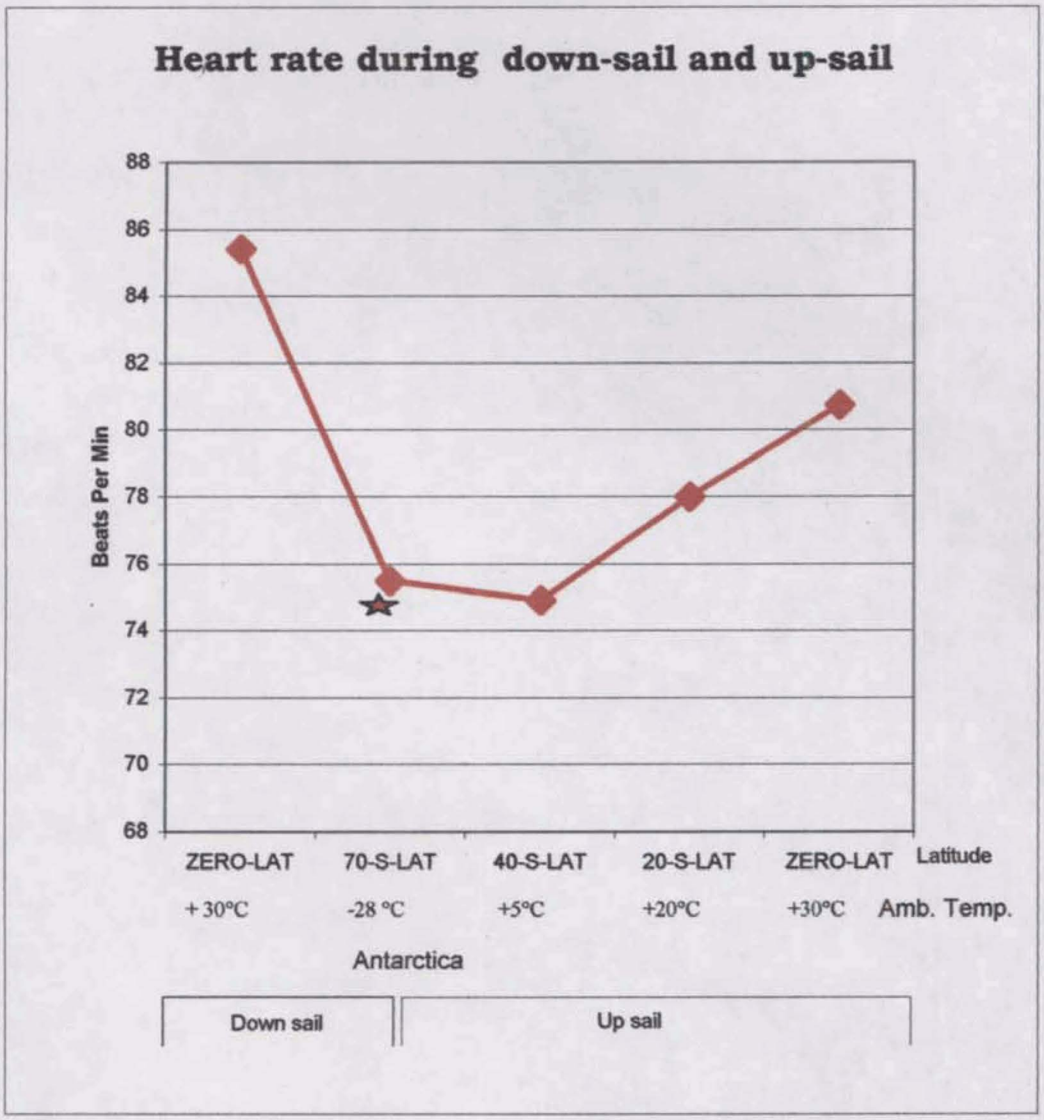
Mean body temperature had shown statistically no significant difference between fluids for the duration of exposure. But, at the end of 60 minutes exposure replinishment with brahmi fluid showed the lowest mean body temperature among the fluids used (fig.32).

Sweat Rate:

Statistically, there was no significant difference between these fluids regarding sweat rate. But, brahmi had the lowest sweat rate in comparison with the other replenishment fluids (fig.33).

Tabular statements with "Standard Deviation" of the above results of part-I and part-II are given in the appendix-I.

Fig – 13



★ Significant with reference to down-sail at zero° latitude ≤ 0.05

Fig - 14

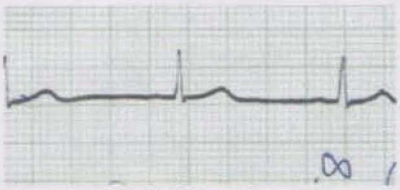

Heart rate during 60 min out-door exposure in last lap of stay
77
76
75
74
73
Beats per Minute
Wind chill index. = 1000 ± 200
Heart Rate
0 - min
20 - min
40 - min
60 - min
Exposure time

Fig - 15

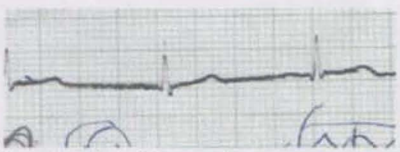
Representative ECG during Antarctica expedition.

Down- sail. At Zero Degree Latitude

Effect of 60 min. exposure to out door during the last lap of stay



Beginning of 60 min exposure



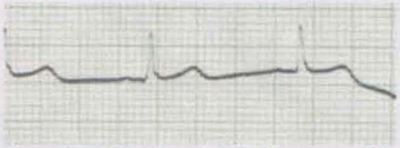
End of 60 min exposure

Scale: 5.mm = 1 Mv
Chart Speed: = 25 mm per Sec.
Duration of recording 2 Sce.

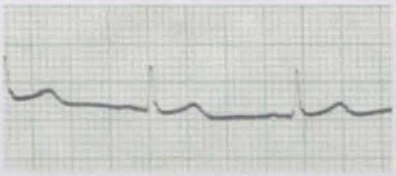
Fig - 16

Representative ECG

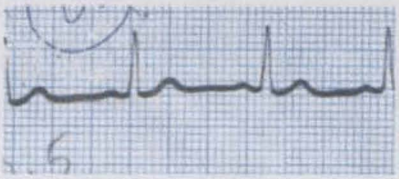
During up-sail:



At 40 degree S Latitude



At 20 degree S Latitude

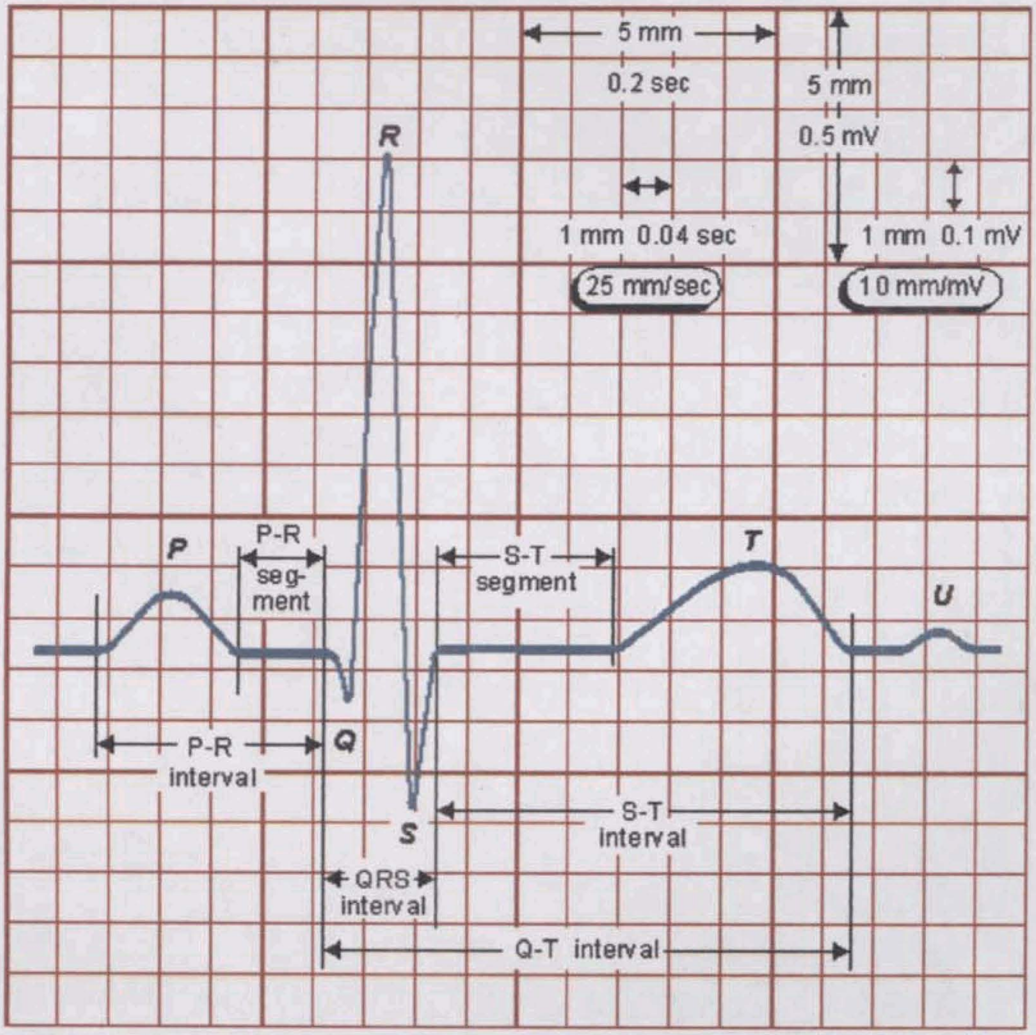


At Zero degree S Latitude

Scale: 5.mm = 1 Mv
Chart Speed: = 25 mm per Sec.
Duration of recording 2 Sce.

Fig – 17

The normal electrocardiogram.

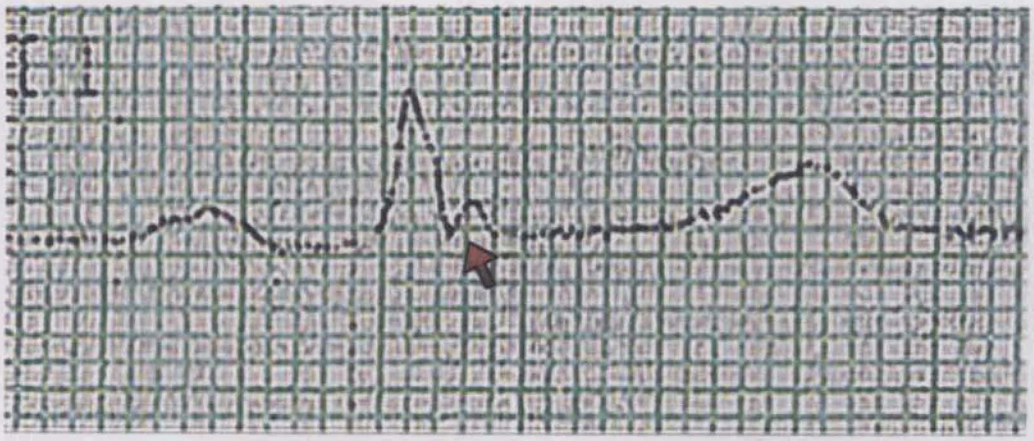


(Schamroth 1982)

Fig – 17a

ECG showing J-wave (12 lead)

Due to hypothermia

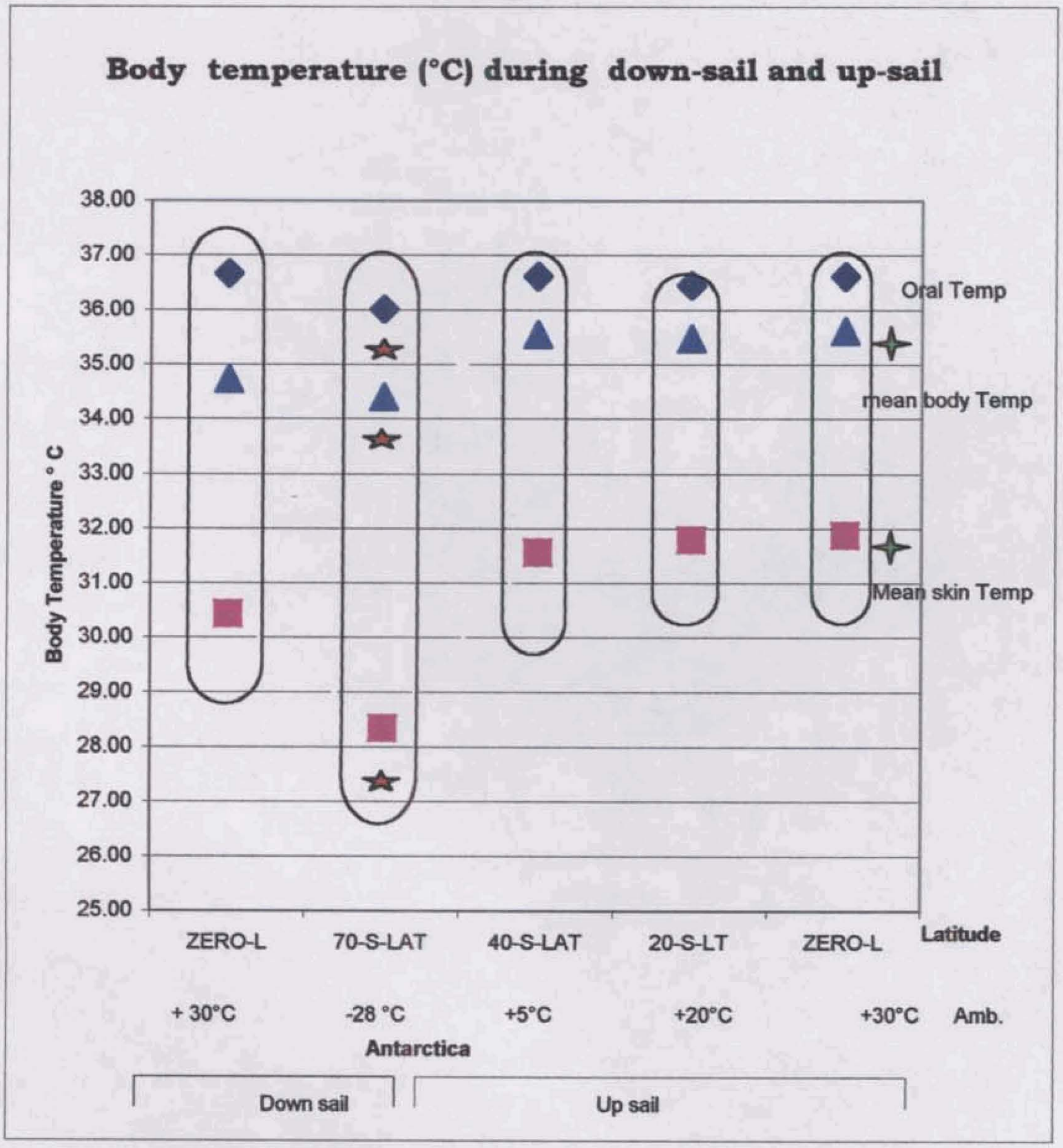


Optioned at 70°-S-Latitude

Scale: 5.mm = 1 Mv
Chart Speed: = 25 mm per Sec.
Duration of recording 2 Sce.

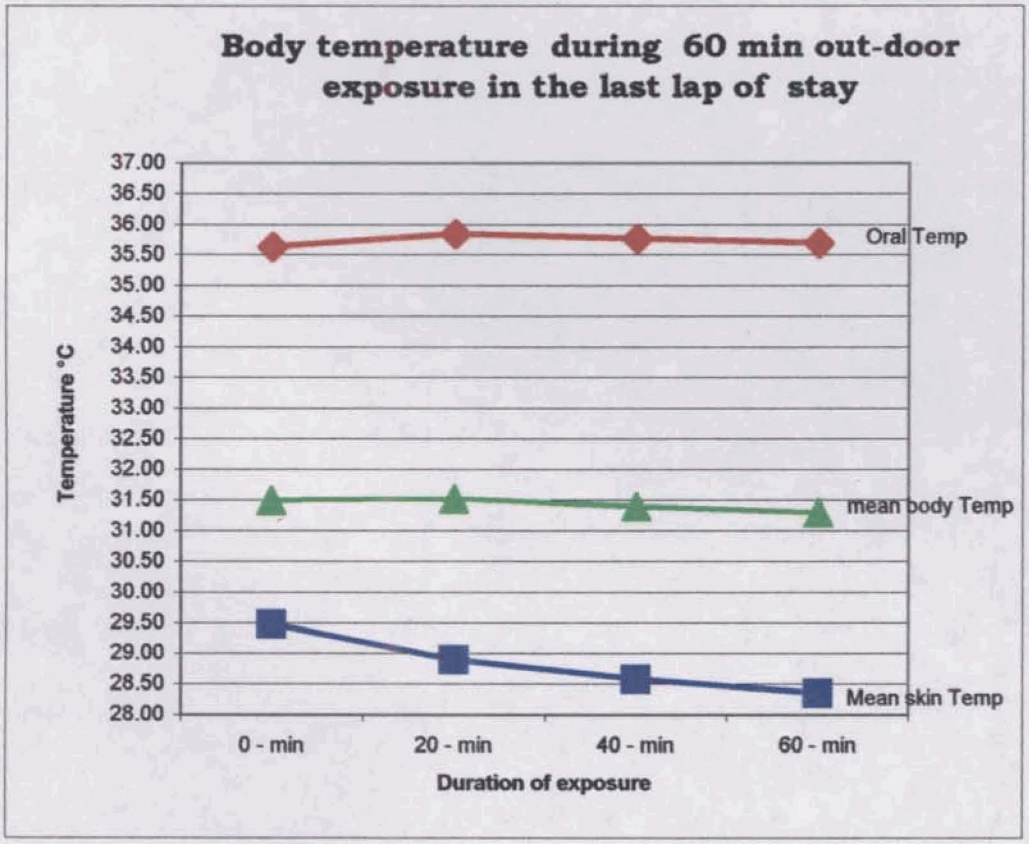
Indication for J wave

Fig - 18



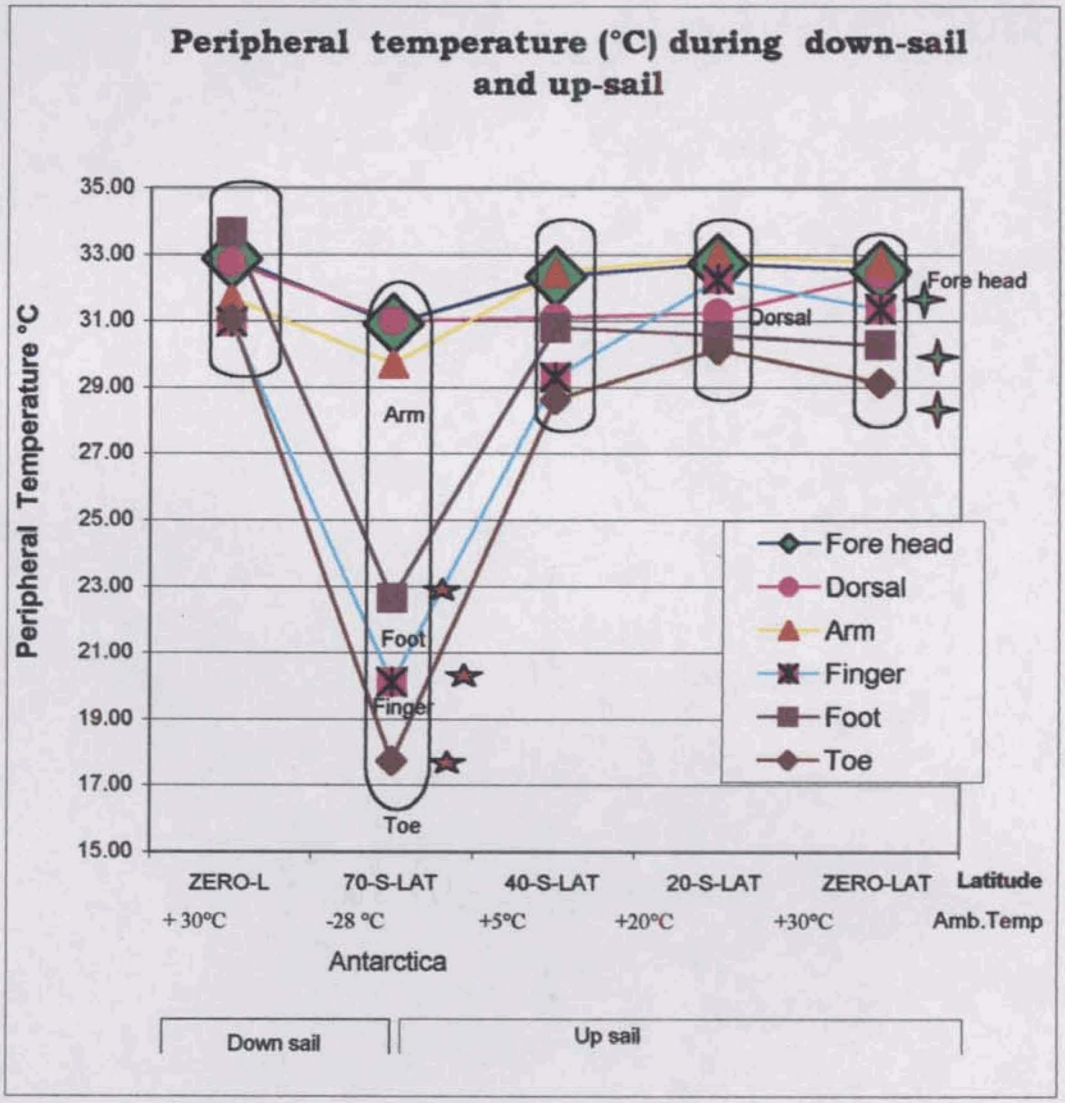
★ Significant with reference to down-sail at zero° latitude ≤ 0.05
✦ Significant with reference to 70°-S- latitude ≤ 0.05

Fig - 19



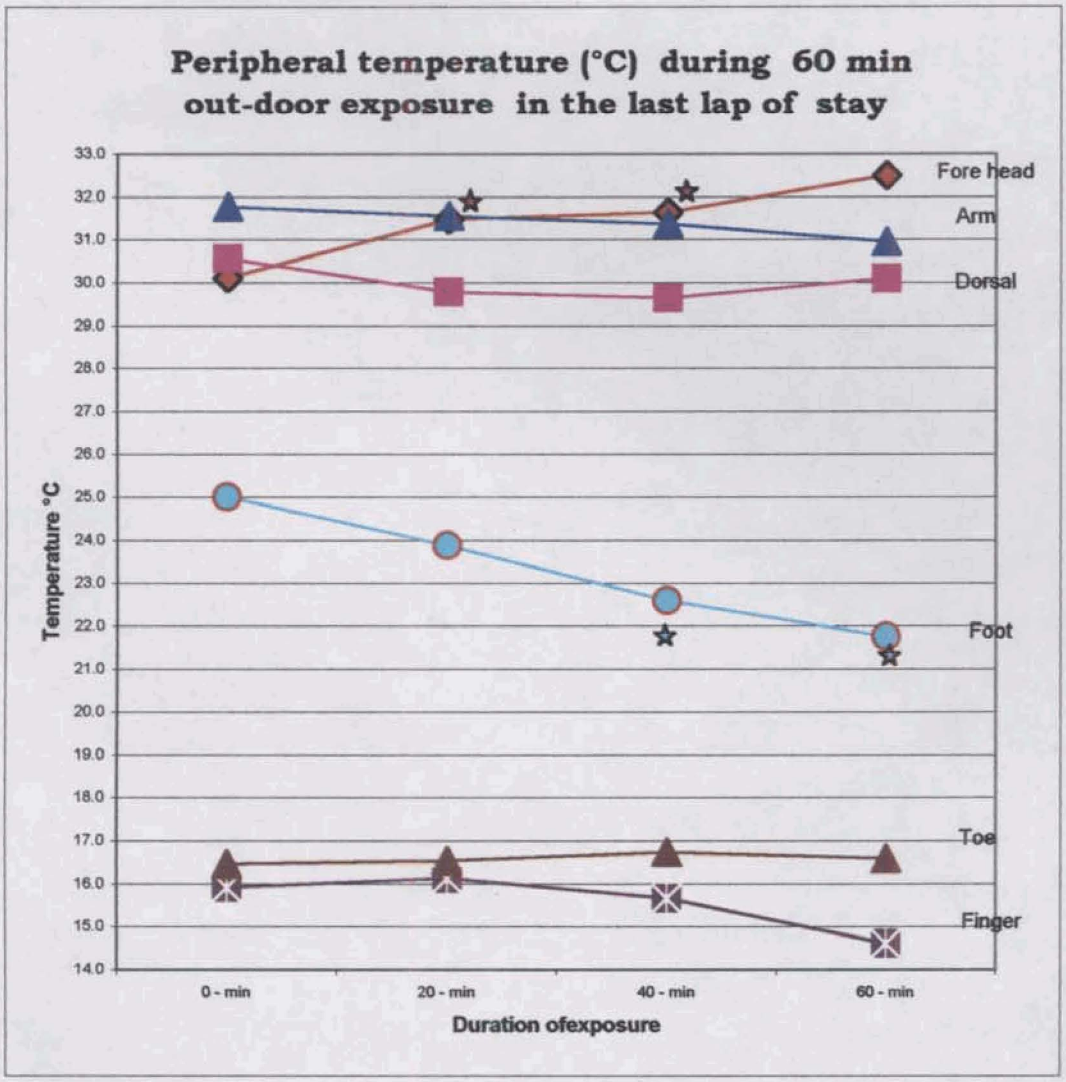
Body temperature during 60 min out-door exposure in the last lap of stay
37.00
36.50
36.00
35.50
35.00
34.50
34.00
33.50
33.00
32.50
32.00
31.50
31.00
30.50
30.00
29.50
29.00
28.50
28.00
Temperature °C
Oral Temp
mean body Temp
Mean skin Temp
0 - min
20 - min
40 - min
60 - min
Duration of exposure

Fig - 20



★ Significant with reference to down-sail at O° latitude ≤ 0.05
✦ Significant with reference to 70°-S- latitude ≤ 0.05

Fig - 21



☆ Statically significant $P \leq 0.05$

Fig – 22

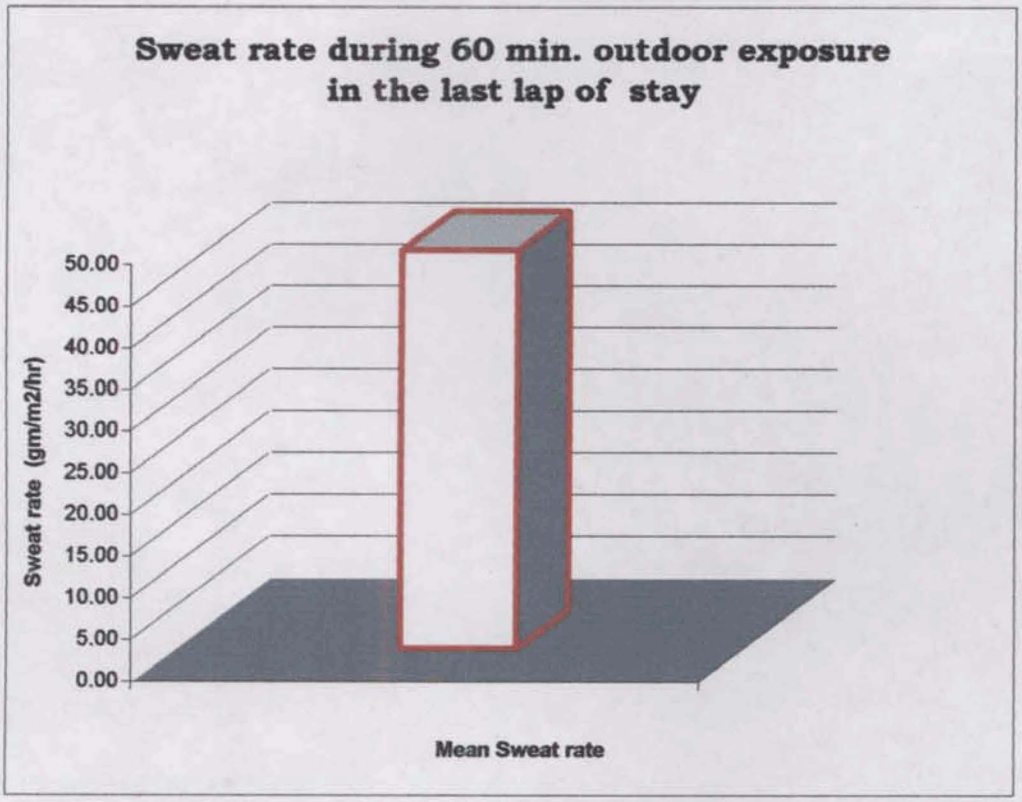
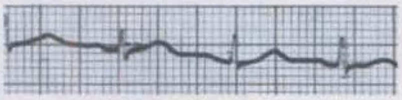

Sweat rate during 60 min. outdoor exposure in the last lap of stay
50.00
45.00
40.00
35.00
30.00
25.00
20.00
15.00
10.00
5.00
0.00
Sweat rate (gm/m2/hr)
Mean Sweat rate

Fig - 24

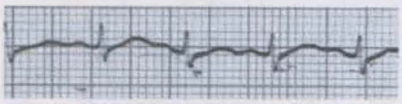
Representative ECG With Out Replenishment Fluids

During Standard-Work-Heat-Test [SWHT]

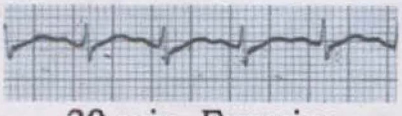


20 min. rest

10 min. Exercise



20 min. Exercise



30 min. Exercise

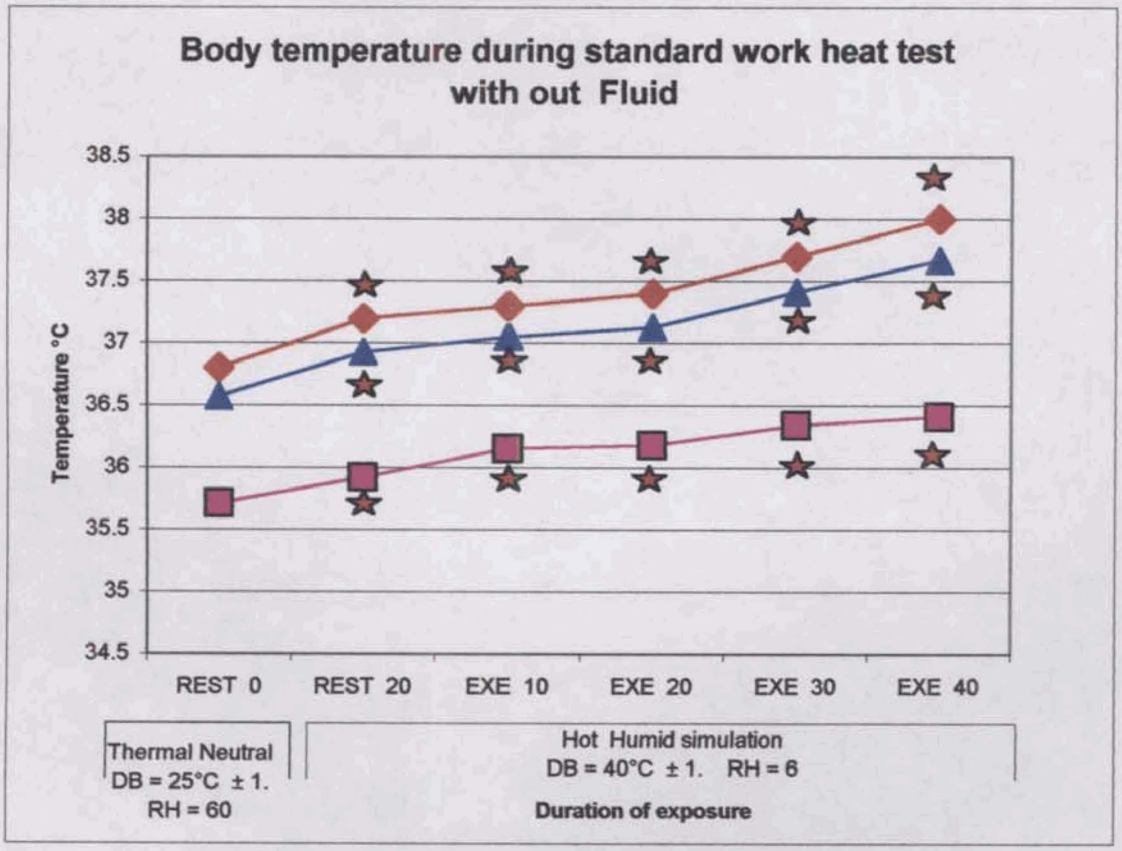
40 min. Exercise

Scale: 5.mm = 1 Mv

Chart Speed: = 25 mm per Sec.

Duration of recording 2 Sce.

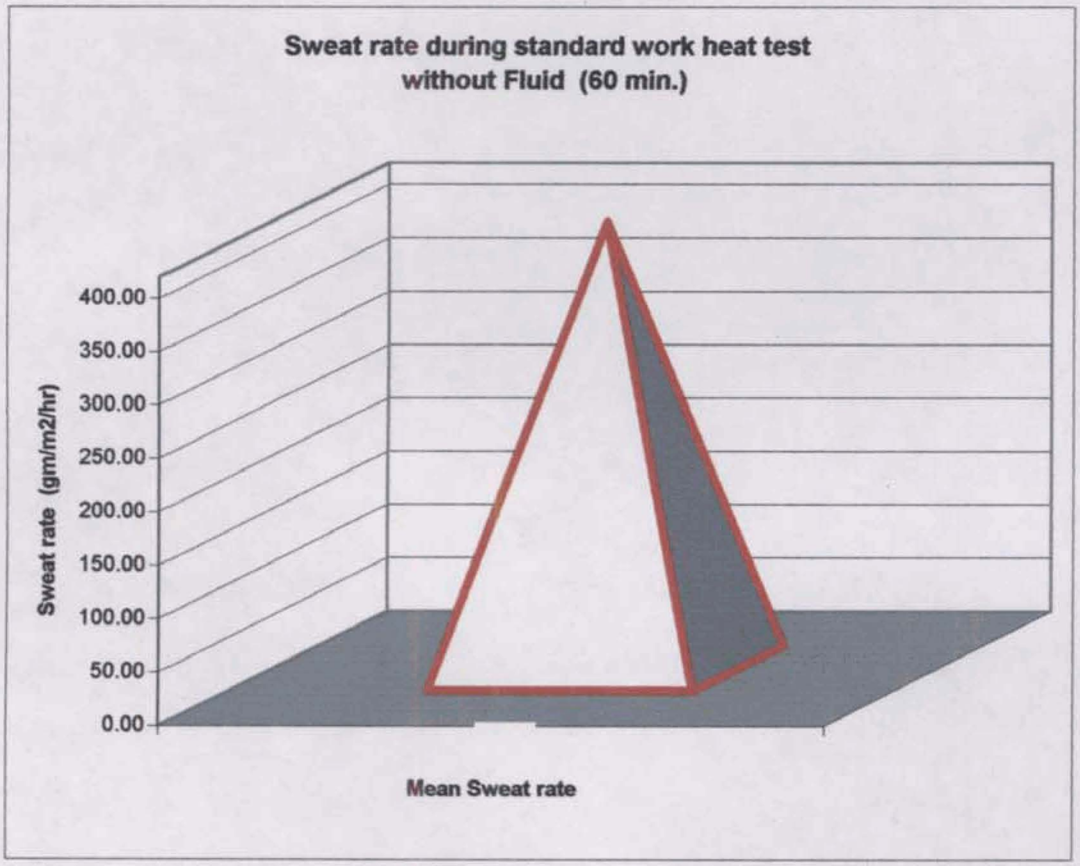
Fig - 25



RES = Rest
EXE = Exercise

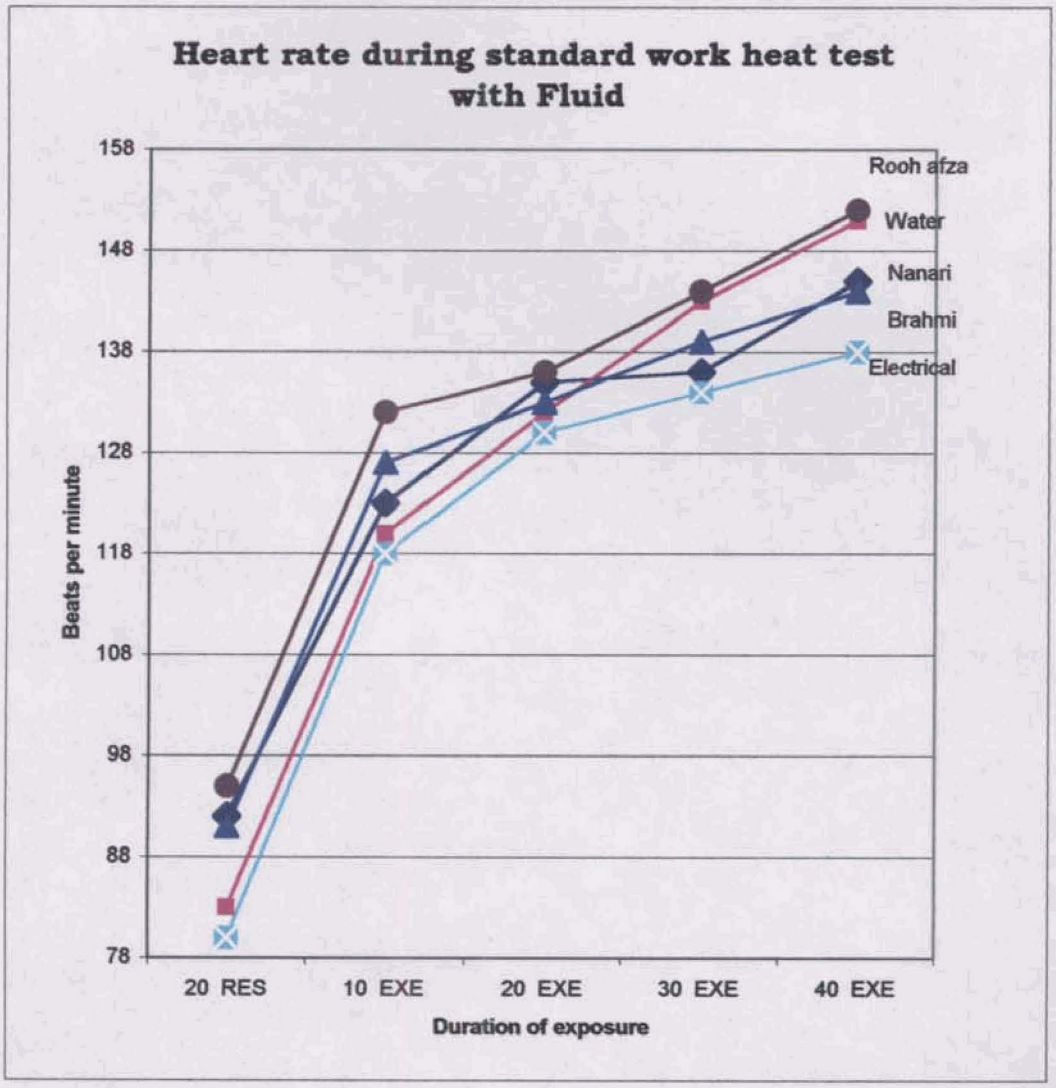
★ Significant P $\leq$ 0.05

Fig - 26



Sweat rate during standard work heat test
without Fluid (60 min.)
400.00
350.00
300.00
250.00
200.00
150.00
100.00
50.00
0.00
Sweat rate (gm/m2/hr)
Mean Sweat rate

Fig - 27

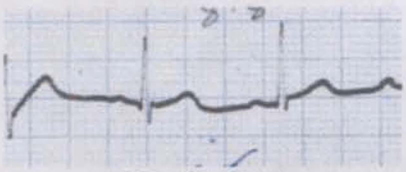


RES = Rest
EXE = Exercise

Fig - 28

Representative ECG With Replenishment Fluids

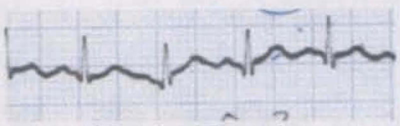
During Standard-Work-Heat-Test[SWHT]



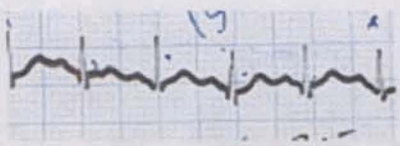
20 min. Rest

10 min. Exercise

20 min. Exercise



30 min. Exercise



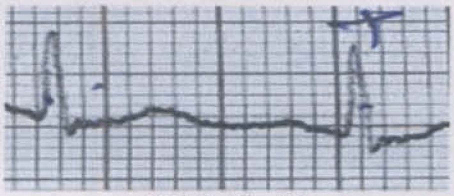
40 min. Exercise

Scale: 5.mm = 1 Mv
Chart Speed: = 25 mm per Sec.
Duration of recording 2 Sce.

Fig - 29

Comparison of ECG
With and without fluid intake

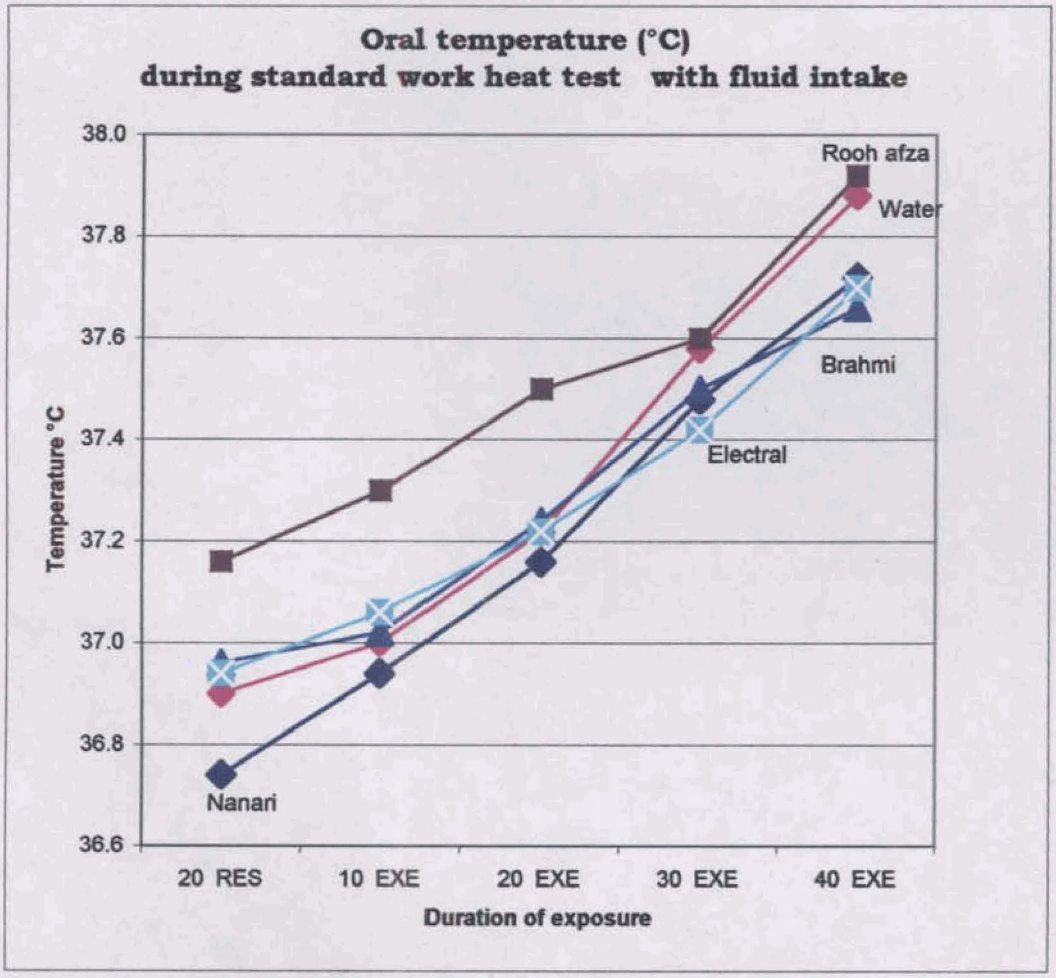
With-out fluid



With fluid

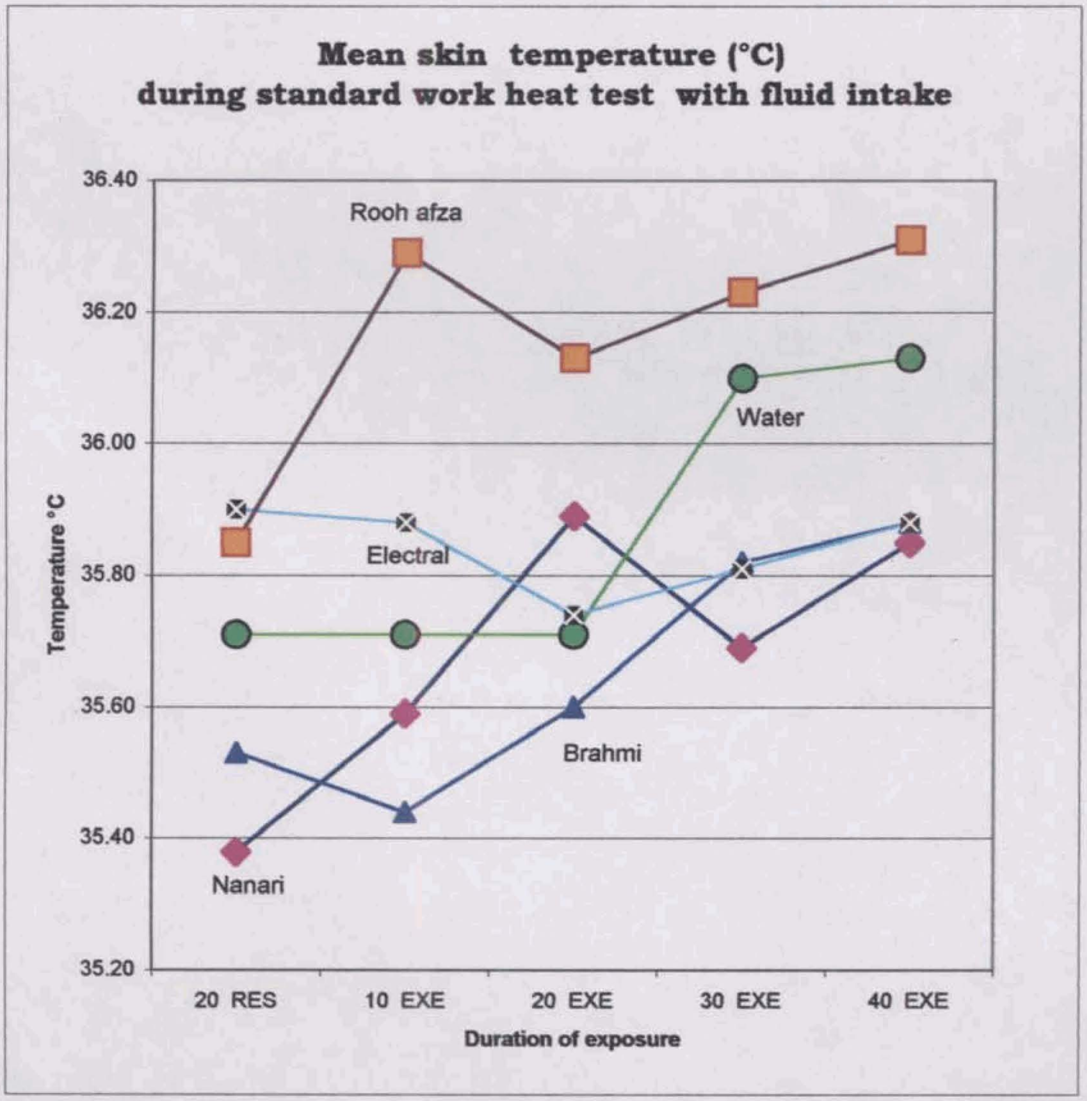
Scale: 5.mm = 1 Mv
Chart Speed: = 25 mm per Sec.
Duration of recording 2 Sce.

Fig – 30



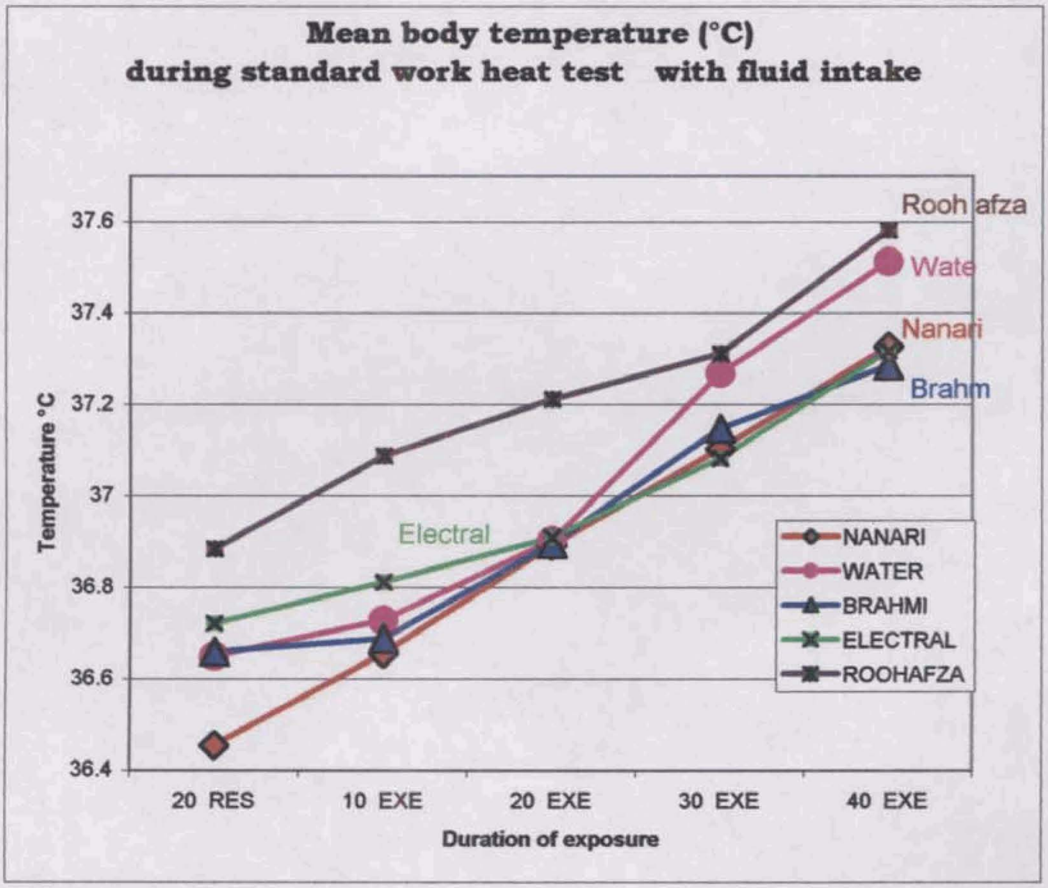
RES = Rest
EXE = Exercise

Fig - 31



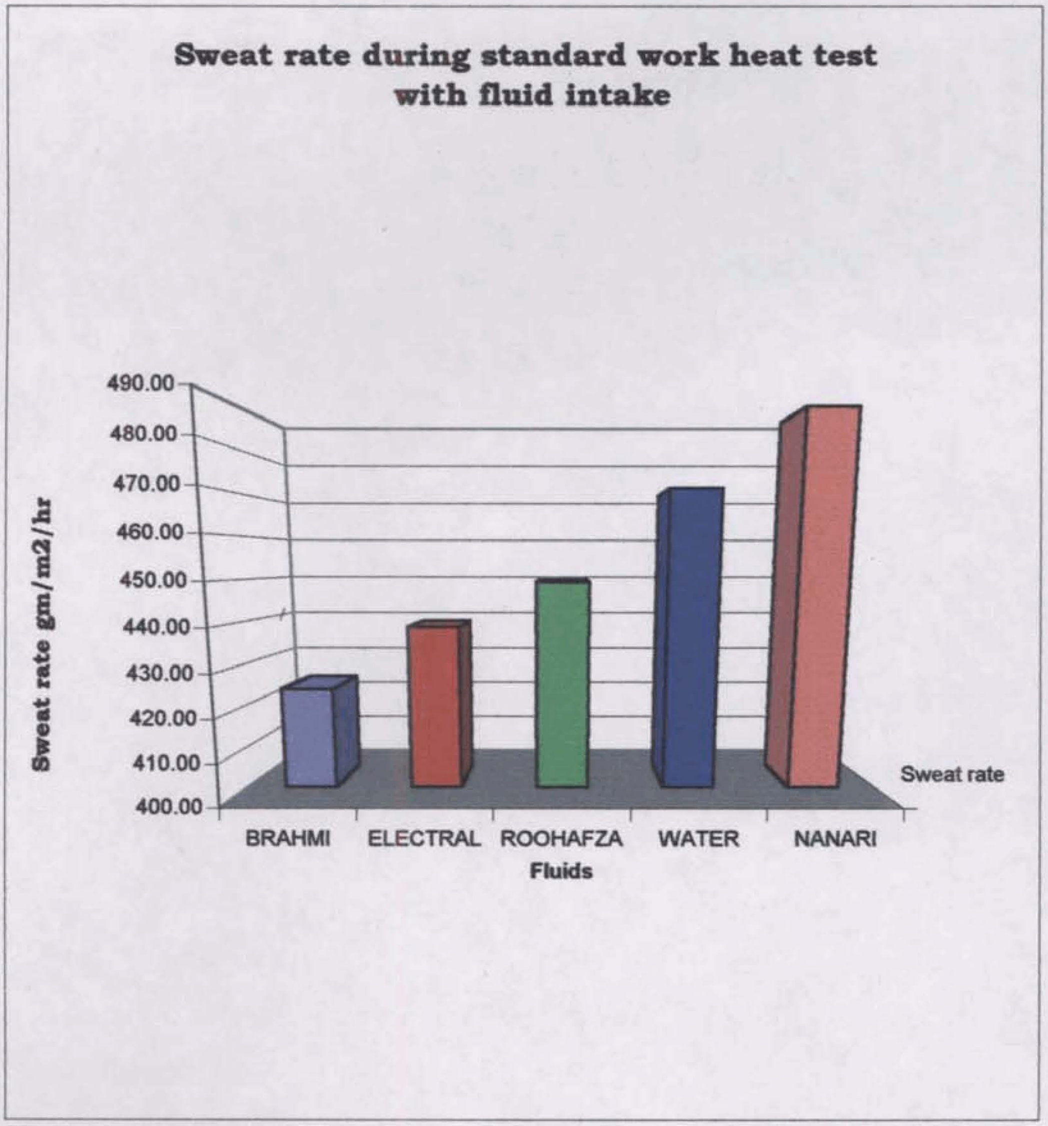
RES = Rest
EXE = Exercise

Fig -32



RES = Rest
EXE = Exercise

Fig - 33


Sweat rate during standard work heat test
with fluid intake
490.00
480.00
470.00
460.00
450.00
440.00
430.00
420.00
410.00
400.00
Sweat rate gm/m2/hr
BRAHMI
ELECTRAL
ROOHAFZA
WATER
NANARI
Fluids
Sweat rate

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA

2004

DISCUSSION

DISCUSSION

PART- I

Study on cold stress

Effect of cold on Cardiac function during voyage

The initial heart rate appeared to be higher than conventional heart rate norms. Reasons might include apprehension and the initial stress of the voyage itself, because all the volunteers were first time voyagers. As Danzl et al.(1995) suggested, in mild or borderline hypothermia, tachycardia and other cardiac responses to sympathetic drive occur. Subsequently, as the temperature falls, a progressive bradycardia along with other ECG changes take place; this is apparently due to parasympathetic overdrive. Finally, at even lower temperatures, severe core cooling results in a direct and global cardiovascular depression (Alings et al.1999 and Lehmann et al. 1998).

In this study, the hypothermic shock on cardiac function was mild regarding heart rate than what was anticipated. The gradual induction to cold environment by sail of ship might have induced the required acclimatisation. The polar clothing ensemble had a protective role. The extent of cardiovascular involvement in hypothermia is a function of the severity of body cooling. The severity of cooling has a sequence of three pathophysiologic stages of mild, moderate, and severe hypothermia (Danzl et at 1995). During up sail, heart rate progressively increased from 40°-S-Ltitude. However during the up- sail heart rate at equatorial region was lesser than the down sail heart rate. It might be due to the residual effect of cold acclimatization.

Suppression of T-wave at 70°-S-Ltitude was observed in comparison to the ECG profile recorded in 0°-Ltitude. This finding showed the effect of cold on heart. Litovsky et al. (1990) and Yan (1996) also reported that apparently, mild hypothermia in his sojourners acted as an, autonomic, amplitude-reduction of T-wave. Studies on accidental hypothermia reported that mild cold impact on ECG is reversible. In the present study, the observed depression of T-Wave was restored during up sail and reached the normal level at the end of the voyage. Similar studies ware reported by Brugada et al. (1992) and Antzelevitch (1998).

Cardiac function during the stay in Antarctica:

Out door expose for one hour, during the stay at 70°-S-Ltitude had a remarkable effect on cardiac profile. But the severity of cold on heart might be reduced due to the possible acclimatization of sojourners. The measurable difference in HR during the initial period of 20 min exposure is in agreement with the trend of tachycardia reported by William Forgey (1985). The initial cold shock leads to vasoconstriction which may result in increased heart rate. In an acclimatized individual this vasoconstriction improves and leads to vasodilatation. From 20 minutes to 40 minute of the exposure, the heart rate remained at the same level.

Some studies had shown that initial tachycardia leads to bradycardia as a sign of hypothermia. In this study, towards the end of 60 minute exposure, the heart rate increased. The clothing might provide a physiological comfort to sojourners. Secondly the sitting posture might have helped the sojourners to conserve some energy to maintain body temperature and in turn the heart rate.

12 lead ECG recordings did reveal the effects of cold stress on cardiovascular function. The recording was made at Maitri MI room as the field laboratory in Aravali hut did not have room heating system. In fact, the temperature in the hut remains same as the out door sometimes even lower due to the still shade- temperature. The temperature at Maitri MI room was thermo neural. But the study assumed, that there should be a residual spell of cold impact of preceding exposure to the cold. The ECG during mild or borderline hypothermia showed a striking ST segment elevation with upward concavity.

The presence of J-wave (Osborn-wave) was observed. The elevation of ST segment as well as the presence of the J-wave are the cardinal symptoms of hypothermic stress on heart. There are similar reports from Litovsky et al. (1990) and Yan (1996), apparently due to, mild hypothermia in his sojourners. ST elevation was regarded to indicate early repolarization syndrome (Kambara 1976, Hurst 1998 and Brady et al. 1999).

The amplitude of J-wave, proved to be a function of the action potential in phase 1 of cardiac electric activity (Hurst 1998). Some of the recent studies, show an intensifying role of the parasympathetic system, as opposed to the sympathetic tone, in the action potential phases 1 and 2 related to J-wave and ST-segment elevation primarily by means of a transient outward (potassium) current predominance over the L-type inward calcium-current in epicardium but not in endocardium (Antzelevitch 1998, Anguera 2000).

The Osborn wave seen under hypothermia is thought to be due to early repolarization (Schamroth 1982, Brugada et al. 1992 and Alhaddad 2000). The presence of Osborn wave is reversible. This finding is in agreement with the earlier studies reported as hypothermic accidents and the impact on ECG (Litovsky 1990, Yan 1996, Antzelevitch 1998, and Gavaliatsis 2000).

Oral temperature during voyage:

A significant reduction of 0.65°C was observed in oral temperature during voyage. This shows the effect of cold on core temperature. So for as homeostasis of temperature regulation is concerned, statistically significant reduction in oral temperature can produce hypothermia. However, this situation did not bring about hypothermia because of gradual induction of sojourners into sub-zero temperature by down sail. This physiological element of cold acclimatization as well as the protection of polar ensemble averted serious hypothermic impact. The reduction in core temperature was recovered during the up sail at 40°-S-Ltitude. At the end of the voyage the oral temperature had been stabilized at 36.62°C. This clearly shows the importance of acclimatization process to withstand the impact of extreme environments LeBlanc 1975).

Bittel (1987) observed cold induced increase in core-temperature and delayed onset of shivering. The present findings of reduced shivering and heat production agree with those of many previous studies, such as those reported by Davias (1963) and Budd (1974, 1989) of cold exposed men.

Livngstone et al (1976, 1989) and Gregory et al (1999) observed, that there are two types of reactions to a general cold stress: the first type is referred to as insulative-hypothermic acclimatization in which the subject does not increase his metabolic rate and allow core temperature and skin temperature to go down. These changes have been reported by Budd (1966), after a stay in Antarctica. In contrast metabolic acclimatization occurs when individuals increase their metabolic rates in response to the cold to maintain body temperature (Scholander et al 1958 and Harnett et al 1983).

Young et al (1986) had concluded that acclimatization was mediated by increased sympathetic activity and an enhanced vascular response to cold. However, because of their finding of reduced mean skin temperature, these authors have suggested that the enhanced vascular response was located in the superficial shell (i.e. skin and subcutaneous fat) and muscle perfusion was increased, whereas our own findings favour the opposite conclusion. We would suggest that these differences in apparent site of vascular changes of acclimatization might be the result of differences in acclimatization as a result of long stay in Antarctica continent and in addition to gradual introduction to cold region by ship's down sail (i.e. 40° to 70°-S-latitude). Another supporting factor was the protection provided by the cold protective ensembles. The protective ensembles collectively offered an average global clothing insulation of 1.17 *Clo*. This is regarded as adequate protection against cold during the period of exposure. (Budd et al.1962, Livingstone et al. 1967 and Rivolier et al. 1988).

The thickness of the subcutaneous fat, which insulates the body from cold, has been shown to have an inverse relationship with the reduction in core temperature (Budd et al 1991) and an increase in metabolic rate after cold exposure. However, none of this relationship was obvious in the result of the present study, although the subjects differ in body fat. Some unpublished data from this institute [Defence Institute of Physiology and Allied Sciences] showed that a measurable weight gain occurred in Antarctica sojourners. Thus it appears that body fat did not significantly contribute to thermal insulation.

Temperature of digits: (Finger and Toe)

Among the peripheral temperatures recorded, digits were affected more by cold. However, cold injury is prevented by increasing the finger blood flow and maintaining the extremity temperature. Similar response was observed in people living in the Arctic and sub-arctic areas who are habituated to cold (Elsner et al 1960 and Leblanc et al 1960). Reducing the local skin temperature of the human hand from a normothermic level results in cold-induced vasoconstriction (CIVC) but the local temperature < 18°C may result in cold induced vasodilatation (CIVD). It is assumed that the arteriovenus anastomoses (AVAs) are responsible for CIVC and CIVD (Bergersn TK et al. 1993).

The results of this study are in agreement with the finding of earlier investigators, (Krog et al 1960, 1978) which indicated that tissue insulation apparently produced sympathetic vasoconstriction. The fall in skin temperature, after 1 and 8 weeks of acclimatization suggests vasoconstriction of the cutaneous blood vessels. This affirms the view that the CIVD response in cold acclimatized subjects is more rapid (Korg et al 1960.) However, the time of onset of vascular response was within the first minute after the maximum vasoconstrictor response when the Temperature of digits fell below 15°C; but the polar gloves prevented the reduction of extremity temperature further down.

Oberg et al (1983) cited onset of vasodilatation as increase in finger blood flow after cold immersion.

This could be considered as a peripheral vasodilator response due to persistent Antarctic cold exposure, although the blood flow was increased by cold acclimatization, the finger skin temperature was lower compared to the normothermic state. This indicates that the amount of cutaneous vasodilatation in hand was insufficient to raise the tissue temperature. Naidu et al (1993) reported that increase in the flow of cooled blood could not bring about skin temperature alterations (Barocroft et al 1943).

When a hand or foot is cooled to 59°F (15°C), maximal vasoconstriction and minimal blood flow occur. If cooling continues to 50°F (10°C), vasoconstriction is interrupted by periods of vasodilatation with an increase in blood and heat flow. This "hunting" response recurs in 5-10 minute cycles to provide some protection from cold. Prolonged, repeated exposure increases this response and offers some degree of acclimatization. Eskimos have a strong response with short intervals in between.

The decrease in digits temperature was observed in the present study even though volunteers were protected by polar gloves. When blood temperature is low the vasodilatation does not warm the skin. This vasodilatation could be a contributory factor for maintaining the strained supply to the skin without the participation of heat conductance, thus preventing cold injuries and hypothermia of digits (Rivolvier 1987).

Part II

Study on heat stress:

The purpose of this study is to determine heat stress imposed on tropical volunteer as well as the efficacy of ergogenic herbal replenishment fluids for improving thermoregulation during endurance in heat. Nanari, Brahmi, Electral, Rooh-afza and water were the five fluids selected for the present study.

Acclimatization:

During first exposures to a hot environment, workers often feel very tired, irritable and 'too hot'. Body temperatures often rise. After repeated exposures these symptoms decrease or disappear. When this occurs a person is considered to be acclimatized. 110 minutes exposure with sub-maximal work in simulated HCC was carried out for 8 consecutive days for acclimation (Barbara et al 1997, Pichan et al (1999 and Margaret et al 2003).

Study without intake of fluid:

The initial heart rate recorded from the thermal room before entering HCC was 72 ± 7.2,while 160 ± 13.3 was observed at the end of standard work heat test for the exposure with out fluid. This is a statistically significant increase from the initial 76 beats per minute. Heart rate is considered as one of the parameters to assess the stress imposed on the heart during heat exposure. Our finding is in agreement with some of the earlier findings.

In the hyperthermic response, generally seen in younger persons, pulse is typically very rapid (160 to 180 beats/minute), cardiac output is normal or increased, blood pressure (BP) is usually normal, and central venous pressure is commonly elevated. There is also a greater fluid loss.

Other cardiovascular manifestations include ECG changes in ST segments and T-waves, premature ventricular contractions, supraventricular tachycardias, and conduction abnormalities.

Chaotic variation in heart rate was observed in this study even at the early stages of exposure. But those did not lead to the pathological situation during exposure.

In this study, the heat induced irregular supraventricular tachyarrhythmias (SVT) were not seen as reported in hyerthermic study of Fisch et al (1997). The atrium "fibrillates", writhing like a bag of worms. The conventional view of the pathogenesis of atrial fibrillation is that there are multiple re-entrant `wavelets' moving through the atrial muscle. AF actually arises from ectopic activity in the muscular cuff surrounding the pulmonary veins where they enter the left atrium. The Ventricular fibrillation with respect to the ECG recording was not seen in the present study.

Study with intake of replenishment fluid:

In the study with fluid replenishment, heart rate varied in the range 138 to 152 at the end of standard work heat test, in response to the five replenishment fluids. These values were in similar range of heart rate observed by Riebe et al (1997) in his study of "effect of oral and intravenous rehydration on rating of perceived exertion". However, the results recorded in the present study were not statistically significant with respect to different replenishment fluids.

Body temperature:

Core temperature of volunteers in the present study with in take of fluid showed statistically insignificant variations with respect to the different replenishment fluids. The values at the end of standard work heat test exposure were in the range of 37.7°C and 37.9°C. The pattern of increase of core temperature during the exposure showed some similarity with the study "hyperhydration: thermoregulatory effects during compensable exercise-heat stress" conducted by Latzka et al (1997).

Mean skin temperature showed a gradual increase during the period of heat exposure. The pattern of increment of mean skin temperature agrees with the end exposure values observed by Latzka et al (1997), even though the observed values were higher in the present study. These results agree with that of Montain et al (1997) who reported no difference in core temperature between glycerol and water hyperhydration trials. Latzks et al summarize their studies as follows. During compensable exercise-heat stress, thermoregulatrory responses were identical regardless of whether subjects were euhydrated. Glycerol hperhydration provided no hydration advantage over water hyperhydration during exercise-heat stress. (Costill et al. 1973, Riebe 1997, Jimene et al 1997, Shirreffs S.M 1997).

However, the results of current experiments showed statistically insignificant physiological variables, such as heart rate, oral temperature, mean skin temperature and sweat rate. But some measurable differences were observed with respect to replenishment fluids. In the case of "Brahmi replenishment fluid," minimum core temperature as well as sweat rate were recorded. In the case of mean body temperature brahmi fluid had the minimum rise. Values are presumably showing some beneficiary effect of heat dissipation during the work in heat (in simulated hot humid environment). In heat stress environment, if a replenishment fluid can bring down core temperature as well as sweat rate to minimum level, it is undoubtedly considered as beneficial (Adolph et.al 1947). The present study also opens up one more important area for further scrutiny. 'Bacopa monnieri' and 'Centella asiatica' are the main herbal constituents of the brahmi replenishment fluid. The water retention property and reduction in core temperature are assumed to be the effect of Bacosites A and B of 'Bacopa monnieri', as well as the Terpinoid compounds of 'Centella asiatica' (Rastogi et al 1993). Further investigation is required to identify the active principles of these brahmi species, Which has beneficial effect in coping with the heat-stress.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

SUMMARY AND CONCLUSIONS

SUMMERY AND CONCLUSIONS.

This study was carried out to understand extreme environmental stress in terms of cold and heat on tropical population with special reference to cardiac function.

Study on cold stress was carried out on members of Indian Antarctica Scientific Expedition. During December 1998 to March 1999 (XVIII Summer Expedition).

Heart rate of sojourners in the tropical region (Zero latitude) decreased when they reached 70°-S-Latitude. The reduction in heart rate was recovered during the end of up sail. Depression in T-wave was observed while sojourners were in Antarctica, due to the impact of cold. Besides, elevation of ST-segment was also observed due to mild hypothermia.

The appearance of J-wave (Osborne wave) was significant finding in volunteers who were exposed to out door environment of sub-zero temperature during their stay in Antarctica. This is an early repolorization symptom due to cold. This is reversible. Body temperature like core, mean-skin and mean-body temperature were lower while sojourners reached 70°-S-Latitude, which were seen rising during up sail 40°-S-Latitude.

Peripheral temperature also showed a reduction in comparison with their tropical values. The same were seen rising during up sail from 40°-S-Latitude. Extremity temperatures showed the lowest viz. Finger, toe and foot.

Those temperatures reached the lowest tolerable limit during the out-door exposure in Antarctica.

Study on heat stress was conducted in human climatic chamber [HCC] (at DIPAS, Delhi) simulated for DB 40°C and RH 60% (34 WBGT). Acclimatized volunteers participated in the study. Initially, experiment was carried out on 2% pre-hypohydrated volunteers. They underwent standard work heat test in HCC with out replenishment. Significant rise in heart rate was observed. Elevation of T-wave as symptom of heat stress was also observed. A progressive rise in oral temperature observed in volunteers during exposures was indicative of the heat stress. Mean skin, and mean body temperature were significantly raised towards the end of exposure.

Experiment was repeated with the same volunteers with intake of different replenishment fluids. Volunteers took replenishment fluid measured for 1% equivalent of their body weight, during the rest time immediately after pre-hypohydration session. In standard work heat test session, the rise in heart rate, elevation of T-wave, and the increase in oral, mean-skin and mean body temperatures were not to the same extent as in the case of initial experiments without replenishment fluids. Out of the replenishment fluids used, Brahmi appears to have some beneficial effect, in reducing heat stress.

In conclusion, appearance of J-wave may be utilized to detect initial hypothermia to avert serious deleterious symptoms that might follow due to cold stress.

Brahmi may be preferred as a good replenishment fluid to counter heat stress. It is hoped that the above findings will be of practical significance to those, working in extreme environments such as Leh, in Himalayas and the desert in Rajasthan.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

REFERENCES

REFERENCE

Adolph EF. (1947) Physiology of man in the desert. *(Adolph EF ed) Interscience, New York.*

Al Mashhadani SA, Gader AG, al Harthi SS, Kangav D, Shaheen FA & Bogus F. (1994) The coagulopathy of heatstroke: alterations in coagulation and fibrinolysis in heatstroke patients during the pilgrimage (Haj) to Makkah. Blood Coagul Fibrinolysis *1994; 5:731-736.*

Alan C, Utter Jie Kang, David C & Nieman. (1999) Effect of carbohydrate ingestion and hormonal responses on ratings of perceived exertion during prolonged cycling and running. *Eur. J. Appl Physiol 80: 92-99.*

Alhaddad I, Khlil M & Brown Jr EJ. (2000) Osborn waves in hypothermia. *Circulation;*101(25):e233-e234.

Alings M & Wilde A. (1999) Brugada syndrome. Clinical data and suggested pathophysiological mechanisms. Circulation, 99: 666-673.

Anguera I, Valls V. Giant. (2000) J waves in hypothermia. *Circulation;*101(13):1627-1628.

Antzelevitch C. (1998) The Brudaga syndrome. J Cardiovasc Electrophysiol, 9: 513-516.

Armstrong LE, Maresh CM, Gabaree CV, Hoffman JR, Kavouras SA, Kenefick RW, Castellani JW & Ahlquist LE. (1997) Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. *J Appl Physiol. 1997 Jun; 82(6): 2028-35.*

Barbara Griefan. (1997) acclimation to three different hot climate equivalent wet bulb globe temperature. Ergonomic, Vol. 40, no.2.223-234

Barcroft H & Edhom OG. (1943) The effect of temperature on blood flow and deep temperature in human forearm. *Jur Physiol (Lond) 102: 105.*

Beller GA & Boyd AE. (1975) Heat stroke: a report of 13 consecutive cases without mortality despite severe hyperpyrexia and neurologic dysfunction. *Mil Med. Jul; 140(7): 464-7.*

Bergersn TK. (1993) A searches for arteriovenous anastomoses in human skin using ultrasound Doppler. *Acta Physiol. Scand. 147: 195-201.*

Bittel JHM. (1987) Heat debt as an index for cold adaptation in man. *Jur. Appl Physiol. 62: 1627-1634.*

Bouchama A, Bridey F & Hammami MM. (1996) Activation of coagulation and fibrinolysis in heatstroke. *Thromb Haemost 1996; 76:909-915.*

Brugada P & Brugada J. (1992) Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome. A multicenter report. *J. Am. Col. Cardiol.*;20(6):1391-1396.

Bud G M. (1974) Physiological research at Australian stations in the Antarctic and Subantarctic, In Human Adaptability to Antarctic Conditions. *Antarctic Research Series, Vol. 22, edited by E. K. E. Gunderson, (Washington, DC: American Geophysical Union), pp. 27-54.*

Budd GM & Warhaft N. (1966) Body temperature, shivering, blood pressure and heart rate during a standard cold stress in Australia and Antarctica. J, Physiol. 186, 216—232.

Budd GM, Brotherhood JR, Hendrie AL & Jefferey SE. (1991) Effects of fitness, fatness and age on men's responses to whole body cooling in air. *Jur. Appl Physiol. 71: 2387-2393.*

Budd GM. (1984) Daly fluid balance, International Biomedical Expedition to the Antarctic (IBEA), poster and abstract no. 20. Presented *at 6th International Conference on Circumpolar, Health, Anchorage, Alaska, 13-18 May.*

Burgess ML, Robertson RJ, Davis JM, Norries JM. (1991) Blood glucose and carbohydrate oxidation during exercise effects of glucose feeding. *Med. Sci. sports Exerc 23: 353-359.*

Burton AC. (1935) Human calorimetry: the average temperature of the tissues of the body. *J. Nutr.* 9: 261-280, 1935.

Cade R. Spooner G, Schlem E, Piekering M & Dean R. (1972) Effect of fluid, electrolyte, and glucose replacement during exercise on performance, body temperature, rate of sweat loss, and compositional changes of extra cellular fluids. *J Sports Med Phys Fitness 12: 150-156.*

Changnon SA & Easterling DR. (2000) Disaster management: U.S. policies pertaining to weather and climate extremes. *Science 2000; 289:2053-2055.*

Chao TC, Sinniah R & Pakiam JE. (1981) Acute heat stroke deaths. *Pathology 1981; 13:145-156.*

Clark RP & Edholm OG. (1985) Man and His thermal environmental. *Edward Arnold, London.*

Costill DL & Sparks KE. (1973) Rapid fluid replacement following thermal dehydration. *J. Med. Physiol .34:299-303.*

Danzi DF & Pozos RS. (1994) Accidental hypothermia. N Engl J Med, 331: 1756-1760.

Danzl Daniel F. (1998) "Disturbances Due to Cold." In *Conn's Current Therapy,* edited by Robert E. Rakel. Philadelphia: W.B. Saunders, 1998.

Danzl Daniel F, Robert S. Pozos, and Murray P. Hamlet. (1995) "Accidental Hypothermia." In *Wilderness Medicine: Management of*

Danzl. (1998) Hypothermia. Rosen: Emergency Medicine: Concepts and Clinical Practice; 4th Edition.

David Jensen. (1924) The principle of physiology. Appleton century crofts. Publishing division of prentice hall INC, USA.

Davis TRA. (1963) Acclimatization to cold in Man. In. Temperature - its measurements and control in science and industry. Part 3 Biology and Medicine. *Edited by C.M. Herzfield and J.D Hardly (New York: Rinhold), pp 443-456.*

Dokkum WV, LBJ Van Dick MJ, Bore LC & Van Der Beek. (1996) Influence of a Carbohydrate Drink on performance of Military personnel in NBC protective clothing. *Aviat Space Environ Med. 67: 819-826.*

DuBois D, and E. F. Dubois. (1916) A formula to estimate the approximate surface area if height and weight be known. *Arch. Intern. Med.* 17: 863-871, 1916.

Edlich RF, Silloway KA & Feldman PS. (1986) Cold injuries and disorders. *current concepts Trauma Care* 1986; .4-11.

El-Kassimi FA, Al-Mashhadani S, Abdullah AK & Akhtar J. (1986) Adult respiratory distress syndrome and disseminated intravascular coagulation complicating heat stroke. *Chest 1986; 90:571-574.*

Elsner, R W, Nellms JD & Irving L. (1960) Circulation of heat to the hands of arctic Indians. *Jur. Appl Physiol. 15: 662-6.*

Fisch C. (1997) Electrocardiography. In Braunwald E, ed: Heart Disease. A Textbook of Cardiovascular Medicine. Fifth Edition. WB Saunders, Philadelphia, p. 136.

Gaffin SL. (1990) Heat stroke, cardiac dysfunction and edema Chest. *Jun; 97(6): 1503.*

Ghaznawi HI & Ibrahim MA. (1987) Heat stroke and heat exhaustion in pilgrims performing the Haj (annual pilgrimage) in Saudi Arabia. *Ann Saudi Med 1987; 7:323-6.*

Giesbrecht G G, Goheen MSL, Johnston CE, Kenny GP, Bristow GK & Hayward JS. (1997) Inhibition of shivering increases core temperature drop and attenuates re-warming in hypothermic humans. *Jur. Appl Physiol. 83: 1630-1634.*

Giesbrecht GG & Bristow GK. (1998) The convective after-drop component during hypothermia exercise decreases with delayed exercise onset. *Aviat Space Environ Med. 69: 17-22.*

Giesbrecht GG, Sessler DI, Mekjavic IB, Schroeder M & Bristow GK. (1994) Treatment of mild immersion hypothermia by direct body- to-body contact. *J Appl Physiol 76: 2373-2379.*

Gisolti CV & Duchman SM (1992) Guidelines for optimal replacement beverages for different athletic events. *Med. Sci Sports Exere. 24: 679-687.*

Gisolti CV, Summers RD, Schedl HP, Bleiler TL & Oppliger RA (1990) Human intestinal water absorption: direct vs. indirect measurements, *AM J. Physiol: G210-G222.*

Goldfinch J, Mc Naughton LR & Davis P. (1988) Bicarbonate indigestion and its effects up on 400-m racing time. *Eur. J Appl Physiol 57: 45-48.*

Gould L, Gopalaswamy C, Kim BS & Patel C. (1985) The Osborn wave in hypothermia. *Angiology. 1985 Feb; 36(2): 125-9.*

Gregory S, and Anderson (1999) Human morphology and temperature regulation. Int. Biometeorol, 43: 99-109.

Gussak I, Antzelevitch C & Bjerregaard P. (1999) The Brugada syndrome: clinical, electrophysiologic and genetic aspects. J Am Coll Cardiol, 33: 5-15.

Gussak I, Bjerregaard P, Egan TM & Chaitman BR. (1995) ECG phenomenon called the J wave: history, pathophysiology, and clinical significance. *J Electrocardiol*. 1995; 28:49-58.

Hardy JD, and E. F. DuBois. (1938) The technique of measuring radiation and convection. *J. Nutr.* 15: 461-475, 1938.

Harrison MH. (1985) Effects of thermal stress and exercise on blood volume in humans. *Physiol. Rev 65: 149-173.*

Hurst JW. (1998) Naming of the waves in the ECG, with a brief account of their genesis. Circulation, 98: 1937-1942.

Jimene C, Melin B, Koulmann N, Charpenet A, Cottet-Emard J, Pequignot JM, Savourey G & Bittel J. (1997) Effects of various beverages on the hormones involved in energy metabolism during exercise in the heat in previously dehydrated subjects. *Eur. J. Appl. physiol. 76: 504-509.*

Kalkstein LS. (2000) Saving lives during extreme weather in summer. BMJ 2000; 321:650-651.

Kalla H, Yan GX & Marinchak R. (2000) Ventricular fibrillation in a patient with prominent J (Osborn) waves and ST segment elevation in the inferior electrocardiographic leads: a Brugada syndrome variant. J Cardiovasc Electrophysiol. 2000; 11:95–98.

Kambara H & Phillips J. (1976) Long-term evaluation of early repolarization syndrome (normal variant RST-T segment elevation). Am J Cardiol, 38: 157-161.

Kellerman AL & Todd KH. (1996) Killing heat. *N Engl J Med 1996; 335:126-127.*

Knochel JP, Reed G, Maxwell & Kleeman. (1994) Disorders of heat regulation. In: Narins RG, ed. Clinical disorders of fluid and electrolyte metabolism. *5th ed. New York: McGraw-Hill, 1994:1549-90.*

Knochel JP. (1990) Catastrophic medical events with exhaustive exercise: "white collar rhabdomyolysis." *Kidney Int 1990; 38:709-719.*

Korg J, Folkow B, Fox RH & Heroux O. (1960). Hand circulation in the cold of lapps and north Norwegian fisherman. *Jur. Appl Physiol. 15: 654*

Korg J. & Wika M. (1978) Studies of hand blood flow of the Igloolik Eskimo. *Med. Biol. 56. 146-186.*

Latzka WA & Sawka MN. (2000) Hyperhydration and glycerol: thermoregulatory effects during exercise in hot climates. *Can J Appl Physiol. 2000 Dec; 25(6): 536-45.*

Latzka William A, Michael N. Sawka, Scott J, Montain, Gary S, Skrinar, Roger A. Fielding, Ralph P. Matott, & Kent B. Pandolf. (1997) Hyperhydration: Thermoregulatory effects during compensable exercise -heat stress. *J.Appl. Physiol. 83 (3): 860-866.*

LeBlanc J, Hildes JA & Heroux O. (1960) Tolerance of Gaspe fishermen to cold water. *J. Appi. Physiol., 15 1031-1034.*

LeBlanc J. (1975) Adaptation In: Man in the Cold. *(Springfield. IL: C.C. Thomas) chapter 5.*

Lehmann KG, Shandling AH & Yusi AU. (1998) Altered ventricular repolarization in central sympathetic dysfunction associated with spinal injury. Am J Cardiol 1989, 63: 1498-1504. 16.

Leithead CS and A. R. Lind (1982) Heat stress and heat disorders. Cassell and & company Ltd. 35 Red Lion Square. Launder W.C.I. Printed in great Britain By chapel River Press Ltd., Andover, Hants.

Litovsky SH & Antzelevitch Ç. (1990) Differences in the electrophysiological response of canine ventricular subendocardium and subepicardium to acetycholine and isoproterenol. A direct effect of acetylcholine in ventricular myocadium. Circ Res, 67: 615-627.

Livingstone SD. (1967) Calculation of mean body temperature. *Can. J. Physiol. Pharmacol.* 46: 15-17, 1967.

Livingstone SD, Nolan RW & Keefe AA. (1989) Changes in cold tolerance during: 100-day polar ski expedition. In Mercer JB (Eds) Thermal physiology. *Elsevier, New York, pp 469-474.*

Livingstone SD. (1976) Changes in cold - induced vasodilatation during Arctic exercises. *J Appl Physiol 40: 455—457.*

Lossius K, Eriksen M, &. Walloe L. (1994) Thermoregulatory flactuations in the heart rate and blood pressure in humans. Effect of cooling and parasympathetic blockade. *J. Auton. Nerv. Syst. 47: 245 -254.*

Lyons TP, Riedesl ML, Meuli LE & Chick TW. (1990) Effects of glycerol-induced hyperhydration prior to exercise in the heat on sweating and core temperature. *Med. Sci. Sports Exere.22: 477-483.*

Mackworth NH. (1953) Finger numbness in very cold winds. *J. Appl. Physiol.* 5: 533-543, 1953.

Majumdar NC. (1973) Indices of heat stress. Defense science laboratory, Delhi, Mehta house, Naraina II, New Delhi

Massett MP, Johnson DG & Kregel KC. (1996) Cardiovascular and sympathoadrenal responses to heat stress following water deprivation in rats. *Am J Physiol. 1996 Mar; 270(3 Pt 2): R652-9.*

Maughan RJ, Owen JH, Shirreffs SH & Leiper JB. (1994) Post-exercise rehydration in man: effects of electrolyte addition to ingested fluids. *Eur J. Appl. Physiol 69: 209-215.*

Miller NC & Seagrave RC. (1974) A model of human thermoregulation during water immersion. Comput Biol. Med 4:165-182.

Mills AW (a). (1956) Finger numbness and skin temperature. *J. Appl. Physiol.* 9: 447-450, 1956.

Mills WJ Jr & R S. Pozos. (1997) Low Temperature Effects on Humans." In *Encyclopedia of Human Biology, edited by Renato Dulbecco. San Diego: Academic Press, 1997.*

Montain SJ, Shippee RL and Tharion. Carbohydrate-Electrolyte solution effects on physical performance of minitary tasks. Aviat. Space and environ Med. Vol. 68. 384-91.

Montain SJ, Sawka MN, Latzka WA & Valeri CR. (1998) Thermal and cardiovascular strain from hypohydration: influence of exercise intensity. *Int J Sports Med. 1998 Feb; 19(2): 87-91.*

Moran DS, Kenney WL, Pierzga JM & Pandolf KB. (2002) Aging and assessment of physiological strain during exercise-heat stress. *Am J Physiol Regul Integr Comp Physiol. 2002 Apr; 282(4): R1063-9.*

Moran DS, Montain SJ & Pandolf KB. (1998) Evaluation of different levels of hydration using a new physiological strain index. *Am J Physiol. 1998 Sep; 275(3 Pt 2): R8 54-60.*

Mount L E. (1974) adaptation to thermal environment man and his productive animals. *Edward Arnold, London.*

Mudambo KSMT, Leese GP, Rennie MJ. (1997) Dehydration in soldiers during walking /running exercise in the heat and the effects of fluid ingestion during and after exercise. *Eur.J.Appl. Physil 76:517–524.*

Nadel ER, Fortney SM & Wenger CB. (1980) Effect of Hydration State on circulatory and thermal regulations. *J.Appl.Physiol .49: 715-721.*

Nadel ER, Mack GW & Nose H. (1990) Influence of fluid replacement beverages on body fluid homeostasis during exercise and recovery. Perspective in Exercise science and Sports Medicine. Fluid Homeostasis during Exercise, *edited by C. V. Gisolfi and D.R. Lamb. Carnel, IN: Benchmark, 1990, vol.3. Chapt.5, 181-205.*

Naidu M. and Sachdeva U. (1993) Effect of local cooling on skin temperature and blood flow of men in Antarctica. *Biometeriology 7: 218-221.*

Nelson N, Eichna LW, Horvath SM, Shelley WB & Hatch TF. (1947) Thermal exchanges of man at high temperatures. *Am J Physiol 1947; 151:626-652.*

Noakes TD (1993) Fluid replacement during exercise. *Exere Sports Rev 21:297-330.*

Norman J N. (1961) Microclimate of man in Antarctica. *Jur. Appl Physiol. 160: 27-28.*

Oberg P A, Temand T & Nilsson GF. (1983). Lesser Doppler flowmetry a non-invasive and continuous method for blood flow evaluation in microvascular studies. *Acta Med Scand. Suppl 687: 17-24.*

Paul Auerbach, Edward Geehr & Macmillan (1983) Management of Wilderness and Environmental Emergencies, 1983.

Peter Tikuisis, Gordon G & Giesbrecht. (1999) Prediction of shivering heat production from core and mean skin temperatures. Eur. *J, Appl. Physiol 79: 221–229.*

Petty Kevin J. (1998) "Hypothermia." In *Harrison's Principles of Internal Medicine,* edited by Anthony S. Fauci, et al. New York: McGraw-Hill, 1998.

Pichan Ganesh, Baburaj TP, Gulab Singh, Bajaj AC, Sreedhran K. (1999) Human performance under various stress conditions. *DIPAS. Reoprt No: 13 / 96.*

Pozos Robert and David Born. (1982) Hypothermia: Causes, Effects, and Prevention, New Century, 1982.

Ramanathan NL. (1964) A new weighing system for mean surface temperature of human body. *J Appl Physiol 19:nn531-533.*

Rastogi P, Ram & Mehrotra BN. (1993) Compendium of Indian medicinal plants. *Publication information directorate. Delhi. Vol 2.*

Ream AK, Reitz BA & C. (1982) Temperature correction of Pco2 and pH in estimating the acid base status: An example of the emperor's new clothes? *Anesthesiology 1982; 56:41-44.*

Regan JM, Macfarane DJ, Taylor NAS. (1996) The role of body core and skin temperatures during heat adaptation. *Acta Physiol Scand 158: 365-375.*

Riebe D, Marshal CM, Amstrong LE, Kenefick RW, Castellani JW, Echegaray M, Clark BA & Camoione DN. (1997) Effect of oral and intravenous rehydration on rating of perceived exertion and thirst. *Med.Sci. Sports. Exerc. Vol.29.No1 pp.117-124.1997.*

Rivolier J, Gold smith R, Lugg DJ & Tylor AJW (Eds) (1988). Man in Antarctica. *Taylor and Francis, Philadelphia. pp 105 - 147.*

Rowell LB. (1974) human cardiovascular adjustment to exercise and thermal stress. *Physiol. Rev 54:75-157.*

Rowell LB. (1983) Cardiovascular aspects of human thermoregulation. *Circ Res 1983; 52:367-379.*

Schafler AE, Kirmanoglou K, Pecher P, Hannekum A & Schumacher B. (2002) Overexpression of heat shock protein 60/10 in myocardium of patients with chronic atrial fibrillation. *Ann Thorac Surg. Sep; 74(3): 767-70.*

Schaller MD, Fischer AP, Perret CH: Hyperkalemia: A prognostic factor during acute severe hypothermia. *JAMA* 1990; 264:1842-1845.

Schamroth L. (1982) An introduction to electrocardiography. Sixth Edition. Blackwell, Oxford, p. 139.

Schneider SM. (1992) Hypothermia. From recognition to rewarming. *Emergency Medicine Reports* 1992; 13:1-20.

Scholander PF, Hammel HT, Hart JS, Le Messurier DH. & Steen J. (1958) Cold adaptation in Australian Aborigines. *Jur. Appl Physiol. 13 (2), 211-218.*

Shirreffs SM & Ronald J Maughan. (1997) Restoration of fluid balance after exercise-induced dehydation: effects of alcohol consumption. *J. Appl Physiol 83(4): 1152-1158.*

Southwick FS & Dalgish PH: (1980) Recovery after prolonged asystolic cardiac arrest in profound hypothermia: A case report and literature review. *JAMA 1980; 243:1250-1253.*

Tam HS Darling RC, Cheh HY & Downey JA. (1978) Sweating response: a means of evaluating the set-point theory during exercise. *J. Appl Physiol 45: 451-458.*

Tanaka M. (1971) Experimental studies on human reaction to cold. *Bull Tokyo Med Dental Univ 16: 169-177.*

Teichner WH. (1957) Manual dexterity in the cold. *J. Appl. Physiol.* 11: 333-338, 1957.

The Backpacker's Field Manual. (1998) Rick Curtis, Random house, New York, 1998.

Van Someran, R. N. M., S. R. K. Coleshaw, P. J. Mincer, and Keatinge WR. (1982) Restoration of thermoregulatory response to body cooling by cooling hands and feet. *J. Appl. Physiol.* 53: 1228-1233, 1982.

Vinje T. (1961) The cooling power in Antarctica. In: *Norsk Polar Institut Arbok (Oslo:Norsk Polar Institute), pp. 7-22.*

Weatherall DJ, Lendingham JGG and Warrell JDD. (1987) Oxford textbook of medicine, Vol-II, Second Edition, ELBS. Oxford university Press.pp-12.

Weinberg AD, Hamlet MP & Paturas JL. (1990) Cold Weather Emergencies: Principles of Patient Management. *Branford, Connecticut, American Medical Publishing Co. 1990. p 10-30.*

Wyndham CH & Loots H. (1969) Responses to cold during a year in Antarctica. *Jur. Appl Physiol. 27: 696-700.*

Yan GX & Antzelvitch C. (1996) Cellular basis for the electrocardiographic J wave. *Circulation.* 1996; 93:372-379.

Yoshimura H, and T. Iida. (1952) Studies on the reactivity of skin vessels to extreme cold. Part II: factors governing the individual difference of the reactivity or the resistance against frost-bite. *Jpn. J. Physiol.* 2: 177-185, 1952.

Yoshimura H & Lida T. (1950) Studies on the reactivity of skin vessels to extreme cold. Part 1. A point test on the resistance against frostbite. *Jpn J Physiol 1: 147 -159.*

Young AJ, Muza sr, Sawka MN, Gonzalez RR, Pandof KB. (1986). Human thermoregulatory responses to cold air are altered by repeated cold water immersion. *Jur. Appl Physiol 60: 1542-1548.*

Zahger D, Moses A and Weiss AT. (1990) Evidence of prolonged myocardial dysfunction in heat stroke. Chest. 1990 Jun;97(6):1503.

Zell S & Kurt K (1985) Severe exposure hypothermia: A resuscitation protocol. *Ann Emerg Med* 1985; 14:339-345.

SOME ASPECTS OF THE PHYSIOLOGY OF TEMPERATURE INDUCED STRESS IN HUMAN VOLUNTEERS WITH PARTICULAR REFERENCE TO CARDIOVASCULAR SYSTEM

Thesis submitted to the University of Calicut in partial fulfilment of the requirements for the award of the degree of

DOCTOR OF PHILOSOPHY IN PHYSIOLOGY

by

Baburaj. T.P.

Defence Institute of Physiology and Allied Sciences
Lucknow Road, Timarpur, Delhi 110 054

and
Department of Life Sciences
University of Calicut
Kerala
INDIA
2004

APPENDIX – I

TABLE- 1

Heart rate (beats per min.) during down-sail and up-sail

Latitude	HR
ZERO-LAT (Down-sail)	85 ±6.35
70°-S-LAT (Antarctica)	76 ±6.5
40°-S-LAT (Up-sail)	75 ±7
20°-S-LAT (Up-sail)	78 ±6
ZERO-LAT (Up-sail)	81 ±4.8

TABLE - 2

Heart rate (beats per min.) during 60 min. out door exposure In last lap of stay in Antarctica

Duration of exposure	HR (Avg)	STDV
0 – min	75	± 4.89
20 – min	75	± 5.64
40 – min	75	± 5.85
60 – min	77	± 5.75

TABLE- 3

Body temperature (°C) during down-sail and up-sail

Latitude	OT	TS	MBT
ZERO-LAT (Down-sail)	36.67 ±0.30	30.43 ±0.52	34.73 ±0.26
70°-S-LAT (Antarctica)	36.02 ±0.41	28.35 ±1.33	34.41 ±0.51
40°-S-LAT (Up-sail)	36.62 ±0.30	31.56 ±0.63	35.56 ±0.21
20°-S-LAT (Up-sail)	36.46 ±0.78	31.80 ±0.48	35.48 ±0.59
ZERO-LAT (Up-sail)	36.62 ±0.43	31.88 ±0.34	35.62 ±0.31

OT = Oral Temperature
TS = Mean-skin temperature
MBT = Mean body temperature

TABLE 4

Body temperature (°C) during 60 min. out door exposure in last lap of stay

Duration of exposure	OT	TS	MBT
0 – min	35.63 ± 0.45	29.48 ±1.94	31.49 ±0.27
20 – min	35.85 ±0.60	28.89 ±1.33	31.52 ±0.42
40 – min	35.77 ±0.62	28.58 ±1.50	31.40 ±0.43
60 – min	35.70 ±0.59	28.35 ±1.51	31.30 ±0.42

OT = Oral Temperature
TS = Mean-skin temperature
MBT = Mean body temperature

TABLE - 5

Peripheral temperature (°C) during down-sail and up-sail

Latitude	FH	DOR	ARM	FING	FOOT	TOE
ZERO-LAT (Down-sail)	32.88 ±0.34	32.76 ±1.50	31.68 ±0.29	30.97 ±1.05	33.65 ±1.90	31.08 ±1.70
70°-S-LAT (Antarctica)	30.92 ±1.97	31.00 ±2.18	29.72 ±3.90	20.12 ±7.59	22.65 ±5.05	17.75 ±6.28
40°-S-LAT (Up-sail)	32.35 ±0.85	31.10 ±1.76	32.47 ±0.89	29.32 ±3.43	30.80 ±2.40	28.63 ±4.12
20°-S-LT (Up-sail)	32.73 ±1.00	31.23 ±1.81	32.96 ±0.85	32.27 ±1.99	30.58 ±1.70	30.14 ±2.74
ZERO-LAT (Up-sail)	32.52 ±0.46	32.40 ±1.21	32.78 ±0.66	31.37 ±5.00	30.27 ±0.82	29.13 ±2.81

FH = Fore Head temperature
DOR = Dorsal temperature
ARM = Arm temperature
FING = Finger temperature
FOOT = Foot temperature
TOE = Big toe temperature

TABLE - 6

Peripheral temperature (°C) during 60 min. out door exposure in last lap of stay

Duration of exposure	FH	DOR	ARM	FING	FOOT	TOE
0 - min	30.1 ±2.71	30.6 ±1.03	31.8 ±2.20	25.0 ±2.03	15.9 ±3.45	16.5 ±2.10
20 - min	31.5 ±2.91	29.8 ±1.92	31.6 ±2.25	23.9 ±2.21	16.1 ±6.40	16.5 ±2.73
40 - min	31.7 ±2.53	29.7 ±2.06	31.4 ±1.98	22.6 ±2.30	15.7 ±5.66	16.7 ±3.41
60 - min	32.5 ±2.14	30.1 ±2.40	31.0 ±1.88	21.8 ±1.89	14.6 ±4.56	16.6 ±3.62

FH = Fore Head temperature
DOR = Dorsal temperature
ARM = Arm temperature
FING = Finger temperature
FOOT = Foot temperature
TOE = Big toe temperature

TABLE - 7

Sweat rate (gm/m^2/hr) during 60 min. out door exposure in last lap of stay

Volunteers	SL (gm)	BSA (m^2)	SR (gm/m^2/hr)
YKM [M]	60	1.50	40.00
RKD [M]	100	1.95	51.28
AR [M]	40	1.65	24.24
PK [M]	100	1.60	62.50
RTK [M]	80	1.58	50.63
KK [F]	100	1.70	58.82
mean	80.00	1.66	47.91
SD ±	25.30	0.16	13.95

SUB = Subject
SL = Sweat Loss
BSA = Body surface area
SR = Sweat rate

Table: 8

Heart rate (beats per minute) during standard work heat test with out fluid

Duration of exposure	HR
REST 0	76 ± 5.2
REST 20	98 ± 6.2
EXE 10	130 ± 8.9
EXE 20	138 ± 9.6
EXE 30	147 ± 13.9
EXE 40	160 ± 13.3

Body temperature(°C) during standard work heat test with out fluid

Duration of exposure	OT	TS	MBT
REST 0	36.8 ± 0.26	35.71 ± 0.45	36.56 ± 0.22
REST 20	37.2 ± 0.21	35.92 ± 0.38	36.92 ± 0.25
EXE 10	37.3 ± 0.16	36.15 ± 0.45	37.04 ± 0.20
EXE 20	37.4 ± 0.27	36.18 ± 0.35	37.12 ± 0.26
EXE 30	37.7 ± 0.29	36.34 ± 0.20	37.41 ± 0.25
EXE 40	38.0 ± 0.26	36.41 ± 0.20	37.67 ± 0.23

Sweat rate ($gm/m^2/hrs$) during standard work heat test with out fluid

	Sweat rate
Mean	418.00
SD	±85.46

TABLE: 9

Heart rate during standard work heat test with fluid in take (Beats/minute)

Duration of exposure	NAN	WATER	BRAH	ELECT	ROOH	ERR V
20 RES	92	83	91	80	95	148.86
10 EXE	123	120	127	118	132	173.50
20 EXE	135	132	133	130	136	141.10
30 EXE	136	143	139	134	144	177.01
40 EXE	145	151	144	138	152	128.00

NAN :	**Nanari ....**	**A**
WATER:	**Water.........**	**B**
BRAH :	**Brahmi.......**	**C**
ELECT :	**Electral......**	**D**
ROOH:	**Rooh Afza...**	**E**
ERR V:	**Error Variance**	

INTER FLUID COMPARISON DURING STANDARD WORK HEAT TEST

Duration of exposure	A vs B	A vs C	A vs D	A vs E	B vs C	B vs D	B vs E	C vs D	C vs E	D vs E
20 RES	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
10 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
20 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
30 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
40 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

NS = Non significant.

TABLE: 10

Oral temperature (°C) during standard work heat test with fluid in take

Duration of exposure	NAN	WATER	BRAH	ELECT	ROOH	ERR V
20 RES	36.7	36.9	37.0	36.9	37.2	0.04
10 EXE	36.9	37.0	37.0	37.1	37.8	0.28
20 EXE	37.2	37.2	37.2	37.2	37.5	0.01
30 EXE	37.5	37.6	37.5	37.4	37.6	0.04
40 EXE	37.7	37.9	37.7	37.7	37.9	0.02

NAN : Nanari .... A
WATER: Water......... B
BRAH : Brahmi....... C
ELECT : Electral...... D
ROOH: Rooh Afza... E
ERR V: Error Variance

INTER FLUID COMPARISON DURING STANDARD WORK HEAT TEST

Duration of exposure	A vs B	A vs C	A vs D	A vs E	B vs C	B vs D	B vs E	C vs D	C vs E	D vs E
20 RES	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
10 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
20 EXE	NS	NS	NS	P<0.1	NS	NS	P<.01	NS	P<.01	P<.01
30 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
40 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

NS = Non significant.

TABLE: 11

Mean skin temperature (°C) during standard work heat test with fluid in take

Duration of exposure	NAN	WATER	BRAH	ELECT	ROOH	ERR V
20 RES	35.38	35.71	35.53	35.90	35.85	0.06
10 EXE	35.59	35.71	35.44	35.44	36.29	0.17
20 EXE	35.89	35.71	35.60	35.60	35.16	0.10
30 EXE	35.69	36.10	35.82	35.82	36.23	0.17
40 EXE	35.85	36.13	35.88	35.88	36.31	0.15

NAN :	Nanari	A
WATER:	Water.........	B
BRAH :	Brahmi.......	C
ELECT :	Electral......	D
ROOH:	Rooh Afza...	E
ERR V:	Error Variance	

INTER FLUID COMPARISON DURING STANDARD WORK HEAT TEST

Duration of exposure	A vs B	A vs C	A vs D	A vs E	B vs C	B vs D	B vs E	C vs D	C vs E	D vs E
20 RES	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
10 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
20 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
30 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
40 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

NS = Non significant.

TABLE: 12

Mean body temperature (°C) during standard work heat test with fluid in take

Duration of exposure	NAN	WATER	BRAH	ELECT	ROOH	ERR V
20 RES	36.45	36.65	36.66	36.72	36.88	0.05
10 EXE	36.66	36.73	36.69	36.81	37.09	0.16
20 EXE	36.89	36.90	36.90	36.91	37.21	0.10
30 EXE	37.10	37.27	37.15	37.08	37.31	0.13
40 EXE	37.33	37.51	37.29	37.32	37.58	0.14

NAN :	Nanari	A
WATER:	Water.........	B
BRAH :	Brahmi.......	C
ELECT :	Electral......	D
ROOH:	Rooh Afza...	E
ERR V:	Error Variance	

INTER FLUID COMPARISON DURING STANDARD WORK HEAT TEST

Duration of exposure	A vs B	A vs C	A vs D	A vs E	B vs C	B vs D	B vs E	C vs D	C vs E	D vs E
20 RES	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
10 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
20 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
30 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
40 EXE	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

NS = Non significant.

TABLE: 13

Sweat rate (gm/sq m/h) during standard work heat test with fluid in take

FLUID	SWEAT RATE (MEAN)	STANDARD DEVIATION
Nanari	453.38	±101.582
Water	469.8	±55.336
Brahmi	424.02	±62.044
Electral	438.96	±108.739
Rooh Afza	449.48	±65.580

Statistically non-significant

APPENDIX – II

BEDFORD "COMFORT VOTE" TECHNIQUE

GRADING SCALES

Record comfort as -3 to +3 corresponding to

+3 Much too warm :
+2 just warm : **Too**

+1 comfortable warm :
0 Neutral : **C**
-1 Comfortably cool :

-2 Just cool : **Too**
-3 Much cool :

Response of all volunteers : -2

Individual items on card

Comfort grade -3 to +3 for each of the four sites : ***trunk, face, hand, feet.***

Numbness, pain. Grade 0 to 3 for each of the above four sites

Individual item points were marked -1 to 0 in the above mentioned four items

Sweating, Shivering. Grade 0 to 3 for your overall response.

Individual item points were marked 1 to 2 in the above mentioned four items

Remarks Note cold spot or other relevant information.

There was no cold spot reported

Activity; Describe in words what you have been doing in the past ten minutes (e.g. Sitting, walking, strenuous exercise), and record numerically (after Effort) Your estimate of level of exhaustion, ranging form 0 (minimal exertion eg. Sitting or standing) to 3 (strenuous exhaustion Example shovelling running.)

Activity recorded : 0

Place (O/I) 'O/I' Short hand for 'out doors/indoors' cross out which ever does not apply, and record (e.g. in tent, caravan, etc.0. After How long/. Record duration of your presence indoors or out doors, if you are at out door, note whether 'in sun' or not (Ø). If you are in sunlight, whether it is week (1), Moderate (2), or strong (3).

Place O, 1 and 2

Hourly comfort vote card

Time: F/N Activity: Effort:
Place: (I/O): How long ? ...1.h ...00..min In sun...1 & 2

	Trunk	Face	Hand	Feet
Comfort				
Cold	*	*	*	*
Numbness		*	*	*
Pain				*

Sweating: NO
Shivering: NO
Clothing Changes. NO

APPENDIX – III

POLAR CLOTHING ENSEMBLE

DMSRDE ensemble

Trunk		**Hand**	
Long Johns		Mitts, win/proof	
Pyjamas		Mitts, Wool (A,F)	
Trousers	Y	Wristlets	
Down pants		Gloves lined Leather.	Y
(A,F)		Glove unlined Leather.	
Wind proof pants		Gloves, wool	y
Singlets		Gloves, silk	
Shirts		**Feet**	
Sweats		Mukluks	
Down parks(A,F)		Vapour barrier boots	
Wind proof park (I/O)	Y	Leather boots	
Vest	y	Camp boots	
Band		Sox, wool	Y
Head		Mukluk inners	
Cap			
Balaclava (cap, Hel)			
Blizzard mask			
Parka hood up	y		
Scarf			

Other items: NIL