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THE EFFECTS OF CARBON DIOXIDE ON SLEEP AND THERMOREGULATION IN COLD ENVIRONMENTS

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"The trouble really began in your sleeping-bag, for it was far too cold to keep a hole open through which to breathe. So all night long our breath froze into the skins, and our respiration became quicker and quicker as the air in our bags got fouler and fouler: it was never possible to make a match strike or burn inside our bags![1]"

Apsley Cherry-Garrard,

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1 Excerpt from A. Cherry-Garrard. (1965). The worst journey in the world. Antarctic 1910-1913. Chatto & Windus: London. p 238.

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DEFINITIONS

T_x	Temperature (°C), where subscript x is one of the following: a - ambient sk - mean skin r - rectal ty - tympanic b - mean body
F_iCO_2	Fraction of inspired carbon dioxide. Expressed as a decimal fraction.
P_xCO_2, P_xO_2	Partial pressure of carbon dioxide and oxygen (mmHg), where x is: a - arterial v - mixed venous A - alveolar I - inspired ET - end tidal
POAH	Pre Optic Anterior Hypothalamus. The major thermoregulatory control centre in the brain.
S_aO_2	% saturation of arterial haemoglobin with O_2
Hypercapnia	Any amount of carbon dioxide in excess of normal levels.
NREM	General classification of sleep stages 1 to 4.
REM	Sleep stage characterized by rapid eye movements and disruption of autonomic outflow.
TST	Total sleep time. The total amount of sleep from the first sleep onset until final awakening.
SE	Sleep efficiency. Total sleep time divided by the total time after initial sleep onset
SOL	Sleep onset latency. The time from lights out until the initial onset of sleep

ABSTRACT

In order to study the effects of mild hypercapnia on sleep and thermoregulation, 5 male volunteers (23.6 $\pm$ 1.96 yrs) were exposed to air containing 0, 2, or 4% CO_2, while sleeping in a double (9.0 clo) or single (4.5 clo) Canadian Forces sleeping ensemble (1 clo = $0.155°C \cdot m^2 \cdot W^{-1}$) at -20°C. Each condition was presented twice in a completely randomized manner on non-consecutive nights. Standard polysomnographic EEG, EMG and EOG measures were monitored as well as rectal (T_r), mean skin ($\bar{T}_{sk}$) and toe (T_{toe}) temperatures. Hypercapnia was associated with enhanced body cooling as indicated by a decreased time to minimal T_r and T_{toe} ($p < .05$). In agreement with current knowledge of sleep in cold environments, sleeping in the single bag resulted in a significantly decreased percentage of REM sleep ($p < .05$) and trends towards decreases sleep efficiency and total sleep time (TST). Slow wave sleep (%SWS) tended to increase in the single bag condition. CO_2 exposure was associated with a trend towards decreased TST and suppression of the cold induced increase in %SWS. The possible effects of body temperatures being mediated through sleep processes as opposed to direct CO_2 effects, and the possible importance of SWS on thermoregulation were discussed.

CHAPTER I

INTRODUCTION

It has been long maintained by both winter sportsmen and the military, that when sleeping in a sleeping bag, one should not draw one's face into the bag, even in the extreme cold (Cottle and Livingstone, 1986). The rationale for this recommendation is that moisture from the exhaled breath may condense in the insulation of the bag, reducing its effectiveness. Moisture accumulation in the insulation of sleeping bags can be significant, increasing the weight by approximately 300 grams over a trek of 4 days (Osczevski, 1983). However, it has been observed that subjects participating in sleeping bag experiments will involuntarily withdraw their heads inside during the night only to emerge again prior to waking. This is done despite being instructed not to do so (S. Livingstone, personal communication, April, 1988). Such an involuntary action may be a protective mechanism to remove the cold stress from the face and upper respiratory tract, reducing the associated discomfort. Furthermore, by breathing the warm, humidified air inside the sleeping bag, respiratory heat loss is reduced. Given that respiratory heat loss may account for up to 25% of one's metabolic rate at rest in the cold (Cain et al., 1990), this strategy could dramatically reduce cold stress. By increasing thermal comfort through these strategies, an increased quality and quantity of sleep may be achieved as cold temperatures are known to have a detrimental effect on sleep (Buguet et al., 1976b; Palca et al., 1986).

Apart from the accumulation of moisture from exhaled breath, there is potential for an additional problem associated with breathing inside a sleeping bag, namely, such an enclosed environment might result in rebreathing expired gases, thereby affecting the composition of the

atmospheric gases. This has been investigated by Livingstone et al. (1988) who demonstrated that rebreathing does occur. They reported levels of ambient O_2 and CO_2 changing and levelling off at 16% and 4%, respectively. A partial inspired pressure of O_2 (P_IO_2) of 16% at sea level is equivalent to a P_IO_2 of 121 mmHg, which has a minimal effect on arterial O_2 saturation. Phillipson and Bowes (1986) reviewed a number of articles which consistently illustrated no disruption of sleep under hypoxia until S_aO_2 decreased to 70 - 80%. However, increasing CO_2 levels as little as 4% has been reported to increase respiratory rate and volume (Reynolds et al., 1972) as well as to disrupt thermoregulatory (Bullard and Crise, 1961) and sleep (Schaefer, 1958) processes. As the degree of hypoxia measured by Livingstone et al. (1988) has not been reported as being a factor of immediate concern, the scope of this investigation and literature review will be limited to physiological effects of increased carbon dioxide levels on sleep and thermoregulatory function.

Winter camping can be an arduous undertaking, requiring physical strength, endurance, and keen judgement and decision making abilities to ensure the well being of oneself and others. Paramount to performing at optimal capacity is the importance of obtaining adequate sleep of sufficient quantity and quality. The detrimental effects of deprivation of total sleep, or its specific stages, on physical, as well as behavioral and cognitive activity have been well documented (as reviewed by Johnson, 1979). Additionally, impairment of thermoregulation has been demonstrated in REM deprived rats (Rechtschaffen et al., 1983). In order to enable adequate sleep in cold environments while avoiding these sleep impairments, insulation is required to provide a thermally comfortable microenvironment. However, even with adequate insulation, it is conceivable that the tendency to sleep with one's head in the sleeping bag with a subsequent build up of carbon dioxide may present unfavourable conditions necessitating changes in current sleeping bag design. Recently, there has been an increase in the popularity of sleeping bags

which have a water vapour impermeable inner liner. The intent of this liner is to prevent the movement of perspiration into the bag's insulation. These liners are also highly impermeable to O_2 and CO_2 as well (Osczevski, 1992). Livingstone et al. (1988) demonstrated that subjects who pulled their head inside these bags could not remain for more than 30 to 40 minutes, as the hypoxia and hypercapnia reached intolerable levels. Thus, it is apparent that the production of mild hypercapnia and hypoxia associated with sleeping with one's head in sleeping bag introduces a problem of both theoretical and practical importance.

STATEMENT OF THE PROBLEM

A review of the related literature has revealed that there is a potential for mild hypercapnia to disrupt normal sleep and thermoregulatory mechanisms. Yet, there is a paucity of literature directly addressing the interactive effects of these variables. A detailed investigation into this question would result in information of significant interest and value to the scientific and general population.

As a result, this investigation was conducted in order to determine the effects of mild hypercapnia on human thermoregulation during sleep in a cold environment. Thermoregulatory responses were represented by measurements of skin and rectal temperatures and indirect calorimetry in response to breathing 0%, 2%, and 4% CO_2 in 21% O_2, balance N_2 gas. Evaluation of sleep parameters was performed through the standard interpretation of EEG recordings according to Rechtschaffen and Kales (1968).

SUBPROBLEM

Additionally, a secondary concern of the effect of different degrees of cold stress on thermoregulatory response during hypercapnia was addressed. This was accomplished by exposing subjects to two levels of insulation which resulted in either a thermoneutral or cold environment.

ASSUMPTIONS AND LIMITATIONS

Temperature and metabolic data are representative of the storage, loss and production of heat in the body, and are the parameters typically measured in literature examining heat balance in mammals. Studies which discuss the relevance of metabolic rates and body temperatures to thermoregulatory processes include those by Heller and Glotzbach (1977), Parmeggiani (1980), and Haskell et al. (1981a).

Factors external to the thermal environment such, as a face mask, thermistors, electrodes and fans, have not been shown to significantly disrupt sleep. While it is well known that some stressors such as light, or noise may interfere with normal sleep, no mention of sleep disturbance due to these aforementioned factors has been made in comparable research (Buguet et al., 1976b; Henane et al., 1977; and Haskell et al., 1981a). Furthermore, it was perceived that the efforts made to ensure the subjects' physical comfort, as outlined in the methodology, would minimize these external factors.

Cold acclimation was not an influence on body temperatures over the duration of the experiment as a result of the randomization of exposure to the experimental conditions and by allowing a

minimum of one day recovery between exposure to the cold environment . Furthermore, it is believed that time period between between exposure to the experimental conditions was not too long, so as to prevent reorientation to the sleeping environment. As a result, the subject was exposed to the cold environment a maximum of three times per week for 4 to 6 weeks.

Differences in sleep or thermoregulatory responses were primarily due to the manipulation of the independent variables (CO_2 or double/single sleeping bag). As many factors as possible which may have disrupted sleep were controlled by the design of the experiment. These controls are detailed in the methodology section.

EXPERIMENTAL DELIMITATIONS

Physiological parameters obtained in this study were gathered under controlled conditions using non-invasive techniques. Despite every effort to minimize the intrusiveness of the experimental apparatus, it was obvious that it was impossible to duplicate conditions found in the field. For these reasons, extrapolation of the results of this experiment beyond the boundaries of this experiment must be made with this knowledge in mind.

HYPOTHESIS I

Based on a comprehensive review of literature, it was hypothesized that increasing the percentage of inspired carbon dioxide from 0% to 4% in air will adversely affect thermoregulation during sleep in a thermally comfortable environment.

HYPOTHESIS II

Altering the thermal stress between a thermally comfortable and cold environment will result in further disruption of thermoregulatory processes during sleep. For example, as thermal stress is increased, any impairment of the thermoregulatory system will be exacerbated.

CHAPTER II

CARBON DIOXIDE IN THE BODY

Introduction

Carbon dioxide is present in the atmosphere at 0.03% and is fairly inconsequential to normal physiological function. The primary source of CO_2 in the body is the production of CO_2 as a byproduct of metabolism. Since an increased partial pressure of CO_2 (PCO_2) in bodily tissue and fluids may result in a disruption of acid/base balance, it is important to remove excessive CO_2 as effectively as possible. When the production of CO_2 exceeds the ability to remove it, one enters a state of acidosis.

Although the balance between metabolic production and the removal of CO_2 by the lungs is usually responsible for the maintenance of the body's PCO_2, it is entirely plausible that external sources of carbon dioxide could impose hypercapnic loading. Humans subjected to enclosed spaces with rather poor ventilation are likely to experience elevated levels of CO_2. Some examples of these environments include submarines, spacecraft, mines, breweries and in fire fighting. These environmental sources provide an additional challenge by increasing the fraction of inspired CO_2 (F_ICO_2). Subsequently, alveolar PCO_2 increases, resulting in respiratory acidosis.

The purpose of this section is to review the relevant basic physiological mechanisms by which CO_2 is handled by the body, and the effects of hypercapnia on systems relevant to this

experiment.

Basic physiology

Carbon dioxide is continually produced in all metabolically active tissue during the process of metabolism. As the intracellular levels of CO_2 rise, it quickly diffuses into the interstitial spaces and then to the blood stream. This is accomplished very rapidly since CO_2 has a very high diffusion coefficient (roughly 20 times that of O_2). Due to this high diffusibility, a very low pressure gradient of 1 to 5 mmHg is maintained between the intracellular, interstitial and intravascular compartments. Mixed venous blood normally has a PCO_2 of 46 mmHg, compared to a normal alveolar PCO_2 of only 40 mmHg. This gradient is sufficient to permit rapid removal of CO_2 from the venous system.

Carbon dioxide levels in the blood are closely regulated primarily due to the process of the combination of CO_2 with amine groups or hydration-dissociation resulting in the formation of H^+ ions. These processes will be described in greater detail in subsequent sections. Without regulation, an increase in arterial PCO_2 would result in a decreased arterial pH, which is associated with the inhibition of metabolism and disruption of enzyme activity. Excess CO_2 in the blood may arise from either metabolic sources in response to extreme physical demands, or any respiratory insufficiency which decreases the alveolar ventilation (V_A) to carbon dioxide production (VCO_2) resulting in an increased VCO_2/V_A ratio. Inhalation of hypercapnic gas is an further means of increasing alveolar and arterial PCO_2 which is of particular relevance to this investigation.

As mentioned previously, the usual carbon dioxide percentage of the atmosphere is

essentially negligible (0.03%). By increasing the F_ICO_2 to only 4%, the ambient PCO_2 is raised to 30.4 mmHg when the atmospheric pressure is the standard 760.0 mmHg. This greatly diminishes the venous/alveolar CO_2 gradient resulting in an increased P_ACO_2. In this situation, respiratory acidosis ensues, and other physiological mechanisms which maintain normal levels of CO_2 in the blood undergo greater strain on their functional capabilities.

Maintenance of normal levels of CO_2 in the blood

Physically dissolved

Just as the membrane permeability coefficient for carbon dioxide is approximately 20 times greater than O_2, CO_2 is also about 20 times more soluble in aqueous solutions. For this reason, the amount of CO_2 which is dissolved in blood plasma can be considerable. The diffusion coefficient for CO_2 is 0.7 ml $CO_2 \cdot L^{-1} \cdot mmHg^{-1}$. Given a normal arterial PCO_2 of 40 mmHg, approximately 28 ml of CO_2 is physically dissolved in each litre of plasma. This amounts to about 5% of the total carbon dioxide carried in arterial blood.

Carbamino compounds

Proteins are able to effectively and reversibly bind CO_2 to their amine groups. Plasma is a 7% solution of plasma proteins such as albumin, globulin and fibrinogen while red blood cells are comprised of about 30% haemoglobin (Hb). The reaction by which carbon dioxide is bound to the amine group is as follows: $R\text{-}NH_2 + CO_2 \rightleftarrows R\text{-}NHCOO^- + H^+$. Due to its pK of 6, the reaction proceeds almost completely to the right. Therefore, although being an effective process for removing CO_2 from the plasma, almost one H^+ ion is formed for every CO_2 bound,

lowering arterial pH. As with CO_2 in solution, approximately 5% of the plasma carbon dioxide is bound in the form of a carbamino compound. Combination of carbon dioxide molecules with haemoglobin results in the formation of carbaminohemoglobin. This union results in the displacement of O_2 molecules and a right shift of the oxygen-haemoglobin dissociation curve. Thus, a slight arterial hypoxia occurs as P_aCO_2 increases.

Bicarbonate ions

By far the most effective method by which CO_2 is carried in the blood is in the form of HCO_3^- ions. This method accounts for up to 90% of all carbon dioxide molecules transported in the arterial blood. The process by which CO_2 molecules are converted to HCO_3^- ions is one of hydration-dehydration and dissociation-association in the following manner:

$$CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows HCO_3^- + H^+$$

Again, the pK of this system is 6.1 so that almost all of the H^+ ions will need buffering if the pH of the blood is to not change significantly. The association-dissociation reaction proceeds very rapidly, however, the hydration-dehydration reaction would proceed very slowly without the catalytic help of the enzyme carbonic anhydrase. Red blood cells are the primary site where this reaction occurs. The reasons for these cells being an ideal location for the rapid proceeding of this reaction are threefold. The first is the presence of carbonic anhydrase to accelerate dehydration. Secondly, Hb is an effective buffer of H^+ ions, rapidly removing them from the intracellular space. And finally, HCO_3^- ions move from the cells to the plasma, allowing the reaction to proceed to the right. In order to compensate for the loss of negative charges and decreased membrane potential, Cl^- ions move into the intracellular space from the

plasma resulting in what is know as the Chloride Shift. Due to these three mechanisms, great quantities of CO_2 can be carried in the blood without a significant decrease in pH below the physiological normal value of 7.4.

Other body stores of CO_2

Although the blood is capable of carrying a great quantity of carbon dioxide in the arterial (PCO_2 = 40 mmHg, 480 ml $CO_2 \cdot L^{-1}$) and mixed venous (PCO_2 = 46 mmHg, 560 ml $CO_2 \cdot L^{-1}$) supplies, by far the greatest amount of storage is in other tissue. As well, extracelluar fluid accounts for approximately 14 litres in the average human, and is capable of storing tremendous amounts of CO_2 as HCO_3^- ions. Bone is also a large storage compartment for CO_2. It has been estimated that the total body stores of CO_2 in the normal resting human may be in excess of 120 litres (Mines, 1986, p. 78).

Regulation of PCO_2 and H^+ in the blood and CSF

As mentioned previously, arterial pH must be closely regulated in order to maintain normal enzyme function. It rarely deviates from a value of 7.4 by more than .15 units in normal, healthy humans. Blood levels outside of the range 6.8 and 7.8 units are incompatible with life (Mines, 1986, p. 84). Therefore, any mechanisms which induce acidosis or alkalosis must be highly regulated.

The pH of any buffer system can easily be described by the ratio of its concentration of ionized and unionized forms. This is represented by the formula $pH = pK_a + \log([A^-]/[HA])$, which is known as the Henderson Hasselbach equation. A common system used to describe the

acid/base balance system of the arterial blood involves the ratio of CO_2 to HCO_3^-. These quotients are derived from the hydration-dissociation equation. The specific Henderson Hasselbach for this system is written as follows:

$$pH_a = 6.1 + \log \frac{[HCO_3^-]}{[(0.03\ mM \cdot L \cdot mmHg^{-1}]PCO_2}$$

where the solubility constant of CO_2 is 0.03 $mM \cdot L^{-1} \cdot mmHg^{-1}$. Therefore, with a normal P_aCO_2 of 40 mmHg and $[HCO_3^-]$ of 24 $mEq \cdot L^{-1}$ the resultant pH is equal to 7.4. Since it is the $[HCO_3^-]/PCO_2$ ratio which is important to pH, the actual values are not important as long as they maintain a 20:1 ratio. As well, due to the large ratio of HCO_3^- to CO_2, PCO_2 can vary markedly before the ratio is significantly affected.

In fluid such as the cerebral spinal fluid (CSF), the bicarbonate buffering system is the only important method of handling excess H^+ ions. The CSF does not have the protein buffering capability of blood due to the impermeability of the blood brain barrier to these molecules. As a result, if the PCO_2 increases in the blood, only CO_2 diffuses into the CSF. This causes the hydration-dissociation equation to shift to the right forming more H^+ ions. Thus, CSF pH is lowered to a greater degree than in the blood, which has the benefit of protein buffers to mop up the excess H^+. Such a situation usually arises in response to an incapacity to remove CO_2 from the blood faster than it is produced. A mismatching of V_A/VCO_2 occurs, resulting in acidosis. A similar result would occur from breathing a hypercapnic atmosphere. Either circumstance increases H^+, HCO_3^-, and PCO_2 above normal levels.

Two primary blood facilitated mechanisms are present to combat elevated PCO_2. The most immediate is achieved by means of an increased V_A by stimulation of carotid receptors, or central receptors located on the ventromedial medulla oblongata. Respiratory compensation lowers blood pH by driving the hydration-dissociation equation to the left as CO_2 is vented to the atmosphere. A slower, but more effective mechanism is renal compensation in response to chronic hypercapnia. Kidneys increase the vascular bicarbonate ion concentration to bring pH towards normal values. However, this only affects the HCO_3^-/PCO_2 ratio, absolute PCO_2 may still remain high despite normal or near normal ventilation rates.

It is possible that this last point may have some relevance to prolonged sleep in a hypercapnic environment. Sleep or thermoregulatory process may be affected by the acute and chronic responses to CO_2. Breathing carbon dioxide has been shown to disrupt normal acid/base balance in the body by elevating the arterial PCO_2. Compensatory mechanisms vary in their response times. Ventilatory and buffering mechanisms exhibit an immediate response to decreased arterial pH, whereas bicarbonate secretion from the kidneys are effective with long term hypercapnic exposure. During the course of a night or consecutive nights exposure to a hypercapnic environment, H^+ ions may initially exert a stimulative effect on sensitive chemoreceptor regions or depress enzyme regulated reactions. If exposure is prolonged, renal compensation may diminish these effects.

THERMOREGULATORY RESPONSES TO CO_2

Subjective perceptions

One of the most common sensations reported by subjects breathing hypercapnic gases is a warm, or flushing sensation which permeates the skin. This phenomena has been known for quite some time as noted by the anecdotal evidence of Boussingault (1855, in Stupfel, 1974) who felt a warm, prickling sensation while walking past carbogaseous volcanic emissions in a sulphur mine.

Of greater relevance to the problem identified by this investigation is the effect of inspired CO_2 on subjective thermal perceptions. It is generally accepted that inspired CO_2 concentrations as low as 2-5% result in general sensations of warmth (Kuno, 1956; Bullard, 1964), which persist even in cold environments (Bullard and Crise, 1961). In an attempt to elucidate the cause of these sensations, Bullard and Crise (1961) measured the thermal discrimination sensitivity of subjects breathing 6% CO_2. They found that despite general sensations of warmth during CO_2 inhalation, local perceptions of warmth or cold were not affected. These findings appear to be inconsistent with the earlier mentioned carbogaseous bath and isolation experiments which described a peripheral effect of carbon dioxide.

Two alternate explanations have been postulated which suggest an alteration of higher control mechanisms. The first proposed by several authors suggests that an increased vascular PCO_2 may alter the thermal integrating and effector mechanisms of the anterior and posterior hypothalamus, thereby lowering the central thermostat (Schaefer and Wünnenberg, 1976;

Jennings, 1979). If the thermostatic threshold is lowered during CO_2 inhalation, then one would expect to see this reflected in lower body temperatures. Although Bullard and Crise (1961) failed to find consistent skin or rectal temperature changes to support this hypothesis, several others have reported significantly lower rectal temperatures with CO_2 exposure (Plewes and Jennings, 1972; Jennings, 1979) as well as decreased VO_2 (Bullard 1961, Kaminski et al. 1985). However, it is difficult to identify a lowered thermostat as being the source of lowered body temperatures since other avenues of heat loss such as sweating, respiratory heat loss, and shivering may be altered. A second explanation proposed by Kuno (1956) and Bullard and Crise (1961) involves the alteration in sensory perceptions as a result of emotional distress due to dyspnea. However, this has only been discussed in speculation.

Sweating

Accompanying the subjective thermal sensation of warmth, sweating may occur, particularly in warm environments. This phenomena has long been noted, and is described in anaesthesia manuals as being indicative of CO_2 retention. Indeed, several studies have clearly demonstrated an augmentation of sweat production in warm environments in response to CO_2 inhalation (Kuno, 1956; Bullard, 1964).

Kuno (1956) describes the work of Adachi (1936) who noted general body sweating occurring when the CO_2 composition of the air inside a rebreathing bag reached 4.3 - 5.0%. Sweating gradually increased and then subsided upon resumption of breathing room air. Kuno (1956) reported that further studies by Adachi (1936) utilizing stable concentrations of CO_2 (5 - 10%) yielded similar results. Unfortunately, no ambient temperatures were described, however, the sweat rate was described as profuse. Twenty to thirty minutes after the outbreak of sweating,

it abruptly subsided despite ventilation rates and associated dyspnea remaining high.

Bullard (1964) attempted similar studies using a steady state 6% CO_2 and temperatures of 27, 38, and 49°C (Figure 1). Again, general body sweating was noted in all conditions, but the augmentation increased with greater ambient temperatures. A subsequent decline in sweat rate occurred after 6 to 15 minutes of continuous hypercapnic inhalation. Despite this decline, sweating never returned to pre hypercapnic baseline values, except at 27°C. Upon return to breathing room air, sweating appeared to be suppressed for 5 - 13 minutes.

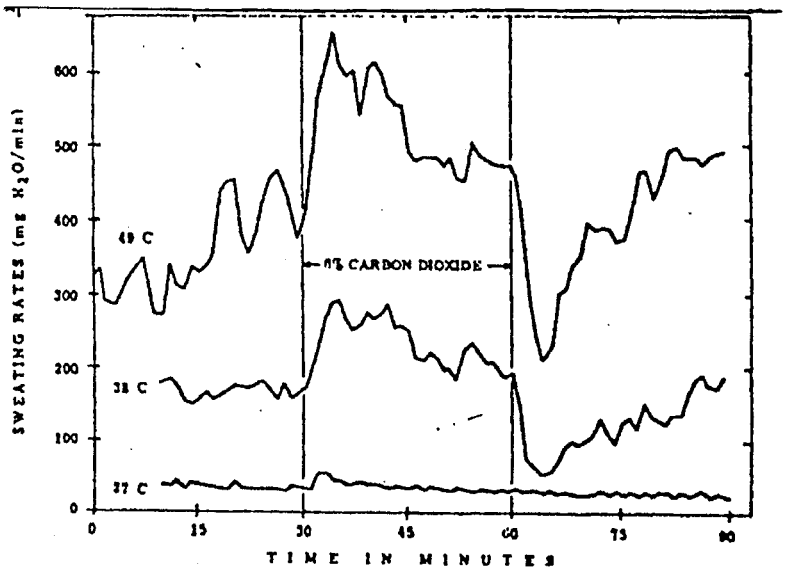

Figure 1. Effects of 6% CO_2 on sweating rates at 27, 38 and 49°C. (Bullard, 1964)

Several explanations have been proposed by the various authors which attempt to account for the increased sweating associated with CO_2 inhalation, yet the exact mechanism remains unknown. Kuno (1956) and Bullard and Crise (1961) mentioned dyspneic distress as a possible

cause, but emotional sweating is primarily confined to the palmar and plantar regions of the hands and feet (Kuno, 1956). All studies reported describe whole body sweating which is characteristic of thermal sweating.

Cutaneous factors such as thermal sensitivity (Bullard, 1964) and effector (sweat gland) activity (Bullard 1964) have also been suggested has having a role in this increase sweat production. Bullard and Crise (1961) found little change in the ability to discriminate thermal sensations while breathing CO_2 in the cold. Although there is little other evidence to support these notions, an altered afferent or efferent output at the cutaneous receptors provides a potential explanation for the observed phenomena of augmentation and suppression of sweat. If sweat rate increases to such an extent that skin and deep body temperatures drop sufficiently, then the central temperature control would suppress the requirement for sweating. Presumably, skin and body temperatures would increase with time and sweating augmentation would reappear. Although, skin and body temperatures have been demonstrated to decrease with CO_2 exposure (Adachi 1936, as referenced in Kuno, 1956; Bullard, 1964), the integration of central and peripheral control on sweating is yet to be ascertained.

Bullard (1964) also entertained the possibility of central thermoregulatory control mechanisms being affected by CO_2 inhalation. He hypothesized that the hypothalamic set point may somehow be shifted downwards resulting in a greater sweating drive for a given temperature above this threshold. Support for this hypothesis is provided by a number of recent studies which indicate that the response of temperature sensitive neurons in the preoptic region of the posterior hypothalamus is decreased with CO_2 inhalation (Schaefer and Wünnenberg, 1976; Wünnenberg and Werner, 1980).

Finally, the effects of CO_2 on the autonomic nervous system output is well known (Price, 1960), which may, in turn, affect temperature sensitive neurons. As well, it is plausible that CO_2 may stimulate subthalamic sweat centres directly. Either one of these factors could potentially increase the efferent output to the sweat glands, increasing their output.

Respiratory heat loss and ventilation

As mentioned earlier, CO_2 is a powerful stimulus to ventilation through its influence on central ventilatory centres and peripheral chemoreceptors. Significant amounts of heat transfer occurs through the process of warming and humidifying air during normal respiration. Historically, it has been assumed that the expired air temperature is 37.5°C and fully saturated (Day, 1968) regardless of the inspired temperature. However, confirmation of this has been difficult due to the long time constants of temperature sensing equipment.

Recent work by Cain et al. (1990), using fast responding temperatures sensors, has illustrated expired temperatures varying according to environmental temperature. Resting expired temperatures ranged from approximately 31°C at - 20°C to approximately 34°C at 20°C. As well, the expired air was near saturation with water vapour. This warming and humidification of inspired air resulted in a total metabolic heat loss of 25 to 30% of resting metabolic and 15 to 20% of working (500 W) metabolic rate. According to the heat loss equation described by Cain et al., respiratory heat loss is directly related to ventilation. Given the relative high loss of metabolic heat, any ventilatory response to a hypercapnic stimulus may result in depletion of body heat content.

Wagner et al. (1983) measured an augmented heat loss while breathing 4% CO_2 at 5 and

29°C. Since shivering, skin blood flow and VO_2 did not change between normal and hypercapnic exposures, they estimated the increased minute ventilation and associated respiratory heat loss as being the primary avenue for the increased heat loss.

Supplementary research into the effects of cold air on ventilatory responses may have made this concern of increased ventilation and respiratory heat loss debatable. Burgess and Whitelaw (1988) found nasal breathing of cold air (2°C) reduced the slope of the warm (32°C) ventilatory response to 2, 4 and 6% CO_2 by 27%, and average V_E by 18%. Similar results were achieved by an earlier study by the same authors (Burgess and Whitelaw, 1984) with 8% CO_2 and -4 to 10°C (Figure 2). One other study did not find cold air directed at the face effective at reducing hypercapnic induced hyperventilation, but subjective distress decreased (Schwartzstein et al., 1987). It is possible that facial cooling was not effective at cooling nasal receptors, and may account for the difference in the results of these two studies.

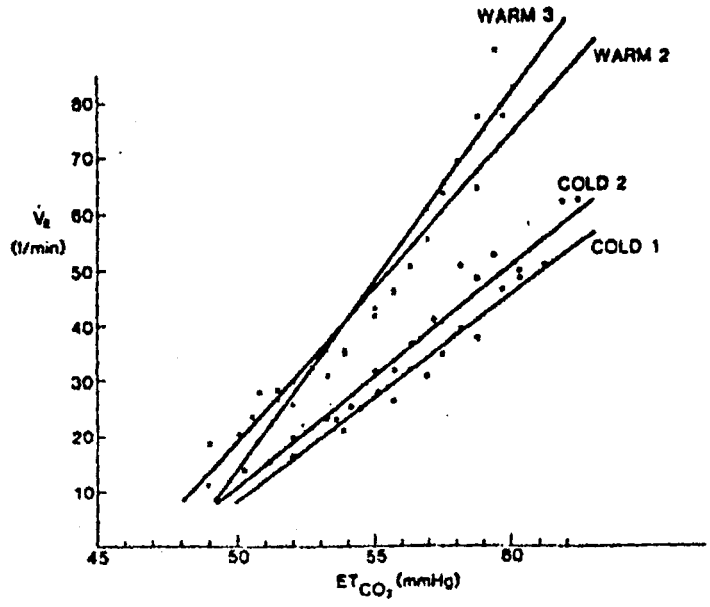

Figure 2. The ventilatory response to 8% CO_2 at -4 and 10°C demonstrating the inter trial variability of a single subject. (Burgess and Whitelaw, 1984).

Increased nasal resistance has been suggested as accounting for a reduced ventilation during hypercapnic inhalation, as Takagi et al. (1969) demonstrated cold air increasing this resistance. Conversely, they also measured a decreased nasal resistance following breathing 7% CO2. This was possibly the result of a vasoconstrictive effect on the nasal mucosa. Subsequent studies found a slight decrease (Burgess and Whitelaw, 1984) or no difference (Burgess and Whitelaw, 1988) in nasal resistance when cold and CO_2 were simultaneously present. An alternative explanation for decreased ventilatory responses lies in nasal receptors or trigeminal nerve afferents. Afferent impulses may feed back to the hypothalamus or respiratory centres in direct response to a cold stimulus, or increased respiratory heat loss, in an attempt to minimize intracranial cooling, or to prevent possible lung damage (Burgess and Whitelaw, 1984).

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CO_2, catecholamines and hypothalamic threshold

Much attention aimed at explaining shifts in thermoregulatory responses has been directed to the investigation of the neurotransmitter norepinephrine. Acidosis induced by carbon dioxide inhalation is known to stimulate the adrenal medullary release of catecholamines epinephrine and norepinephrine (Price, 1960). Norepinephrine is found in the hypothalamus and has been identified by Feldberg and Myers (1964) as being important in temperature regulation.

Intrahypothalamic injections of norepinephrine into guinea pigs (Zeisberger and Brück, 1970) resulted in increases in oxygen consumption, body temperatures and shivering setpoint. These effects were only noted when the site of injection was the anterior hypothalamus. Injection into the POAH or posterior hypothalamus elicited no response. Furthermore, the regions which were sensitive to norepinephrine did not contain thermosensitive neurons, leading to the hypothesis that norepinephrine influences the reference signal inputs for thermoregulation.

Supporting evidence for a hyperthermic effect of norepinephrine was presented by Schaefer et al. (1975). They chronically exposed guinea pigs to an atmosphere of 15% CO_2 and noted a decrease in hypothalamic norepinephrine content in the initial 24 h, which mirrored a decreased VO_2, T_b, and skin blood flow. With compensation to the respiratory acidosis, hypothalamic norepinephrine concentration, metabolic rate, body temperatures, and skin blood flow all increased.

Conversely, there is contradictory evidence suggesting that norepinephrine has a hypothermic effect. Good and Sellers (1957) intravenously injected epinephrine into dogs during cold exposure and noted a decrease or total inhibition of shivering. Injection of both epinephrine

and norepinephrine into the cerebral ventricals of cats (Feldberg and Myers, 1964) resulted in body temperatures decreasing .5 to 1°C despite laboratory temperatures being maintained at 20 to 22°C.

An extensive review of this topic by Hellon (1974) has confirmed that there are interspecies variabilities regarding whether a hypothermic or hyperthermic response to an injection of catecholamines occurs. Many explanations such as, dosage, site of injection, ambient temperature, transmitter function, and species dependent anatomical features have been proposed to account for these differences. Unfortunately, extrapolating the effects of catecholamines on thermoregulation in humans is impossible at this time. However, it is possible that impairment of thermoregulatory mechanisms caused by the disruption of the acid/base balance due to hypercapnic breathing may be mediated through catecholamines acting on the thermosensitive regions of the hypothalamus.

SLEEP AND THERMOREGULATION

Early research

Comfortable environments

Pioneer research into the aspects of thermoregulation during sleep began in the late 1800's when Richet (1898 as referenced in Amdur et al., 1952) observed a diurnal variation in body temperature. Unfortunately, early studies were unable to benefit from modern interpretation of sleep being comprised of stages associated with specific electrophysiological events. As a result, it was often difficult to discern the effects of sleep onset and sleep stages on thermoregulatory parameters. Verification of whether the subjects were actually asleep when measurements were taken was almost impossible. As well, sampling periods were relatively long and few parameters were measured. Without the aid of microcomputers, gathering large amounts of data was labour intensive and impractical. Due to these technological limitations and a relative lack of detailed examination of sleep and thermoregulation, only the most elementary information could be attained.

The general consensus among early researchers was that rectal temperatures decreased during the initial hours of sleep (Day, 1941; Kreider et al., 1959; Kreider and Iampietro, 1960; Anderson and Hellstrom, 1960) followed by a gradual increase prior to awakening (Mellete et al., 1951). Day (1941) noted an average decline in rectal temperature of .55°C after sleep onset, followed by a sudden levelling off with no subsequent rise. This trend agrees with the results of Andersen and Hellstrom (1960).

Metabolic rates mirrored these changes in body temperatures (Kreider et al., 1959) as did skin temperatures (Scholander et al., 1958; Kreider et al., 1959; Kreider and Iampeitro 1960). However, Kreider and Iampietro's (1960) results show a distinct initial rise in skin temperature at sleep onset. It is difficult to ascertain if these latter findings are due to sleep onset, postural changes, or an artifact of transporting the subject from a cool preparation room into a warm bed. If the effects of sleep onset are to be examined, it stands to reason that a period of wakeful adjustment to the sleep environment is essential.

Cold temperatures

Research into the effects of cold temperature and thermoregulation during sleep gathered momentum in the late 1950s with the appearance of a number of studies addressing this question. Pioneer work by Scholander et al. (1958) and Hammel et al. (1959) on Europeans and Australian Aborigines, and Ward et al. (1960) on Kalahari Bushmen and Europeans demonstrated basic yet important information on the effects of cold temperatures during sleep. These were field studies which incorporated the traditional methods of sleeping in the bush as practised by these two aboriginal cultures. Subjects slept either exposed nude (Ward et al., 1960), with light covers (Hammel et al., 1959), or nude with the protection of a windbreak and fire (Scholander et al., 1958) in a desert where night time temperatures commonly fell to approximately 0°C. Common to all of these studies was the fact that skin temperatures and rectal temperatures fell throughout the night. In fact, the Australian Aborigines allowed their skin and rectal temperatures to fall far below that of the Europeans and Bushmen, while simultaneously maintaining a lower metabolic rate. Shivering and muscular activity increased the Europeans metabolic rate 10% to 90% above resting basal, while the Aborigines made little or no metabolic compensation (Scholander et al., 1958). Furthermore, in Scholander's study, foot temperatures of the Aborigines decreased to

12°C - 15°C as they slept, while Europeans experiencing foot temperatures of 15°C - 17°C became distressed and unable to sleep.

The significance of this information lies in the different strategies used to contend with cold stress during sleep. Europeans and Bushmen demonstrated a metabolic adaptation whereas the Aborigines employed an insulative-hypothermic one. Both strategies may be described as adaptive responses allowing the person to tolerate their thermal surroundings. However, metabolic adaptation is gained at great cost to substrate reserves and would require a great caloric intake. One cannot state emphatically that insulative-hypothermia is preferred to metabolic adaptation, as it has been demonstrated that it is an unique individual response (Anderson and Hellestrom, 1960; Livingstone et al., 1989). This must be kept in mind when interpreting pooled data in that either of these strategies may be present in the population sample.

Laboratory studies of Kreider and Iampietro (1960) and Anderson and Hellestrom (1960) essentially confirmed the results of the previously mentioned field experiments. Krieder and Iampietro (1960) examined the effect on sleep of a variety of ambient temperatures ranging from approximately -35°C to +26°C in six young males. Although these temperatures seem extreme, the subjects were given adequate, yet undefined insulation selected to yield graduated degrees of cold stress. It was concluded that decreasing temperatures resulted in a lower minimum rectal and skin temperature as well as a decreased time to reach these minimum temperatures. Surprisingly, decreased ambient temperature had no effect on metabolic rates.

Effects of cold temperature on metabolic rates were illustrated by Anderson and Hellestrom (1960). They defined a nude sleeping thermal comfort zone which had a lower limit temperature of 25°C. Below this temperature, thermoregulatory mechanisms were recruited to

maintain normal thermal steady state utilizing basal heat production. At 20°C metabolic rates increased 20 - 60%, with a trend towards decreased rectal and skin temperatures at lower ambient temperatures. Interestingly, one subject displayed an insulative-hypothermic response to cold similar to that found by Scholander et al. (1958).

Recent research

The most significant advance into the study of thermoregulation during sleep involves the standardization and interpretation of EEG recordings as described by Rechtschaffen and Kales (1968). As previously mentioned, sleep is not a uniform entity, but is composed of various stages based on electrophysiological correlates. Despite the fact that sleep is generally classified in five stages (1,2,3,4 and REM) (Bennet, 1982, p. 223), many authors have elected to combine stages 1 through 4 under the descriptive heading of non-REM (NREM) (Henane et al., 1977; Haskell et al., 1981b). Others have chosen to concentrate on thermoregulatory function during the deeper stages of sleep (3 and 4) since they believe stages 1 and 2 are transitions between wakefulness and deep sleep (Buguet et al., 1976b; Palca et al., 1986). These deeper stages of sleep are then referred to as slow wave sleep (SWS) or deep sleep. Stage 2 is occasionally referred to as spindle sleep in studies involving cats (Parmeggiani, 1969). REM sleep is also treated as a separate entity due to its unique physiological manifestations (Bennet 1982, p. 226). Other terminology frequently employed in the literature for this stage include Paradoxical Sleep (PS), due to its similarity to awake EEG (Buguet et al., 1979a), or Fast Wave Sleep (FWS) in animal models (Parmeggiani et al., 1969; Glozbach and Hellar 1976). Table 1 is summary of the various stages of sleep and their synonyms appearing in the literature. In an attempt to maintain the scientific integrity of the research reviewed in this document, sleep stages will be referred to as described in the particular literature being cited. However, equivalent stages such

as REM and PS will be presented as being indistinguishable.

Table 1. Stages of sleep and the various synonyms for each stage.

Sleep Stage	EEG	Synonyms	
1	2 - 7 Hz, low amplitude	N	
2	Sleep spindles, K complexes	R	Theta(θ), Spindle Sleep
3	20 - 50% large amplitude, <= 2 Hz	E	Delta (δ), Deep Sleep,
4	> 50% large amplitude, <= 2 Hz	M	Slow wave sleep (SWS)
REM	Similar to 1, Rapid Eye Movements (REMs)		Paradoxical Sleep (PS), Fast Wave Sleep (FWS)
Wakefulness	8 - 12 Hz, desynchronized		

Deep body temperatures

Recent literature has confirmed the previous reports of decreased rectal temperatures with sleep onset. This response occurs in both neutral temperatures (Haskell et al., 1981a; Palca et al., 1986) and is exaggerated in cold temperatures (Buguet et al., 1977; Haskell et al., 1981a; Palca et al., 1986) but does not occur in warm (> 35°C) environments (Henane et al., 1977; Haskell et al., 1981a). In fact, the total drop in rectal temperature may by in the order of 1 - 1.5 °C in the cold (Palca et al., 1986). The greatest drop in temperature generally occurs during the first 1 - 2 two hours after sleep onset (Haskell et al., 1981a) and rises 1 - 2 hours prior to waking, although this is not always the case in cold environments (Buguet et al., 1976b; Palca et al., 1986). Figure 3 illustrates the response of rectal temperatures to various ambient temperatures. A lack of morning rise in body temperature is probably due to an inability of

thermoregulatory mechanisms to counter heat loss during sleep. Furthermore, it is possible that many subjects become prematurely aroused from sleep due to the cold stress before the pre-waking rise in body temperature is evident.

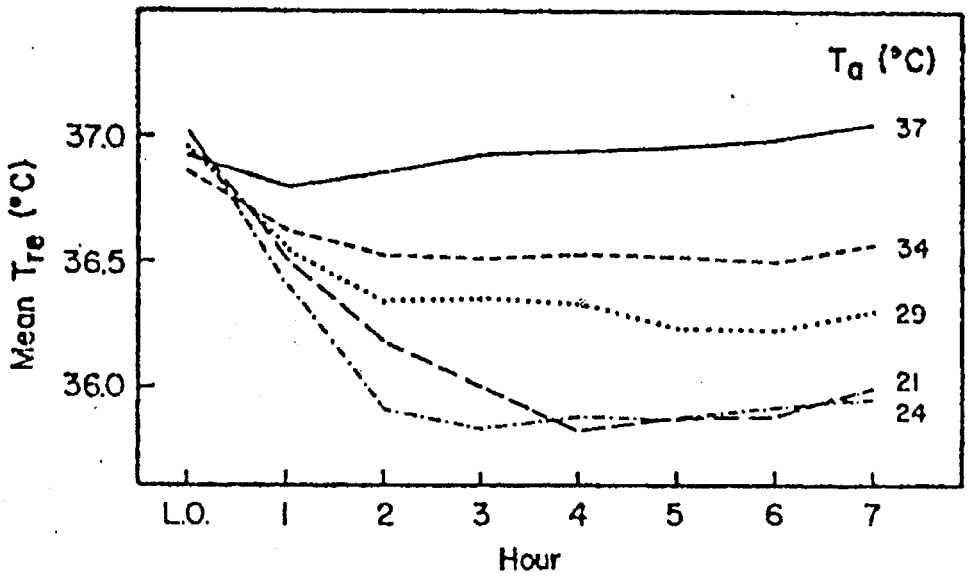

Figure 3. Mean rectal temperatures across the night at 21, 24, 29, 34 and 37°C. (Haskell et al., 1981a).

Although rectal temperature has been the preferred measure representing body temperature, it has been suggested that tympanic temperature is preferable due to its presumed representation of brain and hypothalamic temperature (Baker et al., 1972). A comparison of these two temperatures was made during sleep in an attempt to gauge their response characteristics in sleeping humans (Palca et al., 1986). During nude sleep at 21°C and 29°C both T_{ty} and T_r dropped, however, rectal temperature decreased further in the colder environment. It was concluded that this relative tympanic homeothermy suggests that hypothalamic temperature is more closely regulated than rectal temperature.

Stage REM has demonstrated little or no effect on rectal temperatures (Haskell et al., 1981a; Palca et al., 1986). This is surprising since tympanic temperature has been shown to rise during this stage (Palca et al., 1986). Furthermore, the rise in T_{ty} during REM is greater with lower T_{ty} and ambient temperatures (Haskell et al., 1981a). As a result, Palca et al. (1986) claimed that this stage does not result in an inhibition of thermoregulation in man as it has been demonstrated in other animals (Parmeggiani, 1970; Glotzbach and Heller, 1976).

Skin temperatures

Skin temperatures have also been shown to be affected by sleep. Unfortunately, these measurements are seldom mentioned in the literature. As noted earlier, Kreider and Iampeitro (1960) found a decrease in skin temperature with sleep in cold temperatures. This response is not limited to cold environments as Haskell et al. (1981a) reported a similar response to ambient temperatures greater than 29°C (Figure 4). Geschickter et al. (1966) noted a curious rise in toe temperature upon sleep onset. Once asleep, mean skin temperature drops throughout the night (Buguet et al., 1979a, Haskell et al., 1981a, Palca et al., 1986). Greater decrements occur with lower T_as, particularly in the extremities as opposed to the trunk (Palca et al., 1986). Comparison of the four stages of NREM sleep has little if any influence on the skin temperature profile (Buguet et al., 1979a).

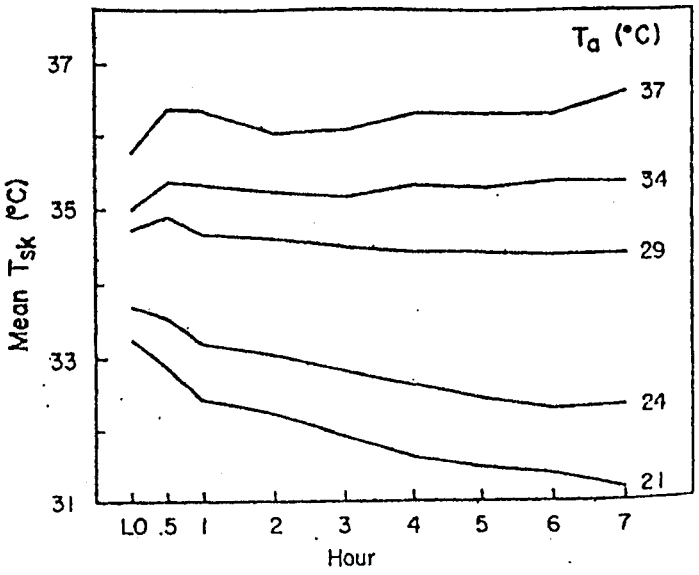

Figure 4. Mean skin temperature across the night at 21, 24, 29, 34 and 37°C. (Haskell et al., 1981a).

It has been demonstrated that stage REM has an influence on skin temperatures. In general, $\bar{T}_{sk}$ rises during this stage (Henane et al., 1977; Buguet et al., 1979a; Haskell et al., 1981a), however this rise is not uniform across the body as demonstrated by Palca et al. (1986). They reported increases in limb temperatures while forehead temperature decreased. Increased skin temperatures have been suggested to be result of the cessation of sweating and decreased evaporative heat loss often associated with REM sleep (Ogawa et al., 1967; Shapiro et al., 1974; Henane et al., 1977; Haskell et al., 1981a; Palca et al., 1986). This effect is clearly illustrated by Figure 5 where REM sleep, as indicated by the horizontal bars, is associated with a rise $\bar{T}_{sk}$ when the rate of sweating weight loss is attenuated. However, increased skin temperatures also occurred in cold environments when a sweating response was not present (Day, 1941; Buguet et al., 1976b; Glotzbach and Heller, 1976; Palca et al., 1986). This latter fact suggests neurogenic vascular mechanisms are responsible for changes of skin temperatures in colder environments.

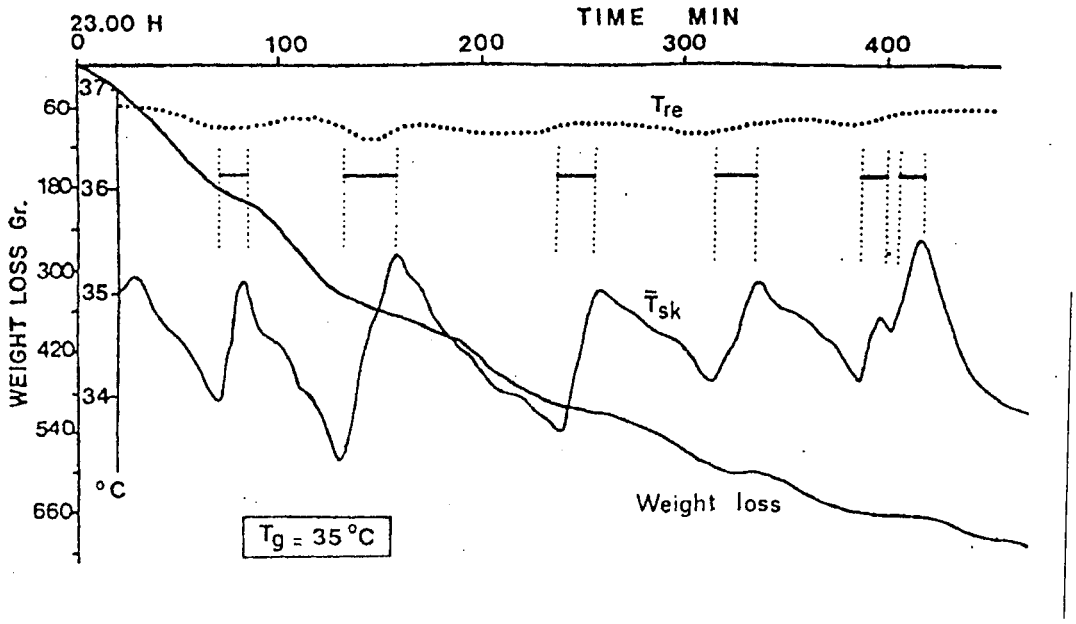

Figure 5. Continuous recording of T_r, $\bar{T}_{sk}$ and weight loss during nocturnal sleep. REM sleep is designated by a solid horizontal line between vertical dotted lines (Henane et al., 1977).

Clues to the neurogenic mechanisms affecting skin temperatures in REM may be derived from the observations of Buguet et al. (1979a). In cold environments, the direction of $\bar{T}_{sk}$ change was not always uniform. Instead, it appeared to be tightly coupled to deep body temperature. When T_r was above 36°C upon entering REM, $\bar{T}_{sk}$ increased, and decreased when T_r was less than 36°C. This relationship between $\bar{T}_{sk}$ and T_r is clearly illustrated by Figure 6. It is not known why 36°C is the pivotal temperature, but it does suggest that changes in skin temperatures and deep body temperature are related during the REM stage of sleep.

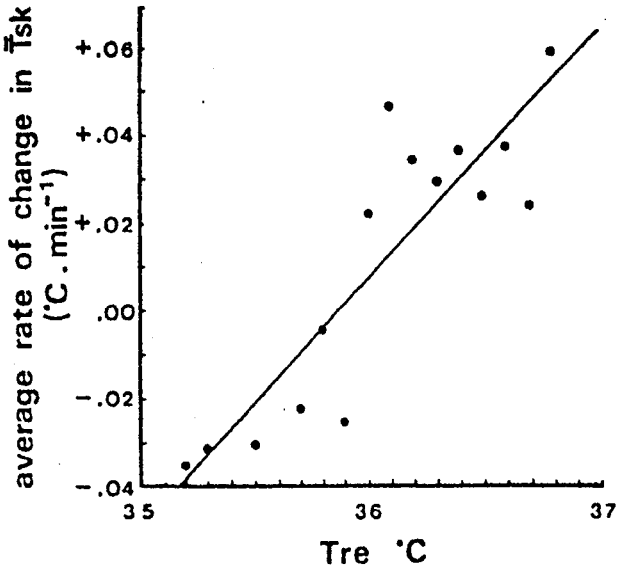

Figure 6. Relation between the average rate of change in $\bar{T}_{sk}$, and level of T_r upon entering REM. (Buguet et al., 1979a).

Currently, the most plausible explanation of changing skin temperatures in REM sleep lies in the alteration of the tonic state of vascular beds. Cardiovascular measurements performed by Franzini et al. (1989) have demonstrated a REM induced shift in blood volume from vasodilated muscular beds to previously vasoconstricted regions such as the splanchnic, renal and arteriovenous anastomoses (AVAs). This was attributed to a drop in sympathetic activity. Parmeggiani (1980) provides further evidence that removal of sympathetic activity during REM and the competition between the tendency to vasodilate or vasoconstrict is responsible for $\bar{T}_{sk}$ changes. Vasomotion is dependent on the degree of sympathetic tone and transmural pressure on the vessels. In a region such as the AVA's, a decreased sympathetic tone would cause vasodilation, with vasoconstriction in the precapillary sphincters of the muscular beds (Åstrand and Rodal, 1977, p. 162). This reduction of sympathetic tone is also associated with a decrease in mean arterial blood pressure (Guyton, 1987, p. 149). Depending on the degree of sympathetic

activity prior to stage REM, the blood flow would either increase or decrease according to the competition between vasodilation and a decreased blood pressure. For example, if sympathetic vasoconstrictor activity is low during SWS, then vasoconstriction may occur during REM. Conversely, high sympathetic activity during SWS would result in REM vasodilation. This is indeed what is observed in the many studies previously mentioned. The problem with this rationale is that it does not satisfactorily explain the results of Buguet et al. (1979a). One would assume that sympathetic activity would be very high at a T_r below 36°C exaggerating the vasodilatory response. Although the removal of sympathetic tone and competition of vascular reactions is a reasonable model for changes in $\bar{T}_{sk}$ during REM, more research is needed to explain such exceptions results.

Metabolic rate

Metabolic rates are known to decrease upon falling asleep, resulting in a 9% to 25% decrease as compared to resting awake values (Shapiro et al., 1984; Fraser et al., 1989). Sleep in cold temperatures also results in decreased metabolic rates, but to a lesser degree as the ambient temperature departs from thermoneutrality (29°C) (Haskell et al., 1981a; Palca et al., 1986). This decrease has been attributed to muscular relaxation or the specific dynamic action (SDA) of food (Geschickter et al., 1966), or circadian and sleep effects (Fraser et al., 1989). The circadian effect can be demonstrated by the gradual decrease in metabolic rate throughout the night usually followed by a gradual rise prior to waking (Haskell et al., 1981a; Palca et al., 1986; Fraser et al., 1989). This is similar to the response of body temperatures described in the previous section. Sleep onset results in a dramatic drop in metabolic rates accounting for up to

50% of the total night's decrease within the first 30 min. of sleep (Fraser et al.,1989) (Figure 7).

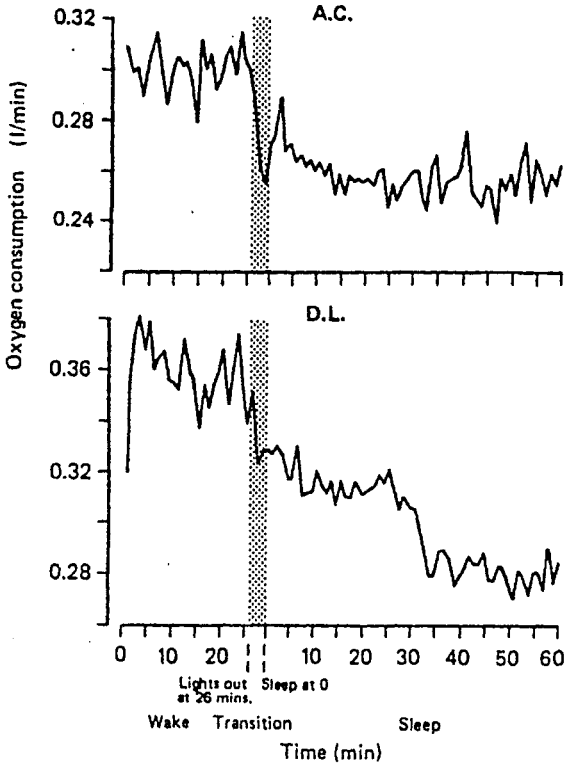

Figure 7. Oxygen consumption at sleep onset in two subjects. (Fraser et al., 1989).

Little is known about the mechanisms of the sleep effect. This is most likely due to the fact that many articles compare stages during sleep and seldom include the wakefulness to sleep transition. It has been suggested that the sudden drop in metabolic rate may be a result of either a resetting of the metabolic threshold or altered ventilation (Fraser et al., 1989). Colrain et al. (1987) demonstrated substantially reduced ventilation rates of 12% to 35% during the wakefulness to stage 2 transition primarily as a result of reduced V_T. This was accompanied by a decreased VO_2 of 2% to 12%. They concluded that metabolic rates during this period are underestimated and should not be reported due to the instability of ventilation during this transitional period. Thus, the exact nature of the sleep onset mechanism remains to be defined.

Metabolic rate during NREM sleep has been demonstrated to be independent of sleep stage (Palca et al., 1986), yet according to other studies, metabolism decreases with increased depth of sleep (Brebbia and Aschuler, 1965; Haskell et al., 1981a; Shapiro et al., 1984). Shapiro et al. (1984) approached this question using direct calorimetry. Their results strongly indicated a graduated decrease in metabolic rate with stage 4 being lower than stage 3 which was in turn lower than stage 2. These results confirmed the findings of Brebbia and Aschuler (1965). However, despite statistically significant differences, the actual differences between sleep stages was physiologically small. As with any comparison of stage effects on other physiological variables, analysis of these data is confounded by the fact that sleep stages are not evenly distributed throughout a sleep period, resulting in a circadian bias which changes throughout the night.

REM sleep in humans has been reported to have either no effect on metabolism (White et al., 1985), similar effects to stage 2 (Shapiro, 1984), or increases compared to NREM sleep (Brebbia and Aschuler, 1965; Haskell et al., 1981a; White et al., 1985). Haskell et al. (1981a) reported an increased VO_2 during REM at temperatures above or below 29°C. No change was evident at the thermoneutral 29°C. A plausible explanation for an increased metabolic rate lies in the fact that cerebral blood flow increases significantly in REM sleep (Franzini et al., 1989). Given that cerebral metabolism accounts for up to 20% of the basal metabolic rate (Sokolof, 1974) an increased cerebral blood flow and neural activity during REM may mask the decreased metabolism associated with the muscular atonia associated with REM. This increase in VO_2 during REM has resulted in the dismissal by Palca et al. (1986) of the frequent assumption that thermoregulation is impaired or absent during this stage.

Sweating

Although not directly relevant to thermoregulation in cold environments, the regulation of sweating gives important additional insight into thermal control mechanisms during sleep. Onset of sleep and sleep in general is associated with an increased sweat rate as compared to waking values (Ogawa et al., 1967; Glotzbach and Heller, 1976). As sleep time progresses, sweating decreases throughout the night (Henane et al., 1977) independent of sleep depth (Ogawa et al., 1967). However, a phasic burst of sweat activity has been demonstrated during the transition from deep to light sleep (Ogawa et al., 1967).

REM sleep has been associated with a cessation of sweating (Ogawa et al., 1967; Shapiro et al., 1974; Glotzbach and Heller, 1976, Henane et al., 1977; Palca et al., 1986), an effect which persists in warm environments, although phasic bursts of sweating have been noted during REM sleep (Ogawa et al., 1967). This sweating is primarily one of psychogenic origin, since it is associated with vivid dreaming in about 80% of the cases.

Shivering

Sleep has been reported to exert a depressant effect on shivering such that it is seldom seen in stage 2 (Haskell et al., 1981a), absent in deep sleep (stages 3 and 4) (Buguet et al., 1976b; Glotzbach and Heller, 1976) as well as REM (Buguet et al., 1976b; Buguet et al., 1979a; Haskell et al., 1981a). Beyond simple reporting of decreased shivering in humans during sleep, little discussion is devoted to it by the literature. Inhibition of shivering during REM sleep is not particularly surprising as muscular atonia is characteristic of this stage. More research is warranted regarding the neural mechanisms associated with a diminished shivering in NREM

sleep.

Animal models

Animal models have provided the study of sleep and thermoregulation with a great deal of fundamental knowledge which could not be obtained with human subjects. There are several reviews by Parmeggiani (1980) and Hellar and Glotzbach (1977) which deal with this topic in a very in-depth manner. A primary reason for the employment of animals in such studies is the relative ease with which such external variables such as activity, diet and environment can be controlled. Furthermore, animal models permit chronic implantation of electrodes and thermodes which can monitor or stimulate regions of tissue, or specific neurons. This provides further details as to the neural control mechanisms of temperature regulation during wakefulness and the various stages of sleep.

Parmeggiani (1970) hypothesized that there is an apparent reciprocal relationship between sleep and circadian rhythms. Since sleep appears to be associated with the lowering of the central thermostat, then any load upon the thermoregulatory system should interfere with sleep. Conversely, sleep under conditions of thermal stress should interfere with thermoregulatory processes. In order to test the second part of this hypothesis, Parmeggiani and Rabini (1970) exposed cats to a variety of thermal conditions ranging from -10°C to +37°C for 3 hours during sleep. During the cold conditions, shivering responses were seen in spindle and SWS, however shivering was essentially abolished upon entering FWS (Figure 8). This cessation of shivering preceded the muscular atonia associated with FWS, suggesting that the inhibition of muscular tone mediated through different neural structures than shivering. At warmer temperatures which initiated panting responses, there was an increased panting threshold which progressed from

wakefulness to spindle sleep to SWS, approaching infinity in FWS. Thus, panting was suppressed during SWS in warm environments and absent during FWS. It was concluded from these results that the occurrence of FWS in lower animals is associated with a disruption of thermal integration and control.

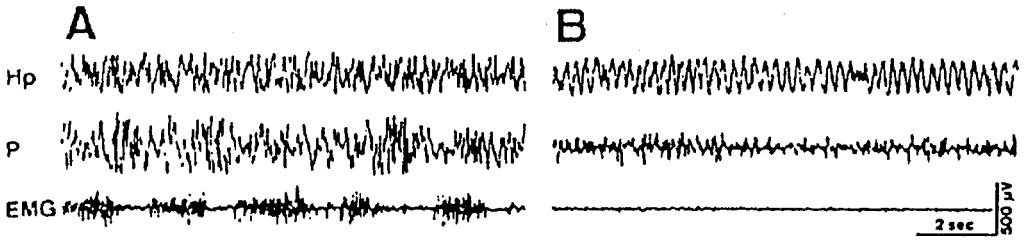

Figure 8. Shivering responses during slow wave sleep (A) and fast wave sleep (B) in the cat. EMG, the electromyogram of the neck muscles. Hp and P, the EEG of the hippocampus dorsalis and parietal regions. (Parmeggiani and Rabini, 1970).

Glotzbach and Heller (1976) attempted a slightly different approach to address this question. Instead of manipulating environmental temperature, thermodes were implanted in the preoptic anterior hypothalamus (POAH) of kangaroo rats, and infusion with warm or cold water manipulated the temperature of this structure during different states of arousal. The POAH is known to be a nucleus of thermosensitive neurons which comprises the major thermoregulatory control centre (Guyton, 1987, p. 548). By decreasing the temperature of the POAH and monitoring the compensatory metabolic response, it was determined that, compared to wakefulness, the temperature threshold for metabolic responses during NREM was decreased as was the gain or proportionality constant. In REM sleep, there was no metabolic response to cooling (Figure 9).

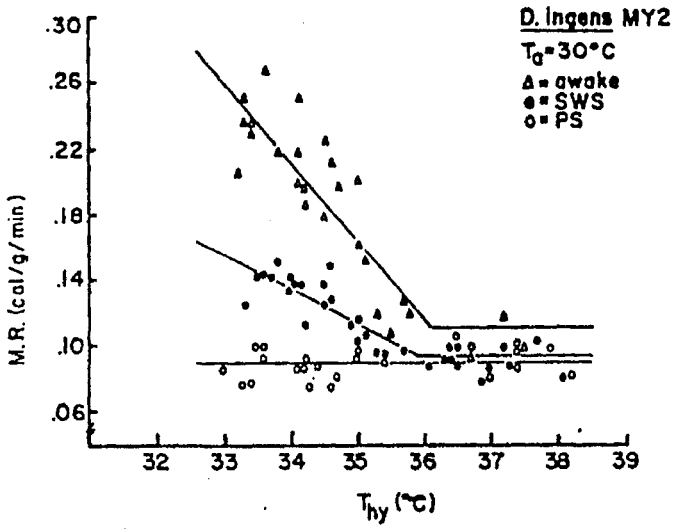

Figure 9. Metabolic rate response to manipulation of hypothalamic temperature of the kangaroo rat during wakefulness, SWS and PS at an ambient temperature of 30°C. (Heller and Glotzbach, 1976).

The relative conclusiveness of these key studies by Parmeggiani (1970) and Glotzbach and Heller (1976) coupled with the support of a large body of related research gives credence to Parmeggiani's (1980) hypothesis regarding the effects of sleep on thermoregulation. It is apparent that these effects are comprised of three components superimposed one on each other, including a tonic (circadian), a dynamic (REM), as well as a NREM sleep component. The tonic, or circadian, component was described by Hammel et al. (1963) as being the result of a changing of the hypothalamic T_b set point controller for various thermoregulatory effectors. For instance, it has been shown by Wenger et al. (1976) that the threshold for such mechanisms as sweating and forearm blood flow change throughout the day. Furthermore, the gains and thresholds for any given response are uniquely affected by their individual rhythms. It is well known that metabolic rate usually increases in response to a cold thermal stress before shivering mechanisms are invoked. This suggests that the set point for increasing metabolic rate is set to a higher T_b than that of shivering. The rhythm of these thresholds as they relate to each other

must be clarified. Buguet et al. (1976a) noted shivering occurring at normal body temperatures. It is possible that these results may be due to measurements being taken when the threshold for initiation of shivering was set to a relatively high T_b.

Electrophysiological evidence has been presented by Heller et al. (1988) indicating NREM and REM thermoregulatory responses are regulated by or mediated through the preoptic anterior hypothalamus. Fine wire electrode and thermodes were chronically implanted in POAH cells of kangaroo rats and unanesthetized cats, respectively. With the onset of NREM sleep, both the number of active cells and their sensitivity to different temperatures decreased. Upon entering REM, all previously active cells became insensitive. This suggests that the down regulation of body temperature during sleep is a process originating in higher centres and is somehow associated with the sleep process. This may partially explain the inhibition of shivering during REM being separate from the muscular atonicity experienced during REM (Parmeggiani, 1970). Shivering is probably mediated through pathways under the regulatory influence of the hypothalamus. However, the exact mechanism by which sleep affects the POAH and effector control has yet to be resolved.

Although one can claim with reasonable confidence that there is an interaction between sleep state and thermoregulatory responses, the exact purpose or function of such a mechanism is not clear. In some animals, thermoregulatory responses are regulated to maintain a lower body temperature with progressively deeper stages of sleep until a poikilothermic state is attained in FWS (Parmeggiani, 1980). Although not as profound, this downward regulation of T_b is evident in humans. It has been proposed that a downward regulation of T_b is a mechanism for energy conservation (Heller and Glotzbach, 1977). If this is an accurate assumption, then lower animals which rely more on physiological adaptation for survival than the behaviourial and technological

responses of man should show an exaggerated temperature response. This hypothesis is supported by the comparison of the human and animal studies. Despite the fact that human research is much more limited in experimental duration, subjects and controls, there are obvious parallels which suggest humans and animals share many of the disruptions of thermoregulatory process during sleep.

EFFECTS OF ENVIRONMENTAL TEMPERATURE ON SLEEP STAGES

According to the statement proposed by Parmeggiani (1970) in the previous section, there is a reciprocal relationship between sleep and thermoregulation. In other words, sleep influences thermoregulatory processes, while thermal stress influences the natural progression of sleep. A corollary to this hypothesis would be that animals sleep best and longest in a thermally neutral environment. A disruption, or downregulation of thermal responses during sleep has already been well described. However, it is also important to describe the influence of thermal stress on sleep patterns. This is particulary relevant for the following two reasons. As an apparent link between sleep and thermoregulation has been established it is possible that a sleep stage alteration may be initiated as a compensatory response to impaired thermoregulation or as an artifact of thermal stress. Secondly, from a operational perspective, to what degree would any disruption of sleep patterns affect performance and recovery during wakefulness, bearing in mind the known adverse effects of sleep deprivation (Bennet, 1982, p. 230). The emphasis of this report will be placed on the first question.

Palca, et al. (1986) confirmed that one sleeps best and longest in a thermoneutral environment. They found nude subjects exposed to five consecutive nights of 21°C and 29°C decreased their total sleep time (TST), as well as their sleep efficiency (SE, ie., the total wake/sleep ratio after sleep onset). All subjects reported feeling cold at 21°C but did not have trouble falling asleep, with the exception of one subject with a sleep onset latency (SOL) on one night of almost 3 hours. The only statistically significant effect of cold temperature on sleep suggested that stage 2 was diminished which resulted in decreased TST. This impairment of stage 2 sleep is supported by other literature (Buguet et al., 1976b; Buguet et al., 1977). Inspection of individual data revealed trends for increased stage 1 and decreased REM in at least

3 of the four subjects. These data concur with the findings of Buguet et al. (1976b).

Several explanations may account for the lack of statistically significant data despite apparent trends in Palca's et al. (1986) report. An obvious point is the relative lack of statistical power due to the limited number of subjects. Second, the cold sleep data were obtained and averaged over 5 consecutive nights. Evidence of acclimation is apparent in some subjects, as cold exposure data drifted in the direction of the baseline values towards the end of the fifth night. This resulted in a tendency to attenuate the mean differences between baseline and cold values. Similar trends occur in studies of 10 consecutive days (Buguet et al., 1975) and 3 consecutive days (Buguet, 1977). In these studies, most of the disruption to sleep was seen during the initial nights. It is possible that one poor night of sleep may contaminate the sleep patterns of a subsequent night resulting in rebound phenomena. As a result, these studies investigate the effects of prolonged cold exposure on sleep. In order to identify the isolated effects of cold on sleep patterns, non-consecutive nights of study are essential.

A consensus as to the effect of cold exposure on deep or slow wave sleep (SWS, or stages 3 and 4 combined) has not been well established. When analyzed separately, stages 3 and 4 are highly variable (Buguet et al., 1976b). For this reason, they are usually combined as SWS (Buguet et al., 1976b; Haskell et al., 1981b; Palca et al., 1986). Several studies have demonstrated an increase (Buguet et al., 1979b), no change (Haskell et al., 1981b; Palca et al., 1986) or a decrease (Buguet et al., 1977) of SWS in cold environments. There are several explanations which may account for the discrepancies between researchers. First, there is a great variability between subjects in their ability to sleep in cold environments. This is not only evident in the earlier studies comparing Europeans to Australian Aborigines (Scholander et al., 1958, Hammel et al., 1959) and Bushmen (Ward et al., 1960), but in current studies within a

Caucasian sample (Buguet et al., 1977; Palca et al., 1986). Palca et al. (1986) noted that the subjects who exhibited the greatest amount of SWS also let their T_r drop to the lowest level during the night. What is not clear is whether low T_r is a function of greater SWS and low heat production, in accordance with the findings of Shapiro et al. (1984). Conversely, it could be that those subjects whose body temperatures dropped in a manner similar to that of the Australian Aborigines, sleep best in cold temperatures, exhibiting less disruption of sleep stages. An alternative explantion also proposed by Palca et al. (1986) suggests SWS is not disrupted as much as other stages of sleep due to the fact that it is most heavily weighted in early sleep before body temperature drops enough to affect sleep progress.

REM sleep is much more consistent in its response to cold as demonstrated by its decrease in almost all subjects (Buguet et al., 1976b; Buguet et al., 1977; Haskell et al., 1981b; and Palca et al., 1986). During studies of prolonged exposure, the most significant disturbance of REM sleep occurs during the first 2 to 3 nights, followed by a gradual recovery on the rest of the nights (Buguet et al., 1977). It has been suggested that due to the disruptive influence of REM on thermoregulation, this stage is selectively curtailed in an attempt to maintain body temperatures (Parmeggiani et al., 1969). Parmeggiani et al. (1969) proposed a model based on animal studies illustrating REM sleep as being the most sensitive to cold stress, with other stages, or transitions between stages, being affected with increasing degrees of cold.

Human studies have not produced as definitive results as those animal studies reviewed by Heller and Glotzbach (1977) and Parmeggiani (1981). In fact, there are many instances of unsuppressed REM in cold-exposed human subjects (Haskell et al., 1981b; Palca et al., 1986). These subjects were primarily those who slept well or claimed to sleep well in cold environments. The most obvious challenge to the theory of REM incompatibility with cold environments is the

previously reported peripheral vasoconstriction, increased metabolic rates, and steady T_r occurring during REM sleep in humans. This suggests that REM sleep is not the poikilothermic state as it is in animals. Recent theories state that the suppression of REM is the result of the nonspecific stress of cold (Haskell et al., 1981b; Palca et al., 1986). For example, Satinoff (1988) states that any treatment which increases stress can decrease REM (e.g. light, noise or hunger). Furthermore, it is well known that REM sleep increases as TST increases (Haskell et al., 1981b). If this is the case, then decreased REM is also a function of decreased TST. Support for this is also given by the subjects of Palca et al. (1986) and Haskell et al. (1981b) who exhibited normal TST and REM periods at low ambient temperatures.

VENTILATION DURING SLEEP

Normal atmosphere

Ventilation has been shown to undergo alteration of control during various sleep stages as compared to wakefulness. Sullivan (1980) described a conceptual hypothesis regarding the influence of sleep state on respiratory control. In general, breathing during quiet wakefulness is dominated by autonomic influences, while active wakefulness is controlled by behaviourial influences. The deep, regular breathing rhythm of NREM sleep is primarily mediated by pulmonary and chemoreceptor afferents. REM sleep is similar to wakefulness in that it is governed by both behaviourial and autonomic influences. This may partially explain the contrast in breathing pattern between REM and NREM, as ventilation becomes highly erratic with bouts of hyperventilation and apneas (hypoventilation) particularly during the phasic portion of REM.

An example of altered ventilatory response mechanisms during normal sleep in humans is illustrated by the work of Reed and Kellog (1958). They reported a decreased sleeping ventilation rate of 2 $L \cdot min^{-1}$ with a concomitant increase of P_ACO_2 by 3 - 5 mmHg. It was concluded that there is a decreased responsiveness of respiratory centres to CO_2 accounting, in part, for the decreased ventilation. Unfortunately, these data were not able to benefit from modern EEG interpretation and may be contaminated by stage effects or frequent periods of arousal reported by their subjects.

More recently, Gothe et al. (1981) compared ventilatory responses during awake and stage 2 sleep state (Figure 10). When ventilation decreased by approximately 0.7 $L \cdot min^{-1}$, which was primarily accounted for by a decreased tidal volume (V_T), end tidal PCO_2 rose from 39.1 to

42.5 Torr. One of the strengths of this report is the fact that ventilatory variables were measured by abdominothoracic displacement. Many studies rely on face masks or mouthpieces to monitor ventilation, or deliver hypercapnic and hypoxic gases. It has been demonstrated that alteration in breathing patterns results from the intervention of this equipment (Hirsch and Bishop, 1982). However, it must be kept in mind that measuring abdominothoracic displacements has limitations as well (Gothe et al., 1981).

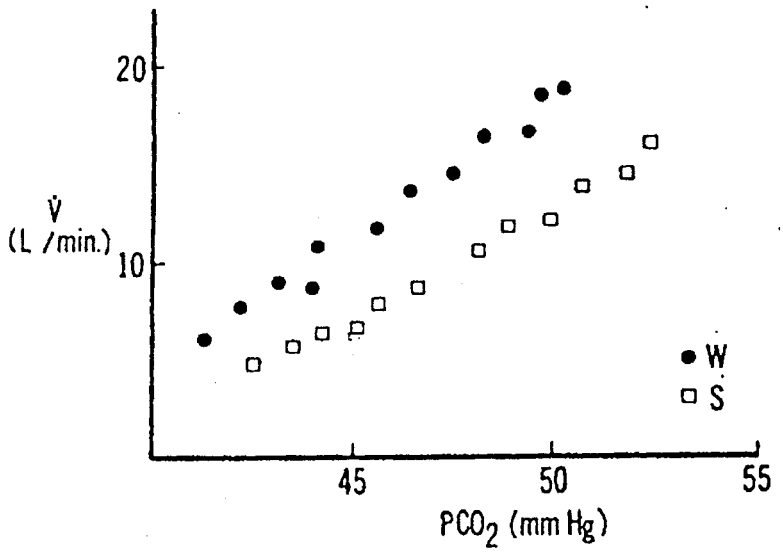

Figure 10. An example of ventilatory response to hypercapnia in 1 subject during awake (●) and stage 2 (□) sleep. (Gothe et al., 1981).

Colrain et al. (1987) initiated a comprehensive investigation to determine if the decreased ventilation associated with NREM sleep is associated with a decreased wakefulness drive. They were able to verify and expand upon the findings of the previous two studies. With the occurrence of stage 2 sleep, ventilation decreased 13 - 35% due in part to a diminished V_T and metabolic rate. It appeared that the critical event associated with the decreased ventilatory drive

was the shift in EEG from the α and β waveforms of quiet wakefulness to the stage 1 θ waveforms. This was interpreted as a removal of waking neural drive to respiration.

Hypercapnia

In order to evaluate respiratory control responses during sleep, hypercapnic inspired gases have been employed. Reed and Kellog (1958) exposed subjects to air containing 2, 4 or 6% CO_2. No change in the slope of the response curve to CO_2 was noted compared to wakefulness, but it was shifted slightly to the right so that higher P_ACO_2's were required to elicit waking ventilation values.

Fuleihan et al. (1963) measured ventilatory responses to 4% CO_2 in air using steady state exposures. They found no significant differences between sleep and wakefulness in end tidal PCO_2 or slope of ventilatory response to CO_2. This type of experiment is limited by the fact that no EEG was measured, and the CO_2 exposures lasted 30 minutes. It is very likely that changing sleep stages effects would confound these data.

More recent research has produced evidence contrary to that of Fuleihan and colleagues. Gothe et al. (1981) exposed subjects to a stepwise increment of CO_2 which raised the inspired fraction by 5% steps until arousal ensued. Each level was maintained for 4 minutes and ventilation parameters and end tidal gases were measured after they had stabilized for at least 2 minutes. Stage 2 sleep decreased the slope of the ventilatory response to hypercapnia by 79 $\pm$ 1% in all but one subject who showed no altered response. As mentioned previously, potential errors associated with this research lie in the fact that tidal volume was measured by thoracoabdominal displacement, not direct flow measurements. A second source of error is the

step increments of CO_2 to which the subjects were exposed. This amount of time does not represent a true steady state, but was necessary to avoid sleep stage changes during an exposure.

Modifications of the rebreathing technique described by Read et al. (1967) have been adopted as the hypercapnic ventilatory response method of choice. This is primarily due to the rapid achievement of equilibrium of the PCO_2 in the rebreathing bag, lung and arterial blood. Thus, end tidal PCO_2 can be measured as being representative of medullary PCO_2 (Berthon-Jones and Sullivan, 1984). As well, rapid measurements decrease the chance of sleep stage changing during the sampling period. Berthon-Jones and Sullivan (1984) utilized this technique to investigate ventilatory responses during NREM (stages 2, 3 and 4) and REM sleep. They found that male subjects decreased from 2.5 $\pm$ 1 $L \cdot min^{-1} \cdot Torr^{-1}$ to 1.49 $\pm$ 0.13 $L \cdot min^{-1} \cdot Torr^{-1}$ (49%) between wakefulness and NREM sleep, with no differences between stages 2 to 4. REM showed a greater reduction to 0.78 $L \cdot min^{-1} \cdot Torr^{-1}$ (69%). One point the authors neglected to address was the fact that REM sleep is characterized by erratic breathing patterns and apneas which may result in an apparent hypoventilation.

Finally, Hedemark and Kronenberg (1982) found no change in slope or position of the CO_2 response curve between wakefulness and SWS. REM data were not available due to the fact that the subjects became aroused from sleep rapidly during rebreathing. Probably the strongest case for the difference in results of this study and others is the fact that 5 out of 8 subjects were female. It has been demonstrated by Berthon-Jones and Sullivan (1984) that female ventilatory responses to hypercapnia are significantly less than males. Unfortunately, gender information was not available which would have allowed comparison of the male and female data.

CO_2 AND AROUSAL

It has been suggested that carbon dioxide may be disruptive to normal sleep. Early anecdotal evidence of this was presented by Schaefer (1949 in Schaefer, 1958) during a 3 - 6 day sojourn in an atmosphere containing 3% CO_2. During the first 24 hours, general excitement and agitation resulted in an decreased ability to fall asleep. The following morning, subjects felt very tired or a "hungover" sensation. After this initial 24 hr period of hyperactivity, a period of decreased attentiveness and irritability ensued. It was not until the third day that a somewhat baseline state was attained. These results may be explained by the remarks of Price (1960) regarding respiratory acidosis having both a depressant (cerebral cortex and myocardium) and excitatory (chemoreceptors, reticular formation and respiratory centres) function. It is now well known that adaptation to chronic respiratory acidosis is achieved via increased bicarbonate excretion. This partially explains the return to normal function by the third day in Schaefer's subjects. However, sleep deprivation due to the excitatory effects of CO_2 may also play an important role in the depressed function on the second day.

Recently, arousal responses to elevated F_ICO_2 have been evaluated during sleep or specific stages of sleep using rebreathing methods. As mentioned earlier, rebreathing methods are chosen because they are believed to be a good index of medullary PCO_2 and are insensitive to changes in cerebral bloodflow. As alveolar PCO_2 increases, arousal from sleep eventually occurs due to the hypercapnic distress. Hedemark and Kronenberg (1982) investigated arousal responses to CO_2 rebreathing during sleep. Although EEG was measured, no mention of sleep stage prior to arousal was given. Regardless, they found that a very narrow range of P_ACO_2 (49.2 $\pm$ 0.4 Torr) resulted in arousal in all subjects, and 86% of the hypercapnic tests. This suggests that arousal from sleep during hypercapnia is highly predictable and controlled,

occurring at a P_ACO_2 only 11.2 $\pm$ 1.1 Torr above resting levels. In contrast, arousal from a isocapnic hypoxic stimulus was not consistent, occurring over a wide P_AO_2 distribution in only 28% of the trials.

A similar study was conducted by Berthon-Jones and Sullivan (1984) who noted arousal occurring at increasing P_ACO_2's as sleep progressed from stage 2 (58.6 $\pm$ 0.9 Torr) to stage 4 (63.8 $\pm$ 0.8 Torr) in male subjects. Arousal from REM sleep occurred at a similar P_ACO_2 as stage 2. However, unlike arousal from NREM, arousal from REM sleep was not always abrupt. Some subjects showed transient arousals, or shifted to NREM before the final arousal. This suggests a REM instability during hypercapnic breathing. It was concluded that the primary stimulus for arousal was a combined effect of medullary PCO_2 and nasopharyngeal detection of CO_2.

CONCLUSIONS

Investigation of the combined effects of CO_2, sleep, and cold environments is a challenging venture fraught with complex relationships between the dependent and independent variables. Figure 13 is a schematic representation of the framework from which this thesis was developed and, in turn, was created through investigation of the relevant literature. From this figure, it is evident that even the simple investigation of the two variables, sleep and thermoregulation, is confounded by the reciprocal effects of environmental temperature and sleep. Although this type of experiment lends itself to simple manipulation of the stressor and analysis of the physiological response, the integrative processes resulting in the physiological output occur in a 'black box'.

Carbon dioxide was revealed to alter normal thermoregulatory responses to cold temperatures. In general, CO_2 increases the avenues of heat loss via augmented ventilation, sweating, altered subjective perceptions and inhibition of shivering. Admittedly, there is dispute in the literature as to the exact contribution of each of these responses to heat loss during sleep. However, there is overwhelming support for the disruption of normal thermoregulatory control during exposure to mild hypercapnia.

Sleep may be thought of as being an altered state of arousal. Thus physiological events occurring during wakefulness may be modified during sleep. For instance, it was shown that the set points for various thermoregulatory processes such as body temperature, metabolic rate, shivering and sweating are altered according to both a circadian rhythm as well as sleep stages. In general, during sleep the body is set to a lower thermal state. In stage REM, this is taken to the extreme with lower mammals which assume a poikilothermic state. Although not as extreme

in humans, REM stage has been demonstrated to disrupt normal thermoregulatory function.

Respiratory function is also altered during sleep. Ventilation rate and tidal volume decreases and arterial PCO_2 rises slightly. Furthermore, the ventilatory responses to CO_2 decrease as compared to wakefulness.

Further complicating the picture is the effect of the environment on sleep. Cold temperatures disrupt normal sleep patterns, decreasing total sleep time and sleep efficiency. NREM is affected in a highly variable way, yet REM is consistently decreased. Whether a decrease in stage REM is merely an artifact of decreased total sleep time or a thermoregulatory control mechanism has yet to be determined.

Finally, a hypercapnic atmosphere has been shown to induce arousal in sleeping subjects. This occurs at a very narrow range of alveolar PCO_2's in a high percentage of subjects. It is evident from this knowledge that CO_2 has the potential to disrupt normal sleeping patterns.

Each of the various components of Figure 11 has been investigated with varying vigour and thoroughness. However, the practical situation of winter camping has been proposed as encompassing the entire gamut of sleep, thermoregulation and hypercapnic environments. Obtaining adequate quantity and quality of sleep in a hostile environment could mean the difference between survival and tragedy. Due to the complex interaction of the variables, and paucity of related literature, it is believed that an investigation into the effects of CO_2 on sleep

and thermoregulation in cold environments is of value to the scientific and general communities.

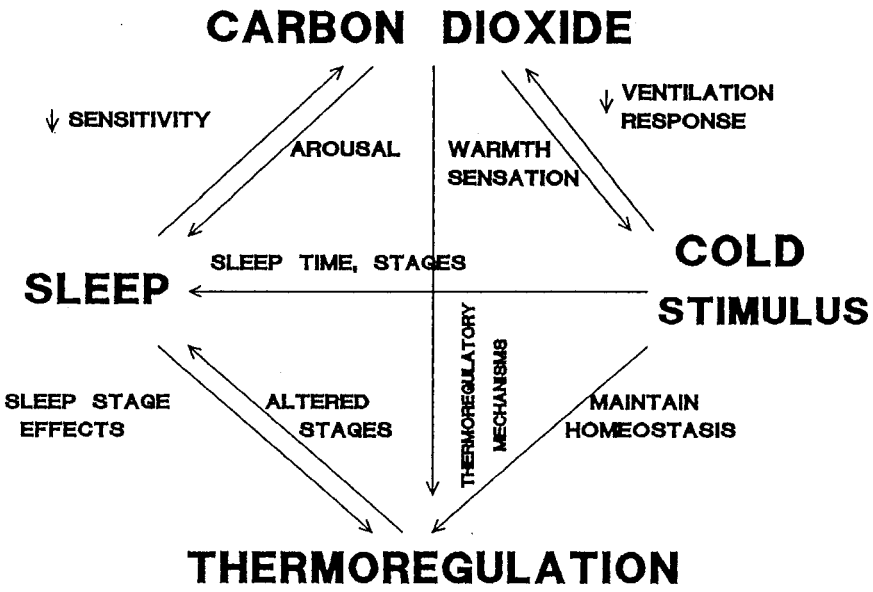

Figure 11. Schematic overview of the complex relationship between sleep, CO_2 and thermoregulation in cold environments.

CHAPTER III

METHODS

Subjects

A total of 5 male volunteers were examined in this study after giving their voluntary informed consent for participation (Appendix A) as required and reviewed by the ethics committees of Defence Research Establishment Ottawa (DREO) and the University of Ottawa. All subjects were members of the Canadian Forces and were chosen for their experience with cold weather camping and wearing a gas mask for extended periods. It was assumed that by selecting subjects familiar with these conditions, a greater probability of successful completion of the test period would result.

Subject screening

Potential subjects were invited to the DREO laboratory to become familiarized with the experimental equipment, and given an opportunity to breathe from the full facial mask. This procedure was intended to identify those volunteers who were not prepared to commit themselves to the duration of the experiment or were uncomfortable with the experimental apparatus.

The subsequent screening procedure was carried out at the University of Ottawa School of Psychology Sleep Laboratory where individuals exhibiting severe medical or psychological

pathologies or sleep disorders could be identified. A detailed sleep behaviour questionnaire and an in-depth interview was initially employed to detect such individuals. As well, all subjects were required to complete the Minnesota Multiphasic Personality Inventory (MMPI) and Spielberger's State Trait Anxiety Inventory (STAI) as further measures to detect psychological disorders as well as susceptibility to stress. Those individuals indicating habitual use of drugs and/or alcohol were not retained. Finally, every candidate was required to submit to a full medical examination to minimize the possibility of health risks complicating their participation in this experiment.

APPARATUS

Environmental chamber

All cold experiments were conducted inside DREO's environmental chamber. This climate controlled chamber measured 4.6m x 6.4m and the temperature could be controlled from -45°C to +45°C $\pm$ 0.1°C. Since the experiments were conducted in very cold conditions (-20°C), it was necessary to locate the data collection hardware in an adjacent laboratory. A window in the wall of the chamber permitted visual monitoring of the subject. As well, ports and electrical junction boxes allowed the connection of breathing tubes and various sensors between the chamber and the laboratory. An intercom system permitted communication between subject and experimenters while the experiment was in progress.

In an attempt to provide a more intimate environment and attenuate the sound of the air circulating fans inside the chamber, a small "igloo" measuring 1.2m x 1.2m x 2.4m had been constructed of 4cm styrofoam panels. A plexiglass viewing window was installed, as well as a

30.5cm port connected to an industrial exhaust fan which ventilated air through the igloo to prevent heat accumulation.

Sleeping ensemble

The sleeping bags employed in this study consisted of a standard issue Canadian Forces Cold Weather Sleeping Bag (NSN 8465-21-842-6079). This bag was composed of two down-filled bags which could be assembled with one inside the other in extreme cold or the inner layer could be used alone in more temperate climates. A double bag yields an insulative value of 9.0 clo (1 clo = 0.155 $°C \cdot m^2 \cdot W^{-1}$) which will maintain thermal comfort for approximately 8 hrs at -20°C (Farnworth et al., 1985). The inner bag alone had roughly half the insulation of the combined ensemble.

Supplemental to the sleeping bag, each subject was supplied with an insulated sleeping hood (NSN 8465-21-842-6081) which protected the head, neck and shoulders, and was maintained in place by two elastic bands under the axilla and a velcro closure in front of the neck. To minimize heat conduction to the floor, a 1.2m x 2.4m x 2cm sheet of plywood was laid down on the flooring, and the subjects slept on a self inflating air mattress (Thermarest Inc., Seattle WA) taped to a 4cm honeycombed, insulated prototype mattress.

Recordings

Standard EEG, EOG, and EMG measurements of sleep were applied according to the methods of Rechtschaffen and Kales (1968). This information was recorded on a six channel Polysomnograph (Grass model #716Y2M). Polysomnograms were scored by an experienced

research associate whose scoring competence had been previously established by the University of Ottawa's School of Psychology's Sleep Laboratory by qualitative comparison to the scoring of a qualified, skilled sleep researcher.

Temperature measurements

Skin temperature was monitored at seven skin sites (Figure 12) as well as the middle finger and great toe on the non-dominant side using thermistors (YSI 44004, Yellow Springs Ohio) affixed to the skin with Blenderm Surgical Tape (3m Co.). Rectal temperature was measured by inserting a rectal thermistor (YSI series 400, Yellow Springs Ohio) approximately 5-8 cm into the rectum as described by Sawka and Wenger (1988). Temperatures were recorded at 1 minute intervals throughout the night and stored on magnetic diskette using an automated data acquisition system (HP 85 computer and HP 3497A Data Acquisition/ Control Unit). Temperatures were visually inspected and data which appeared invalid due to faulty leads, or removal of the thermistors due to subject movement pulling off the adhesive tape or slippage of the recal probe, were removed. In some instances, faulty thermistor leads produced sporadic electrical shorts of 1 or 2 minutes duration. These values could be interpolated and manually corrected by assuming there were no dramatic change in temperatures over this short time.

Skin and rectal temperatures were assigned a weighted value and summated to provide a calculation of mean skin temperature ($\bar{T}_{sk}$) and mean body temperature (T_b) according to the equations of Hardy and Dubois (1938) and Burton (1935), respectively. Each skin temperature was represented as follows:

T_1 = Forehead
T_2 = Forearm
T_3 = Hand
T_4 = Foot
T_5 = Shank
T_6 = Thigh
T_7 = Abdomen
T_8 = Rectal

where

$$\bar{T}_{sk} = .07(T_1) + .14(T_2) + .05(T_3) + .07(T_4) + .13(T_5) + .19(T_6) + .35(T_7)$$

and

$$T_B = .33(\bar{T}_{sk}) + .67(T_8)$$

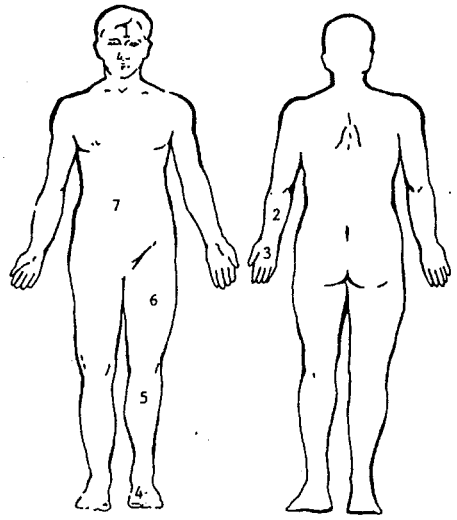

Figure 12. Thermistor placement for measurement of mean skin temperature.

Finger and toe temperatures were measured in a similar manner and will be assumed to represent the vascular state of the extremities (Burton and Edholm, 1955).

Compressed gases

Medical quality breathing gases were supplied to the subject from compressed air cylinders containing 6000-8000 L of gas. Air pressure was governed by a Regulated Demand Valve (Model MD-1, Bendix Aviation Corp., N. Hollywood CA) to ensure minimal resistance on inhalation. The demand valve was connected to approximately 3 m of 3.2 cm diameter, corrugated tubing which passed through the wall of the environmental chamber to the subjects sleeping chamber. This tubing terminated at a flexible connection to an inlet port of a full facial aircrew mask (US XM4) which was worn by the subject. Expired gases were exhausted through approximately 3 m of the corrugated tubing through the chamber wall into a mixing box where expired gases were collected for metabolic analysis. A one way valve situated between the mixing box and the chamber wall prevented refluxive flow of air down this tubing during inhalation. Figure 13 illustrates the flow of gases from the compressed gas cylinders to the subject inside the cold chamber and subsequent venting to the adjacent lab for analysis.

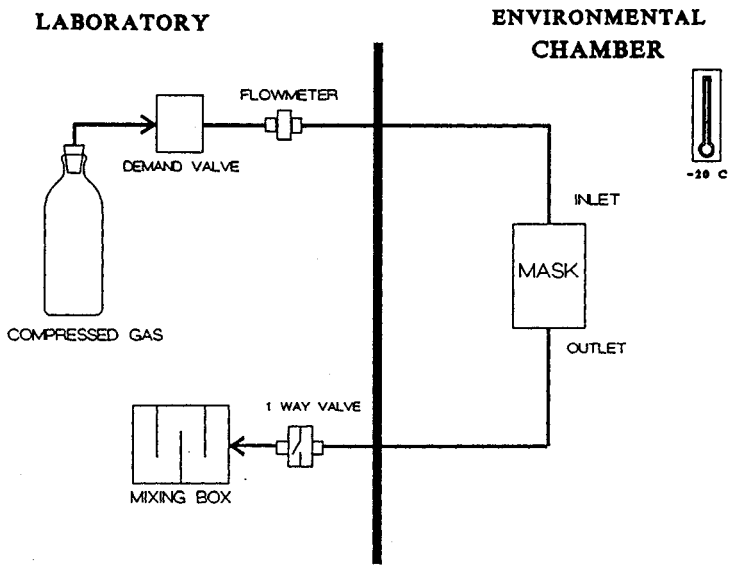

Figure 13. Schematic flow of inspired and expired gases.

Metabolic rates

Metabolic and respiratory parameters were collected and analyzed utilizing an open circuit breathing apparatus. Inspiratory gas volumes were measured by a digital flowmeter (K L Engineering, Sylmar CA) and expired gases were analyzed for F_ICO_2 and F_IO_2 by an Ametek CD 3A CO_2 analyzer and an Ametek S - 3A O_2 analyzer (Ametek/Thermox Instruments Division, Pittsburgh PA). Digital and analog outputs were sampled every minute by a Metrabyte DAS8 A/D converter (Metrabyte Corp., Tauton MA) controlled by an IBM-AT compatible computer. The data were processed by computer to calculate a number of standard metabolic and ventilatory variables which were then stored on diskette.

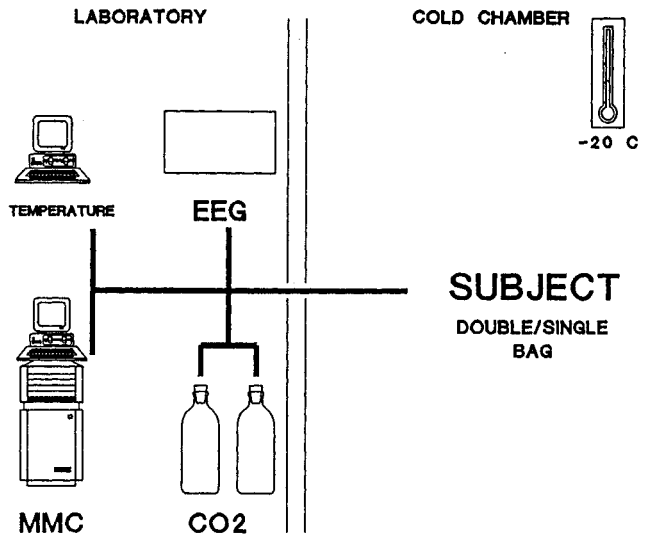

Figure 14. Overview of the experimental apparatus configuration.

EXPERIMENTAL DESIGN

In an attempt to fully elucidate the effects of mild hypercapnia on thermoregulation during sleep while concurrently addressing the practical applications, an experiment was designed which included reasonable levels of CO_2 and involved different challenges to thermoregulatory mechanisms. Exposure of subjects to 0, 2 or 4% CO_2 in air concurs with the range of values measured throughout the night in sleeping bags by Livingstone et al. (1988). An environmental temperature of -20°C, and 50% R.H. is a reasonable condition which would be encountered during cold weather camping, and was selected as the ambient temperature during the environmental chamber tests.

Finally, as noted earlier, a double layered CF sleeping ensemble had been determined to provide adequate insulation to maintain a thermoneutral state for approximately 8 hours at -20°C. It was determined by pilot work that the inner bag by itself would allow several hours sleep while representing a definite cold challenge to the thermoregulatory system. Sleeping bags were selected over nude exposure as this study has direct practical implications and it was perceived that the microenvironment surrounding a person would be different in a sleeping bag as compared to full body exposure. Furthermore, this is a method which has been successfully implemented in several relevant experiments (Buguet et al., 1979a; Livingstone et al., 1988), thereby permitting an extension of previous research. For these reasons the experimental conditions to which the subjects were subjected were as follows:

University of Ottawa Sleep Laboratory

2 Baseline/Screening Nights

Defence Research Establishment Ottawa (DREO)

1) Adaptation Night (0%, Double Bag)
2) 0% CO_2 Balance Air, Double Bag
3) 0% CO_2 Balance Air, Single Bag
4) 2% CO_2 Balance Air, Double Bag
5) 2% CO_2 Balance Air, Single Bag
6) 4% CO_2 Balance Air, Double Bag
7) 4% CO_2 Balance Air, Single Bag

During the baseline testing at the University of Ottawa, EEG was recorded only on the second of the two nights, with the first night serving as a trial to accommodate for any first night effects (Agnew et al., 1966). The subjects were not equipped with thermistors, rectal probe,

sleeping bag or facial mask. A second night served a dual purpose in that baseline sleep architecture as well as any sleep abnormalities such as apneas, myoclonic twitches or abnormal sleep patterns could be further identified. If such abnormalities were detected, then the subject would have been asked to withdraw from the study.

Condition 1 of the DREO experimental chamber nights also served as an accommodation night allowing the subject to adjust to the new surroundings and apparatus. Conditions 2 - 7 were then introduced in a completely randomized manner with at least 1 day rest between exposures to minimize the effects of cold adaptation demonstrated by Kreider et al. (1959), or contamination of the data due to a previous nights exposure. In an attempt to minimize the effect of readaptation to the experimental conditions, subjects were tested 2 to 3 times per week. Paired t-statistics did not reveal any effects between the two trials of the same CO_2 and sleeping bag condition ($p < .05$), thus the two nights were averaged for subsequent analysis.

EXPERIMENTAL PROCEDURE

Baseline data - University of Ottawa

Once subjects had been identified and sufficiently screened, they were asked to report to the University of Ottawa's Sleep Laboratory at approximately 2100h, for the two consecutive nights of sleep. Each subject was advised to refrain from alcohol consumption for the preceding 24 hrs, smoking or caffeine for 12 hrs, and meals for at least 3 hrs. They then proceeded to be instrumented with the standard polysomnographic electrodes (EEG, EMG, and EOG), as well as additional EMG electrodes on the upper non dominant leg,u and then donned their night clothes. A mercury gauge plethysmograph sensor secured to an elastic band was fastened around the

subjects thorax to monitor respiratory movements. The subjects then completed a STAI questionnaire and retired to the laboratory's sleeping room at no later than 2230 h. Sleep chamber temperature was maintained at 20°C and the subjects were free to select any amount of bedding necessary to maintain thermal comfort. During the second night, all physiological variables were constantly monitored. As well, the subject was visually monitored by the use of an infrared sensitive camera. Upon awakening at approximately 0600 h, the subjects completed an additional STAI questionnaire as well as a sleep satisfaction index (Appendix B) which gave an subjective indication of how well they slept.

Environmental chamber - DREO

A preparatory procedure similar to that of the University of Ottawa baseline nights was followed at DREO. Subjects reported to the laboratory at approximately 2100h after abstaining from alcohol, caffeine, smoking and food for the same periods as previously outlined. Upon arrival, they proceeded to be instrumented with a rectal thermistor, skin thermistors and EEG, EOG and EMG electrodes and changed into their Canadian Forces (CF) issue long thermal underwear and cotton socks. After completion of the STAI questionnaire, the subject then donned CF issued duffle socks and an insulated sleeping hood. Prior to entering the environmental chamber, the full facial mask was affixed and securely fastened to the subject by snugly tightening the elastic straps of the attached head harness. An inspection for leakage was made by asking the subject to block both inlet and outlet ports with their hands while forcefully exhaling and inhaling. It was assumed that if no air leakage was noted until well above typically encountered positive and negative pressures, normal unimpeded breathing would not result in leakage. However, with subject movement and cold temperatures it was conceivable that some leakage may have occurred sporadically. Since the compressed air cylinders were governed by

a demand valve, air coming in from the cylinders was under a slight positive pressure and assisted in the prevention of the inflow of ambient air if the face seal leaked during inhalation. Inspiratory leaks were rare and could be detected by sudden changes in expired air composition. Leakage on expiration would not have affected volume measurement since this was determined on the inspired side. It was presumed that leakage on exhalation would not have interfered with the expired gas composition due to the fact that the positive pressure of exhalation would prevent ambient air from entering the collection tubing.

Once inside the chamber, the subjects were quickly led into the sleeping igloo where they climbed into a double or single layered sleeping bag. Inspiratory and expiratory air hoses were connected to the face mask, and thermistor and electrode leads connected to the junction box on the wall of the chamber. Every effort was made to ensure that bedding down time occurred no later than 2300 h. Once it was established that all sensors were giving proper readings, the lights in the chamber were extinguished except for a small amount of diffuse light allowed to filter in through the window from the adjoining laboratory.

Wake up time normally occurred at approximately 0600h or earlier if the subject was unable to sleep any longer. At this time, the subject was disconnected from the experimental apparatus and quickly exited the chamber. The session was terminated with the subject completing a STAI and sleep satisfaction questionnaire. Figure 15 gives an indication of the time and sequence of subject preparation procedures.

2100 h	2115 h	2215 h	2300 h	0600 h
Subject arrives	Placement of EEG electrodes and temperature probes	Subject enters sleeping bag	Lights out	Wake up
Questionnaires completed				Complete questionnaires

Figure 15. Time line of subject preparation and test period.

DATA TREATMENT

Primary analysis

Both the first and second hypotheses were tested through the employment of a repeated measures 2 X 3 (sleeping bag X CO_2) ANOVA. The independent variables tested were:

a) Time to minimum T_b
b) Minimum T_b
c) Time to minimum T_r
d) Minimum T_r
e) Time to minimum $\bar{T}_{sk}$
f) Minimum $\bar{T}_{sk}$
g) Time to minimum T_{toe}
h) Minimum T_{toe}

Single degree of freedom polynomial contrasts (Wilkinson, 1990, p. 275) were performed to identify any trends (linear or quadratic) as a result of significant main effects of CO_2.

Secondary analysis

Subsequent analyses addressed the relationship between sleep stages and body temperatures during sleep, while controlling for each of the six test conditions. The sleep stages identified for analysis included stage 2, slow wave sleep (stages 3 and 4 combined), and REM. After inspecting the resultant data, sleep efficiency and the number of awakenings were also included in the analysis, due to their apparant association with body temperatures.

A general linear regression model was developed which tested the relationship between these sleep parameters and the dependent variables of minimum T_b, T_r, T_{toe}, and $\bar{T}_{sk}$. This model was of the form

$$DV = \text{Constant} + b_1\text{Bag} + b_2CO_2 + b_3\text{Bag}CO_2 + b_4\text{Regressor}$$

Where:

DV = dependent variable
b_x = partial regression slope
Bag = sleeping bag condition, 1 = single, 2 = double
CO_2 = CO_2 condition, 1 = 0%, 2 = 2%, 3 = 4%
Regressor = independent variable

It was anticipated that the data analysis described in this section would provide comprehensive interpretation of the sleep, metabolic and temperature data resulting from this experiment, and provide greater understanding of the complex effects of breathing CO_2 on sleep and thermoregulation in cold environments.

CHAPTER IV

RESULTS

Subjects

In response to our call for subjects, 16 members of the Canadian Armed Forces presented themselves for the initial briefing and pre-screening. One was rejected based on the medical examination, while the remaining 15 proceeded to complete the baseline sleep nights. Unfortunately, due to a myriad of circumstances, such as poor motivation, scheduling difficulties, early postings, and a fire in the laboratory which delayed work for several months, only 5 subjects remained to complete the study. Even amongst these subjects, difficulty was experienced in completing all 13 nights in the cold due to the length of the experiment and transient nature of their posting. Of the 5 subjects, 3 completed the full 13 nights of the experiment, while 1 completed 12 and the other completed 10. This yielded a total of 61 nights of data (Appendix C).

A large drop out rate was not entirely surprising as it was well understood that this study could be quite arduous and required highly motivated subjects. It was perceived that the greatest factor limiting participation was the necessity to wear a full facial mask. This produced a certain degree of claustrophobic anxiety in some subjects. Those who tolerated the mask reported having no subjective problems at all. In response to the varied responses of subjects, each candidate was invited to DREO to try on the mask and rest for at least 1/2 hour. This proved to be successful

in identifying and eliminating several potential subjects who were comfortable with the procedures, but not with the mask.

For the 5 subjects who did remain, their anthropometric characteristics are given in Table 2.

Table 2. Anthropometric Characteristics of Subjects

Subject	Height (cm)	Weight (kg)	Age (yrs)	BMI (kg/m^2)
1	173	68.2	20	22.8
2	170	63.6	25	22.0
3	167	78.0	24	28.0
4	178	67.0	24	21.1
5	170	72.3	25	25.0
=====	======	======	======	======
Mean	171.6	69.8	23.6	23.8
±SD	3.7	4.9	1.9	2.7

Sleep architecture results - University of Ottawa

As mentioned earlier, due to the interdisciplinary nature of this study, the University of Ottawa's Sleep Laboratory produced a separate report detailing findings of their collaborative work at the DREO environmental facilites on the effect of CO_2 and cold on sleep architecture. Complete results are detailed in an DREO contractor report completed by Dr J. Dekoninck (1991). The following is a summary of those findings on the effects of CO_2 and a cold environment on sleep architecture.

As previously mentioned, prior to the main testing in the DREO environmental chamber, the second of two nights sleep at the University of Ottawa's sleep laboratory was recorded. When this baseline laboratory sleep (Appendix D) was compared to the first night exposure to the double bag, 0% CO_2 baseline condition in the DREO environmental chamber, significant decreases ($p < .05$) were noted in total sleep time, sleep efficiency, REM and sleep satisfaction in the chamber. It would have been preferable to have included comparisons between the sleep laboratory and subsequent double 0% nights, since the night compared was the first night in the DREO environmental chamber. This resulted in difficulty determining whether these sleep changes were a result of the experimental procedure or a first night effect.

In the environmental chamber, comparisons between sleep in the single or double layered sleeping bag conditions revealed statistically significant ($p < .05$) decreases in total sleep time, sleep efficiency and REM in the single bag. Sleep satisfaction also tended towards a decrease in the single bag, approaching significance ($p = .08$).

In 4 subjects, slow wave sleep and sleep satisfaction tended to decrease with increasing levels of CO_2. However, CO_2 variations produced no statistically significant differences across all variables and sleeping bag conditions.

It was concluded that sleep in the cold chamber adversely affected most sleep architecture variables as well as subjective perceptions of sleep. Single and double bag effects were also in agreement with previous research, with adverse changes occurring in many sleep variables. Concern was raised by the author as to CO_2's suppression of the natural tendency of slow wave sleep to increase with cold exposure, as this has been previously described as being an important aspect of sleep.

Data file revisions

Examination of the data files revealed that on several nights, recordings were prematurely terminated due to equipment failure, or because the subject experienced an abnormal sleep onset latency (SOL). As the intent of this study was to investigate the effects of CO_2 on sleep and thermoregulation in cold environments, an arbitrary decision was made to implement rejection criteria to eliminate the poorest nights and guarantee adequate sleep data. Only those nights which resulted in at least 3 hours of sleep data and 5 hours temperature data were selected. Of a total of 61 nights of sleep collected, 10 could be rejected solely on the basis of inadequate sleep, while 8 could be rejected on the basis of total time. The total number of files failing to simultaneously satisfy both criteria equalled 6 or approximately 10% of all nights. It was these files which were then rejected. These rejected nights were all single bag conditions. This weakened the database somewhat since each condition was to be repeated and averaged for each subject. Thus in some cases, a subject's data consisted of only a single night of sleep, or no available data at all. A schedule of the revised data is given in Appendix C. Unfortunately, missing cells presented problems in the Repeated Measures ANOVA, further reducing the number of subjects and statistical power.

Re-analysis of sleep data

Following the restructuring of the data file, the data of DeKoninck (1991) were reevaluated to determine if any statistical changes ensued. In brief, the summary of the edited data analysis is given in Table 3 and detailed in Appendix D. The editing of the data base primarily served to readjust mean values towards better sleep, but several interesting differences

did emerge.

As expected, data revisions resulted in an increased total sleep time (TST) in all conditions, however, the difference between the double and single bag only approached significance ($F=5.886$,$p=.072$), with a double layered sleeping bag resulting in greater TST. Despite an apparent trend towards decreasing TST with increasing CO_2 there was no significant effect of CO_2 or an interaction between the sleeping bag conditions.

Other effects of revising the data included a decreased sleep onset latency in the single bag which approached significance ($F=7.435$,$p=.053$). REM sleep remained significantly lower in the single bag ($F=8.58$,$p=.041$). As well, a previously significant main effect of sleeping bag condition on sleep efficiency became non significant. Finally, the trend towards an increased SWS with cold stress, and decreased SWS with increasing levels of CO_2 remained, however, it was not as apparent as in the original data set.

In conclusion, editing the database served to readjust the means in the direction of better overall sleep. As well, minor changes in the results of the statistical analysis ensued. However, the trends and conclusions determined from the original data sets remained intact. It is likely that the use of a greater number of subjects would have increased the statistical power and yielded further significant relationships.

Table 3. Revised sleep variable means (±SD)

	Single Bag	Double Bag	0% CO_2	2% CO_2	4% CO_2
SE	68.5 (21.3)	80.3 (13.0)	76.9 (15.9)	73.2 (20.7)	73.1 (19.8)
TST (min)	245.4 (93.3)	313.1 (58.4)	290.9 (76.9)	284.3 (90.2)	262.5 (90.1)
SOL (min)	14.2 (9.3)	24.5 (24.3)	16.1 (12.3)	25.6 (27.8)	16.3 (12.4)
Stage 2 (%)	70.2 (7.6)	69.8 (9.1)	69.6 (7.4)	70.6 (8.9)	69.8 (9.2)
REM (%)	6.6 (5.2)	11.1 (5.8)*	10.0 (4.1)	8.2 (6.5)	8.4 (7.1)
SWS (%)	10.4 (6.7)	7.2 (4.2)	10.4 (5.0)	9.0 (6.3)	7.1 (5.9)

* - different from single bag ($p < .05$)

THERMAL DATA ANALYSIS

Decision not to use metabolic data

Obtaining accurate metabolic rates proved to difficult under the environmental conditions experienced. The primary difficulties arose from the fact that moisture in the exhaled breath condensed and froze, blocking the tubing or increasing expiratory resistance. Furthermore, the long tubing needed to vent the exhaled air to the mixing box in the adjoining lab, although minimal in length (approximately 2.4 m), when combined with gradual icing provided enough resistance to cause claustrophobic distress in some subjects. An attempt was made to rectify the icing problem by insulating the expired tubing delayed freezing, but was not entirely successful.

Secondly, ventilation rates, as measured by a digital turbine ventilometer, were suspicious. The range of the ventilometer was between 3 and 1000 $L \cdot min^{-1}$. During sleep, ventilation rates as low as 3 to 5 $L \cdot min^{-1}$ are common (Colrain et al., 1987). This, in combination with the often erratic breathing patterns associated with REM sleep, seriously challenged the capabilities of this measurement. In retrospect, the approach of directing 5 to 10 minute samples of expired air to large volume storage and subsequent analysis would have been a superior approach.

These factors combined to affect metabolic measurements to a degree that they were deemed to be unreliable, or suspect. As a result, they were excluded from this study.

Sleep satisfaction

By sleep trial sequence

Sleep satisfaction was evaluated by the order of the night's appearance in the experiment. This was done with the intent of examining the potential effect of the number of trials on subjective perception. In general, subjects tended to score their sleep satisfaction between values of 6 and 8 on a scale of 0 to 10. There appeared to be a trend towards increasing sleep satisfaction with greater number of nights slept (Figure 16) but this is somewhat misleading. The first night was scored lower than (mean = 4.4), but not remarkably different from, the other subsequent nights, due probably to the large variability of this nights score. With the removal of the lowest subject's score (Subject 4, score = 1) this night received an average satisfaction of 5.3, which is still slightly depressed relative to the subsequent scores. In all, 3 of the 5 subjects scored night 1 lowest, suggesting that an adjustment to the environmental conditions was still occurring. It should be reiterated that the first night referred to in this analysis is actually the second night in the environmental chamber, as one adaption night under double bag, 0% CO_2 was previously obtained.

Satisfaction scores on nights 10 through 12 are most likely elevated due to the fact that two subjects were not able to complete these nights as a result of scheduling difficulties. The three remaining subjects included the two highly motivated subjects who tended to score high on all nights, thus these average scores may tend to be biased.

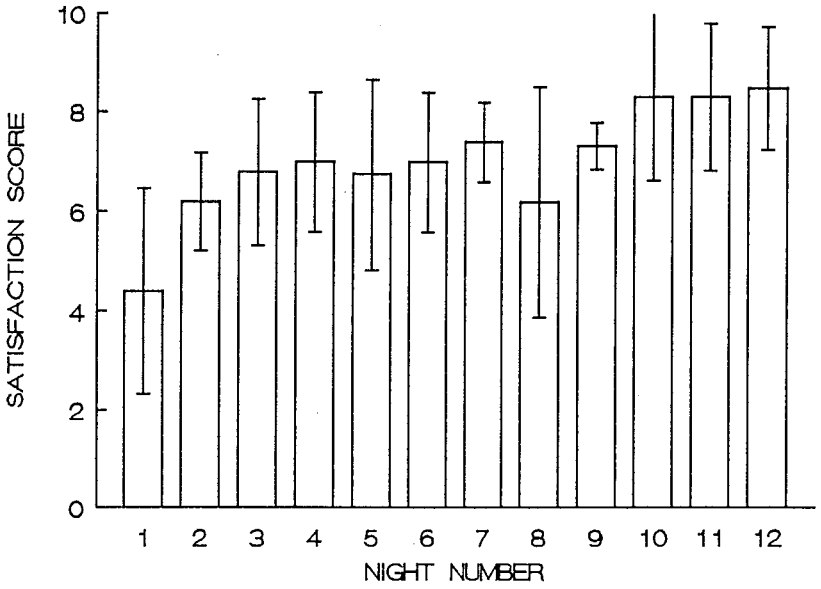

Figure 16. Sleep satisfaction according to the experiment night.

By condition

Comparing the effects of sleeping bag insulation on sleep satisfaction indicated a greater preference towards the double bag condition, although this difference was relatively small (0.8 units) and not significant (Table 4).

Across CO_2 conditions, controlling for insulation did not result in any apparent main effect of CO_2 on subjective perceptions.

Table 4. Sleep satisfaction means (±SD) by group

	Single Bag	Double Bag	0% CO_2	2% CO_2	4% CO_2
Sleep Satisfaction	6.2 (1.6)	7.0 (1.5)	6.6 (1.7)	6.9 (1.3)	6.4 (1.8)

Mean body temperature (T_b)

Hourly readings

Mean body temperatures were reduced to hourly readings for ease of calculating means and presenting temperature trends. Tabulation of the temperatures began 1/2 hour after the initial reading in order to allow stabilization of individual body temperatures. This was done to control for temperature effects of walking through the cold chamber, change of posture and climbing into a cold sleeping bag. Figures 27 and 28 illustrate that skin temperatures are affected most by the transition from the preparation room to the sleeping bag, and are relatively stable after this time period. These data revealed that the strongest effect on T_b was the amount of sleeping bag insulation (Figures 17 and 18). Double bag temperatures were consistently 0.5 to 0.8°C greater than single bag and were maintained at a relatively constant level, with the lowest recorded minimum of 34.5°C in the 0% CO_2 condition by the 6th hour. In the single bag, all subjects T_b's were below 34.5°C by the third hour, and had reached a minimum in the single bag, 0% CO_2 condition of 34.0°C by the 5th hour.

Breathing CO_2 showed no consistent or obvious effects on T_b. Initial temperatures in the single bag seem to be slightly elevated in the CO_2 conditions, but by the second hour there were no discernable differences. In the double bag, 4% CO_2 was associated with a tendency towards higher body temperatures, but of only 0.1 to 0.2°C. There were no apparent effects of 0% and 2% CO_2 on mean body temperature in the double bag.

Statistical analysis revealed a significant interaction between sleeping bag and CO_2 conditions ($F=10.495$, $p=.046$) after 4 hours, with a second order (quadratic) trend ($F=11.59$,

p=.042) (Table 5). As initial temperatures in the single bag were not equal (Figure 18), the drop in T_b at the 4 hour mark was calculated. This new variable demonstrated only a main effect of the sleeping bags (F=18.25, p=.024).

Drop in T_b

The total drop in mean body temperature was measured by the difference between the minimum T_b, and T_b after 30 min. in the bag. This approach was followed to determine if it yielded more information than the minimal temperature because it took into account the initial body temperature. On average, mean body temperature dropped to a greater degree in the single bag as compared to the double bag (Table 5). This is statistically significant (F=21.12, p=.019). There was no effect of CO_2 or any interaction between sleeping bag condition and CO_2 on the drop in body temperature; however, there was a trend towards less drop in T_b with elevated levels of CO_2.

Minimum mean body temperature (minT_b)

Mean body temperature was affected by sleeping bag insulation, resulting in a lower minimum T_b in the single bag (Table 5). This difference was very small, amounting to only 0.6°C, but reached significance (F=26.088 ,p=.015) independent of the level of CO_2 breathed.

Across levels of CO_2, minT_b's were not significantly affected. However, there appeared to be a slight trend towards body temperatures being maintained at a higher level with increasing levels of inspired CO_2.

Time to minimum T_b

Although not significant statistically, the main influence of sleeping bag insulation was for subjects to reach their minimal body temperatures sooner in the single bag as compared to the double bag (Table 5).

Examining the interactive effects between CO_2 and sleeping bags suggest a result parallel to that shown by subjective scoring, in that the greatest difference between the single and double sleeping bag occurred with 0% CO_2, while 2% and 4% CO_2 yielded similar results (Appendix E, Figure 5). In the double bag condition, the time to minimum temperature decreased with increasing inspired CO_2, while with the single bag, time to minimum T_b actually increased from 0% to 2% CO_2 conditions, after which there was little difference between double and single bag when breathing CO_2.

Table 5. Mean values ($\pm$SD) for T_b by grouping

	Single Bag n=12	Double Bag n=12	0% CO_2 n=8	2% CO_2 n=8	4% CO_2 n=8
T_b after 4 h (°C)	34.1 (0.3)	34.7 (0.3)	34.4 (0.4)°	34.4 (0.4)°	34.3 (0.5)°
Change in T_b after 4 h (°C)	-0.9 (0.5)	-0.5 (0.4)*	-0.7 (0.4)	-0.7 (0.4)	-0.7 (0.7)
Drop in T_b (°C)	0.9 (0.5)	0.5 (0.6)*	0.9 (0.7)	0.7 (0.5)	0.6 (0.6)
minT_b (°C)	34.1 (0.4)	34.7 (0.6)*	34.2 (0.7)	34.4 (0.4)	34.5 (0.6)
Time to minT_b (min)	296.5 (51.4)	310.1 (80.8)	313.6 (63.5)	324.4 (52.1)	271.9 (77.9)

* - Significant from single bag ($p < .05$)
o - Significant interaction of bag and CO_2 (quadratic trend, $p < .05$)

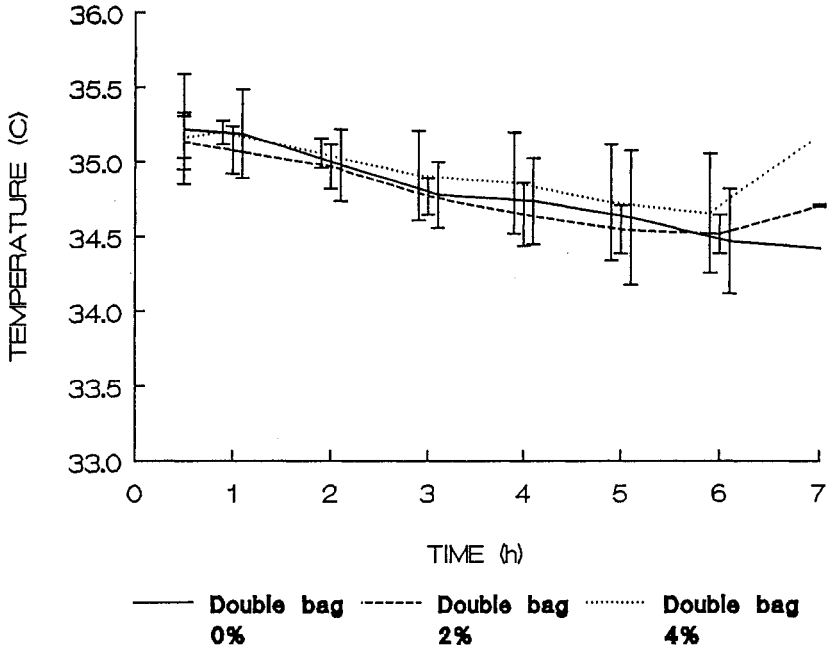

Figure 17. Hourly mean body temperature in each CO_2 condition (double bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

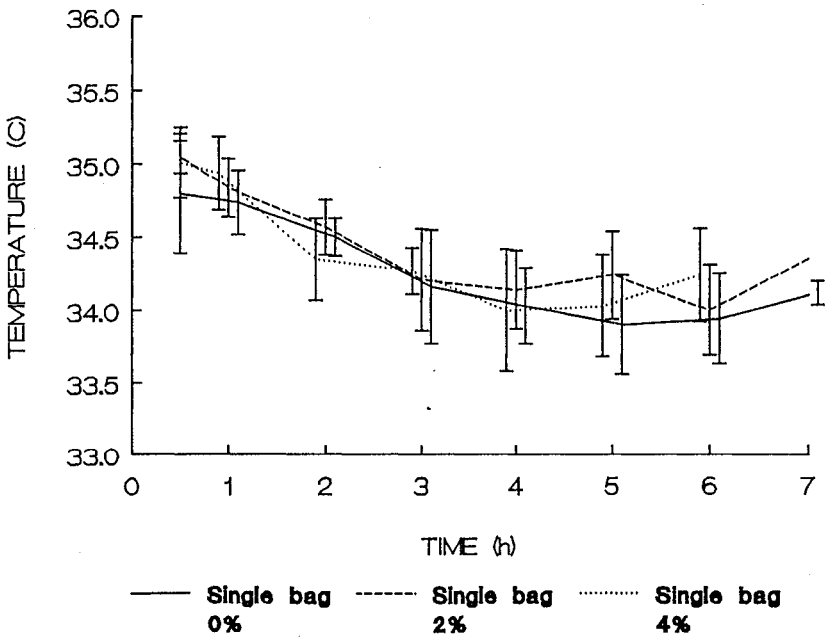

Figure 18. Hourly mean body temperature in each CO_2 condition (single bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

Rectal temperatures T_r

Hourly readings

Rectal temperatures throughout the night were also influenced by the sleeping bag condition. Single bag rectal temperatures were lower by the first hour and remained lower throughout the night (Figures 19 and 20). The greatest drop in T_r appeared to occur during the first 2 to 3 hours. Subsequently, it continued to decline gradually, with the T_r in the double bag being approximately 0.2°C greater than in the single bag. By the fourth hour, the T_r in the double bag had fallen significantly less than in the single bag ($F=24.625$, $p=.016$) (Table 6).

An early morning rise in T_r was not as evident as has been reported in the literature. A slight rise in T_r was common in the double bag condition, but infrequent in the single bag. Hourly data were averaged from available data and may not have included all subjects, particulary as some dropped out towards the 6 to 7 h mark. This may give somewhat misleading results in the latter hours of the trial. All subjects completed at least 4 hours in the chamber, and 4 subjects completed 5 hours.

Drop in T_r

Reducing the insulative value of the double bag by 50% (single bag) resulted in a greater drop of T_r. The average drop of T_r in the single bag compared to the double bag differed by only 0.1°C (Table 6). Although this difference seems to be slight despite the large difference in insulation, it approached statistical significance ($F=8.929$, $p=.058$).

Although the interaction between CO_2 and sleeping bag insulation was not significant, there was a slight trend towards less decrease in T_r with increasing levels of CO_2 in the single bag. In the double bag, the slight trend towards increased drop with elevated CO_2 levels (Appendix E, Table 8) was similarly not significant. There was no influence of the level of CO_2 on the amount of drop of rectal temperatures.

Minimum T_r

Surprisingly, despite the large difference in insulation between sleeping bag conditions, the lower minimum value of rectal temperatures in the single bag as compared to the double bag only approached significance ($F=7.07$, $p=.076$) (Table 6).

Time to minimum T_r

The levels of CO_2 influenced the time to reach minimal T_r such that it reached statistical significance ($F=9.884$, $p=.013$), resulting in a linear trend towards decreased time to minimum temperature ($F=18.797$, $p=.023$) with increasing levels of CO_2.

Sleeping bag condition did not significantly affect the time to minimum T_r although there was a trend to minimal temperatures being reached slightly sooner in the single bag (Table 6).

Table 6. Mean values for T_r ($\pm$SD) for each grouping

	Single Bag n=12	Double Bag n=12	0% CO_2 n=8	2% CO_2 n=8	4% CO_2 n=8
T_r after 4 h (°C)	35.8 (0.1)	35.9 (0.1)*	35.8 (0.2)	35.8 (0.1)	35.8 (0.1)
Change in T_r after 4 h (°C)	-0.8 (0.3)	-0.6 (0.3)*	-0.7 (0.2)	-0.7 (0.3)	-0.7 (0.4)
Drop in T_r (°C)	0.9 (0.3)	0.8 (0.4)	0.9 (0.3)	0.8 (0.3)	0.8 (0.4)
minT_r (°C)	35.6 (0.1)	35.8 (0.2)	35.7 (0.2)	35.7 (0.1)	35.7 (0.2)
Time to minT_r (min)	267.5 (69.3)	284.2 (73.4)	309.9 (62.7)+	259.1 (76.0)+	258.5 (67.2)+

* - Significant from single bag ($p < .05$)
+ - Significant CO_2 main effect (linear trend, $p < .05$)

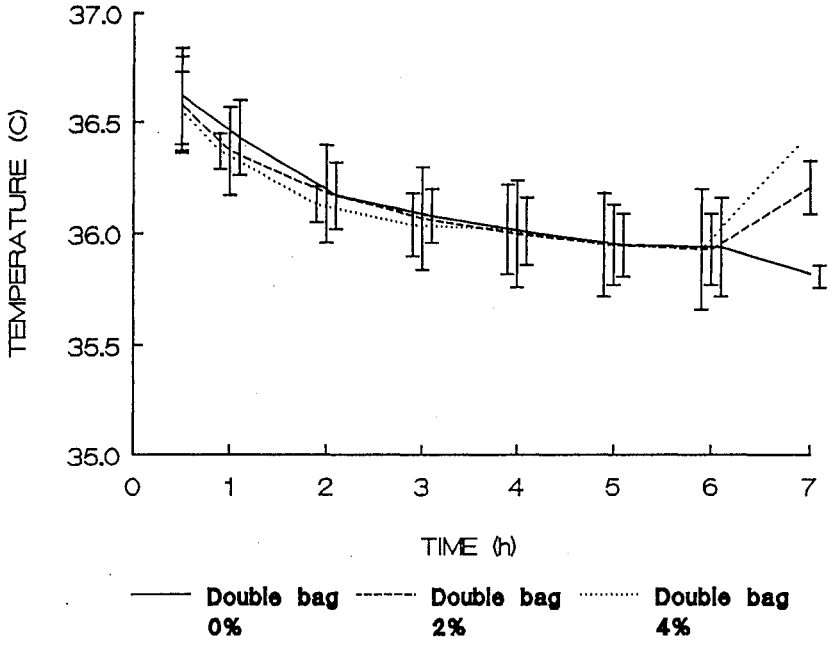

Figure 19. Hourly mean rectal temperature in each CO_2 condition (double bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

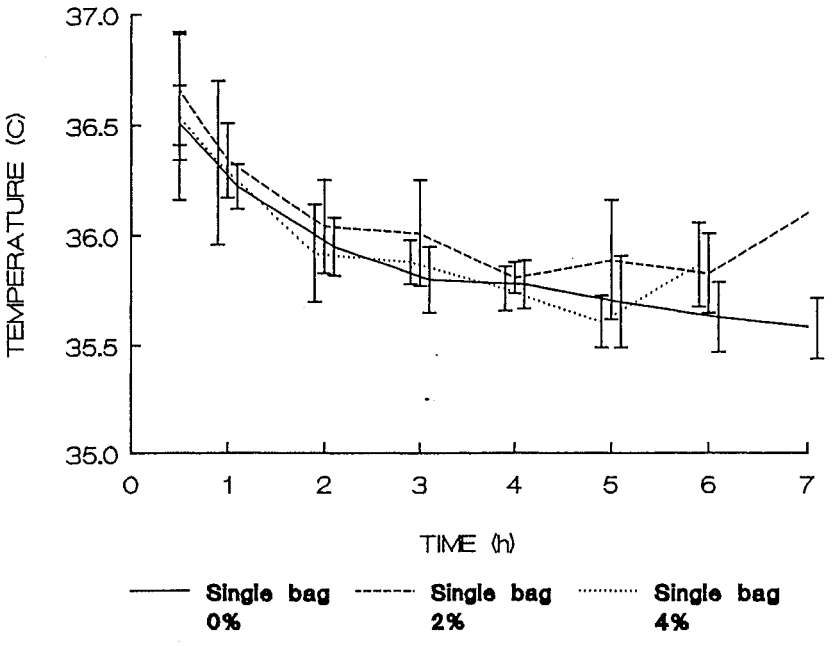

Figure 20. Hourly mean rectal temperature in each CO_2 condition (single bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

Mean skin temperatures ($\bar{T}_{sk}$)

Hourly readings

Mean skin temperatures in the double bag were higher throughout the night relative to the single bag. This difference was significant by the fourth hour ($F=19.051$, $p=.012$) (Table 7). In both sleeping bag conditions, $\bar{T}_{sk}$ was characterized by an slight initial rise followed by a steady decline (Figures 21 and 22). Like mean body temperature, mean skin temperature plateaued in the single bag after 4 hours while double bag temperatures continued to decline. Initial skin temperatures were approximately 0.7°C lower and initial rate of drop of skin temperatures appeared to be greater in the single bag as well. These factors combined to result in skin temperatures approximately 1.5°C lower in the single bag.

To examine the initial rise in skin temperature during the early hours of the trial, $\bar{T}_{sk}$'s were plotted as they changed from the initial temperature (Figures 23 and 24). Both the double and single bag groups show an initial rise of $\bar{T}_{sk}$ in all conditions with the exception of single bag, 2% CO_2. However, while the single bag temperatures decreased below the initial level after only 1 to 2 hours, double bag temperatures remained high for an additional hour and decreased at a lower rate and magnitude.

Plotting the relative change in $\bar{T}_{sk}$ across the night did not reveal any apparent effects of CO_2 in the double bag, but did suggest an enhanced drop in mean skin temperature with CO_2 in the single bag. This difference in temperature drop between the single and double bag was significant by the fourth hour ($F=16.274$, $p=.016$) (Table 7). At first glance, it appeared that increasing levels of CO_2 resulted in an inhibition of the initial rise in skin temperatures in the

single bag. However, Figures 21 and 22 reveal that skin temperatures were initially higher in the CO_2 conditions. Thus, the single bag 0% CO_2 condition did not appear to drop as much since the initial temperatures were lower.

Drop in $\bar{T}_{sk}$

As expected, decreased insulation was associated with a greater decrease in mean skin temperature (Table 7). The corresponding drop in mean skin temperature for single and double bag were -2.1°C and -1.1°C, respectively. Thus, mean skin temperature was maintained at a level 1.0°C higher in the double bag than in the single bag ($F=41.517$, $p=.003$)

The interaction between CO_2 and sleeping bag condition, while not statistically significant, suggests different trends towards a drop in skin temperatures with increasing CO_2 levels. In the double bag, increasing levels of CO_2 seem to be associated with a declining drop in $\bar{T}_{sk}$ while CO_2 appears to accentuate the drop in T_r in the single bag (Appendix E, Table 13).

Minimal $\bar{T}_{sk}$

Mean minimal $\bar{T}_{sk}$ differed greatly between the single and double bag conditions (Table 7). Across all single bag conditions, the average minimal skin temperature reached 30.7°C, whereas the double bag skin temperature was maintained approximately 1.5°C higher at 32.4°C. This difference was significant ($F=41.816$, $p=.003$).

CO_2, independent of sleeping bag insulation was associated with essentially no change in minimal $\bar{T}_{sk}$, and there was virtually no interactive effect between sleeping bag condition and CO_2

except for a very slight tendency for minimum double bag temperatures to be slightly higher in the 4% CO_2 condition (Appendix E, Table 14).

Time to minimum $\bar{T}_{sk}$

Sleeping bag condition resulted in a strong effect on the time to minimum skin temperatures such that skin temperatures reached a minimum almost 50 minutes sooner in the single as compared to the double bag (Table 7) ($F=13.999$, $p=.020$).

There was no significant main effect of CO_2 on the time to minimal $\bar{T}_{sk}$ except for a slight tendency for the time to be slightly diminished with increasing levels of CO_2.

Table 7. Mean values for $\bar{T}_{sk}$ ($\pm$SD) for each grouping

	Single Bag n=15	Double Bag n=15	0% CO_2 n=10	2% CO_2 n=10	4% CO_2 n=10
$\bar{T}_{sk}$ after 4 h (°C)	31.5 (1.1)	33.1 (0.9)*	32.4 (1.3)	32.2 (1.2)	32.2 (1.6)
Change in $\bar{T}_{sk}$ after 4 h (°C)	-1.3 (1.1)	-0.3 (0.8)*	-0.5 (1.0)	-0.9 (0.9)	-1.0 (1.4)
Drop in $\bar{T}_{sk}$ (°C)	2.1 (1.3)	1.1 (0.9)*	1.5 (1.2)	1.7 (1.1)	1.6 (1.4)
min$\bar{T}_{sk}$ (°C)	30.7 (1.2)	32.4 (1.1)*	31.5 (1.4)	31.4 (1.3)	31.7 (1.7)
Time to min$\bar{T}_{sk}$ (min)	282.3 (60.4)	330.5 (44.8)*	313.2 (51.9)	314.0 (68.5)	292.0 (54.8)

* - Significant from single bag ($p<.05$)

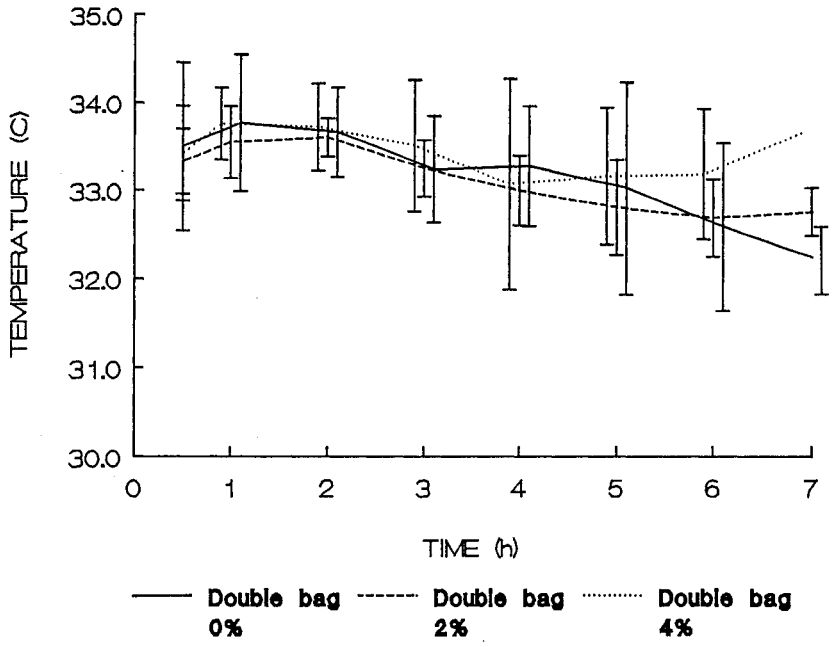

Figure 21. Hourly mean skin temperature in each CO_2 condition (double bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

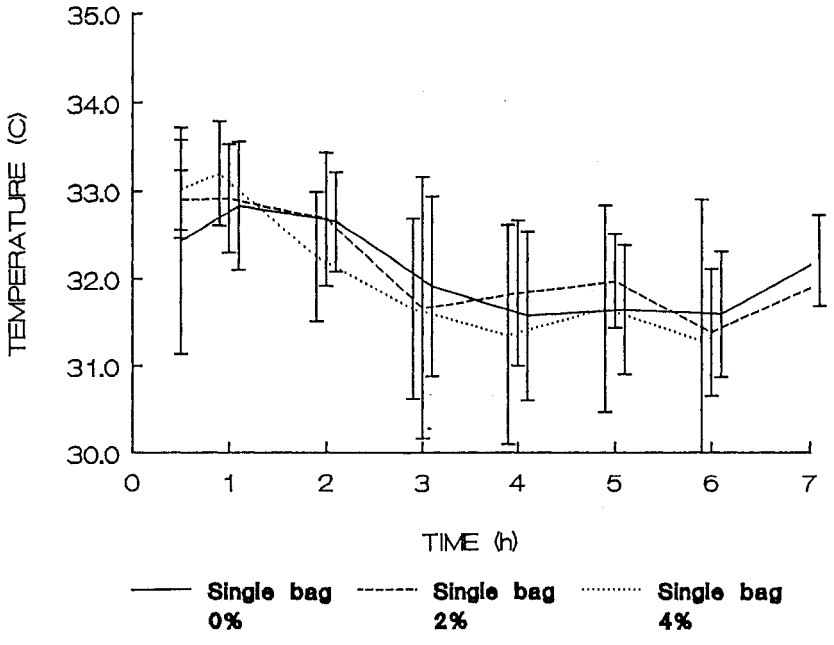

Figure 22. Hourly mean skin temperature in each CO_2 condition (single bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

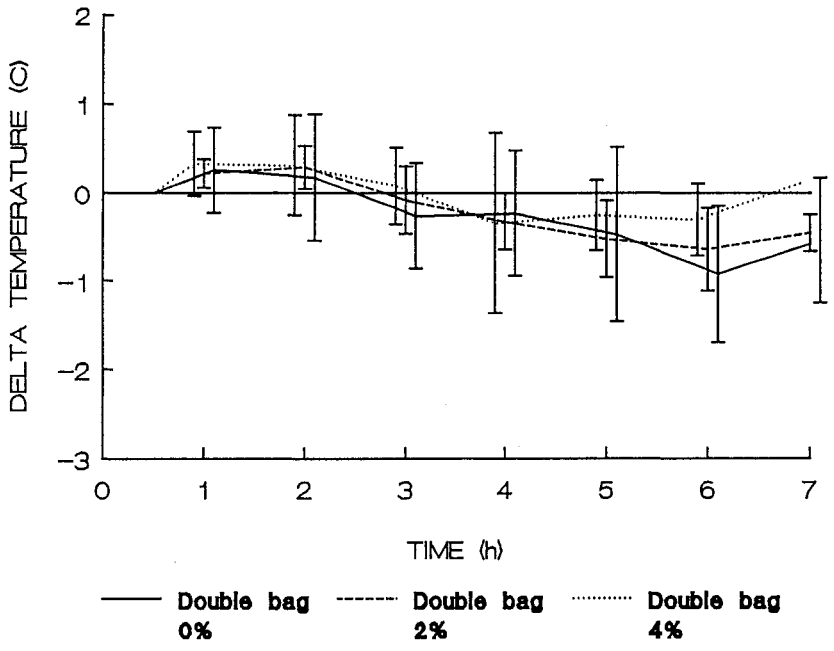

Figure 23. Hourly change from initial mean skin temperature in each CO_2 condition (double bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

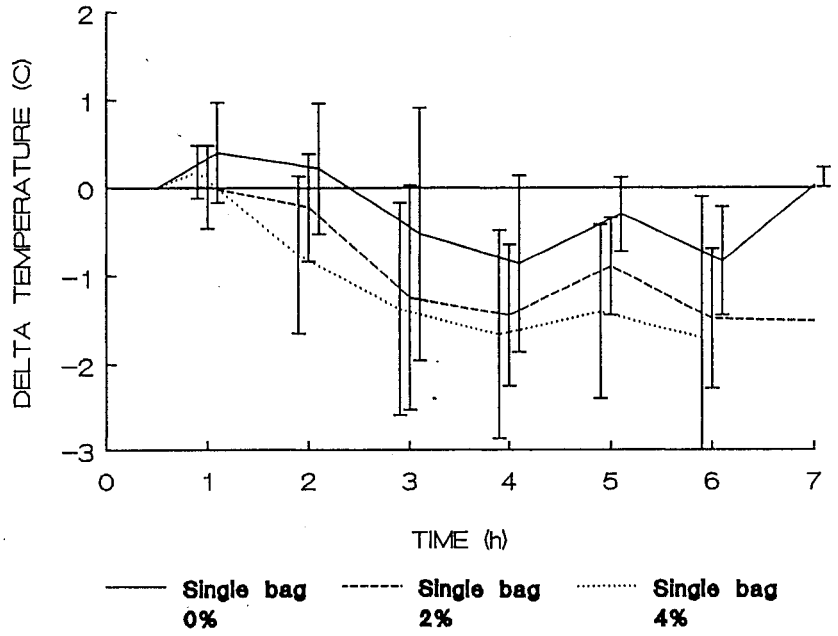

Figure 24. Hourly change from initial mean skin temperature in each CO_2 condition (single bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

Toe temperatures (T_{toe})

Hourly readings

It was anticipated that averaging toe temperatures across the night might be misleading due to the cold induced vasomotor 'hunting response' demonstrated in these anatomical regions. Since individual responses to cold stress makes interpretation of mean values difficult, it was decided to present these data as being representative of general temperature trends. Thus, for the purposes of this investigation the following analysis is based on the averaged data.

For the most part, T_{toe} in the double bag rose slightly during the first 3 to 4 hours and then dropped very slightly towards the end of the night (Figure 25). This temperature increment and decline was very small, averaging approximately 1.0 to 2.0°C. The net result was that the toe temperature was maintained at a comfortable level approximately between 31 and 33°C. There appears to be little influence of the level of CO_2 on this measure with the exception that T_{toe} appeared to be slightly warmer for most of the night in the 4% CO_2 condition.

With the exception of 0% CO_2 in the single bag, toe temperatures began to fall almost immediately (Figure 26). Toe temperatures in this condition rose and remained as high as those in the double bag condition until approximately the 3rd hour when an abrupt drop in temperature began. Within the CO_2 conditions, temperatures began dropping immediately with 4% CO_2 averaging slightly lower values than 2% CO_2. The lowest mean temperature reached was 19.5°C in the 4% condition after 6 hours in the sleeping bag.

Statistical analysis of the toe temperatures at the fourth hour of the night reveal a

significant effect of sleeping bag condition ($F=16.274$, $p=.016$) such that toe temperatures in the single bag were approximately 6.0°C lower than in the double bag (Table 8). There were no main CO_2 or interactive effects.

Drop in T_{toe}

As noted earlier, T_{toe} varied with the sleeping bag insulation such that in the single bag, temperatures dropped approximately 7.4°C lower as compared to the double bag (Table 8). This difference was statistically significant ($F=84.755$, $p=.003$).

In the single bag condition there was a trend towards less decrease in toe temperature with increasing levels of CO_2, while in the double bag, toe temperature fell slightly more in the presence of CO_2 (see Appendix E, Table 18). This interaction was not significant.

Minimum T_{toe}

Minimal toe temperatures over the night were sharply affected by the amount of insulation (Figures 25 and 26). Double bag toe temperatures remained relatively high, while single bag temperatures reached a rather uncomfortable low temperature of approximately 21°C (Table 8). This difference was statistically significant ($F=53.507$, $p=0.005$).

Simple main effects of CO_2 reveal that there was no strong trend towards differences in minimal T_{toe} when sleeping bag insulation was controlled for. Examining the interaction between CO_2 and sleeping bag conditions, one can see that with increasing CO_2 in the single bag, minimal toe temperatures tended to decreased slightly (Appendix E, Table 19). This trend was the

opposite of that present in the double bag condition and the difference between double and single bag conditions was approximately 4.7°C at 0% CO_2, increasing to almost 11.0% at 4% CO_2. However, this interaction was not significant (F=3.093, p=.119).

Time to minimum T_{toe}

CO_2 resulted in a significant main effect (F=16.151, p=.004) which proved to be linear (F=371.252, p=.000), such that increasing levels of CO_2 decreased the time to reach minimum toe temperatures (Table 8). No significant main effect of sleeping bag condition was noted.

Table 8. Mean values for T_{toe} (±SD) For each grouping

	Single Bag n=15	Double Bag n=15	0% CO_2 n=10	2% CO_2 n=10	4% CO_2 n=10
T_{toe} after 4 h (°C)	25.9 (5.8)	32.1 (3.8)*	30.4 (4.2)	28.7 (6.9)	28.0 (6.0)
Change in T_{toe} after 4 h (°C)	-4.5 (4.5)	0.4 (3.4)*	-.73 (2.5)	-2.4 (4.8)	-3.0 (6.1)
	n=12	n=12	n=8	n=8	n=8
Drop in T_{toe} (°C)	9.0 (3.6)	1.6 (2.8)*	5.5 (4.7)	5.9 (4.4)	4.4 (6.1)
minT_{toe} (°C)	21.5 (4.8)	29.6 (4.2)*	25.9 (6.4)	24.8 (6.0)	25.9 (6.4)
Time to minT_{toe} (min)	347.0 (65.5)	344.5 (31.1)	360.9 (43.8)+	346.3 (56.9)+	330.0 (50.6)+

* - Significant from single bag ($p<.05$)
+ - Significant CO_2 main effect (linear trend) ($p<.05$)

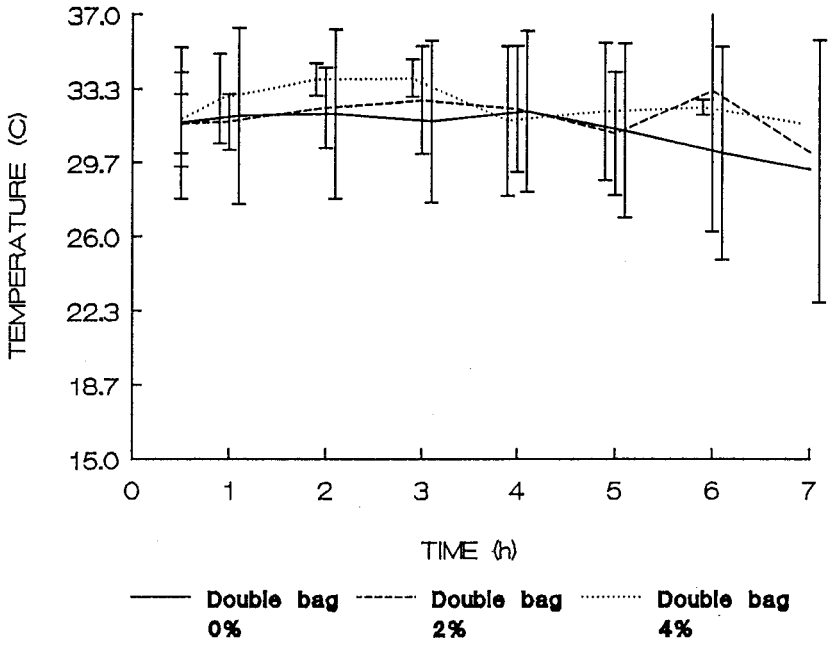

Figure 25. Hourly mean toe temperature in each CO_2 condition (double bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

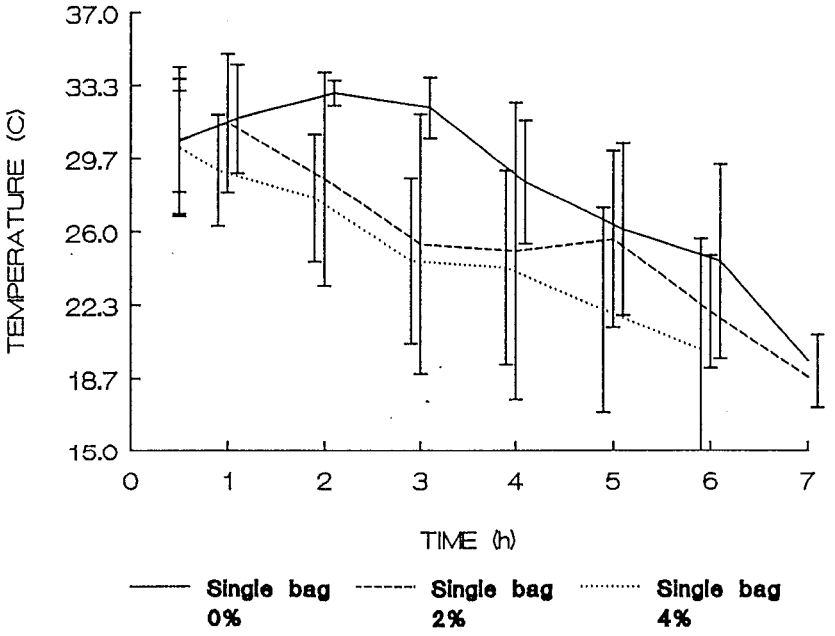

Figure 26. Hourly mean toe temperature in each CO_2 condition (single bag). (Note: each mean and SD represent hourly readings. They have been staggered for clarity of interpretation).

SLEEP AND BODY TEMPERATURES

Introduction

General observations

Averaging the data of 5 subjects affected the data such that the vascular responses which occurred in individual subjects were not evident. To obtain a better understanding of how nightly temperature and sleep profiles interrelated on an individual basis, Figures 27 and 28 detail the typical temperature patterns of one subject's exposure to both a single and double bag, as well as his accompanying sleep profile. From these two plots, it is evident that the temperature responses in the double bag and single bag were very dynamic.

Rectal temperatures demonstrated a steady decline which began prior to sleep onset, was most pronounced in the first 2 hours, and was then followed by a slow steady decrease or plateau. Prior to awakening, a morning temperature rise was noted, which was less pronounced in the single bag condition.

Skin temperatures were characterized by a great deal of vasomotion as evident by fluctuations in mean skin temperature. One prominent event noted was the sudden inflection of $\bar{T}_{sk}$ with sleep onset. Initially, a rapid rise in $\bar{T}_{sk}$ was present for the first 10 to 15 minutes due to the effect of rewarming after walking into the environmental chamber and climbing into a cold sleeping bag. Sleep onset often occurred during this period and was associated with a sudden attenuation of the rise in mean skin temperature.

The possibility of REM sleep being incompatible with normal thermoregulation has been indicated in the literature review. In this study, consistent vasomotion suggesting an impairment of normal thermoregulation was not observed during REM sleep. Accompanying Figures (27 and 28) demonstrate that there were some vascular events coinciding with REM in the single bag, but usually no change was noted.

SWS has also been associated with altered deep body temperature over the night, although, the literature reviewed for this study did not reveal significant vasomotor effects. In this study, individual temperature profiles suggest vascular events at the stage 2-SWS and SWS-stage 2 transitions. In the single bag example (Figure 28) there is a slight increase in $\bar{T}_{sk}$ and T_{toe}, at the first SWS-stage 2 transition, and a much more prominent increase after the second bout of SWS at approximately the 120 minute mark. Vasomotion is also noted in the double bag as well, being particulary dramatic in the toe.

Finally, occasional, fragmented awakenings appeared to be associated with small increments in T_{toe} and $\bar{T}_{sk}$. This phenomenon is particularly evident in the two examples presented in figures 27 and 28. Observing the profile of the single bag toe temperature may lead one to suspect that a rapidly dropping extremity temperature induced wakefulness, which was then followed by a warming or stabilizing of extremity temperatures. Since a common complaint of the subjects was cold feet keeping them awake, the question of the relationship between extremity temperature and termination of the experiment was developed. By examining several prematurely terminated nights in which rapid drop in T_{toe} was noted, it was suggested that low T_{toe} was not responsible for premature arousal, since wakefulness actually preceded this temperature drop (Figure 29).

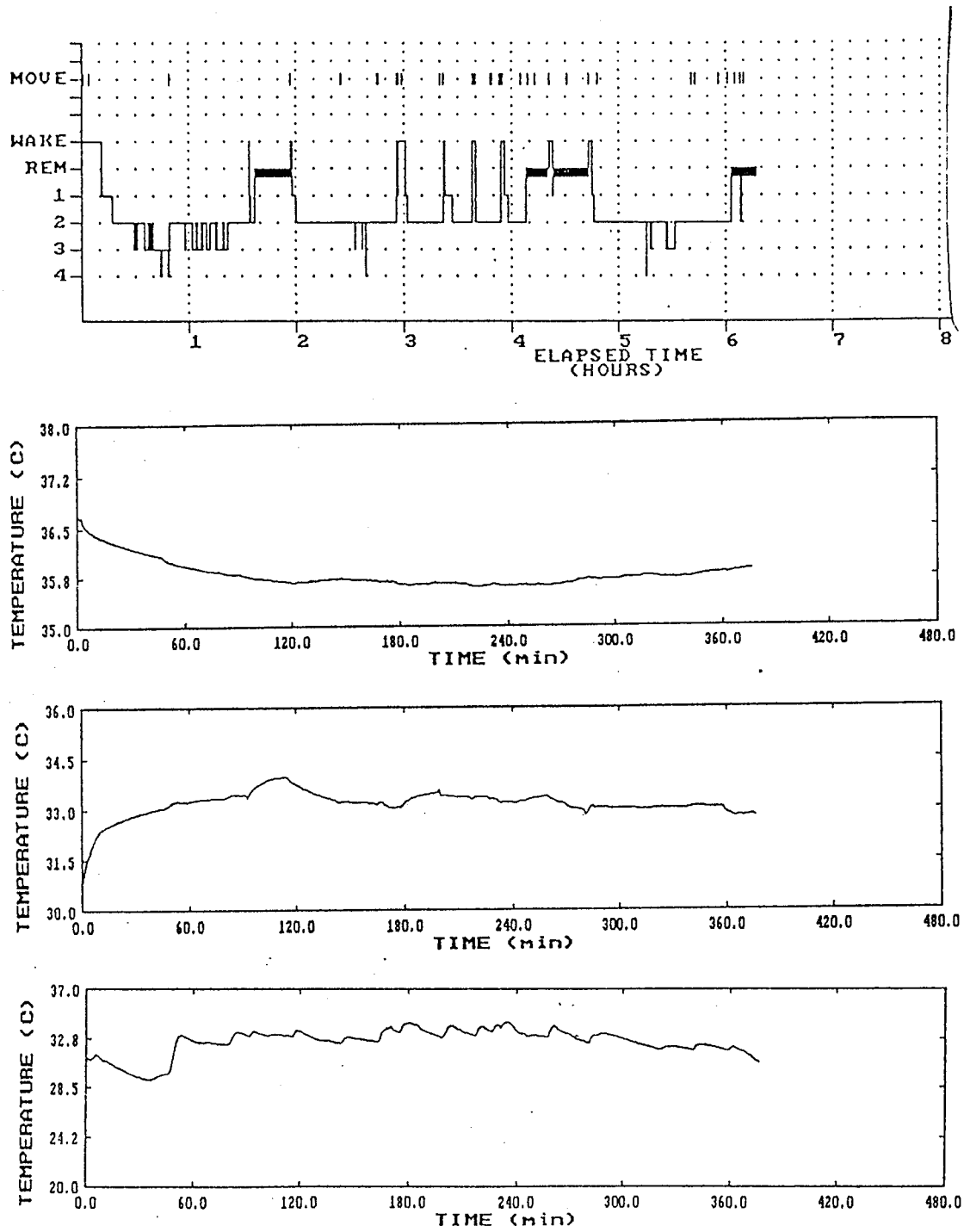

Figure 27. Single night sleep profile, T_r, T_{sk} and T_{toe} in subject 5 (Double Bag)

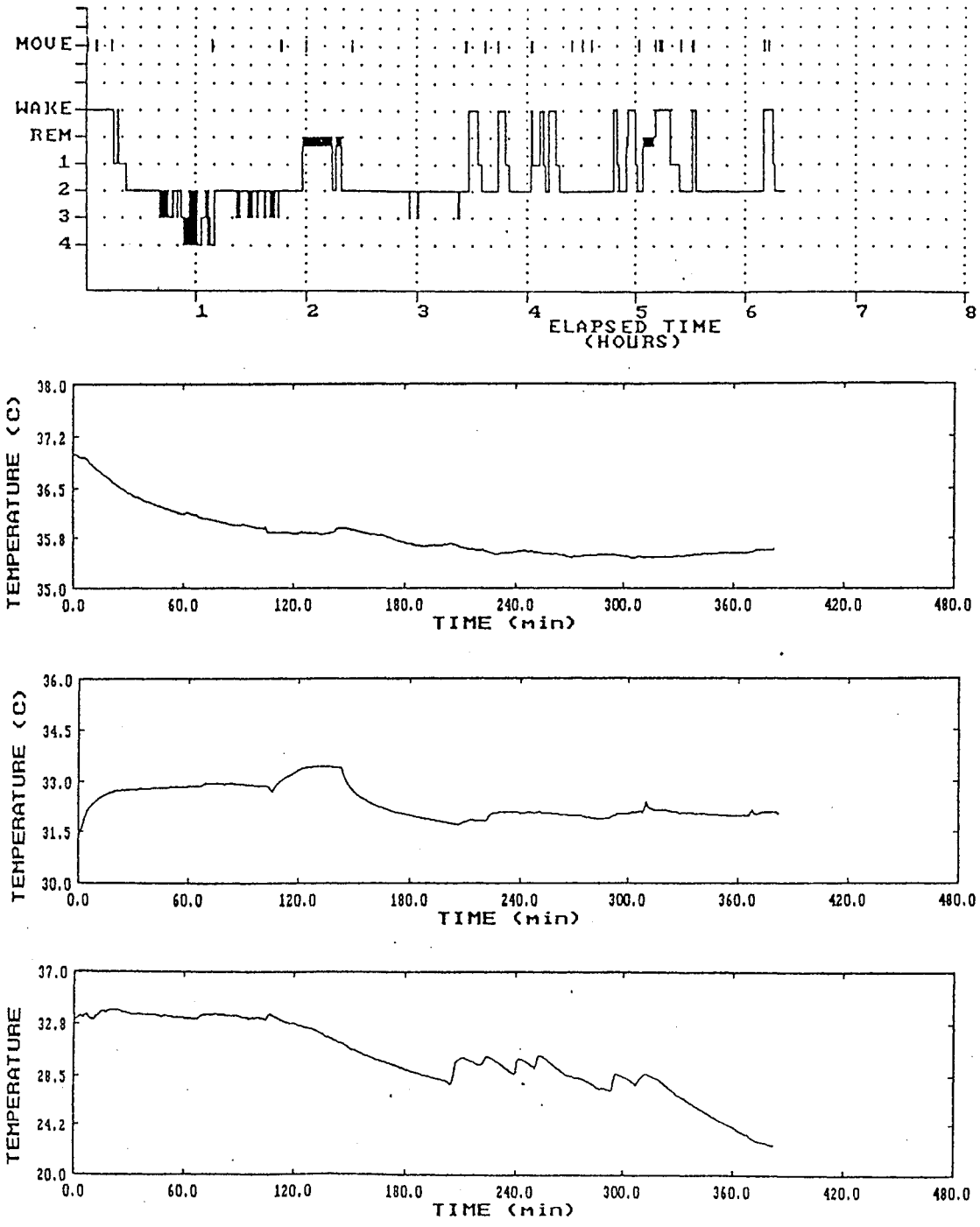

Figure 28. Single night sleep profile, T_r, T_{sk} and T_{toe} in subject 5 (Single Bag)

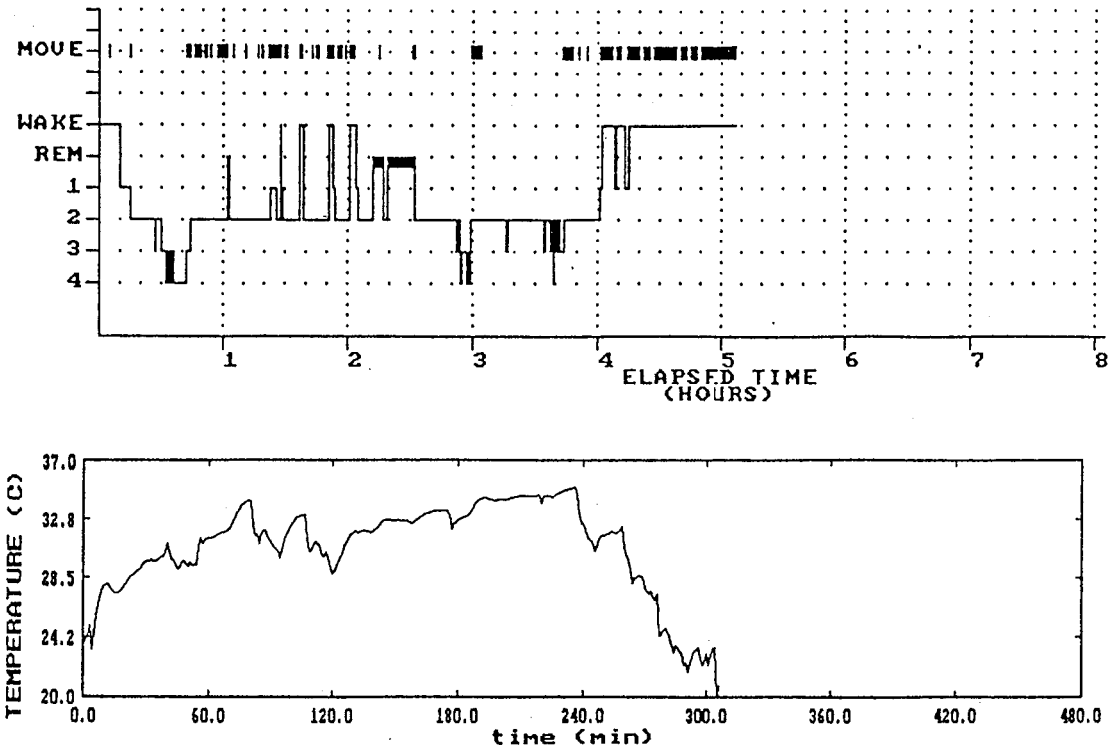

Figure 29. Sleep profile and toe temperature of subject 3 (Single Bag)

In response to these observations of an apparent relationship between sleep stages and vascular responses, it was decided that the previous decision to concentrate data analysis on the relationship between body temperatures and sleep efficiency, %stage 2, %REM and %SWS was valid. In addition, the number of awakenings per night was included in this analysis as it was felt that many fragmented awakenings may not be represented by sleep efficiency, as they are so short yet appear to influence skin and toe temperatures.

As mentioned in Chapter III, a general linear regression model was developed which examined the relationship between body temperatures and sleep parameters while controlling for CO_2 and sleeping bag conditions. The independent variables, or regressors selected are given in Table 9.

Table 9. Table of squared correlation coefficients, and partial correlation slope for each regressor.

	Regressor									
Dependent Variable	Sleep Efficiency		Number of Awakening		% Stage 2		% SWS		% REM	
	r^2	b_4	r^2	b_4	r^2	b_4	r^2	b_4	r^2	b_4
Minimum T_b	.537	+	.507	+	.474	+	.526	-	.403	+
Minimum T_r	.204	-	.243	+	.173	+	.217	-	.186	-
Minimum $\bar{T}_{sk}$	.624	+	.395	+	.444	+	.445	-	.420	+
Minimum T_{toe}	.555	-	.548	-	.483	-	.534	+	.534	+

As this experiment was a completely within repeated measures design, the significance of the model cannot be tested since the primary assumption of independence between observations was violated. Therefore, it is only possible to report the r^2 (explained variance) and *b* coefficient. In Table 9, only the sign of the *b* coefficient was given to demonstrate the direction of its association with the dependent variable.

Without the benefit of hypothesis testing, it is only possible to make inferences regarding the importance of the explained variance of these models. Since it was felt that any model which

could explain the majority of the variance of the dependent variable was of interest, it was arbitrarily decided that an r^2 greater than .50 would be the criterion to be used as a basis for the following discussion.

Mean body temperature

Minimum mean body temperatures appeared to be higher with increasing sleep efficiency as well as an increased frequency of awakenings per night. This suggests that frequent awakenings were not necessarily associated with decreased sleep efficiency. A decreased sleep efficiency was associated with fewer, but longer periods of wakefulness and lower minimal mean body temperature. Conversely, an increased percentage of SWS was associated with a lower minimal body temperature.

Mean skin temperature

When sleep efficiency was entered into the model, over 62% of the variance in minimal skin temperatures could be accounted for. The regressor coefficient for sleep efficiency was positive indicating greater sleep efficiency was associated with higher minimal $\bar{T}_{sk}$.

Toe temperature

Lower minimum T_{toe} was associated with decreased sleep efficiency and decreased number of nightly awakenings. Greater percentages of SWS and REM were associated with higher minimal toe temperatures.

VARIABILITY OF TEMPERATURE RESPONSE BETWEEN TWO SUBJECTS

After examining the data, it was striking to note the large variations between subjects in many of the sleep and temperatures variables. It is well known that there is a wide interindividual response to both thermal stress and hypercapnia. This was also evident in our subject pool, as some subjects demonstrated a much greater subjective and physiological tolerance to the experimental conditions. Pooled data provided mean values which may lie somewhere between these subjects' results, and may have failed to describe all subjects satisfactorily. Two subjects (3 and 5) were selected to illustrate the variability in sleep and temperature response across subjects. The two individuals demonstrated what was termed either a 'good' or a 'poor' subject response. These subjects were selected based on one scoring high, and the other low on the sleep satisfaction questionnaire and were of similar body mass index.

Sleep satisfaction averages for the single and double bags for each subject were as follows; subject 3, single = 5.8, double = 6.3; subject 5, single = 8.8, double = 8.0. Detailed data of each subject are given in Appendix D, Table 1. During the course of the experiment, subject 5 distinguished himself as a good sleeper, while subject 3 was less co-operative and complained frequently. In brief, subject 3 demonstrated less REM, TST, an increased SOL and reduced SE in both sleeping bag conditions.

Figures 30 and 31 demonstrate the difference in mean skin temperature profiles between subjects. It is evident that $\bar{T}_{sk}$ is initially the same for both subjects until approximately the second hour after which subject 3 drops and remains consistently cooler than subject 5. As a result, subject 3 experienced a lower minimum and greater drop of T_b, T_r, $\bar{T}_{sk}$, and T_{toe}. CO_2 effects on thermal results were not evident, and without ventilatory data it is difficult to determine

if CO_2 sensitivity was implicated in the differences. However, the large variance in thermal response between these two subjects is evident.

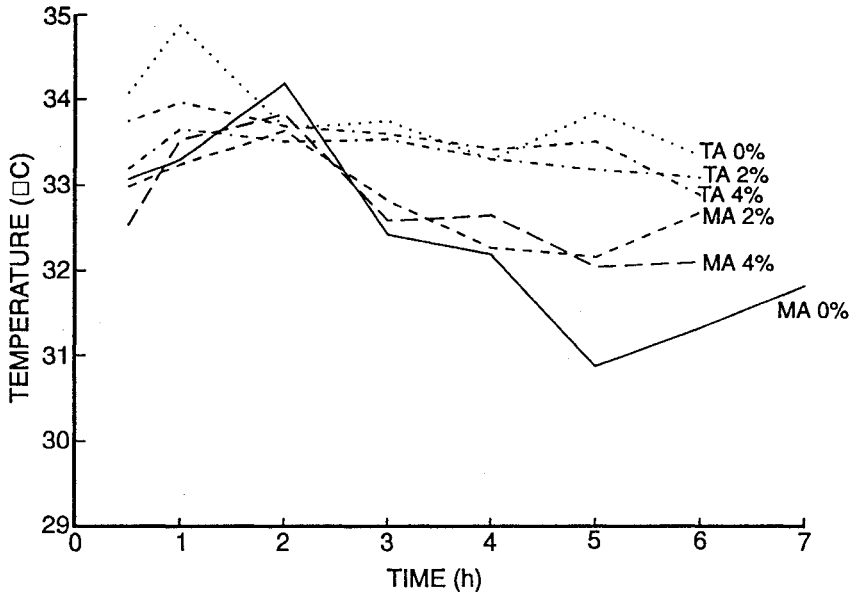

Figure 30. Subjects 3 (MA) and 5 (TA) skin temperatures (double bag).

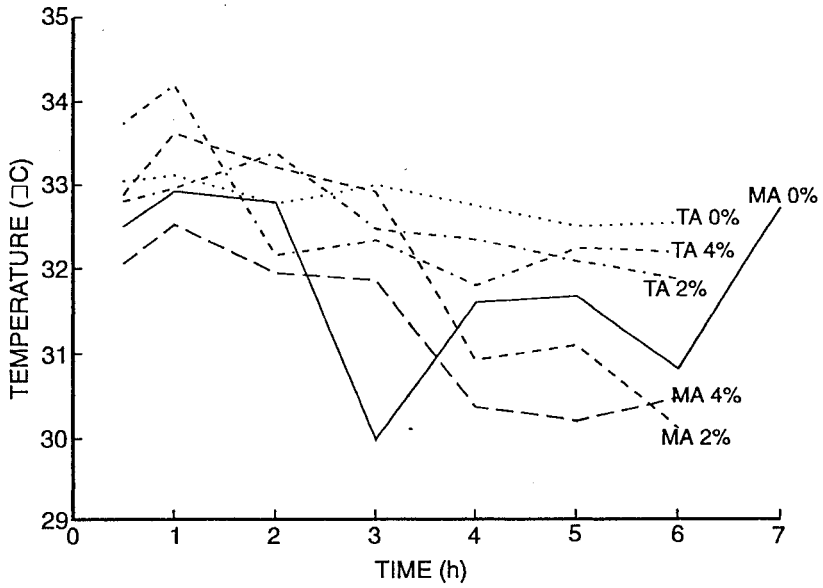

Figure 31. Subjects 3 (MA) and 5 (TA) skin temperatures (single bag).

CHAPTER V

DISCUSSION

Experimental design

A primary concern of this study was the effect of harsh environmental conditions and length of the experiment on both the subjects and experimental equipment. As mentioned earlier, 15 subjects presented themselves for initial briefing and pre-screening, but only 5 actually completed the experiment. The reasons for these subjects withdrawing were varied and are stated in the results. In addition it must be mentioned that these subjects had little prior experience with human physiological experimentation. Despite full subject education and orientation, it must be quite daunting for a lay person to be asked to be instrumented with skin and rectal thermistors, electrodes, don a facemask and breathe air containing up to 4% CO_2. If one is to expect a long term commitment and endurance of some discomfort from participants in a study, a certain amount of trust and rapport must be developed between the experimenter and the subject. It was perceived that good relationships were quickly developed with those subjects who were willing to devote themselves to the experiment. Many of those who dropped out prematurely seemed to be rather intimidated by the experimental equipment and procedures. It is felt that a better participation record could have been achieved by recruiting subjects who were more familiar with the rigours of physiological testing.

Although 5 subjects is a rather small subject pool, and more subjects would have been desirable, this number of subjects is typical of the majority of similar studies upon which many of the premises of this study were founded (e.g. Buguet et al., 1976b; Palca, et al., 1986). Large between and within subject variances, and missing cells decreased power further. Caution must be used in interpreting these results for it is possible that the subjects who remained in this study did so because of their ability to sleep under demanding conditions. These subjects may represent an exceptional population and thereby present a best case scenario.

A second difficulty encountered in conducting this study was the effect of extreme cold on the equipment. Mention has been made in the previous section of the problem of condensation and subsequent icing during collection of expired air for metabolic analysis. It is not known if this could have been remedied with the use of a plexiglass canopy such as those commonly used by others doing similar work (Haskell et al., 1981a; Shapiro, et al., 1984). A mask system was selected due to the necessity of controlling the inspired concentration of CO_2. Unfortunately, a mask has problems associated with it such as discomfort and potential for leakage. Alternately, a canopy system would have made it very difficult to control the composition of the inspired air, and requires a large amount of relatively expensive medical grade gas. It was anticipated that condensation and freezing of sampling lines would have been even greater with this configuration, as the humidified outlet air is usually sampled from the canopy with a small diameter tygon tubing.

Wire leads were also prone to damage in the cold, and breakage was not uncommon, resulting in the loss of data. This was particulary disconcerting when a rectal probe would fail or slip out during the course of the night. A decision was made not to awaken the subject as hypothermia was not determined to be a threat, and disturbing the subject from sleep would have

resulted in an unacceptable disruption to the other data collected. Collection of rectal temperature data was therefore sacrificed in order to maintain good sleep and skin temperature data. Loss of T_r was evident in the missing cells of subject 4 (Appendix E, Tables 6-10).

An alternative procedure might have been to perform the experiment at more hospitable temperatures, adjusting room temperature instead of insulation. This method is common in the literature, with nude subjects sleeping at thermoneutral 25°C (Anderson and Hellström, 1960) or cool 21 to 24°C (Palca et al., 1986; Haskell et al., 1981a) temperatures. However, the conditions of this experiment were explicitly selected to simulate real life conditions in order to address a specific applied research question. It was felt that a nude exposure would result in different temperature profiles than would be obtained in a sleeping bag. This would have limited the application of this study to a field situation.

Sleep stages

Although variations in sleep architecture were not a primary focus of this study, and are described in an ancillary report by DeKoninck (1991), such variations are inseparable from thermoregulatory data. Therefore these data are summarized and discussed in the following paragraphs.

DeKoninck reported that sleep was somewhat disrupted in the environmental chamber resulting in both a decreased quality and quantity. Baseline sleep profile for each subject (Table 10) demonstrates a decreased TST, SE, %REM, %SWS as well as an increased SOL in the chamber.

Varying the amount of insulation appeared to have the strongest effect on sleep with significant main effects or trends towards further impairment on almost all sleep indices measured. Curiously, SOL was slightly elevated in the double bag as compared to the single bag in almost every subject. This is contrary to what has been found in many cold stress studies (Haskell et al., 1981b; Palca et al., 1986).

Table 10. Comparison of subjects baseline/screening sleep patterns with the double bag 0% CO_2 condition of this experiment.

Sleep Variable	Average Sleep Content	Double Bag, 0% CO_2 ($\pm$SD)
Total Sleep Time (min)	399.8 (22.8)	327 (43)
Sleep Onset Latency (min)	9 (4.1)	19 (15)
REM (%)	19.3 (5.2)	11 (3)
Stage 2 (%)	62.5 (8.6)	72 (5)
Slow Wave Sleep (%)	12.3 (2.3)	9 (3)
Sleep Efficiency	.95 (1.5)	.83 (.1)

SWS also tended to increase in the colder condition supporting the findings of Buguet et al. (1979b). Although this effect was nonsignificant, 4 out of 5 subjects demonstrated a trend toward an increase. SWS is an important component of sleep associated with the release of growth hormone (Sassin et al.,1969) and chronic deprivation may result in lethargy, depression, introversion and complaints of muscular aches (Moorcroft, 1989). For these reasons, this inhibition of SWS with CO_2 warrants further attention.

It was perceived that this increase in SWS may be an artifact of a general decreased TST. Palca (1986), stated that a decrease in TST is associated with decreased stages 1, 2 and REM. Since SWS is concentrated in the first third of sleep, sleep must be drastically shortened before it is affected. However, the trend towards a decreased SWS with elevated CO_2 was accompanied by a trend towards a decreased TST. If absolute SWS did not change, a slight increase in SWS% should have occurred. It is probable that any true patterns are disguised in the large variation within and between subjects.

In response to this knowledge, the absolute amount of SWS in each experimental condition was analyzed. Surprisingly, this resulted in no change of SWS in response to either cold or CO_2. Elevated levels of CO_2 appeared to inhibit only the percentage of SWS in both sleeping bag conditions.

Another interesting observation was the fact that CO_2 had no apparent effect on REM sleep. Berthon-Jones and Sullivan (1984) have demonstrated that this stage of sleep was very unstable during hypercapnia and frequently shifted to wakefulness or stage 2 at the onset of rebreathing a bag containing 7% CO_2 in 40% O_2. It is possible that the inspired CO_2 employed in this study was too low to exert any effect. Furthermore, as REM was somewhat depressed or nonexistent in many trials, any CO_2 effects may have already been masked.

It must be acknowledged that some of these sleep impairments may be a function of the experimental design. Subjects were awakened at 0600h if they were not already awake at this time. Since lights out occurred at approximately 2230h to 2300h, the maximum allowed sleep time would be 7h 30min. Second, as mentioned in the methodology section, the cold environment and measuring devices did not constitute as quiet and comfortable environment as

one's own bedroom, adding further to potential stress and sleep disruption. However, the very fact that Stage 4 was reached is supportive of the fact that the environment was not too harsh, as this stage is reputed to be very sensitive to stress (Henane et al., 1977).

Despite this interruption of sleep architecture, the subjective quality of sleep satisfaction was impaired in the single bag, but not to the extent that might have been expected. It must be acknowledged that any subjective psychometric rating system has its inherent shortcomings, and the validity and reliability of this sleep satisfaction score is not known. However, for subjects to rate their sleep quality similarly despite such large differences in insulation and sleep quality is intriguing.

It is possible that these scores were similar purely as a function of using a linear, arbitrary scale, with limited range of numbers. It is well known that instead of a linear relationship, many psychophysical impressions follow a power function

$$J = kI^p$$

Where J = judgement
k = constant
I = intensity
p = power

This may be interpreted as meaning differences between single bag and double bag satisfaction scores may represent a large psychological intensity,but treating the scale as a linear function does not reveal any statistical differences. Proper interpretation of this rating system requires further research to determine the magnitude of the power constant.

Conversely, if these results are valid, then it is possible that mild short term insomnia is

not as disturbing as might have been thought. Wilkinson et al. (1966) conducted a literature review which reported vigilance performance being unaffected by a single night of sleep reduction until total sleep was reduced to 3.5 hours. Moderate performance decrements occurred after 2 days of 5 hour sleep. Thus this experiment, with its single exposures spaced out over several days, may have not greatly altered sleep satisfaction. Consecutive days of sleep reduction may be reflected by lower satisfaction scores.

Body temperatures

Anticipating the effects of breathing CO_2 on thermoregulation during sleep in the cold was very difficult. First, there was a paucity of literature directly dealing with this subject. As a result, it was necessary to extrapolate from literature employing awake subjects exposed to short term CO_2 (Bullard and Crise, 1961; Wagner et al., 1983), as well as other literature examining the effects of chronic exposure to CO_2 (Schaefer and Wünnenberg, 1976), the latter seldom being performed on humans. Complicating this issue was the fact that this literature was not unanimous in its findings. Secondly, it was perceived that CO_2 effects might be mediated through the interruption of normal sleep processes. As mentioned in the previous section, certain aspects of sleep tended to be disrupted with increasing CO_2. This, in turn, may have affected body temperatures. However, it is possible that a disruption of sleep may have been a result of the direct effect of CO_2 on body temperature. At this point in time, it is impossible to identify the exact mechanism whereby CO_2 exerts its influence.

It was apparent that the most significant factor affecting body temperatures was decreasing the insulation of the sleeping bag. This resulted in a lower minimum temperatures, and greater drop of temperature in the single bag as compared to the double bag, agreeing with

the majority of literature (Kreider and Iampietro, 1960; Buguet et al., 1976b). Curiously, there was no statistically significant difference in minimal T_r between the two conditions, nor was there a difference at the time at which this minimum was reached. This was very surprising, as one would expect that single bag T_r would cool faster and by a greater amount. A similar nonsignificant finding has been noticed in one other study involving sleeping bags (Goldman et al., 1960). It must be noted, that the values in this study approached significance and may have become significant with added subjects. However, the absolute difference in T_r between the single and double sleeping bag was still only .1 to .2°C. Considering the normal circadian rhythm of 0.7 to 1.5°C (Mellette et al., 1951), this difference becomes even less remarkable.

T_r would seem to be an inappropriate site to monitor cold stress during sleep, as it is regulated downward over the night due to circadian and sleep effects (Day, 1941; Fraser et al., 1989a), which combined may account for a drop of T_r of 1.5 to 2°C (Haskell et al., 1981) in comfortable temperatures, thus masking any true cold effects. In the present study, the typical drop in T_r was approximately 1.0°C in both sleeping bag conditions. However, this drop may be underestimated as the initial T_r was taken at t = 30 min and the greatest drop typically occurred during the initial 2 h of sleep (Haskell et al., 1981a; Fraser et al., 1989). Furthermore, it was possible that greater wakefulness in the single bag may have prevented a further drop of T_r by increasing shivering and metabolic rate and inducing heat conserving postural effects. Thus, comparisons between the double and single bag conditions may have been complicated by these factors.

CO_2 effects on thermoregulation

In normal waking individuals, CO_2 has been demonstrated to impair normal

thermoregulation by inhibiting shivering, decreasing metabolic rate, increasing skin blood flow and increasing respiratory heat loss. Despite these many potential avenues of heat loss, CO_2 did not exhibit any great effect on the absolute temperatures of the body. One noticeable CO_2 effect, however, was that of decreasing the time to minimum T_r and T_{toe} with increasing hypercapnia. A decreased time to minimum T_r is in agreement with Wagner et al. (1983), and may be due to several factors. CO_2 has been shown to result in decreased vascular resistance leading to a shift in blood volume to the peripheral vasculature and enhanced heat loss (Price, 1960). However, elevated skin temperatures were not evident in our subjects. A more reasonable explanation may lie in the increased respiratory rate and impaired metabolism associated with CO_2 inhalation (Schaefer et al., 1975). Unfortunately, ventilatory and metabolic data were lost in this study, thus preventing the calculation of metabolic heat production and respiratory heat loss. Inhibition or an attenuation of a shivering response may have also been a factor as it has been noted with CO_2 breathing (Bullard and Crise, 1961; Schaefer and Wünnenberg, 1976).

The literature suggests that for the inhibition of shivering, the effect of CO_2 on thermoregulation may have a transient component which is overcome by excessive heat loss. This effect may last as little as 30 min in humans (Bullard and Crise, 1961). Closer examination of the hourly $\bar{T}_{sk}$ data reveals that initial skin temperatures may have been slightly elevated in the single bag, CO_2 conditions. Elevated heat loss in the initial hour of the experiment may have depleted body heat stores resulting in a failure to maintain rectal temperatures. This loss of body heat may be reflected in the single bag with CO_2 conditions where toe temperatures declined rapidly throughout the experiment, as peripheral vasculature has been shown to display a pronounced vasoconstriction with body cooling (Toner and McArdle, 1988).

Relationship between sleep and body temperature

It has been determined that breathing elevated concentrations of CO_2 exert adverse effects on sleep patterns as well as thermoregulation. However, these cannot be discussed without investigating how they are interrelated. Decreasing the insulation of the sleeping bag was associated with decreased sleep efficiency and lower minimum T_b and $\bar{T}_{sk}$. When both sleeping bag condition and CO_2 were controlled for by the linear regression model, both of these temperatures demonstrated an association with decreased sleep efficiency such that between 54 and 62% of the variance in minimal body temperatures could be explained by sleep efficiency. This suggests a close relationship between sleep and skin temperatures.

Both increased sleep efficiency and a greater number of awakenings appeared to be associated with lower minimal toe temperatures. This agrees with the study of Scholander et al. (1958) who noted that Australian Aborigines slept longer and tolerated lower extremity temperatures than Europeans while camping in the outback. The Aborigines awoke only briefly, when their skin temperatures dropped very low, to stoke the camp fire. Europeans maintained higher body temperatures, but were unable to sleep, exhibiting long periods of wakefulness. It appears that during sleep, the sympathetic vasoconstrictor drive to the extremities is reduced allowing temperatures to drop. As an increased number of awakenings is also associated with lower minimal T_{toe}, periodic brief arousals may ensue to increase the toe temperature, without seriously sacrificing sleep. This is supported, in part, by the findings of Scholander and subject 5 in this present investigation (Figure 28).

In this study, increased percentages of SWS appear to be associated with lower minimal mean body temperatures and higher toe temperatures. Thus, it is possible that SWS may play

an more important role in thermoregulation than previously thought. Further evidence for this may be found in the vasomotion which appeared to be quite strong in many subjects during the SWS-2 and stage 2-SWS transitions. Most human experimentation has focused on stage REM as being worthy of special consideration since animal models show dramatic physiological events in this stage. Detailed investigation of various NREM stages is not common but the results of this study suggest that, at least, the deeper stages of sleep may warrant further investigation. Caution must be exercised before discounting the effect of REM on thermoregulation based on the results of this study, as this stage was so greatly diminished that any effects would have been negated over the night.

In summary, CO_2 impaired maintenance of body heat during sleep with trends towards decreased time to minimal $\bar{T}_{sk}$ and significant decreases in time to minimum T_r and T_{toe} with increasing CO_2. Sleep in the cold reduced both the quality and quantity of sleep. CO_2 further reduced the quality of sleep by tending to decrease total sleep time and the percentage of SWS with increasing inspired concentrations. As a link between body temperatures and sleep quality has been suggested, it is still possible that elevated levels of CO_2 may be contraindicated in some persons in situations where sleep and the maintenance of body heat stores is imperative.

CONCLUSIONS

Elevated levels of inspired CO_2, cold temperatures, human thermoregulation and sleep interact in a complex manner which may be complimentary, reciprocal or antagonistic. For these reasons, it is difficult to formulate a simple, concise interpretation of their resultant effects. It was the intention of this study to gain a understanding and appreciation of these complex interactions and to evaluate them in within a pragmatic domain which simulated field conditions as closely as possible while permitting control of environmental variables. It is believed that this was accomplished successfully.

Within the constraints of this study, it was apparent that CO_2 challenged thermoregulation in both comfortable and cold environments by increasing the rate of rectal and extremity (toe) cooling. No strong evidence of an interaction between the cold stimulus and hypercapnia was revealed. However, it was possible that the cold stress was severe enough that any combined effects of CO_2 and cold may have been suppressed.

The quality and quantity of sleep were compromised by both cold temperatures and CO_2. This resulted in a trend towards decreased total sleep time and a CO_2 induced suppression of SWS. Cold temperatures also tended towards the production of reduced REM sleep. Surprisingly, this disruption of sleep was not reflected in subjective ratings. As the physical and cognitive impairments of sleep reduction are well known, it is possible that prolonged exposure to the experimental conditions may result in greater impairment than was evident in this study.

Although, it was not perceived that the levels of CO_2 employed imposed a significant

environmental stress, those who are not adequately protected from the cold may be disposed to hypothermia. Particularly, complications may arise in the person who is exhausted or approaching the onset of hypothermia. In these people, any impairments in sleep or thermoregulation may eventuate in serious consequences. Unfortunately, adequate ventilation may be incompatible with the insulation of an enclosed shelter. In this case, some effort should be made to gain access to fresh air.

The subjects who participated in this study were young, healthy males who demonstrated a tolerance towards sleep in harsh conditions. Extrapolation of these results must be made with caution, with the understanding that the conclusions may err on the side of conservatism.

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4) Ventilation responses to CO_2 should be determined in each subject as there is known to be a wide variation in individual responses. This would provide much needed information as to whether there is a relationship between sensitivity to CO_2 and sleep and thermoregulatory responses.

5) Research is required to elucidate the effects of chronic nightly exposure to CO_2 in order to determine if there is any adaptation or cumulative impairment with time. This is especially important with respect to the trends towards impaired SWS and TST with CO_2.

6) Improvements in sleeping conditions such as an alternative to the face mask, whole body calorimetry and less harsh cold conditions may be necessary to ensure a greater sample size, less extraneous interruption of sleep and allow greater ease with which to collect important metabolic and ventilatory data.

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APPENDIX A

CONSENT FORMS

Consent Form for the Sleep and Thermoregulation Study

Allan Keefe, Graduate Student in Kinanthropology, in conjunction with Dr. S. L. Livingstone of DREO, Dr. J. De Koninck, Professor of Psychology at the University of Ottawa, and Dr. J. Thoden, Professor of Kinanthropology at the University of Ottawa are conducting a study for the Defence Research Establishment of Ottawa in order to measure the effects of CO_2 on thermoregulation during sleep in young male adults. More specifically, the effects will be evaluated when subjects sleep in a sleeping bag in a cold environment. The ethics policy of the University of Ottawa requires that each subject sign an informed consent form.

Outline of the study

- DREO (Defence Research Establishment Ottawa) is located in building 29, Shirley Bay, Ottawa.
- The Sleep Research Laboratory is located in room 424 of Montpetit Hall at the University of Ottawa.
- The study consists of having each participant sleep for 15 nights. One screening night and one baseline night at the University of Ottawa, one adaptation night and six non-consecutive series of two night at DREO in a cold room.
- During the nights in the laboratory at the University of Ottawa, standard polysomnography will be applied in order to detect any sleep abnormalities.
- For the third night, subjects will sleep in a cold room at DREO under standard polysomnography for adaptation purposes. Subjects will breathe 100% air through a face mask applied by DREO.
- Subjects will then sleep again at DREO for six non-consecutive series of two nights with the following six conditions:
  1-Minus 20°C, 100% air, double sleeping bag
  2-Minus 20°C, 100% air, single sleeping bag
  3-Minus 20°C, 2% CO_2 in air, double sleeping bag
  3-Minus 20°C, 2% CO_2 in air, single sleeping bag
  3-Minus 20°C, 4% CO_2 in air, double sleeping bag
  3-Minus 20°C, 4% CO_2 in air, single sleeping bag
  Subjects will not be made aware of the CO_2 content of the air on each night of experimentation even though they are aware of this overall manipulation.
- Electrodes will be installed before each night of recording. These electrodes are applied externally to the skin and may, for some individuals, cause minor irritation to the skin.
- Skin and rectal temperature will be measured following the procedure explained by the experimenter before this form is signed.
- A demonstration of the procedures for electrode installation (including body temperature) will be offered to prospective participants before they accept to participate.
- At all times during the night, a research assistant will be in the laboratory. If for any reason you wish to communicate with the assistant on call, you may do so via an intercom system installed in the bedroom.
- The participants will be required to fill questionnaires concerning their psychological state for every night spent in the laboratories. They will also be required to fill daily sleep questionnaires.

Additional information

- Each subject will undergo a full medical examination prior to the beginning of the experiment. At that time, they will meet with Dr. D. Kilby of the University of Ottawa Health Services (or an associate) who will brief the subject on the effects of breathing minimal levels of CO_2 on the human. Any medical concern during the experiment will be responded to and visits with Dr. Kilby will be scheduled on request. At any rate, a full medical examination will be carried out

on all subjects after the experiment.

Consent Form for the Sleep and Thermoregulation Study (continued)

- Breathing CO_2 concentrations as specified above may cause temporary headaches. However, it is out opinion, based on prior sleep research, that there is no health danger whatsoever for the participant. Nevertheless, if you post health history suggests that any type of complication could occur because of your participation in the study, the experimenter or the physician should be notified prior to the beginning of the research.
- If requested by the participant, a research assistant of the same sex will be provided.
- Each participant will be renumerated $50 per night spent in the laboratory, for a total of %750.
- The participant may withdraw from the present study at any time, even after having signed the consent form. If you are a student, this will in no way affect your academic marks. If you are in the army, this will in no way affect your military career. In case of withdrawal, you will receive $50 for each night spent in the laboratory.
- Individuals participating in the research are assured complete confidentiality regarding the physiological and psychological measures obtained during the study.
- All collected data will be kept confidential and any published data will respect anonymity.
- The participant will need to devote a certain amount of time every morning to his personal hygiene. More specifically, the removal of collodium form the scalp may require a few minutes.
- Subjects will be asked not to sleep (take naps) during the day for research purposes.
- A feedback session will be scheduled after the completion of the study (if desired by the participant), in order to allow the experimenter to answer any questions, as well as offer a more detailed rationale of the study.
- I understand that my participation is part of my militia/military activities and is in not way related to my status as a student (if applicable) of the University of Ottawa.

I understand the nature of this research and accept to participate. A copy of this form has been given to me.

I have explained the nature of the research to the participant, and believe that he has understood it.

Participant's name	Participant's signature	Name of investigator

Witness

Consent Form for the Sleep and Thermoregulation Study (the screening night)

Allan Keefe, Graduate Student in Kinanthropology, in conjunction with Dr. S. L. Livingstone of DREO, Dr. J. De Koninck, Professor of Psychology at the University of Ottawa, and Dr. J. Thoden, Professor of Kinanthropology at the University of Ottawa are conducting a study for the Defence Research Establishment of Ottawa in order to measure the effects of CO_2 on thermoregulation during sleep in young male adults. More specifically, the effects will be evaluated when subjects sleep in a sleeping bag in a cold environment. The ethics policy of the University of Ottawa requires that each subject sign an informed consent form.

Outline of the study

- DREO (Defence Research Establishment Ottawa) is located in building 29, Shirley Bay, Ottawa.
- The Sleep Research Laboratory is located in room 424 of Montpetit Hall at the University of Ottawa.
- The study consists of having each participant sleep for 15 nights. One screening night and one baseline night at the University of Ottawa, one adaptation night and six non-consecutive series of two night at DREO in a cold room.
- This consent form is for the screening night only.
- During the nights in the laboratory at the University of Ottawa, standard polysomnography will be applied in order to detect any sleep abnormalities.
- Electrodes will be installed before each night of recording. These electrodes are applied externally to the skin and may, for some individuals, cause minor irritation to the skin.
- Skin and rectal temperature will be measured following the procedure explained by the experimenter before this form is signed.
- A demonstration of the procedures for electrode installation (including body temperature) will be offered to prospective participants before they accept to participate.
- At all times during the night, a research assistant will be in the laboratory. If for any reason you wish to communicate with the assistant on call, you may do so via an intercom system installed in the bedroom.
- The participants will be required to fill questionnaires concerning their psychological state for every night spent in the laboratories. They will also be required to fill daily sleep questionnaires.
- If requested by the participant, a research assistant of the same sex will be provided.
- Each participant will be renumerated $50 per night spent in the laboratory, for a total of %750.
- The participant may withdraw from the present study at any time, even after having signed the consent form. If you are a student, this will in no way affect your academic marks. If you are in the army, this will in no way affect your military career. In case of withdrawal, you will receive $50 for each night spent in the laboratory.
- Individuals participating in the research are assured complete confidentiality regarding the physiological and psychological measures obtained during the study.
- All collected data will be kept confidential and any published data will respect anonymity.

Additional information

- The participant will need to devote a certain amount of time every morning to his personal hygiene. More specifically, the removal of collodium form the scalp may

require a few minutes.

- Subjects will be asked not to sleep (take naps) during the day for research purposes.
- A feedback session will be scheduled after the completion of the study (if desired by the participant), in order to allow the experimenter to answer any questions, as well as offer a more detailed rationale of the study.
- I understand that my participation is part of my militia/military activities and is in not way related to my status as a student (if applicable) of the University of Ottawa.

Consent Form for the Sleep and Thermoregulation Study (The screening night) continued.

I understand the nature of this research and accept to participate. A copy of this form has been given to me.

I have explained the nature of the research to the participant, and believe that he has understood it.

Participant's name	Participant's signature	Name of investigator

Witness

APPENDIX B

SLEEP SATISFACTION QUESTIONNAIRE

SLEEP SATISFACTION LOG

NAME: _____

DATE

SLEEP SATISFACTION

1 _____	1	2	3	4	5	6	7	8	9	10
2 _____	1	2	3	4	5	6	7	8	9	10
3 _____	1	2	3	4	5	6	7	8	9	10
4 _____	1	2	3	4	5	6	7	8	9	10
5 _____	1	2	3	4	5	6	7	8	9	10
6 _____	1	2	3	4	5	6	7	8	9	10
7 _____	1	2	3	4	5	6	7	8	9	10
8 _____	1	2	3	4	5	6	7	8	9	10
9 _____	1	2	3	4	5	6	7	8	9	10
10_____	1	2	3	4	5	6	7	8	9	10
11_____	1	2	3	4	5	6	7	8	9	10
12_____	1	2	3	4	5	6	7	8	9	10
13_____	1	2	3	4	5	6	7	8	9	10
14_____	1	2	3	4	5	6	7	8	9	10
15_____	1	2	3	4	5	6	7	8	9	10
16_____	1	2	3	4	5	6	7	8	9	10
17_____	1	2	3	4	5	6	7	8	9	10
18_____	1	2	3	4	5	6	7	8	9	10
19_____	1	2	3	4	5	6	7	8	9	10
20_____	1	2	3	4	5	6	7	8	9	10
21_____	1	2	3	4	5	6	7	8	9	10
22_____	1	2	3	4	5	6	7	8	9	10
23_____	1	2	3	4	5	6	7	8	9	10
24_____	1	2	3	4	5	6	7	8	9	10
25_____	1	2	3	4	5	6	7	8	9	10
26_____	1	2	3	4	5	6	7	8	9	10
27_____	1	2	3	4	5	6	7	8	9	10

APPENDIX C

SEQUENCE OF EXPERIMENTAL TRIALS

Table 1. Sequences of Experimental Conditions for Each of the Five Subjects

Subject	1	2	3	4	5
Night					
1	Diagnostic	Diagnostic	Diagnostic	Diagnostic	Diagnostic
2	Normal	Normal	Adaptation	Normal	Normal
3	Adaptation	Adaptation	S, 0%	Adaptation	Adaptation
4	D, 0%	S, 0%*	D, 0%	S, 0%	D, 0%
5	S, 4%	D, 0%	D, 2%	S, 2%	S, 4%
6	S, 0%	D, 2%	S, 2%	S, 4%	D, 0%
7	S, 2%	S, 2%	D, 4%	D, 0%	D, 4%
8	D, 2%	D, 2%	S, 4%	S, 4%*	S, 2%
9	S, 2%	D, 4%	D, 0%	D, 2%	S, 0%
10	D, 0%	S, 4%*	S, 0%	S, 0%	D, 2%
11	D, 4%	D, 4%	D, 2%	D, 4%	S, 2%
12	D, 2%	S, 4%*	S, 2%	S, 2%*	D, 2%
13	N/A	S, 2%*	D, 4%	D, 0%	S, 4%
14	N/A	D, 0%	S, 4%	D, 2%	S, 0%
15	N/A	S, 0%	N/A	N/A	D, 4%

* - Night omitted

APPENDIX D

MEAN SLEEP RESULTS

Table 1. Baseline/Screening Night Sleep Architecture From University of Ottawa

Subjects	1	2	3	4	5	MEAN (±SD)
Sleep Sat.						
SOL	4.0	7.0	NA	15.0	10.0	9.0 (4.1)
SE	97.0	93.0	NA	94.0	94.0	94.5 (1.5)
TST	406.0	361.0	NA	419.0	413.0	399.8 (22.8)
% Stage 2	64.0	51.0	NA	60.0	75.0	62.5 (8.6)
% REM	21.0	22.0	NA	24.0	10.0	19.3 (5.2)
% SWS	11.0	16.0	NA	10.0	12.0	12.3 (2.3)

Table 2. Mean Sleep Satisfaction in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	6.0	4.0	5.5	4.0	9.0	5.7 (2.0)
S2%	5.5	7.0	5.5	6.0	9.5	6.7 (1.7)
S4%	5.0	6.0	6.5	6.0	8.0	6.3 (1.1)
D0%	7.5	8.0	7.0	8.5	6.5	7.5 (0.8)
D2%	6.5	8.0	6.0	7.0	8.0	7.1 (0.9)
D4%	6.0	8.0	6.0	3.0	9.5	6.5 (2.5)

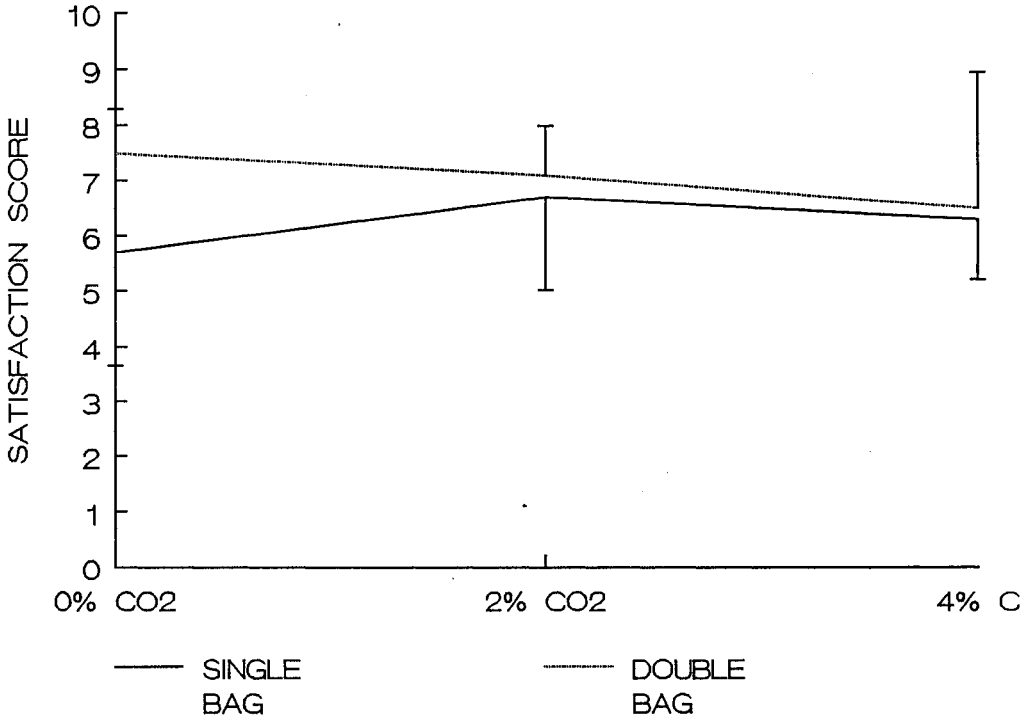

Figure 1. Mean Sleep Satisfaction in Each Condition

Table 3. Mean Sleep Onset Latency (SOL) in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	7.3	5.7	29.5	11.7	11.49	13.1	9.5
S2%	2.5	8.0	37.0	17.3	14.16	15.8	13.2
S4%	5.3	10.0	20.3	16.7	15.5	13.6	5.9
D0%	7.5	12.5	41.7	6.3	27	19.0	15.1
D2%	14.8	5.7	51.8	90	12.83	35.4	36.4
D4%	8.7	15.3	49.3	8.7	13.3	19.1	17.2

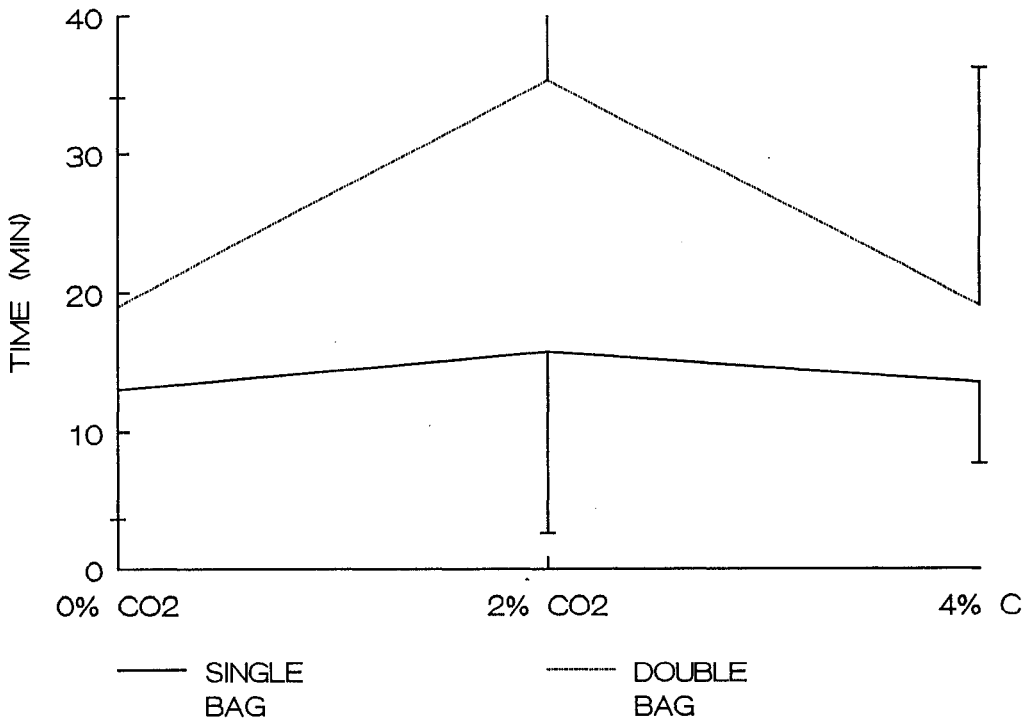

Figure 2. Mean Sleep Onset Latency (SOL) in Each Condition

Table 4. Mean Sleep Efficiency (SE) in Each Conditions

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	80.0	79.0	50.0	50.0	94.0	70.6	19.7
S2%	89.0	75.0	60.5	29.0	90.5	68.8	25.3
S4%	81.0	71.0	65.0	27.0	86.5	66.1	23.4
D0%	94.5	89.0	84.0	73.5	75.0	83.2	9.0
D2%	90.5	84.0	67.5	53.5	92.0	77.5	16.6
D4%	92.0	89.0	69.0	60.0	90.0	80.1	14.6

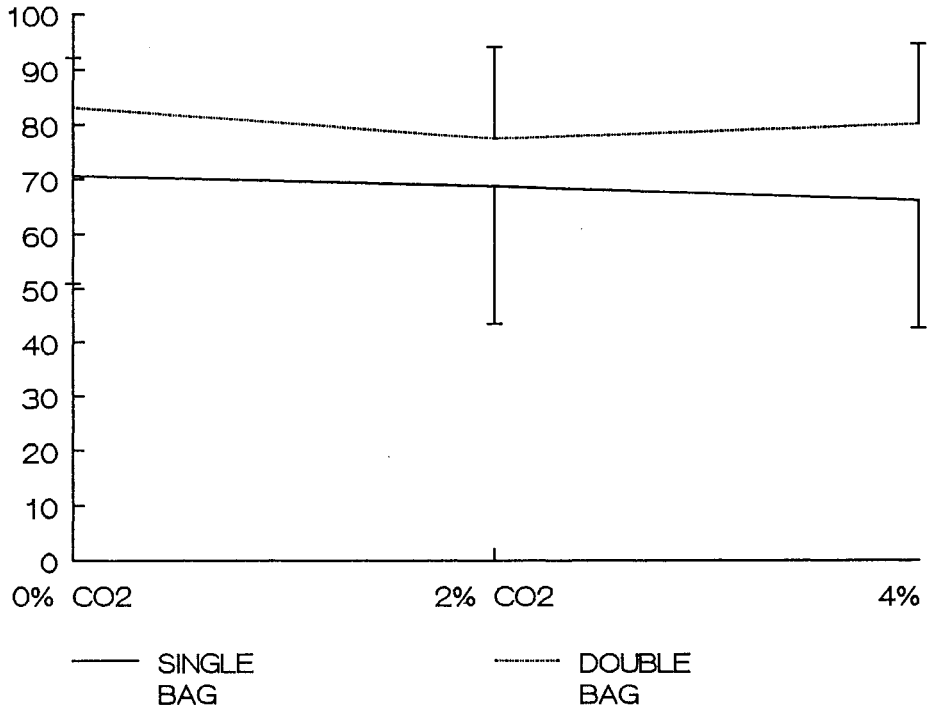

Figure 3. Mean Sleep Efficiency (SE) in Each Conditions

Table 5. Mean Number of Wakings in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	15.0	12.0	9.5	11.0	6.0	10.7	3.3
S2%	14.5	13.0	9.5	5.0	7.5	9.9	3.9
S4%	14.0	5.0	6.0	8.0	9.0	8.4	3.5
D0%	4.5	9.5	5.0	15.0	8.5	8.5	4.2
D2%	7.5	13.0	8.5	20.0	7.0	11.2	5.5
D4%	13.0	11.0	5.5	32.0	5.5	13.4	10.9

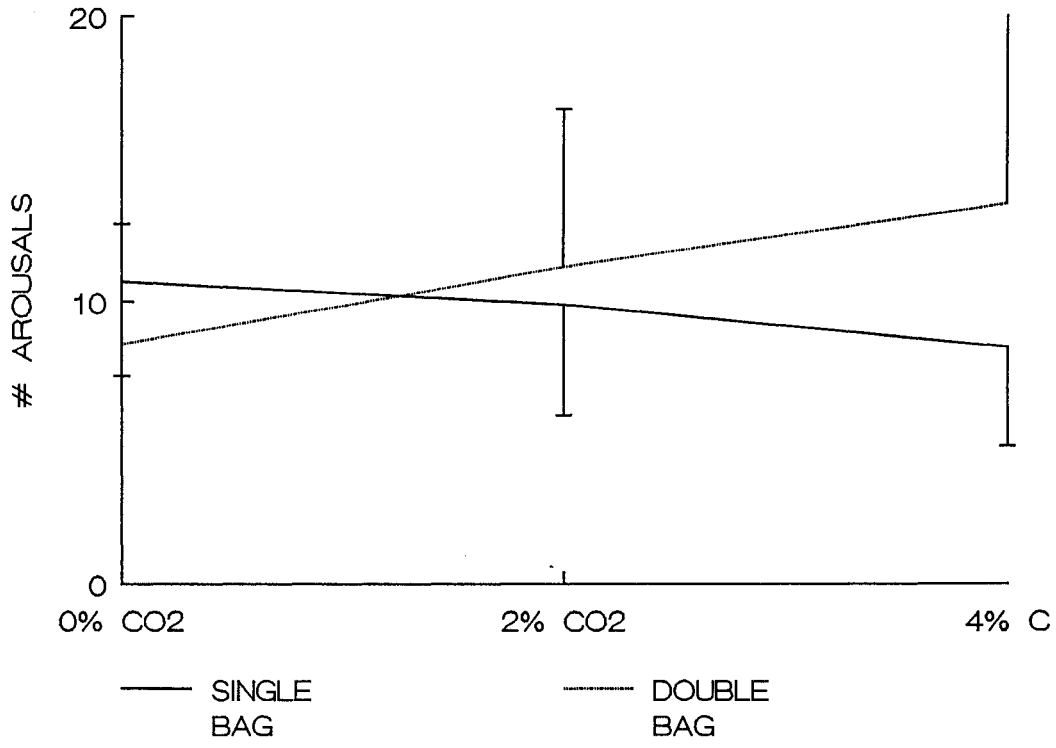

Figure 4. Mean Number of Wakings in Each Condition

Table 6. Mean Total Sleep Time (TST) in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	347.3	220.7	191.2	159.2	354.3	254.5	90.6
S2%	376.7	205.7	222.8	106.0	348.7	252.0	110.9
S4%	293.7	176.0	234.8	97.3	346.2	229.6	97.6
D0%	369.2	335.0	365.0	274.8	292.2	327.2	42.5
D2%	388.0	330.8	256.5	257.0	350.8	316.6	58.4
D4%	378.3	329.0	244.7	186.7	338.2	295.4	77.8

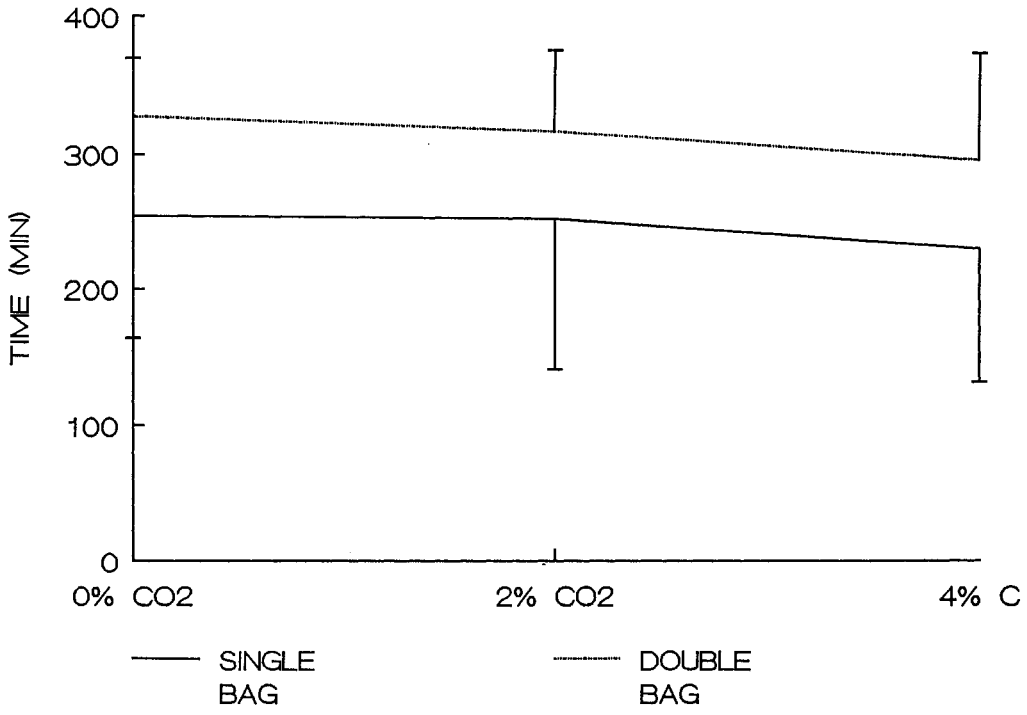

Figure 5. Mean Total Sleep Time (TST) in Each Condition

Table 7. Mean Stage 2 Percentage in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	71.0	74.0	55.5	59.5	75.0	67.0	8.9
S2%	81.5	75.0	71.0	66.0	75.5	73.8	5.8
S4%	80.0	61.0	63.5	69.0	75.0	69.7	7.9
D0%	78.0	67.0	74.5	74.5	66.5	72.1	5.1
D2%	83.5	56.5	62.0	61.5	73.5	67.4	10.9
D4%	86.0	69.0	75.0	56.0	63.5	69.9	11.4

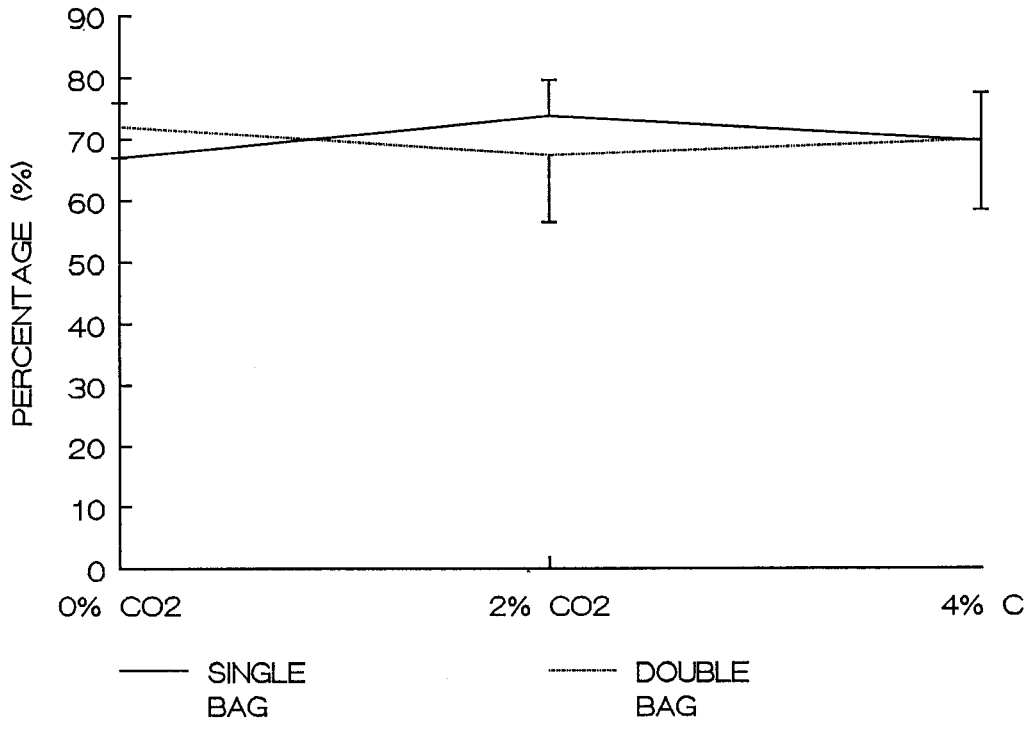

Figure 6. Mean Stage 2 Percentage in Each Condition

Table 8. Mean REM Percentage (REM) in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	14.0	6.0	5.5	4.0	13.5	8.6	4.8
S2%	8.0	6.0	0.0	0.0	10.0	4.8	4.6
S4%	2.0	16.0	9.0	0.0	5.0	6.4	6.3
D0%	10.5	16.0	11.5	7.0	11.5	11.3	3.2
D2%	8.5	23.0	5.5	11.0	10.0	11.6	6.7
D4%	5.0	17.5	11.0	0.0	18.5	10.4	8.0

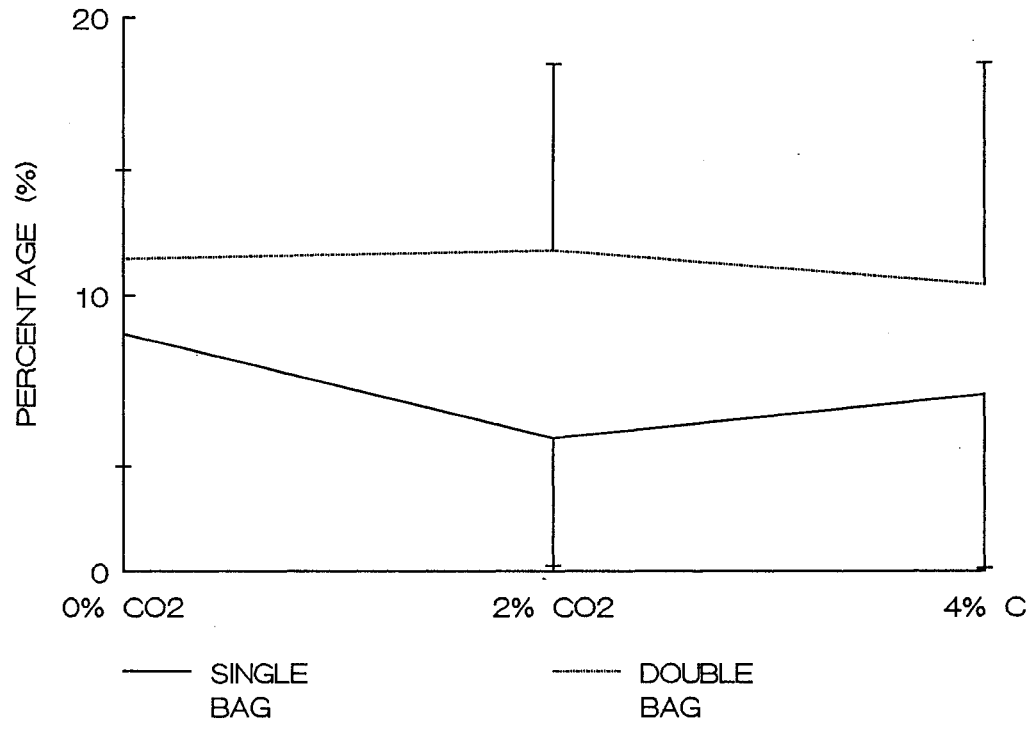

Figure 7. Mean REM Percentage (REM) in Each Condition

Table 9. Mean Slow Wave Sleep (SWS) in Each Condition

Subjects	1	2	3	4	5	MEAN	SD
Condition							
S0%	7.0	9.0	20.5	17.5	6.5	12.1	6.5
S2%	5.0	5.0	11.0	23.0	10.0	10.8	7.4
S4%	6.0	3.0	19.5	2.0	11.5	8.4	7.2
D0%	8.5	6.0	8.5	7.0	13.0	8.6	2.7
D2%	2.5	7.0	14.5	2.0	9.5	7.1	5.2
D4%	4.0	3.5	7.5	1.0	13.0	5.8	4.6

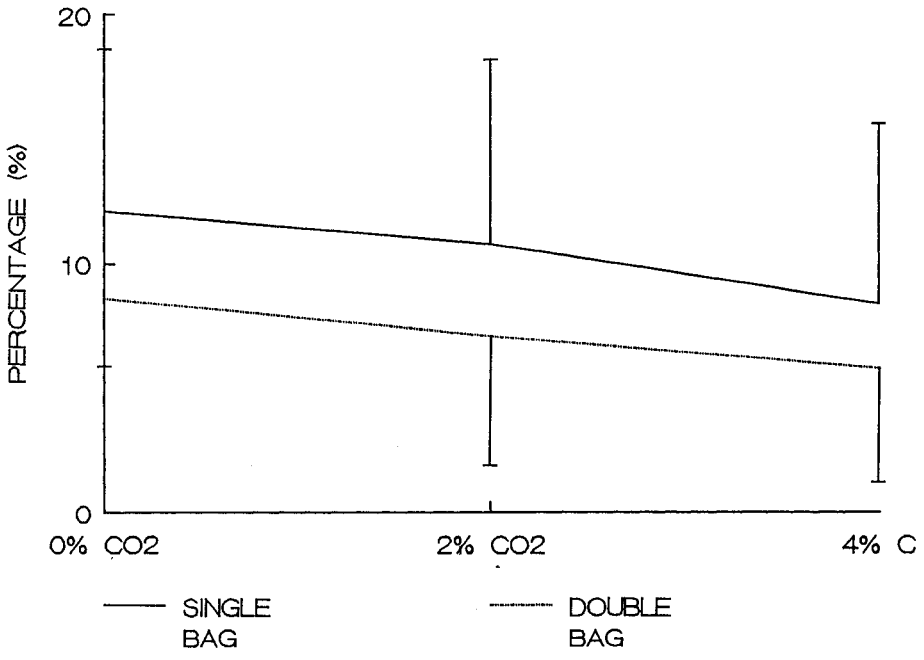

Figure 8. Mean Slow Wave Sleep (SWS) in Each Condition

APPENDIX E

MEAN TEMPERATURE RESULTS

Table 1. Mean T_b After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	34.2	34.0	34.0	33.6	34.4	34.0 (0.3)	34.1 (0.4)
S2%	34.5	34.0	33.8	34.0	34.3	34.1 (0.3)	34.1 (0.8)
S4%	34.7	33.7	33.6		34.0		34.0 (0.1)
D0%	34.7	35.2	34.3	34.8	34.7	34.7 (0.3)	34.7 (0.2)
D2%	34.7	34.7	34.3	34.9	34.6	34.7 (0.2)	34.6 (0.1)
D4%	35.4	34.9	34.5		34.7		34.9 (0.2)

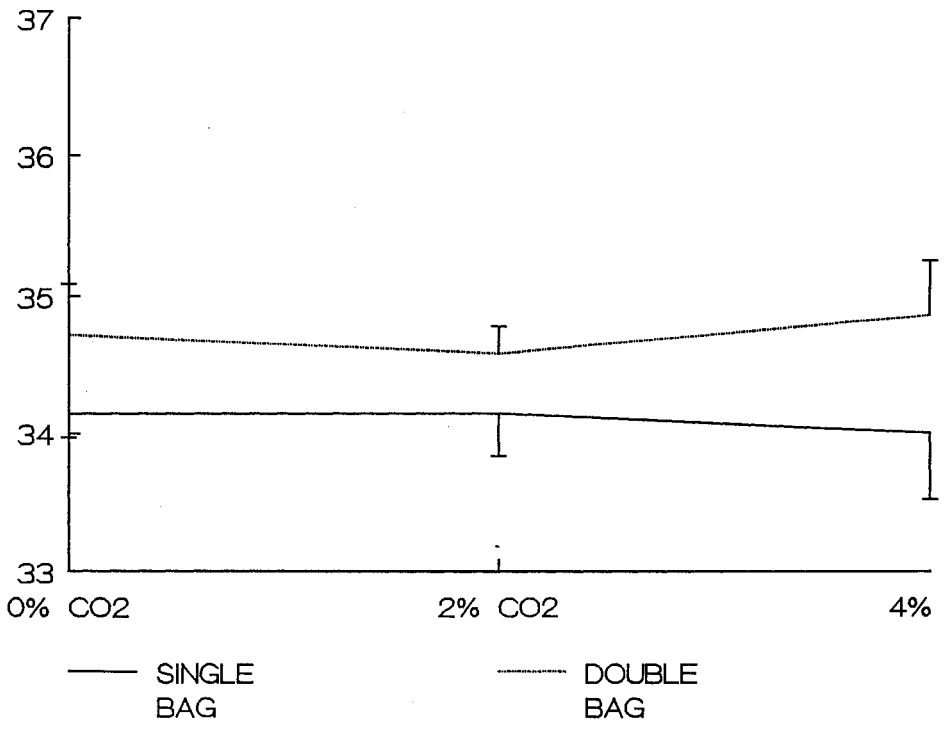

Figure 1. Mean T_b After 4 Hours in the Bag

Table 2. Mean Change in T_b After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	-0.4	-1.4	-1.0	-0.6	-0.4	-0.8 (0.4)	-0.8 (0.5)
S2%	-0.5	-1.1	-1.3	-1.1	-0.6	-0.9 (0.4)	-0.9 (0.4)
S4%	0.1	-1.5	-1.3		-1.3		-1.0 (0.7)
D0%	-0.0	-0.6	-1.0	-0.2	-0.7	-0.5 (0.4)	-0.6 (0.4)
D2%	-0.2	-0.7	-0.9	-0.2	-0.3	-0.5 (0.3)	-0.6 (0.3)
D4%	0.3	-0.5	-0.6		-0.4		-0.3 (0.4)

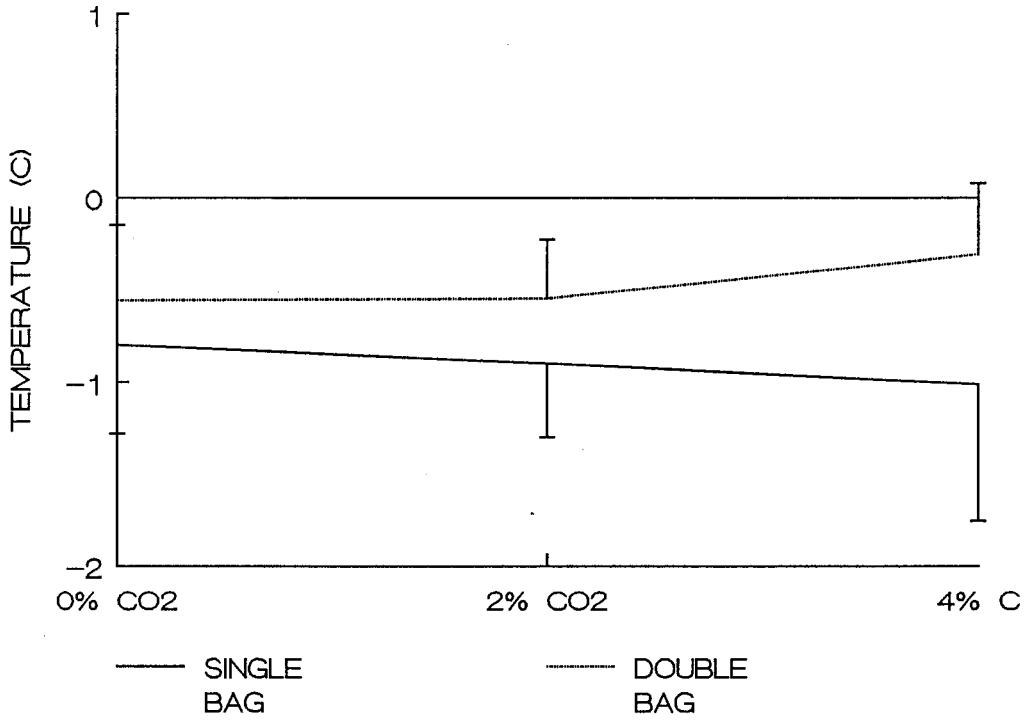

Figure 2. Mean Change in T_b After 4 Hours in the Bag

Table 3. Mean Drop in T_b in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	0.4	1.2	1.6	0.5	0.9	0.9 (0.5)	1.0 (0.5)
S2%	0.5	0.9	1.5	1.3	0.6	0.9 (0.4)	0.9 (0.5)
S4%	-0.2	1.1	1.5		1.1		0.9 (0.7)
D0%	-0.2	0.6	2.1	0.3	0.5	0.7 (0.8)	0.7 (1.0)
D2%	0.1	0.5	1.1	0.5	0.1	0.5 (0.4)	0.4 (0.5)
D4%	-0.2	0.3	0.9		0.3		0.3 (0.5)

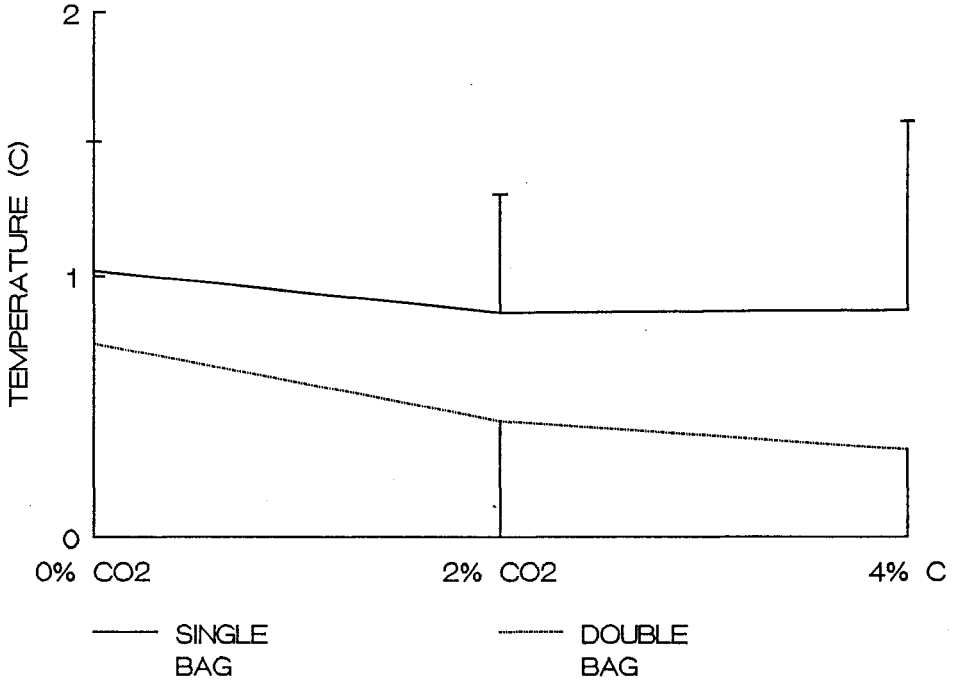

Figure 3. Mean Drop in T_b in Each Condition

Table 4. Mean minimum T_b in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	34.1	34.2	33.4	33.7	34.0	33.9 (0.3)	33.9 (0.3)
S2%	34.5	34.2	33.7	33.9	34.3	34.1 (0.4)	34.2 (0.3)
S4%	34.8	34.0	33.5		34.3		34.1 (0.5)
D0%	34.9	35.2	33.3	34.7	34.8	34.6 (0.8)	34.5 (0.7)
D2%	34.9	34.9	34.2	34.6	34.9	34.7 (0.3)	34.7 (0.3)
D4%	35.3	35.1	34.1		34.9		34.8 (0.4)

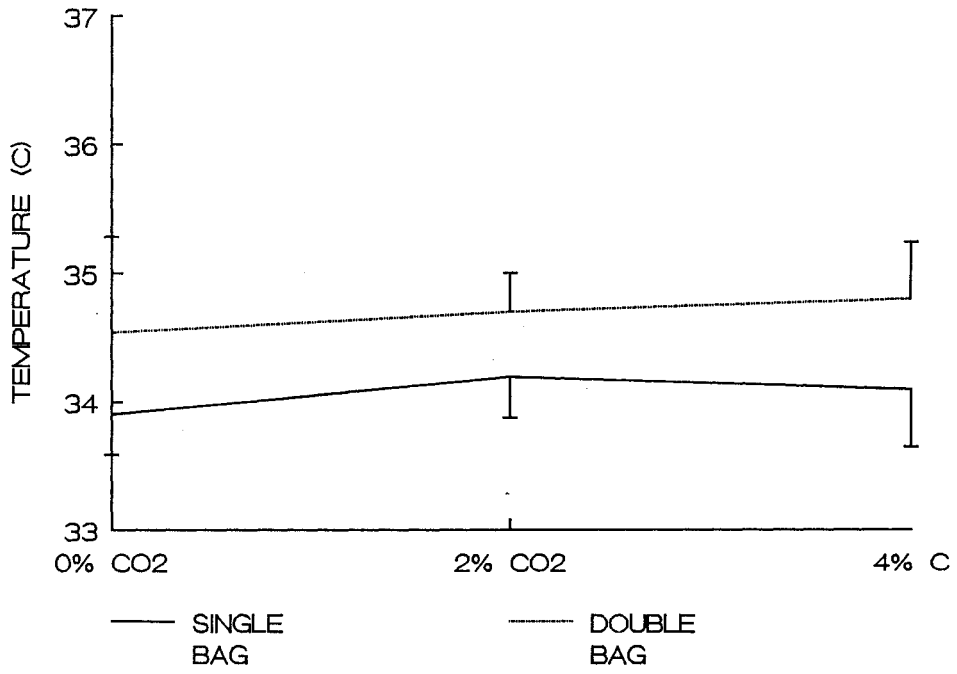

Figure 4. Mean minimum T_b in Each Condition

Table 5. Mean Time to Minimum T_b in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	352.0	254.0	221.5	265.5	309.5	280.5 (50.9)	284.3 (58.0)
S2%	391.5	254.0	336.5	247.0	336.5	313.1 (61.4)	329.6 (56.7)
S4%	306.0	240.0	291.5		265.0		275.6 (29.2)
D0%	277.0	379.0	408.5	369.0	307.0	348.1 (54.3)	342.9 (61.2)
D2%	389.5	337.0	276.5	389.0	273.5	333.1 (57.2)	319.1 (55.3)
D4%	98.0	312.5	310.0		352.5		268.3 (115.2)

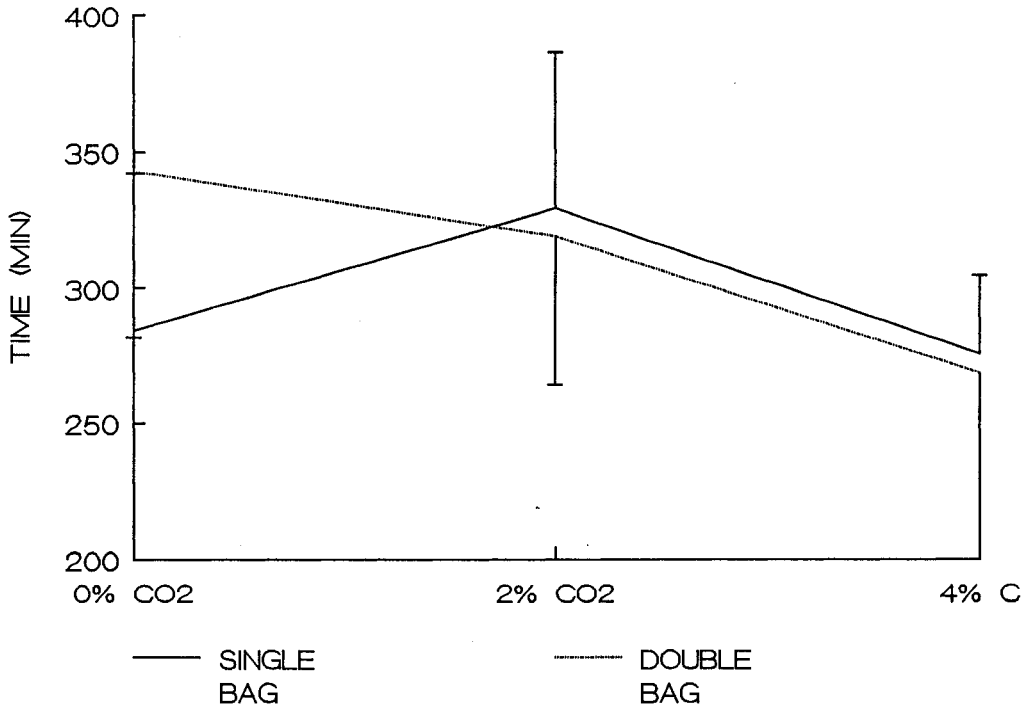

Figure 5. Mean Time to Minimum T_b in Each Condition

Table 6. Mean T_r After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	35.8	35.7	35.7	36.0	35.7	35.8 (0.1)	35.7 (0.0)
S2%	35.9	35.9	35.8	36.4	35.7	35.9 (0.3)	35.8 (0.1)
S4%	35.7	35.9	35.8		35.6		35.8 (0.1)
D0%	35.8	36.2	35.9	36.2	35.9	36.0 (0.2)	36.0 (0.2)
D2%	36.1	35.9	35.8	36.4	35.7	36.0 (0.3)	35.9 (0.1)
D4%	36.4	36.0	35.9		35.8		36.0 (0.2)

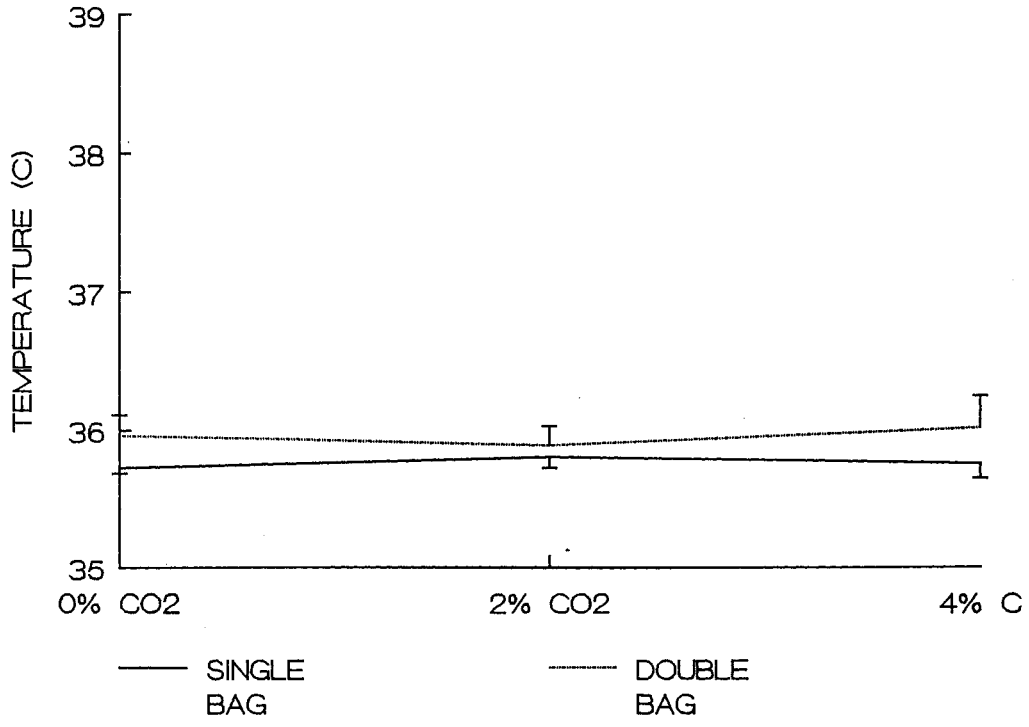

Figure 6. Mean T_r After 4 Hours in the Bag

Table 7. Mean Change in T_r After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	-0.7	-0.8	-1.1	-0.6	-0.5	-0.7 (0.2)	-0.8 (0.2)
S2%	-0.6	-0.7	-1.1	-0.6	-0.7	-0.7 (0.2)	-0.8 (0.2)
S4%	-0.1	-0.9	-1.2		-1.0		-0.8 (0.5)
D0%	-0.5	-0.5	-1.1	-0.4	-0.6	-0.6 (0.3)	-0.7 (0.3)
D2%	-0.3	-0.7	-1.1	-0.3	-0.6	-0.6 (0.3)	-0.7 (0.3)
D4%	-0.1	-0.6	-1.0		-0.5		-0.5 (0.4)

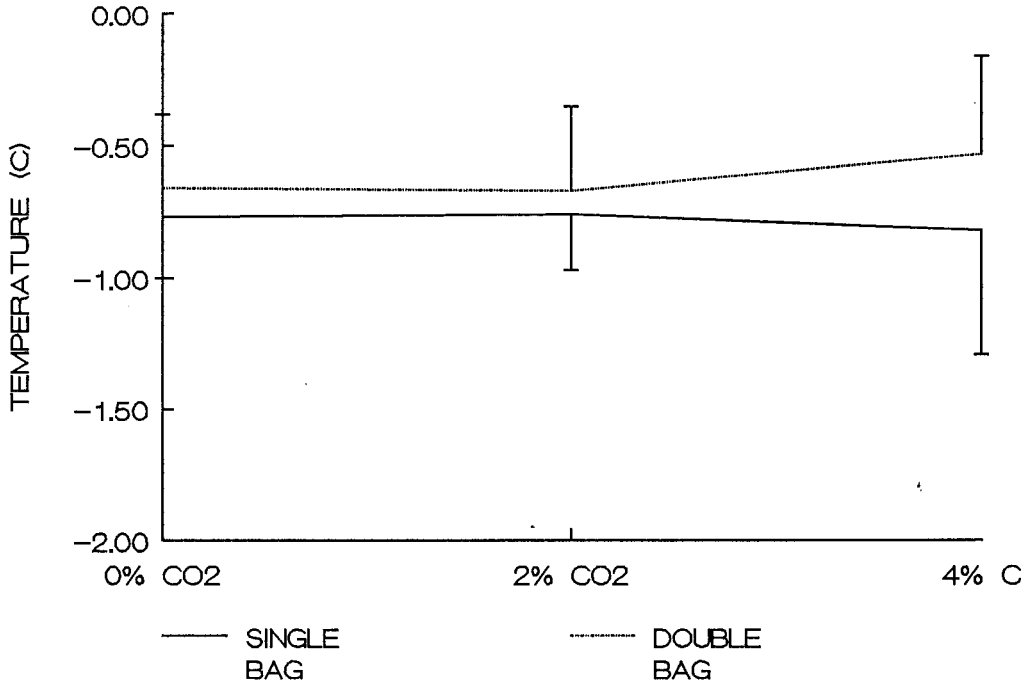

Figure 7. Mean Change in T_r After 4 Hours in the Bag

Table 8. Mean Drop in T_r in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	0.9	0.8	1.4	0.7	0.6	0.9 (0.3)	0.9 (0.3)
S2%	0.7	0.8	1.2	1.0	0.8	0.9 (0.2)	0.9 (0.2)
S4%	0.3	1.0	1.4		1.2		1.0 (0.5)
D0%	0.5	0.6	1.3	0.7	0.9	0.8 (0.3)	0.9 (0.4)
D2%	0.4	0.9	1.2	0.6	0.6	0.7 (0.3)	0.8 (0.4)
D4%	0.2	0.8	1.3		0.6		0.7 (0.4)

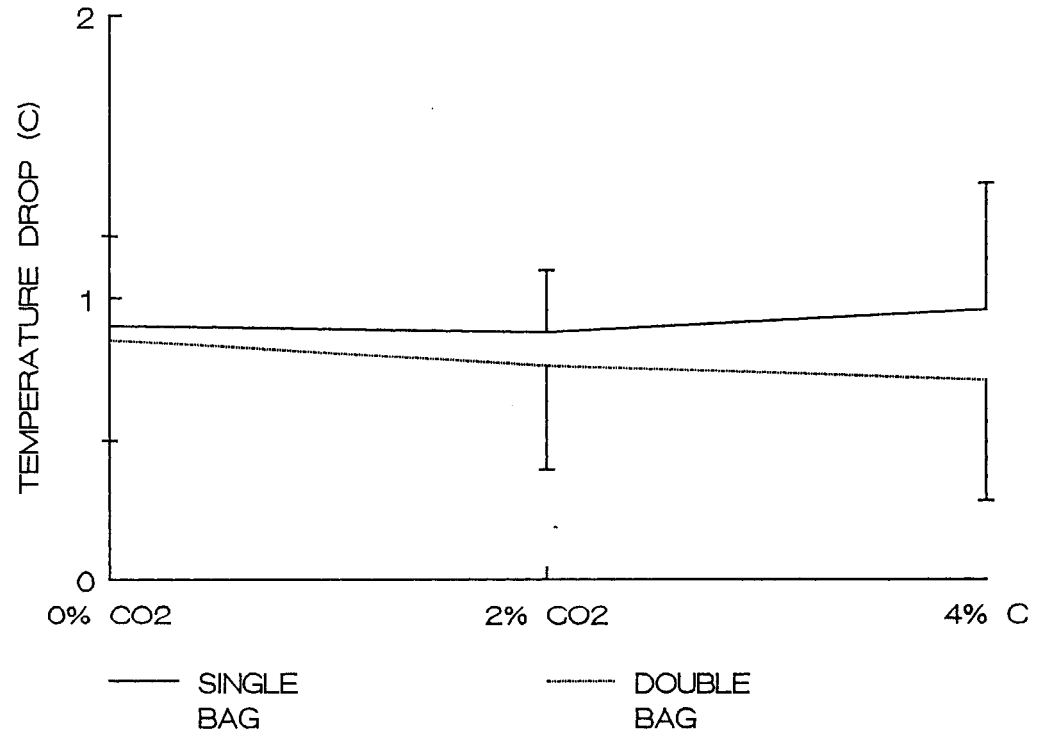

Figure 8. Mean Drop in T_r in Each Condition

Table 9. Mean Minimum T_r in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) N=5	MEAN (±SD) N=4
Trial							
S0%	35.6	35.7	35.4	35.9	35.6	35.6 (0.2)	35.6 (0.1)
S2%	35.8	35.8	35.7	36.0	35.6	35.8 (0.2)	35.7 (0.1)
S4%	35.5	35.8	35.6		35.5		35.6 (0.2)
D0%	35.8	36.1	35.7	35.96	35.6	35.8 (0.2)	35.8 (0.2)
D2%	36.0	35.8	35.7	36.1	35.7	35.9 (0.2)	35.8 (0.2)
D4%	36.2	35.8	35.6		35.8		35.8 (0.3)

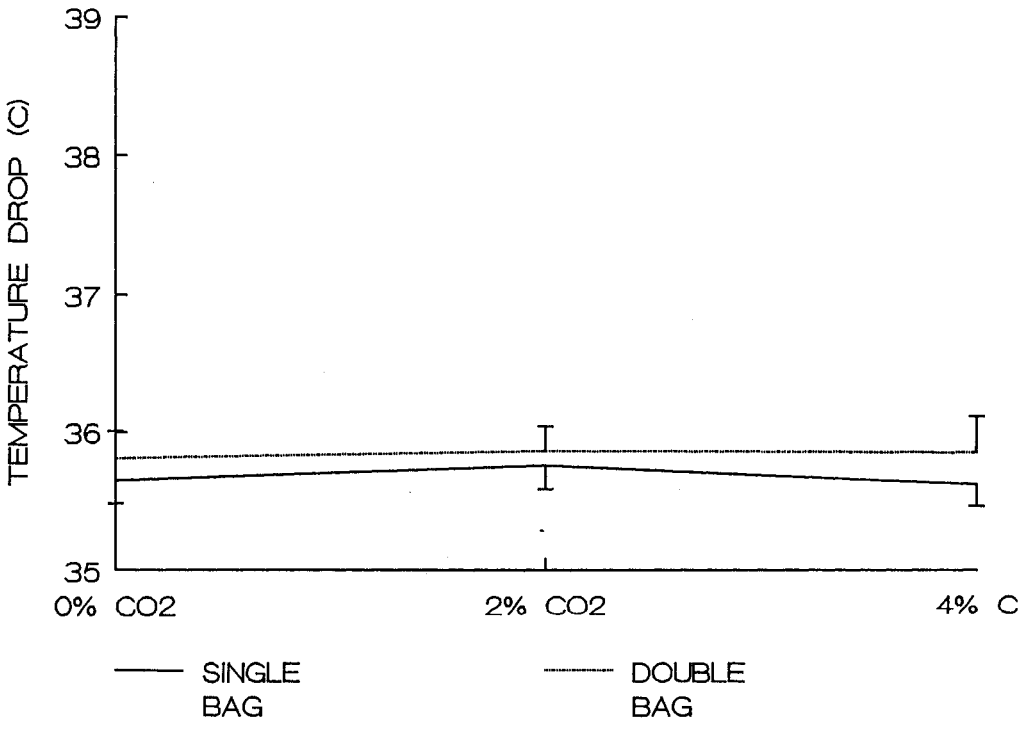

Figure 9. Mean Minimum T_r in Each Condition

Table 10. Mean Time to Minimum T_r in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN(±SD) N=4
Trial							
S0%	351.0	255.0	295.5	246.5	322.5	294.1 (44.3)	306.0 (40.9)
S2%	158.5	139.0	308.0	360.0	320.5	257.2 (101.1)	231.5 (96.0)
S4%	287.0	188.0	268.5		316.5		265.0 (55.0)
D0%	208.0	279.0	379.5	335.5	389.0	318.2 (75.4)	313.9 (86.4)
D2%	255.5	301.0	348.0	399.0	242.0	309.1 (65.3)	286.6 (48.1)
D4%	129.0	304.0	318.0		257.0		252.0 (86.1)

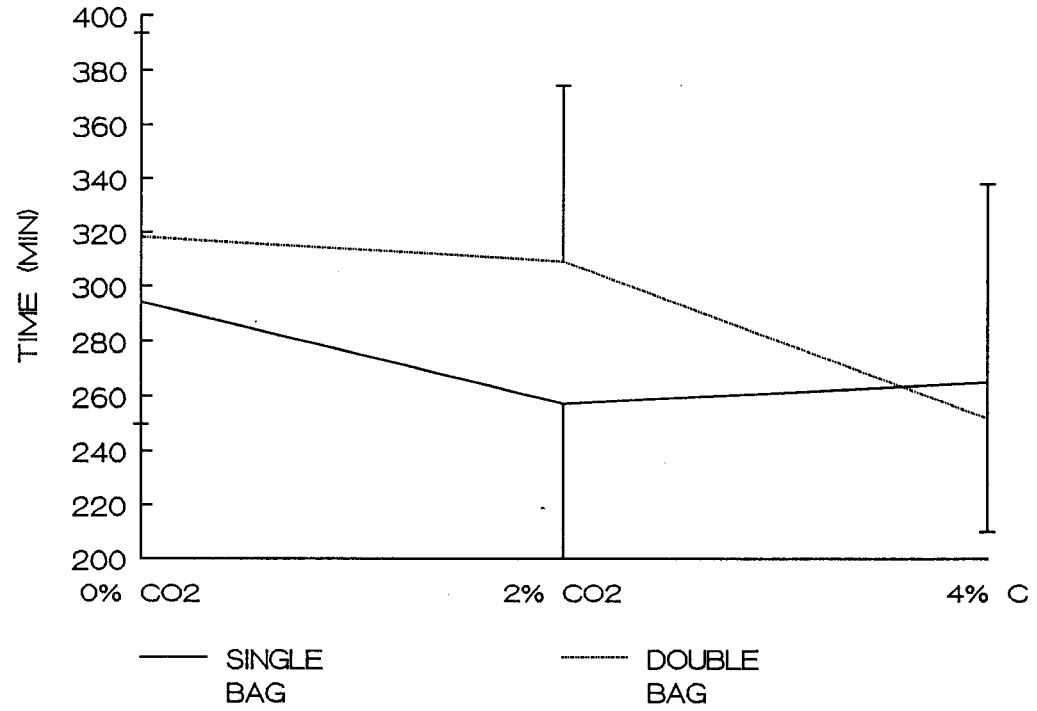

Figure 10. Mean Time to Minimum T_r in Each Condition

Table 11. Mean $\bar{T}_{sk}$ After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	32.0	31.7	31.6	29.8	32.8	31.6 (1.1)
S2%	32.9	31.1	30.9	30.0	32.3	31.5 (1.2)
S4%	33.6	30.3	30.4	30.7	31.8	31.4 (1.4)
D0%	33.3	34.4	32.2	33.2	33.3	33.3 (0.8)
D2%	33.1	33.4	32.3	32.9	33.3	33.0 (0.5)
D4%	34.5	33.9	32.7	31.0	33.4	33.1 (1.3)

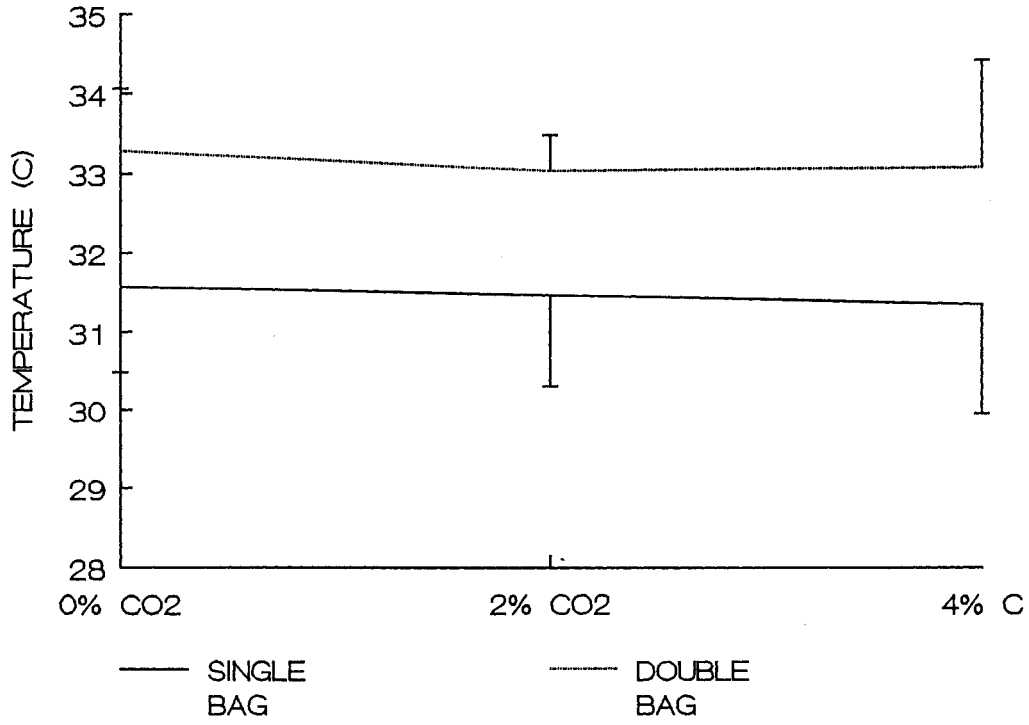

Figure 11. Mean $\bar{T}_{sk}$ After 4 Hours in the Bag

Table 12. Mean Change in $\bar{T}_{sk}$ After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	0.3	-2.7	-0.9	-0.7	-0.3	-0.9 (1.1)
S2%	-0.5	-1.9	-2.0	-2.7	-0.5	-1.4 (0.9)
S4%	0.6	-2.6	-1.7	-2.7	-2.0	-1.7 (1.3)
D0%	0.9	-0.7	-0.9	0.3	-0.8	-0.2 (0.8)
D2%	-0.2	-0.7	-0.7	-0.2	0.1	-0.3 (0.4)
D4%	0.9	-0.3	-0.1	-2.2	-0.3	-0.3 (1.1)

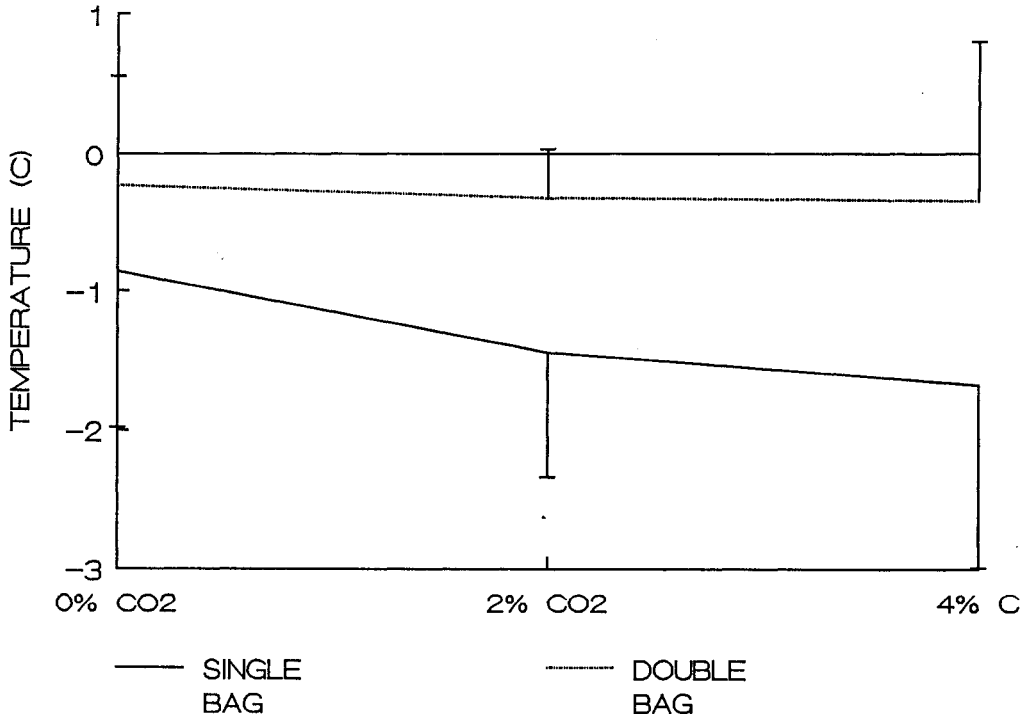

Figure 12. Mean Change in $\bar{T}_{sk}$ After 4 Hours in the Bag

Table 13. Mean Drop in $\bar{T}_{sk}$ in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	0.6	3.2	3.1	0.5	0.8	1.6 (1.4)
S2%	1.2	2.2	4.2	1.7	1.3	2.2 (1.2)
S4%	0.3	2.6	3.1	4.2	2.2	2.5 (1.4)
D0%	-0.5	1.7	2.8	1.6	1.1	1.3 (1.2)
D2%	0.7	1.2	2.1	1.5	0.2	1.2 (0.8)
D4%	0.0	0.6	1.5	0.3	0.9	0.7 (0.6)

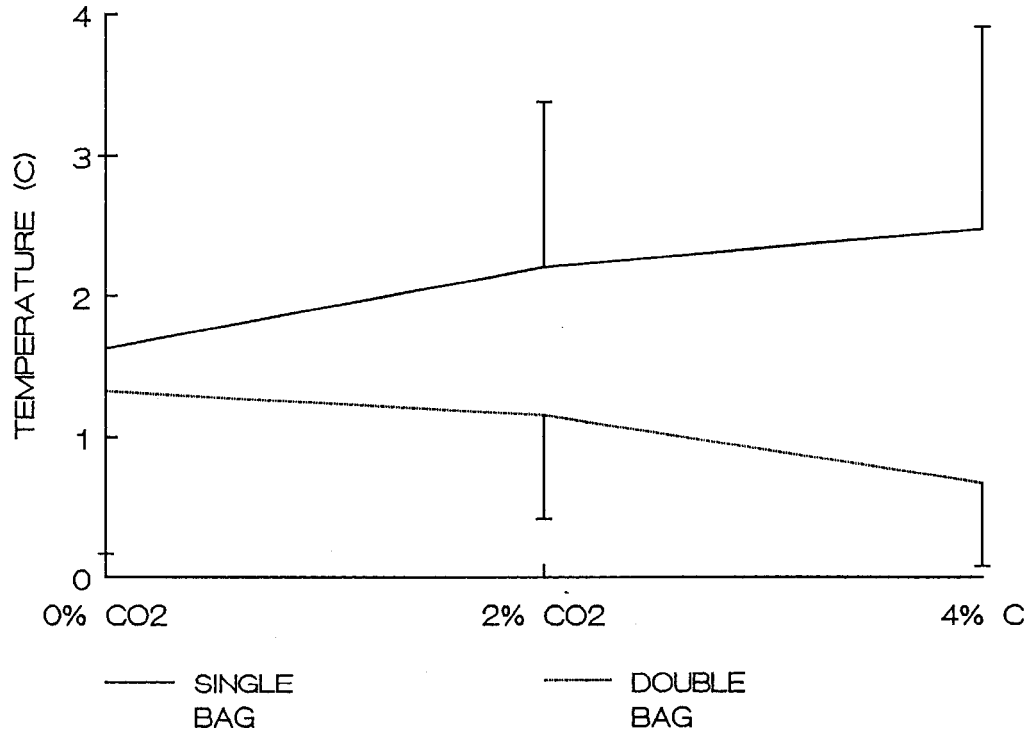

Figure 13. Mean Drop in $\bar{T}_{sk}$ in Each Condition

Table 14. Mean Minimum $\bar{T}_{sk}$ in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	31.1	31.2	29.4	30.0	32.3	30.8 (1.1)
S2%	31.8	30.8	28.7	30.7	31.5	30.7 (1.2)
S4%	32.8	30.3	28.9	29.2	31.5	30.5 (1.6)
D0%	32.9	33.5	30.3	31.3	33.0	32.2 (1.3)
D2%	32.5	32.8	30.9	31.6	33.0	32.3 (0.9)
D4%	33.6	33.5	31.0	32.9	32.9	32.8 (1.0)

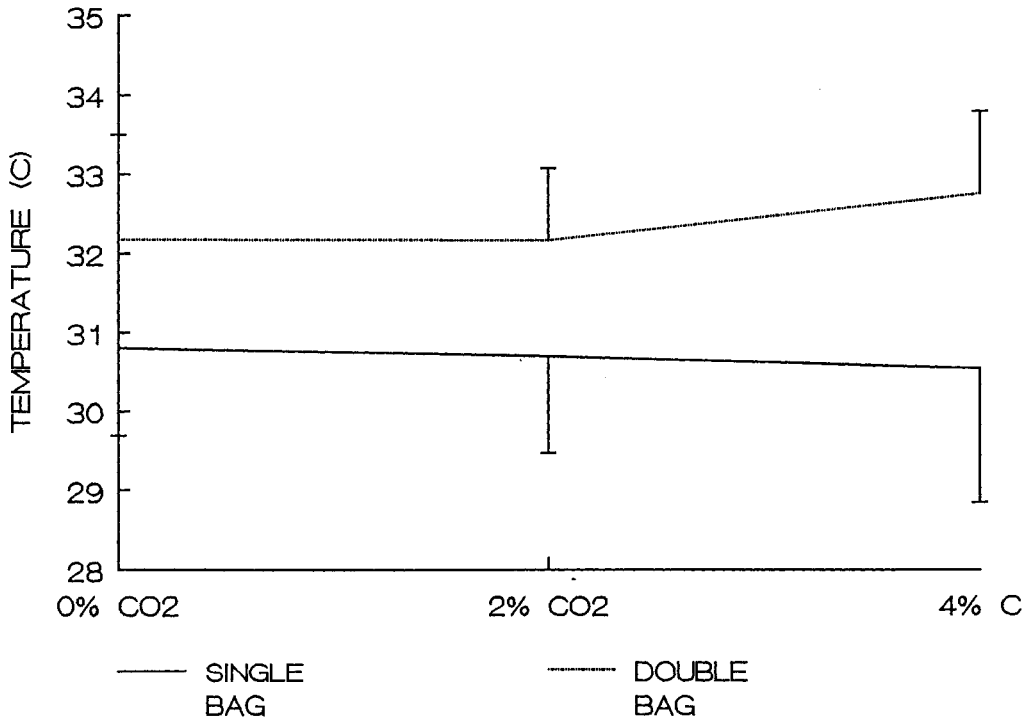

Figure 14. Mean Minimum $\bar{T}_{sk}$ in Each Condition

Table 15. Mean Time to Minimum $\bar{T}_{sk}$ in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	352.0	254.0	220.0	280.0	310.0	283.2 (50.8)
S2%	406.0	254.0	316.0	196.0	290.0	292.4 (77.8)
S4%	185.0	237.0	292.0	347.0	295.5	271.3 (62.0)
D0%	369.0	374.0	324.5	357.5	291.0	343.2 (35.0)
D2%	389.5	355.5	276.5	385.0	271.5	335.6 (57.8)
D4%	342.0	258.0	310.0	287.0	366.0	312.6 (42.9)

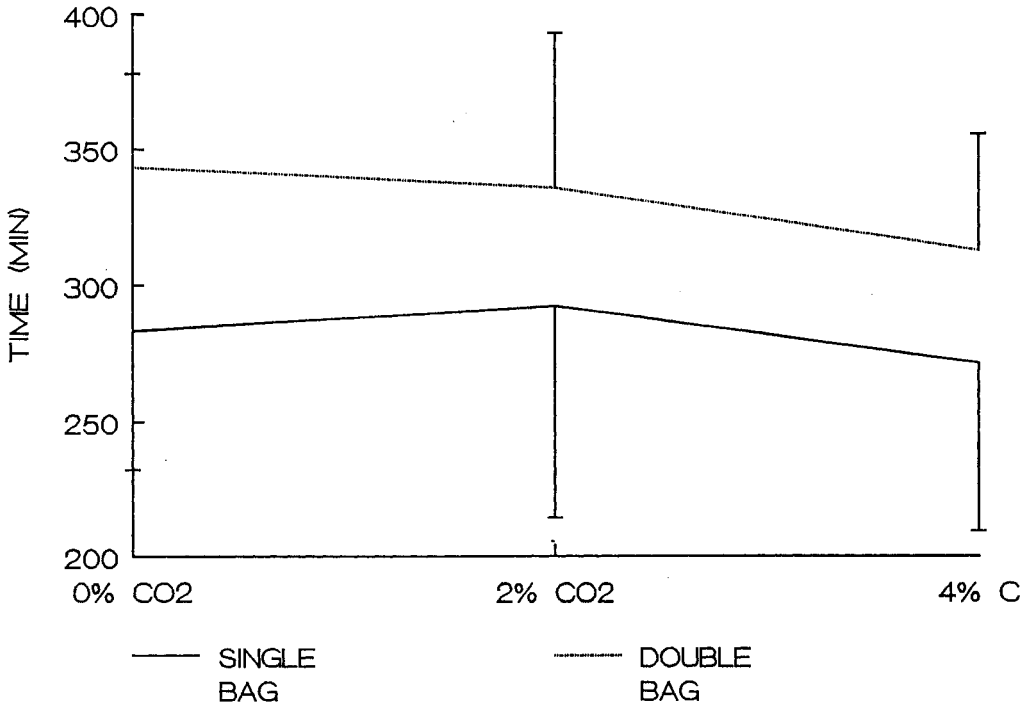

Figure 15. Mean Time to Minimum $\bar{T}_{sk}$ in Each Condition

Table 16. Mean T_{toe} After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	24.6	27.8	27.2	28.8	34.0	28.5 (3.4)
S2%	24.6	15.0	34.5	20.0	31.1	25.0 (7.9)
S4%	19.8	18.2	26.3	23.8	32.3	24.2 (5.4)
D0%	24.3	33.3	34.6	34.6	34.4	32.2 (4.5)
D2%	33.3	26.2	34.3	34.5	33.5	32.4 (3.5)
D4%	34.2	33.7	32.9	24.4	33.7	31.8 (4.2)

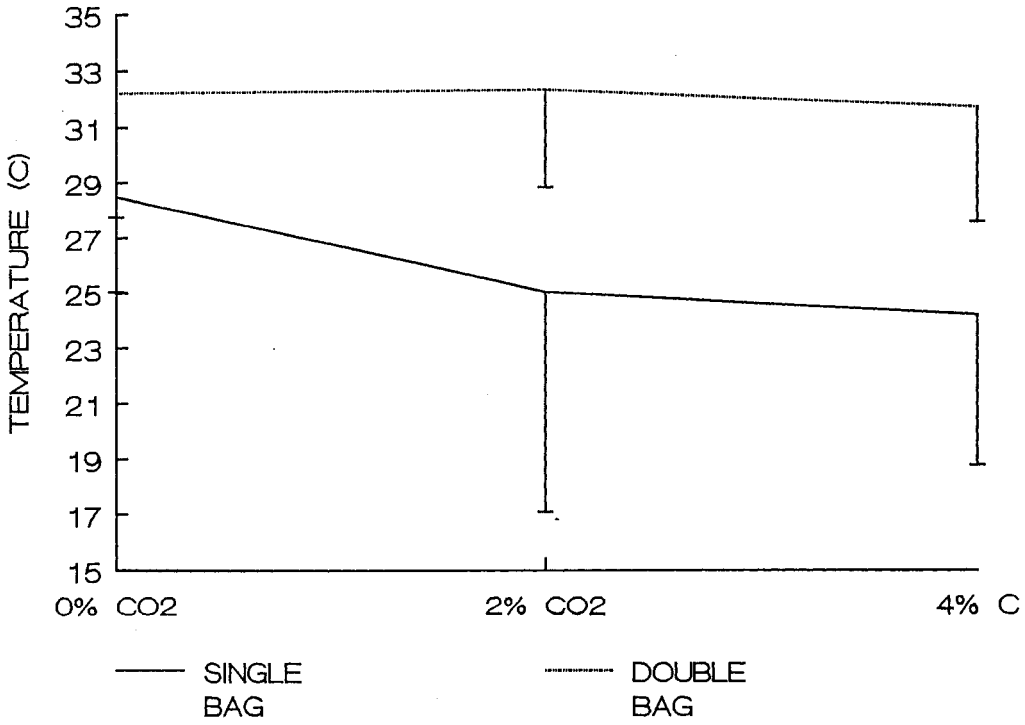

Figure 16. Mean T_{toe} After 4 Hours in the Bag

Table 17. Mean Change in T_{toe} After 4 Hours in the Bag

Subjects	1	2	3	4	5	MEAN (±SD) n=5
Trial						
S0%	-4.8	-2.8	-4.6	2.3	-0.2	-2.0 (3.0)
S2%	-6.0	-8.7	0.7	-11.1	-2.7	-5.5 (4.7)
S4%	-8.9	-10.9	1.4	-9.2	-2.5	-6.0 (5.2)
D0%	0.1	0.6	0.5	1.2	0.5	-0.6 (0.4)
D2%	2.2	-3.2	2.1	0.7	1.9	-0.7 (2.3)
D4%	1.4	5.7	2.7	-9.8	-0.2	-0.1 (5.9)

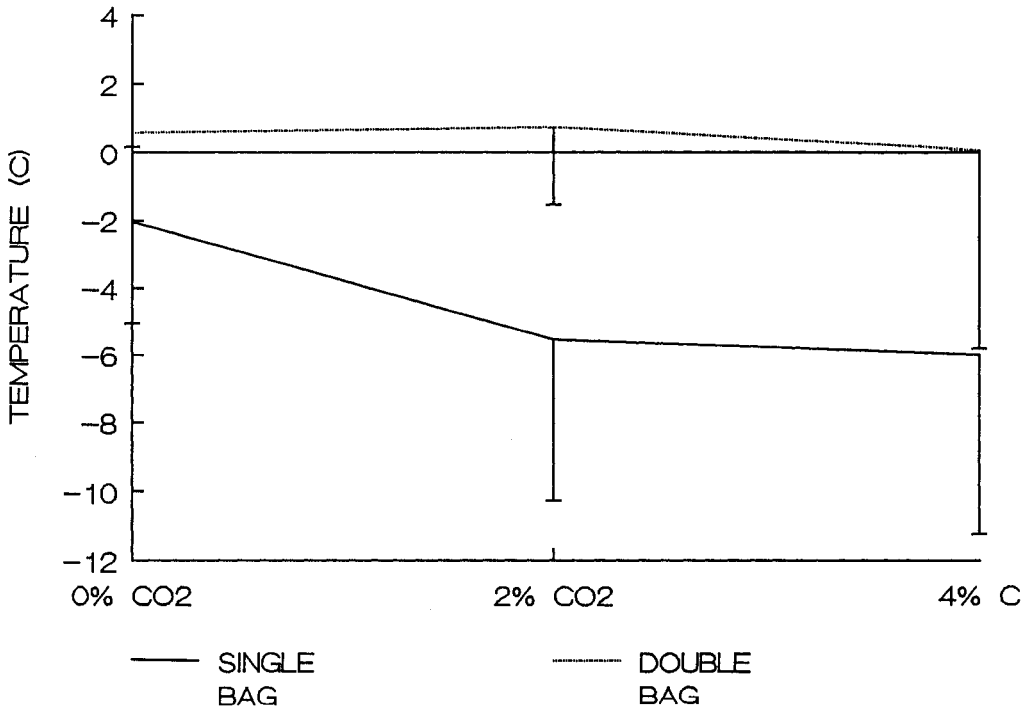

Figure 17. Mean Change in T_{toe} After 4 Hours in the Bag

Table 18. Mean Drop in T_{toe} in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	12.3	4.7	12.5	4.2	4.2	7.6 (4.4)	8.4 (4.6)
S2%	12.1	8.7	10.0	15.1	6.8	10.5 (3.2)	9.4 (2.2)
S4%	12.9	11.0	2.6		10.0		9.1 (4.5)
D0%	4.7	-0.2	4.8	10.1	0.8	4.0 (4.1)	2.5 (2.6)
D2%	2.1	6.6	1.0	4.1	-0.1	2.7 (2.6)	2.4 (2.4)
D4%	1.3	-3.7	-1.0	2.7	2.5	0.4 (2.7)	-0.2 (2.7)

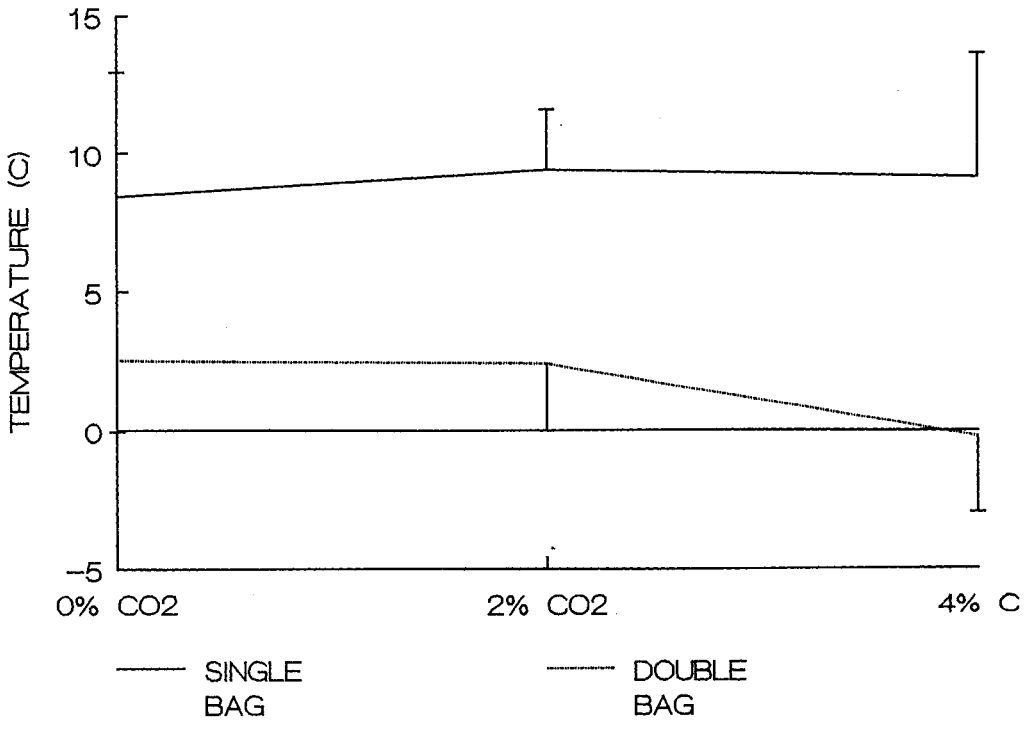

Figure 18. Mean Drop in T_{toe} in Each Condition

Table 19. Mean Minimum T_{toe} in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	17.1	26.0	19.4	22.4	30.0	23.0 (5.2)	23.1 (5.9)
S2%	18.6	15.0	23.7	16.0	27.0	20.1 (5.1)	21.1 (5.3)
S4%	15.8	18.7	22.3		24.7		20.4 (3.9)
D0%	19.5	32.8	29.3	23.2	33.1	27.6 (6.0)	28.7 (6.3)
D2%	29.0	22.7	31.2	29.8	31.6	28.9 (3.6)	28.6 (4.1)
D4%	31.5	31.7	31.2	31.5	31.4	31.5 (0.2)	31.5 (0.2)

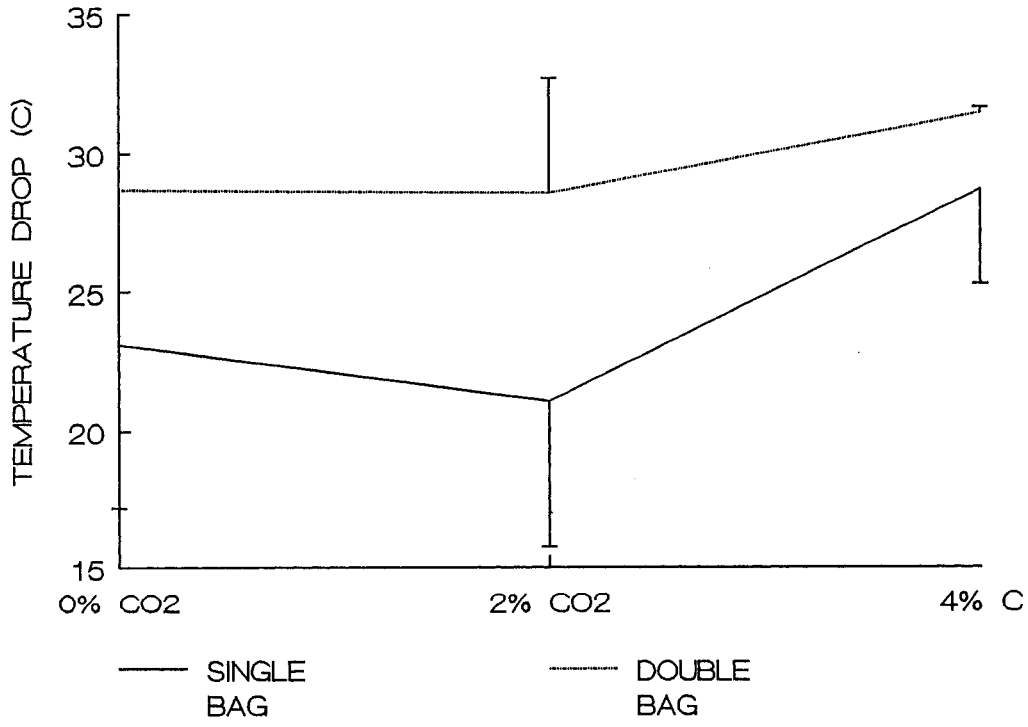

Figure 19. Mean Minimum T_{toe} in Each Condition

Table 20. Mean Time to Minimum T_{toe} in Each Condition

Subjects	1	2	3	4	5	MEAN (±SD) n=5	MEAN (±SD) n=4
Trial							
S0%	432.0	273.0	363.0	274.5	375.0	343.5 (68.8)	360.8 (65.8)
S2%	423.5	238.0	363.5	175.0	373.5	314.7 (103.8)	349.6 (78.9)
S4%	361.0	243.0	317.5		400.5		330.5 (67.5)
D0%	350.5	374.0	350.5	357.5	369.0	360.3 (10.8)	361.0 (12.3)
D2%	352.0	373.5	290.5	345.5	355.5	343.4 (31.3)	342.9 (36.2)
D4%	357.0	348.0	339.0	215.0	274.0	306.6 (60.7)	329.5 (37.7)

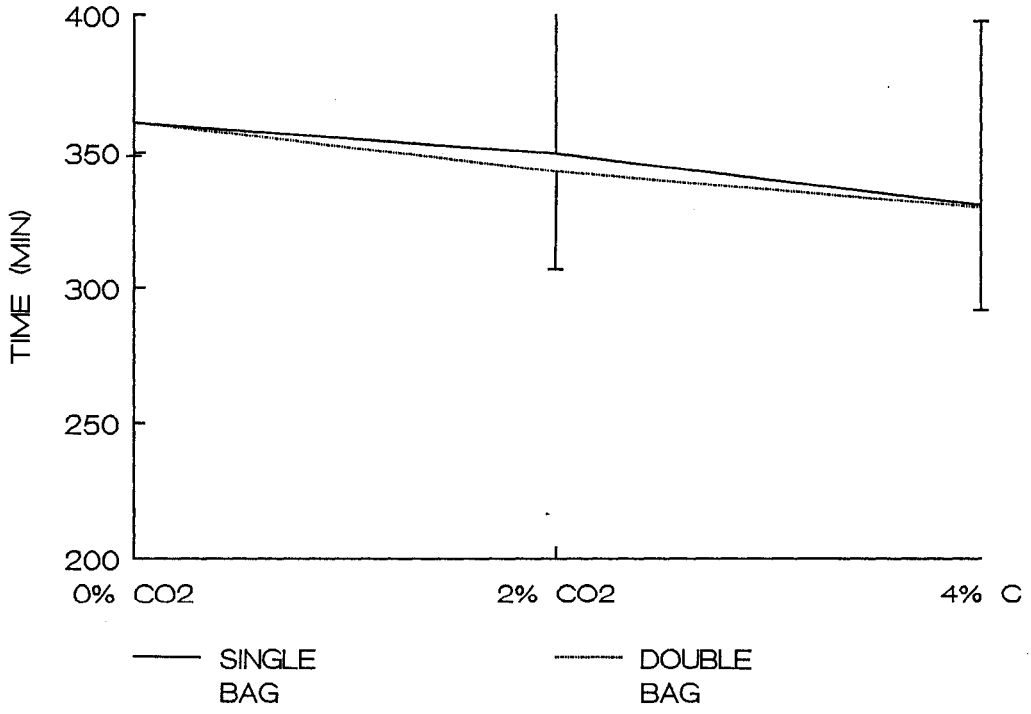

Figure 20. Mean Time to Minimum T_{toe} in Each Condition

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- Fraser et al. (1989). Effect of sleep and circadian cycle on sleep period energy expenditure. 66(2), 830-836. **Figure 3.**
- Bullard (1964). Effects of carbon dioxide inhalation on sweating. 19(1), 137-141. **Figure 1.**
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