POSTOPERATIVE MANAGEMENT OF ANAESTHESIA-ASSOCIATED HYPOTHERMIA WITH A FORCED-AIR CONVECTIVE WARMING DEVICE

by

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DECLARATION

I, Stephanie Ana Maria Jackson declare that this research report is my own work. It is submitted to the University of the Witwatersrand, Johannesburg for the degree of Master of Medicine in the branch of Anaesthesia . It has not been submitted before for any degree or examination at this or any other University.

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September 1997

DEDICATION

In memory of my father Charles Martin Jackson, 1931 - 1996.

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ABSTRACT

This study investigated the postoperative management of hypothermia of intraoperative origin using a forced-air convective warming device. Hypothermia develops during the intraoperative period partly as a result of disordered thermoregulation induced by anaesthesia and partly because of the nature of the operational injury and the surgical environment. Both the hypothermic state and the consequences of physiological attempts to return the core temperature to normal, which take place during the postoperative period, are associated with non-beneficial effects. Attempts to prevent intraoperative decline in core temperature are a part of anaesthesia management. However, most of the traditional options available are inefficient or ineffective, particularly in adults. This study evaluated the performance of a new device, the forced-air convective warmer, in the management of the postoperative hypothermic state. Results showed that when compared to a hypothermic control group the device made a significant difference to the thermal state of a group of hypothermic postoperative patients but only if it was used for at least two hours postoperation.

INTRODUCTION

Hypothermia, defined as a core temperature of less than 36 degrees Centigrade (°C), is a common sequel of operative intervention. Hypothermia is accepted as having beneficial effects in certain circumstances such as neurosurgical and cardiac operations. There are a number of detrimental effects of hypothermia particularly in the postoperative period - such as shivering, cardiovascular instability and arterial hypoxaemia. A number of options are available to the clinician for the purposes of reducing or preventing intraoperative heat loss and promoting heat gain. However the majority are only able to prevent the heat loss that would have occurred if an option was not used (for example, warming cold intravenous fluid) or reduce heat loss by insulating the patient from the environment (for example, metallised plastic sheets). It should be stressed that these options are unable to prevent completely the progressive decline in core temperature that occurs during the intraoperative period. Options which actively add heat to the patient, for example, warm water circulating mattresses suffer from two major drawbacks. They are either inefficient in terms of warming, or the risks associated with their use outweigh their potential advantages.

1.1 Temperature Regulation And Anaesthesia

1.1.1 Normal Temperature Regulation

Homeothermic species require a nearly constant internal body temperature. Significant deviations from normal core temperature cause metabolic functions to deteriorate. Human thermoregulatory defence mechanisms are triggered by as small a change in core temperature as 0.2°C from a desired set point. Hypothermia will occur if the normal regulatory mechanisms are impaired or overwhelmed, for example, when illness, exposure to cold or a combination of exposure and drugs decreases thermoregulatory efficacy. During surgery both exposure to a cold operating room environment and anaesthesia induced inhibition of thermoregulation may produce hypothermia. Prevention and management of temperature related complications are improved by an understanding of both normal and drug influenced thermoregulation.[1,2]

Thermoregulatory control is centred in the hypothalamus which receives information from other parts of the brain, the skin surface, the spinal cord and deep central tissues. The normal core temperature range, 36.5 to 37.5°C, is known as the inter-threshold range. A desired core temperature is maintained via either warm responses such as active vasodilatation, sweating or appropriate behavioural modification (dressing warmly) or cold responses such as vasoconstriction, non-shivering thermogenesis, shivering and behavioural changes (removing warm clothes). The warm or cold responses are triggered at specific core temperatures outside the inter-threshold range (see Table I).

Table I: Threshold temperature of various physiologic responses in the awake, normal person and the anaesthetised person[8]

Physiological Response	Threshold Temperature (°C)	
	Normal	Anaesthetised
Sweating	37.9	40.5
Vasodilation	37.8	40
Vasoconstriction	36.9	36.8
Non-Shivering Thermogenesis	36.7	34.1
Shivering	36.2	33.9

Therefore there are, in essence, three components of thermoregulation: afferent thermal sensing, central regulation and efferent response. Temperature information is obtained from thermally sensitive cells throughout the body. Cold signals travel primarily via A-delta nerve fibres, warm information via unmyelinated C-fibres of the spinothalamic tracts in the anterior spinal cord. Thermal input is derived largely from deep abdominal and thoracic tissues, spinal cord and the brain, so no single tissue can be thought of as a gold standard for temperature determination.

The hypothalamus regulates body temperature by evaluating the thermal information from various body regions. How the body determines absolute threshold temperatures is unknown but the thresholds vary daily, in both sexes, by about 1°C (normal circadian rhythm), and monthly in women by 0.75°C. The hypothalamus responds to temperatures outside the thresholds via the appropriate response mechanisms - either increasing or decreasing metabolic heat production or altering environmental heat loss - thus maintaining a central temperature near 37°C despite varying environmental temperatures.

Examples of physiological thermoregulatory mechanisms are shivering and sweating. Shivering may increase metabolic heat production by 200-600% in adults. However efficiency is less than would be expected because the vasodilatation in muscle leads to heat loss to the environment. Consequently, shivering is the last defence against hypothermia - its temperature activation

threshold is below that for vasoconstriction and non-shivering thermogenesis. Sweating, under the right circumstances, can result in the loss of up to 2 litres of fluid per hour. As sweat evaporates from the body surface, heat loss and body cooling occur. Sweating is the only mechanism by which humans can lose heat in an environment where ambient temperature exceeds core temperature. Active vasodilatation is controlled by release of mediators from the sweat glands and heat stress can increase capillary blood flow up to 7.5 litres per minute.[1]

Overall, the human body gains or loses heat through 4 physical processes:

Conduction: defined as heat exchange between objects or substances at different temperatures that are in direct contact with one another.

Convection: defined as heat exchange between an object and the moving air that surrounds the object.

Radiation: defined as transfer of heat by infrared electromagnetic radiation from one object to a second object that is at a different temperature and not in contact with the first object.

Evaporation: defined as transfer of heat from the body via vaporization of water on the skin and mucous membranes of the mouth and respiratory passages.[3]

The equation describing heat balance is:

$$\Delta h = (M - W) + R + Cn + Cv + Ev$$

where: Δh = net change in heat content

M = metabolic heat production

R = radiation (gain or loss)

W = work heat production

Cn = conduction (gain or loss)

Cv = convection (gain or loss)

Ev = evaporation (gain or loss)

For an anaesthetised person in an operating theatre, work heat production is negligible. Average metabolic heat production is 100 kcal/hr. Heat is usually lost by radiation, conduction, convection and evaporation (i.e. they have negative values). In a thermoneutral environment, heat loss equals heat gain, thus, $\Delta h = 0$. In other words:

$$\Delta h = (M + W) - (R + Cn + Cv + Ev) = 0$$

1.2 Thermoregulation during Anaesthesia

During general anaesthesia the threshold at which a cold response is triggered is shifted down to about 34.5°C depending on the anaesthetic technique. Warm response thresholds increase to 38°C.[2] Thus, under anaesthesia, if core temperature is allowed to fall it is only when a temperature of 34.5°C is reached that the first line of defence against hypothermia (vasoconstriction) will be triggered. Therefore, when the thermoregulatory responses are inhibited, the ability to maintain a normal core temperature is compromised.

The particular conduct of anaesthesia may even influence whether or not a physiological defence against hypothermia can be mounted. For example, if shivering is prevented as a result of the use of non-depolarising muscle relaxants, core temperature may decrease below the trigger threshold for shivering without the appropriate physiological response taking place. If anaesthesia technique prevents all thermoregulatory responses from taking place, then central temperature can only remain normal in a thermoneutral environment.[1]

Since general anaesthesia decreases activation thresholds for responses to hypothermia by about 2.5°C and increases those defending against hyperthermia by 1°C the inter-threshold temperature range is widened. The anaesthetised person becomes poikilothermic and body temperature changes

are determined by redistribution of heat within the body and the difference between metabolic heat production and heat loss to the environment. The thermoregulatory cold threshold in healthy adults varies with the precise drug combinations used to produce anaesthesia. For healthy adults given halothane and oxygen, enflurane and oxygen, or fentanyl and nitrous oxide, the threshold temperature is about 34.5°C. The combination of propofol and nitrous oxide produces more thermoregulatory impairment than typical doses of inhalational agents, having a threshold of approximately 33°C.

Core temperature exhibits a typical pattern during general anaesthesia. Core temperature decreases rapidly for one hour (initial hypothermia) then more slowly for the next two to three hours (linear decrease) and finally becomes constant (plateau phase). During initial preparation for surgery patients are exposed to a cool environment and the skin is cleaned using a solution which evaporates from the skin surface. Following the induction of anaesthesia metabolic heat production decreases and cutaneous vasodilatation occurs. Cold intravenous fluid is administered and the lungs are ventilated with dry gas leading to increased respiratory heat loss. Sessler[4] showed that in dressed volunteers central temperature is stable during a 30 minute control period and then decreases by 1.2°C in the 45 minutes following induction of anaesthesia. Since cutaneous heat loss decreased only slightly during the control period and decreased more rapidly following induction of anaesthesia, skin preparation and fluid evaporation cannot fully explain intraoperative hypothermia. Sessler[4] concluded that decreased heat production and increased heat loss are

insufficient to explain the initial central hypothermia following induction of general anaesthesia and proposed an alternate explanation. Core temperature is a poor measure of mean body temperature and body heat content because the temperature in peripheral tissues, which is about half of the body mass, changes considerably depending on thermoregulatory status, environmental temperature and time spent in a particular environment. Peripheral tissue temperature and heat content is considerably lower than central temperature. Anaesthetic induced vasodilatation only minimally increases cutaneous heat loss to the environment but it does allow mixing of heat in the central and peripheral compartments. The result is peripheral warming at the expense of central temperature. Central hypothermia during anaesthesia and surgery appears to be largely the result of redistribution of heat within the body. Similar internal redistribution of heat causes the initial hypothermia associated with epidural anaesthesia.[1] The slow linear decrease in central temperature typically observed during the second to third hour of anaesthesia results simply from heat loss to the environment exceeding metabolic heat production. When patients are in a relatively warm environment and undergoing minor operations the central temperature plateau seen after the second to fourth hour may be passive, in other words without thermoregulation. In contrast when patients are sufficiently hypothermic this plateau is often accompanied by active thermoregulatory vasoconstriction. Peripheral vasoconstriction constrains metabolic heat to the central thermal compartment preventing further heat loss, sometimes even increasing central temperature. Heat loss

from peripheral tissues may continue unabated but these tissues receive less metabolic heat, thus the temperature plateau does not represent a true thermal steady state and heat loss may continue to exceed heat production.

1.2.1 Heat Exchange during Anaesthesia

Mechanisms of hypothermia due to anaesthesia are:

1 decreased metabolic heat production,

2 redistribution of heat within the body

3 inhibition of central thermoregulation.

Anaesthesia decreases the basal metabolic rate (BMR) even in the absence of induced hypothermia. Observation of continuous mixed venous oxygen saturation (SvO_2) during induction of anaesthesia suggests a decrease in oxygen consumption, because SvO_2 climbs from 75% to 95%. The cold environment of the operating room predisposes to heat loss from the patient by the process of radiation. Skin preparation and wound irrigation produce a decrease in the skin temperature. Cold intravenous solutions contribute to a decrease in core temperature. The cold stress of one unit of bank blood at 4-8°C is equivalent to that of 1 litre of colloid or crystalloid at 16-20°C leading to a decrease in core temperature of about 0.25°C. Exposure of pleural, pericardial and peritoneal surfaces results in dramatic evaporative loss during major abdominal surgery. Although the rate of heat loss is greatest in the first hour of surgery, all the mechanisms of heat loss are exacerbated when surgery is prolonged.

1.2.2 Effect of Various Anaesthetic Agents on Thermoregulation

1.2.2.1 Narcotics

Fentanyl - nitrous oxide anaesthesia decreases the threshold for vasoconstriction by 2.5°C. Narcotics with potent sympatholytic effects such as fentanyl, sufentanil and alfentanil presumably impede sympathetic responses to hypothermia. There do not appear to be important differences in heat exchange when volatile or narcotic anaesthetic techniques are used for cardiac anaesthesia.

1.2.2.2 Muscle Relaxants

Muscle relaxants contribute to heat loss during anaesthesia by reducing muscle tone and abolishing shivering.

1.2.2.3 Volatile Agents

Volatile anaesthetic agents such as halothane depress the threshold for peripheral vasoconstriction by 2.5°C depending on the depth of anaesthesia[5] while isoflurane decreases the threshold by 3 degrees Centigrade.[6] Furthermore, by causing muscle relaxation, enflurane and isoflurane decrease heat production.

1.2.2.4 Regional anaesthesia

Peripheral vasoconstriction responses are overcome by sympathetic blockade and heat generation from muscle is reduced by muscle relaxation. Spinal thermoregulatory centres may be depressed by spinal epidural anaesthesia or narcosis, for example epidural sufentanil inhibits shivering and, consequently body temperature decreases. Information from peripheral thermal receptors may be blocked by local or regional anaesthesia. Inadvertent hypothermia is certainly as common under regional as under general anaesthesia and may be slower to resolve. Shivering during epidural anaesthesia occurs only in hypothermic patients and is always preceded by vasoconstriction above the level of neural blockade.

1.2.3 The Effect of Age on Thermoregulation

Heat loss during anaesthesia is exacerbated at the extremes of age. Elderly people and neonates are less able to protect their body core temperature from environmental influences. The causes of thermoregulatory failure in the elderly include loss of muscle mass, decreased resting muscle tone and increased surface area to body mass ratio. The elderly exhibit a diminished cutaneous vasoconstriction response and have limited cardiovascular reserve. Average heat production between the age of 20 and 40 years is 40 kcal/m^2/hour. In

patients over the age of 60 years this decreases to 30 kcal/m^2/hour. The most rapid decrease in temperature in the operating theatre occurs during the first hour of anaesthesia, at the age of 20 years the decrease averages 0.3°C, in an eighty-year old patient it is about 1.1°C.[7]

Thermoregulatory failure is prominent in the premature and low birth weight neonate. Excessive heat loss occurs because there is an increased surface area to body mass ratio, high respiratory water loss, thin subcutaneous tissue and an immature shivering response. Cold-stressed full term infants are able to double their basal metabolic rate by the process of non-shivering thermogenesis. The lack of brown fat in premature infants deprives them of this advantage. Heat loss is exacerbated by cerebral damage, sedative drugs and hypoglycaemia. Neonatal hypothermia is a serious condition. Its adverse effects include increased oxygen consumption, intra pulmonary haemorrhage, apathy, acidosis, apnoea and ventricular fibrillation.[7]

1.3 *Temperature Monitoring during the Peri-operative Period*

The monitoring of core temperature by an anaesthesiologist is an essential aspect of the care of the peri-operative patient. This view is supported by the Standards for Basic Intraoperative Monitoring published by the American Society of Anaesthesiology, January 1991 in which it is stated " There shall be readily available a means to continuously measure the patient's temperature, when changes in body temperatures are intended, anticipated or suspected the temperature shall be measured".

1.3.1 Intraoperative Temperature Monitoring

During anaesthesia body temperature may be obtained from various sites (See Table III). Core temperature can be determined by a single temperature probe adjacent to the tympanic membrane or in the nasopharynx, pulmonary artery, distal oesophagus or urinary bladder. Skin temperature is considerably lower than central temperature, correlates poorly with central temperature, and is not suitable for intraoperative measurement of temperature. Temperature monitoring usually is not helpful during the first 30 minutes of anaesthesia and it is not necessary when surgery is completed within this period.

1.3.2 Postoperative Temperature Monitoring

The importance of anaesthesia as a cause of intraoperative hypothermia is widely appreciated and it is because anaesthesia impairs the heat regulatory mechanisms that it becomes important to measure changes in core temperature. Only if we understand the physiology of thermal derangement and measure the changes will we be in a position to initiate the correct treatment when it is needed. Hypothermia may be present postoperation because methods available to actively warm patients are not used or are inefficient or hypothermia has been deliberately induced during the intraoperative period. Observations performed in the Department of Anaesthesiology, Johannesburg Hospital, have shown that there is a steady decline in pulmonary artery blood temperature in patients undergoing

abdominal vascular surgery without the use of active intraoperative warming. It is important to realise that thermal changes in both the intraoperative and postoperative periods carry clinical implications for fluid management, haemodynamic management, analgesia and patient comfort.

Various anatomical sites are available for core temperature monitoring. The tympanic membrane monitor uses infrared technology and nasopharyngeal, oesophageal, pulmonary artery blood, rectum and bladder catheters uses thermistor technology. (See Table II).

Table II: The Advantages and Disadvantages of the Various Sites of Temperature Measurement[22]

	Advantages	Disadvantages
1. Tympanic Membrane	Non-invasive method of determining core temperature. Sensor must be applied to, or pointed directly at the tympanic membrane	Risk of perforation of the tympanic membrane, bleeding especially heparinised patients.
2. Nasopharyngeal	Proximity to the internal carotid artery.	Affected by inspired gas temperature, danger of epistaxis.
3. Oesophageal	Convenient, close to the great vessels and heart.	To be placed 24 centimetres below larynx between left atrium and a Readings affected by inspired gas temperature and thoracic surgery.
4. Rectal	A traditional site, good guide to warming after induced hypothermia.	Variable reflection of core temperature affected by faeces, peritoneal lavage, cystoscopy.
5. Bladder	Better reflection of core temperature than rectal. Can be incorporated into urinary drainage catheters.	Affected by rate of urine flow and cannot be used in genito- urinary procedures.
6. Oral	Convenient, simple	Affected by food, hyperventilation and is not practical during anaesthesia.
7. Axillary	Convenient, simple	Must be placed over axillary artery, and is affected by blood pressure cuffs, intravenous solutions. Takes about ten to fifteen minutes to equilibrate.
8. Skin	Can be placed on the anterior abdominal wall. Indicates temperature gradients between the core, periphery (redistribution of heat), cutaneous vascular tone (perfusion, thermoregulation). May be used on fingers, toes, limbs. Used to quantify total body heat.	Forehead temperature correlates modestly with central temperature only when environmental temperature is constant. Skin temperature affected by peripheral perfusion. Inaccurate in presence of vasoconstriction and sweating. Not a reliable warning of malignant hyperthermia because of cutaneous vasoconstriction.
9. Pulmonary artery catheter	Measure of pulmonary artery blood temperature.	Affected by thoracic surgery, extra corporeal circulation and mechan ventilation.

A study conducted by the Department of Anaesthesia, Johannesburg Hospital evaluated a thermistor-bearing urethral catheter as a monitor of core temperature both in the intraoperative and the postoperative period in patients undergoing aortic aneurysmectomy. Ten such patients were recruited and, in addition to standard monitoring, a pulmonary artery catheter and thermistor-bearing urethral catheter were inserted. Pulmonary artery blood and intravesical temperature were measured simultaneously. The study indicated that the thermistor-bearing urethral catheter was a reliable monitor of core temperature in both the intra and postoperative periods (J van Westing & C Clinton - Unpublished data).

1.4 Peri-operative Hypothermia

Hypothermia is defined as a core temperature of less than 36°C and there are various levels of hypothermia.

Early hypothermia: 35 to 35.9°C.

Mild hypothermia: 32-34.9°C

Moderate hypothermia: 28-31.9°C where muscle rigidity occurs and compensatory mechanisms such as shivering are absent. There is an obtunded mental state, all vital signs are depressed and hypotension, decreased intestinal motility, ileus and cardiac arrhythmias are common. The outcome depends on the underlying disease.

Severe or deep hypothermia: 17-27.9°C. Coma is present, brain stem reflexes are absent, hypotension is present, ventricular fibrillation or marked cardiac instability is seen. Oliguria and azotaemia occur and pancreatitis, apnoea or agonal respiratory patterns are seen. Mortality is high, about 25-80 per cent.

Profound hypothermia: 4-16.9°C. The electroencephalogram is isoelectric and apnoea, cardiovascular collapse and disseminated intravascular coagulopathy are present. The mortality is high, about 80-100 per cent.

1.4.1 Preoperative Risk Factors for Hypothermia

1. Extremes of age.
2. Burns more than twenty per cent of the total body surface area.
3. Cardiac dysfunction such as congestive heart failure and myocardial infarction.
4. Central nervous system disease.
5. Cancer associated cachexia.
6. Dermal loss greater than 20 per cent of the total body surface area such as burns, exfoliative dermatitis, ichthyosis and severe psoriasis.
7. Drugs:
   - (i) alcohol,
   - (ii) anaesthetic agents,
   - (iii) barbiturates,
   - (iv) digoxin,
   - (v) narcotics,
   - (vi) tetracyclines,
   - (vii) phenothiazines,
   - (viii) tricyclic antidepressants
   - (ix) rauwolfia derivatives.

8. Exertion.

9. Exhaustion.

10. Exposure.

11. Hepatic disease, malnutrition - cachexia and hypoproteinaemia.

12. Metabolic disorders:

    (i) acidosis,

    (ii) diabetic ketoacidosis,

    (iii) hypoadrenalism,

    (iv) hypoglycaemia,

    (v) hypopitutlarism

    (vi) hypothyroidism or myxoedema.

13. Paget's disease of bone.

14. Pancreatitis.

15. Peripheral vascular disease.

16. Post resuscitation.

17. Renal failure or uraemia.

18. Bacterial infections.

19. Shock of any cause.

20. Smoking.

21. Trauma Injury:

    (i) head injury

    (ii) polytrauma.

22. Accidental environmental exposure.

23. Cold exposure (avalanche, ski slope, post exertion)

24. Immersion in cold water.

25. Inadequate indoor heating (particularly in the elderly).

1.4.2 Intra-operative Hypothermia

1.4.2.1 Operating room and intra-operative extrinsic risks for hypothermia

1. Operating room temperature.
2. Evaporative, conductive, convective, radiant losses.
3. Prolonged surgery or anaesthesia greater than three hours.
4. Inhibition of thermoregulatory mechanisms:
   (i) vasodilatation,
   (ii) muscle relaxation,
   (iii) depressed metabolism,
   (iv) prolonged heat loss greater than heat production.
5. Surgical causation
   (i) open abdominal or thoracic surgery,
   (ii) laparoscopic surgery greater than three hours,
   (iii) vascular surgery.
6. Blood loss.
7. High potential conductive loss - correlates with degree of exposure.
8. Excluded body tissues from normal circulation.

9. Intentional hypothermia:

   (i) cardiopulmonary bypass,

   (ii) cold cardioplegic solutions,

   (iii) hepatic surgery,

   (iv) neurosurgery.

1.4.2.2 Risk Factors for Hypothermia in Trauma Victims

Clinton[9] reviewed hypothermia in the trauma victim, especially the postoperative trauma victim. The review sought to increase awareness of the aetiology and consequences of trauma related hypothermia and to encourage caregivers to improve the care of trauma victims in this regard. The aetiology of hypothermia following traumatic injury depended on:

1. Severity of injury.
2. Type of injury.
3. Environmental exposure.
4. Patient related factors such as:
   (i) extremes of age,
   (ii) nutritional state,
   (iii) body mass.
5. Resuscitation related factors.
6. Surgery related factors such as:
   (i) preparation procedures,
   (ii) site of surgery,
   (iii) duration of surgery.
7. Anaesthesia related factors:
   (i) general anaesthesia
   (ii) regional anaesthesia
   (iii) use of specific drugs

1.4.3 Postoperative hypothermia

1.4.3.1 Incidence of postoperative hypothermia

Slotman *et al.*[10] investigated the effects of operative and postoperative hypothermia on post surgical mortality and morbidity. They reviewed a hundred consecutive patients admitted to a surgical intensive care unit following elective and emergency surgery, and found a 74 per cent incidence of hypothermia. Postoperative hypothermia at time zero was not significantly associated with mortality. For patients who remained hypothermic at 2 hours, mortality was significantly increased compared to that of normothermic patients (7 of 29 patients died versus 2 of 45 patients, $p < 0.01$). Mortality was further significantly increased in patients who remained hypothermic at 4 and 8 hours. When patients with intra-operative hypotension were excluded, mortality in the presence of hypothermia at 4 hours remained significantly increased (4 of 10 hypothermic patients died versus 2 of 54 normothermic patients, $p < 0.01$). Patients over 55 years of age were more likely to be hypothermic than younger patients.

1.4.3.2 Consequences of Hypothermia

Hypothermia has both beneficial and deleterious effects depending on the circumstances under which it arises:

(1) Intraoperative temperatures only 2 to 3°C below normal provide significant protection against cerebral ischaemia and hypoxaemia.

(2) Mild hypothermia provides some protection against malignant hyperthermia.

(3) Mild hypothermia decreases the metabolism of certain drugs.

(4) Temperatures below 33°C reversibly increase the prothrombin index and partial thromboplastin time.

(5) Central hypothermia and peripheral cutaneous vasoconstriction are associated with postanaesthetic muscular activity.[11]

(6) Bleeding time is prolonged by local cutaneous hypothermia but is not directly altered by central temperature changes. Schmied *et al.*[12] found that, *in vitro*, platelet function and the coagulation cascade are impaired by hypothermia. Since the extent to which peri-operative hypothermia influences bleeding during surgery was not known, they tested a hypothesis that mild hypothermia increased blood loss and allogenic transfusion requirements during hip arthroplasty. Blood loss and transfusional requirements were evaluated in sixty patients undergoing unilateral total hip arthroplasties. Patients were divided into two groups on the basis of the final intra-operative core temperature, a normothermic group (36.6 ± 0.4°C) or a hypothermic group (35 ± 0.5°C). Crystalloid, colloid, scavenged red cells and allogenic blood were administered according to a strict protocol. The findings were that intra- and postoperative blood loss was significantly greater in the hypothermic group (2.2 ± 0.5 litres versus 1.7 ± 0.3 litres, $p < 0.001$).

(7) Myocardial ischaemia:

Frank *et al.*[13], found that hypothermia occurs commonly during surgery and can be associated with increased metabolic demands during rewarming in the postoperative period. Although cardiac complications remain the leading cause of morbidity after anaesthesia and surgery the relationship between unintentional hypothermia and myocardial ischaemia during the peri-operative period had not been studied. Frank studied 100 patients undergoing lower extremity vascular reconstruction who received continuous halter monitoring throughout the first twenty-four postoperative hours. Myocardial ischaemia was determined by cardiologists blind to clinical variables.

The patients sublingual temperature on arrival at the intensive care unit immediately after surgery was used to divide the patients into two groups: a hypothermic group (temperature less than 35°C; n = 33) and a normothermic group (temperature greater than or equal to 35°C; n = 67). The relationship between unintentional hypothermia and myocardial ischaemia occurring during the first 24 hours was evaluated by univariate and multivariate analyses. Results indicated that a greater percentage of patients showed electrographic changes consistent with myocardial ischaemia in the hypothermic group (36%, 12 of the 33) than in the normothermic group (13% 9 of 67, p = 0.008). Pre-operative risk factors for peri-operative cardiac morbidity was similar for the two groups. The mean age was 70 ± 2 years and 62 ± 1 year in the hypothermic and normothermic groups respectively (p = 0.001). When subgroup and multivariate analyses were used to adjust for differences in age, temperature remained an independent predictor of ischaemia (odds ratio 1.82 per °C; 95% confidence interval, 1.09 - 3.02). Similarly, the incidence of postoperative angina and arterial oxygen partial pressure (PaO_2) < 80 mmHg was greater in the hypothermic group than in the normothermic group. Frank concluded that unintentional hypothermia is associated with myocardial ischaemia, angina and a PaO_2 < 80 mmHg during the early postoperative period in patients undergoing lower extremity vascular surgery.

(8) Increased morbidity and mortality:

Bush *et al.*[14] undertook a retrospective review of 262 patients undergoing elective abdominal aortic aneurysmectomy to test the hypothesis that postoperative hypothermia (core temperature less than 34.5°C) is associated with increased morbidity and excess mortality rates. Outcome measures included length of stay in the

intensive care unit and in-hospital mortality. Analysis showed that, other than a higher incidence of hypothermia in women ($p < 0.05$), pre-operative risk factors were similar in hypothermic and normothermic patients. After operation patients with hypothermia had significantly greater Apache II scores ($p < 0.001$) and hypothermic non-survivors took significantly longer to rewarm ($p < 0.005$). Patients with hypothermia had significantly greater fluid ($p < 0.005$), transfusion ($p < 0.001$) vasopressor ($p < 0.05$) and inotrope ($p < 0.05$) requirements, and a significantly higher incidence of organ dysfunction (53% vs 28.7%) and death (12.1% vs 1.5%), intensive care unit stay and in-hospital stay was markedly prolonged in hypothermic patients. Multivariate analysis showed that female gender ($P=0.004$) was the only predictor of intra-operative hypothermia, whereas pre-operative hypothermia was significantly predictive of prolonged postoperative hypothermia. Development of organ failure and acute myocardial infarction were independent predictors of death. Bush concluced that, after aortic aneurysmectomy, patients with hypothermia have multiple physiologic derangements associated with adverse outcomes and that although multiple aetiological factors were present, body temperature was a variable that should be controlled during aortic surgery.

(9) Shivering:

Shivering becomes the most obvious mechanism of heat gain as the patient emerges from anaesthesia. As it increases in intensity it progresses from masseter spasm to involvement of the head and neck, upper trunk and finally the whole body with chattering of the teeth. Severe shivering is associated with a rapid rise in temperature and abrupt increases carbon dioxide production and oxygen consumption. If minute ventilation is not increased, acute respiratory acidosis results. If cardiac output does not increase, mixed venous

oxygen saturation decreases leading to hypoxaemia and, possibly, myocardial ischaemia. During mechanical ventilation shivering interferes with ventilation and uniform gas flow. Transmission of intra-pleural pressures spuriously elevates central venous and pulmonary artery pressures. Excessive shivering has the potential to disrupt wounds, promote bleeding, increase intracranial and intraocular pressure and cause patient injury. In awake patients it leads to marked discomfort.

Giffre *et al*[15] compared forced-air convection warmers to warmed cotton blankets and infrared radiant lamps. Shivering and non-shivering patients were observed separately. Shivering patients rewarmed faster than non-shivering patients.

Mort *et al.*[16] found that shivering in the period immediately following cardiac surgery can lead to detrimental haemodynamic changes. Shivering increases the metabolic demand for oxygen and is poorly tolerated in this physiologically compromised population. Increases in core temperature and decreases in skin temperature occur as a result of peripheral-to-central redistribution of heat, shivering and non-shivering thermogenesis. The Bair-Hugger® forced-air convective warming system was evaluated for its ability to limit post cardiac surgery shivering, and to decrease the peripheral-to-central temperature difference. Forty-nine patients undergoing valve replacement and/or coronary artery bypass graft were randomly assigned to be warmed following surgery with either forced-air or warmed cotton blankets. Measurements of central and peripheral temperatures were collected every 20 minutes over 5.5 hours. Electromyograph recordings of pectoral biceps and quadriceps muscle groups were conducted to verify shivering. A visual observation

shivering score ranging from 0-4 where four was continuous shivering were recorded for each study period. Patients who received relaxants following cardio-pulmonary bypass were excluded from the analysis. Overall, 19 of the 25 cotton blanket patients and 8 of the forced-air patients displayed shivering. Three patients, one from the cotton blanket group and two from the forced-air group were shivering upon return to the intensive care unit and were excluded from further shivering assessment. Vigorous shivering (Visual analysis score of 3 or 4) was observed in 17 of the cotton blanket group and 3 of the forced-air group ($p < 0.001$). Total periods of shivering during 5.5 hours were 67 minutes for the cotton blanket group and 12 minutes for the forced-air group ($p < 0.001$). Pharmacological intervention with muscle relaxants to control shivering occurred 11 times in the cotton blanket group but only once in the forced-air group. No difference was noted between pulmonary artery catheter, oesophageal, rectal or nasopharyngeal temperatures in the two groups. Forced-air warming resulted in significant ($p = 0.001$) increases in shoulder, palm, thumb, thigh, ankle and toe temperatures. The authors concluded that forced-air warming therapy was beneficial in limiting the incidence, magnitude and duration of shivering when used in a prophylactic manner to rewarm patients following hypothermic cardiopulmonary bypass.

Shivering-like tremor occurs in about 40 per cent of patients recovering from general anaesthesia and is preceded by central hypothermia and peripheral vasoconstriction indicating that it is thermoregulatory. Peri-anaesthetic tremor increases oxygen consumption by about 200 per cent and exacerbates postoperative pain. Although most forms of peri-anaesthetic tremor resemble normal shivering, electromyographically, pathological clonic/tonic patterns are seen. The tremor can be treated by skin surface

warming, intravenous clonidine or intravenous meperidine administration. Why meperidine is far more effective than equipotent doses of other opiates remains unknown. Tremor during epidural anaesthesia is normal thermoregulatory shivering - triggered by central hypothermia and preceded by vasoconstriction above the level of sympathetic blockade. It can be prevented by skin surface warming above the level of the blockade, intravenous or epidural meperidine, intravenous or epidural sufentanil administration.[17]

(10) Decreased metabolism

(11) Reversible platelet dysfunction,

(12) Inhibition of immune function,

(13) Hypokalaemia,

(14) Increased nitrogen loss,

(15) Acid-base disturbance,

(16) Hyperglycaemia,

(17) Hypertension followed by hypotension,

(18) Bradycardia.

1.4.3.3 Clinical consequences of hypothermia

1.4.3.3.1 Organ System Dysfunction

Cardiovascular system

Thermoregulatory and cardiovascular homeostasis are closely related. Initially, hypothermia-induced elevation of serum catecholamines increases the heart rate and promotes peripheral vasoconstriction. Consequently, venous volume, cardiac output, systemic vascular resistance and mean arterial pressure all increase. In progressive hypothermia reflex bradycardia and myocardial depression lead to a decrease in cardiac output and mean arterial pressure. At 30°C, cardiac output is 30-40 per cent below normal. " J " waves (acute elevation of the ST segment) appear on the electrocardiogram at temperatures between 32 and 33°C and are pathognomonic of hypothermia. Premature atrial contractions and atrial fibrillation occur as a result of atrial distension. Ventricular irritability deteriorating to ventricular fibrillation becomes common by 30°C with increasing incidence at temperatures less than 28°C. Asystole occurs at temperatures less than 15°C. Cardiac irritability may occur early in the presence of pre-existing cardiac disease. Cardioversion is unsuccessful at temperatures less than 30°C. Cardiac drugs vary in efficacy with hypothermia and bradycardia is often resistant to intravenous atropine. Bretylium tosylate may suppress cold cardiac irritability and beta-blocker therapy may improve survival after hypothermia but human studies are preliminary. There is an increase in systemic vascular resistance due to vasoconstriction and increased blood viscosity.

Coagulation

Hypothermia as an independent factor exacerbates blood loss following major trauma by impairing the coagulation cascade, at temperatures less than 34°C. Thrombocytopaenia is caused by reversible platelet sequestration in the liver and hepatic portal circulation. Hypothermia also promotes reversible fibrinolysis because of increased levels of fibrin degradation products. Disseminated intravascular coagulation from peripheral microvascular failure may occur and may be a secondary cause of thrombocytopaenia.

Gastro-Intestinal System

Gastro-intestinal motility decreases at temperatures less than 24°C with ileus being common. Wischnevsky ulcers are common throughout the colon. Acute fulminant haemorrhagic pancreatitis may occur particularly after rewarming. Hepatic microsomal enzymes are depressed by hypothermia and, as a consequence, there is impaired drug metabolism.

Haematological System

Increased blood viscosity from haemo-concentration occurs because of water losses from extravascular fluid redistribution and cold diuresis. Hypothermia produces a decrease in metabolic rate which is directly related to temperature. Oxygen has a greater affinity for haemoglobin (the oxyhaemoglobin dissociation curve shifts to the left) and oxygen extraction is impaired leading to a tissue oxygen debt.

Metabolism

The basal metabolic rate decreases 7% per 1°C decrease in core temperature. Anabolic metabolism accompanies progressive hypothermia, leading to metabolic acidosis. Serum sodium levels decrease while potassium levels increase because of the disruption of the cell membrane sodium-potassium pump. Hyperglycaemia caused by stress-driven gluconeogenesis, depressed insulin release and impaired insulin uptake at peripheral effector sites is [illegible] early in hypothermia and is often accompanied by a mild ketosis.

Neurological System

Progressive hypothermia results in progressive degrees of somnolence and confusion leading to cold narcosis at 28-30°C. Coma is rare above 28°C. Low grade hypothermia has a protective effect against cerebral ischaemia because brain oxygen requirements are decreased more than the decrease in cerebral blood flow favouring oxygen availability. Cerebral blood flow decreases 6-7% for each 1°C drop in core temperature. Recent studies indicate that decreases in cerebral oxygen extraction are independent of the left shifted oxyhaemoglobin dissociation curve during hypothermia. Pupillary and deep tendon reflexes are lost at a core temperature of 25°C. At temperatures less than 18°C the electroencephalogram is usually isoelectric. There is delayed emergence from anaesthesia.

Muscular System

Compensatory active thermogenesis (shivering) and increased muscle tone accompany early to moderate hypothermia at temperatures of 36-32°C. At temperatures less than 30°C compensatory mechanisms are absent and muscle tone is increased. Neuromuscular transmission is impaired, and the neuromuscular junction may be resistant to non-depolarising relaxant drugs.

Renal System

A "cold diuresis" secondary to peripheral vasoconstriction resulting in increased central venous volume and an increased glomerular filtration rate occurs at temperatures of 33-34°C. With further hypothermia renal blood flow decreases progressively resulting in decreased renal perfusion, decreased glomerular filtration rate, oliguria and azotemia. Glomerular filtration rate decreases to 60 per cent of normal at a temperature of 25°C. Acute tubular necrosis is a common finding in victims of fatal hypothermia. Hypothermia may also lead to impaired sodium re-absorption by the renal tubule.

Drug Metabolism

There may also be impaired drug metabolism. Muscle relaxants are metabolised more slowly. Vecuronium doubles its duration of action at 34°C which may be due to delayed metabolism or a pharmacodynamic effect of hypothermia.[7]

Respiratory System

Early hypothermia is associated with hyperventilation. Progressive hypothermia leads to respiratory depression. Respiratory drive may persist at temperatures less than 25°C but anatomical dead space is increased by 50 per cent and physiologic dead space is increased by 28 per cent. Loss of respiratory drive with apnoea occurs at temperatures less than 24°C. There is a diminished ventilatory response to hypoxaemia and hypercarbia. Since there is decreased carbon dioxide production, mechanical hyperventilation produces a respiratory alkalosis.

Wound Infection

Hypothermia inhibits immune function (especially macrophage phagocytosis) and decreases cutaneous blood flow. Kurdz *et al.*[18] showed that mild peri-operative hypothermia may promote surgical wound infection by triggering thermoregulatory vasoconstriction which decreases the subcutaneous oxygen tension. Reduced levels of oxygen in tissues impairs oxidative killing by neutrophils and decreases the strength of the healing wound by reducing the deposition of collagen. Kurdz tested the hypothesis that hypothermia increased susceptibility to surgical wound infection and lengthened hospitalisation. Two hundred patients under-going colorectal surgery were randomly assigned to routine intra-operative thermal care (the hypothermic group) or additional warming (the normothermic group). In a double blind protocol surgical wounds were evaluated daily until patients were discharged from the hospital and then once at two weeks post discharge. Wounds containing culture positive pus were considered infected. Surgical wound infections were found in 18 of 96 patients assigned to routine intra-operative thermal care group but in only 6 of 104 patients assigned to additional warming group ($p = 0,009$). Sutures were removed one day later in the hypothermic group compared to those in the normothermic group ($p = 0.002$). Duration of hospitalisation was approximately 2.6 days longer in the hypothermic group, ($p = 0.01$). Kurdz concluded that hypothermia itself may delay wound healing in patients predisposed to wound infections and that maintaining normothermia intra-operatively was more likely to decrease the incidence of infectious complications in patients undergoing colorectal resection and to shorten their hospitalisation.

1.4.3.3.2 Effect of Hypothermia on mixed venous oxygen saturation

Viale *et al.*[19] found that during muscular exercise there was a negative correlation between the value of mixed venous oxygen saturation (SvO_2) and the level of muscular work. Since the immediate postoperative period is associated with an increase in whole body oxygen demand and in this regard resembles the effects of muscular exercise, a similar correlation might be seen during this period. Viale then investigated postoperative oxygen consumption in patients undergoing coronary artery bypass surgery. The author concluded, from his results, that during the first two postoperative hours after coronary artery bypass surgery VO_2 rarely exceeds 50 per cent of preoperative VO_2 max. Assuming a stable state of myocardial function, SvO_2 measurement may provide an indirect means of an assessment of the exercise test imposed on patients recovering from general anaesthesia.

1.4.3.3.3 Effect of Hypothermia on duration of stay in Post-Anaesthesia Care Unit

To identify and quantify complications occurring in the Post-Anaesthesia Care Unit (PACU), a prospective study by Hines *et al.*[20] evaluated 18 473 consecutive patients entering a PACU at a University teaching hospital using a standardized data collection form. For patients admitted with a core temperature of less than 35°C the duration of the PACU stay was 152 ± 46 minutes compared to 116 ± 65 minutes for patients with a temperature greater than or equal to 36°C ($p < 0.01$).

1.5. Prevention and Treatment of Hypothermia

The initial rapid decrease in core temperature that occurs during epidural or general anaesthesia is difficult to prevent because it results largely from internal redistribution of heat. It can be reduced by warming the skin surface and peripheral tissues before induction of anaesthesia. Such therapy decreases the central-peripheral temperature gradient, however, given that the heat capacity of the peripheral thermal compartment is 150 kilocalories, 1 to 2 hours of vigorous warming is generally required. Less than 10% of metabolic heat is lost by respiration even if patients are ventilated with dry cool gas. Passive airway humidification using heat and moisture exchangers can prevent most of this loss and active gas heating and humidification eliminates all respiratory metabolic heat loss. Heat and moisture exchangers, however, contribute little to maintaining normothermia. Since most of the heat lost is through the skin, although evaporation from large surgical incisions may contribute significantly, simply preventing cutaneous heat loss will prevent the slow linear decline in central temperature that is seen in the anaesthetised patient. Cutaneous heat loss can be passively decreased by covering the skin with cloth or paper surgical drapes, blankets, plastic bags. A single layer of each insulator reduces heat loss by about 30 per cent but the efficacy of different cover types is generally similar. Because cutaneous heat loss is roughly proportional to surface area (heat loss from the head is not disproportionately high in adults) the choice of covering material is far less important than the total surface area covered. Active warming can prevent most cutaneous heat loss and can even reverse the net transfer of heat across the skin. Infrared radiators are less effective than other active warmers because they do not prevent convective loss and much of the skin surface is not parallel to the radiating surface. Radiation is likely to be more effective in infants than adults because the ratio of warming surface size to body size is larger. Circulating water blankets are more effective when placed over rather than under patients because surface contact between skin

and blanket is increased. Usually several blankets can be attached to a single heater permitting use of a full length blanket under a patient and a simultaneous use of a paediatric sized blanket over the chest or legs. Water temperature should not exceed 40°C to minimise the risk of pressure or heat necrosis, and should be set at even lower temperatures in patients with arterial vascular insufficiency.

1.5.1 Basic ..nagement Principles for Hypothermia

1.5.1.1 ... sive rewarming

(i) removal of environmental causes,

(ii) use of a forced-air convective warming device

1.5.1.2 Active external rewarming (for mild to moderate hypothermia)

(i) immersion in heated water up to 40-45°C,

(ii) heated intravenous solutions to 37°C,

(iii) electric blankets at 37°C.

1.5.1.3 Active Core Rewarming (for moderate to severe hypothermia)

This employs an intra-gastric balloon with heated irrigation, heated blood irrigation, haemodialysis or peritoneal dialysis with warmed dialysate, extra-corporeal blood warming or inhalation rewarming with warm gases (less than 43°C). Active rewarming is associated with complications and may have a higher associated morta ty rate compared to passive techniques.

non-shivering patients were observed separately. Shivering patients rewarmed faster than non-shivering patients.

Mort *et al.*[4] found that shivering in the period immediately following cardiac surgery can lead to detrimental haemodynamic changes. Shivering increases the metabolic demand for oxygen and is poorly tolerated in this physiologically compromised population. Increases in central (core) and decreases in peripheral (skin) temperatures occur as a result of peripheral to central redistribution of heat, shivering and non-shivering thermogenesis. The Bair-Hugger® convective warming system was evaluated for its ability to limit post cardiac surgery shivering, and to decrease the peripheral-to-central temperature difference. Forty-nine patients undergoing valve replacement and/or coronary artery bypass graft were randomly assigned to be warmed following surgery with either Bair-Hugger® or warmed cotton blankets. Measurements of central and peripheral temperatures were collected every 20 minutes over 5.5 hours. Electromyograph recordings of pectoral biceps and quadriceps muscle groups were conducted to verify shivering. A visual observation shivering score ranging from 0-4 where four was continuous shivering were recorded for each study period. The patients who received relaxants following cardio pulmonary bypass were excluded from the analysis, a train of four was evaluated in participants to ensure lack of clinical motor blockade. Overall 19 of the 25 cotton blanket patients and 8 of the Bair-Hugger® patients displayed shivering

hypothermia caused by epidural block. These results support the hypothesis that redistribution of heat within the body, not heat loss, is the most important aetiology of hypothermia from epidural anaesthesia.

1.5.2.2 Intraoperative warming

There are various methods available to prevent intra-operative hypothermia such as the use of warming blankets, forced-air devices, space blankets, humidification and warming of inspired gases, warming of intravenous fluid and radiant heat lamps - all of which have been used with varying degrees of success.

Canuc *et al.*[23] investigated the ability of the forced-air convection warmer (Bair-Hugger®) to transfer heat in awake subjects by means of heat flux measurement. During abdominal surgery only the lower limbs are available for skin surface warming. The amount of heat transferred under that particular surgical situation was evaluated. Sixteen adult patients undergoing non-haemorrhagic abdominal surgery were studied. Anaesthesia was induced with thiopentone, fentanyl, enflurane and nitrous oxide in oxygen. Operating room temperature was maintained at 21-22°C and patients were randomly divided into two groups. Eight patients received no additional prevention of hypothermia (control group), the other eight patients had their legs covered by a lower body cover attached to a Bair-Hugger® Model 500 forced-air convection warmer. Core temperature and skin temperature (thorax, arm, midcalf, midthigh) were recorded at 15 minute intervals. Changes in body heat content and mean skin temperature of lower and upper parts of the body were calculated as the average of the thigh and calf temperatures (lower temperature) and from the thorax and arm temperatures (upper temperatures). From the results, the investigators concluded that the Bair-Hugger® was able to provide heat gain in

anaesthetised patients undergoing abdominal surgery even when the warmed skin surface is restricted to the lower limbs.

Gauthier[24] investigated the use of a forced-air warming device in 13 patients undergoing spinal surgery. Patients were anaesthetised using a balanced anaesthetic technique of narcotics, low dose of enflurane and induced hypotension. Core temperature was monitored by an oesophageal temperature probe. A control group of six patients had temperature conservation by warming fluids and inspired gases. The second group of patients were managed fluid and inspired gas warming and with a forced-air warming device, the blanket being applied only to the lower body . Patient core temperature was recorded intraoperatively and on admission to the postoperative care unit as was the duration of stay in the unit. Patients in the control group underwent procedures lasting an average of 290 minutes and had average estimated blood losses of 850 ml. This group showed a range of temperatures from 35.9°C to 35.3°C during the procedure with a net loss of 0.2°C . The average admission temperature on admission to the postoperative care unit was 35.6°C and the average stay was 150 minutes. Patients in the forced-air warming group underwent procedures lasting an average of 313 minutes and had estimated blood losses of 1135 ml. This group had a core temperature ranging from 36.1°C to 37°C with a net gain of 0.6°C. The average postoperative care unit stay was 120 minutes. Temperature gains and the postoperative stay were significantly different when analysed statistically. Traditional management of intra-operative hypothermia has been directed towards the prevention of losses. This data suggested that the use of forced-air warming not only prevented heat loss but may actually result in heat gain during the intraoperative period. In this study the forced-air group had longer procedures, lost more blood, yet were warmer at the end of the procedure and had significantly shorter stays.

Hynson and Sessler[25] compared various warming devices. With Institutional Review Board approval they randomly assigned 12 patients undergoing kidney transplantation to one of four groups. Patients in Group 1 were treated with a circulating water mattress, placed under the subject . Patients in Group 2 were treated with heated humidification of inspired gas, the heater being set at 40°C . Patients in Group 3 were treated with forced-air warming using a lower body cover. Air temperature was set at 43°C . Patients in Group 4 received no specific warming treatment. In all groups intravenous fluids were warmed and fresh gas flows were maintained without a heat and moisture exchanger. Operating room temperatures and preoperative conditions were controlled. Tympanic membrane temperature was measured starting 30 minutes before induction of anaesthesia. During the first 30 minutes of anaesthesia each group averaged a 0.7°C decline in core temperature, after 60 minutes patients in Group 3 evidenced a rise in core temperature. Patients in Group 1 continued to show a slow decrease in core temperature for the next 150 minutes of anaesthesia. Patients in Groups 2 and 4 experienced a rapid decline in core temperature for the duration of anaesthesia. The study concluded that forced-air warming is more effective than the water circulating mattress and that heated humidification of inspired gas offered little protection from intraoperative hypothermia.

1.5.2.3 Postoperative Warming

Lennon *et al.*[26] randomised thirty adult surgical patients admitted to the recovery room with an oral temperature of less than or equal to 35°C into two groups. Group 1 patients were covered with cotton blankets warmed to 37°C and Group 2 patients were treated with a forced-air warming system. Mean oral temperature on admission to the recovery room was the same in both groups (34.3°C). Oral temperature and the presence or

absence of shivering were recorded at 15 minute intervals after application of the selected warming method, patients in Group 2 were warmer at all time intervals, mean temperatures on the forced-air heating group and in Group 1 were respectively 34.8°C and 34.3°C ($p < 0.05$) at 15 minutes; 35°C and 34.2°C ($p > 0.01$) at 30 minutes; 35.2°C and 34.5°C ($p < 0.05$) at 45 minutes; 35.8°C and 34.7°C ($p < 0.001$) at 60 minutes; 36°C and 35°C ($p < 0.01$) at 75 minutes; 36°C and 35°C ($p < 0.01$) at 90 minutes The incidence of shivering was significantly greater in group 1 at 15 and 45 minutes. In addition, time spent in the recovery room was significantly greater in Group 1 than it was in Group 2, 156 minutes versus 99.7 minutes ($p < 0.03$).

Postoperative studies were also performed by Summers *et al.*[27]. Patients who were warmed with a forced-air convective warming device therapy had a higher surface temperature, haemoglobin oxygen saturation and perceived comfort than patients with bath blankets. The authors concluded that forced-air warming was an effective method to promote postoperative thermal comfort and physiological stability. In addition, they concluded that the use of forced-air warmers was a convenient adjunct to care by postanaesthetic care unit nurses.

Giuffre *et al.*[15] conducted a study in a PACU, in which the efficiency of a forced-air warming device was compared to warmed cotton blankets and infrared radiant lamps. Shivering and non-shivering patients were observed separately. Shivering patients rewarmed from surgery faster than non-shivering patients. Patients who received forced-air warming in both shivering and non-shivering groups warmed faster. Forced-air warming therapy reduced the length of stay in the PACU by 77 minutes in the non-shivering patients. Patients who were warmed by alternate methods spent approximately 60% more time in the PACU.

1.5.2.4 Comparison of forced-air convection warming devices in human volunteers.

Diesbrechtph and Ducharme[28] found that peri-anaesthetic hypothermia is common and produces several complications including postoperative shivering, decreased drug metabolism and clearance and impaired wound healing. Forced-air warming transfers more than 50 watts to the body and is an efficient method for either preserving or reversing decreases in core temperature. The authors compared the efficiency of four complete forced-air warming systems, namely:

(a) The Bair-Hugger® 250 power unit together with the Bair Hugger® 300 warming cover (Augustine Medical).

(b) The ThermoCare® TC1000 power unit together with the TC1050 ComfortQuilt® (Gaymar Industries).

(c) The WarmAir® 130 power unit together with the WarmAir® 140 warming tube (Cincinnati Sub-0 product).

(d) The WarmTouch® 5000 power unit together with the CareQuilt® [with connecting hose compressed (short) and extended (long)] (Mallinckrodt Materials).

Six minimally clothed male volunteers were studied supine in a 24.5°C environment. Cutaneous heat flux and skin temperature were measured at 14 area weighted sites using thermal flux transducers. After a 20 minute control periods the volunteers were warmed for 40 minutes with each device. A cotton blanket was placed over each warming cover. All units reached maximum efficiency within 20 minutes. Total heat transfer achieved was as follows:

Bair-Hugger®:	95 ± 7 watts *
WarmTouch® (short hose):	81 ± 6 watts
WarmTouch® (long hose):	68 ± 8 watts
ThermoAir®:	61 ± 5 watts
WarmAir®:	38 ± 6 watts

* = significant difference

Each cover also was tested on a common power unit, the Bair-Hugger® 200. Total heat transferred was statistically significantly better with the Bair-Hugger® 300 warming cover (88 ± 8 watts) than with the ComfortQuilt® (56 ± 6 watts), CareQuilt® (50 ± 7 watts) and the WarmAir® 140 warming tube (43 ± 6 watts).

1.6. Consequences of Postoperative Rewarming

1.6.1 Vasodilatation

Postoperative hypothermia induces vasoconstriction which creates a false impression of the adequacy of patients' intravascular volume status. After 1 to 3 hours, rewarming causes vasodilatation which unmasks underlying hypovolaemia which may then be associated with abrupt hypotension, tachycardia, oliguria and, on occasion, myocardial ischaemia.

2. MOTIVATION FOR A FURTHER STUDY ON THE EFFICACY OF THE FORCED-AIR CONVECTIVE WARMING DEVICE IN HYPOTHERMIC POSTOPERATIVE PATIENTS.

The forced-air convective warmer appears to be a valuable option for the thermal care of the hypothermic patient. This device has been shown to be effective in preventing the intraoperative development of hypothermia when used preoperatively and/or intraoperatively in patients undergoing general anaesthesia.[25, 29, 30] Only a few studies have addressed the use of forced-air warming in the immediate postoperative period[16, 26]. It is not clear whether the patients in those studies received intraoperative thermal management or not. Thus, it would be of value to assess the capability of the device to warm hypothermic postoperative patients who are know not to have received any attempts at intraoperative warming. Therefore, this study was designed to investigate the postoperative use of a forced-air convective warmer in hypothermic postoperative patients who had not received any attempts at intraoperative thermal management.

3. MATERIALS AND METHODS

The study was commenced when approval by the ethics committee of the University of the Witwatersrand had been obtained. Informed consent was obtained from all patients.

Twenty consecutive postoperative patients of both sexes and aged between 18 and 85 years who had not received any active form of intraoperative thermal management and had a rectal temperature of 35.9°C or less on admission to an intensive care unit were enrolled into the study. Although the patients had undergone various different operations (see Table III) all patients had received general anaesthesia only and no attempt by the investigators was made to control the conduct of anaesthesia. Postoperative analgesia for all patients consisted of intravenous morphine given either by nurse administration or by a patient controlled analgesia device delivering intravenous morphine. The patients were randomised into two groups, the control group (10 patients) who were covered from the neck down with two standard hospital cotton blankets and no active warming therapy, and the warmed group (10 patients). Both groups were studied for a period of three hours post operation. Patients in the warmed group were completely covered again from the neck down with a full length blanket connected to the heat source of a forced-air convective warmer ("WarmTouch®", Trigate, Randburg) set to deliver air warmed to a temperature of 42-46°C. The complete unit consists of a two layer paper plastic blanket placed directly over the patient which is in turn connected to the air hose of a heat source. When functioning the heat source forces warmed air between the two layers of the blanket, the patient surface of which contains multiple small perforations. As a result warm air is directed onto the skin surface of the entire body area covered by the blanket.

Temperature probes and self adhesive skin surface temperature sensors (Sher-i-Temp, Brittan Health Care, Johannesburg) were placed in the rectum

and applied to the right thumb, the ventral surface of the right forearm and the orehead, respectively. All sensors were connected to a battery operated temperature display module (Sonatemp, Brittan Health Care, Johannesburg). All temperature values were recorded at 0, 15, 30, 45, 60, 90, 120, 150 and 180 minutes. Environmental temperature in the intensive care unit is controlled between 22 to 24°C.

The presence or absence of shivering during the first three fifteen minute periods of the study was noted by the investigator. However, by the nature of the study design the investigator was not blind to the fact that a warming device was being utilised.

Differences between patient age and gender were analysed using Students Unpaired T-Test and Fisher's Exact Test respectively. The changes in rectal, forehead, forearm and thumb temperatures as a function of time were analysed for the two groups. Temperature differences between the groups at each time interval were evaluated using Students unpaired T-test. The incidence of shivering at each interval was determined by utilising Fisher's exact test. A p-value of less than 0.05 was considered statistically significant.

Table III: Surgical Procedures performed in the Control Group and the Warmed Group.

Control Group		Warmed Group	
Patient No.	Operative Procedure	Patient No.	Operative Procedure
1	Aortic aneurysmectomy	1	Cholecystdocho-duodenectomy
2	Aortic aneurysmectomy	2	Cholecystdocho-duodenectomy
3	Aorto-bifemoral bypass graft	3	Pancreatic exploration
4	Aorto-bi-iliac bypass graft	4	Sigmoid Colectomy
5	Pancreatic exploration	5	Cholecystectomy
6	Gastrectomy	6	Pneumonectomy
7	Common bile duct exploration	7	Removal of frontal sinus tumour
8	Cholecystdocho-duodenectomy	8	Femora-profunda bypass graft (Left)
9	Gastric fundoplication	9	Defunctioning colostomy
10	Nephrectomy (Left)	10	Nephrectomy (Right)

4. RESULTS

With regard to age, gender and the various admission temperatures there was no difference between the two groups (Table IV). The two groups showed various differences in their thermal state within 15 minutes of commencing the study. The temperature changes that took place during the study are detailed in Table V. With respect to rectal temperature, a measure of body core temperature, the warmed group exhibited higher temperatures than the control group at all time intervals from 15 minutes onwards. However, these differences only became statistically significant from 2 hours into the study (Fig. 1). Mean temperatures for warmed and controlled group at 120, 150 and 180 minutes were 36.8 and 36.2, 37.0 and 36.4, 37.5 and 36.6°C respectively. Those results indicate that core rewarming takes place as a consequence of forced-air warming but the time required to reach a significant difference illustrates that there is a delay before heat is transferred from peripheral to core compartments in hypothermic postoperative patients. A possible explanation for this delay is the presence of hypothermia-associated peripheral vasoconstriction.

TABLE IV : Patient Characteristics

	Control Group	Warmed Group	Significance
Age (years)	57.7 (13.7)	66.6 (13.7)	NS
Gender (M : F)	8 : 2	5:5	NS
Admission Temperature (°C)			
Rectal	35.2 (0.4)	35.2 (0.6)	NS
Forehead	30.6 (1.3	31.1 (1.3)	NS
Forearm	31.8 (1.5)	31.3 (1.4)	NS
Thumb	27.9 (2.1)	28.6 (1.9)	NS

M : F = Male : Female

NS = Not significant

Figures in parentheses indicate 1 Standard Deviation

TABLE V: Temperature values obtained from the various anatomical sites in the Control and Warmed Groups following recovery from anaesthesia

Source	Time (minutes)	Temperature (°C) [Mean (±1SD)]	
		Control Group	Warmed Group
Rectum	0	35.2 (0.4)	35.2 (0.6)
	15	35.2 (0.5)	35.3 (0.6)
	30	35.3 (0.6)	35.5 (0.5)
	45	35.5 (0.6)	35.8 (0.4)
	60	36.7 (0.5)	36.1 (0.4)
	90	36.0 (0.5)	36.4 (0.5)
	120	36.2 (0.6)	36.8 (0.5)*
	150	36.4 (0.7)	37.0 (0.7)*
	180	36.6 (0.7)	37.5 (0.8)*
Forearm	0	31.8 (1.5)	31.2 (1.4)
	15	32.1 (1.2)	33.9 (1.0)*
	30	32.3 (1.5)	34.9 (1.3)*
	45	32.5 (1.4)	35.1 (1.2)*
	60	32.8 (1.4)	34.9 (1.1)*
	90	33.0 (1.3)	35.2 (1.0)*
	120	33.2 (1.5)	35.5 (1.1)*
	150	33.5 (1.9)	35.8 (0.9)*
	180	33.9 (1.8)	36.1 (1.0)*
Thumb	0	27.9 (2.0)	28.6 (1.9)
	15	28.8 (3.2)	31.9 (1.7)*
	30	29.4 (2.7)	34.0 (1.8)*
	45	29.8 (2.8)	34.3 (1.6)*
	60	29.9 (2.9)	34.5 (1.5)*
	90	29.8 (3.2)	34.2 (1.5)*
	120	30.0 (3.5)	34.7 (1.3)*
	150	30.1 (3.6)	35.2 (1.1)*
	180	30.5 (3.7)	35.7 (0.8)*
Forehead	0	30.6 (1.5)	31.1 (1.8)
	15	30.9 (1.2)	31.6 (1.1)
	30	31.2 (1.4)	31.7 (1.2)
	45	31.4 (1.3)	32.0 (1.0)
	60	31.5 (1.2)	32.4 (0.8)
	90	31.9 (1.1)	32.8 (1.0)
	120	32.1 (1.2)	33.1 (1.0)
	150	32.2 (1.3)	33.3 (1.0)
	180	32.4 (1.5)	33.5 (1.4)

* Statistically significant difference between Warmed Group and Control Group.

Forehead temperature, which reflects but is not equal to core temperature, showed a similar pattern to that seen with rectal temperature (Fig. 2). Although forehead temperatures were higher in the warmed group than in the control group the differences did not reach statistical significance at any time.

Thumb (Fig. 3) and forearm temperature (Fig. 4) in the warmed group showed an initial dramatic rise from baseline by 15 minutes and then continued to rise slowly throughout the course of the study. When compared to the control group there is a statistically significant difference at time intervals from 15 minutes onwards. These results indicate that the forced-air convective device is effective as a shell or skin surface warmer. An analysis of the forearm-thumb skin temperature difference in the two groups shows that the control group exhibited a mean temperature difference of 3.4°C by the end of the study. The warmed group on the other hand exhibited a temperature difference of only 0.4°C (Fig. 5). The difference between forearm and thumb temperatures seen in the control group is an indication of the maintenance of vasoconstriction to prevent heat loss. Although there is a much smaller temperature differential in the warmed group it cannot be concluded that this indicates vasodilation since the warmed group were covered from the neck down by the blanket. The incidence of shivering assessed simply by visual observation in both groups was noted by the investigator during the course of the study. As previously stated the investigator was aware which patients were being warmed and

which were not. With this bias in mind the investigator noted that the number of patients shivering in the warmed versus control groups during the first three fifteen minute epochs was 3 versus 7, 1 versus 5, 0 versus 4. These figures represent a statistically significant difference for the periods 16-30 and 31-45 minutes but not for the period 0-15 minutes (Fig. 6).

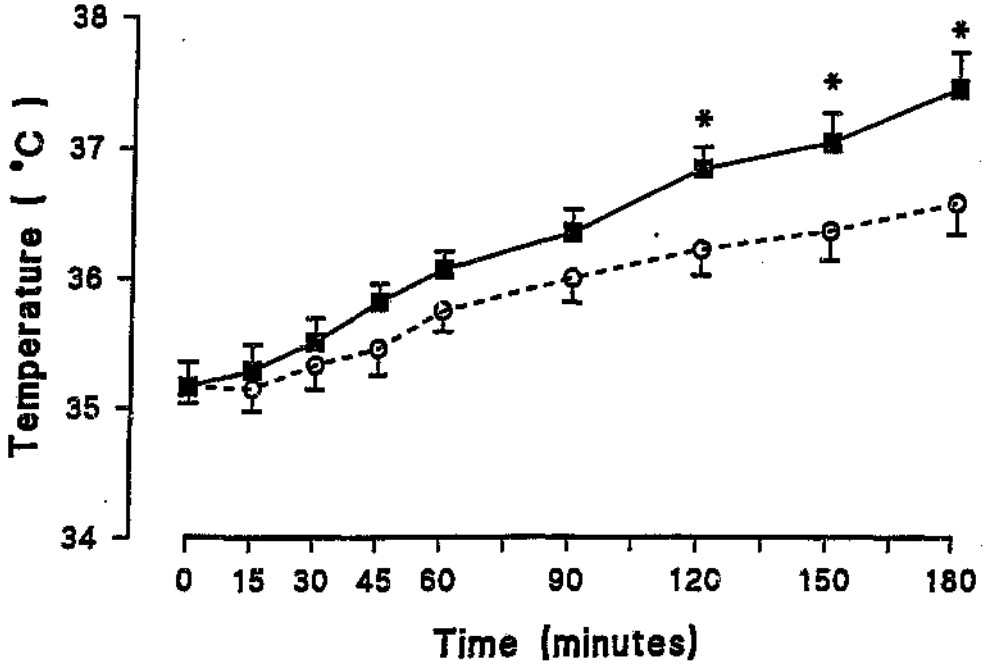

Figure 1: Change in rectal temperature with time in the Warmed Group (■) and the Control Group (○). Results are presented as Mean ± 1 Standard Error of the Mean. °C = Degrees Celsius. * = statistically significant difference.

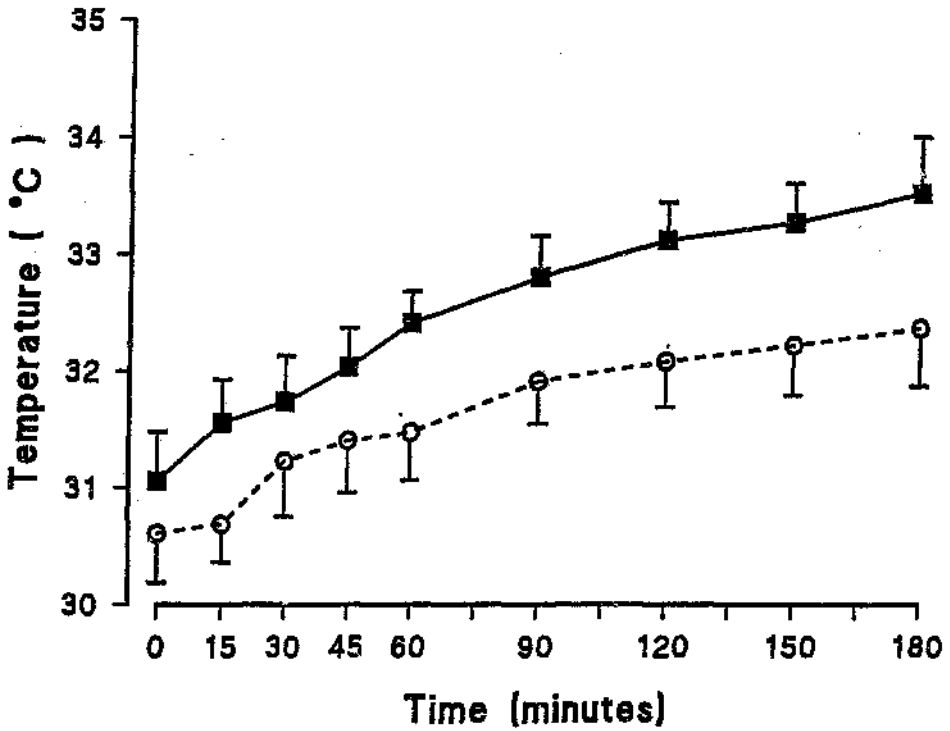

Figure 2: Change in forehead temperature with time in Warmed Group (■) and the Control Group (O). Results are presented as the Mean ± 1 Standard Error of the Mean. °C = Degrees Celsius.

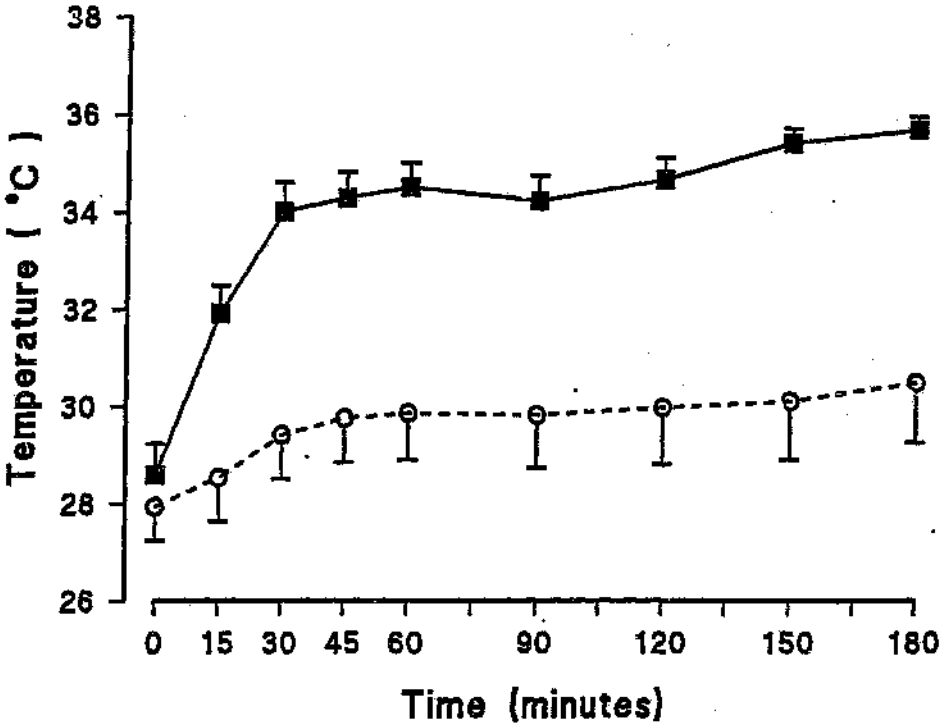

Figure 3: Change in thumb temperature with time in the Warmed Group (■) and the Control Group(○). Results are presented as the Mean ± 1 Standard Error of the Mean. °C = Degrees Celsius.

Note: With the exception of the results at time zero, there is a statistically significant difference between the two groups at all time intervals

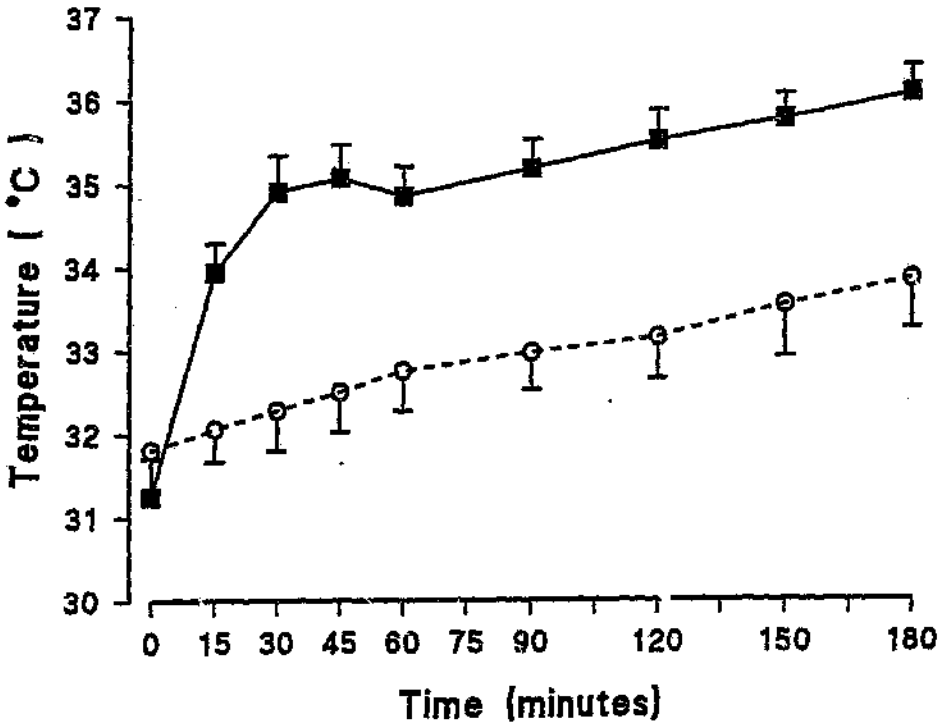

Figure 4: Change in forearm temperature with time in Warmed Group (■) and the Control Group(○). Results are presented as the Mean ± 1 Standard Error of the Mean. °C = Degrees Celsius.

Note: With the exception of the results at time zero, there is a statistically significant difference between the two groups at all time intervals.

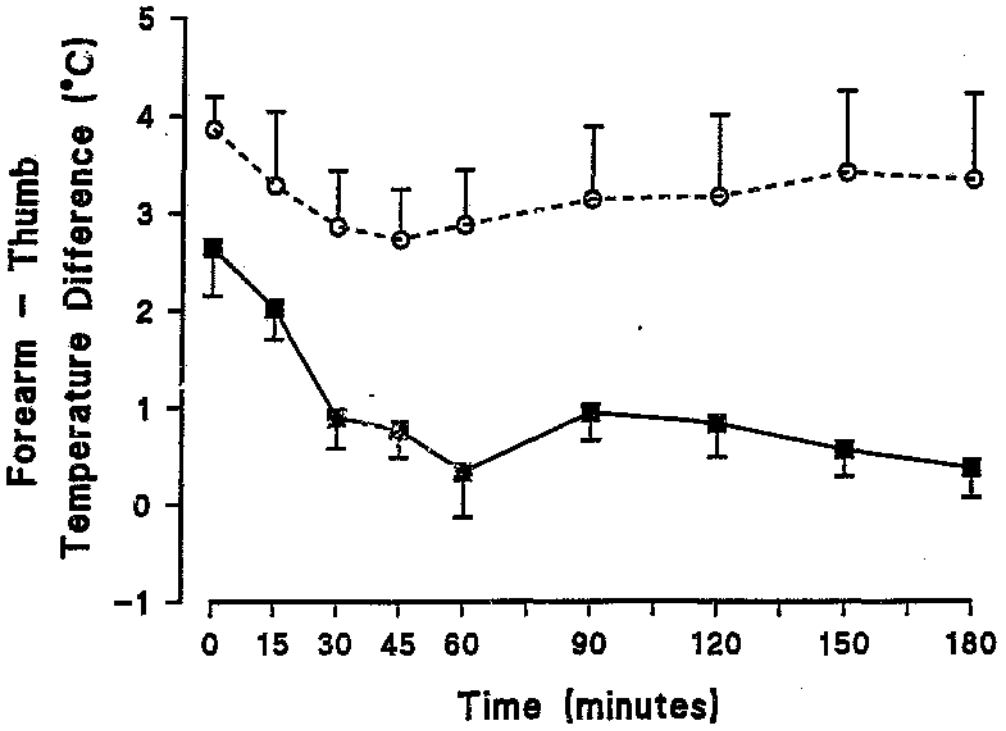

Figure 5: Forearm-thumb temperature difference with time in the Warmed Group (■) and Control Group (○). Results presented as Mean ± 1 Standard Error of the Mean. °C = Degrees Celsius.

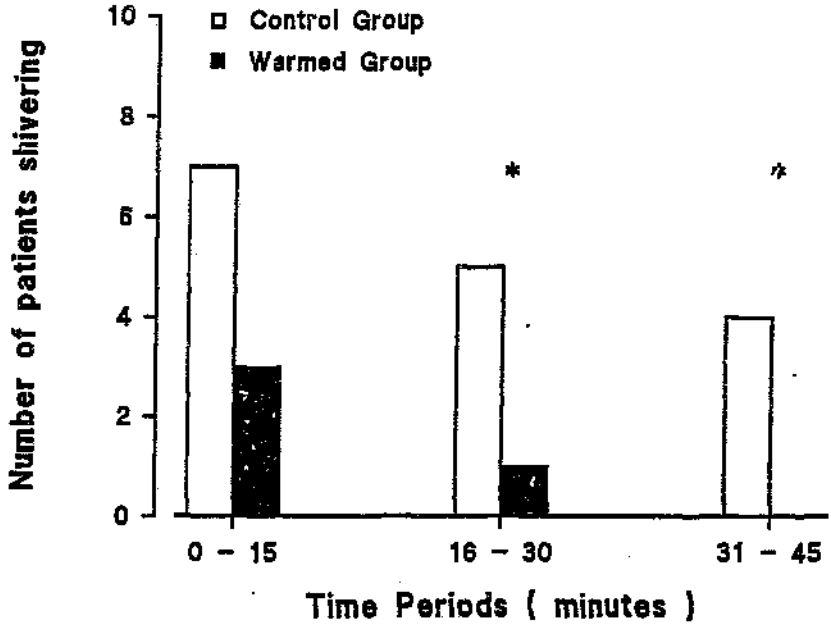

Fig 6. Bar graph illustration of shivering incidence in the Warmed Group (solid bars) and the Control Group (open bars). * = statisically significant difference.

5. DISCUSSION

Hypothermia in the postoperative patient has, until recently, been viewed as a relatively benign complication of the perioperative period. Slotman *et al* [10] noted that hypothermia is associated with adverse effects and showed that, in a mixed group of 100 elective and emergency patients, the incidence of intraoperative and postoperative hypothermia was 77% and 53%, respectively. In particular, those workers observed that if hypothermia remained present at 8 hours postoperation there was an associated 75% mortality. An observation of 50 consecutive elective surgery patients, excluding those having operations involving the head or the neck regions, who received no active intra-operative thermal management showed that 68% of the patients had a core temperature less than 36°C on admission to a recovery area (C. Clinton - unpublished data). The combination of perioperative hypothermia and traumatic injury is associated with an increased mortality rate.[31, 32]

Although the ambient temperature of the operating room and the type of surgery play a role in the genesis of hypothermia, the effect of anaesthesia is more important. Under normal circumstances, thermoregulatory responses will be initiated by decreases in core temperature of as little as 0.1-0.2°C.[5,6] Obviously, anaesthesia utilising vasodilator drugs and muscle relaxants may further obtund or completely prevent a thermoregulatory response, irrespective of the extent of core temperature decline.

As stated, hypothermia is a common complication of the perioperative period that has, in the past, been viewed as relatively innocuous . More recently, some of the deleterious effects have been investigated more thoroughly. While some of the effects may not unduly influence recovery or outcome, others are serious and have the potential to alter postoperative outcome. Frank *et al*[13] have shown that, in patients undergoing lower extremity revascularisation procedures, myocardial ischaemic events in the first 24 postoperative hours are significantly more common in hypothermic patients than in normothermic counterparts - irrespective of whether they received general or regional anaesthesia. In that study the fact that hypothermia was an independent predictor of postoperative myocardial ischaemia was clearly demonstrated . It is easy to imagine a scenario where simultaneous events such as shivering, hypotension and a shift of the oxygen dissociation curve to the left could lead to significant cardiac compromise, especially for the patient with ischaemic heart disease.

Hypothermia in the awake postoperative patient is associated with vasoconstriction . Such vasoconstriction is responsible for varying degrees of hypertension in the immediate postoperative period. Hypertension associated with hypothermia should not receive aggressive pharmacological management since, when re-warming occurs, there may be significant hypotension. On the other hand, mild hypothermia in awake postoperative patients is accompanied by a water diuresis. Later, when core temperature returns to normal and vasoconstriction subsides, this diuresis then manifests

as an ineffective circulating volume with postoperative hypotension. Coagulopathy is particularly important to the surgical team - hypothermia induces prolongation of the bleeding time and reversible platelet dysfunction.[33] Shivering in the postoperative period is associated with patient discomfort, increased wound pain, an increase in oxygen consumption and an increase in carbon dioxide production.

Before the advent of forced-air convective warmers various strategies were utilised intraoperatively for the maintenance of core temperature. Reflective blankets decrease radiant heat loss from patient to environment. Their greatest effect appears to be in patients having peripheral procedures of short duration when at least 60% of body surface can be covered.[34] The warm water circulating mattress has not always been shown to have a significant effect in the adult patient. Its' effectiveness is influenced by operating room temperature and by the fact that only a small surface area of the patient is in contact with the warm surface.[35] The temperature of the blanket required to make a significant difference to core temperature is about 40°C and this carries the risk of thermal injury. Airway humidification with heated humidifiers will balance respiratory heat loss - but this is never very high. Accidental overheating has resulted in respiratory tract thermal injury[36] - thus risk tends to outweigh benefit.

Studies on intraoperative use of forced-air warming devices[25, 29, 30] indicate that core temperature is better maintained than with other conventional methods and call attention to the fact that hypothermic patients are at risk for various complications. For example, this investigation's Control Group exhibited vasoconstriction well into the postoperative period, as evidenced by a forearm-thumb skin surface temperature difference. The use of forced-air warmers in the postoperative period has been associated with faster re-warming[26] and a reduced incidence of shivering.[16] This investigation similarly indicates that hypothermic patients exposed to a forced-air warmer become normothermic sooner, have significant increases in skin surface temperature and have a reduced incidence of shivering compared to a group who are not exposed to the device.

There is evidence, from the literature, that hypothermic postoperative patients should be assisted to rewarm in order to reduce haemodynamic[13] and metabolic[14] complications and to eliminate shivering.[16] The forced-air convective warming device is effective in the management of postoperative hypothermia because the incidence of shivering is reduced and more rapid re-warming takes place. In this investigation of patients who had not received any intraoperative thermal management, 2 hours of continuous use was required to produce statistically significant differences in core temperature. This should not necessarily be interpreted as machine failure although efficiency of various types of forced-air warmers is known to be

different.[25] The particular type of warmer used in this study has been shown to have a heat transfer capability of only 68 ± 8 watts.[24] It could well be that a more efficient forced-air warmer would have produced better results. On the other hand, it could be that patients who have not received intraoperative thermal management are more difficult to warm in the postoperative period than patients who have received some intraoperative thermal care. However, this investigation did not address those two questions. Thus, there is still scope for a comparative study of forced-air convection warming devices in hypothermic, postoperative patients who have and have not received intraoperative thermal care.

This investigation demonstrated that the forced-air convection warmer used:

a) is an effective shell, or skin surface, warming device

b) eliminates shivering in the early postoperative period

c) contributes to faster re-warming of the hypothermic postoperative period, if used for more than 2 hours.

Other studies indicate similar findings. In particular, the simple humanitarian aspect of decreasing postoperative pain by eliminating shivering has been noted.[16, 27] Studies that have examined the use of forced-air warming in the pre-, intra-, and postoperative periods and in combinations of those periods, suggest that there are many actual and potential benefits of eliminating hypothermia - even for the fit, healthy patient who requires anaesthesia and surgery.

Forced-air warming is a simple and easy technique. It can be initiated in the intraoperative period without interfering with the operation. From an analysis of the literature, the risk-benefit ratio of forced-air warming would appear to favour the patient. Similarly, patients who are hypothermic in the postoperative period benefit from forced-air warming.

The author of this text believes that all medical, nursing and para-medical staff involved with intraoperative and postoperative care should appreciate that a simple, safe, effective method is available to combat perioperative hypothermia and that, if not a specific requirement of the surgical technique, hypothermia should be managed actively from the outset.

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