

# THERMAL AND CARDIOVASCULAR CHANGES DURING THREE METHODS OF RESUSCITATION FROM MILD HYPOTHERMIA

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## SUMMARY

**The interrelations among core temperatures (cardiac, esophageal, tympanic, rectal), skin temperature, and cardiovascular function (cardiac output, arterial pressure, heart rate, total peripheral resistance) were studied in a conscious subject during entry into mild hypothermia through cold water (10°C) immersion, and during rewarming by three basic procedures: peripheral heat donation (bath); core heat donation (inhalation); and no exogenous heat (spontaneous). Swan-Ganz catheterization of the heart enabled measurement of cardiac temperature as well as cardiac output by the thermal dilution method.**

During cooling, all sites of core temperature measurement showed similar rates of entry into hypothermia. However, during the rewarming procedures, divergent patterns of temperature change among the four sites occurred. Rectal and tympanic temperatures were not representative of cardiac temperature, but esophageal temperature was, and is therefore most suitable as a criterion for experimental evaluation of the thermal benefit of various core rewarming techniques.

During the first 30 min of rewarming, rates of increase in cardiac temperature for bath, inhalation, and spontaneous procedures varied according to the proportions 4:2:1, respectively. No afterdrop of cardiac temperature occurred with the inhalation or spontaneous procedures, but an afterdrop at this site did occur during the first 15 min of bath rewarming as soon as skin temperature was greater than 30°C. This afterdrop coincided with cardiovascular changes including abrupt decreases in arterial pressure and total peripheral resistance, along with increases in heart rate and cardiac output. Such evidence of increased peripheral circulation was not observed with the inhalation and spontaneous methods.

The findings relate to experimental evaluation of rewarming techniques and principles for resuscitation of hypothermia victims, especially in the first-aid situation.

## **INTRODUCTION**

In recent years, much experimentation and case history analysis has been devoted to strategies for resuscitation of victims of accidental hypothermia in both the first-aid and hospital situations (Hayward and Steinman, 1975; Shanks, 1975; Lloyd, Mitchell and Williams, 1976; Collis, Steinman and Chaney, 1977; Edwards, 1977; Stine, 1977; Jessen and Hagelsten, 1978; Marcus, 1978; Coniam, 1979; Marcus, 1979; Morrison, Conn and Hayward, 1979; Harnett, O'Brien, Sias and Pruitt, 1980; Miller, Danzl and Thomas, 1980; Martyn, 1981; Samuelson, Doolittle, Hayward, Mills and Nemiroff, 1982).

Significant uncertainty still exists concerning the safety or effectiveness of various rewarming techniques, especially for use at the rescue site and during transportation to hospital. Such uncertainty is partially-related to the absence of experimental information on two crucial aspects of this topic.

Heart temperature, which is the most important criterion of resuscitation effectiveness, has not been measured in unanaesthetized humans during evaluation of various techniques for peripheral or core heat donation. Evaluations based upon rectal, tympanic and esophageal temperatures may or may not be valid. Also, major cardiovascular functions, which are necessary for assessing the status of the peripheral circulation during rewarming, have not been studied in this particular context concerning accidental hypothermia. It was the objective of this study to obtain these two types of information concurrently, thereby facilitating their comparison. This combination was potentiated by use of a thermal dilution method for determination of cardiac output, which also provided a means to assess heart temperature.

Mild hypothermia was induced by immersion in cold (10°C) water, which produces large thermal gradients throughout the body of conscious humans capable of maximizing peripheral vasoconstriction. Rewarming involved three basic strategies: peripheral heat donation (bath); core heat donation (inhalation); and no exogenous heat donation ("spontaneous").

The results would be directly relevant to resuscitation from mild hypothermia, and may provide insights into processes and potential problems associated with the more-challenging responsibility for first-aid treatment of severe accidental hypothermia.

## **MATERIALS AND METHODS**

Because heart catheterization was the prime methodology of this study, normal recruitment of subjects was not possible due to the constraints of ethical human experimentation. Instead, one of the investigators volunteered for the procedures. The subject was fully aware of the possible risks associated with heart catheterization and completed formal waiver of responsibility for all

individuals and organizations associated with the experiments. Although results obtained from only one subject must be evaluated with caution, previous studies of immersion hypothermia and rewarming in which this individual was a subject (Hayward, Eckerson and Collis, 1975; Hayward and Steinman, 1975; Fox, Hayward and Hobson, 1979) have always shown him to be representative of responses for the average, adult male. Accordingly, there is a high probability that the results obtained in this study would have good fidelity with the mean response of a population of subjects. The physical characteristics of the subject were: height, 180 cm; weight, 79.5 kg; and mean skinfold thickness (from 4 standard sites), 14 mm. His age was 43 years.

### **Cardiovascular measurements**

Cardiac output was obtained by the thermal dilution technique using a Swan-Ganz catheter inserted in the left brachial vein and advanced through the right heart.

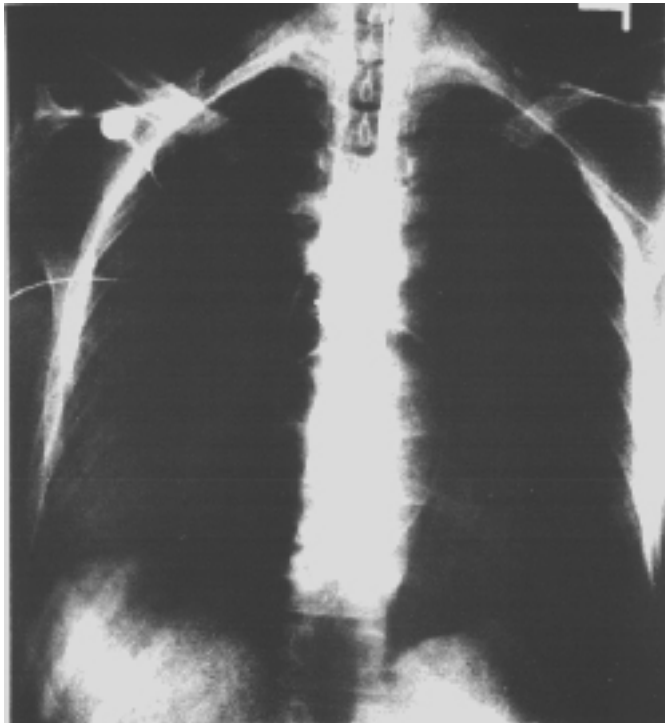


Figure 1 shows the location of the catheter with its tip located in the right pulmonary artery. The thermistor near the catheter tip was connected to a cardiac computer (Edwards Laboratories, Model 9520) for determination of cardiac output. Arterial pressure was obtained using a Tek 414 monitor and Statham P-50 transducer connected to a catheter in the left radial artery. The electrocardiogram was displayed on an oscilloscope with digital display of heart rate.

### **Thermal measurements**

Cardiac temperature was recorded from the thermistor in the Swan-Ganz catheter when this sensor was not involved in the cardiac output determinations. Assignment of the temperature of blood being ejected from the ventricles to represent heart temperature has been utilized previously by Severinghaus (1959) when studying surgical hypothermia. Esophageal temperature was obtained from a thermistor inserted via the nasal passage to the lower esophagus, a distance of 42 cm from the nares. This thermistor also appears in Figure 1 and shows the sensor to be at the level of the ventricles. Rectal temperature was obtained from a thermistor inserted 15 cm beyond the anus. For tympanic temperature, a small, padded thermocouple was placed in close proximity to the tympanum and then

the aural canal was sealed with a wax plug to reduce thermal effects of the environment. Skin temperatures were obtained with thermistors specialized for surface application. These were placed at 4 sites: chest, arm, thigh, and calf. No weightings were utilized in calculating the mean skin temperature.

### **Cooling and rewarming procedures**

The experiments were conducted on 3 consecutive days.

On the morning of day 1, the catheters were inserted by a cardiologist at a hospital location.

Each day the subject would begin the experiment by donning a bathing suit and sitting in a room at 21-22°C while ECG electrodes and thermal sensors were applied. Pre-immersion recordings of all variables were obtained for 10 min, followed by immersion to the level of the neck in a tank of stirred (0.2 m s<sup>-1</sup>) water at 10°C. A sitting posture was facilitated by a chair of mesh-type material suspended in the water. Thermal and cardiovascular measures were recorded at 5-min intervals, except for cardiac output which was obtained on approximately a 20-min schedule.

After 100 min of cooling, the subject quickly (1-2 min) transferred to the rewarming regime.

On day 1, this involved "spontaneous" or "endogenous" rewarming with the provision of no exogenous heat. The subject laid in a sleeping bag with good insulative quality, and body heat production, mainly from vigorous shivering (Hayward, Eckerson and Collis, 1977), was responsible for rewarming. For the next two experiments, exogenous sources of heat also contributed to rewarming.

On day 2, this consisted of a core rewarming procedure via the airway (inhalation rewarming) while the subject was again insulated by a sleeping bag. Heated (43-45°C), water-saturated air was provided for inspiration via an oronasal mask. A device ("Heat-Treat", Canada) which thermoregulates a mixture of steam and ambient air was utilized for this method.

On day 3, rewarming was by a peripheral rewarming method involving immersion (only head and neck out) in a hydrotherapy bath of vigorously-stirred water which was 30°C initially and raised to 41-42°C over the first 10 min. After 30 min, bath temperature was reduced slightly to 38-39°C. By 60 min of rewarming with the three methods, rates of change of core temperature were minimal and measurements were terminated. Statistical analysis of differences between means was by Student's *t* with significance concluded if  $P < 0.05$ .

## RESULTS

### Thermal responses

Cooling phase. Changes in mean skin temperature and core temperatures during entry into mild hypothermia are shown in Figure 2.

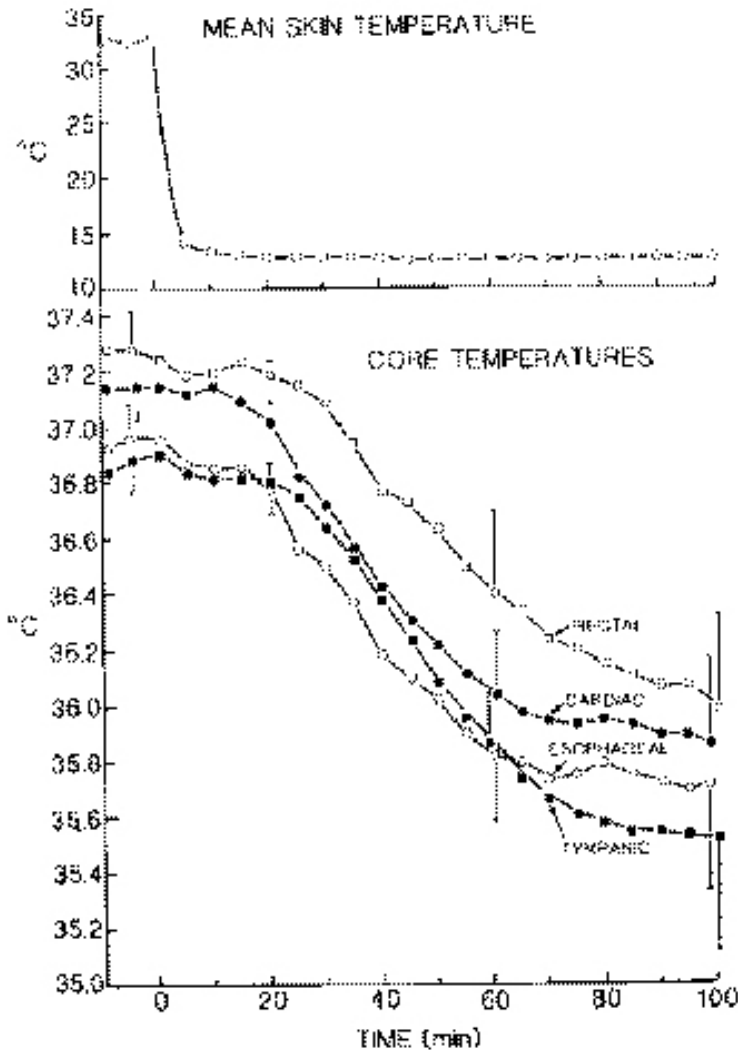


Fig. 2. Mean skin temperature and core temperatures during immersion in 10°C water. Representative standard deviations shown for core temperatures. Standard deviations were too small to illustrate for mean skin temperature.

Each response is the average of the three separate immersions conducted on consecutive days. Mean skin temperature fell rapidly from an average of 32.8 +/- 0.15 (S.D.)°C to less than 13°C by 5 min, followed by further slight reduction to 12.5°C for the remainder of the immersion. Core temperatures averaged from 36.9 to 37.3°C at the beginning of immersion. Consistent core cooling became established after 20 min, with cooling rates of the various sites approximating 1.0°C. h<sup>-1</sup>

These rates are in accord with those found in previous studies (Hayward et al. 1977; Keatinge, 1969) for persons with moderate subcutaneous fatness. The patterns of change of the four core temperatures were essentially identical, with the same order of relationship among the four sites existing at the end of cooling as at the beginning, and the levels not being significantly different ( $P < 0.05$ ).

### Rewarming phase.

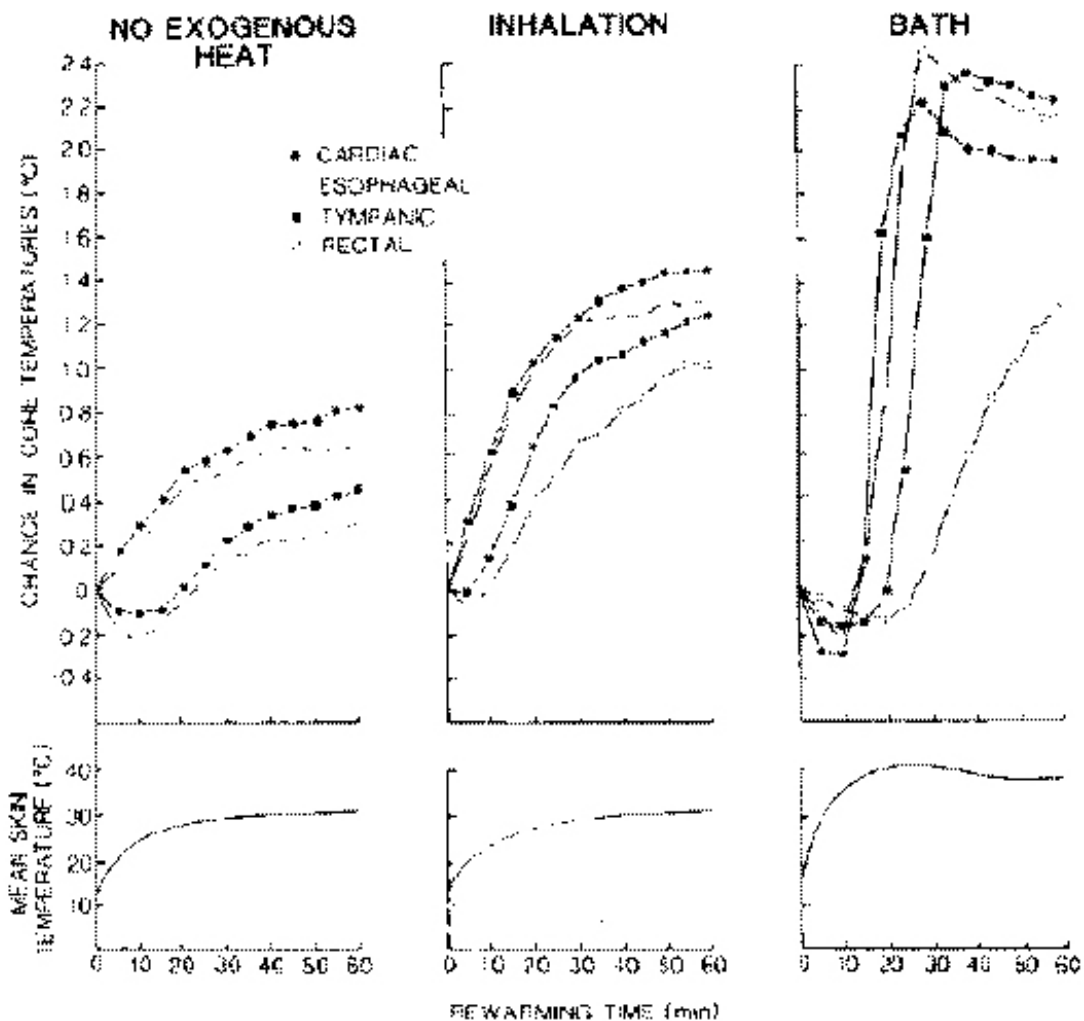


Fig. 3. Changes in core temperatures and mean skin temperature during three rewarming procedures.

Figure 3 presents the changes in core temperatures and mean skin temperature for each of the three rewarming conditions. In each case, esophageal temperature changed with high fidelity to cardiac temperature, especially during the first 30 min when the major alterations occurred. Rectal temperature changes showed poor relationship to those of the cardiac-esophageal pair, with tympanic changes being intermediate. The rectal and tympanic sites always showed an "afterdrop", with the rectal site requiring 15-25 min to regain the temperature at the commencement of rewarming. No afterdrop was observed at the cardiac and esophageal sites, except in the case of bath rewarming, where abrupt falls of approximately  $0.3^{\circ}\text{C}$  occurred within the first 15 min. These falls coincided with

the period that mean skin temperature first increased to above 30°C along with the water temperature of the bath. For the other two conditions, the time courses of increase in mean skin temperature were almost identical to each other and required more than 30 min to reach their maximum near 30°C.

Figure 3 also shows that the rates of core rewarming associated with the three conditions increased in the order: no exogenous heat < inhalation < bath, especially when considering the cardiac and esophageal sites. Over the first 30 min of rewarming, the cardiac-esophageal pair increased 0.6°C for no exogenous heat, 1.2°C for inhalation, and 2.4°C for bath; a 1:2:4 relationship, respectively.

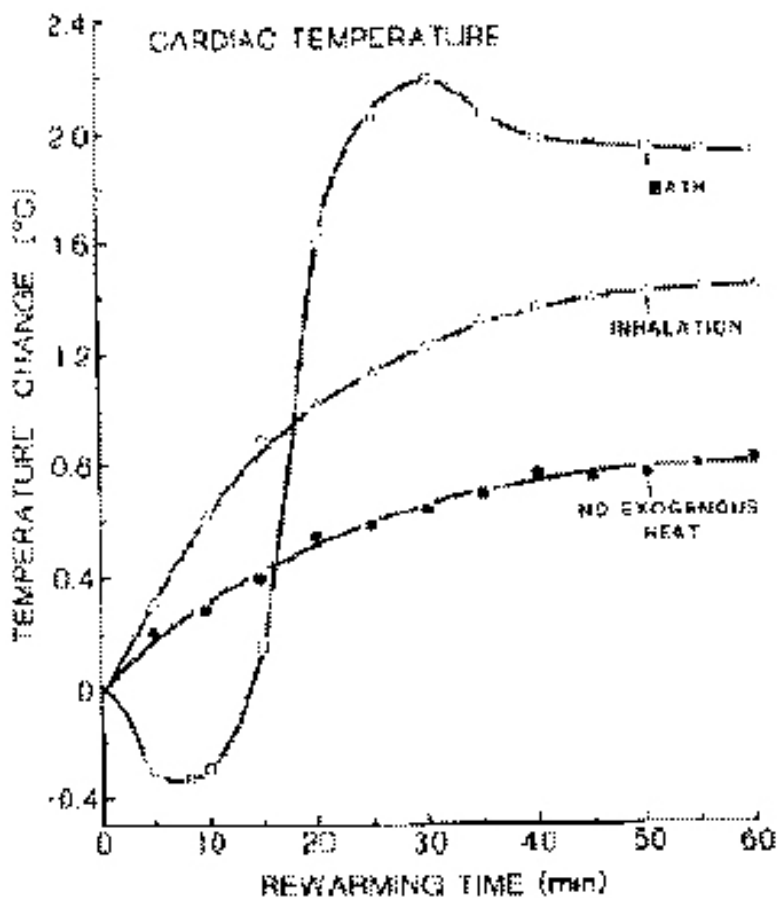


Fig. 4. Changes in cardiac temperature during three rewarming procedures.

Figure 4 isolates the data for change in cardiac temperature during rewarming to facilitate the above comparison of responses to the three methods of treatment. It is clear that the bath treatment provided a very rapid rate of increase of cardiac temperature (approximately 5°C h<sup>-1</sup>) once the period of afterdrop had ended, such that cardiac temperature rose to the normothermic level in the time required (25 min) for rectal temperature to complete its afterdrop.

## Cardiovascular responses

Changes in mean arterial pressure, cardiac output and heart rate during cooling and rewarming are shown in Figure 5.

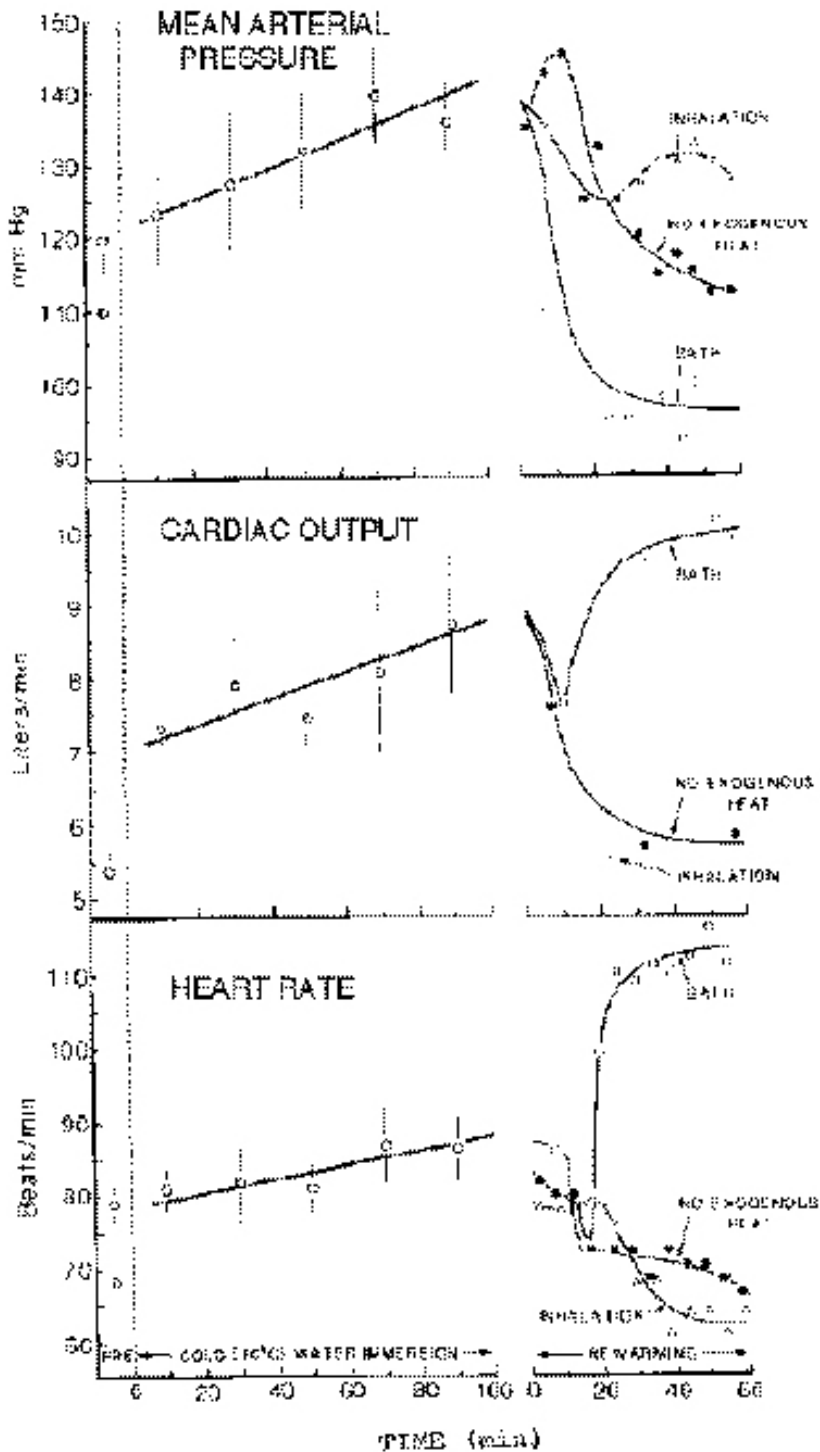


Fig. 5. Cardiovascular responses during cold water immersion and rewarming by three procedures.  $\bullet$  = "normal" pre-immersion values of heart rate and mean arterial pressure.

During pre-immersion, these variables were higher than normal due to the anxiety attending imminent immersion in cold water. More typical pre-immersion

values for this subject are known for mean arterial pressure and heart rate during regular daytime activity as a university professor, and these have been plotted also. Upon cold water immersion, all three variables were elevated above normal by 10-25 %. Thereafter to 100 min, each increased fairly uniformly, with mean arterial pressure reaching 140 mmHg, cardiac output 8.71 min<sup>-1</sup> and heart rate, 85 beats min<sup>-1</sup>. At this stage, cardiac output showed the greatest increase (64 %) from the normal, pre-immersion level, while mean arterial pressure and heart rate had increased approximately 25 %. Pulse pressure during cooling was in the range of 80-100 mmHg. The changes in cardiac output during cooling are appropriate to standard predictions (Altman and Ditter, 1971) in relation to the non-exercising metabolic response (Hayward et al., 1977) to immersion in 10°C water.

During rewarming, similar cardiovascular responses (Figure 5) were observed for the no exogenous heat and inhalation conditions, both showing decreases in cardiac output to near the pre-immersion level by 30 min. This was associated with declines in heart rate and mean arterial pressure to normal levels, although for the inhalation condition, mean arterial pressure remained 10-15 % higher, and heart rate 10-15 % lower than for no exogenous heat. Considerably-different responses occurred with the bath treatment. During the first 15 min, mean arterial pressure showed a large decrease to approximately 95 mmHg, a hypotensive level for this individual. From 20 min onward, further decline was arrested by an abrupt, 55 % increase in heart rate (of about 40 beats min<sup>-1</sup>) which produced a 30-35 % increase in cardiac output. During the first 30 min of rewarming, pulse pressures fell from the elevated range during cooling to approximately 60-70 mmHg for all three conditions.

### **Peripheral resistance and stroke volume.**

These cardiovascular variables were calculated from those measured. During cooling, total peripheral resistance was steady in the range of 15-20 mmHg .1-1 min. During rewarming, total peripheral resistance was near 20 mmHg 1-1 min for the no exogenous heat and inhalation treatments, but fell to 10 mmHg 1-1 min early in the bath treatment. Stroke volume averaged 0.09 liters and no major changes occurred with cooling or rewarming.

## **DISCUSSION**

These results show that the four sites of measurement of core temperature provide similar information on rate of progress into hypothermia, thereby enabling rectal or tympanic temperature to be used for initial assessment of the level of hypothermia when a victim is first removed from severe cold stress. However, it is clear that during the more dynamic events of early resuscitation, when thermal gradients in the body are being reduced or reversed, there are considerable differentials among the various sites used to assess core temperature.

During the early period of treatment, rectal and tympanic temperatures do not adequately reflect temperature changes of the "critical core" as represented by cardiac temperature. Consequently, these two sites should not be used as prime

criteria for experimental evaluation of the effectiveness of various treatments for accidental hypothermia.

On the contrary, deep esophageal temperature is a reliable analogue of cardiac temperature. This was true even when exogenous heat was donated by the airway, and validates previous interpretations concerning inhalation rewarming which were based on esophageal temperature (Hayward and Steinman, 1975).

Due to the inappropriateness of routine use of cardiac catheterization in experimental evaluations of rewarming techniques in conscious humans, esophageal temperature should be utilized for definitive judgment of the effectiveness of core rewarming.

The present findings on temperature differentials in the core during rewarming yield similar conclusions to those derived previously from studies of anaesthetized patients being rewarmed from hypothermic surgery (Cooper and Kenyon, 1957; Severinghaus, 1959). However, in the present study, differences among patterns of change in the various core temperatures are more accentuated. This is probably a result of the strong vasoconstrictive response in conscious humans during cold stress, which produces a larger core-to-shell thermal gradient than in anaesthetized patients entering hypothermia passively.

Reports (Guild, 1978; Marcus, 1978) have concluded (without measuring cardiac temperature) that inhalation rewarming, due to the small *amount* of heat that can be potentially donated, is not more effective than endogenous heat production alone (spontaneous condition). The present results do not support this contention.

Cardiac temperature rose with inhalation at twice the rate observed for no exogenous heat. If "effectiveness" of rewarming is more appropriately evaluated on the basis of heart or deep esophageal temperatures, rather than rectal and aural temperature or amount of heat transferred to the body, then inhalation therapy appears justified.

In cases of severe hypothermia, where ventilation rate is low in semi-comatose victims, the thermal effect of the inhalation method would be reduced, but the same condition would also greatly lower the endogenous heat production, making a small amount of exogenous heat that is donated directly to the thoracic core even more important. This is validated by the work of Shanks (1975).

The benefit of heat donation via the airway in severely hypothermic victims is in addition to the insulative effect of this method of therapy, namely the avoidance of respiratory heat loss. This factor is particularly important when first-aid for hypothermia must be provided in the common rescue situation where the ambient air is very cold.

The combination of airway insulation and heat donation would increase the probability that cardiac temperature could be stabilized, whereas a victim with low endogenous heat production and lack of protection from respiratory heat loss

may not be able to avoid further cooling of the heart to much colder peripheral tissues.

It is important to emphasize that under the conditions of this experiment, no afterdrop of cardiac temperature occurred with either the spontaneous or inhalation treatments.

In contrast, bath rewarming did cause a further decline in cardiac temperature during the initial phase of treatment.

Whereas the afterdrop of rectal temperature which occurred with all treatments is mainly due to continued heat loss down a thermal gradient in the tissues towards the colder periphery (Golden and Hervey, 1977), the mechanism of cardiac afterdrop is more likely due to increased return, centrally, of colder blood from the body shell in conjunction with decreased peripheral vasoconstriction. The cardiovascular findings of this study support this proposition because the decreased peripheral resistance (deduced from the fall in mean arterial pressure and rise in cardiac output) coincided with the period of afterdrop of cardiac and esophageal temperatures. This "circulation-induced" afterdrop was associated with raising of the skin temperature to thermoneutral temperature and above, even though core temperatures were at their lowest levels.

This confirms experimentally, for the first time in conscious humans, the long-suspected increase in peripheral circulation which accompanies "aggressive", external donation of heat to the whole body.

As has already been demonstrated for the thermogenic (shivering) response during hypothermia of the core (Hayward et al., 1977), maintenance of maximal peripheral vasoconstriction is dependent on a cold skin.

If this phenomenon also applies to cases of severe hypothermia (e.g. core temperatures  $<30^{\circ}\text{C}$ ), then the cardiac afterdrop accompanying bath rewarming would increase the probability of attaining a sufficiently low temperature to cause ventricular fibrillation (Keatinge, 1977).

The magnitude of the cardiac afterdrop was relatively small in the strongly-shivering subject of this study, but could be much larger in a comatose victim with severe hypothermia.

Cardiac dysfunction would also be enhanced by probable biochemical disturbances (pH, K<sup>+</sup>) in the blood returning to the heart from peripheral tissues which had been relatively ischaemic during a long cooling phase. Such thermal and chemical challenges to the myocardium would occur just at the time when cardiac excitation was required to compensate for the relative hypotension derived from peripheral dilation in response to warm ( $>30^{\circ}\text{C}$ ) skin temperature. This process or any other factor (such as limb movement) which would increase the return of peripheral blood to the heart would be similarly contraindicated during resuscitation from severe hypothermia, even in a hospital situation where "total physiological control" is often available (Stine, 1977; Martyn, 1981; Samuelson et al., 1982).

It is important to emphasize that these interpretations from the present findings are most relevant to first-aid strategies for management of hypothermia victims at the rescue site or during transport.

This is because the most potentially-dangerous thermal and cardiovascular events are likely to occur during the first 20-30 min immediately after removal of the extreme, external cold stress.

By the time a victim who became hypothermic in the water or on a mountainside is transported to hospital (if that is actually possible), the maximum potential for cardiac dysfunction due to thermal, chemical and mechanical (Golden, 1973) stimuli has probably already occurred.

These results confirm the general recommendation (Samuelson et al., 1982) that unless rescuers are certain that a victim of core cooling has only mild to moderate hypothermia (e.g. rectal temperature 32.0°C), rapid peripheral heating of the whole body and exercise should be avoided, with any sources of exogenous heat being donated to the thoracic and neck regions, the primary aim being to *stabilize* cardiac temperatures and secondarily, to facilitate gradual rewarming of the core.

## REFERENCES

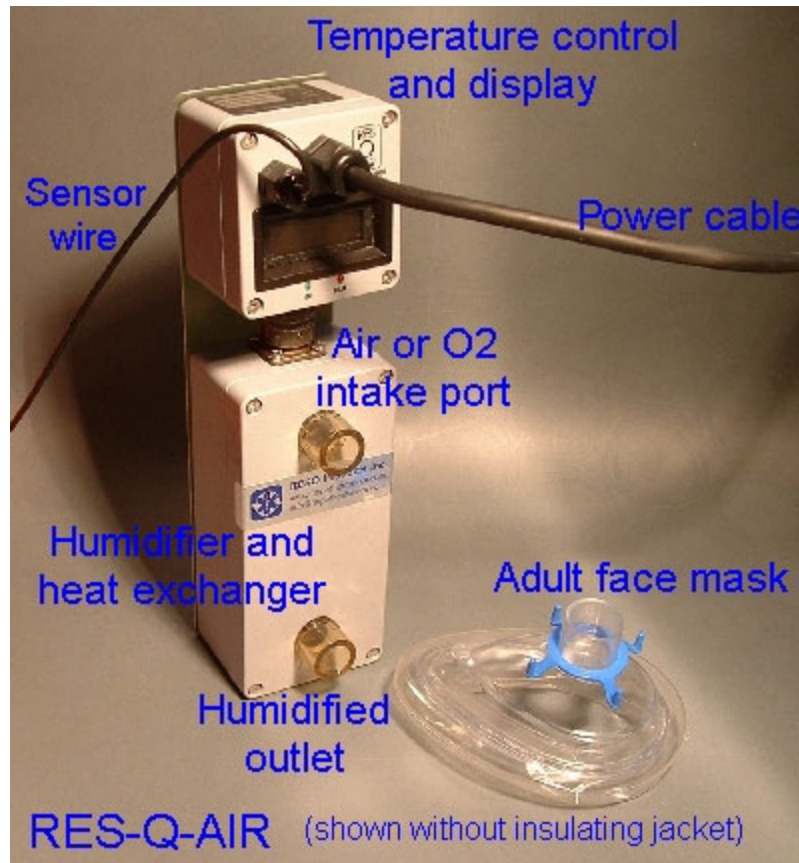
- Altman, P.L. and Dittmer, D.S. (1974) Biology Data Book: Respiration and circulation. Fed. Am. Soc. Exp. Biol., Bethesda, Maryland, p.1695.
- Collis, M.L., Steinman, A.M. and Chaney, R.D. (1977) Accidental hypothermia: An experimental study of practical rewarming methods. *Aviat. Space Environ. Med.*, 48, 625-632.
- Coniam, S.W. (1979) Accidental hypothermia. *Anaesthesia*, 34, 250-256.
- Cooper, K.E. and Kenyon, J.R. (1957) A comparison of temperatures measured in the rectum, esophagus and on the surface of the aorta during hypothermia in man. *Br.J. Surg.*, 44, 616-619.
- Edwards, R.D., Jr. (1977) Accidental hypothermia. *JACEP*, 6, 426-427.
- Fox, G.R., Hayward, J.S. and Hobson, G.N. (1979) Effect of alcohol on thermal balance of man in cold water. *Can. J. Physiol. Pharmacol.*, 57, 860-865.
- Golden, F.St.C. (1973) Death after rescue from immersion in cold water. *J. Roy. Nav. Med. Serv.* 59, 5-7.
- Golden, F.St.C. & Hervey, G.R. (1977). The mechanism of the afterdrop following immersion hypothermia in pigs. *J. Physiol.* 272, 26-27.
- Guild, W..J. (1978). Rewarming via the airway (CBRW) for hypothermia in the field? *J. R. Nav. Med. Serv.*, 64, 186-193.
- Harnett, R.M., O'Brien, E.M., Sias, F.R. and Pruitt, J.R. (1980) Initial treatment of profound accidental hypothermia. *Aviat. Space Environ. Med.*, 51, 680-687.
- Hayward, J.S., Eckerson, J.D. and Collis, M.L. (1975) Thermal balance and survival time prediction of man in cold water. *Can. J. Physiol. Pharmacol.*, 53, 21-32.

- Hayward, J.S., Eckerson, J.D. and Collis, M.L. (1977) Thermoregulatory heat production in man: prediction equation based on skin and core temperatures. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.*, 43, 377-384.
- Hayward, J.S. and Steinman, A.M. (1975) Accidental hypothermia: an experimental study of inhalation rewarming. *Aviat. Space Environ. Med.*, 46, 1236-1240.
- Jessen, K. and Hagelaten, J.O. (1978) Peritoneal dialysis in the treatment of profound accidental hypothermia. *Aviat. Space Environ. Med.*, 49, 426-429.
- Keatinge, W.R. (1969) *Survival in cold water*. Blackwell Scientific Publications, Oxford, Great Britain.
- Keatinge, W.R. (1977) Accidental immersion hypothermia and drowning. *Practitioner*, 219, 183-187.
- Lloyd, E.L., Mitchell, B. and Williams, J.T. (1976) Rewarming from immersion hypothermia: a comparison of three techniques. *Resuscitation*, 5, 5-18.
- Marcus, P. (1978) Laboratory comparison of techniques for rewarming hypothermic casualties. *Aviat. Space Environ. Med.*, 49, 692-697.
- Marcus, P. (1979) The treatment of acute accidental hypothermia: proceedings of symposium held at the R.A.F. Institute of Aviation Medicine. *Aviat. Space Environ. Med.*, 50, 834-843.
- Martyn, J.W. (1981) Diagnosing and treating hypothermia. *Can. Med. Assoc. J.*, 125, 1089-1096.
- Miller, J.W., Danzl, D.F. and Thomas, D.M. (1980) Urban accidental hypothermia: 135 cases. *Ann. Emerg. Med.*, 9, 456-461.
- Morrison, J.B., Conn, M.L. and Hayward, J.S. (1979) Thermal increment provided by inhalation rewarming from hypothermia. *J. Appl. Physiol.: Respirat. Environ. Exercise. Physiol.*, 46, 1061-1065.
- Samuelson, T., Doolittle, W., Hayward, J., Mills, W. and Nemiroff, M. (1982) Hypothermia and cold water near drowning: treatment guidelines. *Alaska Med.*, 24, 106-111.
- Severinghaus, J.W. (1959) Temperature gradients during hypothermia. *Ann. N.Y. Acad. Sci.*, 80, 515-521.
- Shanks, C.A. (1975) Heat gain in the treatment of hypothermia. *Med. J. Aust.* 2, 346-349.
- Stine, R.J. (1977) Accidental hypothermia, *JACEP*, 6, 413-416.

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