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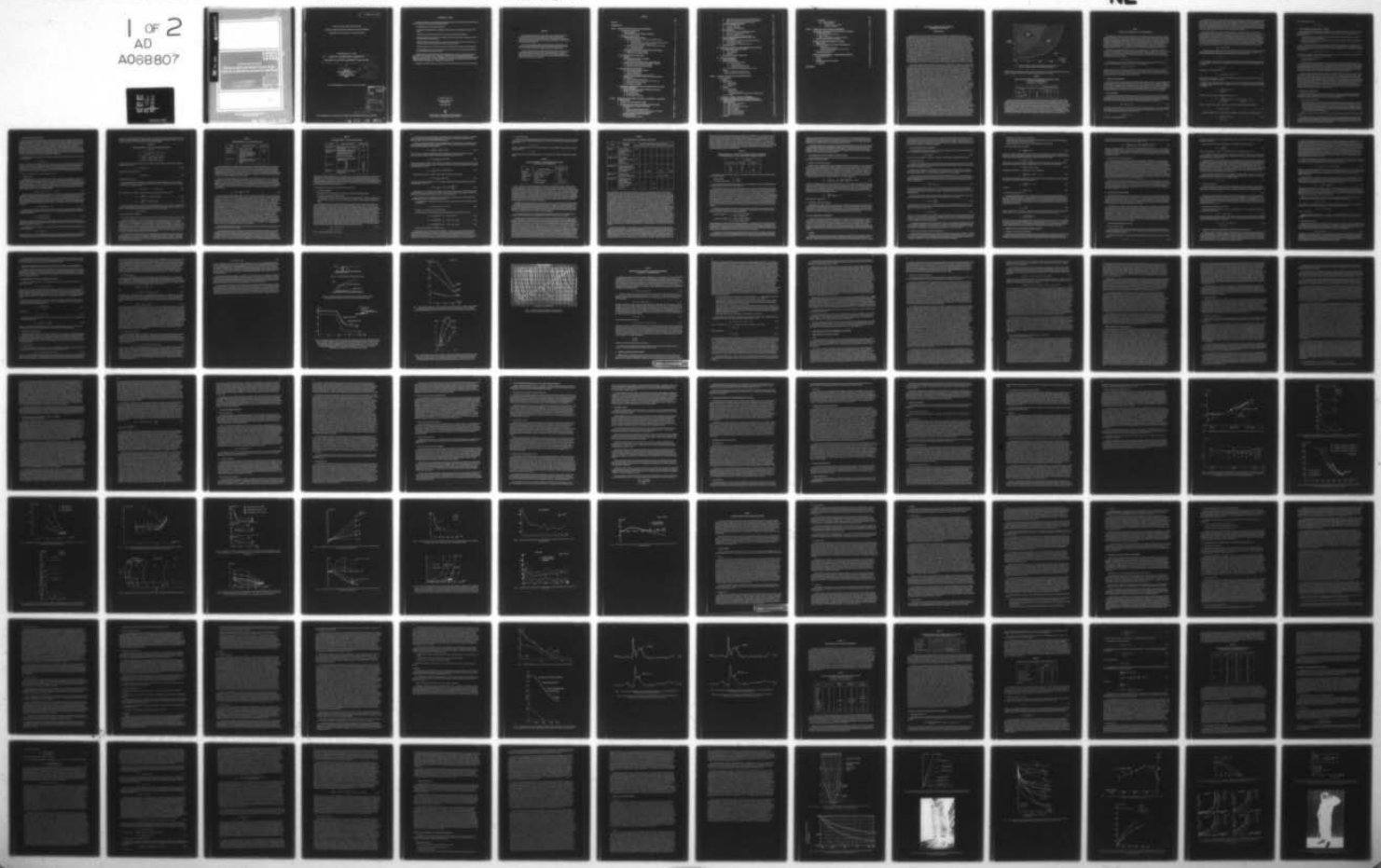

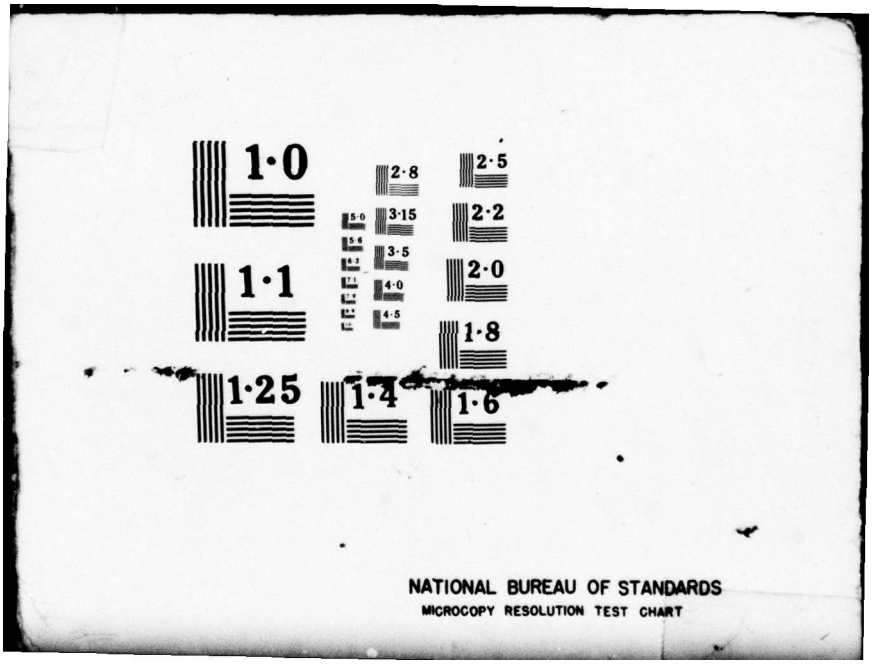
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(6) SURVIVAL AND PROTECTION OF AIRCREW IN THE EVENT OF ACCIDENTAL IMMERSION IN COLD WATER

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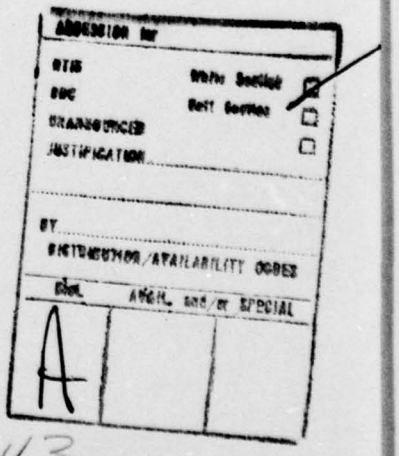

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PREFACE

This monograph, which has been prepared at the request of AGARD, reviews the current status of the problem of protecting an aviator who has become immersed accidentally in cold water. It includes clinical aspects of the treatment of hypothermia as well as theoretical aspects of adaptation to cold and provides information of interest both to flying personnel and technicians.

The document incorporates information from many sources and, in particular, the results of experiments conducted at the Laboratoire de Médecine Aérospatiale du Centre d'Essais en Vol. Most of the items of equipment referred to are currently in use.

Thanks are due to Colonel Tatcher, Medical Officer at the Defence and Civil Institute of Environmental Medicine, Donsview, Canada, who provided reports on accidental immersions experienced in the Canadian Army, and to Dr R.F.Goldman, Director of the Military Ergonomics Division, US Army Research Institute of Environmental Medicine, who was good enought to review the manuscript.

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INTRODUCTION

The importance of the heat loss from the human body when plunged into a cold bath has been known to physiologists ever since the work of Lefevre[203] at the end of the last century. This author did in fact carry out a large number of noteworthy experiments in direct calorimetry on animals and human beings, and described the laws governing the cooling of the body until death occurred. Unfortunately, such work remained on a research level only and, up to the time of the Second World War, most deaths which occurred during accidental immersion in water were attributed to drowning. This assumption was so general that, when the Titanic was lost with 1489 deaths, the effect of cold as the main cause of death was not even mentioned in the reports of the accident, although the temperature of the water was close to 0°C. Yet, as Keatinge[181] points out, as early as 1798, James Currie, when reporting the shipwreck of a small boat in the Bay of Liverpool, had made the assumption that the sailors, who were only partly immersed in the water, had probably died of cold. This observation, however, passed unnoticed. The shipwreck of the Lakonia in 1963 off the coast of Madeira in a calm sea with a temperature of 17 to 18°C is a good illustration of the danger represented by cold water; when help arrived, three hours after the shipwreck, out of the 200 people who were thrown into the water, 113 were found to be dead, floating in their life belts. The descriptions given by the survivors and the rescue workers pointed to hypothermia as the main cause of death. This incident shows that the survival time is only three hours for more than 50% of the people in water at 18°C, a temperature which is considered pleasant on our coasts. Now, 66% of the surface of the oceans has a temperature of less than 25°C, and 47%, a temperature below 20°C. In our temperature zones, the water temperature can be considered to be less than 20°C throughout the year, particularly in the Atlantic. In the event of accidental immersion, survival for a man not wearing protective clothing will therefore be limited to only a few hours.

The problem of survival in cold water became acute during the Second World War. The large number of ships which were sunk, or aircraft which were shot down over the sea in all latitudes forced the General Staffs of the countries at war to look into the question of protecting crews. Research was therefore undertaken to design protective equipment, particularly for air force personnel, and to determine survival time in relation to the temperature of the water in order to improve the organisation of rescue operations. In this connection, Molnar[222], using the US Navy's extremely well-documented information on shipwrecks which occurred between 1942 and 1945, published a tolerance curve based on the temperature of the water and the total period of immersion. This curve, in the view even of its author, is a tolerance limit which few men can exceed and which many cannot even reach. Beyond this time and water temperature limit, death can be expected to occur in 100% of cases. It should be stressed that this curve applies to men wearing clothes which vary a great deal, according to the latitude, from a simple pair of shorts to the thick and relatively water proof clothing worn on deck. To supplement his analysis, Molnar also made use of the information gathered on the criminal experiments performed in Dachau, which had just been made public by Alexander[4], and the results obtained by Spealman[269] on volunteer subjects who were immersed, naked up to the nape of the neck in water at 15°C. This enabled him to show the chances of survival in relation to the temperature of the water and the period of immersion. This work was continued by Barnett[21] who published a survival graph in 1962 which is still in use in a number of countries (Fig.1). This graph is divided into three areas: a safe area, in which all those who were shipwrecked survived; a marginal area, in which the chances of survival are not more than 50%, the victims suffering severe hypothermia which renders them unconscious and exposes them to drowning; a fatal area, within which the chances of survival are practically nil. It should be pointed out that these predictions apply to subjects wearing clothes but not an anti-immersion suit. Great care is required when applying these predictions, as shown in the case of the Lakonia disaster, which falls in the "safe area" of this graph. Other studies have been made on tolerance to accidental immersion in very cold water; one of the most important is that conducted by McCance et al.[213], covering 289 cases. Their data, presented in Table 1, show the number of men rescued and the percentage of deaths in the 24 hours following the rescue as a function of the temperature of the water. The Table suggests that there is risk of total hypothermia only if the water temperature is less than 10°C. A more precise analysis of the 160 persons immersed in water below 10°C, made by these authors, shows the extreme diversity of the tolerance times. Thus, nine out of forty men rescued after one hour's immersion in water at −1.1°C (or 22.5%) died within 24 hours on board the rescue ship. On the other hand, ten men out of twelve (or 83.3%) died after being in the water for 30 minutes at a temperature of 5.6°C, and then spending 12 hours in a dinghy, while two men survived a 90-minute immersion in water at 2.8°C followed by 22 hours in a dinghy. It would seem that the difference in the degree of protection by clothing of the shipwrecked victims cannot, alone, account for this wide spread of the tolerance times. Furthermore, the large number of cases of immersion foot and/or hand which were noted by the authors in these accidental immersions underlines the need for effective protection of the extremities when the temperature of the water is less than 10°C. While knowledge of the tolerance to immersion found from reports of accidents provides an a posteriori proof of the value of protective equipment, it does not provide a means of studying the physio-

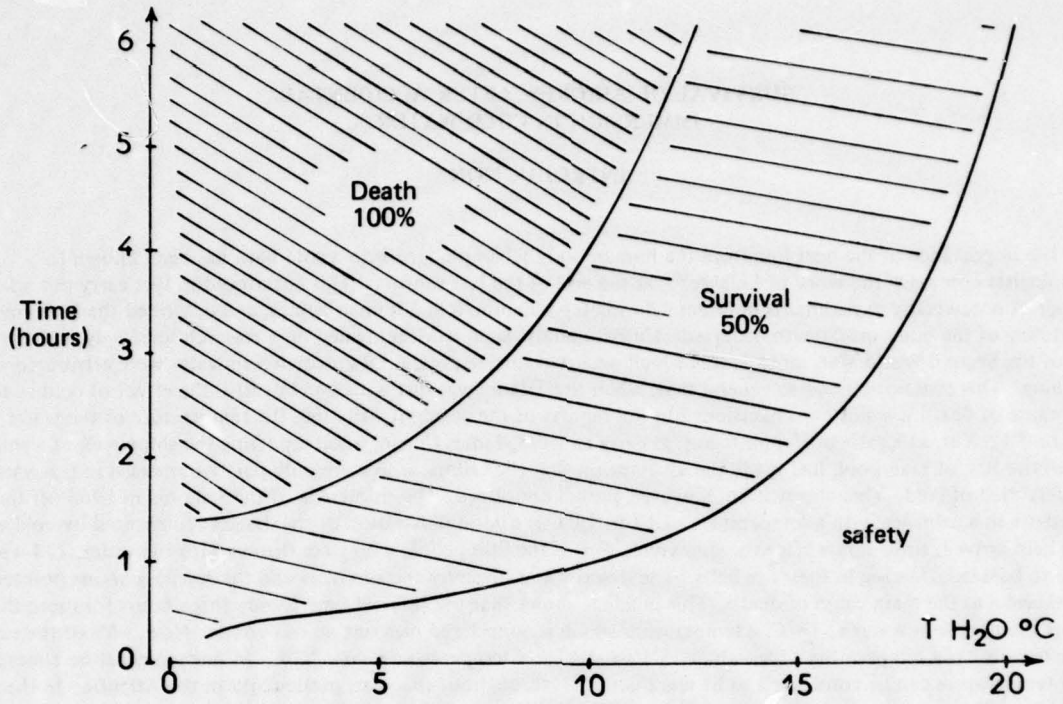

Fig.1 Chances of survival in relation to the temperature of the water in the case of men not wearing an anti-immersion suit (Barnett, Reference 21)

logical reactions of a man, and their variations, as a basis for this tolerance, nor of developing improved protection, since for this, it is necessary to assess the heat exchanges between the body and its environment.

TABLE 1

Number of Men Saved at Different Water Temperatures and Percentage of Deaths within the 24 hours following Rescue

(after McCance et al., Reference 213)

Temperature of the sea (°C)	*Number of men rescued*	*Deaths within 24 hours*	
		Number	*%*
less than 10	160	27	17
10–20	27	0	–
more than 20	102	0	–

During the past twenty years, the increase in intercontinental air traffic, and in overflights of the arctic regions, has exposed crews and passengers with increasing frequency to the risk of accidental immersion in cold water. The problem of affording protection against this danger is therefore becoming one of considerable concern to the aircraft industry. Simultaneously, underwater diving for military and civilian purposes has been increasing. The need to develop a wide variety of protective clothing to meet such requirements stimulated research both from the point of view of the technological aspects of the clothing and from the pathophysiological point of view. In this work on survival after accidental immersion in cold water, an attempt will be made to review the state of the art by considering, in turn, the laws governing heat exchanges, the physiological reactions of the organism to immersion and their limitation, the effects of hypothermia and its treatment, and the basic principles and methods of protection in use at the present time.

Chapter 1

HUMAN HEAT EXCHANGE IN A COLD ENVIRONMENT

After bailing out over the sea, aircrew are first subjected to a more or less prolonged period of immersion, followed generally by exposure to a cold air environment in a life raft. A precise statement of the risks which they run, the men's tolerance of such conditions and the protection needed to ensure their survival, require a knowledge of the various ways in which heat exchanges take place between the body and the surrounding atmosphere, and the laws which govern such exchanges. A full description of these exchanges would be outside the scope of this work; more detailed information can be obtained by consulting such specialist works as: "Physiology of heat regulation and the science of clothing", published by Newburgh (Chapter 3)[138]; "Physiological and behavioural temperature regulation", published by Hardy, Gagge and Stolwijk (Part II)[142]; or physics papers for the use of specialists in heat engineering (Weil[298], Véron[295] ....).

The human body exchanges heat with the environment in four different ways: conduction (K), convection (C), radiation (R) and evaporation (E). During immersion, heat exchange is mainly by conduction and convection; in air, it is by all four ways. Using M to express the metabolic heat production, W the physical work done, i.e. the energy expended in the outside environment, and S, the storage or loss of heat by the body tissues, the thermal balance sheet of the organism can be written as follows:

$$M - W - E \pm R \pm C \pm K = \pm S. \tag{1.1}$$

The components of this balance sheet represent heat flux densities and are usually expressed in W/m^2. If the organism is in stable conditions, S is zero and Equation (1.1) can be rewritten as:

$$\pm R \pm C \pm K = M - W - E, \tag{1.2}$$

in which $(\pm R \pm C \pm K)$ is the external heat stress and $(M - W)$ the internal heat stress. Evaporation (E) is always a loss of heat from the body; the sources of this heat loss are the sweat production which is the body's response to an excess of heat production (M) over heat loss and respiration, while M, normally the energy expenditure of the organism to carry out the work W, is increased when heat loss is excessive.

I. EXCHANGE BY CONDUCTION

Conduction is a transfer of heat through a physical medium without movement of matter. The heat is transmitted by degrees through a liquid, solid or gaseous medium by direct interaction of adjacent molecules. In a liquid or a gaseous medium however, conduction is rarely a pure phenomenon; indeed, in the majority of cases exchanges by conduction and by convection occur simultaneously, and the part played by conduction decreases with an increase in the velocity of the fluid. In solid media, on the other hand, conduction is the only method of heat transfer. This occurs, for example, through the various layers of a protective suit or when the skin comes into contact with colder or warmer objects.

1. Laws of Conduction

In a medium with uniform physical properties, the flow of heat from the warmer surface to the colder surface is in direct proportion to the contact area, to the thermal conductivity of the medium and to the difference in temperature being measured between the two areas; it is inversely proportional to the distance between the two areas and thus to the thickness of the material. In the steady state, this heat flux is expressed by the following relationship:

$$H_K = \frac{k}{d}(T_2 - T_1)\,A \tag{1.3}$$

in which H_K is expressed in watts, k is the thermal conductivity of the medium in $W/m \cdot °C$, d is the distance between the two areas in m, A is the area in m^2, and T_2 and T_1 are the respective temperatures of the two areas in °C. The above relationship is generally expressed as a thermal flux density:

$$K = \frac{k}{d}(T_2 - T_1), \tag{1.4}$$

in which K is then expressed in W/m^2.

The thermal conductivity (k) varies considerably according to the materials used. Metals are very good conductors of heat while air is a very poor conductor. There is no heat exchange by conduction across a vacuum since there is no physical medium. For example, the thermal conductivity of copper (3.75×10^2 W/m·°C is 1.09 times higher than that of silver and 7.3 times higher than that of iron, but it is 365 times better than that of cement, 588 to 760 times better than water (from 0 to 100°C) and 19,000 times better than that of still air. In the case of metals, temperature has practically no effect on thermal conductivity, which, in the case of liquids and gases, increases with temperature; thus the thermal conductivity of water ranges from 0.567 W/m·°C at 0°C to 0.314 W/m·°C at 100°C.

By analogy with Ohm's law for electric current, the expression k/d is called the *thermal conductance* or the *coefficient of heat transfer by conduction* h_k. The latter is expressed in W/m²·°C per metre of thickness of the material. Some authors prefer to use the reciprocal of this coefficient: the thermal resistance or *insulation* by analogy with electrical resistance. This is expressed in °C·m²/W, although another unit is often used, the *clo* coined by Gagge, Burton and Bazett in 1941 (Ref.103). The clo is based on the quantity of insulation required to maintain the thermal balance of a subject with a mean skin temperature of 33.3°C and a metabolic rate of about 58 W/m², who is sitting in a room at 21°C with air movement of 0.05 m/s and a relative humidity of less than 50%. Assuming that 76% of the heat supplied by the subject is lost through his suit, one clo was defined as the insulation which permits the passage of 1.163 W/m² of area with a temperature difference of 0.18°C between the inner and the outer surface of the suit. It follows that:

$$1 \text{ clo} = 0.155 \text{ °C·m}^2\text{/W}.$$

The clo unit is then a combined convection and radiation heat transfer derivation, which can be applied to conduction, but is not, strictly speaking, a conduction expression at all. Chapter 4 presents the insulation values of some of the materials currently used for making protective clothing. Equation (1.4) can then be written:

$$K = \frac{1}{I}(T_2 - T_1). \tag{1.5}$$

This insulation expression can be used to advantage if heat conduction is effected through several parallel layers of different conductivity. In this case the thermal resistance of the whole is then equal to the sum of the resistance of the various layers:

$$I = I_1 + I_2 + I_3 + \ldots + I_n$$

and the heat flux by conduction will then be:

$$K = \frac{1}{I_1 + I_2 + I_3 + \ldots + I_n}(T_2 - T_1). \tag{1.6}$$

Such an equation is frequently used to calculate heat transfer through several layers of clothing based on the thermal conductivity and the thickness of each layer.

Similarly, a study has been made of heat transfer by conduction in a few simple cases. Thus, for an insulating layer of thickness x covering a cylinder of radius r and length l, it can be shown, by assuming the transverse heat exchanges to be negligible, that:

$$K = \frac{2\pi l}{\ln\left(\frac{r+x}{r}\right)} \times k\,(T_2 - T_1). \tag{1.7}$$

This equation can also be written, multiplying the second term by r/r :

$$K = \frac{2\pi l \times r}{r \times \ln\left(\frac{r+x}{r}\right)} \times k\,(T_2 - T_1) = \frac{k}{r} \times \frac{1}{\ln\left(\frac{r+x}{r}\right)}(T_2 - T_1)\,A \tag{1.8}$$

in which A is the area of the cylinder. The insulation of the protective layer around the cylinder will be:

$$I = \frac{r}{k} \times \ln\left(1 + \frac{x}{r}\right). \tag{1.9}$$

Similarly for a sphere of radius r covered by a layer of thickness x, the heat transfer by conduction will be:

$$K = \frac{4\pi}{\left(\frac{1}{x} + \frac{1}{r}\right)} \times k(T_2 - T_1) \tag{1.10}$$

and the thermal insulation will be:

$$I = \frac{r}{k} \cdot \frac{x}{r+x} . \qquad (1.11)$$

2. Practical Application of Heat Exchange by Conduction

These theoretical data on heat exchange by conduction have been extended to the human body. When it is desired to ascertain the heat transfers which occur within the body, it is in fact convenient to reason on the basis of a two compartment model:

- a central cylindrical core, the temperature of which is comparable with the rectal temperature T_{re} and which produces heat uniformly throughout its mass;
- a periphery through which the heat produced in the core flows and the temperature of which is comparable with the mean skin temperature $\bar{T}_s$.

The flow of heat from the core to the periphery depends on these two temperatures and on a coefficient of heat transfer h_b, commonly known as the *thermal conductance of the body*, expressed in $W/m^2 \cdot °C$. By analogy with the laws of conduction developed above, the heat transfers (H) from the core to the periphery can be determined by means of the following equation:

$$H = h_b (T_{re} - \bar{T}_s) . \qquad (1.12)$$

The coefficient h_b appears to contain only the concept of conduction. In fact, as we shall see in the next chapter, this is a complex coefficient which covers two types of heat transfer:

- by conduction through the tissues;
- by blood convection.

The conduction part is a relatively constant term for a given subject, but is difficult to determine since the thickness of the tissues involved is not generally known. The convection part is a variable element depending on the vasomotor state of the subject, particularly at the periphery. Thus peripheral vasoconstriction reduces conductance, while vasodilatation produces increased conductance. This extremely simplified model of the human body clearly cannot cover all the heat exchanges in the body, and one is therefore often obliged to use more complex models with three compartments, or more, such as Stolwijk's model[272] comprising six segments each of which has four compartments.

Apart from the heat exchanges through clothing which will be further developed in Chapter 4, exchanges by conduction are observed whenever the body comes into contact with a solid. Such exchanges are therefore extremely variable according to the position and the activity of the subject. For example, in the standing position the only contact with the ground is by the feet, usually through the relatively insulating soles of shoes. Exchanges by conduction will thus be small, since the area of exchange is generally small; on the other hand, for a subject who is sitting or lying down, this type of heat exchange will be greater. The area in contact with the medium: e.g. seat dinghy, may be as much as 30 to 40%. In a cold environment, it will therefore be very important to use media with a low thermal conductivity in order to reduce losses by this means as far as possible. The hands and face are also likely areas for heat exchange by conduction; contact with objects with a high thermal conductivity, such as metal components, spectacle frames, metal parts of helmets etc. may produce serious frostbite in a cold environment.

II. EXCHANGE BY CONVECTION

This type of heat exchange occurs with an initial conductive heat exchange with a physical medium and a subsequent transfer of matter, an example being the heat transfer between a wall and a fluid (air or water) at a different temperature with a relative displacement of one of them as compared with the other.

There are two types of convection:

- free or *natural* convection, in which the motion of the fluid is due only to variations in its density as a result of heating or cooling by conduction as a result of contact with a body having a different temperature from its own;
- *forced* convection, when the fluid is brought into motion by an outside cause, e.g. a mechanical cause, unless it is the body which is moving inside the fluid. In accidental immersion, while there may be natural convection, it is more often forced convection because of the currents, the movement of the waves or possibly as a result of the water motion generated during swimming.

In addition, convection is *ordinary* or *dead* when there is no change in state of the fluid. It is *live* if there is a change of state.

Exchanges by convection occur both with gases and with liquids. In the case of accidental immersion, this is the main way in which heat is exchanged when the pilot is in the water, but it also exists when he is in the dinghy. In the latter case, the exchanges are mainly with the surrounding air and may be considerable particularly in the unprotected extremities if there is any wind and if the dinghy is not protected.

1. Laws Governing Convective Exchanges

Studies on the laws governing heat exchange by convection have been undertaken principally for industrial use, in particular by heat engineers. The latter have defined the various ways in which such exchanges take place in a number of relatively simple cases: plates, cylinders or spheres surrounded by a fluid, and they have given calculation formulae which have been verified experimentally. To apply these formulae to the heat exchanges between man and his environment is not, however, easy, since the human body is not a simple form. This has prompted some engineers and physiologists to work with analogue models, generally an assembly of cylinders and spheres, representing more or less faithfully the human body. The majority of physiologists have, however, determined the convective heat exchanges on the basis of physiological quantities (metabolism, physiological temperatures, etc.) which vary according to the ambient conditions. Two methods therefore exist for evaluating these exchanges; one is purely physical and the other introduces the reactions of the organism. Their results will be discussed in detail in turn.

Generally speaking, and by analogy with the exchanges by conduction, convective heat transfers can be expressed in the following form

$$C = h_c (\overline{T}_2 - \overline{T}_1) \tag{1.13}$$

in which C is the flow of heat exchanged by convection in W/m^2; $\overline{T}_2$ and $\overline{T}_1$ are respectively the mean temperature of the surface of the body or of the clothing, according to the experimental conditions, and the mean temperature of the fluid; h_c is the coefficient of exchange by convection and is normally expressed in $W/m^2 \cdot °C$. A good deal of research has been done on the coefficient h_c, since it characterises convective exchanges. Relatively low in the case of natural convection in air, it can reach very high values in forced convection in air and extremely high values in water.

1.1 Study of the Coefficient h_c in the Case of Natural Convection

1.1.1 Theoretical study

Comparing Equation (1.13) with the equation for calculating exchanges by conduction (Equation (1.4)), it will be seen that h_c performs the same function as the term k/d in which k is the thermal conductivity of the material through which the conductive exchanges occur and d is its thickness. In the case of convection, the drop in temperature occurs the whole length L of the body, if the latter is vertical; if, on the other hand, it is horizontal, this fall in temperature will depend on the diameter. To show the actual contribution made by convection, it is useful to compare the quantity of heat exchanged by convection with that exchanged by conduction by examining the ratio C/K. This ratio, called the Nusselt number (Nu), is a nondimensional number:

$$Nu = C/K = h_c \times L/k \quad \text{or} \quad h_c \times D/k \,. \tag{1.14}$$

The Nusselt number depends on the nature of the fluid, and the manner in which conductive heat and mass transfers are effected, i.e. it depends on the thermal diffusivity ($\alpha = k/\rho \times c$) where ρ is the density in kg/m^3 and c is the specific heat at constant pressure, in $J/kg \cdot °C$). The units for α are m^2/s. It thus depends on the dynamic viscosity ν which is expressed in m^2/s and also characterizes the movement of matter in the fluid. The dynamic viscosity ν is the ratio of the absolute viscosity of the fluid, μ, expressed in $kg/s \cdot m$ to its density ρ. In convective exchanges the ratio of the dynamic viscosity to the thermal diffusivity is used; this ratio, a nondimensional number, is known as the Prandtl number (Pr):

$$Pr = (\mu/\rho)/((k/\rho) \times c) = \nu/\alpha \,. \tag{1.15}$$

Grashof however observed that, when a fluid was being heated in the vicinity of a wall, the fluid was subjected to a thrust which was partly compensated for by the viscosity forces; the dimensionless Grashof number (Gr) provides a comparison of these forces:

$$\begin{aligned} Gr &= g \times \beta \times \Delta T \times L^3/\nu^2 \\ \text{or} \quad Gr &= g \times \beta \times \Delta T \times D^3/\nu^2 \end{aligned} \tag{1.16}$$

according to the position of the body (vertical: L, horizontal: D) in the water. In both the above equations g is the acceleration due to gravity in m/s^2, β is the coefficient of expansion for the gases only, in s/K, and ΔT is the temperature difference between the wall and the fluid.

The Nusselt number and, consequently, h_c therefore appear as a function of the Prandtl and the Grashof numbers and can be expressed as:

$$Nu = a(Pr \cdot Gr)^n \,. \tag{1.17}$$

An experimental study of the above relationship has been made with many different types of fluids (air, carbon dioxide, hydrogen, water, glycerin, various oils) for bodies of various shapes (spheres, horizontal and vertical wires and tubes,

plates etc.). The various values for the constant were taken at a temperature equal to the arithmetic mean between the wall temperature and the temperature of the fluid. This study made it possible to determine the values of a and n in terms of the product (Pr · Gr). These values are shown in the following Table.

TABLE 1.1

Value of the Coefficients a and n for Various Values of the Product (Pr · Gr)

(Pr · Gr)	a	n
$1 \times 10^{-3} - 5 \times 10^{2}$	1.18	0.125
$5 \times 10^{2} - 2 \times 10^{7}$	0.54	0.25
$2 \times 10^{7} - 1 \times 10^{13}$	0.135	0.33

The coefficient of convective heat transfer can thus be expressed by the general relationship obtained by combining Equations (1.14) and (1.17):

$$h_c = a \times \frac{k}{D} (Gr \cdot Pr)^n \tag{1.18}$$

for bodies in a horizontal position and

$$h_c = a \times \frac{k}{L} (Gr \cdot Pr)^n \tag{1.19}$$

for bodies in a vertical position.

In air, these formulae can be simplified, since the Prandtl number varies only slightly. Its value is in fact 0.76 at −150°C and 0.703 at +100°C. It can therefore be considered to be constant. We then obtain for (Pr · Gr) between 5×10^2 and 2×10^7 the following formula:

$$h_c = 0.49 \cdot \frac{k}{L} \cdot Gr^{0.25} \quad \text{or} \quad 0.49 \cdot \frac{k}{D} \cdot Gr^{0.25} \tag{1.20}$$

Weil[298] suggests even more simplified formulae when the air temperature is in the vicinity of 20°C:

$$h_c = 1.4 \left(\frac{\Delta T}{L}\right)^{0.25} \quad \text{for plates or vertical tubes} \tag{1.21}$$

$$h_c = 1.3 \left(\frac{\Delta T}{D}\right)^{0.25} \quad \text{for horizontal tubes} \tag{1.22}$$

In *water*, the Prandtl number shows much more variation with temperature. From 13.6 at 0°C, its value falls to 7.03 at 20°C; h_c should therefore only be calculated by means of Equations (1.18) and (1.19). While the formula used by Rapp[242] for his cylindrical model in the vertical position in water at 22°C is much simpler:

$$h_c = 0.848\ Gr^{0.25}$$

it is only valid at that 22°C temperature.

1.1.2 Can these Formulae which have been Established for Bodies having Simple Geometric Shapes be Applied to Man?

In air, the use of Equations (1.21) and (1.22) for a subject at rest in an environment at 30°C with a mean skin temperature of 34°C, a body diameter of 0.36 m and a height of 1.75 m gives the following results: for a subject stretched out on a wide mesh netting (i.e. with a maximum area for convective heat exchange) $h_c = 2.37$ W/m²·°C; for a standing subject, $h_c = 1.72$ W/m²·°C. In this model, the body is similar to a cylinder. Rapp[241], using Equation (1.20) applied to a 0.747 m diameter sphere, a shape which corresponds to a sitting subject, finds a value of 2.44 W/m²·°C for a temperature difference of 3.7°C between the surface of the sphere and the air. The theoretical values of h_c are therefore close to each other, but the h_c values determined experimentally on a man by fractional calorimetry, by various authors, show much greater differences as shown in the Table on page 8.

The values quoted by Buettner and Colin et al. are in good agreement with those determined theoretically from a cylinder or a sphere. On the other hand, it is difficult to explain the low values obtained by Hardy et al., whereas the relatively high value found by Winslow can be justified by the slight movements made by his subjects. Thus, it seems that, in still air, the coefficient of heat transfer determined on simple geometric shapes can, in fact be applied to the more complex shape of the human body.

TABLE 1.2

Experimental Values of h_c for Free Convection in Air

Authors	*Experimental conditions*	h_c W/m²·°C
Buettner[60]	– subjects lying on a table	2.5
Hardy et al.[136]	– subjects lying on a net	1.0 to 1.6
Winslow et al.[303]	– sitting subjects making slight movements. Extrapolation of the results to zero wind conditions	3.7
Colin et al[79]	– Subjects lying motionless on a net – Estimated value for sitting subjects	2.7 2.3

In a cold environment, the coefficient of heat transfer increases. Indeed, the Grashof number increases when the ambient temperature decreases as a result of an increase in the density, the thermal diffusivity and the temperature difference between the surface of the skin and the air. In addition, in the case of naked or only lightly clad subjects, shivering creates considerable agitation of the air. Taking a ΔT of 15°C between the skin and the air, for example, by applying Equations (1.21) and (1.22) we obtain values for h_c of 2.4 W/m²·°C for subjects in the vertial position and 3.3 W/m²·°C for subjects in the horizontal position. This latter value is relatively close to that of Winslow and comparable with that calculated by Rapp[241] for a horizontal surface.

In water, h_c for a cylindrical model must be calculated by means of Equation (1.18) or (1.19) according to the position given to the cylinder. It is then necessary to determine the values of the coefficients a and n which affect the product $(Gr \cdot Pr)$. The calculation shows that, for the dimensions of the cylinder quoted above, the value of this product is of the order of 10^9 when the model is in the horizontal position and 10^{10} when it is vertical, for temperature differences between the surface of the cylinder and the water varying from 0.9 to 2.4°C and more. According to Table 1.1, the values of a and n are therefore 0.135 and 0.33 respectively. The equation for calculating h_c can then be written as follows:

$$h_c = 0.135 \frac{k}{D \text{ or } L} (Gr \cdot Pr)^{0.33} . \qquad (1.23)$$

It should however be noted that other values of a and n have been used. Thus Rapp[242], for his cylindrical model of dimensions comparable to those indicated above, in the vertical position in water at 22°C, took the following values for a and n: a = 0.54; n = 0.25. The same applies to Witherspoon et al.[304] but the diameter of their model, based on a weighted mean of the diameters of the various segments of the body, was much smaller (0.144 m). It follows that the product $(Gr \cdot Pr)$ is less than 2.10^7. However, the most important difference in our opinion between these authors would appear to relate to the evaluation of the temperature difference between the surface of the cylinder and the water (ΔT). Rapp, for example, found by calculation a temperature difference of 1.79°C for his cylinder in the upright position in water at 22°C, while the mean temperature difference which we have measured on our subjects at the same water temperature was 2.2°C. For Witherspoon this ΔT, derived from measured values on nude subjects, was only 0.7°C in water at 20°C after an hour. In view of these differences in the data, it is not surprising to find divergent values in the results of the theoretical calculation of h_c by different authors. Thus, in water at 20°C, h_c is stated to be 242 W/m²·°C for a horizontal cylinder 0.36 m in diameter and 1.75 m long with a ΔT of 2.4°C, and 127 W/m²·°C for the same cylinder in the vertical position and the same ΔT. The latter value is relatively close to that suggested by Rapp (94 W/m²·°C), but, on the other hand, very different from that calculated by Witherspoon: 165.2 W/m²·°C after an hour in water at 20°C. Furthermore, Rapp points out that, according to Elenbaas, there is a stagnant layer of water underneath the laminar boundary layer, called a Langmuir film, which requires a conductive resistance acting in series with the convective resistance. If the presence of this layer is taken into account, the value for the overall conductance would be 16.3 W/m²·°C, which would give a ΔT of 6.4°C in water at 22°C. According to the same author the true values of the heat transfer coefficients are between 94 and 16.3 W/m²·°C.

An equally large dispersion exists between the experimental results obtained by fractional or direct calorimetry on human subjects, as shown in the Table on page 9.

The main cause for this dispersion would appear to be the method of measuring skin temperatures, the precision of a surface temperature measurement in water, and the simultaneous use of fluxmeters, as we shall explain when discussing forced convection. It should also be noted that the values of Gee et al., as well as our own values, show that h_c increases in cold water. This increase is greater than that predicted by the product $(Gr \cdot Pr)$ and is due mainly to agitation of the water by shivering. In a recent work, we calculated on the basis of our experimental results the relationship $\log Nu = \log (Gr \cdot Pr)$ in order to determine the coefficients a and n in Equation (1.23) (Boutelier et al.[51]). The

TABLE 1.3

Experimental Values of h_c in Free Convection in Water

Authors	*Experimental conditions*	h_c (W/m²·°C)
Goldman et al.[122]	Measurements on copper manikin in vertical position	46
Boutelier et al.[42] Colin et al.[80]	Subjects naked, immersed to the neck, stretched out in water at thermal neutrality (33°C)	44
	Cold water – with shivering	61
Gee et al.[110]	Measurements on human subjects using fluxmeters:	
	35°C	38
	32°C	96
	28°C	208
	24°C	358
	20°C	537
Nadel et al.[225]	Measurements on human subjects using fluxmeters	230

respective values of these coefficients were: $a = 0.09$ and $n = 0.275$, if the body is approximated by a cylinder 0.36 m in diameter and 1.75 m long. These coefficients are relatively close to those of Weil when the product $(GR \cdot Pr)$ is between 2.10^7 and 1.10^{13} (Table 1.1), but are less than Weil's values. This would lead to the assumption that the body is not perhaps absolutely comparable with a cylinder, either because of its position in the water or, more probably, because the area for the transfer of heat by convection in cold water is certainly less than the total surface area of the body due to the fact that the limbs are kept close to the body.

1.2 Investigation of the Coefficient h_c in the Case of Forced Convection

1.2.1 Theoretical investigation

The coefficient of heat transfer depends on a large number of factors, including the following:

- the distance covered by the fluid along the wall;
- the characteristics of the fluid: thermal conductivity (k), density (ρ), viscosity (ν), velocity (v), temperature (T);
- the characteristics common to the wall and to the fluid and in particular, the angle of attack of the wall by the fluid.

Of all these factors the velocity of the fluid is of particular importance. Indeed, a study of the movements of the fluid in the vicinity of a plate shows that the velocity does not suddenly pass from 0, the velocity at the wall, to the velocity corresponding to the mean fluid velocity. The same is true if the fluid is flowing in a tube. This fact prompts the thought that there is a boundary layer in which the velocity of the fluid varies. The mechanism underlying the formation of this boundary layer is important, since temperature variation, and consequently thermal resistance, is concentrated at this spot. The thicker the boundary layer, the greater the thermal resistance, i.e. the smaller the coefficient of heat transfer. The extent and the constitution of the boundary layer vary according to the type of flow. A study of the velocity distribution of the fluid in the vicinity of the wall of a body has led to a distinction being made between two types of flow: laminar flow and turbulent flow. Figure 1.1 shows that in laminar flow conditions, the velocity distribution in the boundary layer is parabolic, while in turbulent flow conditions it obeys a more complex law despite the existence of a laminar sub-layer. The distinction between these two types of flow depends on the characteristics of the fluid: its density, velocity, absolute viscosity, diameter or length of the wall on contact with which the fluid flows according to the position of the body as compared with the direction of movement of the fluid. A nondimensional number covering all these characteristics has been defined, the Reynolds number (Re), such that:

$$\text{Re} = \rho \times v \times D \text{ or } L/\mu \qquad (1.24)$$

$$\text{or} \quad \text{Re} = v \times D \text{ or } L/\nu$$

where ν is the dynamic viscosity in m^2/h $(= \mu/\rho)$.

It can be seen that the flow pattern changes when the Reynolds number is fairly large. In principle, it is difficult to observe a laminar pattern for Reynolds numbers higher than 2,100; after a not clearly defined transient zone, for Reynolds numbers of more than 3,000 the flow becomes turbulent.

Numerous formulae have been put forward for calculating the Nusselt number and, from it, the coefficient of heat transfer in laminar or turbulent conditions. According to Hardy[138] in air, the equation which gives the best agreement with the experimental results has the following form:

$$h_c = \frac{k}{D \text{ or } L} (1 + a\, Re^{0.5} + b\, Re) \tag{1.25}$$

in which a and b are constants depending on the units used, irrespective, it would seem, of the flow conditions. However for laminar conditions according to Pohlausen the following equation should be used:

$$h_c = 0.664 \times \frac{k}{D \text{ or } L} \times Re^{0.5} \times Pr^{0.33} . \tag{1.26}$$

This formula was adopted by Rapp[242] for his model of water immersion, for Reynolds numbers of the order of 8.6×10^5, which were thus distinctly turbulent conditions. Witherspoon[304] used Fand's formula, which applies when the Reynolds number is between 10^1 and 10^5:

$$h_c = \frac{k}{D \text{ or } L} \times (0.35 + 0.56\, Re^{0.52})\, Pr^{0.33} . \tag{1.27}$$

When the flow is turbulent, in the case of tubes, Colburn's formula can be used for gases:

$$h_c = 0.023 \times \frac{k}{D \text{ or } L} \times Re^{0.8} \times Pr^{0.4} \tag{1.28}$$

for Reynolds numbers between 10^4 and 1.2×10^5. For liquids the Michejew formula seems preferable for Reynolds numbers between 10^4 and 5×10^6:

$$h_c = 0.021 \times \frac{k}{D \text{ or } L} \times Re_f^{0.8} \times Pr_f^{0.43} \times \left(\frac{Pr_f}{Pr_p}\right)^{0.43} . \tag{1.29}$$

The subscripts f and p indicate that the physical constants are taken respectively at the mean temperature of the fluid (subscript f) and of the wall (subscript p). However, this is used only when the fluid temperature is very different from that at the wall, which is not the case for a human body plunged into water.

In air, these formulae can be simplified, since the Prandtl number is practically constant; Rapp[241] suggests using the following equations taken from Jakob, McAdams and Kreith:

– for a horizontal plate, corresponding for example to a man fully stretched out, with a wind parallel to the major axis of the body:

$$h_c = 0.595 \times \frac{k}{L} \times Re^{0.5} \quad \text{for} \quad Re < 10^5 \tag{1.30}$$

the length L to be used for the man would be 0.305 m;

– for a vertical cylinder, corresponding to a standing man, perpendicular to the direction of the wind:

$$h_c = 0.615 \frac{k}{D} Re^{0.466} \quad \text{for} \quad 40 < Re < 4 \times 10^3 \tag{1.31}$$

$$h_c = 0.174 \frac{k}{D} Re^{0.618} \quad \text{for} \quad 4 \times 10^3 < Re < 4 \times 10^4 \tag{1.32}$$

$$h_c = 0.0239 \frac{k}{D} Re^{0.805} \quad \text{for} \quad 4 \times 10^4 < Re < 4 \times 10^5 \tag{1.33}$$

the value of the body diameter D would be 0.33 m.

An examination of these formulae will show how difficult it is to calculate h_c in forced convection conditions, mainly because of the lack of precise information about the flow conditions, particularly in the case of a shape as complex as that of the human body. It can however be noted that h_c is a function of $Re^{0.5}$, and therefore of $v^{0.5}$ (Equation (1.24)) in laminar flow, and of $v^{0.8}$ in turbulent flow. This is however applicable only to tubes or absolutely smooth wires; in the case of the human body, the value of the exponent which affects v or Re may be different.

1.2.2 Experimental results

Relatively few experimental determinations of the coefficient of heat transfer in forced convection in air have been made on a human subject. Most authors have expressed h_c as a function of the velocity of the wind in the following general forms:

$$h_c = a + b\,v^n$$

in which a represents the values of h_c in free convection and $b\,v^n$ is the coefficient for forced convection, or alternatively in the form:

$$h_c = b'\,v^{n'}$$

which can be used in forced convection conditions only. A number of authors' results are contained in the following Table:

TABLE 1.4

Experimental Formulae for h_c Obtained During Tests on Humans in Forced Convection Conditions in Air

Authors	*Experimental conditions*	h_c (W/m²·°C) = f(v) (v in m/s)
Buettner[60]	Subjects stretched out on a table	$7.3\ v^{0.5}$
Winslow et al.[302]	Subjects sitting on a chair	$10.7\ v^{0.5}$
Nelson et al.[229]	Subject standing	$8.7\ v^{0.5}$
Hall[127]	Manikin	$11.6\ v^{0.8}$
Colin et al.[79]	Subjects stretched out	$2.7 + 8.7\ v^{0.67}$
	Subjects standing	$2.7 + 6.5\ v^{0.67}$

The h_c values found with the experimental formulae differ fairly considerably: Buettner's equation gives the lowest values and those of Colin et al. the highest values. This can be explained, at least partly, by differences in the area of exchange, Buettner's subjects being stretched out on a table while Colin's subjects were lying on a large mesh bed which allowed convective exchanges over a larger portion of the area of the body. As h_c is generally expressed in watts per degree and per square metre of the total area of the body, the smaller the area involved in the convective exchange, the smaller the value of h_c. A similar observation can be made when comparing Colin's results with those of Winslow whose subjects were seated, thus exposing only 65% of their body area to convective heat exchanges. An examination of the experimental formulae in Table 1.4 also shows that in the majority of cases the value of the exponent affecting the velocity of the air is 0.5. This would seem to indicate that in spite of Reynolds numbers of more than 3.10^3, the air flow surrounding the body can be considered to be laminar.

Application of the theoretical formulae (1.26) and (1.28) or (1.30) to (1.33), depending on the Reynolds number, to the cylindrical model representing the human body described when investigating free convection, gives much lower h_c values than those found experimentally, particularly when the flow is parallel to the major axis of the body. To account for the experimental values, a very small sized model would be required, as indicated by Plummer (quoted by Winslow[302]). This author has in fact shown that, for a wide range of wind velocities, the human body's heat losses by convection are comparable to those of a cylinder only 0.07 m in diameter.

In water which is in motion, very few experimental determinations have been made of the coefficient of heat exchange, and their values are even more dispersed than in air, as will be seen from Table 1.5 on page 12.

The main causes for these divergences, apart from the variation in the position of the body in the water, must be attributed to the different methods of measurement used by the authors to determine convective heat losses and the average skin temperature. It appears that the convective heat losses measured direct by means of heat fluxmeters placed at various spots on the body (Gee et al.[110], Nadel et al.[225]) are generally higher than those determined by fractional calorimetry. Thus, in still water at 33°C, Gee gives a value for C of about 80 W/m² after 60 minutes of immersion, while metabolism measured in the same ambient conditions by several authors (Carlson et al.[70], Keatinge[181], Boutelier[44], Smith et al.[264]) is approximately 50 W/m². Similar observations can be made at other water temperatures. It is difficult to justify such a difference between heat losses and the production of heat by physiological considerations, particularly in water at 33°C, since the existence of a heat debt in such conditions is in contradiction with the absence of variation in the rectal and skin temperatures observed experimentally. These differences between direct measurement of convective heat losses and their determination by fractional calorimetry can, in our view, be at least partially explained by the fact that the fluxmeter forms an insulating layer on the skin. The temperature of the lower face of the fluxmeter is therefore higher than the temperature of the surface of the skin in contact with the water. The temperature of the upper

TABLE 1.5

Experimental Valules of h_c in Relation to Water Velocity

Authors	*Experimental conditions*	h_c *in* W/m²·°C *for different water velocities in* m/s						
		0.05	0.10	0.15	0.25	0.50	0.75	0.80
Lefevre[201]	Direct calorimetry Water agitated between 5 and 30°C (calculated value)	64						
Goldman et al.[122]	Water agitated Measurements on copper manikin Estimated value	59						
Witherspoon[304]	Measurements on copper manikin, using fluxmeters Subjects perpendicular to the current			206		588		1434
Nadel et al.[225]	Measurements on swimmers with fluxmeters					430	430	
Boutelier et al.[51]	Fractional calorimetry Subjects naked Current parallel to the major axis of the body Thermal neutrality	63	81		147			
	Cold water		111		201			

face of the fluxmeter, on the other hand, varies little, as it is affected by the temperature of the water, the thermal conductivity of which is high. This results in an increased temperature difference between the two surfaces of the fluxmeter. The latter, functioning like a thermopile, will therefore give a higher thermal flux reading than is in fact the case. It would therefore seem that, in the particular case of immersion, certain precautions must be taken when using fluxmeters. The same problem exists, to a lower degree, in the surface measurements of skin temperature, per se. This appears to be the most important cause of the divergences in the experimental determination of h_c in water. Several authors measure skin temperature by fixing temperature pick-ups on the skin with a high thermal conductivity adhesive tape. Now, by comparing the measurements made with thermocouples protected by a permeable insulating layer with those provided by thermocouples implanted in the surface part of the skin, with the tip just touching the surface, it has been shown (Boutelier[44]), that this protection was absolutely necessary (Fig.1.2). The preponderant effect of the temperature of the water due to the high thermal conductivity of this medium, as compared with that of the skin, is thus avoided. In spite of this disagreement between authors in regard to the h_c values, the experimental studies show that the coefficient of heat exchange in water is affected by physiological and morphological factors. Gee et al.[110] (Table 1.3), and Witherspoon et al.[304] observe in fact that the coefficient h_c is higher in cold water than in the vicinity of thermal neutrality. They attribute this increase to the effect of shivering which causes additional agitation of the water and disturbs in particular the boundary layer. This is clearly shown in Figure 1.3 taken from the experimental results obtained on one of our subjects. It will be seen, in particular, that h_c remains constant or even decreases slightly in the water temperature range from 34 to 25°C for still water, and from 34 to 30 or 31°C for water in motion. In this area there is no visible shivering even after two hours of immersion; the heat regulating reactions are almost entirely provided by vasoconstriction of the skin, and a reduction in the convective heat exchange area by keeping the limbs close to the body. It is probably to this last, purely instinctive, reaction that one can attribute the slight decrease in h_c observed in Figure 1.3 for water velocities of 0.1 and 0.25 m/s.

A study of the relationship $C = f(\Delta T)$ carried out on 17 subjects at different water temperatures and velocities enables this effect of shivering to be stated precisely (Boutelier et al.[51]) (Fig.1.4). The slopes of the relationships obtained express a mean coefficient of heat exchange for all the subjects (Table 1.6). In these experiments, the subjects, wearing bathing trunks, were stretched out in the flow so that the current was parallel to the major axis of the body. As in the previous example, it was noticed that, at each of the water velocities, the slope of the curve remained constant and equal to its thermo-neutral value up to a certain value of C (70 to 80 W/m²). Thereafter it varied suddenly, while the temperature difference remained practically constant, and reached a new mean value which was higher than the first,

after a level of losses by convection of between 95 and 110 W/m². This variation in h_c can be explained as follows: when heat losses in water are less than 70 or 80 W/m², shivering does not occur or is very slight and h_c remains equal to its value at thermal neutrality. This corresponds to the water temperature range between 34 and 25°C, or 34 and 30°C depending on the velocity of the water. For heat losses of more than 70 to 80 W/m², h_c varies as a result of shivering; this disturbs the boundary layer and changes the type of convection from free to forced convection if the subjects are in still water; if the water is in motion, the type of flow changes from laminar to turbulent. This variation is rapid and can occur as a result of a very small increase (25 to 30 W/m²) in the heat loss since the boundary layer in water is very thin. Taking into account the high value of the coefficient of exchange, there is very little variation in the temperature difference between the skin and the water.

TABLE 1.6

Experimental Values of h_c in the Absence of Perceptible Shivering and with Shivering, for Unclothed Subjects in a Current of Water Parallel to the Major Axis of the Body

	h_c in W/m²·°C	
Water velocities	*No shivering*	*Shivering*
0	43	53
0.05	63	–
0.10	81	111
0.25	147	201

From the above values it is possible to express h_c as a function of the velocity of the water. The following expressions have been obtained:

– Without shivering: $\bar{h}_c = 273\ v^{0.5}$

– in cold water, with shivering: $\bar{h}_c = 497\ v^{0.65}$

It can be seen that these values obtained for water in the absence of shivering, are about 24 times higher than those determined by Winslow and by Colin et al. in air (Table 1.4).

This study also showed the effect of certain morphological factors on the value of the coefficient of heat exchange. Thus, for a given water velocity and temperature, the coefficient h_c is a function of the inverse of the thickness of the sub-cutaneous fat; it will therefore be higher for a thin subject than for a fat subject in the same conditions. This increase is important, since in water at 26°C and a velocity of 0.10 m/s, h_c may be 1.5 times higher for a thin subject with a 4.5 mm skin fold, than for a fat subject with a 20 mm skin fold. The decrease in the body diameter does not explain this difference: it can perhaps be attributed to greater agitation of the boundary layer by the thin subject, for whom shivering is more intense. The use of a mean value for h_c to predict thermal losses in a given subject may therefore lead to serious errors, particularly at low water velocities. In addition, from a practical point of view, it is easy to understand why the thin person has a much lower tolerance to cold water than a fat subject. Indeed, not only is there less peripheral insulation from the sub-cutaneous fat, but the more pronounced shivering causes an increase in the skin temperature as well as in the h_c. As a result, the convective heat loss for a thin subject may easily be double that for a fat subject in the same conditions.

The application of the theoretical formulae to the cylindrical model described above gave much higher values of h_c than those determined experimentally. If, in fact, we expressed h_c as a function of the velocity of the water, using Equations (1.26) and (1.28) we obtain the following relationships:

– laminar flow conditions: $h_c = 595\ v^{0.5}$ at thermal neutrality

$h_c = 502\ v^{0.5}$ in water at 24°C

– turbulent flow conditions: $h_c = 1782\ v^{0.8}$ at thermal neutrality

$h_c = 1703\ v^{0.8}$ in water at 24°C

These equations are very different from those obtained experimentally. A cylindrical model 8.76 m long and 0.07 m in diameter would have to be used, with the water flowing parallel to the major axis of the body, to make the theoretical results agree with the experimental results; the diameter of this model is the same as that suggested by Plummer for air. It will be noted, in particular, that the coefficient of exchange is lower in cold water than at thermal neutrality, whether the flow is laminar or turbulent, because the model cannot show the effect of shivering.

A comparison of the experimental and the theoretical equations indicates that the flow is laminar in conditions of thermal neutrality ($v^{0.5}$), in spite of the high Reynolds numbers. In cold water, as a result of shivering, the flow would be somewhere between laminar and turbulent ($v^{0.65}$ instead of $v^{0.8}$). This difference in regard to the flow conditions

assumed from the Reynolds numbers may be due to the shape of the body. The very irregular shape probably causes a considerable reduction in the flow velocity of the water in the vicinity of the skin, and thus a decrease in the Reynolds number and an increase in the boundary layer, particularly in certain areas of the body: armpits, groin, lumbar region, popliteal hollow etc., leading to lower h_c values than those predicted by theory.

The comparison made between theoretical data and experimental results throughout this study shows how difficult it is to assimilate the human body to a simple geometrical shape in order to determine the extent of the convection heat exchanges. Further, the reactions of the organism to cold which modify certain physical characteristics of exposure, particularly the velocity of the fluid as a result of shivering, make the problem even more complex, so that its solution can only be found by carrying out systematic experiments.

2. Other Applications of the Laws Governing Convective Heat Exchanges

The heat exchange by convection does not occur solely between the skin and the environment, but is also found in the respiratory and circulatory systems.

2.1 Respiratory Convective Heat Exchanges

Gases are inhaled at a temperature (T_i) which is generally lower than the body temperature. They are rewarmed in the respiratory tract and lungs before being exhaled at a temperature (T_e) which is higher than (T_i). Thus, appreciable heat transfer takes place between the body and the inspired gases, and the body is cooled by this means. The extent of the heat transfer depends on the temperature of the inspired air and the respiratory rate. For a subject resting in a comfortable environment, this type of heat loss is not very great. In a cold atmosphere on the other hand, if respiration is greatly increased as a result of shivering or exercise, respiratory heat loss may become considerable, e.g. of the order of 8-10 W/m² of body surface area at an external temperature of −10°C with a rate of ventilation of 30 l/mn. The heat exchanges by respiratory convection are determined by means of the following equation:

$$C_{res} = Mc_p \left[\dot{V}_{BTPS} \times \frac{1}{22.4} \times \frac{273}{310} \times \frac{P_B - 47}{760} \right] (T_e - T_i)/A_D \qquad (1.34)$$

in which: C_{res} is expressed in W/m², Mc_p is the molar heat of air (22.19 J/°C), $\dot{V}_{BTPS}$ is the ventilation of the subject in dm³/s, 22.4 the molar volume in dm³, P_B the barometric pressure and 47 the water vapour pressure of the alveolar air in mm Hg, T_e and T_i are respectively the temperature of the inspired and expired air and A_D the Du Bois surface area which is 1.8 m² for an average man. The terms in the first brackets express the number of moles of expired gases in standard conditions of temperature and pressure. At sea level, the barometric pressure is approximately 760 mm Hg and the Equation (1.34) can be simplified and becomes:

$$C_{res} = \frac{0.82}{A_D} \times \dot{V}_{BTPS} (T_e - T_i) \qquad (1.35)$$

with C_{res} in W/m², $\dot{V}_{BTPS}$ in dm³/s.

2.2 Circulatory Convective Heat Exchange

Inside the body, the blood, in addition to its nutritive functions, also serves to transport heat and the blood circulation system plays a very important part in temperature regulation. It ensures a relative homogeneity of the deep body temperatures, and vasomotor adjustments of blood flow between core and skin help to maintain a constant temperature of the central core. From this point of view, the peripheral vasomotor phenomena, particularly those in the skin, play a special role by substantially modifying the value of the thermal conductance (h_b) within the body.

III. HEAT EXCHANGE BY EVAPORATION

This type of heat exchange is particularly important in a hot environment when there is sweating. In a cold environment, evaporative heat loss is generally reduced to the minimum, evaporation of the insensible diffusion of moisture through the skin and evaporation of water from the respiratory tract. In the case of accidental immersion, evaporative heat loss may occur during drying of the clothes of the victims after they have got into a dinghy. However, if the previous case is excepted, this type of exchange is not as important as the heat loss by convection and it is therefore not proposed to consider it as fully as the other types, but merely to state general principles. More detailed information can be obtained by consulting works or articles such as those of Hardy[138], Burton[58], Rapp[241], Sibbons[259], Kerslake[182], Snellen et al.[265].

1. Definition

Evaporation is a change of state at the free surface of a liquid which converts matter from the liquid to the gaseous state. The evaporative process consists of three phases: conversion from the liquid to the gaseous state at constant temperature; cooling of the saturated vapour of the surface temperature of the body at ambient temperature; expansion

of this vapour as it passes from the saturation pressure to the ambient water vapour pressure. Each of these three phases requires additional heat; if there is no other heat source the temperature of the liquid falls. This phenomenon is used by the organism to lose heat during sweating. The rate at which evaporation occurs depends on whether the water vapour can be diffused in the ambient air, and therefore on the vapour pressure in the atmosphere. Thus, the term "evaporation" actually covers two phenomena: vaporisation and diffusion.

2. Laws Governing Heat Transfer by Evaporation

The quantity of heat lost through evaporation (E) is equal to the product of the mass of water evaporated per unit time ($\dot{m}$) multiplied by the total heat of evaporation (λ):

$$E = \dot{m} \times \lambda / A \tag{1.36}$$

in which E is expressed in W/m^2, $\dot{m}$ in kg/s and λ in kJ/kg, A being the surface at which evaporation occurs.

2.1 Total Heat of Evaporation

This has three components associated with the physical phenomena constituting the evaporative process. We thus have the latent heat of vaporisation (λ_1), the heat required to cool the vapour at ambient temperature (λ_2) and the heat required for the expansion of the vapour (λ_3).

The latent heat of vaporisation (λ_1) is the quantity of heat required to convert the unit mass of water in the liquid phase to the vapour phase at a given temperature. It varies with temperature, decreasing when the temperature rises. Thus for example at 20°C, λ_1 is equal to 2,454 kJ/kg, and at 30°C, λ_1 is 2,430 kJ/kg.

Cooling the vapour at ambient temperature absorbs heat rather than liberating it. This is due to the fact that the volume of the saturated vapour is greater at ambient temperature than at the temperature of the surface of the body. The method of calculating this heat consists in using the entropy values for the water vapour given in tables in accordance with the following equation:

$$\lambda_2 = \left(\frac{\bar{T}_s - T_a}{2}\right)(S_2 - S_1) \tag{1.37}$$

in which λ_2 is the heat required to cool the vapour, $\bar{T}_s$ is the absolute temperature (degree Kelvin) of the surface, T_a is the absolute temperature of the environment, S_2 and S_1 are the entropy values of the water vapour at ambient temperatures and at the temperature of the surface respectively. This heat represents approximately 1.5% to 2% of the heat of vaporisation.

Finally, the heat required to *expand* the vapour at ambient temperature from the saturation vapour pressure to the ambient vapour pressure can be deduced from Boyle's and Gay-Lussac's laws, and can be written:

$$\lambda_3 = R_w \times T_a \ln \frac{P_{ws}}{P_{wa}} \tag{1.38}$$

R_w being equal to R/M in which R is the universal gas constant and M the molecular weight of the gas, T_a is the absolute temperature of the environment, P_{ws} the saturation vapour pressure and the P_{wa} the ambient water vapour pressure. This equation can also be written:

$$\lambda_3 = R_w \times T_a \ln \frac{1}{\varphi_s} \tag{1.39}$$

in which φ_s is the relative humidity of the environment expressed as a percentage of the saturation vapour pressure at the temperature T_a. A study of the heat exchanges of a completely wet surface, using a wet bulb thermometer, shows that the heat of expansion is taken from the environment and not from the object. It can be seen that the lower the ambient humidity, the higher the heat required for expansion.

The total heat of evaporation is the sum of these different factors. The experimental determinations made by some authors: Snellen[265], Mitchell et al.[221], have shown that it was 2,600 kJ/kg, a value which is distinctly higher than that of the simple heat of vaporisation. For a body which is not completely wet, a total heat of evaporation of 2,470 kJ/kg can be assumed, which corresponds to a relative humidity of the surface of the body of about 70%.

2.2 Determination of the Quantity of Water Vapour Evaporated

This can be done merely by weighing. The variation in the weight of the subject, clothed or otherwise, is measured and then corrected by the variation in the metabolic weight due to respiratory exchanges of CO_2 and O_2. It is interesting to see, however, the particular factors which affect the rate of evaporation. This has been investigated by Dalton who enunciated the following law: the rate of evaporation of a liquid at constant temperature is:

- proportional to the free surface (A) of the liquid;
- proportional to the difference between the saturation vapour pressure (P_{ws}) at the temperature of this liquid and the vapour pressure of the environment (P_{wa});
- inversely proportional to the pressure (P_B) of the atmosphere on top of the liquid.

This law is normally stated by the equation:

$$\dot{m} = k \frac{A}{P_B} (P_{ws} - P_{wa}) \tag{1.40}$$

in which k is a constant depending on the units, on the velocity of the air passing over the surface of the liquid and on the shape of the surface. Furthermore, the passage of water vapour through the boundary layer is a diffusion process, and if h_D is taken as the mass transfer coefficient, the water vapour rate of flow can be written:

$$\dot{m} = h_D (C_{ws} - C_{wa}) A , \tag{1.41}$$

where C_{ws} and C_{wa} are the water vapour concentrations at the surface of the liquid and in the ambient air. These conditions can be expressed as a function of the partial pressures ($C_w = P_w/(R_w \times T)$). We can then write:

$$\dot{m} = (h_D/(R_w \times T)(P_{ws} - P_{wa}) A . \tag{1.42}$$

Transferring this expression for $\dot{m}$ into Equation (1.36), we can write that:

$$E = \frac{h_D}{R_w \times T} (P_{ws} - P_{wa})(\lambda_1 + \lambda_2 + \lambda_3) . \tag{1.43}$$

This equation is obviously fairly complex and therefore, by analogy with the heat exchanges by convection, a coefficient of heat exchange by evaporation has been defined such that:

$$h_e = \frac{h_D}{R_w \times T} (\lambda_1 + \lambda_2 + \lambda_3) \tag{1.44}$$

and we have:

$$E = h_e (P_{ws} - P_{wa}) \tag{1.45}$$

h_e is normally expressed in $W/m^2 \cdot kPa$.

Some authors including Buettner, Nelson et al., Clifford et al., Aikas (quoted by Kerslake) have determined h_e in man in various experimental conditions: subjects standing or lying down, wind speeds varying from 0.15 m/s to 4 m/s. They obtained relations showing that h_e was proportional to $v^{0.5}$ (v being the wind speed). These relations which are related to the wind speed should be compared with those obtained for the coefficient of convection h_c, and as early as 1932 Missenard had pointed out that the coefficients h_e and h_c varied in the same proportions. This remark can be derived from the Lewis's relation:

$$h_D = \frac{h_c}{\rho \times c_p} \tag{1.46}$$

where ρ is the density of moist air and c_p its specific heat. Using this relation in Equation (1.44), the evaporative heat transfer coefficient (h_e) can be expressed as a function of the convective heat transfer coefficient (h_c):

$$h_e = \frac{\lambda_1 + \lambda_2 + \lambda_3}{R_w \times T \times \rho \times c_p} \times h_c . \tag{1.47}$$

The product ($\rho \times c_p$) is a variable depending on the temperature and humidity. It may vary from 1.05 to 1.34 $kJ/m^3 \cdot °C$ between 0 and 50°C and 0 and 100% relative humidity. At ambient temperature $T_a = 25°C$, ($\rho \times c_p$) = 1.21 $kJ/m^3 \cdot °C$. If a total heat of evaporation (λ) of 2,470 kJ/kg is assumed, the ratio h_e/h_c is equal to 15°C/kPa. This value agrees closely with Kerslake's experimentally reported value of 15°C/kPa and Buettner's calculation of 16.4°C/kPa.

3. Application of the Laws Governing Heat Exchange by Evaporation

3.1 Evaporation through the Respiratory Passages

Evaporation by means of the respiratory passages occurs if the humidity of the air expired is greater than that of the air inspired. It is considered indeed that the air expired is saturated at its temperature. This transfer of water with vaporisation involves additional cooling. The mass of water evaporated is equal to the difference between the mass of

water breathed out and the mass of water breathed in. The quantity of inspired and expired water depends therefore on the rate of ventilation and the following can be written (Jacquemin et al.[163]):

$$E_{res} = \frac{\lambda}{A_D}\left[\dot{V}_{BTPS}\left(\frac{1}{22.4} \times \frac{273}{310} \times \frac{P_B}{760}\right)\right]\frac{(P_{w\,ex} - P_{w\,in})}{P_B} \qquad (1.48)$$

where E_{res} is expressed in W/m²; λ is the molar heat of water evaporation (43.98 kJ/mole at $T_a = 25°C$); $\dot{V}_{BTPS}$ is the flowrate of air expired in dm³/s; $P_{w\,ex}$ and $P_{w\,in}$ are respectively the partial water vapour pressures of the expired and inspired air. This heat loss may be high in a cold environment with physical exercise or shivering and much higher than the respiratory heat loss by convection: of the order of 16 W/m² at an external temperature of −10°C with a rate of ventilation of 30 l/mn.

3.2 Evaporation through Clothing after Immersion

The "dry" suits in use at the present time in Air Forces are supposed to be permeable to air but impermeable to water. However, during immersion, the fibres making up the material become soaked, so that when the suit is again exposed to the air, the stored water evaporates and the suit again becomes permeable to air. It is probable that a large part of the heat required for this evaporation comes from the suit and consequently from the body itself. The quantity of heat loss by this mechanism could be calculated by weighing the suit before and immediately after immersion, which would give the mass of water retained by it. But in fact, when the subject is in the dinghy, the mass of water retained by the suit does not fully evaporate, since the victim remains partly immersed and it can be assumed that only about half the mass of water evaporates. The rate of evaporation depends on the differences in the partial water vapour pressure between the surface of the suit and the ambient air, and on the speed of the wind. Slow to begin with, since the partial water vapour pressures are approximately the same, the rate of evaporation accelerates as the suit dries. The heat lost in this way may cause a relatively high degree of cooling, particularly if the victim is poorly protected from the wind and the mist, since the latter contribute to the continuance of the phenomenon.

In addition to this external cause of cooling, there is the effect of *insensible and passive diffusion of the moisture through the skin*. Its occurrence is dependent on the difference in the partial water vapour pressures between the area of skin which is very close to the surface where the skin is saturated and the environment constituted by the suit in the case of a subject wearing protective clothing. In a cold and saturated environment, the extent of insensible perspiration through the skin is small and can be estimated as being of the order of 5 to 6 g/m²·h, or 4.12 W/m² for a partial water vapour pressure difference of 25 mm Hg.

IV. HEAT EXCHANGE BY RADIATION

Heat exchange by radiation is the result of electromagnetic phenomena. It does not require any material medium. In the presence of matter, the radiation is partly or fully absorbed. In the general sense of the term, radiation includes all energy transfers ranging from cosmic rays to metre waves. In fact, when discussing heat exchange by radiation, we are referring to the transfer of energy which occurs by means of a process which depends only on the temperature of the objects involved and on the nature of their surface. The radiation of heat as thus defined extends from the ultraviolet to the far infrared, with a maximum which usually lies in the infrared.

In everyday life, heat exchange by radiation represents a large part of all the heat exchanges occurring between man and his environment, both inside and outside buildings. Inside a building, except in special cases, the majority of objects and walls are at relatively low temperatures and the maximum heat radiation occurs at a wavelength of 8 to 10 micrometres. Outside, a man is subjected to direct solar radiation and to radiation reflected by the sky, the ground and the surrounding objects. Taking into account the temperature of the sun (5,760 K), the maximum emission occurs at a wavelength of 0.5 micrometres. There is considerable energy from the sun and it has been calculated that the earth could receive about 1,400 W/m², the major portion of which comes from direct solar radiation in a range of wavelengths from 0.2 to 3 micrometres. In the case of accidental immersion, the heat exchange by radiation is negligible when the victim is immersed. On the other hand, when he is in the dinghy, it may become considerable, and it therefore seems useful to indicate the basic principles of its calculation.

1. Laws Governing Heat Exchange by Radiation

1.1 Emission of Radiant Energy

Our knowledge in this field has been derived from studying a black body. This has the property of completely absorbing the radiation which reaches its surface. There is therefore no reflected energy and the energy from its surface is due solely to the emission of the latter.

The total heat flux radiation by a surface at uniform temperature is given by the Stefan-Boltzman law, expressed as follows:

$$R = \sigma \cdot T^4 \qquad (1.49)$$

in which R is expressed in W/m^2, σ is the Stefan-Boltzman constant: 5.67×10^{-8} $W/m^2 \cdot K$, and T^4 is the absolute temperature of the surface.

Planck's law is used to calculate the flux radiated at each wavelength (R_λ) at a temperature T:

$$R_\lambda = \frac{C_1 \cdot \lambda^{-5}}{\epsilon(C_2/\lambda T) - 1} \tag{1.50}$$

where λ is expressed in μm, T in K, $C_1 = 3.74 \times 10^{-12}$ W/cm^2, $C_2 = 14{,}385$ $\mu m \cdot K$, and $\epsilon = 2.72$.

It is thus possible to find the wavelength corresponding to the maximum energy radiated (λ_{max}). The latter varies with the temperature T. The higher the temperature T, the lower the value of λ_{max}, as has been shown in the case of solar radiation, for which the λ_{max} is 0.5 μm.

An ideal black body is, however, a theoretical body; the majority of bodies do not possess the characteristic quality of an ideal black body and thus, the energy emitted at a given temperature is less than that of a black body at the same temperature. We then say that its *emissivity* is lower. The ratio of the total energy radiated by the body in question to the total energy radiated by a black body is known as the *emittance* (ϵ); its value thus lies between 0 and 1. The following are examples of some emittance values at 20°C in the infrared region: human skin 0.95 to 0.99; snow: 0.99; rough-surface rubber: 0.98; water or ice: 0.94; polished metals: from 0.10 to 0.02. These few values show that human skin, snow and even ice have a very high emissive power, i.e. are good radiators, while polished metals, which reflect extremely well, have a low emissive power and are poor radiators.

Bodies with a slightly reduced emissivity at all wavelengths are called grey bodies. It has been shown that, in a given temperature range, the Stefan-Boltzman law can be applied to them by multiplying the constant σ by ϵ. The heat flux emitted by the surface of a grey body is thus expressed as follows:

$$R = \epsilon \cdot \sigma \cdot T^4 . \tag{1.51}$$

1.2 Absorption and Reflection

The incident energy flux is absorbed or reflected in varying degrees according to the bodies concerned. Thus, a black body has maximum absorption and zero reflection, while polished metals on the other hand have almost maximum reflecting power. A coefficient of absorption (α) and a coefficient of reflection (ρ) can thus be defined, such that:

$$\rho = 1 - \alpha . \tag{1.52}$$

It has been demonstrated that the coefficient of absorption at the temperature (T) of a surface for radiation of a given wavelength is equal to the emittance of that surface at the same wavelength and the same temperature. In other words, the greater the quantity of energy emitted by a body the greater the quantity of energy absorbed by it. For example, in the infrared region, the human skin will behave practically in the same way as a black body and will become warm rapidly, particularly if $3 < \lambda < 20$ μm. On the other hand, the skin will reflect some of the energy from the sun's radiation, where the $\lambda_{max} = 0.5$ μm.

1.3 Radiative Heat Exchange between Two Surfaces

If the emittance of the two surfaces is ϵ_1 and ϵ_2 at the temperatures T_1 and T_2 respectively, we can write, by applying the Stefan-Boltzman law:

$$R = \sigma \cdot \epsilon_1 \cdot \epsilon_2 (T_1^4 - T_2^4) \tag{1.53}$$

provided that the emittance values are close to 1, as is the case in a number of problems associated with heat exchanges between the human body and its environment. However, when the emittance values are very different from 1, reflection must be taken into account and it can be shown that:

$$R = \frac{1}{\frac{1}{\epsilon_1} + \frac{1}{\epsilon_2} - 1} \times \sigma(T_1^4 - T_2^4) . \tag{1.54}$$

2. Application to Radiative Heat Exchange between the Human Body and its Environment

Several cases have to be considered, according to whether the skin is protected or otherwise, and whether the subject is in or outside a building. In accidental immersion the victim is generally in an individual or a collective dinghy, and therefore in principle may be protected from direct solar radiation. In addition, in cold regions, only a very small part of the skin is exposed to the sun's rays. Heat exchange by radiation then takes place between the surface of the clothing and the walls of the dinghy or, if the dinghy has no roof or protective hood, between the clothing and the environment. Each situation will now be examined in relation to its own particular features.

2.1 The first problem which arises is to determine the *effective radiative exchange area* A_R. Indeed the various regions of the body do not participate in the same manner in the heat exchanges with the surrounding environment. For example, certain parts of the body exchange energy with each other to a much greater extent than with the environment. Such is the case of the inner surfaces of the upper limbs with the trunk, and the inner surfaces of the lower limbs with each other. The posture of the body also has an important effect on this area of exchange. Thus, for a man stretched out on wire netting, with arms and legs slightly apart, Colin et al.[77] found an A_R/A_D ratio of 0.84. This value is very close to the 0.85 reported by Bohnenkamp (quoted by Hardy[138]). For a man who is standing, arms close to his body, the A_R/A_D ratio is 0.78 to 0.80, and in the case of a sitting man, it is 0.70. It may be thought that it is even lower (about 0.6) for the immersion victim sitting in an individual dinghy, in view of the parts of the body which are more or less immersed and those parts in contact with the dinghy. The ratio will be higher (about 0.7) in dinghies which accommodate several people, where there is more space.

2.2 A second problem associated with closed spaces is that of the *ratio between the area* A_R *and the area of the walls* A_W. Equation (1.54) giving the radiative exchange between two areas with emittance values different from 1, is valid only if A_R and A_W are equal. If A_W is greater than A_R, the following must be written:

$$R = \frac{1}{(1/\epsilon_s + 1/\epsilon_r - 1)} \times \sigma(\bar{T}_s^4 - \bar{T}_r^4) A_W/A_R \qquad (1.55)$$

in which ϵ_s and ϵ_r are respectively the emittance values of the skin (or clothing) and the walls, T_s is the mean absolute temperature of the skin (or clothing) and T_r is the mean absolute temperature of the walls. This equation shows that the greater the area of the walls as compared with the radiative heat exchange area of the body, the greater will be the radiative heat exchange.

2.3 The third main point for calculating R is to *determine the emittance values*. When the exchanges occur in the infrared, as in the case of low temperatures, the skin and the majority of the immersion suits have an emittance close to 1, whatever their colour. The same applies to the walls, particularly when they are made of impermeable canvas, as in the case of dinghies. The Stefan-Boltzman law applies and the following can be written:

$$R = \sigma \cdot \epsilon_s \cdot \epsilon_r (\bar{T}_s^4 - \bar{T}_r^4) A_R/A_D \, . \qquad (1.56)$$

The simplest way to reduce losses by radiation is to reduce ϵ_r, by use of a reflecting covering on the inside walls. This method has been put forward for certain dinghies (Hall et al.[131]). Its effectiveness depends on the ratio between the areas A_W/A_R (Equation (1.55)). If A_W/A_R is only slightly different from 1, the losses through radiation can be reduced by 80%. If, on the other hand, the area of the walls is four times greater than A_R, an 80% reduction in the emittance of the walls will not reduce the radiative exchanges by more than 20%.

Equation (1.56) can be simplified in a number of cases. By analogy with the other methods of heat transfer, a coefficient of radiative exchange (h_r) expressed in $W/m^2 \cdot °C$ has been defined, so that we have:

$$R = h_r (\bar{T}_s - \bar{T}_r) \qquad (1.57)$$

h_r is a complex term the expression of which can be simplified when the mean radiant temperature ($\bar{T}_r$) and surface temperature ($\bar{T}_s$) do not differ greatly from each other. It can then easily be shown that:

$$h_r = 4\sigma \cdot \epsilon_r \cdot \epsilon_s \cdot T^3 \cdot A_R/A_D \qquad (1.58)$$

where $T = 0.5 (\bar{T}_s + \bar{T}_r)$.

Equation (1.58) therefore has the form $h_r = a \cdot A_R/A_D$, a being expressed in $W/m^2 \cdot °C$. The value of a varies with temperature. Thus, for a temperature of −10°C (263 K), a = 4.12; for T = 0°C, a = 4.61; for T = 10°C, a = 5.14.

2.4 Solar Radiation Exchanges

When the victim of an accident is in his dinghy, he may be subjected either directly, or indirectly through the walls, to the direct or reflected rays of the sun. As has been seen, this radiation is not completely absorbed by the skin, clothing, or the walls of the dinghy, since the sun emits heat largely in the visible area of the spectrum ($\lambda_{max} = 0.5\ \mu m$). The total heat gain due to direct solar radiation is determined by means of the following equation:

$$R_{so} = A_p \cdot \alpha_s \cdot I_{R_{so}} \qquad (1.59)$$

in which R_{so} is the heat flux received in watts, A_p is the projected area of the body or the dinghy, which varies with the height of the sun above the horizon, α_s is the coefficient of absorption (equal to the emittance for the wavelength involved) and $I_{R_{so}}$ is the direct heat flux emitted by the sun in W/m^2. Formulae for calculating the projected areas have been given for a person in the vertical position in relation to the altitude of the sun (Pugh and Chrenko[240]) but for such a complex shape as a dinghy, only actual experiments can provide data.

In addition to this direct radiation from the sun there is the very variable radiation from the reflections on clouds and water. Unfortunately, as far as we know, there are no estimates of the extent of the heat gain from solar radiation in the particular situation of the victim of an immersion accident sitting in a dinghy.

V. SIMPLIFIED EXPRESSIONS FOR HEAT EXCHANGES AND EVALUATION OF COLD ENVIRONMENTS

These analyses of the various ways in which heat exchanges occur illustrate the complexity of the laws governing these exchanges. Consideration of these laws will however point to solutions for limiting the exchanges in a cold environment although their application to concrete instances, such as those encountered in accidental immersion, is particularly difficult. Numerous attempts have been made to simplify the expression of these laws and find a means of assessing the severity of cold climatic conditions.

1. Simplified Expressions for Exchanges by Radiation and Convection

Combined heat exchange coefficient: when the air temperature is equal to the mean radiant temperature, the following can be written:

$$R + C = (h_r + h_c)(\bar{T}_s - \bar{T}_a) . \tag{1.60}$$

The sum $(h_r + h_c)$ is called the combined heat exchange coefficient (h) (Colin et al.[80]). This coefficient depends on numerous parameters, such as the position of the body and the speed and direction of the wind. The coefficient h is therefore constant, only if these parameters themselves are constant. For a naked subject, sitting in still air, h is approximately 7.15 $W/m^2 \cdot °C$ (Gagge et al.[104], Stolwijk et al.[271]). If the subject is exposed to wind, the combined coefficient, like the coefficient of convective heat exchange, is a function of the wind speed ($v^{0.5}$ or $v^{0.67}$ depending on the authors).

If the radiant temperature is different from the air temperature, this combined coefficient can also be used. It is sufficient in such cases to use the *operative temperature* T_o, a concept developed as early as 1937 by Winslow et al.[302]. This temperature is given by the following equation:

$$T_o = \frac{h_r \times \bar{T}_r + h_c \times \bar{T}_a}{h_r + h_c} \tag{1.61}$$

and we then have:

$$R + C = h(\bar{T}_s - T_o) . \tag{1.62}$$

Gagge and Hardy[105] have also introduced the concept of *standard operative temperature* T'_o. This is calculated by means of the following equation:

$$T'_o = \frac{h}{h_o} T_o + \left(1 - \frac{h}{h_o}\right) \bar{T}_s \tag{1.63}$$

in which h is the combined heat transfer coefficient and h_o the combined coefficient for a subject at rest in still air. This temperature has the advantage of making it possible to compare various experimental situations.

2. Evaluation of a Cold Environment

Although the preceding concepts make for a quicker calculation of the heat exchanges, a number of measurements are necessary which require a wide variety of instruments; e.g. dry and wet bulb thermometer, radiometer, anemometer etc. Quite frequently, such measurements are not all capable of being carried out in practice and, furthermore, they cannot provide an evaluation of the severity of the environment for the human being. For this reason some authors have put forward methods which combine several environmental parameters in order to estimate the thermal stress to which the organism is subjected.

2.1 The Windchill Index

In 1945 Siple and Passel[261] established an index known as the Windchill index, which combined the thermal effects of the wind and the temperature, and is calculated by means of the following expression:

$$K_c = (10\sqrt{v} + 10.45 - v)(33 - T_a) \tag{1.64}$$

where K_c is the Windchill index expressed in $kcal/m^2 \cdot h$, v is the wind speed in m/s and T_a is the air temperature in °C. Graphs, such as those in Figure 1.5, published by the Ministry of the Environment in Canada, show the variation of this index with the wind speed and the ambient temperature. Several areas can be seen, according to the value of the index.

Thus, an index of 700 shows conditions which are comfortable for a person dressed for skiing; at 1,400 W/m², the conditions are unpleasant for indulging in outdoor activities in sunny weather. At 2,300 W/m², conditions are dangerous if one is moving about and the unprotected exposed areas of the body freeze in less than a minute. It should be noted that these charts apply only for wind speeds of more than 8 km/h. The Windchill index is obviously suitable for conditions on land; it could, however, quite easily apply to the conditions encountered in cold seas by accident victims sitting in their dinghy. It is, however inadequate, since it does not allow for solar radiation. In addition, as pointed out by Burton[58], it is theoretically impossible to express the effect of the wind without taking into account the value of the insulation provided by clothing and its more or less good impermeability to the wind. Nevertheless, this index gives a fairly accurate indication of the relative rate of cooling of the uncovered areas of the body – the face and hands.

2.2 The Globe Temperature

The use of the Windchill index could be improved by applying the operative temperature T_o, or better still, the globe temperature T_g. The latter was advocated almost simultaneously in the United Kingdom by Vernon[294] and in France by Missenard[220]. This is the temperature of a globe approximately 15 cm in diameter, painted black, or preferably grey, so that it has the same reflectivity characteristics as clothing. The equilibrium temperature of this globe is reached when its convective heat losses are equal to its radiative heat gains. We can then write:

$$h_{cg}(T_g - T_a) = h_{rg}(\bar{T}_r - T_g) \tag{1.65}$$

and:

$$T_g = (h_{cg} \times T_a + h_{rg} \times \bar{T}_r)/(h_{cg} + h_{rg}) . \tag{1.66}$$

The relation between h_{cg} and the velocity of air is established experimentally and $\bar{T}_r$ calculated from it. In addition, Equation (1.66) is exactly the same as that for calculating the operative temperature T_o. The globe temperature thus gives a direct measurement of the operative temperature if the convective and radiative exchange coefficients are the same as those of the human subject. If the emittance of the globe and of the human subject is the same, we can write that:

$$h_c/h_{cg} = h_r/h_{rg} = A_R/A_D \tag{1.67}$$

A_R being the area of exchange by radiation of a person and A_D the total area of the person, since the term h_r includes the ratio A_R/A_D, whereas in the case of the globe the whole area is involved in radiative heat transfers. For a wind speed of 0.25 m/s, the ratio h_c/h_{cg} is approximately 0.69, a value which is very close to that of A_R/A_D for a seated human subject. It can therefore be said that the globe temperature shows immediately the value of the operative temperature in that situation. For subjects in the standing position, on the other hand, the globe temperature underestimates the effect of the radiant temperature and a much larger diameter globe would have to be used. A theoretical analysis of the validity of the globe temperature was made a few years ago by Kuehn et al.[187]. These authors showed that in order to adjust the globe temperature to the operative temperature two factors could be used: the emittance of the globe and its diameter. The lower the emittance, the larger must be the diameter of the globe. In practice, however, the use of a small diameter globe is recommended. Its emittance must therefore be high, and for this a copper globe covered with matt black paint with an emittance of 0.95 should be used. In such conditions only the diameter of the globe will vary with the position of the subject. For example, for a person wearing clothes, in a semi-stretched out position in a turbulent air flow, and for a person wearing clothes, standing facing the wind, the globe diameter must be 10.5 cm. However, this globe temperature has the disadvantage of not taking into account the effect of the wind in the absence of solar radiation or when the radiant temperature is the same as the air temperature.

2.3 The "Still Shade" Temperature

This has been suggested by Burton and Edholm[58]. To some extent this is an equivalent temperature, the radiation heat gains being reflected in an increase in the ambient temperature and the convective losses by a decrease in this temperature. Thus, in a given situation characterised by a known wind, air temperature, and solar radiation, and a given activity of the subject, the exchanges take place as though the clothed subject were subjected to an equivalent environment in still air with no solar radiation, the temperature of which (T_{so}) differs from that of the air. This temperature is calculated using the formula:

$$T_{so} = T_a - 9.1\,H(1 - I_a) + 9.1 \times 4.6\tau \cdot \alpha \cdot I_a \tag{1.68}$$

in which: T_{so} is expressed in °C, the insulation of the air (I_a) in clo units with the insulation of still air being assumed equal to 1 clo, H, expressed in Met, represents the heat production of the subject per unit area of the body (1 Met = 58 W/m²), τ is a coefficient of transmission by the atmosphere of solar radiation and α is the coefficient of absorption of clothing. The term $(-9.1\,H(1 - I_a))$ is the decrease in the temperature of the air due to convection and the term $(9.1 \times 4.6\tau \cdot \alpha \cdot I_a)$ is the increase in temperature due to radiation. The insulation provided by clothing is included in the term H, the authors having previously determined the total insulation (clothing + air) required as a function of the ambient temperature for subjects having different levels of activity. The equation which they used for the calculation was as follows:

$$I = 0.1476(33 - T_a)/H \tag{1.69}$$

in which I is expressed in clo, T in °C and H in Met. Substituting T_{so} for T_a we can thus calculate the insulation provided by clothing (I_{cl}) required for a given activity level H which will maintain the mean skin temperature at 33°C. I_{cl} is obtained by subtracting from I the insulation provided by still air (1 clo). An instrument, the cold stress meter, has recently been designed for direct measurement of the still shade temperature (Kuehn et al.[188]) in given conditions: H = 2 Met and $\alpha = 0.435$. Such an instrument can be very valuable in estimating the clothing insulation required for a given air environment. Inversely, if I_{cl} is known, it is possible, by applying Equation (1.69), to predict the level of the mean skin temperature.

2.4 Other systems also enable the exchanges to be evaluated. One of the best methods is to use a globe or a cylinder of suitable dimensions, heated and maintained at a constant temperature (37°C) by means of a thermostat. The electrical power consumed would then be a direct measurement of the heat losses. Furthermore, it would be possible to cover this apparatus with known thermal insulating material, which would provide a direct measurement of the thermal insulation required in a given environment for various powers corresponding to different levels of activity.

The short analysis of the methods used to characterise a thermal environment indicates how difficult it is to obtain simple information which can be applied to the various situations encountered in practice. It would appear that efforts must be made to develop a simple instrument which would provide a reliable index for a rapid evaluation of the thermal constraints imposed as a function of the activity of the subjects and of the insulation afforded by the clothing worn. However, it seems no easy task to take into account all the environmental parameters and a current tendency is therefore to make mathematical models, which have greater flexibility of use than the environmental "integrating" instruments.

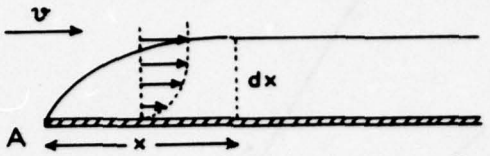

Fig.1.1(a) Laminar boundary layer along a plane surface

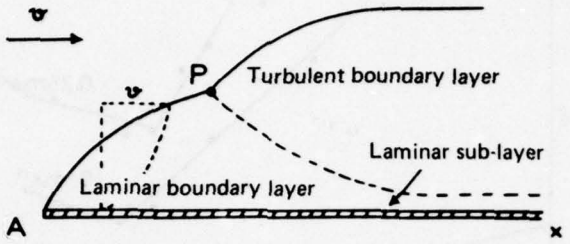

Fig.1.1(b) Turbulent boundary layer. Note the formation, a short distance from the edge of the plate, of a very thin laminar sub-layer and a thick turbulent boundary layer

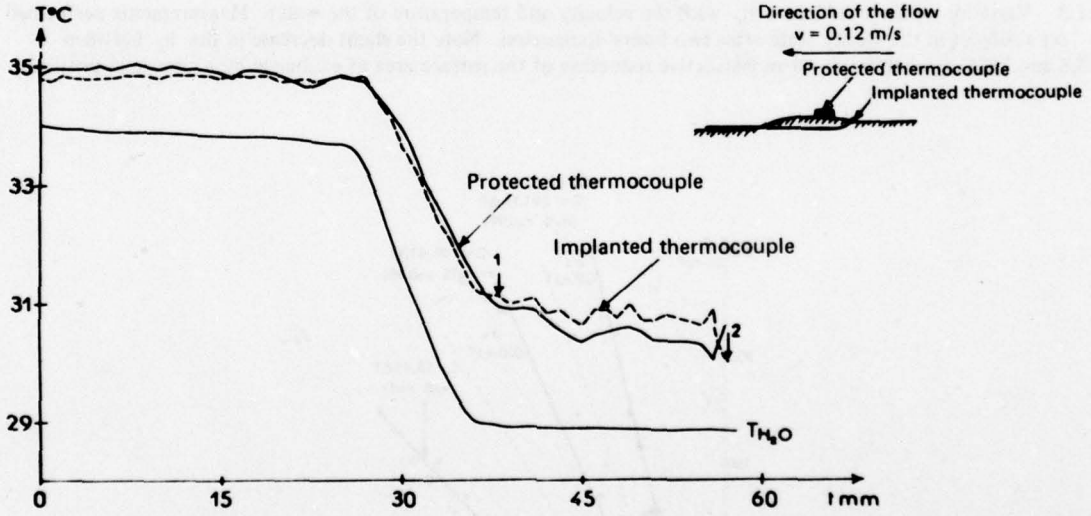

Fig.1.2 Comparison of temperature measurements made with a thermocouple protected by a permeable polystyrene pad 1.5 to 2 mm thick, placed on the skin, and an implanted thermocouple the tip of which was just touching the surface of the skin. There is no significant difference between the two measurements at $\overline{T}_s = 34.8°C$, *but the protected thermocouple reacts slightly more slowly to a temperature variation. At* $T_w = 29°C$, *the protected thermocouple is slightly colder than the implanted thermocouple (0.1°C for the first four minutes). The difference then increases up to 0.3°C because of slight bleeding*

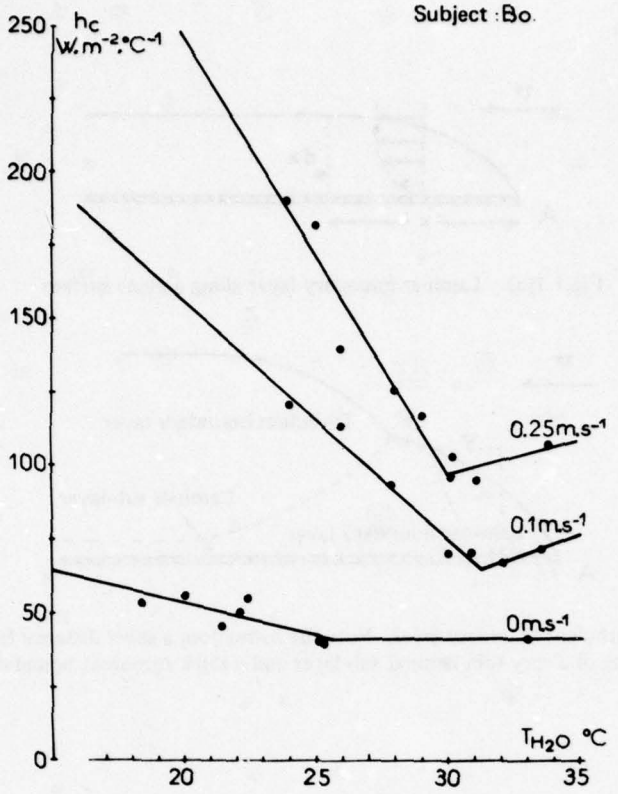

Fig.1.3 Variation of the coefficient h_c with the velocity and temperature of the water. Measurements performed on a subject in the steady state after two hours' immersion. Note the slight decrease in the h_c between 33.8 and 30°C, probably due to an instinctive reduction of the surface area of exchange by a change in posture

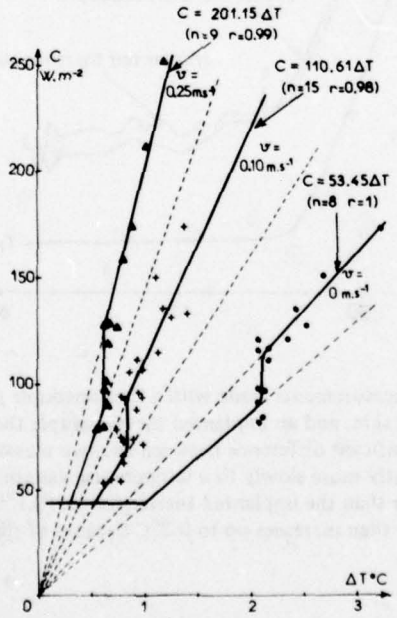

Fig.1.4 *Relationship between the convective losses* C and the temperature difference between the skin and the water ΔT, in the stable state for three water velocities. The thermo-neutral values are shown by a horizontal dashed line. This graph, taken from our experimental results, shows that h_c increases with the velocity of the water and that it is higher in cold water than at thermal neutrality because of the effect of shivering;

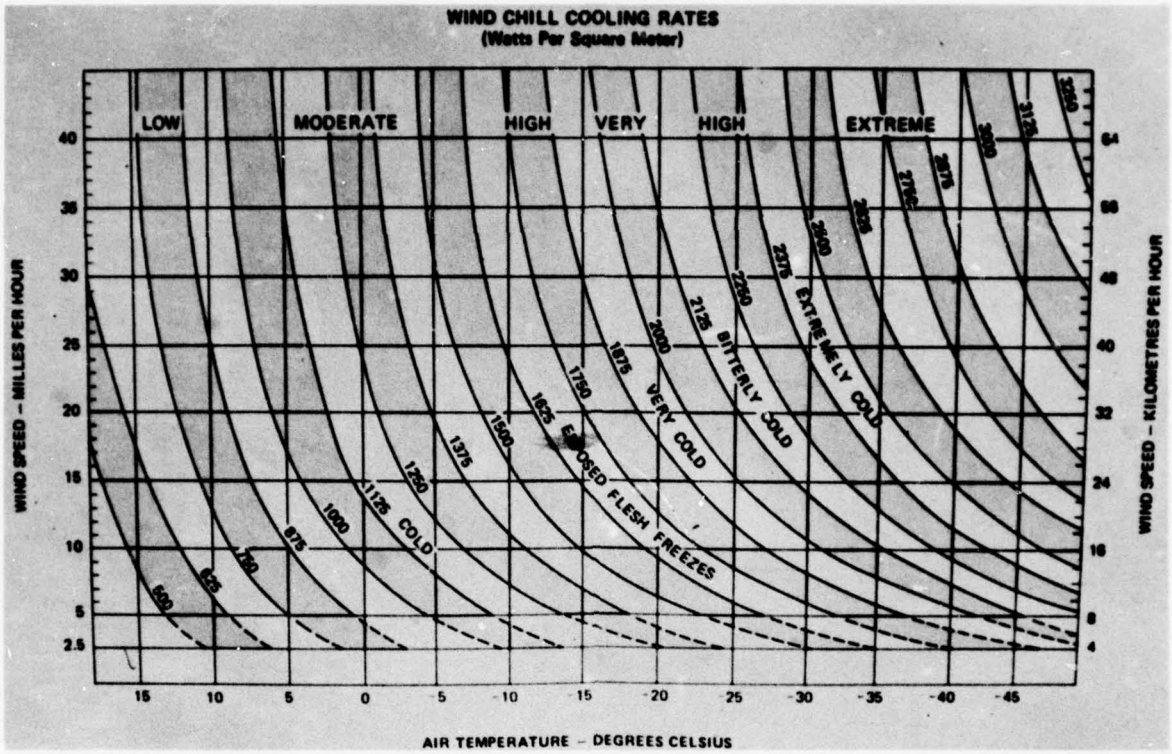

Fig.1.5 Cooling rates calculated by means of the Windchill index
(document supplied by the Canadian Ministry of the Environment)

Chapter 2

PHYSIOLOGICAL REACTIONS TO ACCIDENTAL IMMERSIONS TOLERANCE – ACCLIMATIZATION

Speaking very generally, man uses two methods of combating cold: he warms himself at a fire and/or he increases his insulation against the environment by wearing clothes (or building shelters). The main physiological reactions of the human body exposed to cold are not very different. Warmth is obtained by increasing the production of metabolic heat, either voluntarily by muscular exercise or, involuntarily, by shivering. Circulatory adjustments (vasoconstriction) increase the body's insulation so that the central temperature can be kept, insofar as possible, at a level compatible with the correct functioning of the organs essential to life. These two ways of combating cold can, moreover, be deduced from the equations for calculating the heat balance set out in Chapter 1. Indeed, the body heat balance in a cold environment can be expressed by the following equation:

$$S = M - (R + C + K + W + E) . \tag{1.1}$$

In the case of immersion, radiative and conductive losses are nil if the body is totally immersed, and the evaporative losses are reduced to evaporation from the respiratory tract. For a body at rest, W is zero; by showing the heat losses through the respiratory tract and considering conduction (K) as an integral part of convection (C), it is possible to write the following:

$$S = M - [C + (E_{res} + C_{res})] . \tag{2.1}$$

The balance will be positive, negative or zero according to whether the metabolic rate (M) is higher, lower or equal to the losses. In addition, it is usual to consider the body as made up of two concentric zones: (1) a central core the mean temperature of which is assumed as a first approximation to correspond to the rectal temperature (T_{re}); and (2) a shell, for which the temperature is assumed to be the weighted mean skin temperature ($\overline{T}_s$). This shell provides a certain thermal insulation, the reciprocal of the body conductance (h_b), which regulates the heat exchange between the core and the external environment. In these conditions, the flow of heat from inside the body to the outside is equal to the losses by convection and the following can be written:

$$C = h_b (T_{re} - \overline{T}_s) . \tag{2.2}$$

The heat balance equation then becomes:

$$S = M - [h_b (T_{re} - \overline{T}_s) + (E_{res} + C_{res})] . \tag{2.3}$$

This equation includes only physiological quantities which can be measured during laboratory immersion experiments on volunteer subjects, and can be used as a basis for studying the reactions of these men. It shows, in fact, that an unclothed man immersed in cold water has only two means of compensating for the escape of heat due to a lowering of the skin temperature and to an increase in the core/periphery heat gradient: reducing the conductance (h_b) by vasoconstriction of the skin or increasing his metabolic rate (M). It should be pointed out that the preceding equation can also be applied to a man wearing clothes. All that is necessary is to substitute the mean temperature of the surface of clothes $\overline{T}_{cl}$ for $\overline{T}_s$ and to replace h_b by the following expression:

$$\frac{h_b \times h_{cl}}{h_b + h_{cl}} ,$$

which takes account of the conductance of the clothing h_{cl}.

However, before describing physiological reactions during cold water immersion and indicating the limits of tolerance, it is necessary to clarify the zone of thermal neutrality in water.

I. THERMAL NEUTRALITY ZONE IN WATER

Research studies on determining the water temperature at which an unclothed man is at thermal neutrality are relatively few in number and opinions differ from author to author. Such differences arise from the fact that most of them have tried to determine a critical water temperature rather than a thermally neutral water temperature. For Burton

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and Bazett[57], the critical water temperature is the temperature at which the peripheral conductance h_b is at its lowest value without causing shivering. It is stated to be 33°C in the case of the unclothed subject and to correspond for him to a period in still air at 23°C. This concept of critical temperature was taken up by Rennie et al.[248,249] who make it more precise by adding a concept of time, so that it becomes the water temperature at which subjects can remain motionless without shivering for 3 hours. These authors observed that this temperature was 33°C for American subjects of the male sex and 30°C for Korean women swimmers and fat American women. They also showed that there seemed to be racial differences for the value of this threshold, the Koreans having a lower critical water temperature than that of Americans with the same skinfold, a result which was confirmed by a study by Inoue[162] on the Japanese. Burton and Edholm[58], however, state that the critical water temperature does not correspond with the thermal neutral temperature. They feel that the temperature of the water at thermal neutrality should be 35°C. They even add that, to maintain the heat balance, the water should be at 36°C, when the subject would then have a sensation of thermal comfort. It should be stressed, however, that these authors assumed in their calculations that the temperature of the skin was the same as that of the water, which is obviously impossible. For Craig and Dvorak[84], the criteria for thermal neutrality were a constant average body temperature and a heart beat slightly less than that in a position of rest in air because of the slight bradycardia caused by immersion. They then found a thermo-neutral temperature of between 35 and 35.5°C. These two results illustrate that there is still some lack of precision in regard to the definition of the thermo-neutral zone. One may wonder why this is so since in air the thermo-neutral zone is known to be ±0.30°C, according to the wind speed, the position of the subject and the time of day. In actual fact this inability to fix the zone would appear to be due to the criteria adopted. A metabolic rate equal to the basal metabolic rate is not sufficient. Indeed, it does not increase during short exposure in hot water if the subject is motionless and determinations of the critical water temperature show that a basal metabolism can be observed in fresh water. In a study made by Boutelier et al.[43] to determine the thermo-neutral zone, the following criteria were taken:

- a constant metabolic rate, equal to that observed at thermal neutrality in air, i.e. approximately 47 to 48 W/m²;
- a core/shell conductance close to that found in air at thermal neutrality: 15 to 17 W/m²·°C;
- a slight fall in the rectal temperature during the first 90 minutes of the experiment while the subject is getting into a state of rest. This temperature must be 36.7 to 36.8° during the stable period if the experiments take place in the morning on subjects who have not breakfasted;
- a standard distribution of local skin temperatures, as described by Winslow and Herrington[303], that is, from the coldest to the warmest: the extremities (feet and hands), calf, thigh, forearm, arm, trunk (back, abdomen, chest), taking into account the circulatory adaptation to immersion and the corrective reactions involved. In addition, the average skin temperature $\bar{T}_s$ or $\bar{T}_{s_{im}}$, if the body is not completely immersed, calculated with the coefficients suggested by Hardy must be about 34°C;
- a sensation of thermal confort for the subjects, with complete absence of shivering.

Other conditions can be drawn from the standard fractional calorimetry equations. Indeed, during total immersion, the following can be written, in the stable condition when the heat balance S is zero:

$$C = M - (E_{res} + C_{res}) = h_b (T_{re} - \bar{T}_s) . \tag{2.4}$$

For C to be constant, M, h_b, $(T_{re} - \bar{T}_s)$ must also be constant. In addition, it is known that:

$$C = h_c (\bar{T}_s - T_w) , \tag{2.5}$$

which can be written:

$$\bar{T}_s = T_w + \frac{C}{h_c} \tag{2.6}$$

Now, for a given water velocity and no subject motion, h_c is constant, with the result that the ratio C/h_c is itself constant and the relationship between the average skin temperature and the temperature of the water must have a slope equal to 1. The results obtained on eleven subjects during 31 immersions lasting from 90 to 160 minutes show that for a water velocity of 0.05 m/s, the zone which satisfies best the criteria mentioned above is very narrow and ranges only from 33.1 to 33.4°C (Fig.2.1). This is confirmed by the average local skin temperature distribution (Fig.2.2), in which it will be noted that at $T_w = 33.5$°C, the foot and the hand are warmer than the calf and the thigh, while the arm and the forearm have the highest temperature. This indicates vasodilatation which can be due only to heat. The average thermal characteristics of the subjects in this zone were as follows: $\bar{T}_s = 34 \pm 0.05$°C, $T_{re} = 36.70$°C, $M = 48 \pm 1.7$ W/m² and $h_b = 16.7 \pm 0.9$ W/m²·°C. These characteristics are independent of the environment and are always to be found, whatever the velocity of the fluid. It follows that the thermal neutrality zone varies under the effect of physiological and physical factors. It does not exceed 34°C as an upper limit for a young man in the morning who has had no breakfast.

Among the physiological factors which may have an effect on the displacement of the thermal neutrality zone three main elements deserve consideration: the effect of the circadian rhythm, the extent of the sub-cutaneous adipose tissue, and age.

The circadian rhythm of physiological temperatures and harmonal reactions is a very well known phenomenon in man and there is no doubt that it affects the value of the thermo-neutral temperature. Thus, the latter will be lower in the afternoon and higher at night and in the morning.

The part played by sub-cutaneous adipose tissue is more open to discussion. It might be thought that, even at thermal neutrality, sub-cutaneous fat could play a part by reducing heat loss through the skin, which would have the effect of lowering the skin temperature and consequently the thermal neutrality temperature. This is not, however, the case and we have not found any difference in the value of the thermo-neutral temperature between fat subjects whose skinfold was 20 mm and thin subjects with a skinfold of 4 mm. This result is in line with that found by Cannon and Keatinge[67] who observed that the water temperature threshold for an increase in the metabolic rate was the same for fat men as for thin men. They add that this threshold is determined by the threshold of the skin receptors to cold. The latter is probably at too high a temperature for fat men, who would thus unnecessarily increase their heat production in water at 33°C. This assumption would mean that sub-cutaneous fat acts as an insulator, even at thermal neutrality. For this to be true, it would have to be assumed that the peripheral circulation, which provides the chief means by which internal heat is carried to the surface of the skin, is greater under the layer of sub-cutaneous fat than in the dermis. The multiplicity of dermal blood vessels, their particular anatomical arrangement, with three networks arranged in layers over the whole depth of the dermis and the extent of the arteriovenous anastomoses at all the levels, appear to indicate that these vessels play a prime role in heat exchanges in warm environments and when there is thermal neutrality. The sub-cutaneous fat is therefore short-circuited, and acts as a protective agent only in a cold environment when the surface blood vessels are strongly constricted. It is therefore normal to observe the same thermo-neutral zone for both fat and thin subjects, since the sub-cutaneous fat is involved only in the conductive exchanges through the tissues. This can also explain why the subjective sensation of cold is the same for all subjects, the skin receptors which are sensitive to cold being located mainly in the dermis.

The effect of age is one of the most rarely discussed factors in the literature, although its effect may not be negligible. It is indeed commonly observed that elderly people in a state of rest generally feel cold in environments which younger people would consider comfortable or warm. One of the reasons for this phenomenon is the reduction in the basal metabolism, which is of the order of 12.5 to 15% by the age of 50 to 60. As there would not seem to be any significant deterioration in the flow of blood to the hands, feet and limbs according to Abramson[1], it may be thought that the core/shell conductance is close to that determined by the present author in the neutrality zone (16.7 $W/m^2 \cdot °C$). For the same rectal temperature (36.7°C), the mean skin temperature must be higher than 34°C, as can be deduced from Equation (2.4).

The main physical factors which affect the value of the thermo-neutral temperature in water are the velocity of the water and the wearing of clothes.

The velocity of the water causes an increase in the coefficient of heat exchange h_c and the ratio C/h_c in Equation (2.6) decreases. The neutral temperature therefore approaches the mean skin temperature. Using the values of h_c quoted in Chapter 1, we find, for an average man with the thermal characteristics given above, a thermo-neutral zone which extends from 32.9°C in still water to about 34°C in very disturbed water.

The effect of clothing is more difficult to discuss in view of the wide variety of the insulation values. For example, for a watertight garment with an insulation of 1.45 clo, the surface temperature of the garment will have to be 24.3°C if the subject is to be at thermal neutrality. The water temperature zone corresponding to neutrality will be between 23.3°C and 24.3°C. Below this limit the organism will have to put up some defence against cold.

II. PHYSIOLOGICAL REACTIONS TO IMMERSION IN COLD WATER

1. Metabolic Reactions to Immersion in Cold Water

1.1 Origin

Increasing the production of metabolic heat is the most important means available to the organism in combating cold. Among the majority of the homeothermic animals increased thermogenesis is produced by two separate mechanisms: a process of chemical regulation (in the restricted sense of the term) under the control of the thyroid and suprarenal hormones, and muscular activity, either of an involuntary nature due to shivering, or voluntary, during physical exercise.

The existence of chemical thermogenesis, still called "non-shivering thermogenesis" or NST, was suggested by Rübner as early as 1902. This author had observed that the oxygen consumption of men exposed to cold often increased before the start of shivering. This can, in fact, easily be explained by an increase in muscular tone without its being necessary to postulate non-muscular thermogenesis. Since then, a number of authors (Sellers et al.[258], Carlson[71], Hshieh et al.[155], Hemingway et al.[146]) have shown that this type of thermogenesis exists in several animals: rats, rabbits, dogs, cats. This production of heat, under the effect of adrenaline, originates in the brown fat. However, although chemical thermogenesis is an important means of combating cold in cold acclimatized animals, small mammals and new born infants, the same is not true of the human adult, in whom it seems to have little effect or even to be non-existent. Cannon et al.[66] estimate a 16% increase in oxygen consumption by this means. Iampietro et al.[158] quote values

of 10 to 20%, but Glickman et al.[118] show that the oxygen consumption of a man exposed to cold increases proportionately to the muscular activity and they feel that the production of heat by NST is much lower, and perhaps that no heat is produced at all by NST. Recently, however, Jessen et al.[168] seem to have demonstrated its existence on six patients who had been curarised and exposed to cold. They noted an average increase in oxygen consumption of 23% for a fall of at least 0.5°C in the central temperature in 30 to 45 minutes. In spite of this, non-shivering thermogenesis is in general very slight in man, as compared with that produced by shivering, and it can be considered that the increased production of heat is almost solely of muscular origin, particularly in the case of sudden cooling.

1.2 Characteristics of the Metabolic Response

In accidental immersion the victim may undergo slow cooling if he is wearing an anti-immersion suit and if the water is not icy, or sudden cooling if he has no protective clothing and the water temperature is below 20°C.

In the first case, shivering does not start immediately but appears after a certain time, as pointed out by several authors (Lefevre[204], Horvath et al.[153], Craig and Dvorak[84], Colin and Houdas[75]. The existence of this delay would seem to indicate that there has to be a heat loss in order to produce shivering, or that the latter occurs only after a certain skin temperature value has been reached. Hardy et al.[141] observed a considerable heat loss before the onset of shivering. However, this loss varies from subject to subject and depends on the thickness of the sub-cutaneous fat (Boutelier[44]). The same applies for the average skin temperature which initiates shivering. Thus, Horvath et al.[153] noted in the case of men exposed to an ambient temperature of −3°C in air, that the first shiver appeared at a $\bar{T}_s$ of 29.2°C and overall shivering at a $\bar{T}_s$ of 27.1°C, although Swift (quoted by Horvath) had given 19°C as a threshold skin temperature. We ourselves observed, during numerous tests on anti-immersion suits, that the average threshold value of skin temperatures which initiated shivering varied from one subject to another for the same anti-immersion suit. This temperature was generally of the order of 28°C for thin subjects and 25 to 26°C for fat subjects or those who had been undergoing some kind of training. After the onset of shivering, the metabolic rate increased gradually, but irregularly, shivering being characterised, if the stimulus was not too strong, by irregular muscular activity, which occurred in sudden bursts. The latter grew more frequent as the cooling process became intensified. The metabolic rate then tended towards a steady value so that it balanced the heat losses, or towards a maximum value.

When cooling is sudden, the metabolic response is more complex. Diagrammatically its development is a two-phase process, comprising a phase of rapid variation, followed by a period during which there is a slower and irregular increase (Fig.2.3). The phase of rapid variation is characterised by a sudden increase in the metabolic rate during the period of variation in the skin temperature. This is accompanied by a feeling of tightness in the thorax and violent shivering. The initial metabolic reaction is caused by stimulation of the thermoreceptors in the skin, provoked by the drop in skin temperature. Indeed, the higher the rate of change of skin temperature, the stronger the reaction; it does not take place if the rate of change is less than 0.2°C/mn (Boutelier[44]). Furthermore, it is limited in amplitude if maximum heat production has been achieved by shivering and by the reduction of the convective heat losses following the decrease in the temperature difference between the skin and the water. From the point of view of time, the production of heat develops in a manner comparable with that of the rate of fall in the skin temperature $d\bar{T}_s/dt$, but reaches its peak value slightly later, and then decreases. The amplitude of the initial metabolic reaction varies with the subject and, for the same rate of fall of the skin temperature, is less in fat subjects than in thin subjects. In interpreting this metabolic response, an essential role is assigned to the thermoreceptors on the surface of the skin which provide continuous information on skin temperature values, and their rate of variation to the thermoregulation centres. The work of Iggo[160,161] and Hensel[148,149,150] on the functioning of the temperature receptors in the skin demonstrates that the skin receptors which are sensitive to cold have two types of response: a static response, when the number of spikes per second depends on a given skin temperature, and a dynamic response which is proportional to the rate of change of the skin temperature. The sensitivity curve of the dynamic response is identical with that of the static response. According to Iggo, the characteristics of the discharge from the cold-sensitive receptors in primates are a significant parameter in the coding of thermal information. Indeed, when the skin temperature decreases, a discharge is observed which becomes more regular with a gradual increase in the frequency of the spikes, and the appearance of pulse trains as doublets or triplets. The greater the rate of change of the skin temperature, the greater the number of spikes in the sudden bursts and the shorter the intervals between each burst, thus giving the impression of a continuous discharge. Thus the activity of the thermoreceptors in the skin of primates which react to cold can provide a highly accurate characterization of the rate of change of the skin temperature as well as of its instantaneous value. In addition, the existence of thermoreceptors in all areas of the body, both in the surface tissues and in the deeper tissues, has been demonstrated by several authors (Rawson et al.[244], Reidel et al.[246], Jessen et al.[166]).

Taking these works as a basis, it appears that at thermal neutrality the cutaneous endings which are sensitive to cold are not stimulated; they function with a steady firing rate or even show no response. The rapid fall in skin temperature causes increasing activity of these thermoreceptors, while those which are more deeply located show no response. The central nervous system therefore is continuously informed of the temperature of the skin, its rate of change, and the flow of heat through the skin. It can then trigger the reactions to combat cold, peripheral vasoconstriction and increased metabolism, as a function of the rate of fall of skin temperature and its instantaneous value. As the latter gradually stabilises, the peripheral dynamic stimulation becomes less strong and a decrease in the metabolic response is observed but it does not, however, return to its initial value. These assumptions on the functional mechanism are confirmed in the work of Hammel[134] who recognises the importance of skin temperature as a quantity which triggers the thermoregulatory response. This view is shared by Brown and Brengelmann[52], who considered that the initial metabolic peak is an actual

"overshoot" caused by the rapid change of skin temperature. These authors do not, however, allow for other factors which may affect the response, such as a rectal temperature increase or possible interference by other controlling influences, such as that of respiration.

After this initial response, the metabolic rate increases gradually and irregularly as in the case of slow cooling. It approaches a stable or a maximum value according to the intensity of the stimulation. Recently, Timbal et al.[284] showed that the value of the metabolic response, apart from the initial period, could be predicted with a high degree of accuracy for a nude subject at water temperatures of between 30 and 24°C and a water velocity of 0.10 m/s, by the relationship:

$$M(W/m^2) = 931.44 + 5.97(\bar{T}_{s_o} - \bar{T}_{s_t}) - 23.79T_{re} \qquad (2.7)$$

in which T_{s_o} is the initial average skin temperature, $\bar{T}_{s_t}$ is this same temperature at the instant t, and T_{re} is the rectal temperature. If the initial reaction is taken into account, the effect of the rate of change of skin temperature must be included; the following equation was obtained for 10 subjects:

$$M(W/m^2) = 935.46 - 57.77d\bar{T}_s/dt - 5.01(\bar{T}_{s_t} - \bar{T}_{s_o}) - 23.79T_{re}\,. \qquad (2.8)$$

This relationship suggests that the metabolism is dependent on $d\bar{T}_s/dt$, $\bar{T}_s$ and on the central temperature as represented by T_{re}, a conclusion which is in agreement with the results obtained by Benzinger[29] and Craig and Dvorak[84]. In addition, the relative importance of the weighting coefficients for the various temperatures in Equation (2.8) leads to the conclusion that, ultimately, the internal thermoreceptors play a part which is approximately four times more important than the peripheral receptors in stimulating thermogenesis (i.e., after the initial period) thus confirming the previous data of Webb and Annis[297], Nadel et al.[224], Cabanac et al.[63], Bleichert et al.[36]. Finally this relationship provides an explanation for the increase of metabolism after the initial period. It is known, indeed, that following the sudden reaction at the very beginning of the immersion, and in accord with the intensity of the stimulus, the metabolic rate either returns to a value close to neutrality, with no visible shivering at relatively high water temperatures (29, 30°C), or it increases slowly and irregularly, with sporadic periods of shivering for a greater heat loss. During this period, the skin temperature varies very slightly, if at all; the peripheral thermoreceptors therefore have a stable firing rate which depends on the skin temperature and is lower than during the period of variation of $\bar{T}_s$ (Iggo[161]). The thermoregulatory centres respond to this information by increasing thermogenesis. They also receive information from the deep body thermoreceptors. From the point of view of the centres this information may have a limiting or an additive effect on the peripheral stimulus according to the value of the central temperature. If the latter increases, the increase in metabolism in response to cooling of the skin may be reduced, and a metabolic rate close to neutrality will be observed. If, on the other hand, the central temperature drops, there will be a greater increase in the metabolic rate than that due to peripheral cooling alone. Furthermore, any decrease in body heat debt, produced by the increased metabolism, certainly places a check on the latter.

The relationship (2.8) which expresses M as a function of the skin and rectal temperatures is, however, not complete, since it does not take into account all the factors involved, particularly effects of other regulatory factors on thermoregulation. In addition, the use of a mean skin temperature, calculated from the weighted local temperatures as a function of the area and volume of the various parts of the body, does not allow for the local differences in sensitivity to temperature. A similar criticism can be made about rectal temperature, which responds as only a subdued reflection of the variations of a large number of deep-body temperatures. The thermoregulatory centres do in fact "integrate" the data from a very large number of surface and deep-body thermosensors. It might therefore be preferable to express metabolism as a function of the mean body temperature ($\bar{T}_b$) which, to some extent, is a space/time summation of the various body temperatures; however the weighting coefficients for the various elements of a mean body temperature vary with time, as suggested above.

1.3 Intensity of Shivering in Relation to the Temperature of the Water and the Value of the Maximum Metabolic Rate

These two factors are necessary for predicting the tolerance of a man immersed in cold water and have therefore led to numerous experiments being carried out on nude volunteer subjects immersed up to the neck in baths at different temperatures. The results obtained by the various authors (Carlson[70], Spealman[270], Behnke and Yaglou[28], Beckman and Reeves[26], Cannon and Keatinge[67], Craig and Dvorak[84]) show very wide dispersion. This is due to the fact that the experimental procedures are not always comparable, particularly in regard to the velocity of the water and the duration of the experiments. In addition, the subjects show great variability relating mainly to the thickness of the skin-fold (Cannon and Keatinge[167]). The response of the metabolic rate during shivering, as measured in the stable state or, for low temperatures (15°C), after an hour in the water, is not linear, but an S-shaped curve, as will be seen from Figure 2.4 taken from the experimental results obtained on one of the author's subjects. Indeed, in very cold water the metabolic rate tends towards a maximum value, while in the vicinity of thermal neutrality it increases very little, the cold being combated mainly through vasoconstriction of the skin. From thermal neutrality down to a water temperature of 15 or 20°C, depending on the velocity of the water, the relationships between the metabolic rate and the temperature of the water can be described with a good approximation by parabolic equations of the form $M = a + bT_w + cT_w^2$. These relationships are given in Figure 2.4 for one of our subjects whose skin-fold was 12.9 mm. They are of the same type for the other subjects, but it is difficult to quote a general equation in view of the wide variability of the metabolic responses due mainly to the differences in skin-fold thickness between the various subjects (Fig. 2.5). It will be seen

from this Figure that a very thin subject (4 mm skin-fold) will have a metabolic response in moving water at 26°C which is 2.7 times greater than that of a fat subject with a skin-fold of 20 mm. In addition, the water temperature zone in which peripheral vasoconstriction is sufficient to maintain homeothermia is very narrow for thin subjects: about 1°C, whereas for fat subjects it extends, on average about 4°C, from 34 to 30°C. Finally, it should be noted that for a given water temperature the metabolic rate is higher with increasing water velocity since the coefficient of heat exchange (h_c) increases with water velocity. However, this increase is not as great as the increase of h_c would lead us to expect, since it is partly compensated by the decrease in the temperature difference between the skin and the water. Thus, in water at 24°C, for a 57% increase in h_c between $v = 0.10$ m/s and $v = 0.25$ m/s, the metabolism of a subject increased by only 15.6% as shown in Figure 2.4.

The maximum value of the heat produced by shivering has often been discussed. It is generally agreed that shivering can raise heat production in man to 4 to 5 times its resting value, that is, 200 W/m² approximately (Adolph and Molnar[2], Iampietro et al.[158], Hemingway[147], Behnke and Yaglou[28]). However, higher values were found by Beckman[24] for subjects immersed in water at 10°C. These subjects had a heat production equal to 9 times the basal metabolism. We ourselves observed a metabolic rate of 410 W/m² in one of our subjects who was immersed in water at 15°C; that is 8.2 times the basal rate. It would seem however that these values are fairly exceptional and that such a level of metabolism due to shivering can be achieved only by subjects who have been physically well-trained. When such values are observed, the thermal constraint is very severe and the metabolic rate is insufficient to compensate for the heat losses.

Such a maximum metabolic rate cannot be maintained for very long, since the organism is quickly exhausted. As one aspect of assessing tolerance, it is necessary to know for what length of time a man can maintain a given metabolic level. According to Beckman and Reeves[26] a heat production of 2.2 times the basal metabolism can be maintained for an average of 8 hours by a man who has not undergone training. Colin and Houdas[74,75] think that a heat production of 2.8 times the basal metabolic rate can be sustained for about 3 hours. According to our experimental results, this value appears to be slightly underestimated. There is in fact a very wide degree of variability between subjects and it is not easy to quote precise times, since they depend on the maximum oxygen uptake of the individual, his endurance at a given % of his maximum capacity and his state of fatigue before immersion. An additional element in accidental immersion is the psychological factor which is of extreme importance, since it generally increases the response to the cold stimulus and hastens exhaustion. Ultimately, the reduction in metabolic level after prolonged intense shivering is due to exhaustion, but cooling of the body can also contribute since, as the rectal temperature falls below 35°C, shivering is gradually replaced by muscular rigidity, which becomes marked at about 31 to 32°C. This rigidity persists until deep body temperature falls to about 27°C; thereafter, the body undergoes passive cooling.

The production of heat by shivering is not entirely beneficial. Indeed, rapid muscular shaking has the effect of disturbing the boundary layer of still water in the vicinity of the skin and, consequently, leads to an increase in the coefficient of heat exchange in water (see Chapter 1). In addition, shivering helps to maintain a higher temperature difference between the skin and the water than in the case of passive cooling and increases the losses by convection. Finally the increased oxygen consumption leads to an increase in ventilation and in the heat losses through the respiratory tract. This phenomenon again reduces the effectiveness of shivering. Considered together shivering may actually be detrimental for a thin, lightly clothed individual immersed in cold water.

2. Respiration Reactions to Cold Water Immersion

Several authors have described the respiratory changes in a man subjected to sudden cooling in air (Horvath et al.[153], Cabanac et al.[61], Timbal et al.[280]) or in water (Keatinge et al.[174,177,179] and, more recently, Cooper et al.[83]). From the point of view of regulation, two questions arise: what is the mechanism of the respiratory reaction to thermal shivering, and what effect does ventilation control have on thermoregulation? To answer these two questions, it is necessary to consider the chronological development of the various respiratory and thermal parameters. From the very start of immersion, a sudden increase in ventilation is observed, the extent of which varies with the temperature and velocity of the water, the physical characteristics of the subjects and their degree of training. Thus, in still water at 15°C, we measured maximum flow rates of 80 litres BTPS/mn during the first few minutes of immersion for certain subjects, while others had a ventilation rate of only 30 to 40 l BTPS/mn in the same conditions. This increase in ventilation is due to a considerable increase in the flow volume (V_T), which may be doubled or even tripled, and, to a lesser degree to an increase in the respiratory frequency F_R (Fig.2.6). However, in subjects which have undergone some physical training it is noted, in particular, that the flow volume increases while the frequency remains steady, varies little or increases only after a certain period of latency. The initial ventilatory reaction, which occurs at the same time as the metabolic peak described previously, the drop in the skin temperature, and the sometimes violent shivering which is a concomitant reaction, has all the characteristics of hyperventilation. Indeed it is stronger than that of the oxygen consumption, as illustrated in Figure 2.6, in which the metabolic rate increases 3.3 times as compared with its resting level, while there is a fourfold increase in ventilation. This can also be shown by a study of the respiratory equivalent, or "air convection requirements", to use the terminology suggested by Dejours et al.[89,90], that is, the ratio $\dot{V}_E$ BTPS/$\dot{V}_{O_2}$ STPD (flow-rates expressed in l/mn) during the initial reaction and at the end of the immersion period. It will be observed that this ratio is significantly higher during the initial phase, particularly during the first minute of immersion. In certain subjects it can even double during immersion at 15°C. This means that for an identical oxygen consumption, the ventilatory flow rate is much stronger during the first few minutes than after 20 or 30 minutes in the water. This extremely early reaction depends on a neurogenic stimulus which starts in the skin and acts directly on the respiratory centres (Keatinge and Nadel[179]). These latter authors have in fact shown that the catecholamines could not play a significant part in this

response since the plasma levels of adrenaline and noradrenaline practically never varied. This stimulus would appear to be related to the rate of decrease of the skin temperature, and therefore to the dynamic response of the cold sensitive receptors on the surface of the skin. However, Keatinge and Nadel consider that, if the water is very cold, stimulation of these thermoreceptors is not the sole factor involved; the pain-sensitive receptors in the skin, and emotional factors, may also play a part in the ventilatory reaction. This would partly explain the differences between subjects in the amplitude of this reaction. Hyperventilation results in a significant increase in P_AO_2; there is also a considerable decrease in P_ACO_2 as much as 10 to 15 mmHg. The latter checks hyperventilation and its sudden reduction (see during the third minute in the example in Figure 2.6), is associated with a sharp decrease in the metabolic rate, which may return to a value of approximately 80 W/m^2 while heat losses are still of the order of 300 W/m^2. Thus, the reduction in the ventilatory and the metabolic rates after the first few minutes of immersion would appear to be due not only to a slowing down of the rate of fall of the skin temperature but also to the concomitant hypocapnia. However, in cases where thermal stimulation is very strong, for example in water at 5°C, hyperventilation is not followed by a very high degree of hypocapnia and continues to increase, leading to actual respiratory distress which may result in death in cold water (Keatinge et al.[180]).

After the first few minutes of immersion, the disappearance of hyperventilation brings about a gradual return of the P_ACO_2 to its original level. It oscillates around this value, each decrease corresponding to an increase in the ventilatory and the metabolic rates, and to a bout of shivering (Cabanac et al.[61]). Thus, ventilation regulation would probably affect the metabolic response to cold water immersion chiefly at the beginning of the immersion. Subsequently it appears to be responsible for the oscillations in the metabolic rate, the mean level of which however depends on the extent of the peripheral and central cooling.

3. Circulatory Reactions

Exposure to cold in air and in water generally results in cardiovascular reactions to restrict heat losses and to conserve the heat supplied by the organism. It was in fact shown in Chapter 1 that heat is transferred from the central regions of the body to the periphery by conduction through the tissues and by circulatory convection. Heat transfer by conduction, associated with the thermal characteristics of the tissues (thermal conductivity), can be considered as constant. The exchanges by circulatory convection are the only ones which vary, and are subject to the effect of various factors: physical activity, cold, emotions, etc. During exposure of the body to cold, the main characteristic of the circulatory reactions is peripheral vasoconstriction which causes a reduction in the circulation of blood in the skin and, consequently, reduced heat loss to the environment. This vasoconstriction in the skin leads, however, to changes in cardiac functioning, in addition to those due to increased muscular activity and redistribution of the blood mass.

3.1 Peripheral Vascular Reactions

For a complete understanding of the vasomotor phenomena in the skin which occur when the organism is exposed to a cold environment, a brief review will be given of the anatomical structure of the skin and the distribution of the blood vessels.

The skin consists of three main parts, the epidermis, the dermis and the hypodermis. At the boundary of the dermis and the epidermis is the germinative layer which produces the cells in the epidermis. The dermis is composed of two layers: the reticular layer on the surface, which has a large number of vessels for supporting the papillae – the small projections which extend into the epidermis and which are more or less numerous, depending on the parts of the body concerned; and the connective layer, consisting of a number of intricate connective bundles which give the skin its mechanical protective power. Inside the dermis are various structures: hair roots, sweat glands, free nerve endings, or more specialised nerve structures. The hypodermis is the lowermost layer of the skin and extends as far as the fascia superficialis, the lowermost boundary of the skin covering. It is made up of fibrous cones which divide off segments filled with adipose lobules. This fatty tissue is more or less extensive according to the region of the body and the individual studied. It plays a particularly important part in protection against cold, so that the extent of fatty tissue under the individual's skin explains, to a large extent, the differences in tolerance to cold between subjects (Baker et al.[20], Keatinge[174]).

The cutaneous vascular bed constitutes a large reservoir which may contain 10% of the blood volume in a layer no thicker than 1 mm. The anatomical arrangement of the blood vessels in the skin is highly complex. Their distribution and very variable number in different parts of the body would seem to suggest that their function of feeding the various skin structures is of lesser importance, compared with their thermoregulatory function. The blood vessels consist of networks of arteries and arterioles, the capillaries and networks of veins.

Arteries and arterioles: The arteries in the dermis originate in the sub-cutaneous connective tissue and form, at the boundary between the dermis and the hypodermis, a first plexus: the lowermost arteriolar arch. The latter gives off numerous ramifications to the sweat glands, the sebaceous glands, the hair roots and the adjacent hypodermis as well as vessels which ascend through the various dermal layers: these are the branch arteries. Finally, other arteries, originating in the lowermost plexus, traverse the whole of the dermis to form a second network, called the sub-papillary network, which is located in the upper area of the dermis immediately below the epidermis. This network gives off a large number of very small sized arterioles which extend up into the dermal papillae, perpendicularly to the surface of the skin. At this point there is a pre-capillary sphincter.

Capillaries: Much less numerous than those in the muscles, they form separate loops the number of which varies between 20 and 150 per mm^2, according to the body region. The number of capillaries seems to be related to that of the dermal papillae and varies with age.

Network of veins: The veinlets make up four networks, according to Abramson[1], or three, according to Nelms[228], arranged parallel to the surface of the skin at various depths in the dermis, the last network being located at the junction of the dermis and the hypodermis. This plexus constitutes larger veins which traverse the sub-cutaneous connective tissue and connect with the venae comites of the arterial system. All these dermal veins have smooth muscles in their wall.

Arterio-venous anastomoses: These are highly specialised structures located in the dermis at all levels. They enable the blood to pass direct from the arterioles into the veins, avoiding the capillary bed. These anastomoses, which are richly innervated, are under the control of the adrenergic and vasoconstrictor sympathetic nervous system, but dilate under the action of acetylcholine, which seems to indicate some cholinergic and vasodilatory sympathetic innervation.

This anatomical description helps towards a greater understanding of the phenomena which accompany the effect of cold on the body. If the skin is subjected to a moderately cold environment, there is immediate vasoconstriction of the arterioles in the skin (Burton and Edholm[58]), causing a reduction in the peripheral circulation. This vasoconstriction is proportional to the intensity of cooling and affects one, two, or all three arteriolar plexi; if cooling is more intense, even some of the muscular blood vessels are involved, particularly in such extremities as the hands, forearms, feet and legs. The study of skin temperatures using infrared thermography, carried out by Hayward et al.[142], on subjects who had been immersed for 15 minutes at 7.5°C, demonstrates these facts extremely well. Whenever the skin arterioles close, the arteriovenous anastomoses open, thus putting the dermal capillary bed into short circuit. The flow of blood in the skin can therefore vary considerably. This constriction of the skin arterioles does not entirely originate in the nerves, but may be caused directly by the cold (Keatinge[181]).

In certain cases, skin vasoconstriction may be followed by periodic vasodilation if the skin temperature is very low. This phenomenon, known as paradoxical vasodilation, cold induced vasodilation (CIVD), or the hunting reaction, was initially described by Lewis[206], then by Grant and Bland[123], Aschoff[14,15,16,17], and by several other authors. It is generally confined to the extremities, although it may be produced in a number of skin areas (Fox and Wyatt[99]) and is particularly apparent only where there is localised cooling. It can easily be seen by immersing the hand or finger in a mixture of water and ice and measuring the skin temperature of the immersed area. After a rapid fall to below 5°C, the skin rewarms after a few minutes and may rise to 5 to 10°C. This rewarming is followed by a further cooling of the skin, and the phenomenon is repeated. This alternation of rewarming and cooling is due to sudden bursts of vasodilation which bring a flow of warm blood to the hand. During general cooling of the body, this reaction is greatly reduced (Spealman[267,270], Greenfield and Shepherd[125]). Cannon and Keatinge[67], however, have shown that the peripheral circulation of a man plunged into a bath at 5°C increased after 40 minutes, particularly in the extremities where the increased blood flow took the form of sudden bursts. According to Keatinge, the cold-induced vasodilation threshold during whole-body immersion is a water temperature of 10 to 12°C, which is the same as that found by Lewis in the case of local cooling. The hunting reaction seems to be caused by the opening of the arterio-venous anastomoses and the arterioles, either as a result of an axon reflex with liberation of a vasodilatory substance of the histamine type or, according to Lewis, as a direct result of cold-induced vasodilatory paralysis. The latter releases the blood vessels in the skin, particularly in the extremities, from the vasoconstrictor tone to which they are normally subjected. The resulting flow of warm blood rewarms the tissues and the muscles which recover their contractility. However, it is difficult to understand in that case why the hunting reaction is improved by local acclimatization so that it occurs at an earlier stage and at a higher skin temperature. The resulting rewarming of the skin appears to be a protective mechanism in extreme conditions and is considered to play a part in the prevention of cold injuries which may affect the skin (Leblanc[196–198]). It also promotes some recovery of manual dexterity. In the case of whole-body immersion, however, the vasodilatory reactions which occur over the whole surface of the body increase the heat loss and the drop in the central temperature.

The effectiveness of peripheral vasoconstriction is still further increased by the counter-flow heat exchanges which occur mainly in the limbs. This mechanism, described by Bazett et al.[23], is due to the anatomical arrangement of the deep blood vessels in these areas. Indeed, deep body arteries and veins run side by side and can thus exchange heat, the warm arterial blood being cooled gradually by the veins carrying the cold blood from the periphery. The importance and effectiveness of this mechanism depends on the return conditions in the veins. In a cold environment, this takes place mainly through the deep veins close to the arteries. Thus, over the whole length of the arm, for example, the temperature of the arterial blood may fall by 20°C or more. This veritable thermal "shunt" is a particularly effective mechanism; on the one hand, the arterial blood reaches the extremities at a low temperature, so that the heat loss in the extremities is limited by the fact that the temperature difference between the extremities and the environment is reduced and, on the other hand, the gradual rewarming of the venous blood from the periphery reduces the cooling of the central parts of the organism. The result is that the greater part of the body's heat loss is from the trunk (Cannon and Keatinge[67]).

3.2 Effect of Peripheral Vasoconstriction on General Circulation

The peripheral vascular reactions act on the general circulation by causing compensating adaptation.

They involve, in particular, a redistribution of the blood, which increases in the viscera. The blood flow increases in the mesenteric artery (Reïn[247]) and there is an increase in the volume of the liver, indicating a greater amount of blood in this organ (Glaser et al.[116]). Similarly, the vital capacity decreases and Glaser[114] concludes from this that the lungs could be acting as a blood reservoir. In the case of immersion, this function is still further increased. Gauer et al.[107, 108] were the first to suspect that the increase in the central blood volume during immersion was a direct effect of the hydrostatic pressure, which caused the blood in the capacitive vessels in the lower extremities to move towards the intrathoracic vascular bed. This assumption was later confirmed by Arborelius et al.[12] and Lange et al.[192]. This effect of hydrostatic pressure depends on the position of the subject in the water (Begin et al.[27]); in the sitting or vertical position, the blood volume in the thorax may increase to 700 ml, 25% of which is in the cardiac cavities at thermal neutrality. In cold water, this increase has to be much greater, since the peripheral vasoconstriction affects all the areas of the skin. In the extended position of the body, as for example in accidental immersion with a watertight suit which has considerable buoyancy, hydrostatic pressure has, however, a much smaller effect. Furthermore, cold also results in changes in blood volume, with an escape of plasma water of as much as 15% of the volume of plasma resulting in haemoconcentration (Beckman and Reeves[26]). It would seem that this water is fixed mainly underneath the skin in the sub-cutaneous cellular tissue. This reduction certainly means a smaller increase in the volume of blood in the thorax but the relative importance of the two phenomena has not been established.

These changes in the distribution of the blood mass lead to an increase in the central vascular pressures, the systolic ejection volume and the cardiac output, in spite of a bradycardia which may be partly concealed by the increased heart beat rate caused by muscular activity due to shivering. In addition, the volume receptors are stimulated by the increased blood volume in the thorax. This results in a reduction in the antidiuretic hormone secretion and a particularly marked increase in diuresis during the first few hours of immersion or of exposure to cold (Graveline et al.[124], Kaiser et al.[171]).

3.3 Development of the Thermal Conductance of the Body in Cold Baths – Role of the Sub-Cutaneous Adipose Tissue

In Chapter 1, the thermal conductance (h_b) of the water was defined as the coefficient of heat transfer between the central regions of the body, called the core and the periphery. Heat is transferred to the skin by conduction through the intercellular tissues and by circulatory convection, and, as Burton[58] suggests, it may be thought that these two methods operate in parallel, assuming, however, that the heat exchange takes place in a direction perpendicular to the surface of the body. In this case, the conductance values are added. If we consider that the conduction of heat through the tissues is relatively constant, the only variation will be in that part of the conductance which depends on blood convection under the effect of the vasomotor reactions. Before considering the changes in the thermal conductance of the body in a cold environment, it should be stated that, in the opinion of several authors, following Burton[58], the surface skin temperature during immersion experiments was the same as, or very close to, that of the water. This is not true, even for relatively high water temperatures (see Chapter 1). Furthermore, the surface temperatures vary with the skin areas, since the heat flux is different according to the regions concerned. There is therefore a difference, which may be relatively large, between the skin temperature and the water temperature. To neglect this leads to an underestimation of the conductance value and to minimising the variations as between individuals.

The first conductance values for the skin and the sub-cutaneous tissues are due to Lefèvre[200]. During noteworthy direct calorimetry experiments, this author measured the heat lost by the body in baths at 5, 12, 18, 24 and 30°C and recorded simultaneously the skin and sub-cutaneous temperatures. From this he calculated the conductance of the skin and that of the underlying tissues. He was thus able to show that the skin conductance decreased if the bath temperature fell from 30 to 5°C, while the conductance of the sub-cutaneous tissues showed a sixfold increase. The reduced conductance of the skin can easily be explained by peripheral vasoconstriction, but Burton[58] seemed to find it more difficult to explain the increased conductance of the sub-cutaneous tissues. However, a simple calculation of the overall conductance of the body from the values quoted by Lefèvre shows that the thermal conductance of the body is very low and equal to 7 $W/m^2 \cdot °C$ in a bath at 30°C and that it then increases at lower bath temperatures with the development of thermogenic reactions to combat the cold. This pattern of development was also found by Burton and Bazett[57], who showed that the conductance was very low in a bath at 33°C and had a value of 7.17 $W/m^2 \cdot °C$, but then increased gradually at lower water temperatures with a simultaneous increase in oxygen consumption. Similar results were obtained by Craig and Dvorak[84] and by ourselves (Boutelier et al.[41]) during experiments in still water or water which was only agitated as a result of shivering, although the minimum conductance value was observed at lower temperatures. It can therefore be concluded that shivering or exercise, as a means of combating the cold is of limited effectiveness because of the increased heat transfer capacity of the peripheral area, an observation which agrees with that of Keatinge[175] showing that in water at 15°C, exercise considerably aggravates the drop in the rectal temperature.

The role of skin vasoconstriction is not, however, merely to reduce the exchanges by blood circulation through the skin. If cooling is sufficiently intense, the blood circulation in the skin may be completely short-circuited and the heat exchanges between the core and the skin will take place primarily by conduction through the sub-cutaneous fat, resulting in conductance values which may vary markedly for different subjects in the same environment (Winslow and Herrington[303]). Thus, in cold conditions, these values are always lower for fat subjects than for thin ones; Beckman et al.[26] report that the conductance h_b may show a twenty-fold variation between different subjects subjected to the same cold environment, such differences being mainly due to variation in the thickness of the sub-cutaneous adipose tissue. The importance of the sub-cutaneous layer of fat as a protection against cold has been shown by several authors.

Thus, Pugh and Edholm[239] investigated the physiological reactions of Channel swimmers and showed that they had an unusually thick sub-cutaneous layer of fat and were capable of withstanding much lower water temperatures than thin subjects. Similarly, Baker and Daniels[20] report that, for subjects exposed to the same cold environment, the thicker the skin-fold (i.e. the thicker the sub-cutaneous fat), the lower the surface temperature. Thus, fat subjects have a higher central temperature and a lower skin temperature than thin subjects when subjected to the same environmental conditions. This also leads to the conclusion that in a cold environment, the heat losses of fat subjects are lower than those of thin subjects. The importance of the sub-cutaneous fat in tolerance to cold water immersion has been well illustrated by Cannon and Keatinge[67]. These authors found that the fattest men could stabilise their internal temperature in baths at 12°C, while in the case of thin subjects, stabilisation was observed only at water temperatures at or above 24°C. Further, Keatinge[172] showed that there was a linear relationship between the drop in the rectal temperature and the reciprocal of the skin-fold for a group of volunteer subjects immersed for 30 minutes in water at 12°C; he noted, however, that certain individual differences in the rates of cooling could not be explained by different skin-folds, but corresponded better with fairly intense circulatory reactions in the skin.

From this review of the literature, one can conclude that the thermal conductance of the body decreases rapidly in cold water, reaching a minimum value at a water temperature of between 30 and 33°C, and then remains stable or increases with a lowering of the water temperature, giving very different values depending on the subjects and particularly, the thickness of their skin-folds. A recent study of the thermal conductance of 10 nude subjects, immersed to the chin at different water temperatures, provided data on the variation in conductance as a function of the water temperature and the thickness of the sub-cutaneous fat (Boutelier et al.[46]). The results of this study, presented in Figure 2.7, show that at thermal neutrality the value of the conductance for the immersed part of the body ($h_{b\,im}$) is 16.09 ± 1.89 W/m² · °C, and that there is no significant difference between the subjects; in the cold the development of thermal conductance as a function of water temperature consists of two parts: an area of rapid decrease from neutrality to 30 or 31°C, for the majority of the subjects, and an area of stabilisation or increase, according to whether the subjects have a thick or thin layer of sub-cutaneous fat. The lowest conductance value is a function of the thickness of the sub-cutaneous fat (SCF) expressed in mm:

$$h_{b\,im} = -0.847\ SCF + 13.89 \qquad r = 0.91 \qquad n = 10 \tag{2.9}$$

where SCF mm $= \left(\frac{\text{skin-fold}}{2} - 1\right)$.

It should be noted that in this water temperature range over which the conductance decreases, no relationship could be found between the conductance and the heat losses, as the latter were practically constant. This suggests that over this range the organism combats the lowering of the ambient temperature by reducing its conductance by peripheral vasoconstriction, and does not resort to shivering to increase metabolic rate. This vasoconstriction occurs gradually, which can be explained by the anatomical arrangement of the blood vessels in the skin, but it rapidly reaches its maximum value, since it decreases only within a narrow water temperature range: approximately 33.5 to 30°C. In addition, the thinner the skin-fold, the smaller this water temperature range. For immersion at temperatures between 30 and 24°C, there is no significant relationship between conductance and water temperature for 5 out of 10 subjects; their conductance remained at its minimum value. In the case of the three thinnest subjects, it increased significantly, while for the two fattest subjects it decreased slightly. At the same water temperature, thermal conductance thus showed very different values according to the subjects' degree of sub-cutaneous fat. Thus, thin subjects may have a conductance which is three times higher than that of fat subjects at the same water temperature; furthermore, this conductance has a more marked value than at thermal neutrality, although there is no peripheral vasodilation.

The changes in thermal conductance show the major roles played by skin vasoconstriction and sub-cutaneous fat. One might be tempted to say that h_b reflects the circulatory phenomena at the periphery, but its increase in thin subjects suggests a more complex explanation. It can, in fact, be assumed that the body consists of three sections (Fig.2.8): a core made up of the viscera and the skeleton, a muscular region and a periphery comprising the sub-cutaneous fat and the skin, reduced to the epidermis and a part of the dermis. There are three coefficients of heat transfer for these three sections: h_n for the core, h_m for the muscle and $h_{s.SCF}$ for the periphery. The overall coefficient h_b represents the combined effect of these three conductance values and a very low value for h_b does not mean that the individual coefficients for each of the constituent elements are also very low. A detailed study of these variations is possible only if the mean temperature and the heat production values for each of these regions are known. It is, however, possible to obtain some idea of the development of conductance between the core and the muscular region $h_{n.m}$ if we assume that the heat flux in each region is perpendicular to the surface and that the heat produced in the peripheral region and the reduction in the area between the skin and the surface muscular region are negligible. In addition, to calculate the conductance values of the skin (h_s) and of the fat (h_{SCF}) use is made of the thermal conductivity values given by various authors; for wet skin, 322 W/m² · °C for a thickness of 1 mm, and for fat, 209 W/m² · °C for a thickness of 1 mm (Hardy and Soderstrom[137]). The thickness of the sub-cutaneous fat is determined by measuring the skin-fold (see Equation (2.9)). Figure 2.9 contains the results of such calculations for three subjects: a fat subject, a subject with an 11 mm thick skin-fold, and a thin subject. It was observed that the peripheral conductance ($h_{s.SCF}$) was 16 times less for the fat subject, while the thin subject showed only a threefold reduction. In addition, the minimum value of h_b corresponds to the minimum $h_{s.SCF}$ value only in the case of the thin subject. The minimum $h_{s.SCF}$ value for the other two subjects was reached at higher water temperatures (30 to

32°C) than those at which the minimum h_b value was observed. This means that there is a reduction in conductance in the core/muscle region. In colder water, however, conductance increases in this region, particularly in the case of thin subjects. The reduced conductance between the core and the muscular region can be interpreted as the result of cooling of the surface of the muscular region, which probably participates in peripheral insulation. It is not therefore a question of racial adaptation to cold, as suggested by Inoue[162]; the increase in this conductance for higher thermal losses can probably be explained by a considerable rise in the metabolic rate, a more active blood circulation in the muscular region and a lower temperature difference between the core and the muscular surface because of the heat lost by the core.

The thermal conductance of the body is therefore a complex coefficient; its variation depends on the condition of the peripheral blood vessels, the thickness of the sub-cutaneous fat, and the irrigation and activity of the underlying muscle tissues. The increase observed in cold baths shows that the insulation due to vasoconstriction of the skin may be insufficient to reduce heat loss if there is considerable muscular activity. This finding agrees with that of Keatinge[175] who showed that the rectal temperature of subjects immersed in water at 15°C fell more when they were swimming than when they were resting. This fact emphasises the need to remain as motionless as possible in the case of accidental immersion, in order to reduce heat losses.

4. Variation of Physiological Temperatures

4.1 Mean Skin Temperature

The mean skin temperature of a naked subject falls rapidly at the start of immersion. This fall is steeper, the greater the temperature difference between the skin and the water. However, it is retarded somewhat, in spite of skin vasoconstriction, by the heat produced by the initial metabolic reaction to the immersion. It should be noted that the rate of variation in the skin temperature is greater for fat subjects than for thin subjects when placed in the same environmental conditions. This is due to the improved thermal insulation provided by a thicker sub-cutaneous layer of fat in fat subjects and to a weaker metabolic reaction.

After 15 to 20 minutes for the naked subject, the skin temperature stabilises at a higher temperature than the temperature of the water, even if the latter is stirred. It does, however, show slight variations corresponding to sudden bouts of shivering. The temperature difference between the skin and the water has been the subject of much discussion. Many authors assume, in fact, on the basis of the opinion expressed by Burton et al.[57] that this temperature difference is less than 0.08°C in agitated water. Yet as early as 1898, Lefèvre[200,201] had shown that the skin temperature remained very much above the temperature of the water. After 15 to 20 minutes' immersion, this author found differences of 1.5°C in water at 25°C, 4°C in a bath at 18°C, 7°C in a bath at 12°C, and 12°C when the water temperature was 5°C. Similarly, Behnke and Yaglou showed that the temperature of the trunk was more than 10°C higher than the water temperature in water at 6.5°C. Krog[184,185] made the same observation for the skin temperature of the hand: in water at 0°C, the hand temperature of Lapps and Norwegian fishermen was about 5°C. These various results also show that the temperature difference between the skin and the water increases when the water temperature is lower. This increase is particularly evident if the water temperature is less than 30°C, when there is a linear relationship between the mean skin temperature $\overline{T}_{sim}$ and the temperature of the water (Boutelier[44]). Thus, in water flowing parallel to the major axis of the body at a velocity of 0.10 m/s, we obtained the following mean relationship:

$$\overline{T}_{sim} = 0.926\, T_{H_2O} + 2.95 \,. \qquad (2.10)$$

The correlation (r) was 0.77 for 51 experiments on ten subjects. There were, however, considerable individual differences, with thin subjects having a higher mean skin temperature than the fat subjects because of their higher metabolic reaction and their lower peripheral insulation.

4.2 Local Skin Temperatures

These change in a manner comparable to that of the mean skin temperature (Fig.2.10). In the stable state, they can be divided into three groups from the warmest to the coldest: the trunk, the limbs and the extremities. The chest is the warmest region of the trunk, the back and the abdomen having appreciably the same surface temperature. The temperatures of the limbs are very similar and lower than those in the trunk. The extremities, e.g. hands and feet, are much colder than the rest of the body and, although the temperature difference between the skin in these regions and the water increases with a lowering of the water temperature, this difference remains slight, reaching only 0.8 to 0.9°C in water at 14.5°C. In colder water, the difference is greater and considerably more variable because of the cold induced vasodilatation. These changes in local skin temperatures lead to the conclusion that the loss of heat in water is mainly from the trunk and, to a lesser degree, from the limbs.

4.3 Variation in Rectal Temperature

The rectal temperature (T_{re}) variation, a reflection of the deep body temperatures of the organism, is more complex. During the first few minutes of immersion it remains stable and then begins to rise at about the 5th minute; it reaches a maximum and thereafter decreases, very rapidly if the water temperature is low. This pattern of development is, however, likely to vary considerably from one individual to another. Indeed the variation in the rectal temperature depends on

three main factors: the ambient temperature, the thickness of the sub-cutaneous fat and the extent of the initial metabolic reaction. Thus, it will be seen from Figures 2.11 and 2.12 that the increase in the rectal temperature is proportional to the thickness of the sub-cutaneous fat and that there is no increase if the average thickness is less than 1 mm. In addition, the rectal temperature increase is greater as the temperature of the water is lower. An apparently paradoxical phenomenon can be observed in fat subjects; in moderately cold baths the rectal temperature in the stable state may be higher than the initial rectal temperature but in a very cold bath the final rectal temperature is always lower than the initial T_{re}, with the amount of this drop inversely proportional to the thickness of the skin-fold.

The increased rectal temperature during the transition period has been noted by a number of authors: Lefèvre[201], Burton and Bazett[57], Aschoff[13,18], Keatinge[172,181], Craig and Dvorak[84], Wyndham et al.[305], who have generally attributed this phenomenon to the development of abnormal temperature gradients inside the body and to the effect of vasoconstriction of the skin. Aschoff, for example, suggests that cold causes skin vasoconstriction which reduces the coefficient of heat transfer between the core and the skin. This means a reduction in the total heat loss, to a level less than the metabolic heat production, and hence an increase in the temperature of the core. These authors further emphasise the independence of the core and the periphery from the point of view of heat. Lefèvre[201] stated indeed that at the start of cooling the periphery was poikilothermal, while the deep tissues maintained their homeothermia. Wyndham and McDonald[305] felt that this independence was merely relative and depended on the value of the thermal resistance between the deep tissues and the surface tissues. It can be concluded that the rectal temperature increase was caused by a storage of heat in the core due to increased metabolism and an increase in the peripheral thermal resistance under the effect of vasoconstriction of the skin. An analysis of the results of a number of immersion experiments, using the three compartment model shown in Figure 2.8, enabled us to show that the elevation in the rectal temperature was caused to a large extent, if not entirely, by the metabolic and circulatory reactions during the initial stage (Boutelier[45]). Indeed, because of peripheral vasoconstriction and the intense shivering at the start of the immersion, heat was stored in the muscular region, since the flow of heat to the periphery is greatly reduced by the presence of sub-cutaneous fat. This stored heat produces a rise in the mean muscle temperature, to a higher level than the temperature of the core as represented by the rectal temperature. The result of this is a transfer of heat from the muscles to the core, which is accelerated by the increased circulation between these two regions, and a gradual rise in the rectal temperature. The extent of this rise thus depends, to a large degree, on the thickness of the sub-cutaneous fat (Fig.2.12), resulting in wide differences between fat and thin subjects in the development of the rectal temperature. This mechanism for the variation in the central temperature is in agreement with the work of Burton and Bazett[57] who showed, on a cylindrical model, that the sudden reduction in the conductance of the wall caused a reversal of the temperature gradient and an increase in the central temperature. With regard to the heat loss distribution between the various regions, these observations also make it possible to state that, at the very beginning of immersion, heat losses occur mainly at the expense of the periphery.

The reduced metabolic reaction after the first few minutes of immersion, and its subsequent gradual increase, cause the drop in the muscle temperature and the reestablishment of a normal temperature gradient between the core and the periphery. This reestablishment obviously takes longer in the fat subject. The rectal temperature then starts to drop, the extent of the drop depending on the metabolic value, the conductance values between the various regions, their mean temperature and the extent of the heat exchanges with the environment. The development of the rectal temperature is thus a complex phenomenon and there is no simple relationship between it, the heat losses to the outside and the heat debt undergone by the organism. The main factor which will help to predict the extent of the drop in the rectal temperature is the thickness of the sub-cutaneous layer of fat. This can also be taken as a basis for stating that the distribution of the heat loss between the core and the periphery is not the same for the fat and the thin subject. For the same overall heat loss, the central region (the core) of a fat subject may lose three times less heat than the core of a thin subject.

These arguments suggest that the rectal temperature varies only as the result of the heat exchanges between the various regions of the body and between the periphery and the environment. This variation appears therefore to be a passive phenomenon.

5. Hormonal Reactions

In 1902, Rubner classified the thermogenic mechanisms into two groups: the physical mechanisms which increase body heat production by a visible process, such as shivering or muscular exercise, and the chemical mechanisms which cause the basal metabolism to rise through the operation of exothermal chemical reactions. The latter were thought to be under the control of the endocrine glands, and the physical mechanism under the control of the nervous system. This classification has now been abandoned, since in the cells the two mechanisms are governed by the same chemical processes. Similarly, there is no distinction to be made between control of the body temperature by the nervous system or by hormones; the control exercised by the hormones is subordinate to the control by the nervous system. The neuroendocrine system therefore appears as an intermediate system located between the thermoregulatory nervous centres and the effectors. From a functional point of view, the effector responses to nervous stimuli are quick, whereas the reactions to hormonal stimuli are slower, more sustained and more general. It may therefore be thought that one of the roles of the neuroendocrine system in thermoregulation is to maintain and to intensify the vascular and muscular reactions initiated by a reflex nervous mechanism acting either directly on the effectors or on a more general level, enabling the effectors to use high energy substrates.

Very briefly, hormones can be classified into three types: those involved in the short-term response (a few minutes to a few hours) such as the cathecholamines, adrenaline and noradrenaline; those concerned in the medium term response (several hours) such as the glucocorticoids; and finally, such hormones involved in the long-term adaptation process as the thyroid hormone, sexual hormones and hypophysial regulator. The first two categories of hormones are mainly the ones involved in accidental immersion. Recently in a paper on the neuroendocrine aspects of thermo-regulation, Gale[106] reviewed the considerable work carried out in this field and stressed the importance of the role of the sympathetic adrenal medullary system in controlling body temperature.

5.1 Role of the Sympathetic Adrenal Medullary System

As early as 1926 Cannon et al.[65] reported that the quantity of adrenaline in the blood circulation of cats increased after ingesting iced water, thus showing the part played by the suprarenal medulla in the thermogenesis associated with shivering. Since then, several authors have shown that exposure to cold causes increased adrenaline and noradrenaline secretion. For example, Leblanc and Nadeau[197], after experiments on the rat, concluded that the intensity of shivering depended on the increased secretion of the catecholamines: noradrenaline from the adrenergic nerve endings, and adrenaline from the suprarenal medulla. Similar results were obtained on human subjects exposed to cold environments. The majority of authors found, however, that the noradrenaline secretion increased more than the secretion of adrenaline in moderately cold environments (Tsunashima[288], Feller and Hale[97], Suzuki[273]); the critical temperature under which this increase occurred was 15°C according to Wilkerson[300]. It is now generally assumed that the catecholamines act on the cardiovascular system by increasing the cardiac output and readjusting the blood flow in the organs (Jansky et al.[164]), and by permitting vasoconstriction of the skin (Nagasaka and Carlson[226]). In addition, these hormones act on the adipose tissue to mobilise lipid substrates (Wilson[301], Himms-Hagen[151]) and increase cell oxidation (Chatonnet and Minaire[72]). Finally, they promote shivering by direct action on the nervous system and possibly by liberating acetylcholine at the neuro-muscular junction (Kuba et al.[186]).

5.2 Role of the Corticosuprarenal Hormones

Cold has a stimulating effect on secretion of the glucocorticoids. An increase of cortisol in the plasma (Suzuki[274]), which returns to its point of departure six hours after exposure to cold, is generally observed. There is therefore an increase in the 17-urinary hydroxycorticosteroids; Budd and Warhaft[55] also observed an increase in the 17-ketosteroids. The function of the glucocorticoids is to increase gluconeogenesis by katabolising the proteins, to reinforce the action of adrenaline in mobilisation of the fats, to make the arterioles sensitive to the vasopressor effect of the catecholamines and to cause some rise in blood pressure. Cortisol also increases the excretion of water and helps to inhibit the antidiuretic hormone.

5.3 Effect of the Thyroid Gland

This gland plays a particular role in cold acclimatization by increasing the basal metabolism. In exposure to severe cold, however, the rate of renewal of the thyroid hormones increases and the plasma TSH increases (Schneider[255], Berman et al.[30]).

5.4 Role of the Hypophysis and the Hypothalamus

The activity of the adrenal cortex and the thyroid is controlled by the anterior hypophysis which secretes regulatory hormones whose main purpose is to stimulate production of the hormones in these target endocrine glands. Thus, under the effect of cold, as for any stress, the anterior hypophysis secretes the hormone corticotrophin (ACTH) the concentration of which increases rapidly in the blood. It acts on the adrenal cortex, stimulating the liberation of adrenocortical hormones. Its effect is not, however, restricted to the adrenal gland; it also causes major changes in lipid metabolism, mobilising the non-esterified fatty acids from the lipid depots and increasing the rate of oxidation of the lipids. It also increases the glycogen in the muscles. A similar mechanism exists for the thyroid gland, the activity of which is stimulated by the hormone thyrotrophin (TSH) which also promotes lipolysis.

The activity of the anterior hypophysis is controlled by the hypothalamus, which secretes substances promoting the release of the hormones produced by the anterior hypophysis: the "corticotrophin releasing factor" (CRF) in the case of ACTH and the "thyrotrophin releasing factor" (TRF) for TSH. The view is held that there is some servo-regulatory control by the levels of adrenocortical and thyroid hormones over the neurones which synthesise these releasing factors, via chemoreceptors. Finally this activity of the hypothalamus is itself controlled by the higher centres in the nervous system.

This brief account shows that exposure to cold causes increased activity of the adrenal medulla, the adrenal cortex and, to a lesser extent, the thyroid, the latter two glands being under the control of the hypophysis. Production of these hormones, which increases at all levels of cold stimulus, makes it easier to combat cold, and therefore to maintain a constant body temperature, by their vasomotor effects and its effect on gluconeogenesis and lipolysis, supplying high energy substrates to the muscles.

6. Centres Controlling Physiological Reactions to Cold – Body Temperature Regulation

A review of the many experiments carried out in this field is outside the scope of this report, which will therefore confine itself to a short review of the main results and assumptions, and refer the reader to more specialised accounts by Hemingway[147], Hardy[139], Hammel[134], Hensel[149], Bligh[37,38] or Cabanac[24].

6.1 Thermosensitivity and Afferent Thermal Sensation Receptor

Receptors are usually classified as tegumentary receptors, deep-body receptors and receptors in the central nervous system. The existence of temperature receptors in the skin which are sensitive to cold is no longer a matter of doubt in man, because of the work done by Hensel. Some of the afferent fibres are myelinised. After passing through the spinal ganglion, they enter the dorsal cornua of the marrow, where there are ganglionic cells. The ascending branch then crosses the white commisure and continues to ascend via the lateral spino-thalamic bundle to the thalamus, where it acts as a relay, and from there goes to the sensori-motor cortex. Certain fibres go direct to the hypothalamus.

Deep-body thermosensitive structures have been found in numerous species and in various parts of the body: in the viscera (Rawson and Quick[244]) as well as in the veins (Blatteis[35]). On the other hand there is no direct evidence of thermal sensitivity in the muscles, although indirect arguments have been put forward to prove its existence (Saltin et al.[252]). Finally, evidence has been found to suggest that there are thermosensitive neurones in the central nervous system, particularly in the hypothalamus (Hensel[150], Hellon[145], Cabanac et al.[62]), the bulbous reticular formation (Nakayama and Hardy[227]) and in the spinal cord (Thauer[279], Simon[260], Jessen et al.[165]). The existence of so many and such widely distributed thermosensitive structures enables the control centres to obtain very precise information on the thermal condition of the body.

6.2 Thermoregulatory Centres

The existence of these centres has been demonstrated mainly through lesion and stimulation experiments. Thus, the dorso-median posterior hypothalamus has been shown to contain groups of neurones, the stimulation of which produced shivering and peripheral vasoconstriction (Hemingway[147]). This area is the main control centre of the reactions to cold. Stimulation experiments in adjacent regions showed that the pre-optic area of the anterior hypothalamus exerted an inhibiting influence on the posterior hypothalamus, preventing the occurrence of cold defence reactions. An inhibiting area would therefore seem to exist in the posterior hypothalamus itself. In addition, the dorso-median posterior hypothalamus receives afferent nerves from the higher regions in the central nervous system. These nerve impulses, either inhibiting or exciting, come from the cortex, the septum (Andersson[6]) and the anterior thalamus.

The lesion experiments showed that the posterior hypothalamus was probably not the only control centre, since coarse control of the body temperature persisted after its destruction (Andersson et al.[7]), with cold defence reactions coming into play, but only at much lower temperatures. Thus, according to Bligh[38], there are two control systems: one, for fine control, located in the hypothalamus, and the other coming into operation only when the body temperature deviates considerably from its normal range of variation; these secondary centres are located mainly in the medulla. Nevertheless serious thermoregulatory disorders occur in man and other mammals if the specific regions of the hypothalamus are destroyed. We would emphasise, with Gale[106] that the proximity of the hypothalamic centre for shivering to various nuclei belonging to the sympathetic nervous system would seem to suggest very close functional interrelationships.

6.3 Functioning of the Thermoregulatory System

Body temperature control therefore appears to be a very complex system which uses a variety of systems, each having its own control system, to respond to external or internal stimulation by cold or heat. To explain how it works, reference has been made for some ten years or so to the control models described by automation experts. The most conventional model is the closed loop model. This consists of two elements: a control system (or active system) and a controlled system (or passive system). The control system is made up of two parts: an error detector which detects any difference between the output signal of the passive system and a control signal. This detector therefore supplies an error signal as an input to the control system which feeds a control signal as an input to the controlled system; the latter may also receive disturbance signals directly. The output from the passive system is fed back to the error detector. The aim of this system is to keep the output signal as close as possible to the control signal. If the latter is constant, this is called a "set point", and a control system is involved. If it is variable and the system tries to adjust its output to this variable signal, we have a controlled, or servo-system.

In the field of thermal physiology, most authors consider that a control system with a set point is involved. The fullest development of this concept can be found in the review papers by Hardy[139] and Hammel[134]. Other theories have, however, been advanced and Houdas[154] has recently put forward the idea of a body temperature control system by means of a servomechanism which controls heat exchanges and is not based on a specific set point concept.

On the assumption of a set point control system, observation of homeothermia led to the assumption that the internal body temperature was the controlled element in the system. The concept of internal temperature is, in fact,

a vague one, since there is some heterogeneity in the temperatures of the various organs. It was therefore assumed that the controlled temperature was that of the hypothalamus or the arterial blood reaching the hypothalamus. However, the present tendency is to consider that the controlled element is a function of the various body temperatures, that is, a kind of mean body temperature (Hensel[150]).

The reference signal should also be clarified. In his proposal Hammel takes electrophysiological data showing that a large number of neurones in the hypothalamus discharge constantly at all temperatures, as a basis for assuming that this frequency is the reference signal with which the signals from the thermoreceptors are compared. In fact, according to Hensel, the set point should rather be considered as a function of various reference temperatures for metabolism, vasomotor and evaporation status. Furthermore, as the internal temperature undergoes periodic fluctuation, and may stabilise at higher or lower levels according to circumstances, Hammel has suggested that the set point could be adjusted in relation to the afferent influences. However, the question of which quantity is being controlled and what constitutes the reference signal are matters which are still far from being settled.

7. Other Effects of Cold

In addition to the physiological reactions described above, cold may be the cause of a functional handicap in the extremities, and particularly in the hands. This may have serious consequences in accidental immersion by preventing the victims from using their hands fully.

Manual performance in a cold environment has been the subject of numerous investigations aimed at ascertaining the precise mechanisms responsible for manual performance deterioration. The tests used do not all have the same significance. Some of them are concerned with actual manual dexterity, such as the test applied by Teichner[277], consisting in taking out, turning over and replacing wooden discs 3.8 cm in diameter in holes of the same size. The discs may be grasped only by the part of the disc which extends beyond the edge of the 1.1 cm hole. Other tests involve not only manual dexterity but higher levels of vigilance, consisting of complex coordination tasks, such as the pursuit tests (Teichner and Kobrick[276]), reaction times (Teichner[278]) or a combination of both.

The most valuable physiological parameter to be taken as a reference is the skin temperature of the hand, and particularly of the fingers, and its rate of variation (Gaydos and Dusek[109], Clark and Cohen[73]). There is, in fact, a fairly good correlation between manual performance and these temperatures.

The reasons for the reduction in manual dexterity after exposure to cold are fairly well known, but their respective importance, as well as their closely associated mechanisms often remain obscure. The following can roughly be distinguished: shivering, reduced tactile discrimination, pain, stiffness in the muscles and joints, nerve conduction disorders, freezing. The last two will be dealt with in detail in Chapter 3.

Trembling of the hands under the effect of shivering is due, in actual fact, to a general cooling of the body. Trembling of the whole body right down to the extremities may make it very difficult, even impossible, to perform the simplest task, for example, pressing a button. Additionally, the very unpleasant feeling associated with shivering considerably reduces attention to a task.

Some loss of tactile discrimination has been demonstrated with the anaesthesiometer or Mackworth's V test[214]. This author defined a numbness index which is the difference between the minimum two-point tactile separation perceived at thermal neutrality and in various conditions of cold. This distance increases with cooling of the skin and its value can be used to assess the reduction of tactile sensitivity. When this index reaches a value of 14 mm, the skin is close to the freezing point. This reduction in tactile discrimination is accompanied by a loss of sensitivity to pressure (Mills[219]).

Stiffness in the muscles and joints is felt particularly severely when the hands are required to perform a delicate task. According to Robinson et al.[251] the muscles show increased tension and viscosity. The viscosity of the synovial fluid also increases during cooling (Hunter et al.[157]). This phenomenon is therefore bound to restrict the mobility of joints subjected to cold exposure.

Disturbance of nerve conduction as a result of cold was shown on a nerve fibre isolated by Buchtal and Rosenflack[53] who demonstrated that there is a linear relationship between nerve conduction velocity and temperature. According to Vanggaard[291], the conduction velocity decreases by 15 m/s for a 10°C cooling of the forearm, and if the local temperature reaches 8 to 10°C there is a complete nervous block.

Pain in the hands due to cold results in functional loss of power which is prejudicial to any form of manual activity. According to Blockley[39], sensations in the hands are described as follows, depending on the local skin temperature:

20°C: uncomfortable
15°C: extremely cold
10°C: painful.

From the present author's personal experience it would seem, however, that the temperature difference between the hand and the forearm is a factor in these pain sensations. Similarly, if the temperature of the back of the hand reaches 8°C, the pain becomes unbearable; in such a case, the finger temperature is 5 to 6°C.

It should be noted, finally, that the cooling of the body, even above the generally assumed tolerance limits, causes deterioration in the functioning of the central nervous system which results in a "numbness" of the intellectual faculties, well known to cold water divers.

III. TOLERANCE AND SURVIVAL AFTER IMMERSION IN COLD WATER

The description of the physiological reactions to cold water immersion indicates that, in spite of the important part played by sub-cutaneous fat, the organism's defensive means are limited. Reaching these limits makes it possible to determine the survival time; i.e. the period of immersion for which all the individuals, or some of them, can be recovered without serious or irreversible damage having occurred. Three limits can be distinguished, as indicated by Molnar[222]; survival in 100% of cases, survival of 50% of the victims, and the fatal threshold. Such a study of survival is obviously unsuitable for experimentation and can be made only through the accounts of accidents and shipwrecks. The most important survey in this field was that of Molnar who used the US Navy's Reports of shipwrecks which had occurred during the Second World War. On the basis of these data, Barnett constructed the survival nomogram in Figure 1 (Introduction), still used in a number of countries. It should be pointed out that this is merely an approximate survival estimate, as it concerns clothed men, often wearing very different types of clothing, and does not take account of the morphological differences which may considerably reduce the survival time. Beckman and Reeves[26] have stressed, moreover, that such estimated survival times should be only used with the utmost care when applied to naked or lightly clad subjects.

Since Molnar's work, numerous experiments on voluntary subjects have been performed in an attempt to find the precise limits of voluntary tolerance in relation to physiological and morphological criteria. This research has produced tolerance prediction models which will be discussed in detail in Chapter 4. Voluntary tolerance can be defined as the limit at which a man no longer feels capable of sustaining the thermal stress. It is conditioned by the occurrence of various disorders such as nausea, headache, cramp and/or irregular heart beat, as a result of having reached the extreme skin and/or central temperatures and the maximum heat production, or, if the water is very cold, because of a very strong and uncontrollable hyperventilation reaction, with a considerable increase in the inspiratory phase which may cause drowning in a few minutes (Keatinge[181]). Although the relationship between voluntary tolerance and survival is difficult to determine, it is generally assumed that the limit based on these tolerance criteria is very close to that of 100% survival.

1. Physiological Limits of Voluntary Tolerance

1.1 Skin Temperatures

Several limits have to be taken into consideration; those associated with the mean skin temperature and its rate of change, and those which depend on the local skin temperatures. Iampietro[159], when exposing subjects to a cold air environment with different wind speeds, observed that the voluntary tolerance was 120 minutes if the mean skin temperature reached a mean value of 21.2°C, 60 minutes for a $\overline{T}_s$ of 17.2°C and 20 minutes for a $\overline{T}_s$ of 15°C. In addition, he stated that the development of this mean skin temperature during the first ten minutes made it possible to predict the voluntary tolerance time. We ourselves[40] studied the voluntary tolerance of naked and clothed subjects to cold water immersion, and noted that the subjects requested termination of the exposure when their mean skin temperature reached 23.1 ± 1.8°C. The mean skin temperature could, however, be lower if the exposure time were shorter. This was in fact observed on a subject wearing a dry immersion suit (Mutta), when the $\overline{T}_s$ after 27 minutes' immersion in water at 5°C became 14.7°C. We also noted that the rate of change of the skin temperature during the first five minutes of immersion helped to predict the voluntary tolerance time (Fig.2.13), in spite of a fairly wide dispersion due probably to individual differences and to the very varied experimental conditions. This similarity of results with those found by Iampietro shows that, whatever the ambient environment, there is a relationship between voluntary tolerance time, mean skin temperature and its rate of variation during the initial period of exposure to cold.

An analysis of a few cases in which the tolerance limit was reached at a mean skin temperature of more than 23°C has demonstrated that an extreme local cooling could be the cause of the termination of the experiment. Thus, a skin temperature of approximately 10°C for the back of the hands and the upper part of the feet seems to be the pain threshold for most subjects. This temperature corresponds to a finger temperature of 7 to 8°C, a value which is close to that indicated by Beckman[24,25] for the pain threshold. For the back and the chest, we noted that temperatures of 21 to 22°C represented the tolerance limits for subjects who had undergone some physical training.

1.2 Rectal Temperature

The rectal temperature, which reflects the degree of cooling of the central regions of the body, is a valuable index for determining tolerance limits. A lower limit of 35°C is generally assumed to be the level which must not be exceeded if damage is to be avoided, particularly during the rewarming period. It should be noted that subjects do not experience

any particular sensation when the rectal temperature drops, in spite of the shivering which accompanies this drop. However, below 35°C the metabolism decreases and the central temperature falls more quickly.

1.3 Metabolism

From the point of view of metabolism, Benzinger[29], Behnke and Yaglou[28] and Hemingway[147] assumed that the maximum heat production by shivering does not exceed 200 W/m² and that it cannot be maintained for more than 40 to 60 minutes. The present author, however, observed values of 250 W/m² during one hour on subjects who had not had any physical training. Higher metabolic rates can be measured during shorter periods (Boutelier, unpublished results). For longer periods of immersion (8 hours), a mean heat production of 120 W/m² was found by Beckman and Reeves[26] on 24 nude subjects immersed in water at 23.9°C; only a quarter of these subjects were able to tolerate immersion for 12 hours. Finally, for immersion periods of from 24 to 36 hours, Colin and Houdas[74,75] estimated that no subject could have a metabolic rate of more than 100 W/m², that is, twice the basal rate.

2. Determination of Voluntary Tolerance Time

On the basis of the data quoted above and the heat losses determined on 10 naked subjects at various water temperatures with a water velocity of less than 0.10 m/s flowing parallel to the major axis of the body (Boutelier[44,50]), it was possible to calculate for each subject the water temperatures which he would be theoretically capable of tolerating for 1 hour, 8 hours or 24 hours. In Figure 2.14 the subjects have been divided into three groups according to the thickness of their skin-fold; Group I has an average skin-fold of 20 mm, Group II a skin-fold of 13.2 mm, and Group III a skin-fold of 4.9 mm. The tolerance time of 2 hours was determined when the mean skin temperature became 21 to 23°C (hatched area in Figure 2.14). At this limit the water temperature will be between 19 and 21.6°C. In still water, however, this temperature may be slightly lower at 17 to 18°C. It should be pointed out that Group III, comprising the very thin subjects, showed only a very limited tolerance time in cold water. Indeed, in water at 26°C (v = 0.10 m/s), their rectal temperature was very close to 35°C after two hours' immersion, and they showed signs of fatigue with convective heat losses close to the permissible maximum (170 W/m²). For a tolerance time of one hour, it is difficult to state precise values. The present author has in fact observed some subjects who could tolerate immersion for 90 minutes or more in still water at 12.5°C, while others could not tolerate immersion for longer than 20 minutes in water at 15°C. In the case of the short tolerance times, there is thus a wide degree of variability which cannot be explained by morphological differences alone. These results also show that at best (fat subjects) a tolerance time of 24 hours is reached only in water at 25°C for naked and motionless subjects, the average being found in water at 26.5°C. The good agreement between the present author's results and those of Beckman and Reeves (as indicated by the vertical dashed line on Figure 2.14) should also be noted. The differences between these estimates and those of Molnar (curves 1 and 2 in Figure 2.14) can be explained primarily by the fact that in his shipwreck studies the men were clothed, sometimes warmly, and this increased their tolerance time. Further, these are survival curves and not voluntary tolerance curves. However, the survival times proposed by Molnar seem to be somewhat optimistic, to judge by the shipwreck of the Lakonia during which 133 people died in three hours, the majority from cold in water at 17–18°C.

The question may be asked whether the tolerance time in cold water would not perhaps be improved by increasing heat production through exercise. However, Keatinge[175] has shown that muscular exercise accelerated the drop in rectal temperature in water at 5 and 15°C, while it had no effect on this temperature in water at 25°C. This fact can be explained by a large increase in heat losses because of the increase in the coefficient of heat exchange in water and the increased skin temperature. As a result, it will generally not be to the advantage of victims of a disaster to swim vigorously in cold water, unless they know that help is very close at hand. Finally, considering that the average temperature of the water in the North Atlantic Ocean is lower than 20°C, the period of survival will clearly be extremely limited (between half an hour and 3 hours) in the majority of cases for naked or scantily clad men. Cross-Channel swimmers who stay for 15 to 18 hours in water at temperatures often less than 15°C constitute a remarkable exception, but these are trained individuals who have a very well developed sub-cutaneous layer of fat and who, in addition, cover their bodies with a thick layer of grease.

IV. COLD ACCLIMATIZATION

Since survival after accidental immersion is very limited, the wearing of protective clothing is absolutely essential. However, despite its quality, it provides only relative protection against hypothermia, and it would therefore seem advantageous to try to improve cold tolerance by acclimatization, particularly in the case of crews who fly over cold regions or carry out long missions over oceans. Achieving such improvement can in fact help the victims make the movements which are essential for survival.

1. What is Acclimatization?

Acclimatization must be distinguished from adaptation or habituation. According to Eagan[93], adaptation refers to physiological changes occurring over generations which are transmitted genetically and help to promote survival in a hostile environment.

Acclimatization, on the other hand, is a functional compensation which is established over a period of some days or weeks in response either to complex, natural environmental factors, such as climatic variations (natural acclimatization or acclimatization), or to an artifically controlled, usually simple environmental factor (artificial acclimatization or acclimation) (Eagan[93]).

Habituation is distinguished from acclimatization by its reversibility and by the fact that it involves some loss of normal responses or sensations. There are two kinds of habituation: specific or local habituation, which is familiarization by a given region of the body to a repeated stimulus, and general habituation, which is reflected in a lowering of the responses of the whole body to a repeated stimulus. Any acclimatization experiments will therefore be concerned with the "acclimatization", "acclimation" or "habituation" process, according to whether the environment is more or less complex and whether alteration of the organism's response is a local or general one.

2. Methods of Acclimatization

Cold acclimatization methods have not been as well codified as those for heat acclimatization, in part because they are often painful for the subjects. The methods cover general acclimatization, and local acclimatization or specific habituation.

2.1 General Acclimatization Methods

These are aimed at improving the resistance of the whole body to thermal stress, and can be divided into three groups:

2.1.1 Natural methods

These methods are intended mainly for civil or military groups working in a cold climate. They consist essentially of exposing a group of subjects for several weeks or months to a cold climate in fairly primitive conditions, and with more or less controlled physical activity. Tests performed before and after exposure show any physiological changes produced as a result of the cold experienced. However, physical exercise, training or a change of food can, in certain cases, be held as responsible for any changes observed as the cold exposure per se. Experiments carried out include those by (1) Leblanc[195] in 1956, on clothed men engaged in outdoor activity in the Arctic for up to 12 hours a day, 6 days a week, for 6 weeks; the control tests consisted of an exposure of one hour at 10°C; (2) Scholander[256] in 1958, on 8 subjects who camped for 6 weeks in the Norwegian mountains in a temperature 3 to 5°C, the control tests consisting of an 8 hour night on these subjects, naked, at 20°C; (3) Davis[87] in 1961 on naked subjects exposed for one hour a day to 14°C once a month from October to February and from February to September; (4) Budd[54] in 1962, on soldiers who were spending a year in the Antarctic, the control tests covering a stay of 95 minutes at 10°C.

Mention should also be made of the numerous training exercises in the Far North carried out by the Canadian Army, which served to verify local and general acclimatization (Livingstone[208], Buguet et al.[56]).

2.1.2 Artificial acclimatization in air

These methods comprise a stay in a climatic cold chamber of several hours a day, every day, over a period of several weeks. The large number of experiments performed include for example those by Horvath et al.[152] in 1947, who kept their subjects for one week in a climatic chamber at −29°C; Iampietro et al.[158] in 1957, who exposed subjects wearing shorts during the day and sleeping under a woollen blanket at night for a period of a fortnight to an environment of 15.5°C, with a 0.2 m/s wind; Davis[88] in 1961, who exposed 10 subjects wearing shorts for 8 hours a day, over a period of 31 days, to an environment of 11.8°C; Keatinge[176] in 1961, whose subjects were placed for 7½ hours a day for 19 days in an environment of 6°C; Joy[169] in 1963, who kept his clothed subjects for 8 hours a day for 5 weeks in a 5°C environment with a wind of 0.3 m/s; and Newman[230] in 1968, whose subjects, wearing shorts and shoes, were exposed to an environment of 5°C for 4 hours a day, 5 times a week, for 6 weeks. These experiments included tests before and after the period of acclimatization and, sometimes, during this period.

2.1.3 Artificial acclimatization in water

Since the thermal stresses in water are more severe than in air, the idea was conceived of acclimatizing men in water in order to obtain quicker results. During his polar expeditions, the idea for this method of acclimatization had already occurred to Charcot, who asked his companions to undergo cold training by taking daily baths in very cold water. Experiments in this field have not, however, been on a large scale. We would mention as examples those by Lapp and Gee[193] in 1967, who subjected 8 students to a one hour per day immersion for 8 weeks, during which the bath temperature was lowered gradually from 32 to 21°C, or those by Skreslett et al.[262] in 1968, who carried out observations on 3 subjects diving every day for a month into cold water (0 to 3°C). The tests consisted of immersion for one hour at 0°C, wearing a neoprene suit. Previously, in 1959, Keatinge[172] had studied the effect on metabolism, ventilation, heart rate and blood pressure of repeated short periods of immersion in water at 15°C. Finally, the present author has recently adopted the use of this acclimatization method by baths in water at 15°C. The exposure periods were either for one hour a day for 14 days, or a gradual extension of the immersion period, particularly for the very thin subjects, from 20 to 50 minutes, 5 days a week for 2 weeks (Boutelier et al.[49]). The control tests before and after this acclimatization period consisted of

a two hour immersion in agitated water at 24°C (v = 0.25 m/s), preceded by a 90 minute resting period in thermo-neutral water.

2.2 Local Acclimatization or Specific Habituation Methods

Several surveys of populations living in cold climates have shown that they have a much better tolerance to cold in the extremities, particularly in the hands, than non-habituated people. Macworth[214] showed, in addition, that this improvement occurred in subjects who worked outdoors in a very cold climate (Fort Churchill, Manitoba). These subjects had in fact a much better tactile discrimination in the cold than subjects who normally worked indoors, when exposed to cold. Local habituation to cold was therefore possible. Since then, a large number of experiments have been performed (Glaser et al.[117], Keatinge[176], and Leblanc[196]), either to compare the responses of cold adapted populations with those of non-adapted people, or to assess a more general acclimatization, or again, to carry out habituation experiments. The tests used have been derived from the Yoshimura test and consist of daily repeated immersion of the fingers or hands in icy water.

All the methods described above show that cold acclimatization in man is generally a long and difficult process in people engaged in professional activities.

3. Results

These relate to metabolism, circulatory reactions and changes in the central and skin temperatures. Their variations are generally compared with those of populations who have become naturally adapted to cold climates, such as the Alacalufs, the Australian aborigenes, the Amas, etc. Hammel[133] has attempted to classify the various types of adaptation observed, and differentiates a metabolic type, which has a higher basal metabolism, an insulative type with a lower peripheral tissue conductance and a hypothermic type, which undergoes a more pronounced lowering of the deep-body temperature. He points out, however, that, generally speaking, the types observed are not pure types and that quite frequently there is a mixture of several types. Hensel[149] recently simplified this classification by considering only two types: first a hypothermic type, characterised by a low metabolic response and more pronounced cooling of the body. Acclimatization of this type is economic, since it reduces the temperature difference between the body and the environment, and therefore the heat losses. It is most frequently observed in populations living in a variable environment with cold nights and temperate or hot days, such as the Australian aborigenes. Hensel's second type has an increased basal metabolic response, which is more costly than the previous type from the energy point of view, but helps to maintain body temperatures and, in particular, those of the skin, at a more comfortable level. This type of adaptation is found in populations which wear few clothes and live in a continually cold climate, for example the Alacalufs.

The results of acclimatization experiments can be classified into one or other of the above categories.

Natural and artificial acclimatization experiments in air have provided somewhat contradictory results. Thus Carlson[68,69], Leblanc[195], Davis[88] and Budd[54,55] observed, during the control tests after the acclimatization period, a considerable reduction in shivering resulting in reduced metabolic response both during the period of initial exposure to cold and while the test was in progress. This reduction in metabolism varies from 20 to 25% according to the author concerned. In addition, shivering occurs at a lower skin temperature, and this reaction means a greater drop in the rectal temperature. The skin temperature, on the other hand, does not seem to change significantly. Other authors, such as Glickman[118], Scholander[256], Keatinge[176], and Joy[169] noted, on the other hand, that the metabolic response increased after repeated exposures to cold, either in a cold climatic chamber, or naturally. For Scholander, for example, there was a 20% increase, and after acclimatization, his subjects were capable of sleeping in colder environments, keeping their central and skin temperature at a higher level, particularly in the extremities, at the cost of an increase in their metabolic response; this type of acclimatization is of the metabolic type. The results obtained by Newman[230,231] on Puerto Ricans and Negro Americans are almost similar. He observed a distinct reduction in shivering, but a metabolic rate and a rectal temperature which remained constant throughout the post-acclimatization period (1 hour at 5°C). This seems to indicate the development of non-shivering thermogenesis, unless muscular tension increased without any discernible shivering. The mean skin temperature was also distinctly higher (1°C) after acclimatization.

An increased skin temperature in the extremities (particularly the hands) was noted by several authors in populations working with bare hands in a cold environment; e.g. the Gaspé Fishermen studied by Leblanc[196,198]. Similarly, Carlson et al.[69] had observed on warmly clad subjects, who exposed one hand to cold (−5 to −17°C) for 45 minutes a day, that hand temperature increased gradually. Opposite results were obtained by Livingstone[208] on subjects who had spent two weeks in the Arctic carrying out marches and lying in a tent at temperatures of between −10 and −40°C. This author has studied the effect of cold-induced vasodilation on a finger before and after the subject's stay in the Arctic. He noted that the mean temperature of the finger in icy water was lower after the period in the Arctic than before. The same applied to the temperature at which cold-induced vasodilation (CIVD) occurred. The author feels that short periods of exposure to cold induce general acclimatization but not local habituation.

Thus, in the acclimatization experiments in air, it seems that two types of acclimatization were observed: the insulative type and the metabolic type with the extremities being warmer, reflecting a peripheral circulatory adjustment. Leblanc[199] has suggested that these differences are due to the fact that in some of the experiments, exposure to cold was continuous, inducing acclimatization of the insulative and hypothermic type, whereas in others, exposure to cold was

intermittent, inducing a metabolic type acclimatization. It is however possible that this assumption cannot be verified, as shown by the results of the acclimatization experiments in water.

Results of acclimatization experiments in water:

Skreslett and Aarefjord[262] investigated the reactions of three subjects wearing neoprene suits, diving almost daily, for 45 days, into very cold water. Control tests in water at 1 to 2°C were carried out every two weeks; the first test was performed on the first day. The results showed that, before acclimatization, the increase in the metabolic reaction to immersion was very marked, and compensated for the heat loss, thus preventing the rectal temperature from falling. During the intermediate period (2nd and 3rd test), the metabolism increased only slightly and remained distinctly lower than that observed during the first test; the rectal temperature decreased more sharply. The authors consider that this stage corresponds to habituation of the central nervous system. Finally, during the last test, they noted that the central temperature was maintained, in spite of the very slight increase in metabolism. This stage apparently corresponds to real acclimatization, of the insulative type. The skin temperature of the trunk, the scapular region and the thighs was lower after acclimatization, but that of the extremities was higher.

The results obtained by the present author on seven naked subjects immersed every day in water at 15°C for 13 days or for 2 weeks, at the rate of 5 immersion periods per week, are slightly different. In certain subjects the metabolism measured over the last 10 minutes of immersion decreased gradually (Fig.2.15), and on the 13th day, it was 25% less than that on the first day. In addition, the initial metabolic and ventilatory reaction to immersion disappeared and was replaced by about 10 minutes without shivering. This period varied with the subjects, probably as a function of their skin-fold thickness. During the control tests in water at 24°C (v = 0.25 m/s), a comparable reduction in metabolism was observed during the initial phase and throughout the test (Fig.2.16). The rectal temperature changes were fairly significant and a sharper fall was observed after acclimatization than before (Fig.2.17). This development indicated a hypothermic type acclimatization; however, not all the subjects responded in this manner. Some of them showed no reduction in metabolism, but rather an increase at the end of the acclimatization period. Their rectal temperatures dropped more slowly and they were able to tolerate immersion for twice as long in water at 15°C. Furthermore, the nausea and cramps which they suffered during the first few baths gradually disappeared. These subjects seemed to exhibit a metabolic type acclimatization.

These few results obtained during immersion show that, as in air, two types of acclimatization can be observed. Contrary to the view held by Leblanc[199], this does not appear to be due to different cold exposure procedures. It would seem, furthermore, that cold acclimatization in water is acquired more quickly than in air.

It should be noted in addition that all these methods enable the subject to recover sufficient manual dexterity to perform relatively difficult tasks.

Acclimatization would therefore seem to be a useful solution for improving man's tolerance in a cold environment in air and in water. But there is still the problem of deciding which method to adopt. In the military aviation field, the choice will depend on two factors: the availability of crews, and the missions to be flown by them. If the latter include flying over regions with a rigorous climate, artificial acclimatization in air seems to be preferable. On the other hand, if it is a question of protection against accidental immersion only, acclimatization by means of cold baths seems a very acceptable solution, since it can be carried out as part of their sea survival training.

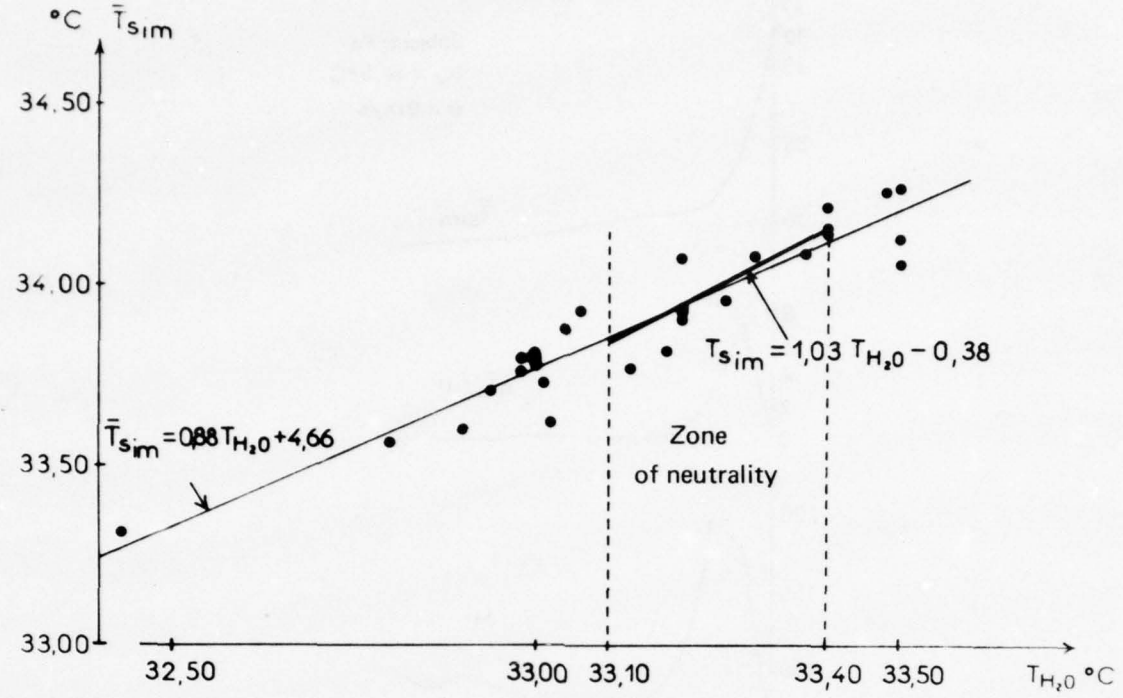

Fig.2.1 Relationship between mean skin temperature $\bar{T}_{sim}$ and water temperature. Thermal neutrality zone

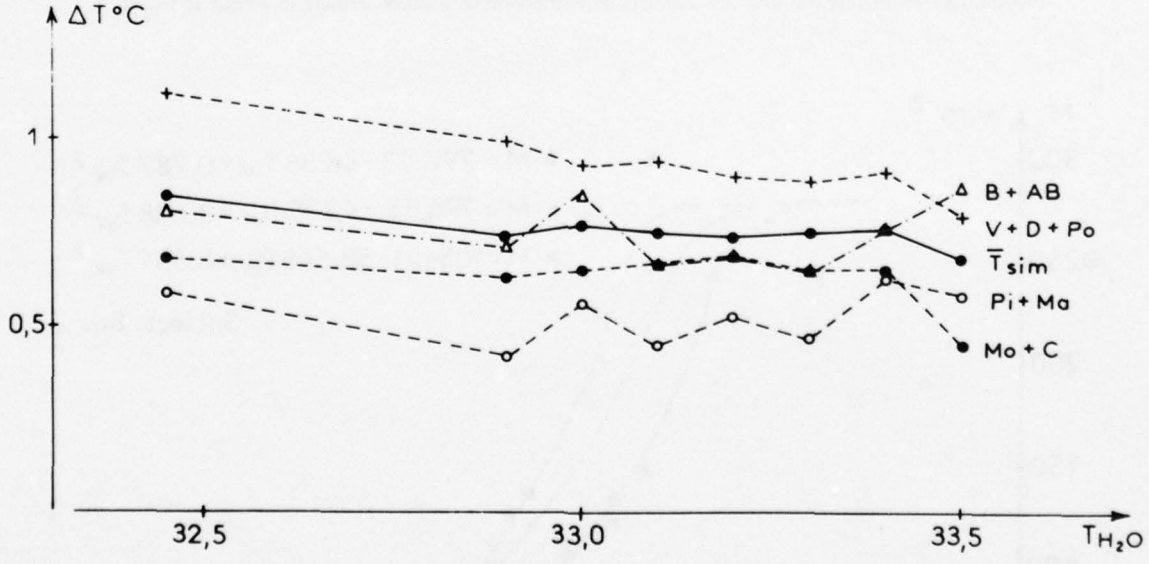

Fig.2.2 Development, as a function of the water temperature, of the temperature differences between the skin and the water according to the regions concerned. B: arm, AB: forearm, V: abdomen, D: back, Po: chest, Pi: foot, Ma: hand, Mo: calf, C: thigh, $\bar{T}_{sim}$: mean skin temperature immersed

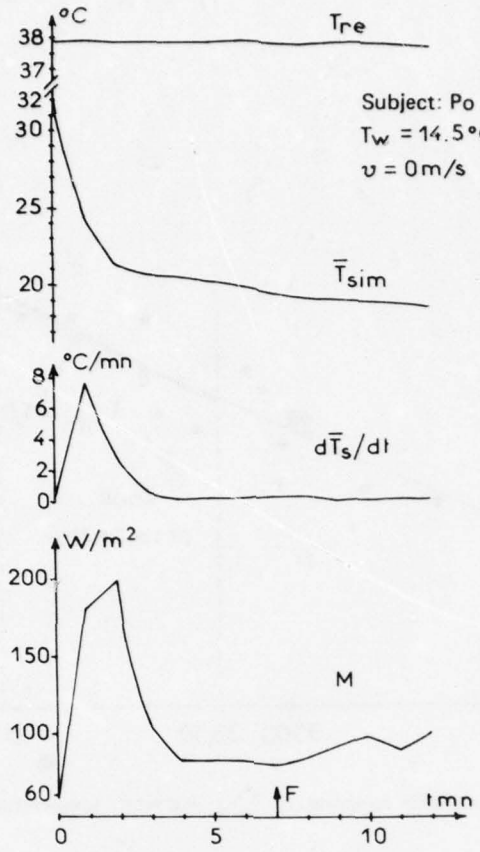

Fig.2.3 Development of the rectal temperature T_{re}, the mean skin temperature $\overline{T}_{sim}$, its rate of variation and the *metabolism M during the first few minutes* of immersion of a naked subject in a bath at 14.5°C

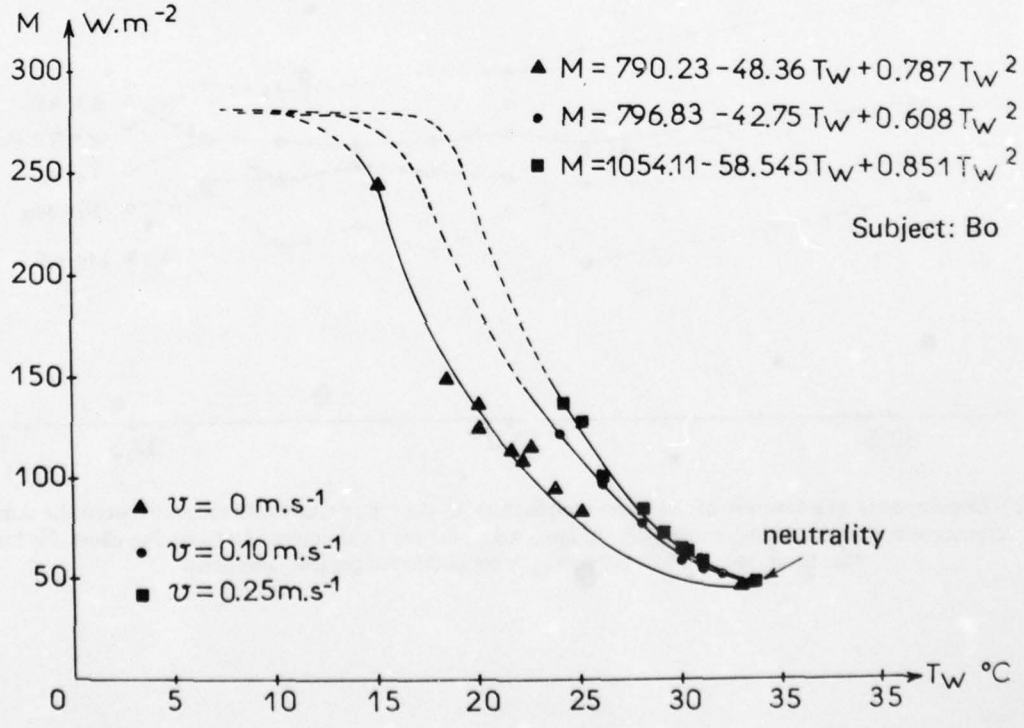

Fig.2.4 Maximum metabolic value or its value in steady state as a function of the water temperature for different water velocities (flow parallel to the major axis of the body)

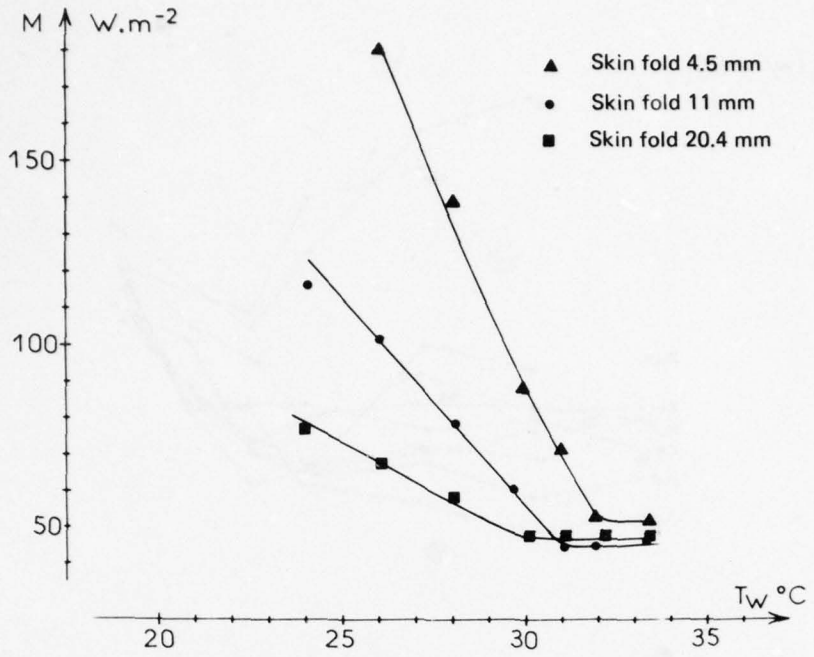

Fig.2.5 Metabolism in steady state as a function of the water temperature at a constant velocity (v = 0.10 m/s) for three subjects whose skin fold is different

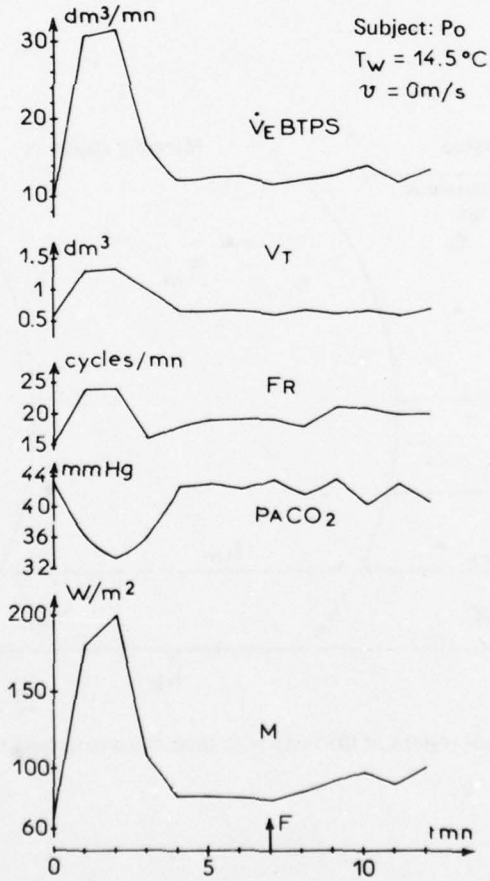

Fig.2.6 Development of the ventilatory parameters and the metabolism during the first few minutes of immersion in still water at 14.5°C. The arrow F denotes the recurrence of shivering which had ceased at the third minute

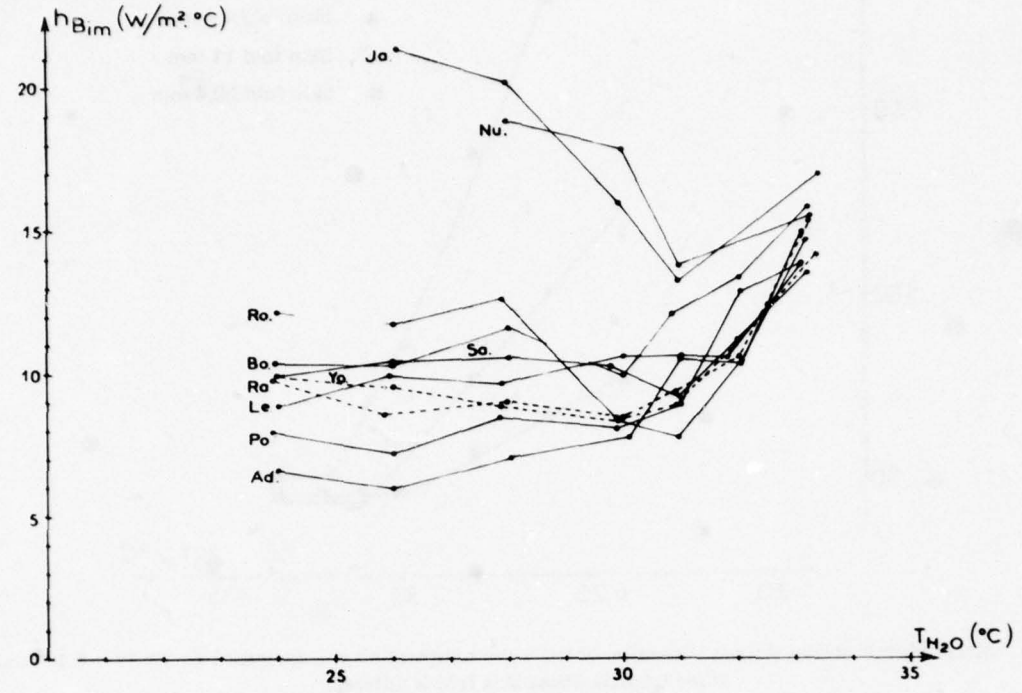

Fig.2.7 Variation in the thermal conductance of the body as a function of the water temperature. Note the high values for this conductance in cold water for two thin subjects Ja and Nu

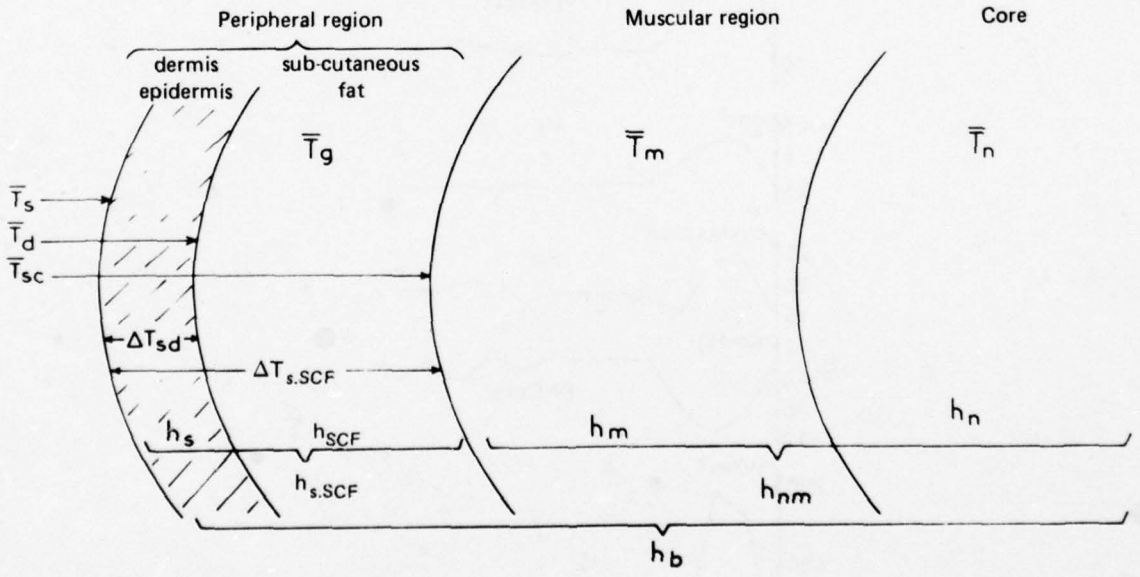

Fig.2.8 Diagram showing the various regions of the body with their mean temperatures and their conductance values

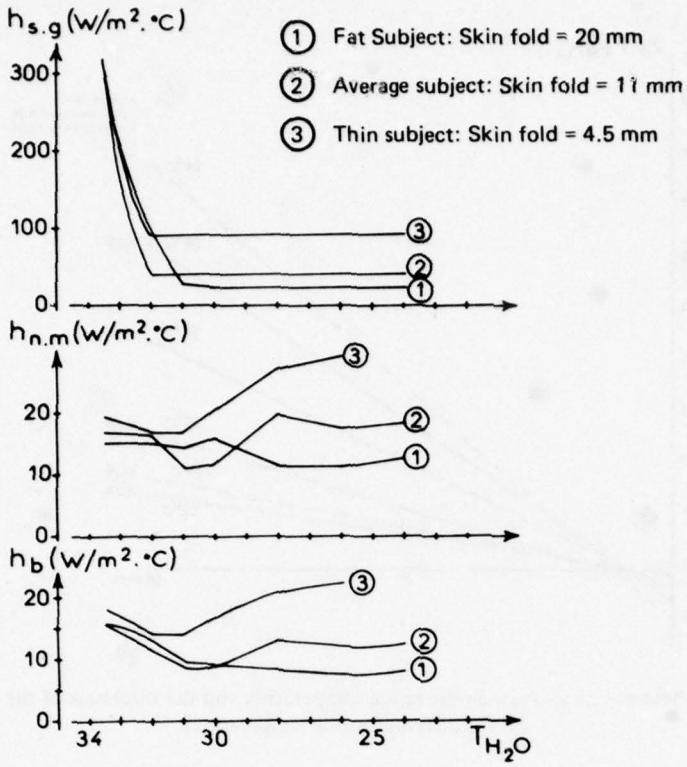

Fig.2.9 Development, in relation to the temperature of the bath, of the conductance values of the periphery $h_{s.SCF}$, the core/muscle region $h_{n.m}$ and the general conductance h_b of a fat subject (1), of an average subject (2) and of a thin subject (3)

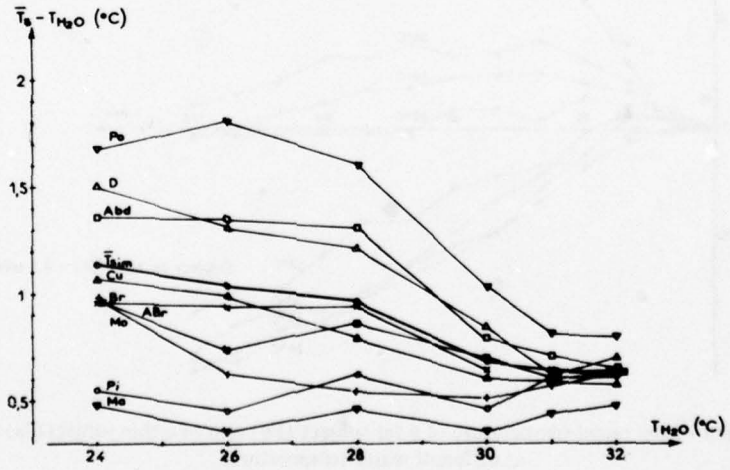

Fig.2.10 Development, as a function of the bath temperature, of the temperature difference between the skin in the various regions of the body and the water in steady state. Po: chest, D: back, Abd: abdomen, Cu: thigh, Br: arm, ABr: forearm, Mo: calf, Pi: foot, Ma: hand, $\overline{T}_{sim}$: mean temperature for immersed skin

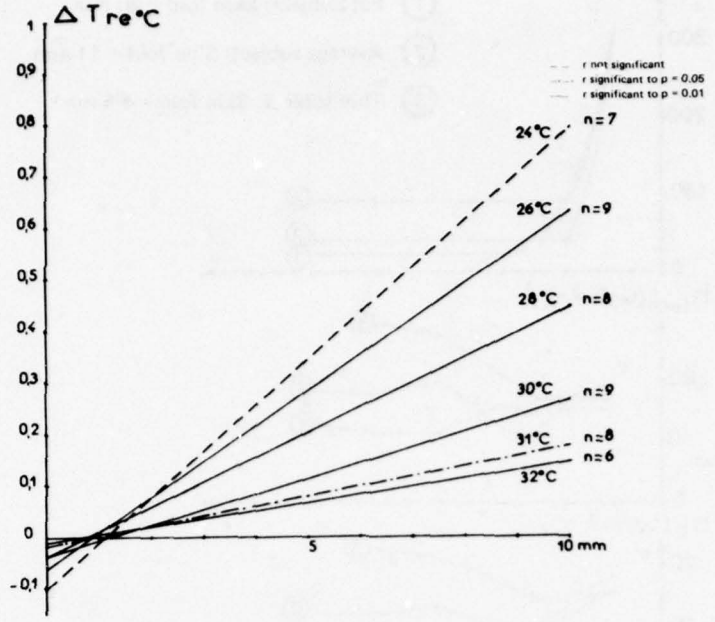

Fig.2.11 Relationships between an increase in the rectal temperature and the thickness of the sub-cutaneous layer of fat for different water temperatures

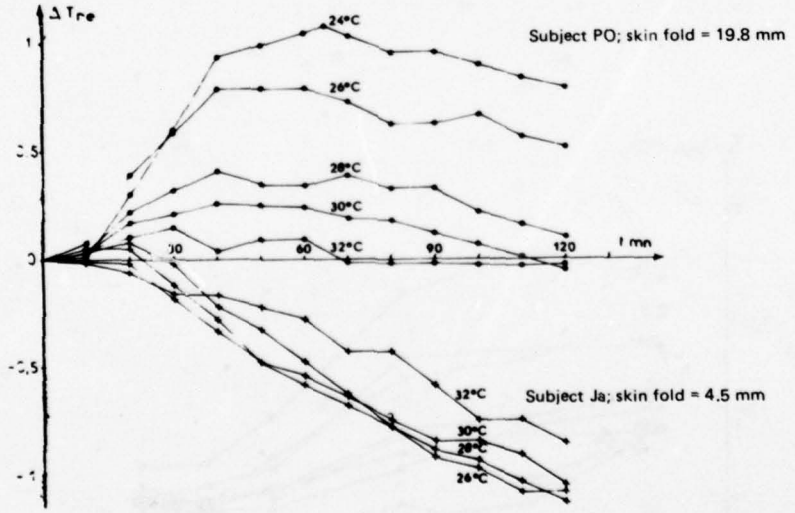

Fig.2.12 Development of the rectal temperature of a fat subject (Po) and of a thin subject (Ja) as a function of time at different water temperatures

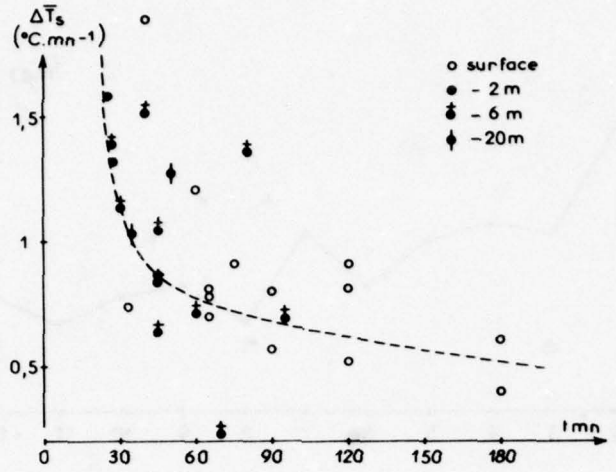

Fig.2.13 Relationship between the voluntary tolerance time and the rate of variation of the mean skin temperature during the first 5 minutes of immersion in various experimental conditions

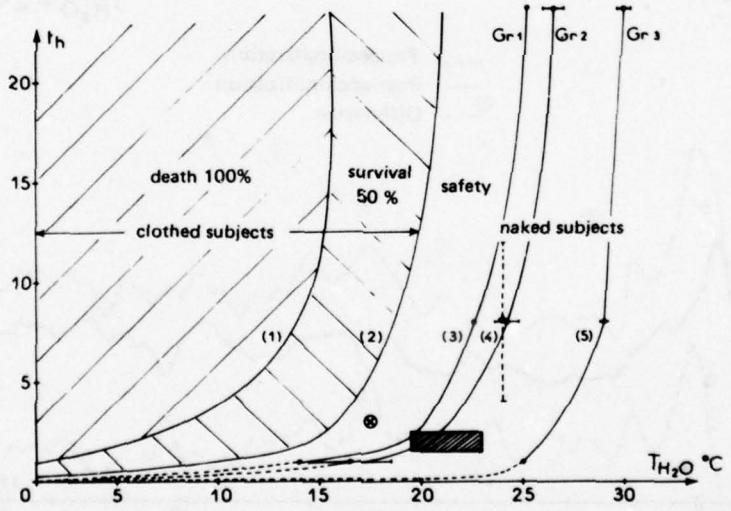

Fig.2.14 Molnar's survival nomograms (curves 1 and 2) and tolerance nomograms for naked subjects in moving water (curves 3, 4, 5). The nomogram 3 is for naked subjects with skin-fold 20 mm, the nomogram 4 is for subjects with skin-fold 13 mm and the nomogram 5 is for subjects with skin-fold 4.9 mm. The vertical dotted line shows the results obtained by Beckman and Reeves, and the hatched rectangle, the voluntary tolerance limit associated with reaching the minimum $\overline{T}_s$. The circle with a cross in the centre indicates the Lakonia shipwreck in which 133 victims died.

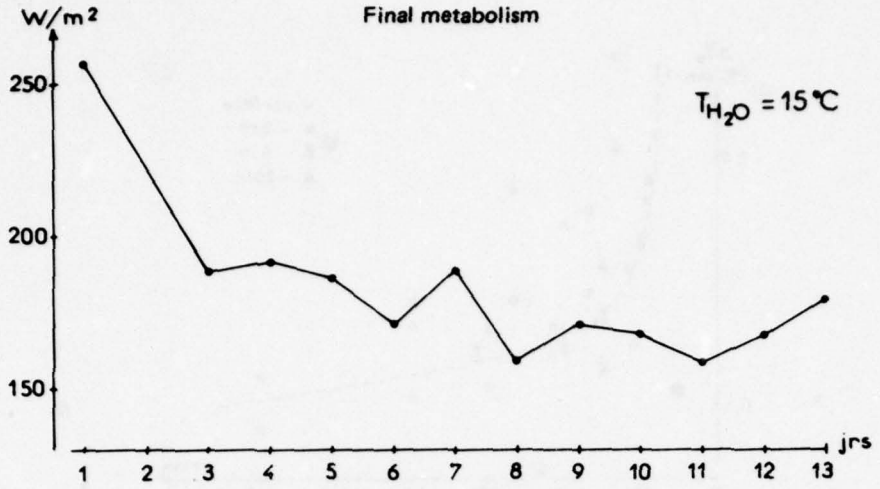

Fig.2.15 Variation, during acclimatization, of the metabolism of a subject after 1 hour in still water at 15°C (unpublished results)

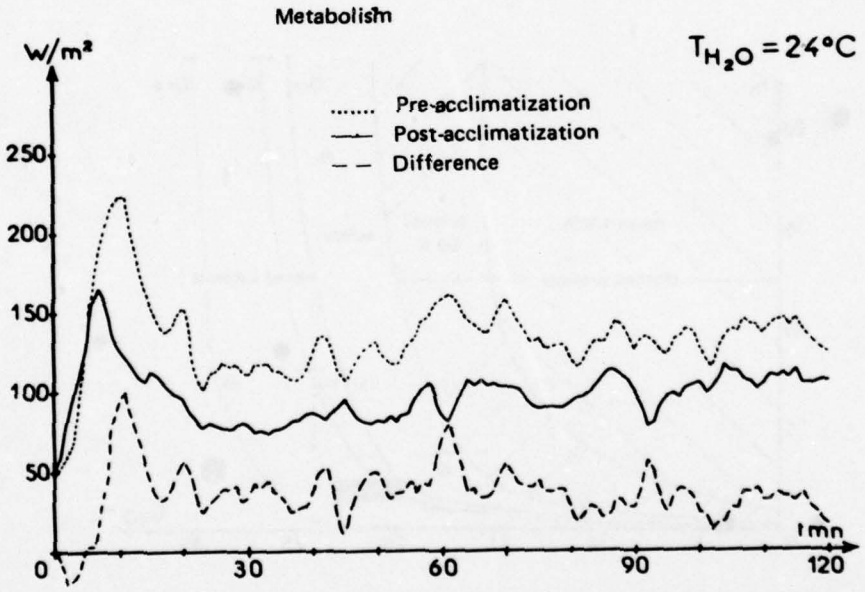

Fig.2.16 Development of the metabolism of a subject during pre and post acclimatization tests in water at 24°C (v = 0.25 m/s)

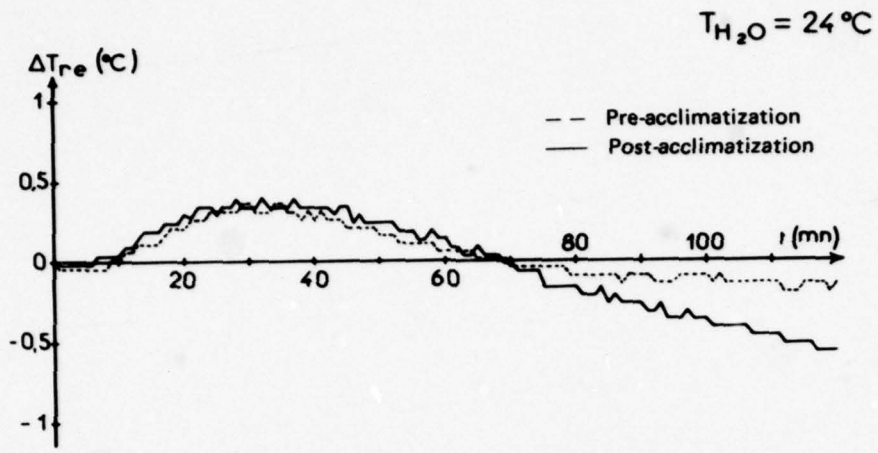

Fig.2.17 Development of the rectal temperature of a subject during pre and post acclimatization tests in water at 24°C (unpublished results)

Chapter 3

INJURIES CAUSED BY COLD AND THEIR TREATMENT

In the Northern hemisphere aircrews involved in an accident are generally exposed to cold ambient conditions whether in the water or in dinghies. In addition, in spite of the effectiveness of protective clothing, circumstances beyond their control may aggravate such exposure. For example, they may lose their dinghy or find themselves unable to get into it if the weather is rough; they may have lost some of their equipment, such as gloves or helmet, or their equipment may have become damaged when they ejected. Now, the defensive reactions of the organism are limited and the victims, particularly in water, may be found to have a number of disorders or lesions due to damp cold. The extent and the severity of such injuries will depend on the ambient conditions and on the period of exposure. Furthermore, and in spite of wearing a life jacket, such lesions may be complicated by an initial period of drowning if the sea is rough.

Since the Second World War a great number of studies on injuries associated with exposure to cold in the air and in the water have been made both from the pathophysiological point of view and from the clinical and therapeutic aspects. It is standard practice to classify injuries into two groups: local injuries which affect only certain parts of the body which are uncovered or insufficiently protected, such as the face, hands and feet, and injuries of a general type associated with a more or less profound hypothermia of the body. It is clear, however, that both these types of injuries can be observed simultaneously.

1. LOCAL INJURIES

These can be lesions, without freezing of the tissues, which may occur at temperatures usually higher than 0°C, or lesions accompanied by freezing. In increasing order of severity we have cramp, immersion foot and hand, and frostbite.

1.1 Cramp

Nukada[235] and Lind[209] have observed that sustained voluntary contraction could be maintained longer in water at 18°C than in water at 34 or 40°C. This is probably due to a slowing down, as a result of the cold, of the rate of relaxation of actomyosin. Maintaining this contraction may have a deleterious effect by causing the occurrence of cramp, particularly in the legs. This is observed either during immersion for long periods in moderately cold water, or at an early stage of immersion in cold water (of the order of 15°C). Thus, in the experiments conducted by Beckman and Reeves[26], 13 of the 24 subjects immersed in water at 23.9°C experienced severe and definite cramp after only 2 hours. In our own experiments (the results have not been published) some of our subjects, the thinner ones, when immersed up to the neck in the horizontal position in still water at 15°C, suffered severe cramp during the first 20 minutes of immersion. Fat subjects immersed at the same water temperature developed cramp much later, usually after one hour or longer. In these conditions, thin subjects are affected by much more violent shivering. It would therefore seem that the intensity of the muscular activity associated with shivering is a factor which predisposes towards cramp, especially since the muscles of the limbs, and of the legs in particular, are relatively hypoxic because of the extensive peripheral vasoconstriction. Other reasons have been put forward to explain this occurrence of cramp. Some authors think that there is a slowing down of the rate of destruction of acetylcholine at the motor end-plate, and of the rate of repolarisation of the latter (Li[207], Bigland[33]) under the direct action of the cold conditions. All these mechanisms probably contribute simultaneously to the production of cramp.

According to Keatinge[181], cramp is not one of the main causes of death in immersions following an accident. However, it is bound, in our view, because of the pain it causes, to considerably reduce the resistance capacity of the victim and hasten a fatal outcome.

1.2 Immersion Foot

This lesion is very comparable to that suffered by numerous soldiers during the winters of 1916 and 1917 during the First World War and known as "trench foot". The prior condition for the occurrence of such a lesion is prolonged exposure to damp cold. It may occur after some 12 hours in a damp environment at +10°C. In accidental immersions, such lesions have frequently been observed after a prolonged period in a dinghy (Critchley[85], MacCance[213]). Local compression, e.g. by shoes, is an aggravating factor. Ungley[289] has shown that this injury could arise in the absence of wetness, and stresses the fact that the cold was the main factor involved, leading to severe injuries to the nerves. He has also suggested that this type of lesion should be called post-cooling vasoneuropathy.

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1.2.1 Clinical Evidence

Immediately after rescue, the limbs affected are numb and have lost their power. If immersion has occurred in water which is close to 0°C, they are red while at less cold temperatures they are yellowish white or have black and blue spots, and are swollen and cold. The arterial pulses are absent. The limbs are insensitive and motor paralysis normally occurs. The blood flow in this region is reduced to a minimum and, indeed, is almost non-existent. This is the *ischaemic* stage.

Two to five hours after the patient has been placed in a warm atmosphere, the second stage appears. This is characterised by excessive *hyperaemia.* The blood flow in the affected limbs increases considerably and the pulses are strong; the legs become warm, red and painful. There is no sweating. The swelling increases and blisters form in the majority of cases. At this stage there is a partial return of sensitivity and motility, although anaesthesia still persists in the injured areas, as well as muscular weakness. This stage may last for weeks or months.

In the final stage, the blood flow to the legs returns to normal, or almost to normal. The affected members then become very sensitive to temperature. Cold causes intense vasoconstriction, and heat a high degree of vasodilation with excessive sweating. Some decrease in sensitivity is often observed but the most serious disorder at this stage is the loss of muscular power with contracture of the affected muscles.

In a few very severe cases the blood supply does not return in certain areas, probably because of thrombosis of the arteries which irrigate these areas. This produces gangrene. Such cases are not, however, frequent and although Ungley et al.[289] have confirmed the existence of these thromboses, an additional trauma must often also be present.

1.2.2 Pathophysiology

The main cause of immersion foot is an affection of the nerves and the muscles due to cold. Thus the sensori-motor paralysis found in the first stage is probably due to a direct effect of the cold on the nerves and the muscles. This direct action of the cold has been demonstrated in several experiments. Bicford[31], quoted by Keatinge, cooled the skin above the cubital nerve and showed that this first produced a sensation of cold in the finger controlled by this nerve, and then a loss of motility, followed by a loss of all sensation. The temperature at which these disorders occur is always above the freezing point of the tissues. Nervous conduction ceases, in fact, at about 9°C. According to Li[207], it seems that the cold affects the neuromuscular junction. Biopsies performed on such injuries have shown marked degenerescence of the nerves which could account not only for the persistent loss of sensitivity, but also the excessive vascular reactions to cold conditions, as well as the high degree of sweating in warm conditions. These reactions are comparable to sympathectomy of the limb.

However, paralysis and contracture of the muscles cannot be attributed to degenerescence of the motor nerves. There is therefore a direct cold effect on muscles which is aggravated by ischaemia due to intense vasoconstriction. In early cases, some muscular degeneration is observed, while in cases at an advanced stage the muscles have become atrophied (Burton and Edholm[58]). Friedman (quoted by Burton) has described the vascular reactions during the second stage of lesions. The blood vessels are very much congested and dilated, and their walls are thin and may rupture. The red blood corpuscles escape from the vessels. Sometimes a collection of hyalinised red corpuscles is found which could form a thrombus. Endarteritis obliterans is also observed at a later stage. Finally, intense vaso-constriction may cause necrosis of the fatty tissues.

The time factor is extremely important in the genesis of immersion foot. Indeed, during the first few hours of immersion, vasodilation in a cold environment provides a certain degree of protection. This phenomenon, however, grows gradually less as the body gets colder. Thus, the blood supply to the hand may not increase if the subject becomes cold. Schwiegk (quoted by Burton) has stressed the importance of the general cooling of the body on the occurrence of immersion foot. It should finally be noted that the hands may be affected with the same lesions as the feet.

1.2.3 Treatment

This is primarily of a preventive nature. The severity of these lesions indicates the need for effective protection of the extremities. For this purpose, neoprene boots attached to some anti-immersion suits seem to offer a very satisfactory solution.

Curative treatment: According to Keatinge, there is no evidence to suggest that one kind of treatment is better than another, and he therefore recommends following the instructions given by Ungley et al.[289]. These authors advise active rewarming of the patient's trunk, while allowing the limbs to get warm again spontaneously. This minimises the risk of lesions through ischaemia due to premature rewarming of the limbs, which might increase the oxygen requirements of the tissues before restoration of an adequate supply of blood to the legs. In no case must a radiant source of heat be used on the injured area, for fear of the risk of burning due to the local loss of sensitivity. To avoid any pain when the blood flow returns to the limbs, an analgesic may be given. The extent of the oedema will be reduced by keeping the legs in a raised position. If there is necrosis, the infection can be combated by antibiotics.

1.3 Frostbite

Cases of frostbite are rare during accidental immersions in our part of the world, but overflight of arctic regions may expose aircrews to this risk, particularly if they are in a dinghy. Frostbite may then be observed on the uncovered parts of the body, especially the face, but the hands may also be affected if gloves have been lost or damaged.

1.3.1 Pathophysiology

Very little is still known about frostbite pathophysiology, although a number of established facts do exist. We would note first of all with Burton[58] that it is difficult to define a temperature which is lethal for the cells, since several cellular forms can survive at very low temperatures. The nerves and the striated muscles are the most sensitive structures to cold and can withstand only short periods of exposure to temperatures of from 0 to 5°C. The skin is more resistant and can quite safely withstand a temperature of −1.9°C for 7 minutes, although its real freezing point is about −0.5°C (Keatinge et al.[173]). However, the skin can become cold at much lower temperatures without freezing. Thus, Molnar et al.[223] exposed the second phalanx of a finger to an air temperature of −10 to −15°C, with a 10 m/s wind, and showed that at the same skin temperature (approximately −7°C) either frostbite or vasodilation from the cold could be observed. In three of their experiments neither frostbite nor vasodilation was noted. This phenomenon of "supercooling" without causing frostbite still remains unexplained. It is possibly due, as suggested by Devries et al.[91] following their work on fish in the Antarctic, to a glycoprotein which lowers the freezing point. Furthermore, the skin temperature at which freezing occurs is extremely variable. Freezing occurs in 62% of cases at between −5 and −9.3°C, which confirms the work done some time ago by Lewis et al.[205]. Because of this particular resistance of the skin to freezing, lesions in deeper structures, such as the nerves and the muscles and the vascular endothelium, can be found but without any damage to the overlying skin. It should be noted finally that the aponeuroses, connective tissue, tendons, bones and red blood corpuscles are very resistant to cold.

Observation of the death of cells at certain temperatures does not explain the reasons for their death. It was formerly assumed (Lewis et al.[205]) that cell destruction was caused mainly by the mechanical action of the ice crystals formed during the freezing process. These may occur in one of two forms, depending on the speed of freezing: either as small crystals which are distributed at numerous central points in the extracellular spaces, if freezing is rapid, or large crystals in the case of a slow rate of freezing. Large crystals may also form if the injured parts are rewarmed slowly after rapid freezing. It is these large crystals which may in fact cause mechanical lesions of the cell walls. It will therefore be better to rewarm the lesions rapidly, in order to avoid this risk. This mechanical action is not generally, in fact, the main cause of cell destruction and some cells, such as the red blood corpuscles, can withstand freezing at very low temperatures (−79°C) and completely recover their functions after rewarming. Nowadays, most authors (Burton[58], Busby[59], Lorentzen[212]) attribute an essential pathogenic role to the vascular phenomena observed during cooling and rewarming. The intense vasoconstriction of the arterioles caused by the cold leads to hypoxia of the tissues in the relevant area. This reaction is reduced by the occurrence of sudden periods of vasodilation, but in the long run, this phenomenon tends to disappear and all the more rapidly if the victim is tired, depressed by anxiety, or if general hypothermia has set in. Severe vasoconstriction with complete cessation of the circulation of the blood takes place and it seems as though "the exposed area were being sacrificed for the protection of the centres". Following considerable animal research, Kulka[189] showed that arterial and arteriolar vasoconstriction was followed by excessive dilation of the veins and capillaries causing exudation of plasma and stasis of the erythrocytes. The arterio-venous shunts open, and this is followed by segmentary vascular necrosis and extensive thrombosis. In addition, Mundth, quoted by Burton, has shown that the first pathological evidence in response to frostbite was deterioration of the intravascular stability of the cells with the formation of clusters of platelets and erythrocytes.

In addition to these vascular phenomena, which are probably of primary importance, cold has a direct effect on the biochemistry of the tissues. Indeed, the dissociation curve for oxyhaemoglobin moves to the left at low temperatures, reducing the supply of oxygen and modifying the metabolism both qualitatively and quantitatively. These changes are generally small when freezing is rapid. However, prolonged exposure of certain tissues to low temperatures, but which still are higher than the freezing point, might possibly cause an irreversible imbalance in the functioning of the metabolic enzyme systems responsible for the lesions observed. This concept is not unanimously accepted and requires confirmation.

Another mechanism may be invoked to explain the death of tissues without dislocation of the cells: intracellular dehydration (Meryman[218]). This is due to the formation of ice crystals in the extracellular spaces, involving an increase in the osmotic pressure of the extracellular fluid which remains in the liquid state, and the passage of the intracellular water to the outside. This results in an increased intracellular concentration of electrolytes and other constituents of the cell. Such changes in concentration may cause lesions by chemical means such as the formation of chemical bonds which do not exist naturally, and modifications of the permeability of the membrane resulting in an escape of potassium and certain intracellular enzymes, and penetration of sodium.

1.3.2 Clinical Evidence

Clinical evidence of the results of exposure to cold is spread over a very long period as compared with the actual period of exposure. In the usual approach, four periods have to be taken into consideration: the period of exposure to cold, rewarming, the much longer period between rewarming and complete recovery, and finally, any sequelae.

During exposure to cold, lesions generally occur insidiously and reliance should not be placed on premonitory symptoms as a basis for thinking that precautions should be taken which may, moreover, be difficult to apply in practice in accidental immersions. At most, paraesthesia is noted initially, with a sensation of pricking, and tingling, followed by a lack of sensitivity in the affected areas (face, fingers). The fingers feel as though they were "dead". After a short erythematous phase, the skin becomes pallid and waxy. If there is freezing, the tissues become hard and brittle. There is no sensation or pain at the actual site of the lesion, which is colourless and generally well defined. At this stage, a clinical examination will hardly reveal enough to make a prognosis except perhaps an indirect and by no means precise estimate based on the duration and the intensity of the attack.

The period during which the patient's body is being rewarmed is generally marked by a return of the blood circulation accompanied by local rewarming, filtering of plasma through the walls of the blood vessels, together with the occurrence of oedema and particularly of pain which is sometimes extremely intense. Here again it is difficult to make a prognosis. However, consideration of the skin temperature is of definite value, since it will indicate the areas to which warmth is not returning and which will die because the circulation is inadequate or has been stopped irreversibly.

It is only during the third period that it is possible to assess the severity of the frostbite. This phase is of very variable duration and depends on the extent and depth of the lesions. The following four degrees of increasing severity are usually distinguished (Busby[59]):

In the first degree the injured area exhibits erythema and oedema. During the rewarming period the skin becomes marbled and bluish, then red, hot and dry. The hyperaemia whitens only slightly under pressure and the capillaries fill slowly or not at all. The sensation of burning or pricking, sometimes intense, which is present in the first instance, gives way to more severe pain. In the most benign cases, the pain persists for a few hours, after which the symptoms disappear gradually without leaving any sequelae. In more severe cases, the above symptoms are more pronounced and may last several days. After that, aching of the affected area, with paraesthesia, cyanosis, hyperhidrosis and coldness, may begin 2 to 3 weeks after the frostbite occurred, and last for several months. As a general rule, oedema is initiated within 3 hours after the rewarming period but clears up after 10 days. Peeling of the surface layers of the skin begins 5 to 10 days after the frostbite and goes on for a few weeks.

The second degree is characterised by the formation of blisters. The same symptoms as in the first degree are observed, but oedema is less extensive and disappears in 3 to 5 days. The tactile sense is quite often lost, and there is throbbing pain for from 3 to 20 days. The blisters form between 6 and 12 hours after rewarming. These vesicles are most frequently found on the back of the fingers, hand or big toe, and on the heel. They remain localised on the epidermis which may be regenerated from the deeper layers. They may contain blood or a transudate. They gradually dry up and black scabs form in 10 to 24 days after the rewarming period. When these scabs are gone they leave the skin fragile and thin and slightly keratinised.

The third degree is characterised by necrosis of the skin and the sub-cutaneous tissue. After rewarming, the skin does not retain its heat and cools, with persistent loss of sensibility to touch. Vesicles may appear on the periphery of the damaged tissues. Oedema is severe, but disappears in 6 days. Throbbing pain, with a sensation of burning, occurs during the second week and continues for about 5 weeks. The skin in the damaged area becomes dry and black, and a scab forms. This disappears slowly, leaving behind slightly vascularised granular tissue. The regeneration of epithelial tissues in this area is a slow process and complete healing will take 2 to 3 months. Hyperhidrosis and cyanosis appear between the 4th and 10th week after the frostbite and may persist for several months. At this stage, infection is a complication which is always to be feared.

The characteristic feature of a fourth degree injury is the destruction of the whole of the affected area, including the bones. As soon as it is rewarmed, the skin becomes marbled and cyanotic. Oedema appears one hour later and reaches its maximum in 6 to 12 hours. The extent of the lesion cannot be fully appreciated until the oedema has disappeared. The formation of a scab or gangrene cannot be seen until 2 to 3 weeks after the frostbite occurred. In the severest cases, the affected tissues rapidly develop dry gangrene and mummification. The demarcation between the living and the dead tissues will not be apparent until about a month later and the bones will not be involved until two months after the frostbite or even later. A rare but serious complication from extensive frostbite accompanied by a state of shock is disseminated intravascular coagulation. This consists of petechial and purpuric lesions, haemorrhagic bubbles, extensive gangrene and distal cyanosis (Basset et al.[22]). The blood mixture can generally help to make a diagnosis (thrombocytopenia, a drop in fibrinogen and the presence of fibrin degradation products).

A final balance sheet of any sequelae can be established only after complete recovery. The following can then be observed:

- *trophic disorders* with hyperhidrosis, cyanosis, chronic oedema and sometimes recurrent ulcers. In the serious forms, there is painful decalcification with, as seen on X-rays, images of loss of epiphysial substance;
- *nervous disorders*: neuralgia, anaesthesia or on the other hand hyperaesthesia to cold, the sensation of coldness being felt as painful;
- *vascular disorders*, sometimes constituting an actual arterial syndrome, as revealed by arteriography.

1.3.3 Treatment

As the pathophysiology of this condition has not been fully established, treatment is primarily based on the signs of cessation of the circulation in the damaged area and on the evidence of the vascular lesions, together with their consequences. It covers the following stages according to the development of the lesions.

The body must be rewarmed as early as possible after loosening of the clothing or equipment likely to cause constriction. In the case of accidental immersion, however, this cannot be done until after the patient has been treated for hypothermia. Numerous animal experiments (Finneran et al.[98], Fuhrman et al.[101]) and the encouraging results obtained from experiments on humans have shown that rapid rewarming reduced the extent of the necrosis by reducing the area in which there was total stoppage of the blood supply. In addition, the increased permeability of the blood vessels following frostbite is not so great with rapid rewarming as with slow rewarming (Aturson[19]). The best method of achieving this rapid rewarming is to plunge the injured part in a bath with a water temperature of between 37 and 42°C. In no case must the injured area be massaged or rubbed, and care must be taken to avoid rough handling, which can only aggravate the injury.

The effect of drugs on vascular phenomena: intra-arterial injection of vasodilatators, anti-aggregates, anti-coagulants, is subject to much discussion. It would seem however that their effectiveness depends on their being administered at an early stage, even before rewarming, and then being repeated over a period of several days. For example, to be at all effective, heparin has to be injected within 16 hours of exposure to cold. It is extremely important, on the other hand, to avoid any further infection of the wound and to ensure the gradual cleansing of the dead tissues, hence the application of appropriate antibiotics, anti-tetanus serotherapy and antiseptic copper sulphate baths, colour washes etc. (Basset et al.[22]). Finally, pain-killers can be given to lessen the pain.

At a later date, surgical excision of the gangrenous tissues can be considered. This must not be done too early, since surprising recoveries are possible if one only knows how to wait. Thus, as Keatinge[181] so rightly summarises it: "When the lesions have been rewarmed, when analgesics have been given and when the tissues have been protected from infection, no additional treatment is required, except that of protecting the patient from the more interfering members of the medical profession."

2. INJURIES OF A GENERAL NATURE: HYPOTHERMIA

The local injuries described above may be complicated by much more serious disorders caused by the excessive drop in the central body temperature. In accidental immersions, hypothermia represents a major risk for pilots and passengers at relatively high water temperatures, in spite of the protective equipment developed since the Second World War. The literature is very rich in examples in this connection, whether as a result of shipwrecks (Keatinge[181]), aircraft accidents or helicopter accidents. One of the most striking examples is possibly the one quoted by Kreider[183] when describing an accident which occurred in 1956 to a USAAF aircraft in the Atlantic near Cape Cod. Of the 19 crew members, 12 survived the impact of the aircraft on the water, but only 3 were recovered alive after being in the water for 11½ hours at a temperature of 11°C. The other victims had died of cold in spite of their anti-immersion suits and life jackets.

There is a great wealth of data on hypothermia from many different sources:

Animal experiments (Lefevre[200, 201, 202, 203], Adolph[3], Giaja et al.[111, 112, 113], Malmejac[215]): These have helped to indicate the chronology of the disturbances in the physiological systems which occur during the development of hypothermia, as well as the limit temperatures at which certain organs – heart, brain, etc. – cease to function. These data, which are essential for an understanding of the phenomena involved, must, however, be applied with care when extrapolating to humans, particularly with reference to the limit temperatures.

Human experiments (Behnke et al.[28], Keatinge[181]): These are necessarily limited to a very moderate degree of hypothermia, with the exception of the criminal experiments conducted at Dachau and reported by Alexander[4].

Artificial hypothermia practised on humans even before the Second World War for therapeutic purposes, and subsequently, for surgical reasons: This has provided useful information, although anaesthesia modifies the physiological responses, notably by inhibiting shivering. In addition, control of respiration avoids disturbing the blood pH value, so that the reactions are different from those observed in cases of accidental hypothermia.

Accidental hypothermia which occurs on the ground, particularly in winter, and observed in hospital conditions (Fruehan[100], Justin-Besançon et al.[170], Hudson et al.[156], Nicolas et al.[232], Péquignot et al.[237]): The victims on such occasions are, however, generally undernourished, and old (88% of the subjects are over 50), often chronic drunkards or sometimes drug addicts. The clinical picture is therefore generally complex and it is frequently very difficult to distinguish what is due to hypothermia from what causes it. Furthermore, complications frequently occur in organisms which are often deficient. Any transposition of such data to hypothermia due to an accidental immersion which occurs in relatively young and healthy subjects must therefore be subject to great care. A number of recent publications (Keatinge[181], Golden[119, 120, 121], Tansey[275]), do, however, make it possible to obtain the latest information on this question.

2.1 Definition and Classification of Cases of Accidental Hypothermia

Accidental hypothermia is an unintentional lowering of the central body temperature below 35°C (Golden[120]). According to the degree of coldness, four degrees of hypothermia can be distinguished (Nicolas et al.[232]): *slight*, when the rectal temperature is between 35 and 34°C; *moderate*, if it is between 34 and 32°C; *serious*, if it is between 32 and 25°C; and *profound* or *major* if it is lower than 25°C. The lowest temperatures recorded in the literature are 16°C (Laufman[194]) and 17°C (Anderson et al.[5]).

According to the period of exposure, accidental hypothermia can therefore be classified under three types (Golden[120]):

- acute, for a period of exposure of less than 6 hours;
- sub-acute, for a period of exposure of between 6 and 24 hours;
- chronic, for a period of exposure of more than 24 hours.

This classification based on the period of exposure to cold gives some indication of the rate of cooling of the body, which is important from the therapeutic point of view, since disturbances of the fluid balance and the electrolytes are relatively slight in acute hypothermia. In accidental immersions the first two types of hypothermia may be observed, depending on the state of the protective clothing, the possibility or otherwise of getting into a dinghy, and the ambient conditions: water and air temperature, wind etc. Situations of this kind are often complicated by partial drowning, particularly during the evacuation of helicopters or in rough seas.

2.2 Physiological Reactions and Symptomatology of Acute and Sub-Acute Hypothermia in Man

2.2.1 Changes in Body Temperature

Changes in the response of the organism to cooling are generally described in terms of changes in the central temperature of the body (most frequently the rectal temperature). Lefevre[201], as early as 1898, described the route taken by the thermal topography from the skin to the liver during the complete cooling of a homeothermal animal, the rabbit, immersed in a bath at a temperature of 5°C (Fig.3.1).

The general development of the curves indicates that there were three main phases. During the first period, the rectal temperature remained stable or rose slightly. The muscular temperature followed the same course, while the skin temperature fell rapidly. In the second period, there was a rapid fall in the rectal and the muscular temperatures, while the skin temperature remained stable. Finally, during the third period, the rectal, muscular and skin temperatures decreased together until there was complete failure of the organism; the animal essentially became a poikilotherm. In humans (Fig.3.2), there is a comparable change in the rectal temperature. From the first few minutes of being immersed in cold water, the rectal temperature rises more or less sharply according to the subjects and the extent of coldness (see Chapter 2), then falls fairly rapidly and tends to become stable at a lower level, but one which is more than 35°C if the thermal constraint is not too great. If the latter is so great that the organism is unable to withstand it, the rectal temperature drops to 35°C. Below this value the drop in temperature is accelerated, and the decrease takes on an exponential development. Death generally occurs at between 28 and 24°C if no treatment is given. These changes are accompanied by a number of symptoms indicating the objective nature of the severe degradation of the main functions: metabolism, circulation, respiration, excretion, functioning of the central nervous system.

2.2.2 Effect on Metabolism

The effects of cold on the body's metabolism when the rectal temperature is more than 35°C have been described in the previous Chapter. The metabolism is all the higher if the stresses are greater, with a maximum of 5 or 6 times the value of the basal metabolism, or approximately 300 W/m^2. This level of heat production by shivering cannot be maintained for a long time without causing fatigue. A certain muscular rigidity replaces shivering and the subjects suffer cramp in the muscles of their calves and thighs. These disorders may appear while the rectal temperature is still relatively high (35.5 to 36°C), as the author observed during experiments on volunteer subjects who were immersed naked in still water at 15°C. This confirms the observations made by Dill and Forbes[92] that muscular rigidity could occur when the rectal temperature was normal. It is considered, however, that as a general rule the maximum metabolic response can be maintained down to a rectal temperature of 34.8 to 35°C, below which value there is a gradual reduction in the metabolic rate. When the rectal temperature reaches about 32°C, shivering is no longer detectable clinically, although some muscular tremor still persists. The dominant symptom at this stage of hypothermia is the rigidity which affects all the muscular groups and makes breathing very difficult. The calorific value of this rigidity of the muscles is not known, but it is assumed that the basal metabolic rate is reached at a rectal temperature approaching 30°C. Muscular rigidity generally ceases at about 27°C. From 30°C onwards it is possible to consider that the metabolic rate obeys van t'Hoff's law, that is, that it decreases by half for a drop in body temperature of 10°C.

2.2.3 Effect on the Cardio-Vascular System

This effect is extremely important, since death during hypothermia is due to cardiac failure.

Following an increase in the central arterial and venous pressure, accompanied by intense peripheral vasoconstriction and an increase in the frequency of the heart beat and in the cardiac output at the beginning of the immersion period, bradycardia proportional to the drop in the rectal temperature sets in when the latter is less than 34°C. As a general rule it is sinusal (Nicolas et al.[232]); moderate until a rectal temperature of 28–30°C is reached, it then becomes greater under the effect of the cold which acts directly on the nodal tissue. Very low frequencies of less than 30 can thus be observed (23 and 25 in two observations quoted by Nicolas et al.[232] for a rectal temperature of 24°C).

The electrocardiogram reveals this bradycardia. A lengthening of the PR space is in fact observed, which, in adults, increases from 160 milliseconds to 200 or 220 milliseconds. The latter value is probably the upper limit of the PR space lengthening, but Nicolas et al.[233] found a value of 280 milliseconds in a patient whose rectal temperature was 24°C. There is considerable lengthening of the QT interval to a value which is higher than the ideal value fixed in relation to bradycardia. This lengthening is +37% of the ideal value. It is almost always found to occur during hypothermia and is not related to the depth of the latter. It is remarkably well tolerated, even when it reaches extreme values and is quickly reversible during the rewarming period.

Deformation of the pattern of the QRS complex (Fig.3.3) during hypothermia has been known for some time. This is an enlargement of the complex caused by a projection in the ascending branch of R, which, in certain cases, quickly joins the isoelectrical line. It may also take the form of a dome of variable amplitude; the projection is called a "raising" of the point J, while the dome has been given the name "J wave" or "Osborn wave". According to its origin, one or other of these two forms can be observed. In the view of many authors, the depth of the hypothermia plays an important part. For example, the J wave or the projection would nearly always be found in cases of hypothermia of less than 30°C. The interpretation given to this hypothermia wave is not clear: Bigelow[32] and Osborn[236] interpret it as a lesion current related to acidosis and regard it as a pre-fibrillatory aspect. Other authors think it is caused by a ventricular conduction disorder or that it is an auricular repolarisation wave. Some people regard it therefore as a depolarisation delay while yet others interpret it as a disturbance of repolarisation. However, according to Nicolas and Bouhour, this wave has no prognostic significance whatever. Finally, there is an excessive levelling of the ST segment and sometimes an inversion of the T wave.

Bradycardia is frequently accompanied by irregularity of the heart beat. For example, at the very beginning of the cooling period, extrasystoles can be observed. Auricular fibrillation, considered by most authors as occurring very often (60 to 80% of cases, according to Fruehan[100]), may appear at a rectal temperature of 33°C, but the critical temperature is probably lower: 27 to 28°C. Auricular tachysystoly is also observed and, at about 28°C, auriculo-ventricular dissociation. Below 28°C, ventricular fibrillation is the greatest risk to be feared as its reversibility is a very uncertain process. It is often triggered by mechanical stimuli during the handling of victims of an accident during their recovery. It may appear spontaneously below 25°C and prevent the functioning of the cardiac pump. The aetiology of ventricular fibrillation is very complex. According to Keatinge, it does not appear to be due to the direct effects of cold on the cellular membrane of the cardiac muscle, but rather to an inadequate supply of energy to this muscle. This view seems to be confirmed by Nicolas and Bouhour[234], who found, after examination under the electron microscope, that the cardiac cell was seriously affected on an ultrastructural scale. This consisted mainly of mitochondrial changes in which the mitochondria increased in volume and there was a vacuity in the matrix of the sarcosome. In addition, there was very little glycogen and very few intracellular lipids. Lesions of this type point to the existence of cell damage, affecting mainly the energy components of the cell. They are reversible, but reversibility is gradual and takes more than 24 hours after the return of the normal body temperature. Such lesions may therefore be the cause of loss of contractility during the rewarming process and of unexpected irregularity of the heart beat. Other factors which predispose to the occurrence of ventricular fibrillation have been invoked, among which anoxia would appear to be the most important. This is due to a serious reduction of ventilation below 27 to 28°C, having as a corollary the retention of CO_2 and the gradual production of acidosis, causing fibrillation. Anoxia is furthermore a cause of potassium leaving the cellular spaces, this ion imbalance being a factor predisposing fibrillation.

The cardiac output decreases simultaneously with the drop in body temperature in parallel with the reduced oxygen consumption. In acute hypothermia, the main reason for this reduction is bradycardia, since the systolic ejection volume remains normal or even slightly above normal. If hypothermia is prolonged, there is another factor which contributes to a fall in cardiac output: this is hypovolemia, associated with an extravascular escape of plasma or a storing of plasma in certain vascular compartments. This hypovolemia is demonstrated by a reduction in the systolic ejection volume associated with low filling pressures – right auricular pressure and pulmonary capillary pressure – and by measurement of the total volume of blood (Harari et al.[135]). If the rectal temperature is 25°C, the cardiac output is reduced by about 70% as compared with its value for a normal body temperature.

Blood pressure is often normal or only slightly lower, down to a rectal temperature of about 27 or 28°C. On the other hand it is very low below 25°C, and is often difficult to take by means of the arm-cuff.

The central venous pressure is very variable, but does not undergo any major modification.

Peripheral resistance shows an opposite change from that of the cardiac output, increasing as the body gets colder. However, if the rectal temperature falls below 25°C it decreases, showing that there has been a failure of vasomotor tone.

2.2.4 *Effect on Respiration*

At the start of cooling, ventilation increases considerably. Golden[119] has observed frequencies of from 60 to 70 cycles/minute in subjects immersed naked in water at a temperature of 10°C. A detailed analysis of the ventilatory reactions on immersion in very cold water has been made by Keatinge et al.[180], who showed that good swimmers, even when clothed, were unable to swim more than 250 metres in water at 4.7°C. One of their subjects, who was fairly thin, could not do more than 30 metres. The limiting factor in this case was not hypothermia but ventilation. The frequency was 56 to 60 cycles/minute with an increase in the inspiratory phase, a feeling of breathlessness, thoracic constriction and an inability to control respiration voluntarily. This reflex respiratory response, initiated at the skin by stimulation of the peripheral receptors to cold, causes real distress which, according to Keatinge, is responsible for a number of fatal accidents in cold water. In the case of accidental immersion in cold water, however, such a phenomenon should occur only rarely if the crews are wearing their anti-immersion suits, although this possibility cannot be excluded during helicopter accidents, when the crew has to evacuate the aircraft under the water. Hyperventilation leads to alkalosis which, according to Cooper et al.[83], produces a certain disorientation caused by a reduction in the flow of blood to the brain. When hypothermia develops, some respiratory depression is observed, but this is generally not severe enough to be dangerous if the rectal temperature is sufficiently high to avoid cardiac failure. The respiratory depression becomes appreciable only when the rectal temperature falls to about 28°C and will cease only at about 20°C, although Alexander[4] has mentioned earlier stoppages during the cases of hypothermia produced at Dachau. The reduced ventilation may be due to reduced oxygen requirements or to an even more marked reduction in the CO_2 eliminated, to the depressing effect of cold on the respiratory centre or again, to the fact that the peripheral and central chemoreceptors are less sensitive first of all to the hypoxic stimulus and then to the hypercapnic stimulus. Finally, it should be pointed out that the solubility of CO_2 increases under the effect of cold, thus giving rise to the development of acidosis. Broncho-dilatation is also present, which increases the anatomical dead space by 50% and the physiological dead space by 6 to 28% for a rectal temperature of 25°C. Pulmonary compliance is not affected.

The rate of circulation in the lungs decreases, in the same way as the cardiac output. Pulmonary resistance is very high. Below 25°C the volume of blood in the lungs increases and there is a tendency to pulmonary oedema and to alveolar haemorrhages. These lesions may be aggravated if there has been slight drowning. Indeed, inhaled sea water has an osmotic pressure approximately four times that of blood. It will therefore tend to cause the exudation of plasma through the already irritated alveolar walls and oedema of the lungs, in spite of the small quantities of water generally inhaled. Fuller[102] has in fact found that pneumonia and pulmonary oedema were the most frequent causes of death following drowning.

2.2.5 *Effect on the Blood*

The blood mass decreases during hypothermia, affecting mainly the plasma. The haematocrit value therefore increases, and at a relatively early stage, as the author has observed it during immersion in water at 15°C while the rectal temperature was still very much higher than 35°C. There is an increase in the viscosity of the blood and leucopenia and thrombocytopenia are also present. The bleeding time increases and the shape of the platelets alters. Particularly pronounced below 26°C, these changes are completely reversible.

Glycaemia is high in relation to disturbances of the glucidic metabolic rate. When the body starts to get cold, the hyperglycaemia appears to be associated with shivering; it is not observed when there is no shivering. Below 30°C, there is a reduction of insulin activity. Glucose is no longer entering the cells and the glucose already there cannot be metabolised, and so passes into the extracellular fluid.

There is an increase in the free fatty acids from the very beginning of exposure to cold, and this increase persists for 8 hours. It would therefore seem that the lipids are used in preference to the glucosides which are rapidly exhausted by the intense shivering at the start of the cooling period.

There is a considerable change in the supply of oxygen to the blood. Pa_{O_2} is always very low and often less than 70 mmHg. Sa_{O_2} is also reduced and has a value of between 85 and 95%. This reduction is partly compensated by the increased solubility of the oxygen in the plasma. The increased affinity of the haemoglobin for oxygen because of alkalaemia and the reduction in Pa_{O_2} make the supply of oxygen to the tissues a variable process. However the consumption of oxygen by the tissues is less during hypothermia and the blood flow rates are reduced, so that it is difficult to say whether there is actually any hypoxia of the cells. For example, the supply of oxygen to the myocardium seems to be adequate during hypothermia (Malmejac[215]). The presence of hyperlactataemia, however, noted by numerous authors could be considered as a sign of hypoxia of the tissues.

The acid/base balance also undergoes major modifications. Cooling by a simple physical phenomenon leads to an increase in the pH value. The latter is, however, relatively low (+0.015 pH for a drop of 1°C), since the bicarbonates increase and, although the Pa_{CO_2} becomes less, the CO_2 dissolved in the plasma increases.

Changes in the blood electrolytes are frequently found, although precise data are lacking in this connection, probably because of variations in the physiological responses during cooling. Thus, Nicolas et al.[234] report that either hyponatremia, hypernatremia, or even normal natremia can occur. Similarly, kalemia is very variable, although hyperkalemia is fairly rare.

Hyperamylasemia is often observed, but is not always the sign of pancreatic affection.

2.2.6 *Effects on Renal Functioning*

The rise in the central venous pressure with its depressant effects on the secretion of ADH results in considerable diuresis at the start of hypothermia. Later on, the reduced cardiac output causes less blood to flow to the kidneys. Glomerular filtration is reduced, but diuresis may not be affected or even increased. Keatinge thinks that in such case this is a physiological compensation for the relative increase in the central blood volume as a result of peripheral vasoconstriction. Disturbances of the tubular transfers cause excretion of a part of the glomerular filtrate normally reabsorbed. Thus, there is less reabsorption of the water, which would appear to be due to the persistence of natriuresis and, in deep hypothermia (rectal temperature of 25°C), to loss of tubular sensitivity to vasopressin. Glycosuria may also be present, in spite of normal glycaemia. Finally, urinary excretion of the H^+ ions is reduced. An accumulation of lipids in the distal tube is also noted. These changes are reversible on rewarming, but only after a certain lapse of time. No concomitant histological lesions seem to exist.

2.2.7 *Effect on the Nervous System*

The brain is the part of the nervous system which is most sensitive to a drop in temperature. After a hyperexcitability phase at the beginning of cooling, cerebral activity deteriorates as soon as the rectal temperature reaches 34°C, resulting in mental confusion with amnesia and disorientation. Below 33°C, the patient is semi-conscious. He sinks into a coma at about 30°C, but this threshold varies very much from one individual to another.

The electroencephalogram starts to change when the central temperature falls below 35 to 32°C. A reduction in wave amplitude is noted, first of all in the occipital regions and then, much later, in the frontal and wall regions. At about 30°C, theta and delta shaped waves appear in the frontal region. The decline in the electrical activity of the brain continues at a regular rate during cooling until, below 25°C, a trace corresponding to a stage III coma appears to be the rule. The theta rhythm disappears, as do also the delta waves; at about 20°C there is complete electrical silence. These changes regress without sequelae on rewarming and the higher nervous functions completely recover, as shown by Malmejac's experiments on monkeys. This author showed, in fact, that monkeys subjected to profound cooling recovered, after rewarming, the conditioning reflexes acquired previously. Similar observations have been made in man. The only thing is that the more profound the cooling, the slower the recovery.

At the beginning of cooling (drop in central temperature of 1 to 3°C), hyperexcitability of the medulla is observed, but this gradually becomes less if cooling is more pronounced. The osteotendinous reflexes are often quick, even polykinetic, down to about 32°C. Below this temperature, they become slow, incomplete, and disappear when the rectal temperature is between 30 and 28°C. The skin reflexes also disappear at about this temperature.

The pupils contract in moderate hypothermia, but if the central temperature falls below 30°C, the photomotor reflexes disappear and bilateral mydriasis is observed.

2.2.8 *Effect on Endocrine Glands*

These glands are affected only in profound hypothermia. Cold has a depressant effect on endocrine gland functioning:

Hypophysial activity is reduced and ACTH synthesis decreases gradually and finally stops if the central temperature reaches 28°C.

Hypothermia reduces the secretion of corticosteroids, but the adrenal cortex gland reacts to an injection of ACTH at central temperatures of less than 25°C. It would therefore seem that the reduction in adrenocortical secretion is due to reduced stimulation from the ante-hypophysis.

Less adrenaline and noradrenalin is secreted during hypothermia, but the sensitivity of the receptors, particularly those in the heart, increases.

The activity of the thyroid gland is reduced, as is also the secretion of insulin.

2.3 Diagnosis

A diagnosis of hypothermia in accidental immersion is relatively easy. It should, however, be distinguished from drowning. Hypothermia can be confirmed if the skin is cold, livid, and exhibits the symptoms previously described and if there is a low body temperature. To measure the body temperature, it is desirable to use special probes, e.g. oesophageal probes, or, if these are not available, a thermometer on which the scale goes down to 20°C. However, in the majority of cases, it is not possible to take the temperature of a victim at the time of his recovery from the water, and a diagnosis should therefore be based on the symptoms present when estimating the depth of the hypothermia. Classifying hypothermia into three stages can help to estimate the central temperature (Hagelsten and Jessen[126]). These three stages are as follows:

- *an excitation stage*, in which there is violent shivering, peripheral vasoconstriction and slight nervous symptoms: disorientation, confusion, introversion and even amnesia. The heart and respiratory rates become faster. At this stage, the rectal temperature is between 35 and 34°C.
- *an adynamic stage* between 33 and 30°C, in which there is bradycardia and cardiac arrhythmia. The rate of respiration remains high and shivering is replaced by muscular rigidity. The victim is semi-conscious.
- *a stage of torpor and paralysis* below 30°C, which may terminate in a comatose state which is difficult to distinguish from death. The victim is unconscious, he has no reflexes, his pupils, which are dilated, do not react to light, the respiratory rate is very low: two to three movements a minute with a very low amplitude. The pulse is imperceptible and blood pressure cannot be measured with an armband. Heart sounds cannot be heard with the stethoscope. This condition could suggest death, but survivals have been described among patients who have been regarded as dead, hence the rule that resuscitation must always be tried. This is due to the particular resistance of the brain to hypoxia below 30°C. It is therefore now generally agreed that the usual criteria for death are not strictly applicable in the case of hypothermia. Death by accidental hypothermia is then defined as being the impossibility of reviving the victim by rewarming (Hagelsten et al.[126], Golden[120]).

2.4 Treatment

2.4.1 Preventive Treatment

In the water, cooling is a very rapid process. It is therefore important to make use of equipment which is watertight and which provides good thermal insulation (see Chapter 4). A life raft, or dinghy, considerably increases the chances of survival, although there is always the possibility that it may be lost or that the state of the sea or of the victim is such as to make it impossible to get into it. In such cases, there is a great risk of hypothermia. Everything will depend on the speed of action of the rescue services and the signalling devices available to the victim: radio beacon, mirror etc. There has been much discussion of the usefulness of swimming. Glaser[115] considered that it was preferable to swim vigorously rather than allow oneself to float in the water without moving. He considered that the heat produced by swimming should be sufficient to balance or at least to reduce the heat loss. This opinion must be rejected. Physical activity, indeed, causes the skin temperature to rise and increases the coefficient of heat exchange in water h_c. The heat losses will therefore increase in large proportions (see Chapter 1). In addition, the metabolic cost of sustained swimming is of the order of 350 to 400 W/m², which is not enough to make up for the heat losses. Finally, swimming uses up the energy reserves, thus hastening the occurrence of failure of the organism. The harmful effect of swimming in cold water in the case of clothed subjects was demonstrated by Keatinge[172, 175]. This author showed that the fall in the rectal temperature was three times higher after 20 minutes of immersion in water at 5°C for clothed subjects doing work, than for subjects similarly clothed but resting in the water. Victims are therefore advised to remain as motionless as possible in the water until the arrival of help, unless they can swim quickly to a place of refuge. If victims are unable to get into a dinghy, they will find it helpful, according to Hayward et al.[143], to remain together as close as possible to each other, holding to each other's shoulders in order to conserve their heat better. This method would increase the survival time appreciably by reducing by 66% the rate of fall of the rectal temperature. A similar method can be adopted in multiseat dinghies. If the victim is alone, the same authors advise him to get into a curled-up position in the water, with arms folded and lying flat along the side walls of the thorax, thighs up and legs bent. This position slows down the lowering of the rectal temperature by 69% in water at 9–10°C.

Apart from the cold, problems of food and drink may arise if help is long in arriving. Life rafts are normally supplied with survival rations and a certain amount of drinking water. The quantity of water may, however, be insufficient. If this is so, it is absolutely essential to avoid drinking sea water, the salinity of which (35‰) is distinctly higher than that of the organism (10‰). The absorption of sea water leads to serious disorders: intestinal disorders with diarrhoea, dehydration caused by the hypersalinity of the liquid absorbed, which aggravates the haemoconcentration produced by the cold, and finally mental disorders with suicidal tendencies. The best plan is to ration the drinking water: 500 ml a day per person is sufficient. This quantity can be reduced to 100 ml a day, if necessary.

2.4.2 Curative Treatment

From the very start a decision has to be made whether to begin treatment on the spot in conditions which are often precarious, or to transfer the patient to a hospital centre. This decision is obviously governed by the seriousness of the patient's condition and the distance to the hospital. In accidental immersion two methods of rescue are possible: by helicopter or by boat. Recovery by helicopter is generally quick and the journey to a hospital centre or a well equipped treatment centre is relatively short. Recovery by a rescue boat on the other hand may mean long delays before appropriate treatment can be given. According to Golden[120, 121], if the journey takes more than 30 minutes, it is better, if possible, to give first aid on the spot. Indeed there are two major risks during transport of the patient: aggravation of hypothermia, and ventricular fibrillation as a result of being handled a great deal.

Action to be taken on the spot. After removing the victim from the water with the utmost care, the first thing is to prevent further heat losses due to evaporation of the water in the suit. For this purpose, not even damp clothing should be removed, to avoid unnecessary handling, but the victim should be placed in a plastic bag or, failing this, wrapped in blankets. It is absolutely essential to avoid using friction or massage, and no alcohol must be given. The airways must be kept free and oxygen given. The patient must be transported with his head slightly inclined downwards

so as to reduce the risk of hypotension due to rewarming of the peripheral tissues and to the relative suppression of the vasoconstriction caused by the cold.

Complications may occur during transport. In relatively light forms of hypothermia the victim may be conscious or semi-conscious at the time of recovery, but may lapse into unconsciousness a few minutes afterwards. This condition is due to the continuing fall of the central temperature after the rescue, known as the "after drop". This is the result of the return of the cooled peripheral blood to the central regions. This continuing drop in the central temperature is greater in fat subjects than in thin ones, in view of the greater mass of peripheral tissues which are once more being irrigated. If the journey is short, it is therefore better not to try to rewarm the victim with hot water bottles or other means. The return of the cold peripheral blood to the heart may cause ventricular fibrillation and cardiac arrest. If such is the case, external massaging of the heart should be carried out immediately but at a slower rate than that normally used for a subject with normal body temperature. However, in profound hypothermia it is difficult to diagnose cardiac arrest, particularly as the victim is still fully clothed. Care must therefore be taken not to make a hasty diagnosis of cardiac arrest, since massage at this stage of hypothermia may result in ventricular fibrillation if this has not yet occurred. Breathing may also have ceased. If this follows cardiac arrest, it must be treated by resuscitation using expired air. Here again, one must proceed with extreme care.

Treatment of hypothermia in a hospital. The main treatment is to rewarm the patient. Methods of rewarming have been the subject of much discussion, some authors being in favour of rapid rewarming and others of slow rewarming. The rate of rewarming depends in fact mainly on the type of hypothermia – acute, sub-acute or chronic. In acute hypothermia there is very little electrolytic disturbance, and we are concerned primarily with a thermal problem. The best treatment is therefore rapid rewarming of the patient. Two methods are used: active external rewarming and central rewarming.

Active external rewarming consists in applying heat externally either by means of electric blankets or a number of hot water bottles, or by a hot bath. The latter method is the one most frequently used. It consists of immersing the patient in a bath at 40–41°C, if he is naked, or at 43–44°C if he is clothed (Golden[121]). This method achieves a rise in temperature of from 1 to 1.3°C per hour. A number of variants of this method are used. Jessen[167] recommends plunging the patient first of all in a bath at 34–35°C to avoid a too severely painful skin reaction on rewarming and the risk of ventricular fibrillation. After 10 minutes' immersion, the bath temperature is raised to 42°C and rewarming is stopped when the central temperature reaches 34°C. For Golden, rewarming in a bath consists in immersing the patient fully clothed in water at 43–44°C, keeping the limbs out of the water so as to prevent too rapid a return of the peripheral cold blood to the heart (Keatinge[182]). The clothing is cut off in the water to reduce handling to a minimum. During the whole of this period, the patient must be kept under supervision and respiratory and circulatory aid given if necessary. Oxygen (95% O_2 + 5% CO_2) is given through a respirator to meet the increased oxygen requirement of the organism. The patient is kept in the bath until he states that he is feeling warm again. He is then *carried* to a previously warmed bed where he is given hot sweet drinks. This method of rewarming in a hot bath gives spectacular results, particularly in cases of profound or moderate acute hypothermia. All the authors agree, however, in pointing out the frequent risk and the suddenness of rewarming collapses, which are difficult to treat and very often complicated by a relatively large drop in the central temperature, even as much as 3°C according to Alexander[4], and causing a loss of consciousness and even cardiac arrest. Patients with a lot of adipose tissue must be kept under particularly close supervision from this point of view. In the event of collapse on rewarming, the patient must be taken out of the bath and his head bent forwards until he recovers consciousness. In serious cases, the intravenous administration of 500 cc of plasma warmed to 38°C has proved very useful. He is then put back into the bath with the limbs kept in an elevated position. Ventricular fibrillation, followed by cardiac arrest may occur if the central temperature is less than 28°C because of the "after drop". This possibility is particularly likely to occur when the cold, acidic, hypoxic and hypoglycemic blood at the periphery returns to the heart. According to Linton and Ledingham[210], defibrillation is only of limited value if the central temperature is below 28°C. However, if the sinusal rhythm does not reappear when the central temperature reaches 30°C, direct current defibrillation must be undertaken immediately. In the event of respiratory depression, artificial ventilation must be of a moderate nature to avoid hypocapnia which would have disastrous effects on the dissociation of the oxygen in the blood and on the flow of blood to the brain. If there has been partial drowning, intermittent positive pressure ventilation, combined with a heated humidifier, should be applied. A probe should not be inserted in the trachea unless absolutely necessary as it may cause reflex bradycardia and trigger ventricular fibrillation.

Since active external rewarming may result, as we have just seen, in a number of injuries associated with intense peripheral vasodilation at a time when the myocardium has not yet been rewarmed and thus cannot increase the flow to the heart, several authors recommend active internal rewarming. This method has the main advantage of rewarming the central core first of all. A number of techniques are employed:

either: peritoneal dialysis and the artificial kidney, a method which is used primarily for cases of hypothermia with a toxic origin, since it promotes the elimination of the toxic product if the latter can be dialysed;

or: circulation outside the body with heat exchangers, a method applied mainly in very profound hypothermia (below 25°C). With this technique, rewarming is very rapid and the patient's central temperature may reach 37°C in less than an hour, according to Truscott et al.[287] and Towne et al.[286]. Nicolas et al.[232] think that the internal rewarming method should be used only for hypothermia less than 28°C; for less severe hypothermia passive external rewarming should prove beneficial;

or, finally, rewarming through the airway. This method can also be used as an adjuvant to rewarming in a hot bath. This is a very old technique, since Kay, as early as 1834, recommended rewarming the air blown into the lungs of victims of drowning or asphyxia, and it is a method which has recently been revived by various authors. Lloyd[211] has put forward a hot gas method which uses the heat produced by the reaction of CO_2 with soda lime, and has described portable apparatus based on this principle. This author thinks that oxygen/helium mixtures should provide better results still, because of the high coefficient of heat transfer of helium. According to him, this method does not cause an after drop of the central temperature and the heart is the first organ to improve its functioning. Hayward and Steinman[144] recently compared the gas rewarming method (humidified oxygen) with that of the hot bath. Out of 10 subjects who were cooled to 35°C, there was no significant difference in the after drop of the rectal temperature, but the inhalation of the hot gas caused a more rapid rise in the temperature of the oesophagus and the tympanum, indicating early rewarming of the cardiac region and of the brain. The authors recommend the simultaneous use of both methods, restricting the bath rewarming method to the trunk regions.

In sub-acute accidental hypothermia a rapid rise in body temperature would considerably increase the oxygen requirement of the tissues, while the still cold heart is unable to meet this demand. In addition, the already existing water/electrolyte imbalance would be aggravated. The best treatment is therefore *slow rewarming* at ambient temperature, plus an auxiliary treatment. This consists in correcting:

- *metabolic acidosis*, which increases during rewarming, by the intravenous injection of 4.2% bicarbonate of soda at 38°C;
- *hypoglycaemia*, by injections of 5% glucose serum, which also corrects hypovolemia;
- *electrolyte disorders*, by additions of sodium and potassium.

Antibiotics are absolutely essential as soon as there is the least sign of infection, either localised (frostbite), or general.

Complications

According to Nicolas and Bouhour[233], asystolic type damage which occurs during rewarming or even after a return to normal temperature is fairly frequent. To avoid its occurrence, they recommend that an electrosystolic endoscope should always be inserted as soon as the temperature goes beyond 31°C.

Fatal complications resulting from bronchopneumonia occur with a frequency which varies from author to author. They may be the cause of a third of the deaths. The initial stages of drowning with inhalation of sea water makes a prognosis very difficult.

Delayed acute nephrosis has been known to occur after sub-acute hypothermia.

To summarise: Hypothermia and lesions in the extremities due to cold, e.g. immersion foot and hand or frostbite, constitute the main risks in accidental immersion in cold seas. Their seriousness depends on the value of the protective equipment available and the speed with which help arrives. In the majority of cases, hypothermia is acute (less than 6 hours) and rapid rewarming by external or internal means is the best method of treatment. In sub-acute hypothermia (more than 6 hours), slow rewarming followed by correction of biochemical disorders, is advocated by numerous authors. Stress should be laid on the fact that resuscitation must always be attempted, even on subjects who appear to be dead.

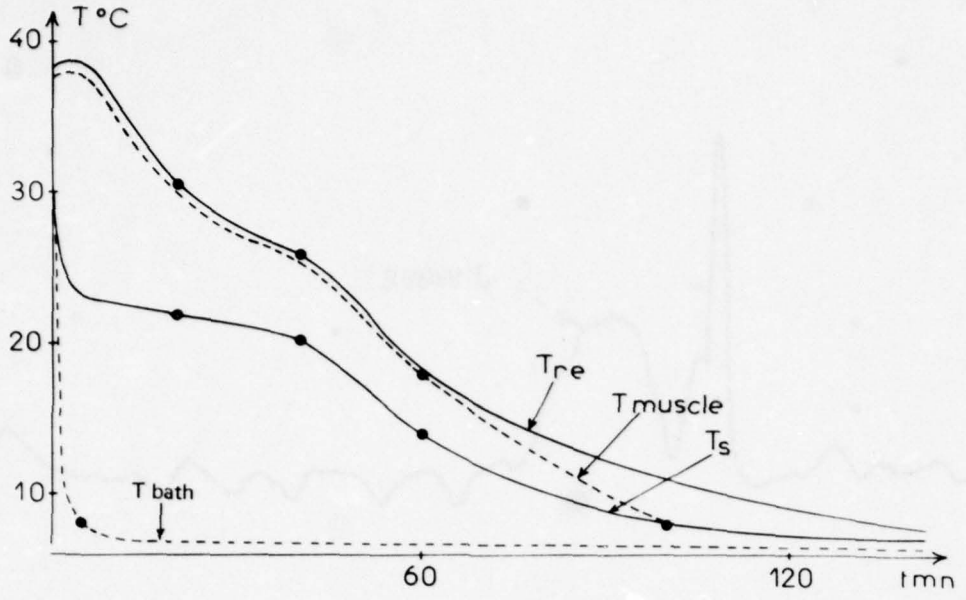


Fig.3.1 Thermal topography of the body of a rabbit during a total cooling experiment (Lefèvre[201])

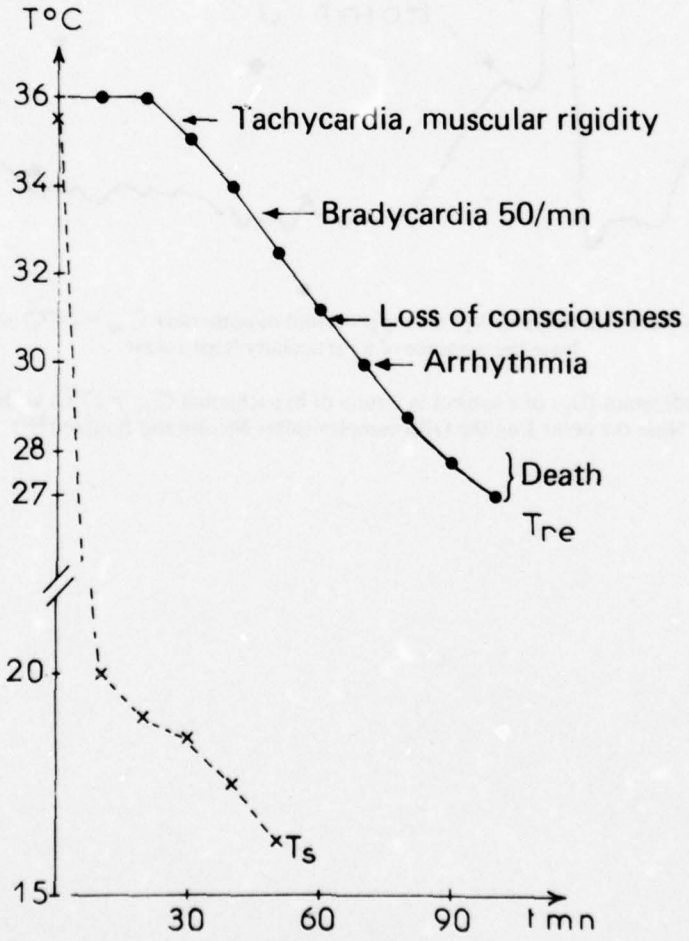


Fig.3.2 Development of rectal and skin temperatures of men immersed in cold water (2 at 12°C) and symptoms accompanying the fall in the rectal temperature (Figure based on data taken from the report by Alexander[4])

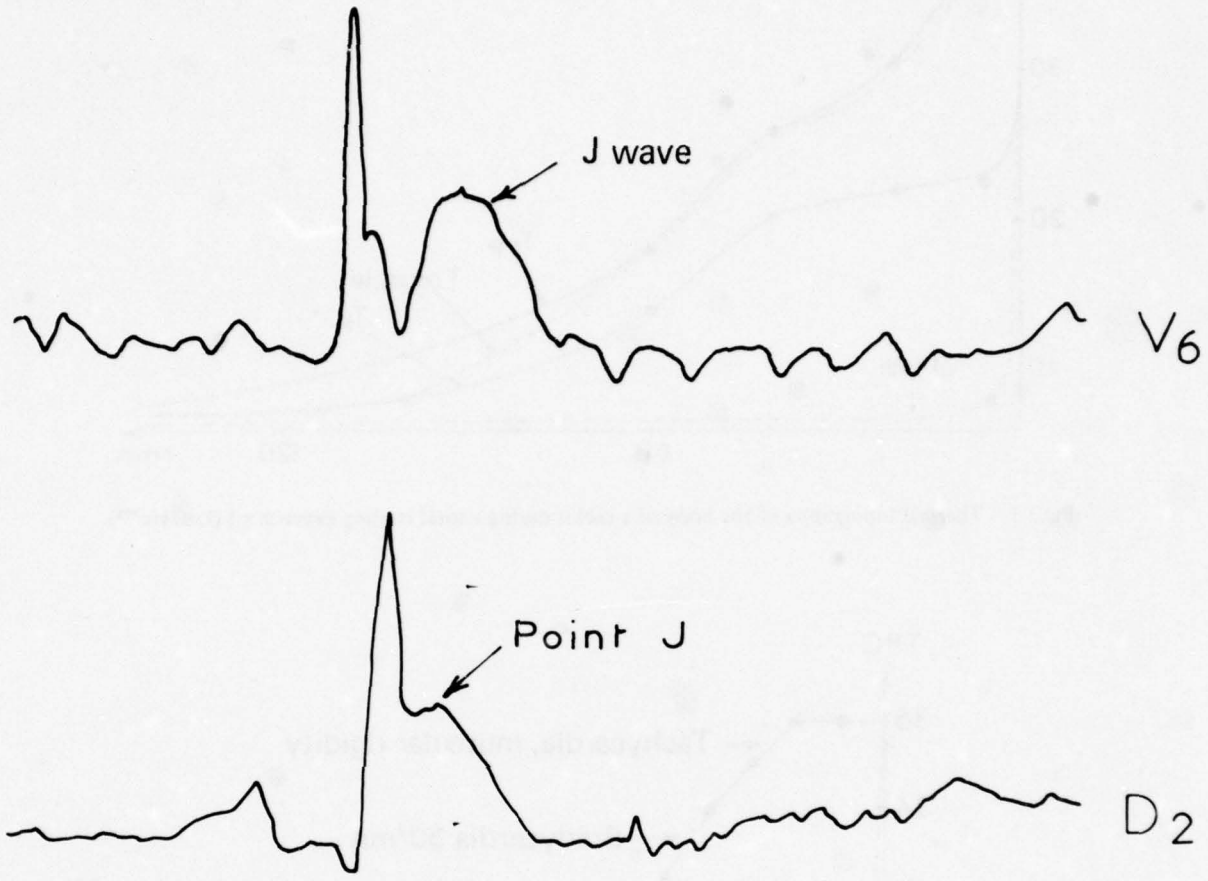
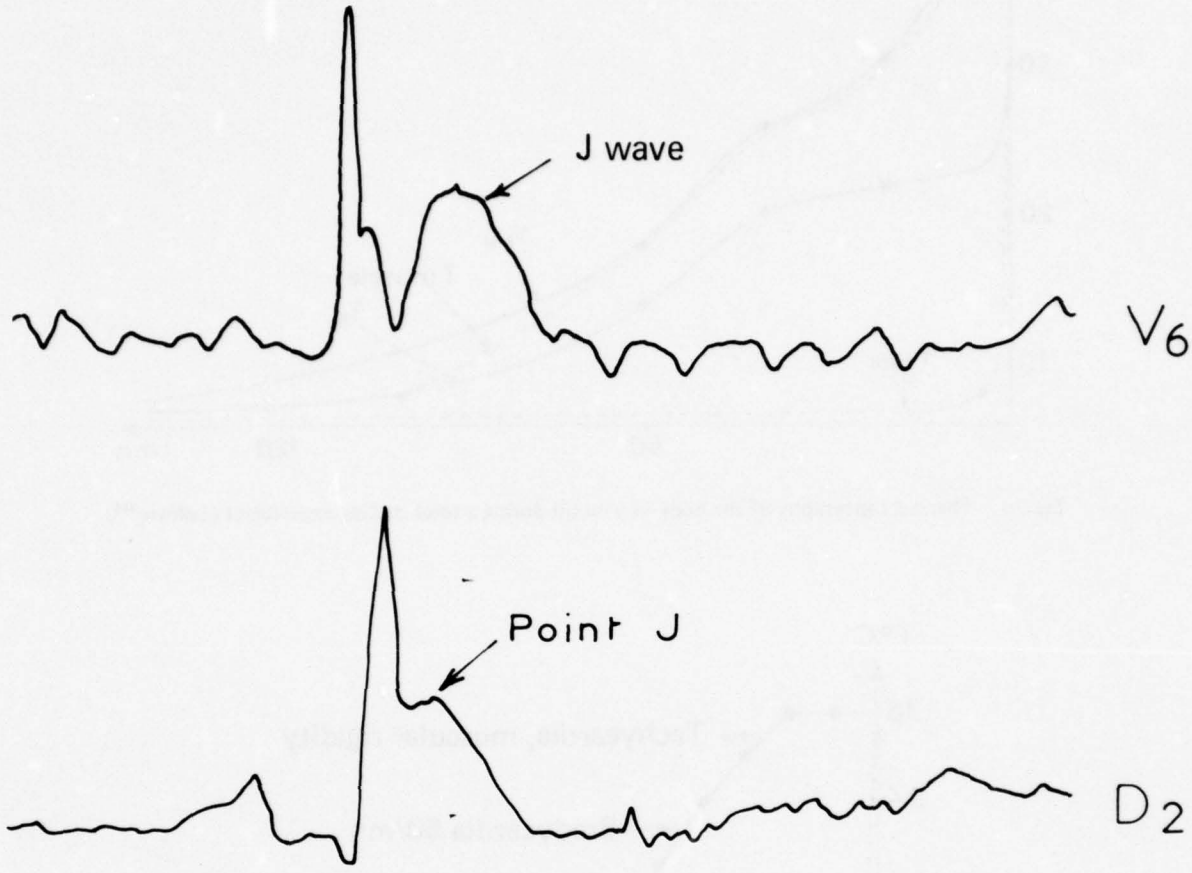


Fig.3.3 Top: Human electrocardiogram (V_6) during profound hypothermia (T_{re} = 24°C) with bradycardia at 23. Note the presence of a particularly large J wave.

Bottom: Electrocardiogram (D_2) of a subject in a state of hypothermia (T_{re} = 27°C) with bradycardia at 32. Note the point J on the QRS complex (after Nicolas and Bouhour[233])



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Bottom: Electrocardiogram (D_2) of a subject in a state of hypothermia (T_{re} = 27°C) with bradycardia at 32. Note the point J on the QRS complex (after Nicolas and Bouhour[233])

Chapter 4

PROTECTION AGAINST ACCIDENTAL IMMERSION

An analysis of the physiological reactions of a man exposed to cold water immersion has shown that the organism's means of defence were very limited. Indeed the latter can ensure the maintenance of a constant body temperature only in a range of water temperatures of between approximately 34 and 24°C (Beckman and Reeves[26], Boutelier et al.[45]). Below that temperature, although there are considerable individual differences related mainly to variations in the thickness of the layer of sub-cutaneous fat (Keatinge[181], Boutelier et al.[46]), the survival time of the unprotected man is limited. Military aircrews carrying out missions are frequently obliged to fly over seas or oceans, the surface temperature of which is generally less than 20°C. Thus, in summer, the temperature of the Pacific Ocean, depending on the latitude, is between 10 and 15°C off the coast of Canada and between 15 and 20°C off the coast of the USA. In winter, these temperatures are much lower: between 0 and +10°C in the case of Canada and between 10 and 15°C for the USA. The temperatures in the Atlantic Ocean are approximately the same: 0 to 15°C in summer for Canada, and 15 to 25°C for the USA. In winter, these temperatures vary from −2 to +5°C off the Canadian coasts and from 10 to +20°C off the US coasts. In the European theatre of operations, the seas or oceans most frequently crossed are the Baltic Sea, the North Sea and the Channel, the Atlantic Ocean off the coasts of France and the Western Mediterranean. The following Table gives the monthly and annual mean value of the number of days on which the temperature of the surface waters in these regions is less than 15°C.

TABLE 4.1

Monthly and Annual Mean Value of the Number of Days on which the Temperature of the Water is Less than 15°C

(AC/243-D/249, AC/243 (LTSS D/17))

Month	*Western Baltic*	*Heligoland Bight*	*Atlantic Ocean (off Brest)*	*Western Mediterranean*
January	31	31	31	31
February	28	28	28	28
March	31	31	31	31
April	30	30	30	26 to 30
May	31	31	31	8
June	25	25	25	occasionally
July	4	6	occasionally	occasionally
August	4	occasionally	occasionally	0
September	19	3	occasionally	occasionally
October	31	22	20	2
November	30	30	30	30
December	31	31	31	31
Total	295	268	257	187

It will thus be seen that the water temperature exceeds 15°C during only two months (July and August) in the Western Baltic, while in the Channel, the Heligoland Bight and the Atlantic off Brest, it varies between 15 and 18°C during three months in summer (July, August, September). During the remaining months the water temperature in these areas is between about 5 and 12°C. In the Western Mediterranean the temperature of the water is of the order of 17 to 22°C for about 6 months of the year (from May to the beginning of November). It should, however, be pointed out that this temperature may suddenly drop due to the wind (mistral).

Figure 4.1, taken from the Oceanographic Atlas of the North American Ocean, gives some idea of the changes in the mean temperatures of the water in the Atlantic Ocean and in the Western Mediterranean. In these conditions, the survival of aircrews, wearing conventional clothing (nomex underwear, flying suit and life jacket), is limited, as can be seen from the estimates by Saunders[253] reproduced in the following Table:

TABLE 4.2

Estimated Survival Time in Hours in European Waters for a Man Wearing a Life Jacket but no Protective Clothing Against the Cold

Sea areas	J	F	M	A	M	J	J	A	S	O	N	D
Western Baltic	1.5	1.5	1.5	3	3	6	6	12	6	6	3	1.5 to 3
North Sea Channel	3	3	3	3	3	6	6	6-12	6	6	3-6	3
Bay of Biscay	6	6	6	6	6	12	12	12	12	12	6	6
Western Mediterranean	6	6	6	6	12	12	limited through exhaustion			12	12	6

It should be stressed that the values quoted in this Table are only the mean values; a number of victims probably did succumb within this period, particularly in winter in the coldest waters (the Baltic and the North Sea). This appears in the very complete study by McCance[213], who showed the coldness of the air and of the water as the main cause of death during disasters when the temperature of the water was less than 10°C. The wearing of a protective suit against accidental cold water immersion therefore appears to be a necessity for crews who fly over these areas. The only crews who can dispense with it are those flying over the Mediterranean from May to October.

The problems of developing such suits are complex, since it is necessary to cater for the large number of requirements, which are sometims contrary to the main aim in view, imposed by flying conditions. Indeed such suits must be capable of keeping the victim on the surface of the water and providing him with adequate thermal protection both in the water and in an air environment. Furthermore, they must be compatible with wear of other aeronautical clothing (e.g. anti-G suit), and must not hinder flight control operations by restricting movement or inducing excessive perspiration. In view of these requirements, the protection afforded by an acceptable suit will be necessarily limited. It is therefore important to state, at the design stage of the suit, the conditions in which it will be used, particularly the environmental conditions against which it is desired to provide protection, and the required duration of this protection. The water temperatures and their seasonal variations have been indicated above. It should, however, be emphasised that in countries with a temperate climate there may be a great difference between the pleasant temperature of the air, particularly in the Spring, and that of the much colder water. In such case the only criterion to be taken into consideration when requiring the wearing of anti-immersion suits is the temperature of the water. Determining the length of time for which protection is necessary is a more difficult matter. Indeed the inclusion of a life raft in the survival pack would seem to indicate a duration in water of only a few minutes. In actual fact, provision must always be made for the possible loss of the dinghy, its destruction by a bullet in war time, or the impossibility of getting into it, either because the sea is too rough or because the pilot is wounded. Other reasons may also be invoked. Thus Every and Parker[95], in an analysis of the problems encountered by US Navy aircrewmen in South East Asia, mention that a number of pilots became entangled in the cords of their parachute and consequently could not get into their dinghy. The length of time during which aircrew personnel remain in the water will depend in fact on the time taken to recover them, and hence on the organisation of the rescue services. For example, in South East Asia, the same authors state that almost all the aircrewmen (99%) were recovered in the first two hours. In peacetime, the period of immersion is generally short; it rarely exceeds 30 to 60 minutes when the accident occurs near the coast (about 10 to 20 miles), as witness the recent reports of accidents involving Canadian Army pilots in Canada and in Europe (DCIEM documents). However, it is wise to anticipate that the period in the water may be much longer, either because bad weather prevents immediate recovery or because it has not been possible to locate the aircraft. This fact emerges from the analysis made by McCance of a large number of rescue operations; indeed in this study it is noted that some victims spent more than two hours in the water, followed by up to three days in a dinghy. Anti-immersion suits must therefore provide effective thermal protection for several hours in the water plus approximately one day or more in an air environment.

THEORETICAL BASIS OF PROTECTION BY MEANS OF CLOTHING

1. Anti-Immersion Protection

Heat exchanges between a clothed human body and the ambient environment takes place by conduction through the clothing, and in the case of immersion, the following equations can be written if the stable condition has been reached:

$$\frac{(M - H_{res})\,A_D - H_{head} \times A'}{A_D - A'} = h_{cl}(\bar{T}'_s - T_{cl}) = h_c(\bar{T}_{cl} - T_w) \qquad (4.1)$$

in which M is the metabolism of the subject, H_{res} and H_{head} are respectively the heat loss through the respiratory passages and the head, expressed in W/m^2. A_D is the area of the body and A' the area of the head, expressed in m^2. h_{cl} is the thermal conductance of the clothing and h_c the coefficient of heat exchange in water (see Chapter 1).

These two coefficients are expressed in $W/m^2 \cdot °C$. $\overline{T}_s$ and $\overline{T}_{cl}$ are respectively the mean skin temperature under the clothing and of the outside surface of the clothing, and T_w is the water temperature.

The conductance h_{cl} is often replaced by its reciprocal, insulation or thermal resistance $I_{cl}(I_{cl} = 1/h_c)$. The latter is expressed in $°C \cdot m^2/W$, or in clo (see Chapter 1).

From the equation quoted above it is possible to calculate the thermal insulation theoretically necessary to protect a man in water at various temperatures. Thus, a subject with a metabolic rate of $60\ W/m^2$, heat losses through the respiratory passages and the head, estimated at 12% of his metabolic rate, and a mean skin temperature of 33°C, should wear a suit which has a mean thermal insulation of $0.608°C \cdot m^2/W$, or 3.9 clo in water at 0°C, which is only slightly rough ($h_c = 60\ W/m^2 \cdot °C$). In water at 10°C, with the subject in the same condition, the insulation must be $0.409°C \cdot m^2/W$ or 2.6 clo. Conversely, if the insulation value of the suit is known, the water temperature which the subject will be able to withstand in given conditions can be determined. These insulation values enable a choice to be made from among the materials which can be used to make anti-immersion suits so that they are not too thick. Thus, the best thermal insulation is a vacuum, but its use raises considerable technological problems since it is impossible to give a suit its essential flexibility. The following Table taken from the data published by Beckman[24] and the ASHRAE[11] gives the insulation values of air and of a few currently used materials:

TABLE 4.3

Insulation Provided by Some Materials for a Constant Thickness of 1 cm

Materials	*Insulation*	
	$°C \cdot m^2/W$	Clo
Still air at 27°C	0.380	2.45
Cotton (cotton wool)	0.579	3.73
Non-compressed wool	0.263	1.70
Normal wool suit	0.108	0.70
Foam neoprene with nitrogen	0.187	1.21
Cotton (linen or hemp)	0.175	1.13
Rubber	0.054	0.35

According to these values, to obtain complete insulation in water at 0°C, the mean thickness of the suit would have to be 1.05 cm, if made of cotton wool; 2.3 cm if made of non-compressed wool; 3.2 cm if made of foam neoprene with nitrogen, but 11.3 cm if made of rubber! These estimates are in fact only theoretical, since a number of factors may cause a variation in the insulation provided by a suit. Among these factors we shall single out for discussion the effect of the geometry of the body, wetting, compressibility of the materials and the wind.

1.1 Effect of the Geometry of the Body

The insulation values given in the above Table were generally measured on samples of material, and therefore, for flat elements. In these conditions the insulation is proportional to the thickness of the material:

$$I_{cl} = \frac{x}{k}, \tag{4.2}$$

in which x is the thickness of the material in metres and k its thermal conductivity in $W/m \cdot °C$. By analogy with what happens in electrical circuits comprising several resistors in series, the global insulation or thermal resistance of a flat protective element, made up of several layers of materials of different conductivity, will have the following value:

$$I_{cl} = \frac{x_1}{k_1} + \frac{x_2}{k_2} + \frac{x_3}{k_3} + \ldots . \tag{4.3}$$

This no longer applies if the protective suit is cylindrical or spherical. The insulation is then no longer exactly proportional to its thickness and, for equal thickness, is of a lower value than in the case of a flat suit. This is due to the fact that the outer surface, where heat exchanges with the environment take place, increases with the curvature. If the suit and the skin are on two parallel planes, for each skin area element there is an exactly identical exchange area. On the other hand, if there is a radius of curvature, the area of exchange through clothing for the same skin area element increases with a reduction in the radius and hence there is a greater heat loss for an identical skin area. Indeed, for a cylinder of radius r and length l, covered with a material of thickness x and thermal conductivity k, the heat flow transferred by convection through the material, assuming the transverse heat exchanges are negligible, has the following value (see Chapter 1):

$$K = \frac{2\pi l}{\ln\left(\frac{r+x}{r}\right)} \times k(T_2 - T_1) \tag{1.7}$$

T_2 being the wall temperature of the cylinder and T_1 the outside temperature of the material.

The insulation I_{cl} is given by the formula:

$$I_{cl} = \frac{r}{k} \ln(1 + x/r) . \tag{1.9}$$

For a sphere of radius r covered with a protective layer of thickness x, it is demonstrated that the conductive heat transfer is:

$$K = \frac{4\pi}{\frac{1}{x} + \frac{1}{r}} \times k(T_2 - T_1) \tag{1.10}$$

and the thermal insulation:

$$I_{cl} = \frac{r}{k} \times \frac{x}{(r+x)} . \tag{1.11}$$

The insulation given by a protective layer around a cylinder or a sphere, as compared with that provided by a flat layer of the same thickness, can also be calculated:

$$\frac{I_{cl\ cylinder}}{I_{cl\ flat}} = \frac{r}{x} \times \ln\left(1 + \frac{x}{r}\right)$$

or:

$$I_{cl\ cylinder} = I_{cl\ flat}\left[\frac{r}{x} \ln\left(1 + \frac{x}{r}\right)\right] = I_{cl\ flat} \times A \tag{4.4}$$

For a sphere:

$$\frac{I_{cl\ sphere}}{I_{cl\ flat}} = \frac{r}{r+x}$$

$$I_{cl\ sphere} = I_{cl\ flat}\left[\frac{r}{r+x}\right] = I_{cl\ flat} \times A' . \tag{4.5}$$

The terms A and A′ are corrections applied to the value of the insulation of a flat protective layer. Van Dilla, Day and Siple[290] have studied the development of these terms as a function of the ratio of the thickness of the protective layer over the radius of the cylinder or of the sphere (Fig.4.2).

It will be noted that these terms decrease when the ratio x/r increases, the decrease being more pronounced for the sphere than for the cylindrical shape. Thus, for a constant thickness of the protective layer, the insulation decreases when the radius of the element to be protected decreases. These theoretical data can be applied to the human body by considering the latter as formed of a set of cylinders: the trunk, limbs, fingers, and of spheres: the head, finger tips. Thus, a foam neoprene suit 3.2 cm thick, the insulation of which is 3.63 clo for a flat surface, will have an insulation of 3.31 clo over the chest (radius: 16 cm), 2.94 clo on the calf of the leg (radius: 6.4 cm) and only 2.8 clo over the arms (radius: 5 cm). Conversely, the same authors studied the effect of an increased thickness of the ideal protective material (I_{cl} = 2.87°C · m²/W or 1.85 clo thickness) when placed on a flat surface or around cylinders and spheres of different diameters (Fig.4.3). They concluded that for thicknesses of about 5.5 cm or less, the insulation was approximately proportional to the thickness, in the case of flat surfaces and cylinders with a diameter of 30.5 cm, which corresponds to the trunk. On the other hand, for small diameter cylinders (2.5 cm) corresponding to the fingers, increasing the thickness of the insulating material beyond a value of 5 cm made only a very slight difference. This effect was even zero for 1.3 cm diameter spheres (finger tips), starting with a thickness of 2.5 cm. They deduced from this that the best method of protecting the hands was to use mitts, since their radius of curvature was much greater than that of the fingers of gloves.

1.2 Effect of Wetting

The insulation values quoted in Table 4.3 were measured in air with a relatively low water vapour pressure. However, it is a well known fact that increased water content reduces the insulation of a suit to a great degree. This is the result of several mechanisms: first, the thickness of damp material is generally less than when it is dry; second, if the air imprisoned in the suit is replaced by water the thermal conductivity of the material is considerably increased; finally, in the case of some suits which become impermeable on contact with water, impregnation of the surface fibres

by water increases the thermal conductivity of the material. This wetting may arise for two reasons: sweating or the ambient conditions, such as rain, mist or immersion if the suit is not watertight or, if it is watertight, tearing of the suit, for example during ejection. This effect of wetting on insulation has been well investigated by Hall and Polte[127, 129], who measured, on a copper manikin in the vertical position, in air and in water, the reduction in insulation of a watertight anti-immersion suit, due to the penetration of a fairly large quantity of water. The purpose of the measurements in air was to simulate the effect of sweating, and of those in air, the effect of water penetration as a result of deterioration of the watertight suit. Their results are summarised in Table 4.4.

TABLE 4.4

Variation of the Insulation of a Watertight Suit as a Function of Wetting in the Internal Layers (Hall and Polte[129])

Environment	*Water content* g/m²	*Insulation* clo	*Insulation reduction* %
Air	0	2.71	0
	181	2.62	3.3
	229	2.42	10.7
	312	2.24	17.3
	476	2.12	21.8
	652	1.99	26.6
	940	1.79	33.9
Water	0	1.45	0
	99	1.36	6.2
	222	1.24	14.5
	337	1.18	18.6
	436	1.03	29
	690	0.94	35.2
	1051	0.73	49.6

It will be noted that the effect of wetting on insulation is about 25% less in air than in water. This difference can be explained by the fact that the air content of the suit is less in water than in air, because of the hydrostatic pressure which compresses the suit. The increased quantity of water as compared with the total quantity of air retained in the fibres of the material is therefore greater in water, giving an equally greater reduction in the insulation of the suit. In view of the flying suits now being used and the air conditioning systems in aircraft, it may be considered that aircrews are not generally subjected to high thermal stresses. They will therefore not experience a great deal of sweating and the effect of the latter on the reduction in insulation will be relatively small (less than 10%). In immersion, on the other hand, any tearing of the watertight suit will have much more serious results. Thus, even a slow seepage of 200 g/h will reduce insulation in two hours by approximately 30%.

1.3 Effect of Compressibility of the Protective Materials

Immersion causes an increase in the pressure exerted on the suit. As the latter is flexible, this has the effect of expelling the air imprisoned within the clothing layers, and of reducing the thickness of the anti-immersion suit. A kind of patchwork pattern occurs, which is particularly evident on the abdomen and lower limbs (Fig.4.4), with a reduction in diameter of the parts of the body immersed. It should be pointed out that this patchwork pattern occurs whatever the position of the body in the water, and that it is responsible for the greater part of the insulation reduction. It is, however, difficult to assess precisely the effect of hydrostatic pressure, since in certain cases there is also the effect of the absorption of water by the surface fibres of the watertight suit. These two factors can be evaluated by comparing the insulation values in air and water given in Table 4.4. In the case of the suit used by Hall and Polte in their investigation, the resulting average insulation reduction was 49.3%. Beckman et al.[25] found approximately the same insulation reduction values for manikins immersed in the vertical position, wearing a damp neoprene suit: 48.6% for 0.6 cm thick neoprene and 41% for 0.5 cm thick neoprene. According to Hall[129], the insulation loss due to hydrostatic pressure may vary from 10 to 50% according to the compression strength of the suit.

2. Protection Against the Cold in the Life Raft

Studies made by a number of authors (Pittman et al.[238], Andrae[9] and Veghte[292]) have shown that the thermal stress imposed on aircrews in these conditions is much lower than in the water. This is due to a lower rate of cooling in air than in water because of the difference in conductivity between the two environments, to an increase in the insulation of the suit by elimination of hydrostatic pressure effects and to the additional insulation afforded by the life raft. It is

difficult, however, to analyse the heat exchanges in such a situation. Indeed, taking into account the position of the victim in the dinghy, it can be assumed that only one part of the body exchanges heat by radiation and convection with the environment. In single-seat dinghies, this part may be relatively small (about 40%) because of the quantity of water which is inevitably taken in. On the other hand, it is a great deal more than 50% in multi-seat dinghies. The lower part of the body and the back exchange heat by conduction with the walls and the bottom of the dinghy. Finally, the lower limbs, which are often immersed, lose heat by convection with the water contained in the dinghy. It is therefore difficult to find a simple expression for the exchanges based on the main parameters, especially as the exchange surfaces between the suit and the environment in the dinghy and between the dinghy and the external environment are very different in dimension and form. Since an analytical study of the exchanges is scarcely possible, an estimate is most frequently made of the overall protection afforded by the suit, the air and the wall of the dinghy in different environments. This gives an approximate assessment of the effect of temperature, wind, radiation and hygrometry. Particular attention should be paid to the effectiveness of the protection for the extremities and the head. These regions, which are generally less well protected than the rest of the body because of their geometry, are very sensitive to damp cold and serious lesions may occur (immersion foot and hand) if the time spent in the dinghy is prolonged and the ambient temperature sufficiently low (McCance[213]).

This investigation of the general principles of thermal protection has shown the very great reduction in insulation under the effect of hydrostatic pressure and humidity, and therefore the need for a watertight suit, and the difficulty of providing effective protection for certain areas of the body because of their geometry. It has also illustrated how difficult it is to provide completely effective protection in the water, taking into account the need to consider the requirements relating to flying the aircraft.

METHODS OF ASSESSING PROTECTIVE CLOTHING

The purpose of these methods is to assess the efficiency of the thermal protection afforded by a suit in cold water and in the dinghy, and in a cold air environment, and to provide an estimate of the probable survival time, depending on the temperature of the water and the air. They must also be capable of indicating any thermal stress and mechanical interference imposed on aircrew personnel during flying through the wearing of such a suit.

1. Since the watertight suit (dry-suit) is the solution universally adopted at the present time for aviation use, the first tests to which anti-immersion suits must be subjected are *tightness tests.* These are extremely important, since the least penetration of water into the suit causes a very high reduction in its insulation. In the Aerospace Medicine Laboratory of the Flight Test Centre at Brétigny these tests are carried out as follows: the subject, fully equipped, but without a life jacket, jumps into a tank filled with water at 20°C and dives to a depth of 2 m. He remains at this depth for 10 minutes, bending his limbs and trunk in various ways. He then comes up to the surface where he remains motionless for 20 minutes. The under-suits are examined and weighed before and after the experiment, to give an indication of how much water may have penetrated the suit. This is an extremely severe test, since the hydrostatic pressure of 200 mbar and the bending movements can show up any tightness defects which might possibly not be evident at the surface of the water.

2. Study of the Effectiveness of Protective Clothing Against Cold

Since the protective clothing has to give protection both in water and in air, the tests have to be carried out in these two conditions. Their purpose is to determine the insulation and heat losses through the suit, as well as the probable survival time of a man in such conditions, taking into account the physiological tolerance limits discussed in Chapter 2. Tests are generally performed in the laboratory where numerous measurements can be made, but are often usefully supplemented by tests at sea, in actual operational conditions.

2.1 Immersion Tests

Determining the overall insulation of a suit requires the application of fractional calorimetry using human subjects (the method most widely adopted), or heated copper manikins (the method used at Wright Patterson – Hall[127], and at Natick – Goldman[122]). The principle is the same in both cases. Once the stable state has been reached, it consists of finding the ratio of the temperature difference between the skin and the external surface of the suit to the power supplied to the skin by the metabolism, in the case of the human subject, or by heated resistors, in the case of the manikin. For the human subject the following can be written:

$$I_{cl} = \frac{(\overline{T}'_s - T_{cl})(A_D - A')}{(M - H_{res})A_D - H_{head} \times A'} \qquad (4.6)$$

in which $\overline{T}'_s$ is the mean skin temperature under the suit and $\overline{T}_{cl}$ the mean temperature of the external surface of the suit. M is the metabolic rate, H_{res} the heat losses through the respiratory passages and H_{head} the heat losses through the non-immersed head.

In the case of the manikin:

$$I_{cl} = \frac{(\bar{T}_s' - T_{cl})(A_D - A')}{W \times A_D - H_{head} \times A'} \tag{4.7}$$

in which W is the electrical power supplied to the manikin.

These two equations can be simplified if the insulation of the water I_w is known. The following can be written:

$$I_{cl} + I_w = \frac{(\bar{T}_s' - T_w)(A_D - A')}{(M + H_{res})A_D - H_{head} \times A'} . \tag{4.8}$$

However, this means that the coefficient of heat exchange in water has to be determined. Now, this coefficient varies with a number of parameters, among which the shape of the body, its position and the velocity of the water are the most important (see Chapter 1). This may lead to errors of from 10 to 15% in the insulation value of the suit. The mean skin temperature is calculated from a number of local skin temperatures (10, or more, depending on the authors making the tests), the values of which are weighted in relation to the skin area of the region represented, according to the method used by Hardy and Dubois[136]. The mean temperature of the surface of the suit is calculated by the same method. The metabolism and heat losses through the respiratory passages are obtained by analysing the expired gases, and by measuring the ventilation, the temperatures of the gases inspired and expired and the ambient hygrometry. The heat loss through the head is estimated by means of the following equation:

$$H_{head} = h_{(R+C)}(T_f - T_a)A'/A_D , \tag{4.9}$$

where $h_{(R+C)}$ is the combined coefficient of heat exchange by radiation and convection in air: 7.5 $W/m^2 \cdot °C$ approximately, in calm air; T_a is the temperature of the air above the water and T_f is the temperature of the forehead. A' is the area of the head not protected. The experimental procedure normally used is as follows: the subject equipped with skin temperature sensors and a central temperature sensor (generally the rectal temperature) puts on the protective suit. The latter has temperature sensors attached to its external surface. After a period of resting in air, he gets into a temperature-controlled bath, where he remains motionless until the physiological temperatures and the metabolic rate have stabilised. The stable state can be considered to have been reached when the central and skin temperatures and the metabolism have been stable for at least 20 minutes. Such experiments must therefore be performed in a bath with a relatively high temperature (13 to 15°C). A variant of this procedure consists in attaching heat fluxmeters to the outside of the suit. These enable local heat fluxes to be determined and a more precise estimate to be made of the effectiveness of the suit. However, the interpretation of the results obtained is not an easy matter. Indeed these sensors act as thermopiles, i.e. their output voltage depends on the temperature difference between their external and internal faces. Simply placing the fluxmeter on a surface creates additional insulation of that surface with respect to the environment. It follows that the temperature of the internal surface of this sensor is higher than the temperature of the external surface of the suit not covered by the fluxmeter. The heat fluxes measured are therefore probably an overestimate. In practice, however, these instruments provide some information on the relative amount of the heat losses in the various regions of the body.

Such tests have to be supplemented by immersion in baths at lower temperatures in order to determine the probable survival time when the suit is worn. Complete thermal protection in very cold water can in fact be achieved only at the cost of such an increased thickness of the suit as to make it incompatible with the flying requirements. In the absence of a dinghy, aircrews involved in an accident will therefore undergo a heat loss, the extent of which will depend on the temperature of the water and the degree of insulation of the suit. The tests are usually carried out on volunteer human subjects, using the same procedure as that previously described; the duration of the experiments is determined by the point at which the voluntary tolerance limits are reached: minimum skin and rectal temperatures (see Chapter 2), cramp, nausea, headache etc. The bath temperatures selected for these experiments vary according to the authors concerned: between 0 and 4°C in the USA and in Sweden (Veghte[293], Pittman et al.[238], Andrae[9]). In France the tests are performed at various temperatures: 13, 9, 6 and 4°C (Timbal et al.[282]). This enables the curves showing survival in relation to water temperature to be plotted with greater accuracy. The results of these experiments are analysed to meet two major concerns: assessment of the effectiveness of the protective suit in relation to the different areas of the body and determination of the survival time. The effectiveness of the protection provided is assessed according to the development and distribution of the local skin temperatures. It is based on the following criteria: no local temperature must be less than or equal to the minimum local temperatures indicated in Chapter 2: 21 to 22°C for the trunk (back, chest, abdomen), 21 to 23°C for the mean skin temperature and 10°C for the temperature of the back of the hand or of the foot. In addition, as far as the limbs are concerned, the temperature difference between any two adjacent regions, e.g. thigh and foot, must be less than 10°C, otherwise painful phenomena will appear which are very comparable to those observed when the temperature of the bottom of the foot falls below 10°C. Such criteria should avoid the occurrence of cold injury in the extremities (immersion foot and hand). Figure 4.5 taken from our experimental results is an example of the variation and distribution of local skin temperatures of a subject wearing an ARZ 14 under-suit, the ARZ 842 dry suit and a life jacket, immersed up to the base of the neck in a bath at 9°C.

Determining the survival time as a function of the water temperature is a more complicated task, since it depends on the tolerance limits chosen for the physiological temperatures and the body heat loss. It is generally assumed that there is 100% survival when the rectal temperature is 35°C or over, although some authors use slightly higher or slightly lower values: Andrae[9] 35.5°C and Hall[132] 34.7°C. The limits of the mean skin temperature vary to a greater extent from author to author. Andrae estimates that there is 100% survival if $\bar{T}_s$ is 24.5°C for a T_{re} of 35.5°C, but Hall's values are higher, since they are based on the more or less painful character of cooling and not on an actual survival limit: 31.1°C for $T_{re} = 35.9$°C, 30°C for $T_{re} = 35.6$°C and 28.3°C for $T_{re} = 34.7$°C. This latter value for $\bar{T}_s$ would appear to be too high in view of the rectal temperature, the very low temperature of the water and the inadequate insulation provided by the suits. The maximum value which the present author would assume for the mean skin temperature is 22°C, since when this is reached, the mean voluntary tolerance time is about two hours (Iampietro[159], Boutelier[50]).

Evaluation of the maximum heat loss compatible with survival in 100% of cases is a problem for which it is difficult to find a solution. The object is to determine the quantity of heat which the body can lose to reach the limit temperatures indicated above. Burton and Bazett[57] suggested in 1936 that the body heat loss should be calculated from the variation in its average temperature:

$$-Q_s = -\Delta\bar{T}_b \cdot C_p \cdot m_b \cdot A_D^{-1} \tag{4.10}$$

in which $\Delta\bar{T}_b$ is the variation in the average temperature of the body, C_p its average specific value: 3.47 kJ/kg·°C (Burton[57]), m_b its weight in kg and A_D its area in m². According to these authors, $\Delta\bar{T}_b$ can be found from the rectal temperature and the mean skin temperature by applying to each of them an appropriate weighting factor, the general equation giving $\bar{T}_b$ having the form:

$$\bar{T}_b = xT_{re} + (1 - x)\bar{T}_s . \tag{4.11}$$

Burton[57], using the results of 40 experiments in which the heat loss was determined by direct calorimetry, calculated the value of x by a statistical method. The best value of x is the one which, statistically, gives the best agreement between the loss calculated from the body temperatures and the loss measured absolutely. He thus found the following equation for calculating $\bar{T}_b$:

$$\bar{T}_b = 0.70T_{re} + 0.30\bar{T}_s . \tag{4.12}$$

The author stresses, however, that x is susceptible to fairly wide variation according to the subjects (between 0.60 and 0.75). Hardy and Dubois[136], using the same method on two naked subjects, stretched out, in a neutral environment or close to neutrality, obtained for x a value of 0.80, a value which was also found by Stolwijk and Hardy[271] in a warm environment. When analysing the results obtained on 13 subjects using partitional calorimetry, Colin et al.[81] found $x = 0.66$ at thermal neutrality and $x = 0.79$ in a warm environment. In addition, they showed that x varied linearly as a function of the heat stored when the latter had reached a mean value of 113 kJ/m². In a cold environment, x has been determined on very few occasions. Hardy and Dubois[136] suggested a value of 0.5 for x in the case of subjects in an air environment less than 24°C. During several cold-water immersion experiments, the present author observed that the heat loss calculated from the rectal temperature and the mean skin temperature, applying the factor $x = 0.5$, was much higher than that found during direct measurement. An investigation covering 10 naked subjects who had been involved in 72 immersion experiments at water temperatures of between 33 and 24°C, during which the heat loss had been measured by partitional calorimetry, showed that the factor x applied to the rectal temperature varied in relation to the environment (Boutelier et al.[47]). The general equation showing the variation in the mean body temperature between a neutral environment and a cold environment can therefore be written as follows:

$$\Delta\bar{T}_b = \bar{T}_{b_i} - \bar{T}_{b_f} = [x_0T_{re_i} + (1 - x_0)\bar{T}_{s_i}] - [x'T_{re_f} + (1 - x')\bar{T}_{s_f}] \tag{4.13}$$

in which x_0 is the factor affecting the rectal temperature at thermal neutrality and x' the factor which affects this same temperature in a cold environment. The subscripts i and f denote an initial temperature (i) or a final temperature (f). These two factors were determined statistically and the following equations for calculating $\bar{T}_b$ were obtained:

$$\bar{T}_{b_i} = 0.73T_{re_i} + 0.27\bar{T}_{s_i} \tag{4.14}$$

at thermal neutrality, x varying from 0.62 to 0.76 according to the subjects.

$$\bar{T}_{b_f} = 0.79T_{re_f} + 0.21\bar{T}_{s_f} \tag{4.15}$$

in a cold environment, x' varying from 0.68 to 0.79.

The use of these factors gave a relatively precise calculation of the heat loss, but the latter was overestimated in the case of values less than 110 kJ/m². The relationship between the loss calculated using these factors and the loss measured is as follows:

$$Q_{s\,\text{calculated}} = 0.72Q_{s\,\text{measured}} + 46.54 \text{ in kJ/m}^2 \tag{4.16}$$

($r = 0.72$ for $n = 72$).

It is curious to note that the mean value of the factor affecting the rectal temperature increases in a cold environment, whereas a decrease should have been expected, as suggested by Hardy and Dubois. In actual fact, this is true only for small losses of less than 110 kJ/m² and probably reflects the effect of peripheral vasoconstriction which, to a certain extent, makes it possible to preserve the heat content of the core. The loss thus occurs mainly at the expense of the periphery. For greater losses on the other hand a gradual reduction in the weighting factor for the rectal temperature (Fig.4.6) is observed, the loss occurring at the expense of the core and the periphery. From a practical point of view, to ascertain as exactly as possible the mean value of the heat loss compatible with survival in 100% of cases, taking into account the limits given above for rectal and skin temperatures, the variation in the mean body temperature is calculated from the Equations (4.14) and (4.15), and the loss, from Equation (4.10). The latter is then corrected using the relationship (4.16). For example, for a man weighing 75 kg with an area of 1.90 m², a variation in the mean body temperature of 3.65°C is found, assuming 37°C as the initial rectal temperature and 33°C as the initial mean skin temperature. The maximum loss ($Q_{s\,max}$) is then 630 kJ/m², or 16 kJ/kg. This value is higher than that suggested by Andrae and Hall: 10 kJ/kg, but these authors assume higher rectal or skin temperatures and use non-variable weighting factors in their calculations. The loss thus calculated is obviously an approximation and can vary in high proportions, up or down, according to the initial temperatures of the subjects and their body weight. The survival time is therefore estimated by assuming that from the last 30 minutes of the experiment, if this is sufficiently long (two hours or more) to avoid the period of rapid variation of body temperatures at the beginning of the immersion, the heat loss occurs at a constant rate. The survival time is calculated by means of the following equation:

$$t_{hours} = \frac{Q_{s\,max}\,(J/m^2) - Q'_{s\,end\ of\ experiment}\,(J/m^2)}{S(W/m^2) \times 3600} + t' \qquad (4.17)$$

t′ being the duration of the experiment in hours. Proceeding in this manner, the survival times are underestimated, since the loss does not in fact develop linearly, as is shown in Figure 4.7 taken from our experimental results (Boutelier et al.[48]). It decreases gradually as the body gets cooler, the heat losses being reduced because of the decrease in the temperature difference between the body and the water, and the increased metabolism. For very low water temperatures and/or suits which provide only slight protection, estimating survival time by the method described above may lead to serious errors, since the physiological temperatures and the metabolic rate vary rapidly. An approximately linear development of the rectal temperature after the first 20 or 30 minutes of immersion, together with the metabolic rate can therefore be taken as a basis. The latter is compared with the maximum metabolism values which may be caused by shivering during a given time: 300 W/m² for about 30 minutes (Boutelier et al.[44]); 245 W/m² for 40 to 120 minutes (Adolph and Molnar[2]); 120 W/m² for 8 hours (Beckman and Reeves[26]). Some authors also determined, by analogy with the Molnar nomogram, a period of immersion for which the survival of 50% of the victims can be expected, and a period beyond which only a few would have any chance of survival (fatal threshold). These determinations are based mainly on the presumed development of the rectal temperature below 35°C, at which point adverse physiological effects, such as loss of consciousness, the cessation of shivering (T_{re} = 30 to 31°C), cardiac and respiratory arrest (T_{re} = 27 to 25°C) have been reported. The estimated heat loss corresponding to these thresholds using the method described above is a rough approximation, as pointed out by Smith and Hames[263], in the absence of information about the skin temperature and the weighting factor affecting the rectal temperature.

Other methods, using models, have been put forward for predicting immersion tolerance, when the insulation value of protective suits is known. One of the first attempts of this kind was carried out by Smith and Hames in 1962. These authors constructed a nomogram for estimating survival time from the temperature of the water and the overall insulation of the peripheral body tissues and the suits. They first of all determined the heat losses from the following equation:

$$C = \frac{5.55\,(T_c - T_w)\,A}{I_t}$$

in which 5.55 is a constant for converting insulation to "clo", T_c is the central temperature assumed to be 37°C and T_w, the temperature of the water. By fixing a value for metabolism, they deduced the heat loss, the variation in the mean body temperature and the survival time. In actual fact, and in the view even of the authors, considering the central temperature as constant when calculating the exchanges is an extreme simplification, since this temperature gradually decreases when heat loss occurs. In general, therefore, the heat losses are overestimated, particularly as the value of the insulation of the water does not seem to have been taken into account. Furthermore, the use of a fixed metabolic rate is not realistic, as this rate varies in relation to the skin temperature and the central temperature. When the latter falls below 35°C, the metabolic rate decreases gradually and reaches approximately the basal value at about 30–31°C. This variation in metabolism obviously has an effect on the heat loss, and thus on the survival time. The Smith and Hames nomogram can therefore provide only a very approximate, and probably, underestimated survival time.

A prediction model based on effective thermal insulation, a fixed metabolic rate of 87 W/m², a mean body area and weight of 1.8 m² and 70 kg respectively was also put forward by Hall[127] following several experiments on a copper manikin. This model helps to predict the tolerance times, but not the survival times, for an average subject wearing an anti-immersion suit to reach precise heat loss limits: 209 kJ/m² or 5.4 kJ/kg; 291 kJ/m² or 7.5 kJ/kg and 419 kJ/m² or 10.8 kJ/kg. These loss levels correspond respectively to slight, moderate and severe cooling, to judge from the values of the rectal temperatures indicated by the author: 35.9°C for a loss of 5.4 kJ/kg; 35.6°C for a loss of 7.5 kJ/kg and 34.7°C

for a loss of 10.8 kJ/kg. The same comments can be made about this model as those expressed about the Smith and Hames nomogram with regard to the fixed metabolic rate at a level which is very low in view of the drop in the rectal temperature. It is also felt that the mean skin temperature values quoted by the author: 31.1; 30 and 28.3°C respectively, are too high compared with the rectal temperature levels given above, as illustrated by the experimental results obtained by Boutelier et al.[44] on unclothed subjects. The heat losses are therefore probably overestimated and the tolerance times underestimated. However, this model is of great importance, since it allows for the various factors which can affect clothing insulation: hydrostatic pressure, humidity etc.

These few considerations on models show that the latter must be used with care when determining survival, as their authors do in fact point out. To develop a model which provides a relatively precise prediction of survival time, the present author feels it is essential to take account not only of physical factors, such as the coefficient of heat exchange in water, clothing insulation, humidity, hydrostatic pressure, but also of the morphological and physiological factors. Among the morphological factors can be quoted: the geometry of the body, which can be simplified and reduced to an equivalent cylinder; the body weight and area and the thickness of the skin-fold, which conditions the rate of cooling of the body. The physiological factors include: thermal conductance between the core and the periphery, variation in metabolism in dependence on the mean skin temperature and the rectal temperature, and the relation between the heat losses through the respiratory passages and metabolism. A model based on these principles was recently constructed in the CEV Aerospace Medicine Laboratory (Timbal et al.[285]) following the experimental results obtained on unclothed subjects exposed to cold environments in air and in water. It is possible, using this model, to determine the development of the rectal temperature of unclothed subjects in relation to the ambient temperature (air or water) and the thickness of the skin-fold (Fig.4.8), or the time taken for the rectal temperature of subjects with different skin-fold thicknesses to reach critical values: 35, 30, 25 and 20°C, depending on the temperature of the water (Fig.4.9). The respective development of the rectal temperature and the mean skin temperature is calculated from the difference between the heat lost through the skin and the respiratory passages and the production of heat due to shivering. The latter, which depends on the rectal temperature and the mean skin temperature (Benzinger[29]), is determined by the following experimental relationship (Timbal et al.[284]):

$$M(W/m^2) = 935.46 - 57.77\frac{d\bar{T}_s}{dt} - 5.01(\bar{T}_s - \bar{T}_{s_o}) - 23.79T_{re} \qquad (4.18)$$

in which $dt = 1$ mn and $\bar{T}_{s_o} = 34°C$. Assumptions were also made for the metabolic response due to shivering. It was assumed that the latter could not be maintained for more than 90 minutes when it reached a high level: 5 times the basal metabolism (Adolph and Molnar[2]). It may then begin to decrease even before the rectal temperature has reached a value of less than 35°C. The maximum heat production value was therefore fixed at 1,215 kJ/m². When the rectal temperature drops below 35°C, the metabolism decreases proportionally to the fall in the rectal temperature and reaches its basal value (45 W/m²) when $T_{re} = 30°C$. This temperature corresponds to the cessation of any shivering and to a total loss of consciousness. Below this value, the metabolic rate continues to decrease according to Van t'Hoff's law. In spite of certain imperfections and simplifications, the results obtained with this model, as far as rectal temperature development is concerned, are in good agreement with those found in the literature. They show clearly the diversity of the limits of survival associated with morphological factors, the most important of which is the thickness of the sub-cutaneous fat. Relatively minor modifications in the method of transferring heat from the core to the periphery will shortly make it possible to adapt this model for use in investigating the survival times of a man wearing a protective suit.

2.2 Tests in Cold Air in a Dinghy

The immersion tests have to be supplemented by a study of tolerance and survival in the dinghy with which the aircrews are provided. In these dinghies the victims are exposed to a cold and damp air environment, and possibly a strong wind. The bottom of the dinghy however often contains water, particularly those made to take only one person. The heat exchanges, as has been said, therefore take place in the two environments: air and water. In addition, the complex shape of the dinghy and the position of the subject make it difficult to apply the physical laws of heat exchanges. Most laboratories have therefore adopted the solution of investigating the physiological reactions of the subjects in the dinghies (body temperatures, metabolic rate) in order to deduce the additional protection afforded by the dinghy, and the survival times. The experimental procedure most frequently used is as follows: the subjects, wearing an anti-immersion suit, are subjected to prior immersion for 5 to 10 minutes in a cold bath, and then take their place in the dinghy which is located in a cold air environment. The characteristics of the environment vary according to the authors. In the CEV Aerospace Medicine Laboratory the tests are carried out at an ambient temperature of −10°C with a wind of 20 km/h and the dinghy resting on the floor of the climatic test chamber. In other laboratories (USA, Sweden) the tests are performed in a climatic chamber containing a tank filled with sea water at 0°C. The air environments used are as follows: Sweden (Andrae[9]): $T_{air} = 0°C$, wind – 36 km/h; USA: $T_{air} = 1.7°C$, wind varying from 2.7 to 9.4 km/h (Hall et al.[131]) or $T_{water} = 4°C$, $T_{air} = 4°C$, wind – 18.5 km/h (Pittman et al.[238]). As in the immersion tests, the duration of the experiments is limited by reaching the minimum skin or rectal temperature or when these temperatures and the metabolic rate of the subjects have become stable. Stabilisation, and even a rise in temperatures is indeed frequently found, particularly in well protected dinghies, as can be seen from Figure 4.10 taken from our experimental results (Timbal et al.[281]). Particular attention has to be paid to the temperature of the extremities and of the head, since the amount of heat lost through these areas is considerable, as was proved by the experiments conducted by Hall et al.[127] on copper manikins. Although the cooling of these areas does not actually endanger the lives of the victims,

it can, however, cause serious injury (immersion foot and hand) if they have to remain for a long time in the dinghy and if the temperature of the water is below 10°C (McCance, quoted by Whittingham[299]). Tests on dinghies are generally on a comparative basis, to show what improvements have been made to dinghies or to compare the products of various manufacturers. An excellent method of testing the efficiency of the protection afforded by dinghies is that suggested by Hall et al.[127]. These authors use a very advanced heated manikin, with no protective suit, and study the heat transfer of the manikin when protected only by the dinghy. They compare the results with those obtained in the same environment without a dinghy. The difference in the results of the two tests is used to calculate the insulation provided by the dinghy. Such tests, conducted in different ambient conditions and with a variety of protective suits, can help to develop a tolerance prediction model (Hall[132]), with, however, the previously mentioned restrictions in regard to the fixed metabolic rate.

These laboratory experiments have to be supplemented by tests at sea (Veghte[292, 293]), which bring to light any differences between actual and artificial environments, particularly in regard to the effect of the waves and direct or reflected solar radiation. They also serve to test the qualities of the dinghies from the point of view of stability, resistance to wind, handling and ease of use of the various protective elements. Finally, certain factors other than thermal factors, which set a limit to human tolerance, such as nausea or vomiting, can also be evaluated and provide corrections for the estimates of tolerance times based solely on body cooling.

3. Comfort during Normal Use

To be complete, tests on anti-immersion suits must include a study of comfort during normal use in flight. This study covers three main points: ease of putting on and taking off the suit, freedom of movement of the arms, legs and trunk, and thermal comfort. The first point is judged by the speed with which a subject can put on and take off the suit without assistance from another person. This implies in particular that the watertight fasteners are correctly positioned so that they can be easily manipulated by the subject himself (Fig.4.11). This test is very important for aircrews on operational alert.

Freedom of movement is checked with the subject standing and sitting, in the laboratory and in flight. The suit must not, indeed, in any way hinder the operations required for flying the aircraft or for ejecting. For this reason the suit is relatively close-fitting and often has pleats to give plenty of room over the back and the joints, mainly those of the knees. It should, however, be noted that the more close-fitting a suit, the more it loses its efficiency from the point of view of thermal protection, because of the reduction in the radii of curvature, as illustrated in Figure 4.12 taken from our experimental results (Timbal et al.[281]). A compromise must therefore be made between the idea of comfort and the thermal protection requirements.

Thermal comfort considerations deserve special attention. Indeed, since anti-immersion suits are intended to protect against cold, they necessarily involve some limitation of the heat exchanges between the body and the environment. When worn in flight they therefore constitute a constraint, the limits of which must be evaluated. These limits are determined by the occurrence of sweating which reduces the effectiveness of the thermal protection (Hall[129, 130]). In our Aerospace Medicine Laboratory this study is conducted in a climatic test chamber at ambient temperatures of 20, 25 and 30°C; the temperature of the walls is the same as that of the air and the wind speed is 0.4 m/s. Each test lasts one hour, the subject remaining seated and doing no work. His skin and rectal temperatures are recorded continuously and the mean skin temperature is automatically calculated every minute. The beginning of sweating is indicated by a slight fall in the skin temperature. Weighing the subject before and after the experiment will show the amount of sweat secreted, and thus make it possible to state precisely the extent of the thermal stresses imposed. In addition, weighing the suit before and after the experiment will enable the quantity of sweat absorbed by the suit and the decrease in its insulation to be evaluated. The limit of thermal confort is determined by the ambient temperature at which the period of initiation of sweating is less than one hour (generally, if $\overline{T}_s = 34.8$ to 35°C). The environments that can be tolerated are obviously higher, although this range of ambient temperatures must be avoided because of its effect in reducing the insulation of the suit.

DESIGN AND DEVELOPMENT OF ANTI-IMMERSION EQUIPMENT

This equipment, as we have seen, is made up of two elements each of which will be considered in turn: the anti-immersion suit, on the one hand, and the equipment for keeping the victim on the surface of the water, i.e. the life jacket, and the individual or collective dinghy on the other.

1. The Anti-Immersion Suit

This item of equipment must meet the following three requirements:

- provide effective thermal protection in water and in air;
- be comfortable to use in flight;
- be neither too large nor too heavy, since weight and volume are limiting factors on board military aircraft.

To fulfil these requirements, two alternative general principles have been adopted when designing the suits: either to use a material which is not watertight but which retains suitable insulating properties in water, these being known as "wet suits"; or to provide complete watertightness and insulation with suits known as "dry suits".

The *wet suits* were developed at the end of the Second World War with the increasing widespread use of neoprene foams and, as early as 1944, Spealman[266] was recommending the use of foam neoprene boots to ward off the risk of "immersion foot". This type of clothing was developed primarily for divers following the work by Bradner in 1951 on the insulation of clothing made of unicellular foam neoprene. However, in view of the difficulties encountered in developing clothing which was watertight, insulating and resistant, experiments were carried out in the USA to evaluate the thermal protection afforded by suits made of 0.48 cm and 0.64 cm thick unicellular neoprene (Beckman et al.[25]). The results obtained showed that a subject wearing a suit of this type having a thickness of 0.48 cm could tolerate immersion for 4 hours in water at 10°C. The tolerance time fell to 2 hours in water at 4.4°C and to 1.5–1.3 hours in salt water at −2°C. In all cases, the restriction on the duration of the immersion was due to the pain experienced in the extremities as a result of the cold. The authors came to the conclusion that adequate insulation of the subjects would have required the use of 2.54 cm thick neoprene. But the suit would then have become heavy and would have lost its flexibility, leading to severe restriction of movement. Further, in normal flight conditions, it would have imposed a considerable thermal load on the subject, since its insulation in air would have been about 6 clo. To overcome this disadvantage and keep a relatively small thickness for the suit, the authors suggested that the suit should be provided with an additional source of heat, but this complicated it and made it considerably heavier. The basic principle of a wet suit was therefore quickly abandoned.

"*Dry suits*" were also being designed as early as the end of the Second World War and resulted in a number of developments. The suits consisted of two garments: an over-suit which was watertight but had no insulating properties, and an under-suit which provided good insulation. To keep pilots cool in flight, it was necessary to wear a ventilated garment under the under-suit, which was incorporated in the latter. This made the whole equipment very thick, since the compressible under-suit had to retain a certain insulating power during immersion, in spite of the effect of hydrostatic pressure. Equipments of this type included the Mark V A suit in use in the US Navy between about 1960 and 1967. This suit consists of a very thick insulating and ventilated suit (Fig.4.13) and a watertight suit made of neoprene impregnated nylon to which are bonded fine rubber socks. Tightness at the neck and wrists is provided by a neoprene impregnated flexible material which fits tightly around the skin in these areas. Watertight anti-immersion mitts, a waterproof hood and a life jacket complete the equipment (Fig.4.14). Tests on this equipment (Colin et al.[78]) showed that the subjects could tolerate immersion up to the neck for at least 1.5 to 2 hours in water at 4°C, providing the suit was absolutely watertight. In air, the effectiveness of the protection afforded is remarkable, since the equipment enables subjects to remain for several hours in an environment at −30°C with a 15 km/h wind, doing moderate work. In spite of its very real insulating qualities, this suit had a number of disadvantages, for example, insufficient protection for the feet, sealing faults in the connection couplings in the ventilated suit, and it was very big. At the same period the EFA-11 (Fig.4.15) was being developed in France. This was a relatively watertight foam neoprene suit for wear over a ventilated garment. It combined the watertightness and good thermal insulation properties of neoprene and enabled wearers to tolerate immersions of 2.5 hours in water at 5°C without difficulty (Colin et al.[76]).

These two types of suits obviously required the installation in the aircraft of relatively large additional apparatus for ventilation within the suit. They could therefore be worn only in single seater or twin seater aircraft, which left the problem of protecting a larger aircrew, such as that in bomber or transport aircraft, still unsolved. As a result, while maintaining the principle of the dry suit, manufacturers tried to develop a suit which let in air but was watertight. This attempt led to the development of a cotton base material with a relatively tight weft. This material allows the air to pass through and facilitates evaporation of the sweat. In normal flying conditions in a temperate climate, it does not therefore require the use of a ventilated suit. In addition, contact with the water makes the cotton fibres swell and it thus, in theory, becomes completely impermeable to water. The majority of the anti-immersion suits in use at present are designed on this principle. This is the case in particular of the Mark 10 Beaufort suit and its derivatives, used in several countries, and of the Aerazur 842 and 847 suits which are used mainly in France. The Mark 10 suit (Figures 4.15 and 4.16) is a double walled suit made of "ventile" material which has a cotton base. The two walls are thicker below the waist to make them more resistant to possible damage and also to avoid slight oozing of the water through the fibres under the effect of hydrostatic pressure. The feet are protected by neat, watertight slippers which are bonded to the suit. As a protection from the cold, the user can wear one or two pairs of woollen socks under these slippers. The wristbands and collar are lined at the edges with treated latex, to avoid skin irritation. A watertight zip fastener at the top of the suit goes from the right shoulder to the left hip. A transverse gusset in the back facilitates donning the suit and any extensive bending movements of the trunk. This gusset is normally sealed by a zip fastener. The suit can also have a light hood made of polyurethane coated material and mitts. Various modifications have been made by users in the countries in which this suit has been marketed. For example, in Sweden (Andrae[9]), the fm 66 suit has three layers of material instead of two and the zip fastener goes from the left shoulder to the right shoulder, passing behind the neck, and then almost straight down to the right groin. It also includes a flexible cowl attached to the suit. In Canada, tests have been carried out with a small neoprene collar, as a lining for the latex collar. In France, the firm of Angevinière has developed the SIA 10-2 suit (Figures 4.18 and 4.19) which differs from the Mark 10 by having a semi-circular zip which seals the top of the suit and passes behind the neck. The suit is easier to put on with the zip in this position and the front panel can be pulled down, if there is a long stay on the ground, thus improving the wearer's comfort. In addition, a double strip of flexible material held in position by two zip fasteners at the sides protects the latex collar and prevents possible tearing during ejection. These suits are intended to be worn with the insulating under-suit Mark 2

(Fig.4.20). This is made of a fur-like raised nap material, which is flexible and very effective from the point of view of keeping out the cold. A number of immersion tests have been carried out on this suit (Andrae[9], Timbal et al.[281, 282]). The results can be summarised as follows: in water at 0°C, with an air environment at 0°C and a 10 m/s wind, the limit of survival in 100% of cases will be reached in 1.7 h (Andrae). In water at 4°C with no wind, voluntary tolerance close to the survival limit is of the order of 4 to 5 hours. After 90 minutes immersion, the mean skin temperature is still 26.4°C, the lowest temperature being that of the foot: 16.5°C. In water at 6°C, voluntary tolerance is about 8 hours, and in water at 9°C, it should be more than 20 hours. If, however, the Mark 10 suit is worn without the Mark 2 under-suit, over a simple flying suit, voluntary tolerance is reduced to about 4 hours in water at 13°C. The wearing of the watertight suit without an under-suit should therefore be prohibited when the water temperature is less than 15°C. The buoyancy provided by this suit is good and the victim, with his life jacket, assumes an almost horizontal position, which reduces the effect of the hydrostatic pressure on the insulation. From the point of view of thermal comfort, the complete equipment allows moderate activity without sweating in a 20 – 22°C environment, if there is not much solar radiation.

The ARZ 842 suit (Fig.4.21) has been in use for the last ten years or so, mainly in France. It, too, is made of a cotton material which will admit air and keep out water, but is thicker, fuller and less flexible than the previous suits. A tight circular fastener, which extends fairly low over the chest, makes for ease of putting on. This suit has neoprene boots, giving effective thermal protection for the feet. It is intended to be worn over a raised nap under-suit which is not very thick (the ARZ 14 (Fig.4.22)) to which slippers are attached. Its thermal insulation is low, compared with the Mark 2 under-suit, but is sufficient, however, to ensure survival for about six hours in water at 6°C (Boutelier et al.[48]). In spite of its good thermal insulation, the lack of flexibility of this suit and its size have led the manufacturer to design a new model quite recently, which is more flexible and more close-fitting: the ARZ 847 (Fig.4.23). This suit has characteristics comparable to those of the Mark 10 and the SIA 10-2 and differs from them externally only by the position of the zip fastener (which makes it possible to let the top of the suit fall backwards (Fig.4.24)), two side gussets and neoprene boots which are bonded to the legs of the trousers. Providing less insulation than the ARZ 842, it affords, together with the Mark 2 under-suit, a degree of protection which is close to that given by the Mark 10, and is distinctly more comfortable to wear. For water at a temperature of less than 10°C, this suit must therefore be used with an under-suit which gives greater insulation than the ARZ 14. Its excellent buoyancy is comparable with that of the Mark 10.

2. Equipment to Keep the Victim Floating

2.1 Life Jacket

The purpose of a life jacket is to keep the head of the victim out of the water and to prevent any rotation of the body in the water if the subject is unconscious. The required characteristics of a life jacket have been stated by Scardino[254]. It must have a lift of 17.5 kg, allow free movement, come to the surface and turn an unconscious man face upwards within 5 seconds of entering the water after a drop of three metres; it must maintain the body of the victim in an inclined position at an angle of inclination of between 30 and 60°, and finally it must not burn and must be impermeable to oil derivatives. For aviation use, other requirements have to be specified: the life jacket must not interfere with the cords of the ejector seat or parachutes or hinder the extraction of the latter. Furthermore, it must accommodate a number of accessories essential for survival and for signalling. Taking all these constraints into account, the development of a good life jacket is thus a fairly complex operation. The jackets used in the aviation field generally consist of a two compartment bladder, forming a collar with a wide opening so that it can be used over all types of suits. This collar keeps the head in the correct position and at the same time protects the neck from too rapid cooling. The bladder comes down as far as the base of the chest, forming two separate pockets. The latter have different shapes according to the type of life jacket: either approximately cylindrical, or becoming wider towards the base of the chest. In this area the pockets must also be relatively free in order to give the body some stability and prevent it from turning over. The bladder is attached to a base which has several pockets containing the signalling kit. It has a capacity of 9 to 20 litres and is inflated by means of one or two carbon dioxide capsules. There is also a tube, which is sealed by a non-return valve, for completing the inflation of the bladder by mouth, if necessary.

2.2 Dinghies

These are of two types: individual dinghies used on fighter aircraft, and collective dinghies which can take from 3 to 30 people. The individual dinghies are very small. When inflated they are about 1.6 m long and from 0.8 to 0.9 m wide. These dinghies generally consist of a pneumatic part in the shape of a polygon which is longer towards the front end. It is larger at the back of the dinghy which has to take the greater part of the weight of the victim. It is inflated by means of carbon dioxide, the capacity of the cylinder varying according to the model. Inflation can be carried out correctly, even at very low temperatures. During evacuation by parachute, the dinghy hangs underneath the victim, to whom it is attached by means of a strap hooked on to the life jacket, and is inflated automatically during descent or manually by pulling on the inflation control cable. The dinghy also has a bottom part which can be inflated by mouth, and a covering consisting of a waterproof front panel and a hood in the light model (Fig.4.25), or a covering which can be inflated by mouth (Fig.4.26). This provides the pilot with excellent protection against cold and wind, a fact which is especially necessary as the current anti-immersion suits are permeable to air. Their insulation is therefore reduced by the wind. Two handrails in the form of straps fixed to each side of the dinghy help the victim to climb into it, which he does from the front. The dinghy is stabilised by means of two air pockets bonded to the bottom. A floating anchor made of cloth is hooked on to the dinghy to reduce drift. These dinghies provide very considerable thermal

protection, as has been shown by the numerous tests carried out in the laboratory and at sea. For example, for an ambient temperature of −10°C and a 12 m/s wind, the external temperature of the walls of the covering is −6 to −9°C and that of the internal walls from −2 to −5°C for the lighter model. This enables the victim, wearing the anti-immersion suit, a warm under-suit and gloves, to keep his skin temperature around 24°C. In the same ambient conditions, the dinghy with the inflatable bottom and covering has an internal wall temperature of between +2 and +14°C according to the areas concerned, and the victim, wearing the same equipment as before, can keep his mean skin temperature at about 29°C. In both cases, however, the hands require protection by mitts.

The collective dinghies are designed on the same principle as the individual dinghies. They have an inflatable bottom and a waterproof roof held in position by inflatable masts. They are equipped with a very comprehensive survival kit. In addition, for very cold climates, various improvements in thermal protection have been made by using a wall which reflects infrared radiation inside the dinghy, and an additional propane heating device (Hall et al.[131]).

This analysis of thermal protection against accidental immersion has shown that the present achievements both in anti-immersion suits and dinghies give aircrews quite a long period of survival, even in a very cold environment. It has underlined the difficulty of reconciling good thermal protection with the requirements associated with flying, particularly in high-performance aircraft, and the resulting interference with the comfort of the crew. This has prompted those in command to specify the conditions in which anti-immersion suits are to be used. An analysis of the papers given at the 70th Meeting of the AFFSC (E) in 1973 shows that the regulations concerning the wearing of anti-immersion suits vary from country to country. In the Federal Republic of Germany, for example, the wearing of the suit is compulsory if the temperature of the water is less than 15°C or, whatever the temperature of the water, if crews are making night flights over the sea. In France, the anti-immersion suit must be worn from the 15th October to the 15th May, if the temperature of the water is less than 12°C. For the Danish and Norwegian Air Forces, and also the RAF, wearing of the suit is compulsory if the water temperature is less than 10°C, but this limit is 15°C for the Royal Navy. In Canada the regulations are more precise. The anti-immersion suit must be worn the whole time North of 60° latitude North, from the 1st September to the 1st May from 48° to 60° latitude North and from the 1st October to the 1st April South of 48° latitude. In addition, wear of the suit is compulsory at all latitudes if the water temperature is less than 13°C or if the sum of the air and water temperatures is less than 43°C. Some of these regulations appear particularly optimistic from the point of view of tolerance to immersion. In particular, it would be desirable to have common regulations for all countries living at the same latitudes and for the wearing of the anti-immersion suit to be compulsory whenever the water temperature is 15°C.

Finally, it should be emphasised that the estimates of survival times have not taken account of psychological factors. Now, this is extremely important and may considerably reduce the defence capabilities of the organism. To lessen the psychological impact of accidental immersion as far as possible, it is essential for crews to undergo hard training in survival at sea, so that they can make the best possible use of the equipment at their disposal. Such training should also be repeated fairly frequently. In the USA (Lafitte[191]) sea survival training includes both theoretical courses and practical instruction during which aircrewmen carry out training in descent into the water, parasailing, being dragged on their back or their stomach to simulate the dragging of the parachute under the effect of the wind when landing on the sea, and getting into the dinghies and remaining there for two to three hours. This type of training is tending to become general practice in Air Forces in all countries. In some of them, which have to carry out missions in very cold regions, the training is more rigorous and the period of exposure in the dinghies may last from 16 to 18 hours (Blake[34]). For helicopter crews, training also includes evacuation exercises. Indeed when this type of aircraft falls into the sea it sinks rapidly and turns over under the weight of the rotor. The crew members must then wait, strapped to their seats, until the machine is completely filled with water, before they can evacuate it. Throughout this period they must on no account inflate their life jackets, as they would then remain imprisoned in the cockpit. This wait, however short, is a very testing period and may lead to panic, so that it is particularly beneficial for crews to have had prior training.

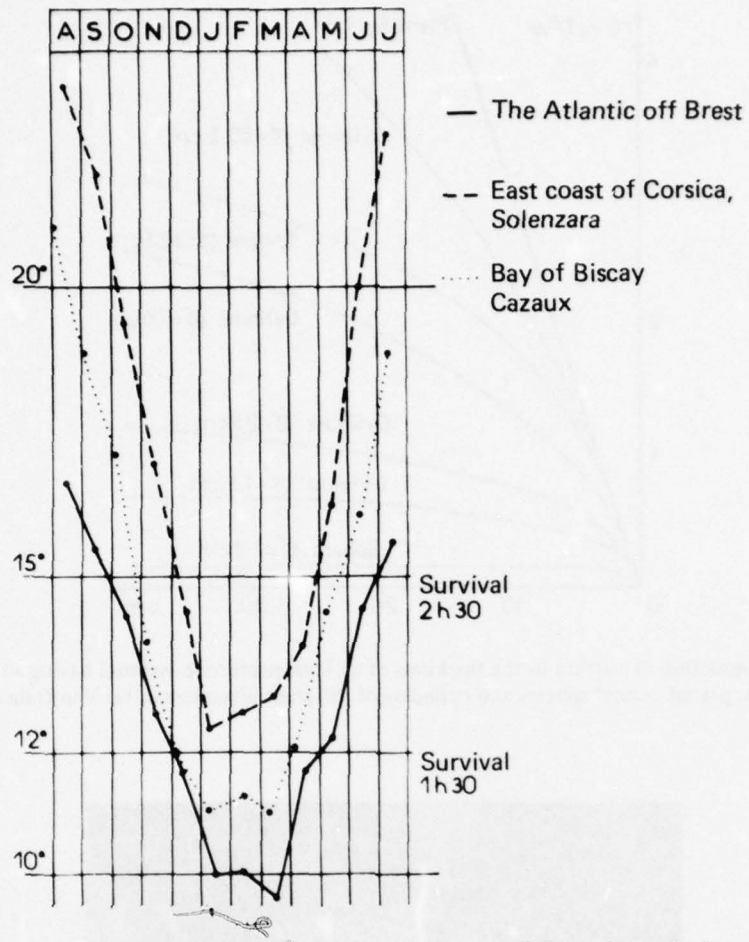

Fig.4.1 Development of the water temperature in the Atlantic Ocean (off Brest and in the Bay of Biscay) and in the Western Mediterranean, according to the month of the year

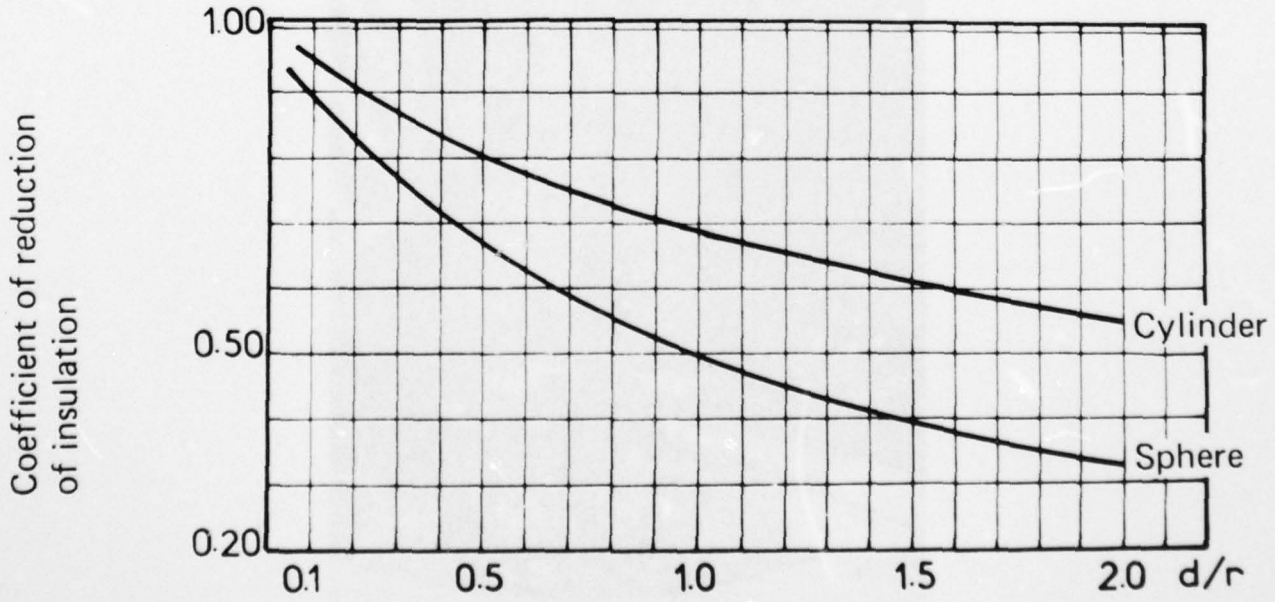

Fig.4.2 Effect of the radius of curvature on the reduction of insulation in the case of the cylinder and the sphere (d is the thickness of the suit and r the radius of curvature)

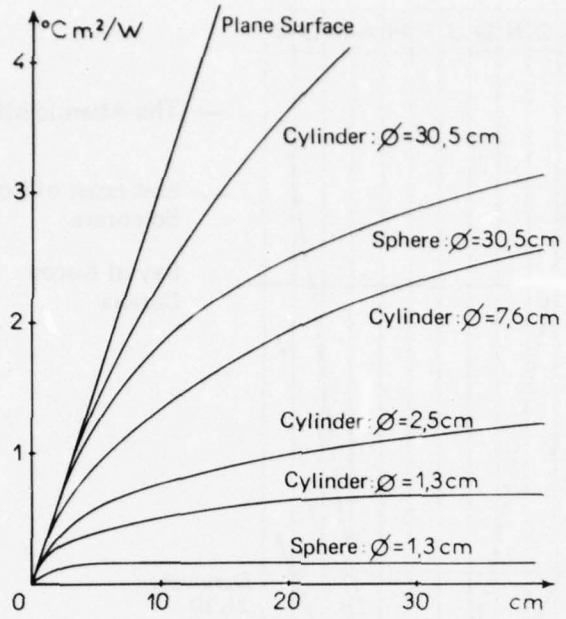

Fig.4.3 Variation in insulation in relation to the thickness of an ideal protective material having an insulation of 1.85 clo for 1 cm thickness, placed around spheres and cylinders of different diameters (after Van Dilla and Siple, 1949)

Fig.4.4 Test in water of the SIA 10.2 anti-immersion suit, showing the patchwork pattern on the lower limbs and the abdomen

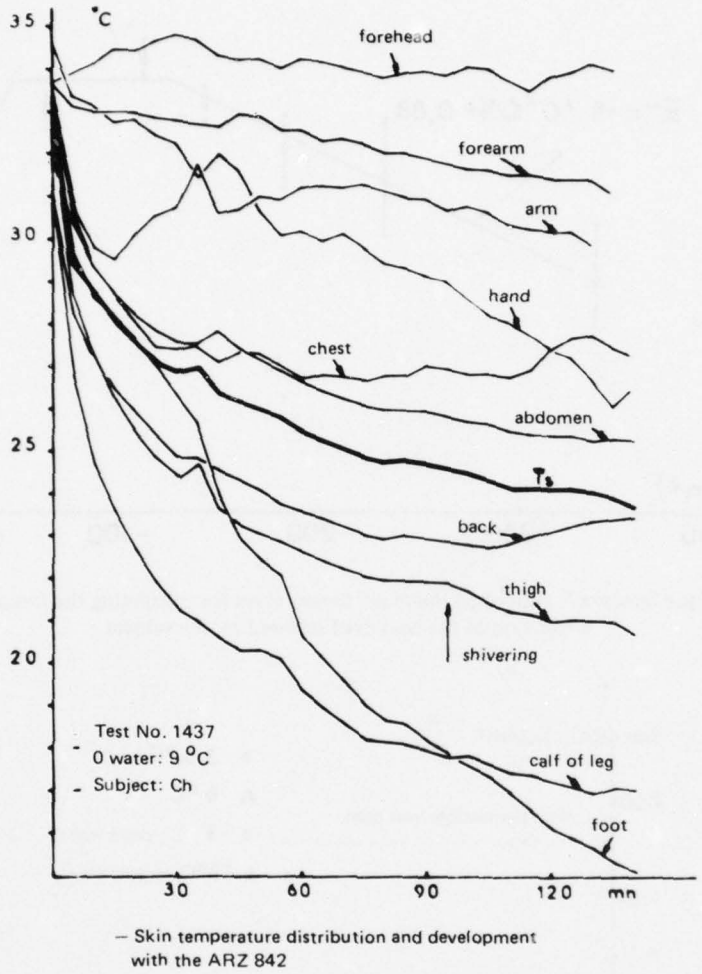

Fig.4.5 Development of local skin temperatures and the mean skin temperature of a motionless subject wearing the ARZ 14 under-suit and the ARZ 842 anti-immersion suit, immersed in water at 9°C

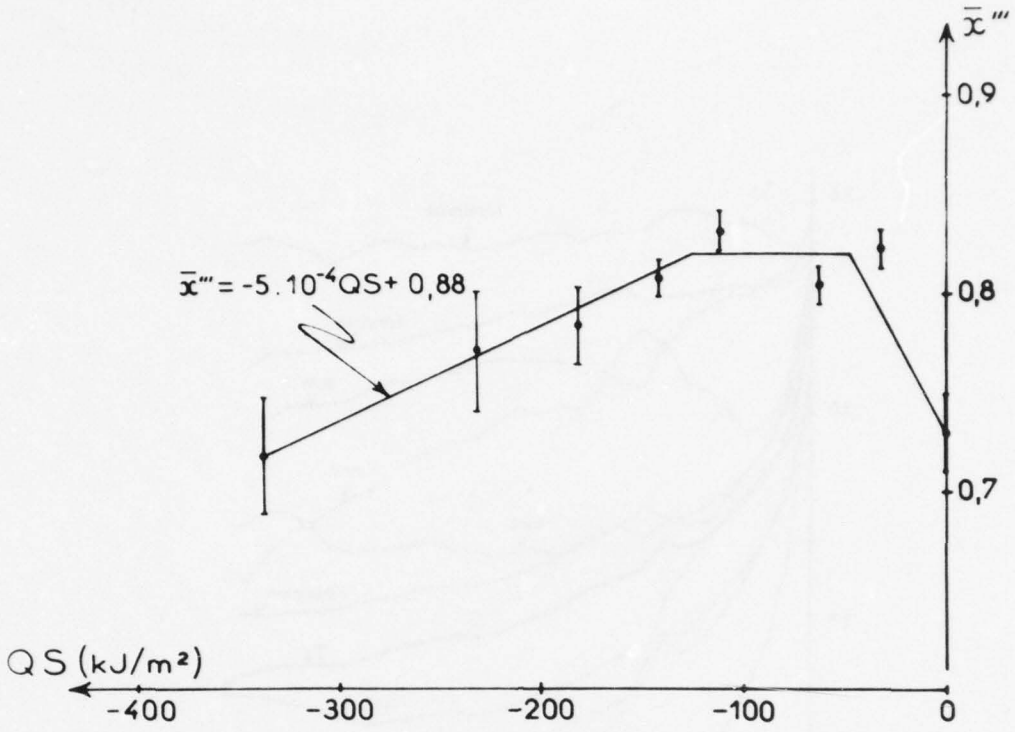

Fig.4.6 Variation of the factor x''' applied to the rectal temperature for calculating the mean body temperature as a function of the heat debt suffered by the subject

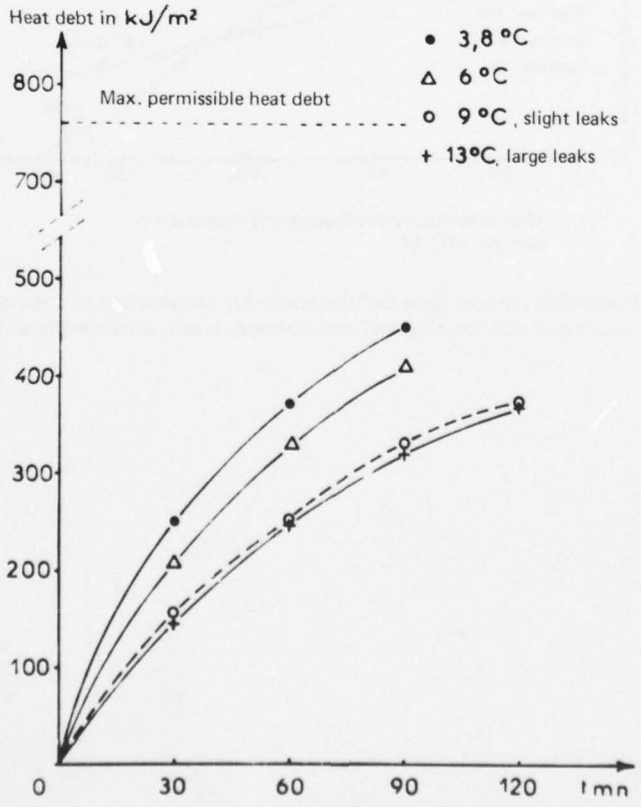

Fig.4.7 Development of the heat debt suffered by a subject wearing the ARZ 842 anti-immersion suit at various water temperatures. Note the extent of penetration of water into the suit compared with the reduction in insulation and the constitution of the heat debt

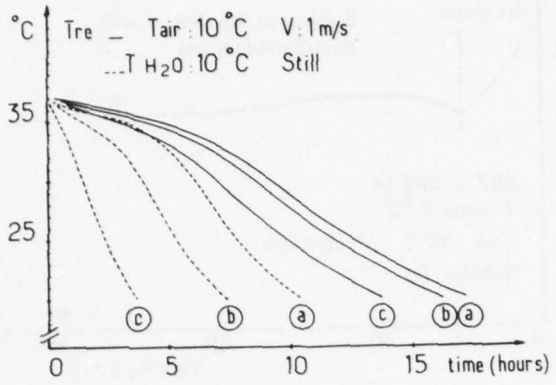

Fig.4.9 Estimated time taken for the rectal temperature of subjects with skin-folds of different thicknesses to reach 35, 30, 25 or 20°C, as a function of the water temperature (after Timbal et al.[285])

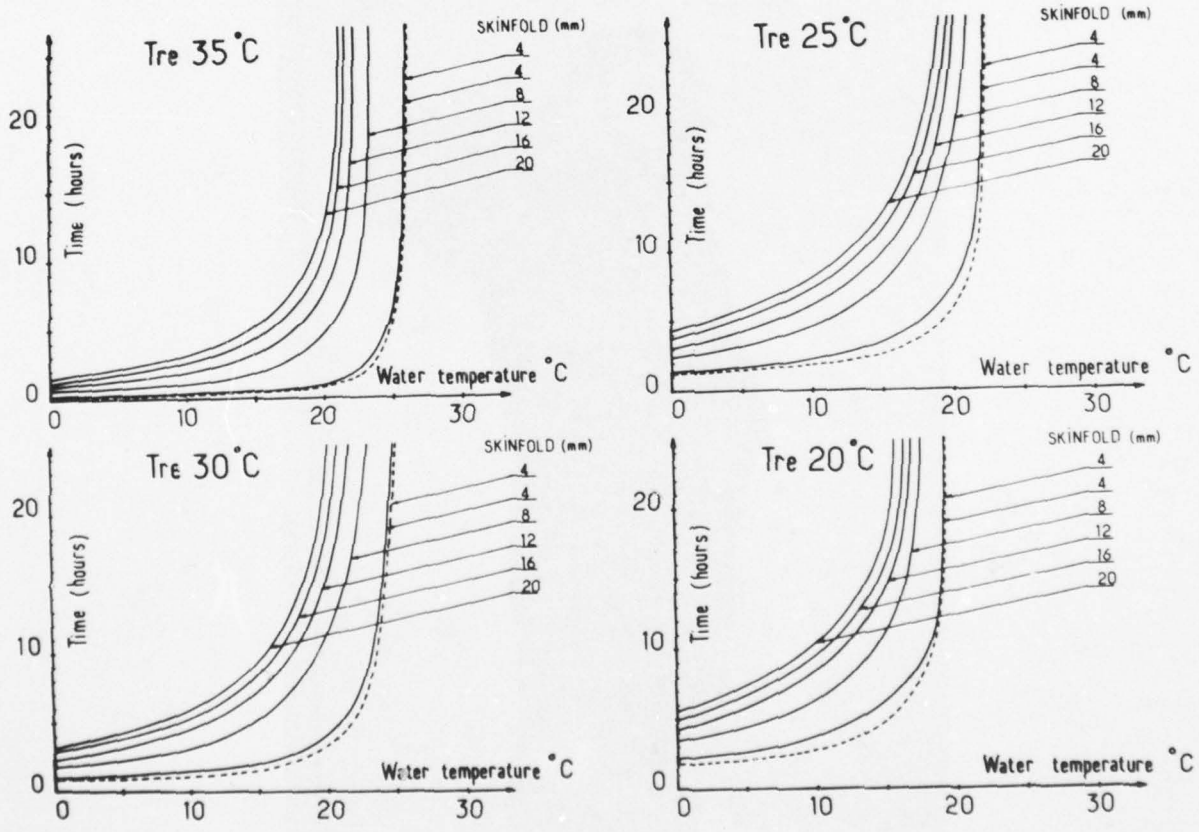

Fig.4.8 Presumed rectal temperature development of naked subjects: (a) fat, with skin-fold: 20 mm; (b) average, with skin-fold: 10 mm; or (c) thin, with skin-fold: 4 mm, in air at 10°C, and in water at the same temperature (after Timbal et al.[285])

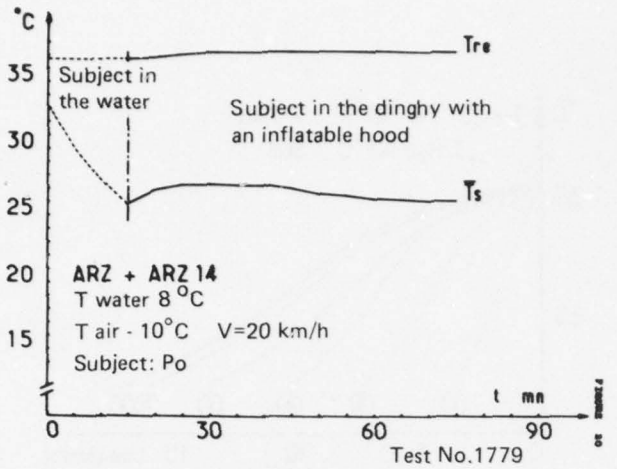

Fig.4.10 Development of rectal temperature and mean skin temperature of a subject wearing the ARZ 14 under-suit and an ARZ 847 anti-immersion suit, immersed in water at 8°C for 15 minutes, and then sitting in a dinghy with an inflatable hood in an air environment of −10°C, with a 20 km/h wind

Fig.4.11 Subject wearing the SIA 10.2 suit. Note the position of the zip fastener around his neck, enabling the subject to put it on without assistance

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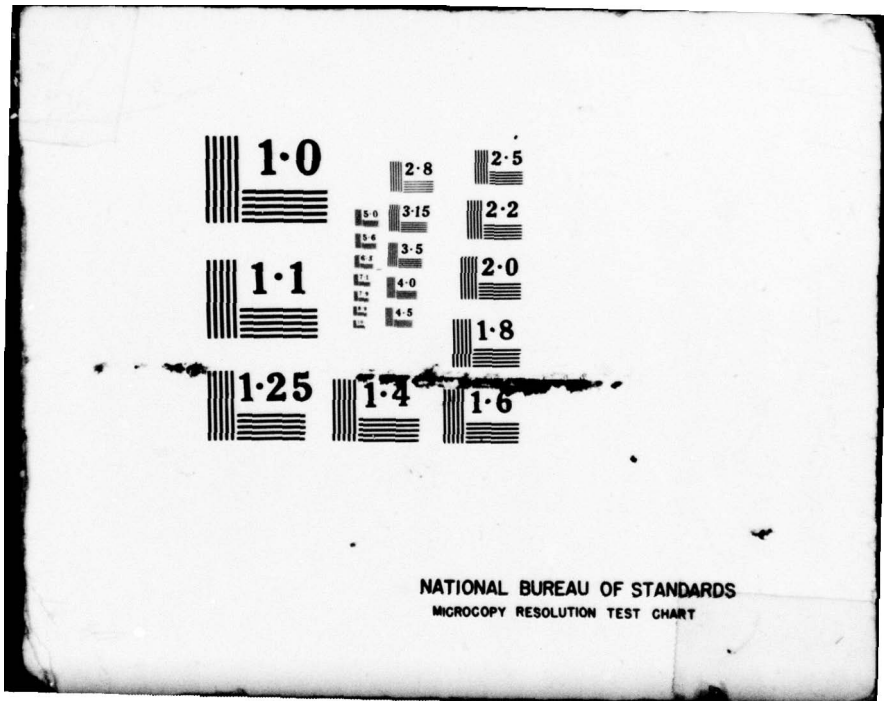
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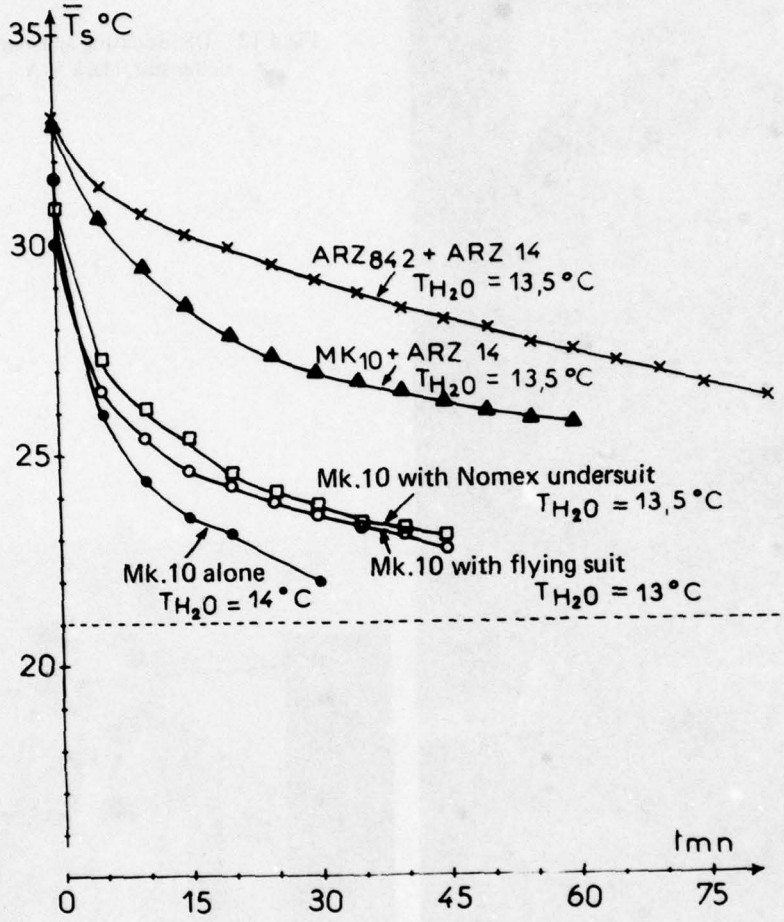

Fig.4.12 Mean skin temperature development of a subject wearing either the ARZ 842, relatively large, or the Mark 10, which is much more close-fitting, with the same ARZ 14 under-suit. This Figure provides a good illustration of the effect of the reduction in radius of curvature of the body

Fig.4.13 US insulating and ventilated under-suit, Mark V A

Fig.4.14 US anti-immersion suit, Mark V A

Fig.4.15 EFA 11 foam neoprene anti-immersion suit

Fig.4.16 Mark 10 Beaufort anti-immersion suit

Fig.4.17 Mark 10 Beaufort anti-immersion suit (back view) showing the gusset for ease of sitting down

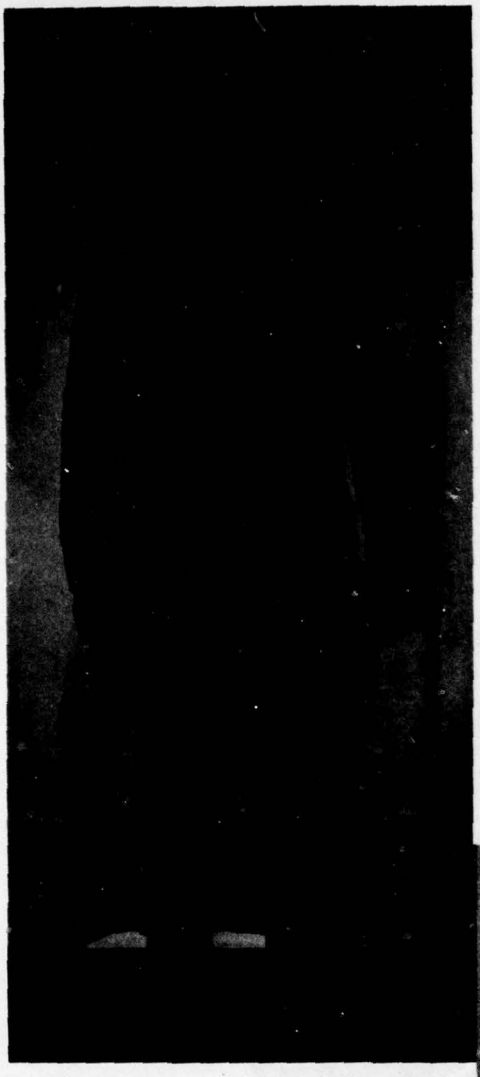

Fig.4.18 SIA 10.2 anti-immersion suit

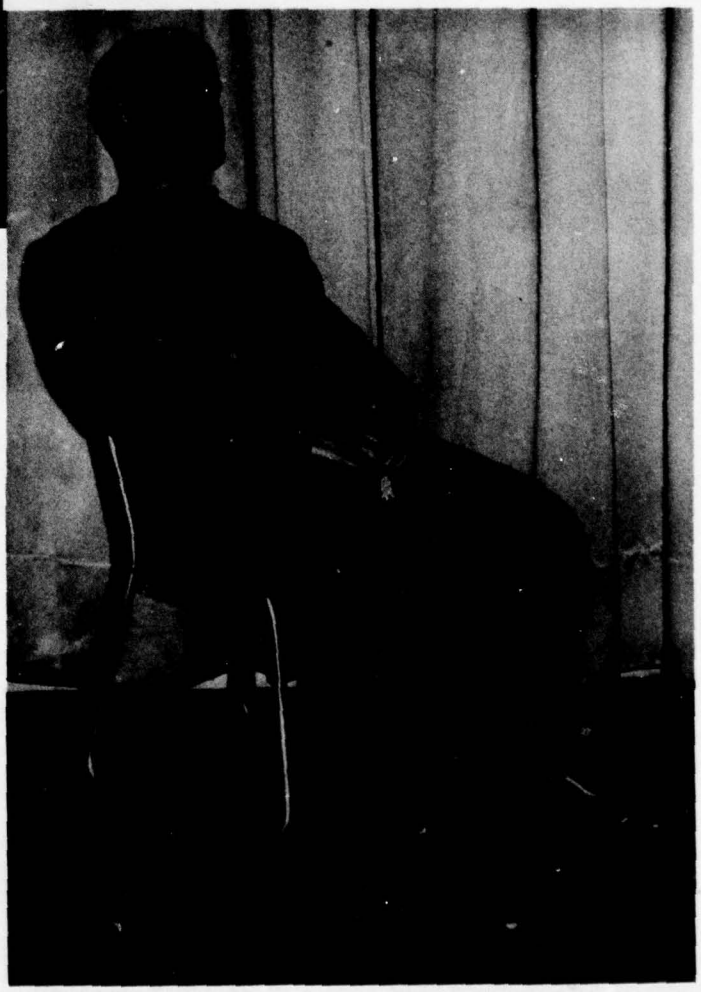

Fig.4.19 SIA 10.2 anti-immersion suit with the top of the suit dropped forward for the pilot's comfort while on ground alert

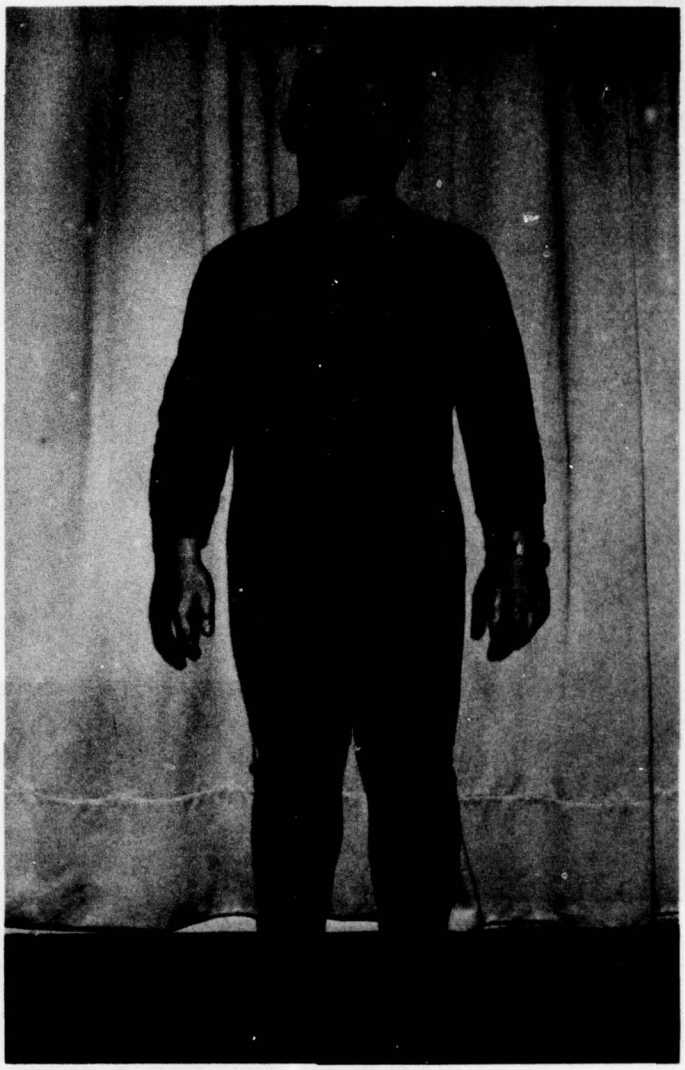

Fig.4.20 Mark 2 insulating under-suit to be worn under the Mark 10 and SIA 10.2 suits

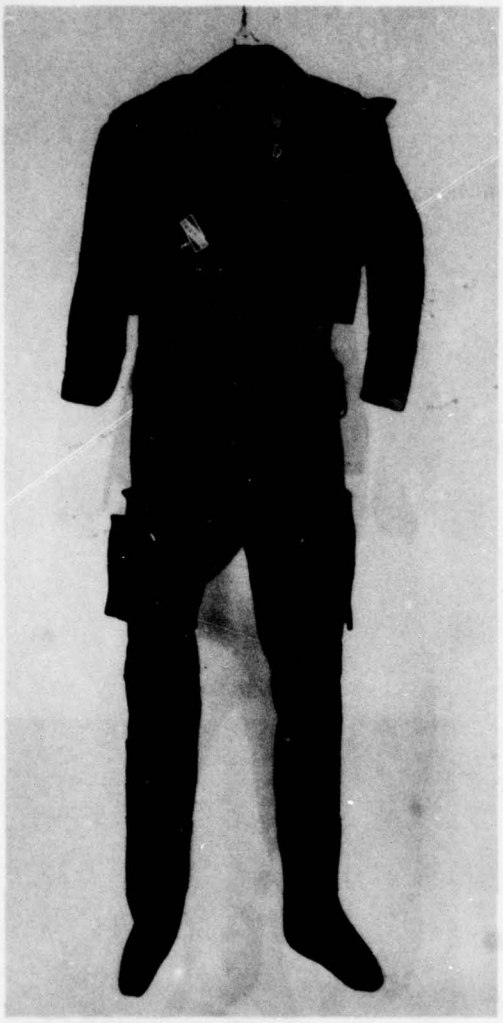

Fig.4.21 ARZ 842 anti-immersion suit. Note the neoprene slippers which are very effective in cold water

Fig.4.22 ARZ 14 under-suit to be worn under the ARZ 842 or 847 suits

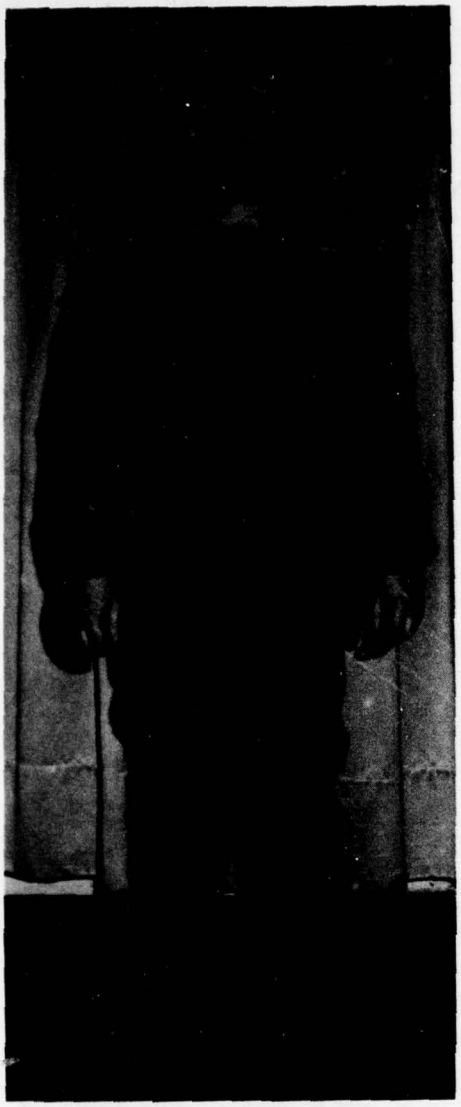

Fig.4.23 Front view of ARZ 847 suit

Fig.4.24 ARZ 847 suit with the top of the suit dropped backwards for the pilot's comfort while on ground alert

Fig.4.25 Individual dinghy, lightweight model, hood and front panel not inflatable

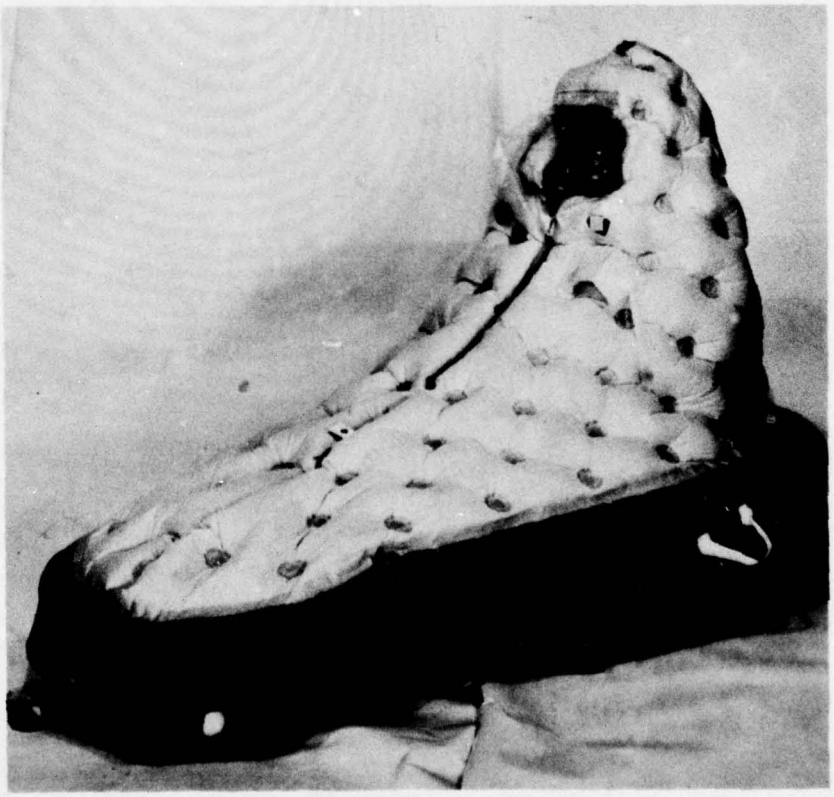

Fig.4.26 Individual dinghy with inflatable bottom and hood

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14. Abstract

The survival of aircrews in the case of accidental cold water immersion is limited by the extent of thermal losses. In this AGARDograph, the physical laws governing thermal exchanges in both air and water are described. The state-of-the-art in the fields of physiological reactions, tolerance, acclimatization, cold induced accidents and their treatment is reviewed. Finally, the major items of protective equipment used in aeronautics and the methods applied to test their effectiveness are described.

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