

Editorial Review

The initial responses to cold-water immersion in man

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INTRODUCTION

The British Isles contain and are surrounded by cold water: the surface temperatures around the coast vary between 5 and 9°C in February and 13 and 17°C in August [1]. The possibility of accidental immersion in cold water is an ever-present risk, which results in the death of between 400 and 1000 people each year [2, 3]. Immersion deaths represent the fourth most common cause of accidental death in the adult population and the third most common cause in children [3].

In recent years the threat associated with accidental immersion in cold water has tended to be thought of in terms of hypothermia, or, a fall in the core temperature of victims. This, however, is at variance with the finding that approximately 60% of the annual open-water immersion deaths occur within 3 m of a safe refuge, and two-thirds of those who die are regarded as 'good' swimmers [2]. Such statistics do not suggest protracted immersion and core cooling as the primary cause of death; instead other, more rapid responses and mechanisms are likely to be responsible.

Golden & Hervey [4] have outlined four stages of cold immersion associated with particular risk. These are: (1) initial responses to immersion (0–3 min), (2) short-term immersion (3–15 min), (3) long-term immersion (> 30 min) and (4) post immersion. Normally, the possibility of hypothermia does not occur until stage (3) (30 min); before this, the immersed individual must survive the initial responses to immersion. These can be broadly divided into two different groups which have been given the generic titles of the 'diving response' and the 'cold-shock response'. Since the first comprehensive study of diving vertebrates was published by Bert in 1870 [5], there have been many other excellent papers on this topic [6–9]; the present review will concentrate, therefore, on the cold-shock response to immersion. This includes cardiovascular, ventilatory and metabolic components, and, unlike many other 'reflex' responses, does not appear to serve any beneficial purpose. Instead, it can represent

the first, and possibly the greatest, threat to the lives of individuals immersed in cold water.

THE 'COLD-SHOCK' RESPONSE

Cardiovascular responses

Several authors have reported that healthy subjects demonstrate a tachycardia within 2–3 s of exposure to cold water [10–17]. Cooper *et al.* [12] reported that naked subjects immersed in water at 10°C responded with an average increase in heart rate of approximately 20 beats/min greater than that seen on immersion in water at 27°C, whereas Hayward & Eckerson [15] recorded a mean increase in heart rate of 49% (87–129 beats/min) over pre-immersion levels after the immersion of lightly clothed females in water at 0°C. The corresponding value for males was 42% (81–115 beats/min), and both increases were greater than those seen on immersion in warm water.

Exposure to cold water has also been reported to increase cardiac output [11, 18]: after 1 min of ice-water showering of the chest, Keatinge *et al.* [11] measured increases in the cardiac output of two subjects of 59% (7.9–12.4 l/min) and 100% (6.5–13.0 l/min) over pre-exposure levels.

In addition to the cardiac effects, exposure to water temperatures below approximately 33°C can induce significant peripheral vasoconstriction and consequent reductions in peripheral blood flow [19, 20]. As a result of the cardiac and vascular responses to cold-water exposure, arterial and venous pressures are increased [21], with arterial pressure increasing within 2–3 s of exposure to cold water and reaching maximal levels within 30 s [11]. The mean systolic and diastolic arterial pressures of subjects have been reported to increase from a resting level of 130/76 mmHg (17.3/10.1 kPa) to 175/93 mmHg (23.3/12.4 kPa) after 1 min of ice-water showering [11].

These early cardiovascular responses to immersion in cold water can place a significant strain on the system: a rapid increase in blood pressure and cardiac output increases the work required of the heart, the left ventricle

in particular. This can result in greater ventricular irritability, cardiac irregularities and, on rare occasions, precipitate ventricular fibrillation [10, 22], especially when the fibrillation threshold is lowered due to excitation of the ventricular myocardium [11, 23]. Both ventricular and atrial ectopic beats, sometimes multifocal in origin, have been reported on immersion in cold water [10, 11]. However, the sinus arrhythmias reported by Rochelle & Horvath [14] were thought to be related to the respiratory, rather than cardiovascular, responses occurring at the time.

The initial cardiovascular responses to cold-water immersion represent only a slight risk for healthy individuals. The risk is greater for people with hypertension or coronary heart disease: individuals with poor coronary blood flow are more likely to develop myocardial ischaemia after sudden increases in cardiac workload. Coronary blood flow can be reduced still further as the time available for diastolic filling is shortened due to the tachycardia associated with the cold-shock response.

A raised peripheral resistance induces cardiac overload by increasing the afterload. For susceptible individuals this can be associated with cardiac irregularities. In addition, sudden elevations in blood pressure on immersion increase the likelihood of vessel rupture in hypertensive or aneurysmal individuals. This latter group are particularly at risk because they may be unaware of their condition.

Respiratory responses

For the healthy population it is the cold-shock respiratory responses which represent the greatest threat to survival. Several authors have reported that sudden cold-water immersion produces an 'inspiratory gasp' in subjects followed by uncontrollable hyperventilation, indicated by a significant fall in the end-tidal partial pressure and arterial tension of carbon dioxide [11–17, 21, 24–27]. The inspiratory gasp occurs almost immediately upon immersion: Goode *et al.* [13] measured a mean gasp of 2.0 litres lasting 1.5 s when eight male subjects were immersed in water at 28°C. In water at 11°C the corresponding values were a 3.0 litre gasp lasting 1.2 s.

On average, head-out immersion in cold water produces about a fourfold increase in ventilation during the first minute of exposure; this generally results from a greater increase in tidal volume than respiratory frequency [15, 25, 26]. There is significant variation in this response due to factors such as water temperature and subject differences. For example, increases in minute ventilation of 600% [12] and over 1000% [16] have been obtained after immersion of naked individuals in water at 10°C, and females have been reported to respond to immersion in cold water with a 35% smaller increase in minute ventilation than males [26]. Other authors [15, 27] have failed, however, to identify any difference between the sexes with regard to the cold-shock response.

Two dangers of the ventilatory responses are the respiratory alkalosis and hypocapnia which result from profound

hyperventilation. Significant reductions in the alveolar partial pressure and arterial tension of carbon dioxide can occur during the first minutes of exposure to cold water [10–12], reaching minimum levels within 15 s of exposure [11]. The mean arterial tension of carbon dioxide of 10 seated subjects has been reported to fall from 39.8 mmHg (5.31 kPa) at rest to 27.8 mmHg (3.71 kPa) after 1 min of ice-water showering of the chest [11].

Reductions in blood carbon dioxide levels are quickly followed by hypocapnia in sensory and motor nerve cells; this results in a decrease of their activation threshold [28]. Large falls in the arterial tension of carbon dioxide have been associated with ventricular fibrillation in dogs and man [29–32]. Such falls can also result in cerebral hypoxia caused by reductions in cerebral blood flow and a shift to the left in the haemoglobin dissociation curve (Bohr effect). These alterations probably account for the tetany, disorientation and clouding of consciousness demonstrated by subjects during the first minutes of cold-water immersion [12, 33, 34].

The loss of control of ventilation makes breathing while swimming very difficult: a swim stroke/respiration asynchrony occurs [35] which can produce inefficient swim stroke mechanics, resulting in the inhalation of water and swim failure. Even competent warm-water swimmers may only be able to swim for a few minutes in cold water [35–38]. Swimming performance is also likely to be adversely affected by the subjective feelings of dyspnoea [21, 25] and consequent panic initiated by cold-water immersion.

The dyspnoea is not thought to be caused by bronchoconstriction or changes in lung mechanics: evidence of only a small (17%) increase in airway resistance and no decrease in pulmonary compliance has been found after 30 s of ice-water showers [25]. The dyspnoea may be caused by a direct effect of the afferent impulses which initiate the respiratory responses [25], or by increases in the work required from the respiratory musculature. Alternatively, it may result from the abrupt and transient shift which occurs in end-expiratory lung volume [21, 25]. This shift is thought to result from greater reflex stimulation of inspiration than expiration, and not from alterations in lung mechanics [25]. It is maximal within two to three breaths after exposure [25], continues for between 1 and 3 min depending on water temperature, and then slowly declines. The time-course of the shift has been found to approximate the period when subjects report being breathless [21].

During ice-water showers the average inspiratory shift in end-expiratory lung volume has been reported to be 0.44 litre; this compares with 0.36 litre observed during showers with water at 15°C and no significant effect with showers at higher water temperatures [25]. A mean inspiratory shift in end-expiratory lung volume of 2.3 litres was found in eight naked subjects immersed in water at 5, 10 and 15°C (M. J. Tipton, unpublished work). In most subjects this resulted in tidal breathing occurring within 1 litre of total lung capacity. The larger shifts observed in this study were probably due to the head-out immersions employed; these would produce greater

haemodynamic effects and cold stress than cold-water showering.

Perhaps the most dangerous consequence of the initial responses to cold water is the reduction in maximal breath-hold time caused by the inspiratory 'gasp' response. This effect has been noted by several authors [25, 27, 39, 40]; Hayward *et al.* [27] found that over a 0–15°C range of water temperature, the maximum breath-hold time of subjects was reduced to 25–30% of that seen before submersion, and to 30–60% of that seen on immersion in water at thermoneutral temperatures. In the study of Tipton & Vincent [40] the mean maximum breath-hold time of 18 normally clothed individuals fell from 45 s in thermoneutral air to 9.5 s on submersion in water at 5°C; the maximum breath-hold time of one subject was reduced to 0.2 s.

The cold-shock reduction in breath-hold time significantly increases the risk of an immersion victim aspirating water; this risk is increased still further if the immersion occurs in choppy water or involves a period of forced submersion in a capsized or sinking craft. In such situations death has been attributed to drowning [41, 42], or to vagal arrest of the heart after inhalation of water and stimulation of receptors in the nasopharynx and glottis [43].

Metabolic response

The metabolic rate of subjects increases during the first minutes of cold-water immersion [12, 15–17]. Hayward & Eckerson [15] reported that the metabolic rate of 21 lightly clothed male and female subjects increased by a factor of four during the first minute of immersion in water at 0°C. Although such an elevation will increase carbon dioxide production and reduce, to an extent, the degree of hypocapnia resulting from hyperventilation [10, 12], it may also increase the risk of cardiac irregularities by increasing the work required of the heart.

Hormonal response

It is generally believed that prolonged exposure to cold is a powerful stimulus for hormonal secretion in man [44–46]. The hormonal response to cold exposure lasting only a few minutes is less clear; Johnson *et al.* [45] reported that the plasma noradrenaline levels of six subjects had almost doubled after 2 min of immersion in water at 10°C, a finding supported by the work of Buhning & Spies [23]. Other authors [47, 48] have reported significant increases in plasma levels of noradrenaline and adrenaline after 1–2 min immersions of one hand or one foot in water at 0–5°C.

In contrast, Manager *et al.* [49] reported that the cold-pressor response does not affect catecholamine levels and Keatinge *et al.* [11] found no evidence of the release of adrenal catecholamines during 2 min ice-water showers, in which plasma adrenaline levels were not altered and plasma levels of noradrenaline were only slightly increased. The unreliability of early analytical techniques may have contributed to the variation in the findings reported above. The lack of agreement precludes any firm

conclusions from being drawn; in general, however, the evidence suggests that rapid increases in the plasma level of noradrenaline may occur on sudden exposure to cold water as a result of activation of the sympathetic nervous system, but rapid increases in the level of adrenaline are less likely to occur. If this is the case, then cold may be unique among unpleasant stimuli in not initiating a rapid release of adrenaline from the adrenal medulla [50].

MECHANISMS OF THE COLD-SHOCK RESPONSE

The evidence presented in the previous section, together with the immediacy with which the cold-shock response is initiated, suggests that it is neurogenic in nature, and tends to preclude any significant contribution from circulating hormones. On the basis of both the magnitude of the response observed on warm compared with cold-water immersion, and the relationship observed between the rate of change of skin temperature and respiratory drive [51], it can be concluded that the cold-shock response is initiated by afferent sensory information arising at the peripheral receptors: more specifically, those receptors which alter their rate of firing in response to cooling [12, 13, 17].

A cold stimulus applied to the skin inhibits warm receptor activity and activates several other types of receptors, including thermosensitive mechanoreceptors, thermal nociceptors and cold receptors [52, 53]. The depth of cold receptors in the tongues of cats has been shown to be 0.18–0.22 mm [54], placing them in a superficial subepidermal layer. This superficial location helps explain the rapidity with which the cold-shock response is initiated, and why subcutaneous fat fails to reduce the magnitude of the response [12, 13].

Knowledge of the pathways associated with the transmission and processing of thermosensory information is based on work in animals. Keatinge & Nadel [25] obtained greater increases in ventilation when water at 0°C, compared with 25°C, was poured over the trunks of four cats decerebrated just below the anterior border of the anterior colliculi, and over four hypothalamic cats. Ventilation was not found to increase when the experiment was repeated with two cats decerebrated through the posterior part of the posterior colliculi. The authors concluded that the reflex respiratory responses to cold are mediated at midbrain level and the cerebrum is not essential for the response in the cat.

It was suggested that emotional factors may complicate this response in humans; this was subsequently confirmed when greater ventilatory responses were obtained from subjects immersed in open-water compared with corresponding laboratory conditions [12]. It would also appear that some unhabituated subjects can consciously suppress the cold-shock ventilatory response [13], in some cases by as much as 75% (M. J. Tipton, unpublished work).

It has been hypothesized that thermoafferents from the peripheral cold receptors increase ventilation by directly stimulating the respiratory centre [10, 13]. The sensitivity of the central chemoreceptors has been reported to remain unchanged [12]. Part of the ventilatory response

may result from reflex stimulation at a spinal level of α -motoneurons innervating the intercostal muscles and diaphragm [51].

The increase in heart rate is thought to be due to the reflex stimulation of the sympathetic nervous system; increased sympathetic activity can occur as a direct consequence of rapid falls in skin temperature [45, 46] and has both inotropic and chronotropic effects on the heart [11, 55]. The response is thought to be mediated through the tegmentum of the midbrain and hypothalamus [56]; the speed with which it is initiated suggests that uncomplicated pathways are involved.

The sympathetic nervous system is also involved in the peripheral vasoconstriction and blood pressure responses which occur on immersion in cold water [11, 19, 57]. Work with rats [58] suggests that cold-induced cutaneous vasoconstriction is integrated in the preoptic area of the hypothalamus and may depend on connections between this area and the pressor area of the medulla via the medial forebrain bundle.

The diffusion of noradrenaline released from the terminals of sympathetic nerves after skin or hypothalamic receptor stimulation, can result in peripheral smooth muscle contraction and blood vessel constriction [59]. In addition to sympathetic vasoconstrictor efferents, peripheral blood vessel vasoconstriction can also be caused by a direct effect of cooling [60]. The hypothesized mechanism involves depolarization of arterial smooth muscle as a result of cooling [57] and, as a cause of peripheral cold-induced vasoconstriction, it is thought to be at least as important as the sympathetic input [50].

Frey *et al.* [55] examined the cardiovascular responses of six subjects to immersion of one foot in water at 4°C. They reported that this manoeuvre initiated a prompt tachycardia and increased peripheral vasoconstriction. Using propranolol plus atropine they determined which of the responses to foot immersion obtained after 16–30 s of immersion were neurally mediated. They concluded that the early cardiovascular responses indicated widespread sympathetic outflow to the heart and vessels, probably accompanied by vagal withdrawal. The arterial pressure response to immersion of the foot was not found to be directly influenced by the drugs used; it was therefore concluded that this response resulted from an increase in peripheral resistance caused by stimulation of α -adrenoceptors. This conclusion supported the earlier work of Obrist *et al.* [61].

The increase in metabolic rate on immersion in cold water is probably largely determined by the general increase in muscle tension that accompanies such cold exposure. It will also be raised as a result of increased oxygen utilization by the respiratory musculature during any hyperventilation occurring at this time. It has also been suggested [51] that some of the initial increase in oxygen consumption after immersion may be due to the hydrostatic and haemodynamic responses which occur after immersion at any temperature and produce increases in pulmonary perfusion [62–66].

The cold-shock response subsides after 2–3 min and returns towards pre-immersion levels. The ventilatory response peaks within the first 30 s and declines to

approximately twice pre-immersion levels by the third to fifth minute of immersion [10–13, 15, 67]. The dynamic phase of the metabolic response is complete by approximately 3 min of immersion, at which time it has been reported to be 50% higher than pre-immersion values [15]. The subsidence of the cold-shock response is thought to be due to the habituation of the peripheral receptors [10], and the decay of their dynamic response to cold. The return of the heart rate response towards pre-immersion levels is also likely to be influenced by a baroreceptor reflex response to increasing arterial pressure; this would explain why it habituates more quickly than the other responses [55].

FACTORS MODIFYING THE COLD-SHOCK RESPONSE

Hydrostatic and haemodynamic effects of immersion

Immersion in water increases the hydrostatic pressure acting on the body. The resulting haemodynamic alterations occur within six heart beats of immersion to the neck [64] and include a 500–700 ml increase in central blood volume [62, 63], an increase in central venous pressure [20] and an increase in ventricular diastolic filling and preload of the heart [20]. Some of these responses are likely to modify the cardiac responses observed on immersion in cold water; they may also impose limitations on pulmonary ventilation [51, 65, 66].

Water temperature

One would expect lower water temperatures to produce faster rates of fall of skin temperature and, consequently, a greater dynamic response from the peripheral receptors, resulting in a larger cold-shock response. Down to moderately low water temperatures an inverse relationship does exist between the water temperature in which naked subjects are immersed and the magnitude of their cold-shock response [10–17].

At low water temperatures, however, this relationship ceases and the maximum possible physiological response is evoked. The temperature at which this occurs is a matter for debate. Hayward & Eckerson [15] concluded that the pattern and magnitude of the metabolic, respiratory and heart rate responses obtained on immersion in water at 0°C were similar to those observed in water at 5–15°C. This finding is both supported [13] and contradicted [12] by the results of other investigators, and may be dependent on the response being examined [10].

Clothing

In addition to the rate of change of skin temperature, the magnitude of the cold-shock response initiated on immersion in cold water is also determined by the surface area exposed to the cold stimulus [40, 68]. These temporal and spatial effects can, to varying extents, be altered by clothing.

Although normal clothing can attenuate the cold-shock response [10, 69], it can still reach potentially hazardous

levels in individuals [15, 40]. Specialist protective clothing provides significantly better protection [16, 40, 51] by reducing the rate of fall of skin temperature and the surface area wetted. Only a small number of authors have attempted to determine which body areas are particularly important for the initiation of the cold-shock response. As part of a wider study, Keatinge & Nadel [25] examined the ventilatory response evoked by sudden cooling of different regions of the body with showers at 15°C. They obtained the greatest response when the front of the trunk and back of the chest were cooled. A smaller response was initiated by cooling the middle and lower back and extremities, even though the surface area of the extremities was larger than that cooled during torso exposure.

These findings were thought to be due to cold habituation of the extremities and the paucity of cold receptors in these regions. This latter suggestion was based on the psychophysical evidence of cold-spot identification; although each cold spot may correspond to a single cold-receptor ending [70], interpretation of such evidence requires some caution as electrophysiological studies have revealed constant cold- and warm-receptor discharge at perceptual thermoneutrality [52, 71].

More recently, Tipton & Golden [16] examined the amelioration of the cold-shock response after regional insulation of the body surface. They reported that limb protection significantly reduced the mean heart rate and ventilatory responses observed in 12 subjects during the first minute of immersion in water at 10°C, when compared with the responses recorded during naked immersion in water at 10°C. Torso protection significantly reduced ventilation on immersion, but failed to lower the heart rate response when compared with naked immersion. The authors concluded that there may be differences in the relative importance of different body areas for the initiation of some of the responses included in the cold-shock response.

Exercise and aerobic fitness

The performance of gentle exercise during the first minutes of immersion does not prevent cold-shock hyperventilation and falls in the end-tidal partial pressure of carbon dioxide from occurring [12]. At moderate and maximal levels of exercise the fall in carbon dioxide partial pressure can be reduced or reversed [10, 12] and hyperpnoea rather than hyperventilation therefore occurs.

A cross-adaptation between fitness and the thermoregulatory responses to exposure to cold in man has been reported by several authors [10, 72–75]. Fewer investigators have examined the relationship between fitness and the cold-shock response. Hull *et al.* [48] found that aerobic fitness was inversely related to the heart rate of male but not female subjects during a cold-pressor test, and Golden & Tipton [76] reported an inverse relationship between the aerobic fitness of subjects and their cold-shock, cardiac and ventilatory responses to immersion in water at 15°C.

The mechanism behind this cross-adaptation is not known, but training the aerobic system is thought to alter the balance between the activity of the sympathetic and parasympathetic nervous systems, such that vagal cholinergic drive is increased while sympathetic adrenergic drive is inhibited [77–79]. As the autonomic nervous system is involved in the initiation of the cold-shock response, it is possible that exercise-induced alterations in the responsiveness of this system could attenuate the cold-shock response of individuals.

Cold habituation

Only a few authors [10, 12, 14, 17] have examined the alterations occurring in the cold-shock response of man after repeated exposure to cold. Keatinge & Evans [10] reported that eight immersions in water at 15°C reduced or abolished the initial respiratory, heart rate and metabolic responses to immersion, and increased the end-tidal levels of carbon dioxide and thermal comfort of 12 subjects. In contrast, Rochelle & Horvath [14] could find no significant differences between the heart rate and ventilatory responses of regular surfers and non-surfers during the first minute of immersion in water at 19°C.

Golden & Tipton [17] confirmed the findings of Keatinge & Evans [10] and described the time-course of adaptation of the heart rate and ventilatory response over eight immersions in water at 15°C. The profile of the adaptation of these two responses was found to differ; the initial ventilatory response to immersion was reduced by greater amounts in the early compared with later immersions, whereas the heart rate response was reduced by greater amounts in the later rather than earlier immersions. Additionally, by the later immersions the heart rate response was found to approximate that recorded on immersion in water at thermoneutral temperatures, whereas the ventilatory response remained significantly higher (250%).

These results suggest that by the later cold-water immersions the heart rate response was almost completely habituated, but the ventilatory response was not; either because it has a longer time-course of adaptation or because it does not possess the same capacity for adaptation.

The frontal areas of the cerebral cortex have been shown to be involved in the habituation of the heart rate response of rats to cold immersion of their tails [80] and it has been hypothesized [81] that the frontal cortex accelerates plastic changes occurring at a subcortical level: the reticular formation has been implicated [82]. As with physical training, repeated exposure to severe cold may result in a diminution of the sympathetic response to cold and an enhancement of the vagal response [83]. Whatever the precise mechanism, once established, habituation of the cold-shock response can last several months [82].

Pre-heating

It has been reported [12, 26] that pre-heating in a sauna, so as to increase skin but not core temperature,

significantly attenuates the ventilatory responses of subjects to a subsequent immersion in cold water. As skin temperature was found to fall at the same rate on immersion, with or without a prior sauna, the authors attempted to explain their findings by postulating the existence of deep skin receptors. It was suggested that after the sauna, deep skin receptors did not cool as quickly during the initial minutes of immersion and, as a consequence, the afferent stimulus to breathe was reduced [12].

During another study [67] this suggestion was supported when the percentage increase observed in ventilation on immersion in cold water was found to correlate with changes in deep skin temperature (1–2 mm), but not surface skin temperature. In this study, however, the authors were unable to demonstrate an attenuating effect of saunas on the ventilatory responses to cold-water immersion.

THE COLD-SHOCK RESPONSE AND THE DIVING RESPONSE

The other initial response to cold-water immersion is the diving response. This is most prominent, and has been most extensively investigated in diving mammals. The major components of the diving response have been identified as apnoea, bradycardia and selective vasoconstriction [5–9]; the main effect of this response is to extend underwater time by redistributing blood flow to tissues which are most susceptible to hypoxia, such as the brain and myocardium.

Despite suggestions that it can lead to vagally mediated cardiac arrhythmias and arrest in man [7, 84, 85] and is

unlikely to influence the ease with which immersion victims are resuscitated [27, 86], some authors [87, 88] believe the diving response may be implicated in the survival of individuals, especially children, after many minutes of submersion. This contrasts with the cold-shock response which is potentially hazardous.

The diving response is thought to be initiated after stimulation of the ophthalmic division of the trigeminal nerve by cold-water immersion of the face [6, 89]. During an accidental head-out immersion, therefore, the victim is likely to demonstrate the cold-shock response, initiated as it is by falling skin temperature. However, when the accidental immersion involves whole-body submersion the stimulus for both the cold-shock and the diving response is present. Under such conditions it is the cold-shock response which has been reported to predominate in children [86] as well as in adults [27].

SUMMARY AND CONCLUSION

This review is summarized in Fig. 1. It is concluded that the cold-shock response can result in the death or serious incapacitation of an individual long before general hypothermia develops. As such, this response is probably responsible for the majority of annual open-water immersion deaths.

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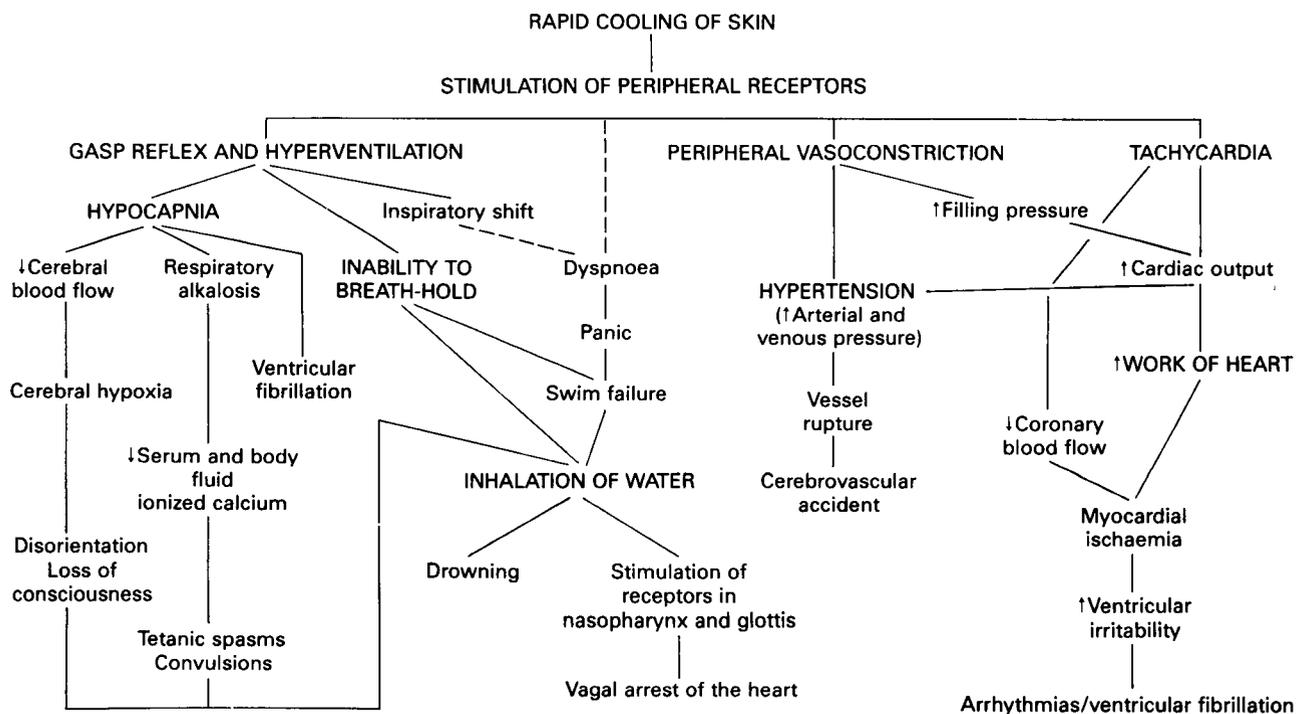


Fig. 1. Summary of the cold-shock response. For references, refer to the text. †, Increase; ↓, decrease.

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