CUTANEOUS REACTIONS TO COLD

THESIS

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بسم الله الرحمن الرحيم

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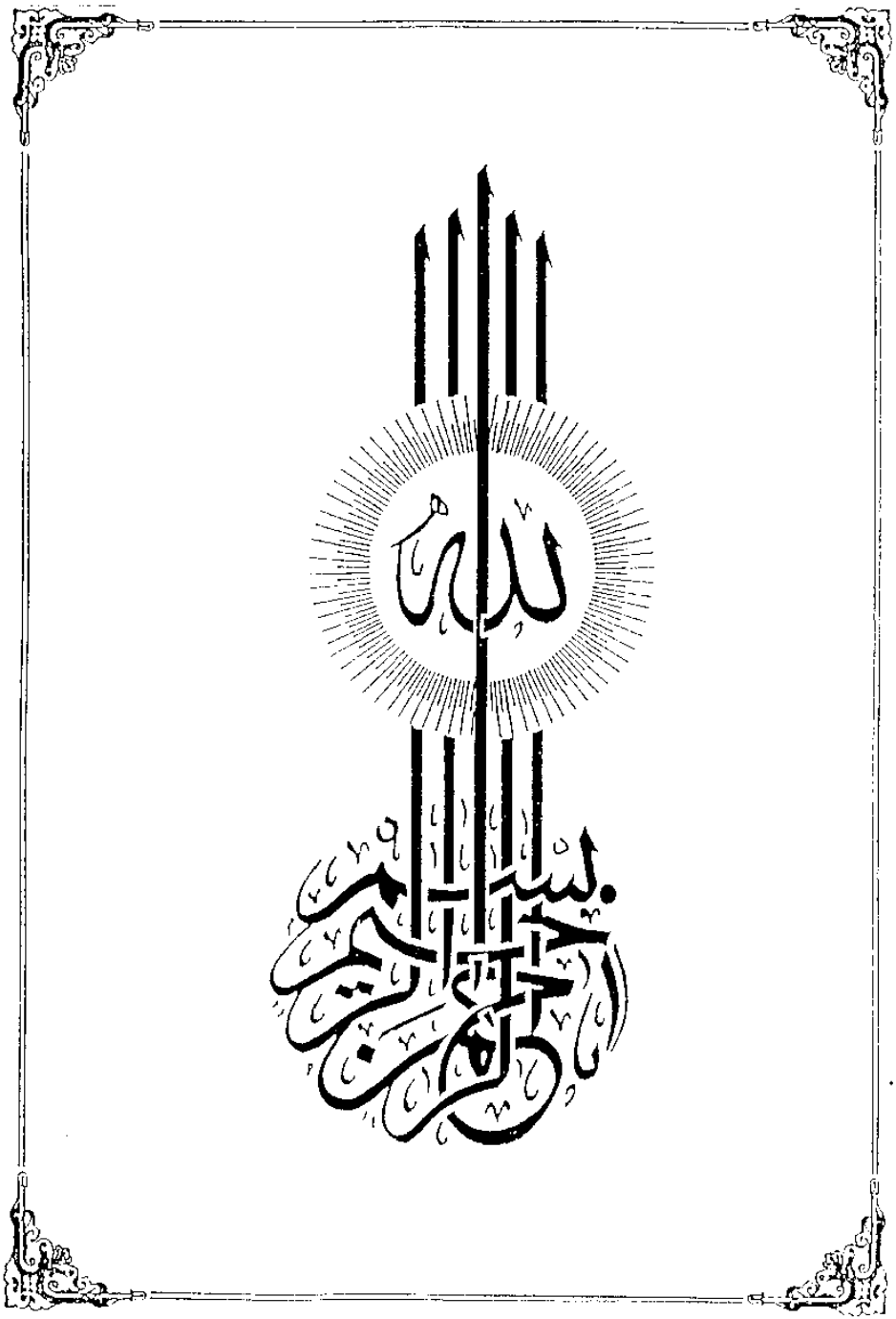

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TO MY PARENTS

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INTRODUCTION

INTRODUCTION

Cold is one of the most common stresses to which living creatures are exposed. All through human history, cold played a major share in changing the destination of many nations. The cold winter of Russia defeated the army of Napoleon and killed much more of his men than did the Cossacks. Again, such cold weather protected Moscow from invasion by the Nazi army.

The normal response to cold air is, at first, rapid cooling of the extremities accompanied by a slight increase in internal body temperature (Teichner, 1966). Rapid cooling of the extremities is a result of both surface-to-air heat loss and reduced flow of warm blood due to sympathetic vasoconstriction. The increase in body temperature is the result of reduced heat loss to the periphery, while maintaining body heat production. This occurs provided cold conditions are not extreme and internal body temperature can be maintained by use of clothing, metabolic heat production, or physical activity (Sampson, 1984). Thus the primary function of the cutaneous blood flow is temperature regulation which serves the organism as a whole and not the skin itself. However mechanisms do exist which afford a degree of local protection against skin

damage. One such mechanism is the "hunting reaction" of Lewis, which consists of a peripheral vasodilation-vasoconstriction cycle. In this latter, after the initial cooling, there is a rewarming of the extremities due to vasodilation followed again by vasoconstrictive cooling, then rewarming, and so on in an alternating fashion (Dana et al., 1969). However, when the individual is immobile for prolonged periods, when there is insufficient protective clothing, or when he is highly stressed, this reaction gives way to steady and progressively more severe vasoconstriction, with an increased risk of cold injury (Teichner, 1966; Hanson and Goldman, 1969).

The skin is provided with a profuse blood supply to fulfil its role in thermoregulation. In digital skin, blood flow can range from less than 0.5 ml per minute through each 100 ml of tissue during intense vasoconstriction to more than 50 ml with the subject warm. The oxygen requirement of digital skin has been calculated as 0.8 ml of blood per minute. So, even in the normal person vasoconstrictor control is probably powerful enough to reduce the blood supply to the point of anoxia (Fox, 1968).

The control of blood flow through the skin depends partly on the central control through the sympathetic vasomotor nerves and partly on a direct local effect of temperature. Over the greater part of the body surface, cutaneous vasomotor control is mediated by both vasoconstrictor nerves and a vasodilator

mechanism, whereas in the extremities only vasoconstrictor control is present (Fox and Edholm, 1963).

The sympathetic outflow to the cutaneous arterioles, arteriovenous anastomoses and veins is continuously adjusted by the hypothalamic thermoregulatory centers. When the body temperature increases, the sympathetic outflow to the skin blood vessels decreases, and the sweat glands are activated and produce kinins; as a result, the arterioles, the arteriovenous anastomoses, and the veins open. Large amounts of blood are shunted to the latter, which act as a capacious radiator for the physical exchange of heat. When the body temperature decreases, the sympathetic outflow to the skin is augmented, sweat secretion is inhibited, and the dissipation of heat is restricted (Shepherd and Vanhoutte, 1980).

Local cooling greatly augments the arterial and venous responses to sympathetic nerve activation. The enchanced arteriolar constriction limits the amount of blood flowing through the skin. The augmented venoconstriction directs the venous return to the deep venae comitantes which run alongside the deep arteries. Unlike cutaneous vessels, deep vessels dilate when they are exposed to the colder blood coming from the skin. This favors "the counter-current" exchange of heat which warms up the venous blood returning to the body core (Vanhoutte and

Shepherd, 1970 ; Vanhoutte and Lorenz, 1970; Shepherd and Vanhoutte, 1980).

The augmented response of cutaneous blood vessels to the sympathetic nerve activity during moderate cooling is due to two factors. First, lowering the temperature delays the disappearance of the adrenergic neurotransmitter from the junctional cleft between the adrenergic nerve endings and the cutaneous vascular smooth muscle, because it inhibits neuronal uptake, enzymatic degradation, and diffusion of norepinephrine (Janssens and Vanhoutte, 1978, 1979). Thus, the amount of transmitter in the vicinity of the alfa-adrenergic receptors of the vascular effector cells increases. Further, moderate cooling greatly augments the responsiveness of the cutaneous vascular smooth muscle to norepinephrine, because the affinity of their alpha-adrenergic receptors significantly increases; this increase in affinity holds not only for norepinephrine, but also for alpha-adrenergic antagonists (Vanhoutte and Shepherd, 1970; Janssens and Vanhoutte, 1978). At the same time moderate cooling augments the sensitivity of the vascular smooth muscle from the beta-adrenergic effects of catecholamines, which in normal conditions persumably tempers the increased responsiveness to the alpha-adrenergic effect of these substances (Vanhoutte and Shepherd, 1970; Shepherd and Vanhoutte, 1980).

An important effect to cold is the increase of blood viscosity which plays a part in some of the abnormal reactions to this cold (Ryan and Copeman, 1969). Low temperatures also induce platelet aggregation (Kattlove and Alexander, 1971) and show the dissociation of oxyhemoglobin to hemoglobin (Champion, 1979).

A correlation between pain intensity and degree of vasoconstriction have been reported. Nociceptor for cold pain are most probably situated in deeper skin structures and are excited by temperature ranging approximately from 3 to 20° C(Kreh et al., 1984).

Under experimental conditions, all injuries due to cold are similar in pathogenesis and differ primarily in degree. The vascular changes are characterized by initial arterial and arteriolar constriction, followed by venous and capillary dilation. This results in decreased blood flow, increased endothelial leakage and stasis. Arterial venous shunting occurs, and blood bypasses the frozen region. Segmental vascular necrosis and massive thrombosis then develop, with gangrene appearing in the areas devoid of circulation (Zalar and Harber, 1985).

Diseases caused or aggravated by cold are either reactions to abnormal cold or abnormal reactions to cold.

A working classification proposed by Champion (1979) will be followed and its various items outlined:

1.0. Reactions to abnormal cold:

- 1.1. Frostbite.
- 1.2. Trench foot (Immersion foot).

2.0. Abnormal reactions to cold:

- 2.1. Pernio (Chilblain).
- 2.2. Acrocyanosis.
- 2.3. Livedo-reticularis.
- 2.4. Raynaud's syndrome
- 2.5. Cryoglobulinemia.
- 2.6. Cryofibrinogenemia.
- 2.7. Crystalglobulinemia.
- 2.8. Cold agglutinins syndrome.
- 2.9. Paroxysmal cold hemoglobinuria.
- 2.10. Cold urticaria.
- 2.11. Cold erythema.
- 2.12. Cold panniculitis.
- 2.13. Asteatotic eczema.
- 2.14. Cutaneous reactions to cold in newborn:
  - 2.14.1. Sclerema neonatorum.
  - 2.14.2. Subcutaneous fat necrosis.

AIM OF THE WORK

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Egypt is generally a warm country most of the year, except for a relatively short winter. It is expected that cutaneous reactions to cold should be rare, however, they are not infrequently met with. This is not astonishing if we put into consideration that houses and public places are not provided with heating as in cold countries with prolonged winter.

The aim of this work is to review the cutaneous reactions to cold in a concise manner.