HYPERHIDROSIS — AN AETIOLOGICAL INTERPRETATION

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Summary

Increased Sweating on palms, soles and axillae is a very common problem. This paper describes various aetiological factors in the different types of hyperhidrosis. The role of autonomic nervous system and other factors in hyperhidrosis is discussed. Role of anxiety in causation of palmo-plantar hyperhidrosis is also discussed. Neurophysiological mechanism of anxiety involved in palmo-plantar hyperhidrosis is described.

Hyperhidrosis is excessive production of sweat. In theory when there is over or under production of sweat it should be possible to determine whether the change is in the sweat glands due to pharmacologically active agents acting on the glands, to abnormal stimulation of sympathetic pathway between the hypothalamus and the nerve-ending, or to over activity of one of the three different 'Centres' responsible for thermo-regulatory, mental and gustatory sweating. Patients with hyperhidrosis usually fall into the following groups :—

I. Those with asymmetrical sweating.

II. Those with generalised sweating.
   Thermal
   Emotional

(I) Asymmetrical hyperhidrosis

Excessive sweating may be due to neurological lesions involving any part of the sympathetic pathway from brain to nerve ending. Lesion may be present in the cerebral cortex, basal ganglia, spinal cord or peripheral nerves. It is usually present as an isolated phenomenon in the absence of other neurological signs or symptoms. Asymmetrical sweating may also occur reflexly from visceral disturbances, around an area of anhidrosis as in leprosy or due to axon reflex stimulation around a leg ulcer. Affected areas may be small or extensive as when one half of the body may sweat continuously or more than the other half with mental activity. Sometimes psychological disturbances account for such distribution but more often the causes remain a mystery.

Facial sweating affecting either right or left side of face has been reported by Tarlov and Herz[1], Tankel[2] and Pearce[3]. In these cases, the sweat glands were found to be hypersensitive to cholinergic drugs.

Certain localised areas of hyperhidrosis on face have been shown to occur after stellate ganglionectomy. This is due to the development of nervous pathways from epibranchial placodes[4].

Mellinkoff and Mellinkoff[5] and Mellinkoff[6] reported two instances of

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gustatory sweating of knees and wrists without any demonstrable hypersensitivity to cholinergic drugs.

II. Generalised or symmetrical hyperhidrosis

(a) Thermal

Generalised sweating occurs during or after many infective processes and may be due to some instability of heat regulating centre in hypothalamus after febrile illnesses. Thermoregulatory sweating, unlike emotional sweating occurs usually during sleep. A similar mechanism may account for the hyperhidrosis associated with alcohol intoxication, gout and vomiting[7].

The mechanism of generalised hyperhidrosis associated with diabetes, hyperthyroidism, hyperpituitarism, acromegaly, obesity, pregnancy, menopause, concussion, parkinsonism and malignant diseases is not known.

Sympathetic nervous system diseases involving vasoconstriction of skin blood vessels with sweating occur; as for example in hypoglycaemia, dumping syndrome, alcohol and drug withdrawal, shock, syncopal states, pheochromocytoma and carcinoid syndrome.

In response to thermal stimulation, body sweats profusely first on forehead, neck, larger areas of ventral and dorsal surfaces of trunk, lumbar regions and the back of hands[8]. The sides of chest and extremities sweat less and inner surface of thigh still less.

(b) Localised or Emotional or Idiopathic Hyperhidrosis.

In this type the most frequent sites involved are the palms and soles and intertriginous areas namely axillae, inguinal folds and perineal areas. Other common sites of involvement are forehead, tip of nose and sternal area.

Hyperhidrosis of palms and soles may be continuous or phasic. When continuous it is worse in the summer and not clearly precipitated by mental factors. When phasic it is usually precipitated by minor emotional or mental activity with no significant seasonal variation. The hands may be cold and may show a tendency to acrocyanosis.

The chief cause of localised hyperhidrosis is said to be emotional. Based on the responses of human eccrine sweat glands to various stimuli they are divided into 3 categories[9].

(i) Glands in palms and soles which respond to psychic stimuli.

(ii) Glands in Axillae and Forehead which respond to thermal and psychic stimuli.

(iii) Glands in the rest of the body which respond to thermal stimuli.

The primary idiopathic sweating is by definition a diagnosis of exclusion. Hyperhidrosis of palms and soles in association with generalised hyperhidrosis may be produced secondary to recognised entities like hyperthyroidism, hypoglycemia, hypoxia, obesity, menopause, pregnancy and pheochromocytoma[10]. States of fear, rage and tension can induce an increase of sweat secretion.

Emotional or mental activity increases palmo-plantar hyperhidrosis. Thermal stimuli may aggravate this condition. Mental or emotional stimuli are usually said to trigger off this type of sweating. In some patients deep seated emotional disturbances may be found; in some there may be no primary emotional disorder.[11] We have studied patients of palmo-plantar hyperhidrosis and have found that their anxiety scores are not different from those of the normal[12].

Hyperhidrosis of palms, soles and axillae can occur in any combination. Troublesome hyperhidrosis of soles occurs in young adults. When this is associated with vasomotor changes, the skin is sodden, cold and cyanotic. To this, the name of "symmetrical-lividity" is sometimes applied[13]. In our study on 32 hyperhidrotics, eight patients were found to have symmetrical lividity[14].

Axillary sweating is continuous or more commonly phasic and may be precipitated by heat or mental activity. It is very common on undressing[13].

MacKenna[15] has tried to correlate hyperhidrosis of palms and soles (along with acne-excorie) with personality of patients suffering from morbid anxiety. Engles and Wittkower[16] have correlated certain emotional states with various forms of cutaneous disorders: aggression with generalised pruritus, sexuality (both Homo and Heterosexuality) with genital and anal pruritus, anxiety with hyperhidrosis, shame with rosacea, anger with urticaria and the longing for love with atopic dermatitis.

It should be stressed that none of the disorders mentioned above are considered to be strictly psychogenic. All are multicausal determined by a combination of constitutional and acquired factors of which emotion is but one; one weighing more or less heavily in the given individual at a given time. Rook and Wilkinson[17] have classified hyperhidrosis among dermatoses frequently provoked or perpetuated by demonstrable psychosomatic mechanism.

Norton and Hall-Smith[18] stated that 40% of their total number of skin patients suffer from anxiety states. These authors felt that a high percentage of skin patients have abnormal personality. The most common type of personality in the whole series was the "anxious— obsessional" personality. These patients were found to be over-conscientious, orderly, quiet and cautious worriers with preference for routine peaceful existence.

Sainsbury[19] found that in general, psychosomatic patients have higher than average levels of neuroticism and this was confirmed by Barendregt[20] and Bendian[21]. Srivastava, Bhat and Singh[22] found that skin patients in general have higher neuroticism than normals.

Neurophysiologically, anxiety is associated with a 'tuning' of the sympathetic adrenergic system in response to repeated alerting stimuli. Such stimuli successively lower the threshold for new stimuli and the heightened sympathetic system then dominates the parasympathetic system[23]. The resulting excessive discharge in the visceral and motor systems produce clinical manifestations the nature of which depend upon the individual's pattern of anxiety. Although there is an overall physiological overactivation, considerable individual differences have been found and the physiological reaction profile is highly specific to an individual[24].

Malmo[24] postulated that pathological anxiety is related to an over-arousal state. Lader and Wing[25] also suggest that morbid anxiety is the experience of abnormally prolonged and excessive C. N. S. activity. Elaborating this further, Eysenck[26] suggested that all emotional states are probably states of high arousal and it is the subjective experience and the overt behaviour which delineate the emotional pattern. Conversely all arousal states need not be 'emotional' as cortical arousal can take place due to non-emotional stimuli also.

A measure of autonomic anxiety is palmar sweat gland activity measured indirectly as the electrical resistance (or conductance) of the skin. The

palmar sweat glands serve to optimize tactile sensitivity and grip. Alteration in skin resistance in response to auditory, visual or tactile stimuli always take the form of transiently increased sweat gland activity (lowered resistance) and are termed 'galvanic-skin-responses (G. S. R.'s) or Psychogalvanic reflexes (P. G. R's). Spontaneous fluctuations in the skin resistance are a usual feature of the tracings and take the form of small diminutions in resistance in the absence of any apparent external stimuli[27].

Thus it is seen that palmo-plantar hyperhidrosis is not a simple symptom but a manifestation of extremely complicated neurophysiologic and psychodynamic processes in a patient.

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TRUE or FALSE ?

The epidermis of the aged is more permeable than that of the young as a result of which risk of primary irritant contact dermatitis is increased in the old people.

(Answer page No. 15)