Pathophysiology and Management Approaches for Hypohidrosis and Anhidrosis

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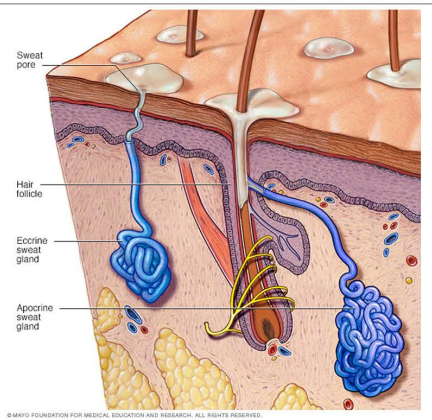
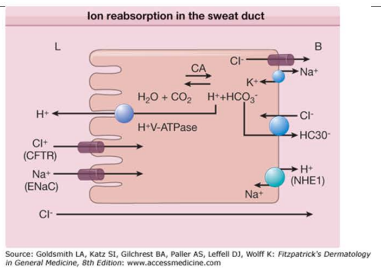
Eccrine sweating glands are divided into functional units: 1) a bulbous secretory coil that generates precursor fluid, and 2) a reabsorptive duct that transports the fluid to the skin surface. The secretory coils reside in the deep dermis or hypodermis and consist of clear cells, dark cells, and myoepithelial cells. Clear cells are the primary serous secreting cells and the likely source of secretory disorders of the gland. Dark cells are responsible for exocytosis of many of the non-ionic constituents found in sweat and are associated with certain anhidrotic sweat glands, and in addition have secretory components of their own. Myoepithelial cells surround the secretory coil and contract to provide the structural rigidity necessary to generate pressure within the gland. The reabsorptive duct contains a two-cell thick structure that modifies the fluid being transported to the skin surface.



Disorders of sweat output are expressed as systemic or focal (i.e., confined to a specific skin area), and as increased (hyperhidrosis), decreased (hypohidrosis), or lack (anhidrosis) of sweat output when compared to sweating requirements for thermoregulation. Hyperhidrosis has previously been described, and is estimated to affect 15.3 million Americans, severely affecting these patients' quality of life. In contrast, hypohidrosis and anhidrosis incidence rates and etiologies are less clear. Neurologic conditions, such as multiple sclerosis, can impair activation of sympathetic nerves, and other disorders, such as peripheral neuropathy, can reduce sympathetic neural transduction to sweat glands. The sweat glands themselves can be occluded (malaria rubra), damaged (tattoos), dysfunctional (cystic fibrous), or missing altogether (congenital anhidrosis). Exogenous compounds, such as anticholinergics, can prevent acetylcholine from activating the sweat gland. Endogenous factors, such as increased plasma osmolality, or physiological adaptations, such as detraining or bedrest, can also decrease the capacity to sweat, resulting in hypohidrosis. The purpose of this review is to clarify what is known about these disorders resulting in low or no sweat output, identify common themes and potential mechanisms of action, and describe some possible management approaches for the health care practitioner. If left untreated, hypohidrosis can predispose an individual to heat illnesses, such as heat stroke, as well as skin barrier dysfunctions, as sweat secretions are needed for the control of flora, immune defense, and skin hydration.



Classification of Hypohidrosis and Anhidrosis Causes

- Neurologic – Multiple Sclerosis, Harlequin Syndrome, Ross Syndrome, Diabetes Mellitus
- Sweat Gland Dysfunction – Cystic Fibrosis, Miliaria, Cholinergic Urticaria, Atopic Dermatitis, Congenital Anhidrotic Ectodermal Dysplasia
- Exogenous Sources – Anticholinergics, Opioids, TCAs, Antiepileptics, Antihistamines
- Endogenous Sources – Aging, Acclimation to Climate, Dehydration, Tattooing, Bedrest

Image Citation

https://www.mayoclinic.org/diseases-conditions/hyperhidrosis/multimedia/sweat-glands/img-20007980

https://accessmedicine.mhmedical.com/Content.aspx?bookid=392&Sectionid=41138789