very limited manner. More often, however, the local or circumscribed manifestation of NF suggests, if not declares, an atypical or distinctive form of NF, that is, a form of NF not accounted for by the rubrics NF-I or NF-II.

At this juncture a second problem must be considered: how to evaluate the patient with known or presumed NF. (For the sake of brevity, patient ages will not be considered/emphasized in this editorial.) Three rationales for undertaking such an evaluation must be acknowledged: to determine assignment of a diagnosis; to respect assignment of a diagnosis; and to respect ignorance.

1. **To Determine Assignment.**—If the type of NF is unclear, and specifically if assignment of NF-I or NF-II is not readily apparent, an evaluation to identify specific features of NF-I (eg, Lisch nodules, optic pathway gliomas) or NF-II (eg, acoustic neuromas, cataracts) must be undertaken. Positive findings will presumably establish the diagnosis, while negative data may leave a specific diagnosis wanting.

2. **To Respect Assignment.**—If the type of NF is already established, respect for the full array of its manifestations may require that the extent and nature of its complications and presymptomatic features be clarified through a comprehensive evaluation.

3. **To Respect Ignorance.**—Even if we know the correct diagnosis (eg, NF-I or NF-II), there are still many things about the disorder we are ignorant about. For example, consider the recent emphasis on iris Lisch nodules for NF-I[5] and the more recent appreciation of cataracts as part of NF-II.[6] However, if the diagnosis is other than either of these two forms of NF, then we know even that much less about the disorder, and a comprehensive evaluation respecting that ignorance is even more compelling.

Clearly, the trend is toward the identification of more and more cases of NF that do not neatly fit into the two diagnostic categories we know best. One of our tasks for the past few years is to sort out these various forms as accurately as possible, to understand the pathogenesis of NF, and to provide optimally accurate data for direct patient management and genetic counseling. This is not merely the task of the NF specialist; it is the responsibility of all of us who assume any responsibility for the NF patient and his or her family.

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Treatment of Hyperhidrosis

Human beings have two functionally distinct sets of eccrine sweat glands. One populates the entire skin with the exception of the palms and soles. These thermally responsive glands play a key role in heat adaptation. In fact, thermally responsive eccrine glands are a uniquely human attribute. The other set of eccrine glands responds only to emotional stimulation and is analogous to the glands in frictional skin (paws) of other mammals. These emotionally responsive glands are present primarily in the palms and soles. Eccrine glands in the axillae are unusual in their responsiveness to both thermal and emotional stimulation.

See also p 893.

The rate of sweating, whether of emotional or thermal origin, displays great individual variability. Copious thermally stimulated sweat as a result of exercise is considered "healthy," whereas excessive emotionally stimulated sweating is regarded as a sign of weakness and socially unacceptable by most

persons. The dermatologist is, therefore, often consulted by patients who complain of excessive axillary and/or palmoplantar sweating. For many, simple topical measures such as the use of an alcoholic solution of aluminum hexahydrate (Drysol) suffices. There remains, however, a significant number of patients who are unresponsive to topical therapy. Although logical, systemic anticholinergic therapy is useless because toxicity must be achieved to lessen sweating. Similarly, tranquilizers are generally unrewarding, although the hyperhidrosis is triggered by emotional stress. In most cases, significant lethargy and drowsiness must occur before sweating is decreased. In other words, both the sweat glands and the mind must be rendered nonfunctional. This problem can be overcome in some cases by prescribing a tranquilizer, eg, diazepam (5 mg at bedtime for a week or two). Once the patient has become "accustomed" to the drug it can usually be taken before anticipated periods of stress without producing somnolence while still maintaining its calming effect.

If hyperhidrosis is confined to the axillae, excision of the main sweat gland–bearing areas can be easily accomplished. However, many patients are unwilling to undergo surgery and obviously excision is not a reasonable approach to excessive sweating of the palms and/or soles. For palmar hyperhidrosis, sympathectomy has been advocated for many decades and the technique has improved in recent years so that it is a reasonably simple and safe procedure. It should be emphasized, however, that permanent nonfunction of the sweat glands may lead to scaling and fissuring of the palms, which can be a far more distressing problem for the patient than hyperhidrosis.

In this issue of the ARCHIVES, Stolman[1] describes his experience with a well-established but still infrequently utilized technique to inhibit sweat gland function. The inhibiting effect of iontophoresis on sweating was first described 50 years ago,[2] but its dermatologic debut can be attributed to the classic study of Shelley et al[3] about a decade later. In recent years, Levit[4] has been its major advocate and its increasing popularity can be attributed to his efforts. Despite a considerable amount of research, the mechanism of iontophoretic inhibition of axillary and palmoplantar sweating is entirely unknown. Although there is considerable speculation based on studies of other areas of skin, this is of little relevance because the skin of the axillae and palm differs markedly in its response from the skin of the back or forearm. For example, the usual aluminum chlorhydrate antiperspirant agents are capable of totally inhibiting sweating on the forearm but are only partially effective in the axillae, even under optimal conditions. Similarly, the application of an iontophoretic current to the skin of the back induces microscopically detectable plugs in the distal sweat pore and miliaria rubra, but these do not occur in similarly treated palmar skin.[5] But, whatever the mechanism, there is no question that iontophoresis works well in most patients.

Two basic devices are commercially available. The Fischer unit (R. A. Fischer, Glendale, Calif) popularized by Levit was used in Stolman's study. Because the hand or foot to be treated is completely immersed in water within a plastic tray, the Fischer unit has the advantage of being able to treat all emotionally responsive eccrine glands including those on the lateral borders of the fingers or toes. Its major disadvantage is that treatment must be professionally supervised. In addition, to complete the circuit, a foot must be immersed while a hand is being treated and vice versa, so that current is flowing through the body.

The other commercially available unit is the Drionic, which has been shown to be highly effective in two recent controlled studies.[6,7] It has the advantages of being relatively inexpensive and can be used at home without professional supervision. A Drionic unit is also available for use in the axillae, which are not treatable with the Fischer unit. To date, no studies have been performed that critically and objectively compare the efficacy of the two devices, and these would be most welcome.

Given the rapid evolution in the electronics industry, it is likely that increasingly more effective, safer, and more convenient iontophoretic devices will be introduced for the treatment of hyperhidrosis. But, even today, we have available adequate instruments for the treatment of the majority of our patients with distressing hyperhidrosis.

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