At long last, hidradenitis suppurativa, a disease first described a century and a half ago and one of the most devastating among the thousands of dermatologic entities, has finally received recognition by a text dedicated solely to this hideous disorder.

This publication reminds us that this disease, neglected for most of its history, would merit classification by taxonomists as a “nomen dubium et confusum,” about which there has been no consensus regarding etiology, pathogenesis, histopathology, bacteriology, genetics, etc.

Major dermatologic texts the world over give rudimentary, incomplete, misleading, even fallacious and contradictory accounts of this baffling, multifarious, polymorphic disorder.

The historicity of hidradenitis suppurativa, which has been described under a variety of Latinized names, is in itself a fascinating, convoluted story, demonstrating once again that the only path to enlightenment and the resolution of controversies is through serious basic investigations. It is worth recounting too how patent fallacies can be perpetuated by the pronouncements of influential clinical figures, schooled in the descriptive morphology of dermatologic diseases. All this has thankfully changed owing to investigations by authoritative contributors to this volume.

The disease was named by Verneuil, a French surgeon, who in 1864 held that the primary event in pathogenesis was inflammation of the sweat glands [1]. This concept held sway over more than a century, during which numerous reports were in concordance. It was not till 1939 that Brunsting identified the apocrine, rather than the eccrine sweat glands as the specific target of the disease, although he did not quarrel with the prevailing idea that the primary event was inflammation of the apocrine glands [2]. In a later paper in 1952 [3] he presciently recognized that hidradenitis suppurativa had some similarities to acne vulgaris regarding clinical manifestations. The latter idea was further fleshed out in 1951 by Kierland, also of the famous Mayo Clinic, who perceived that hidradenitis suppurativa was not solely restricted to localization of the apocrine glands. He saw a relationship between acne conglobata and dissecting cellulites of the scalp, which sometimes occurred concomitantly [4]. Later workers confirmed Kierland’s concept that hidradenitis suppurativa was an umbrella term encompassing a variety of clinical expressions.

It was Shelley and Cahn’s report in 1955 that gave scientific credence to the belief that inflammation of the apocrine glands was the primary pathogenic event [5]. They sought to validate the concept by inducing the disease experimentally, historically a successful strategy embodied in Koch’s postulates. They plucked the axillary hairs of 12 male volunteers and immediately covered the area with an occlusive adhesive tape impregnated with belladonna, the latter presumably to suppress secretory activity. In 3 of the 12 volunteers, they histologically demonstrated hyperkeratotic plugging and dilatation of only one apocrine sweat duct in each specimen, associated with a severe inflammatory infiltrate engulfing and destroying the gland. Notably there was no involvement of the surrounding eccrine, apocrine or sebaceous
glands. They concluded that hidradenitis suppurativa represented an infection of the obstructed apocrine duct by the resident microflora of the axilla in the absence of known pathogens. Their findings were so commanding and persuasive that a host of reports for many years afterwards by many different observers thoroughly endorsed the concept, which was thereby elevated to the status of a dogma.

Interestingly, no attention was paid to an early blunt dissenting opinion in 1957 by Tiber Benedek, a Chicago dermatologist, who opined that the experiments from the famous Department of Dermatology of the University of Pennsylvania School of Medicine in Philadelphia were “poorly conceived” and that the results bore no resemblance to the native disease either bacteriologically or histopathologically, noting also that there was no evidence that the disease had been reproduced clinically [6]. Additionally Xu and Cook’s substantial histologic studies 30 years later completely failed to demonstrate keratinous plugging of the apocrine ducts at any stage [7]. Curiously, the prevailing dogma was not weakened in any way by the sharp criticisms.

On the contrary, in 1956, Pillsbury, Shelley, and Kligman of the University of Pennsylvania School of Medicine in Philadelphia proclaimed in their popular 1956 text, *Dermatology* that “hidradenitis suppurativa is a severe, chronic, recurrent suppurative infection of the apocrine sweat glands, resulting from poral closure and secondary bacterial infection” [8]. It was not until decades later that a steady stream of reports in the world literature failed to verify the Philadelphia doctrine that obstructive hyperkeratotic plugging of the apocrine duct was the initiating pathogenic event, or that infection was a common complication [9–11]. The Philadelphia concept has not withstood the test of time and is no longer tenable, as described by later investigators. James Leyden, a younger member of the Philadelphia group, one of the co-authors of this volume, has joined the chorus of dissenters against the views of his eminent predecessors, to which the name of Kligman has perforce been added!

Another of the authors, G.B.E. Jemec, is clearly a leading authority who has published more than a half-dozen reports on the histopathology, bacteriology, and clinical aspects of the diverse manifestations of this polymorphic disease [12–14]. In fact, all of the contributors to this volume have worked in the field, with credible bona fides. Jemec compared 60 consecutive biopsy samples of hidradenitis suppurativa patients with 30 normals, the first large controlled study, which failed to verify the ancient concept.

Some redeeming remarks are in order regarding the Philadelphia triad, Pillsbury, Shelly and Kligman, who perhaps deserve honorable mention for elaborating on Kierland’s perceptive observations that hidradenitis suppurativa was more than a disease of apocrine glands but belonged to a family of related conditions [3]. They presented a unifying concept which led them to coin the term “the follicular occlusion triad,” relating acne conglobata, hidradenitis suppurativa and dissecting cellulitis of the scalp into one nosologic grouping. This notion has now achieved universal acceptance. Plewig and Kligman added another component, the pilonidal sinus, comprising what is now called the follicular occlusion tetrad [15]. Finally, it was left to Plewig and Steger to coin the term acne inversa to acknowledge that hidradenitis suppurativa, while part of the occlusion tetrad, was a clinical entity, emphasizing its localization to the axilla, anogenital area, and the buttocks [16]. By contrast, acne vulgaris favors the face and trunk. So, hidradenitis logically became the inverse form of acne vulgaris. The first comprehensive account of acne inversa may be found in the third edition of *Acne and Rosacea* by Plewig and Kligman in 1993. The most elaborate account of acne inverse is given by Jansen and Plewig [17].

For the last and final word on acne inversa, the interested scholar should read Plewig’s elaborate treatise entitled “Acne inversa, acne keloidalis nuchae, abszedierende follikulitis der kopfhaut” [18]. He goes to great pains to emphasize that acne vulgaris originates in sebaceous follicles while acne inversa invokes terminal hair-bearing follicles.

Attention is called to a fascinating paper by Sellheyer and Krahl with the provocative title “Hidradenitis suppurativa is acne inversa! An appeal to finally abandon a misnomer” in which