The report by Barton et al. [1] in this issue of Dermatology is not only an innovative work related to dysplasia and premalignancy but also addresses the basic concepts that attend such matters.

There are laymen and experts in dermatopathology who use the term dysplasia. Others consider that there is no need and no reason to use that designation. In the past, many talented individuals spent their time in contributing to this controversy by discussing words instead of biologic and clinical concepts. The study by Barton et al. [1] is an attempt to objectivate epidermal dysplasia if such a term is suitable. Unfortunately, dysplasia is a time-honored term [2,3] that conceals controversial concepts. For Barton et al. [1], dysplasia ‘signifies that the tissue in question has entered a phase of irreversible or otherwise that will lead to neoplasia’. This is at variance with other authors who separate dysplasia from intraepithelial neoplasia because the latter usually does progress to invasive cancer in time, whereas dysplasia tends either to regress or to remain stationary, very few cases having a tendency to progress [4].

In fact, much of the controversy arises when dysplasia is considered as a synonym for atypia, because dysplasia is also used for other diseases in which no malignant potential whatsoever is implied, i.e. fibrous dysplasia of the bone, bronchoalveolar dysplasia or renal dysplasia [5].

Trying to get out dogmatic interpretations, it should therefore be more appropriate to define nuclear and architectural atypias rather than mixing up all observations in a concept of dysplasia. Even then, it is difficult to define what we should evaluate in terms of potential risk of malignancy. This is particularly evident in solar keratosis, the example chosen by Barton et al. [1]. For many dermatopathologists indeed, it seems impossible to predict which solar keratosis will form a real cancer on the basis of the extent of disturbed epithelial growth pattern and cellular abnormalities.

It also appears that invasive squamous-cell carcinomas arise as readily in solar keratoses, in which atypias are confined to the lower portion of the epidermis, as they do in Bowen’s disease, in which atypias involve the entire thickness of the epidermis.

Therefore, our understanding of the progress of carcino-genesis still remains limited by our inability to distinguish reversible atypical hyperplasia from irreversible intraepithelial neoplasia. Furthermore no morphological criterion allows the recognition of fully cancerized cells capable of invasion. As a consequence, we cannot impose a signified, all-encompassing nomenclature on the complex and unknown biology of cancer. Quantifying some aspects of a controversial concept of dysplasia was the aim of the work of Barton et al. [1]. The issue is promising for those who believe in measurements. Others will probably add this information to the bulk of contesting terminologic and philosophic papers dealing about premalignancy. We are looking
forwards for a better understanding of the linkage between the ‘dysplasia index’ and its biologic or clinical outcomes. Many other attempts are currently presented with diverse approaches [6, 7]. This moving field will probably help the dermatopathologist of the future.

References


162
Piérard
Editorial