In the previous chapters, I have attempted—by referencing published works and lectures—to outline my journey in the development of views on spinocerebellar ataxias: from the initial conceptual conflict between “syndrome” and “disease,” through the issues surrounding the validity of neurological clinical examination itself, to a humble return to historical literary sources with detailed descriptions of neurological findings and disease progression in patients, upon which the original clinical entities were based.

In an effort to find orientation within this complex field, I created several more or less successful schemes of possible diagnostic procedures, explored rare forms of metabolic diseases, and examined hundreds of patients. A fundamental and unexpected problem at the beginning was the lack of knowledge and the relatively difficult acceptance of newly discovered neurogenetic entities by the broader neurological and genetic communities.

The chapter of neurodegenerative diseases has never been particularly “popular” due to the complexity of broad differential diagnostics and the near-total absence of causal therapeutic options.

It is true that today, DNA analysis methods offer much broader possibilities for disease verification, yet uncertainty regarding targeted therapy remains. As a result of current advancements in all fields of medical and related sciences, there is a gradual unification of perspectives on originally distinct neurodegenerative, neurometabolic, and neurogenetic diseases—i.e., diseases once diagnosed at the level of clinical symptoms or syndromes, later at the biochemical level (such as enzyme deficiencies or activator dysfunctions), and now at the level of gene mutations.

However, the pace of progress is so dynamic that its practical implementation into the daily routines of general practitioners or subspecialists remains very challenging.