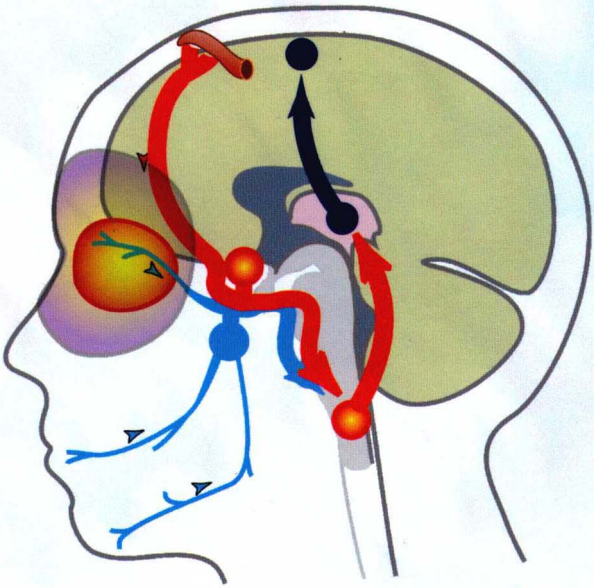

NEUROBIOLOGICAL BASIS OF MIGRAINE

EDITED BY

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The New York Academy of Sciences

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Neurobiological Basis of Migraine

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Foreword

When I studied psychology between 1969 and 1975, I took a course on psychosomatic diseases. The professor presented migraine as a typical example of disease which was clearly a psychological problem without a biological basis. There were compelling arguments, like migraine attacks triggered by stress and a strong co-morbidity with anxiety disorders. How much has changed since these times?

When I started to see migraine patients as a young neurology resident, it became immediately clear to me that migraine was clearly more than a psychological problem. Why had the psychologists neglected the results from twin studies? The phenotype of migraine attacks was extremely homogeneous across patients.

Now is the time to summarize the progress in the neurobiological basis of migraine we have made in the last 40 years. The editors have recruited the best scientists and clinicians in the field of migraine research for a display of amazing research results. We are now able to assign all phases of a migraine attack, from prodromes, aura, headache, autonomic symptoms, photo- and phonophobia and postdromes, to anatomical structures, modifications in the pain transmission and modulation system and higher cortical functions.

A major challenge is still the treatment of acute migraine attacks and migraine prevention. Triptans were developed as attack treatment, under the assumption that they would constrict dilated vessels in the dura and the base of the brain. Later, it turned out that they have major effects on pain transmission in the trigemino-thalamic pathways. We desperately need more effective and better tolerated drugs for migraine prevention. The migraine-preventive properties of available medications like beta-blockers, flunarizine, valproic acid, topiramate, amitriptyline and onabotulinum-toxin A were detected "by chance" when these drugs were used for other indications in patients with migraine. CGRP was identified as a major player in the pathophysiology of migraine. At present, four antibodies against CGRP or the CGRP receptor are under development for migraine prevention. This is a good example of translational research, where observations from pathophysiological studies have resulted in new treatment approaches.

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Who should read this book? Anyone who is interested in migraine as a disease and in migraine patients. I hope that many young researchers and clinicians will become motivated to move into the very promising field of headache research.

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