Matthias Seidel, J Pain Relief 2018, Volume 7
DOI: 10.4172/2167-0846-C1-020

Neurogenic information in autoimmune diseases and chronic pain syndromes

Matthias Seidel
Pain Clinic Basel, Switzerland

Pain is a key symptom in many musculoskeletal diseases. Persistent nociceptor stimulation activates the peripheral and central nervous system (figure 1A, 1B)), and induces the release of neurotransmitters such as substance P (SP) or calcitonin-gene related peptide (CGRP). Such molecules mediate peripheral tissue inflammation with reddening, swelling, hyperalgesia or allodynia (1). These events are termed neurogenic inflammation (NI). Nerve growth factor (NGF) is a primary stimulator of SP and CGRP. A variety of degenerative and autoimmune inflammatory diseases are characterized by NI and either show a robust upregulation of neuronal mediators or clinical features of disease exasperation after stimulation of the nervous system (2). The latter include internal or external Köbner phenomenon in psoriatic arthritis, pathergy in Behçet's disease, UV light-induced dermatitis in systemic lupus erythematosus or chronic regional pain syndrome (CRPS) that is typically induced by trauma. Further strong evidence for NI in musculoskeletal diseases is provided by clinical trials with molecules that block neuron-derived molecules. Recently, erenumab has been approved for the treatment of chronic migraine (3). This monoclonal antibody binds to the CGRP receptor. Treatment with monoclonal antibodies against NGF such as tanezumab (4) or fasinumab (5) have been effective in chronic pain syndromes including osteoarthritis or low back pain. In conclusion, NI is now more increasingly recognized as an important mechanism in clinical medicine.

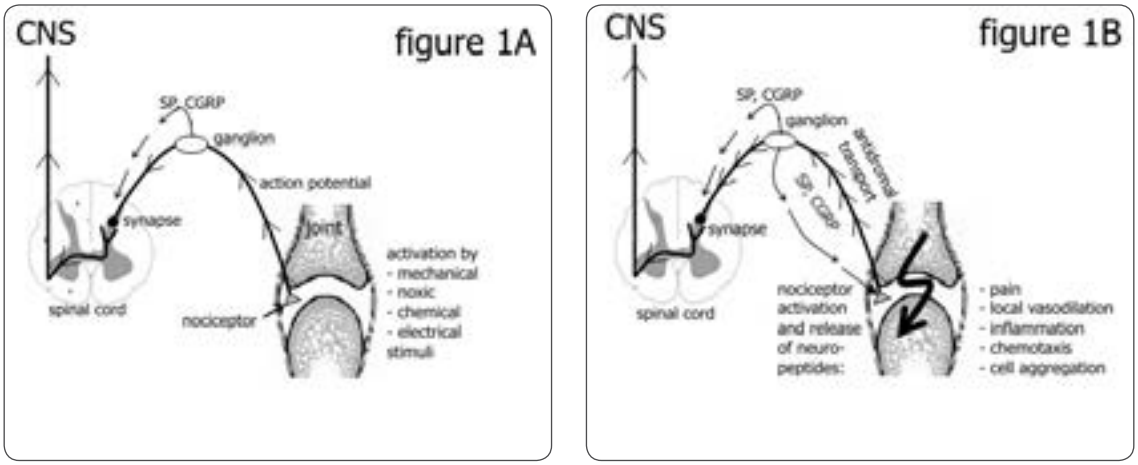

Figure: Principles of pain transmission under physiological and chronic conditions. Neurotransmitters (e. g. SP or CGRP) are released at the synapse to conduct the action potential to the CNS (1A). In case of chronic pain, neuropeptides are transported to and released from the nociceptor (1B). The molecules are powerful inductors of local inflammation.

Recent Publications

1. Seidel M, Tsalik J, Vetter H, Müller W. (2007) Substance P in Rheumatic Diseases. Current Rheumatology Reviews. 3: 17.
2. Seidel MF, Herguijuela M, Forkert R, Otten U. (2009) Nerve growth factor in rheumatic diseases. Doi: 10.1016/j.semarthrit.2009.03.002.

3. Goadsby PJ, Reuter U, Hallström Y, Broessner G, Bonner JH, Zhang F, Sapra S, Picard H, Mikol DD, Lenz RA. (2017) A Controlled Trial of Erenumab for Episodic Migraine. The New England Journal of Medicine. 377(22): 2123.
4. Seidel MF, Wise BL, Lane NE. (2013) Nerve growth factor: an update on the science and therapy. Osteoarthritis Cartilage 21(9): 1223.
5. Tiseo PJ, Kivitz AJ, Ervin JE, Ren H, Mellis SJ. (2014) Fasinumab (REGN475), an antibody against nerve growth factor for the treatment of pain: results from a double-blind, placebo-controlled exploratory study in osteoarthritis of the knee. Pain 155(7): 1245.

Biography

Matthias Seidel has obtained his graduate medical and neuroscience training in Italy, Germany and the United States. He received his medical degree in Essen (Germany) and then served as a Marie-Curie-Fellow in Créteil (Paris/France). He is licensed in Internal Medicine and Rheumatology and has worked in several rheumatology departments in Bonn (Germany), Basel and Biel/Bienne (both Switzerland) where he was recently appointed a chief position. His major scientific interests are neurogenic inflammation in clinical medicine, biomarker molecules and rare diseases in rheumatology.

DrMatthiasSeidel@gmail.com

Notes: