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Tomic S[1,2], Petkovic I[2], Pucic T[1], Resan B[2,3], Juric S[1,2], Rotim T[4]. Cervical dystonia and quality of life. Acta Neurol Belg. 2016 Dec;116:589-592.

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Cervical dystonia is focal dystonia characterized by involuntary movement of the neck muscle, which leads to abnormal head posture. It can be accompanied with pain and tremor. In this study, we evaluated the presence of depression and anxiety in patients with cervical dystonia and the influence of dystonia symptoms on the quality of life. Psychiatric symptoms were evaluated by use of the Beck Depression Inventory (BDI-II) and Beck Anxiety Inventory. Toronto Western Spasmodic Torticollis Rating Scale (TWSTRS) was used to evaluate the cervical dystonia symptoms. Quality of life was assessed by the craniocervical dystonia questionnaire (CDQ-24) and short form 36 health survey (SF-36). Nineteen patients were analyzed. Most of the patients had mild cervical dystonia (mean TWSTRS 23.89). Depression was present in 42.1 % and anxiety in 57.9 % of the patients. Disability due to cervical dystonia correlated with the occurrence of depression ($\rho = 0.534$) and anxiety ($r = 0.652$). Disability was found to significantly influence the stigma, emotional state, pain, daily activity, social life, physical function, and physical and mental disability. Pain influenced some aspects of body pain, physical function, and physical and mental disability. Being associated with disability and pain, cervical dystonia decreases the quality of life in many aspects. Disability also influenced depression and anxiety, which were present in half of study patients. In addition to follow up for cervical dystonia symptoms, patients with cervical dystonia should also be assessed for psychiatric symptoms on routine clinical check-ups. In addition to botulinum toxin, psychopharmaceuticals should be considered as a treatment option in these patients.

Habek M[1,2,3], Crnošija L[1], Lovrić M[4], Junaković A[2], Krbot Skorić M[2], Adamec I[2]. Sympathetic cardiovascular and sudomotor functions are frequently affected in early multiple sclerosis. Clin Auton Res. 2016;26:385-393.

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OBJECTIVE: The aim of this study was to determine the prevalence of autonomic dysfunction using the composite autonomic scoring scale (CASS) and heart rate variability (HRV) in patients with clinically isolated syndrome (CIS) and to correlate autonomic dysfunction with other measures of MS disease activity. METHODS: CASS, HRV and plasma catecholamines during supine and tilted phase were performed in 104 CIS patients. MRI findings were analyzed for total number of lesions and the presence of brainstem and cervical spinal cord lesions. RESULTS: Autonomic dysfunction (CASS >1) was present in 59.8 % of patients, parasympathetic dysfunction in 5 %, sympathetic in 42.6 % and sudomotor in 32.7 % of patients. Patients with autonomic dysfunction on CASS had lower level of norepinephrine in the supine position compared to patients without autonomic dysfunction (1.06 ± 0.53 vs. 1.37 ± 0.86, $p = 0.048$). The CASS score showed positive correlation with s-HF ($r = 0.226$, $p = 0.031$), s-SDNN ($r = 0.221$, $p = 0.035$), t-HF ($r = 0.225$, $p = 0.032$), and t-HFnu ($r = 0.216$, $p = 0.04$), and a negative correlation with t-LF/HF ($r = -0.218$, $p = 0.038$). More patients with MRI brainstem lesions had a positive adrenergic index ($p = 0.038$). Patients with MRI brainstem lesions also had a lower t-SDNN (26.2 ± 14.2 vs. 32 ± 13.3, $p = 0.036$) and a lower t-LF (median 415.0 vs. 575.5, $p = 0.018$) compared to patients without these lesions. Patients with adrenergic index ≥ 1 had a significantly higher standing heart rate compared to patients with an adrenergic index of 0 (96 ± 13.5 vs. 90 ± 12, $p = 0.032$). CONCLUSION: Autonomic (primarily sympathetic) dysfunc-

tion is present in a large proportion of early MS patients and it seems to be related to brainstem involvement.

Ikić Matijašević M[1,2], Flegar D[1,2], Kovačić N[2,3], Katavić V[2,3], Kelava T[1,2], Šućur A[1,2], Ivčević S[1,2], Cvija H[1,2], Lazić Mosler E[2,3], Kalajzić I[4], Marušić A[5], Grčević D[1,2]. Increased chemotaxis and activity of circulatory myeloid progenitor cells may contribute to enhanced osteoclastogenesis and bone loss in the C57BL/6 mouse model of collagen-induced arthritis. Clin Exp Immunol. 2016;186:321-335.

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Our study aimed to determine the functional activity of different osteoclast progenitor (OCP) subpopulations and signals important for their migration to bone lesions, causing local and systemic bone resorption during the course of collagen-induced arthritis in C57BL/6 mice. Arthritis was induced with chicken type II collagen (CII), and assessed by clinical scoring and detection of anti-CII antibodies. We observed decreased trabecular bone volume of axial and appendicular skeleton by histomorphometry and micro-computed tomography as well as decreased bone formation and increased bone resorption rate in arthritic mice in vivo. In the affected joints, bone loss was accompanied with severe osteitis and bone marrow hypercellularity, coinciding with the areas of active osteoclasts and bone erosions. Flow cytometry analysis showed increased frequency of putative OCP cells (CD3$^-$ B220$^-$ NK1.1$^-$ CD11b$^{-/lo}$ CD117$^+$ CD115$^+$ for bone marrow and CD3$^-$ B220$^-$ NK1.1$^-$ CD11b$^+$ CD115$^+$ Gr-1$^+$ for peripheral haematopoietic tissues), which exhibited enhanced differentiation potential in vitro. Moreover, the total CD11b$^+$ population was expanded in arthritic mice as well as CD11b$^+$ F4/80$^+$ macrophage, CD11b$^+$ NK1.1$^+$ natural killer cell and CD11b$^+$ CD11c$^+$ myeloid dendritic cell populations in both bone marrow and peripheral blood. In addition, arthritic mice had increased expression of tumour necrosis factor-α, interleukin-6, CC chemokine ligand-2 (Ccl2) and Ccl5, with increased migration and differentiation of circulatory OCPs in response to CCL2 and, particularly, CCL5 signals. Our study characterized the frequency and functional properties of OCPs under inflammatory conditions associated with arthritis, which may help to clarify crucial molecular signals provided by immune cells to mediate systemically enhanced osteoresorption.

Labor M[1,2], Vrbica Ž[3,4], Gudelj I[5], Labor S[1,2], Jurić I[6], Plavec D[2,7]. Exhaled Breath Temperature as a Novel Marker of Future Development of COPD: Results of a Follow-Up Study in Smokers. COPD. 2016;13:741-749.

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Although only less than one-third of smokers develop COPD, early marker(s) of COPD development are lacking. The aim of this research was to assess the ability of an average equilibrium exhaled breath temperature (EBT) in identifying susceptibility to cigarette smoke so as to predict COPD development in smokers at risk. The study was a part of a multicenter prospective cohort study in current smokers (N = 140, both sexes, 40-65 years, ≥20 pack-years) with no prior diagnosis of COPD. Diagnostic workup includes history, physical, quality of life, hematology and highly sensitive CRP, EBT before and after smoking a cigarette, lung function with bronchodilator test, and 6-minute walk test. Patients without a diagnosis of COPD and in GOLD 1 stage at initial assessment were reassessed after 2 years. COPD was additionally diagnosed based on lower level of normal (LLN) lung function criteria. Utility of EBT for disease progression was analyzed using receiver operator curve (ROC) and logistic regression analyses. Change in EBT after smoking a cigarette at initial visit (ΔEBT) was significantly predictive for disease progression (newly diagnosed COPD; newly diagnosed COPD + severity progression) after 2 years (p < 0.05 for both). ΔEBT had an AUC of 0.859 (p = 0.011) with sensitivity of 66.7% and specificity of 98.1% for newly diagnosed COPD using LLN criteria. We conclude that EBT shows potential for predicting the future development of COPD in current smokers. This was best seen using LLN to diagnose COPD, adding further evidence to question the use of GOLD criteria for diagnosing COPD.