Camptocormia

Camptocormia in Parkinson's disease: new insights

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Camptocormia—either a central disorder or an outcome of peripheral mechanism

Of the several axial deformities, stooped posture is the most common postural abnormality. Other more extreme forms include drop head, Pisa syndrome and camptocormia (bent spine). The first two are usually related to multisystem atrophy, but camptocormia is now known as one of the extreme posture impairments in patients with Parkinson's disease. Although early reports often attributed this symptom to a conversion disorder or malingering, it is now accepted also as one of the axial features of Parkinson's disease. Since the first description,[1] many case reports and series have been published, further characterising this phenomenon and exploring its pathogenesis. Camptocormia in Parkinson's disease is defined by marked anteroflexion of the trunk, which abates in the recumbent position, with no or minimal response to levodopa. The clinical features of patients with Parkinson's disease and with camptocormia often include old age, predominantly male sex, long disease duration and early axial involvement. Interestingly, nearly all patients have spondyloarthrotic changes of the spine, rendering it a risk factor for developing camptocormia. Although abundant clinical data have been acquired, there is a paucity of physiological clues to explain the pathogenesis of this enigmatic symptom. Only limited data exist on the topographical representation of the axial muscles in the basal ganglia. Magnetic resonance imaging and single-photon emission computed tomography of the brain with dopamine transporters do not differ in patients with Parkinson's disease with or without camptocormia. There seem to be two schools of thought that attempt to provide mechanisms underlying camptocormia. The first considers camptocormia to be primarily a central disorder—that is, focal action dystonia of the spine.[2] This type of dystonia might be due to a disorder of the striatum and its projections to the reticulospinal tract, or the thalamus, where the trunk is markedly represented. Electromyographic findings of dystonic discharges from paraspinal muscles, and a few case reports of patients in whom bent spine was improved after deep-brain stimulation of the pallidum or subthalamic nucleus,[3] support this theory. The other theory connects camptocormia to peripheral mechanisms—in particular, myopathy of the antigravity muscles associated with trunk extension.[4] This is supported by findings of hypodensity on computed tomography scans, myopathic changes in biopsy specimens and electromyograms of the paraspinal muscles, and some improvement with steroid treatment. The extensive central and peripheral evaluation carried out by Bloch *et al*[5] (***see p 1223)*** and Lepoutre *et al*[6] (***see p 1229)*** are steps towards better understanding the pathogenesis underlying camptocormia. The conclusion from these studies is that both concepts do not necessarily contradict, and might even be linked, as atrophy of the paraspinal muscles might be secondary to a primary action dystonia of the spine.

With currently available technologies, our ability to further characterise the pathophysiology of abnormal postures in patients with Parkinson's disease and in those with other extrapyramidal syndromes is limited. Therefore, an animal model of abnormal posturing is required to fully understand the nature of these perplexing phenomena and to offer successful treatment.

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REFERENCES

1 **Djaldetti R**, Mosberg-Galili R, Sroka H, *et al*. Camptocormia (bent spine) in patients with Parkinson's disease—characterization and possible pathogenesis of an unusual phenomenon. *Mov Disord* 1999;**14**:443–7.
2 **Azher SN**, Jankovic J. Camptocormia: pathogenesis, classification, and response to therapy. *Neurology* 2005;**65**:355–9.
3 **Micheli F**, Cersosimo MG, Piedimonte F. Camptocormia in a patient with Parkinson disease: beneficial effects of pallidal deep brain stimulation. Case report. *J Neurosurg* 2005;**103**:1081–3.
4 **Schabitz WR**, Glatz K, Schuhan C, *et al*. Severe forward flexion of the trunk in Parkinson's disease: focal myopathy of the paraspinal muscles mimicking camptocormia. *Mov Disord* 2003;**18**:408–14.
5 **Bloch F**, Houeto J-L, du Montel ST, *et al*. Parkinson's disease with camptocormia. *J Neurol Neurosurg Psychiatry* 2006;**77**:1223–8.
6 **Lepoutre A-C**, Devos D, Blanchard A, *et al*. A specific clinical pattern of camptocormia in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 2006;**77**:1229–34.