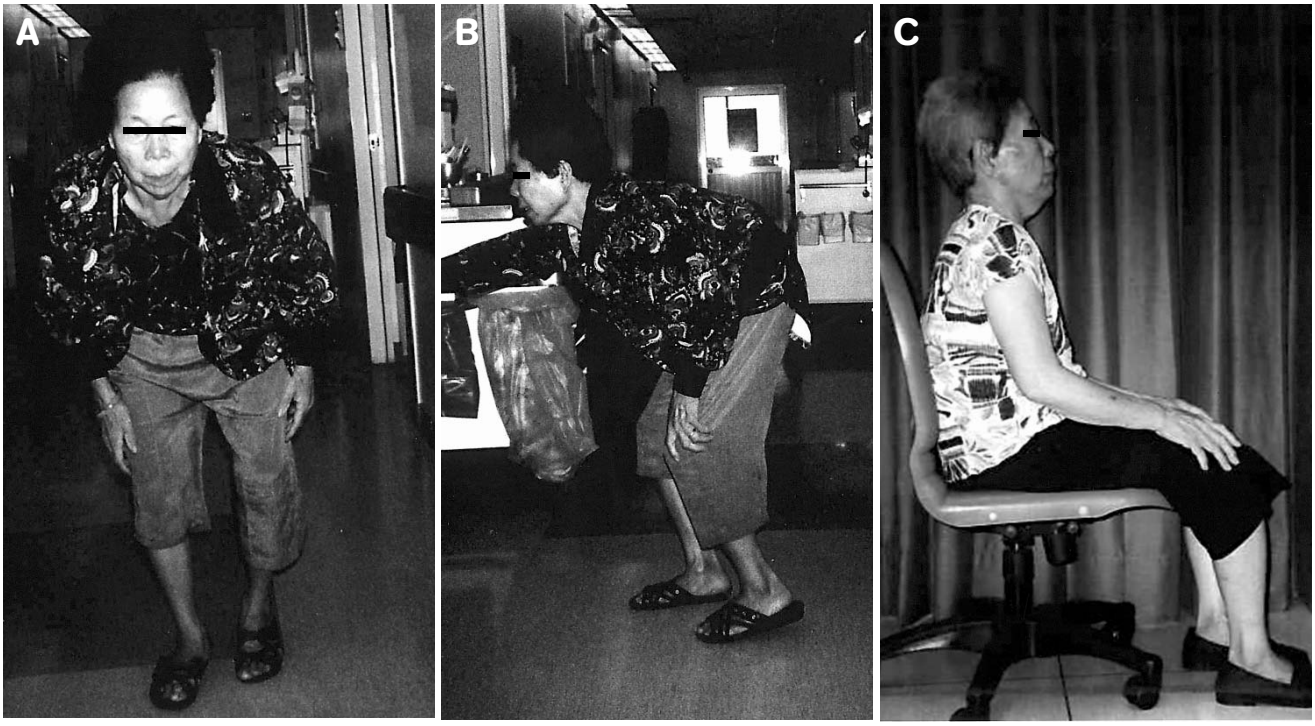
Camptocormia in Parkinson's Disease

Juei-Jueng Lin



Camptocormia is characterized by pronounced forward flexion of the thoracolumbar spine, which increases while walking and disappears in recumbent position. In most of the reported patients, this phenomenon was the result of a psychogenic conversion reaction[1,2]. Aside from a psychiatric cause, other etiologies include cerebral infarction, CNS infection , spinal tumors, spinal hemorrhages, spine disorders (e.g.. ankylosing spondylitis, spinal stenosis), neuromuscular disorders (e.g.. myopathies, muscular dystrophies), metabolic disorders, paraneoplastic syndromes, drug-induced and movement disorders (Parkinson's disease [PD], post encephalitis parkinsonism [PEP], dystonia)[3]. Camptocormia presented in patients with PD was described for the first time in 1999 by Dajaldetti et al[4]. They suggested that this phenomenon in PD might be the result of striatal, especially putaminal, damage. A 72-year-old female was diagnosed as suffering from PD on the basis of resting tremor, rigidity and bradykinesia 13 years ago. Her clinical symptoms were slow progression and responded well to levodopa therapy. Seven year later she developed extreme posture by the painful spasm of paraspinal

From the Department of Neurology, Chushang Show-Chwan Hospital, Nantou, Taiwan.
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Reprint requests and correspondence to: Juei-Jueng Lin, MD. Department of Neurology, Chushang Show-Chwan Hospital, No. 75, Sec. 2, Chi-Shang Rd., Chushang Jenn, Nantou, Taiwan.

muscles, resulting in the posture more anteriorly flexed to the point that her trunk was almost horizontal to the floor (Fig. A and B). She could walk with her trunk bent forward and the picture disappeared when she sat on the chair (Fig. C) or lay down in bed. The clinical picture resembled to be camptocormia. She also had no difficulty lying down fully extended. When asked to extend herself while standing, she could voluntarily and temporarily overcome the problem and straighten up. Meanwhile, the extent of abnormal posture was aggravated by some activities of daily living, including in dressing and hygiene. There was no diurnal fluctuation in its severity, neither correlation between the appearance of abnormal posture and the regimen of levodopa administration (time or dosage). At the onset of camptocormia, she was treated with levodopa (with peripheral decarboxylase inhibitor, madopar (600 mg/day), pergolide (0.75 mg/day) and amatadine (200 mg/day). The score for the Activity of Daily Living of the Unified Parkinson's Disease Rating Scale (UPDRS) was 17 (part II, maximum=52) and was 34 in Motor Examination (part III, maximum=108). A series of studies including MRI of the brain, X-ray of the thoracolumbar spine, biochemistries of the blood, electrophysiological studies and electromyography of the paraspinal muscles all revealed non-significant finding. She had received rehabilitation and medical treatment including trihexyphenidyl, baclofen, dantrolene, diazepam, clonazepam, and increasing dose of levodopa for her camptocormia, but none was effective. The present case illustrated an unusual manifestation of camptocormia in PD, which was unrelated to medication. The abnormal posture increased during walking, disappeared in recumbent position and was overcame by extend herself while standing that was similar to sensory tricks in dystonia[5]. This behavior suggested the camptocormia in PD might share some feature suggesting action dystonia.

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