

Brown and Baker [1] described a predilection for involvement of the triceps, deltoids, extensors of the wrist and fingers, and of the iliopsoas, hamstrings, and peroneal muscles in this disorder. It was their impression that regardless of the regions of the body otherwise involved, the triceps muscles were generally the most affected. In 1971 we [3] reported that 23 of 50 patients with Guillain-Barré syndrome had weakness that predominated in selected muscle groups; these were the thigh flexors, hamstrings, foot dorsiflexors, or wrist extensors. Particular affinity for triceps involvement was not observed.

Since then we have paid particular attention to evidence of selective weakness in Guillain-Barré syndrome. Sixteen of the subsequent 20 patients with Guillain-Barré syndrome admitted to our service also showed dissociated muscle weakness, most prominently in the lower extremities. In 14 of the 16 patients the weakest muscle groups were the hamstring muscles. This was especially noteworthy when hamstring function was compared with that of the quadriceps. There was dissociation between these two groups, with little weakness of the quadriceps and marked weakness of the hamstrings.

In contradistinction, in only 1 instance among our last 15 patients with polymyositis, was any appreciable dissociation found between quadriceps and hamstrings, and in this patient the hamstrings were the weaker. Among the last 20 patients with a diagnosis of diabetic mononeuritis multiplex, the well-known predilection for quadriceps involvement over that of the hamstrings was found in all but 1 instance. In this single case the predilection was for the hamstrings, the same as in Guillain-Barré syndrome.

Occasionally in porphyric neuropathy and often in lead neuropathy, extensor muscles are mainly involved [4]. In periodic paralysis, extensor muscles are more affected than flexors, while in myasthenia gravis the flexor thigh muscles are often the weakest [5]. However, selective hamstring involvement is unusual in any of these conditions.

Dissociated weakness, preferentially involving the hamstring muscles in contradistinction to the quadriceps, appears to be a valuable clue to the clinical diagnosis of Guillain-Barré syndrome.

*Neurology Service
Veterans Administration Medical Center
50 Irving St, NW
Washington, DC 20422*

References

1. Brown JR, Baker AB: The diagnosis of Guillain-Barré disease. *Am J Med* 2:45-52, 1947
2. Masucci EF: Idiopathic polyneuritis. In Conn HF, Conn RB (eds): *Current Diagnosis*. Sixth edition. Philadelphia, Saunders, 1980, pp 912-915
3. Masucci EF, Kurtzke JF: Diagnostic criteria for the Guillain-Barré syndrome. *J Neurol Sci* 13:483-501, 1971
4. Ridley A: Porphyric neuropathy. In Dyck PJ, Thomas PK, Lambert EH (eds): *Peripheral Neuropathy*. Philadelphia, Saunders, 1975, vol 2, p 949
5. Simpson JA: Myasthenia gravis and myasthenic syndromes. In Walton JN (ed): *Disorders of Voluntary Muscle*. Third edition. Edinburgh and London, Churchill/Livingstone, 1974, p 657

Lithium Carbonate in Pseudobulbar Palsy

E. Wayne Massey, MD, and Stephen Lowe, MD

Lithium carbonate has been of therapeutic value in a variety of psychoneurological entities and has recently been shown to be efficacious in emotionally unstable individuals [4]. We have seen good results in treating the emotional lability of four patients with pseudobulbar palsy. One patient is described.

A 57-year-old man was referred with a left paraparesis of approximately four weeks' duration. Three years previously he had had a right hemiparesis and associated mild, nonfluent aphasia which had improved enough that he was able to work part-time. With the onset of his second stroke he developed pseudobulbar palsy with hypernasal speech, oropharyngeal spasticity, signs of cortical bulbar tract involvement, and emotional lability. On his transfer to the Rehabilitation Unit it was difficult to elicit a good therapeutic response due to the patient's emotional lability. Therefore lithium carbonate, 300 mg twice daily, was initiated and a blood level of 0.42 mg/dl was obtained within 72 hours (therapeutic range, 0.05 to 1.61). He remained without heightened emotional lability in the subsequent six weeks and experienced no untoward side effects from the lithium carbonate.

The rationale for using lithium in affective disorders is reasonably well established, but its use in aggressive patients [5] or emotionally unstable patients with personality disorders has been unclear. One proposed mode of psychopharmacological action is derived from the observation that an epileptoid disorder [1, 3] may be present, although lithium's anticonvulsant properties are controversial [2]. In patients with pseudobulbar palsy, lithium carbonate may function as a mood stabilizer.

*Department of Neurology
Duke University Medical Center
Durham, NC 27710*

References

1. Bach-y-Rita G, Lion JR, Climent CE, Ervin FR: Episodic dyscontrol: a study of 130 violent patients. *Am J Psychiatry* 127:1473-1478, 1971
2. Jus A, Villeneuve A, Gautier J, Pires A, Cate JM: Some remarks on the influence of lithium carbonate on patients with temporal lobe epilepsy. *Int J Clin Pharmacol* 7:67-74, 1973
3. Monroe RR: *Episodic Behavior Disorders*. Cambridge, MA, Harvard University Press, 1970
4. Rifkin A, Quitkin F, Carrillo C, Blumberg AG, Klein DF: Lithium carbonate in emotionally unstable character disorders. *Arch Gen Psychiatry* 27:519-523, 1972
5. Sheard MH: Effect of lithium in human aggression. *Nature* 230:113-114, 1971