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Freezing of Gait that disappears: Should there be rest before rehab?

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Freezing of gait (FOG) is a leading cause of falls and is associated with poor quality of life in Parkinson's disease (PD)[1]. The pathophysiology is not understood, however, one possibility is a loss of automaticity of gait and changes in connectivity between the mesencephalic locomotor region (MLR) and frontal cortical structures[1]. Treatment of FOG is problematic from a medical and surgical standpoint. This has led to the development of rehabilitation approaches including the development of compensatory techniques to “reset”

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the motor system and overcome freezing phenomena[2]. With impairment of automatic pathways this approach moves gait to being a learned task. However, while promising, these also have demonstrated inconsistent results. Thus, once FOG occurs, it continues to progress leading to significant disability and a wheelchair bound state.

We observed an unusual phenomenon where two patients (see table) in whom levodopa-nonresponsive FOG was a significant burden and disappeared after a catastrophic illness that led to prolonged hospitalization and bedridden state. One developed a ruptured appendix followed by a fractured hip while the other had urosepsis. Bedridden states of 2 and 6 weeks leading an inability to walk were followed by rehabilitation and, in turn, recovery that included sustained abolition of FOG over 2-3 years. Both were left with some level of postural instability but could walk independently.

While it is well known that immobilizing advanced PD patients, even for a few days, can lead to a significant setback from an ambulatory standpoint, both of our patients recovered over time. We posit the question: does immobilization for weeks allow for an optimization of rehabilitation therapy to lead to sustained “resetting” of the motor system and improvement of FOG?

There is precedent for such an approach. In occupational dystonia immobilization of the affected limb for four weeks followed by active practice (playing musical instrument) lead to sustained improvement for up to 12 months[3]. The response was thought to be related to inactivity-dependent plastic changes reversing functional abnormalities on the cortical level including shrinkage of motor cortical representation which is abnormally large in occupational dystonia. It was referred to as a “detraining” of the movement. The authors further stated that the combination of immobilization and rehabilitation might be additive in the response. While FOG is not a form of dystonia they share several similarities that might suggest a similar response to this strategy including both disorders can be influenced by tricks or cues and both are associated by co-activation of agonist and antagonist muscles.

It has been demonstrated that corticospinal plasticity occurs with immobilization using a bed rest model[4]. Ninety days of bed rest in healthy subjects resulted in a decreased corticospinal excitability demonstrated with transcranial magnetic stimulation (TMS) and functional MRI (fMRI) with recovery to normal activity which was delayed in at least one patient to over two weeks. Alterations in afferent signaling from immobilization of the legs induced this reorganization of the cortex by altering cortical excitability and post bed rest increased corticospinal excitability was thought to reflect motor relearning. Casting the legs after a fracture had a similar effect after at least nine days[5]. Such cortical plasticity may be relevant to FOG as imaging studies associate FOG with increased functional connectivity between the MLR and the supplementary motor area[1]. These findings suggest that increased cortical control of gait in FOG subjects may be a maladaptive response. As such, a period of immobility may aid in releasing cortical control, while follow-up systematic gait rehabilitation can induce functional reorganization of these pathways leading to improvement in FOG.

Such changes have not been examined in PD with FOG. Questions remain to be answered before embarking on the study of immobilization followed by gait training including: how long a period of immobility would be needed to reverse FOG? How safe is it to immobilize PD patients for prolonged periods? Would bed rest be required or could patients simply refrain from walking? What would be the retraining method with the best outcome? Such an intervention of rest before rehabilitation would be simple to test. The recovery of our patients who were advanced and seriously ill suggest such an approach could be safe and further investigation in a prospective trial is warranted.

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Table

Case #	Age/sex	PD Duration	FOG Duration	FOG Situations	Other troublesome PD Features	Acute Illness	Duration Bedridden	Physical Therapy
1	79/female	16 yrs	6 yrs	Straight walk, initiation, doorway, approaching destination hesitation	Dyskinesia	Ruptured appendix, fractured hip	8 weeks	Home
2	86/male	20 yrs	7 yrs	Initiation, Turning, Destination hesitation	Dyskinesia	Urosepsis	2 weeks	Inpatient