Effect of Rhythmic Initiation Along With Rhythmic Activity Facilitation on Functional Mobility in Early Parkinson's Patients

[1] Dr. Akshata Adhikari, [2] Dr. G Varadharajulu, [3] Dr. Suraj Kanase
[1]MPT, Department of Neuro Physiotherapy, Krishna College of Physiotherapy, Krishna Vishwa Vidyapeeth, Karad.
[2]Dean, Krishna College of Physiotherapy, Krishna Vishwa Vidyapeeth, Karad.
[3]Professor / Head of Department of Neuro Physiotherapy, Krishna College of Physiotherapy, Krishna Vishwa Vidyapeeth, Karad.
Corresponding Author: Dr Suraj B Kanase, Professor/HOD, Department of Neurophysiotherapy, KVV, Karad
Email: drsurajkanase7@gmail.com

Abstract

Background:

Parkinson's Disease (PD) is the second most prevalent neurodegenerative disorder worldwide. The incidence of PD is estimated to range between 410 and 529 cases per 100,000 person-years. The condition gradually impairs motor functions, leading to symptoms such as akinesia, postural instability, gait disturbances, fatigue, dysphagia, and speech changes. While studies have shown that Proprioceptive Neuromuscular Facilitation (PNF) techniques have benefits in treating PD, though particularly rhythmic initiation has not been examined individually. Hence the study aims to evaluate the effectiveness of rhythmic initiation, alongside conventional therapy to determine its potential for inclusion in standard treatment protocols to improve patient recovery and independence.

Methodology:

The study was conducted on 12 individuals diagnosed with early-stage Parkinson's Disease, selected based on predetermined inclusion and exclusion criteria. A brief demographic profile was collected from each participant before the initial assessment. The outcome measures utilized were SPDDS and UPDRS. Participants were randomly allocated into two groups: Group A (control group), which received conventional physiotherapy, and Group B (experimental group), which received conventional physiotherapy in addition to rhythmic initiation and rhythmic activity facilitation techniques. The intervention was administered for 45 minutes per day, four days per week, over a period of six weeks. Outcomes were analyzed by comparing the differences in pre-intervention, mid-intervention (3rd week), and post-intervention assessments both within and between groups.

Results:

Data was analyzed using repeated measures ANOVA and unpaired t-tests. The control group showed UPDRS improvement from 30.8 to 18.6 and SPDDS from 43.6 to 31.5 over six weeks. The experimental group improved from 31.8 to 16.6 (UPDRS) and 41.6 to 25.83 (SPDDS). P-values indicated significant improvements: 0.0133 (UPDRS) and 0.0263 (SPDDS) in the control group; 0.0024 (UPDRS) and 0.0121 (SPDDS) in the experimental group. T-values were 0.5489 (UPDRS) and 2.436 (SPDDS); F-values were 5.725 (UPDRS) and 11.649 (SPDDS), indicating greater improvement in the experimental group.

Conclusion:

This study demonstrated that combining rhythmic initiation with rhythmic activity facilitation significantly improved functional mobility in patients with Parkinson's disease.

Keywords: PNF, Rhythmic initiation, Parkinson's Disease, Functional Mobility, ADL

How to cite this article: Adhikari A, Varadharajulu G, Kanase S. Effect of Rhythmic Initiation Along With Rhythmic Activity Facilitation on Functional Mobility in Early Parkinson's Patients. Int J Drug Deliv Technol. 2026;16(8s): 990-996; DOI: 10.25258/ijddt.16.8s.111

Introduction

Parkinson's Disease (PD) is the second most common neurodegenerative disorder after Alzheimer's disease. It was first described in 1817 by James Parkinson in his classic work, *Essay on the Shaking Palsy*. The disease was initially referred to as "paralysis agitans," but later, in the 19th century, Jean-Martin Charcot recognized Parkinson's contributions by renaming the condition "maladie de Parkinson," or Parkinson's disease (PD).[1,3] There are two forms of Parkinson's disease (PD): familial and sporadic. Familial PD is genetically inherited, following either an autosomal dominant or recessive pattern, while sporadic (idiopathic) PD is believed to result from gene-environment interactions.

Genetically linked PD accounts for approximately 10–15% of all PD cases, with the remaining majority classified as sporadic. To date, seven causal genes have been identified in familial PD: Alpha-synuclein (SNCA), Leucine-rich repeat kinase 2 (LRRK2), Parkin RBR E3 ubiquitin protein ligase (PARK2), Phosphatase and tensin homolog-induced kinase 1 (PINK1), Parkinson protein 7 (PARK7). These genes, along with specific metabolites and PD-associated biomarkers, have been explored as potential avenues for the early detection of PD.[2]

Early Onset Parkinson's Disease (EOPD) is defined as the onset of parkinsonian features before the age of 40 years and accounts for approximately 3–5% of all Parkinson's disease (PD) cases. EOPD is further classified into two categories:

- Juvenile Parkinson's Disease: onset before 21 years of age
- Young Onset Parkinson's Disease (YOPD): onset between 21 and 40 years of age.[4]

Although the prevalence of PD in India is relatively low at 70 per 100,000 population, the country's large population of over 1 billion translates to an estimated 7 million individuals living with PD. in Bangalore district, South Karnataka, India, in 2004, the crude prevalence rate of Parkinsonism was found to be 33 per 100,000, while the age-adjusted prevalence was 76 per 100,000. A study among the Parsi community in Mumbai, a small and relatively stable ethnic group, revealed a higher prevalence of 192 per 100,000, compared to the general Indian population.[5]

At the cellular level, one of the defining histopathological features of Parkinson's Disease (PD) is the presence of Lewy bodies and Lewy neurites. In Parkinson's Disease (PD), alongside mitochondrial dysfunction, impairment of the ubiquitin-proteasome system (UPS) and autophagy-lysosomal pathway (ALP) disrupts protein degradation, leading to the accumulation of toxic α-synuclein aggregates. Genetic mutations, such as in LRRK2 and GBA, further compromise these pathways, accelerating disease progression by impairing cellular clearance mechanisms. Neuroinflammation plays a critical role in Parkinson's Disease (PD) pathogenesis. Activated microglia in the substantia nigra release pro-inflammatory cytokines and reactive species, creating a toxic environment that promotes neuronal damage. Astrocyte dysfunction and dysregulated glutamate homeostasis further exacerbate neurodegeneration. Emerging evidence also points to adaptive immune involvement, suggesting a potential autoimmune component in PD.[6,7]

Among some hallmark motor symptoms of Parkinson's Disease, rigidity is often described by patients as "heaviness" or "stiffness" in the limbs. It affects both agonist and antagonist muscles and is consistent regardless of movement type or speed. Two forms exist: cogwheel rigidity, a jerky resistance linked to coexisting tremor, and lead-pipe rigidity, a constant resistance throughout motion. Typically starting asymmetrically in proximal muscles like the shoulders and neck, rigidity progresses to the extremities and face over time. If untreated, it may lead to reduced ROM, contractures, and postural deformities, significantly impacting mobility and quality of life. Bradykinesia, or slowness of movement, is a core symptom of Parkinson's Disease, primarily due to insufficient muscle force recruitment during movement initiation. Although tremor, rigidity, and weakness contribute, the main issue is in movements generated internally rather than with external cues. Emotional states like anxiety can worsen bradykinesia, while positive emotional cues may improve it temporarily. The symptom is closely linked to dopamine deficiency, with reduced neuronal density in the substantia nigra observed in parkinsonism. Dysfunction in the putamen and globus pallidus, parts of the basal ganglia, is believed to underlie the reduced muscle force during movement onset. Rest tremor is the most common and noticeable symptom of Parkinson's Disease, typically starting unilaterally at 4 to 6 Hz and often affecting the hand, with the "pill-rolling" tremor being classic. It can also affect the lips, chin, jaw, and legs but rarely involves the neck, head, or voice, differentiating it from essential tremor. Rest tremor disappears with voluntary movement or during sleep. Alongside rest tremor, many PD patients experience postural tremor, which can be more disabling. Postural instability is a key feature of later-stage Parkinson's Disease, often leading to falls and increasing the risk of hip fractures. Contributing factors include other parkinsonian symptoms, orthostatic hypotension, sensory decline, and impairments in integrating visual, vestibular, and proprioceptive input. Patients with PD often have abnormal postural responses and struggle to maintain their center of mass within their base of support. Tasks requiring narrow stances or divided attention worsen instability, leading to muscle coactivation and rigid posture, which impairs balance recovery.3,8

Functional mobility is the ability to move independently and safely to perform daily activities like walking, standing, and climbing. In Parkinson's Disease (PD), where mobility is often impaired, it serves as a key indicator of treatment effectiveness and disease progression. As noted by Forhan & Gill, reduced functional mobility increases the risk of falls, loss of independence, and institutionalization, underscoring its importance in improving quality of life for PD patients.[9]

Proprioceptive Neuromuscular Facilitation (PNF) is a treatment concept that focuses on utilizing the body's sensory and motor systems to promote functional movement. The approach emphasizes the importance of neurophysiological principles in facilitating movement patterns that are naturally occurring in the body.[10] PNF techniques aim to improve functional movement by facilitating or inhibiting muscle activity and promoting strength and relaxation. They use concentric, eccentric, and static contractions with tailored resistance. One method involves rhythmic limb motion, starting passively and advancing to active resistance, to support movement initiation, coordination, proprioception, motion learning, and relaxation. This technique is especially useful for patients with difficulty initiating movement, uncoordinated or dysrhythmic patterns like ataxia or rigidity, or abnormal muscle tone. It starts with passive movement guided by verbal cues to establish rhythm. As the patient engages, resistance is gradually added while maintaining the rhythm, progressing toward independent, functional movement control.[11,12]

Proprioceptive Neuromuscular Facilitation (PNF) techniques have been shown to improve dynamic balance and gait in PD patients. However, there is limited research on the specific use of the rhythmic initiation technique of PNF to enhance functional mobility. Existing literature suggests that PNF is comparable or superior to other therapies in improving movement in PD, though more high-quality studies are needed to confirm its effectiveness. This study aims to evaluate the impact of PNF techniques combined with conventional therapy on patient recovery and independence, with the goal of supporting the integration of PNF into standard treatment protocols.

Materials and Methods:

This was an experimental study designed as a randomized controlled trial (RCT). A simple random sampling method was used to allocate participants into groups. The study was conducted at Krishna College of Physiotherapy, KVVDU, Karad, over a duration of one year. The intervention was administered three days per week for a period of six weeks. Initially, 19 participants were recruited; however, 7 patients were lost to follow-up, resulting in a final sample size of 12, with 6 participants in each group. Participants included both males and females aged between 40 and 85 years. All participants were clinically diagnosed with Parkinson's Disease, on stable pharmacological treatment, and within stages 1 to 3 of the Hoehn and Yahr grading scale. Participants were excluded if they had undergone deep brain stimulation or stereotactic neurosurgery, had medical conditions interfering with endurance training (e.g., unstable angina, cardiomyopathy, uncontrolled metabolic disorders), sustained a fracture or dislocation of the lower limb, or had any central nervous system disease other than Parkinson's Disease. Both the groups were assessed initially, after 3 weeks and after 6 weeks with UPDRS and SPDDS scales. Data analysis was performed using SPSS software version 25. Within-group comparisons were analyzed using repeated measures ANOVA, while between-group comparisons were evaluated using the unpaired t-test.

Intervention:

Group A received conventional treatment which included relaxation techniques, breathing exercises stretching and flexibility exercises; whereas group B received treatment in the initial weeks that included foundational movements such as passive rolling, supine-to-sit transitions, and bridging are introduced using RI with verbal and tactile cues to build body awareness and basic motor control. Rhythmic cues, verbal timing are employed to initiate and coordinate movement. As the program advances, functional transitions like sit-to-stand and rhythmic gait training using marching, arm swings, and stepping with auditory stimuli are emphasized. Mid-phase weeks include dynamic walking, turning, tandem steps, and stair climbing, gradually increasing complexity and reducing assistance while maintaining rhythm. The final weeks focus on integrating these movements into real-life contexts with obstacle navigation, rhythmic dual-tasking, and community ambulation challenges.

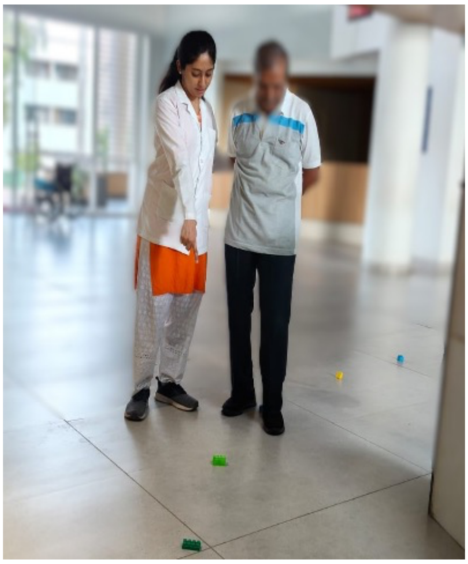

Fig 1: Marching in parallel bar

Fig 2 : Walking with floor markers

Figure 3: ADL activities with ADL box

Figure 4: Pelvic Bridging

Results:

Total 12 patients were included and divided equally into 2 groups.

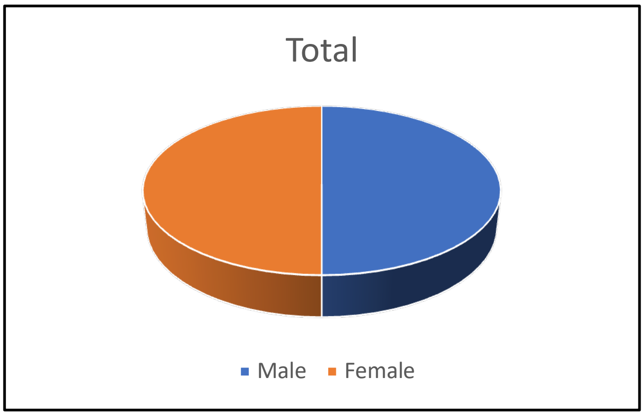

Fig 5 : Gender distribution

The graph shows that an equal male female distribution was followed in the study.

Within group analysis

UPDRS Group A

UPDRS	Pre	3rd week	6th week	P value	F value
Group A	30.8	23.8	18.16	<0.001	32.255

Table No 1: 1st week, 3rd week and 6th week mean scores of UPDRS for group A

SPDDS for group A

SPDDS	Pre	3rd week	6th week	P value	F value
Group A	43.6	36	31.5	0.0192	32.25

Table No 2: 1st week, 3rd week and 6th week mean scores of SPDDS for group A

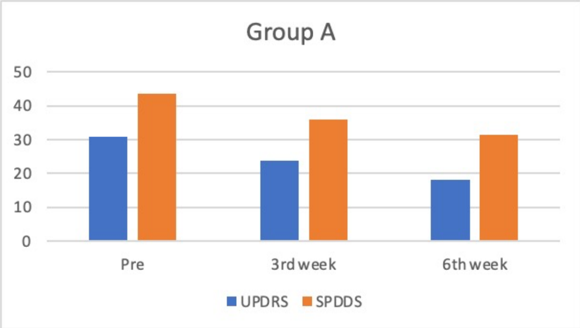

Fig 6: 1st week, 3rd week and 6th week mean scores of SPDDS and UPDRS for group A

Interpretation: Group A demonstrates steady functional improvement over 6 weeks, with significant reductions in both motor symptoms (UPDRS) and disability (SPDDS), though the decline in SPDDS is slightly less pronounced compared to UPDRS.

UPDRS Group B

UPDRS	Pre	3rd week	6th week	P value	F value
Group B	31.83	22.8	16.6	0.0042	9.938

Table No 3: 1st week, 3rd week and 6th week mean scores of UPDRS for group B

SPDDS for group B

SPDDS	Pre	3rd week	6th week	P value	F value
Group B	41.6	30.16	25.83	0.0005	17.475

Table No 4: 1st week, 3rd week and 6th week mean scores of SPDDS for group B

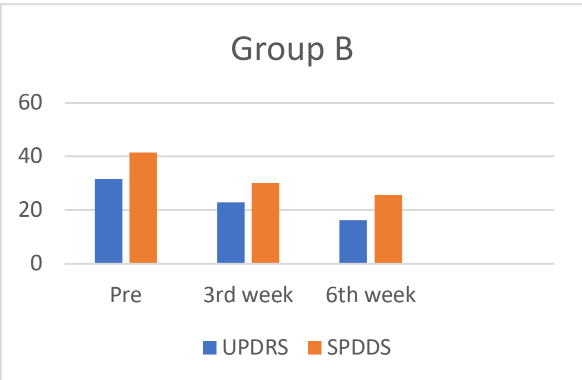

Fig 7: 1st week, 3rd week and 6th week mean scores of SPDDS and UPDRS for group B

Interpretation: Group B shows consistent functional improvement across 6 weeks, as reflected by declining UPDRS and SPDDS scores.

Between the group analysis

UPDRS scale for group A and group B

UPDRS	pre	post	P value	F value	T value
group A	30.8	18.16	0.004	5.725	0.5487
group B	31.83	16.6			

Table No 5: 1st week and 6th week mean scores of Group A and Group B for UPDRS

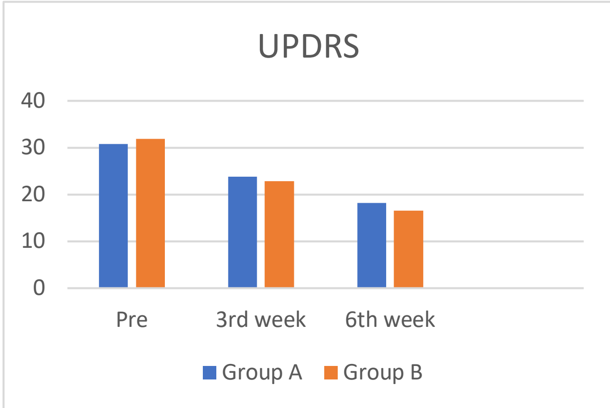

Fig 8: 1st week and 6th week mean scores of Group A and Group B for UPDRS

Interpretation: Both interventions are effective in reducing UPDRS scores over 6 weeks. Group B shows a slightly greater improvement, suggesting that its intervention method may be more effective.

SPDDS scale for group A and group B

SPDDS	pre	post	P value	F value	T value
group A	43.6	31.5	0.0351	11.649	2.436
group B	41.6	25.83			

Table No 6: 1st week and 6th week mean scores of Group A and Group B for SPDDS

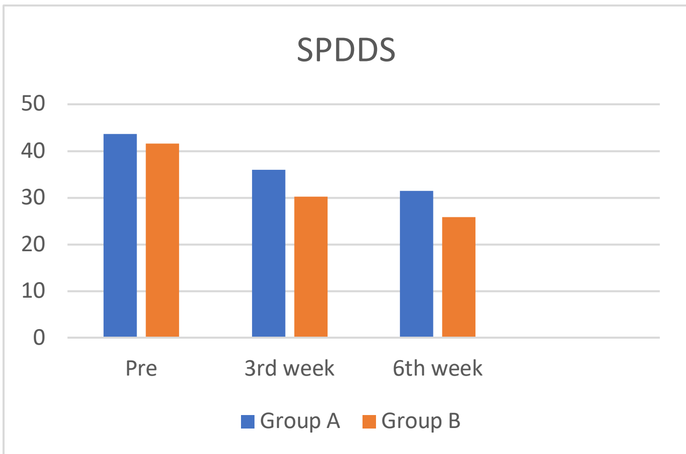

Fig 9: 1st week and 6th week mean scores of Group A and Group B for SPDDS

Interpretation: The graph shows a decreasing trend in the values of the scale indicating that there is significant improvement in both the groups however group B shows remarkable improvement than group A.

Discussion

The study investigated the effect of rhythmic initiation and rhythmic activity facilitation on patients with early-stage Parkinson's disease (PD). A total of 12 participants meeting the inclusion criteria were recruited. Group A (control) received conventional therapy, while Group B (experimental) received conventional therapy combined with rhythmic initiation and rhythmic activity facilitation. The analysis of values from baseline to the sixth week revealed statistically significant improvements in both groups. However, the experimental group demonstrated greater significance in outcomes compared to the control group.

The primary goals of physiotherapy for individuals with Parkinson's disease include patient education, enhancing motivation and self-efficacy, preventing physical inactivity and sedentary behavior, reducing fear of falling, alleviating pain, and improving overall physical capacity. Additional objectives involve delaying the onset of activity limitations, improving balance and transfers, managing freezing of gait, and enhancing manual function. A survey of Indian physiotherapists revealed that 50.8% recommend patients exercise for 30 minutes daily, while 26.2% suggest 45-minute sessions. In terms of medication timing, most physiotherapists recommend beginning exercise 60 minutes after taking medication. [13]

We observed significant positive effects of rhythmically cued exercise interventions on motor function in patients with Parkinson's disease. These results are consistent with previous research, which has highlighted the potential of such interventions to improve motor symptoms. The most notable improvements were observed in motor-related assessments. However, no significant changes were found in cognitive or mental state measures. These findings are in line with the review by Lötzke et al., which reported similar motor benefits from tango-based interventions in individuals with Parkinson's disease. Rhythmically cued exercises are believed to stimulate multiple brain regions involved in motor control. Specifically, they activate the cerebral cortex, enhance the activity of brainstem reticular structures and basal ganglia, and strengthen the connectivity between the brainstem, basal ganglia, and frontal lobe. Additionally, such interventions may stimulate dopaminergic pathways, including the striatum, thereby supporting residual dopaminergic activity and improving function within the basal ganglia-thalamocortical network—ultimately contributing to better motor performance in Parkinson's disease.[14]

External rhythmic cues, such as auditory and visual stimuli, have been shown to enhance walking speed, step length, and step frequency in individuals with Parkinson's disease, particularly during single-task conditions. However, research by Morris and others has shown that these benefits tend to diminish under dual-task conditions, indicating that the effectiveness of cueing strategies depends on sustained attentional engagement. In Parkinson's disease, movement often shifts from being internally generated to relying on external feedback. Rhythmic cues help compensate for this by engaging attentional strategies and feed-forward motor control mechanisms, facilitated by executive processes under frontal cortical control. These cues effectively bypass the impaired internal cueing pathways associated with basal ganglia dysfunction by offering consistent temporal signals that promote movement. They also reduce the cognitive load by minimizing the effort required to select, initiate, and sustain motor responses, thus freeing attentional resources. Supporting this approach, Carvalho et al. (2015) reported positive outcomes in the use of Proprioceptive Neuromuscular Facilitation (PNF) to improve functional independence among elderly individuals with Parkinson's disease. Their findings demonstrated immediate improvements in muscle strength and flexibility following PNF interventions. Mortari et al. (2009) further reinforced these results by showing significant gains in thigh muscle strength after PNF sessions. These findings underscore the potential of functionally oriented movement stimulation and targeted muscle training in enhancing motor control in Parkinson's patients. Likewise, Lima and Rodrigues de Paula (2012) highlighted muscle training as an effective therapeutic approach for addressing muscular impairments associated with Parkinson's disease.[15] Compared to the neutral position, the facilitation position modified the sequence of muscle activation, thereby enhancing the efficiency of joint movement. Intensive repetition of movements, induced by facilitation techniques such as Proprioceptive Neuromuscular Facilitation (PNF), stretch reflex, and skin-muscle reflex, contributed to significant improvements in voluntary motor control. [16]

Proprioceptive Neuromuscular Facilitation (PNF) has been shown to temporarily reduce EMG activity, vertical jump height, and ground reaction time. Despite these short-term effects, PNF remains effective for submaximal activities such as jogging. Nelson et al. (2005) found that PNF surpassed traditional strength training in improving muscle power, strength, and range of motion (ROM) in untrained individuals over an 8-week period. As individuals age, changes in soft tissue and neural responsiveness may affect the long-term efficacy of PNF. While significant ROM gains are often observed after a single session, sustaining these benefits requires consistent and correct application. Over time, the magnitude of improvement may diminish, highlighting PNF's value for immediate results.[17] Continued research is needed to better understand the mechanisms by which PNF influences physiological and functional outcomes in Parkinson's disease.

Conclusion

The present study, showed that the effect of rhythmic initiation along with rhythmic activity facilitation helped in improving functional mobility of the parkinsons disease patients.

Acknowledgement:

The authors would like to thank our college and Department Of Neurophysiotherapy of Krishna college of Physiotherapy, KVV, Karad.

References:

1. Wirdefeldt K, Adami HO, Cole P, Trichopoulos D, Mandel J. Epidemiology and etiology of Parkinson's disease: a review of the evidence. Eur J Epidemiol. 2011 Jun;26 Suppl 1:S1-58. 10.1007/s10654-011-9581-6
2. Ball N, Teo WP, Chandra S, Chapman J. Parkinson's Disease and the Environment. Front Neurol. 2019 Mar 19;10:218 doi: 10.3389/fneur.2019.00218
3. Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry. 2008 Apr;79(4):368-76. 10.1136/jnnp.2007.131045
4. Abbas MM, Govindappa ST, Sudhaman S, Thelma BK, Juyal RC, Behari M, Muthane UB. Early Onset Parkinson's disease due to DJ1 mutations: An Indian study. Parkinsonism Relat Disord. 2016 Nov;32:20-24. 10.1016/j.parkreldis.2016.04.024
5. Surathi P, Jhunjhunwala K, Yadav R, Pal PK. Research in Parkinson's disease in India: A review. Ann Indian Acad Neurol. 2016 Jan-Mar;19(1):9-20. 10.4103/0972-2327.167713
6. Obergasteiger J, Frapporti G, Pramstaller PP, Hicks AA, Volta M. A new hypothesis for Parkinson's disease pathogenesis: GTPase-p38 MAPK signaling and autophagy as convergence points of etiology and genomics. Mol Neurodegener. 2018 Aug 2;13(1):40. 10.1186/s13024-018-0273-5
7. Collier TJ, Kanaan NM, Kordower JH. Ageing as a primary risk factor for Parkinson's disease: evidence from studies of non-human primates. Nat Rev Neurosci. 2011 Jun;12(6):359-66. 10.1038/nrn3039
8. O'sullivan SB, Schmitz TJ, Fulk GD. Physical rehabilitation. 6th ed. Philadelphia, Pa: F.A. Davis Company; 2014.
9. Bouça-Machado R, Maetzler W, Ferreira JJ. What is Functional Mobility Applied to Parkinson's Disease? J Parkinsons Dis. 2018;8(1):121-130. doi: 10.3233/JPD-171233.
10. Wang JS, Lee SB, Moon SH. The immediate effect of PNF pattern on muscle tone and muscle stiffness in chronic stroke patient. J Phys Ther Sci. 2016 Mar;28(3):967-70. doi: 10.1589/jpts.28.967.
11. Adler SS, Beckers D, Buck M. PNF in practice: an illustrated guide. 4th ed. Berlin: Springer; 2014.
12. Siff MC. Modified PNF as a system of physical conditioning. NSCA J. 1991;13(4):73–77.
13. Agarwal V, Yadav N, Sharma A, Rawat A. Current practice patterns of Indian physiotherapists in the rehabilitation of Parkinson's disease: a cross-sectional survey study. Obstet Gynaecol Forum. 2024 Aug 6;34(3s):2807–14.
14. Huang X, Dong K, Gan C, Xu Z, Lei D, Dong X, Liu H, Chen X. Effect of Rhythmically Cued Exercise Interventions on Functions in Patients With Parkinson Disease: A Meta-Analysis. Phys Ther. 2024 Mar 1;104(3) doi: 10.1093/ptj/pzad158.
15. Carvalho EL, Cruz KLT, Queiroz IP, Silva F, Maciel TS, Arêas FZS. Influences of neuromuscular facilitation in the hamstrings and quadriceps muscles of Parkinsonian elderly. Man Ther Posturol Rehabil J. 2016 Nov 18;1–8. https://doi.org/10.17784/mtprehabjournal.2016.14.369
16. Kumar S, Kumar A, Kaur J. Effect of PNF technique on gait parameters and functional mobility in hemiparetic patients. J Exerc Sci Physiother. 2012;8(2):67–73.
17. Hindle KB, Whitcomb TJ, Briggs WO, Hong J. Proprioceptive neuromuscular facilitation (PNF): its mechanisms and effects on range of motion and muscular function. J Hum Kinet. 2012 Mar;31:105–13. doi: 10.2478/v10078-012-0011-y