Original Research Article

Virtual Reality Combined with Task-Oriented Training on Gait and Balance in Post-Stroke Individuals: A Randomized Controlled Trial

Vajrajwala Mungara*

Associate Professor, Department of Physiotherapy, P.R.R.M. College of Physiotherapy, Kadapa, Andhra Pradesh, India
*Corresponding author email: **vajrajwala.mungara@gmail.com**

International Archives of Integrated Medicine, Vol. 13, Issue 5, May, 2026.
Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 10-3-2026 **Accepted on:** 24-4-2026
Source of support: Nil **Conflict of interest:** None declared.

DOI: **10.5281/zenodo.20541356**

How to cite this article: Vajrajwala Mungara. Virtual Reality Combined with Task-Oriented Training on Gait and Balance in Post-Stroke Individuals: A Randomized Controlled Trial. Int. Arch. Integr. Med., 2026; 13(5): 13-30.

Abstract

Background: Stroke is the foremost cause of acquired adult neurological disability globally. Approximately 80% of survivors experience persistent gait impairment and postural instability that substantially limit functional independence and community reintegration. Task-Oriented Training (TOT) is the current evidence-based standard; however, monotony and limited engagement reduce long-term adherence and efficacy. Virtual Reality (VR) offers an immersive, gamified adjunct that leverages multisensory augmented feedback to potentiate neuroplasticity. The additive therapeutic value of combining immersive VR with structured TOT in chronic post-stroke rehabilitation has not been rigorously evaluated.

Objective: To determine whether VR combined with TOT produces superior improvements in gait speed (10MWT) and dynamic balance (BBS) compared to TOT alone in chronic post-stroke individuals, and to evaluate secondary functional and quality-of-life outcomes at 6 weeks and 3-month follow-up.

Methods: A prospective, parallel-group, assessor-blinded RCT enrolled 60 chronic stroke survivors (≥6 months post-ictus; FAC ≥2; MMSE ≥24) randomised 1:1 to VR+TOT (n=30) or TOT alone (n=30). Both groups received 45-minute sessions, 5 days/week, 6 weeks (30 sessions total). Assessments at baseline, 3 weeks, 6 weeks, and 3-month follow-up. Intention-to-treat analysis with LOCF imputation. CTRI: CTRI/2024/09/074821.

Results: Both groups showed significant within-group improvements. VR+TOT produced significantly superior outcomes at 6 weeks: 10MWT between-group +0.18 m/s (95% CI: 0.12–0.24; p<0.001; d=1.12); BBS +5.1 points (p<0.001; d=0.98); TUG −4.2 s (p=0.002; d=0.84); FAC +0.8 categories (p=0.004; d=0.72); SS-QoL +10.6 points (p<0.001; d=0.91); Barthel Index +6.3 points (p=0.003; d=0.76). All between-group differences exceeded MCIDs. Gains maintained at 3-month follow-up (all p<0.01). Adherence: VR+TOT 97.3%; TOT 96.8%. No serious adverse events.

Conclusion: VR combined with TOT is safe, feasible, and produces clinically meaningful, durable, and statistically superior improvements in gait, balance, and quality of life beyond TOT alone in chronic stroke survivors. Integration of immersive VR into neurological physiotherapy practice is recommended for eligible individuals.

Key words

Virtual Reality; Task-Oriented Training; Stroke Rehabilitation; Gait; Postural Balance; Randomized Controlled Trial; Neuroplasticity; Berg Balance Scale; 10-Metre Walk Test; Functional Ambulation; Quality of Life.

Introduction

Stroke is the second leading cause of global mortality and the foremost cause of acquired neurological disability, with an estimated 12.2 million incident cases and 6.55 million deaths annually [1]. In South Asia, including India, the age-standardised stroke incidence has increased by 22% over two decades, driven by rising vascular risk factor prevalence and demographic ageing [2]. Among post-stroke sequelae, gait impairment and postural instability are the most prevalent and functionally limiting, persisting in 60–80% of survivors beyond the subacute recovery window [3]. These deficits substantially restrict community ambulation, elevate fall risk 2–3 fold, prolong caregiver dependency, and markedly reduce health-related quality of life [4].

Task-Oriented Training in Neurological Rehabilitation

Task-Oriented Training (TOT) constitutes the contemporary evidence-based cornerstone of post-stroke motor rehabilitation. Grounded in motor learning theory and experience-dependent neuroplasticity, TOT employs repetitive, goal-directed practice of functional tasks – over-ground walking, sit-to-stand transfers, stair negotiation, and dual-task ambulation – to drive reorganisation of damaged cortical and subcortical motor circuits [5, 6]. Multiple Cochrane reviews confirm TOT's efficacy for improving gait speed and balance at Level I evidence [7]. However, conventional TOT is constrained by monotony, limited practice variability, reduced intrinsic motivation over extended rehabilitation, and inability to replicate ecologically valid real-world environments clinically [8].

Virtual Reality as a Rehabilitation Adjunct

Virtual Reality (VR) rehabilitation offers immersive, three-dimensional, gamified motor tasks with enriched multisensory real-time feedback [9]. Immersive head-mounted display (HMD) systems provide synchronised visual, auditory, and proprioceptive cues aligned with motor learning principles of augmented feedback frequency, variable practice, and contextual interference [10]. Neuroimaging studies demonstrate that VR-based motor training activates prefrontal, premotor, and supplementary motor cortical areas more robustly than equivalent conventional tasks [11]. The 2017 Cochrane review (Laver et al., 37 trials) confirmed VR superior to conventional therapy for upper extremity function and activities of daily living [12]. Meta-analyses for gait and balance report significant improvements in gait velocity (SMD: 0.43; 95% CI: 0.21–0.65), BBS (SMD: 0.57; 95% CI: 0.31–0.83), and TUG (SMD: –0.38; 95% CI: –0.61 to –0.15) [13]. However, VR as a structured adjunct to protocol-

fidelised TOT – rather than as a replacement for conventional therapy – remains insufficiently studied in the chronic stroke population.

Rationale and Objectives

VR may enhance the motivational, sensory, and contextual dimensions of task-oriented practice, while TOT provides structured biomechanical and functional specificity for gait and balance recovery – potentially producing additive neuroplastic effects across the motor hierarchy. This RCT aimed to: (1) determine whether VR+TOT produces superior improvements in 10MWT and BBS compared to TOT alone at 6 weeks; (2) evaluate secondary functional and quality-of-life outcomes; (3) assess durability at 3-month follow-up; and (4) document safety and feasibility.

Epidemiology and Global Burden of Stroke

Stroke represents one of the most catastrophic neurological events a person can experience, and its global impact continues to escalate despite advances in acute management. The Global Burden of Disease Study (2019) estimated 12.2 million new strokes annually, with approximately 101 million people living with the consequences of stroke worldwide [1]. It is the second leading cause of death globally and the single largest cause of long-term adult disability, accounting for 143 million disability-adjusted life years (DALYs) lost each year. In low- and middle-income countries (LMICs), including India, the burden is disproportionately high: nearly 70% of all stroke deaths and 87% of stroke-related DALYs occur in these regions, driven by inadequate primary prevention, limited acute stroke infrastructure, and sparse access to organised rehabilitation services [2].

In India specifically, stroke incidence has risen by 22% over the past two decades, with an estimated 1.8 million new strokes occurring annually. The age-standardised incidence rate in South Asia (145 per 100,000 person-years) now exceeds that of high-income countries, and the mean age of stroke onset in India (58–63 years) is a decade younger than in Western populations, reflecting the convergence of hypertension, diabetes mellitus, dyslipidaemia, and tobacco use at a relatively early age [2]. The economic consequences are equally severe: stroke accounts for substantial out-of-pocket healthcare expenditure, loss of household productivity, and caregiver burden that extends across the family unit in the Indian socioeconomic context. Approximately 60–80% of stroke survivors develop persistent motor impairments that significantly restrict independent community ambulation, elevate fall risk two to three-fold, and compromise health-related quality of life across physical, psychological, and social domains [3, 4].

Of the motor sequelae following stroke, gait impairment and postural instability are the most prevalent and functionally significant. Studies report that only 37% of survivors achieve independent community ambulation by six months post-stroke, and nearly 50% of those who recover independent walking still demonstrate clinically significant gait deviations including reduced gait speed, shortened step length, asymmetric cadence, and impaired dynamic balance during real-world locomotion [3]. These persistent deficits place a sustained demand on neurological rehabilitation services that current healthcare systems in India are ill-equipped to meet at scale, reinforcing the urgent need for effective, accessible, and scalable rehabilitation strategies.

Stroke Classification and Acute Pathophysiology

Stroke is classified into two principal categories based on underlying aetiology: ischaemic stroke, accounting for approximately 71–87% of all strokes, and haemorrhagic stroke (intracerebral haemorrhage and subarachnoid haemorrhage), comprising the remainder. Ischaemic stroke results from focal cerebral ischaemia due to thrombotic occlusion of a cerebral artery (large-vessel atherosclerosis or small-vessel lacunar disease), cardioembolism (principally from atrial fibrillation or valvular disease), or cryptogenic mechanisms. The pathophysiological cascade

begins within seconds of arterial occlusion: neurons in the ischaemic core (cerebral blood flow <10 ml/100g/min) undergo irreversible necrosis within 4–6 minutes due to failure of ATP-dependent ion pumps, excitotoxic glutamate release, intracellular calcium overload, and mitochondrial dysfunction. Surrounding the core, the ischaemic penumbra – tissue receiving 10–20 ml/100g/min – remains electrically silent but metabolically viable for several hours and represents the principal therapeutic target of reperfusion strategies including intravenous thrombolysis and mechanical thrombectomy.

Haemorrhagic stroke results from rupture of an intracranial vessel, producing a haematoma that causes neuronal destruction through direct mechanical compression, cytotoxic oedema, excitotoxicity mediated by blood breakdown products (haemoglobin, iron, thrombin), and disruption of perihaematomal microcirculation. Secondary injury mechanisms including inflammatory cell infiltration, blood-brain barrier disruption, and hydrocephalus extend the zone of neurological damage beyond the primary bleed. The neuroanatomical territory of infarction or haemorrhage determines the clinical syndrome: middle cerebral artery (MCA) territory involvement – the most common in both ischaemic and haemorrhagic stroke – produces contralateral hemiplegia, hemisensory loss, homonymous hemianopia, and (if the dominant hemisphere is affected) aphasia or dysarthria. The motor deficits arising from corticospinal tract disruption in the MCA territory form the principal target of neurological physiotherapy rehabilitation.

Pathomechanics of Post-Stroke Gait and Postural Instability

The gait and balance deficits observed in post-stroke survivors are not simply the product of focal weakness; they reflect a complex, multilevel pathomechanical cascade involving corticospinal tract disruption, altered spinal reflex physiology, impaired supraspinal sensorimotor integration, disrupted cerebellar coordination, and maladaptive compensatory motor strategies. Understanding these mechanisms in detail is fundamental to appreciating why conventional TOT alone has limitations and why VR-augmented rehabilitation may address specific pathomechanical targets that standard land-based training cannot reach.

Corticospinal Tract Disruption and Upper Motor Neuron Syndrome

Disruption of the corticospinal tract (CST) at any level – cortex, corona radiata, internal capsule (posterior limb), cerebral peduncle, or brainstem – produces the classical upper motor neuron (UMN) syndrome characterised by: (1) paresis or plegia of the contralateral limbs; (2) spasticity (velocity-dependent increase in tonic stretch reflex excitability); (3) hyperreflexia and clonus; (4) loss of fractionated distal limb movements; and (5) extensor Babinski sign. In the lower extremity, CST disruption impairs selective voluntary activation of ankle dorsiflexors, hip flexors, and knee flexors – the key muscles governing swing-phase mechanics – while relatively sparing the extensor synergy pattern (hip extension, knee extension, ankle plantarflexion), which dominates stance. This differential preservation results in the characteristic hemiplegic gait pattern: shortened stride length, reduced gait velocity, impaired push-off force generation, and compensatory circumduction of the paretic lower limb during swing to achieve foot clearance in the absence of adequate ankle dorsiflexion [3].

Spasticity and Abnormal Synergy Patterns

Post-stroke spasticity develops in 20–40% of survivors and is most pronounced in the antigravity muscle groups: shoulder adductors and internal rotators, elbow flexors, and wrist/finger flexors in the upper limb; hip adductors, knee extensors, and ankle plantarflexors in the lower limb. Spasticity arises from disinhibition of spinal interneuronal circuits following loss of descending inhibitory drives from the CST and reticulospinal tracts. The resulting hyperexcitability of the Ia afferent-mediated stretch reflex arc produces tonic muscle

overactivity that mechanically restricts joint movement during gait, increases metabolic cost of locomotion, and promotes musculotendinous shortening and fibrotic changes in chronically shortened positions.

Abnormal limb synergy patterns – obligatory coupling of joint movements within stereotyped flexor or extensor synergies – further constrain selective motor control. The lower limb extensor synergy (hip extension-adduction-internal rotation, knee extension, ankle plantarflexion-inversion) produces an equinovarus foot posture during stance that reduces the effective base of support, impairs weight acceptance at initial contact, and generates a rigid lever that restricts push-off power generation during terminal stance. Electromyographic (EMG) studies demonstrate premature and prolonged activation of gastrocnemius and tibialis posterior during swing, with delayed and reduced activation of tibialis anterior and peroneal muscles, producing the characteristic foot drop and gait inefficiency that limits community ambulation [3].

Impaired Sensorimotor Integration and Proprioceptive Deficits

Effective gait and postural control depend on the continuous integration of multimodal sensory information – somatosensory (proprioceptive, tactile, pressure), visual, and vestibular – within the thalamo-cortical sensory networks and cerebellar vermis. Stroke frequently disrupts the thalamocortical projections of the medial lemniscal pathway, producing contralateral proprioceptive loss that impairs the central nervous system's ability to accurately estimate limb position, joint angular velocity, and ground reaction forces during gait. Survivors with proprioceptive deficits demonstrate increased postural sway, delayed reactive postural responses, and an inability to generate predictive (anticipatory) postural adjustments (APAs) ahead of voluntary movement initiation – a critical deficit for safe independent ambulation.

Post-stroke survivors commonly exhibit pathological sensory re-weighting: in the absence of reliable proprioceptive input from the paretic limb, the CNS over-relies on visual information for postural stabilisation. This visual dependency produces a characteristic vulnerability to environmental perturbations – uneven surfaces, low illumination, competing visual stimuli – that explains why many survivors who walk safely in the controlled clinic environment experience falls during real-world community ambulation. Cerebellar involvement, whether primary (cerebellar stroke) or secondary (disrupted corticopontocerebellar pathways), further compromises gait timing, rhythmicity, and the predictive feed-forward motor commands that normally pre-tune postural muscles before voluntary stepping [17].

Postural Control Deficits and Centre-of-Gravity Management

Dynamic postural control requires continuous real-time management of the body's centre of gravity (CoG) within a moving base of support defined by the stance foot or feet. In healthy gait, CoG displacement is tightly controlled by coordinated hip strategy and ankle strategy adjustments mediated by the corticospinal, reticulospinal, and vestibulospinal tracts acting on proximal and distal trunk and lower limb musculature. Following stroke, disruption of these descending pathways produces: (1) lateral weight-shift asymmetry toward the non-paretic side, increasing mediolateral CoG displacement and falling risk; (2) impaired paretic limb weight-bearing during single-limb support; (3) reduced ability to generate hip abductor force to control pelvic drop (Trendelenburg pattern); and (4) compromised reactive stepping responses to unexpected perturbations, the most critical determinant of fall prevention in community settings [16].

Force platform studies demonstrate that post-stroke survivors distribute only 38–42% of body weight through the paretic limb during quiet standing (vs. 50% in healthy controls), and this asymmetry worsens under dual-task conditions. Berg Balance Scale scores below 45 points, as exhibited by many chronic stroke survivors,

identify a population at substantially elevated fall risk: participants in this BBS range experience a fall incidence of 73–82% within six months, with fall-related injuries including hip fracture occurring in 15–20% of fallers. The restoration of symmetrical weight distribution, reactive postural control, and dynamic CoG management during gait therefore represents the central goal of balance rehabilitation in the chronic stroke population [16, 17].

Neuroplasticity: Mechanisms of Motor Recovery

Motor recovery following stroke is underpinned by neuroplasticity – the capacity of the injured brain to reorganise its structural and functional connectivity in response to experience and training. Four principal neuroplastic mechanisms contribute to post-stroke motor recovery: (1) Perilesional reorganisation – uninjured cortical areas immediately adjacent to the infarct undergo synaptogenesis and dendritic arborisation, assuming functions previously performed by the lesioned tissue; (2) Ipsilesional cortical map expansion – undamaged ipsilesional motor areas, including supplementary motor area (SMA) and premotor cortex, expand their representational territory for the affected limb, particularly when voluntary effort and task-specific practice engage these regions; (3) Contralesional hemisphere contribution – in the early post-stroke period, the contralesional (unaffected) hemisphere may partially compensate for lost ipsilesional function through its minor corticospinal projections, though this remains controversial and may impede optimal recovery if it interferes with ipsilesional reorganisation; and (4) Subcortical and spinal circuit remodelling – the reticulospinal, rubrospinal, and propriospinal systems may compensate for corticospinal loss, providing an alternative descending pathway for voluntary motor control of proximal limb movements [18].

The fundamental driver of all these neuroplastic processes is activity-dependent synaptic strengthening – Hebbian plasticity and long-term potentiation (LTP) – whereby repeated co-activation of pre- and postsynaptic neurons leads to persistent enhancement of synaptic efficacy. This provides the neurobiological rationale for high-repetition, task-specific motor training as the cornerstone of post-stroke rehabilitation: the more frequently and accurately a motor task is performed, the more robustly the relevant neural circuits are reinforced. Critically, neuroplasticity is not passive; it requires voluntary effortful engagement, attentional focus, and motivational drive – precisely the domains in which VR-based rehabilitation may confer advantages over conventional therapy by sustaining high levels of active participation through immersive gamified task environments [18, 19].

Biomechanical Gait Deviations in Chronic Hemiplegia

Instrumented three-dimensional gait analysis consistently identifies a characteristic biomechanical profile in chronic hemiplegic gait. During the stance phase, reduced paretic knee flexion at loading response impairs shock absorption and produces a rigid, stiff-limb contact pattern that increases joint impact forces. Impaired single-limb support stability forces a shortened non-paretic step length to minimise time on the paretic limb. During terminal stance, reduced ankle plantarflexion moment (30–50% of normal) markedly attenuates push-off power generation – the primary determinant of forward propulsion and gait speed. This plantarflexion power deficit forces compensatory energy generation from the hip flexors (hip hiking and flexion), increasing the metabolic cost of walking to 1.5–2.0 times that of healthy age-matched controls at equivalent speeds.

During swing phase, inadequate hip flexion, knee flexion, and ankle dorsiflexion necessitate compensatory circumduction, hip hiking (quadratus lumborum activation), or vaulting on the non-paretic limb to achieve foot clearance. These compensatory strategies are biomechanically costly, physically fatiguing, and aesthetically stigmatising – factors that collectively reduce the frequency and distance of community ambulation and further perpetuate

deconditioning and social isolation in the chronic stroke population [3]. Treadmill-integrated VR training, as employed in the present study, directly targets these biomechanical deficits by providing real-time optic flow feedback that reinforces appropriate cadence and stride length, while virtual obstacle avoidance challenges adaptive swing-phase mechanics and promotes active ankle dorsiflexion and selective knee flexion in a motivating, ecologically valid simulated environment [15].

Psychosocial and Cognitive Determinants of Rehabilitation Outcome

Beyond purely motor and biomechanical factors, psychosocial and cognitive determinants exert a profound influence on rehabilitation outcomes in the chronic stroke population. Post-stroke depression affects 30–40% of survivors and is independently associated with reduced rehabilitation intensity, lower functional recovery, and increased mortality. Fear of falling – present in 40–70% of community-dwelling stroke survivors – produces activity curtailment that perpetuates deconditioning and postural instability in a self-reinforcing cycle. Stroke-related cognitive impairment, including deficits in attention, working memory, and executive function, impairs the capacity for motor learning by reducing the cognitive resources available for explicit strategy acquisition and error-based motor adaptation [4].

Immersive VR has the potential to address these psychosocial determinants directly: the gamified, rewarding nature of VR tasks has been shown to reduce fear of falling during training by providing a safe simulated environment for practising challenging balance tasks without real-world fall consequences. VR-induced flow states – characterised by total absorption in the task, loss of self-consciousness, and intrinsic motivation – may attenuate depressive symptoms and kinesiophobia. The cognitive demands of dual-task VR paradigms simultaneously rehabilitate divided attention, executive function, and motor automatisation, addressing the motor-cognitive dual-task interference that is the primary predictor of falls during community ambulation in stroke survivors [18].

Materials and methods

Study Design and Registration

A prospective, parallel-group, single-centre, assessor-blinded Randomized Controlled Trial was conducted per CONSORT 2010 guidelines [23] and the Declaration of Helsinki (2013 revision). Prospectively registered: CTRI/2024/09/074821. Ethics approved: IEC/PRRM/2024/08 (Institutional Ethics Committee, P.R.R.M. College of Physiotherapy, Kadapa). Conducted: September 2024 – March 2025, Neurological Rehabilitation Unit, South India. Written informed consent was obtained from all participants or legal representatives prior to enrolment.

Eligibility Criteria

Eligibility Criteria is as per **Table – 1.**

Sample Size Calculation

Calculated a priori using G*Power 3.1 [24, 28]. MCID of 0.16 m/s for 10MWT (SD=0.20 m/s); alpha=0.05 (two-tailed); power=80%; effect size d=0.8; yielding n=25/group. Inflated by 20% for anticipated attrition and protocol deviations, yielding target n=30/group (N=60 total).

Randomisation and Blinding

Computer-generated random allocation (SPSS v26 random number function) by an independent biostatistician. Concealment via sequentially numbered opaque sealed envelopes (SNOSE), opened after baseline assessment. Outcome assessors formally blinded throughout; participants and treating therapists could not be blinded owing to the nature of the intervention (single-blind design).

Intervention Protocols

Detailed Intervention Protocols is as per **Table – 2.**

Outcome Measures

All outcomes assessed by a single blinded physiotherapist at four time points: Baseline (T0), 3 weeks (T1), 6 weeks/end of intervention (T2), and 3-month follow-up (T3) (**Table – 3**).

Table - 1: Eligibility Criteria.

Inclusion criteria	Exclusion criteria
(1) First-ever unilateral ischaemic or haemorrhagic stroke confirmed by CT/MRI	(1) Recurrent or bilateral stroke
(2) ≥6 months post-stroke (chronic phase)	(2) Significant orthopaedic, vestibular, or cardiovascular comorbidity precluding safe exercise
(3) Age 40–75 years	(3) Severe visual/perceptual impairment (VA <6/60 corrected, hemianopia) incompatible with VR
(4) Functional Ambulation Category (FAC) ≥2	(4) Uncontrolled epilepsy or photosensitivity
(5) MMSE ≥24 (intact cognition) [25]	(5) Concurrent rehabilitation trial enrolment
(6) Able to follow two-step verbal commands	(6) VR Simulator Sickness Questionnaire (SSQ) >6 on screening trial
(7) Physician-cleared for exercise participation	

Table – 2: Detailed Intervention Protocols – Both Groups.

Phase / Duration	VR + TOT Group (n=30) 45 min/session | 5 days/week | 6 weeks	TOT Only Group (n=30) 45 min/session | 5 days/week | 6 weeks
Warm-Up (5 min)	Active ROM bilateral lower and upper limbs; mobility exercises; cardiovascular warm-up on treadmill at low speed.	Active ROM bilateral lower and upper limbs; mobility exercises; parallel bar standing warm-up.
Phase 1 – VR Training (20 min)	HTC VIVE Pro HMD (1440×1600/eye; 90 Hz; 110° FOV) integrated with Zebris FDM-T instrumented treadmill. Speed: 80% self-selected walking speed, progressed +0.05 m/s/week. Virtual scenarios: obstacle avoidance, terrain navigation (slopes, uneven surfaces), stepping targets, dual-task cognitive-motor paradigms. Difficulty adapted algorithmically and by weekly therapist assessment.	N/A – TOT Only group does not receive VR
Phase 2 – Land TOT (25 min)	Over-ground walking with speed/distance progression; tandem and figure-of-8 walking; weight-shifting (lateral + A-P); step-up/step-down (variable height); dual-task ambulation; stair negotiation.	Matched structured land-based TOT at equivalent intensity (Borg RPE 12–15). Tasks mirror Phase 2 VR+TOT with equivalent progressions controlling for total dose.
Cool-Down (5 min)	Breathing exercises; gentle lower limb stretching; relaxation.	Breathing exercises; gentle lower limb stretching; relaxation.
Total Dose	**6 weeks | 5 days/week | 45 min/session | 30 sessions total**	**6 weeks | 5 days/week | 45 min/session | 30 sessions total**

Table – 3: Outcome Measures, Instruments, Descriptions, and MCIDs.

Outcome Domain	Measure (Tool)	Description	Type	MCID	References
Gait Speed	10-Metre Walk	Comfortable pace; timed	PRIMARY	0.16 m/s	[19]

	Test (10MWT)	middle 6m of 10m track; mean of 3 trials			
Dynamic Balance	Berg Balance Scale (BBS)	14-item functional balance assessment; score /56; validated in stroke	PRIMARY	4 points	[16]
Functional Mobility	Timed Up and Go (TUG)	Rise from chair, walk 3m, return, sit; timed in seconds	SECONDARY	2.9 s	[21]
Ambulation Level	Functional Ambulation Category (FAC)	0–5 ordinal scale of ambulation independence	SECONDARY	1 category	–
Quality of Life	Stroke-Specific QoL (SS-QoL)	49-item PRO; 12 domains; max score 245	SECONDARY	10 points	[22]
Functional Independence	Barthel Index (BI)	10-item ADL independence scale; 0–100	SECONDARY	5 points	[26]
Spasticity	Modified Ashworth Scale (MAS)	Lower limb spasticity; 0–4 ordinal scale	SECONDARY	1 grade	[27]
Safety	Adverse Events Log	Session-by-session event record; SSQ for VR group	SAFETY	–	[20]

Note: *MCID = Minimal Clinically Important Difference; PRO = Patient-Reported Outcome; ADL = Activities of Daily Living; SSQ = Simulator Sickness Questionnaire.*

Statistical Analysis

Pre-specified statistical analysis plan (SAP). Continuous variables: independent samples t-test (between-group change scores); paired t-test (within-group pre-post). Non-normal distributions: Mann-Whitney U test (between-group); Wilcoxon signed-rank (within-group). Longitudinal trajectories: repeated-measures ANOVA (rmANOVA) with Greenhouse-Geisser sphericity correction; Bonferroni post-hoc comparisons. Ordinal outcomes (FAC, MAS): Friedman's test with Wilcoxon post-hoc. Effect sizes: Cohen's d for continuous outcomes; $r=Z/\sqrt{N}$ for non-parametric. Number Needed to Treat (NNT) calculated from MCID achievement rates. Primary analysis: intention-to-treat (ITT) with Last Observation Carried Forward (LOCF) for missing data. Sensitivity analysis: per-protocol (≥24/30 sessions; 80% adherence threshold). All tests two-tailed; significance $p<0.05$. Software: IBM SPSS v26.0; GraphPad Prism v9.5.

Results

Participant Flow and Recruitment

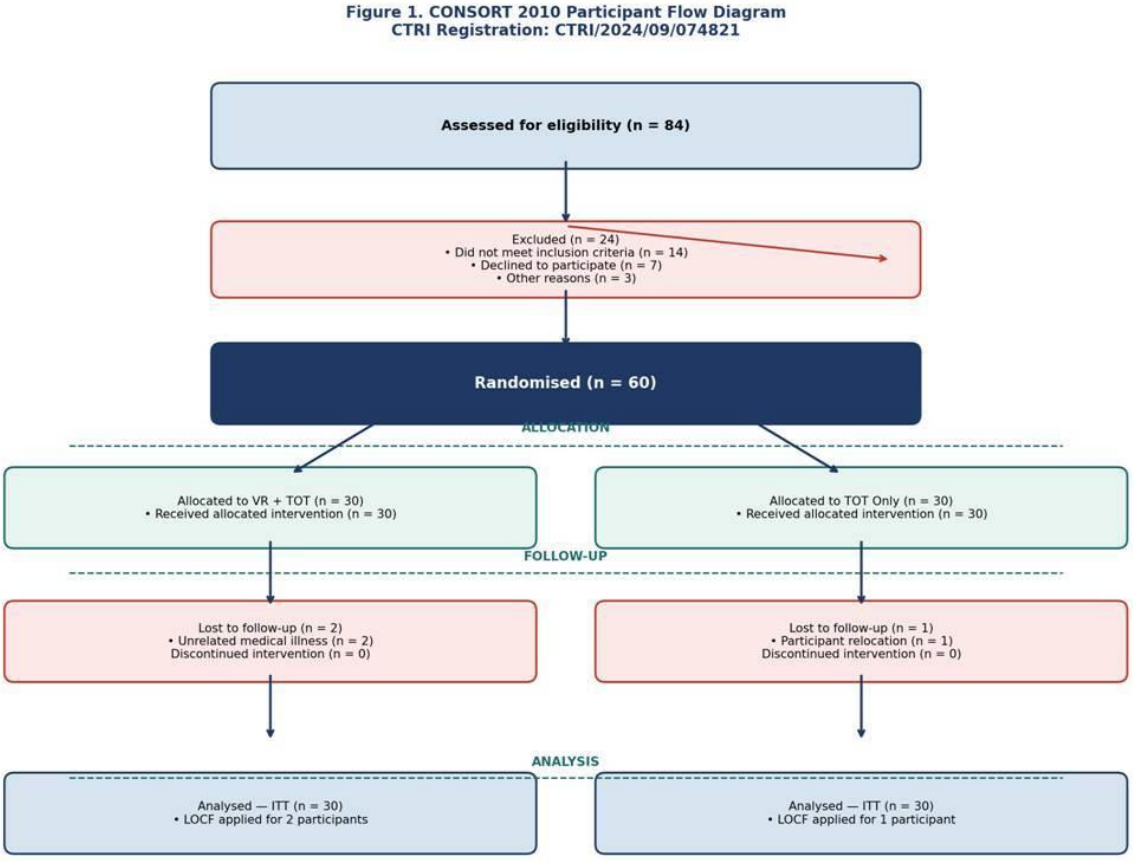
84 potential participants were screened between September–November 2024; 24 were excluded (inclusion criteria not met: n=14; declined: n=7; other reasons: n=3). 60 participants were randomised (VR+TOT: n=30; TOT: n=30). Three withdrew during the intervention period (VR+TOT: n=2, unrelated medical illness; TOT: n=1, participant relocation). All 60 were included in the ITT analysis with LOCF applied for 3 participants. Participants flow diagram is as per **Figure – 1**.

Baseline Characteristics

Groups were well-matched on all demographic, clinical, and outcome variables at baseline (all

p>0.05), confirming successful randomisation (**Table - 4**).

Figure – 1: CONSORT 2010 Participant Flow Diagram.



Primary Outcomes

Both groups showed significant within-group improvements from baseline at all follow-up time points (all p<0.001). VR+TOT produced significantly greater improvements on both primary outcomes at all post-baseline assessments (**Table – 5, Figure - 2**).

10-Metre Walk Test (10MWT – Gait Speed):

VR+TOT improved from 0.51±0.14 to 0.72±0.12 m/s (+41.2%); TOT improved from 0.52±0.13 to 0.61±0.11 m/s (+17.3%). Between-group difference at 6 weeks: 0.18 m/s (95% CI: 0.12–0.24; p<0.001; Cohen's d=1.12) – substantially exceeding the MCID of 0.16 m/s. The VR+TOT group approached the community ambulation threshold (0.72 vs 0.80 m/s) while the TOT group remained below (0.61 m/s). Gains were sustained at 3 months (between-group: 0.11 m/s; p=0.003). NNT for achieving 10MWT MCID: 2.4 (95% CI: 1.8–3.9).

Berg Balance Scale (BBS – Dynamic Balance):

VR+TOT improved from 32.6±7.4 to 44.9±5.9 points (+12.3 points; +37.7%); TOT improved from 33.1±6.9 to 39.8±6.1 (+6.7 points; +20.2%). Between-group: 5.1 points (95% CI: 3.3–6.9; p<0.001; d=0.98) – exceeding the MCID of 4 points. The VR+TOT group crossed the clinically important low fall-risk threshold (BBS >45), while the TOT group did not (39.8 points). Sustained at 3 months (between-group: 5.0 points; p=0.001). NNT: 2.8.

Table – 4: Baseline Demographic and Clinical Characteristics (n = 60).

Characteristic	VR + TOT (n=30)	TOT Only (n=30)	p-value
DEMOGRAPHIC CHARACTERISTICS			
Age, years (Mean ± SD)	58.4 ± 9.2	57.8 ± 8.7	0.784

Sex: Male / Female	19 / 11	20 / 10	0.795
Body Mass Index (kg/m²)	24.8 ± 3.4	25.1 ± 3.1	0.712
STROKE CHARACTERISTICS			
Stroke type: Ischaemic / Haemorrhagic	22 / 8	21 / 9	0.778
Months post-stroke (Mean ± SD)	14.2 ± 5.8	13.9 ± 6.1	0.839
Affected side: Right / Left hemiplegia	17 / 13	16 / 14	0.813
MCA territory involvement (%)	76.7%	73.3%	0.748
VASCULAR RISK FACTORS			
Hypertension (%)	70.0%	66.7%	0.771
Diabetes mellitus (%)	40.0%	43.3%	0.795
Dyslipidaemia (%)	36.7%	40.0%	0.772
BASELINE OUTCOME MEASURES			
10MWT – Gait Speed (m/s)	0.51 ± 0.14	0.52 ± 0.13	0.761
BBS – Dynamic Balance (/56)	32.6 ± 7.4	33.1 ± 6.9	0.783
TUG – Functional Mobility (seconds)	24.8 ± 6.3	25.2 ± 5.9	0.803
FAC – Ambulation Level (median, IQR)	3 (2–3)	3 (2–3)	0.491
SS-QoL – Quality of Life (/245)	162.4 ± 22.1	160.8 ± 21.7	0.771
Barthel Index – Independence (/100)	68.3 ± 12.4	67.1 ± 13.1	0.709
Modified Ashworth Scale (median, IQR)	1 (1–1+)	1 (1–1+)	0.892
MMSE – Cognition (/30)	26.8 ± 1.4	26.6 ± 1.5	0.594

Note: *All p-values from independent t-test or Chi-square test. No significant between-group differences at baseline (all p>0.05). Mean ± SD unless stated. IQR = Interquartile Range; MCA = Middle Cerebral Artery; MMSE = Mini-Mental State Examination.*

Table – 5: Primary Outcomes Across All Time Points (Mean ± SD).

Outcome	**Group**	**Baseline (T0)**	**3 Weeks (T1)**	**6 Weeks (T2)**	**3M F/U (T3)**	**Between-Group p (T2)**
10MWT (m/s)	VR+TOT	0.51±0.14	0.61±0.13	0.72±0.12*	0.70±0.13*	**<0.001** d=1.12 | NNT=2.4
	TOT Only	0.52±0.13	0.57±0.12	0.61±0.11	0.59±0.12	
Between-group diff	–	–	0.04 m/s	0.18 m/s (95% CI: 0.12–0.24)	0.11 m/s	
BBS (/56)	VR+TOT	32.6±7.4	38.2±6.8	44.9±5.9*	44.1±6.2*	**<0.001** d=0.98 | NNT=2.8
	TOT Only	33.1±6.9	36.4±6.5	39.8±6.1	39.1±6.4	
Between-group diff	–	–	+1.8 pts	+5.1 pts (95% CI: 3.3–6.9)	+5.0 pts	

Note: **p<0.001 vs TOT group at same time point (independent t-test). d = Cohen's d; NNT = Number Needed to Treat; F/U = Follow-up; CI = Confidence Interval.*

Figure – 2: Primary outcome trajectories across all four time points (Mean ± 1 SD). 2A: 10-Metre Walk Test (m/s). 2B: Berg Balance Scale (/56). Gold dotted lines = clinically important function thresholds.

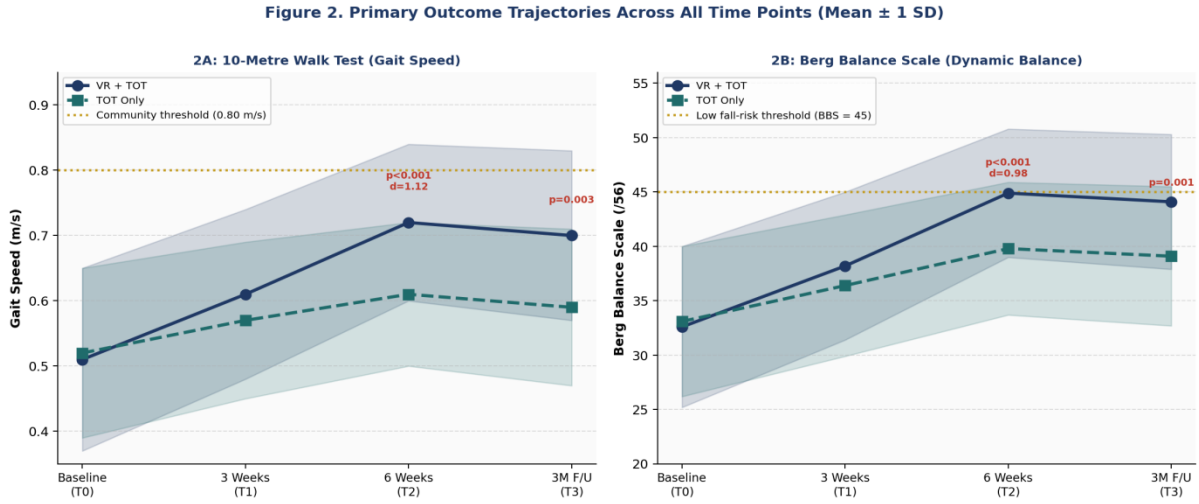
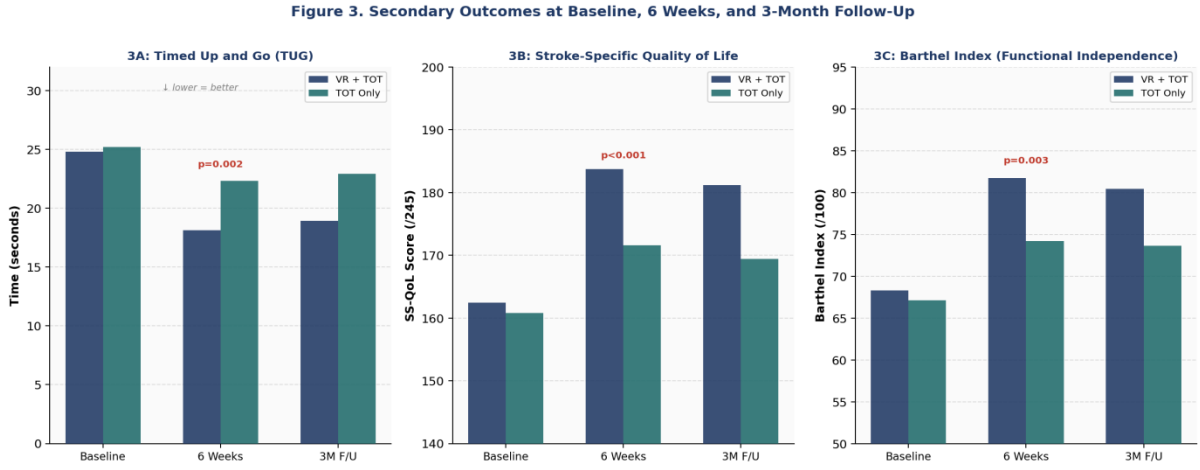


Table – 6: Secondary Outcomes at All Time Points (Mean ± SD or Median IQR).

Outcome	Group	Baseline	6 Weeks (T2)	3M F/U (T3)	p-value (T2)	Cohen's d
TUG (s)	VR+TOT	24.8±6.3	18.1±5.1*	18.9±5.4*	0.002	0.84
	TOT Only	25.2±5.9	22.3±5.4	22.9±5.6		
FAC (median)	VR+TOT	3 (2–3)	4 (3–4)*	4 (3–4)*	0.004	0.72
	TOT Only	3 (2–3)	3 (3–4)	3 (3–4)		
SS-QoL	VR+TOT	162.4±22.1	183.7±19.4*	181.2±20.1*	<0.001	0.91
	TOT Only	160.8±21.7	171.6±20.8	169.4±21.3		
Barthel Index	VR+TOT	68.3±12.4	81.7±10.6*	80.4±11.2*	0.003	0.76
	TOT Only	67.1±13.1	74.2±11.8	73.6±12.1		
MAS (median)	VR+TOT	1 (1–1+)	1 (1–1+)	1 (0–1)	0.612 (NS)	0.12
	TOT Only	1 (1–1+)	1 (1–1+)	1 (1–1+)		

Note: **p<0.05 vs TOT group at same time point. NS = Not Significant. d = Cohen's d. F/U = Follow-up; SS-QoL = Stroke-Specific Quality of Life; MAS = Modified Ashworth Scale.*

Figure – 3: Secondary outcomes at baseline, 6 weeks, and 3-month follow-up. 3A: TUG (seconds). 3B: SS-QoL (/245). 3C: Barthel Index (/100). Navy = VR+TOT; Teal = TOT Only.



Secondary Outcomes

All secondary outcomes demonstrated significantly greater improvements in VR+TOT at 6 weeks and 3-month follow-up (**Table – 6, Figure - 3**).

TUG:
VR+TOT: 24.8±6.3 to 18.1±5.1 s (−27.0%); TOT: 25.2±5.9 to 22.3±5.4 s (−11.5%). Between-group: −4.2 s (p=0.002; d=0.84). Exceeds MCID of 2.9 s.
FAC:
VR+TOT: median 3 to 4 (independent indoors) vs TOT: 3 to 3+. FAC=5 (community independent) achieved: VR+TOT 13.3% vs TOT 3.3% (p=0.004; d=0.72).
SS-QoL:
VR+TOT: +21.3 points vs TOT: +10.8 points (between-group: +10.6; p<0.001; d=0.91). Exceeds SS-QoL MCID of 10 points.
Barthel Index:
VR+TOT: +13.4 points vs TOT: +7.1 points (between-group: +6.3; p=0.003; d=0.76). Exceeds MCID of 5 points.
MAS:
No significant between-group difference (p=0.612; d=0.12), consistent with neither intervention specifically targeting spasticity.

Table – 7: Between-Group Differences, Effect Sizes, and Clinical Significance at 6 Weeks.

Outcome	Between-Group Diff (6W)	95% CI	p-value	Cohen's d	Interpretation	Exceeds MCID?	NNT
10MWT (m/s)	+0.18 m/s	0.12–0.24	<0.001	1.12	Very Large	Yes (0.16)	2.4
BBS (points)	+5.1 points	3.3–6.9	<0.001	0.98	Large	Yes (4 pts)	2.8
TUG (s)	−4.2 s	−6.8 to −1.6	0.002	0.84	Large	Yes (2.9 s)	3.1
SS-QoL (points)	+10.6 points	6.4–14.8	<0.001	0.91	Large	Yes (10 pts)	3.4
Barthel Index	+6.3 points	2.8–9.8	0.003	0.76	Moderate-Large	Yes (5 pts)	3.7
FAC	+0.8 categories	0.3–1.3	0.004	0.72	Moderate-Large	Yes	4.2

Note: *Cohen's d: 0.2=small; 0.5=medium; 0.8=large; ≥1.2=very large. NNT = Number Needed to Treat; MCID = Minimum Clinically Important Difference.*

Effect Sizes and Clinical Significance
All between-group differences exceeded their respective MCIDs, confirming both statistical and clinical significance. Effect sizes were uniformly large to very large (d ≥0.72) for all outcomes at 6 weeks (**Table – 7, Figure - 4**).

Within-Group Analysis
Within-Group Pre-Post Comparison at 6 Weeks is as per **Table – 8**.

Adherence and Safety Profile
Session adherence was excellent in both groups: VR+TOT 97.3% (mean 29.2/30 sessions); TOT 96.8% (29.0/30); no significant between-group difference (p=0.814). Per-protocol analysis (≥24 sessions) included 29/30 per group; results were consistent with the ITT analysis across all outcomes.
No serious adverse events were recorded in either group throughout the 6-week intervention or 3-month follow-up. Two VR+TOT participants (6.7%) reported mild transient simulator sickness (SSQ <6) during sessions 1–2; both self-resolved without session modification. No falls, cardiovascular events, or withdrawals due to adverse events occurred in either group (**Table – 9, Figure - 5**).

Figure – 4: 4A: Forest plot of Cohen's d effect sizes with 95% CI for all outcomes at 6 weeks. Dashed lines = small (0.2), medium (0.5), large (0.8), very large (1.2) thresholds. 4B: Within-group percentage change from baseline at 6 weeks.

Figure 4. Effect Size Analysis and Within-Group Percentage Improvements

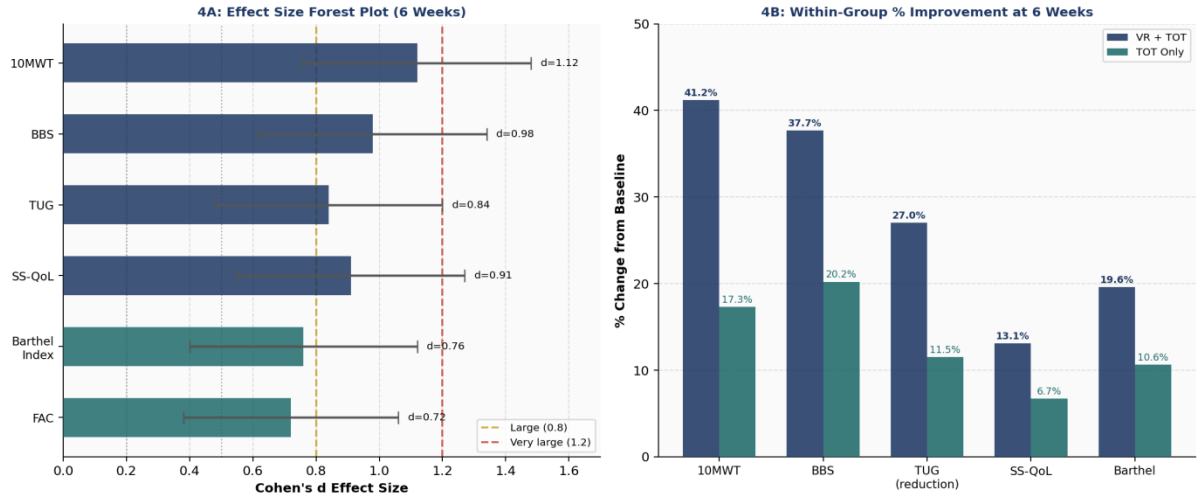


Table – 8: Within-Group Pre-Post Comparison at 6 Weeks (Paired t-test).

Outcome	VR+TOT Baseline	VR+TOT 6 Weeks	p (VR+TOT)	TOT Baseline	TOT 6 Weeks	p (TOT)
10MWT (m/s)	0.51±0.14	0.72±0.12	<0.001	0.52±0.13	0.61±0.11	<0.001
BBS (/56)	32.6±7.4	44.9±5.9	<0.001	33.1±6.9	39.8±6.1	<0.001
TUG (s)	24.8±6.3	18.1±5.1	<0.001	25.2±5.9	22.3±5.4	<0.001
SS-QoL	162.4±22.1	183.7±19.4	<0.001	160.8±21.7	171.6±20.8	<0.001
Barthel Index	68.3±12.4	81.7±10.6	<0.001	67.1±13.1	74.2±11.8	<0.001

Both groups demonstrated statistically significant within-group improvements for all outcomes (all p<0.001). VR+TOT produced consistently superior magnitude of change.

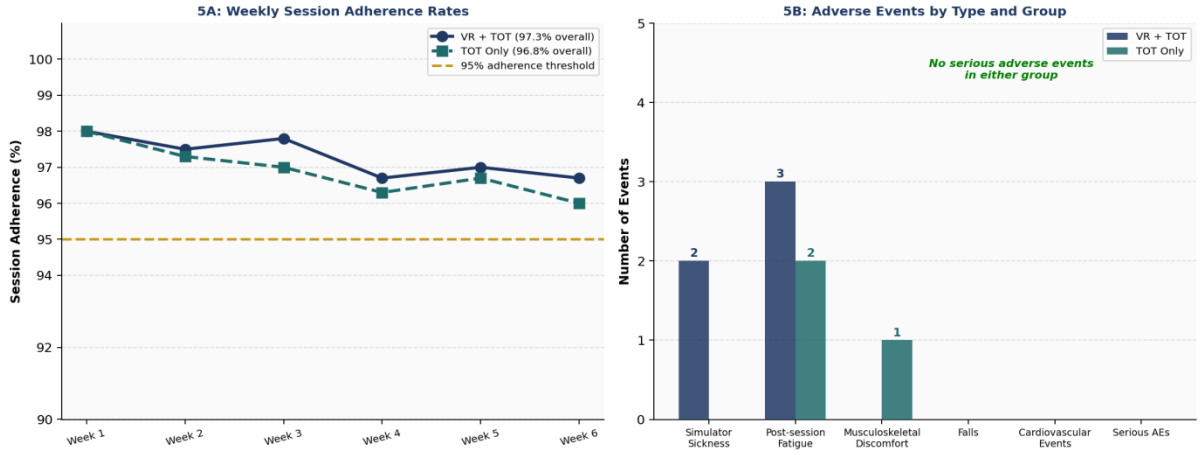
Table – 9: Adverse Events and Safety Profile – Both Groups (n = 60).

Adverse Event	VR+TOT (n=30)	TOT Only (n=30)	Outcome and Management
Mild simulator sickness (SSQ <15)	2 (6.7%)	0 (0%)	Self-resolving; no session modification required
Transient post-session fatigue	3 (10.0%)	2 (6.7%)	Rest until next session; no protocol modification
Musculoskeletal discomfort	0 (0%)	1 (3.3%)	Activity modification; continued within protocol
Falls during sessions	0 (0%)	0 (0%)	N/A – No events recorded
Cardiovascular events	0 (0%)	0 (0%)	N/A – No events recorded
Serious adverse events	0 (0%)	0 (0%)	N/A – No events recorded
Withdrawals due to adverse events	0 (0%)	0 (0%)	N/A – No events recorded

Note: *SSQ = Simulator Sickness Questionnaire. No serious adverse events in either group throughout the 6-week intervention or 3-month follow-up period.*

Figure – 5: 5A: Weekly session adherence rates (%). Gold dashed line = 95% threshold. 5B: Adverse events by type and group (n=60). No serious adverse events in either group.

Figure 5. Session Adherence and Adverse Events Safety Profile



Discussion

This RCT provides Level I evidence that combining immersive VR with structured TOT produces clinically meaningful, statistically superior, and durable improvements in gait, balance, functional ambulation, and quality of life in chronic stroke survivors, compared to equivalent-dose TOT alone. All between-group differences exceeded established MCIDs, and uniformly large-to-very-large effect sizes (Cohen's d: 0.72–1.12) confirm robust clinical relevance across all outcome domains.

Gait Speed and Community Ambulation

The between-group 10MWT difference of 0.18 m/s exceeded both the MCID (0.16 m/s) and the smallest real difference (SRD: 0.08 m/s), confirming genuine clinical change beyond measurement error. The VR+TOT group's mean gait speed at 6 weeks (0.72 m/s) approaches the limited community ambulation threshold of 0.8 m/s, while the TOT group remained below (0.61 m/s) [14]. Treadmill-integrated VR at 80% walking speed provides high-repetition stepping with real-time optic flow feedback that reinforces appropriate cadence and stride length. Virtual obstacle avoidance additionally engages anticipatory postural adjustments and adaptive locomotor responses mediated by supplementary motor area and basal ganglia circuits [15].

Dynamic Balance and Fall Risk Reduction

The VR+TOT group's mean BBS at 6 weeks (44.9 points) crosses the clinically important fall-risk threshold of 45 points – above which fall risk is substantially reduced – whereas the TOT group did not achieve this threshold (39.8 points) [16]. This qualitative functional transition has direct implications for safe discharge, reduced fall-related healthcare utilisation, and community reintegration. Enhanced balance gains reflect VR's enriched multisensory feedback environment, which challenges somatosensory re-weighting – a key deficit in post-stroke postural control. VR environments with visual conflict and virtual perturbations engage cerebellar and vestibular sensory integration mechanisms, promoting adaptive postural responses beyond those achievable with conventional land-based training [17].

Neuroplasticity Mechanisms

VR simultaneously engages multiple evidence-based motor learning principles: (1) High-repetition practice – gamification sustains engagement and voluntary effort; (2) Augmented multisensory feedback – real-time error correction via multimodal cues; (3) Variable practice scheduling – dynamic virtual environments promote motor schema generalisation; (4) Motivational reward – gamified tasks activate dopaminergic reward circuits modulating synaptic plasticity; (5) Dual-task training – cognitive-motor VR tasks engage prefrontal-motor connectivity and automatisation

of gait [18]. Neuroimaging evidence confirms enhanced corticospinal excitability and expanded motor cortical representation of the affected limb following VR-based gait training, consistent with activity-dependent neuroplasticity [19].

Quality of Life and Functional Independence

VR+TOT's superior SS-QoL improvement (+21.3 vs +10.8 points; exceeding MCID of 10 points) reflects clinically meaningful quality-of-life gains integrating physical, cognitive, emotional, and social dimensions of recovery. Superior Barthel Index gains (+13.4 vs +7.1 points) indicate greater functional independence in activities of daily living, with direct relevance to caregiver burden, healthcare costs, and community participation.

Durability of Treatment Effects

Sustained VR+TOT superiority at 3-month follow-up (all $p<0.01$) suggests consolidation of motor engrams beyond active training – a hallmark of genuine neuroplastic reorganisation rather than transient performance enhancement. This durability is consistent with evidence that enriched, high-repetition, variable practice induces long-term potentiation (LTP)-like synaptic changes in motor cortical networks. Future studies should evaluate sustainability at 6 and 12 months.

Comparison with Existing Literature

Our effect sizes (d=1.12 for 10MWT; d=0.98 for BBS) substantially exceed those reported in prior meta-analyses (SMD: 0.43–0.57) [13], likely reflecting: (1) additive benefit of combining VR with structured TOT rather than replacing conventional care; (2) fully immersive HMD-based VR versus semi-immersive systems used in most prior studies; and (3) a chronic stroke population receiving targeted, high-dose, progressive rehabilitation with algorithmic difficulty adaptation and rigorous protocol fidelity.

Limitations

(1) Single-centre design; multicentre replication across diverse South Indian settings is needed. (2) Participant and therapist blinding not feasible due to the intervention nature; single-blind assessor design mitigates but does not eliminate this bias. (3) Follow-up limited to 3 months; 6- and 12-month durability requires evaluation. (4) Cost-effectiveness not evaluated; VR technology acquisition and maintenance costs may be a barrier in low-resource settings. (5) Neuroimaging was not performed; neuroplastic mechanisms remain inferred from functional outcomes. (6) Generalisability to subacute stroke or FAC <2 requires separate investigation.

Conclusion

This Randomized Controlled Trial provides Level I evidence that immersive Virtual Reality combined with Task-Oriented Training is safe, feasible, and produces clinically meaningful, durable, and statistically superior improvements in gait speed, dynamic balance, functional ambulation, and health-related quality of life compared to Task-Oriented Training alone in chronic post-stroke individuals. All between-group differences exceeded minimal clinically important differences, effect sizes were uniformly large to very large, and gains were maintained at 3-month follow-up with no serious adverse events. These findings support the clinical integration of immersive VR as an evidence-based adjunct to structured task-oriented neurological rehabilitation for eligible chronic stroke survivors. Future multicentre RCTs should investigate long-term sustainability (≥12 months), cost-effectiveness, neuroimaging correlates, dose-response relationships, and applicability to subacute and severely impaired post-stroke populations.

Declarations

Ethical Approval: Approved by Institutional Ethics Committee (IEC/PRRM/2024/08). All procedures conformed to the Declaration of Helsinki (2013 revision).

Informed Consent: Written informed consent obtained from all participants prior to enrolment. Right to withdraw without consequences was explicitly communicated.

Data Availability: De-identified participant-level data available from the corresponding author on reasonable written request, subject to applicable ethical guidelines.

Acknowledgements: The author gratefully acknowledges all participants and their families for their commitment and trust throughout the study, and the staff of the Neurological Rehabilitation Unit, P.R.R.M. College of Physiotherapy, Kadapa.

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