

## THE TRIPLE FEEDBACK DISRUPTION (TFD) MODEL: A PROPOSED MODEL LINKING UNILATERAL NEGLECT, APRAXIA AND LEARNED NON-USE AFFECTING MOVEMENT AFTER STROKE

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### Abstract

**Background:** Stroke remains a leading cause of long-term disability worldwide, with upper-limb dysfunction significantly reducing independence and participation. Beyond motor weakness, complex cognitive-sensorimotor impairments such as unilateral spatial neglect, limb apraxia, and learned non-use play a substantial role in determining rehabilitation outcomes. Evidence suggests these impairments frequently coexist and interact, magnifying disability, yet their interrelationships remain poorly understood.

**Objective:** This review aimed to synthesize current evidence on the interaction among unilateral neglect, limb apraxia, and learned non-use following stroke, and to propose the Triple Feedback Disruption (TFD) Model, a conceptual framework explaining how disruptions in sensory feedback, motor planning, and reinforcement learning form a self-perpetuating cycle limiting functional upper-limb recovery.

**Materials and Methods:** A narrative review was conducted using PubMed, Google Scholar, Scopus, and Web of Science, covering studies published from 2015 to 2025. Search terms included *neglect, apraxia, learned non-use, stroke rehabilitation, and sensorimotor integration*. Eligible studies involving adults post-stroke discussing any of the three impairments or their rehabilitative implications were qualitatively synthesized.

**Results:** Twenty-nine studies were included. Findings indicate strong evidence of interaction among the three impairments, with neglect reducing sensory engagement, apraxia limiting motor planning, and learned non-use reinforcing behavioural disuse. Co-occurrence of these deficits predicts poorer therapy response and functional outcomes compared to isolated impairments.

**Conclusion:** The TFD Model provides a unified explanation for persistent upper-limb dysfunction after stroke, highlighting the need for integrated rehabilitation targeting sensory-attention, motor-planning, and behavioural reinforcement simultaneously. This framework may guide future multimodal intervention trials and personalised rehabilitation strategies.

**Keywords:** Stroke Rehabilitation, Unilateral Neglect, Limb Apraxia, Learned Non-Use, Sensorimotor Integration

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## 1.0 Introduction

Stroke is one of the primary causes of adult disability globally, with many survivors experiencing lasting upper-limb impairments that restrict independence, participation, and overall quality of life [1]. Beyond simple muscle weakness, stroke often results in complex interactions of sensorimotor and cognitive deficits, which can limit functional recovery and reduce responsiveness to standard rehabilitation approaches [2]. Gaining a deeper understanding of how these deficits interact is therefore crucial for developing targeted interventions that restore meaningful upper-limb function.

Unilateral spatial neglect, commonly referred to as “neglect,” is a multifaceted disorder affecting attention and spatial awareness, in which individuals fail to perceive, respond to, or utilize information on the side opposite their brain lesion [2]. Apraxia is a higher-order motor disorder in which patients cannot plan or execute learned, purposeful movements despite retaining sufficient strength and primary sensation [3]. Its presence predicts worse rehabilitation outcomes, independent of basic motor deficits [4,3]. Critically, apraxia impairs the translation of intention into precise, timely motor commands, leading to failed attempts, task errors, and reduced practice opportunities that would otherwise drive motor relearning [3]. Learned non-use is a behavioral and neuroplastic phenomenon in which patients gradually stop attempting to use the affected limb because initial attempts are inefficient, frustrating, or unrewarding [5]. Over time, habitual reliance on the unaffected limb develops, and neural representations of the paretic limb weaken, reducing spontaneous use even when some motor function returns [1]. Learned non-use effectively converts remaining motor capacity into functional loss and is a central

target of behavioral therapies such as constraint-induced movement therapy (CIMT) [5]. Evidence suggests that the presence of cognitive barriers, including attention and planning deficits, further increases the likelihood of arm non-use and reduces responsiveness to conventional training [6,1].

Empirical research demonstrates that neglect, apraxia, and learned non-use frequently co-occur and interact. Observational data indicate that patients with neglect are more likely to exhibit proprioceptive impairments and poor upper-limb performance, which increase the risk of developing learned non-use [7]. Disturbances in proprioception and multisensory processing associated with neglect reduce the accuracy of feedback needed for online motor control and error-based learning [8]. Prospective cohort studies and early rehabilitation investigations show that both apraxia and neglect, independently and together, predict poorer functional outcomes and limit gains during inpatient therapy, suggesting overlapping effects on recovery trajectories [1]. In a multi-centre early rehabilitation cohort, the severity of both apraxia and neglect significantly predicted discharge functional status [4]. Studies examining predictors of arm non-use highlight cognitive factors such as attention and planning, alongside higher-order motor deficits like apraxia, as key contributors to persistent non-use, indicating that behavioral avoidance of the limb is influenced not only by motor limitations but also by sensory-attentional and planning systems [1].

Taken together, this evidence suggests that deficits in sensory registration (neglect), motor planning and execution (apraxia), and behavioral reinforcement (learned non-use) may mutually reinforce one another, creating a persistent cycle of disuse and impaired motor learning. While previous work has

documented pairwise relationships (e.g., neglect ↔ reduced limb use; apraxia ↔ poorer ADL performance), there is a lack of conceptual frameworks and empirical studies that simultaneously examine all three phenomena or test their dynamic interactions during rehabilitation. Therefore, this study aims to review relevant literature to develop and evaluate the Triple Feedback Disruption (TFD) model, which based on the narrative synthesis will hypothesize that unilateral neglect, limb apraxia, and learned non-use interact in a feedback loop that sustains upper-limb dysfunction after stroke.

## 2.0 Methodology of the Narrative Review

This narrative review aimed to synthesize current evidence on the interplay between unilateral neglect, limb apraxia, and learned non-use following stroke, with the goal of informing the development of the proposed TFD Model. While narrative reviews do not follow the strict protocols of systematic reviews, methodological transparency was maintained to ensure rigor, reproducibility, and reliability of the findings (Figure 1).

### 2.1 Search Strategy

A thorough literature search was conducted using major scientific databases, including PubMed, Google Scholar, Scopus, and PEDro. The search period spanned January to February 2025 and focused on studies published between 2015 and 2025 to capture the most recent evidence. Keywords and Boolean operators were applied in multiple combinations, including: {“*unilateral spatial neglect*” OR “*hemispatial neglect*” AND *stroke* AND *upper limb*}, {*apraxia* AND *stroke* AND *motor planning*}, {“*learned non-use*” OR “*nonuse*” AND *stroke* AND *rehabilitation*}, {*interaction* OR *relationship* OR *association* AND *neglect* AND *apraxia* AND *learned non-use*}, and {*sensorimotor integration* AND *stroke* AND *recovery*}.

Medical Subject Headings (MeSH) terms such as *Apraxias*, *Stroke Rehabilitation*, *Motor Cortex*, *Sensory Feedback*, and *Arm* were incorporated to enhance search sensitivity. Additionally, reference lists of key studies and review articles were manually searched to identify further relevant publications.

### 2.2 Inclusion and Exclusion Criteria

Studies were eligible for inclusion if they examined neglect, apraxia, or learned non-use in adult stroke populations. They needed to investigate rehabilitation outcomes, recovery mechanisms, neurophysiological aspects, or the interactions among these impairments. Included study designs comprised observational, interventional, cohort, or review articles, published in English between 2017 and 2025.

Studies were excluded if they focused exclusively on children or non-stroke neurological populations, presented qualitative or anecdotal findings without clinical or quantitative evidence, or did not address upper-limb functional outcomes.

### 2.3 Study Screening and Selection

Titles and abstracts retrieved through the database search were first screened for relevance, followed by a full-text review of eligible articles. Studies meeting the inclusion criteria were synthesized qualitatively. Any disagreements about study selection were resolved through consensus discussions among the reviewers.

Given the heterogeneity of study designs and outcome measures, a narrative synthesis approach was adopted instead of meta-analysis. 29 articles were found to be eligible for inclusion, the findings were organized thematically, first examining individual impairments, then exploring evidence of their interactions, and finally discussing implications for rehabilitation.

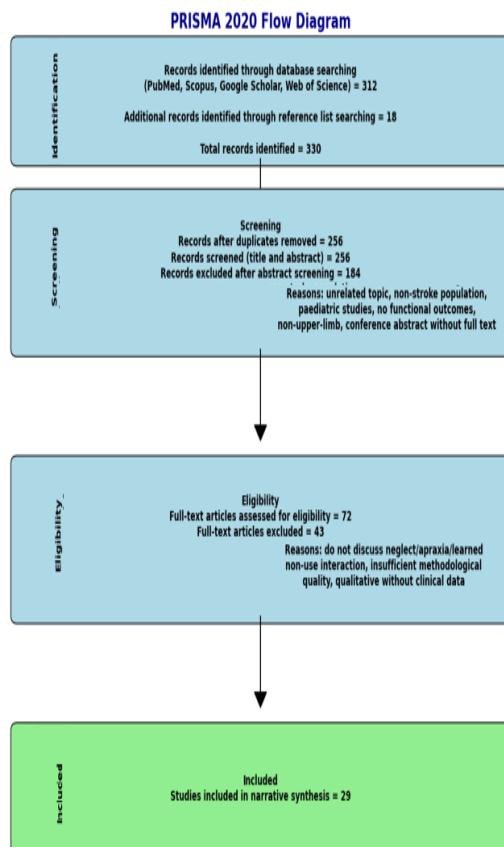


Figure 1: PRISMA flow chart

### 3.0 Results

**3.1 Neurophysiological Basis of Upper Limb Impairment After Stroke**  
 Upper-limb deficits after stroke result not only from muscle weakness but also from disruption of neural networks responsible for sensorimotor integration, spatial attention, movement planning, and reinforcement learning. Damage to cortical and subcortical regions, the cerebellum, and white matter tracts impairs coordinated movement. Recovery relies on intact neural feedback loops, and when these are disrupted, movements become inefficient, quality declines, and patients increasingly compensate with the unaffected limb [9]. Damage to the sensorimotor network reduces motor drive and plasticity, while injury to frontoparietal attention networks impairs spatial perception and attentional control needed for precise limb movements.

Spatial impairments, such as unilateral neglect, often result from impaired multisensory integration (visual, tactile, proprioceptive) in parietal areas, especially in the right hemisphere [10]. This impairment reduces awareness of the affected limb, degrading the sensory feedback needed for real-time error correction and motor adaptation. Imaging and connectivity studies show that neglect is linked to weakened functional connections between parietal and premotor areas, which may explain why individuals with neglect struggle to initiate or monitor movements effectively [9,10].

Similarly, limb apraxia is related to interruption in networks that support the planning, sequencing, and internal modeling of learned actions, particularly in the left inferior parietal lobule and premotor cortex [11]. When connections between intention

and execution are disrupted, patients may retain strength and sensation but lose the ability to produce well-organized, purposeful movements. Meta-analytic and lesion studies suggest that these left-hemisphere regions are critical for ideomotor and ideational apraxia [12]. Apraxia limits effective motor practice and inhibits learning, because patients cannot reliably translate their goals into appropriately timed motor commands.

Although learned non-use is primarily behavioral, it reflects underlying neurophysiological change. Repeated failures or inefficient movements with the paretic limb reduce attempts to use it, which over time weakens its cortical representation and synaptic connectivity [10,11]. This creates a maladaptive feedback loop: negative experiences depress the brain's engagement of the impaired limb, pushing behavior toward more successful compensatory patterns using the intact side. Reinforcement learning frameworks help explain this: early failure generates negative

reinforcement, biasing behavior toward the unaffected limb and suppressing motor circuits in the damaged hemisphere.

Thus, stroke disrupts the integrity of three interconnected systems:

1. Sensory-attentional processing (neglect)
2. Motor planning and execution (apraxia)
3. Reinforcement-based motor learning (learned non-use)

These systems normally interact through feedback-driven adaptation essential for motor recovery. When disrupted simultaneously, a self-sustaining cycle of impairment emerges, explaining persistent disability despite adequate motor potential. This neurophysiological framework provides the rationale for the TFD Model (Figure 2), which conceptualizes how these interconnected deficits interact to restrict functional arm use.

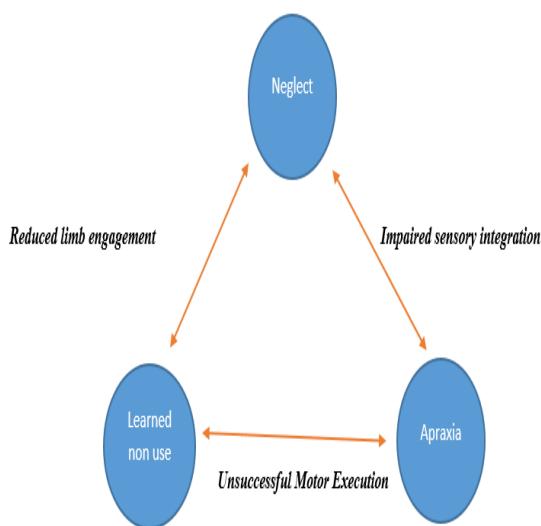


Figure 2: Vicious cycle of TFD Model

### 3.2 Unilateral Spatial Neglect in Post-Stroke Patients

Unilateral spatial neglect is one of the most disabling cognitive-perceptual disorders following stroke. It is characterized by an impaired ability to detect, orient to, or respond to stimuli on the side of space opposite the cerebral lesion, despite intact primary visual and sensory pathways [2]. The condition occurs most frequently after right-hemisphere stroke, resulting in left-sided neglect, although it can also arise following damage to the left hemisphere. Neglect reflects disturbances in multi-modal spatial representation rather than loss of sensory reception, affecting visual, tactile, proprioceptive, and auditory awareness [1]. As a result, individuals with neglect may behave as if the affected side of their body or environment does not exist, significantly compromising independence and participation in daily life. Prevalence estimates vary widely due to differences in assessment tools and timing of evaluation. A systematic review found the overall prevalence of spatial neglect after unilateral stroke to be about 29%, with higher rates following right-brain damage (38%) versus left-brain damage (18%) [13]. Other studies report early-stage prevalence up to ~43% after right-hemisphere injury and about 20% after left-hemisphere lesion [2]. USN is strongly linked with longer hospital stays, reduced gains from rehabilitation, slower and less complete motor recovery even when muscle weakness is equivalent and greater caregiver burden [14].

From a neurophysiological perspective, neglect arises from dysfunction across widespread attention networks, particularly the right-hemisphere dorsal and ventral attention systems and parietal cortex, resulting in deficits in vision, proprioception,

and multimodal spatial representation. By downplaying sensory input from the affected side, neglect reduces spontaneous use of the contralesional limb and limits the sensorimotor experiences necessary for recovery [15].

### 3.3 Limb Apraxia After Stroke

Limb apraxia is a higher-order motor cognitive disorder resulting from brain injury, most commonly stroke, and is characterised by a failure to perform learned, purposeful movements despite preserved basic motor, sensory, and comprehension abilities. This impairment goes beyond weakness or paralysis and reflects disruption in the planning, sequencing, and execution of skilled actions [16]. Limb apraxia is classically divided into ideomotor and ideational (or conceptual) subtypes. Ideomotor apraxia involves impaired translation of an intended movement into the correct motor programme (e.g., inability to pantomime tool use), whereas ideational apraxia refers to breakdown of the conceptual knowledge required to carry out a multi-step action sequence (e.g., making coffee) [16]. Prevalence estimates in stroke populations vary, with earlier studies reporting up to 30–50% of survivors affected; more recent reviews suggest a substantial number of patients demonstrate clinically meaningful praxis deficits in the upper limb, yet many go undetected in standard rehabilitation settings [16].

The neural substrates of apraxia span a wide network, including the left inferior parietal lobule, premotor cortex, supplementary motor area, and their interconnections with sensory and motor output regions. Damage to these networks interrupts the translation of intention into movement, disrupts the representation of tool use actions, and interferes with spatial-temporal sequencing of action units [3]. This results in mis-

grasping, improper tool use, inefficient movement trajectories, and increased error rates during task execution. The functional consequences are profound: patients with apraxia frequently struggle with activities of daily living (ADLs), such as grooming, dressing, and utensil use, independently of primary motor paresis.

### 3.4 Learned Non-Use Phenomenon After Stroke

Learned non-use is a behavioural and neuroplastic phenomenon following neurological injury in which a stroke survivor gradually abandons use of the affected upper limb despite retaining some motor capability. Originally described by Edward Taub in primate de-afferentation experiments, the concept has been extended to human stroke rehabilitation. When early attempts to move the limb repeatedly fail (frustration, inefficiency, discomfort), negative reinforcement leads the individual to increasingly rely on the unaffected limb. Over time, this compensation becomes habitual, suppressing attempts to use the affected limb even when neurological recovery might permit functional movement [17]. Learned non-use involves maladaptive re-organisation within motor networks. Reduced attempts to use the paretic limb lead to diminished corticospinal excitability, weakened synaptic pathways, and contraction of the cortical representation of that limb. For example, studies show reduced motor cortical maps and altered interhemispheric connectivity in individuals exhibiting arm non-use [1].

Learned non-use is distinguished from pure biomechanical inability: patients may demonstrate adequate capacity in structured assessments but fail to use the affected arm spontaneously in real-life settings. This capacity–performance gap is now recognized as a key behavioural marker in motor

recovery research [17]. Clinically, learned non-use contributes to persistent deficits in independence, limits participation, increases caregiver burden, and reduces engagement in rehabilitation. Importantly, it often predicts long-term functional outcomes more strongly than initial paresis severity [1].

## 4.0 Unique Features of the TFD Model

To the best of our knowledge this is the first model to explicitly connect these three conditions as a single interdependent system. Prior models typically treat them as isolated phenomena. It also emphasizes the role of disrupted feedback systems in maintaining and exacerbating the triad. It also integrates behavioral, neuroanatomical, and physiological evidence into a cohesive mechanism.

## 4.1 Clinical Implications

The TFD Model suggests that treating one condition can disrupt the vicious cycle and improve the others:

- **For Neglect:** Using sensory reactivation therapies (such as prism adaptation, visual scanning) to re-establish feedback loops. Techniques to improve attention and awareness of the neglected side, can reduce the impact of neglect [18].
- **For Learned Non-Use:** introducing Constraint-Induced Movement Therapy (CIMT) can enforce use and enhance cortical representation. CIMT addresses learned non-use by constraining the unaffected limb and encouraging use of the affected limb, thereby strengthening neural connections [6].
- **For Apraxia:** by implementing errorless motor learning and guided practice to improve motor planning. Motor imagery exercises and repetitive task-specific training can help patients with apraxia relearn motor planning and execution [19].

Within the TFD Model, limb apraxia occupies the motor-planning disruption node.

Specifically, when praxis systems fail, attempted movements become inefficient or erroneous, leading to frustration or reduced success, which in turn reduces reinforcement and practice opportunities. In other words: poor motor planning → increased movement errors → decreased movement attempts → increased learned non-use. Moreover, apraxia may also amplify sensory-attentional deficits by making movement monitoring more difficult and increasing reliance on external cues. Therefore, incorporating apraxia assessment and targeted intervention is critical for breaking the cycle envisioned in the TFD Model.

## 5.0 Discussion

The present narrative review has synthesized evidence that three critical post-stroke impairments, unilateral spatial neglect, limb apraxia, and learned non-use do not simply co-exist by coincidence but rather form an interacting network of deficits that undermines functional upper-limb recovery. This has important theoretical, clinical and translational implications.

First, the evidence affirms that each of the three impairments independently predicts worse outcomes. For instance, severity of visuospatial neglect has been shown to significantly predict poorer functional recovery and prolonged rehabilitation stays [1]. Similarly, apraxia severity predicts cognitive and functional outcomes beyond motor impairment [4]. Learned non-use, in turn, is increasingly identified as a behavioural mediator linking motor capacity to actual arm use: patients may retain physical ability yet fail to use the limb, because of behavioural suppression of movement attempts [17].

Second, and more importantly, the literature increasingly supports interaction among these impairments. Studies show that patients

presenting with neglect are more likely to have apraxia and that both deficits contribute to non-use of the affected limb. For example, the study by Latarnik et al. [4] found that apraxia and neglect severity were significant predictors of early rehabilitation outcome. The fact that neglect and apraxia share attention- and action-based neural networks fosters a mechanistic bridge between perceptual-attentional disruption and motor-planning failure.

Third, the behavioural consequence, learned non-use can itself be seen as the outcome of a disrupted feedback loop: when attentional registration (neglect) is poor, movement attempts are less frequent and less successful; when motor planning (apraxia) is poor, the attempted movements are inefficient or fail; together these reduce successful movement practice and reinforcement, leading to habitual disuse of the limb (learned non-use). This chain of events supports the TFD model's central proposal that sensory-feedback disruption, motor-planning disruption and reinforcement disruption are linked in a self-sustaining cycle.

The TFD Model offers a conceptual framework that integrates these three domains in a single cycle. By doing so, it helps explain why some stroke survivors fail to recover meaningful upper-limb function despite ostensibly adequate motor capacity and conventional therapy. Specifically, it predicts that unless all three nodes of the cycle are addressed, attentional/sensory registration, motor planning, and reinforcement of use rehabilitation gains may be limited and transient.

The model also anticipates that the severity or presence of one node may amplify the impact of the others: for example, severe neglect may mitigate the benefits of motor-planning interventions, and pronounced

apraxia may blunt the effect of behavioural reinforcement strategies. This is consistent with clinical observations: patients with neglect and apraxia often respond poorly to pure motor therapies or standard constraint-induced training.

Clinically, the findings and model collectively suggest that assessment of post-stroke patients should routinely include screening for neglect, apraxia and arm-use behaviour (i.e., non-use) rather than focusing on motor strength/impairment alone. Rehabilitation programmes may need to be redesigned to incorporate multimodal interventions targeting:

- Enhancement of sensory-attentional awareness (e.g., visual scanning, prism adaptation, multimodal stimulation)
- Motor-planning training (e.g., tool use training, gesture imitation, strategy coaching)
- Behavioural reinforcement of affected-limb use (e.g., shaping, forced use, feedback systems, real-world integration)

By combining these components, therapy may break the vicious cycle: improved attention allows more meaningful movement practice; improved planning makes practice more effective; increased successful practice reinforces use and further consolidates neural reorganisation.

Several limitations must be noted. The empirical literature is still somewhat limited in terms of large-scale, longitudinal or interventional studies that explicitly address all three domains simultaneously. Many studies focus on single impairments (neglect or apraxia or non-use) and rarely track their dynamic interaction over time [3].

## 6.0 Conclusion

By synthesising the literature on neglect, apraxia and learned non-use, and by embedding it in the integrated framework of the TFD Model, this discussion highlights that persistent upper-limb dysfunction after stroke is rarely attributable to a single impairment. Rather, a network of disruption in sensory-attentional registration, motor planning, and behavioural reinforcement collaborates to maintain functional loss. Recognising and addressing this complex interplay offers new avenues to optimise rehabilitation and improve meaningful recovery of limb use.

## 7.0 Recommendation

1. Future studies should empirically evaluate integrated rehabilitation protocols that concurrently address neglect, apraxia, and learned non-use. Comparative trials against single-domain interventions would provide evidence on potential synergistic benefits and optimal therapy dosage.
2. Interventional studies are required to validate the effectiveness of the proposed clinical implications and therapies showing how improvement in one domain will improve others.
3. There will be need to use imaging method (such as fMRI) to identify overlapping and distinct neural pathways linking the three conditions so as to validate the model.
4. To properly predict the model, there will be need to develop computational models to simulate how disruptions in one domain affect the others.

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## Authors' contributions

**IAA** draft the methodology and designed the manuscript, proposes and develop the model. **AA** conceived the idea and supervised the review, **HYA** and **MAB** draft the assisted in screening the relevant articles and proof read the entire manuscript. **AHG** and **ZA** draft the conclusion and discussion section of the manuscript. All authors approve the final version of the study.

### Competing Interests

All authors declare no conflict of interest

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