



REVIEW ARTICLE

Advances in the application of web-based cognitive stimulation in improving cognitive function in patients with cognitive impairment after stroke

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ABSTRACT

Stroke is a leading cause of long-term disability worldwide, with post-stroke cognitive impairment (PSCI) affecting over 70% of survivors and significantly diminishing their independence and quality of life. In Indonesia, the prevalence of PSCI exceeds 60%, the highest in Southeast Asia, creating a critical demand for effective and accessible rehabilitation strategies. This review addresses the effectiveness of web-based cognitive stimulation applications in enhancing cognitive recovery, specifically during the first three months post-stroke, which is identified as the "golden period" for recovery due to heightened cortical plasticity. In this review, we systematically searched and analyzed articles published in English from the date of inception up to December 31st, 2025, using PubMed, Science Direct, Google Scholar, Cochrane Central Library, and SpringerLink. Relevant search terms included "POST-STROKE COGNITIVE IMPAIRMENT" AND ("COGNITIVE REHABILITATION" OR "COGNITIVE STIMULATION" OR "WEB-BASED COGNITIVE TRAINING"). The rationale for this review stems from the need to overcome barriers in traditional rehabilitation, such as high clinician workload and limited accessibility, by utilizing technology-based interventions that facilitate telerehabilitation. Major studies and paradigms reviewed include the Montreal Cognitive Assessment (MoCA-Ina) for sensitive screening, as well as specific training tasks such as the Stroop Effect and Go/No-Go paradigms. Key findings from some researchers demonstrate significant improvements in MoCA-Ina scores following computerized training, while some meta-analyses suggest that Go/No-Go tasks are highly effective in improving inhibitory control. The review concludes that web-based applications offer a practical and effective solution for individualized, repetitive cognitive training that can be accessed without specialized software installation. Furthermore, these digital tools support continuity of care after hospital discharge. To maximize clinical impact, future research must focus on integrating these prototype applications into standardized clinical practice and establishing a clearer consensus on the diagnostic timing of post-stroke dementia.

Keywords: Stroke, cognitive impairment, cognitive stimulation, web-based application.

ABBREVIATIONS

A β	: Amyloid beta
AD	: Alzheimer's disease
ADL	: Activities of Daily Living
C	: Color
CW	: Color Word

DALYs	: Disability-Adjusted Life Years
EF	: Executive Function
GBD	: Global Burden Disease
IL-1 β	: Interleukin-1 beta
IL-6	: Interleukin-6
MCI	: Mild Cognitive Impairment
MMSE	: Mini-Mental State Examination
MoCA	: Montreal Cognitive Assessment
APP	: Amyloid-beta Precursor Protein
MoCA-Ina	: Montreal Cognitive Assessment Indonesian Version
PSD	: Post-Stroke Dementia
PSCI	: Post-Stroke Cognitive Impairment
PSCID	: Post-Stroke Cognitive Impairment and Dementia
SVD	: Small Vessel Disease
SVZ	: Subventricular Zone
TNF- α	: Tumor Necrosis Factor-alpha
W	: Word
WCST	: Wisconsin Card Sorting Test
WHO	: World Health Organization

INTRODUCTION

Stroke is a significant health problem worldwide and a leading cause of long-term disability and death. Cognitive impairment refers to a range of deficits in cognitive functions, including memory, attention, executive function, language, and visuospatial abilities. Understanding the relationship between stroke and cognitive impairment is crucial for effectively managing symptoms and improving the quality of life of stroke survivors.⁽¹⁾

According to the Global Burden of Disease (GBD) study, stroke ranks third as the leading cause of death, responsible for 7.3 million deaths, accounting for 10.7% of all deaths.⁽²⁾ Globally, ischemic stroke accounts for 65.3% (62.4%–67.7%) of stroke cases, intracerebral hemorrhage for 28.8% (28.3%–28.8%), and subarachnoid hemorrhage for 5.8% (5.7%–6.0%)⁽³⁾. Stroke is also the fourth most common cause of disability-adjusted life years (DALYs), accounting for 160.5 million DALYs.⁽²⁾ In Southeast Asia, there were 11.93 million cases with 880,000 deaths in 2021.⁽²⁾

Recent studies have shown that cognitive impairment commonly occurs after stroke, with cognitive deficits found in more than one-third of stroke survivors.⁽⁴⁾ Numerous studies have demonstrated a strong link between stroke and cognitive impairment. Cognitive deficits experienced by stroke survivors can significantly impact their daily functioning, independence, and overall well-being.⁽¹⁾

Although the development of effective stroke treatments has shown improvements in stroke outcomes, post-stroke cognitive impairment and dementia (PSCID) remain highly prevalent and often leads to disability. Cognitive deficits are present in over 70% of stroke survivors, depending on the type of stroke, definition, and time point of assessment. PSCID is associated with disability and morbidity, placing a significant burden on patients, caregivers, and the healthcare system.^(5,6) In Indonesia, the prevalence of PSCID exceeds 60%, indicating the highest prevalence of PSCID in Southeast Asia. However, the overall prevalence of PSCID does not differ substantially from global prevalence rates.⁽²⁾

Cognitive recovery may be limited in patients with multiple comorbidities, polypharmacy, advanced age, and a history of prior cognitive decline. This cognitive impairment can progress to post-stroke dementia (PSD). In fact, the temporal pattern of PSD based on clinical observations varies widely. Cognitive impairment may begin at the onset of stroke and remain stable, begin at stroke onset and progressively worsen, develop after recurrent strokes, or emerge more than 3 to 6 months after stroke.⁽⁷⁾

Rehabilitation is currently the most effective intervention for improving recovery in stroke patients, and its effectiveness is influenced by the time interval between stroke onset and the start of therapy.⁽⁷⁾ Several previous studies have highlighted that the first 3 months after stroke are a golden period for functional recovery, due to the

persistence of cortical plasticity. Cortical plasticity is supported by cortical reorganization and structural changes in the brain. Ischemia induces the growth of new dendrites and axons, both in the perilesional cortex and in areas remote from the lesion.⁽⁸⁾ Therefore, it is crucial to initiate therapy as soon as possible.

In recent years, technology-based interventions have shown potential in cognitive rehabilitation. Clinical trials have confirmed benefits in restoring cognitive abilities and reducing recovery time. The advantages of device-based rehabilitation include optimizing treatment duration, reducing clinician workload, and feasibility for telerehabilitation. These devices provide individualized and repetitive activities with immediate feedback, promoting independence, tracking progress and adherence, and enabling continuity of care after hospital discharge.^(9,10) In Indonesia, technology-based cognitive rehabilitation interventions are still quite rare. Existing studies are still prototypes in the form of applications that can be installed on software.⁽¹¹⁾ Website-based applications offer the advantage of being accessible from anywhere with an internet connection and do not require downloading or installation, making them more practical and potentially more helpful for stroke survivors to perform daily rehabilitation exercises.⁽¹²⁾

METHODS

In this review, we searched the literature for articles on post-stroke cognitive impairment and cognitive stimulation published in English and Indonesian. Relevant articles were retrieved from several online databases such as PubMed, Science Direct, Google Scholar, Cochrane Central Library, and SpringerLink. The keywords used to perform a literature search from the date of inception up to December 31st, 2025, were “POST-STROKE COGNITIVE IMPAIRMENT” AND (“COGNITIVE REHABILITATION” OR “COGNITIVE STIMULATION” OR “WEB-BASED COGNITIVE TRAINING”). All searches were performed using the same keywords. Any duplicate records were removed after obtaining the initial results. Relevant articles were sorted by screening their titles and abstracts. Lastly, the relevance of the remaining records was assessed based on the inclusion and exclusion criteria. Inclusion criteria comprised original research articles, systematic reviews, meta-

analyses, and clinical trials published in English or Indonesian that specifically examined cognitive outcomes following stroke and the application of technology-based cognitive interventions. Exclusion criteria included opinion pieces, conference abstracts without full text, and studies focusing exclusively on non-stroke neurodegenerative conditions such as Alzheimer’s disease without vascular components criteria.

Initially, 1896 articles were found to match inclusion criteria, but eventually only 52 articles were selected for analysis and synthesis. Articles were removed due to duplication, lack of access, and irrelevance to the topic, as seen in Figure 1.

Clinical overview of cognitive impairment after stroke

Stroke represents a significant global health burden in terms of morbidity and mortality. An estimated 15 million people experience a stroke each year worldwide. The incidence and prevalence of stroke vary across populations and regions.⁽¹⁾ Cognitive impairment is a relatively common complication in stroke patients. Roughly, around 39-47% of stroke survivors experience cognitive deficit.⁽¹³⁾ A large international cohort study showed that stroke is associated with acute cognitive decline and accelerated long-term decline in global cognition and specific cognitive domains compared with individuals without stroke. In this study, stroke survivors experienced a reduction in global cognition ranging from -0.17 to -0.22 standard deviations compared with adults without stroke. Domains significantly affected included processing speed, executive function, and language.⁽⁴⁾

Post-stroke cognitive impairment (PSCI) encompasses any degree of cognitive impairment resulting from a stroke, while post-stroke dementia (PSD) represents the most severe end-stage of PSCI. Post-stroke dementia is dementia that occurs after a stroke.⁽⁷⁾

An acute stroke in the brain with pre-existing microvascular changes (SVD) and neurodegenerative changes can trigger a series of pathological events leading to post-stroke cognitive impairment and dementia (PSCID). The presence of a stroke, combined with pre-existing cerebrovascular pathology or Alzheimer’s disease/dementia, may predispose to PSCID, although the mechanisms and interactions of these factors remain unclear. In addition, there is still no clear consensus regarding the definition and timing of PSCID diagnosis.⁽¹⁴⁾

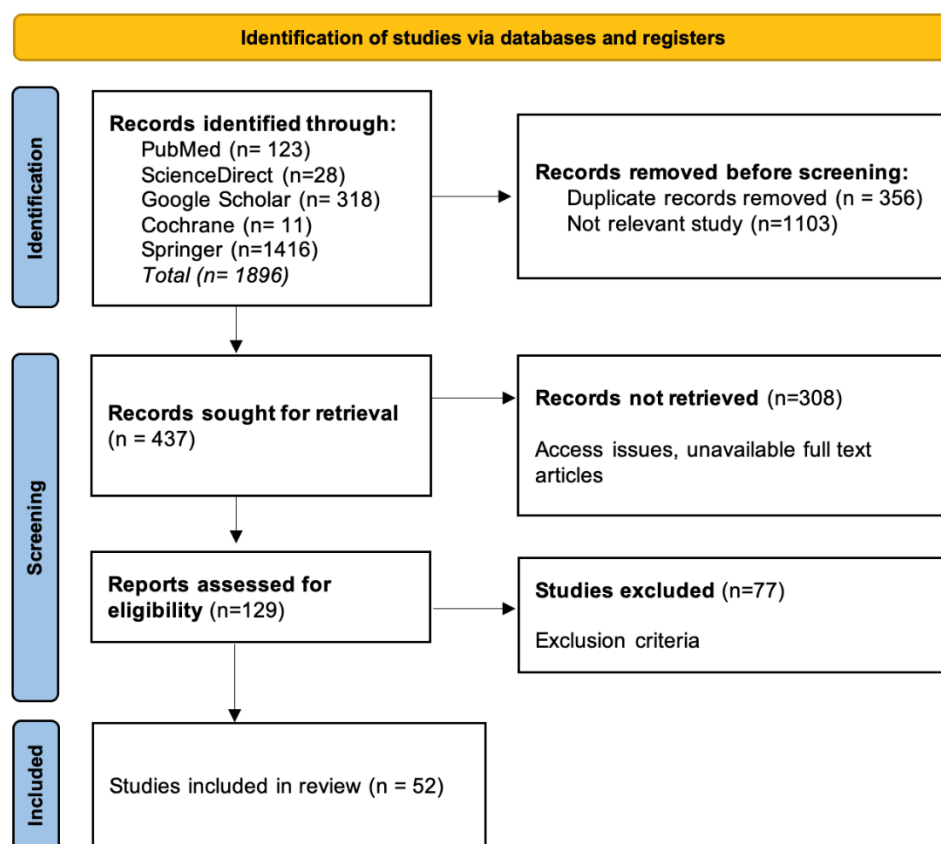


Figure 1. Identification of studies via databases and registers

Cognitive impairment in stroke

The pathophysiology of cognitive impairment after stroke is complex and involves multiple mechanisms that contribute to the disruption of brain structure and function. Ischemic stroke, which accounts for most strokes, occurs when blood flow to a specific region of the brain is blocked, leading to tissue damage. Cerebral infarction results in oxygen and glucose deprivation, triggering a series of pathological events.⁽¹⁾ The ischemic cascade involves excitotoxicity, oxidative stress, inflammation, and apoptosis, leading to neuronal cell death and tissue damage. In response to cerebral ischemia, inflammation is activated in the brain.⁽¹⁵⁾ Activated microglia and astrocytes release pro-inflammatory cytokines, such as interleukin-1 β (IL-1 β), tumor necrosis factor-alpha (TNF- α), and interleukin-6 (IL6).⁽¹⁶⁾ Neuroinflammation exacerbates neuronal damage, disrupts synaptic connections, and contributes to neurodegeneration, ultimately leading to cognitive impairment.⁽⁵⁾

Ischemic stroke can damage white matter, which plays a crucial role in cognition. Axonal

injury, demyelination, and disruption of white matter pathways disrupt neuronal connectivity and communication. White matter lesions, such as leukoaraiosis and lacunar infarcts, are frequently observed in post-stroke cognitive impairment and contribute to executive dysfunction, information processing deficits, and cognitive slowing.⁽¹⁾

Stroke can also trigger neurodegenerative processes, including the accumulation of abnormal protein aggregates, such as amyloid beta (A β) plaques and tau tangles, such as those observed in Alzheimer's disease (AD).⁽¹⁷⁾ Cerebral ischemia increases the production of amyloid-beta precursor protein (APP) processing and alters tau phosphorylation, although the extent to which concurrent AD pathology drives post-stroke cognitive decline is still debated.⁽¹⁸⁾ These pathological features in stroke survivors may contribute to cognitive impairment and increase the risk of developing mixed dementia, which is characterized by both vascular and neurodegenerative pathology.⁽¹⁹⁾

Historically, vascular dementia has been associated with large vessel infarctions; however, small vessel disease can also cause cognitive

impairment and dementia. Microinfarcts can be found in 20–40% of individuals who die after age 80. Meanwhile, macroscopic and/or microinfarcts can be found in >50% of elderly people who die after the age of 90 years.^(20,21)

The number, location, and size of macroscopic infarcts influence their potential association with cognitive impairment.⁽²²⁾ In general, multiple infarcts, larger infarcts, and infarcts in cortical regions are more likely to be associated with dementia.⁽¹⁹⁾ Although dementia can be caused by multiple infarcts, a single infarct in a specific brain region can also substantially impact cognitive function.⁽²²⁾ These strategic infarcts have been found in the thalamus, cortical regions of the parietal and temporal lobes, and the genu of the internal capsule. Small subcortical (lacunar) infarcts, especially if multiple infarcts occur or are present in individuals with other subcortical vascular diseases such as arteriosclerosis or white matter disease, increase the risk of dementia.^(22,23)

Macroscopic and microscopic hemorrhages, if present in sufficient quantities, may also lead to cognitive impairment. Due to the imaging characteristics of hemosiderin (which can be detected in the brain after a hemorrhage), microscopic hemorrhages can be detected more easily using neuroimaging than pathological analysis. In fact, the number and location (e.g., lobar) of microscopic hemorrhages detected using neuroimaging have been associated with cognitive impairment and an increased risk of stroke and mortality.^(21,24)

Stroke affects cognitive domains, including attention, memory, language, and orientation. The most affected domains at the time of stroke are attention and executive function.⁽²⁵⁾ Emerging evidence suggests that many stroke survivors may have pre-stroke impairments in processing speed and executive function. These impairments have been linked to structural brain changes resulting from the same cerebrovascular disease and associated risk factors that contribute to stroke.⁽²⁶⁾

Executive function (EF) refers to higher-level cognitive operations that direct lower-level processes toward goal-oriented actions. This class of cognitive functions encompasses a wide range of abilities, including planning, problem-solving, criterion-setting, multitasking, working memory, cognitive control, inhibition, shifting, and monitoring. These functions are highly interrelated but also separable and independent. The complexity of EF makes it highly sensitive to

brain changes resulting from stroke. Impaired EF is expected to have a ripple effect on other cognitive functions and reduce the ability to regain independence in daily activities.⁽²⁷⁾ Furthermore, it affects the ability to adapt to new or challenging situations, such as when alternative movement strategies are needed to compensate for limb weakness or when pre-planning sentences is necessary to rehabilitate verbal communication, executive dysfunction significantly impacts patients' quality of life and independence, resulting in daily difficulties across a range of neurological conditions. A study by Tarantino et al.⁽²⁸⁾ found that only the group of patients receiving cognitive training showed improved performance on attention matrices and phonemic fluency tests. Effect sizes calculated using a pretest–posttest control group design revealed moderate training effects on digit span, phonemic fluency, Wisconsin Card Sorting Test (WCST) errors, and Stroop interference.⁽²⁸⁾

One of the fundamental elements of post-stroke neuroplasticity is cortical reorganization, a process by which impaired brain function migrates to uninjured brain regions.⁽⁸⁾ In rats, within 1–3 days after stroke, stimulation of the limb contralateral to the stroke produced activity in the ipsilateral cortex, indicating reorganization of sensory input to the intact hemisphere. At 1–2 weeks post-stroke, activity shifts back to the injured hemisphere, with the remaining perilesional cortex taking over the functions of the damaged brain.^(8,29) Although this temporary shift of activity to the opposite hemisphere may be adaptive, the extent of the shift of function back to the injured hemisphere correlates with the rate of behavioral recovery.⁽⁸⁾

Neural plasticity and functional recovery after stroke are also supported by structural changes in the brain. In animal models, ischemia induces the growth of new dendrites and axons, primarily in the perilesional cortex but also in areas distant from the lesion. Growth factor signals that promote synaptogenesis can be detected as early as 3 days after stroke and peak at 7–14 days^(8,29). Stroke also triggers angiogenesis, which is well established within 10 days after stroke in rats and results in the development of collateral vessels to support the ischemic penumbra. These newly growing blood vessels may also serve as a scaffold to support the migration of neural stem cells from their reservoir in the subventricular zone (SVZ) to the infarct area.⁽⁸⁾

Diagnosis

To evaluate various cognitive domains and detect cognitive impairment in stroke patients, several cognitive assessment tools can be used, such as the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA). Both tests are screening tests capable of assessing various cognitive functions, including attention, memory, language, and executive function.⁽¹⁾

1. Montreal cognitive assessment (MoCA)

The MoCA is a one-page cognitive screening tool used to assess attention and concentration, executive function, memory, language, visuoconstructional skills, conceptual thinking, calculation, and orientation in approximately 10 minutes. The maximum score is 30 points. A score below 26 indicates cognitive impairment. One point is added for participants with less than 12 years of education.⁽³⁰⁾

The MoCA consists of 30 items that assess several cognitive domains, namely: a. Executive function: assessed with trail-making B (1 point), phonemic fluency task (1 point), and two items of verbal abstraction (1 point). b. Visuospatial: assessed with clock drawing task (3 points) and describing a 3-dimensional cube (1 point) c. Language: mentioning 3 animal names (lion, camel, rhinoceros; 3 points), repeating 2 sentences (2 points), language fluency (1 point) d. Delayed recall: mentioning 5 words (5 points), mentioning again after 5 minutes (5 points) e. Attention: assessing alertness (1 point), sequential subtraction (3 points), digit forward and backward (1 point each) f. Abstraction: assessing the similarity of an object (2 points) g. Orientation: assessing mentioning the date, month, year, day, place and city (1 point each).⁽³¹⁾

2. Montreal cognitive assessment Indonesian version (MoCA-Ina)

The MoCA-Ina itself has been translated into Indonesian and tested for validity.^(31,32) The MoCA-Ina was first developed by Husein et al.⁽³²⁾, and at that time, it underwent transcultural validation, finding it valid and reliable according to the World Health Organization (WHO) transcultural validation criteria. Content, construct, and criterion-related validation were conducted because the MoCA test itself met these criteria. The MoCA-Ina was then further validated for each item in stroke patients by Panentu and Irfan⁽³¹⁾ who found validity with Pearson $r = 0.529$

and $p = 0.046$, and reliability with Pearson $r = 0.963$ and $p = 0.000$. Therefore, the MoCA-Ina is valid and reliable for screening stroke patients.

3. Advantages of the MoCA over the MMSE

Both the MoCA and the MoCA-Ina have several advantages over the MMSE in detecting cognitive impairment, particularly in the executive domain. The MMSE is often considered to have lower sensitivity for mild cognitive impairment compared to the MoCA. The MoCA was designed to identify mild cognitive impairment more sensitively, with an emphasis on executive function, visuospatial function, and short-term memory. A study conducted by Silalahi et al.⁽³³⁾ showed that of the 49 patients studied, a statistically significantly higher proportion of patients was diagnosed with cognitive impairment using the MoCA assessment than using the MMSE (81.6% compared to 34.7%, $p < 0.001$).

Several reviews have identified MoCA as one of the clinically feasible instruments for detecting cognitive impairment in stroke patients.^(34,35) Its diagnostic accuracy is established by comparison with a gold standard, such as a neuropsychological test battery consisting of domain-specific cognitive tests. Because such extensive testing is rarely feasible in the acute phase, brief screening is recommended as the practical alternative, and the MoCA is able to detect deficits that can be unnoticed during routine clinical observation.⁽³⁰⁾

Cognitive intervention

A systematic review by Mendes et al.⁽³⁶⁾ described several cognitive interventions that can be performed on patients with and without vascular cognitive impairment, including Compensatory Cognitive Training, Cognitive Remediation, Cognitive Enrichment, Cognitive Activation, Brain Training, Cognitive Stimulation, Cognitive Training, and Cognitive Rehabilitation.

1. Compensatory cognitive training

Compensatory cognitive training is the use of low-tech intervention strategies (internal, external, or environmental) to compensate for cognitive impairments in order to reduce their impact on activities of daily living (ADL) and quality of life. These interventions aim to improve frontal mediation function or to compensate for specific cognitive functions negatively affected by aging. This intervention is suitable for patients with

severe cognitive impairment who also have functional impairments.^(36,37)

2. Cognitive remediation

Cognitive remediation is a therapy that uses a series of techniques designed to teach "thinking skills," which can be considered a form of cognitive rehabilitation. This therapy involves training in a series of tasks designed to improve cognitive abilities and social functioning. The targeted domains depend on the patient's needs but may include attention, working memory, planning, and executive function.⁽³⁶⁾ Cognitive remediation primarily focuses on global cognitive training (e.g., theater training) or specific cognitive competencies (e.g., memory) and utilizes a variety of intervention techniques/methods, such as paper-and-pencil exercises, group activities, workshops, videos, or computers. Cognitive remediation therapy is most commonly used in patients with schizophrenia, psychosis, and anorexia nervosa.^(36,37)

3. Cognitive activation

Unlike Cognitive Remediation, Cognitive Activation interventions are aimed at healthy individuals or individuals with MCI who are independent and functional. Therefore, the presence of a therapist is often replaced by a computer system. The primary goal of cognitive activation is to maintain the patient's cognitive function.^(36,37)

4. Cognitive training

Cognitive Training involves guided practice and repetitive training of cognitive functions using computerized or written methods. This cognitive intervention aims to correct cognitive deficits while identifying, tracking, and monitoring the user's cognitive performance. Cognitive training trains specific rationalization functions, such as memory, executive function, processing speed, language, and attention, through repetitive practice. This training can be performed on adult patients, both with and without cognitive impairment. The media used for cognitive training vary widely, from computer games, web-based exercises, television programs, to tablet-based training through interactive applications.^(36,38)

5. Brain training

Brain training is based on the use of repetitive programs ("brain games") that focus on the performance of cognitive tasks over a period of

time to improve cognitive deficits. This type of intervention should include everyday activities such as video games, music, computer training, or physical exercise.^(36,39,40)

Cognitive training for post-stroke cognitive impairment

Post-stroke cognitive impairment is an important factor influencing patient rehabilitation outcomes and the impact of physical dysfunction. Early cognitive rehabilitation training can not only improve cognitive dysfunction in post-stroke patients but also promote recovery of activities of daily living (ADL).^(41,42)

The rehabilitation period is generally divided into 3 periods: very early rehabilitation, early rehabilitation, and late rehabilitation. Very early rehabilitation is defined as rehabilitation interventions provided within 24 hours of stroke. Early rehabilitation refers to interventions provided within the first two weeks after stroke, while late rehabilitation is defined as rehabilitation provided after the first two weeks after stroke.⁽⁷⁾

A study conducted by Nurcaya et al.⁽⁴³⁾ found that statistically significant differences were observed in pretest and posttest MoCA-Ina scores between the intervention group ($p = 0.000$) and the control group ($p = 0.003$), both of which at $p < 0.005$. When comparing the differences in MoCA-Ina scores before and after the intervention, statistical tests showed a statistically significant difference between the two groups ($p < 0.05$). Other research also showed that stroke patients with MoCA scores < 26 before early cognitive training who underwent two weeks of cognitive training three times a week, found that their cognitive function improved significantly. However, only seven of the 22 respondents returned to normal cognitive function, with a MoCA score > 26 . On the other hand, some studies suggest that, in comparison to the cutoff of 26, a MoCA cutoff score of 23 may indicate better overall diagnostic accuracy in cognitive function.^(43,44)

1. Stroop effect task as an instrument of cognitive training

The cognitive interventions described above represent different categories of intervention, each defined by its aim, structure, and mode of delivery. The Stroop effect task, in contrast, is a task-level instrument that can be embedded within a cognitive training programme. Weng et al.⁽⁴⁵⁾ reported that cognitive training incorporating

interference-control tasks improved working memory in older adults with mild cognitive impairment, and a recent meta-analysis of randomised controlled trials found that cognitive training produced significant gains in inhibitory control as measured by the Stroop task in older adults.⁽⁴⁵⁾ Contemporary computerised training platforms for post-stroke cognitive impairment routinely include Stroop-type interference tasks within their exercise libraries.^(46,47)

The Stroop effect task was originally a neuropsychological test widely used for both experimental and clinical purposes.⁽⁴⁸⁾ However, when used as a cognitive training exercise, the Stroop effect task has been shown to improve cognitive abilities in the domains of executive function and working memory.⁽⁴⁹⁾

This test assesses the ability to inhibit cognitive interference, which occurs when processing one feature of a stimulus influences the simultaneous processing of another attribute of the same stimulus. The test was first introduced by Stroop in 1935. In the original version, subjects were asked to read three different tables as quickly as possible. Two of these represented the "congruent condition," in which participants were asked to read color names (hereafter referred to as color words) printed in black ink (word/W) and name various colors (color/C). In contrast, in the third table, called the color-word (CW) condition, the color words were printed in an inconsistent color (e.g., the word "red" was printed in green ink). Thus, in this incongruent condition, participants were asked to name the color of the ink instead of reading the word. In other words, participants were asked to perform a less automatic task (namely, naming the color of the ink) while inhibiting interference arising from the more automatic task.⁽⁴⁸⁾

Another variation of the Stroop task is the numerical Stroop task, in which the subjects are

asked to compare the physical size and numerical value of two numbers, ignoring irrelevant dimensions. Each trial begins with a focal point (a plus sign) displayed in the center of the screen for 500 ms, after which a pair of numbers appears and remains visible until the participant responds or 2,000 ms have elapsed. After a response, the screen goes blank for 300 ms, and then the next trial begins. A computer measures reaction time in milliseconds from the onset of the stimulus, as shown in Figure 2.⁽⁵⁰⁾

2. Go/No-Go task as an instrument of cognitive training

The Go/No-Go task was initially developed as an assessment tool for inhibitory control. The latter is an executive function that involves controlling one's attention, behavior, thoughts, and/or emotions to overcome strong internal tendencies or external lures. In these tasks, participants are typically required to perform a rapid motor response (e.g., pressing a key on a keyboard as quickly as possible) when a specific stimulus (i.e., a target) is displayed on a computer screen and to withhold this response for another stimulus (i.e., a non-target; also called a distractor or lure).

Because this test is a simple task, the go/no-go paradigm typically involves a large number of trials with only a small number of no-go trials. This design ensures that "Go" responses become dominant, thereby increasing the likelihood of commission errors (i.e., incorrectly pressing the button during No-Go trials, also referred to as false alarms). Reaction time, the number of correct Go trials (hits), and omission errors (i.e., failure to press the button during Go trials, also called misses) can also be measured, however, these are generally not considered primary indices of inhibitory control or its impairment.⁽⁵¹⁾

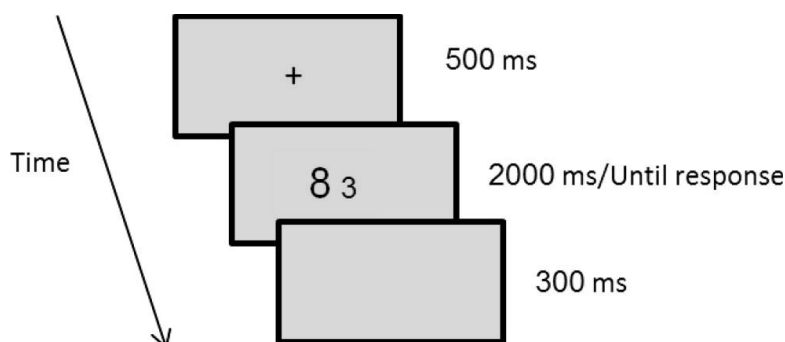


Figure 2. Example of numerical Stroop task⁽⁵⁰⁾

Go/No-Go tasks can also be used to train cognitive function. A meta-analysis conducted by Li et al. in 2022 revealed that response inhibition training (of which the go/no-go task is one) can improve cognitive function. This study also compared response inhibition training with other methods, such as the stop signal task, and found that the go/no-go task was superior in improving cognitive function compared to the stop signal task.⁽⁵²⁾

CONCLUSIONS

In this review, we discussed post-stroke cognitive impairment (PSCI) that represents a massive global health burden and severely impacts the patient's independence and quality of life. There are several intervention and cognitive training methods that can be used to reduce cognitive impairment in post-stroke patients. The primary purpose of this review was to evaluate the effectiveness of website-based applications as cognitive stimulation training, particularly during the first three months post-stroke which is recognized as the "golden period" for recovery. Our review concludes that computerized cognitive training utilizing tasks such as the Stroop effect and Go/No-Go paradigms significantly improves cognitive performance, as evidenced by validated tools such as the MoCA-Ina. Further research in this field should focus on transitioning prototype applications into standardized clinical practice, particularly in regions such as Indonesia where these tools remain rare.

Conflict of Interest

All authors declare that there is no conflict of interest. The research was conducted using the authors' resources.

Authors' Contributions

DRTS: Conception and design, acquisition of data, analysis and interpretation of data; Drafting the article; Final approval of the version to be published. IPEW: Conception and design; Critical revision of the article; Final approval of the version to be published. KW: Conception and design; Critical revision of the article; Final approval of the version to be published. All authors have read and approved the final manuscript.

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The data used to support the findings of this study are included in the article.

Declaration of AI Usage in Scientific Writing

The authors confirm that they did not use any AI tools during the preparation of this manuscript. The entire written content is based on the authors' original literature review and synthesis.

REFERENCES

1. Elendu C, Amaechi DC, Elendu TC, et al. Stroke and cognitive impairment: understanding the connection and managing symptoms. *Ann Med Surg (Lond)* 2023;85:6057–66. doi:10.1097/MS9.0000000000001441.
2. Mtambo ML, Ragunathan T, Mohan D, Warren N, Su TT, Quek KF. Post-stroke cognitive impairment and dementia research in Southeast Asia: a scoping review. *Asian J Psychiatr* 2025;107:104460. doi:10.1016/j.ajp.2025.104460.
3. Feigin VL, Brainin M, Norrving B, et al. World Stroke Organization: global stroke fact sheet 2025. *Int J Stroke* 2025;20:132–44. doi:10.1177/17474930241308142.
4. Lo JW, Crawford JD, Lipnicki DM, et al. Trajectory of cognitive decline before and after stroke in 14 population cohorts. *JAMA Netw Open* 2024;7:e2437133. doi:10.1001/jamanetworkopen.2024.37133.
5. Rost NS, Brodtmann A, Pase MP, et al. Post-stroke cognitive impairment and dementia. *Circ Res* 2022;130:1252–71. doi:10.1161/CIRCRESAHA.122.319951.
6. Dichgans M, Leys D. Vascular cognitive impairment. *Circ Res* 2017;120:573–91. doi:10.1161/CIRCRESAHA.116.308426.
7. El Hussein N, Katzan IL, Rost NS, et al. Cognitive impairment after ischemic and hemorrhagic stroke: a scientific Statement from the American Heart Association/American Stroke Association. *Stroke* 2023;54:e272–91. doi:10.1161/STR.0000000000000430.
8. Coleman ER, Moudgal R, Lang K, et al. Early rehabilitation after stroke: a narrative review. *Curr Atheroscler Rep* 2017;19:59. doi:10.1007/s11883-017-0686-6.
9. Fonte C, Damora A, Abbruzzese L, et al. Self-administered cognitive rehabilitation using an electronic device in subacute stroke patients: a proof-of-concept study on safety, feasibility, and

- preliminary efficacy. *Neuro Sci* 2025;6:109. doi:10.3390/neurosci6040109.
10. Jeon H, Kim DY, Park SW, et al. A systematic review of cognitive telerehabilitation in patients with cognitive dysfunction. *Front Neurol* 2025;15:1450977. doi:10.3389/fneur.2024.1450977.
11. Khairunisa Y, Nurwicaksono BD, Kuswoyo D. Game memoria sebagai inovasi digital terapi kognitif pencegahan demensia bagi lansia: studi pengujian prototipe skala industri. *Amalee Indones J Community Res Engage (Indonesian)* 2025;6:489–500. doi:https://doi.org/10.37680/amalee.v6i2.
12. Niyomyart A, Ruksakulpiwat S, Benjasirisan C, et al. Current status of barriers to mHealth access among patients with stroke and steps toward the digital health era: systematic review. *JMIR Mhealth Uhealth* 2024;12:e54511. doi:10.2196/54511.
13. Ma Y, Yang Y, Wang X, et al. Prevalence and risk factors of poststroke cognitive impairment: a systematic review and meta-analysis. *Public Health Nurs* 2025;42:1047–59. doi:10.1111/phn.13503.
14. Rost NS, Meschia JF, Gottesman R, et al. Cognitive impairment and dementia after stroke: design and rationale for the Discovery study. *Stroke* 2021;52:e499–516. doi:10.1161/STROKEAHA.120.031611.
15. Tuo QZ, Zhang ST, Lei P. Mechanisms of neuronal cell death in ischemic stroke and their therapeutic implications. *Med Res Rev* 2022;42:259–305. doi:10.1002/med.21817.
16. Iadecola C, Buckwalter MS, Anrather J. Immune responses to stroke: mechanisms, modulation, and therapeutic potential. *J Clin Invest* 2020;130:2777–88. doi:10.1172/JCI135530.
17. Pluta R, Ułamek-Kozioł M, Januszewski S, Czuczwar SJ. Participation of amyloid and tau protein in neuronal death and neurodegeneration after brain ischemia. *Int J Mol Sci* 2020;21:4599. doi:10.3390/ijms21134599.
18. Mok VCT, Lam BYK, Wong A, Ko H, Markus HS, Wong LKS. Early-onset and delayed-onset poststroke dementia – revisiting the mechanisms. *Nat Rev Neurol* 2017;13:148–59. doi:10.1038/nrneurol.2017.16.
19. Agrawal S, Schneider JA. Vascular pathology and pathogenesis of cognitive impairment and dementia in older adults. *Cereb Circ Cogn Behav* 2022;3:100148. doi:10.1016/j.cccb.2022.100148.
20. Jellinger KA. Pathology and pathogenesis of vascular cognitive impairment—a critical update. *Front Aging Neurosci* 2013;5:17. doi:10.3389/fnagi.2013.00017.
21. Van Der Flier WM, Skoog I, Schneider JA, et al. Vascular cognitive impairment. *Nat Rev Dis Primers* 2018;4:18003. doi:10.1038/nrdp.2018.3.
22. Mok VCT, Cai Y, Markus HS. Vascular cognitive impairment and dementia: mechanisms, treatment, and future directions. *Int J Stroke* 2024;19:838–56. doi:10.1177/17474930241279888.
23. Weaver NA, Kuijff HJ, Aben HP, et al. Strategic infarct locations for post-stroke cognitive impairment: a pooled analysis of individual patient data from 12 acute ischaemic stroke cohorts. *Lancet Neurol* 2021;20:448–59. doi:10.1016/S1474-4422(21)00060-0.
24. Arvanitakis Z, Capuano AW, Leurgans SE, Bennett DA, Schneider JA. Relation of cerebral vessel disease to Alzheimer's disease dementia and cognitive function in elderly people: a cross-sectional study. *Lancet Neurol* 2016;15:934–43. doi:10.1016/S1474-4422(16)30029-1.
25. Huang YY, Chen SD, Leng XY, et al. Post-stroke cognitive impairment: epidemiology, risk factors, and management. *J Alzheimer's Dis* 2022;86:983–99. doi:10.3233/JAD-215644.
26. Skidmore ER, Eskes G, Brodtmann A. Executive function poststroke: concepts, recovery, and interventions. *Stroke* 2023;54:20–9. doi:10.1161/STROKEAHA.122.037946.
27. Boyd LA, Hayward KS, Ward NS, et al. Biomarkers of stroke recovery: consensus-based core recommendations from the Stroke Recovery and Rehabilitation Roundtable. *Int J Stroke* 2017;12:480–93. doi:10.1177/1747493017714176.
28. Tarantino V, Burgio F, Toffano R, et al. Efficacy of a training on executive functions in potentiating rehabilitation effects in stroke patients. *Brain Sci* 2021;11:1002. doi:10.3390/brainsci11081002.
29. Joy MT, Carmichael ST. Encouraging an excitable brain state: mechanisms of brain repair in stroke. *Nat Rev Neurosci* 2021;22:38–53. doi:10.1038/s41583-020-00396-7.
30. Stiekema APM, Vreven LWA, Hummel RSO, et al. The Montreal Cognitive Assessment detects cognitive deficits that go unnoticed during clinical observation in the acute phase after stroke. *Brain Inj* 2024;38:687–91. doi:10.1080/02699052.2024.2341039.
31. Panentu D, Irfan M. Uji validitas dan reliabilitas butir pemeriksaan dengan Montreal Cognitive Assessment versi Indonesia (MoCA-Ina) pada insan pasca stroke fase recovery. *J Fisioter (Indonesian)* 2013;13. doi:10.47007/fisio.v13i1.646.
32. Husein N, Lumempouw S, Ramli Y, Herqutanto. Uji validitas dan reliabilitas Montreal Cognitive Assessment versi Indonesia (MoCA-Ina) untuk skrining gangguan fungsi kognitif. *Neurona (Indonesian)* 2010;27:13 hal.
33. Silalahi VR, Milenia V, Aritonang M. Post stroke cognitive impairment in rural hospital: a comparative analysis of MoCA-Ina and MMSE.

- Neurona (Indonesian) 2025;42: 4 hal. <https://doi.org/10.52386/neurona.v42i1.806>.
34. Shi D, Chen X, Li Z. Diagnostic test accuracy of the Montreal Cognitive Assessment in the detection of post-stroke cognitive impairment under different stages and cutoffs: a systematic review and meta-analysis. *Neurol Sci* 2018;39:705–16. doi:10.1007/s10072-018-3254-0.
 35. Quinn TJ, Richard E, Teuschl Y, et al. European Stroke Organization and European Academy of Neurology joint guidelines on post-stroke cognitive impairment. *Eur J Neurol* 2021;28:3883–920. doi:10.1111/ene.15068.
 36. Mendes L, Oliveira J, Barbosa F, Castelo-Branco M. A conceptual view of cognitive intervention in older adults with and without cognitive decline—a systemic review. *Front Aging* 2022;3:844725. doi:10.3389/fragi.2022.844725.
 37. Lenze EJ, Bowie CR. Cognitive training for older adults: what works? *J Am Geriatr Soc* 2018;66:645–7. doi:10.1111/jgs.15230.
 38. Chaikham A, Putthinoi S, Lersilp S, Bunpun A, Chakpitak N. Cognitive training program for Thai older people with mild cognitive impairment. *Procedia Environ Sci* 2016;36:42–5. doi:10.1016/j.proenv.2016.09.007.
 39. Simons DJ, Boot WR, Charness N, et al. Do "brain-training" programs work? *Psychol Sci Public Interest* 2016;17:103–86. doi:10.1177/1529100616661983.
 40. van Heugten CM, Ponds RWHM, Kessels RPC. Brain training: hype or hope? *Neuropsychol Rehabil* 2016;26:639–44. doi:10.1080/09602011.2016.1186101.
 41. Xuefang L, Guihua W, Fengru M. The effect of early cognitive training and rehabilitation for patients with cognitive dysfunction in stroke. *Int J Methods Psychiatr Res* 2021;30:e1882. doi:10.1002/mpr.1882.
 42. Kim S, Park SW, Jeong T, Kang MS, Kim DY. AI-driven cognitive telerehabilitation for stroke: a randomized controlled trial. *Front Neurol* 2025;16:1636017. doi:10.3389/fneur.2025.1636017.
 43. Nurcaya, Arafat R, Sjattar EL. Effectiveness of early cognitive exercise intervention on improvement cognitive function of stroke patients in the acute phase. *J Vasc Nurs* 2024;42:60–4. doi:10.1016/j.jvn.2023.11.010.
 44. Salvadori E, Cova I, Mele F, Pomati S, Pantoni L. Prediction of post-stroke cognitive impairment by Montreal Cognitive Assessment (MoCA) performances in acute stroke: comparison of three normative datasets. *Aging Clin Exp Res* 2022;34:1855–63. doi:10.1007/s40520-022-02133-9.
 45. Olegário RL, Fernandes SR, de Moraes R. Efficacy of cognitive training on executive functions in healthy older adults: a systematic review with meta-analysis of randomized controlled trials. *Psychol Health* 2025;40:714–41. doi:10.1080/08870446.2023.2267610.
 46. Soni AK, Kumar M, Kothari S. Efficacy of home based computerized adaptive cognitive training in patients with post stroke cognitive impairment: a randomized controlled trial. *Sci Rep* 2025;15:1072. doi:10.1038/s41598-025-85511-3.
 47. Gao M, Huang L, Yi J, et al. The effectiveness of computerized cognitive training in patients with poststroke cognitive impairment: systematic review and meta-analysis. *J Med Internet Res* 2025;27:e73140. doi:10.2196/73140.
 48. Scarpina F, Tagini S. The Stroop color and word test. *Front Psychol* 2017;8:557. doi:10.3389/fpsyg.2017.00557.
 49. Weng W, Liang J, Xue J, et al. The transfer effects of cognitive training on working memory among Chinese older adults with mild cognitive impairment: a randomized controlled trial. *Front Aging Neurosci* 2019;11:212. doi:10.3389/fnagi.2019.00212.
 50. Dadon G, Henik A. Adjustment of control in the numerical Stroop task. *Mem Cognit* 2017;45:891–902. doi:10.3758/s13421-017-0703-6.
 51. Meule A. Reporting and interpreting task performance in go/no-go affective shifting tasks. *Front Psychol* 2017;8:701. doi:10.3389/fpsyg.2017.00701.
 52. Li W, Shang Y, Zhuang W, Mai W, Cheng W, Chen Z. Effectiveness of response inhibition training and its long-term effects in healthy adults: a systematic review and meta-analysis. *Front Neurosci* 2022;16:813975. doi:10.3389/fnins.2022.813975.

