

# We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index  
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?  
Contact [book.department@intechopen.com](mailto:book.department@intechopen.com)

Numbers displayed above are based on latest data collected.  
For more information visit [www.intechopen.com](http://www.intechopen.com)



# Movement Rehabilitation in Physiotherapy after Stroke: The Role of Constraint-Induced Movement Therapy

*Auwal Abdullahi*

## Abstract

Stroke is increasingly becoming a global health problem. This is because it may lead to death, Long-term disability such as in motor function, and significant burden to the patients and their families. The disability can be prevented or rehabilitated using a physiotherapy technique known as constraint-induced movement therapy (CIMT). The CIMT comprises of task practice with the affected limb, constraint of the unaffected limb, and transfer package to foster compliance and increase the amount of task repetition. It helps to reestablish normal motor control through facilitating changes in physiological functions of the brain, improvement in real-world arm use, and movement precision and quality. However, its protocols vary. Some protocols use number of hours and others use number of repetitions to determine the intensity or the amount of task practice. This chapter argued that CIMT is effective, but the protocols that use a number of hours of task practice are not clear and are resource intensive; and as such they could interfere with the process of clinical decision making. Consequently, it proposed the use of a number of repetitions of task practice to determine the intensity or the amount of task practice and extending the application of CIMT to those with severe impairments after stroke.

**Keywords:** stroke, motor recovery, neuroscience, constraint-induced movement therapy, rehabilitation

## 1. Introduction

This chapter reviews the roles played by constraint-induced movement therapy (CIMT) in the rehabilitation of movement impairment following stroke. The chapter discusses these issue under three major sections and a conclusion section. The sections are overview on stroke, control of human movement, and the role of CIMT. The section on overview on stroke comprises of definition of stroke, etiology of stroke, pathophysiology of stroke, epidemiology of stroke, and consequences of stroke. The section on control of human movement consists only of nervous system control of movement in health and disease. The section on the role of CIMT consists of constraint-induced movement therapy: historical background and neuroscientific basis, components of CIMT, protocols of constraint-induced movement therapy, effects of constraint-induced movement therapy, and constraint-induced

movement therapy: the future perspective. Information from all the sections to be discussed is needed by the physiotherapist in order to be able to make an effective clinical decision during application of CIMT in people with stroke.

## **2. Overview on stroke**

### **2.1 Definition**

World over, stroke has become a major public health issue. It is defined as “a rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 h or leading to death, with no apparent cause other than that of vascular origin” [1]. This definition seems outdated since presently there are advances in prevention, diagnosis, and management of stroke, which led to so many people surviving stroke and living with long-term disabilities. Additionally, stroke is a compendium of neurovascular syndromes with diverse presentations, and the 24 h time limit is not sacrosanct as minutes or few hours of stroke event can lead to severe and/or permanent damage [2]. Consequently, attempts were made to reassess the definition of transient ischemic attack and stroke [2, 3]. A high-powered committee of experts deliberated and defined the conditions; thus, stroke is classically characterized as a neurological deficit attributed to an acute focal injury of the central nervous system (CNS) by a vascular cause, including cerebral infarction, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH), and is a major cause of disability and death worldwide [2]. This definition included the vascular causes of the condition. Thus, from the foregoing, it can be seen that stroke results from various vascular events and can manifest as varied forms of neurological signs and symptoms.

### **2.2 Etiology**

As stated in the definitions by Hatano and Sacco and his colleagues, the causes of stroke are of vascular origins [1, 2]. These sources are ischemia and intracerebral and subarachnoid hemorrhage, which may cause cerebral infarction. Ischemia simply means reduction in blood supply to brain cells, which could be due to occlusion as a result of the presence of either thrombus (a blood clot) or embolus (a moving blood clot) or stenosis (narrowing or reduction in the caliber of the arteries) [4]. The embolus usually may originate from the arteries or the heart as a result of conditions such as atrial fibrillation, sinoatrial disorder, recent acute myocardial infarction (AMI), marantic or subacute bacterial endocarditis, cardiac tumors, and valvular disorders, both native and artificial [5]. The intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH), which mean rupture or leakage of the tiny or very small blood vessels in the brain, are mainly caused by the elevated or increased intracranial pressure and sustained weakening of walls of blood vessels usually as a result of a long-standing high blood pressure [6]. Although majority of stroke cases are due to ischemia, in very rare cases, hemorrhagic and ischemic stroke can occur concurrently [7]. However, when stroke occurred as either ischemic or hemorrhagic, the latter usually leads to poor prognosis including mortality than ischemic stroke [8].

### **2.3 Pathophysiology**

The human brain under normal circumstances receives 20% of the cardiac output [9]. This output is responsible for cerebral blood flow (CBF) whose normal value in adults is about 50–55 ml/100 g/min. This makes the brain tissues to be rich and abundant in oxygen and glucose necessary for major metabolic processes at all times.

Thus, damage to the brain tissues due to reduced blood flow depends on the extent of the reduction to the tissues and probably for how long the reduction persists. If the CBF reduced to about  $14 \pm 2$  ml/100 g/min, the damage is usually reversible, and the affected area is known as ischemic penumbra. The penumbra is a region of functionally impaired, but structurally intact, tissues. Thus, this area of penumbra is the one that offers hope for recovery in case of therapy and rehabilitation. On the other hand, if the CBF reduced to about 6 ml/100 g/min, the damage is usually irreversible, and the affected area is known as the core. The core is the region of infarction. This is because the human brain is not endowed with respiratory reserve; it has low capacity for oxygen reserve, and this makes it to heavily depend on aerobic respiration. Any event that will alter its circulatory process and interfere with the aerobic respiration can cause damage to the brain cells. However, the abundant collateral circulation in the brain ensures that the effect of the damage is limited to some parts of the brain.

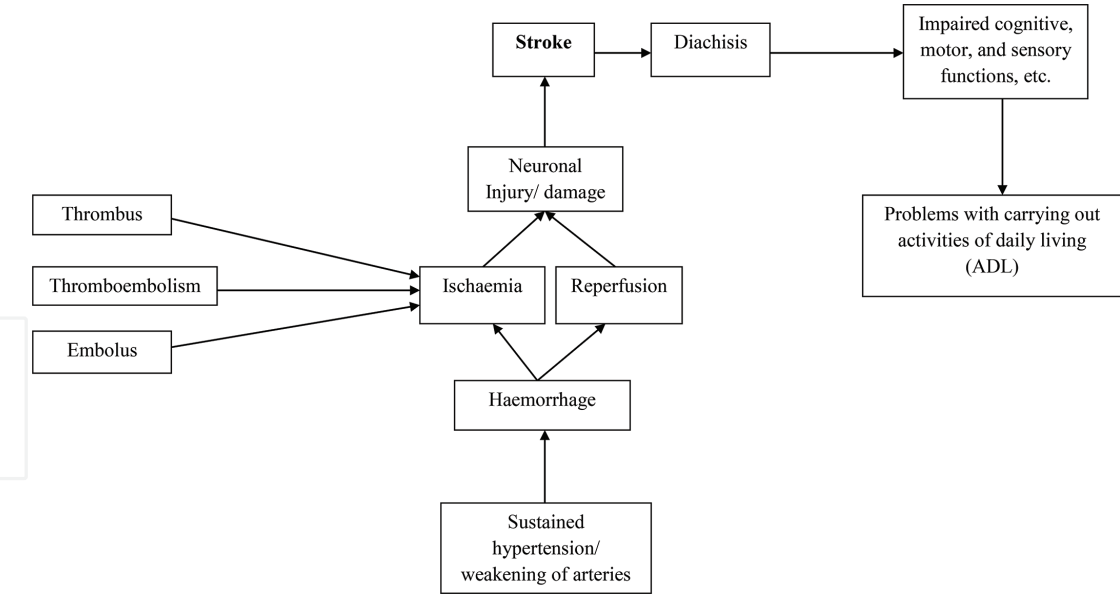
The events that bring about the alteration or impairment in blood circulation in the brain come in two ways: ischemia and hemorrhage. Ischemia is usually in the form of thrombosis in large and small vessels, embolism with or without cardiac and/or arterial factor, systematic hypoperfusion, and venous thrombosis. The mechanism of brain cell damage due to ischemia is through depletion of oxygen and glucose with attendant reduced capacity to produce energy-releasing compounds such as the adenosine triphosphate (ATP) [10]. The molecule ATP is important for cellular metabolic processes. This creates a vicious cycle of lack of essential molecules such as oxygen and glucose for the survival of the cells and eventually causes cellular death. The extent of the damage is, however, a factor of the location, severity, and the duration of ischemia. That is to say, the longer the duration of the ischemia, the more extensive the damage will be.

The mechanisms of cellular death due to ischemia often come in many ways. One of these ways comes through the damage or dysfunction of the mitochondria. The mitochondria are the energy storage or power house of the cells [11]. Dysfunction or damage of the mitochondria will result in depletion of energy required to initiate and sustain physiologic processes such as the normal function of ion gradient, which results in ionic imbalance. The ionic imbalance pertains to the loss of potassium in exchange of sodium, chloride, and calcium ions [12]. This ionic imbalance leads to intracellular accumulation of fluid, which manifests as swelling of neuronal cells and glia that will eventually lead to the death of these cells. Another way in which neuronal cell death can occur due to ischemia is through excessive release of excitatory neurotransmitters such as the glutamate and aspartate. These neurotransmitters release will further complicate the damage to the brain cells. Other ways through which cellular death occur include production of oxygen-free radicals and reactive oxygen species (ROS), and apoptosis. Free radicals and the ROS react with a number of cellular and extracellular elements including the vascular endothelium and cause cell death. Apoptosis occurs in response to release of proapoptotic molecules such as cytochrome c and apoptosis-inducing factor from mitochondria due to gene expression of Bcl-2 and p53.

The whole process resembles blocking the water supply to a flower. If the blockage continues, the flower begins to shrink gradually and eventually dies off. Thus, whatever the pathophysiologic process that takes place, the result is that ischemia or reperfusion causes cell death, and this in turn causes impairment in brain functions [13]. The pathophysiologic process of stroke is simplified in **Figure 1**.

## 2.4 Epidemiology of stroke

Many people around the world suffer stroke. According to the World Heart Federation, annually about 15 million people suffer stroke, out of which about 40% die and 33.33% live with long-term disability [14]. This shows that about 60%



**Figure 1.**  
*Schematic representation of pathophysiology of stroke and its consequences.*

survives stroke probably due to improved stroke management over the last few decades. Equally, the percentage of those who live with long-term disability also calls for public health concern. Thus, improving rehabilitation services for stroke survivors is much needed. However, the incidence of stroke seems to vary between different regions of the world. In many developed countries, the incidence of stroke is declining even though the actual number of strokes is increasing because of the aging population [14, 15]. In contrast, in the developing countries, the incidence of stroke is increasing. For example, about 1.3 million people suffer stroke every year with about 75% surviving it, and the incidence is predicted to increase with high incidence of death in Latin America, the Middle East, and sub-Saharan Africa in a few decades to come probably due to lack of standard care for stroke [14].

Many factors can put one at the risk of developing stroke. The risk factors for stroke can be divided into modifiable and nonmodifiable ones. The modifiable risk factors include high blood pressure, heart disease, hypercholesterolemia, physical inactivity, smoking, alcohol consumption, diabetes mellitus, psychosocial stress and depression, and kidney diseases [16]. The nonmodifiable risk factors include gender, race, and genetic factors. For gender as a nonmodifiable risk factor, there are varied reports on whether stroke occurs more in women or men and vice versa, though they are mostly in favor of high incidence in women. According to a study by Petrea and colleagues, stroke occurs more in women than men, though there is no significant difference between men and women in terms of stroke severity, subtype, and case fatality [17]. These variations may probably represent the social structure of a given population. Consequently, the incidence between men and women may be because of one gender reporting to the clinic more than the other in a given population. Secondly, the distribution of gender in the population can also play a role. In addition to all that was mentioned, women tend to have poorer functional outcomes following stroke [18]. Furthermore, whatever the cause of a stroke, its incidence imposes a huge financial burden on patients and their families and can cost governments millions of dollars as direct and indirect costs depending on the country and the particular healthcare system.

## 2.5 Consequences of stroke

Stroke results in impairment of brain functions, cognitive, motor, and sensory/ perceptual functions [13]. Examples of these impairments include decreased

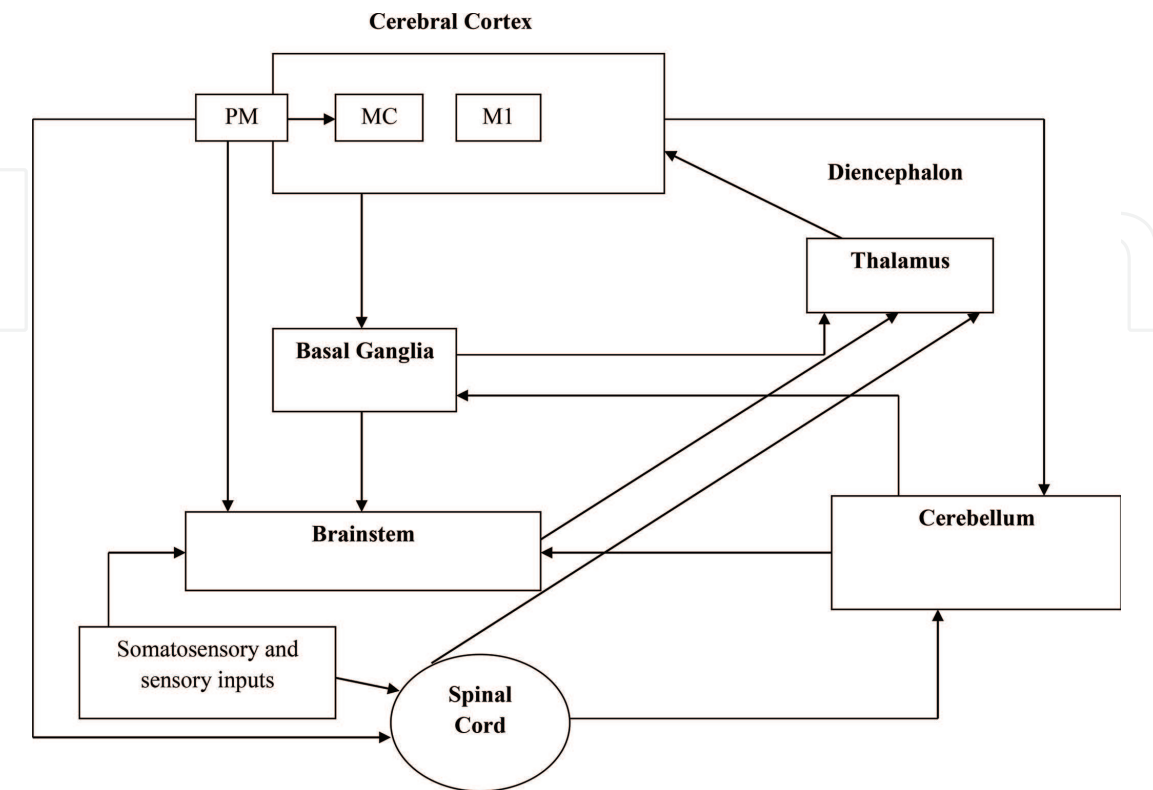


movement quality, impaired movement coordination, gait and balance problems, vascular dementia, memory impairment, emotional disturbances, and hemispatial neglect. The motor impairment is significant since movement is important for humans' daily functioning. The consequences of impairment of brain functions following stroke include difficulty in carrying out activities of daily living such as washing, dressing, bathing and difficulty in carrying out previously enjoyed leisure activities [19]. Normal functions of the brain are required in our daily life activities and participation. Therefore, rehabilitation is important to help patient recover or live an independent life as much as possible. In physiotherapy practice, CIMT is used for the rehabilitation of movement impairment after stroke.

### 3. Nervous system control of movement in health and disease

One of the major functions of the nervous system is the control of human movement, which is essential for our daily functioning and participation. This control occurs in top-down, bottom-up, and parallel manner using different parts of the central nervous system. The CNS structures responsible for the control of human movement include the cerebral cortex, basal ganglia, brainstem, cerebellum, and thalamus that are contained in the diencephalon and spinal cord, which are complemented by the peripheral feedback [20, 21]. The functions of these various structures are summarized in **Figure 2**.

The cerebral cortex functions for cognition, perception, and behavior. The cerebral cortex integrates these aforementioned functions to help execute human movements. It consists mainly of three important structures that are closely related with each other, the motor cortex, the premotor area, and the primary motor area. The main function of the motor cortex is sending inputs/commands through the corticospinal tract and corticobulbar system to the brainstem and the spinal cord [20]. The premotor area functions alongside other parts of the brain in movement



**Figure 2.**  
*Schematic representation of nervous control of human movement. PM, premotor cortex; MC, motor cortex; M1, primary motor cortex.*

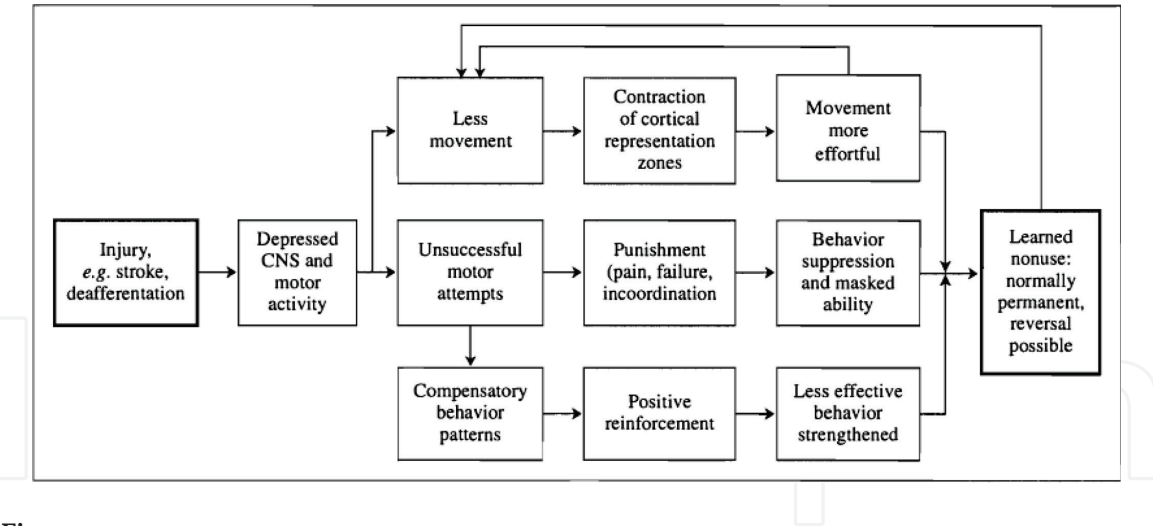
programming, selection of what to do, and identification of objects in space [20, 22]. It sends most of its outputs directly to the motor cortex, and some to the brainstem and the spinal cord [23]. The basal ganglia are a collection of nuclei at the base of the brain that are involved in higher-order cognitive functions like planning the strategies of movement [24]. These nuclei receive inputs from most parts of the cerebral cortex and send back their outputs to the motor cortex by the way of the thalamus.

The brainstem is important for the control of posture and locomotion. Postural control is essential for proper execution of movement. This is possible due to the presence of important nuclei in the brainstem, which include vestibular nuclei, red nuclei, and reticular nuclei [20, 21]. The brainstem receives somatosensory inputs from the skin and muscles of the head and sensory inputs from the vestibular and visual systems. Similarly, nuclei in the brainstem control outputs to the neck, face, and eyes. The cerebellum receives inputs from the spinal cord and the cerebral cortex detailing about movement and planning of movement, respectively. In addition, it sends output to the brainstem. The main functions of the cerebellum are comparing the intended movement with sensory signals and update the movement commands when the movement goes abnormally. Secondly, it helps in modulating range and force of movements. The thalamus is contained in the diencephalon, and most of the outputs from the basal ganglia, the spinal cord, the brainstem, and the cerebellum are processed via it. The spinal cord is at the lower level of the movement control hierarchy. It receives somatosensory information from the muscles, joints, and skin and sends information to the higher centers. However, following stroke, the functions of these various structures of the brain become impaired in such a way that movement is no longer well planned, coordinated, modulated, and integrated. The aim of rehabilitation is to reestablish these various integrated functions for normal movement control. Consequently, constraint-induced movement therapy (CIMT) is used to reestablish the normal nervous control of human movement.

## **4. Role of constraint in movement rehabilitation after stroke**

### **4.1 Constraint-induced movement therapy: historical background and neuroscientific basis**

Movement is important in every human's endeavors; it is essential for the performance of most of our daily activities. However, after a stroke, our ability to move may be impaired. This is due to the impairment in normal movement control as a result of damage to some areas of the brain. To help reestablish normal movement control following stroke, constraint-induced movement therapy is used [25, 26]. Constraint-induced movement therapy (CIMT) is a movement rehabilitation technique, which aims to counteract learned nonuse acquired after stroke [26, 27]. The learned nonuse phenomenon is an acquired behavior exhibited by patients following stroke that makes them not to use the affected limb in functional activities. It is said to be due to failure to carry out tasks after unsuccessful attempts as a result of depression of functions of the central nervous system, pain or fatigue following the injury. This acquired behavior may set off a vicious cycle of impairments comprising of decreased movement, decreased cortical representation of the affected part of the body, and compensatory behavior that may also decrease use of the affected part [28]. The mechanisms of learned nonuse phenomenon are depicted in **Figure 3**. Please refer to the articles by Taub and his colleagues for more details on the learned nonuse [27, 28]. Although, the description of the learned nonuse phenomenon by Taub has been challenged, modified, and expanded to include psychosocial and structural factors that can lead to the acquired behavior [29], his explanation has provided researchers



**Figure 3.**  
*The learned nonuse phenomenon (reproduced with permission from Edward Taub).*

and clinicians with the foundation for designing restorative rehabilitation techniques enabling people with stroke regain functions. The examples of these techniques have been seen in forced use and CIMT.

The origin of CIMT has been credited to the work of Edward Taub, though he acknowledged previous researchers' contribution to the understanding of this phenomenon [25]. Here, monkeys received surgical abolition of somatic sensation of one or the two forelimbs. Thereafter, they were trained based on some learning principles. The abolition of somatic sensation conditioned the monkeys to never use the limbs again. However, the animals were forced to use the affected limb as soon as the unaffected limb is constrained. Additionally, it was noted that the use of the affected limb was further strengthened when the animal was rewarded with food after a successful attempt. The use of the limb was achieved through breaking task into manageable components in a process known as shaping. Subsequently, this protocol was first translated to a patient with chronic stroke in which his unaffected upper limb was constrained for about 90% of the waking hours and patients with chronic stroke and brain injury [26, 30]. This variant of CIMT is known as forced use. The current and widely used variant of CIMT was started on chronic patients who received constraint of the unaffected limb for 90% of the waking hours and task practice with the affected limb for 6 h for 10 days [27]. So far, there are many variants of CIMT in forms of randomized controlled trials (RCT), single case experimental studies, case reports, and so forth. In fact, as of now, there are not less than 500 studies of CIMT including a highly rigorous multicenter RCT known as the EXCITE trial [31].

In addition to the explanations above, thus far CIMT has been extended to the rehabilitation of many conditions such as cerebral palsy, brachial plexus injury, spinal cord injury, multiple sclerosis, aphasia, and hearing impairments [32–36]. All these reported improvement in the outcomes of interest. Furthermore, currently CIMT is used for both upper and lower limbs [37, 38]. However, the studies in lower limbs are still few, and therefore, more studies need to be carried out to determine the effect of CIMT in lower limb motor impairment after stroke. This will help in translating the opportunities gained with upper limb CIMT and possibly further researchers' understanding of the mechanisms of recovery of motor function following stroke.

## 4.2 Components of constraint-induced movement therapy

The basic components of CIMT include repetitive task/shaping practice with the affected limb, constraint of the unaffected limb, and transfer package [39]. Task practice involves carrying out the usual everyday tasks or activities such as



brushing, cooking, washing plates, playing tennis, kicking or throwing a ball, and picking up a cup and taking it to the mouth to drink from it [40, 41]. In shaping practice, similar tasks as in task practice are carried out; however, they are broken down into manageable components in which the participant will have to master a component before proceeding to another [40–42]. For example, when a participant is to learn how to pick up a cup and take it to the mouth to drink from it, he will be taught to learn how to grasp the cup, then pick it up, and the rest follows. Constraining the unaffected limb involves use of slings, mitts, or any possibly practicable orthotic device to prevent the use of the limb [42]. Sometimes, this can be achieved through conscious restriction of the use of the unaffected limb by the patient. The transfer package is any method used to foster compliance with task/shaping practice and the constraint components of CIMT [41]. The methods used include the use of logbook, diary, everyday administration of motor activity log, monitoring by caregivers, and practice outside the laboratory (e.g., at home).

In the literature, importance of repetitive task/shaping practice in being an essential component responsible for improvement following CIMT has been underscored. However, according to Taub and colleagues, transfer package is very key to improvement in function following CIMT [43]. This finding probably confirms the importance of task or shaping repetition as transfer package is meant to foster compliance and use of the affected limb outside laboratory. In essence, transfer package helps to achieve more repetition. Similarly, constraint ensures that use of the constrained limb is restricted, while the use of the affected limb is maximized. This is also akin to encouraging more repetitions with the affected limb. Pooling all these arguments together, we can see that the other components of CIMT, constraint, and transfer package work to provide high repetition of task or shaping practice. This is more especially that it has been reported for the upper limbs that there was no significant difference whether constraint is used or not during CIMT [44]. However, studies on CIMT for the lower limb need to determine whether use of constraint is necessary, tolerable, or even practicable. Some studies on upper limb have indicated some compliance or tolerability of the participants with constraint [45, 46].

### **4.3 Protocols of constraint-induced movement therapy**

Constraint-induced movement therapy has evolved over the years to have many different protocols. The protocols involve either modification of the task/shaping practice, constraint or both components of CIMT; or even the process or setting of its administration. These protocols include using hours as measure of intensity of task/shaping practice, using number of repetitions as measure of intensity of task/shaping practice, home-based CIMT, use of automated constraint-induced therapy extension (AutoCITE), and CIMT without constraint [38, 47–50]. See **Table 1** for the details of these different protocols. The protocol that uses number of repetitions is relatively a new approach. Therefore, its details are represented in **Table 2**.

One of the concerns about the standard CIMT is the issue of compliance since it seems to have a long duration of tasks/shaping practice of 6 h or less [51]. However, studies such as those by Kaplon and his colleagues and Stock and his colleagues have all indicated that the time spent practicing task/shaping fell short of what is being claimed [52, 53]. Additionally, many studies have shown that the shorter duration CIMT, the protocol that uses less than or 3 h of task/shaping practice, provides better outcomes [54, 55]. In contrast, the protocol using number of repetition has been touted to be feasible, easier, and may provide better compliance and complements the transfer package component as it takes on the elements of self-management [45, 56]. Recently, it has been shown to be as effective as the one using number of hours [48, 49]. Furthermore, a virtual reality-amplified arm training of just 30 minutes

Protocol	Task/shaping practice	Constraint	Transfer package
Use of hours	Practice for 0.5–6 h for $\geq 10$ days	Constraint for 2 h or more or for 90% of the waking hours	Logbook, diary, everyday administration of motor activity log, monitoring by caregivers, practicing at home, etc.
Without constraint	Same as above	No any constraint	Same as above
Home-based CIMIT	Same as above, but practice is done at home	Same as above	Same as above
Use of AutoCITE	Same as in use of hours, but practice is administered through the use of a computerized mechanical system	Same as above	Same as above
Use of number of repetitions	300–600 repetitions in two or three sessions per day	Constraint for 90% of the waking hours	Same as above

**Table 1.**  
*Different protocols of CIMIT.*

proved very effective at improving motor outcomes at 6 weeks postintervention and 3 months follow-up [57]. This technique known as reinforcement-induced therapy uses virtual reality system with the sole aim of optimizing proper practice with the affected arm during rehabilitation. Similarly, an automated delivery of CIMIT has been used [51]. This form of delivery of CIMIT is aimed at fostering compliance with the CIMIT protocol. Therefore, the goal of future CIMIT protocols should be aimed at improving compliance in order to achieve the massed practice of the technique. This type of protocol as reported in some previous studies can be administered in the form of a distributed practice [48, 58]. A distributed practice is a type of practice in which the practice is divided into sessions per day [42]. Probably, distributed practice will help do away with overwhelm; fatigue and possibly anxiety patients may have and help encourage them perform the required intensity of practice per day.

**4.4 Effects of constraint-induced movement therapy**

Effectiveness of a given rehabilitation intervention can be said to be sound if it is evaluated on different outcomes. This will enable the intervention to have a strong theoretical basis. For CIMIT, its effects have been investigated on impairments such as

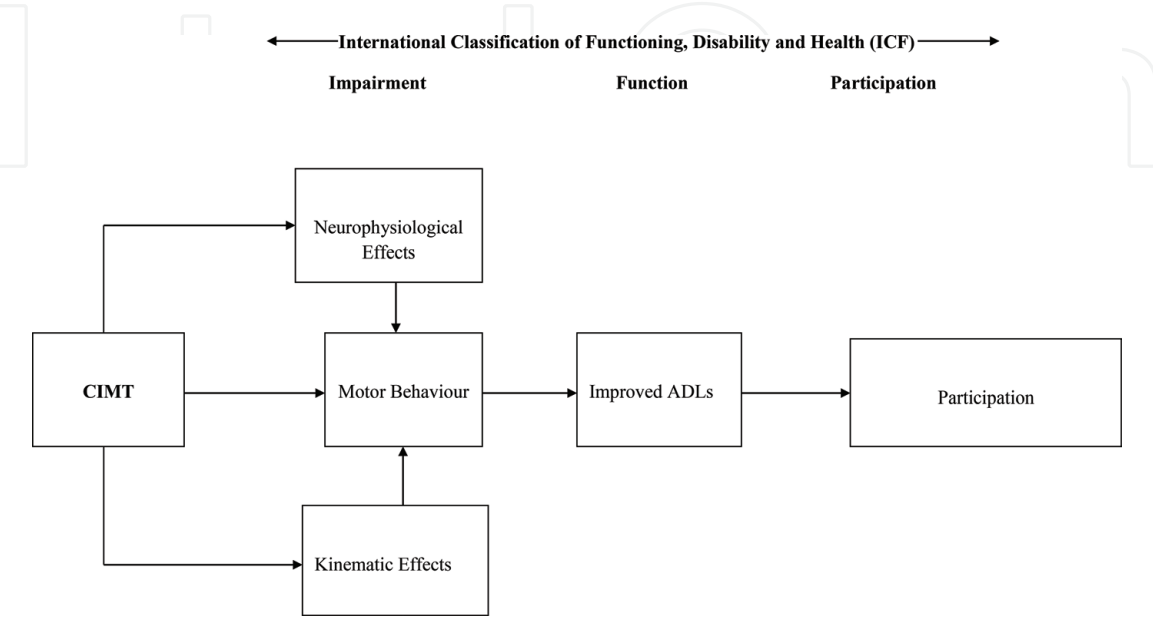
Tasks	No. of repetitions per session	No. of sessions per day	No. of days per week	No. of weeks
(1) Writing the letter A	20	2 or 3	5 or 7	2 or more
(2) Picking up a cup, taking it to the mouth, and drinking from it	20	2 or 3	5 or 7	2 or more
(3) Drawing a circle	20	2 or 3	5 or 7	2 or more
(4) Transferring an object from one place to another on a table	20	2 or 3	5 or 7	2 or more
(5) Imitation of teeth brushing with the middle or the index finger	20	2 or 3	5 or 7	2 or more

**Table 2.**  
*Example of the protocols of tasks/shaping practice for the CIMIT using number of repetitions.*

neurophysiological and behavioral, real-world arm use, motor function, activities of daily living, quality of life, and kinematics [31, 55, 59–64]. Recently, it has been demonstrated that neurophysiological changes following CIMT well correlate with motor function [48, 64]. The examples of effects of CIMT and the effects based on the International Classification of Functioning, Disability and Health (ICF) model are summarized in **Table 3** and **Figure 4**, respectively. These effects are measured using outcome measures such as Wolf motor function test (WMFT), action research arm test (ARAT), nine peg hole test (NPHT), motor activity log (MAL), motor function subscale of Fugl-Meyer assessment (FM), upper limb self-efficacy test (UPSET), functional magnetic resonance imaging (fMRI), and transcranial magnetic stimulation (TMS) [27, 30, 65–72]. The WMFT, the ARAT, the FM, and the JPHT are measures of motor function [30, 68, 69, 73]. The fMRI and TMS are measures of neurophysiological functions [70, 72]. The MAL is a measure of real-world arm use [27, 70, 71]. The UPSET is a measure of confidence in the use of the upper limb after stroke [67]. However, the effects are restricted to only those who have mild to moderate impairment. Consequently, the inclusion criteria used during CIMT include patients who have 10° and 30° of interphalangeal and wrist joints extension, respectively, or those that have some level of motor activity enough to enable them practice some tasks with the limb [40, 59, 73]. Thus, the existing protocol of CIMT is not a one size fits all kind of rehabilitation technique, though the need to investigate how its application can be extended to all forms of degree or level of impairment arises.

Effects	Examples
Neurophysiological effects	Increase in gray matter, increase in cortical map, increase in cortical activation, decreased transcallosal inhibition, improvement in central conduction time, improvement in resting motor threshold, and prolongation of cortical silent period
Kinematics	Efficient temporal and spatial movements, improved preplanned movement and control
Behavioral	Improved real-world arm use, improved use of the hand in activities requiring fine motor control, improved motor function, improved gait and balance

**Table 3.**  
*Effects of constraint-induced movement therapy (CIMT).*



**Figure 4.**  
*Schematic representation of the effects of constraint-induced movement therapy (CIMT) in stroke patients. ADL, activities of daily living.*

#### **4.5 Constraint-induced movement therapy: the future perspective**

At the moment, CIMT is administered only to the patients who have mild to moderate impairment. These are those who can to some extent perform some motor activity with the affected part. However, it is possible to integrate mental practice and motor imagery in the existing protocols of CIMT for the benefits of those with severe impairment bearing in mind the neurophysiological effect of CIMT and its relationship with motor function [64], and the role of mirror neurons when actions of a second or third person are observed or when task performance is imagined [74]. This perspective is for the benefit of those who may have severe impairment—those who do not have any appreciable motor activity enough to enable them perform any task. These forms of CIMT may be called passive CIMT (pCIMT) and imagery CIMT (iCIMT), respectively. For the pCIMT, the task/shaping practice should be carried out in high repetition with the affected limb similar to the repetition in the standard CIMT, and the unaffected limb should be constrained for 90% of the waking hours or less depending on the therapist's clinical decision. However, the transfer package component may be initiated or deferred until the patients improve in their motor ability that they can use to practice. This too depends on the therapist's clinical decision and reasoning. Similarly, for the iCIMT, the patient should be made to observe a second person or third person performing task/shaping practice to the required number of repetition as in the normal CIMT protocol. Here, the unaffected limb may or may not be constrained.

Another possible perspective is to use the number of repetition as a measure of intensity of task/shaping practice during CIMT. Already, there are indications that these types of protocols seem to be easier and have similar effectiveness compared to the one using number of hours [48, 49]. This is because number of repetition of task is important for motor recovery [45, 51, 58].

#### **4.6 A hypothetical case to help enhance problem solving, clinical decision making, and clinical reasoning in constraint-induced movement therapy**

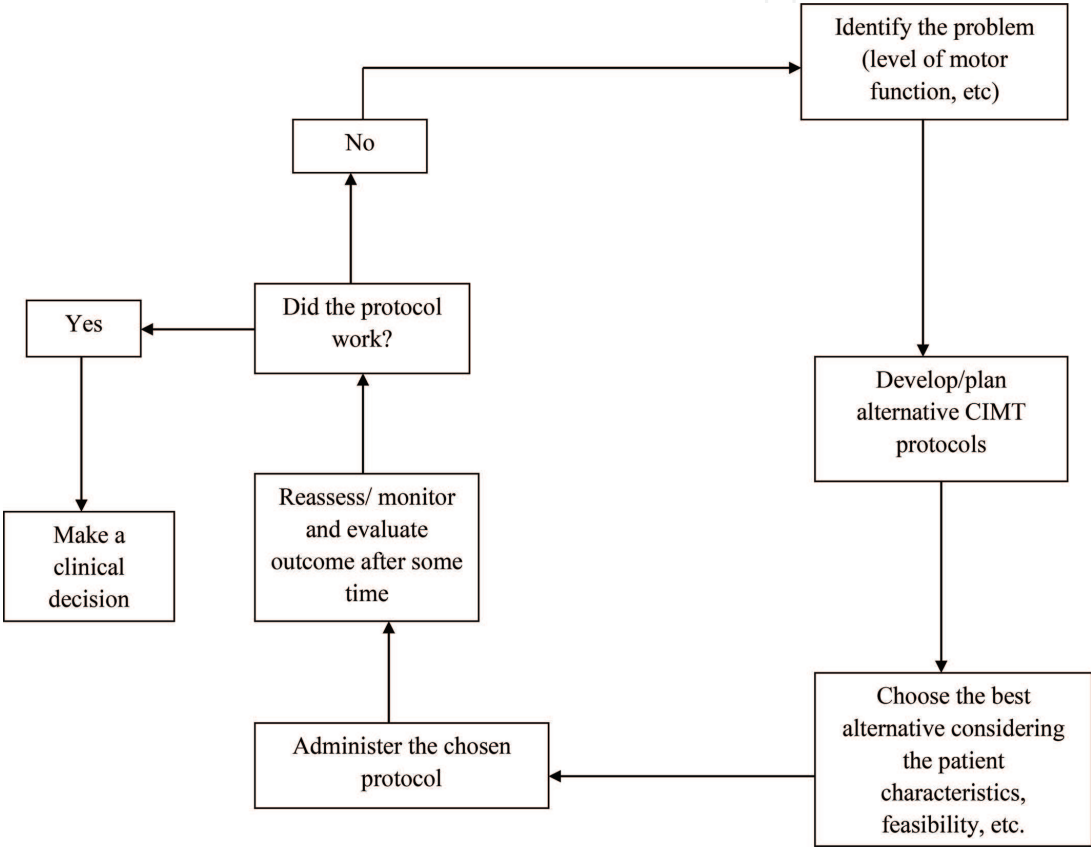
Mr. MM is a 67-year-old community dwelling man who had a stroke 10 days earlier. Before his stroke, he was a very active subsistent farmer. He woke up the morning he had the stroke and realized that he was unable to move any part of his body. Upon realizing his condition, his family rushed him to a nearby secondary health facility where he was diagnosed with a stroke and started receiving care accordingly. According to clinical assessments and reports, he had ischemic stroke involving the left hemisphere. Mr. MM's main presentations were inability to sit or stand and walk without help from the formal or informal caregivers, and inability to use his right upper limb for any activity. Other presentations include inability to maintain his position in sitting without help.

In order to design a rehabilitation program using CIMT say for the upper limb for this patient, we need to first of all identify his problems. We can identify his problem by assessing his level of activity with the upper limb using, for example, Wolf motor function test or motor activity log, and being aware of the degree of freedom at the joints of the upper limb [27, 31]. Similarly, we can assess the range of motion (ROM) in the individual joints and assess the limb for spasticity, for instance, using modified Ashworth scale (MAS) [75]. Thereafter, we can develop alternative protocols of CIMT for him. For instance, we can decide to plan 6 h or less of task/shaping practice for the right limb with constraint for 90% or less of the waking hours, or we can plan for 300 or more of task/shaping practice for the right limb with constraint for 90% or less of the waking hours divided into two or more sessions per day. Additionally, we may decide to manage the patient at home, in the



clinic, or provide him with the skills to self-manage. In particular, self-management is important for positive outcomes after stroke [76, 77].

After making an informed clinical decision on the choice of the best alternative or protocol, we can then administer it and reassess or monitor and evaluate its progress after sometime. If it works fine, we can decide to continue with it; if it does not provide any appreciable outcome, we can decide to choose another alternative protocol. This is the clinical reasoning. However, the existing studies on CIMT do not especially emphasize on clinical reasoning, rather they give a straightjacket prescription of what should be done throughout the period of care whether the patients improve better or not. Clinical reasoning is important to physiotherapy practice [78]. The process of problem solving, clinical decision making, and clinical reasoning in constraint-induced movement therapy is represented in **Figure 5**.



**Figure 5.**  
*Problem solving, clinical decision making, and clinical reasoning in CIMT.*

## 5. Conclusion

Constraint-induced movement therapy (CIMT) plays a major role in the rehabilitation of movement after stroke. Its effects range from improved real-world use of the arm, motor function, neurophysiological functions, kinematics, and quality of life. The problem is that CIMT is not done for all categories of patients with stroke. It is done for only those with mild to moderate impairment. Additionally, there are varied protocols of CIMT. Some protocols use number of hours and some others use number of repetitions of tasks/shaping practice. The protocols that use number of hours of task practice are not clear and are resource intensive, and as such, they could interfere with the process of clinical decision making. Consequently, the use of number of repetitions of task practice to determine the intensity or the amount of task practice may be more appropriate. Secondly, there is a need to extend the use of

CIMT application to those patients with severe impairments after stroke probably by asking the patients to wear constraint on the unaffected limb and imagine they are practicing tasks with the affected limb. However, skills in problem solving, clinical decision making, and clinical reasoning are required by the physiotherapist in order to make an effective use of CIMT. These skills may be acquired through reflective practice and continuing professional development.

### **Conflict of interest declaration**

The author does not have any conflict of interest to declare.

### **Author details**

Auwal Abdullahi

Department of Physiotherapy, Faculty of Health Sciences, Bayero University Kano, Nigeria

\*Address all correspondence to: [aabdullahi.pth@buk.edu.ng](mailto:aabdullahi.pth@buk.edu.ng)

### **IntechOpen**

© 2018 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

## References

- [1] Hatano S. Experience from a multicentre stroke register: A preliminary report. *Bulletin of the World Health Organisation*. 1976;**54**(5):541-553
- [2] Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, et al. An updated definition of stroke for the 21st century: A statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013;**44**(7):2064-2089. DOI: 10.1161/STR.0b013e318296aeca
- [3] Easton JD, Saver JL, Albers GW, Alberts MJ, Chaturvedi S, Feldmann E, et al. Definition and evaluation of transient ischemic attack: A scientific statement for healthcare professionals from the American Heart Association/American Stroke Association Stroke Council; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular Radiology and Intervention; Council on Cardiovascular Nursing; and the Interdisciplinary Council on Peripheral Vascular Disease. *Stroke*. 2009;**40**:2276-2293. DOI: 10.1161/STROKEAHA.108.192218
- [4] Del Zoppo JD, Hallenbeck JM. Advances in the vascular pathophysiology of ischemic stroke. *Thrombosis Research*. 2000;**98**(3):73-81
- [5] Helgason CM, Wolf PA. American heart association prevention conference IV: Prevention and rehabilitation of Stroke. *Circulation*. 1997;**96**:701-707. DOI: 10.1161/01.CIR.96.2.701
- [6] Frizzell JP. Acute stroke: Pathophysiology, diagnosis, and treatment. *AACN Clinical Issues: Advanced Practice in Acute and Critical Care*. 2005;**16**(4):421-598
- [7] Balci K, Utku U, Asil T, Unlu E. Simultaneous onset of hemorrhagic and ischemic strokes. *The Neurologist*. 2007;**13**(3):148-149
- [8] Vermeer SE, Algra A, Franke CL, Koudstaal PJ, Rinkel GJE. Long-term prognosis after recovery from primary intracerebral hemorrhage. *Neurology*. 2002;**59**(2):205-209
- [9] Mishra LD. Cerebral blood flow and anaesthesia. *Indian Journal of Anaesthesia*. 2002;**46**(2):87-95
- [10] Kalogeris T, Baines CP, Krenz M, Korthuis RJ. Cell biology of ischemia/reperfusion injury. *International Review of Cell and Molecular Biology*. 2012;**298**:229-317. DOI: 10.1016/B978-0-12-394309-5.00006-7
- [11] Sims NR, Anderson MF. Mitochondrial contributions to tissue damage in stroke. *Neurochemistry International*. 2002;**40**(6):511-526
- [12] Deba P, Sharma S, Hassan KM. Pathophysiologic mechanisms of acute ischemic stroke: An overview with emphasis on therapeutic significance beyond thrombolysis. *Pathophysiology*. 2010;**17**:197-218. DOI: 10.1016/j.pathophys.2009.12.001
- [13] Hachinski V, Iadecola C, Petersen C, Breteler MM, Nyenhuis DL, Black SE, et al. National institute of neurological disorders and stroke—Canadian stroke network vascular cognitive impairment harmonization standards. *Stroke*. 2006;**37**:2220-2241. DOI: 10.1161/01.STR.0000237236.88823.47
- [14] World Heart Federation (WHF). Global Burden of Stroke [Internet]. 2017. Available from: <http://www.world-heart-federation.org/cardiovascular-health/stroke/> [Accessed: May 25, 2018]
- [15] Chong J, Sacco R, Ovbiagele B, Nguyen-Huynh MN. Stroke epidemiology: Advancing our

understanding of disease mechanism and therapy. *Neurotherapeutics*. 2011;**8**(3):319-329. DOI: 10.1007/s13311-011-0053-1

[16] O'Donnell MJ, Xavier D, Liu L, et al. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (The Interstroke Study): A case-control study. *Lancet*. 2010;**376**:112-123. DOI: 10.1016/S0140-6736(10)60834-3

[17] Petrea RE, Beiser AS, Seshadri S, Kelly-Hayes M, Kase CS, Wolf PA. Gender differences in stroke incidence and poststroke disability in the framingham heart study. *Stroke*. 2009;**40**:1032-1037. DOI: 10.1161/STROKEAHA.108.542894

[18] Niewada M, Kobayashi A, Sandercock PA, Kaminski B, Czlonkowska A. Influence of gender on baseline features and clinical outcomes among 17,370 patients with confirmed ischaemic stroke in the international stroke trial. *Neuroepidemiology*. 2004;**24**:123-128

[19] Pound P, Gompertz P, Ebrahim S. A patient-centred study of the consequences of stroke. *Clinical Rehabilitation*. 1998;**12**(4):338-347

[20] Shumway-Cook A, Woollacott MH. *Motor Control: Theory and Practical Applications*. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2001

[21] Haas B. Motor Control. In: Everett T, Kell C, editors. *Human Movement*. Edinburgh: Churchill Livingstone; 2010. pp. 47-60

[22] Gerloff C, Corwell B, Chen R, Hallett M, Cohen LG. The role of the human motor cortex in the control of complex and simple finger movement sequences. *Brain*. 1998;**121**(Pt 9):1695-1709

[23] Swenson RS. Chapter 11: The Cerebral Cortex. Review of Clinical and Functional Neuroscience [Internet]. 2006. Available from: <https://www.dartmouth.edu/~rswenson/NeuroSci/> [Accessed: June 6, 2018]

[24] Lanciego JL. Basal ganglia circuits: what's now and next? *Frontiers in Neuroanatomy*. 2012;**6**:4. DOI: 10.3389/fnana.2012.00004

[25] Taub E, Berman AJ. Avoidance conditioning in the absence of relevant proprioceptive and exteroceptive feed back. *Journal of Comparative and Physiological Psychology*. 1963;**56**:1012-1016. DOI: 10.1037/h0048315

[26] Ostendorf CG, Wolf SL. Effect of forced use of the upper extremity of a hemiplegic patient on changes in function: A single-case design. *Physical Therapy*. 1981;**61**(7):1022-1028. DOI: 10.1093/ptj/61.7.1022

[27] Taub E, Miller NE, Novack TA, Cook IEW, Fleming WC, Nepomuceno CS. Technique to improve chronic motor deficit after stroke. *Archives of Physical Medicine and Rehabilitation*. 1993;**74**(4):347-354

[28] Taub E, Uswatte G, King DK, Morris D, Crago JE, Chatterjee A. A placebo-controlled trial of constraint-induced movement therapy for upper extremity after stroke. *Stroke*. 2006;**37**(4):1045-1049

[29] Sunderland A, Tuke A. Neuroplasticity, learning and recovery after stroke: A critical evaluation of constraint-induced therapy. *Neuropsychological Rehabilitation*. 2005;**15**:81-98

[30] Wolf SL, Lecraw DE, Barton LA, Jann BB. Forced use of hemiplegic upper extremities to reverse the effect of learned nonuse among chronic stroke and head-injured patients. *Experimental Neurology*. 1989;**104**(2):125-132



- [31] Wolf SL, Winstein CJ, Miller JP, Taub E, Uswatte G, Morris D. Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: The EXCITE randomized clinical trial. *JAMA*. 2006;**296**(17):2095-2104. DOI: 10.1001/jama.296.17.2095
- [32] Okamoto H, Fukushima M, Teismann H, Lagemann L, Kitahara T, Inohara H, et al. Constraint-induced sound therapy for sudden sensorineural hearing loss—Behavioral and neurophysiological outcomes. *Scientific Reports*. 2014;**4**:3927. DOI: 10.1038/srep03927
- [33] Matuti GD-S, Pires CVG, Rafael Garcia RE, Oliveira CB. Constraint induced movement therapy in patients with incomplete tetraplegia after spinal cord injury. *Archives of Physical Medicine and Rehabilitation*. 2015;**96**(10):85-e86. DOI: 10.1016/j.apmr.2015.08.287
- [34] Buesch FE, Schlaepfer B, de Bruin ED, Wohlrab G, Ammann-Reiffer C, Meyer-Heim A. Constraint-induced movement therapy for children with obstetric brachial plexus palsy: Two single-case series. *International Journal of Rehabilitation Research*. 2010;**33**(2):187-192. DOI: 10.1097/MRR.0b013e3283310d6e
- [35] Mark VW, Taub E, Uswatte G, Morris DM, Cutter GR, Adams TL, et al. Phase II randomized controlled trial of constraint-induced movement therapy in multiple sclerosis. Part 1: Effects on real-world function. *Neurorehabilitation and Neural Repair*. 2018;**32**(3):223-232. DOI: 10.1177/1545968318761050
- [36] Burkhardt J, Sheridan J, Villavecchia P, Hollander L, Garbarini JG. Effectiveness of constraint-induced movement therapy for functional use in children with spastic hemiplegic cerebral palsy: A systematic review. *American Journal of Occupational Therapy*. 2017;**71**:7111520311p1. DOI: 10.5014/ajot.2017.71S1-PO6088
- [37] Mark VW, Taub E, Uswatte G, Bashir K, MD CGR, Bryson CC, et al. Constraint-induced movement therapy for the lower extremities in multiple sclerosis: Case series with 4-year follow-up. *Archives of Physical Medicine and Rehabilitation*. 2013;**94**:753-760. DOI: 10.1016/j.apmr.2012.09.032
- [38] Etoom M, Hawamdeh M, Hawamdeh Z, et al. Constraint-induced movement therapy as a rehabilitation intervention for upper extremity in stroke patients: Systematic review and metaanalysis. *International Journal of Rehabilitation Research*. 2016;**39**(3):197-210. DOI: 10.1097/MRR.0000000000000169
- [39] Morris DM, Taub E, Mark VW. Constraint-induced movement therapy: Characterizing the intervention protocol. *Europa Medicophysica*. 2006;**42**:257-268
- [40] Kim H, Yoo EY, Jung MY, Kim J, Park JH, Kang DH. The effects of mental practice combined with modified constraint-induced therapy on corticospinal excitability, movement quality, function, and activities of daily living in persons with stroke. *Disability and Rehabilitation*. 2017;**9**:1-9. DOI: 10.1080/09638288.2017.1337817
- [41] Taub E. The behaviour analytic origins of constraint-induced movement therapy: An example of behavioural neurorehabilitation. *The Behaviour Analysis*. 2012;**35**:155-178
- [42] Wolf SL. Revisiting constraint-induced movement therapy: Are we too smitten with the mitten? is all nonuse “learned”? and other quandaries. *Physical Therapy*. 2007;**87**(9):1212-1223. DOI: 10.2522/ptj.20060355

- [43] Taub E, Uswatte G, Mark VW, Morris DM, Barman J, Bowman MH, et al. Method for enhancing real-world use of a more-affected arm in chronic stroke: The transfer package of CI therapy. *Stroke*. 2013;**44**(5):1383-1388. DOI: 10.1161/STROKEAHA.111.000559
- [44] Brogårdh C, Vestling M, Sjölund BH. Shortened constrained induced movement therapy in subacute stroke-no effect of using a restraint: A randomized controlled study with independent observers. *Journal of Rehabilitation Medicine*. 2009;**41**:231-236
- [45] Abdullahi A, Shehu S, Dantani BI. Feasibility of high repetitions of tasks practice during constraint induced movement therapy in an acute stroke patient. *International Journal of Therapy and Rehabilitation*. 2014;**21**(4):190-195. DOI: 10.12968/ijtr.2014.21.4.190
- [46] Collin A, Grimee M, Libois PY. The actimetry as assessment method of patients' compliance and effectiveness of constraint induced movement therapy. *Annals of Physical and Rehabilitation Medicine*. 2015;**58**(1):e8-e9
- [47] Abu Taria H, Almalty A-M, Sbeih Z, Al-Oraib S. Constraint induced movement therapy for stroke survivors in Jordan: A home-based model. *International Journal of Therapy and Rehabilitation*. 2010;**17**(12):638-646. DOI: 10.12968/ijtr.2010.17.12.638
- [48] Abdulahi A. Number of repetitions as measure of dose of shaping practice in acute stroke: Preliminary results of a randomized controlled trial. *Neurorehabilitation and Neural Repair*. 2018;**32**(4-5):402-403. DOI: 1177/1545968318765498
- [49] Abdulahi. Effects of number of repetitions and number of hours of shaping practice during constraint-induced movement therapy: A randomized controlled trial. *Neurology Research International*. 2018. DOI: 10.1155/2018/5496408
- [50] Lum PS, Taub E, Schwandt D, Postman M, Hardin P, Uswatte G. Automated constraint-induced therapy extension (AutoCITE) for movement deficits after stroke. *Journal of Rehabilitation Research and Development*. 2004;**41**(3A):249-258
- [51] Viana R, Teasell R. Barriers to the implementation of constraint-induced movement therapy into practice. *Topics in Stroke Rehabilitation*. 2012;**19**(2): 104-114. DOI: 10.1310/tsr1902-104
- [52] Kaplon RT, Prettyman MG, Kushi CL, Winsten CJ. Six hours in the laboratory: Quantification of practice time during constraint induced therapy. *Clinical Rehabilitation*. 2007;**21**(10):950-958. DOI: 10.1177/0269215507078333
- [53] Stock R, Thrane G, Askim T. Norwegian constraint-induced therapy multisite trial: Adherence to treatment protocol applied early after stroke. *Journal of Rehabilitation Medicine*. 2015;**47**:816-823
- [54] Nijland R, Kwakkel G, Bakers J, van Wegen E. Constraint-induced movement therapy for the upper paretic limb in acute or sub-acute stroke: A systematic review. *International Journal of Stroke*. 2011;**6**(5):425-433. DOI: 10.1212/WNL.0b013e3181ab2b27
- [55] Dromerick AW, Lang CE, Birkenmeier RL, Wagner JM, Miller JP, Videen TO, et al. Very early constraint-induced movement during stroke rehabilitation (VECTORS): A single-center RCT. *Neurology*. 2009;**73**(3):195-201
- [56] Birkenmeier RL, Prager EM, Lang CE. Translating animal doses of task-specific training to people

with chronic stroke in 1-hour therapy sessions: A proof-of-concept study. *Neurorehabilitation and Neural Repair*. 2010;**24**(7):620-635. DOI: 10.1177/1545968310361957

[57] Ballester BR, Maier M, San Segundo MR-M, Castañeda V, Duff A, Verschure PFMJ. Counteracting learned non-use in chronic stroke patients with reinforcement-induced movement therapy. *Journal of NeuroEngineering and Rehabilitation*. 2016;**13**:74. DOI: 10.1186/s12984-016-0178-x

[58] Abdullahi A, Shehu S, Abdurrahman Z, Bello B. Determination of optimal dose of tasks practice during constraint induced movement therapy in a stroke patient with severe upper limb pain. *Indian Journal of Physiotherapy and Occupational Therapy—An International Journal*. 2015;**9**(1):198. DOI: 10.5958/0973-5674.2015.00039.8

[59] El-Helow MR, Zamzam ML, Fathalla MM, et al. Efficacy of modified constraint-induced movement therapy in acute stroke. *European Journal of Physical and Rehabilitation Medicine*. 2015;**51**(4):371-379

[60] Yu C, Wang W, Zhang Y, et al. The effects of modified constraint-induced movement therapy in acute subcortical cerebral infarction. *Frontiers in Human Neuroscience*. 2017;**11**:265. DOI: 10.3389/fnhum.2017.00265

[61] Ro T, Noser E, Boake C, et al. Functional reorganization and recovery after constraint-induced movement therapy in subacute stroke: Case reports. *Neurocase*. 2006;**12**:50-60. DOI: 10.1080/13554790500493415

[62] Wu CY, Lin KC, Chen HC, Chen IH, Hong WH. Effects of modified constraint-induced movement therapy on movement kinematics and daily function in patients with stroke: A kinematic study of motor control

mechanisms. *Neurorehabilitation and Neural Repair*. 2007;**21**(5):460-466

[63] Page SJ, Sisto S, Johnston HV, Levine P. Modified constraint-induced therapy after subacute stroke. *Neurorehabilitation and Neural Repair*. 2002;**16**:290-295

[64] Abdullahi A. Neurophysiological effects of constraint-induced movement therapy and motor function: A systematic review. *International Journal of Therapy and Rehabilitation*. 2018;**25**(4):167-176. DOI: 10.12968/ijtr.2018.25.4.167

[65] Abdullahi A. Upper limb self-efficacy test (UPSET): A measure of confidence in the use of upper limb after stroke. *Advances of Science for Medicine*. 2016;**1**(2):10-18

[66] Lin J-H, Hsu M-J, Sheu C-F. Psychometric comparisons of 4 measures for assessing upper-extremity function in people with stroke. *Physical Therapy in Sport*. 2009;**89**(8):840-850

[67] Wolf SL, Catlin PA, Ellis M, Archer AL, Morgan B, Piacentino A. Assessing wolf motor function test as outcome measure for research in patients after stroke. *Stroke*. 2001;**32**(7):1635-1639

[68] Uswatte G, Taub E, Morris D, Vignolo M, McCulloch K. Reliability and validity of the upper-extremity motor activity log-14 for measuring real-world arm use. *Stroke*. 2005;**36**(11):2493-2496

[69] vanderLee JH, Beckerman H, Knol DL, DeVet HCW, Boute LM. Clinimetric properties of the motor activity log for the assessment of arm use in hemiparetic patients. *Stroke*. 2004;**35**(6):1410-1414

[70] Zittel S, Weiller C, Liepert J. Citalopram improves dexterity in chronic stroke patients. *Neurorehabilitation and Neural Repair*. 2008;**22**(3):311-314

- [71] Dobkin BH, Firestine A, West M. Ankle dorsiflexion as an fMRI paradigm to assay motor control for walking during rehabilitation. *NeuroImage*. 2004;**23**:370-381
- [72] Sawaki L, Butler AJ, Leng X, Wassenaar PA, Mohammad YM, Blanton S, et al. Differential patterns of cortical reorganization following constraint-induced movement therapy during early and late period after stroke: A preliminary study. *Neurorehabilitation*. 2014;**35**(3): 415-426. DOI: 10.3233/NRE-141132
- [73] Cunningham DA, Varnerin N, Machado A, Bonnetta C, Janini D, Roelle S, et al. Stimulation targeting higher motor areas in stroke rehabilitation: A proof-of-concept, randomized, double-blinded placebo-controlled study of effectiveness and underlying mechanisms. *Restorative Neurology and Neuroscience*. 2015;**33**(6):911-926. DOI: 10.3233/RNN-150574
- [74] Braun SM, Beurskens AJ, Borm PJ, Schack T, Derick Wade DT. The effects of mental practice in stroke rehabilitation: A systematic review. *Archives of Physical Medicine and Rehabilitation*. 2006;**87**(6):842-852. DOI: 10.1016/j.apmr.2006.02.034
- [75] Kaya T, Karatepe AG, Gunaydin R, et al. Inter-rater reliability of the modified ashworth scale and modified modified ashworth scale in assessing poststroke elbow flexor spasticity. *International Journal of Rehabilitation Research*. 2011;**34**:59-64
- [76] Korpershoek CL, van der Bijl J, Hafsteinsdottir TB. Self-efficacy and its influence on recovery of patients with stroke: A systematic review. *Journal of Advanced Nursing*. 2011;**67**(9):1876-1894. DOI: 10.1111/j.1365-2648.2011.05659.x
- [77] Jones F, Riazi A. Self-efficacy and self-management after stroke: A systematic review. *Disability and Rehabilitation*. 2011;**33**(10):797-810. DOI: 10.3109/09638288.2010.511415
- [78] Baker SE, Painter EE, Morgan BC. Systematic clinical reasoning in physical therapy (SCRIPT): Tool for the purposeful practice of clinical reporting in orthopaedic manual therapy. *Physical Therapy*. 2017;**97**:61-70