INTRODUCTION

Acute ischemic stroke (AIS) is the leading cause of adult disability in the USA. AIS most commonly occurs when a blood vessel is obstructed either by thrombosis or embolic leading to irreversible brain injury and subsequent focal neurologic deficits ***(Bansal et al., 2013).***

Stroke has recently declined from the third to the fourth leading cause of death in the United States Nonetheless; it remains the second leading cause of death worldwide. Therefore, continued research into the pathophysiology and potential emerging therapeutic interventions for cerebrovascular disease are more important than ever ***(Towfighi and Saver, 2011).***

Across population about 87% of stroke are ischemic Since 1950, stroke death rates in the US and in many other countries have dramatically declined In 1950, the age-adjusted death rate from stroke in the US was 180.7/100,000; by 2005, it had fallen to 46.6/100,000. In 1950, stroke mortality for ages 75–84 was approximately 1,500/100,000 by 2005, this had declined to 335/100,000 ***(Boysen et al., 2009)***.

In 2005, there was an estimated 16 million first-ever strokes worldwide and 5.7 million deaths attributed to stroke. In the absence of population-wide intervention, it is estimated that by 2015 the number of first-ever stroke cases will rise to 18 million and the number of stroke-attributed deaths will rise to 6.5 million. These numbers will further rise to 23 million first-ever strokes and 7.8 million deaths by 2030 ***(Johnston et al., 2009).***

The incidence of stroke among Egyptians is not accurately known due to lack of reliable surveys. Yet it is generally estimated to be 2.1 per 1,000 inhabitants, 78% of which are ischemic ***(Abdulghani and Etribi, 2003).***

Available facts and figures may easily explain why the economic burden of stroke is requiring increasing attention for more effective health care planning and resources allocation. An international comparison of stroke cost studies showed that, on average, 0.27% of gross domestic product was spent on stroke by national health systems, and stroke care accounted for ~3% of total health care expenditures ***(Evers et al., 2004).***

In the United States, the total direct and indirect cost of stroke for 2008 is estimated at $65.5 billion. Direct costs, which include the cost of physicians and other health professionals, acute and long-term care, medications and other medical durables, account for 67% of total costs, while the remaining 33% is due to indirect costs, which consider lost productivity resulting from morbidity and mortality ***(Rosamond et al., 2008)***.

In the 27 European union (EU)countries, total annual cost of stroke is estimated at €27 billion: €18.5 billion (68.5%) for direct and €8.5 billion (31.5%) for indirect costs. A further sum of €11.1 billion is calculated for the value of informal care including informal care in the total amount, percentages would change to 48.6% for direct, 22.3% for indirect and 29.1% for informal care costs ***(Carlo, 2009).***

Quality of life (QOL) has been defined as “an individuals’ perception of their position in life in the context of the culture and value systems in which they live and in relation to their goals, expectations, standards and concerns” ***(Madden et al., 2006)***.

There are a number of factors which seem to be contributing towards a decline in QOL of stroke patients. Advanced age, the severity of motor impairment or paralysis, lack of perceived social supports, inability to return to work, supratentorial lesion locations, impaired cognition, and the presence of comorbid health problems have been associated with a decline in QOL and should be taken into account when making an analysis of stroke results. Several authors have reported a strong association between physical disability, dependency in activities of daily living and QOL. Dependency in activities of daily living has been shown to be associated with physical functioning and the general health domains of

QOL, but not to predict psychological and socioeconomic aspects of QOL ***(Krančiukaitė and Rastenytė, 2006)***.

The correlation between age, sex and QOL has remained obscure. ***Anderson et al. (1996)*** showed that women had a better stroke outcome in terms of social functioning and mental health, but most authors report QOL either to be independent of gender or lower in females. Failure to maintain or re-establish social ties, except for those with family members, seems to be an important determinant of poor QOL in long-term survivors of stroke, whereas high levels of social support have been shown to be related to a better outcome. On the other hand, too much support from the spouse may lead to over protection and under stimulation and lead to a less favorable outcome. To ensure a good outcome, the support of the family is not enough; the support of society is also needed, so that stroke victims feel cared for, loved, valued, and esteemed and are ready to accept assistance from others if needed ***(Krančiukaitė and Rastenytė, 2006)***.

Stroke severity was evaluated with National Institutes of Health Stroke Scale (NIHSS) and categorized as mild (0-6), moderate (7-15), or severe (16-38**) *(Tsenq and Chanq, 2006)*.**

The most common deficit after stroke is hemiparesis of the contralateral upper limb, with more than 80% of those with stroke experiencing it acutely and more than 40% chronically ***(Cramer, 1997).***

Up to 80%of people who have a stroke experience sensory loss in their affected arm. This sensory loss puts the arm at risk for injury and impacts functional use of the arm and the survivors' level of independence during daily activities ***(Doyle et al., 2010).***

In many patients with severe stroke the affected upper limb never becomes useful, even after therapy only about 15 percent of those suffering from severe stroke recover hand functions. The outcome of patients with severe upper limb (UL) paresis is poor ***(Higgins et al., 2005).***

Loss of independence of upper limb function contributes enormously to functional disability, affecting quality of life and independence in 'basic' (washing, grooming, feeding, dressing, etc.) and 'instrumental' activities (shopping, home/financial management, etc.) of daily living ***(Hunter and Crome, 2002).***

Although stroke often results in some degree of long-term impairment and disability, most patients experience some natural recovery of neurological functioning and improvement in ability to perform daily activities ***(Roth et al., 1998).***

There are varying degrees of spontaneous improvement in arm paresis over the first 6 months after stroke. The degree of improvement at 6 months is best predicted by the motor deficit at 1 month despite standard rehabilitative interventions in the ensuing 5 months. However, despite good motor power, some patients do not attain functional use of the affected upper extremity. Quantitative studies of reaching movements in patients suggest that arm paresis consists of higher- order motor planning and sensorimotor integration deficits that cannot be attributed to weakness or presence of synergies ***(Krakauer, 2005)***.

Neurologic and functional recovery is dependent on a large variety of factors such as initial stroke severity, body temperature and blood glucose in the acute phase of stroke, stroke in progression, and treatment and rehabilitation in a dedicated stroke unit. The most important factor for recovery remains the initial severity of the stroke; the ability to perform basic activities of daily living initially is reduced in three out of four patients with stroke. Most often affected is the ability to transfer, dress, and walk. The prognosis of patients with mild or moderate stroke generally is excellent. Patients with severe stroke have a very variable recovery. Although the prognosis of patients with the most severe stroke is generally poor. Functional recovery generally is completed within 3 months of stroke onset. Patients with mild stroke, however, recover within 2 months, patients with moderate stroke within 3 months ***(Olsen et al., 1999)*.**

AIM OF THE WORK

To verify the clinical deficits that predict functional recovery of the upper extremity in acute ischemic stroke patients.

CHAPTER 1
IMPACT OF POST STROKE DIFFERENT DISABILITIES ON FUNCTION

In this context we will concentrate on disabilities concluded in our study

Impact of motor disability on function

Individuals are more likely to have residual impairments that could affect daily living. More than 80% of individuals with stroke experience hemiparesis, and of those people who initially have upper-extremity paresis, it is estimated that 70% have residual impairment. The upper limb makes a significant contribution to most activities of daily living (ADL), Activities of daily living are considered essential to independent living and includes activities such as dressing, eating, and carrying and impairments can compromise participation in many of these essential and meaningful tasks ***(Jocelyn & Janice, 2007)***.

Even mild impairment of upper extremity function after stroke results in significant limitations in daily function and has been demonstrated to negatively impact health-related quality of life ***(Peter et al., 2009).***

The weaker the paretic upper limb, the worst the score on upper-limb performance measures. Strength involves the capacity to generate sufficient force for movement. Weakness in upper-limb muscles could impair stabilization of proximal arm segments, limit reaching ability, confine hand usage, and affect upper-limb control and coordination ***(Jocelyn & Janice, 2007)***.

Impact of neglect & Anosognosia on function

In everyday life, patients with neglect often fail to shave or dress the left side of their body, fail to attend to events and people situated on the left, and collide with objects to their left. Several authors have found that presence of neglect predicts poor functional recovery and inability to manage activities of daily living (ADL) after stroke ***(Jehkonen et al., 2006).***

Individuals with anosognosia for hemiplegia (AHP) have motor impairments leading to gait and self-care deficits but are unaware of the impairments and the deficits in functioning. Because these individuals believe there is nothing wrong with their motor functioning, they do not follow appropriate precautions, resulting in safety risks. Additionally, they do not understand the need for therapeutic interventions, leading to refusals to participate in rehabilitation overall, AHP has been found to be associated with longer rehabilitation stays and poorer functional outcomes following stroke ***(Kortte and Hillis, 2009)***.

Impact of spasticity on function

Spasticity can maintain an abnormal resting limb posture leading to contracture formation. In the arm, severe flexion deformity of the fingers and elbow may interfere with hand hygiene and dressing, as well as affecting self-image. Spasticity, therefore, indirectly affects many aspects of self-care through the maintenance of abnormal limb posture. Articular and periarticular pain caused by the abnormal resting position and immobility of the joints can exacerbate spasticity. Exaggerated reflex responses to cutaneous stimuli may cause painful flexor or extensor spasms, which can interfere with seating, transferring and cause sleep disturbance. In some patients, heterotopic calcification may cause pain and further encourage abnormal limb posture ***(Bhakta, 2000)***.

Impact of somatosensory alteration on function

Functionally, the problems resulting from sensory deficits after stroke can be summarized as:

(1) Impaired detection of sensory information,

(2) Disturbed performance of motor tasks that require somatosensory information, and

(3) Diminished rehabilitation outcomes for the upper limb ***(Hunter and Crome, 2002)***.

The development of secondary complications such as sores, abrasions, and shoulder-hand syndrome has been associated with the impairment of sensation ***(Rand et al., 2001).***

Sensory impairment has also been found to be directly associated with the development of shoulder pain and subluxation ***(Suethanapornkul et al., 2008)***.

The spontaneous use of the upper limb has been noted to significantly decrease when cutaneous sensory processing is impaired ***(Rand et al., 2001)***.

This continued disuse of the affected extremity leads to a further decrease in skilled movement, particularly for functional skills that require a constant sustained muscle contraction ***(Dannenbaum and Jones, 1993)***.

This further contributes to the pattern of learned nonuse. The quality of upper limb movements is also impaired in the presence of sensory impairments ***(Nowak et al., 2007)***.

Stroke survivors were found to have impairments in force control, fine motor manipulation of objects, sensory ataxia, decreased grasp, and changes in comprehension patterns, all of which have been found to be associated with sensory impairment ***(Nowak et al., 2007)***.

Impact of apraxia on function

Furthermore, several studies directly demonstrated the ecological relevance of apraxia by showing that clinical measures of apraxia correlated significantly with the patients' ability to perform several activities of daily living (ADLs), including mealtime behavior, bathing, toileting, and grooming, as well as dressing and brushing one's teeth. Consistently, apraxia significantly impacts upon neurorehabilitation: with respect to several ADLs, the severity of apraxia determined the dependency of stroke patients on their caregivers after discharge from the rehabilitation clinic. Likewise, stroke patients suffering from apraxia less frequently return to work than stroke patients without apraxia ***(Dovern et al., 2012)***.

Impact of Ataxia on function

Ataxia becomes especially prominent during fast goal-directed movements. ***(Wild and Dichgans, 1993)*** and is most pronounced during multi-joint arm motion, with patients showing a decomposition of multi-joint motion and intention tremor in the terminal phase of the movement. Movement slowness has also been measured in patients with chronic cerebellar lesions, especially during fast movements ***(Topka et al., 1998).*** In patients with chronic conditions, movement slowness or bradykinesia usually has not been considered to be a genuine cerebellar deficit but has been interpreted as a patient's deliberate compensation strategy of attempting to avoid high accelerations and the associated high passive interaction torques that the patient may not be able to control when attempting to move the hand to a target in space ***(Bastian et al., 1996).*** That is, to avoid an intention tremor at the end of a goal-directed movement, a cognitive strategy is to move slowly toward it. Here, we report that patients in the acute stage showed signs of motor slowing and not of dyscoordination. Patients performed pointing movements at lower peak velocities and accelerations than did healthy controls. It seems unlikely that this slowing resulted from adopting a compensation strategy. Moreover, it underlines the notion that acute lesions of neural structures involved in motor control lead initially to slowness and signs of bradykinesia ***(Nowak, 2008).***

CHAPTER 2
FUNCTIONAL RECOVERY AFTER ISCHEMIC STROKE

What is functional recovery?

Restoration of ability to perform function in the same way as before injury and successful task completion as typically done by individuals who are not disabled. ***(Levin et al., 2008)*** often, patients that have experienced a stroke exhibit continued functional recovery for many years following their initial injury ***(Cramer, 2008).***

Mechanisms of functional recovery post stroke

Most patients experience some natural recovery of neurologic functioning and improvement in ability to perform activities of daily living**. *(Roth et al., 1998)*** which is recovery that is not rehabilitation-guided ***(Cramer, 2008)***.

A number of mechanisms that have been hypothesized in the literature may contribute to the predictability.These mechanisms are often described as involving either restitution or substitution of function ***(Kwakkela et al., 2004).*** **The restitution** model suggests that the lesioned area recovers as a result of tissue repair, while its function is assumed by other cortical and subcortical structures, either adjacent to or remote from the damaged area. This theory is known as ‘vicariation of function’. To understand this theory, it requires an appreciation of several short-term and long-term physiological processes, such as the unmasking of previously present but functionally inactive connections, axonal and dendritic regeneration (i.e., collateral sprouting), synaptogenesis and denervation hypersensitivity. Based on these mechanisms of repair, **Von Monakow** hypothesized in 1914 that neurological recovery is largely due to a reactivation of functionally suppressed areas remote from, but connected to, the area of primary injury.This process is known as resolution of diaschisis ***(Nudo et al., 2001)***. Recent positron emission tomography (PET) scan studies showed that immediately after an acute stroke, restitution of no infarcted penumbral areas around the infarcted area, i.e., resorption of extracellular and intracellular oedema and necrotic tissue by reperfusion of viable areas, may contribute to spontaneous functional recovery in the early stages after stroke ***(Seitz et al., 1999)***.

The substitution model, by contrast, suggests that functional recovery after stroke occurs largely by behavioural compensation, by which patients learn to compensate for their acquired deficits ***(Nudo et al., 2001)***.

There are currently strong indications that both phenomena, i.e., restitution as well as substitution, are largely responsible for the functional improvement observed after