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STROKE REHABILITATION

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ABSTRACT

Despite advancements in acute stroke management, stroke remains one of the leading causes of disability in Singapore. Stroke management typically commences in an acute hospital for acute stabilisation, followed by inpatient rehabilitation and outpatient rehabilitation. With the trend towards early supported discharge of stroke patients, general practitioners (GPs) are crucial in providing comprehensive and evidence-based post-stroke care, which includes management of co-morbidities and common post-stroke complications. This review aims to cover the common post-stroke complications the GP may encounter when providing care for the stroke survivor. The review will also cover the role of rehabilitation in stroke recovery and the types of rehabilitation resources and services available in Singapore.

Keywords: stroke; recovery; stroke rehabilitation

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INTRODUCTION

Although there have been advancements in acute stroke management, stroke remains a chronic lifelong condition with an enormous burden on the patient, the family, and the healthcare systems. As the ageing population continues to increase, the number of stroke episodes as well as the burden of stroke is expected to increase significantly, posing challenges to limited healthcare resources. According to the Singapore Stroke Registry Annual Report 2021, the number of stroke episodes increased from 6,143 to 9,680 between 2011 and 2021.[1] There was also a significant increase in crude incidence rate from 194.9 per 100,000 population to 283.9 per 100,000 population during this period.[1]

Besides encouraging the population to inculcate healthy lifestyle habits and being proactive in preventive care to minimise cardiovascular risk factors, the incorporation of a comprehensive stroke rehabilitation programme is the key to reduce the burden of stroke. This review aims to provide GPs information on the common post-stroke complications, the role of rehabilitation in stroke recovery, and the types of rehabilitation resources and services available in Singapore.

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EPIDEMIOLOGY OF STROKE AND COMMON RISK FACTORS

Globally, stroke is the second leading cause of death and the third leading cause of disability.[2] Stroke imposes a huge economic burden on the society, with an estimated global cost of stroke being over USD891 billion.[3] In Singapore, cerebrovascular diseases remain as the fourth leading cause of death, with a prevalence of 5.8 percent in 2022.[4] The estimated annual direct and indirect costs of stroke in Singapore amounted to USD4 billion.[5] Hence, it is important to minimise the risk factors of stroke.

The common risk factors of stroke can be categorised into the following:

- Non-modifiable: age, prior history of stroke, history of transient ischaemic attacks (TIAs), family history of stroke or TIA
- Modifiable: Hypertension, diabetes mellitus, hyperlipidaemia, smoking, physical inactivity, diet, abdominal obesity, atrial fibrillation, heart disease, e.g., valvular heart disease and dilated cardiomyopathy, hypercoagulable states, oral contraceptive use

Of note, modifiable risk factors of hypertension, current smoking, abdominal obesity, diet, and physical inactivity accounted for more than 80 percent of the global risk of both ischaemic and haemorrhagic strokes.[6]

COMMON POST-STROKE COMPLICATIONS AND THEIR MANAGEMENT

Motor and Sensory Impairment

The majority of patients with stroke have motor symptoms or signs (80-90 percent).[7] This is mainly due to damage to the primary crossed corticospinal tract. Other reasons could be related to muscle atrophy from learnt disuse.[8]

Motor assessment should include strength assessment, active and passive range of motion, tone, gross and fine motor coordination, balance, apraxia, and mobility. Motor function can be improved with strengthening, balance, gait training, functional electrical stimulation (FES), robot-assisted movement therapy, constraint-induced movement therapy, and body-weight-supported treadmill training.

Somatosensory deficits have a reported prevalence ranging from 50 to 80 percent of stroke survivors.[9] The production of a movement requires the use of sensory information. Thus, somatosensory deficits affect integration of sensory inputs such as body position in space, texture, weight, and shape of object to be handled, which in turn impacts motor activity planning.[10] Impaired sensation affects self-care and participation in activities.[11]

Sensory assessment should include assessment of different sensations (sharp/dull, temperature, light touch, proprioception, and vibration). Sensory re-education is used to retrain sensory pathways or stimulate latent pathways. This may also involve the use of transcutaneous electrical nerve stimulation. Adaptive or compensatory techniques are also incorporated into the rehabilitation programme.

Post-stroke Cognitive Impairment

The American Heart Association/American Stroke Association (AHA/ASA) defines post-stroke cognitive impairment (PSCI) as "any severity of cognitive impairment, regardless of cause, noted after an overt stroke".[12] PSCI is common and can affect up to 60 percent of stroke survivors within the first year after stroke.[12] Mild stroke survivors are usually discharged after the acute admission as they appear to have recovered functionally in most physical aspects but they may have subtle cognitive impairments that do not appear apparent.[13,14] PSCI is associated with poor outcomes, which includes physical disability, poor sleep, and impaired quality of life (QOL).[12] The exact mechanism of PSCI is unclear but is likely related to preexisting microvascular and neurodegenerative changes.[12]

Currently, there is no gold standard for cognitive screening after stroke. However, several cognitive screening tests have been suggested by the Canadian Stroke Best Practices to screen for PSCI.[15] Locally, clinicians will be familiar with the use of Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MOCA). MOCA is generally recommended over the MMSE especially in subacute phases after stroke, because it has less of a ceiling effect and is more sensitive to mild cognitive impairment.[12]

Treatment of PSCI involves both non-pharmacological and pharmacological interventions. Good control of cardiovascular risk factors, e.g., hypertension, diabetes, hyperlipidaemia, atrial fibrillation is the key. This should include lifestyle modification such as smoking cessation, moderate alcohol intake, healthy diet, and physical activity. Cholinesterase inhibitors such as donepezil, rivastigmine, galantamine, and the N-methyl-D-aspartate (NMDA) receptor antagonist may be considered.[15]

The choice of interventions that are considered for rehabilitation of PSCI should be individualised according to the severity of the impairment and the patient's specific clinical profile. These may also include compensatory strategies and/or direct remediation/cognitive skill training.[15]

Bowel and Bladder Dysfunction

Bowel and bladder dysfunction are common and cause significant distress to stroke survivors. Post-stroke urinary incontinence (PSUI) has an approximate prevalence ranging between 40 to 80 percent.[16] Urinary incontinence is an indicator of stroke severity and is a prognostic indicator of poorer functional outcomes with higher odds of institutionalisation and mortality.[17] The common types of PSUI are urge incontinence, stress incontinence, overflow incontinence, functional incontinence, and mixed incontinence, which is a combination of urge and stress incontinence.[18]

It is important to exclude exacerbating factors such as urinary tract infection, faecal impaction, and medication such as anticholinergic and diuretic. Management of PSUI depends on the type of incontinence and the patient's clinical profile. The bladder scanner can be used to assess for urinary retention. Timed voids and temporary use of external catheters such as urosheath in men or intermittent catheterisation may be required. Behavioural modifications such as bladder retraining and Kegel exercises can help both urge and stress incontinence. In some cases, anticholinergic medications such as oxybutynin chloride may be used to relieve urgency and frequency. An indwelling catheter should only be used where it is absolutely necessary, and all other options have been explored.

The common bowel problems a stroke survivor may experience include: constipation, faecal incontinence, faecal impaction, and incomplete emptying.[19] Constipation is one of the most common bowel dysfunction in stroke survivors, with incidence ranging between 29 to 79 percent.[20] The common risk factors for post-stroke constipation are impaired mobility, dehydration, reduced dietary fibre intake, use of polypharmacy, cognitive impairment, and toilet access.[21,22] Post-stroke faecal incontinence has an estimated incidence ranging between 10 to 40 percent and is associated with age, diabetes, stroke severity, poor mobility, use of anti-cholinergic medications, and dependence of others for toilet access.[21,23] Faecal incontinence is associated with increased risk of institutionalisation and higher mortality.[21] Treatment of constipation incudes addressing dietary fibre intake, hydration, toilet access, review of mediations such as the use of opioids, antacids and iron; and prescription of stool softeners and laxatives. As for treatment of diarrhoea, it is important to exclude faecal impaction and to review medications such as antibiotic and laxatives. In some cases, loperamide may be prescribed.

Post-stroke Depression

Post-stroke depression (PSD) is common after stroke and affects approximately 30 percent of stroke survivors.[24] However, it is often underdiagnosed. The onset of PSD is usually within the first three months after stroke but may occur at any time.[15] The most consistent predictors of PSD are: physical disability, stroke severity, premorbid depression, and cognitive impairment.[25] PSD is associated with poorer functional recovery, impaired QOL, and increased mortality.[25]

Although there are currently no local clinical practice guidelines (CPGs) on screening of PSD, CPGs issued by the Canadian Stroke Best Practices, National Clinical Guideline for Stroke for the United Kingdom and Ireland, and ASA recommend screening for PSD.[15,26,27] Screening could be conducted at various stages throughout the continuum of stroke care, e.g., upon transfer to inpatient rehabilitation unit or during follow-up appointments with GPs.[15]

The diagnosis of PSD is based on the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria, which defines post-stroke mood disorders due to stroke into three sub-types: (i) major depressive-like episode; (ii) mood disorder due to stroke with depressive features; and (iii) mixed mood features.[28] A patient with a diagnosis of mood disorder due to stroke with a major depressive-like episode is characterised by the presence of depressed mood or loss of interest or pleasure along with four other symptoms of depression lasting two or more weeks. A patient with a diagnosis of mood disorder due to stroke with depressive features must have depressed mood or loss of interest or pleasure, as well as at least two but fewer than five symptoms of depression lasting two or more weeks.[28]

Treatment of PSD is usually complex and requires a multi-disciplinary approach including pharmacological and non-pharmacological approaches. For the treatment of PSD, there have been no specific local guidelines regarding the use of any specific antidepressant. The guidelines from Australia, Canada, and ASA also do not recommend any particular antidepressant as there is no evidence that particular antidepressants are superior to others.[15,25,27,29] Based on the Singapore Ministry of Health CPG 2012, SSRIs are generally recommended as the first line for treatment of depression in adults and elderly due to better tolerance, safety in overdose, and favourable risk-benefit ratio.[30] However, SSRIs, especially citalopram, can prolong the QI interval, and this risk appears to be dose-dependent, with increased susceptibility in those with cardiac disease and other QT-prolonging risk factors.[31] This could be a consideration in post-stroke patients, especially those with multiple cardiac co-morbidities. Most side effects of SSRIs are dose-related and are attributed to serotonergic effects.[32] The prescribing physician will need to be aware of the common side effects such as nausea and vomiting, of which nausea may be reduced by coadministration with food. Psychological interventions may be considered.[15,25,26,29]

Post-stroke Pain

Post-stroke pain (PSP) is common after stroke and its reported prevalence can be up to 55 percent.[33] It is often missed because of its variable characteristics and underlying concurrent medical conditions. Patients may also have difficulty reporting the symptoms due to impairments of either cognition or communication after stroke. PSP is associated with cognitive and functional decline, impaired QOL, fatigue, and depression.[33] Risk factors of development of PSP include: older age at stroke onset, premorbid depression, spasticity, reduced upper extremity movement, sensory deficits, and strokes in the thalamus and brainstem.[33] The common types of PSP are: central post-stroke pain, spasticity-related pain, complex regional pain syndrome, and shoulder pain.[34]

Central Post-stroke Pain

Central post-stroke pain (CPSP) is a central neuropathic pain syndrome that occurs after a stroke that is characterised by pain and sensory abnormalities in the affected body parts that correspond to the injured cerebral area.[35] The prevalence of CPSP in stroke ranges between 1 to 12 percent.[35] Development of CPSP is frequently associated with sensory impairment.[35] CPSP can be related to a lesion at any level of the somatosensory pathway of the brain with resultant maladaptive neuroplastic changes within the central nervous system (CNS) causing abnormal sensory perception.[36] The clinical features of CPSP are variable and often similar to the other central and peripheral neuropathic pain syndromes.[35] Although diagnostic criteria for CPSP have not been established, an algorithm to improve the diagnosis has been suggested by Klit et al[35]:

Mandatory criteria for CPSP:

- Pain within an area of the body that corresponds to the lesion of the CNS
- Onset of pain at or soon after stroke
- Confirmation of a CNS lesion by imaging or negative or positive signs confined to the area of the body corresponding to the lesion
- Other causes of pain such as nociceptive or peripheral neuropathic pain have been ruled out or considered highly unlikely

Supportive criteria for CPSP:

- No primary relation to movement, inflammation, or other local tissue damage
- Descriptors such as burning, stinging, pins and needles, dull, electric shocks, although all pain descriptors are applicable
- Allodynia or dysesthesia to touch or cold

Multidisciplinary approach is recommended, which may include psychological treatment (biofeedback, relaxation), mirror therapy, and pharmacological treatment (gabapentinoids, antidepressants).[36]

Complex Regional Pain Syndrome

Complex regional pain syndrome (CRPS) is a clinical syndrome characterized by pain, sensory, motor, vasomotor, and trophic changes.[37] The incidence of CRPS in poststroke patients has been reported to be as high as 50 percent.[38] Post-stroke CRPS has been classified as type 1 due to lack of overt nerve damage.[37] To make a clinical diagnosis of CRPS, the following criteria (Budapest criteria) need to be met[39]:

- The patient has continuing pain, which is disproportionate to any inciting event
- The patient must have at least one symptom in 3 of the 4 following categories:
  a. Sensory: Hyperesthesia and/or allodynia
  b. Vasomotor: Temperature asymmetry and/or skin colour changes

c. Sudomotor/oedema: Oedema and/or sweating changes and/or sweating asymmetry.
d. Motor/trophic: Decreased range of motion and/or motor dysfunction (weakness, tremor or dystonia) and/or trophic changes (hair, skin, nail)

- The patient must have at least one sign in 2 of the 4 following categories:

a. Sensory: hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement)
b. Vasomotor: Temperature asymmetry between limbs and/or skin colour changes/asymmetry
c. Sudomotor/oedema: Oedema and/or sweating changes and/or asymmetry
d. Motor/trophic: Decreased range of motion and/or motor dysfunction (weakness, tremor, or dystonia) and/or trophic changes (hair, skin, nail)

In addition, there is no other better diagnosis that explains the symptoms.

As there is no specific diagnostic test for CRPS, CRPS is diagnosed clinically. However, investigations may be used to exclude other differential diagnoses such as infection, acute rheumatological condition and deep vein thrombosis. Additional objective testing such as triple phase bone scan or a trial of sympathetic ganglion block is not required to make the diagnosis of CRPS but, in some cases, may be used to support the clinical diagnosis.[40]

Treatment of CRPS requires a multi-modal approach, which involves medical, psychological, and rehabilitation treatments. Pharmacological agents such as gabapentin, pregabalin, and amitriptyline have been used to treat CRPS.[41] Studies involving non-steroidal anti-inflammatories (NSAIDs) have shown mixed results. An initial trial of oral corticosteroids may be considered for patients with acute phase CRPS to dampen the inflammatory component believed to be common in the acute phase.[40] However, the optimal dosage remains uncertain.[42] Physical therapy includes graded motor imagery, reduction of oedema, desensitisation, promotion of normal positioning, and increased functional use of the affected limb.[41]

Shoulder Pain

Shoulder pain occurs in 75 percent of stroke survivors and is associated with poorer motor recovery.[43,44] The development of post-stroke shoulder pain is multi-factorial and the aetiology can be related to one of the above causes that were discussed earlier. It could also be due to musculoskeletal causes such as rotator cuff tears/tendinopathy, and subacromial bursitis. In the early stages of stroke recovery, when there is flaccidity and weakness of the upper extremity, the shoulder is prone to injury.[33] Flaccidity of the shoulder muscles predisposes to glenohumeral subluxation, which can result in shoulder pain. Weakness in the surrounding muscles and ligaments of the shoulder joint can also result in instability, which may lead to impingement and rotator cuff tears.[34] Very often, the patient may have multiple pathologies. During the flaccid stage of stroke recovery, protection and stabilisation of the shoulder joint are essential. A gentle range of motion exercises of the shoulder should start as soon as the patient is medically stable.

If there is any clinical suspicion, an X-ray of the shoulder should be considered to rule out an occult fracture or space-occupying lesion. An ultrasound of the shoulder can be used to assess for rotator cuff injury or adhesive capsulitis.

Treatment of shoulder pain depends on the aetiology. For the treatment of shoulder subluxation, the use of arm slings as well as lap boards and arm troughs in wheelchairs can be considered. Pharmacological options such as paracetamol and topical or oral NSAIDs have been used as a first line. FES to the posterior deltoid and supraspinatus have been reported to reduce shoulder subluxation after stroke.[45, 46] For the treatment of rotator cuff disorders and subacromial bursitis, besides the use of NSAIDs, subacromial corticosteroid injection may be considered in some patients to reduce the inflammation so as to allow the patient to participate in rehabilitative and strengthening exercises.[47]

Post-stroke Spasticity

Spasticity is defined as "a motor disorder characterised by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neuron syndrome."[48] Post-stroke spasticity (PSS) may occur in 30 to 80 percent of cases.[49] On one hand, PSS can cause pain, impair function, and affect care as well as QOL. On the other hand, PSS can aid in function: for instance, spasticity of the knee extensor may allow the patient to stand and transfer. Hence, indications for treatment of PSS are to: relieve spasticity-related pain, improve function, hygiene, or ease of care, prevent, or slow the development of contractures and joint deformities, and lastly improve cosmesis.

Treatment involves regular stretching of the limbs to maintain a good range of motion and minimise the risk of contractures; the use of orthosis to achieve prolonged stretching and to prevent further deformity; proper positioning of the affected limb; and recognise and prevent noxious stimuli that may exacerbate spasticity such as constipation, urinary retention, infection, pressure sore, and ingrown toenail. For optimal management of PSS, a comprehensive approach involving both pharmacological and non-pharmacological interventions should be considered. When contemplating pharmacological intervention, it is important to consider the pattern of spasticity (generalised or focal) and the patient's profile. Several oral treatment options for generalised spasticity are available such as baclofen, tizanidine, and gabapentin. For focal spasticity, botulinum toxin injection and alcohol neurolysis can be considered.

ROLE OF REHABILITATION IN STROKE RECOVERY

World Health Organisation defines "rehabilitation" as "a set of interventions designed to optimise functioning and reduce disability in individuals with health conditions in interaction with their environment".[50] Rehabilitation also aims to improve and facilitate natural mechanisms of recovery.

Within hours of acute ischemic injury to the brain, mechanisms of repair are initiated that involve dendritic growth, axonal sprouting, and the formation of new synapses.[51] In early recovery, local processes including resolution of post-stroke oedema, reperfusion of ischaemic penumbra, and reversal of diaschiasis are involved. Diaschiasis refers to the concept that damage to one part of the brain could lead to a loss of function in another area due to its functional and structural connection to the ischemic area.[52]

In the later stages of neurological recovery, reorganisation of cortical networks happens. Studies have demonstrated that stroke induces modulations in local and distant cortical and subcortical activity, alterations in inter-hemispheric interactions, shifts in cortical representational maps, and changes in functional and effective brain connectivity.[53]

Most of the spontaneous recovery happens in the first 3-6 months after the acute neurological event.[54,55] However, recent research has demonstrated that the time window for recovery extends beyond one year post-stroke.[56] Generally, post-stroke patients recover 70 percent of their maximum motor recovery potential three months after a stroke (proportional recovery rule).[57-59] Exceptions to this proportional recovery rule includes damage to the cortico-spinal tract, which results in poorer recovery from impairment.[57,60]

Based on the principles of neural plasticity, there is a need to provide timely, task-specific, multiple repetitions and high intensity stimulation or training.[61] Earlier access to rehabilitation appears to improve functional outcomes, reduce the length of hospital stay, and increase the likelihood of discharge to home.[62,63] Hence, all patients admitted to hospital with acute stroke should have an initial rehabilitation assessment, carried out by rehabilitation professionals, as soon as possible after admission. It is generally recommended to start stroke rehabilitation as soon as the patients are medically stable and are able to participate.[64,65] Most guidelines recommend a minimum of three hours of therapy a day for at least five days a week.[26,64-66]

Large amounts of task-specific practice have been shown to induce neural plasticity and bring about functional improvement.[67] Intensive and high-dose stroke therapy in stroke rehabilitation has been shown to improve functional outcomes.[68] High-dose and intensive training may include the incorporation of constraint-induced movement therapy, robotics, and circuit therapy in suitable patients.[68]

However, caution and individualised clinical judgement are to be exercised especially in severe strokes. The AVERT trial showed that very early, more frequent, and higher dose of mobilisation (VEM) intervention decreased the odds of a favourable outcome three months post-stroke when compared with usual care (UC) group.[69] Of note, the median time between stroke onset and initial mobilisation in both groups were less than 24 hours (22.4 hours in UC group vs 18.5 hours in VEM group).[69] Subsequent analyses from the AVERT study suggested that more frequent but shorter mobility sessions increased the odds of favourable outcome at three months, after accounting for age and stroke severity.[70] The Australian guidelines recommend commencement of out-of-bed activity within 48 hours of stroke onset unless contraindicated.[66]

An "enriched environment", which is an environment that provides greater opportunity for physical activity and motivation, has been shown to facilitate motor recovery as well as neural plasticity in animal studies.[71,72] Patients with stroke who participated in an enriched environmental programme within an inpatient neurorehabilitation unit demonstrated significant improvements in functional and cognitive abilities when compared with patients who participated in routine ward activity programmes.[73] Rehabilitation facilities are ideal enriched environments when they are housed in stimulating and specialised centres managed by a multidisciplinary team of medical professionals.

REHABILITATION SERVICES AND RESOURCES IN SINGAPORE

Stroke management typically starts in an acute hospital for acute stabilisation. In Singapore, upon acute admission, most stroke patients are under the care of acute stroke units, which are managed by a specialised multidisciplinary team of healthcare professionals who provide acute stroke treatment, early recognition of medical stroke-related complications, and early rehabilitation.[74] All patients admitted with an acute stroke should receive an assessment of their rehabilitation needs.[64,65,75,76]

In Singapore, inpatient rehabilitation service is either carried out in acute hospital rehabilitation units or in community hospitals. Rehabilitation facilities are made up of a multidisciplinary team of healthcare professionals who are dedicated to realistic goal-setting and engaging in multimodal disability and impairment assessment, medical management, and functional training. The team addresses the various post-stroke sequelae including sensorimotor and balance impairments, dysphagia, cognitive-communication impairments, mood disorders, visual and hearing impairments, and hemi-spatial neglect. Regular multidisciplinary meetings are carried out to discuss the rehabilitation goals, rehabilitation intervention, functional improvement, discharge planning, and arrangements for outpatient rehabilitation. Recommended feasible goals are planned together with the patients and their caregivers to

enable a seamless transition to outpatient rehabilitation, aiming for maximum independence upon discharge.[77]

Outpatient rehabilitation can be conducted either at the hospital outpatient clinic or at the day rehabilitation centre. Home therapy can be arranged for those who have difficulties getting out of their house. Government subsidies for community rehabilitation services are available for patients who are financially needy. There are various local support groups for both post-stroke patients as well as for their caregivers, e.g., Singapore National Stroke Association (SNSA), Stroke Support Station (S3), and Aphasia SG. For post-stroke patients who have the potential to return to work, referral to employment/vocational rehabilitation programmes such as SPD and ABLE can be considered.

Stroke survivors who wish to resume driving after a period of rehabilitation should first be assessed by a specialist such as a neurologist on whether they are medically fit to drive in accordance with the Singapore Medical Association's guidelines for driving, e.g., there is no history or recent episodes of epilepsy, no mental or coordination impairment that is severe enough to interfere with control of the vehicle, and no significant visual disturbance such as diplopia. This will be followed by an assessment by a certified driver-assessor therapist. The driving assessments are available via doctors' referral at the various local hospitals: Driving Assessment and Rehabilitation Programme – DARP by Tan Tock Seng Hospital; Driving Assessment and Rehabilitation Service – DARS by Ng Teng Fong General Hospital; and SGH Driving Evaluation and Rehabilitation Service – SGH DrivERS by Singapore General Hospital. The certified driver-assessor therapist is often an occupational therapist who will perform both off-road and on-road assessments to determine whether the stroke survivor is able to operate a motor vehicle safely. Upon completion of the above assessments, the stroke survivor will need to be reviewed by the specialist to advise whether he/she can resume driving safely and to obtain the specialist's endorsement on the driving report.

CONCLUSION

With the advancements in acute stroke management as well as an ageing population, the number of stroke survivors is expected to increase. A comprehensive, well-organised, and integrated post-stroke care plan includes optimisation of cardiovascular risk factors and chronic diseases as well as management of the common post-stroke complications while working closely with the rehabilitation teams. To achieve the best functional outcomes in stroke survivors, it is important for the GP to identify their rehabilitation needs and able to readily re-refer them to the rehabilitation teams and the various specialists when necessary for further assessment and intervention.

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LEARNING POINTS

- **Stroke remains one of the leading causes of disability in Singapore. A comprehensive post-stroke care plan includes optimisation of cardiovascular risk factors and chronic diseases as well as management of the common post-stroke complications while working closely with the rehabilitation teams.**
- **Most of the spontaneous recovery happens in the first 3-6 months after the acute neurological event. However, recent research has demonstrated that the time window for recovery extends beyond one year post-stroke.**
- **Based on the principles of neural plasticity, it is essential to provide timely, task-specific, multiple repetitions and high-intensity stimulation or training. All patients admitted to hospital with acute stroke should have an initial rehabilitation assessment, carried out by rehabilitation professionals, as soon as possible after admission. Caution and individualised clinical judgement are to be exercised especially in severe strokes.**
- **Common post-stroke complications such as post-stroke pain, spasticity, post-stroke cognitive impairment, post-stroke depression, and bowel and bladder dysfunction should be recognised and managed to improve function and quality of life of the stroke survivors.**