

## **Guide for Stroke Rehabilitation planning**

**Implementation plan- Ravi will make the proforma into a redcap survey.**

**Expect 100 data sets. Data will have proforma details are fields, along with modules selected and value of each in that case.**

**After obtaining 100, we can publish the work, and Ravi is free to work on other care plan topics**

### **GUYS we have to work on this part**

#### **4. Managing the Stroke Rehabilitation Triage Process**

##### **Predictors of Functional Outcomes**

Best predictors of functional recovery and discharge home are age and initial stroke severity (level 3 evidence)

#### **5. The Efficacy of Stroke Rehabilitation**

##### **Acute Care**

acute stroke care= reduction in death/dependency/ institutionalization, ≠ reductions in mortality/ length of stay vs alternative care. (Level 1a evidence)

Acute stroke care≠ reduction in functional disability vs alternative interventions. (Level 1a evidence)

##### **Combined Acute and Rehabilitation Stroke Units**

Combined acute and rehabilitation stroke units= reductions in death/dependency, institutionalization, and length of stay, ≠ reduced mortality vs general medical wards. (Level 1a evidence)

Combined stroke units= improved functional outcome vs general medical wards. (Level 1a evidence)

##### **Subacute Rehabilitation Units**

Specialized, interdisciplinary subacute rehabilitation= reductions in mortality and death/dependency, ≠ reduced institutionalization or length of stay, vs conventional care on a general medical ward. (Level 1a evidence)

For Severe stroke patients, specialized stroke rehabilitation reduces mortality but does not result in improved functional outcomes or reduced institutionalization compared to conventional care. (Level 1a evidence)

Patients with moderately severe stroke, specialized rehabilitation improves functional outcomes but does not reduce mortality compared to conventional care. (Level 1a evidence)

Patients with mild stroke, specialized rehabilitation does not improve functional outcome or reduce mortality compared to conventional care. (Level 1a evidence)

Patients with severe or moderately severe stroke who receive treatment on a stroke rehabilitation unit have a lower risk of being dependent or dead/dependent compared with patients who receive little or no rehabilitation. (Level 1b evidence)

### **Mobile Stroke Teams**

mobile stroke teams do not reduce mortality, combined death/dependency, institutionalization, or length of stay. (Level 1a evidence)

### **Overall**

Specialized stroke care is associated with reductions in the odds of mortality, combined death/dependency, institutionalization, and length of stay. (Level 1a evidence)

## **6. The Elements of Stroke Rehabilitation**

### **Care Pathways in Stroke Rehabilitation**

Integrated care pathways are not associated with long term (>6 months) reductions in death or dependency, but may improve these outcomes in the short term (<3 months), when compared to conventional care. (Level 1a evidence)

Integrated care pathways are not associated with reduced length of stay, readmission rate, or complication rate, when compared to conventional care.

Greater adherence to care guidelines, treatment protocols, and organizational quality is associated with better clinical outcomes. (Level 1a evidence)

### **Timing to Stroke Rehabilitation**

Earlier admission to rehabilitation results in improved overall functional outcomes. (Level 1a evidence)

Very early mobilization (VEM) post stroke (within the first 24 hours) results in improved outcomes when there are more frequent short in duration out-of-bed sessions and that VEM results in poorer outcomes when early mobilization session are more prolonged. (Level 1a evidence)

### **Intensity of Therapy**

Greater intensities of physiotherapy and occupational therapy results in improved functional outcomes. (Level 1a evidence)

Amount of therapy needed to result in a significant improvement in motor outcomes is 17 hours of physiotherapy and occupational therapy over a 10 week period of time. (Level 1a evidence)

Additional caregiver-supported therapy results in improved functional outcomes compared to conventional therapy alone. (Level 1a evidence)

There is conflicting evidence that greater intensity of speech language therapy results in improved aphasia outcomes.

### **Durability of Rehabilitation Gains**

greater functional improvements are made in specialized stroke units vs general medical units in the long term. (Level 1a evidence)

Functional outcomes achieved through stroke rehabilitation are maintained for up to one year post stroke. (Level 1a evidence)

By five years post-stroke functional outcomes plateau and may decline. By ten years, overall functional outcome scores significantly decline although it is unclear to what extent the natural aging process and comorbidity may contribute to these declines. (Level 1b evidence)

## 7. Outpatient Stroke Rehabilitation

### **Early Supported Discharge**

Stroke patients with mild to moderate disability, discharged early from an acute hospital unit, can be rehabilitated in the community by an interdisciplinary stroke rehabilitation team and attain similar or superior functional outcomes when compared to patients receiving in-patient rehabilitation. (Level 1a evidence)

Cost associated with early-supported discharge is lower when compared to usual care; however, savings are generally not dramatic or consistent across the studies. (Level 1a evidence)

### **Outpatient Rehabilitation Provided Within the First 6 Months of Stroke Onset**

Additional outpatient therapy improves performance of ADLs. (conflicting Level 1a evidence)

### **Outpatient Rehabilitation Provided at Least One Year Following Stroke**

regarding the association between home based therapy for chronic stroke survivors and improvements in performance on ADLs and mobility. (conflicting Level 1a evidence)

### **Rehabilitation in the Home or in the Hospital**

home-based and hospital-based outpatient stroke rehabilitation programs are equally effective in achieving modest gains in ADL following inpatient rehabilitation. (Level 1a evidence)

## **The team**

**Dr. Ravi Sankaran**

**Dr. Unnikrishnan Ramachandran**

**Dr. Nitha Jayaram**

**Dr. Noufal Ali**

**Dr. Shadiya Beegum**

**Dr. Vidya G**

---

## IAPMR Kerala chapter

---

## Foreword

This evidence based 'Guide for Stroke rehabilitation planning' is the result of a long felt need to have uniformity and standardization among Physiatrists and also the way to update our individual stroke rehab planning. It is based on the EBRSR by Dr Robert Teasell MD, a Canadian Physiatrist. I first came across one of the early versions of this and was impressed by how many topics of concern to me it hit in one go. As I read deeper I realized it was tailor made for a western audience. Some of the things mentioned were not feasible for our set up. Another problem I found was 'How did one know when to apply what?'. This is an answer to those issues

We all know rehabilitation is vast and complex. A cookie cutter approach has little value in something like neuro-rehabilitation, yet it is the need of the hour. Our allied medical specialties have guidelines and protocols. As a result they can publish and grow. Our allied paramedics have very little regularity of practice. We sit somewhere in between. This is a means for us to improve ourselves, through our patients.

This by no means is an end product and needs to be revised periodically based on updated recommendations as they come, and user feedback based on experience at the ground. It would be very fruitful to see how this can be adapted and used at all levels of rehab care and at the community level. A uniform approach opens up possibilities of multicenter collaborative research in the field of Stroke rehab, and a chance to collate data on stroke rehab in our state.

The authors would like to thank the IAPMR Kerala chapter President Dr. Muralidharan P.C, Secretary Dr. Selvan and the executive committee for their support in embarking on this project. We thank all the senior physiatrists who had kindly reviewed the guideline and gave their valuable input which has helped us refine this.

It is still a work in progress.

*A journey of thousand miles starts with a step.*

## **Mission**

To develop a comprehensive guide for Stroke rehabilitation planning, based on available evidence, which is user friendly, and applicable to different rehabilitation settings.

## **Vision**

Establish uniform standards of rehabilitation care across different centers in the state.



## **Table of contents**

Post-stroke cognitive disorders

Aphasia and apraxia

Depression

Dysphagia

Perceptual disorders

Upper extremity dysfunction

Lower extremity dysfunction

Hemiplegic shoulder pain

Medical complications

Severe stroke

Young stroke

Community reintegration

Nutrition post stroke

Part 2: Comprehensive care

Plan integration

Managing triage process

The elements of rehabilitation

Efficacy of rehabilitation

OPD rehabilitation

## **Introduction**

Stroke is one of the leading causes of acquired motor dysfunction around the world. Around 800000 people are affected every year in US. Unfortunately such statistics are not available for India, and a systematic review done in 2017 highlighted the lack of quality epidemiological data and the need to have state and central level initiatives to collect such data.<sup>1</sup> Advances in acute care management has ensured that many more patients survive and are left with varying degrees of deficits. The advent of newer interventions in Neurology like the thrombolytic therapy has improved the survival and outcome of stroke. All these make it necessary for Physiatrists to have a common rehab guideline, which needs to be updated regularly.

Stroke rehabilitation is a multidimensional process, considering the complex interplay of various impairments in brain functions, which affect the functional independence, activities and participation of stroke patients as productive individuals in society. Even though stroke rehabilitation is established at many centers at different levels, there is a lack of uniformity in approach. Only a small proportion of stroke patients have access to comprehensive rehabilitation. There is also a lack of proper follow up of these patients once they are discharged from the tertiary level centers.

Comprehensive rehabilitation centers have a complete team with Physiatrist as the lead. They help streamline the complex process, and also in preventing possible complications right from ICU stage to discharge, which could otherwise result in unnecessary delay in rehabilitation and increase in costs. The importance of this cannot be stressed. Why? There is increasing awareness regarding health care costs optimization, length of patient stay etc. from the patient/ party side

Quantifying the impact of stroke on a patient's functioning based on the biopsychosocial model of ICF is what needs to be addressed. Impairments in bodily structure or function, Activity restriction, and interaction of the contextual factors- environment and personal factors determine participation in society. Measures of body function tend to be more objective, easier to define, and easier to measure compared with other levels of the World Health Organization's ICF, but these measures have less relevance to a patient's participation and independence. While many outcome measures cover impairment and activity limitations, participation restriction is a challenge to measure. For one, this aspect of community integration, and vocational rehabilitation in our settings can best be described as unorganised and chaotic. This again points to the need for step down levels of care and follow up.

Although many scales are described in literature, we may need to zoom in on the one most appropriate for our settings (i.e. Stroke impact scale)

## **Levels of Care**

The developed world boasts of an organised level of rehab care in stroke, where after acute care further rehabilitation management continues till goals are attained. Thereafter the patient can be managed and followed up at the secondary levels and also at the community level.

In Kerala, such is absent both in the private sector or in Government PMR centers. Follow up and treatment continuation is sub optimal at community level once the patient is discharged.

So while we continue our efforts to standardise and improve tertiary level care in stroke rehabilitation, it is also important that we envisage a referral and community level follow up system for the stroke patients. Use of telemedicine could be one immediate option at hand.

With the increasing burden of lifestyle diseases, we are likely to witness an increasing number of vascular events like CVA, with a resultant increase in people with compromised abilities. In the background of the challenges caused by recurrent pandemics like the SARS COVID, this assumes more significance and points to an urgent need to establish comprehensive rehabilitation centers.

This rehab planning guide is primarily based on the modules of EBRSR, 18<sup>th</sup> edition, with inputs from AHA stroke guideline. What we have attempted is to identify deficits and wherever possible define a metric for that deficit. Recommendations are then classified as “Recommended”, “Neutral”, and “Not recommended” based on strength of evidence and linked with the metric. We have used the Oxford EBM pyramid for stratification of available data.

Part one shows a quick summary of all the modules with the deficits and the recommendations. Part two gives in detail the deficits, metrics and the recommendations. Appendix lists the relevant scales used, with references at the end. Having a quick reference short list of important recommendations, will allow the busiest of Psychiatrists to fully assess rehab needs within an hour. It will also be an instant reference for those new to neuro-rehabilitation. More seasoned clinicians are advised to fill the proforma and go directly to the modules.

The oversimplified table of care neglects much of the complexity of rehabilitation. For this reason those interested in seeing more options, learning more of the science/evidence, or just interested in general are encouraged to review the full modules in Part two.

## **Part One**

### **Snapshot of the recommendations**

## Proforma

Name              Age              Profession              Dominant hand

Stroke location              Stroke duration

Co-morbidities

Stroke etiology: Unknown/ Ischemic/ Hemorrhagic/ Uncommon

ASPECTS              NIHSS at \_\_\_ months              mRS-

## Deficits and treatment options listed according to domains involved

### Post stroke cognitive disorders

To use the below table first do a MOCA or MMSE. Select treatment based on the deficits detected

Domains affected	Recommendations
Executive function	Citicoline Combined Aerobic and resistance exercise
Attention and Orientation	Selegiline Citicoline Computer based training Attention specific training Combined Aerobic and resistance exercise
Memory	SSRI Computer training TDCS
Vascular Cognitive impairment	Donepezil Galantamine Nimodipine Rivastigmine
General Cognition	rTMS

### Aphasia and Apraxia

To use the table below first do the following: PASS and Western Aphasia Battery. With PASS rate severity as Mild / Moderate/ Severe. Select treatment based on the deficits detected.

Domains affected	Recommendations

Speech	Intensive Language-Action Therapy PACE- Promoting Aphasic Communication Effectiveness Group therapy Melodic Intonation Therapy
Comprehension, communication	Volunteer-Facilitated Speech and Language Therapy Training conversation partners Group therapy
Communication, Language, Word retrieval	Computer-based aphasia therapy Telerehabilitation and Speech and Language Therapy Filmed Language Instruction Constraint-Induced Therapy for Aphasia
Word retrieval	Semantic and phonological cues Melodic intonation therapy
Naming	Repetitive Transcranial Magnetic Stimulation (rTMS) Transcranial Direct Current Stimulation (tDCS) Theta burst stimulation
Medications – Aphasia	Amphetamine Dextran-40 Donepezil Memantine Galantamine
Ideomotor apraxia	Strategy training

### Depression in Stroke

We don't suggest a screening scale for this condition. The clinician should be able to detect it with history and exam. Select treatment based on the deficits detected.

Domains	Recommendation
Depression and cognitive improvement	Escitalopram Nortriptyline
Depression and Functional recovery	Fluoxetine Nortriptyline Methyl Phenindate
General Pharmacotherapy for Depression	Fluoxetine Citalopram Escitalopram Nortriptyline Mianserin Milnacipran Duloxetine Reboxetine Mirtazapine Methylphenidate Vitamin B therapy administered over a long period
Non pharmacological Management of depression	Motivational interviewing Active care management program

	Psychosocial-behavioural therapy in combination with antidepressants Art therapy Goal achievement program Transitional care program Specialized, therapeutic exercise program Combined speech and orofacial therapies Hyperbaric oxygen therapy Repetitive transcranial magnetic stimulation Acupuncture
Anorexia + Depression	Mirtazipine
Neuropathic pain	Venlafaxine Duloxetine

## Dysphagia

The advised screening tool is GUSS. Select treatment based on the deficits

<b><i>Domains affected</i></b>	<b><i>Recommendations</i></b>
Aspiration Pneumonia	Dietary Modifications – thickened fluids Swallowing Treatment Programs Non-Oral Feeding
Prevention of Pneumonia Post-Stroke	ACE inhibitors Metoclopramide Cilastazol

detected.

## Perceptual disorders in Stroke

It is up to the clinician to detect perception disorders. They may be visual/ tactile/ spatial neglect/ hemianopsia. Select treatment based on the deficits detected

<b><i>Domains affected</i></b>	<b><i>Recommendations</i></b>
Visual neglect and hemianopsia	Visual scanning Feedback strategies Prism glasses
Tactile neglect	Sensory Stimulation Intervention Feedback strategies
Spatial neglect	Limb activation Feedback strategies Vestibular Galvanic Stimulation

## Upper extremity

How to use this: quickly get a Brunnstrom and Ashworth score. Find out the appropriate goals using the table below. Select treatment based on the deficits detected.

Performance		Range for identifying acceptable goals				
BRS	MAS	Improve grip	Reduce contracture	Manage problematic hypertonia	Improve reduced PRoM	Improve poor function
1	0	X x	X x	X x	X x	X x
2	1	Ok	Ok	Ok	Ok	Ok
2	1+	Ok	Ok	Ok	Ok	Ok
2	2	Ok	Ok	Ok	Ok	Ok
2	3	Ok	Ok	Ok	Ok	Ok
3	3	Ok	Ok	Ok	Ok	X x
3	4	Ok	Ok	Ok	Ok	X x
4	2	Ok	Ok	Ok	Ok	Ok
4	1+	Ok	Ok	Ok	Ok	Ok
4	1	Ok	Ok	Ok	Ok	Ok
4	0	Ok	Ok	Ok	Ok	Ok
5	1+	Ok	Ok	Ok	Ok	Ok
5	1	Ok	Ok	X x	X x	Ok
5	0	Ok	Ok	X x	X x	Ok
6	1	Ok	Ok	X x	X x	Ok
6	0	Ok	X x	X x	X x	Ok
7	0	Ok	X x	X x	X x	Ok

Domains affected	Recommendations
To improve grip	linked tasks add light objects for them to manipulate
Contracture	Splinting Positioning Stretching
Spasticity	Splinting Positioning Stretching Antispastic agent Botulinum toxin Functional retraining of muscles
PRoM	Splinting Positioning Stretching
Function	CIMT Repetitive task training FES



### Lower extremity (Gait and Balance)

To use the below table first determine the Functional Ambulation Category for your patient. Select treatment based on the deficits detected.

<b>FAC</b>	<b>Recommendations</b>
0	Neater wheelchair
0-3	FES/ TENS/ NMES/ IFT/ Thermal stimulation/ Local vibration Ankle/foot sensorimotor stimulation
0-4	Ankle Foot Orthosis (Standard and Dynamic) Hybrid Assistive Limb
0-5	Action observation with gait training Caregiver- Mediated Programs Mirror therapy Strength and Resistance Training Combined resistance and aerobic training
2-4	Robotic walker
3-4	Taping in combination with rehabilitation
3-5	Aquatic therapies Community- and home-based exercise programs

<b>Domains affected</b>	<b>Recommendations</b>
Motor Function (including Gait & Balance) Therapeutic Approach  Medications	Compensatory and restorative approaches  Methylphenidate Droxidopa Citalopram Fluoxetine Cerebrolysin dalfampridine
Contracture	Splint and tilt table Both prevent ankle contracture
Spasticity	Botulinum toxin-A/ combined with AFO Thermocoagulation Phenol and ethyl alcohol(Botox unaffordable pts) Baclofen Tolperisone

### Hemiplegic shoulder pain

To use the table below determine VAS and MAS. Select treatment based on the deficits detected.

<b>Domains affected</b>	<b>Recommendations</b>
General measures	Avoid pulling on that limb Proper Positioning
Hypertonia	Positioning

	ROM exercises Icing Botulinum toxin
Hypotonia	Positioning pillow elevation sling strapping, Physical Modalities - TENS, NMES, FES, High voltage Galvanic stimulation
Contracture	Stretching, ROM exercises
Pain <ul style="list-style-type: none"> <li>• MSK pain</li> <li>• Central Pain</li> <li>• CRPS/ RSD</li> </ul>	Medications Modalities Acupuncture SSNB Intra articular steroid Hyaluronic acid  Medications CBT Opioids NS referral  Medications Modalities Acupuncture SSNB Stellate block

## **Part Two**

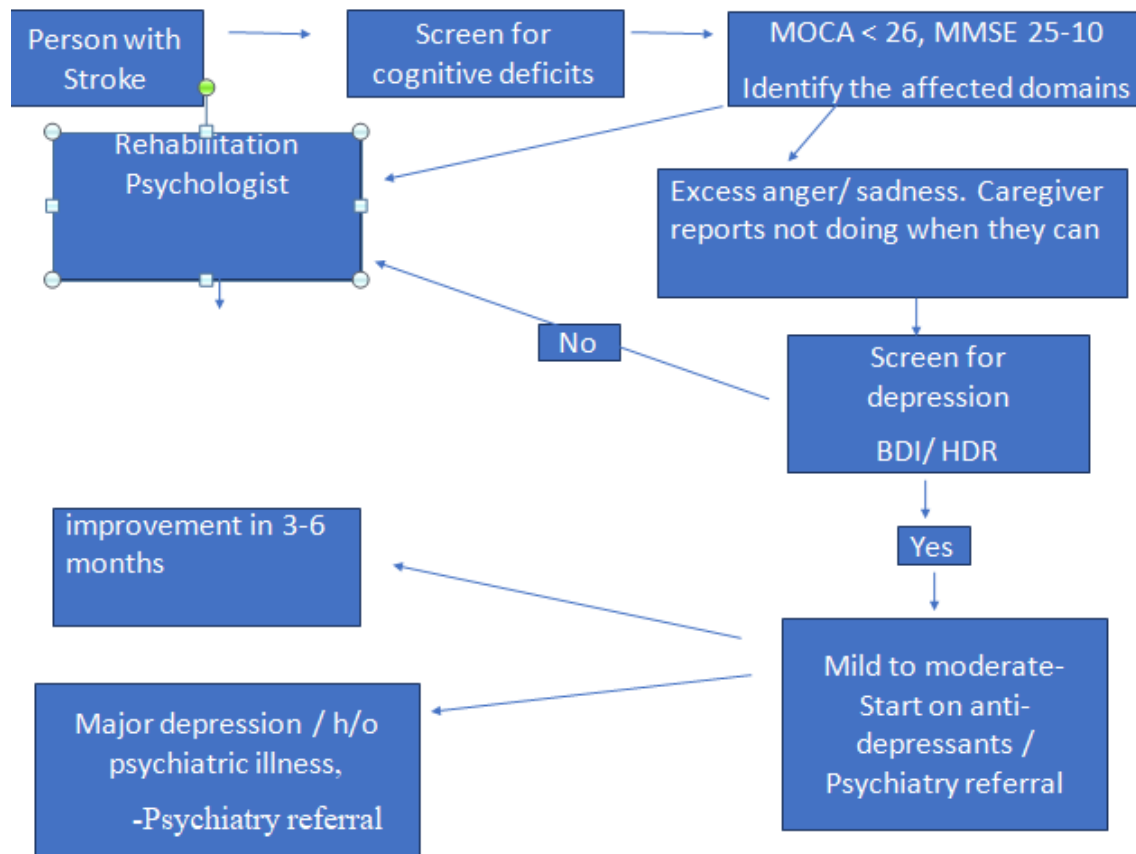
### **Complete modules with recommendations**

## Post Stroke Cognitive disorders

- About 30 % of the of people experience depression after stroke.<sup>2</sup>
- The cognitive problems post stroke varies from 30% -80%.<sup>3</sup>
- Furthermore, cognitive impairments continue to deteriorate in 11% of stroke survivors during the first year after stroke.<sup>4</sup>
- The main domains which are affected are Executive/ visuo-spatial, memory, attention, orientation. After a stroke, as many as 55% of people have deficits in episodic memory, up to 40% show deficits in executive functions, and 23% of people have language deficits.<sup>5</sup>
- More than 60% of stroke survivors reports mild to severe cognitive impairment up to 10 years after stroke.<sup>6</sup>
- Guidelines for neuro-rehabilitation are mainly focused on compensational strategy training<sup>7</sup>. These strategies do not aim to restore brain functions (i.e., restitution), but aim to compensate for the lost function by using remaining intact functions. Some postulate that depending on the amount of remaining connectivity, different types of intervention are needed notably restitution or compensation. Mildly damaged brain networks might reconnect by everyday life activities, and no special intervention is necessary. Severely affected brain networks may not be able to reconnect at all. Therefore, in severe cases compensational interventions are required that make use of preserved networks. For moderately affected networks, restitution-based interventions may be needed to stimulate the relevant parts of the impaired network.
- Cognitive deficits are a major contributor to disability and decreased quality of life after stroke.<sup>8</sup>
- The presence of cognitive impairment has been associated with institutionalization and increased caregiver burden and health care costs.<sup>9</sup>
- Dementia is a strong predictor of mortality after stroke.<sup>10</sup>
- Cognitive recovery of stroke patients has the potential to significantly reduce the burden of this disease.

### A. Workflow

Screening all stroke patients for identification of cognitive deficits forms the first step. In the presence of cognitive impairment the criteria below can be used to decide on whether to see if rehabilitation strategies, maintenance therapy or referral is needed.



### Prerequisites/ Inclusion criteria for rehabilitation

- Any abnormal cognitive parameters suspicion
- MOCA < 26
- MMSE - 25- 10
- No active participation in therapy
- Family or self-complaints of cognitive impairment
- No severe dementia – MMSE < 10

## Clinical history and exam/ baseline measures

- Duration from stroke (acute/ sub acute/ chronic)
- Pre-existing Psychiatric condition, evidence of complex stroke (CADASIL, other neuro-degenerative conditions)
- Family and Social support system
- Occupation and feasibility to return to it
- Screening- Hamilton depression rating scale
- Domain specific histories – correlation with anatomical lesion – Disinhibition, personality change, Apathy, Not cooperating with therapy, low mood and motivation

## B. Decision Tree

- Treatment plan- Patient led, Psychiatrist guided
- Goal Attainment Scale (GAS) Approach

- Over how much time? (3 months usually)
- What do we do until then? Titrate medications and therapy
- What next if they do or don't achieve? No improvement – compensation

### Executive Functions

MOCA - Visuospatial – Trail, copy cube, Clock drawing - ..../5

#### Recommended

Treatment	Conclusion
Citicoline	Improves Attention, executive function, orientation (1a)
Exercise CARET	Improves Attention and Executive functions (2)

#### Neutral

Treatment	Conclusion
Computer training	Inconclusive, useful +/- (1b)

#### Not recommended

Treatment	Conclusion
rTMS to the left DPC	Improves executive function (4)

### Attention And Orientation

MOCA – Attention /Orientation ....6/.../6 ( Digit span)

Subtraction, Day ,place,city,date ,month

#### Recommended

Treatment	Conclusion
Selegeline	Improves Attention, Executive Function (1b)

Attention specific training	Improves auditory and visual attention (1b)
Exercise CARET	Improves Attention and Executive functions (1a)
Citicoline	Improves Attention, executive function, orientation (1a)
Computer training	Improves attention and working memory (1b)

#### Not recommended

TDCS	Attention, concentration, figural memory, logical reasoning, reaction behavior (4)
------	--

### MEMORY

MOCA – Delayed recall+ Attention 11/30 ( Digit span, A marking, Subtraction)

#### Recommended

Treatment	Conclusion
SSRI	Improves Delayed/immediate memory (1b)
Computer training	Improves attention and working memory (1b)
TDCS	improvements in working memory and attention (2)

#### Neutral

Treatment	Conclusion
Group based interventions	Improves subjective everyday memory and functional goal attainment (2)
Cognitive rehabilitation for memory	Improves subjective measures of memory in the short term. (1a)

**Vascular Cognitive Impairment / Vascular Dementia** - multi-infarct dementia, single strategic infarct dementia and subcortical dementia

Scale - change in problem behaviour, ADL

#### Recommended

Treatment	Conclusion
Donepezil	Improves cognition (1a)
Galantamine	Limited data (1a)

Nimodipine	Cognition and global functioning (1b)
Rivastigmine	Cognition generally improved in mixed dementia (older individuals and medial temporal atrophy)

#### Neutral

Treatment	Conclusion
Memantine	Murine studies – promising- relieve the impaired memory and decrease the neural lesions caused by cerebral ischemia (?)
Citicoline	Improves memory and behaviour not attention (Cochrane review) Mixed evidence (1a)

General Cognition	
Recommended	
Treatment	Conclusion
rTMS	Lack of supportive evidence

## Aphasia and Apraxia

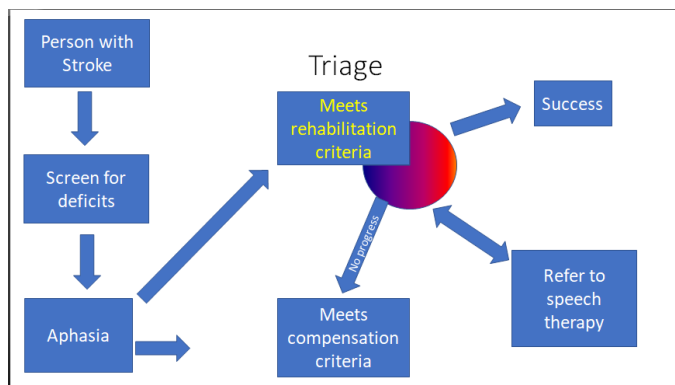
### Problem statement

Patients with aphasia from acute disorders, such as stroke, generally show spontaneous improvement over days, weeks, and months. In general, the greatest recovery occurs during the first 3 months, but improvement may continue over a prolonged period, especially in young patients and in global aphasics (Pashek G.V., and Holland A.L.: Evolution of aphasia in the first year post-onset. Cortex 1988; 24: pp. 411-423)

Aphasia recovers best when left hemisphere areas, either in the direct language cortex or in adjacent areas, recover function. Right hemisphere activation seems to be a “second best” type of recovery.<sup>11, 12</sup>

A community-based study of primary caregivers of stroke patients showed that those looking after aphasic patients (compared with those looking after non-aphasic patients) had significantly increased caregiver task difficulty, caregiver depressive symptoms, and, more negative stroke-related caregiver outcomes.<sup>13</sup> Their final dataset included 57 trials comprising 3002 subjects. The headline finding is that speech and language therapy (SALT) compared with no SALT improves functional outcomes, but the SMD is small at 0.28. The largest effect size is for studies focusing on treating speech production (1.28).<sup>14</sup>





## Eligibility

Rehabilitation program criteria: Aphasia / Dysarthria.

Best results in first three months, anywhere up to 1 year from event otherwise

Maintenance program criteria: no progress for max 6 months, > 1 year duration from stroke

Triage: Mild/ Moderate/ Severe Aphasia, use scale below

Three representative domains of the Progressive Aphasia Severity Scale (PASS) (Sapolsky et al., 2010)

PASS Domain	0 normal	0.5 questionable/very mild impairment	1 mild impairment	2 moderate impairment	3 severe impairment
<b>FLUENCY:</b> Degree to which speech flows easily or is interrupted by hesitations, fillers, pauses; reduced fluency is associated with decreased phrase length and words per minute (WPM)	Normal flow of speech.	Speech contains occasional blank pauses or use of fillers (umm); reduced WPM and/or phrase length.	Speech is in short phrases, interrupted with pauses or groping for words but there are occasional runs of fluent speech.	Dysfluencies in most utterances; phrase length rarely exceeds three words.	Severely dysfluent speech; phrase length rarely exceeds one word. May not speak.
<b>SYNTAX AND GRAMMAR:</b> Use of word forms (run, ran), functor words (the, an), and word order when forming phrases and sentences in most used modality (speech or writing)	No difficulty in the use of grammar and syntax.	Occasional agrammatism or paragrammatism (i.e., odd sentence structure such as, "I my car drive in your house."); may complain it is effortful to combine words into phrases or sentences	Frequent agrammatism; sentence structures are simple, frequent misuse/omission of grammatical words or morphology	Utterances contain mostly content words with rare use of syntactic word groupings, functor words, or morphological markers	Single word utterances or no speech/writing.
<b>SINGLE WORD COMPREHENSION:</b> Ability to understand spoken or written single words	No difficulty understanding single words in conversation or testing.	Occasional difficulty understanding low frequency words (e.g., cork); may question the meaning of words (e.g., "What is a _?").	Displays lack of word comprehension several times in a brief conversation but able to carry on reasonably meaningful conversation.	Understands some high frequency and/or familiar words. Questions the meaning of many words in conversation.	Minimal comprehension of single words.

	Conclusion
Language Therapy	Intensive Language-Action Therapy significantly improves symptoms of aphasia (1a)
	Promoting Aphasic Communication Effectiveness is an effective treatment for aphasia...(2)
	19.3hrs of speech therapy program may improve performance on comprehensive language assessments compared to standard therapy (6.9hrs). (1b)
Volunteer-Facilitated Speech and Language Therapy	Volunteers can provide speech and language therapy and achieve similar outcomes in terms of comprehension and communicative ability when compared to speech- language pathologists. (1b ,2)
Group Therapy	Group treatment may improve communicative ability (1a)
	Immediate group therapy may improve language impairment when compared to deferred group therapy (2)
Training Conversation/Communication Partners	Training conversation partners to acknowledge and reveal competence of individuals with aphasia may enhance the conversational skill of both parties when compared to delivering an informative video presentation to conversation partners (1b)
Computer-Based Treatments	Computer-based aphasia therapy may improve word retrieval ability in the short-term (1a)
	Computer-based aphasia therapy may improve communicative ability and language function when compared to no treatment (limited level 2)
Reading comprehension focused computer-based treatment	may improve communicative ability and language skills assessed at the impairment level when compared to a cognitive rehabilitation focused computer-based treatment (2)
Telerehabilitation and Speech and Language Therapy	Remote assessment when compared to face-to-face assessment; however, preliminary findings suggest that the interventions are comparable (limited level 2)
	Teleconferencing for remote speech and language treatment is comparable to face-to-face treatment in individuals with aphasia following stroke. (limited level 2)
	Filmed Language Instruction There is level 1b evidence that supplementary-filmed programmed language instruction combined with speech therapy may be as effective as traditional speech therapy for aphasia recovery post-stroke

melodic intonation therapy	may improve responsive speech when compared to no language treatment (limited level 2)
Constraint-Induced Therapy for Aphasia	There is conflicting and level 1a evidence for the effectiveness of constraint-induced aphasia therapy (CIAT) on language performance, as compared to conventional treatment or placebo. There is limited level 2 evidence that CIAT administered by experienced therapists may be as effective as CIAT administered by trained lay persons for aphasia recovery
	CIAT may be as effective as the PACE treatment for the improvement of confrontational word retrieval in individuals with aphasia or other language disturbances caused by stroke (limited level 2)
Music Based Therapies	Melodic intonation therapy may be as effective as standard language therapy for the improvement of word retrieval ability or performance on comprehensive language assessments (1b and limited level )
Repetitive Transcranial Magnetic Stimulation (rTMS)	rTMS may improve performance on comprehensive language assessment as well as on tests of naming abilities. (level 1a)
theta burst stimulation	may improve naming abilities among individuals with aphasia as compared to sham stimulation. (limited level 2)
Transcranial Direct Current Stimulation (tDCS)	Anodal tDCS applied over the left frontal cortex is associated with improved naming performance in individuals with chronic post-stroke aphasia. ( level 1a and limited level 2)
	tDCS in combination with naming therapy is more effective than naming therapy alone in improving aphasic symptoms. (level 2)
	high definition tDCS is more effective than traditional sponge tDCS in treating aphasia. (level 2)
Gesture Training	Gesture training may be associated with improvements in ideomotor apraxia extending to activities of daily living. These effects may be sustained for at least 2 months following the end of treatment (1b)
Unilateral Forced Nostril Breathing	unilateral forced nostril breathing may improve anxiety and language (limited Level 2)
Specific Treatment for Word-Retrieval Deficits	Both semantic and phonological cues may aid in lexical retrieval abilities; however, it is unclear whether there is a difference between the uses of the two types of cues (1a limited level 2)
Specific Treatment for Global Aphasia	Speech and language therapy may be helpful for individuals with global aphasia post-stroke (limited level 2)

Treatment of Ideomotor Apraxias and Ataxia	Strategy training is effective in the treatment of apraxias post-stroke. Training effects may include improvement in performance of activities of daily living that appear to be sustained over time (1a)
Amphetamines	Dextroamphetamine may improve aphasia recovery when combined with speech and language therapy (1b)
Piracetam	may be helpful for arm and leg motor movement, and the rate of perfusion compared to placebo (1b)
Dextran-40.	There is level evidence that Dextran-40 may result in better outcomes than the non-treatment control (1b)
Donepezil	donepezil may produce some improvement on global language function, this improvement is reported only during active treatment and may not extend to everyday communication ability. (1b )
Memantine	memantine therapy effective in treatment of chronic aphasia. Combination therapy using constraint-induced language therapy and memantine may provide additional benefit than either therapy used independently (1a)
Galantamine	galantamine may have a beneficial effect on post-stroke aphasia; however, Galantamine has not been studied sufficiently in aphasia recovery (1b)
<b>Neutral recommendation</b>	
	Conclusion
Language therapy	Effect on communicative ability when compared to a non-aphasia therapy program(limited and conflicting 1a and level 2)
	Whether Immediate language therapy improve communicative ability when compared to deferred therapy (conflicting)
	Immediate group therapy rather than deferred therapy for the effect on communicative ability is conflicting
Community-Based Treatment Programs	Effectiveness of a community-based language program on communicative ability when compared to a recreational activities program (conflicting level 1b)
Audio-visual naming training	Effect of on word retrieval ability when compared to audio only naming training (conflicting and limited level 2)
	Speech rehabilitation involving biological feedback may be helpful for aphasia recovery; however, the use of video clips alone may not result an improvement. Further research regarding filmed language instruction is required (limited level 5)

Music therapy	effect on repetition (conflicting)
	Addition of music therapy to a standard aphasic therapy program is effective in the remediation of language function (conflicting level 2)
rTMS	Effectiveness on test components such as comprehension and repetition (conflicting)
tDCS	tDCS is as effective as sham-tDCS (conflicting level 2)
Effect of picture-naming therapy	when combined with gesture therapy on word retrieval abilities ( conflicting level 1b limited level 2)
Specific Treatment for Alexia in Aphasia	There is limited evidence that specific therapy for alexia in aphasic patients may improve language function and reading ability post-stroke
Bifemelane	Bifemelane may improve comprehension and naming; however more research is needed (1b)
<b>Not Recommended</b>	<b>Conclusion</b>
General language therapy	may not improve communicative ability, performance on comprehensive language assessments, comprehension or oral expression when compared to no treatment (1a and 2)
	Comparisons between similar types of aphasia therapy may not result in differences for the improvement of communicative ability, comprehension, language and cognitive impairment, non-verbal reasoning, verb acquisition and performance on comprehensive language assessments (2 and 4)
Intensity of Speech and Language Therapy	Intensive language therapy may not improve performance on comprehensive language assessments, cognitive and language tasks or communicative ability when compared to standard language therapy (1a, conflicting level 2 )
	Immediate language therapy may not improve reading comprehension, auditory comprehension or non-verbal reasoning when compared to deferred therapy (1b and 2)
Group Therapy for Aphasia Post-Stroke	Group therapy may not improve conversational ability, non-verbal reasoning, verbal expression, auditory comprehension or fluency as compared to individual treatment (1a)
	Group treatment, individual treatment and combined group and individual treatment may not produce different results in terms of word retrieval (1b)
community-based program	may not improve performance on comprehensive language assessments.
Patient and Caregiver Education	Caregiver and patient education program may improve knowledge of aphasia but not activity

	level, community integration or family functioning when compared to no treatment (limited level 2)
Computer-Based Treatments	Do not improve language function or word retrieval ability in the long-term when compared to standard language therapy (1a)
melodic intonation therapy	May not improve repetition when compared to no language treatment ( limited level 2)
Unilateral Forced Nostril Breathing	not attention level, spatial ability, auditory comprehension or depression
Piracetam.	Piracetam may be no better than placebo for comprehensive language assessment, and specific language outcomes, including semantic and phonological outcomes (1a)
	There is level 1b evidence that piracetam combined with language therapy may be no better than placebo for comprehensive language assessment and other language performance outcomes
Bromocriptine	Bromocriptine may be no better than placebo for treating aphasia poststroke (1a)
Levodopa	Levodopa may not be an effective adjunct to speech and language therapy (1a and level 2)
Moclobemide	Moclobemide may not improve verbal communicative abilities of individuals with aphasia. (1b)

## Post stroke depression

### Problem statement

- Approximately 1/3 individuals experience depression post stroke. Generally, incidence decreases and recovery increases over time, although some individuals may experience persistent depression and others may develop late onset depression.
- Although post stroke depression is one of the most common complication after stroke, few guidelines exist regarding assessment, treatment and prevention of PSD
- Neuroinflammation and decrease neurogenesis and plasticity may play an important role in the mechanism of PSD. The strongest predictors of PSD are stroke severity, early physical disability, and severity of loss of functioning. Nevertheless, populations at risk for PSD are yet to be identified.<sup>15</sup>
- Depression has a significant negative impact on physical function and cognitive function post stroke.

### Triage

Person with  
stroke



Apply Patient Health  
Questionnaire 9

PHQ 9 score	Depressive severity	Suggested Treatment
0-4	None-minimal	None
5-9	Mild	Watchful waiting; repeat PHQ-9 at follow-up
10-14	Moderate	Psychiatry referral, counselling, follow-up and/or pharmacotherapy
15-19	Moderately severe	Psychiatry referral, Active treatment with pharmacotherapy and/or psychotherapy
20-27	Severe	Immediate initiation of pharmacotherapy and, if severe impairment or poor response to therapy, expedited referral to a mental health specialist for psychotherapy and/or collaborative management

- Inclusion criteria- all stroke patients
- Exclusion criteria – aphasic patient
- Screening – with PHQ9
- History to get – pre existing mental illness, lack of sleep, suicidal thoughts

Recommended

TREATMENT	Conclusion
Fluoxetine	Reduced risk of post stroke depression (1a)  improves post-stroke functional recovery, but not cognitive or motor function (1a)

	improves long-term survival post stroke (1b)
Citalopram	reduces depressive symptoms post stroke (1b)
Escitalopram	Improvements in both working memory and attention (1b)
Nortriptyline	May improve cognitive performance more than standard care alone (1b)  reduces depressive symptoms post stroke (1a)  improves post-stroke functional recovery (1b)  improves long-term survival post stroke (1b)
Mianserin	reduces depressive symptoms post stroke when paired with imipramine or desipramine (1b)
Milnacipran	may improve cognitive performance (1b)
Duloxetine	Reduces depressive symptoms post stroke (1b)
Reboxetine (noradrenaline reuptake inhibitor)	Reduces depressive symptoms post stroke (1b)
Mirtazapine	reduced risk of post- stroke depression (2)
Methylphenidate	Reduces depressive symptoms post stroke (1b)  improves post-stroke functional recovery (1b)
Motivational interviewing	improves mood and reduces depressive symptom (1b)
Vitamin B therapy administered over a long period	reduced risk of post-stroke depression (1b)
Active care management program	enhances the effectiveness of pharmacologic treatment for post stroke depression (1b)
Behavioural therapy with antidepressants	Reduces depressive symptoms post stroke (1b)
Art therapy	reduces depressive symptoms post stroke (1b)
Goal achievement program	reduces depressive symptoms post stroke (1b)
Transitional care program	reduces depressive symptoms post stroke (1b)
Specialized, therapeutic exercise program	reduces depressive symptoms post stroke (1b)



Combined speech and orofacial therapies	reducing depressive symptoms post stroke (1b)
Hyperbaric oxygen therapy with fluoxetine 45 MTS 5 /WK 4 WKS	reduces depressive symptoms post stroke (1b)
Hyperbaric oxygen therapy	reduces depressive symptoms post stroke (1b)
Repetitive transcranial magnetic stimulation (rTMS)	reduces depressive symptoms post stroke (1b)
Acupuncture	in reducing depressive symptoms post stroke (1b)

#### Neutral

Modality	Conclusion	
Venlafaxine (SNRI)	Reduces depressive symptoms post stroke (4)	
Valdoxan (Melatonin agonist)	reduces depressive symptoms post stroke. (4)	
Statins	may reduce depressive symptoms post stroke (2)	
Pioglitazone with fluoxetine.	reduces depressive symptoms post stroke (2)	
Music-listening therapy	improves mood post stroke (2)	
Autogenic training	reduces psychological tension post stroke (4)	
Circuit training	reduces depressive symptoms post stroke (1b)	
Exercise with technological enhancements.	reduce depressive symptoms post stroke (1b)	
Group exercise programs	reduce depressive symptoms post stroke (1b)	
Adaptive physical activity program	reduces depressive symptoms post stroke (1b)	
Electroconvulsive therapy (ECT)	reduces depressive symptoms post stroke. (4)	

Transcranial direct current stimulation (tDCS)	reduces depressive symptoms post stroke. (4)
Meridian acupressure	reduces depressive symptoms post stroke (2)

Not recommended

	Conclusion
Sertraline	regarding the efficacy of sertraline in reducing the does not reduce depressive symptoms post stroke (1b)  risk of post- stroke depression (1b)
Mianserin	not associated with reduced risk of post- stroke depression (1b)
Selegiline	does not reduce depressive symptoms post stroke (1b)
Community outreach (using post mail or telephone calls)	Does not reduce depressive symptoms (1b)
Home visits from nurses and therapists	do not reduce depressive symptoms (1b)
fish oil supplementation	does not impact mood post stroke (1b)
self-management program	does not reduce depressive symptoms post stroke (1b)
Customized occupational therapy	does not reduce depressive symptoms post stroke (1b)
Ecosystem focused therapy	does not reduce depressive symptoms post stroke (1b)
Music therapy	does not reduce depressive symptoms post stroke (2)
music-movement	does not reduce depressive symptoms post stroke (2)

therapy	
Deep unilateral nostril breathing	does not reduce depressive symptoms post stroke (4)
resistance training	does not reduce depressive symptoms post stroke (1a)
Speech therapy	does not reduce depressive symptoms (1b)

## Dysphagia

### 15. Dysphagia and Aspiration Following Stroke

#### Problem statement

The incidence of aspiration in the acute phase of stroke varies from 16% to 52%. Silent aspiration occurs in 8% to 27% of acute stroke patients. Of identified aspirators, 20% to 67% developed silent aspiration.<sup>16</sup> Factors indicative of the development of aspiration include: a delayed swallow reflex, reduced peristalsis, respiratory tract infection, abnormal volitional coughing and cough with swallow, dysphonia, soft palate dysfunction, and facial hypesthesia.<sup>17</sup>

Incidence of Dysphagia in the Acute Phase post stroke is between 3.5% and 65%. Age, diabetes, neurological status, and lesion location may be associated with an increase in the rate of dysphagia.<sup>18</sup>

The prevalence of dysphagia in the dysfunction of the pharyngeal phase of swallowing seems to be high. Functional disturbances may vary based on lesion location. Specific measures of pulmonary function seem to be inhibited by dysphagia.

Decreased functional neurological connectivity may be associated with the presence of dysphagia and lead to complications of swallowing.<sup>19</sup>

Prognostic Indicators of Dysphagia Post-Stroke are: the presence of dysarthria, dysphonia and aspiration, abnormal cough and cough after swallow, National Institute of Health Stroke Scale scores  $\geq 12$ , level of consciousness assessment, intubation and bi-hemispheric infarcts, cognitive dysfunction, disuse syndrome, fever and length of hospital stay (inversely related). /3<sup>20</sup>

Relationship between Pneumonia and Dysphagia/Aspiration: both are associated with an increased risk of developing pneumonia, proportional to the severity of aspiration/ 1a<sup>21</sup>

Incidence and Development of Pneumonia: Stroke severity, level of consciousness, age, oral hygiene and other factors contributing to the aspiration of bacterial laden secretions and refluxed material are major indicators of an increased risk of pneumonia.<sup>22</sup>

Dysphagia Screening Protocols: swallow screening may reduce the incidence of pneumonia among patients with dysphagia when compared to no screening protocol or usual care/ 2 <sup>23</sup>

Tube feeding via Nasogastric tube can be given for first 3 to 4 weeks post stroke, but after that time period, patient should be counselled for PEG (Percutaneous Endoscopic Gastrostomy)insertion.<sup>24,25</sup>

## Metric MMASA

### Dysphagia Screen

The Modified Mann Assessment of Swallowing Ability(MMASA)

ENSURE PATIENT APPROPRIATE FOR SCREENING: i. Diagnosis of acute stroke ☐ ii. GCS  $\geq 12$  ☐ iii. No previous dysphagia/ head and neck surgery ☐

INSTRUCTIONS: Circle the most appropriate clinical findings for each indicator. Further information regarding each indicator provided over. Calculate the total score by adding the points for each indicator.

INDICATOR	CLINICAL FINDINGS (POINTS)			
1. Alertness	2 coma/ non responsive	5 difficult to rouse	8 Drowsy/ fluctuating	10 alert
2. Cooperation	2 non cooperative	5 reluctant	8 fluctuating	10 cooperative
3. Auditory comprehension	2 no response to speech	4 occasional motor response if cued	6 follows simple conversation with repetition	8 follows ordinary conversation with little difficulty
4. Expressive dysphasia	1 unable to assess	2 no functional speech sounds / single words	3 expresses self in limited manner short phrases / words	4 mild difficulty finding words or expressing ideas
5. Dysarthria	1 unable to assess	2 speech unintelligible	3 speech intelligible but obviously defective	4 slow with occasional hesitation or slurring
6. Respiration	2 Suspected chest infection/ suctioning vent dependent	4 coarse basal creps	6 fine basal creps	8 sputum upper airway other condition
7. Saliva	2 gross drool	4 some drool consistently	6 drooling at times	8 frothy / expectorated
8. Tongue movement	2 no mov't	4 minimal mov't	6 incomplete mov't	8 mild impairment in range
9. Tongue strength	2 gross weakness	5 Obvious unilateral weakness	8 minimal weakness	10 full ROM
10. Palate	2 No elevation or spread	4 Minimal mov't Nasal regurg/air escape	6 Unilaterally weak	8 Slight asymmetry, but mobile
11. Gag	1 no gag	2 absent unilaterally	3 diminished unilaterally	4 diminished bilaterally
12. Voluntary cough	2 no attempt / unable to assess	5 attempt inadequate	8 Cough attempted, but hoarse	10 NAD
TOTAL SCORE				
Score $\geq 95$ : Start oral diet and progress as tolerated. Monitor first oral intake and refer to SPEECH AND LANGUAGE THERAPY if any difficulties				
Score $\leq 94$ : NBM, consider inserting NGT as appropriate, and refer to SPEECH AND LANGUAGE THERAPY for formal swallow assessment				

North Bristol NHS Trust 2009

Deficits:

Classify according to MMASA subsections

Detection

Clinical Screening Methods

Screening tools	Conclusion
presence of post-swallow vallecular residue	greater risk of penetration-aspiration (4)
Water swallowing test	There was a wide range of sensitivity (47.8-100%) and specificity (50-100%) values for the water swallowing test and its variations. (4)
Swallowing provocation test.	There was a wide range of sensitivity (first-step=71.4-100%; second-step=13-76.4%) and specificity (first-step=38-100%; second-step=70.3-100%) values for the swallowing provocation test.
GUSS screening tool	The GUSS screening tool has 100% sensitivity and 69% specificity to predict aspiration risk.
Combination of the Water Swallowing Test and oxygen	Combination of the Water Swallowing Test and oxygen desaturation test may result in an improvement in the predictive accuracy of detecting aspiration and pneumonia over either of these

desaturation test	<p>screening tests conducted alone.</p> <p>There is no ideal volume of water that is used to assess dysphagia on the water swallowing test.</p>
Bedside Clinical Examinations	There was a wide range of sensitivity (68-97%) and specificity (53-86%) values for the different bedside clinical examinations.
Video fluoroscopic Modified Barium Swallow Examination	<p>Video fluoroscopic Modified Barium Swallow studies are considered the gold standard for dysphagia/aspiration diagnosis.</p> <p>Scintigraphy and video fluoroscopic (VFS) results associate with swallowing function.</p> <p>Scintigraphy has good predictive values for VFS results (70-95%).</p> <p>Sensitivity and specificity values for scintigraphy in predicting laryngeal penetration and/or aspiration were between 17-77% and 69-92%, respectively. (3)</p>
Flexible Endoscopic Evaluation of Swallowing	<p>Incidence of pneumonia after flexible endoscopic evaluation of swallowing (FEES) is used versus facial oral tract therapy or video fluoroscopy. (1b)</p> <p>Incidence of pneumonia may be reduced by FEES versus no assessment. FEES leads to increased instrumental assessment and on standard diet at discharge (4)</p> <p>There is evidence from a large case series study indicating that the incidence of pneumonia may be reduced when dysphasic patients are assessed with FEES versus no assessment. Additionally, FEES may be responsible for a higher proportion of patients treated with instrumental assessment and on standard diet at discharge which may be related to longer periods of non-oral feeding and length of stay in hospital.</p>
Pulse Oximetry	sensitivity 13% and specificity 39%
Ultrasonography	<p>Ultrasonography is video fluoroscopy</p> <p>Ultrasonography identifies significant differences between factors involved in the diagnosis of dysphagia with sensitivity (70-73.3%) and specificity (66.7-66.7%) (2)</p>

#### Aspiration Pneumonia

	Conclusion
Diagnosis	Criteria that may be most useful in the identification of pneumonia

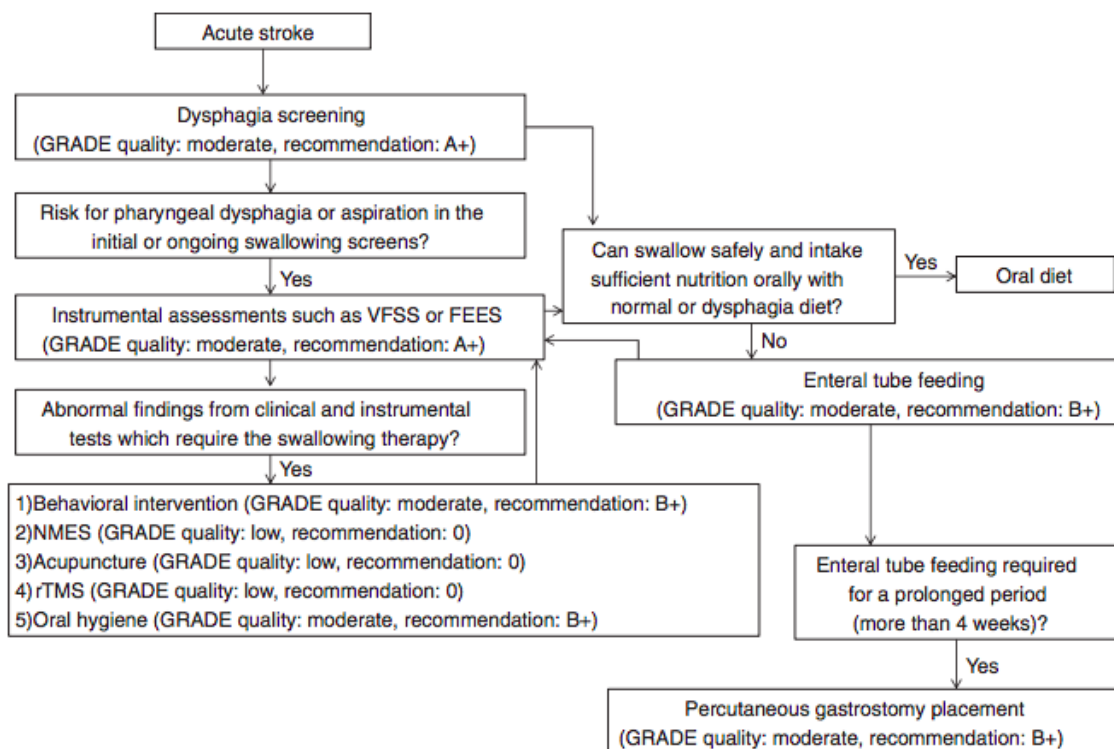
	<p>include: abnormal chest x-ray, temperature &gt;100°F, WBC &gt;10,000, arterial hypoxemia (PO<sub>2</sub> &lt;9.3kPa), PO<sub>2</sub> &gt;10torr, production of purulent sputum, crackles on auscultation, tachypnea &gt;22 breaths/min, tachycardia, bronchial breathing.</p> <p>Studies included required affirmative outcomes on two or three of these indicative measures for a positive diagnosis of pneumonia.</p>
--	--

Recommended	Conclusion
Dietary Modifications	<p>Supporting diets involving thickened liquids improves overall swallow safety and reduce incidence of aspiration pneumonia versus lower viscosity diets (1b)</p> <p>While thin fluids increase of total fluid intake there is an increase in aspiration pneumonia (2)</p>
Oral hygiene	Reduce aspiration pneumonia (1b)
Swallowing Treatment Programs	<p>High intensity swallowing therapy with dietary prescription= better recovery of normal diet and swallowing ability vs a lower intensity therapy or usual care (1b)</p> <p>Swallowing therapy and physical therapy are effective in reducing dysphagia (2)</p>
Non-Oral Feeding	Controlled infusion rate in enteral feeding based on the individual patient's gastric residual volume (GRV) may improve the incidence of regurgitation and aspiration versus no monitoring of the infusion rate (2)
Selection of Feeding Tubes	NG feeding may decrease the incidence of death and poor functional outcome (1b)
PEG insertion	Can consider from 3 to 4 weeks, when appropriate, to reduce the risk of microaspiration and other complications (1b)
Mode of Nutritional Intake	Oral intake versus tube feeding at discharge improved functional independence during acute care (4)
Electrical Stimulation	<p>NMES is effective in treating dysphagia, more so when combined with traditional therapies</p> <p>Electrical stimulation may improve swallowing function and the incidence and severity of penetration-aspiration when compared to thermal-tactile stimulation (2)</p>
Thermal Application	swallowing efficiency is improved, specifically among patients with supranuclear lesions after dry swallow preceded by ice massage of the oral cavity (1b)
Pharmacotherapy	<p>nifedipine may be associated with improved swallowing function versus placebo (1b)</p> <p>treatment with cabergoline, amantadine, imidapril, or cilostazol may reduce the incidence of aspiration and subsequent pneumonia when compared to no treatment (2)</p>
Transcranial Direct Current Stimulation	may improve functional severity of dysphagia when compared to sham stimulation (1a)
Repetitive Transcranial	may improve penetration and aspiration, swallowing function

Magnetic Stimulation	and functional disability compared to sham stimulation (1a)
Low-Risk Feeding Strategies for Dysphagia	Individuals with dysphagia should feed themselves to reduce the risk of aspiration. If hand-over-hand support is not viable and full feeding assistance is necessary, low-risk feeding strategies should be provided by trained personnel.
Prevention of Pneumonia Post-Stroke	ACE inhibitors reduces the relative risk of developing pneumonia vs placebo or other antihypertensive agents Metoclopramide improve incidence of pneumonia and resultant days on antibiotic treatment, episodes of aspiration, and swallowing outcome vs placebo with no observed effect on mortality (1a) Cilostazol improves the incidence of pneumonia vs not giving it (4)

Neutral Recommendation	Conclusion
Non-Oral Feeding	Conflicting evidence oral feeding vs nasogastric tube feeding increases the incidence of aspiration pneumonia among dysphasic patients (2)
Selection of Feeding Tubes	It is unclear which method of tube feeding (gastrostomy tube vs. nasogastric tube) is associated with a greater increase in the incidence of pneumonia
Acupuncture	combined with physical therapy is better than physical therapy alone (2)

Not Recommended	
Swallowing Treatment Programs	oral strength training may not be beneficial (1a)
Electrical Stimulation	Transcutaneous pharyngeal electrical stimulation does not improve swallowing function vs traditional swallowing therapy (1a)
Pharmacotherapy	cilostazol prescribed with aspirin may not have an effect on swallowing function compared to aspirin alone (2)



## Perceptual Disorders

### Problem Statement:

The major forms of visual perceptual deficit (and their clinical manifestations) are as follows: agnosia (inability to recognize an object by sight despite adequate cognition, language skills, and visual acuity/field; may include denial of illness); alexia (inability to recognize or comprehend written or printed words); apraxia (inability to execute purposeful movement, leading to dressing difficulty); ataxia (inability to visually guide limbs; mis localization when reaching or pointing for objects); depth perception (inability to judge depths and distances); figure-ground discrimination (inability to distinguish foreground from background); form perception/constancy (inability to judge variations in form); spatial relations (inability to perceive the position of two or more objects in relation to self and to each other); unilateral spatial neglect (inability to attend to or respond to meaningful sensory stimuli presented in the affected hemisphere, also known as hemi-inattention and hemi spatial neglect). Studies have reported incidence rates from 23% to 46% for unilateral neglect; unilateral neglect also negatively impacts functional recovery and long-term outcomes. (Jutai et al., 2003) Neglect is important in the rehabilitation for mobility; a crucial factor in the risk for falls is the patient's inclination toward initiating behaviours that place the patient in danger<sup>26</sup>



Screening Tests: Neglect - clock draw, line bisection, extinction (tactile and visual),  
Hemianopsia – Confrontation test,perimetry

Rx options

Recommended	
Approach/Modality	Conclusion
Perceptual training interventions	There is conflicting level 1a and level 2 regarding the evidence for perceptual training interventions on perceptual functioning.
Visual scanning	There is level 1a evidence that treatment utilizing primarily visual scanning techniques may improve perceptual impairment post-stroke with associated improvements in function.
Computer-based or virtual reality treatment	There is level 1b evidence that computer-based or virtual reality treatment for neglect may improve visual perception and alleviate right-hemisphere bias when compared to conventional rehabilitation or no treatment.
Computer-based or virtual reality treatment	There is limited level 2 evidence that computerized visual perception training may be no more effective than occupational therapy for patients with hemianopia.
Limb activation	There is level 1a and level 2 evidence that limb activation may alleviate rightward bias and improve motricity when compared to conventional rehabilitation.
<b>Sensory Stimulation Intervention</b>	Electrical somatosensory stimulation as a supplement to visual scanning training is associated with greater benefit than visual scanning training alone. (1b)
Feedback strategies	Visuomotor feedback may be beneficial in the treatment of neglect. (1b)
Prisms	Rightward shifted prisms may be effective for neglect and hemianopia. (1a)
<b>Vestibular Galvanic Stimulation</b>	Galvanic vestibular stimulation may improve unilateral spatial neglect. There is conflicting level 1a evidence with regards to the effect of right cathodal versus left cathodal galvanic vestibular stimulation on unilateral spatial neglect.
<b>Neck Muscle Vibration</b>	Neck muscle vibration therapy in association with visual exploration training may be effective in improving both symptoms of neglect and performance of activities of daily living. (1a)
<b>Repetitive Transcranial Magnetic Stimulation</b>	Transcranial magnetic stimulation (rTMS) and theta burst stimulation (TBS) may improve neglect and functional ability. (1a)
Trunk rotation	Trunk rotation when combined with visual scanning is of benefit in the treatment of spatial neglect. (2)

<b>Nicotine Therapy</b>	Nicotine may improve unilateral neglect and target information processing when compared to placebo treatment (1b)
<b>Acetylcholinesterase Inhibitors Therapy</b>	Rivastigmine in conjunction with cognitive training may accelerate the rate of improvement of unilateral spatial neglect associated with therapy. (1b)
<b>Optokinetic Stimulation</b>	Optokinetic stimulation may have a positive impact on unilateral neglect when compared to scanning or alertness training (1b)

Neutral Recommendation	
Approach/Modality	Conclusion
<b>Sensory Stimulation Intervention</b>	Sensory cues for movement may have a positive effect on neglect, although evidence is inconclusive. (1b)
Feedback strategies	Auditory feedback for left eye movement may not improve visual inattention or bias in eye movement. (2)
<b>Eye-Patching and Hemispatial Glasses</b>	Conflicting evidence use of right half-field eye patches for left visual neglect. (Level 1b evidence conflicting) Monocular occlusion may not improve visual neglect or alleviate rightward bias. (2)
<b>Eye-Patching and Hemispatial Glasses</b>	Conflicting evidence of the effect of bilateral half-field eye patches on functional ability. (1b)
<b>Caloric Stimulation</b>	At present, there is little evidence regarding the effectiveness of caloric stimulation as a treatment intervention for visuospatial neglect post-stroke.
<b>Music Therapy</b>	Presently, there is little evidence to support the use of music as treatment for unilateral spatial neglect in right hemispheric patients.

<b>Transcranial Direct Current Stimulation</b>	Transcranial direct current stimulation is associated with improvement on tests of neglect; however, limited Level 2 and Level 4 evidence suggests that transcranial direct current stimulation may not be beneficial for neglect
--	---

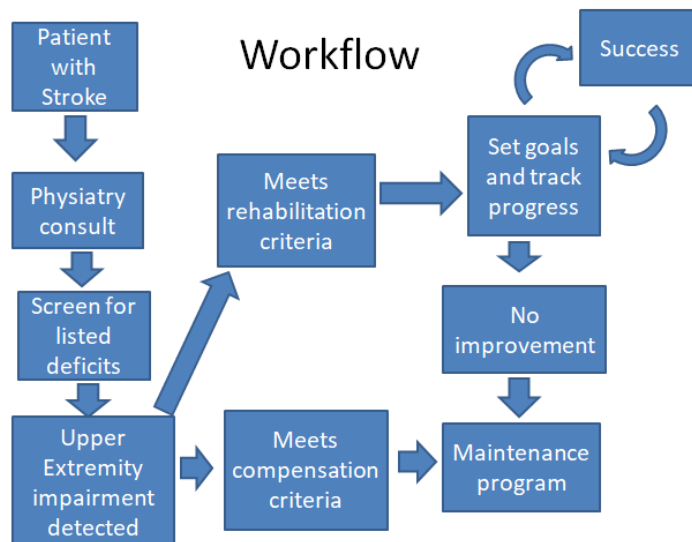
Not Recommended	
Approach/Modality	Conclusion
Transfer of training approach	approach may not produce different results on measures of neglect and functional ability when compared to a functional approach to perceptual training. (1b)
<b>Family Participation</b>	There is limited level 2 evidence that family participation in rehabilitation may not be associated with additional improvements in perceptual impairment and functional ability when compared to conventional rehabilitation.
<b>Prism Treatment</b>	Improvements seen in visual-spatial tasks may not be sustained over time. (1a)
	Improvements in visual-spatial tasks following prism treatment are not associated with improvement in functional ability. (1b)
Optokinetic stimulation	Optokinetic stimulation may not have an effect on functional outcome. (1a)
Trunk rotation	Trunk rotation therapy may not have a positive effect on unilateral spatial neglect or performance of activities of daily living. (1b)
Trunk rotation	Trunk rotation in combination with half-field eye-patching is similarly ineffective. (1b)
<b>Transcutaneous Electrical Nerve Stimulation</b>	Transcutaneous electrical nerve stimulation does not improve visual neglect. (1b)
<b>Dopaminergic Medication Therapy</b>	Dopamine agonist rotigotine may not improve perceptual impairment or motor function. (1b)
<b>Caloric Stimulation</b>	At present, there is little evidence regarding the effectiveness of caloric stimulation as a treatment intervention for visuospatial neglect post-stroke. (1b)
<b>Music Therapy</b>	Presently, there is little evidence to support the use of music as treatment for unilateral spatial neglect in right hemispheric patients.

## Upper extremity

## Problem statement

Approximately 70–80% of patients with stroke experience upper extremity impairments, and 5 years after stroke onset, approximately 56% of patients continue to report pronounced hemiparesis. It is taken for granted that the maximal recovery from stroke occurs over the first three to six months. After this little improvement is possible, especially at the level of impairment.<sup>27,28,29,30,31</sup> When we consider the arm and hand deficits after stroke, we find that upper extremity paresis impacts the ability to perform bimanual tasks.<sup>32</sup> This loss of functionality can lead to difficulties in activities of daily living (ADLs) and participation in the community.<sup>33,34</sup> Long-term spasticity may lead to tendon contractures and limb deformities that can cause significant pain and functional impairment. Depending on the location of the spasticity, this can increase the dependence on caregivers.<sup>35</sup>

### Part 1:



### Part 2: Decision tree 1- Triage

Prerequisites/ Inclusion criteria for rehabilitation

- at least moderate handicap on admission (Rankin scale 3)
- MMSE score **of 7** or higher (no severe dementia)
- Anything other than Brunnstrom arrest at 2-3 for >6months
- If otherwise the goal of compensation with preserved limb and maintenance therapy for involved limb

Clinical history and exam/ baseline and outcome measures

**BRS-** (shoulder/ elbow/ wrist)

- **MAS-** (shoulder/ elbow/ wrist)
- Contractures- PRoM (shoulder/ elbow/ wrist)
- Selective Voluntary Motor Control- ARoM (shoulder/ elbow/ wrist) and Fugl Meyer-2
- Spasticity pattern: (adducted shoulder, flexed elbow, pronated forearm, flexed wrist, clenched fist, thumb in fist)
- Other- Clonus, Neglect, VAS for pain, spastic dystonia

### Part 3a: Decision tree 2- Custom tailored program

Treatment plan- Patient led, Physiatrist guided

Goal Attainment Scale (GAS) Approach

- Map out a plan how to achieve felt need. If not possible plan the easiest achievable next step.
- Examples:i.e.**BRS 3/ MAS 4**, wants to use his hand to drink water from a cup.
  - Step 1 reduce contracture to get 10 degrees on active wrist extension
  - Step 2 reduce hypertonia for the same goal
  - Step 3 therapy to attain crude grasp
  - Step 4 reassess

Follow-up plan/ outcome measures

- How do we measure this? (BRS/ MAS/ Fugl Meyer/ Tardieu) Once a month physical meeting (x3) and weekly tele-meeting/ activity diary
- Over how much time? (4 weeks per step, total of 3 months)
- What next if they do or don't achieve? if they achieve ask what more do they want.
- If no change by 3months then maintenance

Deficits mapped to metrics –

Now that we know what the patient wants and what is possible. Next, we should identify what prevents them from getting their desired gain. We have to match the clinical exam scores (BRS and MAS) to the following deficits: impaired grip, contracture, problematic hypertonia, reduced PRoM, poor overall limb function.

Brunnstrom staging marks motor recovery while Ashworth tracks hypertonia. While both can be measured together, they don't consistently move in tandem. First please note the two values. Using the superimposed BRS/ MAS image below with the associated chart determine the goals you can work on. (figure 2 and table 1)

Examples:

i.e. If the BRS is 1 and MAS is 0 you can't work on impaired grip, there likely is no contracture to reduce, no hypertonia to address, nor PRoM or function to improve. That means you work to prevent complications.

i.e. If the BRS is 5 and MAS is 1 you can improve impaired grip and function, and reduce contracture, but may not have to focus on reducing hypertonia, nor improve PRoM. This is the time where you can refer to the supplementary tables for treatment options.

None of the measurement correlations below are set in stone. They act as a rough guide within which we can fine tune the plan.

### **Part 3b-** What to do, when, how, why, and to whom?

Having reviewed the existing literature and comparing it to our setting, the committee recommends the following as starting points for care of upper extremity deficits in stroke.

#### Impaired grip

Rehabilitation begins at Brunnstrom 2 and MAS 1

Basic prescription should include: PRoM for contracture and hypertonia, motor re-education, gripper toy, occupational therapy, dynamic splints (spring/ elastic loaded extensor splint).

#### Contracture with hypertonia

Rehabilitation begins at BRS 2 and MAS 1 and ends at BRS 6 MAS 0

Basic prescription should include: PRoM, chemo denervation of finger long flexors (MAS 2-3)

#### Restricted PRoM (non-contracture)

Rehabilitation begins at Brunnstrom 2 and MAS 1 and ends at BRS 5 and MAS 1

Basic prescription should include passive stretching, positioning, splinting

#### Hypertonia

Rehabilitation begins at Brunnstrom 2 and MAS 1 and ends at BRS 5 and MAS 1

Basic prescription should include: oral antispastics as needed, Botox or ECSWT if not better, mCIMT

Impaired function Rehabilitation begins at Brunnstrom 2 and MAS 1, Between BRS 3 with MAS 3 and 4 not much can be done, but after that rehab can resume

Basic prescription should include: arm and leg training, repetitive task training,

**\*\*With this if you don't see progress on follow up please consult the below tables for more treatment options.**

<b>Recommended</b>		
Deficit- grip		
Modality/ Approach	Conclusion	BRS/ MAS
PRoM	Improve RoM	1-3/ 0-1+
Strength training	Will make grip stronger (1a)	4 or more/ 2 or less
Deficit- contracture		
Passive RoM	Prevent contracture (n/a)	1/ 0-1+
Stretching	Stretching improves passive range of motion in the upper extremity and reduce pain(1b)	2-3/ 2 or more
Active RoM for non-synergy muscles	Restore muscle balance(n/a)	4-7/ 2 or less
Deficit- hypertonia		
PRoM	Reduce tone/ n/a	2-3/ 1-4
Botox	Will reduce hypertonia (1a)	2-3/ 2+ or more
mCIMT	Will reduce hypertonia(1a)	2-3/ 2+ or more
ECSWT	Will reduce hypertonia(1a)	2-3/ 2+ or more
tolperisone	Will reduce hypertonia(1b)	2-3/ 2+ or more
Device based	Will reduce hypertonia	2-3/ 2+ or

		more
Nerve block	Will reduce hypertonia(4)	2-3/ 2+ or more
Mirror therapy	Will reduce hypertonia(1a)	2-3/ 2+ or more
<b>Deficit – UE function</b>		
Passive RoM	Motor re-education/ n/a	1-3/ any
NDT	Will induce short term recovery and shorter hospital stay, like standard care(1a)	4/ 2 or less
Bilateral arm training	Will improve motor function equal to unilateral training, conventional training (CIMT and Estim), even if Rhythmic stimulation is used(1a)	4/ 2 or less
Arm and leg training	Will improve arm function, task practice, and strength(1a)	4/ 2 or less
Additional therapy	Will improve arm function and independence same as conventional therapy(1a)	4/2 or less/ 2 or less
Repetitive task training	Will improve function better than conventional training(1a)  Improves general task-related training, and reaching movements with bilateral arm training(1a)  will improve dexterity/ reaction time if combined with active stimulation(1b)	4/2 or less/ 2 or less
Trunk restraint	Will improve function, alone or as an adjuvant to CIMT(1a)	4/2 or less/ 2 or less
Sensori-motor training	Focal or whole-body vibration therapy will improve motor function(1a)  Mesh glove therapy will improve motor function and dexterity (1a)	4/2 or less/ 2 or less
Mental practice	Will improve motor function(1a) <i>unclear if it improves ADLs</i>	4/2 or less/ 2 or less
CIMT	Will improve motor function(1a)	4/2 or less/ 2 or less
mCIMT	improve motor function vs conventional(1a)	4/2 or less/ 2 or less
Feedback therapy	Will improve function	4/2 or less/ 2 or less
Music therapy	Will improve some aspects of motor function(1a)	4/2 or less/ 2 or less
NMES	improve motor function, range of motion, and manual dexterity combined with conventional therapy or delivered alone/ subacute(1a)  improve range of motion and manual dexterity alone	4/2 or less/ 2 or less



	or in combination with other therapies/ chronic/effective(1b)	
Physiotherapy	Brunnstrom hand manipulation treatment > motor relearning program (1b)	4/2 or less/ 2 or less
Strength training	improve motor function and shoulder range of motion(1a)	4/2 or less/ 2 or less
<b>Robotic Rehabilitation</b>		
MIT Manus	Will improve motor function (2)	Any
ARMin	improves motor function(2)	
Bi-Manu-Track	will not improve motor function, is not effective in the acute/ conflicting evidence in subacute/ but in chronic phase is effective (2)	
NeReBot	not effective in chronic phase, conflicting evidence acute phase(2)	
GENTLE	will not improve motor functionin chronic phase(2)	
Amadeo	will improve motor function in acute but not chronic has conflicting evidence in chronic phase(2)	
Music Glove		
Mirror-Image Motion Enabler Robots (MIME)	are effective in the acute phase and are not effective in the subacute phase(2)	
ARM Guide	will not improve motor function(2)	
Continuous Passive Motion	will not improve motor function in acute phase. But will improve motor function in the chronic phase(2)	

(CPM)		
-------	--	--

<b>Neutral Recommendation</b>		
Modality	Conclusion	BRS/ MAS
Deficit- hypertonia		
Botox w/ electric stimulation	Conflicting data regarding reduction of hypertonia(1b)	2-3/ 2+ or more
Deficit – UE function		
Sensori-motor training	Conflicting evidence that Thermal stimulation will improve motor function(1a)	4/2 or less
Mirror therapy	conflicting evidence that it will improve wrist and hand function Combined with conventional therapy equal to Bobath method(1b)	4/2 or less
Action observation	conflicting evidence it will improve function/ but with BCI+ FES will improve motor function(1b)	4/2 or less
Botox	May improve motor function in correct patient(1b)	4/2 or less
Brain computer interface	Conflicting evidence to improve motor function(1a)	4/2 or less

<b>Not Recommended</b>		
Modality	Conclusion	BRS/ MAS
Deficit- contracture		
Splint	Will not prevent development of contracture (1a)	2-3/ 2 or more
Deficit- hypertonia		
Estim alone	Will not reduce wrist or elbow hypertonia(1a)	2-3/ 2+ or more
Splinting	Will not reduce hypertonia(1a)	2-3/ 2+ or more
Feedback therapy	Will not reduce hypertonia(1a)	2-3/ 2+ or more
<b>Deficit – UE function</b>		
<b>Sensori-motor training</b>	to improve motor function electroacupuncture is not more effective than a usual care(1a)	4/2 or less
Hand splinting,	Will not improve motor function(1a)	4/2 or less

taping, orthoses		
Music therapy	Will not improve muscle strength vs conventional rehabilitation(1a)	4/2 or less
Telerehabilitation	Is not effective for improving upper limb motor function (1b)	4/2 or less
Virtual reality	Will not Improve motor function(1a)	4/2 or less
EMG biofeedback	Does not improve motor function or spasticity/ not effective(1a)	4/2 or less

### Part 3c- What to do otherwise?

Every patient with an upper limb deficit will need exercise, but they will not always need a therapist to deliver this. The maintenance group patients can receive care from trained family members. Educating the family empowers them to communicate with us. If there is a need, we can modify the plan from there. Some patients will have no further motor recovery after their stroke, due to the severity of damage. If they are not improving over three months, we call this Brunnstrom arrest, and they should be considered for maintenance therapy. Acute exacerbations in care, noncompliance with exercise or other therapeutics can confound this so it is always worthwhile to see if a patient meets rehabilitation criteria later on. In the meantime, we suggest setting the following goals: prevent contracture, manage pain, prevent subluxation or worsening, prevent CRPS. The ways to do this are as follows: PRoM by family/ caregiver, injections to reduce tone, orthotic support.

Addendum

Figure 1: The pathogenesis of spasticity

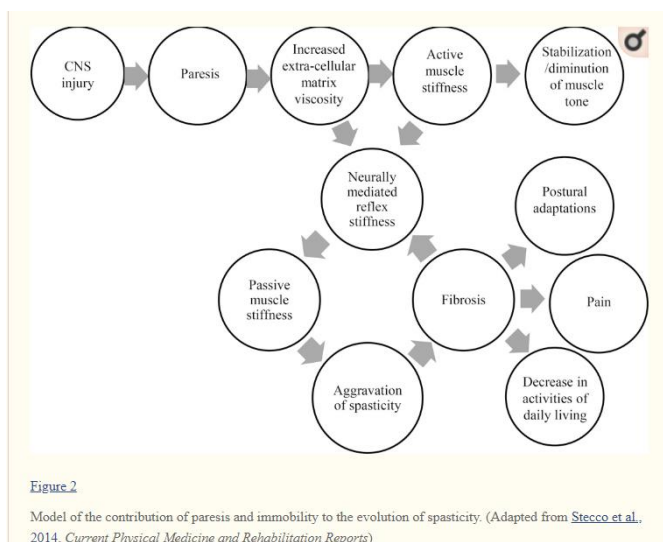


Figure 2: correlation of BRS to MAS

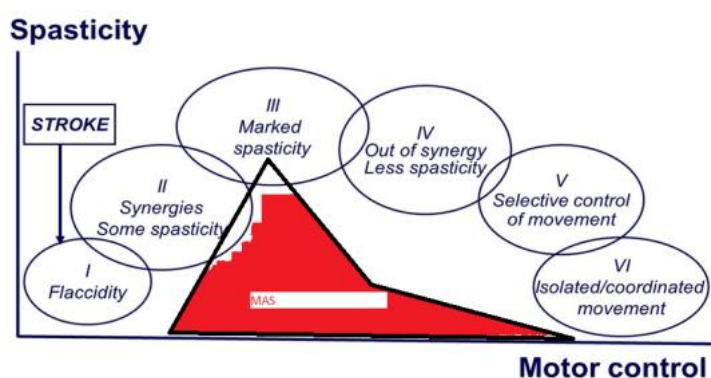


Table 1: detailed overlay of BRS to MAS relationship with respect to treatment timings and appropriateness

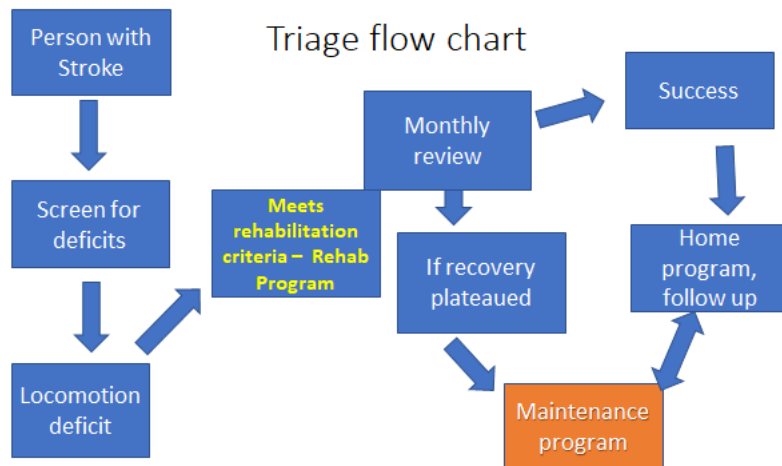
BR S	1	2	2	2	2	3	3	4	4	4	4	5	5	5	6	6	7
MA S	0	1	1 +	2	3	3	4	2	1 +	1	0	1 +	1	0	1	0	0
			Work on Impaired grip														
			Reduce Contracture													X	
			Manage Problematic Hypertonia											X			
			x	Improve Reduced PRoM										X			
			Poor function			X		Improve Poor function									

## Lower Extremity

### Problem Statement

Walking dysfunction occurs in more than 80% of stroke survivors.<sup>36</sup> Despite rehabilitation efforts, 25% of all stroke survivors have residual gait impairments that

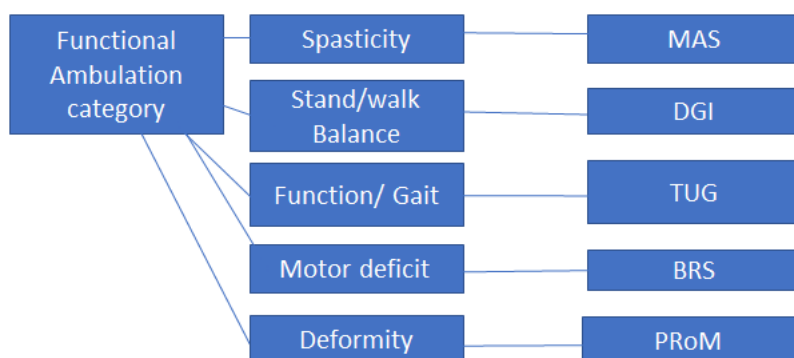
require full physical assistance before hospital discharge.<sup>37</sup> Consequently, gait impairments cause difficulties in performing activities of daily living and mobility. Gait abnormality is characterized by a pronounced clinical presentation of gait asymmetry, as compared to healthy people.<sup>38</sup> Stroke survivors usually have decreased stance phase and prolonged swing phase of the paretic side. Further, the walking speed is decreased and the stride length is shorter.<sup>39</sup> These gait abnormalities along with muscle weakness place stroke survivors at a high risk of falls.<sup>40</sup> Falls usually occur during walking in community-dwelling stroke survivors. Thus, improving walking safety and speed is the major goal for stroke survivors to prevent falls and to improve quality of life.<sup>39</sup>



## Prerequisites

- A. Inclusion: Neurological deficits of the lower limb after a vascular cerebral event,
- B. Medically stable
- C. Functional deficits- dependence on ADL, affected mobility
- D. Ability to comprehend and participate in rehab
- E. Caution: cardiac EF min 40%, Resp capacity (single breath count minimum 15) for active exercises, intensity and duration to be adjusted and monitored in cardio-respiratory compromised patients
- F. Pre-existing condition, co morbidities, previous stroke, evidence of complex stroke (cognitive, speech, swallowing issues)
- G. Family and Social support system
- H. Economic status of the patient
- I. Occupation and feasibility to return to it
- J. Screening- contractures, pressure sore, deformities impeding walking- ?
- K. What history points to specific locomotion issues? ( bed mobility, Sitting balance, Standing / Gait FAC )

<b>FAC</b>		<b>O</b>	<b>1,2,3</b>	<b>4,5</b>
Spasticity		MAS	MAS	MAS
Balance (stand/ walk)		Sitting (static/ dynamic)	BBS, standing/ walking	TUG
Motor function		BRS	BRS/ FM	FM
Deformity		PROM (all joints)	PROM (all joints)	PROM (all joints)
Gait		xxxxxxxxxx	xxxxxxxxxx	DGI, TUG



### Recommended ( based on available evidence)

#### **Motor Function ( including Gait, Balance)**

<b>Approach</b>	<b>Conclusion</b>	<b>FAC</b>
Therapeutic Approach Compensatory and restorative approaches	improve motor function, although neither approach is superior (1a)	NA
Action observation In combination with gait training	improves gait and balance when compared to gait training alone (1b)	0-5
Aquatic therapies	improves gait when compared to conventional therapy. (1a)	3-5
Caregiver-Mediated	improve gait and balance when compared	0-5

Programs	to usual care in chronic stroke (1b)	
Mirror therapy	improves gait and mobility when compared to conventional rehabilitation (conflicting 1b)	0-5
Combined with FES		
Strength and Resistance Training	improves muscle strength (1a)	0-5
Progressive resistance training for the lower limbs		
Combined resistance and aerobic training (walking)	for the lower limbs improves gait and endurance. (1a)	4-5
aerobic exercise	improves gait (1a) when compared to conventional rehabilitation	2-5
community- and home-based exercise programs incorporating aerobic exercise	Improve gait and balance when compared to conventional rehabilitation (1a,2)	3-5
Wheelchair	Neater wheelchair attachment improves efficiency of motor skills and activity performance when compared to a standard wheelchair (1b)	0
Walking Aid	single-point canes improve gait speed and endurance when compared to quad canes and hemi-walkers (1b)	3,4
Robotic walker	improves gait speed and endurance when compared to no device (1b)	2-,4
quad canes	improve balance when compared to single-point canes(2)	2,3
taping in combination with rehabilitation	improves balance and gait when compared to rehabilitation alone. (2)	3,4
Electromechanical Devices End-effector systems (e.g. Gait Trainer)	improve lower limb motor function in acute/subacute stroke, but not chronic stroke, when compared to conventional training (1a)	2-4
Ankle Foot Orthosis	Improves gait when compared to no device (1a)	0-4
Standard ankle foot orthosis	as effective in improving gait as an individualized ankle foot orthosis. (1b)	0-4
dynamic ankle foot orthosis	is more effective in improving gait than a static ankle foot orthosis. (limited Level 2)	0-4
Hybrid Assistive Limb	improves gait and balance when compared to conventional training (2)	0-4
Electromechanical Devices End-effector systems (e.g. Gait Trainer)	improve lower limb motor function in acute/subacute stroke, but not chronic stroke, when compared to conventional training.( 1a)	0-4
FES during	improves gait, balance, and	0-3

conventional rehabilitation	independence when compared to rehabilitation alone (1a)	
FES during gait training	improves gait when compared to gait training alone (1a)	0-3
FES during cycling training	improves gait and balance when compared to cycling alone but not to conventional rehabilitation (1a)	0-3
TENS	improves gait, balance, and muscle strength when compared to sham or no stimulation (1a)	0-3
neuromuscular electrical stimulation in combination with gait/balance training	improves gait/balance when compared to stimulation or training alone (1a)	0-3
Neuromuscular electrical stimulation alone	improves gait and balance when compared to sham or no stimulation (1b)	0-3
interferential current therapy	improves gait and balance when compared to sham stimulation(1b)	0-3
Other Sensorimotor Stimulation	improves lower limb motor function and reduces spasticity when compared to no/sham stimulation(1a)	0-5
Thermal stimulation		
Local vibration	improves gait and balance when compared to sham stimulation( 1b )	0-3
repetitive peripheral magnetic stimulation	improves ankle/foot strength and range of motion when compared to sham stimulation (1b)	0-3
Ankle/foot sensorimotor stimulation	improves weight distribution when compared to no stimulation (1b)	0-3
Repetitive Transcranial Magnetic Stimulation	Repetitive transcranial magnetic stimulation improves balance, gait, independence, and lower limb motor function when compared to sham stimulation (1a)	0-3
Noradrenergic Agents methylphenidate	when compared to placebo, improves functional independence (1a)	
droxidopa	improves functional outcomes when compared to no medication(limited2)	
Serotoninerbic Agents	citalopram improves neurological status, when compared to placebo(1b)	
.	fluoxetine improves lower limb motor function when compared to placebo (1a)	



Other Medications	cerebrolysin improves lower limb motor function in severe stroke when compared to placebo (1b)	
	dalfampridine improves gait speed when compared to placebo (2)	NA
<b>Contracture</b>		
Splint and tilt table	Both prevent ankle contracture (1b)	
<b>Spasticity with MAS &gt;3</b>		
Botulinum toxin	Reduces lower limb spasticity when compared to placebo (1a)	
Botulinum toxin-a	higher dosage of ( $\geq 300$ U) reduces lower limb spasticity compared to a lower dosage (100-200U) (1a)	
	similar reductions in lower limb spasticity regardless of injection location (1a)	
	combined with AFO reduces lower limb spasticity compared to botulinum toxin alone, with taping, or stretching (1a, 2)	
	reduces lower limb spasticity when compared to phenol nerve block. (1b)	
	USG guided injection better when compared to electrical stimulation or palpation to reduce spasticity (1b)	
Thermocoagulation	reduces lower limb spasticity compared to AFO or sham treatment (1b)	
phenol and ethyl alcohol	There is limited evidence (2) that both are equally effective in reducing lower limb spasticity	
Tolperisone	reduces lower limb spasticity and improves functional independence when compared to placebo (1b)	

### **Neutral Recommendation ( Based on available evidence)**

<b>Approach</b>	<b>Conclusion</b>	<b>FAC</b>
<b>Motor Function ( incl Gait, Balance)</b>		
Horse riding simulation	conflicting evidence that hippotherapy improves gait and	

(Hippotherapy)	balance when compared to conventional rehabilitation or standard gait training (1a,2)	
Functional strength training	Conflicting (1b , 2) evidence as to whether it improves gait	
isokinetic strength training	For lower limbs improves motor function (conflicting 1b,2 )	0-5
eccentric resistance training	for the lower limbs improves motor function Conflicting(1b,2)	0-5
Progressive resistance training for the lower limbs	conflicting (1b , 2) evidence for improvement in balance, gait, or endurance.	0-5
aerobic exercise,	Improvement in balance is conflicting (4,5)	
Intensity of exercise	high-intensity aerobic exercise better improves gait than low-intensity aerobic exercise / (Conflicting 2)	
Ankle Foot Orthosis	improve balance (conflicting)	0-4
Exoskeletal systems (e.g.Lokomat, LokoHelp, AutoAmbulator, Walkbot)	Improves lower limb motor function when compared to conventional training (conflicting Level 1a)	
FES during treadmill training	improves gait and balance when compared to treadmill training alone(conflicting Level 1b)	0-3
Other Sensorimotor Stimulation Whole-body vibration	improves gait or balance when compared to no/sham stimulation (conflicting 1b)	0-3
Dopaminergic Agents levodopa	Improves lower limb motor function and functional independence when compared to placebo or no medication (conflicting1b)	
Serotoninerbic Agents	Improves functional independence	

Fluoxetine	when compared to placebo. (Conflicting 1b)
	does not improve neurological recovery when compared to placebo(1a)

### Not Recommended ( based on available evidence)

Approach	Conclusion
<b>Motor Function (incl Gait, Balance)</b>	
Caregiver-Mediated Programs	do not improve mobility or independence when compared to usual care in acute stroke. (1a)
Self-management programs	do not improve gait and balance when compared to usual care. (1a)
Functional strength training for the lower limbs	does not improve muscle strength (1a) ,
Cane length	does not impact gait speed (2)
Electromechanical Devices  End-effector systems (e.g. Gait Trainer)	Does not improve lower limb motor function in chronic stroke, when compared to conventional training (1a)
Portable, localized exoskeletal devices (e.g.Stride Management Assist, Bionic Leg, Anklebot	do not improve gait or balance when compared to conventional training. (1a)
FES during robot-assisted	does not improve gait or balance when compared to robot-

training	assisted gait training alone (1a)
	does not improve gait or balance when compared to ankle foot orthosis (1a)
Transcranial Direct Current Stimulation	transcranial direct current stimulation does not improve gait or balance when compared to sham stimulation. (1a)
Galvanic Vestibular Stimulation	galvanic vestibular stimulation does not improve pusher behaviour or lateropulsion(1b)
Noradrenergic Agents	Both amphetamines and methylphenidate do not improve lower limb motor function when compared to placebo(1a)
Dopaminergic Agents	Ropinirole does not improve gait, functional independence, or motor function when compared to placebo (1b)
<b>Serotonergic Agents</b> Citalopram	Does not improve walking ability or functional independence, when compared to placebo(1b)
Escitalopram	does not improve gait, strength, or motor function when compared to placebo (1b)
Fluoxetine	does not improve neurological recovery when compared to placebo(1a)
Other Medications .	PF-0304923 does not improve gait speed when compared to placebo (1b)
<b>Spasticity</b>	
Botulinum toxin	is less effective than tibial nerve neurotomy in reducing spasticity (1b).
	combined with taping does not reduce lower limb spasticity when compared to botulinum toxin combined with sham taping or stretching (1a)
Phenol nerve block	less effective than botulinum toxin (1b)

## Hemiplegic shoulder pain

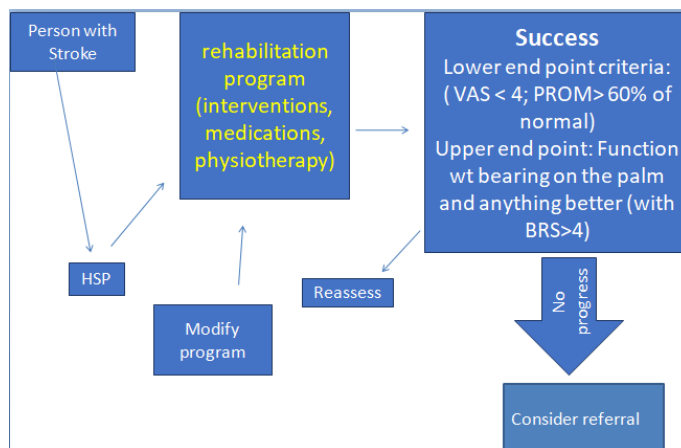
## Problem statement

Estimates of the prevalence of PSSP varies widely from 16 to 80%, depending on when and how it is measured; but it is clear that its prevalence increases with time since stroke.<sup>41,42</sup> It has been associated with prolonged hospital stay.<sup>43</sup> A variety of factors may be responsible, for eg, joint pathology, adhesive capsulitis, subluxation of the head of the humerus, injury to rotator cuff tendons, spasticity of surrounding muscles, central poststroke pain, and complex regional pain syndrome.<sup>44,45</sup> However, the etiology of hemiplegic shoulder pain (HSP) remains uncertain.

It is highly probable that PSSP is multifactorial with different factors contributing at different stages of the illness.<sup>46</sup> As an example, early flaccidity may cause subluxation of glenohumeral joint, which in turn causes stretching of the shoulder capsule and ligaments. Hypertonicity develops later, which together with the immobilisation effects of the sling, continue the pain. Later, sympathetically mediated pain may occur.

### Triage flow chart

Receive patient- dx HSP- identify components (flaccid, spastic, contracture, pain, CRPS)- address deficits- review and modify plan



### Prerequisites:

- Inclusion: all w/ HSP

Stroke duration any

- Exclusion: Contraindications for interventions including multiple uncontrolled medical issues
- History to get: Pre-existing trauma or neuro- MSK disease, lack of sleep, pain interfering with ADLs
- Screening- **VAS**, MAS, BRS, PROM, AROM, FM2

	<b>VAS <math>\leq</math>4</b>	<b>VAS &gt;4 interfering with routine/ patient preference</b>
Adhesive Capsulitis	Exercises +/- NSAIDs	Interventions/ medication
Subluxation	Sling, Positioning, exercises, electrical stimulation	Interventions / medication
Rotator cuff tendonitis	Exercises	Interventions /medication
Central pain	CBT, acupuncture, neuropathic medications	Medication including IV ketamine
Spasticity	Exercises, Medications, Icing	Botulinum toxin, Phenol (with MAS > 2/ BRS 2 -3)
Autonomic pain/ CRPS	Medications, Physical agents	Interventions

### **Setting the plan**

- Goal Attainment Scale (GAS) Approach

### **Review and follow-up plan**

- Review monthly once for six months. Till our treatment options have exhausted
- Review the plan and modify it
- Reasons for not achieving goal - Pain not controlled; cognitive issue; apraxia; family; Brunnstrom arrest

### Treatment of pain

<b><u>Recommended</u></b>		
Modality	Conclusion	Metric
Shoulder Sling exercise	Reduce pain and subluxation /1b and 2	VAS<4
Strapping of shoulder	Reduce pain/1a	VAS<4
Active therapies for shoulder	<p>Passive ROM exercises not more effective than self ROM exercises in improving motor function, joint stability, spasticity or pain/1b</p> <p>Stretching and joint stabilising exercises more effective than conventional exercises in improving the motor function/ 2</p> <p>Bobath therapy more effective than cryotherapy in reducing shoulder pain/2</p>	VAS<4
Electrical stimulation of shoulder	<p>Intramuscular NMES effective, when compared to cuff sling shoulder, in reducing shoulder pain (doesn't improve subluxation, spasticity or motor function) for up to one year post treatment</p> <p>PNS (Peripheral Nerve Stimulation) effective in reducing shoulder pain/ 1b</p> <p>IES(Interferential Electrical Stimulation) effective in reducing shoulder pain/ 1b</p> <p>ESWT (Extracorporeal Shock Wave Therapy) is effective in reducing shoulder pain /1b</p> <p>FES (Functional Electrical Stimulation ) is effective in reducing subluxation and improving motor function/ 2</p> <p>TENS (Transcutaneous Electrical Nerve Stimulation) at high intensity effective in improving passive ROM of shoulder/2</p> <p>TENS is more effective than USG therapy in improving muscle strength and ROM/2</p> <p>HVPGS (High Voltage Pulsed Galvanic Current) effective in reducing subluxation and shoulder</p>	VAS<4

	displacement/ 2	
Botulinum toxin injections (Botox 100 U)- Pec major, Subscapularis, Infraspinatus	Reduce chronic hemishoulder pain and increase ROM when compared to intraarticular Triamcinolone Acetonide injection /1b	VAS>4
Steroid injections	Reduce shoulder pain/1a	VAS>4
Hyaluronic acid injections	Reduce shoulder pain/ 1b As effective as triamcinolone acetonide/2	VAS>4
Supra scapular nerve block	Reduce shoulder pain(does not improve ROM)/- /effective than saline injections or ultrasound therapy; comparable to intra articular steroid injection /1b and 2	VAS>4
Segmental neuro myotherapy	Improves motor function (not shoulder pain)/- /effective when compared to oral pain medications /1b	VAS<4
Complementary and alternative therapies	Acupuncture reduces pain, improves ROM and motor function/Effective than standard therapy/ 1a  Massage therapy, alone, or with Acupuncture reduces shoulder pain/1b and 2  Acupressure and aromatherapy in reducing pain/ effective than dry acupressure/1b	VAS<4

<b><u>Not recommended</u></b>		Conclusion
Surgery	Surgical resection of subscapularis and pectoral muscle improves ROM/4  Biceps tenodesis through delto pectoral approach reduces pain and subluxation /4	VAS>4
Positioning of shoulder	Does not reduce pain/1a	
Strapping of shoulder	Doesn't reduce spasticity, disability, ROM restriction or improve motor function	
Active therapies for shoulder	Doesn't reduce, but increase shoulder pain when compared to exercises alone/ 2  ROM exercises +USG therapy, positioning not more effective than ROM exercises alone/ 1b	



Electric stimulation	Surface NMES effective in reducing subluxation and improves ROM, but doesn't reduce pain / 1a and 2	

### **Nutritional Interventions Following Stroke**

#### Problem statement

The prevalence of malnutrition varies from 6 - 62% post stroke, depending on timing of assessment and criteria used to define malnutrition.<sup>47,48</sup> There is currently no “gold standard” for the assessment of nutritional status, and various methods of detection may be used. The metabolic requirements and the resting energy expenditures (REE) depend on the type of stroke with subarachnoid hemorrhage (SAH) requiring the most caloric intake when compared to ischemic strokes and intracerebral hemorrhage (ICH)<sup>49</sup>

In acute ischemic stroke patients, malnutrition on admission has been strongly associated with poor 1-month and 3-months outcome. Undernutrition as a predictor of poor clinical outcomes in acute ischemic stroke patients.<sup>50, 51</sup>

Intervention	Conclusion
Metabolic Rate Following Stroke	There is insufficient evidence regarding malnutrition during the acute phase of stroke.
Gastrointestinal Impairments Following Stroke	There is insufficient evidence regarding the development of significant gastrointestinal impairments poststroke.
constipation post stroke.	There is limited evidence suggesting that constipation can develop post stroke
Nutritional Intake Following Stroke	Patients consume 67% of their daily recommended intake during the first week post stroke, and up to 85% of their calorie requirements and 86% of their protein requirements during the first few weeks post stroke.
Glucose Regulation Following Stroke	

Glucose-potassium insulin injections	significantly reduce glucose levels and systolic blood pressure post stroke; no clinical benefits were observed. (1b )
Metformin	Ineffective in reducing glucose levels poststroke. (1b)
treadmill exercise	significantly reduces insulin levels but not glucose levels post stroke. (level 2)
impaired glucose regulation post stroke	Significantly greater risk for mortality than those with normal glucose regulation; no differences in dependency or stroke recurrence were observed. (level 3)
Vitamin D supplementation	single dose of Vitamin D2 (100,000 units) significantly increases 25-OH vitamin D levels for up to 16 weeks; no effects on blood pressures, cholesterol levels, and albumin levels were observed. (1b)
	Monthly dose of oral Vitamin D (60,000 units), along with a single injection of Vitamin D (600,000 units) and a daily dose of oral calcium (1g), increases 25-OH vitaminD levels and reduces risk of mortality when compared to no supplementation; no effects on odds of good outcome were observed (1b)
Lipid Profiles Following Stroke	
Atorvastatin 80mg/d	Effective in reducing total cholesterol and LDL levels and increasing HDL levels post stroke( 1b)
Enteral Feeding	gastric tube feeding such as PEG is associated with fewer mechanical complications and greater consumed intake post stroke compared to NG feeding. (1b)
Enteral protein supplementation	Does not differ significantly from standard enteral nutrition in its effect on malnutrition, based on biochemistry and/or body composition (1b)
Early enteral feeding	does not differ significantly from late or delayed enteral feeding in its effects on poor outcome post stroke. (1b, 2)

Oral nutritional Supplementation	Improves the calorie-protein intake of patients post stroke (1a)
	does not reduce the risk of death or dependency post stroke. (1a)
ALAnerv nutritional supplement (Alpha lipoic acid)	May significantly reduce total lipid levels and increase HDL levels compared to conventional treatment post stroke (1a)
Dysphagia Treatment	High-intensity dysphagia therapy results in improved swallowing function and less time to resuming a normal diet post stroke (1b)
Dysphagia therapy	does not reduce the risk of death or dependency post stroke, regardless of treatment intensity or diet type (1b, 2)
Long-Term Enteral Feeding	The one-year survival rate of patients with PEG feeding tubes post stroke varied from 16 - 67%, and functional recovery was reported in 2 - 28% of these patients.
NG tube feeding (long-term) post stroke	results in greater levels of malnutrition than oral feeding. (level 2)
Total Parenteral Nutrition	No evidence regarding the efficacy of total parenteral nutrition in the treatment of patients post stroke.

## Community Reintegration

This part follows a different format than the others. There are no assigned metrics. As the data is predominantly from countries with an established healthcare system, the suggestions are not easily applied to our setting. What follows is a set of problems, the result of an intervention, what is immediately practical in our setting, and finally what can be done to improve the situation.

When planning discharge social support and destination are critical to regular follow-up. Patients visiting tertiary centres may come from areas far away. Hiring an ambulance for follow-up visits will be challenging, hence establishing a referral network of likeminded Physiatrists is critical. Services that focus on the transition from hospital to homes/ subacute/ are valued high by the patients and caregivers. Regardless stroke survivors still have social barriers that negatively impact the reintegration within the community and in their homes. Measures that affect discharge destination are: admission FIM (motor and cognitive), age, and marital status. Currently each centre has its own unique way of ensuring continuity of care. Lack of homogeneity in this covers both the highlights and weaknesses of each.

Setting up a referral network, with a patient registry, and tele-med monthly can improve this. The goal should be to establish a PMR stroke rehabilitation network.

With respect to social support and functional status studies show high levels of social support may facilitate improved functional gains, mood, and social interactions. Moderate amounts of instrumental support and high amounts of emotional support are also effective. In India this is often not a problem. Patients whose children are abroad, unmarried individuals not in a family unit, those who can't afford care

Social Support improves physical recovery, and quality of life, while reducing psychological distress. The benefit seems linked to: presence, size, and its perceived effectiveness. The benefits are greater functional gains, less depression and improved mood and social interaction. This correlates to extent of services. A pet may improve physical, psychological, and social recovery. This depends on the individual.

In our setting optimally a caring family assumes role of social support. Stroke survivors should have a social support group centered around a PMR centre per taluk, with 2x/year meeting/ mela. Digital program, entertainment program during which QoL and medical screening can be checked. This must be lead by a PMR Dr.

Studies are ambivalent about the effect of Social Workers to improve independence or social activity by counseling, giving information and education to patients and families. What can we do better? 1:1/ group activity/ mela

Specialized Social Support Network Interventions do not improve perceived social support or functional recovery, even if it includes patient's social support network. Regardless some improvement in physical performance and instrumental activities of daily living for healthier, non-frail stroke survivors is seen. Similar programmes are useful. They can be conducted at taluk PMR centres.

Home-Based Support and Care Management do not improve social activity, mood, quality of life or physical independence. They do improve mental health though. Participation in a social worker led program of care coordination featuring frequent, regularly-scheduled contact is effective. Increasing knowledge about stroke and satisfaction with services is effective when delivered by stroke liaison worker or case manager. It will reduce caregiver burden or strain when delivered as social support interventions. It may improve mental health and reduce hospital readmission when delivered by occupational therapist led home-visits. We suggest making a standardized home care program and training course. This can be monitored and supported at rehab melas. A Physiatrist and MSW together can manage about 20 patients. The mela can function as a CBR. Active Case Management by an MSW does not improve social activity, quality of life, and mood.

Discharge Planning Programs to improve caregiver preparedness, quality of care, and patient outcomes if individualized, caregiver-oriented discharge planning are not effective, but may improve caregiver satisfaction with discharge needs.

Patient Education Programs to improve patients' and caregivers' well-being delivered to caregiver are not effective. They do improve stroke knowledge as a community-based nurse-led education program.

And do improve psychological functioning in both stroke individuals and their partners, as psychoeducational interventions. Re-integration guidelines are effective. We suggest using tele-health videos and interactive sessions?

Community Based Rehabilitation Programs will improve walking performance thereby mitigating the impact of stroke. As community walking programs they are more efficient than usual care. If patients can be linked through taluk PMR leaders they can form walking groups at local parks.

Day Services given within 6 months of stroke is associated with improved participation in leisure activities. This is often for FAC 5 patients with no CST lesions. They can be brought together for badminton, caroms, table tennis etc.

Bridges made a Self-Management Program to promote self-efficacy which was found to be not effective.

What was found was self-management programs work best when targeting specific rather than broad goals.

We know caregiving takes its toll on the caregiver. Commonly identified effects are: increasing psychological distress, increased financial burden, decreased social contact and activity, increased risk for depression, increased caregiver stress, strain or burden and an overall decrease in quality of life. Decreased social contact and activity in itself may contribute to increased caregiver strain, increased risk of depression and decreased life satisfaction. Reports concerning the influence of patient characteristics vary with the effect in question. Age, severity of stroke and stroke-related impairments, functional status and cognitive status do influence caregiver outcomes. The positive consequences of caregiving include improved appreciation of life, feeling needed or appreciated and development of a more positive outlook. Maintaining a positive attitude has been identified as an important coping strategy. Family Caregiver and Social Support Interventions improve stroke-related knowledge and family structure however, it may not have an impact on caregiver psychological health. We suggest improving caregiver QoL with counseling, skill training session, hands-on demo of DME, craft session. During melas addresses caregivers in separate sessions.

A personalized patient program in which the caregiver is included, improves social support and self-efficacy. These programs do not improve measures of function or affect though. Interactive educational resources and professional support accessed via online chat sessions, phones, message boards and educational videos may reduce depression in caregivers but has no impact on mastery, self-esteem, or

caregiver's outcomes. A caregiver-mediated home-based program involving exercise may improve measures of daily living in stroke patients.

Perceived family dysfunction is common post stroke. However, family function affects treatment adherence, performance of ADLs and social activity. Stroke patients do better with well-functioning families. Effective communication, good problem solving or adaptive coping, and strong emotional interest in each other characterize well-functioning families. This can be addressed in caregiver sessions during a mela.

#### Information Provision and Education

Psychoeducational interventions have no significant effect on the burden or health of caregivers but may benefit family functioning. Providing information and education through multimedia is better than single mode materials alone. Skills training is associated with a reduction in depression. Problem-solving intervention for caregivers is associated with a reduction in depression, life changes, and health. These benefits may not be maintained beyond 6 months. Training in basic nursing skills improves outcomes of depression, anxiety and quality of life for both the caregiver and the stroke patient. All these can be done during a mela

#### Perceived Need for Information, Education and Training

Although the receipt of information is of great importance to stroke patients and their families/caregivers, relatively few receive adequate information about topics they perceive to be important. Caregivers rarely receive adequate training in skills they require to care for the stroke survivor. Healthcare professionals involved in stroke care may acknowledge the importance of education for patients and carers; however, relatively few provide adequate information based upon the information needs of the recipients. In addition, written materials should be suited to the educational/reading level of the intended recipient. A care framework should be established with the Physiatrist as the locus of a communication network. Nurses doing home visits can implement and provide realtime feedback for patients, provide education, etc. The education plan should be in phases starting from: immediate at DC, at f/u 1mo , f/u 6mo, f/u 12 mo. The patients can be stratified by mRS and the modules provided accordingly.

Deterioration in social and leisure activities is common post-stroke and is greatest in women, the young and those who are better educated. Perceptions about how others view their disabilities and perceptions about how they will be able to cope post-stroke may influence the degree of social isolation experienced. When considered individually, there appears to be conflicting evidence as to the benefit of leisure therapy post-stroke and following discharge. However leisure therapy is associated with modest improvement in leisure activity. Participation in a leisure education program focused on awareness and competency development is associated with improvement in number and duration of activities and reduction in depressive symptoms. Participation in group education and exercise programs result in improved physical outcomes, but not social/leisure participation outcomes. Ideal leisure activity? Carom? Card games, reading, painting, 1 handed craft? In Mela we screen for patient interest and have appropriate craft option training

## Sexuality

A decrease in sexual activity is very common post-stroke. There is general agreement that sexual drive is still present and the main barriers to sexual activity are physical impairments and psychological factors, in particular a changed body image and lack of communication. Inappropriate sexual behaviour following stroke is not well studied. There may be an association between inappropriate sexual behaviour and the presence of right frontal lobe stroke and cognitive impairment. Sexual rehabilitation programs may not be effective in remediating sexual function. Sexual issues should be discussed during rehabilitation and addressed again after transition to the community when the stroke survivor and significant other are ready. These should be individualized per patient, start with providing information. The first question to ask is: Are you concerned about your sex life at present?

Patients for whom there is concern about their ability to drive need to be identified and proper assessment and treatment initiated. Determination of ability to drive should not rely solely on neuropsychologic testing or road test evaluation. Rather, a 2-step process is recommended in which the patient is first screened for readiness to participate in an on-road evaluation. In addition, provision of contextual driving therapy may be associated successful on-road evaluation. At taluk melas screen on when to restart driving.

Visual attention-retraining program is no more effective than traditional visuoperception retraining in improving the driving performance of patients with stroke. Simulator training program involving use of appropriate adaptations and driving through complex scenarios similar to real life is associated with improvement in driving fitness and successful on road evaluation. Dynavision training is not effective in improving the results of on-road assessments in individuals with stroke.

A substantial proportion of stroke survivors who were employed prior to the stroke event do not return to work. Factors influencing return to work include the severity of functional limitations, age and type of pre-stroke employment. A structured workplace intervention can improve return to work rates.

Those who worked prior to their stroke should, if their condition permits, be encouraged to be evaluated for their potential to return to work.

The physical limitations of stroke have a direct impact on the patient's ability to reintegrated back into the community. Accepting and adapting to a post-stroke status can mitigate the negative effects that come as a result of stroke. The individual characteristics of stroke patients such as optimism, determination, competitiveness, resilience and initiative can facilitate community reintegration.

Emotional and social support from family, friends and professionals plays a crucial role in reintegration success. Physical barriers and the lack of environmental accessibility limit one's ability to return in the community.

## **Young stroke**

The incidence of stroke in young patients varies from 3 to 44 out of 100,000 and has increased over time.

Unknown etiology	one third of strokes in young people
Hemorrhagic Etiology	most common causes include hypertension, arteriovenous malformation, ruptured aneurysm, or a combination of these factors.
Ischemic Stroke	majority of strokes in young patients are ischemic. Cardiac embolism is a frequent cause for patients younger than 40, while atherosclerosis is a common cause for patients aged 40-49.
Uncommon Etiologies	likely in stroke patients under the age of 30. migraines, non-atherosclerotic vasculopathy, mitral valve prolapse, multifocal intracranial stenosis, extracranial dissection, and cardioembolism

## **Modifiable risk factors**

<b>Risk factors</b>	
Smoking hypertension	most considerable risk factors
Hyperlipidemia, diabetes mellitus, and elevated plasma homocysteine level	risk factors for stroke in the young population, particularly for those older than 35.
Protein C and protein S deficiency	
Drug use	uncommon risk factor for stroke in general but is more common in the younger population
Alcohol-related stroke events	Related to the amount consumed: one to two alcoholic beverages daily may reduce the risk of stroke, while excessive alcohol consumption can be a significant risk factor for stroke.
Migraine with aura	young women at an elevated risk
Oral contraceptives	minor role in risk of stroke in the young population when paired with other factors
Chlamydia pneumoniae	risk factor for stroke in the young population

## **Non-Modifiable risk factors**



<b>Risk factors</b>	
Previous stroke	less common than in older patients
Gender	young patients under the age of 35 more likely to be female and above the age of 35 more likely to be male.
Atrial fibrillation	uncommon and understudied
Mitral valve prolapse	minimal risk factor and infrequent sole etiology
family history of stroke and patent foramen ovale	unclear
Pregnancy and postpartum state	unique periods of elevated stroke risk in young female patients

### Recovery and Prognosis

- better neurological recovery, less functional disability, and greater long-term survival

### Rehabilitation

- similar to that of older patients, with the main differences being the nature of neurological recovery and associated social issues.
- rehabilitation programs with an emphasis on socialization and community integration could be effective for young patients.

### Severe stroke

Definition: unconsciousness with severe unilateral or bilateral paresis at onset; early FIM® score <40 or motor FIM® score <37; high risk for failure to return home due to physical, cognitive, perceptual, and communication difficulties, or a combination of the above.

### Stroke Severity and Rehabilitation Outcomes.

More severe strokes, as determined upon admission, are associated with poorer outcomes after rehabilitation when compared with less severe strokes

### Benefits of Rehabilitation for Severe Strokes

<b>Modality</b>	<b>Conclusion</b>
specialized	-1a evidence that specialized interdisciplinary stroke rehabilitation

interdisciplinary stroke rehabilitation	reduces mortality in patients with severe stroke when compared to general rehabilitation programs.
	level 1b and limited level 2 evidence suggesting that patients with severe stroke who are admitted to specialized interdisciplinary stroke rehabilitation programs are more likely to be discharged home.
	level 1a and level 2 evidence regarding the effect of specialized interdisciplinary stroke rehabilitation programs on hospital length of stay.
	level 4 evidence regarding functional gains of persons with severe stroke following specialised interdisciplinary inpatient stroke rehabilitation.

## Medical complications of Stroke

Problem statement-

- Medical complications after stroke are common and influence outcome.
- Potentially serious complications include pneumonia, UTI, GI bleed, MI, DVT and pulmonary embolism.
- The presence of any in-hospital medical complication, many of which are preventable, has been associated with a significantly increased risk for 30-day readmission.

### Common medical complications of stroke

COMPLICATIONS	PERCENT
Falls	25
Urinary Tract Infection	24
Chest Infection	22
Pressure Sores	21
Depression	16
Shoulder Pain	9
Deep Vein Thrombosis	2
Pulmonary Embolism	1

### Serious medical complications of stroke

COMPLICATIONS	PERCENT
---------------	---------

All pneumonia	5
Aspiration pneumonia alone	3
Heart failure	3
Gastrointestinal bleeding	3
Cardiac arrest	2
Angina/cardiac ischaemia	1
Deep vein thrombosis	1
Pulmonary embolism	1
Hypoxia	1
Urinary tract infection	1
Sepsis	1
Cellulitis	1
Peripheral vascular disease	1
Dyspnea	1
Pulmonary edema	1
Dehydration	1

## 1. Bladder dysfunction

### Recommended

Intervention	Conclusion
Pelvic floor training	Improves muscle control and reduces urinary incontinence when compared to standard care (level 1a) Conflicting evidence as to whether it improves health-related quality of life (level 1b)
Traditional Chinese medicines	Reduce urinary incontinence but do not improve functional outcomes (level 1a)
Transcutaneous electrical nerve stimulation (TENS)	Reduces urinary incontinence when compared to no treatment, and that stimulation is more effective at 20Hz than 75Hz (level 1b)

Functionally-oriented rehabilitation program	Reduces urinary incontinence and improves wellbeing when compared to a conventional Bobath approach (limited level 2).
--	--

## Not Recommended

Intervention	Conclusion
Time of day for catheter removal	does not impact subsequent urinary incontinence (level 1b).
Bladder reconditioning prior to catheter removal	does not impact subsequent urinary incontinence (limited level 2)
Indwelling urinary catheters	are associated with worse outcomes, including urinary tract infections (level 3)

## 2. Bowel dysfunction

### Recommended

Intervention	Conclusion
Bowel program	Nursing program consisting of an assessment, educational material, diagnostic results, and treatment recommendations reduce constipation and fecal incontinence post stroke when compared to routine care(level 1 b)
Diakenchuto	traditional Japanese medicine, Diakenchuto, reduces constipation post stroke when compared to routine care(level 1b)
Tui-pushing and point sticking	protocol of tui-pushing and point sticking reduces constipation post stroke when compared to routine care(level 1 b)
Suppository use	bowel training is most efficient when coinciding with previous bowel regimens, but schedule of suppository use did not have an effect (limited level 2)

## 3. Venous thromboembolism

## Recommended

Intervention	Conclusion
IPC	Intermittent pneumatic compression reduces the incidence of deep vein thrombosis when compared to standard care (level 1 a), although there is limited Level 2 evidence that suggests otherwise.
Unfractionated heparin	*reduces the incidence of deep vein thrombosis when compared to placebo(level 2) *no more effective than intermittent pneumatic compression or neuromuscular electrical stimulation in reducing the incidence of deep vein thrombosis( level 2)
Graded compression stockings	Thigh-high graded compression stockings reduce the incidence of deep vein thrombosis when compared to below-knee stockings(level 1b)

## Neutral

Intervention	Conclusion
LMWH vs Unfractionated heparin/aspirin/placebo	Conflicting Level 1a evidence as to whether low molecular weight heparin is more effective than unfractionated heparin, aspirin, or placebo in reducing the incidence of deep vein thrombosis, without increasing the risk of bleeding complications.

## Not recommended

Intervention	Conclusion
Graded compression stockings	no more effective than standard care in reducing the incidence(level 1a)

## 4. Seizures

### Recommended

Intervention	Conclusion
are similar in reducing the rate of recurrent post-stroke seizures, but carbamazepine is more	are similar in reducing the rate of recurrent post-stroke seizures, but carbamazepine is more poorly tolerated (level 1b and 2)

poorly tolerated(level 1b and 2)	
----------------------------------	--

Not recommended

Intervention	Conclusion
Valproic acid	does not prevent post-stroke seizures when compared to placebo, but may confer neuroprotective effects(level 1b)

## 5. Osteoporosis

Recommended

Intervention	Conclusion
Bisphosphonates	preserve bone mineral density post stroke when compared to placebo, although there is limited Level 2 evidence that suggests otherwise (level 1a)
Vitamin D	preserves bone resorption and reduces the rate of fractures when compared to placebo. (level 1 b)
vitamin K	preserves bone mineral density and enhances bone metabolism when compared to no treatment (limited level 2)

Neutral

Intervention	Conclusion
Calcitonin	There is limited Level 2 evidence that calcitonin does not enhance bone metabolism when compared to placebo.

Not recommended

Intervention	Conclusion
Vitamin B	Does not reduce the rate of fractures when

	compared to placebo. (level 1b)
--	---------------------------------

## 6. Central pain

### Recommended

Intervention	Conclusion
Lamotrigine	reduces central pain post stroke when compared to placebo. (level 1 b)
Gabapentin	reduces central pain post stroke when compared to placebo (level 1 b)
Propofol	reduces central pain post stroke when compared to placebo (level 1b)
Levorphanol	high-dose levorphanol is more effective than low-dose Levorphanol in reducing central pain post stroke (level 1 b)
Apitoxin	apitoxin is more effective than saline during acupuncture in reducing central pain post stroke. (level 1 b)
rTMS	Level 1b and Level 2 evidence that high-frequency repetitive transcranial magnetic stimulation is more effective than low-frequency stimulation in reducing central pain post stroke

### Neutral

Intervention	Conclusion
Pregabalin	conflicting Level 1b evidence as to whether pregabalin is more effective than placebo in reducing central pain post stroke.
Amitriptyline	conflicting Level 1b evidence as to whether amitriptyline is more effective than placebo in reducing central pain post stroke.
rTMS	Level 1b and Level 2 evidence as to whether repetitive transcranial magnetic stimulation is more effective than sham stimulation in reducing central pain post stroke.

### Not recommended

Intervention	Conclusion
Levetiracetam	no more effective than placebo in reducing central pain post stroke. (level 1 b)

carbamazepine	no more effective than placebo in reducing central pain post stroke (level 1b).
Duloxetine	no more effective than placebo in reducing central pain post stroke (level 1b).
Ketamine	no more effective than placebo in reducing central pain post stroke (level 1b)
Morphine	no more effective than placebo in reducing central pain post stroke (level 1b)
Naloxone	no more effective than placebo in reducing central pain post stroke (level 2)

## 7. Fatigue

### Recommended

Intervention	Conclusion
Modafinil	reduces fatigue post stroke when compared to placebo (level 1b)
OSU-6162	reduces fatigue post stroke when compared to placebo (level 1b)
Cognitive therapy	combination of cognitive therapy and graded activity training reduced fatigue post stroke when compared to cognitive therapy alone (level 1b)
Astragalus membranaceus	a traditional Chinese herbal medicine, yields a short-term reduction in fatigue post stroke when compared to placebo.
Mindfulness	mindfulness-based stress reduction reduces fatigue post stroke when compared to no therapy (level 2)

### Not recommended

Intervention	Conclusion
Antidepressants	do not reduce fatigue post stroke (level 1b and level 2)
fatigue management program	does not reduce fatigue post stroke when compared to a stroke education program (level 2)



## 8. Insomnia

Acupuncture	intradermal acupuncture reduces insomnia when compared to sham acupuncture (level 1a)
Music therapy	acupuncture combined with music therapy reduces insomnia when compared to acupuncture alone (level 1b)

## Bibliography

1. (Incidence & prevalence of stroke in India: A systematic review. Suresh kumar Kamalakannan et al., Indian J Med Res. 2017 Aug; 146(2):175-185)
2. ( LuisaTerroni, Matildes F.M. Sobreiro Association among depression, cognitive impairment and executive dysfunction after stroke Dement Neuropsychol 2012 September;6(3):152-157, DOI: 10.1590/S1980-57642012DN06030007).
3. ( Jia-Hao Sun<sup>1</sup>, Lan Tan<sup>1,2,3</sup>, Jin-Tai Yu<sup>1</sup>Post-stroke cognitive impairment: epidemiology, mechanisms and management ,Ann Transl Med 2014;2(8):80).
4. (Tham et al., 2002)
5. (Lincoln NB, Gladman JR, Berman P, Luther A, Challen K. Rehabilitation needs of community stroke patients. DisabilRehabil. 1998;20(12):457–63. )
6. (Maaijwee et al., 2014
7. (Cicerone et al., 2011)
8. (Hachinski V. The 2005 Thomas Willis Lecture: stroke and vascular cognitive impairment: a transdisciplinary, translational and transactional approach. Stroke. 2007; 38(4):1396. [PubMed: 17347469] )
9. (Moorhouse P, Rockwood K. Vascular cognitive impairment: current concepts and clinical developments. Lancet Neurol. 2008; 7(3):246–255. [PubMed: 18275926])
10. (Wolfson C, Wolfson DB, Asgharian M, M'Lan CE, Ostbye T, Rockwood K, Hogan DB. Clinical Progression of Dementia Study Group. A revaluation of

- the duration of survival after the onset of dementia. *N Engl J Med.* 2001; 344(15):1111–1116. [PubMed: 11297701]
11. (Heiss W D, Kessler J., Theil A, et al: Differential capacity of left and right hemispheric areas for compensation of post stroke aphasia. *Ann. Neurol.* 1999; pp430-438.
  12. Thompson CK, and den Ouden DB: Neuroimaging and recovery of language in aphasia. *Curr.Neurol.Neurosci. Rep* 2008; pp. 475-483)
  13. (Bakas T, Kroenke K, Plue LD, Perkins SM, Williams LS. Outcomes among family caregivers of aphasic versus nonaphasic stroke survivors. *RehabilNurs.* 2006;31(1):33–42.)
  14. (Brady MC, Kelly H, Godwin J, Enderby P, Campbell P. Speech and language therapy for aphasia following stroke. *Cochrane Database Syst Rev.* 2016;(6)
  15. (Cerebrovascular Disorders (D Jamieson, Section Editor)Published: 25 June 2019) Treatment of Post-Stroke Depression
  16. (Armstrong JR, Mosher BD. Aspiration pneumonia after stroke: intervention and prevention. *Neurohospitalist.* 2011;1(2):85-93. doi:10.1177/1941875210395775)
  17. (Armstrong JR, Mosher BD. Aspiration pneumonia after stroke: intervention and prevention. *Neurohospitalist.* 2011;1(2):85-93. doi:10.1177/1941875210395775)
  18. (Arnold M, Liesirova K, Broeg-Morvay A, et al. Dysphagia in Acute Stroke: Incidence, Burden and Impact on Clinical Outcome. *PLoS One.* 2016;11(2):e0148424. Published 2016 Feb 10. doi:10.1371/journal.pone.0148424)
  19. (Arnold M, Liesirova K, Broeg-Morvay A, et al. Dysphagia in Acute Stroke: Incidence, Burden and Impact on Clinical Outcome. *PLoS One.* 2016;11(2):e0148424. Published 2016 Feb 10. doi:10.1371/journal.pone.0148424)
  20. (Armstrong JR, Mosher BD. Aspiration pneumonia after stroke: intervention and prevention. *Neurohospitalist.* 2011;1(2):85-93. doi:10.1177/1941875210395775)
  21. (Armstrong JR, Mosher BD. Aspiration pneumonia after stroke: intervention and prevention. *Neurohospitalist.* 2011;1(2):85-93. doi:10.1177/1941875210395775)
  22. (Armstrong JR, Mosher BD. Aspiration pneumonia after stroke: intervention and prevention. *Neurohospitalist.* 2011;1(2):85-93. doi:10.1177/1941875210395775)
  23. (Lee KM, Kim HJ. Practical Assessment of Dysphagia in Stroke Patients. *Ann Rehabil Med.* 2015;39(6):1018-1027. doi:10.5535/arm.2015.39.6.1018)
  24. (Geeganage C, Beavan J, Ellender S, Bath PM. Interventions for dysphagia and nutritional support in acute and subacute stroke. *Cochrane Database Syst Rev.* 2012;10:CD000323.
  25. Dennis MS, Lewis SC, Warlow C; FOOD Trial Collaboration. Effect of timing and method of enteral tube feeding for dysphagic stroke patients (FOOD): a multicentre randomised controlled trial. *Lancet.* 2005;365:764–772. doi: 10.1016/S0140-6736(05)17983-5.)
  26. (Rapport et al., 1993)

27. (Broeks JG, Lankhorst GJ, Rumping K, Prevo AJ. The long-term outcome of arm function after stroke: results of a follow-up study. *Disabil Rehabil.* 1999;21:357–364.)
28. (Kwakkel G, Kollen BJ, van der Grond J, Prevo AJH. Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. *Stroke.* 2003;34:2181–2186.)
29. (Nakayama H, Jørgensen HS, Raaschou HO, Olsen TS. Recovery of upper extremity function in stroke patients: the Copenhagen Stroke Study. *Arch Phys Med Rehabil.* 1994;75:394–398.)
30. (Sunderland A et al. Enhanced physical therapy for arm function after stroke: a one year follow up study. *J. Neurol. Neurosurg. Psychiatr.* 1994;57:856–858.)
31. (Wade DT, Langton-Hewer R, Wood VA, Skilbeck CE, Ismail HM. The hemiplegic arm after stroke: measurement and recovery. *J. Neurol. Neurosurg. Psychiatr.* 1983;46:521–524)
32. (Dean C, Mackey F. Motor assessment scale scores as a measure of rehabilitation outcome following stroke. *Australian Journal of Physiotherapy* 1992;38:31–35.)
33. (Gillot, A.J.; Holder-Walls, A.; Kurtz, J.R.; Varley, N.C. Perceptions and experiences of two survivors of stroke who participated in constraint-induced movement therapy home programs. *Am. J. Occup. Ther.* **2003**, 57,168–176.)
34. (Lawrence, E.S.; Coshall, C.; Dundas, R.; Stewart, J.; Rudd, A.G.; Howard, R.; Wolfe, C.D. Estimates of the prevalence of acute stroke)
35. (Brainin M, Norrving B, Sunnerhagen KS, et al. Poststroke chronic disease management: towards improved identification and interventions for poststroke spasticity-related complications. *Int J Stroke.* 2011;6:42–46.)
36. (Duncan et al., <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6088193/#B16>> 2005)
37. (Hendricks et al., <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6088193/#B24>> 2002).
38. (Olney and Richards, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6088193/#B26>> 2002)
39. (Dobkin, <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6088193/#B14>> 2005; Batchelor et al., <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6088193/#B4>> 2012).
40. (Roy CW et al Shoulder pain in acutely admitted hemiplegics *Clin Rehabil* 1994; Ward AB. Hemiplegic shoulder pain. *J Neurol Neurosurg Psychiatry* 2007;
41. VanOuwenaller C, Laplace PM, Chantraine A. Painful shoulder in hemiplegia. *Arch Phys Med Rehabil* 1986)
42. (Black-Schaffer RM, Kirsteins AE, Harvey RL. Stroke rehabilitation. Co-morbidities and complications. *Arch Phys Med Rehabil.* 1999)
43. (Bohannon RW, Larkin PA, Smith MB, Horton MG. Shoulder pain in hemiplegia: Statistical relationship with five variables. *Arch Phys Med Rehabil.* 1986)
44. (Geurts AC, Visschers BA, van Limbeek J, Ribbers GM. Systematic review of aetiology and treatment of post-stroke hand oedema and shoulder-hand syndrome)

45. (Griffin JW, hemiplegic shoulder pain, Phys Ther 1986)
46. A review of the relationship between dysphagia and malnutrition following stroke Foley NC, Martin RE, Salter KL, Teasell RW :J Rehabil Med. 2009 Sep; 41(9):707-13.,
47. Prevalence of malnutrition and its risk factors in stroke patients residing in an infirmary :Chai J, Chu FC, Chow TW, Shum NC ; Singapore Med J. 2008 Apr; 49(4):290-6.
48. Malnutrition in Stroke Patients: Risk Factors, Assessment, and Management Toni Sabbouh, MD1 and Michel T Torbey, MD, MPH, FCCM, FAHA, FNCS1 :Neurocrit Care. 2018 Dec; 29(3): 374–384.
49. Yoo SH, Kim JS, Kwon SU, Yun SC, Koh JY, Kang DW -rch Neurol. 2008 Jan; 65(1):39-43 . Impact of premorbid undernutrition on outcome in stroke patients.
50. Davis JP, Wong AA, Schluter PJ, Henderson RD, O'Sullivan JD, Read SJ-Stroke. 2004 Aug; 35(8):1930-4.