EVIDENCE-BASED REVIEW OF STROKE REHABILITATION (18th Edition)

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Executive Summary

The Stroke Rehabilitation Evidence-Based Review (SREBR) reviews techniques, therapies, devices, procedures and medications associated with stroke rehabilitation. The purpose of the Evidence-Based Review of Stroke Rehabilitation was to fulfil the 12th recommendation of The Stroke Rehabilitation Consensus Panel Report that supported the continuing review of stroke rehabilitation research with the *"purpose of maintaining timely and accurate information on effective stroke rehabilitation, identifying ideas for further research, supporting continuous peer-review and encouraging improved evidence-based practice."* The aim of the SREBR was to:

- Be an up-to-date review of the current evidence in stroke rehabilitation.
- Provide a comprehensive and accessible review to facilitate best-practice.
- Provide specific conclusion based on evidence that could be used to help direct stroke care at the bedside and at home.

Since its original publication in April 2002, the SREBR has undergone eighteen major revisions and now includes articles published up to December 2016. To date, we have included over 2,300 randomized controlled trials (RCTs).

Methods

For the first edition of the SREBR a literature search using multiple databases (MEDLINE, EBASE, MANTIS, PASCAL, and Sci Search) was conducted to identify all potential trials published from 1970-2001, regardless of study design. The search was restricted to the English language and excluded animal studies. Search terms included, but were not restricted to: *"stroke", "cerebrovascular accident", "cerebrovascular disorder", "rehabilitation", "physiotherapy", "occupational therapy", "speech therapy", "recreation therapy"*.

From 2001 onwards, the authors of each of the modules have conducted their own searches. Databases used include EMBASE, CINAHL, PubMED, ProQuest, PsycINFO, AMED, and Scopus. Key terms were tailored to identify potential trials within each subsection of every module. Depending on the breadth of the current evidence, searches may have been restricted to randomized controlled trials, since they are given the greatest emphasis when formulating conclusions. This review was restricted to published works. Although it was not confined to the results from randomized controlled trials (RCT), these articles received priority when formulating conclusions. Systematic reviews and meta-analyses were also incorporated in the content of the modules. The 18th version of the SREBR contains published literature up to December 2016.

Data Extraction and Quality Assessment Tool

Two abstractors, each blinded to the others' results reviewed each article independently. Reviewers collected data relating to the study methodology, outcome measures, results, and final conclusions as well as quantitatively evaluated the study's methodological quality using the Physiotherapy Evidence Database (PEDro) scale, developed by the Centre for Evidence-Based Physiotherapy (CEBP) in Australia.

The PEDro Scale consists of 10 quality ratings each receiving either a yes or no score:

1. Subjects were randomly allocated to groups (in a crossover study, subjects were randomly allocated an order in which treatments were received).
2. Allocation was concealed.
3. The groups were similar at baseline regarding the most important prognostic indicators.
4. There was blinding of all subjects.
5. There was blinding of all therapists who administered the therapy.
6. There was blinding of all assessors who measured at least one key outcome.
7. Measures of at least one key outcome were obtained from more than 85% of the subjects initially allocated to groups (*).
8. All subjects for whom outcome measures were available received the treatment or control condition as allocated or, where this was not the case, data for at least one key outcome was analysed by "intention to treat".
9. The results of between-group statistical comparisons are reported for at least one key outcome.
10. The study provides both point measures and measures of variability for at least one key outcome.

(*) For the purposes of this review, follow-up was considered adequate if all of the subjects that had been originally randomized could be accounted for at the end of the study period.

The maximum score a study could receive was 10. Two independent raters reviewed each article. Scoring discrepancies were resolved through discussion.

Formulating Conclusions Based on Levels of Evidence

There are many systems currently available to summarize a body of knowledge and to establish levels of evidence. Some of these are increasingly complex, requiring a specialized body of knowledge for correct interpretation. With our focus on ease and accessibility, we intentionally chose a system that was simple and straight-forward. The levels of evidence used to summarize the findings are based on the levels of evidence developed by Sackett et al. (2000). For the purpose of this review, a simplified version of the categories used by Sackett et al. (2000) was adopted. Instead of the original 10 scoring categories, we developed a scoring system ranging from a level 1 evidence to a level 5 evidence, and added descriptions to each category to help designate the appropriate level of evidence based on the type of research design. In the Version 4.0 of this grading scheme used in this review, the evidence level of 1 category is further divided into 2 subcategories to distinguish between a single RCT with a PEDro score ≥ 6 (Level 1b), and 2 or more RCTs with PEDro scores ≥ 6 (Level 1a).
The modified Sackett Scale version 4.0 consists of the following levels of evidence:

- **Level 1a**
  - More than one higher RCT with PEDro score ≥ 6. Includes within subjects comparison with randomized conditions and cross-over designs.
- **Level 1b**

- One higher RCT with PEDro score ≥ 6.

- **Level 2**
  - Lower RCT(s) with PEDro score <6.
  - Prospective controlled trial(s).
  - Prospective cohort (longitudinal) study using at least 2 similar groups with one exposed to a particular condition.

- **Level 3**
  - A retrospective case control study comparing conditions, including historical cohorts.

- **Level 4**
  - A prospective pre-post trial with a baseline measure, intervention, and a post-test using a single group of subjects.
  - A prospective post-test with two or more groups (intervention followed by post-test and no re-test or baseline measures) using a single group of subjects.
  - A retrospective case series usually collecting variables from a chart review.

- **Level 5**
  - An observational study using cross-sectional analysis to interpret relations.
  - A clinical consensus (expert opinion) without explicit critical appraisal, or based on physiology, biomechanics or "first principles".
  - A case report involving one subject.

Meta-analyses, conducted by the authors of this review have also been included in modules 8, 15, 16, 17 and 18.

Using this system, conclusions were easily arrived at when the results of multiple studies were in agreement. However, interpretation became difficult when the study results conflicted. In cases where RCTs also differed in terms of methodological quality, the results of the study (or studies) with the higher PEDro score(s) and statistical power (i.e. large sample size) were more heavily weighted to arrive at the final conclusions. However, there were still some instances where interpretation remained problematic. For instance, the authors needed to make a judgement when the results of a single study of higher quality conflicted with those of several studies of inferior quality. In these cases, we attempted to provide a rationale for our decision and to make the process as transparent as possible. In the end, the reader is encouraged to be a "critical consumer" of all of the material presented.

Levels of Evidence

Levels of evidence were generated based on the modified Sacket's Scale described above for literature presented in modules 4 through to 22, with the exception of module 20 which summarized rehabilitation outcome measures.

Modules

4) Managing the stroke rehabilitation triage process
5) The efficacy of stroke rehabilitation
6) The elements of stroke rehabilitation
7) Outpatient stroke rehabilitation
8) Secondary prevention of stroke
9) Lower extremity interventions
10) Upper extremity interventions

11) Hemiplegic shoulder pain & complex regional pain syndrome
12) Post-stroke cognitive disorders
13) Perceptual disorders
14) Aphasia and apraxia
15) Dysphagia and aspiration following stroke
16) Nutritional interventions following stroke
17) Medical complications post stroke
18) Post-stroke depression and mood disorders
19) Community reintegration
21) Rehabilitation of younger patients post stroke
22) The rehabilitation of severe stroke

The following brief summaries highlight the information provided in the SREBR and provide conclusions regarding treatments involved in stroke rehabilitation. The entire evidence-based review is available at: http://www.ebrsr.com

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4. Managing the Stroke Rehabilitation Triage Process

Predictors of Functional Outcomes
The two most powerful predictors of functional recovery and eventual discharge home are age and initial stroke severity, with the latter being the most important. However, this does not preclude the use of additional factors to determine appropriate stroke rehabilitation destination during triage.

Severity of Stroke and Impact of Rehabilitation
There is Level 3 evidence that severity of stroke predicts ability to participate and benefit from stroke rehabilitation.

Impact of Age on Recovery
There is Level 3 evidence that severity of stroke predicts ability to participate and benefit from stroke rehabilitation.

5. The Efficacy of Stroke Rehabilitation

Acute Care
Based on the results from meta-analyses, there is Level 1a evidence that acute stroke care is associated with a reduction in death/dependency and institutionalization, but not with reductions in mortality or length of stay, when compared to alternative care.

There is Level 1a evidence that acute stroke care is not associated with a reduction in functional disability when compared to alternative interventions.

Combined Acute and Rehabilitation Stroke Units
Based on the results from meta-analyses, there is Level 1a evidence that combined acute and rehabilitation stroke units are associated with reductions in death/dependency, institutionalization, and length of stay, but not with reduced mortality, compared to general medical wards.

There is Level 1a evidence that combined stroke units are associated with improved functional outcome compared to general medical wards.

Subacute Rehabilitation Units
Based on the results from meta-analyses, there is Level 1a evidence that specialized, interdisciplinary rehabilitation provided in the subacute phase is associated with reductions in mortality and death/dependency, but not with reduced institutionalization or length of stay, compared to conventional care on a general medical ward.

There is Level 1a evidence that for the subset of more severe stroke patients, specialized stroke rehabilitation reduces mortality but does not result in improved functional outcomes or reduced institutionalization compared to conventional care.

There is Level 1a evidence that for patients with moderately severe stroke, specialized rehabilitation improves functional outcomes but does not reduce mortality compared to conventional care.

There is Level 1a evidence that for patients with mild stroke, specialized rehabilitation does not improve functional outcome or reduce mortality compared to conventional care.

There is Level 1b evidence that 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.

Mobile Stroke Teams
Based on the results from meta-analyses, there is Level 1a evidence that mobile stroke teams do not reduce mortality, combined death/dependency, institutionalization, or length of stay.

Overall
There is Level 1a evidence that overall, specialized stroke care is associated with reductions in the odds of mortality, combined death/dependency, institutionalization, and length of stay.

6. The Elements of Stroke Rehabilitation

Care Pathways in Stroke Rehabilitation
There is level 1a, level 2, and level 3 evidence that 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.

There is level 1a, level 2, and level 3 evidence that integrated care pathways are not associated with reduced length of stay, readmission rate, or complication rate, when compared to conventional care.

There is level 1b and level 2 evidence that greater adherence to care guidelines, treatment protocols, and organizational quality is associated with better clinical outcomes.

Timing to Stroke Rehabilitation
There is level 1a evidence that earlier admission to rehabilitation results in improved overall functional outcomes.

There is level 1a evidence that 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.

Intensity of Therapy
There is level 1a evidence that greater intensities of physiotherapy and occupational therapy results in improved functional outcomes.

There is level 1a evidence that the 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.

There is level 1a evidence that additional caregiver-supported therapy results in improved functional outcomes compared to conventional therapy alone.

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

Durability of Rehabilitation Gains
There is level 1a evidence that relatively greater functional improvements are made by patients rehabilitated on specialized stroke units when compared to general medical units in the long term.

There is level 1a evidence that functional outcomes achieved through stroke rehabilitation are maintained for up to one year post stroke.

There is level 1b evidence that 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.

7. Outpatient Stroke Rehabilitation

Early Supported Discharge

There is level 1a evidence that 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.

There is level 1a evidence that the cost associated with early-supported discharge is lower when compared to usual care; however, savings are generally not dramatic or consistent across the studies.

Outpatient Rehabilitation Provided Within the First 6 Months of Stroke Onset

There is conflicting level 1a evidence that additional outpatient therapy improves performance of ADLs.

Outpatient Rehabilitation Provided at Least One Year Following Stroke

There is conflicting level 1a evidence regarding the association between home based therapy for chronic stroke survivors and improvements in performance on ADLs and mobility.

Rehabilitation in the Home or in the Hospital

There is level 1a and level 2 evidence that home-based and hospital-based outpatient stroke rehabilitation programs are equally effective in achieving modest gains in ADL following inpatient rehabilitation.

8. Secondary Prevention

Risk Factor Management

There is level 2 and level 4 evidence that urgent assessment and initiation of treatment following transient ischemic attack is associated with reduced hospital costs, length of stay and risk for early stroke.

There is conflicting level 1b evidence that treatment of patients using an accelerated protocol in an emergency department observation unit results in shorter lengths of stay and reduced costs, but does not result in an improved risk for stroke when compared to inpatient admission for transient ischemic attack.

There is level 1a evidence that personalized secondary preventative care management programs may not improve risk factor management.

There is level 1b evidence that the addition of a positive affirmation intervention to educational materials focussed on self-management and level 2 evidence that a detailed history of medication provided to the GP versus only a basic record of medication at discharge may improve adherence to statins, antihypertensive and antithrombotic medications.

There is level 1b and level 2 evidence that a pharmacist-led educational intervention, a stroke prevention group workshop or post-discharge management of risk factors conducted using a model of

shared care may improve long-term benefits in terms of blood pressure reduction, reduced lipid levels, reduced body mass and increased physical activity.

There is level 1b evidence that recording stroke-related events with an electronic support tool or pharmacist-led care management with direct prescription of medication (versus nurse-led management) may not improve stroke or cardiovascular risk management.

There is level 2 evidence that specialist nurse follow-up three months post-stroke or administration of the PROTECT program may improve health outcomes and short-term risk of myocardial infarction, respectively.

There is level 1b evidence that standardized discharge orders are not associated with improved secondary prevention treatment at six months' post-discharge.

Hypertension

There is level 1a evidence that incidence of cardiovascular events, fatal or nonfatal stroke and mortality were reduced by commonly used antihypertensive agents. Furthermore, larger reductions of BP were associated with greater reductions in risk.

There is level 1b evidence that a reduction in blood pressure is associated with a decreased risk of stroke particularly among patients with a previous history of intracerebral haemorrhage.

There is level 1a evidence that the use of an ACE-I and diuretic together may result in the greatest reductions of stroke, myocardial infarction and all vascular events compared to ACE-Is, diuretics and β-receptor agonists used alone.

There is level 1a evidence that diuretics at high doses, diuretics at low doses (i.e. Thiazides, Chlorthalidone, and Indapamide), beta-blockers, calcium antagonists, ACE inhibitors, angiotensin receptor blockers, and centrally acting drugs are more effective than the control therapy at reducing the relative risk of stroke.

There is level 1a evidence that only Chlorthalidone at low doses and ACE inhibitors are superior to the control therapy at lowering the relative risk of coronary heart disease.

There is level 1a evidence that Chlorthalidone at low doses, beta-blockers, ACE inhibitors, angiotensin receptor blockers, and centrally acting drugs are more effective than the control at reducing the occurrence of heart failure.

There is level 1a evidence that a composite of stroke and coronary heart disease can be significantly lowered by diuretics delivered at high and low doses (i.e. Thiazides, Chlorthalidone, and Indapamide), beta-blockers, calcium antagonists, ACE inhibitors, angiotensin receptor blockers, and centrally acting drugs, relative to control therapy.

There is level 1a evidence that a composite of stroke, coronary heart disease, and heart failure can be significantly lowered by diuretics delivered at high and low doses (i.e. Thiazides, and Chlorthalidone), beta-blockers, calcium antagonists, ACE inhibitors, angiotensin receptor blockers, and centrally acting drugs, relative to control therapy.

There is level 1a evidence that cardiovascular death can be significantly reduce by Thiazides at low doses, calcium antagonists, and centrally acting drugs, while all-cause mortality can only be significantly reduced by the use of low dose Indapamide and calcium antagonist, when compared to control therapy.

There is level 1b evidence that combination therapy with telmisartan (angiotensin receptor blocker) and Ramipril (ACE inhibitor) is associated with increased symptoms of hypotension, syncope, and renal dysfunction.

Versus placebo, there is level 1b evidence that ramipril (ACE inhibitor) and nitrendipine may reduce the incidence of cardiovascular and stroke events as well as subsequent mortality (particularly among diabetics). Additional level 1b evidence suggests that aspirin may improve odds of stroke among patients with pre-existing ischemic heart disease and BP ≤80mmHg while vorapaxar (PAR-1 receptor inhibitor) may not improve stroke or cardiovascular risk.

There is level 1b evidence that chlorthalidone (diuretic) may be superior to both doxazosin (α-adrenergic blocker) for stroke and cardiovascular risk management.

There is level 1b evidence suggesting that captopril (ACE-inhibitor) may reduce the incidence of stroke when compared to beta-blockers and/or diuretics. Additional level 1b evidence suggests that perindopril (ACE-I) may significantly improve blood pressure while also reducing risk of stroke however, this drug may have no effect on cardiovascular endpoints.

Management of Diabetes and Associated Macrovascular Complications

There is level 1a and level 1b evidence that pioglitazone may not be associated with a relative reduction in the risk of stroke; however, it may be effective at lowering the composite risk of stroke, myocardial infarction, and death.

There is level 1b evidence that in patients with no history of previous stroke, pioglitazone was not effective at reducing the risk of stroke however, in patients with a history of stroke, the use of pioglitazone was associated with a reduction in the risk of a recurrent stroke.

There is level 1a evidence that intense glucose lowering therapy is not significantly different than standard therapy for reducing the risk of stroke. Intensive glucose lowering therapy may only be an effective treatment for type 2 diabetes and for patients with a history of macrovascular events.

There is level 1b evidence that empagliflozin was not significantly different than placebo therapy at reducing the relative risk of stroke; however, more research is needed to identify the mechanism of action of metformin and potential benefits on cardiovascular health.

There is level 1a evidence that metformin has no additional benefits on cardiovascular health other than reducing blood glucose levels for the treatment of type 2 diabetes.

There is level 1a evidence that treatment of hypertension in diabetic patients reduces the risk of stroke. Furthermore, tighter control of blood pressure is associated with greater reduction of risk for stroke compared to “less tight” therapy; however, greater risk of adverse events may be associated with aggressive therapy.

There is level 1b evidence that perindopril (angiotensin converting enzyme inhibitor, ACE-I) administered with indapamide (diuretic) may not be superior to placebo therapy at reducing the incidence of macrovascular or cerebrovascular events.

There is level 1b evidence that nitrendipine (ca-channel blocker, CCB) improves risk of cardiovascular events and mortality compared to placebo.

There is level 1b evidence that ramipril (ACE-I) alone improves a combined outcome of myocardial infarction, stroke and cardiovascular mortality.

There is level 1a evidence suggesting that ACE-Is may improve the incidence of major vascular events, especially myocardial infarction, when compared to CCBs.

There is level 1b evidence that amlodipine besylate (CCB) or lisinopril (ACE-I) may not reduce the risk of cardiovascular mortality or nonfatal myocardial infarction when compared to chlorthalidone (diuretic) among patients with diabetes.

There is level 1b evidence that treatments with CCB and ACE-I provide no additional benefit over conventional therapy in terms of preventing the occurrence of macrovascular events including stroke in individuals with Type 2 diabetes.

There is level 1b evidence that valsartan (angiotensin receptor blocker) is as effective as amlodipine (CCB) at reduction of risk for macrovascular events or cardiac complications. Use of this amlodipine may be associated with increased risk for hospitalization due to heart failure.

There is level 1a evidence that all hypertensive medications reduce the risk of stroke, especially among patients with diabetes.

There is conflicting level 1b evidence regarding the effectiveness of pravastatin for the prevention of stroke and composite endpoints of coronary and cardiac complications.

There is conflicting level 1b evidence regarding the efficacy of atorvastatin in the secondary prevention of stroke and cardiovascular complications.

There is level 1b evidence that simvastatin may reduce the odds of stroke as well as the incidence of major coronary and atherosclerotic events when compared to placebo.

There is level 1b evidence that a structured care intervention for hyperlipidemia using atorvastatin and strict implementation of guidelines may decrease mortality, coronary morbidity, and incidence of stroke versus usual care.

There is level 1a evidence that statin treatment in patients with diabetes may reduce the risk of stroke; however, in patients with diabetes and existing coronary heart disease, statin treatments only reduced the risk of subsequent coronary heart disease but not stroke.

There is level 1a evidence that fibrate treatment may not reduce the risk of stroke or coronary events.

There is conflicting level 1b evidence regarding the effect of gemfibrozil on lowering the risk of stroke in patients with diabetes.

There is level 1a evidence that fenofibrate and simvastatin combination therapy or fenofibrate treatment alone may not be more efficacious in the prevention of stroke and cardiovascular events when compared to simvastatin monotherapy or placebo. Additional level 1b evidence suggests that unaccompanied fenofibrate administration may decrease the risk of nonfatal myocardial infarction.

There is level 1b evidence that bezafibrate may not improve incidence of myocardial infarction or stroke.

Hyperlipidemia

There is level 1a evidence that statin therapy is effective at lowering the risk of further strokes however, it may not reduce the risk of intracerebral hemorrhage.

There is level 1a evidence that intensive statin therapy may be more effective than less intense therapy in reducing risk for ischemic stroke events.

There is level 1a evidence that statin therapy may not reduce stroke-related mortality, however the evidence is unclear regarding its effects on all-cause mortality.

There is level 1b evidence that withdrawal of statin treatment at the time of acute stroke is associated with increased risk for death and dependency when compared to continuous statin use.

There is level 1b evidence that pre-treatment with atorvastatin may not improve ischemic or haemorrhagic stroke outcome when compared to placebo.

There is level 2 and level 3 evidence that pre-stroke treatment with statins may improve functional disability on the Barthel Index but may not improve stroke severity on the National Institutes of Health Stroke Scale when compared to no statin pre-treatment. Conflicting level 2 and level 3 evidence suggests no consistent data for functional independence on the Modified Rankin Scale or mortality up to 6 months.

Macrolide Antibiotics the Prevention of Cardiovascular Events

There is level 1a and level 1b evidence that azithromycin or roxithromycin (macrolide antibiotic) may not decrease the incidence of cardiovascular events

Lifestyle Modification

There is level 1a evidence that engaging in physical activity is associated with substantial benefits in terms of a reduced risk for stroke and cardiovascular disease. A dose-response relationship may exist between exercise and stroke risk. Conflicting level 1a evidence from a meta-analysis of 10 cohort studies suggests that this relationship may only be significant for men.

There is level 1a evidence that moderate to high levels of leisure and occupational activity may be beneficial for a reduced rate of cardiovascular disease compared to low level exercise.

There is level 1b evidence that a detailed, personalized activity program with regular verbal instruction and encouragement does not effectively increase level of physical activity when compared to the provision of basic information regarding physical activity and no training program.

There is level 1a evidence that low-fat, low-cholesterol diets rich in fruits, vegetables and low-fat dairy products are effective in reducing blood pressure when compared to control diets low in fruits and vegetables, and with average fat content.

There is level 1a evidence that Mediterranean type diets (rich in whole grains, fruits, vegetables, legumes, walnuts, almonds, and alpha-linolenic acid) may improve blood pressure and reduce risk of cardiovascular events including stroke when compared to a prudent type diet.

There is level 1a evidence that the use of vitamin C and vitamin E together may reduce atherosclerotic progression.

There is level 1a evidence that vitamin E may not affect the incidence of cerebrovascular accidents, and all-cause/cardiovascular mortality while use of β-carotene may be associated with an increase in cardiovascular and all-cause mortality when compared to control.

There is conflicting level 1b evidence suggesting variable efficacy of daily antioxidant vitamins (vitamin E, vitamin C and β-carotene) when used alone on clinical cardiovascular endpoints including stroke, and mortality. Additional level 1b evidence suggests a beneficial effect of combinatorial therapy with ascorbic acid (vitamin C) and vitamin E on stroke risk.

There is level 1a evidence that vitamin B therapy may improve flow-mediated dilation (FMD) in the short-term however, no long-term effects on FMD or carotid intima-media thickness are observed.

There is conflicting level 1a evidence regarding the effect of B-vitamins (folic acid, vitamin B6 and B12) on cardiovascular outcome or risk of stroke.

There is level 1a evidence that supplementation with folic acid and vitamins B_6 and B_{12} is associated with significant reductions in plasma homocysteine levels (tHcy) up to one year from baseline.

There is level 1b evidence that folic acid alone may have no effect on a combined cardiovascular outcome when compared to standard care.

There is level 1b evidence that high dose vitamin B therapy concurrent with antiplatelets may increase risk of stroke versus low dose therapy. There may be no effect on incidence of stroke or a cardiovascular composite endpoint among patients not supplementing vitamin therapy with antiplatelets.

There is level 1b evidence that homocysteine-lowering therapy with B-vitamins may not improve the risk of recurrent stroke, stroke severity or functional outcome when compared to placebo.

There is level 1b evidence that high dose homocysteine-lowering therapy may improve risk of stroke, myocardial infarction or death in patients ≥67 years old versus low dose treatment.

There is level 1a evidence that smoking or exposure to environmental tobacco smoke may increase risk of stroke in a dose-dependent manner.

There is level 1b evidence that an intensive smoking cessation program providing a period of counselling and support may be as effective as a minimal intervention providing a single 30-minute session of counselling only.

There is level 1a evidence that light (1-2 drinks per day) alcohol consumption reduces the risk for ischemic stroke while heavy drinking (>5 drinks per day) and binge-drinking increase the risk of haemorrhagic stroke in a linear dose-dependent fashion.

There is level 1b evidence that a multi-factorial behavioural intervention focussing on eating habits and smoking cessation may substantially improve smoking cessation, mortality, and serum cholesterol and glucose concentrations, and reduce the risk of cardiovascular events.

There is level 1b evidence that a program of e-counselling that promotes self-directed lifestyle change in the area of diet, exercise and smoking cessation may be associated with reductions in systolic blood pressure and total cholesterol.

There is level 2 evidence that the Secondary Stroke Prevention Program (STOP) may improve stroke knowledge, smoking cessation and alcohol use when compared to usual care.

Antiplatelet Therapy

There is level 1a and level 2 evidence that ASA therapy effectively reduces the risk for recurrent stroke and should be initiated as soon as it is safe following the onset of the stroke event and maintained over the long-term.

There is level 1a evidence that treatment with clopidogrel may be as effective as ticlopidine in terms of prevention of secondary vascular events, including stroke.

There is level 1b evidence that clopidogrel may be similar to ASA with regard to safety.

There is level 1a evidence that treatment with ticlopidine may be associated with a significantly greater risk for adverse events, including hepatic dysfunction, than clopidogrel.

There is level 1a evidence suggesting that cilostazol is superior to aspirin monotherapy in reducing the risk of recurrent stroke and hemorrhagic events however, it is unclear whether its use results in an increased risk of gastrointestinal bleeds.

There is level 1b evidence that the use of Lotrafiban (a glycoprotein IIb/IIIa inhibitor) in the secondary prevention of stroke may be associated with excessive bleeding incidents.

There is level 1a evidence suggesting that administration of clopidogrel and ASA dual therapy is significantly more effective than ASA monotherapy at reducing the risk of stroke, particularly among patients with early (<30d) brain ischaemia.

There is level 1a evidence suggesting that combination clopidogrel and ASA therapy increases the risk of major bleeding relative to ASA therapy alone.

There is level 1a evidence that the use of dipyridamole in combination with ASA may be associated with reduced risk for recurrent vascular events including stroke, non-fatal MI, and non-fatal stroke when compared to placebo.

There is level 1a evidence that dipyridamole in combination with ASA may be more effective than ASA monotherapy when used in the prevention of recurrent stroke.

There is level 1a evidence that use of combination therapy of dipyridamole and ASA may be associated with increased occurrence of headaches and diarrhea when compared to ASA alone.

There is level 1a evidence that combination therapy with dipyridamole and ASA is associated with a lower incidence of bleeding events compared to combination therapy with clopidogrel and ASA.

There is level 1a evidence that clopidogrel in combination with ASA may provide more effective platelet inhibition than ASA in combination with dipyridamole.

There is level 1b evidence that combined ASA + extended release dipyridamole therapy is less likely to cause major bleeding events.

There is level 1b evidence that major bleeding events are more common among patients using aspirin monotherapy compared to those using a combination therapy consisting of aspirin, clopidogrel, and dipyridamole.

There is level 1a evidence that triple antiplatelet therapy with aspirin, clopidogrel and cilostazol is comparable to dual therapy consisting of aspirin and clopidogrel regarding its effect on all-cause death, non-fatal MI, ischaemic stroke, and bleeding events.

There is level 1a evidence that combination therapy of clopidogrel and aspirin or dipyridamole and aspirin has no additional benefit on functional outcomes compared to either ASA or clopidogrel monotherapy.

There is level 1b evidence that early initiation of dipyridamole + ASA therapy has no impact on functional outcome relative to early ASA monotherapy.

Anticoagulants

There is level 1a evidence that treatment with oral anticoagulant therapy of moderate intensity is not superior to antiplatelet therapy in preventing death, recurrent ischemic stroke, or myocardial infarction however, it may result in a greater risk for bleeding.

Atrial Fibrilation

There is level 1a evidence that the use of anti-coagulation therapy, particularly with adjusted dose warfarin, may substantially reduce the risk of primary and secondary stroke in individuals with atrial fibrillation.

Anticoagulant Therapy

There is level 1a evidence that treatment with ASA 300 – 325 mg/day may be associated with reduced risk of stroke when compared to no treatment in individuals with atrial fibrillation. However, anticoagulant therapy (dose-adjusted warfarin) may be more effective in preventing strokes among individuals with atrial fibrillation than antiplatelet therapy (ASA).

There is level 1b evidence that oral anticoagulation therapy may be more effective than ASA + clopidogrel in the prevention of stroke in individuals with atrial fibrillation. However, for patients not

eligible for oral anticoagulation, ASA + clopidogrel may be associated with reduced risk for stroke when compared to ASA monotherapy.

There is level 1b evidence that use of ASA + clopidogrel may be associated with increased risk for bleeding events compared with ASA monotherapy. Risk for major bleeding events with dual therapy may be similar to that reported for oral anticoagulation with vitamin-K antagonists.

Alternative Therapies

There is level 1b evidence that Indobufen may be as effective as warfarin, but is associated with a reduced risk of bleeding events. It is currently not used in the Canadian clinical practice.

There is level 1a evidence that treatment with the direct thrombin inhibitor ximelagatran/melagatran may not be inferior to treatment with warfarin. Ximelagatran treatment is associated with risk for liver injury and due to concerns with safety, it has been withdrawn from the market and its development terminated.

There is level 1a evidence that a dabigatran may be more effective in preventing stroke than warfarin. With respect to dabigatran prescription, a higher dose (150mg b.i.d) appears to be more effective than a lower dose (110mg b.i.d) at reducing the risk of ischemic stroke however, it also increases the risk of major bleeding. The risk or mortality is comparable amongst the two doses and based on a composite of major ischemic, hemorrhagic, and fatal events, both doses demonstrate a similar net clinical benefit. This effect is observed up to 5 years of treatment.

There is level 1b evidence that treatment with fixed dose rivaroxaban (20 mg p.o. o.d.) is not superior to dose-adjusted warfarin for the prevention of stroke in high risk individuals with atrial fibrillation. Treatment with rivaroxaban may also be associated with less risk for intracranial bleeding when compared with dose-adjusted warfarin.

There is level 1b evidence that treatment with apixaban may be superior to ASA for the reduction in risk of stroke in individuals with AF and for whom a vitamin K antagonist is considered unsuitable.

There is level 1b evidence that treatment with apixaban may be superior to dose-adjusted warfarin for the prevention of stroke or systemic embolism in high risk individuals with atrial fibrillation.

There is level 1b evidence that treatment with apixaban may be associated with reduced risk for death from any cause and for major bleeding events when compared to treatment with dose-adjusted warfarin.

Drug Management

There is level 1a evidence that the use of patient decision aids may be associated with an increase in patient knowledge and a decrease in uncertainty regarding treatment.

There is level 2 evidence that incorporating narrative information in the form of patient anecdotes may help increase patient knowledge and belief in the importance of laboratory testing.

There is level 1b evidence that, among high risk patients with atrial fibrillation, use of patient aids may be associated with a temporary increase in the use of appropriate warfarin-based therapy.

There is level 1a evidence that self-management programs are associated with a reduced risk of thromboembolic events and mortality. However, these programmes are more likely to be feasible for a small, select group of patients only.

There is level 1a evidence that self-testing and self-management programmes may not be associated with increased risk of bleeding events.

There is level 2 evidence suggesting that a coordinated, multidisciplinary approach may result in improved adherence to specific targeted guidelines.

Other Cardiac Diseases
There is level 1a evidence that patent foramen closure does not reduce the risk of recurrent stroke, death, or TIA relative to traditional medical therapy in patients with cryptogenic strokes and patent foramen ovale.

Carotid Endarterectomy
There is level 1a evidence that carotid endarterectomy may be an effective procedure to reduce the risk of stroke in individuals with symptomatic carotid artery stenosis of 70-99%.

There is level 1a evidence that carotid endarterectomy may be an effective procedure to reduce the risk of stroke in individuals with asymptomatic carotid artery stenosis of ≥ 60% however, the operative risks associated with the procedure outweigh the benefit if they exceed 3%. Current guidelines do not recommend regular revascularization in asymptomatic patients.

There is level 1a evidence that CEA may be an effective procedure to reduce stroke risk in individuals with 50-69% stenosis if done soon after the event (< 14 days). Risk of procedure needs to be weighed on an individual patient basis.

There is level 1b evidence that early CEA may not be associated with increased risk for stroke or death. Pooled analysis suggests that benefits associated with CEA may decrease as time from the qualifying ischemic event increases especially in patients with moderate (50-69%) carotid stenosis.

There is level 1b evidence that nursing-led coordinated case management may be associated with short-term improvements in knowledge of stroke warning signs and self-reported lifestyle and dietary changes.

Carotid Artery Angioplasty and Stenting
There is level 1b evidence that CAS procedures may result in a decrease incidence of carotid territory stroke.

There is level 1a evidence that both CAS and CEA procedures may be equally effective in preventing strokes. Both procedures generate comparable rates of restenosis.

There is level 1b and level 2 evidence that carotid angioplasty with cerebral protection may not provide additional benefits relative to CAS without protection.

There is level 1a evidence that CAS may be associated with a greater 30-day and longer term (≥ 12 months) risk for stroke than CEA.

There is level 1a evidence that CEA may be associated with a greater 30-day risk for myocardial infarction and cranial neuropathy however, in the long-term the risk of recurrent stroke is similar between CAS and CEA.

There is level 1a evidence that the risk for death and stroke may be higher in patients over 70 years of age with symptomatic stenosis treated with CAS compared to those treated with CEA.

9. Lower Extremity Interventions

Therapeutic Approach
There is Level 1a, Level 2, and Level 3 evidence that the compensatory and restorative approaches improve motor function, although neither approach is superior.

Balance Retraining

There is Level 1a evidence that training focused on balance, stability, and mobility but not weight shifting can improve balance when compared to conventional rehabilitation.

There is Level 1a evidence trunk training improves balance when compared to conventional rehabilitation.

There is Level 1a evidence that sit-to-stand with asymmetrical foot positioning improves balance when compared to symmetrical foot positioning.

There is Level 1a evidence that virtual reality-enhanced balance training improves balance when compared to conventional rehabilitation or standard balance training.

There is Level 1a and Level 2 evidence that balance training with a Balance Trainer device improves balance when compared to conventional rehabilitation or standard balance training.

There is Level 1a and Level 2 evidence that aquatic therapy improves balance when compared to conventional rehabilitation.

There is Level 1b and Level 2 evidence that dual task training improves postural sway but not overall balance when compared to standard balance training.

There is Level 1b and Level 2 evidence that motor imagery-enhanced balance training improves balance when compared to standard balance training.

There is Level 1b and Level 2 evidence that Tai Chi improves some aspects of balance when compared to conventional rehabilitation.

There is Level 1b and Level 2 evidence that visual feedback-enhanced balance training improves balance when compared to standard balance training, although there is limited Level 2 evidence that suggests otherwise.

There is Level 1b evidence that visual deprivation balance training does not improve balance when compared to conventional rehabilitation.

There is Level 2 evidence that Pilates improves centre of pressure when compared to usual care.

There is conflicting Level 1b and Level 2 evidence as to whether Wii-based balance training improves balance when compared to conventional rehabilitation or standard balance training.

There is conflicting Level 1b and Level 2 evidence as to whether sit-to-stand training improves balance when compared to conventional rehabilitation.

There is conflicting Level 1b and Level 2 evidence as to whether force platform biofeedback improves balance when compared to conventional rehabilitation.

Falls Prevention Training

There is Level 1b evidence that a falls prevention programs does not reduce the rate of falls when compared to usual care.

Task-Specific Training

There is Level 1a and Level 2 evidence that task-specific training for the lower limbs improves gait and balance when compared to conventional training or task-specific training of the upper limbs.

There is Level 2 evidence that task-specific training on stairs/ramps improves aspects of balance when compared to training on flat surfaces.

There is conflicting Level 1b evidence as to whether task-specific circuit training improves gait and balance when compared to conventional training.

Treadmill Training
There is Level 1a and Level 2 evidence that treadmill training improves mobility when compared to overground gait training or conventional rehabilitation, although there is Level 1b evidence that suggests otherwise.

There is Level 1a and Level 2 evidence that treadmill training with modifications (e.g. incline, sideways, handrail) improves gait when compared to treadmill training alone.

There is Level 1a and Level 2 evidence that body weight supported treadmill training does not improve balance when compared to overground gait training or conventional rehabilitation.

There is conflicting Level 1a and Level 2 evidence as to whether body weight supported treadmill training improves mobility when compared to overground gait training or conventional rehabilitation.

Virtual Reality Training
There is Level 1a and Level 2 evidence that virtual reality combined with treadmill training may improve gait and balance when compared to treadmill training, overground gait training, or conventional therapy.

There is Level 1a and Level 2 evidence that virtual reality interventions, both immersive and non-immersive, improve gait and balance when compared to conventional therapy.

There is Level 1a and Level 2 evidence that Nintendo Wii does not improve gait or balance when compared to conventional therapy or standard care, although there is limited Level 2 evidence that suggests otherwise.

Feedback
There is Level 1a and Level 2 evidence that auditory feedback, in combination with gait training, improves gait and balance when compared to training alone.

There is Level 1a and Level 2 evidence that visual feedback, in combination with gait training, does not improve gait or balance when compared to training alone.

EMG Biofeedback
There is Level 1b evidence that EEG biofeedback improves gait when compared to sham feedback.

There is conflicting Level 1a and Level 2 evidence as to whether EMG biofeedback improves gait as an adjunct to conventional rehabilitation.

Rhythmic Auditory Stimulation
There is Level 1a and Level 2 evidence that rhythmic auditory stimulation in combination with gait training improves gait and balance when compared to gait training alone.

Dual Task Training
There is Level 1a and Level 2 evidence that dual task training improves gait and stability when compared to single task training.

Mental Practice
There is Level 1a evidence that mental practice improves gait and balance when combined with functional training, but not general rehabilitation, in comparison to training alone.

Mirror Therapy

There is Level 1b and Level 2 evidence that mirror therapy does not improve gait when compared to sham therapy or conventional rehabilitation.

There is Level 1b evidence that mirror therapy combined with repetitive transcranial magnetic stimulation improves mobility, stability, and balance when compared to mirror therapy combined with sham stimulation.

There is conflicting Level 1b and Level 2 evidence as to whether mirror therapy combined with functional electrical stimulation improves gait and mobility when compared to conventional rehabilitation.

Action Observation
There is Level 1b and Level 2 evidence that action observation in combination with gait training improves gait and balance when compared to gait training alone.

Aquatic Therapy
There is Level 1a and Level 2 evidence that aquatic therapies improves gait when compared to conventional therapy.

Horse Riding Simulation
There is conflicting Level 1a and Level 2 evidence as to whether hippotherapy improves gait and balance when compared to conventional rehabilitation or standard gait training.

Self-Management Programs
There is Level 1a evidence that self-management programs do not improve gait and balance when compared to usual care.

Caregiver-Mediated Programs
There is Level 1b evidence that caregiver-mediated programs improve gait and balance when compared to usual care in chronic stroke.

There is Level 1b evidence that caregiver-mediated programs do not improve mobility or independence when compared to usual care in acute stroke.

Strength and Resistance Training
There is Level 1a evidence that progressive resistance training for the lower limbs improves muscle strength, but there is conflicting Level 1b and Level 2 evidence as to whether it improves balance, gait, or endurance.

There is Level 1a evidence that functional strength training for the lower limbs does not improve muscle strength, but there is conflicting Level 1b and Level 2 evidence as to whether it improves gait.

There is Level 1a evidence that combined resistance and aerobic training for the lower limbs improves gait and endurance.

There is conflicting Level 1b and Level 2 evidence as to whether isokinetic strength training for the lower limbs improves motor function.

There is conflicting Level 1b and Level 2 evidence as to whether eccentric resistance training for the lower limbs improves motor function.

Aerobic Exercise
There is Level 1a and Level 2 evidence that aerobic exercise improves gait when compared to conventional rehabilitation, but the evidence regarding balance is conflicting.

There is Level 1a and Level 2 evidence that community- and home-based exercise programs that incorporate aerobic exercise improve gait and balance when compared to conventional rehabilitation.

There is conflicting Level 2 evidence as to whether high-intensity aerobic exercise better improves gait than low-intensity aerobic exercise.

Wheelchair
There is Level 1b evidence that a Neater wheelchair attachment improves efficiency of motor skills and activity performance when compared to a standard wheelchair.

There is level 1b evidence that encouraging wheelchair self-propulsion does not improved functional independence.

Walking Aid
There is Level 1b evidence that single-point canes improve gait speed and endurance when compared to quad canes and hemi-walkers.

There is Level 1b evidence that a robotic walker improves gait speed and endurance when compared to no device.

There is Level 2 evidence that quad canes improve balance when compared to single-point canes.

There is Level 2 evidence that cane length does not impact gait speed.

Ankle Foot Orthosis
There is Level 1a and Level 2 evidence an ankle foot orthosis improves gait when compared to no device, while the evidence regarding balance is conflicting.

There is Level 1b and Level 2 evidence that a standard ankle foot orthosis is as effective in improving gait as an individualized ankle foot orthosis.

There is limited Level 2 evidence that a dynamic ankle foot orthosis is more effective in improving gait than a static ankle foot orthosis.

Taping
There is Level 2 evidence that taping in combination with rehabilitation improves balance and gait when compared to rehabilitation alone.

Electromechanical Devices
There is Level 1a evidence that 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.

There is Level 1a and Level 2 evidence that portable, localized exoskeletal devices (e.g. Stride Management Assist, Bionic Leg, Anklebot) do not improve gait or balance when compared to conventional training.

There is conflicting Level 1a and Level 2 evidence as to whether exoskeletal systems (e.g. Lokomat, LokoHelp, AutoAmbulator, Walkbot) improves lower limb motor function when compared to conventional training.

There is Level 2 evidence that the Hybrid Assistive Limb improves gait and balance when compared to conventional training.

Functional Electrical Stimulation
There is Level 1a and Level 2 evidence that functional electrical stimulation during conventional rehabilitation improves gait, balance, and independence when compared to rehabilitation alone.

There is Level 1a and Level 2 evidence that functional electrical stimulation during gait training improves gait when compared to gait training alone.

There is Level 1a and Level 2 evidence that functional electrical stimulation during robot-assisted training does not improve gait or balance when compared to robot-assisted gait training alone.

There is Level 1a and Level 2 evidence that functional electrical stimulation does not improve gait or balance when compared to ankle foot orthosis.

There is Level 1a evidence that functional electrical stimulation during cycling training improves gait and balance when compared to cycling alone but not to conventional rehabilitation.

There is conflicting Level 1b evidence as to whether functional electrical stimulation during treadmill training improves gait and balance when compared to treadmill training alone.

Neuromuscular Electrical Stimulation
There is Level 1a evidence that neuromuscular electrical stimulation in combination with gait/balance training improves gait/balance when compared to stimulation or training alone.

There is level 1b evidence that interferential current therapy improves gait and balance when compared to sham stimulation.

There is Level 1b and Level 2 evidence as to whether neuromuscular electrical stimulation alone improves gait and balance when compared to sham or no stimulation.

Transcutaneous Electrical Nerve Stimulation
There is Level 1a evidence that transcutaneous electrical nerve stimulation improves gait, balance, and muscle strength when compared to sham or no stimulation.

Other Sensorimotor Stimulation
There is Level 1a evidence that thermal stimulation improves lower limb motor function and reduces spasticity when compared to no/sham stimulation.

There is Level 1b evidence that local vibration improves gait and balance when compared to sham stimulation.

There is Level 1b evidence that repetitive peripheral magnetic stimulation improves ankle/foot strength and range of motion when compared to sham stimulation.

There is Level 1b evidence that ankle/foot sensorimotor stimulation improves weight distribution when compared to no stimulation.

There is conflicting Level 1b and Level 2 evidence as to whether whole-body vibration improves gait or balance when compared to no/sham stimulation.

Repetitive Transcranial Magnetic Stimulation
There is Level 1a evidence that repetitive transcranial magnetic stimulation improves balance, gait, independence, and lower limb motor function when compared to sham stimulation.

Transcranial Direct Current Stimulation
There is Level 1a evidence that transcranial direct current stimulation does not improve gait or balance when compared to sham stimulation.

Galvanic Vestibular Stimulation
There is Level 1b evidence that galvanic vestibular stimulation does not improve pusher behaviour or laterpulsion.

Noradrenergic Agents
There is Level 1a evidence that amphetamines do not improve lower limb motor function when compared to placbeo.

There is Level 1a evidence that methylphenidate improves functional independence, but not lower limb motor function, when compared to placebo.

There is limited Level 2 evidence that droxidopa improves functional outcomes when compared to no medication.

Dopaminergic Agents
There is conflicting Level 1b and Level 2 evidence as to whether levodopa improves lower limb motor function and functional independence when compared to placebo or no medication.

There is Level 1b evidence that ropinirole does not improve gait, functional independence, or motor function when compared to placebo.

Serotonergic Agents
There is Level 1b evidence that citalopram improves neurological status, but not walking ability or functional independence, when compared to placebo.

There is Level 1b evidence that escitalopram does not improve gait, strength, or motor function when compared to placebo.

There is Level 1a evidence that fluoxetine improves lower limb motor function when compared to placebo.

There is Level 1a and Level 2 evidence that fluoxetine does not improve neurological recovery when compared to placebo.

There is conflicting Level 1b and Level 2 evidence as to whether fluoxetine improves functional independence when compared to placebo.

Other Medications
There is Level 1b evidence that cerebrolysin improves lower limb motor function in severe stroke when compared to placebo.

There is Level 1b evidence that PF-0304923 does not improve gait speed when compared to placebo.

There is Level 2 evidence that dalfampridine improves gait speed when compared to placebo.

Spasticity and Contractures: Contracture Prevention
There is Level 1b evidence that both a splint and a tilt table prevent ankle contracture in acute stroke.

Spasticity and Contractures: Botulinum Toxin
There is Level 1a evidence that botulinum toxin reduces lower limb spasticity when compared to placebo.

There is Level 1a evidence that a higher dosage of botulinum toxin (≥300U) reduces lower limb spasticity when compared to a lower dosage of botulinum toxin (100-200U).

There is Level 1a evidence that botulinum toxin yields similar reductions in lower limb spasticity regardless of injection location.

There is Level 1a evidence botulinum toxin combined with taping does not reduce lower limb spasticity when compared to botulinum toxin combined with sham taping or stretching.

There is Level 1a and Level 2 evidence that botulinum toxin combined with ankle foot orthosis reduces lower limb spasticity when compared to botulinum toxin alone, with taping, or with stretching.

There is Level 1b evidence that botulinum toxin reduces lower limb spasticity when compared to phenol nerve block.

There is Level 1b evidence botulinum toxin reduces lower limb spasticity when compared to transcutaneous electrical nerve stimulation or therapeutic ultrasound.

There is Level 1b evidence that botulinum toxin injection guided by ultrasonography reduces spasticity when compared to injection guided by electrical stimulation or palpation.

There is Level 1b evidence botulinum toxin combined with functional electrical stimulation does not reduce lower limb spasticity when compared to botulinum toxin combined with sham stimulation.

There is Level 1b evidence that botulinum toxin is less effective than tibial nerve neurotomy in reducing spasticity.

Spasticity and Contractures: Nerve Block
There is level 1b evidence that thermocoagulation reduces lower limb spasticity when compared to ankle foot orthosis or sham treatment.

There is Level 1b evidence that phenol nerve block is less effective than botulinum toxin in reducing lower limb spasticity.

There is limited Level 2 evidence that phenol and ethyl alcohol are equally effective in reducing lower limb spasticity.

Spasticity and Contractures: Oral Medications
There is Level 1b evidence that tolperisone reduces lower limb spasticity and improves functional independence when compared to placebo.

There is Level 1b evidence that baclofen and tizanidine reduce lower limb spasticity, but there is no significant difference between treatments.

There is conflicting Level 1b and Level 2 evidence regarding the effectiveness of dantrolene in reducing lower limb spasticity and improving functional independence.

Spasticity and Contractures: Intrathecal Medications
There is Level 1b evidence that intrathecal baclofen reduces lower limb spasticity when compared to placebo.

Spasticity and Contractures: Electrical Stimulation
There is Level 1a and Level 2 evidence that transcutaneous electrical stimulation reduces lower limb spasticity when compared to sham stimulation or no treatment.

There is conflicting Level 1b and Level 2 evidence regarding the effectiveness of neuromuscular/functional electrical stimulation in reducing lower limb spasticity.

Spasticity and Contractures: Physical Therapy
There is conflicting Level 1b evidence as to whether rehabilitation program in combination with anti-spastic medications reduces lower limb spasticity when compared to medications alone.

Acupuncture
There is Level 1a evidence that acupuncture does not improve lower limb motor function or functional independence when compared to sham acupuncture or no active treatment, although there is Level 2 evidence that reported otherwise.

There is Level 1a evidence that electroacupuncture does not improve lower limb motor function or functional independence when compared to sham electroacupuncture, transcutaneous electrical nerve stimulation, or no active treatment.

There is Level 1a evidence that acupuncture does not improve lower limb motor function or functional independence when compared to or in combination with physiotherapy.

There is Level 1b and Level 2 evidence that modified acupuncture improves aspects of lower limb motor function when compared to traditional acupuncture.

Meridian Acupuncture
There is Level 1b evidence that acupressure improves lower limb motor function and functional independence when compared to no active treatment.

Chinese Herbal Medicine
There is conflicting Level 1b evidence as to whether various traditional Chinese herbal medicines improve lower limb motor function and functional independence when compared to placebo or no active treatment. There is level 1a evidence that electroacupuncture may not improve motor function or ADL.

10. Upper Extremity Interventions

Consensus Panel Treatment and Recommendations
There is consensus opinion that in severely impaired upper extremities (less than stage 4) the focus of treatment should be on compensation.

For those upper extremities with signs of some recovery (stage 4 or better) there is consensus that attempts to restore function through therapy should be made.

Neurodevelopmental Techniques
There is level 1a evidence that neurodevelopmental techniques are not superior to other therapeutic approaches.

There is level 1b evidence that when compared to the Bobath treatment approach, Motor Relearning Programme may be associated with improvements in short-term motor functioning, shorter lengths of hospital stay and better movement quality.

There is level 1b evidence that Brunnstrom hand manipulation treatment is preferable over a motor relearning program.

Bilateral Arm Training
There is level 1a evidence that bilateral training is not more effective than unilateral training for upper limb motor function outcomes.

There is level 1a evidence that bilateral training is not more effective than conventional therapies such as modified constraint induced movement therapy and cutaneous electrical stimulation.

There is level 1a evidence that bilateral arm training with rhythmic auditory cueing (BATRAC) is not more effective than unilateral arm training.

Arm and Leg Training
There is level 1a evidence that arm function training, task practice, and strength training provide significant functional improvements in the arm after stroke in comparison to similar leg training.

Additional/Enhanced Therapy

There is level 1a evidence that additional upper limb therapy is not superior to conventional therapy at improving upper extremity motor function or functional independence.

There is level 1b evidence that a therapist-supervised in-home program is not more effective than usual care at improving upper limb motor function.

Strength Training
There is level 1a evidence from a meta-analysis that strength training increases grip strength following stroke.

There is level 1a evidence that strength training improves upper limb motor function and shoulder range of motion.

Repetitive/Task-Specific Training Techniques
There is level 1a evidence that task-related practice may be superior to conventional training at improving upper extremity motor function.

There is level 1b evidence that task-related training may not be superior to resistive training or bilateral arm training at improving general upper limb motor function; however, it may improve reaching arm movements.

There is level 1b evidence that combining task practice with active stimulation may improve manual dexterity and reaction time.

Trunk Restraint
There is conflicting level 1a evidence regarding the efficacy of trunk restraint therapy on upper extremity function when combined with constraint induced movement therapy or delivered alone.

Sensorimotor Training
There is level 1a evidence that transcutaneous electrical nerve stimulation (TENS) improves upper limb motor function.

There is level 1a evidence that focal or whole-body vibration therapy improves upper limb motor function.

There is level 1a evidence that peripheral nerve/afferent stimulation does not significantly improve overall upper limb motor function.

There is level 1a evidence that mesh glove therapy improves motor function and dexterity based on the Box and Block test.

There is level 1b evidence that thermal stimulation is effective for upper limb motor function.

There is level 1a evidence that electroacupuncture is not more effective than an active control for improving upper limb motor function.

Mental Practice/Motor Imagery
There is level 1a evidence that mental practice therapy is effective for improving upper extremity motor function; however, the evidence for its effect on activities of daily living is limited and conflicting.

There is level 1a evidence that motor imagery is not effective for improving upper extremity motor function.

Splinting
There is level 1a that hand splinting/taping/orthoses do not improve upper extremity motor function.

Constraint-Induced Movement Therapy
There is level 1b and level 2 evidence that there is no benefit of CIMT in the early stage of stroke for improving upper limb motor function or dexterity.

There is level 1a evidence that CIMT in the chronic phase of stroke may help improve upper extremity motor function. The evidence regarding the ideal frequency of CIMT is currently unclear.

There is level 1a evidence that mCIMT in the early phase of stroke may improve adaptation strategies as it optimizes already preserved function. However, mCIMT does not improve neurological impairment in the early stage of stroke.

There is level 1a evidence that mCIMT in the chronic phase of stroke may improve upper limb function relative to conventional therapy.

Mirror Therapy
There is level 1a evidence that mirror therapy improves upper limb motor function following stroke, especially for the wrist and hand.

There is level 1b evidence that Mirror therapy in combination with conventional therapy is not superior to the Bobath method for upper limb motor function.

There is conflicting level 1a evidence regarding the effect of mirror therapy on spasticity.

Feedback Therapy
There is level 1a evidence that feedback is effective for improving upper limb motor function, and that it is ineffective for improving spasticity.

Action Observation
There is conflicting level 1a evidence regarding the effect of action observation on upper motor function.

There is level 1b evidence that action observation with brain–computer interface-based functional electrical stimulation is effective for improving upper limb motor function.

Music Therapy
There is level 1a and level 1b evidence that music therapy can improve some aspects of upper extremity motor function but not muscle strength when compared to conventional rehabilitation.

Telerehabilitation
There is level 1a evidence that telerehabilitation interventions are not effective for improving upper limb motor function.

Exercise Therapy
There is conflicting evidence regarding the effectiveness of additional exercise therapy for improving upper limb motor function.

Robotic Devices
There is level 1a and 2 evidence in the acute phase and level 1a evidence in the chronic phase that MIT-Manus/InMotion therapies are no more effective than a control for improving upper limb motor function in the chronic phase.

There is level 2 evidence that Mirror-Image Motion Enabler Robots (MIME) are effective in the acute phase, level 2 evidence that (MIME) are not effective in the subacute phase, and level 1a conflicting evidence for the effectiveness in the chronic phase for improving upper limb motor function.

There is conflicting level 1b and 2 evidence for the use of ARMin during the chronic phase for improving upper limb motor function.

There is level 2 evidence that ARM Guide is not effective for improving upper limb motor function.

There is level 1b evidence during the acute phase that Bi-Manu-Track is not effective, level 1a conflicting evidence for the subacute phase, and level 1a evidence during the chronic phase that Bi-Manu-Track is effective for improving upper limb motor function.

There is conflicting level 2 evidence for the use of NeReBot during the acute phase, and level 1b evidence that NeReBot is not effective during the chronic phase for improving upper limb motor function.

There is level 2 evidence that Continuous Passive Motion (CPM) is not effective during the acute phase, and there is level 2 evidence that CPM is effective during the chronic phase for improving upper limb motor function.

There is level 1a evidence that the use of GENTLE during the chronic phase is not effective for improving upper limb motor function.

There is level 1b evidence that the use of Amadeo during the acute phase is effective, while there is level 1b evidence that the use of Amadeo during the chronic phase is not effective for improving upper limb motor function.

There is conflicting level 1a evidence regarding the effectiveness of MusicGlove during the chronic phase.

Virtual Reality
There is level 1a evidence that virtual reality does not improve upper limb motor function in the chronic stroke phase.

Computer Brain Interface Technology
There is level 1a evidence that computer brain interface technology is not effective for improving upper limb motor function post-stroke.

Splinting
There is level 1a evidence that splinting does not reduce the development of contracture nor reduce spasticity in the upper extremity.

Stretching Programs to Prevent Contracture Formation
There is level 1b evidence that a nurse-led stretching program may improve range of motion in the upper extremity and reduce pain in the chronic stage of stroke.

There is level 1b and 2 evidence that a hand stretching device may improve spasticity in the upper limb.

Botulinum Toxin Injections
There is level 1a evidence that treatment with botulinum toxin significantly reduces spasticity in the upper extremity in stroke survivors.

There is level 1a evidence that treatment with botulinum toxin does not improve upper limb motor function.

Electrical Stimulation Combined with Botulinum Toxin Injection
There is level 1a evidence that electrical stimulation combined with botulinum toxin injection is associated with reductions in spasticity.

There is level 1b evidence that modified constraint induced movement therapy combined with botulinum toxin injection is associated with reductions in spasticity.

Nerve Block and Spasticity
There is level 4 evidence that nerve blocks with ethyl alcohol improves elbow and finger passive range of motion and can decrease spasticity in the upper extremity in stroke survivors.

Physical Therapy in the Treatment of Spasticity
There is level 1a evidence that physical therapy may not improve motor function or contracture.

Electrical Stimulation
There is level 1a evidence that neuromuscular electrical stimulation may not reduce wrist or elbow spasticity.

Shock Wave Treatment
There is level 1a evidence that extracorporeal shock wave therapy improves upper limb spasticity.

Centrally Acting Muscle Relaxants
There is level 1b evidence that tolperisone may reduce spasticity following stroke.

EMG/Biofeedback
There is level 1a evidence that EMG/biofeedback therapy does not improve upper extremity motor function or spasticity.

Neuromuscular Electrical Stimulation
There is level 1a and level 2 evidence that FES/NMES may improve upper limb motor function, range of motion, and manual dexterity when offered in combination with conventional therapy or delivered alone in subacute stroke. The evidence is also indicative of a beneficial effect on range of motion and manual dexterity when FES/NMES was offered to chronic stroke patients either alone or in combination with other therapies.

Despite improvements in both stages of stroke recovery, level 1b evidence indicates that delivering FES early (<6 months) may be more beneficial at recovering impaired motor function than delivering FES after 6 months post-stroke.

There is level 1b evidence that EMG-NMES in the subacute phase is not more effective than usual care for patients with an unfavourable prognosis based on voluntary finger extension.

There is level 1a evidence that high intensity NMES or FES exercise is no more effective for improving upper limb motor function than low intensity NMES or FES in the subacute phase.

There is level 1a and level 2 evidence that both EMG-triggered and cyclic approaches to NMES/electrical stimulation may improve upper limb motor function and range of motion in subacute and chronic stroke patients; however, evidence indicates no superior benefit of EMG-triggered NMES over cyclic or passive NMES at improving upper limb motor function in chronic (level 1a) and subacute (level 1b) stroke patients.

There is level 1b evidence that Contralaterally Controlled FES is not superior to cyclic NMES for improving upper limb motor function, although it may improve dexterity.

There is level 1b evidence that coupling continuous NMES with repetitive facilitative exercise may be beneficial at improving general upper extremity function and range of motion during elbow extension but not during shoulder or wrist flexion in subacute stroke patients.

There is level 1b evidence that high frequency NMES may be superior to low frequency NMES at improving endurance of thumb adduction, lateral pinch strength and manual dexterity in chronic stroke individuals.

Invasive Motor Cortex Stimulation
There is level 1a evidence that motor cortex stimulation does not improve upper limb motor function.

There is level 1b evidence that vagus nerve stimulation can improve overall upper limb motor function, but not dexterity or grip strength.

Repetitive Transcranial Magnetic Stimulation
There is level 1a conflicting evidence regarding the effectiveness of low-frequency (1Hz) rTMS for the improvement of upper limb motor function and dexterity.There is also level 1a evidence that inhibiting rTMS does not improve upper limb spasticity when compared to sham stimulation.

There is level 1a evidence that high-frequency rTMS (≥5 Hz) improves upper limb motor function, dexterity, and grip strength when compared to sham stimulation.

There is level 1a evidence that there is no significant difference between inhibitory and excitatory rTMS for improving upper limb motor function or grip strength.

There is level 1b evidence that dual rTMS (the combination of both inhibitory and excitatory rTMS) improves upper limb motor function, but not grip strength when compared to sham stimulation.

Theta Burst Stimulation
There is level 1b and level 2 evidence that iTBS improves upper limb motor function, but not dexterity, in the acute or subacute period after stroke.

There is conflicting level 1a evidence that iTBS improves upper limb motor function and dexterity in the chronic phase after stroke. There is level 1b and level 2 evidence that iTBS improves spasticity in the chronic phase after stroke.

There is level 1a evidence that cTBS does not improve upper extremity motor function or dexterity following stroke.

Transcranial Direct Current Stimulation
There is level 1a evidence that anodal tDCS does not improve upper limb motor function, spasticity, or grip strength. There is conflicting level 1a evidence regarding whether anodal tDCS improves dexterity.

There is level 1a conflicting evidence for the effectiveness of cathodal tDCS for improving upper limb motor function, dexterity grip strength, and activities of daily living.

There is level 1a evidence that anodal and cathodal tDCS do not significantly differ on measures of motor function, dexterity, or on measures of independence/daily living.

There is level 1a evidence that dual tDCS (both anodal and cathodal tDCS administered at the same time) is effective for improving dexterity. There is level 1a conflicting evidence regarding the effectiveness of dual tDCS for improving grip force.

There is level 1b evidence that coupling methylphenidate with tDCS may improve hand function relative to when tDCS or methylphenidate are delivered alone.

There is level 1b evidence that combining tDCS with computer brain interface training may not improve spasticity or upper extremity motor function.

Stimulants

There is level 1a evidence that delivering stimulants in combination with additional therapy may improve upper extremity function; however, level 1b evidence suggests that grip strength may not improve.

There is Level 1b evidence that stimulants may only be effective at improving impaired upper limb function in the short term.

Levodopa
There is level 1b evidence that Levodopa may not improve arm and hand function however, level 2 evidence suggests that reaction time may be improved.

Antidepressants
There is level 1a evidence that fluoxetine and nortriptyline may improve overall disability and upper extremity motor function.

There is level 1a that citalopram, reboxetine and lithium carbonate may enhance impaired arm and hand function however, level 1b evidence indicates that citalopram may not be effective at improving hand grip strength.

Steroids
There is level 1b evidence that intra-articular steroid injections may not improve pain or range of motion of the upper extremity; however, limited level 2 evidence provides conflicting findings.

Antibiotics
There is level 1b evidence that d-cycloserine delivered in combination with constraint-induced movement therapy may not improve upper extremity motor function.

Ozonated Autohemotherapy
There is limited level 2 evidence that ozonated autohemotherapy may improve general motor disability.

Peptides
There is level 1a evidence that Cerebrolysin improves upper limb motor function.

There is level 1b evidence that Cerebrolysin improves dexterity and measures of independence/daily living.

Neuroprotectants
There is level 1b evidence that NeuroAid does not improve upper limb motor function.

There is level 1b evidence that phosphodiesterase-5 inhibitor does not improve dexterity, grip strength, or level of independence/daily living.

Statins
There is level 1b evidence that Atorvastatin improves level of independence / activities of daily living.

Acupuncture
There is level 1a evidence from high-quality, high-powered studies that acupuncture does not improve upper extremity motor function or performance of activities of daily living however.

There is conflicting level 1a evidence regarding the effect of acupuncture on spasticity.

Meridian Acupressure
There is level 1a and limited level 2 evidence that meridian acupressure may improve spasticity, upper limb motor function, range of motion of the upper limb, and performance of activities of daily living.

Traditional Chinese Herbal Medicine

There is level 1b conflicting evidence regarding the effectiveness of Astralagus Membranaceus for improving functional independence and performance in activities of daily living after hemorrhagic stroke.

There is level 1b evidence that Tokishakuyakusan improves functional independence and performance in activities of daily living in the chronic stage of stroke.

Massage Therapy
There is level 1a evidence that Chinese or Thai massage therapy does not improve functional independence or performance on activities of daily living.

There is level 1b evidence that Chinese or Thai massage therapy does not improve upper limb motor function, spasticity, or quality of life.

Treatment of Hand Edema
There is level 1b evidence that intermittent pneumatic compression does not reduce hand edema or strength in the upper extremity following stroke.

11. Hemiplegic Shoulder Pain & Complex Regional Pain Syndrome

Hemiplegic Should Pain
Factors associated with hemiplegic shoulder pain include older age, longer disease duration, poor arm function, muscle imbalance, rotator cuff tear, subscapularis/pectoralis spasticity, glenohumeral subluxation, bursitis, tendonitis, adhesive capsulitis, and complex regional pain syndrome.

Shoulder Subluxation
Shoulder subluxation may occur early on in the hemiplegic arm due to flaccid supporting shoulder musculature and can be exacerbated by external forces.

Shoulder subluxation may be associated with hemiplegic shoulder pain, although patients with shoulder subluxation may not experience pain and patients with pain may not have subluxed shoulder.

Contracted/Frozen Shoulder
Spastic muscle imbalance of the shoulder adductors and internal rotators, particularly the subscapularis and pectoralis major, appears to be associated with hemiplegic shoulder pain.

Adhesive capsulitis and its associated limited range of movement appear to be associated with hemiplegic shoulder pain.

Rotator Cuff Disorders and Hemiplegic Shoulder Pain
Rotator cuff disorders do not appear to be associated with hemiplegic shoulder pain.

Frequency of Hemiplegic Shoulder Pain
The reported frequency of hemiplegic shoulder pain varies from 9% to 72%, which may be influenced by heterogeneity in the type and timing of assessment.

Functional Impact of Hemiplegic Shoulder Pain
There is Level 4 evidence that hemiplegic shoulder pain may be associated with poorer upper limb motor function and lower quality of life; the association with functional outcomes is less clear.

Positioning of the Hemiplegic Shoulder
There is Level 1a evidence that sustained positioning and static stretching does not reduce pain, increase range of motion, or improve motor function of the hemiplegic shoulder.

Slings for the Hemiplegic Shoulder

There is Level 1b and Level 2 evidence that slings reduce subluxation and pain of the hemiplegic shoulder.

Strapping/Taping the Hemiplegic Shoulder
There is Level 1a evidence that shoulder strapping/taping reduces hemiplegic shoulder pain; however it may not improve spasticity, disability, range of motion, or motor function.

Active Therapies for the Hemiplegic Shoulder
There is Level 1b evidence that continuous passive range of motion exercises are not more effective than self-range of motion exercises at improving motor function, joint stability, spasticity, or pain in the affected limb.

There is Level 1b evidence that supplementing range of motion exercises with ultrasound or positioning is not more effective than the exercises alone.

There is limited Level 2 evidence that aggressive range of motion exercises (e.g. overhead pulleys) increase hemiplegic shoulder pain when compared to the exercises alone.

There is limited Level 2 evidence that stretching and joint stabilizing exercises improve motor function of the affected limb when compared to conventional exercises.

There is limited Level 2 evidence that Bobath therapy reduces hemiplegic shoulder pain when compared to cryotherapy but not standard therapy.

There is limited Level 2 evidence that 'monkey chair and band' therapy improves motor function, range of motion, and pain in the affected limb when compared to standard therapy.

Electrical Stimulation of the Hemiplegic Shoulder
There is Level 1a and Level 2 evidence that surface neuromuscular electrical stimulation (NMES) reduces subluxation and improves range of motion of the hemiplegic shoulder, but does not reduce pain, when compared to sham or no stimulation.

There is Level 1b evidence that intramuscular neuromuscular electrical stimulation (NMES) reduces hemiplegic shoulder pain for up to 12 months post-treatment when compared to a cuff sling, but does not improve subluxation, spasticity, or motor function.

There is level 1b evidence that peripheral nerve stimulation (PNS) reduces hemiplegic shoulder pain when compared to no stimulation.

There is Level 1b evidence that interferential electrical stimulation (IES) reduces hemiplegic shoulder pain when compared to sham stimulation.

There is Level 1b evidence that extracorporeal shockwave therapy (ESWT) reduces hemiplegic shoulder pain when compared to no stimulation.

There is Level 2 evidence that functional electrical stimulation (FES) reduces subluxation and improves motor function of the hemiplegic shoulder when compared to no stimulation.

There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) at high intensity improves passive range of motion of the hemiplegic shoulder when compared to sham stimulation.

There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) improves muscle strength and range of motion when compared to ultrasound therapy.

There is limited Level 2 evidence that high-voltage pulsed galvanic stimulation (HVPGS) reduces subluxation and joint displacement in the hemiplegic shoulder when compared to no stimulation.

Botulinum Toxin Injections for the Hemiplegic Shoulder
There is Level 1a evidence that high doses of botulinum toxin (500U) improve pain and range of motion, but not spasticity, in the hemiplegic shoulder.

There is Level 1a evidence that low doses of botulinum toxin (100-150U) do not improve pain, spasticity, or range of motion in the hemiplegic shoulder.

Steroid Injections for the Hemiplegic Shoulder
There is conflicting Level 1a and Level 2 evidence regarding the effectiveness of triamcinolone acetonide injections in reducing hemiplegic shoulder pain.

Hyaluronic Acid Injections for the Hemiplegic Shoulder
There is Level 1b evidence that hyaluronic acid reduces hemiplegic shoulder pain when compared to standard care.

There is Level 2 evidence that hyaluronic acid is as effective as triamcinolone acetonide in reducing hemiplegic shoulder pain.

Suprascapular Nerve Block for the Hemiplegic Shoulder
There is Level 1b and Level 2 evidence that suprascapular nerve block injections reduce hemiplegic shoulder pain, but do not improve range of motion, relative to saline injections or ultrasound therapy.

There is limited Level 2 evidence that suprascapular nerve block is not superior to intraarticular steroid injections in reducing hemiplegic shoulder pain.

Segmental Neuromyotherapy for the Hemiplegic Shoulder
There is Level 1b evidence that segmental neuromyotherapy improves hemiplegic upper limb motor function, but not hemiplegic shoulder pain, when compared to oral pain medication.

Surgery of the Hemiplegic Shoulder
There is limited Level 4 evidence that surgical resection of the subscapularis and pectoralis muscle tendons improves range of motion in the hemiplegic shoulder.

There is limited Level 4 evidence that biceps tenodesis through a deltopectoral approach reduces pain and subluxation in the hemiplegic shoulder.

Complementary & Alternative Therapies for the Hemiplegic Shoulder
There is Level 1a evidence that acupuncture reduces pain, increases range of motion, and improves motor function in the hemiplegic shoulder when compared to conventional therapy.

There is Level 1b and Level 2 evidence that massage therapy, alone or with acupuncture, reduces hemiplegic shoulder pain.

There is limited Level 1b evidence that a combination of acupressure and aromatherapy is more effective than dry acupressure in reducing hemiplegic shoulder pain.

Complex Regional Pain Syndrome (CRPS)
Peripheral changes due to complex regional pain syndrome include pain, edema, dystrophy, immobility, and vasomotor instability of the affected upper limb.

Central changes due to complex regional pain syndrome include sensory cortical processing, motor cortex disinhibition, and disrupted body schema.

Pathophysiology of CRPS
The pathophysiology of complex regional pain syndrome is poorly understood, although several theoretical peripheral and central etiologies have been proposed.

Frequency of CRPS
The reported frequency of complex regional pain syndrome post stroke varies from 10% to 48%, which may be influenced by heterogeneity in the type and timing of assessment.

Diagnosis of CRPS
Several CRPS diagnostic tests exist, although none will identify all patients with CRPS.

Pharmacological Interventions for CRPS
There is Level 1a evidence that oral or intravenous corticosteroids reduce pain, swelling, and severity of complex regional pain syndrome.

There is Level 1b evidence that stellate ganglion nerve blocks reduce pain and swelling in complex regional pain syndrome, which may be enhanced by ultrasound guiding.

Mirror Imagery Therapy for CRPS
There is Level 1a evidence that mirror imagery therapy reduces pain and improves upper limb motor function in complex regional pain syndrome when compared to placebo or standard care.

Exercise for the Prevention and Treatment of CRPS
There is Level 1b evidence that a combination of aerobic exercise and physiotherapy reduces pain and improves psychosocial outcomes in complex regional pain syndrome when compared to conventional physiotherapy.

There is limited Level 2 evidence that passive range of motion exercises prevent the development of complex regional pain syndrome when compared to standard care.

Calcitonin for the Prevention of CRPS
There is limited Level 2 evidence that intramuscular injections of calcitonin within four weeks of stroke prevent the development of complex regional pain syndrome.

12. Post-Stroke Cognitive Disorders

Depression and Cognitive Impairment
There is conflicting level 5 evidence regarding the link between post-stroke depression and cognitive and functional impairment.

Treatment of Hypertension and Prevention of Cognitive Decline
There is level 1a evidence indicating no statistical association between lowering of blood pressure and a reduction in the risk for the development of dementia.

There is level 1a evidence that antihypertensive medication may prevent recurrence of stroke, but not reduce cognitive decline or dementia.

There is level 1b evidence that reducing risk factors detrimental to brain health such as cholesterol levels, blood pressure, and BMI may have no significant effect on cognitive performance.

Remediation of Attention Deficits
There is mixed level 1a and level 2 evidence regarding the effect of computerized training for attention tasks on the performance of specific attention tasks.

There is mixed level 1a evidence that cognitive rehabilitation may improve divided attention but not global measures of attention and standardised attentional assessments.

There is level 1b evidence that Attention Process Training may improve aspects of visual and auditory attention.

There is level 1b evidence that an intensive, computerized training program may result in improvements in both working memory and attention.

There is level 1b evidence that visual attention retraining using the Useful Field of View may be more effective than conventional computerized visuoperceptual training at improving the on-road driving performance of individuals with right-sided lesions.

Remediation of Memory Deficits

There is level 1a evidence that compensatory strategies may be effective at improving memory outcomes, including imagery-based, process-oriented, and self-efficacy training.

There is level 1b evidence that home visits combined with mailed letters containing resources and information may result in an improvement of self-reported health status for both patients and caregivers after 6 months compared to mailed letters only.

There is level 1b and level 2 evidence that mental imagery may improve relearning of activities of daily living in patients with acute stroke and minimal cognitive deficits.

There is limited level 2 evidence that patients in group-based interventions may not improve memory abilities any better than patients who did not receive intervention while on a waiting list.

Rehabilitation of Executive Function and Problem Solving

There is level 1b evidence that an analogical problem solving skills approach may increase problem solving abilities and performance of extended activities of daily living.

There is level 1b evidence that self-regulation training may increase executive control over motor but not cognitive function, although these findings may be biased.

There is level 2 evidence that goal management training may be beneficial in the rehabilitation of executive function.

There is level 1b and level 2 evidence that acupuncture can be an effective intervention in the remediation of cognitive deficits.

There is level 1b evidence that problem-solving therapy is not an effective intervention for the improvement of executive functioning.

There is level 1b evidence that tailored occupational cognitive interventions do not significantly affect return to work or executive functions.

There is level 2 evidence that visual training in combination with other interventions can improve executive functions.

The standardization of both intervention and outcome measures would help resolve the conflicts seen between individual studies.

Multi-Modal Interventions

There is mixed level 1b evidence that physical therapy in combination with mindful interventions can improve cognition and quality of life post-stroke.

There is level 1b evidence that acupuncture combined with nimodipine can improve cognitive functioning short term.

There is level 1b evidence that standard care combined with computerized training may improve cognitive performance more than standard care alone.

There is limited level 2 evidence that virtual reality training combined with computerized training may improve cognitive performance more than computerized cognitive training alone.

Electroacupuncture and Transcutaneous Electrical Nerve Stimulation
There is level 1b evidence that electroacupuncture may improve attention, praxis, perception and orientation, but not thinking, organization memory and mental health.

There is level 1b evidence that high-intensity TENS may not be more effective than low-frequency TENS at improving cognitive function.

Music Therapy
There is level 1b evidence that self-regulated music therapy may have a positive impact on verbal memory and focused attention in individuals with left hemisphere stroke.

Exercise Programs
There is conflicting level 1a evidence regarding the effect of exercise therapy on cognitive rehabilitation post stroke.

There is level 1b evidence that robotic table verticalization can demonstrate greater improvements in both electrophysiological and cognitive measures compared to verticalization delivered by a therapist.

There is level 1a and level 2 evidence that exercise programs with a focus on resistance, balance and aerobics can result in significant cognitive gains.

Repetitive Transcranial Magnetic Stimulation
There is level 1b evidence that high-frequency, low-frequency and sham rTMS are not significantly different at improving cognitive performance.

There is level 1b evidence that rTMS in general compared to sham therapy can significantly improve cognitive functioning.

There is level 4 evidence that rTMS to the left DPC may be associated with improvements in executive function following stroke.

Transcranial Direct Current Stimulation
There is level 2 evidence that anodal tDCS to the left dorsolateral prefrontal cortex may be associated with improvements in working memory and attention.

Aspirin
There is level 1b evidence that aspirin is effective in stabilizing and/or improving cognitive outcomes in patients with multi-infarct dementia.

Phosphodiesterase Inhibitor
There is level 1b evidence that phosphodiesterase-5 inhibitor may not be effective in remediating cognitive deficits.

Donepezil
There is level 1a evidence that donepezil taken for 24 weeks may improve cognitive function in patients with probable or possible vascular dementia.

There is level 1a evidence that treatment with donepezil is associated with improvement in global function for individuals with probable or possible vascular dementia.

Rivastigmine
There is conflicting level 1a evidence regarding treatment with rivastigmine and its effect on vascular dementia and cognitive decline.

There is level 2 evidence that treatment with rivastigmine is associated with more stable cognitive performance and improved behavioural outcomes among patients with vascular dementia.

Galantamine
There is level 1a evidence that treatment with galantamine is associated with improvements in cognitive and global function. However, the benefits associated with treatment are more clearly demonstrated among patients with mixed dementia than vascular dementia.

Nimodipine
There is level 1a evidence that nimodipine may not be beneficial in the treatment of vascular dementia.

There is level 1b evidence that treatment with nimodipine may slow cognitive deterioration in patients with vascular dementia.

Memantine
There is level 1a evidence that treatment with memantine is associated with stabilization or improvement of cognitive function in patients with vascular dementia.

Pentoxifylline
There is level 1a evidence that treatment with pentoxifylline is associated with cognitive benefits in patients with multi-infarct dementia.

Citicoline
There is conflicting level 1a evidence regarding the effect of citicoline in the long term management of cognitive function post stroke.

Antidepressants
There is level 1a and level 2 evidence that treatment with antidepressants may be associated with and improvement in cognitive functioning in patients without post-stroke depression.

Selegiline
There is level 1b evidence that selegiline may improve cognitive function post stroke, with benefits lasting as long as six weeks.

Xueshuan Xinmai
There is level 1b evidence that XXMT may be an effective treatment for memory remediation but not attention or language deficits.

Vincopocetine
There is level 2 evidence that IV vinpocetine is effective in improving cognition and quality of life.

Prevention of Delirium Post Stroke
There is level 2 evidence that a multi-component approach to the management of known risk factors may be associated with reduced incidence and duration of delirium. However, this has not been demonstrated within the stroke population; further research is required.

Treatment of Delirium Post Stroke
There is limited level 4 evidence regarding the impact of short-term treatment with rivastigmine on post-stroke delirium. Further research is required.

13. Perceptual Disorders

Treatment of Perceptual Deficits
There is conflicting level 1a and level 2 regarding the evidence for perceptual training interventions on perceptual functioning.

There is level 1b evidence that a transfer of training approach may not produce different results on measures of neglect and functional ability when compared to a functional approach to perceptual training.

Family Participation
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.

Visual Scanning
There is level 1a and level 2 evidence that treatment utilizing primarily visual scanning techniques may improve perceptual impairment post-stroke with associated improvements in function.

Computer-Based Rehabilitation
There is level 1b and level 2 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.

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 Treatment
There is level 1a and level 2 evidence that limb activation may alleviate rightward bias and improve motricity when compared to conventional rehabilitation.

Sensory Stimulation Interventions
There is level 1b evidence that sensory cues for movement may have a positive effect on neglect, although evidence is inconclusive.

There is level 1b evidence that use of electrical somatosensory stimulation as a supplement to visual scanning training is associated with greater benefit than visual scanning training alone.

Feedback Strategies
There is level 1b and limited level 2 evidence that visuomotor feedback may be beneficial in the treatment of neglect. Further study is required to establish the degree to which treatment effects generalize to other behaviours and to determine the durability of effect.

There is limited level 2 evidence that the auditory feedback for left eye movement may not improve visual inattention or bias in eye movement.

Prism Treatment
There is level 1a and level 2 evidence that the use of rightward shifted prisms may be effective for neglect and hemianopia.

There is level 1a evidence that any improvements seen in visual-spatial tasks may not be sustained over time.

There is level 1b and limited level 2 evidence that improvements in visual-spatial tasks following prism treatment are not associated with improvement in functional ability.

There is limited level 2 evidence that terminal prismatic adaptation may alleviate rightward bias and improve visual perception to a greater degree than concurrent prismatic adaptation.

Eye-Patching and Hemispatial Glasses
There is conflicting level 1b and level 2 evidence regarding the use of right half-field eye patches for left visual neglect.

There is limited level 2 evidence that monocular occlusion may not improve visual neglect or alleviate rightward bias.

There is conflicting level 1b and level 2 evidence with regards to the effect of bilateral half-field eye patches on functional ability.

Caloric Stimulation
At present, there is little evidence regarding the effectiveness of caloric stimulation as a treatment intervention for visuospatial neglect post-stroke.

Vestibular Galvanic Stimulation
There is level 1a evidence that 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.

Optokinetic Stimulation
There is level 1a evidence that optokinetic stimulation may have a positive impact on unilateral neglect when compared to scanning or alertness training; however, level 2 evidence suggests that optokinetic stimulation may not have additional benefit.

There is level 1a evidence that optokinetic stimulation may not have an effect on functional outcome.

There is level 2 evidence that optokinetic stimulation may not improve neglect when compared to standard rehabilitation.

Trunk Rotation Therapy
There is level 1b evidence that trunk rotation therapy may not have a positive effect on unilateral spatial neglect or performance of activities of daily living.

There is level 1b evidence that trunk rotation in combination with half-field eye-patching is similarly ineffective.

There is level 2 evidence that trunk rotation when combined with visual scanning is of benefit in the treatment of spatial neglect. Further study of trunk rotation therapy is indicated.

Neck Muscle Vibration
There is level 1b evidence that 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.

Music Therapy
Presently, there is little evidence to support the use of music as treatment for unilateral spatial neglect in right hemispheric patients. Further investigations are required.

Transcutaneous Electrical Nerve Stimulation
There is level 1b evidence that transcutaneous electrical nerve stimulation does not improve visual neglect.

There is level 2 evidence that transcutaneous electrical nerve stimulation may improve reading and writing post-stroke.

Repetitive Transcranial Magnetic Stimulation
There is level 1a evidence that repetitive transcranial magnetic stimulation (rTMS) and theta burst stimulation (TBS) may improve neglect and functional ability.

Transcranial Direct Current Stimulation
There is level 1b evidence that 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.

Dopaminergic Medication Therapy
There is level 1b evidence that the dopamine agonist rotigotine may not improve perceptual impairment or motor function.

Acetylcholinesterase Inhibitors Therapy
There is level 1b evidence that the use of rivastigmine in conjunction with cognitive training may accelerate the rate of improvement of unilateral spatial neglect associated with therapy.

Nicotine Therapy
There is level 1b evidence that nicotine may improve unilateral neglect and target information processing when compared to placebo treatment.

14. Aphasia and Apraxia

Language Therapy
There is level 1a evidence that Intensive Language-Action Therapy significantly improves symptoms of aphasia.

There is level 2 evidence that Promoting Aphasic Communication Effectiveness is an effective treatment for aphasia.

There is level 1a and level 2 evidence that general language therapy may not improve communicative ability, performance on comprehensive language assessments, comprehension or oral expression when compared to no treatment.

There is limited and conflicting level 1a and level 2 evidence for the effect of language therapy on communicative ability when compared to a non-aphasia therapy program.

There is level 2 and level 4 evidence that 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.

Intensity of Speech and Language Therapy
There is level 1a that intensive language therapy may not improve performance on comprehensive language assessments, cognitive and language tasks or communicative ability when compared to standard language therapy; however, level 2 evidence is conflicting.

There is level 1b evidence that 19.3hrs of speech therapy program may improve performance on comprehensive language assessments compared to standard therapy (6.9hrs).

Volunteer-Facilitated Speech and Language Therapy
There is level 1b and level 2 evidence that volunteers can provide speech and language therapy and achieve similar outcomes in terms of comprehension and communicative ability when compared to speech- language pathologists.

There is level 1b and level 2 evidence that immediate language therapy may not improve reading comprehension, auditory comprehension or non-verbal reasoning when compared to deferred therapy; however, the evidence for communicative ability is conflicting.

Group Therapy for Aphasia Post-Stroke
There is level 1a evidence that group treatment may improve communicative ability but not conversational ability, non-verbal reasoning, verbal expression, auditory comprehension or fluency as compared to individual treatment.

There is level 1b evidence that group treatment, individual treatment and combined group and individual treatment may not produce different results in terms of word retrieval.

There is limited level 2 evidence that immediate group therapy may improve language impairment when compared to deferred group therapy; however, evidence for the effect on communicative ability is conflicting.

Community-Based Treatment Programs
There is conflicting level 1b evidence in reference to the effectiveness of a community-based language program on communicative ability when compared to a recreational activities program; however, evidence suggests that the community-based program may not improve performance on comprehensive language assessments.

Training Conversation/Communication Partners
There is level 1b evidence that 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.

Patient and Caregiver Education
There is limited level 2 evidence that a caregiver and patient education program may improve knowledge of aphasia but not activity level, community integration or family functioning when compared to no treatment.

Computer-Based Treatments in Aphasia
There is level 1a evidence that computer-based aphasia therapy may improve word retrieval ability in the short-term but not language function or word retrieval ability in the long-term when compared to standard language therapy.

There is limited level 2 evidence that computer-based aphasia therapy may improve communicative ability and language function when compared to no treatment.

There is level 2 evidence that a 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.

There is conflicting and limited level 2 evidence in reference to the effect of audio-visual naming training on word retrieval ability when compared to audio only naming training.

Telerehabilitation and Speech and Language Therapy
There is limited level 2 evidence for the use of remote assessment when compared to face-to-face assessment; however, preliminary findings suggest that the interventions are comparable.

There is limited level 2 evidence that the use of teleconferencing for remote speech and language treatment is comparable to face-to-face treatment in individuals with aphasia following stroke.

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.

There is limited level 5 evidence that 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.

Music Based Therapies
There is level 1b and limited level 2 evidence that melodic intonation therapy may be as effective as standard language therapy for the improvement of word retrieval ability or performance on comprehensive language assessments; however, evidence regarding its effect on repetition is conflicting.

There is limited level 2 evidence suggesting that melodic intonation therapy may improve responsive speech but not repetition when compared to no language treatment.

There is conflicting level 2 evidence that the addition of music therapy to a standard aphasic therapy program is effective in the remediation of language function.

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.

There is limited level 2 evidence that 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.

Repetitive Transcranial Magnetic Stimulation (rTMS)
There is level 1a evidence that treatment with rTMS may improve performance on comprehensive language assessment as well as on tests of naming abilities. However, there is conflicting evidence for its effectiveness on test components such as comprehension and repetition.

There is limited level 2 evidence that theta burst stimulation may improve naming abilities among individuals with aphasia as compared to sham stimulation.

Transcranial Direct Current Stimulation (tDCS)
There is level 1a and limited level 2 evidence that anodal tDCS applied over the left frontal cortex is associated with improved naming performance in individuals with chronic post-stroke aphasia.

There is level 2 evidence that tDCS in combination with naming therapy is more effective than naming therapy alone in improving aphasic symptoms.

There is level 2 evidence that high definition tDCS is more effective than traditional sponge tDCS in treating aphasia.

There is conflicting level 2 evidence that tDCS is as effective as sham-tDCS.

Unilateral Forced Nostril Breathing
There is limited Level 2 evidence that unilateral forced nostril breathing may improve anxiety and language but not attention level, spatial ability, auditory comprehension or depression.

Specific Treatment for Word-Retrieval Deficits
There is level 1a and limited level 2 evidence that 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.

There is conflicting level 1b and limited level 2 evidence regarding the effect of picture-naming therapy when combined with gesture therapy on word retrieval abilities.

Specific Treatment for Global Aphasia
There is limited level 2 evidence that speech and language therapy may be helpful for individuals with global aphasia post-stroke.

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.

Piracetam
There is level 1a evidence that piracetam may be no better than placebo for comprehensive language assessment, and specific language outcomes, including semantic and phonological outcomes.

There is level 1b evidence that piracetam may be helpful for arm and leg motor movement, and the rate of perfusion compared to placebo.

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
There is level 1a evidence that bromocriptine may be no better than placebo for treating aphasia post-stroke.

Levodopa
There is level 1a and level 2 evidence that the use of levodopa may not be an effective adjunct to speech and language therapy.

Amphetamines
There is level 1b evidence that dextroamphetamine may improve aphasia recovery when combined with speech and language therapy.

Bifemelane
There is level 1b evidence that Bifemelane may improve comprehension and naming; however more research is needed.

Dextran-40
There is level 1b evidence that Dextran-40 may result in better outcomes than the non-treatment control.

Moclobemide
There is level 1b evidence that the use of Moclobemide may not improve verbal communicative abilities of individuals with aphasia.

Donepezil
There is level 1b evidence that 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.

Memantine
There is level 1a evidence for the effectiveness of memantine therapy on the treatment of chronic aphasia. Combination therapy using constraint-induced language therapy and memantine may provide additional benefit than either therapy used independently.

Galantamine

There is level 1b evidence that galantamine may have a beneficial effect on post-stroke aphasia; however, Galantamine has not been studied sufficiently in aphasia recovery.

Nao-Xue-Shu
There is level 2 evidence that western therapies supplemented with Nao-Xue-Shu oral liquid have improved language and comprehension function compared to western therapies alone.

Treatment of Ideomotor Apraxias and Ataxia
There is level 1a evidence that 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.

Gesture Training
There is level 1b evidence that 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.

15. Dysphagia and Aspiration Following Stroke

Pathophysiology of Dysphagia
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.

Aspiration Associated with Dysphagia
There is limited level 4 evidence suggesting that the presence of post-swallow vallecular residue may result in a greater risk of penetration-aspiration.

Incidence of Aspiration Post-Stroke
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.

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.

Tested factors that may not be predictive of aspiration include: poor oral motility and bedside evaluations (which were associated with the identification of non-aspirators).

Incidence of Dysphagia in the Acute Phase of Stroke
The incidence of dysphagia appears to be quite variable following acute stroke with between 3.5% and 65% of patients affected, depending on the sample studied and the method of assessment used.

Age, diabetes, neurological status, and lesion location may be associated with an increase in the rate of dysphagia.

Prognostic Indicators of Dysphagia Post-Stroke
There is level 3 evidence that potential prognostic indicators of dysphagia include: the presence of dysarthria, dysphonia and aspiration, abnormal cough and cough after swallow, National Institute of Health Stroke Scale scores ≥12, level of consciousness assessment, intubation and bi-hemispheric infarcts, cognitive dysfunction, disuse syndrome, fever and length of hospital stay (inversely related).

Defining Aspiration Pneumonia
Criteria that may be most useful in the identification of pneumonia include: abnormal chest x-ray, temperature >100°F, WBC >10,000, arterial hypoxemia (PO2 <9.3kPa), PO2 >10torr, production of purulent sputum, crackles on auscultation, tachypnea >22 breaths/min, tachycardia, bronchial breathing.

Studies included required affirmative outcomes on two or three of these indicative measures for a positive diagnosis of pneumonia.

Relationship between Pneumonia and Dysphagia/Aspiration
There is level 1a evidence that dysphagia and aspiration may both be associated with an increased risk of developing pneumonia. This association appears to be proportional to the severity of aspiration.

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.

Dysphagia Screening Protocols
There is level 2 evidence that the introduction of swallow screening may reduce the incidence of pneumonia among patients with dysphagia when compared to no screening protocol or usual care.

Prevention of Pneumonia Post-Stroke
There is level 1a evidence from a meta-analysis that the use of angiotensin-converting enzyme inhibitors reduces the relative risk of developing pneumonia when compared to placebo or other antihypertensive agents.

There is level 1b evidence that metoclopramide may improve incidence of pneumonia and resultant days on antibiotic treatment, episodes of aspiration, and swallowing outcome in dysphasic patients following stroke compared to placebo. There was no observed effect on mortality.

There is level 4 evidence that cilostazol may improve the incidence of pneumonia when compared to patients not given the drug.

Clinical Screening Methods
A large number of different screening methods exist for dysphagia with a wide variation of sensitivity (0-100%), specificity (50-92%) and predictive values.

There was a wide range of sensitivity (47.8-100%) and specificity (50-100%) values for the water swallowing test and its variations.

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.

The GUSS screening tool has 100% sensitivity and 69% specificity to predict aspiration risk.

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 screening tests conducted alone.

There is no ideal volume of water that is used to assess dysphagia on the water swallowing test.There is level 4 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.

Bedside Clinical Examinations
There was a wide range of sensitivity (68-97%) and specificity (53-86%) values for the different bedside clinical examinations.

Videofluoroscopic Modified Barium Swallow Examination
Videofluoroscopic Modified Barium Swallow studies are considered the gold standard for dysphagia/aspiration diagnosis.

There is level 3 evidence that scintigraphic and videofluoroscopic (VFS) results may be associated with swallowing function. Furthermore, scintigraphy provided good predictive values for VFS results (70-95%).

Sensitivity and specificity values for scintigraphy in predicting laryngeal penetration and/or aspiration were between 17-77% and 69-92%, respectively.

Flexible Endoscopic Evaluation of Swallowing
There is conflicting level 1b and level 2 evidence regarding the reported incidence of pneumonia after flexible endoscopic evaluation of swallowing (FEES) is used versus facial oral tract therapy or videofluoroscopy.

There is level 4 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.

Pulse Oximetry
It is unclear whether pulse oximetry is a useful tool in the detection of dysphagia and aspiration following stroke. The low sensitivity and specificity values reported (minimum 13% and 39%, respectively) call into question its clinical validity.

Ultrasonography
There is level 2 evidence that both ultrasonography and videofluoroscopy provide comparable results.

There is level 2 evidence that ultrasonography may be able to identify significant differences between factors involved in the diagnosis of dysphagia while approaching high levels of sensitivity (70-73.3%) and specificity (66.7-66.7%).

Dietary Modifications
There is level 1b and level 2 evidence supporting diets involving thickened liquids improving overall swallow safety and reducing incidence of aspiration pneumonia versus lower viscosity diets.

There is level 2 evidence suggesting that thin fluids may be associated with an increase of total fluid intake however, it is also associated with an increase in aspiration pneumonia.

Swallowing Treatment Programs
There is level 1b evidence supporting high intensity swallowing therapy with dietary prescription for better recovery of normal diet and swallowing ability in patients with dysphagia post-stroke compared to a lower intensity therapy or usual care.

Regarding formal dysphagia therapy, there is level 1a evidence that oral strength training may not be beneficial, while there is level 2 evidence that swallowing therapy and physical therapy are effective in reducing dysphagia.

There is level 2 evidence that acupuncture combined with physical therapy is more effective in treating dysphagia than physical therapy alone.

Non-Oral Feeding
There is conflicting level 2 evidence for whether oral feeding or nasogastric tube feeding increases the incidence of aspiration pneumonia among dysphasic patients.

There is level 2 evidence that a 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.

Selection of Feeding Tubes
There is level 1b evidence from a large, multicentre RCT that nasogastric tube feeding may decrease the incidence of death and poor functional outcome. The same study suggests that the type of tube feeding may not affect incidence of pneumonia however, there is level 1b evidence from a lower powered RCT suggesting a positive effect of gastro-enteric tubes.

There is conflicting level 1b evidence regarding the effect of gastrostomy tubes on mortality, proportion of prescribed feed delivered, and weight gained.

It is unclear which method of tube feeding (gastrostomy tube vs. nasogastric tube) is associated with a greater increase in the incidence of pneumonia.

Mode of Nutritional Intake
There is level 3 evidence that oral intake versus tube feeding may be related to stroke severity.

There is level 4 evidence that oral intake versus tube feeding at discharge may be associated with lower age and improved functional independence during acute care.

Electrical Stimulation
There is conflicting level 1a, level 1b, and level 2 evidence that transcutaneous pharyngeal electrical stimulation may not improve swallowing function when compared to traditional swallowing therapy.

There is level 2 evidence from multiple RCTs that NMES is effective in treating dysphagia, more so when combined with traditional therapies.

There is level 2 evidence that electrical stimulation may improve swallowing function and the incidence and severity of penetration-aspiration when compared to thermal-tactile stimulation.

Thermal Application
There is conflicting level 1b and level 2 evidence regarding the effect of intensity and presence of thermal application on the incidence of aspiration and penetration.

There is level 1b evidence that swallowing efficiency is improved, specifically among patients with supranuclear lesions after dry swallow preceded by ice massage of the oral cavity.

Pharmacotherapy
There is level 1b evidence that nifedipine may be associated with improved swallowing function versus placebo. Level 2 evidence indicates that cilostazol prescribed with aspirin may not have an effect on swallowing function compared to aspirin alone.

There is level 2 and level 4 evidence that treatment of dysphagia with cabergoline, amantadine, imidapril, or cilostazol may reduce the incidence of aspiration and subsequent pneumonia when compared to no treatment.

Transcranial Direct Current Stimulation
There is level 1a evidence suggesting that transcranial direct current stimulation (tDCS) may improve functional severity of dysphagia when compared to sham stimulation.

Repetitive Transcranial Magnetic Stimulation
There is level 1a evidence that repetitive transcranial magnetic stimulation (rTMS) may improve penetration and aspiration, swallowing function and functional disability compared to sham stimulation.

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.

16. Nutritional Interventions Following Stroke

Prevalence of Malnutrition Following Stroke
The prevalence of malnutrition varies from 6 - 62% post stroke, depending on timing of assessment and criteria used to define malnutrition.

There is currently no “gold standard” for the assessment of nutritional status, and various methods of detection may be used.

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 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
There is level 1b evidence that glucose-potassium insulin injections significantly reduce glucose levels and systolic blood pressure post stroke; no clinical benefits were observed.

There is level 1b evidence that administration of Metformin is ineffective in reducing glucose levels post stroke.

There is level 2 evidence that treadmill exercise significantly reduces insulin levels but not glucose levels post stroke.

There is level 3 evidence that patients with impaired glucose regulation post stroke are at significantly greater risk for mortality than those with normal glucose regulation; no differences in dependency or stroke recurrence were observed.

Vitamin D Deficiency Following Stroke

There is level 1b evidence that a single dose of Vitamin D2 (100,000 units) significantly increases 25-hydroxyvitamin D levels for up to 16 weeks; no effects on blood pressures, cholesterol levels, and albumin levels were observed.

There is Level 1b evidence that a 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-hydroxyvitamin D levels and reduces risk of mortality when compared to no supplementation; no effects on odds of good outcome were observed.

Lipid Profiles Following Stroke
There is level 1b evidence that Atorvastatin 80mg/d is effective in reducing total cholesterol and LDL levels and increasing HDL levels post stroke.

Enteral Feeding
There is level 1b evidence that gastric tube feeding such as PEG is associated with fewer mechanical complications and greater consumed intake post stroke compared to NG feeding.

There is level 1b evidence that enteral protein supplementation post stroke does not differ significantly from standard enteral nutrition in its effect on malnutrition, based on biochemistry and/or body composition.

There is level 1b and level 2 evidence that early enteral feeding does not differ significantly from late or delayed enteral feeding in its effects on poor outcome post stroke.

Oral Supplementation
There is level 1a evidence that oral nutritional supplementation improves the calorie-protein intake of patients post stroke.

There is level 1a evidence that oral nutritional supplementation does not reduce the risk of death or dependency post stroke.

There is level 1a evidence that the ALAnerv nutritional supplement may significantly reduce total lipid levels and increase HDL levels compared to conventional treatment post stroke.

Dysphagia Treatment
There is level 1b evidence that high-intensity dysphagia therapy results in improved swallowing function and less time to resuming a normal diet post stroke.

There is level 1b and level 2 evidence that dysphagia therapy does not reduce the risk of death or dependency post stroke, regardless of treatment intensity or diet type.

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.

There is level 2 evidence that long-term NG tube feeding post stroke results in greater levels of malnutrition than oral feeding.

Total Parenteral Nutrition
There is currently no evidence regarding the efficacy of total parenteral nutrition in the treatment of patients post stroke.

17. Medical Complications Post Stroke

Bladder Dysfunction

There is Level 1a evidence that pelvic floor training improves muscle control and reduces urinary incontinence when compared to standard care, but there is conflicting Level 1b evidence as to whether it improves health-related quality of life.

There is Level 1a evidence that traditional Chinese medicines reduce urinary incontinence but do not improve functional outcomes.

There is Level 1b evidence that transcutaneous electrical nerve stimulation reduces urinary incontinence when compared to no treatment, and that stimulation is more effective at 20Hz than 75Hz.

There is Level 1b evidence that the time of day for catheter removal does not impact subsequent urinary incontinence.

There is limited Level 2 evidence that a functionally-oriented rehabilitation program reduces urinary incontinence and improves wellbeing when compared to a conventional Bobath approach.

There is limited Level 2 evidence that bladder reconditioning prior to catheter removal does not impact subsequent urinary incontinence.

There is limited Level 3 evidence that indwelling urinary catheters are associated with worse outcomes, including urinary tract infections.

Bowel Dysfunction

There is Level 1b evidence that a 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.

There is Level 1b evidence that a traditional Japanese medicine, Diakenchuto, reduces constipation post stroke when compared to routine care.

There is Level 1b evidence that a protocol of tui-pushing and point sticking reduces constipation post stroke when compared to routine care.

Venous Thromboembolism

There is limited Level 2 evidence that bowel training is most efficient when coinciding with previous bowel regimens, but schedule of suppository use did not have an effect.

There is 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.

There is Level 2 evidence that unfractionated heparin reduces the incidence of deep vein thrombosis when compared to placebo.

There is Level 2 evidence that unfractionated heparin is no more effective than intermittent pneumatic compression or neuromuscular electrical stimulation in reducing the incidence of deep vein thrombosis.

There is Level 1a evidence that intermittent pneumatic compression reduces the incidence of deep vein thrombosis when compared to standard care, although there is limited Level 2 evidence that suggests otherwise.

There is Level 1a evidence that graded compression stockings are no more effective than standard care in reducing the incidence of deep vein thrombosis post stroke.

There is Level 1b evidence that thigh-high graded compression stockings reduce the incidence of deep vein thrombosis when compared to below-knee stockings.

Seizures
There is Level 1b and Level 2 evidence that lamotrigine, gabapentin, and carbamazepine are similar in reducing the rate of recurrent post-stroke seizures, but carbamazepine is more poorly tolerated.

There is Level 1b evidence that valproic acid does not prevent post-stroke seizures when compared to placebo, but may confer neuroprotective effects.

Osteoporosis
There is Level 1a evidence that bisphosphonates preserve bone mineral density post stroke when compared to placebo, although there is limited Level 2 evidence that suggests otherwise.

There is Level 1b evidence that vitamin D preserves bone resorption and reduces the rate of fractures when compared to placebo.

There is Level 1b evidence that vitamin B does not reduce the rate of fractures when compared to placebo.

There is limited Level 2 evidence that vitamin K preserves bone mineral density and enhances bone metabolism when compared to no treatment.

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

Central Pain
There is Level 1b evidence that lamotrigine reduces central pain post stroke when compared to placebo.

There is Level 1b evidence that gabapentin reduces central pain post stroke when compared to placebo.

There is Level 1b evidence that propofol reduces central pain post stroke when compared to placebo.

There is Level 1b evidence that high-dose levorphanol is more effective than low-dose Levorphanol in reducing central pain post stroke.

There is Level 1b evidence that apitoxin is more effective than saline during acupuncture in reducing central pain post stroke.

There is Level 1b evidence that levetiracetam is no more effective than placebo in reducing central pain post stroke.

There is Level 1b evidence that carbamazepine is no more effective than placebo in reducing central pain post stroke.

There is Level 1b evidence that duloxetine is no more effective than placebo in reducing central pain post stroke.

There is Level 1b evidence that ketamine is no more effective than placebo in reducing central pain post stroke.

There is Level 1b evidence that morphine is no more effective than placebo in reducing central pain post stroke.

There is 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.

There is Level 2 evidence that naloxone is no more effective than placebo in reducing central pain post stroke.

There is conflicting Level 1b evidence as to whether pregabalin is more effective than placebo in reducing central pain post stroke.

There is conflicting Level 1b evidence as to whether amitriptyline is more effective than placebo in reducing central pain post stroke.

There is conflicting 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.

Fatigue
There is Level 1b evidence that modafinil reduces fatigue post stroke when compared to placebo.

There is Level 1b evidence that OSU-6162 reduces fatigue post stroke when compared to placebo.

There is Level 1b evidence that a combination of cognitive therapy and graded activity training reduced fatigue post stroke when compared to cognitive therapy alone.

There is Level 1b evidence that astragalus membranaceus, a traditional Chinese herbal medicine, yields a short-term reduction in fatigue post stroke when compared to placebo.

There is Level 1b and Level 2 evidence that antidepressants do not reduce fatigue post stroke.

There is Level 2 evidence that mindfulness-based stress reduction reduces fatigue post stroke when compared to no therapy.

There is Level 2 evidence that a fatigue management program does not reduce fatigue post stroke when compared to a stroke education program.

Insomnia
There is Level 1a evidence that intradermal acupuncture reduces insomnia when compared to sham acupuncture.

There is Level 1b that acupuncture combined with music therapy reduces insomnia when compared to acupuncture alone.

18. Post Stroke Depression and Mood Disorders

Prevalence of Post-Stroke Depression
Approximately a third of 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.

Assessment of Post-Stroke Depression
Diagnosis of post-stroke depression should be conducted by a mental healthcare professional in Structured Clinical Interview as per the criteria outlined in the DSM-V.

Screening for post-stroke depression can be conducted using a variety of validated assessment tools. However, the Patient Health Questionnaire 9 has shown relatively high sensitivity, specificity, and clinical utility.

Detection of post-stroke depression is often inconsistent, which may be due to the heterogeneity of screening tools.

Compliance with guidelines for screening is generally poor, which may be due to lack of time and knowledge.

Risk Factors for Post-Stroke Depression

There is Level 4 and Level 5 evidence that risk factors for post-stroke depression include prior depression, functional impairment, cognitive deficit, and stroke severity.

There is conflicting Level 4 and Level 5 evidence as to whether variables such as age, sex, socioeconomic status, cardiovascular comorbidities, and stroke severity are risk factors for post-stroke depression.

There is conflicting Level 4 and Level 5 evidence as to whether lesion location is a risk factor for post-stroke depression.

Consequences Associated with Post-Stroke Depression
There is Level 2 and Level 3 evidence that depression has a significant, negative impact on functional outcomes post stroke.

Physical Function and Post-Stroke Depression
There is Level 2 and Level 3 evidence that that depression has a significant, negative impact on physical functional post stroke.

Cognitive Function and Post-Stroke Depression
There is Level 2 evidence that that depression has a significant, negative impact on cognitive function post stroke.

Mortality and Post-Stroke Depression
There is conflicting Level 2 and Level 3 evidence as to whether depression post stroke is associated with an increased risk of mortality.

Pharmacotherapy and the Prevention of Post-Stroke Depression
There is Level 1a evidence that early initiation of fluoxetine is associated with reduced risk of post-stroke depression when compared to placebo.

There is Level 1b evidence that early initiation of escitalopram is associated with reduced risk of post-stroke depression when compared to placebo.

There is Level 1b evidence that early initiation of nortriptyline is associated with reduced risk of post-stroke depression when compared to placebo.

There is Level 1b evidence that early initiation of milnacipran is associated with reduced risk of post-stroke depression when compared to placebo.

There is Level 1b evidence that early initiation of duloxetine is associated with reduced risk of post-stroke depression when compared to no antidepressant medication.

There is Level 1b evidence that early initiation of mianserin is not associated with reduced risk of post-stroke depression when compared to placebo.

There is conflicting Level 1b evidence regarding the efficacy of sertraline in reducing the risk of post-stroke depression when compared to placebo.

There is Level 2 evidence that early initiation of mirtazapine is associated with reduced risk of post-stroke depression when compared to no antidepressant medication.

Care Provision and the Prevention of Post-Stroke Depression
There is Level 1a evidence that community outreach, using post mail or telephone calls, does not reduce depressive symptoms when compared to standard care.

There is Level 1b evidence that a pre-discharge home visit by an occupational therapist reduces short-term depressive symptoms when compared to a pre-discharge hospital interview.

There is Level 1b evidence that motivational interviewing improves mood and reduces depressive symptoms when compared to standard care.

There is Level 1b and Level 2 evidence that home visits from nurses and therapists do not reduce depressive symptoms when compared to information provision or standard care.

There is conflicting Level 2 evidence regarding the effectiveness of coordinated or integrated care programs on reducing depressive symptoms when compared to standard care.

Omega-3 Fish Oil
There is level 1b evidence that fish oil supplementation following does not impact mood post stroke.

B-Vitamins
There is level 1b evidence that Vitamin B therapy, administered over a long period, is associated with reduced risk of post-stroke depression.

Heterocyclic Antidepressants
There is Level 1a evidence that heterocyclic antidepressants are as effective as fluoxetine in reducing depressive symptoms post stroke.

There is Level 1a evidence that nortriptyline reduces depressive symptoms post stroke when compared to placebo.

There is Level 1b evidence that mianserin reduces depressive symptoms post stroke when paired with imipramine or desipramine, but is more effective with imipramine.

There is Level 2 evidence that desipramine does not reduce depressive symptoms post stroke when compared to placebo.

Selective Serotonin Reuptake Inhibitors
There is Level 1a evidence that fluoxetine is no more effective than heterocyclic antidepressants in treating depressive symptoms post stroke.

There is Level 1b evidence that citalopram reduces depressive symptoms post stroke when compared to placebo.

There is Level 1b evidence that sertraline does not reduce depressive symptoms post stroke when compared to placebo.

There is conflicting Level 1b evidence regarding the effectiveness of fluoxetine in treating depressive symptoms post stroke when compared to placebo.

Adjunctive Light Therapy
There is Level 1b evidence that adjunctive high-intensity light therapy is more effective than moderate-intensity light therapy in treating depressive symptoms post stroke.

Noradrenaline Reuptake Inhibitors
There is Level 1b evidence that reboxetine reduces depressive symptoms post stroke when compared to placebo.

Serotonin and Noradrenaline Reuptake Inhibitors
There is limited Level 4 evidence that venlafaxine reduces depressive symptoms post stroke.

Psychostimulants
There is Level 1b evidence that methylphenidate reduces depressive symptoms post stroke when compared to placebo.

There is Level 3 evidence that methylphenidate is as effective as nortriptyline in reducing depressive symptoms post stroke.

GABA Receptor Modulators
There is level 1a evidence that nefiracetam does not reduce depressive symptoms post stroke when compared to placebo.

Monoamine Oxidase Inhibitors
There is Level 2 evidence that selegiline does not reduce depressive symptoms post stroke when compared to placebo.

Melatonin Agonist
There is limited Level 4 evidence that valdoxan reduces depressive symptoms post stroke.

Statins
There is limited Level 2 evidence that statins may reduce depressive symptoms post stroke when compared to no medications.

Antidiabetics
There is limited Level 2 evidence that pioglitazone with fluoxetine reduces depressive symptoms post stroke when compared to metformin with fluoxetine.

Alternative Medicine
There is Level 1b evidence that treatment with the herbal preparation, Free and Easy Wanderer Plus, is as effective as fluoxetine and more effective than placebo in reducing depressive symptoms post stroke.

Care Management
There is Level 1b evidence that an active care management program enhances the effectiveness of pharmacologic treatment for post stroke depression.

Stroke Recovery and Pharmacologic Treatment of Post-Stroke Depression
There is Level 1a evidence that antidepressants improve post-stroke functional recovery when compared to no medication.

There is Level 1a evidence that fluoxetine improves post-stroke functional recovery, but not cognitive or motor function, when compared to placebo.

There is Level 1a evidence that nortriptyline improves post-stroke functional recovery when compared to placebo.

There is Level 1a evidence that trazodone does not improve post-stroke functional recovery when compared to placebo.

There is Level 1b evidence that methylphenidate improves post-stroke functional recovery when compared to placebo.

There is Level 1b evidence that maprotiline does not improve post-stroke functional recovery when compared to placebo.

There is Level 1b evidence that desipramine does not improve post-stroke functional recovery when compared to fluoxetine or trazodone.

There is limited Level 2 evidence that selective serotonin reuptake inhibitors yield greater improvement in functional recovery when initiated pre stroke than post stroke.

Mortality and Pharmacological Treatment of Post-Stroke Depression

There is Level 1b evidence that early treatment with nortriptyline or fluoxetine is associated with improved long-term survival post stroke when compared to placebo.

There is Level 2 evidence that antidepressants are associated with improved short-term survival post stroke when compared to no medications.

There is conflicting Level 2 evidence regarding the effect of selective serotonin reuptake inhibitors, initiated before or after stroke, on post-stroke mortality.

Cognitive-Behavioural Interventions
There is conflicting Level 1a evidence as to whether cognitive behavioral therapy reduces depressive symptoms post stroke when compared to attention placebo or usual care.

There is Level 1a evidence that problem-solving therapy is does not reduce depressive symptoms post stroke when compared to usual care.

There is Level 1b evidence that aphasic behavioural therapy reduces depressive symptoms post stroke when compared to usual care.

There is Level 4 evidence that group-based cognitive behavioural therapy reduces depressive symptoms post stroke in the short term.

Combined Therapy
There is Level 1b evidence that psychosocial-behavioural therapy in combination with antidepressants is more effective than antidepressants alone in reducing depressive symptoms post stroke.

Supportive Interventions
There is Level 1b evidence that a goal achievement program reduces depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that a transitional care program reduces depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that a self-management program does not reduce depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that customized occupational therapy does not reduce depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that ecosystem focused therapy does not reduce depressive symptoms post stroke when compared to an education program.

Music Therapy
There is Level 2 evidence that music therapy does not reduce depressive symptoms post stroke when compared to usual care.

There is limited Level 2 evidence that music-listening therapy improves mood post stroke when compared to language-listening therapy and usual care.

There is limited Level 2 evidence that music-movement therapy does not reduce depressive symptoms post stroke when compared to usual care.

Art Therapy
There is Level 1b evidence that art therapy reduces depressive symptoms post stroke when compared to standard care.

Relaxation Therapy

There is limited Level 4 evidence that deep unilateral nostril breathing does not reduce depressive symptoms post stroke.

There is limited Level 4 evidence that autogenic training reduces psychological tension post stroke.

Physical Activity
There is Level 1a evidence that resistance training does not reduce depressive symptoms post stroke when compared to relaxation training or usual care.

There is Level 1b evidence that a specialized, therapeutic exercise program reduces depressive symptoms post stroke when compared to usual care.

There is Level 1b evidence that yoga does not reduce depressive symptoms post stroke when compared to usual care.

There is conflicting Level 1b and Level 2 evidence as to whether aerobic exercise reduces depressive symptoms post stroke when compared to usual care.

There is conflicting Level 1b and Level 2 evidence as to whether circuit training reduces depressive symptoms post stroke when compared to basic exercise or usual care.

There is conflicting Level 1b and Level 2 evidence as to whether exercise with technological enhancements reduce depressive symptoms post stroke when compared to standard exercise.

There is conflicting Level 1b, Level 2, and Level 4 evidence as to whether group exercise programs reduce depressive symptoms post stroke when compared to individual exercise or usual care.

There is conflicting Level 2 evidence as to whether an adaptive physical activity program reduces depressive symptoms post stroke when compared to usual care.

Speech Therapy
There is Level 1b evidence that speech therapy does not reduce depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that combined speech and orofacial therapies are more effective in reducing depressive symptoms post stroke than speech therapy alone.

Hyperbaric Oxygen Therapy
There is Level 1b evidence that hyperbaric oxygen therapy with fluoxetine reduces depressive symptoms post stroke when compared to either intervention alone.

There is Level 1b evidence that hyperbaric oxygen therapy reduces depressive symptoms post stroke when compared to the psychoactive medication Deanxit.

Electroconvulsive Therapy
There is limited Level 4 evidence that electroconvulsive therapy (ECT) reduces depressive symptoms post stroke.

Repetitive Transcranial Magnetic Stimulation
There is Level 1a evidence that repetitive transcranial magnetic stimulation (rTMS) reduces depressive symptoms post stroke.

Transcranial Direct Current Stimulation
There is limited Level 4 evidence that transcranial direct current stimulation (tDCS) reduces depressive symptoms post stroke.

Acupuncture

There is Level 1b evidence that a combination of acupuncture and herbal medicine reduces depressive symptoms post stroke when compared to standard care.

There is Level 1b evidence that acupuncture is no more effective than sham acupuncture in reducing depressive symptoms post stroke.

There is Level 1b evidence that dense cranial acupuncture reduces post-stroke depressive symptoms in the short term when compared to non-invasive cranial acupuncture.

There is Level 1b and Level 2 evidence that acupuncture reduces post-stroke depressive symptoms in the short term when compared to antidepressants.

There is Level 2 and Level 4 evidence that electroacupuncture reduces depressive symptoms post stroke.

Acupressure
There is limited Level 2 evidence that meridian acupressure reduces depressive symptoms post stroke when compared to standard care.

Reiki Treatment
There is Level 1b evidence that reiki treatment does not reduce depressive symptoms post stroke when compared to sham reiki or no treatment.

Prevalence of Post-Stroke Emotionalism
Approximately a fifth of individuals experience emotionalism post stroke. The majority of individuals develop emotionalism in the acute phase of stroke and recover in the chronic phase.

Risk Factors for Post-Stoke Emotionalism
There is Level 4 and Level 5 evidence that cognitive deficit and anterior lesions are risk factors for emotionalism post stroke.

There is conflicting Level 4 and Level 5 evidence as to whether factors such as age, sex, stroke severity, functional impairment, and lesion laterality are risk factors for post-stroke emotionalism.

Treatment of Post-Stroke Emotionalism
There is Level 1a evidence that selective serotonin reuptake inhibitors improve symptoms of emotionalism post stroke when compared to placebo.

There is Level 1a evidence that tricyclic antidepressants improve symptoms of emotionalism post stroke when compared to placebo.

Guidelines for Post-Stroke Mood
Screening for post-stroke depression should be conducted in all individuals following stroke using a validated tool and throughout the continuum of care.

Assessment of post-stroke depression should be conducted by an experienced health professional in individuals with a high probability of clinically significant depression.

Treatment with an appropriate antidepressant for a period of 6 to 12 months should be considered for individuals diagnosed with a depressive disorder; regular monitoring of response by a health professional is required.

Treatment with psychotherapy as an adjunct to antidepressants is a reasonable consideration, given demonstrated efficacy in primary depressive disorders; other non-pharmacological interventions require more research.

Prophylactic treatment for post-stroke depression using antidepressants is not recommended.

19. Community Reintegration

Social Support and Discharge Destination
Rehabilitation programs that focus on the transition from hospital to homes are highly valued by the patients and caregivers; however, many stroke survivors still expressed social barriers that negatively impact the reintegration within the community and in their homes.

Factors such as admission FIM (motor and cognitive), age, and marital status were found to be significantly associated with discharge destination.

Social Support and Functional Status
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 may appear to be most beneficial to stroke patients.

Social Support and Quality of Life
The presence and size of social support networks as well as the perceived effectiveness of social support networks have a positive influence on physical recovery, psychological distress, and quality of life post stroke.

Higher levels of support are associated with greater functional gains, less depression and improved mood and social interaction.

The size and perceived effectiveness of social support networks are important predictors of discharge destination.

Having a pet was found to facilitate physical, psychological, and social recovery after a stroke.

Social Work Interventions
There is level 1a evidence that social work interventions providing counselling along with information and education for stroke patients and their families are not associated with improvements on measures of independence or social activity.

Specialized Social Support Network Interventions
There is level 1b evidence that a specialized social support intervention that includes the stroke patient's social support network is not effective in improving perceived social support or functional recovery. Subgroup analyses suggest that there may be some benefit in terms of physical performance and instrumental activities of daily living for healthier, non-frail stroke survivors.

Home-Based Support and Care Management
There is level 1a evidence that home-based support and care management interventions are not associated with improved social activity, mood, quality of life or physical independence. However, there is level 1b evidence that participation in a social worker led program of care coordination featuring frequent, regularly-scheduled contact may result in improved mental health.

There is level 1a evidence that involvement with a stroke liaison worker or case manager is associated with increased knowledge about stroke and satisfaction with services.

There is level 1a evidence that social support interventions may be associated with a reduction in caregiver burden or strain.

There is conflicting level 1b evidence regarding the efficacy of occupational therapist led home-visits on mental health and hospital readmission.

Active Case Management

There is level 2 evidence that active case management does not improve social activity, quality of life, and mood.

Discharge Planning Programs
There is limited level 2 evidence that individualized, caregiver-oriented discharge planning does not improve caregiver preparedness, quality of care, and patient outcomes, but may improve caregiver satisfaction with discharge needs.

Patient Education Programs
There is limited and conflicting level 2 evidence regarding the effect of caregiver training programs on the patients' and caregivers' well-being.

There is limited level 2 evidence that community-based nurse-led education programs for patients may improve stroke knowledge.

There is limited level 2 evidence that psychoeducational interventions can improve psychological functioning in both stroke individuals and their partners.

There is limited and conflicting level 2 evidence regarding the effect of providing re-integration guidelines to patients.

Community Based Rehabilitation Programs
There is limited level 1b evidence that community walking programs are more efficient than usual care at improving walking performance and the impact of stroke on the patient.

Day Services
There is level 1b evidence that early attendance (within 6 months of stroke) at a day service is associated with improved participation in leisure activities.

Patient Self-Management Programs
There is level 1a evidence that the Bridges Self-Management Program is not effective in the short term rehabilitation of self-efficacy.

There is level 1b evidence that self-management programs may be very beneficial in improving self-efficacy post-stroke given targeted interventions to improve specific areas of efficacy. However, more research is needed.

Effects of Caregiving on the Caregiver
Commonly identified effects of caregiving on the caregiver include increasing psychological distress, increased financial burden, decreased social contact and activity, increased risk for depression, increased carer stress, strain or burden and an overall decrease in quality of life.

Decreased social contact and activity in itself may contribute to increased carer strain, increased risk of depression and decreased life satisfaction.

Reports concerning the influence of patient characteristics vary with the effect in question. However, age, severity of stroke and stroke-related impairments, functional status and cognitive status have been reported as influencing caregiver outcomes.

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.

The Family Caregiver and Social Support Interventions
There is level 1a evidence that group-based programs and support may improve stroke-related knowledge and family structure however, it may not have an impact on caregiver psychological health.

There is level 1a evidence that a personalized patient program in which the caregiver is included and that is designed to provide social support for patients who have sustained a stroke improves social support and self-efficacy. There is level 1b evidence that such programs do not improve measures of function or affect.

There is level 1b evidence that 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.

There is level 1b evidence that a caregiver-mediated home-based programs involving exercise may improve measures of daily living in stroke patients.

Family Interactions and Stroke

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.

Information Provision and Education

There is level 1a evidence from a meta-analysis that psychoeducational interventions have no significant effect on the burden or health of caregivers but may benefit family functioning.

There is level 1a evidence of a positive benefit, associated with the provision of information and education through a variety of intervention types. Education sessions may have a greater effect on outcome than the provision of information materials alone.

There is level 1a evidence that skills training is associated with a reduction in depression.

There is level 1b evidence that a 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.

There is level 1b evidence that training in basic nursing skills improves outcomes of depression, anxiety and quality of life for both the caregiver and the stroke patient.

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.

Social and Leisure Activities Post Stroke

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.

Leisure Intervention and Social Participation

When considered individually, there appears to be conflicting evidence as to the benefit of leisure therapy post-stroke and following discharge. However, based on the information from a meta-analysis using pooled data from the same RCTs, there is level 1a evidence that leisure therapy is associated with modest improvement in leisure activity.

There is level 1b evidence that 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.

There is level 1a evidence that participation in group education and exercise programs result in improved physical outcomes, but not social/leisure participation outcomes.

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.

There is level 2 evidence that sexual rehabilitation programs may not be effective in remediating sexual function.

There is level 3 evidence that sexual issues should be discussed during rehabilitation and addressed again after transition to the community when the stroke survivor and significant other are ready.

Driving Assessment

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.

Interventions and Driving Performance

There is level 1b evidence that a visual attention-retraining program is no more effective than traditional visuoperception retraining in improving the driving performance of patients with stroke.

There is level 1b evidence that a 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.

There is level 1b evidence that Dynavision training is not effective in improving the results of on-road assessments in individuals with stroke.

Returning to Work Post 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.

There is level 1b evidence that structured workplace intervention can improve return to work rates.

There is level 3 evidence that stroke survivors who worked prior to their stroke should, if their condition permits, be encouraged to be evaluated for their potential to return to work.

Factors Influencing Community Reintegration

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.

21. Rehabilitation of Younger Patients Post Stroke

Incidence of Stroke for Younger Individuals
The incidence of stroke in young patients varies considerably across reports, ranging from 3 to 44 out of 100,000.

The incidence of stroke in young patients is notably lower than in older patients.

The incidence of stroke in young patients has increased over time.

Unknown Etiology
Up to one third of strokes in young people are of unknown etiology. However, this proportion is decreasing as diagnostic methods improve.

Hemorrhagic Etiology
The most common causes for hemorrhagic stroke in young patients include hypertension, arteriovenous malformation, ruptured aneurysm, or a combination of these factors.

Ischemic Stroke
The 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
Uncommon etiologies are likely in stroke patients under the age of 30.

There are many uncommon etiologies that have been recognized as risk factors for stroke in young patients, including but not limited to migraines, non-atheroclerotic vasculopathy, mitral valve prolapse, multifocal intracranial stenosis, extracranial dissection, and cardioembolism.

Modifiable Risk Factors
Smoking and hypertension are the most considerable risk factors for stroke in the young population.

Hyperlipidemia, diabetes mellitus, and elevated plasma homocysteine level are risk factors for stroke in the young population, particularly for those older than 35.

Drug use is an uncommon risk factor for stroke in general but is more common in the younger population.

Alcohol-related stroke events in the young population are relative 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 is a risk factor for stroke in the young population, with young women at an elevated risk.

Oral contraceptives play minor role in risk of stroke in the young population when paired with other factors.

Further research is required to determine whether chlamydia pneumoniae is a risk factor for stroke in the young population.

Non-Modifiable Risk Factors
Previous stroke in young patients is less common than in older patients.

Sex appears to be related to age at stroke onset, with young patients under the age of 35 more likely to be female and above the age of 35 more likely to be male.

Race appears to be an important risk factor for stroke in young populations, with elevated risk for young Black patients.

Atrial fibrillation appears to be an uncommon and understudied risk factor for stroke in young patients.

Mitral valve prolapse appears to be a minimal risk factor and infrequent sole etiology for stroke in young patients.

The significance of family history of stroke and patent foramen ovale as risk factors for stroke in young patients is unclear.

Pregnancy and postpartum state are unique periods of elevated stroke risk in young female patients.

Recovery and Prognosis for Young Stroke Patients
Young patients have better neurological recovery, less functional disability, and greater long-term survival post stroke than older patients.

Impaired cognitive performance and recurrent stroke may be associated with post-stroke recovery in young patients.

Rehabilitation of Young Stroke Patients
Rehabilitation of young patients post stroke is similar to that of older patients, with the main differences being the nature of neurological recovery and associated social issues.

Stroke rehabilitation programs with an emphasis on socialization and community integration could be effective for young patients.

Family Stress
Younger patients tend to achieve higher levels of functional recovery and independence post stroke than older patients, which commonly puts less stress on caregivers and close relations.

Younger patients tend to experience different social and adjustment issues post stroke compared to older stroke patients.

Caregivers reported more emotional distress when caring for patients exhibiting more depressive symptoms and greater cognitive impairment post stroke.

Institutionalization
Institutionalization is required infrequently in young stroke patients as a result of better prognosis and greater availability of caregivers.

Functional improvement was found to be more significant when young stroke patients were discharged home than when they were institutionalized.

Return to Work for Young Stroke Patients
Vocational issues are important for young patients post stroke.

Inability to return to full employment is associated with more severe stroke, cognitive impairment, poor functional recovery, and working class position.

Reported rates of return to work one year post stroke range from 7% to 75%.

Future Needs of Younger Stroke Patients
Young patients need to be aware of possible long-term health consequences post stroke, including recurrent stroke and sexual impairment.

Depression, anxiety, and fatigue can occur in young patients post stroke.

Young patients need to be connected with support organizations and individuals with similar experiences.

22. The Rehabilitation of Severe Stroke

Stroke Severity and Recovery
Animal studies, combined with human neuroimaging, demonstrate that recovery post-stroke is largely dependent on peri-lesional intact cortical areas which subsume a similar function and can take over the lost function. Larger strokes have reduced potential for this to occur.

Neuroimaging studies suggest that although increased bilateral activity may occur following a stroke, this does not necessarily translate into functional recovery. A combination of residual activity, compensatory actions by surrounding regions, and cortical reorganization may play a role in the activity observed.

Although anatomical integrity of the brain may explain part of the recovery, recent studies suggest that cortical connectivity may better predict clinical change in the first three months after a stroke. More studies are needed to investigate the cortical connectivity patterns in patients post-stroke.

Issues in Severe Stroke Rehabilitation
Despite having the greatest number of impairments and the most severe disabilities, patients often have limited access to rehabilitation.

Limited access to rehabilitation may be a result of many factors but in particular concerns about reduced potential for functional gains comparable to those individuals with moderate sized strokes.

Rehabilitation of individuals with severe stroke is associated with a greater use of rehabilitation resources.

Definition of Severe Stroke
Stroke severity has been defined in a variety of ways. Common definitions are 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.

Funding Models and Severe Strokes
Severe strokes may be the most negatively affected by the type of funding models employed.

Severe Stroke Admission to ICUs
Severe strokes are seldom admitted to intensive care units as compared with other types of critically ill or injured patients.

Patients with critical health issues in addition to severe stroke appear to have lower mortality rates when admitted to intensive care. Further research is needed to establish other specific outcome gains.

It is currently unclear whether stroke type influences the extent of the benefits that the ICUs may offer.

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
There is level 1a evidence that specialized interdisciplinary stroke rehabilitation reduces mortality in patients with severe stroke when compared to general rehabilitation programs.

There is 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.

There is conflicting level 1a and level 2 evidence regarding the effect of specialized interdisciplinary stroke rehabilitation programs on hospital length of stay.

There is conflicting level 4 evidence regarding functional gains of persons with severe stroke following specialised interdisciplinary inpatient stroke rehabilitation.

Functional outcomes suggest that rehabilitation of patients with severe stroke should emphasize discharge planning and reduction of post-stroke complications.

Slow-Stream Rehabilitation
Some data suggest that slow-stream stroke rehabilitation may result in less favourable outcomes when compared to the more intensive stroke rehabilitation program.

The utilization of slow stream rehabilitation should be dictated by the tolerance of the individual patient for therapy and not by preconceived notions about the amount of therapy that patients can successfully tolerate.

Severe Stroke Rehabilitation Ethics
More research needs to be conducted in the area of severe stroke prognosis.

Trial treatments may assist in creating a more accurate basis for ethical decision-making.

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