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Exercise for stroke prevention

Peter L Prior,[1,2] Neville Suskin[1,2,3]

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[1]Lawson Health Research Institution, London, Ontario, Canada
[2]St. Josephs Health Care Cardiac Rehabilitation and Secondary Prevention Program, London, Ontario, Canada
[3]Department of Medicine (Cardiology), Schulich School of Medicine & Dentistry, Western University, London, Ontario, Canada

Correspondence to
Dr Neville Suskin;
neville.suskin@lhsc.on.ca,
nsuskin@uwo.ca

ABSTRACT

We review evidence concerning exercise for stroke prevention. Plausible biological reasons suggest that exercise would be important in preventing stroke. While definitive randomised controlled trials evaluating the impact of physical activity (PA) and exercise on preventing stroke and mortality are lacking, observational studies, small randomised controlled trials and meta-analyses have provided evidence that PA and exercise favourably modify stroke risk factors, including hypertension, dyslipidaemia, diabetes, sedentary lifestyle, obesity, excessive alcohol consumption and tobacco use. It is, therefore, important to understand the factors associated with poststroke PA/exercise and cardiorespiratory fitness. Positively associated factors include self-efficacy, social support and quality of patients' relationships with health professionals. Negatively associated factors include logistical barriers, medical comorbidities, stroke-related deficits, negative exercise beliefs, fear of falling, poststroke fatigue, arthropathy/pain and depression. Definitive research is needed to specify efficacious behavioural approaches to increase poststroke exercise. Effective techniques probably include physician endorsement of exercise programmesto patients, enhancement of patient–professional relationships, providing patients an exercise rationale, motivational interviewing, collaborative goal-setting with patients, addressing logistical concerns, social support in programsmes, structured exercise programming, individualised behavioural instruction, behavioural diary recording, reviewing behavioural consequences of exercise efforts, reinforcing successful exercise performance. Exercise programming without counselling may increase short-term activity; simple advice or information-giving is probably ineffective. Older patients or those with cognitive impairment may need increased structure, with emphasis on behaviour per se, versus self-regulation skills. We support the latest American Heart Association/American Stroke Association guidelines (2014) recommending PA and exercise for stroke prevention, and referral to behaviourally oriented programmes to improve PA and exercise.

WHY EXERCISE SHOULD REDUCE THE RISK OF STROKE

There are a number of biological reasons why exercise might be beneficial in preventing stroke. Habitual exercise has been shown to have beneficial effects on a number risk factors for stroke, namely hypertension, dyslipidaemia, diabetes, physical inactivity, obesity, excessive alcohol consumption and tobacco use.[1–6] In addition, exercise increases high-density lipoprotein cholesterol (HDL-C), improves blood rheology and coronary artery endothelial function.[7] Moderately or highly active persons had a lower risk of stroke incidence or mortality than those with a low level of activity.[4] Stroke risk can be reduced with regular leisure-time physical activity (PA) in individuals of all ages and both sexes.[8] A 10-year cohort study in over 16 000 healthy men demonstrated an inverse association between greater baseline cardiorespiratory fitness and stroke mortality, with those in the high-fitness groups experiencing a 68% lower risk of stroke and death than those in the lowest-fitness group.[9] Moreover, the inverse association between aerobic fitness and stroke mortality remained after adjustments for cigarette smoking, alcohol consumption, body mass index, hypertension, diabetes mellitus and family history of coronary artery disease. Habitual exercise moderates the association between moderate-to-vigorous intensity exercise and onset of acute stroke: the risk of stroke acutely following moderate to vigorous exercise was significantly lower in subjects who had previously been physically active compared with those who had not (adverse rate ratio 2 vs 6.8).[10]

The above-mentioned benefits of exercise are consistent with the growing body of evidence that interventions (such as exercise training) that promote plaque stability and favourable changes in vascular wall function have important implications for the medical management of patients after stroke.[11]

SECONDARY PREVENTION: EVIDENCE OF EXERCISE BENEFITS IN PATIENTS WHO HAD A STROKE

Randomised controlled trial (RCT) mortality and morbidity (including recurrent stroke) evidence is lacking to substantiate beneficial effects of structured exercise training following transient ischaemic attack (TIA) or mild non-disabling stroke. However, we and others have provided emerging evidence that such a strategy results in improvement of exercise capacity and stroke risk factors.[12–16] In a prospective cohort study, we demonstrated that a 6-month duration cardiac rehabilitation (CR) programme, including pre–post exercise stress testing and recommended four times per week

(twice per week supervised sessions) of moderately intense exercise training, was feasible, safe and effective in 80 subjects. Favourable and significant intake-to-exit changes were observed in: mean aerobic capacity (+31%; p<0.001), total cholesterol/HDL ratio (−11.6%; p<0.001), waist circumference (−2.4 cm; p<0.001), body mass index (−0.5 kg/m^2 p=0.003) and body weight (−1.4 kg; p=0.001). Mean systolic (−3.2 mm Hg) and diastolic (−2.3 mm Hg) blood pressures changed favourably, but non-significantly. Compared with intake, 11 more individuals (25.6% increase) completed CR in the lowest-mortality risk category of the Duke Treadmill Score (p<0.001). Only three adverse events occurred and were reported to our ethics board, none of which was deemed related to the exercise intervention.[12]

The landmark RCT 'Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis' demonstrated that, with respect to stroke or death, aggressive medical management, including exercise and activity counselling, outperformed the combination of angioplasty and stenting plus aggressive medical management in patients with severe intracranial atherosclerotic stenosis following a TIA or stroke.[17] Moreover, in a substudy that examined the independent impact of each medical intervention, including exercise and activity counselling, only the achievement of a Physician-based Assessment and Counseling for Exercise score of at least 4 (participation in moderate physical exercise but less than four times per week or vigorous exercise but less than three times per week) was independently associated with a 40% lower risk of stroke, myocardial infarction or vascular death at 3 years.[18] Although the substudy examined this association only within the intensive medical arm alone, it was prespecified and included over 220 subjects, reinforcing the body of literature supporting the benefits of increased PA post-TIA or stroke to reduce the risk of subsequent vascular events.

Tang *et al* conducted a systematic review and meta-analysis on RCTs (18 trials with 930 subjects overall) evaluating the efficacy of exercise training in subjects at any time post-TIA or stroke, and demonstrated significant improvements of blood pressure, fasting blood glucose, fasting insulin and HDL-C; however, changes in exercise capacity were not reported.[19] In another recent meta-analysis comprising 22 RCTs evaluating lifestyle interventions in over 2500 patients post-TIA, no significant effect was found on cardiovascular morbidity or mortality or lipids profile; however, from subgroup analyses focused on trials with exercise training that lasted longer than 4 months, interventions that used at least three behaviour change techniques (BCTs) showed significant reductions in systolic blood pressure.[20] These studies point to the need for larger definitive studies exploring the impact of PA and structured exercise training on mortality and morbidity post-TIA or stroke.

INACTIVITY AND LOW FITNESS AMONG TIA AND STROKE SURVIVORS

Notwithstanding the potential secondary prevention benefits of PA and exercise, stroke survivors may be insufficiently active to increase cardiorespiratory fitness.[21] From systematic reviews and meta-analyses, free-living community-dwelling stroke survivors showed a mean of 4355 steps/day, lower than healthy elderly, with a preponderance of low-intensity compared with high-intensity PA.[22] Stroke survivors performed less than half the PA of age-matched normative values, as measured in steps/day.[23] Peak exercise capacity among survivors of generally mild strokes, as measured by the gold standard, peak oxygen consumption, was 26%–87% lower than healthy age-matched and sex-matched individuals, without clear evidence of improvement over time.[24] A more recent study of over 100 subjects 1–2 months post-TIA revealed their peak exercise oxygen consumption at the fifth percentile of age-related and sex-related normative values. Interestingly enough, while lower levels of PA, increasing age, female sex, history of cardiovascular disease and pulmonary disease were associated with lower oxygen consumption at peak exercise, stroke history and characteristics were not.[25]

Therefore, given their lower levels of PA and cardiorespiratory fitness, it is vital to identify key barriers and motivators affecting stroke survivors with respect to PA and exercise, to identify and apply strategies to address these and to apply or develop effective BCTs and integrated interventions to enhance PA and exercise.

FACTORS ASSOCIATED WITH PA AND EXERCISE AFTER STROKE

From their systematic review and meta-analysis based on community-dwelling samples, Field *et al* concluded that poststroke PA was associated positively with walking ability, balance and cardiorespiratory fitness and negatively with low mood, but not with age or demographic variables.[22] Among 61 stroke survivors with hemiparetic gait attending CR, barriers to attendance were mainly logistical, including severe weather, transportation, language-related barriers, costs, family responsibilities or lack of social support, with patients who were challenged socioeconomically or physically by stroke-related deficits as more susceptible to these.[26]

Self-efficacy, a key construct from Bandura's social-cognitive theory, is an individual's perception of or confidence in his or her own ability to complete a behavioural sequence to achieve a specified goal.[27] From a 2012 structured review of psychological and social factors, Morris *et al* identified self-efficacy, beliefs about PA and social support as important to PA after stroke. Self-efficacy was the most common and reliable predictor of exercise behaviour. Consequently, factors enhancing or impairing self-efficacy should tend to increase or decrease PA and exercise, respectively. Feelings of depression, fatigue and low motivation decreased the desire for PA, while

'self-determination' and goal achievement were associated with greater PA engagement.[28]

Based on community-dwelling samples, Thilarajah *et al* recently reported that modifiable factors associated with lower PA were worse physical functioning, fatigue, low cardiorespiratory fitness, low self-efficacy with respect to falls and balance, depression, and lower health-related quality of life. Non-modifiable factors included age and female sex, each only weakly associated with lower PA. Time elapsed from stroke was not related to PA. Findings concerning side of infarct, neglect and cognition were inconclusive.[29] These authors emphasised that physical function accounted for only half of the variance in poststroke PA levels; thus, while physical functioning needs to be maximised, attention to psychological factors, including depression and self-efficacy, is required to achieve optimal PA levels[29]—a conclusion consistent with findings from earlier reviews.[22 28]

Falls are common after stroke. A recent systematic review based principally on poststroke community samples reported frequencies of all fallers ranged from 23% to 55%, and of recurrent fallers from 5% to 42%.[30] An earlier review had reported that 37%–73% fall during the first 6 months after hospital discharge.[31] From meta-analysis, self-efficacy with respect to each of balance (r^2=0.14) and falls (r^2=0.16) was reported to have significant positive correlations with poststroke PA.[29] Risk factors associated with falling include impaired mobility, reduced balance, use of sedative or psychotropic medications, impaired self-care, depression, cognitive impairment and previous falling.[30] For older adults, there is strong evidence for efficacy of falls prevention interventions including at least two components, typically strengthening and balance training, delivered in groups or at home, with greater effects associated with higher training intensity.[32] Unfortunately, there is insufficient evidence concerning the efficacy of falls prevention strategies, including exercise, among stroke populations, and this is clearly an area where more research is needed.[31 33] Recent investigations are promising. For example, perturbation training may reduce post-stroke falls risk.[34] Some have recommended that evidence-based falls risk assessment should be undertaken, considering both individual and environmental factors.[31] While acknowledging the insufficiency of evidence with respect to falls prevention in this population, we concur with this recommendation, at least with respect to monitoring and safety considerations in PA and exercise interventions for stroke prevention.

Fatigue is frequent after stroke, with prevalence ranging from 23% to 75%.[35 36] However, the relationship of fatigue to exercise and PA is unclear. Authors of one systematic review found insufficient evidence to associate fatigue with either physical fitness or PA, although considered it very plausible that exercise could be an effective fatigue treatment.[37] Consistent with this, among 136 participants, lower daily step count and anxiety at 1 month poststroke each independently predicted greater fatigue at 6 and 12 months, suggesting PA could be a therapeutic target for poststroke fatigue.[38] Fatigue may adversely affect physical performance self-efficacy rather than performance per se.[35] Poststroke fatigue is multifactorial, having been associated with functional disability, prestroke fatigue, medical comorbidities, sleep disturbances, nutritional problems, depression, cognitive dysfunction and damage to particular brain areas.[36] From a systematic review and meta-analysis of psychological factors, poststroke fatigue was consistently associated with depressive symptoms, even in patients without clinical depression, or when using depression scales without fatigue items; the association with anxiety approached significance. From the same review, narrative evidence suggested that poststroke fatigue was associated with perceived loss of control, poor coping styles and other emotional and behavioural symptoms.[39] Interestingly, among 21 stroke survivors, exertional and chronic fatigue were predicted independently by peak O_2 uptake and depression, respectively.[40] In cerebrovascular populations, prevalence of obstructive sleep apnoea has been estimated at 61.9%,[41] pointing to the importance of assessment and treatment of this condition.[36 41] In an RCT, 73 patients at least 4 months poststroke were assigned to receive cognitive therapy with or without graded activity training. Both groups showed significantly improved fatigue scores compared with a 3-month baseline period, with the combined intervention superior to cognitive therapy alone.[42] However, a recent Cochrane review concluded that there was insufficient evidence on the efficacy of any intervention to treat or prevent poststroke fatigue.[43] We conclude that patients after stroke should be screened, assessed and treated as appropriate for obstructive sleep apnoea. With respect to PA and exercise, more research is needed to clarify their relationship to, and potential for, treatment of poststroke fatigue.

Poststroke chronic pain syndromes, including those with a musculoskeletal aetiology such as shoulder subluxation, are common, having been reported in up to 50% of stroke survivors.[44] Not surprisingly, patients with musculoskeletal pain syndromes are less likely to be functionally independent.[45] Prevention, prompt identification and treatment of musculoskeletal poststroke pain syndromes are important to maintain mobility.[46]

Depression affects about one-third of stroke survivors.[47] Outside of the stroke literature, in addition to its widely known deleterious effects on mood and interest or motivation,[48] depression is associated with reduced self-efficacy,[49] lower treatment adherence[50] and exercise capacity among patients with cardiovascular disease,[51] and worse response to exercise training in CR.[52] Poststroke depression has been associated with lower PA[28 29] and worse gait balance.[53] Emotional distress measured with an anxiety/depression/stress scale was associated with lower quadriceps strength, worse balance, reduced walking and stair-climbing speed among 45 community-dwelling chronic stroke survivors.[54] Evidence concerning effects of depression on exercise programming adherence and outcomes is sparse. Depression was associated with absence of gait improvements among 145 patients who had a stroke for

the first time and undergoing rehabilitation.[55] Depression symptoms were inversely associated with strength gains among subjects randomised to 12-week strength training or usual care at least 1 year after ischaemic stroke.[56] In other secondary RCT analyses however, strength, balance, endurance and upper extremity therapeutic exercise training improved impairments and functional limitations, independently of depressive symptoms.[57]

Depression is also associated with neuropsychological impairments, including slowing particularly of effortful or deliberate cognitive processes, attention, working memory and executive functions.[58] Depression and stroke-related executive dysfunction may interact unfavourably with respect to cognition and depression prognosis, and their co-occurrence has been associated with greater vascular and degenerative brain pathology compared with either one alone.[59] As chronic disease self-management approaches are grounded in behavioural self-regulation,[60] poststroke depression and executive dysfunction may interact to pose challenges to their effectiveness.

Recent recommendations of the American Heart Association/American Stroke Association (AHA/ASA, 2017)[47] include use of brief, accurate psychometric screening instruments to identify depressed stroke survivors, particularly in contexts of collaborative care enabling timely intervention, treatment and follow-up. Antidepressant pharmacotherapy and/or brief psychosocial interventions may be effective in managing poststroke depression. Meta-analytic evidence suggests that structured exercise programming, particularly at higher intensities, improves poststroke depressive symptoms in both subacute and chronic periods.[61] Meeting PA guidelines has been associated with lower risk of mild depressive symptoms.[62] Among patients post-TIA/mild non-disabling stroke in comprehensive CR, psychological service recipients, referred in part on the basis of psychometric results at intake, improved significantly more than non-recipients in mean depression score.[63]

Programmes seeking to promote exercise for secondary stroke prevention should use effective screening approaches to identify patients at risk of falling, fatigue related to obstructive sleep apnoea and other factors, pain and depression, which are prevalent after stroke. Table 1 summarises the factors associated with PA and exercise after stroke.

CHANGING PA AND EXERCISE BEHAVIOUR

Britain's National Institute for Health and Care Excellence (NICE) has published evidence-based public health

Table 1 Factors associated with physical activity or exercise after TIA or stroke

Factor	Probable positive association	Weak/ inconclusive association	Probable negative association	Modifiable?
Self-efficacy	X			Yes[64 70]
Self-determination, goal achievement	X			Yes[64 70]
Social support	X			Yes[64]
Supportive relationships with health professionals	X			Yes[28 77 78]
Infarct side		X		No
Neglect		X		More evidence needed[92]
Cognition		X		More evidence needed[93 94]
Logistic: weather, transportation, language, financial challenges			X	Partially[70]
Comorbidities, stroke-related deficits			X	Yes
Low cardiorespiratory fitness			X	Yes[72 73]
Negative PA/exercise beliefs			X	More evidence needed[69 84 95]
Fear of falling			X	More evidence needed[31–34]
Fatigue, low motivation			X	More evidence needed[36 41–43]
Pain/arthropathy			X	Partially[44]
Depression			X	Yes[47 61–63]

PA, physical activity; TIA, transient ischaemic attack.

guidelines for individual-level interventions to change behaviours associated with chronic disease, including increasing PA.[64] These recommend use of proven BCTs when designing interventions, including working with the client to develop goals and planning for behaviour and outcomes; feedback and monitoring, including self-monitoring of behaviour and outcomes; incorporating or developing social support; ensuring techniques match clients' needs; clearly defining a rationale for BCTs; ensuring that novel interventions are evaluated; considering use of remotely delivered interventions, for example, telephone, internet, text-messaging or 'apps', assuming evidence of effectiveness.

Various theoretical models[27 65 66] assume that *intention* causes health behavioural change.[67] This might seem obvious, yet we should recall the old adage observing that 'the road to hell is paved with good intentions'. Outside of stroke populations, meta-analysis has shown that interventions to increase intention had medium-to-large effects on intention (d_+=0.66; 95% CI 0.51 to 0.82) but only small-to-medium effects on subsequent behaviour change (d_+=0.36, 95% CI 0.22 to 0.50), across various health domains.[67] BCTs to increase PA produced a medium-sized effect on intention to engage in PA (d_+=0.57, 95% CI 0.46 to 0.68), but only a small effect on behaviour change (d_+=0.29, CI 0.15 to 0.42).[68] In a prospective cohort study of 100 patients post-TIA or minor stroke, self-efficacy with respect to behaviour change, belief in effectiveness of behaviour change to confer stroke risk reduction (response efficacy) and fear of another stroke, measured by standardised questionnaires at baseline, were each independently associated with the intention to increase PA, the first of these most strongly. Intention only tended non-significantly to predict actual behaviour change at 3 months as measured with a standardised self-report questionnaire.[69] Therefore, while PA intentions may contribute to behaviour change, they do not suffice to account for it. Clinically, these findings caution against restricting selection of patients for behavioural interventions to those deemed to be 'ready', for example, based on assignment to a particular stage of change,[65] as this ascertainment would essentially be intention-based. Indeed, the causal relationship between intention and behaviour may be bidirectional: intention may emerge from behavioural engagement.[67] Further research is needed to clarify whether or how PA and exercise interventions should explicitly target both intention and behaviour.

A self-efficacy/behaviour change discrepancy was seen in a 2014 systematic review involving non-clinical older adult samples. BCTs were effective for increasing self-efficacy (d=0.37, 95% CI 0.22 to 0.52; small-to-medium effect) and PA (d=0.14, 95% CI 0.09 to 0.20; small effect). Interestingly, BCTs based on self-regulation were associated with relatively *lower* gains in self-efficacy and PA, including providing normative information about others' behaviour, setting behavioural goals, behavioural self-monitoring, performance feedback, planning social support and relapse prevention. BCTs associated with relatively greater self-efficacy gains included setting graded tasks, self-monitoring of behavioural effects (for example, heart-rate response to PA), providing information on where and when to perform PA and motivational interviewing. Relatively greater PA gains were associated with barrier identification and problem solving, reward contingent on successful behaviour and modelling/demonstrating behaviour. These authors suggested that relatively weaker benefits with self-regulation techniques may have reflected reduced executive control or acceptability of the interventions, among older compared with middle-aged and younger adults.[70] While not derived from a stroke population, these findings highlight the importance of adjusting behavioural interventions to account for personal preferences, age and cognitive abilities, considerations which may assume even greater importance in stroke populations. BCTs associated with greater PA effects in older adults placed relatively less emphasis on intention to change; and greater weight on motivation, and on behaviour per se, including instructing or modelling, antecedents and consequences. These findings are consistent with results from a questionnaire study, in which compared with healthy matched controls, stroke survivors expressed greater preferences for exercise to be structured, group-based, at a facility such as a gym and for exercises to be demonstrated; a preference which might have reflected lowered self-confidence.[71]

The foregoing conclusions receive some support from two reviews of interventions on cerebrovascular samples. A systematic review and meta-analysis of comprehensive rehabilitation or secondary prevention interventions, including lifestyle or pharmacological components, initiated within 90 days of a TIA/minor stroke found evidence from individual studies of increased aerobic capacity. Noting limited evidence, frequently poor study quality and a need for further robust RCTs in this area, the authors concluded that the provision of individualised instruction about specific behaviours and behavioural goal-setting were key BCTs. Discussion of behavioural consequences might also be an important BCT associated with improved exercise capacity.[72] Authors of a 2014 systematic review of poststroke interventions to promote long-term participation in PA identified two types: tailored counselling with or without supervised exercise and tailored supervised exercise with advice, for example, to be active. Data-pooling was not possible due to heterogeneity in outcomes and timing of assessments. Counselling interventions included group or individual approaches, and one or more of mapping, setting or monitoring of PA goals, motivational interviewing or counselling, or follow-up visits or calls to enhance adherence. The authors concluded that tailored counselling with or without exercise was likely to be effective at promoting long-term PA participation after stroke, probably due to its capacity to address barriers to PA and to provide motivational support. While exercise with advice had the potential to increase fitness, it was unlikely to promote long-term PA participation.[73] This is

consistent with the results from a 2012 structured review of psychological and social factors which found that while supportive relationships with health professionals were positively associated with greater PA, simple advice and information were not.[28]

Very recent RCTs have tested self-management interventions after stroke. A targeted self-management approach based on social cognitive theory, delivered by trained peer dyads to African-American male stroke survivors after stroke or TIA in one individual and four group sessions over 3 months, versus usual care, produced favourable changes in blood pressure, glycosylated haemoglobin and HDL, but not in PA or other health behaviours.[74] Regular individualised PA coaching monthly for 18 months, with goal-setting based on exercise preferences, exercise scheduling, diary recording of PA and choice from a variety of exercise settings, versus standard care, did not improve motor functioning or 6 min walk test performance. While exercise and PA measured from diaries were treated as adherence measures rather than outcomes, compared with standard care, the intervention group recorded significantly more vigorous activity at 6, 12 and 18 months, and more moderate activity at 6 and 12 months.[75]

With respect to alternative interventions, recent systematic reviews and meta-synthesis of quantitative and qualitative data provided evidence that patients with cardiovascular disease including stroke survivors, who used mobile applications showed improved exercise capacity and PA, although overall study quality was considered low.[76] Nonetheless, these findings accord with NICE recommendations to consider remote interventions, if there is evidence to do so.[64]

Patient–health professional relational and communication factors are probably important in behavioural uptake and adherence. Among medical populations, meta-analytic evidence indicates that the quality of the patient–clinician relationship has a small but significant effect on healthcare outcomes such as blood pressure or validated pain scores.[77] Direct, verbal physician endorsement has increased adherence to CR programmes,[78] and may therefore also be important in stroke survivors' participation in exercise.

Overall, despite a number of systematic reviews and meta-analyses, uncertainty persists concerning optimal BCTs and integrated frameworks among stroke survivors. Several recent trial protocols,[79–81] to a significant degree grounded in social-cognitive theory and self-management, bode well for greater clarity. So too do recent efforts to establish reliable taxonomies of basic health BCTs which may be deployed within complex or comprehensive interventions across various populations. Such approaches are intended, for example, to enable meta-regression investigations to identify effective BCTs for modifying health behaviours, including those directed at PA.[82 83]

While self-regulation and self-management approaches have become key elements of behaviour change interventions, we caution that, compared with younger or non-stroke populations, older stroke survivors may require greater structure, guidance and professional support in achieving meaningful increases in PA and exercise. The 2014 AHA/ASA statement[84] on PA and exercise for stroke survivors include efforts to remove physical or environmental barriers such as cost or lack of transportation, access to exercise instructors with suitable knowledge and training, provision of social support such as through group interventions, professional advice to increase PA together with efforts to enhance self-efficacy and positive outcome expectations concerning PA and exercise, allowing patients to establish personal goals that may help with exercise and reassurance that exercise is likely to reduce rather than increase fatigue and risk of recurrent stroke.

In summary, this review has found that the following BCTs are probably effective in increasing exercise and PA. We have attempted to order these pragmatically, in terms of programming:

- Physician endorsement to patients of exercise programmes, their value and importance.
- Enhancement of the patient–professional relationship regarding communication and rapport.
- Explaining the rationale for PA and exercise.
- Motivational interviewing.
- Collaborative goal-setting with patients, considering their preferences.
- Identification of, and problem-solving with respect to, barriers such as logistical concerns.
- Development and incorporation of social support in programmes.
- Structured exercise programming.
- Individualised behavioural instruction, including demonstration or modelling of exercise.
- Behavioural diary recording.
- Review and discussion of behavioural consequences of exercise and PA efforts.
- Rewarding or reinforcing successful performance of exercise.
- Mobile applications ('apps').

While exercise programmes without counselling may be somewhat helpful in behaviour change, simple advice or information given alone is unlikely to be helpful, although study quality[73] or publication bias could potentially weaken such conclusions.

GUIDELINES AND RECOMMENDATIONS

Acknowledging that additional validation by RCTs are needed, the most recent AHA/ASA Stroke Prevention Guidelines included one revised and one new class IIa recommendation for participation in regular PA or exercise training programmes. The revised recommendation addressed frequency and intensity of exercise: 'At least 3 to 4 sessions per week of moderate- to vigorous-intensity aerobic physical exercise. Session duration should be approximately 40 min. Moderate-intensity exercise was defined as sufficient to break a sweat or noticeably raise heart rate (eg, walking briskly, using an exercise bicycle). Vigorous-intensity exercise includes activities

such as jogging.' The new recommendation addressed the key aspect of the aforementioned behaviour change as being a driver for successful transition from sedentary to active lifestyle by recommending 'referral to a comprehensive, behaviourally oriented program'.[85] In addition, the 2014 statement from the AHA/ASA recommends training heart rates should be set at least 10 bpm below the ischaemia ECG or angina threshold.

Early mobilisation and exercise recommendations for individuals postdisabling stroke is beyond the scope of this review and have been well described in a statement from the AHA. Also described in that statement are strategies to evaluate subjects' safety for exercise and general exercise prescription principles which are reiterated below.

Pre-exercise evaluation

In general, the following are considered absolute contraindications to exercise testing and training [86]:

- Acute myocardial infarction (within 2 days) or unstable angina.
- Uncontrolled cardiac arrhythmias causing symptoms or haemodynamic compromise.
- Symptomatic severe aortic stenosis.
- Uncontrolled symptomatic heart failure.
- Acute pulmonary embolus or pulmonary infarction.
- Acute myocarditis or pericarditis.
- Acute aortic dissection.

Although the likelihood of experiencing a fatal cardiac event during exercise training is extremely small,[87] it is usually associated with the presence of coronary artery disease. Because up to 75% of stroke victims have coexisting cardiac disease,[88] it is recommended that patients who have had a stroke undergo ECG-monitored graded exercise testing as part of a medical evaluation before beginning an exercise programme.[86] Generally, graded exercise testing in patients who have had a stroke should be conducted in accordance with contemporary guidelines as detailed elsewhere.[86] Briefly, the exercise test protocol for the stroke survivor should assess functional capacity and the cardiovascular response to exercise. The graded exercise test should include continuous 12-lead ECG monitoring and recordings at rest prior to exercise, every minute during exercise, peak exercise and at 1 min intervals for 6 min during recovery. Symptoms of chest pain, leg fatigue and dyspnoea should be quantified using an appropriate instrument such as the 6–20 Borg Scale.[86 89] The stress test should be terminated according to usual indications as follows: when the subject cannot continue due to symptoms (such as fatigue, dyspnoea or chest pain) or if it is deemed medically necessary due to any of the following clinical findings: >2 mm of horizontal or down-sloping ST segment depression,; persistent ≥10 mm Hg decline in systolic blood pressure, a hypertensive (systolic blood pressure >250 mm Hg, diastolic blood pressure >115 mm Hg) blood pressure response or the development of significant arrhythmias.[86]

Exercise testing is usually safe, even in very deconditioned patients, with less than one potentially life-threatening complication such as myocardial infarction or arrhythmia, occurring per every 1000–10 000 tests.[90] While studies have reported that patients can be screened for and have exercise training implemented safely within 24 and 48 hours following acute stroke,[91] it has been recommended that usual cardiac stress testing strategies can be deployed as soon as 3 weeks after the stroke or TIA, or if an exercise ECG is not performed, lighter-intensity exercise should be prescribed and the reduced exercise intensity may be compensated for by increasing the training frequency, duration or both.

CONCLUSIONS

Definitive RCT evidence is lacking concerning the mortality and morbidity (including stroke) benefits of increased PA and exercise. Nonetheless, a substantial body of evidence shows that exercise improves risk factors associated with initial or recurrent stroke. Mean levels of PA and cardiorespiratory fitness are low in relation to age-normative or age-matched values. Therefore, increasing PA and exercise is important for secondary prevention after stroke. Certain precautions and contraindications notwithstanding, exercise evaluation and programmes are usually safe after TIA or stroke. Effective behaviour change strategies are crucial to successfully increasing and maintaining PA and exercise. Such strategies will address key logistic barriers, stroke-related deficits, fatigue, safety issues and fear related to falls risk, pain and crucially, individual psychological differences such as self-efficacy, exercise-related beliefs, availability of social support and depression. While information, advice-giving and exercise programmes may contribute to enhancement of PA and exercise behaviour, they are not sufficient. Programme delivery and design should consider patients' priorities and goals, as well as their cognitive functioning. While teaching self-regulation knowledge and skills is important, older patients, or those with worse executive impairments, may benefit from a programmatic shift of emphasis towards PA and exercise behaviour per se, for example, through modelling, structure and reinforcement. Structured exercise programmes should be integrated with evidence-based BCTs and frameworks.

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