REVIEW / SYNTHÈSE

Evidence-based risk assessment and recommendations for physical activity clearance: stroke and spinal cord injury[1]

E. Paul Zehr

Abstract: Physical activity (PA) has potential benefits after stroke or spinal cord injury (SCI), especially in improving efficiency and functional capacity in activities of daily living. Currently, many who could benefit from PA may be routinely excluded from participation because of myths related to functional capacity and the concern for harm. The purpose of this review was to evaluate the literature for reports of adverse events during exercise after stroke or SCI, and to provide recommendations regarding exercise participation in supervised and unsupervised environments. Studies were evaluated for quality, and the summary level and quality of evidence were evaluated using the AGREE rubric, modified to address the main outcome measure of adverse events. Levels of exercise stress were evaluated for aerobic activities, using an established rubric. Included in the current analysis were 32 studies for stroke and 4 for SCI. In aggregate, this yielded a total of 730 experimental participants with stroke and 143 with SCI. It should be noted that almost all studies were not designed to examine naturally occurring adverse events from PA. Significant contraindications to unsupervised exercise include manifestation of autonomic dysreflexia in SCI and cardiovascular comorbidity after stroke. There are clear benefits of exercise training on physiological outcomes in stroke and SCI, but the relation between outcomes and safety remains unclear. However, taken on balance, the risk-to-benefit ratio favors the recommendation of exercise. This recommendation is based on studies in which participants were almost universally screened for participation in supervised environments. Thus, the grading of evidence for finding adverse events to support this conclusion is inadequate.

Key words: rehabilitation, physical therapy, neurotrauma, functional capacity, safety.

Résumé : À la suite d'un accident vasculaire cérébral ou d'une lésion médullaire (SCI), l'activité physique (PA) peut procurer des bienfaits particulièrement en ce qui concerne l'amélioration de l'efficacité et de la capacité fonctionnelle pour les activités de la vie courante. À l'heure actuelle, plusieurs personnes qui pourraient tirer profit de la pratique de PA sont exclues d'office de la pratique, et ce, à cause de mythes concernant leur capacité fonctionnelle et le souci de les protéger. Cette étude se propose d'une part de fouiller dans la documentation existante des mentions d'événements indésirables associés à l'exercice physique à la suite d'un accident vasculaire cérébral et d'une lésion médullaire et d'autre part, de formuler des recommandations au sujet de la pratique de l'activité physique dans des milieux supervisés ou non. On évalue la qualité des études, puis on évalue aussi le niveau et la qualité des données probantes au moyen de la rubrique AGREE modifiée afin de cibler particulièrement la mention des événements indésirables. On évalue le stress d'effort des activités aérobies au moyen de la rubrique d'évaluation admise. Pour l'analyse, on retient 32 études portant sur l'accident vasculaire cérébral et 4 études sur la SCI. Au total, ces études sont réalisées auprès de 730 sujets expérimentaux ayant subi un accident vasculaire cérébral et 143 sujets ayant été victimes d'une SCI. Il faut mentionner que presque toutes les études n'ont pas été conçues pour analyser les événements indésirables associés à la pratique de PA dans le quotidien. Parmi les contre-indications d'importance à la pratique de PA sans supervision, on compte la manifestation de dysréflexie autonome chez les lésés de la moelle épinière et la comorbidité consécutive à l'accident vasculaire cérébral. L'entraînement physique procure des bienfaits évidents sur le plan physiologique à la suite d'un accident vasculaire cérébral et d'une lésion médullaire, mais il reste à clarifier l'association entre ces observations et la sécurité du patient. Néanmoins, à peser le pour et le contre, le ratio risque / bénéfice favorise la recommandation en faveur de la pratique de l'activité physique. Cette recommandation découle des ob-

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E.P. Zehr. Rehabilitation Neuroscience Laboratory, University of Victoria, PO Box 3015 STN CSC, University of Victoria, Victoria, BC V8W 3P1, Canada; International Collaboration on Repair Discoveries (ICORD), Vancouver, BC, Canada; Centre for Biomedical Research, University of Victoria, Victoria, BC, Canada; Division of Medical Sciences, University of Victoria, Victoria, BC, Canada; School of Exercise Science, Physical and Health Education, University of Victoria, Victoria, BC, Canada.

E-mail for correspondence: pzehr@uvic.ca

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servations faites dans des études où on avait autorisé presque tous les participants à la pratique de l'activité physique dans un milieu sous supervision. En conséquence, le niveau d'évidence concernant l'observation d'événements indésirables pour appuyer cette conclusion est insuffisant.

[Traduit par la Rédaction]

Lay synopsis

Exercise after stroke or spinal cord injury (SCI) could be good for many people. Exercise could help improve ability to perform daily activities. However, exercise can also be harmful, and it is important to know whether it is safe for people after injury. The purpose of this review was to look through and evaluate scientific papers of exercise after stroke or SCI. The papers were examined for any possible harmful events that occurred. This could then be used to make recommendations about the safety of exercise for participants. This analysis showed that there are some serious considerations related to exercise after stroke or SCI. For example, changes in the way the nervous system controls heart rate and blood pressure after serious SCIs are important to be aware of. After stroke, decreased activity levels and worsening of cardiovascular disease are the main concerns. However, there are clear benefits of exercise in improving function after stroke or SCI. This, combined with the low incidence of subsequent harmful events, suggests that, in general, exercise is recommended. However, in many cases of stroke or SCI, exercise participants should be medically evaluated and perform exercise under qualified supervision.

Introduction

Exercise after stroke or spinal cord injury (SCI) can be quite beneficial, particularly as the adaptations may help improve efficiency and functional capacity associated with activities of daily living. Despite that, many individuals with neurological dysfunction who could benefit from physical activity may be routinely prevented from participation because of incorrect beliefs related to functional capacity and the concern for harm. The focus of this review is on the risk of adverse events for physical activity participation in the neurological pathologies of cerebrovascular accident (stroke) and SCI. For the purposes of this review, neurological dysfunction after stroke or SCI will be broken into 2 broad classes of neurological impairment, based on time after injury: acute (less than 6 months postinjury), and chronic (>6 months postinjury).

The single most common pathological outcome of stroke or SCI is a reduced ability to activate muscle and produce movement. This progressively leads to a corresponding reduced overall level of physical activity and concomitant deconditioning. A common feature is a global decrease in strength, which can be manifested differentially. For example, weakness in many muscles after incomplete SCI can coexist with maintained or even enhanced activity in spastic muscles. As such, a key concern during activity is the risk of falling or cardiovascular events. Thus, overall functional capacity is decreased and, because of low levels of physical activity, those with neurological dysfunction typically have low oxidative capacities and increased fatigue. Reduced activity in people with neurological dysfunction has additional secondary effects, such as osteoporosis, cardiovascular comorbidities, and overall decreased functional capacity. An important consideration for physical activity after neurological dysfunction is that the physical activity prescription should consider elements of functional relearning (e.g., rehabilitative motor practice for walking recovery), in combination with physiological health benefits. As such, there is considerable potential benefit for participation in physical activity, which includes, for example, neurological rehabilitation and physiological conditioning, but there are also secondary concerns that must be addressed.

Purpose

The purpose of this paper was to review the evidence for the occurrence of adverse events related to and contraindications for exercise participation, and to provide recommendations for exercise clearance for those with stroke or SCI. The recommendations are intended to cover both independent activities and those under the supervision of allied healthcare practitioners, including certified exercise professionals. It was hypothesized that, in the absence of comorbidities, neither stroke nor SCI would be substantiated as significant contraindications to physical activity participation. This paper is part of a larger project designed to assess exercise adverse events and clearance; it consists of multiple systematic reviews of numerous clinical conditions, and is aimed at an evidence-based revision of the Physical Activity Readiness Questionnaire (PAR-Q) and the Physical Activity Readiness Medical Evaluation (PARmed-X) screening tools.

The following section was written by the consensus panel that guided the overall revision of the physical activity clearance process. This information is reprinted in each of the systematic review papers, so that these reviews can stand alone from the paper describing the overall consensus process (Jamnik et al. 2011).

Physical activity participation is recommended and beneficial for all asymptomatic people and for people with chronic disease (Warburton et al. 2006*a*, 2007). However, physical activity participation of people with certain chronic disease conditions or constraints may need to be restricted. The PAR-Q is a screening tool completed by people who plan to undergo a fitness assessment or to become much more physically active; for example, when initiating physical activity participation that is beyond a person's habitual daily activity level or when beginning a structured physical activity or exercise program. Screening is also recommended when a person is joining a health club, commencing a training program with a fitness professional, or joining a sports team. If a person provides a positive response to any question on the PAR-Q, he or she is directed to consult with his or her physician for clearance to engage in either unrestricted or restricted physical activity.

The PARmed-X is a screening tool developed for use by

physicians to assist them in addressing medical concerns regarding physical activity participation that were identified by the PAR-Q. Recent feedback from physical activity participants, fitness professionals, and physicians has brought to light substantial limitations to the utility and effectiveness of physical activity participation screening by the PAR-Q and PARmed-X. In short, the exercise clearance process is not working as intended and at times is a barrier to physical activity participation for people who may be most in need of increased physical activity. The aim of this project was for experts in each chronic disease, together with an expert panel, to revise and increase the effectiveness of the PAR-Q and PARmed-X screening process, using an evidence-based consensus approach that adheres to the established Appraisal of Guidelines for Research and Evaluation (AGREE).

An important objective of this project was to provide evidence-based support for the direct role of university-educated and qualified exercise professionals in the exercise clearance process. An example of a qualified exercise professional is the Canadian Society for Exercise Physiology Certified Exercise Physiologist, which is the highest nationally recognized certification in the health and fitness industry. It recognizes the qualifications of people who possess advanced formal academic preparation and practical experience in health-related and performance-related physical activity and exercise science fitness applications for both nonclinical and clinical populations.

The AGREE instrument was developed by a group of researchers from 13 countries to provide a systematic framework for assessing the quality and impact on medical care of clinical practice guidelines (AGREE Collaboration 2001, 2003). The AGREE collaboration published the rigorous development process and associated reliability and validity data of the AGREE instrument, based on a large-scale study focused primarily on clinical practice guidelines (AGREE Collaboration 2001, 2003). The AGREE instrument is now a commonly used tool for assessing clinical practice guidelines and other health management guidelines (Lau 2007). The AGREE guidelines were applied in this project to assess the formulation of risk stratification and physical activity participation clearance recommendations for each of the critical chronic diseases. One of the authors of this project (J.M.) is an AGREE instrument expert, and she was responsible for evaluating the compliance of the overall process to the AGREE guidelines.

In addition to adhering to the AGREE process, the Level of Evidence (1 = randomized control trials (RCTs); 2 = RCTs with limitations or observational trials with overwhelming evidence; 3 = observational studies; 4 = anecdotal evidence) supporting each physical activity participation clearance recommendation and the grade (A = strong; B = intermediate; C = weak) of the physical activity participation clearance recommendation were assigned by applying the standardized Level and Grade of Evidence detailed in the consensus document (Warburton et al. 2011*b*).

In this series of articles, each chronic disease condition was considered in reference to a continuum of risk, from lower-risk to intermediate- (moderate) and higher-risk categories (Warburton et al. 2011*a*, 2011*b*). Particular attention was paid to the short-term (acute) risks of physical activity and exercise, compared with the long-term (chronic) benefits on the chronic disease. Physical activity participation may transiently increase the risk acutely, while leading to physiological and psychological adaptations that markedly reduce the long-term risk. Adverse events were considered to be any adverse change in health status or a side effect related to to physical activity or exercise participation.

Cerebrovascular accident (stroke)

Cerebrovascular accident, or stroke, is the term used to describe damage to the brain that results from ischemia arising secondarily from a blockage of blood flow to the brain. Strokes can be classified into 2 broad categories — ischemic and hemorrhagic — which account for 70%–80% and 20%–30% of incidents, respectively. Each of these categories can be further subdivided. Ischemic stroke consists of both thrombotic (arising after arterial narrowing in atherosclerosis) and embolic (arising from a blockage due to a blood clot formed elsewhere in the body). Hemorrhagic strokes are classified as either cerebral (blood vessel rupture in the brain, often secondary to high blood pressure) or subarachnoid (ruptured vessel on the brain surface, often secondary to head trauma).

After ischemic stroke, an important concern regarding ongoing health status and risk is derived from the likely subtype. The TOAST criteria are commonly used to classify ischemic stroke into 5 groups (Adams et al. 1993, 2003): large artery atherosclerosis (in which a major cerebral artery has narrowing of >50%, and which are often preceded by transient ischemic attack); cardioembolism (in which the cerebral artery occlusion arises form embolus in the heart or aorta); small-artery or lacunar stroke (where symptoms are consistent with smaller, discrete lesions, producing pure motor hemiparesis), which are associated typically with diabetes or hypertension; uncommon cause (e.g., occurring in hemotalogical disorders or with hypercoagulation); and undetermined cause.

Stroke ranks as the third leading cause of death in North America, and ranks first as cause of disability (Kelly et al. 2007). There are approximately 55 000 cerebrovascular accidents each year, and stroke accounts for approximately 15 000 deaths per year in Canada (Heart and Stroke Foundation 2010). In the United States, stroke is the third leading cause of death (accounting for 1 of every 16 deaths) and the leading cause of long-term disability, with more than a million people reporting impaired ability to conduct activities of daily living (Rosamond et al. 2008). Worldwide, the World Health Organization reports that ~15 million people succumb to stroke each year, with roughly one third of the incidents being fatal and one third leading to permanent disability (Kelly et al. 2007).

Spasticity after stroke can have a major impact on functional capacity for physical activity, including activities of daily living. The hallmark of spasticity is exaggerated stretch reflex responses, arising because of increased velocity sensitivity of the monosynaptic stretch reflex arc. This gives rise to inappropriate expression of these reflexes, which can produce reciprocating excitation, known as clonus, around a joint and within antagonistic muscles. Both of these severely impair movement and coordination, and reduce efficiency during exercise. The notion that exercise, and in particular resistance exercise, might exacerbate spasticity has now been

clearly refuted (for review, see Morris et al. 2004; Patten et al. 2004).

Aerobic capacity and overall strength are reduced, while effort associated with activity is increased after stroke (Ramas et al. 2007). During functional tasks like walking, energy expenditure related to hemiparesis after stroke can be almost double that found in uninjured participants (Corcoran et al. 1970; Gersten and Orr 1971; Ivey et al. 2006). Peak aerobic capacity in stroke, obtained during leg cycling or treadmill protocols, ranges from ~12 to 18 $mL \cdot kg^{-1} \cdot min^{-1}$. This can be contrasted with age-matched (e.g., ~60–70 years) inactive healthy participants, whose peak aerobic capacity can range between 25 and 30 $mL \cdot kg^{-1} \cdot min^{-1}$ (Ivey et al. 2006).

Because of the typical unilateral locus of the supraspinal lesion, stroke typically presents with asymmetrical effects. This poststroke overall weakening applies to both sides, but is more prevalent on one side, and hemiparesis is commonly observed (Patten et al. 2004). The distribution of muscle weakness after stroke is widespread, can range between ~20% and 90% of the less affected side, and may be more evident in distal muscles than in proximal ones (Patten et al. 2004). For example, this is clearly evident at the ankle. In a recent study of 16 stroke survivors, on the more affected side, maximal torque for plantarflexors and dorsiflexors was 55% and 38%, respectively, of the less affected side (Barzi and Zehr 2008). This overall weakness negatively affects activities of daily living, including such things as rising from a chair and walking (Morris et al. 2004; Zehr and Loadman, in press).

Perceived risk of exercise after stroke

A main concern about exercise after stroke is the risk of events arising from cardiovascular comorbidities (Adams et al. 2003; Ivey et al. 2008). The leading causes of mortality in stroke survivors have been identified as recurrent stroke and cardiovascular disease (Gordon et al. 2004). So-called asymptomatic, or silent, cardiovascular disease is a significant concern in stroke (Potempa et al. 1996). When coronary angiography was performed on 200 patients with transient ischemic attack or stroke but no clinical diagnosis of coronary artery disease, 40% were identified as having advanced or severe coronary artery disease (Hertzer et al. 1985). Indeed, Roth (1993) performed an exhaustive review of the literature, and found that there was evidence of cardiovascular disease in almost 75% of stroke patients. It has been suggested that the long-term risk of coronary artery disease in stroke is approximately twice that of neurologically intact and age-matched participants (Adams et al. 2003).

Accordingly, there is incomplete evidence related to exercise testing and training in the stroke population. The data available on exercise testing in chronic stroke participants suggests that it can be safe, valid, and reliable (Dobrovolny et al. 2003; Ivey et al. 2005). Ivey et al. (2005) report that in 95% of cases, in their experience, stroke participants can tolerate and achieve ~85% of the age-predicted maximal heart rate. However, these data were obtained under medically supervised conditions, and it must be noted that in ~29% of participants without diagnosed coronary artery disease, asymptomatic myocardial ischemia was detected during exercise testing (Macko et al. 1997).

Spinal cord injury

Damage to the spinal cord can occur as a direct result of physical trauma, such as a motor vehicle accident, fall, or sports injury. Additionally, diseases that create spinal tumours or cysts can lead to spinal cord damage. In any event, spinal cord damage leads to an interruption of communication between the supraspinal regions and the spinal segments distal to the lesion. There are more than 85 000 Canadians with SCI, and this number is projected to increase at a rate of ~3000 per year (Farry and Baxter 2010). There is a similar incidence per capita in the United States, where between 250 000 and 400 000 people have SCI, with ~13 000 new injuries occurring each year. The overall average annual estimate for the incidence of SCI in the developed world ranges from 15 to 40 per 1 000 000 people (Mehrholz et al. 2008). Half of those with SCI will be left with incomplete motor impairment (Mehrholz et al. 2008).

An SCI results from a lesion that may give rise to partial or total interruption of the integrity of the sensory or motor axons in the spinal tracts. As a result, there are effects on the somatosensory system, as well as the autonomic nervous system. There are considerable ranges of impairment, depending on where the injury in the spinal cord is localized. Over the years, a variety of classifications for SCI have been used. Currently, the most commonly used classification is that of the Neurological Standards Committee of the American Spinal Injuries Association (2002). This so-called ASIA impairment scale has 5 classifications, from A to E. An injury that is sensory and motor complete as low as sacral segments S_4–S_5 is categorized as ASIA A. ASIA B consists of an incomplete injury with no motor function, but in which some sensory function is maintained below the level of the lesion, and affects the S_4–S_5 segments. In the initial recovery period after an injury, many individuals are categorized as ASIA B before some neurological recovery occurs and they transition to motor incomplete status (ASIA C or D). ASIA C is an incomplete injury with motor function below the level of the neurological lesion, and with more than half the key muscles below the lesion unable to produce active contraction against gravity (muscle grade of less than 3). An ASIA D injury is one in which motor function is present below the lesion level, and the ability to produce active contraction against gravity (have a muscle grade of 3 or greater) in at least half the key muscles below the lesion is preserved.

In addition to the ASIA system of categorization, a useful but gross classification of physical impairment after SCI are the terms paraplegia and quadriplegia. When injury occurs in the cervical segments or the most rostral thoracic segment (T_1), the result is quadriplegia, with possible impairment of the arms, trunk, legs, bladder, bowel, and sexual function (Figoni et al. 2002). Paraplegia describes the constellation of outcomes arising from injuries at thoracic levels T_2–T_{12}, with impairment of the trunk, legs, and organs of the pelvis. Damage to lumbar or sacral segments L_1–S_4 results in paraplegia affecting the legs and pelvic organs. Residual physical capacity is a product of the degree of impairment arising from the level and completeness of the injury. As such, it is possible for someone to have, for example, an incomplete high cervical injury with good physical capacity or a complete low lumbar injury with poor physical capacity. However, the anticipated level of physical functional capacity for someone

with quadriplegia and someone with paraplegia is typically markedly different.

As a result of all this, extreme physical deconditioning is a serious concern after SCI. It was recently reported that cardiovascular disease has surpassed renal and respiratory disease as the leading cause of mortality in people with chronic SCI (Myers et al. 2007). There is a higher prevalence and earlier onset of coronary artery disease in the population with SCI than in the neurologically intact population, which is significantly affected by the hyperlipidemia, obesity, and diabetes that occur in higher rates in this population (Myers et al. 2007). Based on a long-term survey of the sociological and physical well-being of people with SCI in Japan, Nakajima and Honda (1988) found that there was 2.8, 5.5, and 3.7 times the average incidence of hypertension, diabetes mellitus, and heart disease, respectively, compared with the incidence found in the neurologically intact population. Physical activity, a main modifiable risk factor for cardiovascular disease, is obviously directly and negatively affected by the severity of the SCI.

Perceived risk of exercise in SCI

In addition to overt somatosensory and motor impairment tied to any spinal injury, in quadriplegia a major concern is interruption of autonomic function. In particular, and more so with higher injuries, cardiac and adrenal sympathetic innervation is affected, leading to vasomotor paralysis and corresponding hypotension and autonomic dysreflexia (Jacobs and Nash 2004; Krassioukov et al. 2009). Thus, in addition to the more obvious reduced ability to voluntarily activate large muscle groups to perform exercise after SCI, a serious secondary consequence is the inability to drive the autonomic and cardiovascular systems to perform aerobic activity (Figoni et al. 2002). For example, reduced catecholamine production from the adrenal medulla, combined with inadequate activation of the skeletal muscle pump, impairs cardiac output (Figoni et al. 2002), resulting in circulatory hypokinesis (Jacobs and Nash 2004). A possibly dangerous outcome — more prominent in higher thoracic (above T_6) and cervical injuries — is autonomic dysreflexia. Autonomic dysreflexia is a syndrome arising from impairment of autonomic reflexes, which can give rise to possibly harmful hypertension (arterial peak of ~200–300 mm Hg), headache, unusual sweating, bradycardia, and shivering (Figoni et al. 2002). For exercise response, a key outcome of this syndrome is a reduced heart rate response; peak heart rates in SCI with quadriplegia may be ~120 beats·min^{-1} (Jacobs and Nash 2004).

Methodology

The search terms and outcomes used to obtain the studies are found in Table 1. Each study was evaluated for quality, using a tool modified from that developed by Downs and Black (1998). For this evaluation, 5 questions were asked for each study: Was the dose and (or) type of physical activity similar to the aims of the PAR-Q?; Did the study participants represent the population from which they were recruited (that is, not excessively screened)?; Was the training environment (supervised or unsupervised) similar to the environment that this population would access under normal PAR-Q conditions?; Was any sample attrition of any consequence to the results obtained?; and Was the sample large enough for generalizations about adverse events? Studies were scored with 1 point for each "yes" and 0 points for each "no". Quality assessment scores were determined to be high for a score of 5, medium for a score of 3 or 4, and low for a score of 1 or 2.

The summary level and quality of evidence were evaluated using the AGREE rubric, modified (Table 2) to address the main outcome measure of interest — adverse events.

Levels of exercise stress were evaluated for aerobic activities, using the rubric established by Warburton et al. 2006*b* (see Table 3) and endorsed by the Canadian Society for Exercise Physiology and Health Canada (see Table 4). For descriptors associated with resistance activities, see Table 5.

Results

As shown in Table 1, a total of 576 potential citations were identified. On secondary screening, the number meeting inclusion criteria fell to 439. It should be noted that almost all studies were not designed to examine naturally occurring adverse events from physical activity.

A total of 32 studies related to outcomes after stroke were evaluated, as were 3 trials related to outcomes after SCI. In aggregate, this yielded a total of 730 experimental participants with stroke and 108 with SCI. All studies that were included involved exercise in a supervised environment. It should be noted that no studies were rated 4 or 5 for quality assessment; typically, they were rated between 2 and 3 (actual range, 1–3).

Physical activity after stroke

The data are described with reference to 3 categories of physical activity after stroke: treadmill training for locomotor rehabilitation; resistance training for strength improvement; and mixed fitness training. Several studies used combined approaches (that is, strength and cardiovascular training); these are categorized as mixed fitness training. However, the focus was to partition out studies that purely evaluated gait training or strength training in the other sections.

Treadmill training for locomotor rehabilitation after stroke

A growing number of studies have investigated treadmill training as a vehicle for improving overground walking capacity after stroke (Moseley et al. 2005). This systematic review of the literature supports the approach taken in a recent Cochrane review on this topic (Moseley et al. 2005), in which 15 studies were included, encompassing 622 participants. The summary of the participant characteristics, physical activity dose exposure, and reported adverse events are shown in Table 6. It is important to note that the key concern is the cardiovascular events. As shown in Table 6, there were adverse cardiovascular events in only 0.03% (2 of 622) of all possible conditions (found in only 1 trial) (Kosak and Reding 2000). Of these, 1 was exacerbation of the stroke pathophysiology in the control condition, and 1 was an acute myocardial infarction that occurred 2 days after the trial had finished.

It is unclear whether treadmill training should be categorized purely as a so-called "task-specific" practice, or if it can concomitantly be considered "exercise". Difficulty arises

Table 1. Search strategy in MEDLINE 1950–2008.

Search no.	Search terms	No. hits
Search for neurological dysfunction		
1	(Cerebrovascular accident) OR (stroke) OR CVA (text words)	115 655
2	(Spinal cord injury) OR (SCI) OR (neurotrauma) (text words)	29 172
3	Stroke (medical subject heading, all subheadings and categories included)	29 138
4	Spinal cord injuries, trauma, nervous system (medical subject heading, all subheadings and categories included)	85 559
5	Autonomic dysreflexia (medical subject heading, all subheadings and categories included)	210
6	OR/1–5	218 416
Search for physical activity		
7	Exercis* OR (physic* activ*) OR exert* OR (physic* fit*) OR sports (text words)	363 820
8	Walk* or jog* or swim* locomotion* gait (text words)	49 797
9	(Weight lift*) OR (strength train*) OR (resistance train*) OR (circuit weight train*) OR (aerob* train*) (text words)	6 698
10	Exercise (medical subject heading, all subheadings and categories included)	45 038
11	Exertion (medical subject heading, all subheadings and categories included)	0
12	Physical education (medical subject heading, all subheadings and categories included)	10 143
13	Training (medical subject heading, all subheadings and categories included)	0
14	Physical fitness (medical subject heading, all subheadings and categories included)	16 056
15	Sports (medical subject heading, all subheadings and categories included)	18 162
16	OR/7–15	405 606
Filter for RCTs and CCTs		
17	RANDOMIZED-CONTROLLED-TRIAL in PT	0
18	RANDOMIZED-CONTROLLED-TRIALS	63 797
19	RANDOM-ALLOCATION	63 529
20	DOUBLE-BLIND-METHOD	98 973
21	SINGLE-BLIND-METHOD	12 521
22	17 or 18 or 19 or 20 or 21	222 827
23	TG-ANIMAL not (TG = HUMAN and TG = ANIMAL	9
24	22 not 23	222 827
25	CLINICAL-TRIAL in PT	0
26	Explode CLINICAL-TRIALS	557 549
27	(Clin*near trial*) in TI	0
28	Clin* trial*	619 012
29	(Singl* or doubl* or trebl* or tripl*) near (blind* or mask*)	1 114 813
30	(29 in TI) or (29 in AB)	0
31	PLACEBOS	28 297
32	Placebo* in TI	0
33	Placebo*	5
34	Random*	585 985
35	Random* in AB	0
36	RESEARCH-DESIGN	65 656
37	25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36	1 939 539
38	TG = ANIMAL not (TG = HUMAN and TG = ANIMAL)	0
39	37 not 38	1 939 539
40	39 not 24	1 716 712
41	COMPARATIVE-STUDY	1 414 101
42	Explode EVALUATION-STUDIES	113 306
43	FOLLOW-UP-STUDIES	377 761
44	PROSPECTIVE-STUDIES	260 630
45	Control* or prospectiv* or volunteer*	2 460 728
46	(45 in TI) or (45 in AB)	0
47	41 or 42 or 43 or 44 or 45	3 786 737
48	TG = ANIMAL not (TG = HUMAN and TG = ANIMAL	0
49	47 not 48	3 786 737
50	49 not (24 or 39)	2 855 833
51	48 or 24 or 39	4 795 372
52	6 and 16 and 22 and 37 and 47	638
53	Limit 52 to English language and humans	576

Note: CCT, controlled clinical trial; CVA, cerebrovascular accident; RCT, randomized controlled trial; SCI, spinal cord injury; TG, triglyceride.

Table 2. Grading of Evidence (revised Appraisal of Guidelines for Research and Evaluation (AGREE) rubric).

Level and Grade of Evidence	Criteria
1A	RCT; no screening; excellent reporting and documentation, including adverse events and drop outs, strong external validity, no adverse events (overwhelming evidence that benefits outweigh the risks)
1B	RCT; no screening; excellent reporting and documentation, including adverse events and drop outs, strong external validity, possible adverse events (unclear if benefits outweigh the risks)
2A	RCT or clinical trials (quasi experimental); documentation, including adverse events and drop outs with other limits (weak external validity), no adverse events (benefits outweigh the risks)
2B	RCT or clinical trials (quasi experimental); documentation, including adverse events and drop outs with other limits (weak external validity), possible adverse events (unclear if benefits outweigh the risks)
3A	Observational studies (passive prospective, cross-sectional); documentation of adverse events, no adverse events reported (benefits outweigh the risks)
3B	Observational studies (passive prospective, cross-sectional); documentation of adverse events, possible adverse events (unclear benefits outweigh the risks)
3C	Observational studies (passive prospective, cross-sectional); documentation of adverse events, possible adverse events (risks may outweigh benefits)
4C	Inadequate or no data (adverse events not documented)

Table 3. Harmonization of different expressions of relative intensities for aerobic exercise prescription for activities lasting 30 to 60 min.

Intensity	%HRR	%HR_{max}	RPE	METs	METs min·wk^{-1}	Breathing rate	Body temperature	Example activity
Sedentary	<20	<50	<10	<3	<450*	Normal	Normal	Sitting watching TV, working on the computer
Light effort	20–39	50–63	10–11	3–4	450–600	Slight increase	Starting to feel warm	Dusting, light gardening
Moderate effort	40–59	64–76	12–13	5–6	750–900†	Greater increase	Warmer	Brisk walking
Vigorous effort	60–84	77–93	14–16	7–10	1050–1500‡	More out of breath	Quite warm	Jogging
Very hard effort	>84	>93	17–19	>10	>1500	Greater increase	Hot	Running fast
Maximal effort	100	100	20	>10	>1500	Completely out of breath	Very hot, perspiring heavily	Sprinting all-out

Note: The shaded area identifies intensity levels that are required for health. Adapted from (Warburton et al. 2006*b*). %HHR, % heart rate reserve; %HR_{max}, % maximum heart rate; MET, metabolic unit relative to resting metabolism; RPE, rating of perceived exertion.

*3 METs × 30 min × 5 days = 450 MET min·week^{-1}.

†6 METs × 30 min × 5 days = 900 MET min·week^{-1}.

‡10 METs × 30 min × 5 days = 1500 MET min·week^{-1}.

Table 4. Rubric used for quantification of exercise intensity levels for aerobic activities (from the Canadian Society for Exercise Physiology and Health Canada's recommendations for aerobic activities).

Very light effort	Light effort (60 min)	Moderate effort (30–60 min)	Vigorous effort (20–30 min)	Maximum effort
Strolling	Light walking	Brisk walking	Aerobics	Sprinting
Dusting	Volleyball	Biking	Jogging	Racing
	Easy gardening	Raking leaves	Hockey	
	Stretching	Swimming	Basketball	
		Dancing	Fast swimming	
		Water aerobics	Fast dancing	
How does it feel? How warm am I? What is my breathing like?				
No change from resting state	Starting to feel warm	Warmer	Quite warm	Very hot–perspiring heavily
Normal breathing	Slight increase in breathing rate	Greater increase in breathing rate	More out of breath	Completely out of breath
Range needed to stay healthy				
Less than is required	Where you want to be	Where you want to be	Where you want to be	More than is required

Note: Source: Handbook for Canada's Physical Activity Guide to Healthy Active Living. Health Canada. Available from www.paguide.com.

when categorizing the relative exercise stress involved in the course of treadmill training, since few studies report specific details about estimated work performed or the physiological cost of the treadmill training. In only 1 study were the physiological measures of exercise strain reported (Macko et al. 2008). In Macko et al. (2008), the treadmill slope across the 6-month exercise treatment phase was increased (from 0.% to ~2.2% grade), so that a threshold of 60%–70% of heart rate reserve was obtained during training. No adverse events were reported using this protocol. Moseley et al. (2005) calculated an overall risk difference from control using a 95% confidence interval; the risk for treadmill training was not different from 0.

Table 5. General intensity classification for resistance activities and example activities of daily living for older adults.

Intensity classification	Resistance activities (%1RM)	Example activities
Very light effort	<30	Watering the lawn or garden
Light effort	30–49	General housecleaning, ironing
Moderate effort	50–69	Raking leaves, vacuuming
Hard effort	70–84	Wood splitting, shovelling snow
Very hard effort	>84	Carrying groceries upstairs
Maximal effort	100	Lifting a heavy load that you can only lift once

Note: Data in adapted from information in Warburton et al. (2006*a*) and American College of Sports Medicine (1993). %1RM, percentage of 1-repetition maximum.

Resistance training for strength improvement after stroke

The actual volume of literature specifically addressing resistance or strength training after stroke has been small until recently (Morris et al. 2004). This may be largely because of the erroneous assumption that resistance training can exacerbate spasticity (see comments in Patten et al. 2004). In the studies shown in Table 7, very few details on either adverse events or adherence are reported. Weiss et al. (2000) found that they had 90% attendance for their 12-week supervised intervention. Also, most studies involved the prescreening of participants.

Mixed fitness training after stroke

The studies reviewed in this section and included in Table 8 represent those that did not attempt to control for either more aerobic training or pure strength training. Overall, there is no evidence of adverse events, and nomusculoskeletal injuries, falls, and cardiovascular events were disclosed or reported.

A recent Cochrane review was published by Saunders et al. (2009), which suggests that fitness training improves locomotor ability after stroke. Overall, more research is needed to further clarify the benefits and necessary thresholds to improving fitness after stroke.

Physical activity during treadmill training for locomotor rehabilitation after SCI

The studies in this section were restricted to recent RCTs. There were 3 RCTs included in this analysis (Postans et al. 2004; Field-Fote et al. 2005; Dobkin et al. 2006). These studies comprised a total of 222 people, and the details of each intervention can be found in Table 9. For the 1 ongoing trial (Field-Fote et al. 2005), data were extracted from the review of Mehrholz et al. (2008). The level of primary spinal cord lesions in participants ranged from C_3 to L_4, with ASIA grades of A to D.

It is important to note that these studies report adverse events and dropouts. Further, as with the issue identified above for stroke, almost all body-weight supported treadmill training studies did not provide physiological measure of metabolic or physiological cost. Instead, the intensity of treatment is presented in terms of the duration of the treadmill training. The longest duration was 60 min (Field-Fote et al. 2005). Across the 3 trials, the dropout rates were 0% to 20%, and adverse event rates were 0% to 4% (see Table 9). The adverse events were generally due to orthopedic (tendon or joint pain) injuries. No cardiovascular events were identified. However, as with the points above for stroke, and as listed in Table 9, participants were prescreened, with considerations related to autonomic dysfunction ((Dobkin et al. 2006) or cardiovascular disease (Postans et al. 2004; Field-Fote et al. 2005).

Discussion

Risk-to-benefit ratio of exercise after stroke or SCI

Actual risk of exercise after stroke?

The most rigorous information on the risks associated with physical activity participation after stroke comes from rehabilitation studies that examine exercise doses that are typical for activities of daily living. Most studies are from rehabilitation interventions in which participants have been prescreened. Furthermore, most of these interventions do not involve cardiovascular stress that is beyond activities of daily living, and are well below those needed to be considered exercise or sufficient to evoke training effects. It has been commented upon numerous times in the literature that the level of exercise stress typically found in commonly used rehabilitation protocols is insufficient to offset physical deconditioning or to sustain long-term health benefits (Gordon et al. 2004; Ivey et al. 2006). Because of the attendant and probable cardiovascular comorbidity, graded exercise testing with electrocardiograph monitoring has been recommended for stroke participants, following on from treatment guidelines for participants after a myocardial infarction and using submaximal protocols (Gordon et al. 2004). However, in many cases, it is impractical or impossible for stroke survivors to have an exercise test before beginning an exercise intervention program (Gordon et al. 2004). Therefore, lighter and lower-intensity (and lower risk) activities are recommended in conjunction with medical prescreening (Gordon et al. 2004; Ivey et al. 2008). These are summarized in Table 10.

That low-intensity exercise, such as that occurring during treadmill training, is well tolerated in stroke concurs with the conclusion of the Ottawa Panel report (Ottawa Panel et al. 2006). It suggested that, particularly in contrast to pharmacological intervention, noninvasive physical rehabilitation after stroke presents few adverse effects or contraindications. A similar general conclusion was reached by van de Port et al. (2007). They suggested that lower limb cycling exercise, strengthening, and gait training were safe and of utility after stroke. However, these conclusions need to be tempered by the conclusions of other authors, such as Meek et al. (2003). They conducted a systematic review of the literature to identify studies that involved cardiovascular exercise training after stroke. Only 3 trials were identified as rigorously controlled, and the conclusion was that "insufficient evidence was identified to establish if cardiovascular exercise has a positive effect on disability, impairment, extended activities of daily living, quality of life and case fatality post stroke" (p. 6).

Table 6. Summary of trial design, exercise dose, and adverse events during treadmill rehabilitation exercise training after stroke.

Study	Quality (Level of Evidence)	Participants		Dose		Effort	Adverse events
		Experimental group	Control group	Experimental group	Control group		
Ada et al. 2003	3 (2B)	n = 13; mean age 66.0 y; 28.0 mo post	n = 14; mean age 66.0 y; 26.0 mo post	3×30 min per wk for 4 wk	3×30 min per wk for 4 wk; low-intensity home program	Light	EXP = 2 (1 fall, 1 back pain)
da Cunha et al. 2002	3 (2A)	n = 6; mean age 57.8 y; 15.7 d post	n = 7; mean age 58.9 y; 19 d post	5×20 min per wk for 2–3 wk	5×20 min per wk for 2–3 wk; task-oriented gait training	Light	0
Dean et al. 2000	3 (2B)	n = 6; mean age 66.2 y; 2.3 y post	n = 6; mean age 62.3; 1.3 y post	3×60 min per wk for 4 wk	3×60 min per wk for 4 wk; upper limb training	Light	EXP = 1 (muscle soreness)
Eich et al. 2004	3 (2B)	n = 25; mean age 62.4 y; 6.1 wk post	n = 25; mean age 64.0 y; 6.3 wk post	5×30 min per wk for 6 wk	5×30 min per wk for 6 wk; nontask oriented	Light	0
Jaffe et al. 2004	2 (2A)	n = 10; mean age 58.2 y; 3.9 y post	n = 10; mean age 63.2 y; 3.6 y post	3×60 min per wk for 2 wk	3×60 min per wk for 2 wk; task-oriented walking	Light	0
Kosak and Reding 2000	3 (2B)	n = 22; mean age 74.0 y; 39.0 d post	n = 34; mean age 70.0 y; 40.0 d post	5×45 min per wk for 2–3 wk	5×45 min per wk for 2–3 wk; nonwalking	Light	EXP = 1 (acute MI after training ended); CON = 1 (stroke exacerbation)
Laufer et al. 2001	2 (2A)	n = 13; mean age 66.6 y; 32.6 d post	n = 12; mean age 69.3 y; 35.8 d post	5×20 min per wk for 3 wk	5×20 min per wk for 3 wk; task oriented	Light	0
Liston et al. 2000	2 (2B)	n = 18; mean age 79.1 y		3×60 min per wk for 4 wk	3×60 min per wk for 4 wk; task oriented	Light	EXP = 2 (1 knee pain; 1 hospitalized after initial training and perished in hospital)
Macko et al. 2008	3 (2B)	n = 32; mean age 63.0 y; 35 mo post	n = 29; mean age 64.0 y; 39.0 mo post	3×40 min per wk for 6 mo	3×40 min per wk for 6 mo; task oriented	Vigorous	EXP = 11 (minor falls with no complications)
Nilsson et al. 2001	3 (2A)	n = 36; mean age 54.0 y; 22.0 d post	n = 37; mean age 56.0 y; 17.0 d post	5×30 min per wk for 9–10 wk	5×30 min per wk for 9–10 wk; task oriented	Light	0
Pohl et al. 2002	3 (2B)	n = 40; mean age 57.6 y; 16.6 wk post	n = 20; mean age 61.6 y; 16.1 wk post	3×30 min per wk for 4 wk	3×45 min per wk for 4 wk; nontask oriented	Light	Exp = 1 (vertigo)
Richards et al. 1993	2 (4C)	n = 10; mean age 69.6 y; 8.3 d post	n = 8; mean age 67.3 y; 8.8 d post	5×105 min per wk for 5 wk	5×105 min per wk for 5 wk; nontask oriented	Light	NA
Scheidtmann et al. 1999	3 (2A)	n = 30; mean age 57.7 y; 52.2 d post		5×30 min per wk for 3 wk		Light	0
Visintin et al. 1998	3 (4C)	n = 50; mean age 66.5 y; 68.1 d post	n = 50; mean age 66.7 y; 78.4 d post	4×20 min per wk for 6 wk	4×20 min per wk for 6 wk; task oriented	Light	NA
Werner et al. 2002	2 (2A)	n = 15; mean age 59.7 y; 7.4 wk post	n = 15; mean age 60.3 y; 6.9 wk post	5×20 min per wk for 2 wk	5×20 min per wk for 2 wk; task oriented	Light	0

Note: CON, control group; EXP, experimental group; MI, myocardial infarction; NA, not applicable.

Table 7. Summary of trial design, exercise dose, and adverse events during resistance training after stroke.

Study	Quality (Level of Evidence)	Participant		Dose		Effort	Events
		Experimental group	Control group	Experimental group	Control group		
Inaba et al. 1973	3 (2A)	n = 77; mean age 56.0 y; less than 3.0 mo		5×0.5RM, 10×1RM 5× per wk for 4–8 wk; knee and hip flex–ext; dorsiflex		Hard	
Bourbonnais et al. 2002	3 (2A)	n = 25; mean age 45.5 y; 35.0 mo post		6–8RM, 3× per wk for 6 wk; arm and leg training		Moderate	
Bütefisch et al. 1995	3 (2B)	n = 27; mean age 61.5 y; 5.0–19.0 wk post		2×15 min of repetitive activation with variable loads 5× per wk for 4 wk; wrist and hand		Very light	
Engardt et al. 1995	2 (3A)	n = 20; mean age 63.0 y; 27.0 mo post		10 reps of isokinetic, eccentric, and concentric knee flex–ext (60, 120. 180°·s^{-1}) 2× per wk for 6 wk; knee extensors		Light	
Sharp and Brouwer 1997	2 (2B)	n = 15; mean age 67.0 y; >6.0 mo post		3 sets of 6–8 reps of isokinetic (30, 60, 120°·s^{-1}) knee flex–ext 3× per wk for 6 wk		Moderate	
Weiss et al. 2000	2 (3A)	n = 7; mean age 70.0 y; >1.0 y		3 sets of 8–10 contractions at 70% RM, 2× per wk for 12 wk; hip and knee flex–ext		Moderate	
Kim et al. 2001	2 (3A)	n = 10; mean age 60.4 y; 4.9 y post	n = 10; mean age 61.9 y; 3.2 y post	30 min of 3×10 reps at maximal effort 3 d per wk for 6 wk; hip–knee–ankle on paretic limb	Same as EXP but unresisted	Moderate	
Badics et al. 2002	3 (4C)	n = 56; 3.0 wk to 10.0 y post		Arm and leg resistance training; 5×20 reps at 30%–50% of maximal strength for 4 wk			
Winstein et al. 2004	3 (4C)	n = 60; 2.0–35.0 d post		Arm training with free weights or grip apparatus			
Karimi et al. 1996	2 (3B)	n = 10; mean age 62.3 y; 2.0–33.0 y post		10 maximal reps of isokinetic (60, 120, 180°·s^{-1}) 3× per wk for 8 wk; knee and hip flex–ext,dorsiflex–plantarflex		Moderate	
Giuliani et al. 1992	2 (3B)	n = 20; 61.5 y; 4.0 wk post		5× per week for 4 wk; isokinetic knee flex–ext (60, 90, 120°·s^{-1})		Moderate	
Ouellette et al. 2004	3 (2B)	n = 21; mean age 65.8 y; 31.8 mo post	n = 21; mean age 66.1 y; 25.6 mo post	3×8–10 reps at 70% of 1RM 3× per wk for 12 wk; hip–knee–ankle flex–ext exercise with pneumatic resistance or free weights; supervised	3 d per wk; bilateral range of motion and upper body flexibility	Moderate	EXP = 3 (inguinal hernia, angina, stent); CON = 1 (ECG abnormalities)

Note: ECG, electrocardiograph; ext, extention; flex, flexion; rep, repetition; RM, repetition maximum.

Table 8. Summary of trial design, exercise dose, and adverse events during fitness training after stroke.

	Quality (Level of	Participants		Dose			
Study	Evidence)	Experimental group	Control group	Experimental group	Control group	Effort	Events
Duncan et al. 1998	2 (4C)	n = 10 (ischemic); mean age 67.3 y; 66 d post	n = 10; mean age 67.8 y; 56 d post (8 ischemic, 2 hemorrhagic)	Mixed cardio and resistance; 90 min ×3 d per wk for 12 wk (8 wk supervised, 4 wk alone); HR and BP monitored but not reported	Usual care	Moderate	Not reported
Glasser 1986	2 (4C)	n = 10; age range 40.0–75.0 y; 3–6 mo post	n = 10; age range 40.0–75.0 y; 3–6 mo post	Cardio on isokinetic ergometer; 2× per d, 5 d per wk for 10 wk; no incrementing of workload; sessions progressed from 10 to 30 min over first 5 wk	Therapeutic exercise and gait training, 1 h×2 sessions per d, 5 d per wk for 5 wk	Moderate?	Not reported
Potempa et al. 1995	2 (4C)	n = 19; age range 43.0–70.0 y; 216 d post	n = 23; age range 43.0–70.0 y; 216 d post	Cardio leg cycling erg, 30 min per d, 3 d per wk for 10 wk at 30%–50% maximal effort, incrementing to maximum sustainable over 4 wk; supervised	Passive range of motion, 30 min per d, 3 d per wk for 10 wk	Vigorous?; get actual HR, BP, workload etc. from file	Not reported
Teixeira-Salmela et al. 1999	1 (2A)	n = 7; mean age 65.9 y; 9.2 y post	n = 6; mean age 69.4 y; 6.4 y post	Mixed cardio and leg strength training 60–90 min per d, 3 d per wk for 10 wk; cardio progressing from 50%–70% maximal work rate; strength with Theraband 3×10 reps, progressing from 50%–80% 1RM	None	Vigorous; very hard effort	None reported
Cuviello-Palmer 1988	1 (4C)	n = 10; mean age 71.8 y; 16.8 d post	n = 10; mean age 69.5 y; 13.2 d post	Isokinetic cardio; 1–2×7 min per d for 6 d per wk for 3 wk; progressing to 12 min; HR<20 beats·min^{-1} above resting	Usual care	Moderate	Not reported

Note: BP, blood pressure; HR, heart rate.

Table 9. Summary of trial design, exercise dose, and adverse events during treadmill rehabilitation training after SCI.

Study	Study design; quality (Level of Evidence)	Participants		Dose		Effort	Adverse svents or dropout
		Experimental group	Control group	Experimental group	Control group		
Dobkin et al. 2006	Multicentre RCT, quality-screened (autonomic); 3 (2B)	n = 87; mean age 26 y; acute 26 d post (incomplete, ASIA B–D)	n = 88; mean age 24 y; acute 30 d post (incomplete, ASIA B–D)	~60 min per d×5 d per wk for 12 wk BWSTT	~60 min per d×5 d per wk for 12 wk of overground walking training	Moderate	20% dropout (n = 13); of 10 who dropped out prior to completing half of the intervention, 2 found it too difficult and 2 had tendon or joint injuries
Field-Fote et al. 2005	RCT, quality-screened for CV disease; 3 (2A)	n = 27; mean age 42–48 y; chronic 4–7 y post injury	Data are collapsed for all BWSTT groups and shown in EXP	~45 min per d of BWSTT 5 d per wk for 12 wk	NA	Moderate	0 dropouts
Postans et al. 2004	RCT, quality-screened (medical stability, standing balance); 2 (2B)	n = 14 acute iSCI (ASIA C or D); age range 19–71 y; 12 wk post	Single-case experimental design	~25 min per d of BWSTT 5 d per wk for 4 wk	NA	Light–moderate	14% dropout (n = 2); 1 due to orthopedic problems

Note: ASIA, American Spinal Injuries Association impairment scale; BWSTT, body-weight supported treadmill training; CV, cardiovascular; iSCI, incomplete spinal cord injury; NA, not applicable.

Actual risk of exercise after SCI?

Risks associated with physical activity participation after SCI are mainly related to associated comorbidities, such as the presence of cardiovascular disease and osteoporosis. In the case of higher-level lesions, the cardiovascular risks may be compounded by the manifestation of autonomic dysreflexia. Regardless of the nature of the central nervous system lesion, the common pathological outcome of neurological dysfunction is an interruption in the ability to activate muscle and produce movement. A direct effect of this is a corresponding reduced level of physical activity. Coincident with this, deconditioning is a reduced ability to respond to and meet the challenges of homeostasis. Reduced activity in neurological dysfunction has additional important secondary effects, which include osteoporosis, cardiovascular comorbidities, and an overall decreased functional capacity. As such, there is considerable benefit of participation in physical activity. Two specific considerations that are of considerable relevance to numerous pathologies are spasticity and generalized weakness. Additional issues arise due to the spectrum of pharmacological agents (e.g., antispastic medications, such as baclofen, dantrolene, and tizanidine) involved in the management of the impact of dysfunctions, such as spasticity. Because of the effect of the lesion in reducing the ability to activate skeletal muscle and the secondary reduction in physical activity, those with neurological dysfunction typically have low oxidative capacities and increased fatigue.

A key aspect of exercise prescription after neurological dysfunction is the aspect of functional relearning. This involves not just performing an exercise, such as leg or arm cycling, for the physiological benefit of activity, but also for retraining and enhancing the recovery of motor activity. This may seem difficult in contrast to the additional concern for balance control. Also, it is important to realize that, contrary to much dogma, functional gains can be expected well (years, decades) after any traumatic incident.

Overall, exercise in neurological dysfunction could be beneficial, particularly as the adaptations may help improve the efficiency and functional capacity associated with activities of daily living. Despite that, routinely, many individuals who could benefit from physical activity may be excluded from participation because of incorrect stereotypes related to functional capacity and the concern for harm. For example, there persists the perception that exercise may exacerbate spasticity, and that strength training should be avoided after stroke. This is balanced against valid concerns, such as the presence of autonomic dysreflexia after SCI.

There are potential benefits of exercise training on physiological outcomes in stroke and SCI. The relation between these outcomes and survival outcomes remains unclear. However, combined with the very low incidence of adverse events identified, it suggests that the risk-to-benefit ratio favors the recommendation for exercise. It must be emphasized that this recommendation is based on studies in which participants were almost universally screened, with many exclusions from participation. Thus, the grading of evidence for finding adverse events to support this conclusion is inadequate. It is also important to realize that most exercise training studies after stroke or SCI are conducted in supervised rehabilitation centres. This, combined with considerable subject bias due to

Table 10. Risk assessment for exercise after stroke and spinal cord injury.

Dysfunction	Main concern	Risk level	Level of activity	Supervision
Stroke	Cardiovascular comorbidity; re-incidence	Lower	Very light	Self-directed
		Lower	Light	Self-directed
		Intermediate	Moderate	QEP-directed
		Higher	Vigorous	MD-supervised (QEP-directed)
		Higher	Very hard effort	MD-supervised (QEP-directed)
		Higher	Maximal effort	MD-supervised (QEP-directed)
Spinal cord injury	Autonomic dysreflexia; cardiovascular	Lower	Very light	Self-directed
		Lower	Light	Self-directed
		Lower	Moderate	Self-directed
		Intermediate	Vigorous	Self-directed
		Intermediate	Very hard	QEP-directed
		Higher	Maximal	MD-supervised (QEP-directed)

Note: The table should be read according to what the level of risk is for a given level of activity under a certain level of supervision. For example, for stroke, the risk is lower when the level of activity is light, so that self-monitoring by the stroke survivor is appropriate. However, the risk is higher for a vigorous level of activity and should be supervised by a physician. MD, physician; QEP, qualified exercise professional.

a significant proportion of participants being deemed ineligible on the basis of medical or other criteria, means that the generalizability of the current recommendations to participants in everyday scenarios outside of a supervised clinical trial environment are unclear.

Caveat and limitations of scope

A key limitation and impediment to an estimation of risk for physical activity participation in neurological dysfunction is an absence of proper trials to evaluate risk. That is, in almost every instance, participants in studies related to exercise in neurological dysfunction are prescreened for any contraindications, and then medically supervised. Even in instances such as rehabilitation, where participants are unscreened, the relative exercise stress is often equivalent to activities of daily living. Therefore, it remains difficult to gauge the extent of exercise stress that can be safely tolerated. As such, the failure to identify significant adverse events in the many populations addressed here cannot be taken as a full assessment of all adverse events that may occur in unscreened and medically unsupervised scenarios. This is particularly the case for stroke, where significant cardiovascular comorbidities are not only associated with and arise subsequent to the dysfunction, but are also often the underlying cause of the pathology.

Conclusions

Overall, given the limitations outlined above, it appears that low-intensity exercise (~1.5× the level of activities of daily living) may be safe for medically stable participants who are 6 months beyond the cerebrovascular accident or SCI. That is, such exercise appears to pose a moderate risk. It should be clearly understood, however, that the boundary between the minimal level of activity required to produce a significant training effect and that for significant risk of adverse events remains to be fully explored.

It is suggested that new questions be added to the PAR-Q, explicitly asking about the history of neurological insult of stroke or SCI, such as: Have you been diagnosed with a stroke or SCI within the past 6 months?

It is suggested that the PARmed-X be revised to include a question related to autonomic risk, such as: Has the participant had a diagnosed complete SCI at T_6 or above or an incomplete cervical injury? If so, additional evaluation and screening for cardiovascular implications arising from autonomic dysreflexia are required.

Finally, in the cases of both stroke and SCI, age by itself does not appear to be a significant risk factor.

Recommendations

Recommendation no. 1: Individuals in the acute stage (less than 6 months from injury onset) of a stroke or spinal cord injury must be medically cleared to participate in physical activity under the direct supervision of a qualified exercise professional (Level 3, Grade A).

Recommendation no. 2: Owing to the risk of cardiovascular comorbidity and underlying pathophysiology, people with chronic stroke should only perform vigorous physical activity under the supervision of a qualified exercise professional (Level 3, Grade A).

Recommendation no. 3: Medical screening is explicitly needed for those with stroke and cardiovascular comorbidities, for those with complete spinal cord injury at T_{12} or higher and for those with incomplete spinal cord injury at T_6 or higher because of concerns related to autonomic dysreflexia. Exercise in this group should only be performed under the supervision of a qualified exercise professional (Level 4, Grade B).

The decision trees associated with stroke and SCI, and which account for the recommendations and conclusions above, can be found in Figs. 1 and 2, respectively.

Areas of research requiring additional evidence

The assessment of the evidence of risk and adverse events during exercise after stroke or SCI is confounded by a lack of rigour in reporting the physiological cost of physical activity and uncertainty about the extent and nature of adverse events. Accordingly, there are 2 major recommendations for future investigations. First, the measurement and reporting of level of exercise stress experienced during rehabilitation or exercise training in those with stroke or SCI must be increased. Enhanced measurement and reporting, including even rudimentary and superficial measures (such as heart rate or rat-

Fig. 1. Clinical decision tree for exercise screening after stroke, showing (A) initial Stage 1 Physical Activity Readiness Questionnaire (PAR-Q) application and, if necessary, (B) Stage 2 Physical Activity Readiness Medical Evaluation (PARmed-X) application. Diamonds indicate points of decision generating yes or no answers. R#1 and R#2 indicate specific outcomes that map onto recommendations contained in the main text. Please refer to the main text for level and grade of evidence supporting each recommendation.

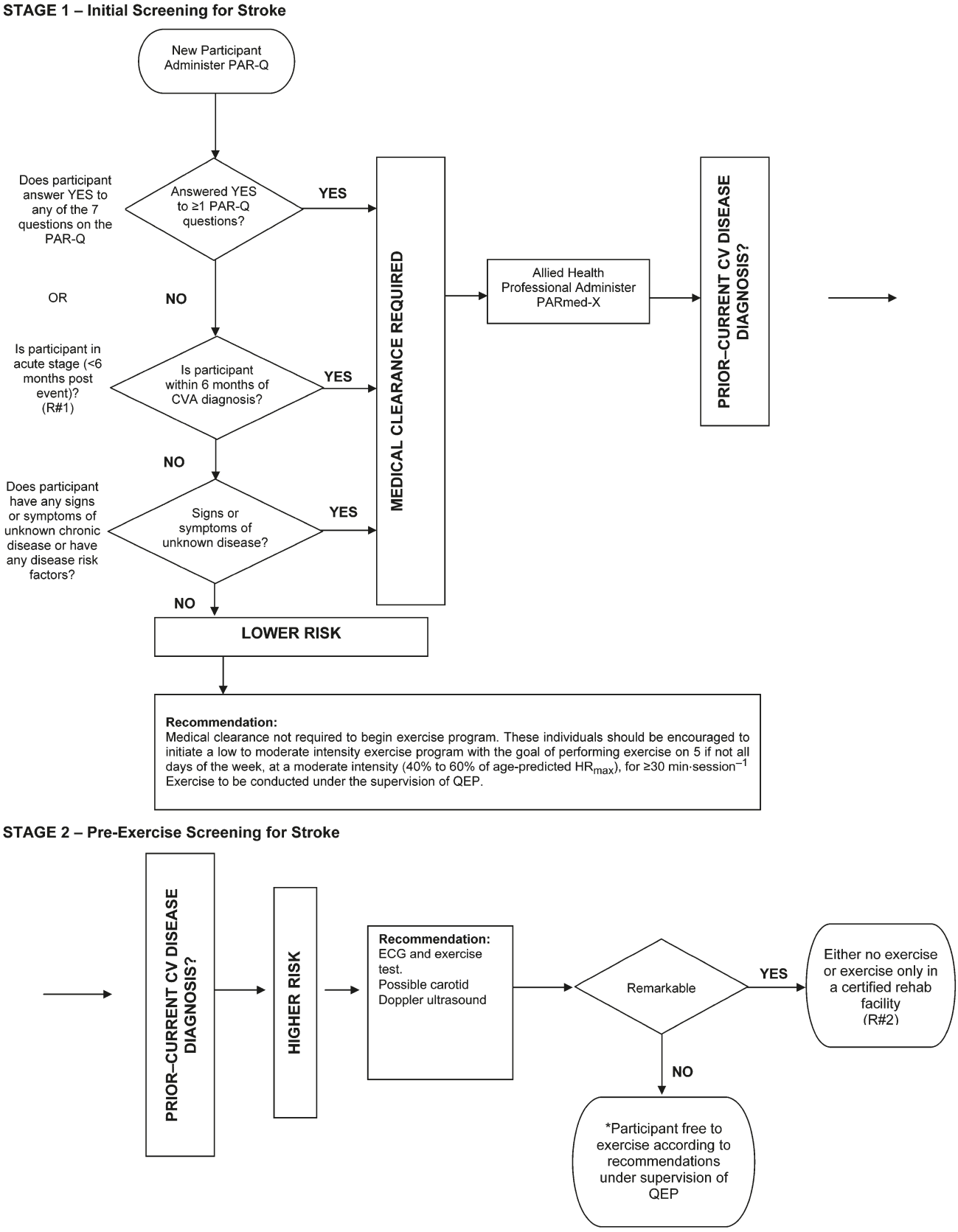
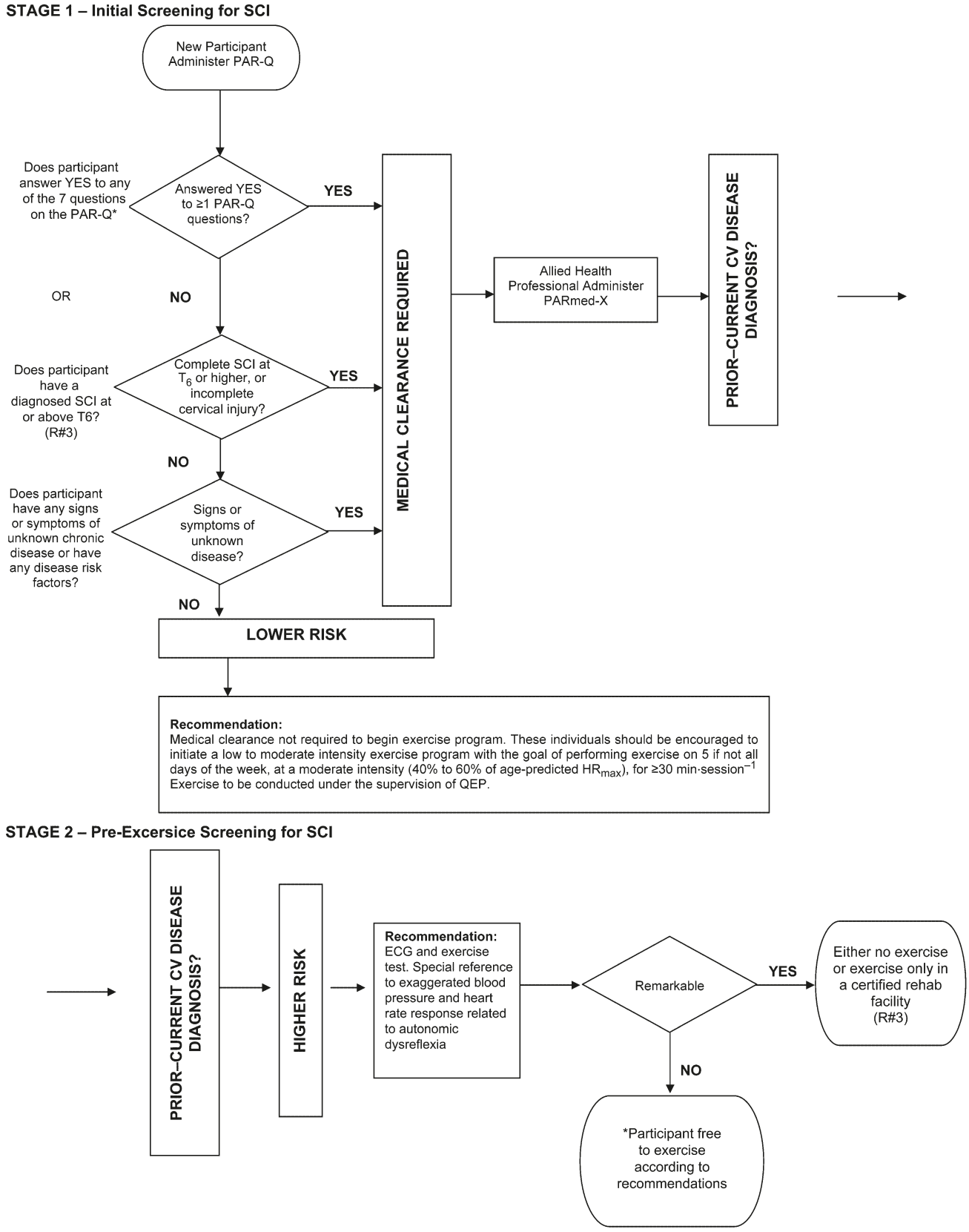


Fig. 2. Clinical decision tree for exercise screening after spinal cord injury showing (A) initial Stage 1 PAR-Q application and, if necessary, (B) Stage 2 PARmed-X application. Diamonds indicate points of decision generating yes or no answers. R#3 indicates specific outcome that maps onto recommendations contained in the main text. Please refer to the main text for level and grade of evidence supporting this recommendation.



ings of perceived exertion), would be an advance. Second, appropriate and detailed reporting of exercise data outcomes and adverse events must be increased. Currently, it is difficult to determine the actual risk of adverse events, owing to excessive exclusion criteria or inadequate reporting. The vast majority of articles in the field of exercise in neurological dysfunction do not do any form of formal adverse event monitoring, and it is often unclear when an adverse event is mentioned if it was determined only through an ad hoc process. Related to this, large-scale randomized trials that document valid adverse events in unscreened populations are needed. Additionally, it is still unclear what risks are associated with the performance of exercise that is of sufficient intensity to gain physiological training benefits after stroke or SCI.

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