



## Maximal muscle power after stroke: a systematic review

### Practice Points

- Stroke is a significant cause of disability, and stroke rehabilitation aims to reduce the impact of stroke-related deficits on activities of daily living and promote independence.
- Previous studies have shown that physical fitness is reduced following stroke. Both aerobic capacity and muscle strength are low following stroke, exacerbating disability and making daily tasks more challenging.
- Muscle power, defined as the maximum rate at which work can be applied, is another component of physical fitness. It appears to be important for functional activities in daily life, such as stair climbing and chair rising.
- It appears that muscle power is low in both the affected and unaffected side following stroke. Furthermore, it appears that low muscle power may be associated with poor walking performance.
- The pathophysiology of this is likely to be multifactorial, with the poststroke neurological deficit and the effects of aging playing a role. Additionally, disability after stroke leads to physical inactivity and it is likely that this reversible cause also plays a crucial role in reduced muscle power after stroke.
- Muscle power is a component of physical fitness that is amenable to training. Thus, clinicians should consider incorporating exercises to improve muscle power into stroke rehabilitation programs.

Maximal muscle power, defined as the maximum rate at which work can be applied, reflects both the force a muscle can produce and velocity at which it can apply that force. This article systematically reviewed all studies reporting maximal muscle power of the affected and unaffected limbs after stroke. Medline and EMBASE were searched (inception to 16 July 2012) and the reference lists were scrutinized. Cross-sectional studies, longitudinal studies and intervention trials reporting baseline data were included. From 2216 citations, six ( $n = 171$ ) met the inclusion criteria. Our findings showed explosive muscle power is low in both the affected and unaffected limbs of stroke patients and may also be associated with poorer walking performance. As low power is responsive to training, these observations support a rationale for using exercise to improve power and highlight the need for intervention studies to investigate the effect of power training on the physical function of stroke patients.

**Keywords:** muscle power • physical fitness • rehabilitation • stroke

Stroke rehabilitation aims to limit the impact of stroke-related deficits on activities of daily living through a combination of problem solving and therapeutic approaches [1]. Interventions to improve physical fitness are incorporated into

the rehabilitation of stroke survivors as they are thought to benefit patients in a number of ways. First, the physical impairments experienced following a stroke may require increased effort to participate in physical tasks, and this

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may increase fatigue and encourage inactivity. This leads to reduced fitness, making tasks more challenging and exacerbating disability [2]. Furthermore, low physical fitness is also a risk factor of stroke [3,4]. Recurrence of stroke is a significant problem; therefore, fitness training is also included in stroke rehabilitation programs, with the aim to reduce recurrence [5].

Physical fitness is defined as a set of attributes that people have or achieve that allow them to perform physical activity without undue fatigue [6]. It encompasses several concepts including cardiorespiratory fitness, strength, explosive power, balance and flexibility. The effect of stroke on cardiorespiratory fitness and strength has been extensively researched and considerable evidence states that both are reduced following stroke [7–10]. To date, no review has collated evidence about maximal explosive muscle power after stroke. Maximal explosive muscle power is defined as the greatest rate of work achieved during a single resisted, ballistic and muscle contraction. It reflects both the force a muscle can produce and the velocity at which it can apply that force [11,12]. Several different methods could be used to measure muscle power in stroke patients, including isokinetic dynamometers for power measurement in individual muscle groups, a Nottingham power rig for lower limb extension power and portable strain gauged calibrated dynamometers for determining hand grip power [13]. A vertical jump onto a force platform and pneumatic resistance training equipment can also be used to directly measure muscle power [14]. The unit for measuring power is Watts (W).

Some researchers consider power to be a more functionally relevant measure of muscle performance than strength, as it reflects the dynamic movements required in many daily activities [14]. Studies into healthy elderly individuals have shown that muscle power declines with age and is lower in women than men [15]. This decline can result in them being unable to generate the power required for many daily activities; for example, to mount a 30-cm step, you must be able to generate a lower limb extensor power of 1.5 W/kg [16]. Power below this level may limit the ability to climb stairs and contribute to loss of independence.

To our knowledge, there is no published narrative or systematic review of muscle power after stroke. Our primary aims are to use systematic review methodology to report maximal muscle power in the affected and unaffected upper and lower limbs of people with stroke and to determine if muscle power is low after stroke. Our secondary aim is to determine whether muscle power is related to functional ability after stroke.

## Methods

We aimed to include all published studies that measured maximal skeletal muscle power in stroke survivors.

Medline and EMBASE electronic databases were searched from inception to 16 July 2012 using MESH headings and key words ([Supplementary Appendix 1](#); please see online at [www.futuremedicine.com/doi/suppl/cpr.13.97](http://www.futuremedicine.com/doi/suppl/cpr.13.97)). Our inclusion criteria were:

- Studies that included at least ten stroke survivors, at any time since stroke;
- Cross-sectional studies, longitudinal studies and baseline data from controlled and uncontrolled intervention trials;
- Studies that directly measure maximum skeletal muscle power. All forms of direct measurement were considered;
- Only full-text articles were considered. Conference proceedings and abstracts were not included as they usually provide limited data so do not allow the quality of the study to be accurately appraised. The abstracts of non-English articles were read, but we were unable to translate them so we could not include them in the review. Where the results of a study were published in more than one article, the article with the most complete set of data was selected.

The titles and abstracts of articles identified in the literature search were first screened by one author (RL Knight) and irrelevant studies were removed. Full-text copies of the remaining articles were obtained and the inclusion criteria were applied. One author (RL Knight) assessed all the articles for inclusion. The two other authors (G Mead and DH Saunders) were each issued half the articles and independently applied the inclusion criteria. Any uncertainty was discussed with the third author (G Mead or DH Saunders). The reference lists of all included articles were searched for further relevant studies.

A data extraction form was devised by one author (RL Knight) and was piloted on one of the included studies. Data relating to participant demographics (total number of participants, age, time since stroke, pathological type of stroke and ambulatory status), outcome measure (method of measurement and muscle groups tested) and results (results of muscle power measurement and percentage of healthy norms for both the affected and unaffected sides) was extracted from each included study by one author (RL Knight). To ensure accuracy, the other two authors (G Mead and DH Saunders) were each allocated three of the papers and independently repeated the data extraction process. Where the data were presented in graphical form only, the authors were contacted and asked to provide the data. Failing this,

numerical data was interpolated using a ruler. Data relating to the association between muscle power and walking performance was also extracted from articles that reported comfortable and maximum walking speed of the participants. Included articles were critically appraised for bias by one author (RL Knight) using the Downs and Black checklist (Supplementary Appendix 2) [17]. Not all items in the checklist were relevant so some were omitted to create a modified version that was more suitable for this present review. When reporting our findings, the PRISMA checklist for the preferred reporting of systematic reviews was adhered to [18].

## Results

### Study selection

Figure 1 shows how studies were selected. The title and abstract of 2216 articles were examined; 2183 were excluded after reviewing the title and abstract. Two further studies were identified through searches of reference lists. Of these 35 relevant studies, three were abstracts and two were not available in English, so 30 full-text articles were reviewed. Eight fulfilled the inclusion criteria, but two [19,20] were excluded as they reported data from the same participants. Thus, six articles, recruiting a total of 171 participants, were included in the review.

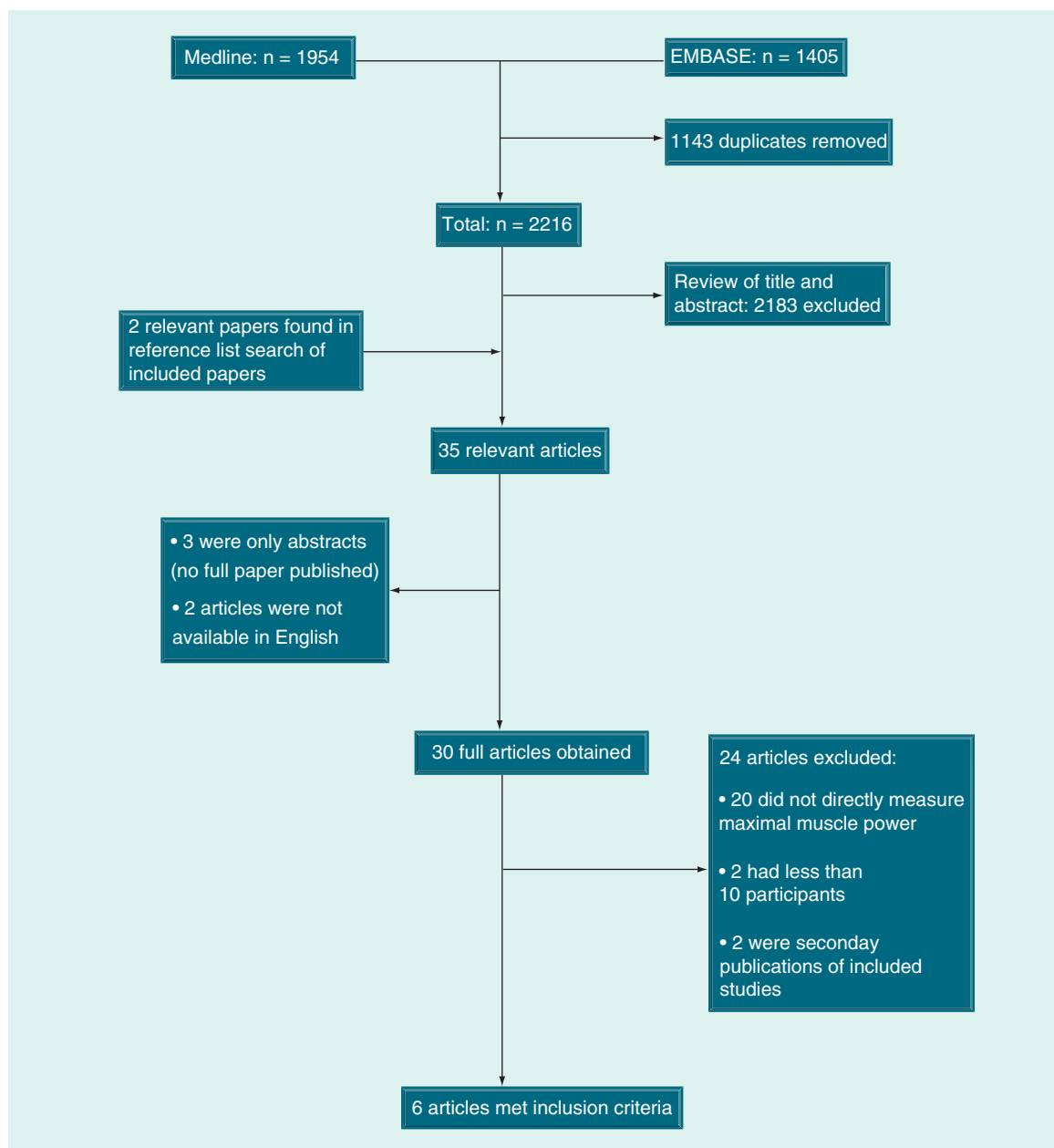


Figure 1. Flow diagram of study inclusion.

### Characteristics of studies

The characteristics of included studies are reported in [Table 1](#). Four of the studies were cross-sectional [21–24], one of which included a group of healthy people as controls, and two were randomized controlled trials [11,25]. The number of participants in each study ranged from ten [23] to 66 [11], and the mean age of the participants ranged from 56 [23] to 72 years [11]. Three studies [21,24–25] reported mean time since stroke and ranged from 2.3 [21] to 57.0 [25] months; two studies [11,23] reported time since stroke as a range between 24.3 and 29.2 months [23] and 2.8 and 9.3 months [23]. Only one study [23] reported stroke severity using the hand dimension of the Chedoke–McMaster Stroke Assessment; all participants were considered to be level 4 or 5, which corresponds to a moderate impairment [26].

Five studies [11,21,22,24,25] reported ambulatory status. Three stated participants could ambulate independently with or without a walking aid [11,21,22]. One study [25] required participants to have a gait velocity between 0.15 and 1.4 m/s, but did not specify if they could be assisted or use walking aids. Another [24] reported that all participants were level 2, 3 or 4 for the Functional Ambulation Categories test, which corresponds to a low level of physical assistance, supervision only and independent ambulation on flat ground, respectively [27].

### Method of power measurement

The method of power measurement is reported in [Table 2](#). Two studies measured power using a Nottingham Power Rig [11,22], two used an isokinetic

dynamometer [21,24], one used a pneumatic resistance machine [25] and one a custom built jig [23]. Five of the six studies measured muscle groups of the lower limb, including leg extensors [11,22], knee extensors [21,24], knee flexors [24] and total leg power [25]. The final study measured power in the extensor and flexor muscles of the metacarpophalangeal joints (MCPJs) [23].

### Power of the affected side

As shown in [Table 2](#), two studies reported maximal lower limb extension power in the affected limb. One reported a mean of 1.07 W/kg [22] and the other reported a median of 0.92 W/kg [11]. The other four studies reported power in W and did not correct for body mass. One study found mean total leg power of the affected side to be 190.5 W [25]. Two studies measured mean knee extension power and it was found to range from 10.10 to 70.2 W [21,24]. Mean knee flexor power was measured by one study and found to be between 12.00 and 60.00 W [23]. One study measured mean power of the MCPJ flexors of the affected hand and it was found to be between 0.15 and 0.80 W [23]. Valid data was not reported for MCPJ extension as participants were unable to perform the task.

### Power of the unaffected side

In the unaffected limb, mean maximal lower limb extension power was reported as 1.99 W/kg by one study [22], and a median of 1.05 W/kg by another [11]. One study found mean total leg power of the unaffected side to be 649.1 W [25]. Mean knee extension power of the unaffected side ranged from 26.26 to

Table 1. Study and participant characteristics.

Study (year)	Study type	Total number of participants (M/F)	Age, mean (SD)	Time since stroke, mean (SD) unless otherwise stated <sup>†</sup>	Ambulatory status	Ref.
Bohannon (1992)	CS	20 (10/10)	63.7 (14.9)	2.3 (3.6) months	Independent ± walking aid	[21]
Conrad and Kamper (2012)	CS	10 (6/4)	56 (8)	Range: 24.3–292.0 months	Not reported	[23]
Dawes <i>et al.</i> (2005)	CS	14 (8/6)	46 (8)	Not reported (>6 months)	Independent >4 min ± walking aid	[22]
Lee <i>et al.</i> (2008)	RCT	48 (28/20)	63.2 (9.0).	57.0 (54.2) months	Gait velocity between 0.15 and 1.4 m/s	[25]
Prado-Medeiros <i>et al.</i> (2012)	CS	Stroke: 13 (9/4) Control: 13 (9/4)	Stroke: 53.6 (8.1) Control: 53.1 (8.2)	47.2 (29.9) months	Functional Ambulation Categories test level 2, 3 or 4	[24]
Saunders <i>et al.</i> (2008)	RCT	66 (36/30)	71.85 (9.91)	Range: 2.8–9.3 months	Independent ± walking aid	[11]

<sup>†</sup>Where the authors did not report time since stroke in months, the approximate number of months has been calculated.  
CS: Cross-sectional study; F: Female; M: Male; RCT: Randomized controlled trial; SD: Standard deviation.

**Table 2. Methods and outcomes of measuring power.**

Study (year)	Methods	Results of muscle power measurement (Watts, unless otherwise stated), mean (SD, unless otherwise stated)		Ref.			
		Affected side	Unaffected side				
	Method of measurement	Muscle groups tested	Affected side	Unaffected side	Affected side	Unaffected side	
Bohannon (1992)	Isokinetic dynamometer	Knee extensors	70.2 (56.4)	164.6 (105.2)	Not reported	Not reported	[21]
Conrad and Kamper (2012)	Custom made jig that simultaneously controlled the motion of the MCPJs with an optical-encoded tachometer and torque transducer to measure angular rotation, velocity and force, respectively	MCPJ flexors and extensors	Extension <sup>†</sup> Flexion: • 10°/s: 0.15 (1.45) • 60°/s: 0.53 (0.84) • 120°/s: 0.80 (0.83)	Extension: • 10°/s: 0.08 (0.90) • 60°/s: 0.43 (0.73) • 120°/s: 0.46 (0.68) Flexion: • 10°/s: 0.22 (1.77) • 60°/s: 1.39 (1.71) • 120°/s: 1.76 (1.81)	Not reported	Not reported	[23]
Dawes <i>et al.</i> (2005)	Nottingham leg extensor power rig	Leg extensors	1.07 (0.50) W/kg	1.99 (0.85) W/kg	Not reported	56% of healthy male norms, 83% of healthy female norms	[22]
Lee <i>et al.</i> (2008)	Pneumatic resistance machine	Leg extension, knee extension and knee flexion then combined to form total leg power	190.5 (102.2)	649.1 (242.9)	Not reported	Not reported	[25]
Prado-Medeiros <i>et al.</i> (2012)*	Isokinetic dynamometer	Knee extensors and flexors	Knee extensors: • Con: 60°/s: 24.24 (8.1) • Con: 120°/s: 36.36 (14.1) • Ecc: 60°/s: 10.10 (8.1) • Ecc: 120°/s: 13.13 (11.1) Knee flexors: • Con: 60°/s: 12.00 (5.0) • Con: 120°/s: 12.0 (8.0) • Ecc: 60°/s: 31.00 (13.0) • Ecc: 120°/s: 60.00 (27.0)	Knee extensors: • Con: 60°/s: 66.67 (14.1) • Con: 120°/s: 109.09 (24.2) • Ecc: 60°/s: 26.26 (12.1) • Ecc: 120°/s: 36.36 (18.2) Knee flexors: • Con: 60°/s: 37.00 (11.0) • Con: 120°/s: 60.00 (15.0) • Ecc: 60°/s: 58.00 (18.0) • Ecc: 120°/s: 98.00 (27.0)	Knee extensors: • Con: 60°/s: 27% • Con: 120°/s: 27% • Ecc: 60°/s: 19% • Ecc: 120°/s: 16% Knee flexors: • Con: 60°/s: 24% • Con: 120°/s: 16% • Ecc: 60°/s: 41% • Ecc: 120°/s: 45%	Knee extensors: • Con: 60°/s: 73% • Con: 120°/s: 82% • Ecc: 60°/s: 48% • Ecc: 120°/s: 44% Knee flexors: • Con: 60°/s: 74% • Con: 120°/s: 81% • Ecc: 60°/s: 76% • Ecc: 120°/s: 74%	[24]
Saunders <i>et al.</i> (2008)	Nottingham leg extensor power rig	Leg extensors	Median: 0.92 W/kg IQR: 0.53–51.49 W/kg	Median: 1.05 W/kg IQR: 0.73–71.56 W/kg	42%	54%	[11]

<sup>†</sup>The participants were not able to maintain an extension force during the trials testing power in the MCPJ extensors. This resulted in a net flexion movement; therefore, the results have not been included as they do not reflect the task the participants were asked to perform.  
<sup>\*</sup>When measuring muscle power, these studies fixed the maximum velocity the participants were able to apply to the mass. Still constitutes maximal power as participants were instructed to make a maximal effort.  
 Con: Concentric muscle contraction; Ecc: Eccentric muscle contraction; IQR: Interquartile range; MCPJ: Metacarpophalangeal joint; SD: Standard deviation.

164.6 W [22,25]. Mean knee flexor power was between 37.00 and 98.00 W [24]. In the unaffected hand, mean MCPJ flexor power was between 0.22 and 1.76 W and mean MCPJ extensor power was between 0.08 and 0.46 W [23].

### Comparison with healthy people

Two studies reported power on the affected side compared with healthy control or normative data [11,24]. It was found to be between 16 and 45% of normal. It was reported by three studies on the unaffected side and it was 44 to 76% of normal [11,22,24].

### Association with functional ability

Three studies (Table 3) examined the association between muscle power and comfortable walking speed: two found a positive correlation [11,21] and one found a negative correlation [22], although this was not a significant finding. One study [21] also found a positive correlation between muscle power and maximum walking speed. These findings suggest low muscle power predicts poor function.

### Discussion

To our knowledge, this is the first systematic review to report maximal skeletal muscle power after stroke. Not surprisingly, power is lower in the affected side than the unaffected side. Furthermore, power appears to be reduced in both the affected and unaffected limbs compared with healthy controls. Results from two of the included studies suggest low muscle power may be associated with reduced comfortable and maximal walking speed. These findings reflect what happens to muscle strength after stroke [7,28–30].

First, consideration should be given to the limitations of this review. Owing to restricted resources, we only searched two databases. However, we placed an emphasis on broad terms when designing our search strategy and scrutinized the reference lists of all

included articles to minimize the risk of missing relevant studies. We were not able to translate non-English articles so they may be under-represented in the review. Finally, only six articles met our inclusion criteria; this limits the scope of our findings and the conclusions we draw. This is particularly relevant when applying our findings to muscle power in the upper limb, as only one included study [23] investigated this.

Furthermore, the limitations of the studies included in this review warrant a mention. Only one study [23] attempted to quantify stroke severity and they reported the severity of deficit in the hand only; thus, we lack a full picture of the functional status and limitations of the participants. We can, however, deduce from the ambulatory status data that most of the participants included in the studies had a moderate-to-high level of physical function, and in the studies that studied power in the hand all participants had moderately impaired hand function. Additionally, all included studies excluded participants with severe cognitive deficits. Therefore, it is unlikely the participants are truly representative of all stroke patients as it appears those suffering from severe impairments were excluded. Furthermore, despite most stroke patients being elderly [31], the mean age of the participants ranged from 56 to 72 years. This does not reflect the age range at which stroke commonly occurs. Thus, these findings may not generalize to older or more disabled stroke populations.

The reliability of the method of measurement used in some of the studies should be considered. Two studies [11,22] used a Nottingham power rig, which research has shown is an acceptable measure of explosive lower limb extension power [32] with good test–retest and intraobserver reliability. One study [21] reported good test–retest reliability. The remaining three studies [23–25] did not report on the reliability of their outcome measure. Future research should state whether their outcome measures are reliable in stroke patients.

Table 3. Association between power and walking.

Study (year)	Comfortable walking speed		Maximum walking speed		Ref.
	Power affected side	Power unaffected side	Power affected side	Power unaffected side	
Bohannon (1992)	$r = 0.752$ , $p = 0.000^{\dagger}$	$r = 0.613$ , $p = 0.004$	$r = 0.735$ , $p = 0.000$	$r = 0.507$ , $p = 0.023^{\S}$	[21]
Dawes <i>et al.</i> (2005)	$r = 0.37$ , NS <sup>‡</sup>	$r = -0.28$ , NS	NR	NR	[22]
Saunders <i>et al.</i> (2008)	$r^2 = 0.28$ , $p < 0.001^{\dagger}$	$r^2 = 0.41$ , $p < 0.001$	NR	NR	[11]

<sup>†</sup>Two studies found a positive association between comfortable walking speed and power of both the affected and unaffected sides.

<sup>‡</sup>One study found no associations between power and comfortable walking speed.

<sup>§</sup>One study reports finding a positive correlation between maximum walking speed and power on the affected and unaffected sides.

NR: Not reported; NS: Not significant.



Only three studies [11,22,24] compared their findings to healthy norms. One of these studies [24] included a healthy control group to calculate these from, which is generally considered best practice. The remaining two compared their findings to a set of population norms [16] which, although these were determined using the same method and equipment, are likely to represent a less accurate reflection of the participants as a control group would as the measurements were made in a different setting and at a different time. Due to wide variation in the methods used to measure muscle power, we did not compare the findings of the remaining three studies to these population norms. Future research in this area could be improved by the inclusion of an age- and gender-matched control group to allow stronger and more accurate conclusions to be drawn regarding the difference in muscle power between stroke patients and healthy individuals.

Finally, the methods used to recruit participants to the studies may also be a source of bias. One study used a convenience sample [21] and another relied on consultant referrals [22], both of which allow self-selection of fitter patients. Two studies [23,24] did not report how they recruited participants and only two [11,22] of the six studies adjusted for differences in participants' body mass. For the studies investigating power in the lower limb, body mass has a considerable effect on the variation in power observed between participants so it should be corrected for.

The pathophysiology of low muscle power is probably multifactorial. First, the direct pathological effects of stroke make a significant contribution to power loss in the affected side. Loss of cortical control and hemiparesis result in reduced activation of motor neurons supplying the affected side. Muscle atrophy, reduced cross sectional area and altered fiber-type composition have also been observed [33,34]. Reduced motor neuron activation may also contribute to the decline in muscle power observed in the unaffected limb as approximately 10% of descending motor fibers are thought to remain on the ipsilateral side [35,36].

It is likely that inactivity also plays a significant role in determining muscle power after stroke. The muscle weakness, ataxia and hemisensory loss that occur in stroke can affect an individual's ability to participate in physical activity [37]. The resulting sedentary lifestyle leads to deconditioning and a decline in muscle power on both sides. Bed rest has also been shown to reduce muscle power so it is possible that bed rest in the acute stages of stroke may also effect muscle power generation [38]. Furthermore, physical inactivity is a risk factor of stroke [38], so low muscle power might have existed prior to the occurrence of stroke due to a pre-existing sedentary lifestyle.

Finally, the effects of aging on muscle power may also contribute. Muscle power is known to decline by 3.5% per year between 65 and 89 years of age [15]. Almost 75% of stroke patients are over 65 years of age [31], so it is likely that aging contributes to the low levels of muscle power observed in stroke patients. However, as we have found that power is reduced compared with age-matched controls, it is clear stroke has an independent effect.

An association between muscle power and walking ability is hinted at by our findings, suggesting power of the lower limbs may be an important determinant of function after stroke. Previous work has shown that muscle power in elderly and disabled people is linked to functional activities including stair climbing and chair rising [13,31]. In fact, evidence suggests muscle power may be a more influential predictor of functional performance than both strength and aerobic capacity [14,39]. However, more research is warranted before valid conclusions can be drawn.

## Conclusion

Our findings show skeletal muscle power is low after stroke in the affected and unaffected side. It also appears muscle power may be a determinant of walking ability in stroke patients, in-keeping with previous research that has shown a strong link between muscle power and functional ability in other patient groups [14,16,31–32]. This suggests that exercise interventions for stroke patients that aim to improve their maximal muscle power may be beneficial to their function; however, large intervention trials are required before firm conclusions can be drawn about the effect of power training on the functional capabilities of stroke patients.

## Future perspective

It would appear that muscle power is an important determinant of functional ability after stroke. However, further studies investigating muscle power after stroke are warranted. Furthermore, our review has highlighted concerns in the methodology of previous studies that future work should aim to address. Researchers should strive to recruit participants that accurately reflect the whole stroke population by investigating maximal muscle power in those with mild, moderate and severe strokes. They should also include a control group and ensure their outcome measures are reliable. Longitudinal studies to investigate how muscle power in stroke patients evolves over time would also be of value to determine the stage of stroke rehabilitation that interventions to improve power should be introduced. Finally, large intervention studies are needed to determine whether fitness training focused on improving power leads to improved function after stroke.

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The authors have no relevant affiliations or financial involvement with any organization or entity with a financial

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