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REVIEW ARTICLE (META-ANALYSIS)

Specific Brain Lesions Impair Explicit Motor Imagery Ability: A Systematic Review of the Evidence

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Abstract

Objective: To determine which neurologic disorders/lesions impair or restrict motor imagery (MI) ability.

Data Sources: CINAHL, Cochrane, Embase, MEDLINE, Web of Science, PsychINFO, Physiotherapy Evidence Database, and Grey Literature were searched between May 8 and May 14, 2014. Keywords and Medical Subject Headings from 2 concepts (MI and lesion) were exploded to include related search terms (eg, mental practice/mental imagery, neurologic damage/lesion).

Study Selection: Two independent reviewers assessed the 3861 studies that resulted from the database search. The studies were assessed for relevancy using the following inclusion criteria: use of explicit kinesthetic MI; neurologic lesion location identified; and use of an MI ability assessment tool.

Data Extraction: Twenty-three studies encompassing 196 participants were included. The 23 studies used 8 different methods for assessing MI ability. MI assessment scores were then normalized to facilitate comparison across studies.

Data Synthesis: Lesion locations comprised many brain areas, including cortical (eg, parietal and frontal lobes), subcortical (eg, basal ganglia, thalamus), and cerebellum. Lesion etiology primarily was comprised of stroke and Parkinson disease. Several participants presented with lesions resulting from other pathologies. Subjects with parietal lobe damage were most impaired on their ability to perform MI. Subjects with frontal lobe and basal ganglia damage also consistently showed impairment in MI ability.

Conclusions: Subjects with damage to specific brain structures, including the parietal and frontal lobes, showed impaired MI ability. As such, MI-based neurorehabilitation may not be efficacious in all patient populations. Therefore, decisions related to the use of MI in neurorehabilitation should, in part, be based on the patient's underlying pathophysiology.

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Motor imagery (MI), the mental practice of a movement without actual execution, is emerging as a useful adjunct in rehabilitation for patients with neurologic injury.[1-3] Current neurorehabilitative strategies generally rely on motor execution (ME) to stimulate, and subsequently rewire, damaged neural networks via neuroplasticity.[4] Neuroplasticity, or rewiring of motor networks, ultimately leads to the recovery of movement. Interestingly, research has shown that MI and ME activate similar brain regions, providing the basis for why MI has the potential to recover lost motor function via neuroplasticity similar to ME.[5,6] Unlike ME, MI negates the necessity for movement, allowing patients with little to no motor function to engage in neurorehabilitation. Therefore, MI provides a gateway therapy for patients with severe impairment and a means to increase the therapeutic dose as an adjunct to traditional ME-based interventions.[1,7,8] Clinicians are using MI therapy as a tool for patients with neurologic disorders, including acquired and traumatic brain injury, Parkinson disease (PD), cerebral palsy, among others.[9-12]

The literature describes MI as visual or kinaesthetic.[13] Although visual MI requires a subject to imagine watching themselves (or another individual) perform a movement (ie, imagining from a third-person perspective), kinesthetic MI requires the subject to mentally rehearse performing the movement (ie, imagining from a first-person perspective).[14] MI is further characterized as implicit or explicit.[15] Briefly, imagined movement is the cognitive mechanism behind implicit MI, whereas imagined movement is the sole purpose of explicit MI. For example, an individual would engage in implicit MI during a mental rotation task where they are required to assess photographs of hands in different positions and determine the handedness.

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Conversely, individuals engage in explicit MI for the sake of mentally rehearsing the movement. These 2 categories of MI describe different aspects of the cognitive task and are therefore not mutually exclusive; it is possible for MI to be both visual and implicit, kinesthetic and explicit, or vice versa. Although MI tasks used in neurorehabilitation are primarily explicit, both visual and kinesthetic MI is implemented. This review will solely focus on explicit, kinesthetic MI because it is the closest relative to ME with respect to brain activity.[1,16,17]

There is mixed support for the implementation and use of MI in rehabilitation. Only some researchers have found MI to be efficacious in driving functional recovery of movement. Page et al[18] found that MI successfully induced plastic changes in the cortex with subsequent movement improvement in the affected limbs of patients poststroke. Conversely, a randomized controlled trial by Ietswaart et al[19] found no benefit for MI in stroke rehabilitation. Moreover, a meta-analysis conducted by Braun et al[20] reported that only 6 of 14 studies showed beneficial effects of MI in stroke rehabilitation.

The discrepancy in the literature concerning the efficacy of MI-based rehabilitation may be partially explained by the lack of control for MI ability (ie, including participants in the study who have an impaired ability to do MI). In other words, a patient's brain damage may prevent successful MI performance accounting for the absence of an MI-based treatment effect observed in other studies.[19,20] Indeed, it has been well substantiated that parietal lobe damage impairs MI performance.[21,22] Although damage to the parietal lobe evidently impairs MI, the impact of neurologic damage elsewhere on MI ability remains to be systematically evaluated. Moreover, many studies have reported MI impairment after stroke.[23-26] Overall, the body of MI literature lacks reports of MI impairment after neurologic damage specifically relating degree of MI impairment to lesion location.

In light of this gap in the MI literature, this review examines the literature investigating MI in individuals with neurologic disorders to determine which disorders and/or lesions impair or even prevent an individual from performing MI. Understanding which neurologic disorders and lesions impact MI performance would allow clinicians to individually tailor rehabilitation by taking into consideration a patient's underlying pathophysiology and therefore ability to perform MI. This approach would ensure MI-based therapy is prescribed appropriately in an evidence-informed manner.

To achieve this objective, we systematically searched the MI literature and reviewed all studies that measure MI ability in adult patients with neurologic disorders and identified lesion location(s). We anticipate our findings will help to inform decisions regarding the use of MI-based therapy in neurorehabilitation.

Methods

Study selection criteria

The literature search was conducted between May 8 and May 14, 2014, by a reference librarian. Seven electronic databases were searched, including CINAHL, Cochrane, Embase, MEDLINE, Web of Science, PsychINFO, Physiotherapy Evidence Database, and the Grey Literature (eg, Canadian Public Policy Collection, Cochrane Central Register of Controlled Trials) from inception to present. The search was limited to adult (≥18y) participants. Keywords and Medical Subject Headings from 2 concepts (MI and lesion) were exploded to include related search terms (eg, mental practice/mental imagery, neurologic damage/lesion). The resulting 3861 titles/abstracts were exported to a reference manager database, and the duplicates were removed. A total of 1809 titles/abstracts remained.

In 2 phases, 2 independent reviewers selected and analyzed the studies for inclusion. In both phases, a third reviewer resolved disagreements about study inclusion. The multiphase approach ensured a broad, comprehensive literature search that would identify all potentially relevant sources. A representative search, including all keyword and Medical Subject Headings combinations and filters, is presented in supplemental appendix S1 (available online only at http://www.archives-pmr.org/).

List of abbreviations:

KVIQ	**Kinesthetic and Visual Imagery Questionnaire**
ME	**motor execution**
MI	**motor imagery**
M1	**primary motor cortex**
PD	**Parkinson disease**

Phase 1: assessing citations for relevancy

Two reviewers assessed the titles/abstracts of the 1809 citations. Criteria for inclusion were use of kinesthetic MI and participants with a neurologic disorder/lesion. Each reviewer completed a spreadsheet wherein he or she recorded both citation information and decision for inclusion or exclusion. Following the end of phase 1, 305 citations remained.

Phase 2: further refining the literature

The full texts of the remaining citations were retrieved and imported into the reference database. After discarding citations for which the full text was unavailable or not published in English, 251 studies remained. In this phase, both reviewers assessed the full texts to ensure that studies measured MI ability and reported specific lesion locations. Each reviewer completed a spreadsheet wherein his or her reason for inclusion or exclusion was recorded. For this phase, studies using both standardized (eg, mental chronometry) and idiosyncratic (questionnaires generated and used solely by the study's authors) measures of MI ability were included. Lesion locations were defined as indicating specific brain structures (eg, parietal lobe) rather than general brain regions (eg, right hemisphere). This criterion allowed assessment of damage to specific brain regions on MI ability. In keeping with this aim, we also excluded studies describing patients with neurologic disorders affecting areas outside the brain (ie, spinal cord injury) or neurologic disorders with an underlying pathology beyond specific structures (ie, multiple sclerosis). After phase 2, 50 studies remained.

Phase 3: assessing for suitability and comparability of studies

In this phase, studies describing implicit MI (eg, mental rotation tasks) were discarded given that explicit MI is more relevant to MI-based rehabilitation.[1,16,17] To standardize and therefore permit comparison of MI ability across studies, studies using idiosyncratic MI ability assessments were discarded. Studies that prescreened participants for MI ability (ie, only included participants who could perform MI) were also discarded. Finally, studies describing patients with ≥4 lesion locations were discarded given the difficulty

Table 1 MI Ability Assessment Questionnaire values indicative of MI ability, MI impairment, and MI inability, as calculated by the MI Ability Assessment Scale

Questionnaire	Likert Scale (points)	Total Scale	Total No. of Items	Able	Impaired	Unable
MIQ	7	18–126	18 (9 visual, 9 kinesthetic)	≥95	63–94	≤62
MIQ-R	7	8–56	8	≥42	28–41	≤27
MIQ-RS	7	14–98	14 (7 visual, 7 kinesthetic)	≥74	49–73	≤48
MIQ-RS (kinesthetic only)	7	7–49	7	≥37	25–36	≤24
KVIQ-10	5	10–50	10	≥38	25–37	≤24
KVIQ-20	5	20–100	20	≥75	50–74	≤49
KVIQ	5	34–170	34 (17 visual, 17 kinesthetic)	≥128	85–127	≤84
KVIQ (Heremans et al[37])	5	34–170		≤76	77–119	≥119
VMIQ	5	24–120	24	≤54	55–84	≥85
VMIQ-2	5	12–60	12	≤27	28–42	≥43
MC*				0%–25%	26%–50%	>50%

Abbreviations: MC, mental chronometry; MIQ, Movement Imagery Questionnaire; MIQ-R, Movement Imagery Questionnaire-Revised; MIQ-RS, Movement Imagery Questionnaire-Revised Second Edition; VMIQ, Vividness of Movement Imagery Questionnaire; VMIQ-2, Vividness of Movement Imagery Questionnaire: Revised Edition.

* See Mental chronometry section within Standardizing MI ability.

in ascribing MI ability impairment to a specific lesion location. After these exclusions, 23 studies remained.[3,9,14,21-23,27-43]

Because many systematic reviews derive the level of evidence supporting a given intervention, they include a quality appraisal of the individual studies, which informs the level of evidence assigned. Unlike these reviews, the current study did not seek to assign a level of evidence to any intervention, nor did it investigate the intervention component (if one was present) of the included studies. As such, the items contained in typical quality appraisal instruments do not apply, and an appraisal of study quality was not performed.

Standardizing MI ability

Comparing MI ability across studies is difficult given the numerous methods for assessing MI ability (tables 1 and 2). Furthermore, MI ability assessment scores are not used to ascribe MI ability or lack thereof; rather, they are used for evaluation of statistically significant differences in subjects within studies using the same measure. To facilitate comparison of MI ability across studies, we devised a method for normalizing MI ability assessment scores, termed the MI Ability Assessment Scale. The MI Ability Assessment Scale is helpful for determining ability, impairment, or inability using only the numerical value of an assessment score. As one could imagine, normalizing scores from various MI ability assessments is a challenging task given the different approaches used. With that said, the MI Ability Assessment Scale represents a data-driven approach to normalizing these scores, therefore permitting the review of MI literature en masse. To be clear, the MI Ability Assessment Scale is not an assessment tool for determining MI ability. Instead, it was developed as a means for standardizing differing assessment scores and combining data in systematic reviews. The MI Ability Assessment Scale is subsequently described.

MI Ability Assessment Scale

The MI Ability Assessment Scale uses the following premise: scores <50% correspond to being unable to perform MI (MI unable); scores ≥50% but <75% correspond to being able yet impaired when performing MI (MI impairment); and scores ≥75% correspond to being able to perform MI without impairment (MI able) (fig 1). For methods of MI ability assessment where lower scores indicate better ability, the reverse applies. Assigning 2 levels of ability for subjects who are MI able (ie, MI able with and without impairment) allows for differentiating lesions that are impairing versus those that prevent MI performance altogether. The following sections describe how MI ability assessment scores were measured against the MI Ability Assessment Scale illustrated in figure 1.

Mental chronometry

Although mental chronometry scores are most often presented as the temporal ratio of imagined to executed movement, there are multiple methods for presenting mental chronometry data (table 3). To compare mental chronometry scores and apply them to the MI Ability Assessment Scale (ie, out of 100), all mental chronometry scores were converted to the imagined to executed movement ratio and then into a temporal discrepancy percentage. For example, an imagined to executed movement ratio of .96:1 has a 4% temporal discrepancy (see step 1 in table 3). Table 3 describes the steps taken to convert all mental chronometry data into a temporal discrepancy percentage. Temporal discrepancy percentage scores can then be applied to the MI Ability Assessment Scale previously described (see the bottom of fig 1).

MI questionnaires

The top portion of figure 1 illustrates the scores on the MI questionnaires that represent the threshold scores for MI ability, MI impairment, and MI inability. The 50% score on a Likert scale is

Table 2 Summary of included articles organized by lesion type/neurologic disorder

Lesion Type	Reference	Measure of MI Ability	No. of Subjects
Stroke	Butler et al[3]	VMIQ, MIQ-R, MC	3
	Confalonieri et al[27]	MIQ-RS	11
	Danckert et al[21]	MC	1
	Dunsky et al[28]	MIQ	1
	Gaggioli et al[29]	VMIQ	1
	Guttman et al[30]	MIQ	13
	Jackson et al[9]	KVIQ, MC	1
	Li[31]	FPA	12
	Malouin et al[23]	MC	37
	Schwoebel et al[32]	MC	1
	Shindo et al[33]	BCI	8
	Tam et al[34]	BCI	5
PD	Cunnington et al[35]	FPIS	6
	Dominey et al[36]	MC	7
	Heremans et al[37]	MC, MIQ-R, KVIQ	14
	Peterson et al[38]	KVIQ	28
	Randhawa et al[39]	KVIQ, MIQ	11
	Thobois et al[40]	MC	9
Temporary (TMS)	Fleming et al[41]	FPA	15
Surgical	Grealy et al[42]	MC	1
	Kagerer et al[43]	MC	4
Herpes (n=1) Surgical (n=1)	Sirigu and Duhamel[14]	MC	2
Multiple			
Stroke (n=3) DPS (n=1) Surgical (n=1)	Sirigu et al[22]	MC	5

Abbreviations: BCI, brain-computer interface; DPS, degenerative pyramidal syndrome; FPA, Final Position Assessment; FPIS, Florida Praxis Imagery Scale; MC, mental chronometry; MIQ, Motor Imagery Questionnaire; MIQ-R, Movement Imagery Questionnaire-Revised; MIQ-RS, Movement Imagery Questionnaire-Revised Second Edition; TMS, transcranial magnetic stimulation; VMIQ, Vividness of Movement Imagery Questionnaire.

ambiguous given the absence of a natural zero (ie, a score of 1 is the lowest score) and the ambiguity of the value of each interval (ie, a score of 2 is not necessarily twice the score of 1). Furthermore, ascribing a midpoint on a Likert scale is also confounded by the nature of the questionnaires. Briefly, the scale used for the Kinesthetic and Visual Imagery Questionnaire (KVIQ) is as follows: 5 (image as clear as seeing); 4 (clear image); 3 (moderately clear image); 2 (blurry image); and 1 (no image). Although a low score on the KVIQ (eg, a score of 2) indicates a blurry image, it does not indicate that the participant did not successfully imagine

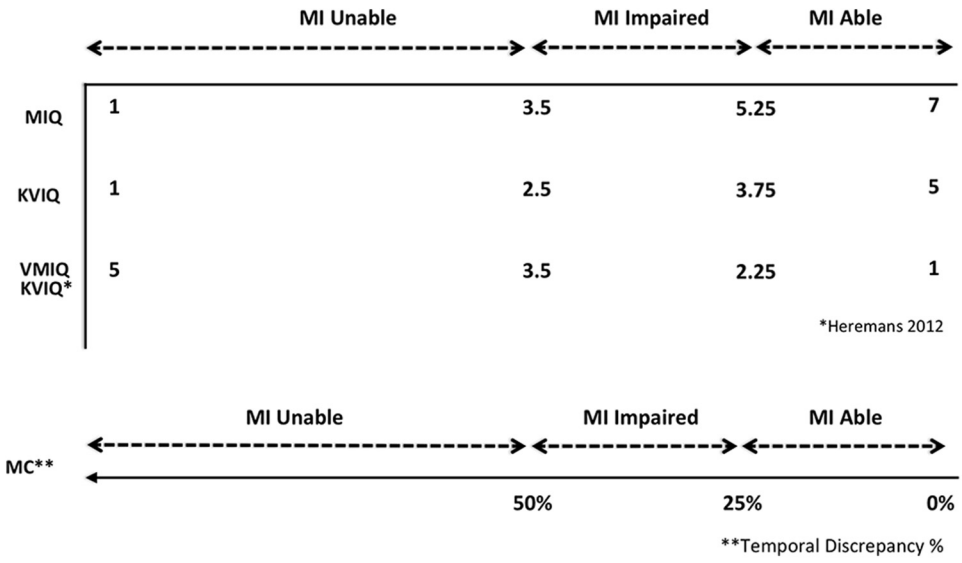

Fig 1 Visual depiction of the MI Ability Assessment Scale. The top portion shows MI ability, inability, and impairment on a Likert scale of self-reported questionnaires. The bottom portion shows MI ability, inability, and impairment on temporal discrepancy scores from MC data. Abbreviations: MC, mental chronometry; MIQ, Motor Imagery Questionnaire; VMIQ, Vividness of Movement Imagery Questionnaire. * Heremans et al.[37] ** Temporal discrepancy %.

Table 3 Protocol for calculating temporal discrepancy using differentially presented MC data

Step	Data	Rule	Example
1	I/E ratio given	Calculate percent discrepancy by using the absolute value of the I/E ratio subtracted by 1, then multiply by 100	I/E ratio: .96 → |0.96−1|x100=4%
2	Multiple I/E ratios given for 1 subject	Calculate mean I/E ratio. Proceed to step 1.	I/E ratios: 1.21; .88 → |1.21−1|+|0.88−1|=.33
3	Multiple I/E times given for 1 subject	Convert each set of I/E times to a ratio. Proceed to step 2.	I/E times (s): 15/18; 14/19 → .83; .74
4	I/E times given separately	Convert to I/E ratio by dividing imagined movement duration by executed movement duration. Proceed to step 1.	Imagined movement: 15s Executed movement: 12s → I/E=15/12=1.25
5	I/E ratios given for multiple conditions	Proceed to step 2.	Pre- and post-MI rehabilitation; right- and left-limb I/E ratios

Abbreviations: I/E, imagined movement/executed movement; MC, mental chronometry.

the movement despite having low vividness. Similarly, a score of 1 on the Motor Imagery Questionnaire rates MI as difficult to imagine, which again does not imply that the participant was unable to imagine the movement. These stipulations pose a difficulty in translating Likert scale scores to a score out of 100 on the MI Ability Assessment Scale.

To support the MI Ability Assessment Scale for assigning ability to questionnaire scores (see fig 1) despite the difficulty posed by the nature of these questionnaires, we compared our values to unpublished KVIQ data collected from nondisabled participants from 2 studies in our laboratory. This comparison capitalizes on the assumption that nondisabled individuals are able to perform MI, and therefore their KVIQ scores indicate approximate values for MI ability. As such, KVIQ data of healthy subjects was used in the development of the MI Ability Assessment Scale. Figures were calculated by averaging both the kinesthetic and visual subscales across all sessions (ie, baseline KVIQ assessment, session 1). We averaged both the visual and kinesthetic subscales because KVIQ scores are typically presented as one averaged value. In the first study (N=18), the average KVIQ score was 3.84 out of 5, whereas the second study (N=15) reported an average of 3.75 out of 5 (Gionfriddo, unpublished data, May 2014; Boe & Bardouille, unpublished data, August 2014). These scores are well in line with the MI Ability Assessment Scale illustrated in figure 1 in that the average KVIQ score for nondisabled participants fell into the MI able category.

Other assessments

We identified 3 additional MI ability assessments other than mental chronometry and questionnaire-based approaches. These include brain-computer interface and MI adherence assessments (Florida Praxis Imagery Scale and Final Position Assessment). These methods, unlike mental chronometry and questionnaires, did not require standardizing using our protocol. Completion of a task using brain-computer interface is only possible if the participant successfully performs MI. The individual scores for the participants in the studies using the Florida Praxis Imagery Scale were not reported; however, it was noted that each individual scored very well. Finally, the Final Position Assessment is a pass/fail assessment. Participants for which assessment scores were provided as pass/fail were considered MI able given their passing score on the assessment.

Results

Study selection

Figure 2 summarizes how studies were selected for inclusion in this systematic review. The 23 studies comprised 196 participants, including 97 patients poststroke, 75 patients with PD, 7 surgical patients, 1 patient with herpes encephalitis, 1 patient with degenerative pyramidal syndrome, and 15 healthy subjects in whom temporary lesions were created using transcranial magnetic stimulation (see table 2). The 23 studies included 8 different methods for assessing MI ability, including subjective questionnaires, mental chronometry, brain-computer interface, and MI adherence assessments (see table 2). A complete review of MI assessment methods has previously been described.[44]

MI ability and lesion location

MI ability in patients with stroke and non−PD-derived lesions

Table 4 provides an overview of MI ability broken down by lesion location in each of the 97 patients poststroke and 24 patients with lesion types other than stroke and PD. Table 4 shows that patients with 1 lesion location, who have damage to either the parietal lobe or the basal ganglia, demonstrate an increased likelihood of compromised MI (ie, either MI impairment or MI inability). In contrast, patients with 1 lesion location and damage to either the cerebellum or subcortical structures other than the basal ganglia are less likely to be impaired. There were 21 patients whose strokes were reported as arterial supply rather than affected (lesioned) brain region. Given the large cortical area supplied by the major arteries in the brain (eg, middle cerebral artery), combined with the individual variance in collateral blood supply, the neural damage after occlusion of these arteries is not consistent across patients. As such, we were unable to further examine the effect of lesions resulting from these occlusions on MI ability. These 21 patients are included in table 4 for reporting purposes, but they were excluded from further analysis.

Table 5 reports the MI abilities in both patients with stroke and without stroke with any number of lesion locations and that have damage to either the parietal or frontal lobes. Table 5 also reports specificity within the structures (ie, posterior frontal lobe vs frontal lobe) when available. Although table 5 presents a confound—MI ability is reported without consideration of multiple

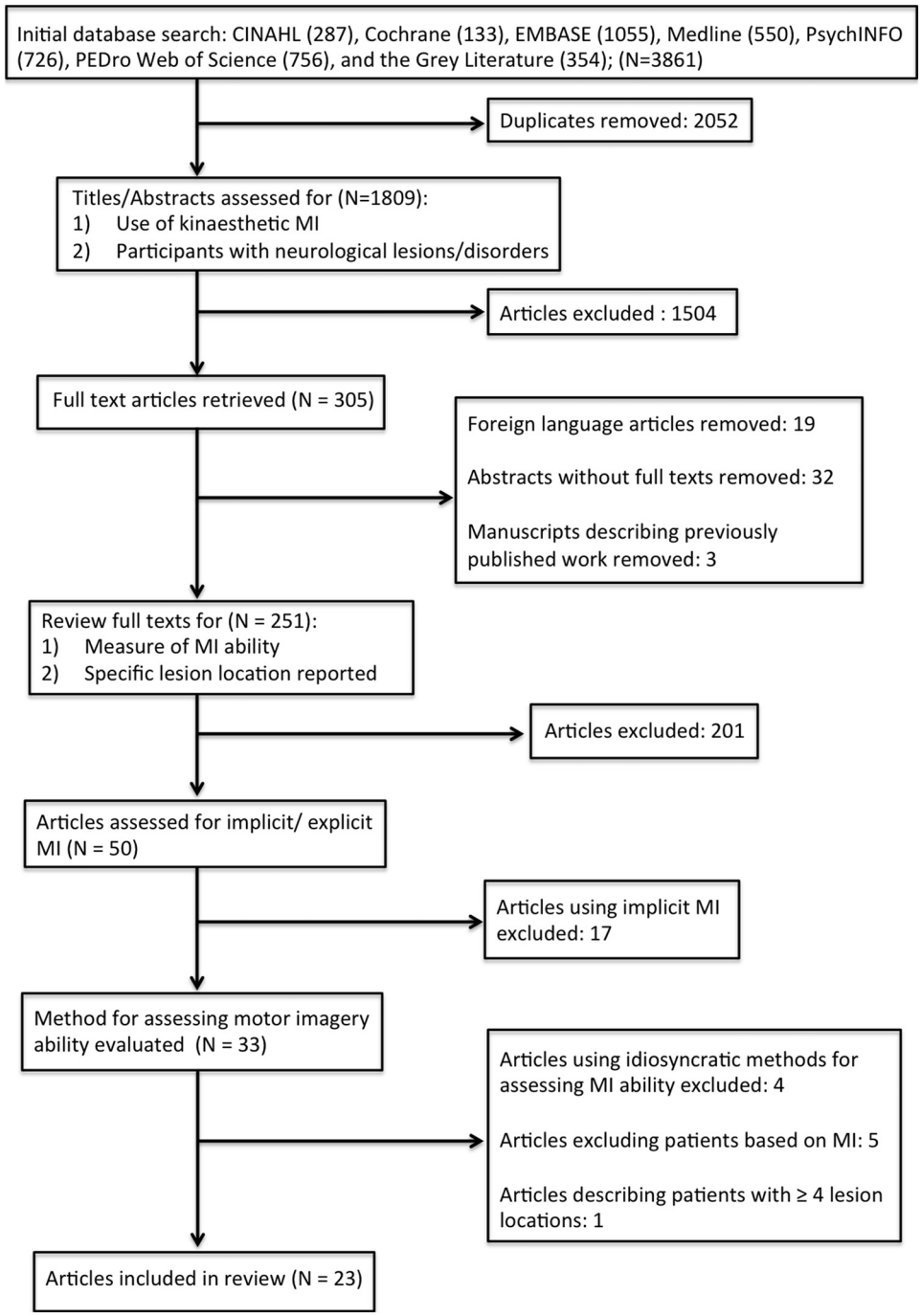

Fig 2 Flowchart summarizing the number of articles included and excluded in each stage of article selection. Abbreviation: PEDro, Physiotherapy Evidence Database.

lesion locations—it is a better account of the number of patients with lesions in each location. The results presented in table 5 (and visually depicted in fig 3) further confirm our initial finding that parietal lobe damage severely impairs MI ability. Figure 3 shows how parietal lobe damage not only impairs MI ability (middle column), but also that it is responsible for the highest percentage of participants who are unable to perform MI. Our results also indicate that frontal lobe damage impairs MI ability (see table 5 and fig 3). Interestingly, the impairment does not appear to stem from the primary motor cortex (M1) or posterior frontal lobe lesions (see table 5). That is, 6 of the 10 patients with M1/posterior frontal lobe lesions were able to perform MI, whereas only 4 were impaired, and none were unable to perform MI. Moreover, when taking into consideration only those with lesions solely in the M1/posterior frontal lobe, all 3 patients were able to perform MI without impairment. Unfortunately, the results were void of participants with damage exclusively to the anterior frontal lobe. The limited number of patients with damage exclusively to individual frontal lobe subregions (ie, M1, premotor cortex, prefrontal lobe) also prevented further analysis.

Table 4 MI ability in patients with stroke- and nonstroke-derived lesions categorized into number of lesions and lesion location

Number and Lesion Location	Able	Impaired	Unable	Total
Stroke: 1 location				
Frontal	2	2		4
Parietal	2		5	7
Cerebellum	1			1
B.G.	8	10	1	19
Thal.	5	1	1	7
I.C.	6	1		7
S.C.	1			1
Pons/medulla	1	1		2
Subcortical	2			2
Total				50
Stroke: 1 location (arterial supply)				
MCA territory	7	4	6	17
Vertebrobasilar artery	2			2
Subarachnoid space	2			2
Total				21
Stroke: 2 locations				
Frontoparietal	1	4		5
Frontotemporal	1			1
Temporal, B.G.			1	1
Parietotemporal		1		1
Temporooccipital		1		1
Frontal, B.G.	2	4		6
EC, B.G.	1			1
Parietal, C.R.	1			1
Thal., C.R.	1			1
C.P., thalamus		1		1
MCA territory, Thal.	1			1
I.C., C.R.	1	1		2
Claustrum, E.C.		1		1
Total				23
Stroke: 3 locations				
Frontotemporal, B.G.			1	1
Frontoparietal, B.G.	1			1
I.C., B.G., C.R.		1		1
Total				3
Nonstroke lesions				
Frontal	1 (DPS)			1
Parietal		15 (TMS)	2 (surgical)	17
Cerebellum	3 (surgical)	1 (surgical)	1 (surgical)	5
Temporal	1 (herpes)			1
Total				24

NOTE. Values are n for number of participants.
Abbreviations: B.G., basal ganglia; C.P., cerebral peduncles; C.R., corona radiata; DPS, degenerative pyramidal syndrome; E.C., external capsule; I.C., internal capsule; MCA, middle cerebral artery; S.C., semioval center; Thal, thalamus; TMS, transcranial magnetic stimulation.

MI ability in patients with both PD-derived and non–PD-derived basal ganglia damage

Table 6 reports MI ability in patients with basal ganglia damage from both PD-derived and non–PD-derived pathologies. With similar organization to table 5, table 6 presents MI abilities for patients with any number of lesion locations, and locations are further specified within the basal ganglia (eg, caudate nucleus). Table 6 and figure 3 show that basal ganglia damage is more likely to impair rather than prevent MI ability. Damage to the right or left basal ganglia does not appear to be more impairing (see table 6). In non-PD patients, damage to the putamen appears to be driving the impairment of MI performance in that 13 of the 17 patients with compromised MI have putamen damage (see table 6).

Table 6 also shows that most patients with PD are able to perform MI without impairment. Specifically, of the 75 patients with PD, 66 were able to perform MI without impairment. Our results do not appear to show any relation between ability to perform MI and PD disease severity, as assessed using the Hoehn and Yahr[45] scale for PD severity (see table 6) and the Unified Parkinson Disease Rating Scale[45] (data not shown). These results are limited because only 3 of the 7 studies with patients with PD provided the Hoehn and Yahr/Unified Parkinson Disease Rating Scale scores. In most cases, participants with PD with Hoehn and

Table 5 MI ability in patients with ≥1 lesion location, including the parietal or frontal lobes*

Lesion Location	Able	Impaired	Unable	Total
Parietal	5	20	7	32
Bilateral/unspecified	1	15	3	19
Right	1	3	2	6
Left	3	2	2	7
Frontal	7	10	1	18
Bilateral/unspecified	1	4	0	5
Right	2	3	0	5
Left	4	3	1	8
Posterior frontal/M1	6	4	0	10
Posterior frontal/M1 only	3	0	0	3

NOTE. Values are n for number of participants.
* Precise lesion locations reported when available.

Yahr/Unified Parkinson Disease Rating Scale scores above a threshold were excluded from the studies. Nonetheless, our results indicate good MI ability in patients with PD.

Discussion

In keeping with our aim to investigate the impact of brain damage on MI ability, we identified 3 structures that when damaged impair MI ability: the parietal lobe, frontal lobe, and basal ganglia. Specifically, we show MI ability is greatly impacted by parietal lobe damage and moderately impacted by frontal lobe damage, albeit outside the posterior region (see fig 3). Furthermore, we show that damage to the basal ganglia, specifically the putamen, impairs MI ability in patients with non−PD-derived pathology. Although previous studies have demonstrated impaired MI ability for certain brain lesions, this systematic review adds comprehensive data to our current understanding of the impact of brain damage on MI ability.

Parietal lobe

The results clearly show that parietal damage impairs MI ability. Of the 32 participants with parietal lobe lesions, 17 were MI compromised. This is unsurprising given the parietal lobe's critical role in producing mental images.[5,22,46] The parietal lobe is thought to coordinate premotor areas with the dorsal stream such that intended movements are appropriately carried out in the individual's surroundings.[47] The parietal lobe is also thought to aid in the inhibition of the M1 during MI, albeit indirectly through its connections with the frontal lobe, specifically, the supplementary motor area.[32,48,49] In support of this, Schwoebel et al[32] describe a patient with parietal lobe damage whose MI was unconsciously accompanied by ME of the imagined task. They suggest that their patient's unintended movements during MI are attributed to the loss of parietal lobe inhibition on the M1. Given the parietal lobe's

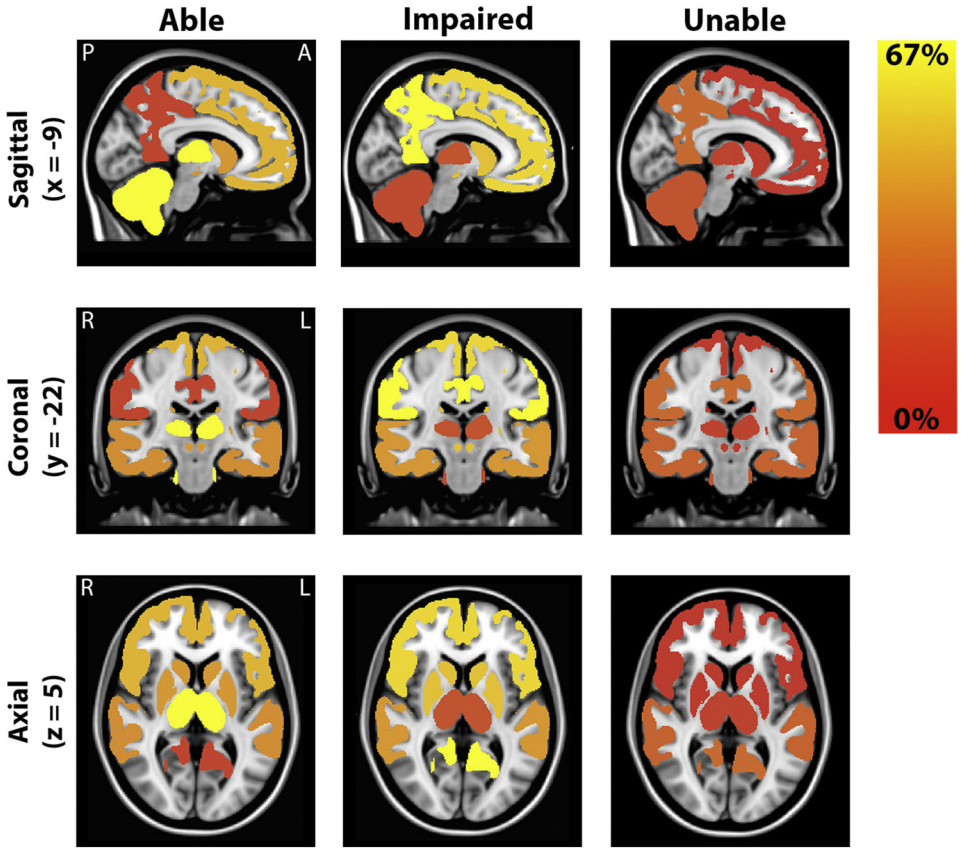

Fig 3 Motor imagery ability, impairment, and inability in 6 brain regions (parietal lobe, frontal lobe, basal ganglia, cerebellum, temporal lobe, thalamus) in both patients with stroke and nonstroke/non-PD patients. The red-yellow color bar represents the percentage of participants able to perform MI (left column), impaired when performing MI (middle column), and unable to perform MI (right column). Abbreviations: A, anterior; L, left; P, posterior; R, right.

Table 6 MI ability in patients with both PD-derived and non–PD-derived basal ganglia damage*

Clinical Characteristics	Able	Impaired	Unable	Total
B.G. laterality (non-PD)				
Bilateral/unspecified	3	0	0	3
Right	4	7	1	12
Left	6	8	1	15
Total	13	15	2	30
B.G. structure (non-PD)				
Lenticular nucleus†			1	1
Putamen	2	13	1	16
Globus pallidus	1			1
Caudate	1	0	0	1
Unspecified	9	2	0	11
Total	13	15	2	30
PD total	66	5	4	75
Disease severity (PD)				
H&Y stage 1	8	2	1	11
H&Y stage 1.5	2	1		3
H&Y stage 2	4	1	1	6
H&Y stage 2.5	1			1
H&Y stage 3	2	1	1	4

NOTE. Values are n for number of participants.
Abbreviations: B.G., basal ganglia; H&Y, Hoehn and Yahr scale for PD severity.
* Precise lesion locations reported when available.
† Putamen and globus pallidus.

role in guiding motor activity in relation to spatial information (ie, dorsal stream functions), we further suggest that the patient is experiencing interference of frontoparietal communication, which is necessary for planning and coordinating MI. Impairment of MI after parietal damage may be attributed to the loss of any of its functions during MI, including its role in facilitating the planning and coordination of imagined movements and/or its ability to indirectly inhibit the M1.[5,22,46]

Frontal lobe

The results also show that frontal lobe damage impairs MI ability. The frontal lobe, notably the prefrontal and premotor areas, play an important role during MI.[5,46,49] The prefrontal region is involved in generating attention during MI tasks, and the premotor cortex is involved in generating a motor plan.[5] Of the 18 participants with frontal lobe damage, 10 were impaired, and only 1 was unable to perform MI. This finding suggests that frontal lobe damage impairs rather than prevents MI ability. Moreover, this finding distinguishes between regions that are either critical to, or helpful for the production of mental motor images. The implications for rehabilitation are that patients with parietal lobe lesions may struggle with MI-based therapy, whereas patients with frontal lobe lesions may benefit from MI-based therapy. Unfortunately, given the scarcity of studies that control for (or report) MI ability, it remains to be investigated the extent MI ability impairment lessens its beneficial effects in rehabilitation. Specifically, the lack of sensitivity of MI assessment tools precludes investigation of the degree of MI impairment (as opposed to inability) on the effectiveness of MI for rehabilitation. Future work using metrics based on objective measures of MI ability (eg, brain activity) may be useful in addressing this gap in the literature.

Our results also suggest that damage to the posterior frontal lobe (including the M1) does not appear to impair MI ability (see table 5 and figure 3). This finding is particularly interesting given the disagreement in the literature regarding the involvement of the M1 during MI.[27,50] Briefly, it has yet to be elucidated whether the M1 is or is not involved in MI or if it is necessarily inhibited to imagine without execution.[51] It is possible that the absence of MI impairment after M1 damage supports the hypothesis that the M1 is not involved in MI. It is also possible that M1 damage negates the necessity for the inhibition of the M1 that is potentially necessary for MI. However, our results did not include participants with damage specifically to the prefrontal region. It is therefore possible that the 11 MI-compromised participants whose lesion locations were unspecified beyond frontal lobe had posterior frontal lobe damage.

Basal ganglia

Non–PD-derived basal ganglia lesions were also shown to impair MI performance (see fig 3). Of the 19 patients with lesions strictly to the basal ganglia, 10 were impaired, and 1 was unable to perform MI (see table 6). Of the 30 total participants with basal ganglia lesions, only 13 were able to perform MI without impairment (see table 6). Similar to frontal lobe damage, basal ganglia damage appears to impair rather than prevent MI ability. Only 2 of the 30 patients with basal ganglia damage were unable to perform MI, whereas 15 were able yet impaired (see table 6).

Remarkably, of the 17 MI-compromised patients whose lesion locations within the basal ganglia were specified, 14 had damage to the putamen (see table 6). This finding suggests that damage to the putamen drives the impairment in MI after basal ganglia damage. Given this finding, it is surprising that only 9 of the 75 patients with PD were MI compromised. There are several possible explanations for this finding. First, the etiology of PD—the selective death of dopaminergic cells originating in the substantia nigra—does not parallel basal ganglia damage caused by stroke. Unfortunately, our sample was void of non-PD patients with lesions specifically to the substantia nigra; this data would have helped determine whether substantia nigra damage consistently spares MI ability. Another explanation for the spared MI ability is the homogeneity of disease severity in our cohort of patients with PD. Although we did not see any impact of PD severity on MI ability (see table 6), our PD population was limited to patients with low disease severity. Of the 6 reported studies with patients with PD, only 3 reported individual data on disease severity and MI ability. Furthermore, all 6 studies had at least 1 exclusion criterion that may have influenced our results. Specifically, 4 studies excluded patients with severe PD (Hoehn and Yahr stage>3); 2 studies excluded patients with dyskinesia and/or severe tremor; and 3 studies excluded patients with other neurologic disorders or cognitive dysfunction. PD severity is connected to comorbidity with other neurologic disorders and cognitive dysfunction (eg, dementia[52]). Consequently, the latter exclusion criterion indirectly excluded patients with severe PD.

Given the limitations described, our results obtained from studies of patients with PD may not be an accurate account of the impairment in MI ability associated with PD. Moreover, given the impairment of MI in non–PD-derived putamen damage, and the putamen's reliance on dopaminergic transmission, it is likely that patients with PD may experience MI impairment as their disease progresses. The lack of patients across the spectrum of PD severity prevented further analysis.

Other brain regions

Although our results yielded only 1 or 2 patients with lesions in other brain regions, there were enough participants with lesions to the cerebellum, internal capsule, and thalamus to be discussed. Six patients had damage exclusively to the cerebellum: 5 lesions that resulted from surgery and 1 from stroke (see table 4). Of these 6 patients, 4 were able to perform MI without impairment. This finding is surprising given the substantiated role of the cerebellum during MI.[16,46,53,54] It is possible, however, that the participants with cerebellar damage were able to perform MI because of the nature of the MI task. The cerebellum has been shown to have the greatest activation during the initial learning stage of a novel task and much less activation during performance of a learned skill.[55] Consequently, patients with cerebellar damage are less likely to have compromised MI ability when assessed using a learned rather than novel task. The small number of patients with cerebellar damage, however, limits our ability to confirm whether or not cerebellar lesions impair MI ability.

Our results also included 7 subjects each with damage solely to the thalamus and internal capsule (see table 4). Of the 7 with thalamic damage, 5 could perform MI without impairment. Similarly, 6 of the 7 subjects with damage to the internal capsule were able to perform MI without impairment. Given that information is relayed between subcortical and cortical regions via the internal capsule and that the cortex is primarily responsible for generating MI, it is not surprising that patients with internal capsule damage were still able to perform MI. In keeping with this statement, it is also not surprising that thalamic damage did not impair MI because the thalamus is primarily involved in relaying sensory information to the cortex.

Study limitations

The primary limitation of this review stems from the absence of MI ability assessments in most of the literature. Only 50 of 251 studies assessed MI ability and reported specific lesion locations (see fig 2). Studies that fulfilled only one of these criteria were excluded. Furthermore, the methodologic shortcomings of assessing for MI ability present a second limitation. Specifically, numerical scores on standardized assessments are not indicative of MI ability versus disability. Although we overcame this shortcoming by developing the MI Ability Assessment Scale, our system does not circumvent all the shortcomings. For example, studies that implement multiple assessments of MI ability demonstrate how participants can show good MI ability on one assessment and poor ability on another. Furthermore, MI ability assessments are not consistently given to participants either before or after MI practice. Our results, therefore, represent a medley of MI ability scores assessed in both novice and practiced imagers. Because MI is not a common skill, the ability to perform MI will vary between individuals who have never engaged in the skill versus athletes, for example, who engage in MI during training.

A third limitation of this study lies in our interpretation of MI ability in participants with multiple lesion locations. A trade-off exists between including a greater number of participants (ie, including those with multiple lesion locations) and reporting precise results that allow MI ability/impairment to be equated with specific lesion locations. For example, 5 patients in this study had frontotemporal lesions: 1 of which was able to perform MI without impairment, whereas the other 4 were impaired on their MI ability (see fig 3). It is possible that although only a single lesion location contributed to the impaired MI performance, we are unable to tease out this information.

Despite the limitations, this study adds new information relating the ability to perform MI with brain lesion location. Knowledge of this relation is important because MI use in rehabilitation is increasing, and clinicians need to be aware of possible limitations in performing MI-based therapy to both prescribe it appropriately and assess its effectiveness. Prospectively collecting data relating MI ability to lesion location would address several (but not all) of the limitations noted; with that said, such a study would have limitations. For instance, although including patients with a single identified lesion aids in relating MI impairment to the lesion, it would also reduce external validity because patients undergoing rehabilitation rarely have single identifiable lesions; rather, they present similar to those patients included in the current review.

Conclusions

Although previous reviews have explored which regions of the brain are involved in MI,[5,27] we have shown which regions, when damaged, impair or prevent MI performance. Our results suggest that parietal lobe damage prevents MI ability, whereas frontal lobe and basal ganglia damage impair MI ability. Although the patients with PD and cerebellar damage in our study show unimpaired MI ability, this finding should be interpreted in the context of methodologic limitations. The aforementioned conclusions should be considered in light of the fact that lesion location is not the sole factor that determines MI ability. Indeed, previous studies have identified age, experience with MI, clinician guidance, and working memory capacity as important factors influencing the ability to perform MI and therefore its effectiveness as a neurorehabilitative tool.[56,57] Nevertheless, our findings address the importance of assessing MI ability and considering lesion location before implementing or studying MI-based therapy in clinical populations. The lack of control for MI ability in previous work may be an alternative explanation for the negligible impact of MI-based treatment previously observed.[19,20]

Keywords

Imagery (psychotherapy); Nervous system diseases; Parkinson disease; Rehabilitation; Stroke

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Supplemental Appendix S1 Representative Search Strategy

Search strategy for CINAHL

(Concept 1 AND Concept 2) NOT (MH "Child+" NOT MH "Adult+")

287 results; May 14, 2014

Concept 1

(MH "Guided Imagery")

TI imagery N2 psychotherap* OR AB imagery N2 psychotherap*

TI (imag* N3 (motor* OR mental* OR movement*)) OR AB (imag* N3 (motor* OR mental* OR movement*))

TI guided N2 imagery OR AB guided N2 imagery

TI "mental practice" OR AB "mental practice"

TI "mental training" OR AB "mental training"

Concept 2

(MH "Stroke+")

(MH "Brain Diseases+")

(MH "Brain Injuries+")

(MH "Neurodegenerative Diseases+")

TI lesion* OR AB lesion*

TI stroke* OR AB stroke*

TI brain N2 infarct* OR AB brain N2 infarct*

TI (lacunar OR (brainstem OR brain stem) N4 (stroke* OR infarct* OR syndrome*)) OR AB (lacunar OR (brainstem OR brain stem) N4 (stroke* OR infarct* OR syndrome*)) OR (brainstem OR brain stem)) N4 (stroke* OR infarct* OR syndrome*)

TI apoplexy OR AB apoplexy

TI CVA OR AB CVA

TI ((cerebrovascular OR cerebral OR vascular) N3 (accident* OR apoplexy OR stroke*)) OR AB ((cerebrovascular OR cerebral OR vascular) N3 (accident* OR apoplexy OR stroke*))

TI stroke* N3 acute OR AB stroke* N3 acute

TI ((brain OR cereb* OR cortical) N3 (disease* OR abscess* OR traum* OR wound* OR fracture* OR injur* OR contusion* OR damage* OR neoplasm* OR cancer* OR tum?r* OR maligna* OR lacerat* OR concuss* OR hemorrhage*)) OR AB ((brain OR cereb* OR cortical) N3 (disease* OR abscess* OR traum* OR wound* OR fracture* OR injur* OR contusion* OR damage* OR neoplasm* OR cancer* OR tum?r* OR maligna* OR lacerat* OR concuss* OR hemorrhage*))

TI ((neurodegenera* OR neurologic*) N3 (disease* OR disorder*)) OR AB ((neurodegenera* OR neurologic*) N3 (disease* OR disorder*))

TI parkinson's OR AB parkinson's

TI locked-in syndrome OR AB locked-in syndrome