1

2

Can motor imagery and hypnotic susceptibility explain Conversion Disorder with motor symptoms?

5

6

7 Alexander J. Srzich[1,2], Winston D. Byblow[1,2], James W. Stinear[1,2], John Cirillo[1,2], J. Greg Anson[1,2]
8
9
10
11
12
13 [1]Movement Neuroscience Laboratory, Department of Exercise Sciences, the University of Auckland,
14 New Zealand, [2]Centre for Brain Research, the University of Auckland, New Zealand
15
16
17
18
19 Section and Senior Editor:
20 Abbreviated title: Motor imagery, hypnotic susceptibility, and Conversion Disorder
21 Number of figures: 1
22 Number of tables: 1
23 Number of pages: 46
24
25
28
29
30
31
32 Correspondence to: Greg Anson, Department of Exercise Sciences, the University of Auckland,
33 Private Bag 92019, Auckland 1142, New Zealand. Email: g.anson@auckland.ac.nz
34
35

1 Abstract

2

3 Marked distortions in sense of agency can be induced by hypnosis in susceptible individuals,

4 including alterations in subjective awareness of movement initiation and control. These

5 distortions, with associated disability, are similar to those experienced with Conversion

6 Disorder (CD), an observation that has led to the hypothesis that hypnosis and CD share

7 causal mechanisms. The purpose of this review is to explore the relationships among motor

8 imagery (MI), hypnotic susceptibility, and CD, then to propose how MI ability may

9 contribute to hypnotic responding and CD. Studies employing subjective assessments of

10 mental imagery have found little association between imagery abilities and hypnotic

11 susceptibility. A positive association between imagery abilities and hypnotic susceptibility

12 becomes apparent when objective measures of imagery ability are employed. A candidate

13 mechanism to explain motor responses during hypnosis is kinaesthetic MI, which engages a

14 strategy that involves proprioception or the "feel" of movement when no movement occurs.

15 Motor suppression imagery (MSI), a strategy involving inhibition of movement, may provide

16 an alternate objective measurable phenomenon that underlies both hypnotic susceptibility and

17 CD. Evidence to date supports the idea that there may be a positive association between

18 kinaesthetic MI ability and hypnotic susceptibility. Additional evidence supports a positive

19 association between hypnotic susceptibility and CD. Disturbances in kinaesthetic MI

20 performance in CD patients indicate that MI mechanisms may also underlie CD symptoms.

21 Further investigation of the above relationships is warranted to explain these phenomena, and

22 establish theoretical explanations underlying sense of agency.

23

1. Introduction

Disturbances in sense of agency, e.g., the sense that "I" am the one causing or generating an action (Gallagher, 2000), are a key feature of hypnosis. Susceptible individuals report marked changes in subjective experience when hypnotised. They perceive a suggested stimulus when there is no stimulus present, i.e., hallucinate. Conversely, they will not perceive a stimulus that is present after it has been suggested there is nothing there. Individuals will also carry out a suggested movement and experience it as involuntary, or will be unable to move after it has been suggested that they are paralysed. These experiences do not appear to be feigned and indicate that the sense of agency of an hypnotised individual has been disturbed. Unobserved hypnotised participants continue to respond to hypnotic suggestions, whereas participants instructed to simulate being hypnotised do not (Kirsch, et al., 1989). Patterns of cortical blood flow during attempted movement of the left leg were different for intentionally feigned compared with hypnotically induced left leg paralysis (Ward, et al., 2003). Electrodermal skin conductance responses recorded to assess the veracity of reported involuntary arm movements indicate that participants truthfully reported their experiences (Zamansky & Ruehle, 1995). These findings support the authenticity of the reported changes in subjective experience and the associated alterations in sense of agency.

A disturbance in sense of agency is a key feature of Conversion Disorder (CD). According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V), CD is characterised by one or more symptoms of altered voluntary motor or sensory function (American Psychiatric Association, 2013). The DSM-V further specifies that clinical

findings provide evidence of incompatibility between the symptom and recognised

neurological or medical condition, and that the symptom or deficit is not better explained by

another medical or mental disorder. The symptom or deficit must also cause clinically

significant distress or impairment in social, occupational, or other important areas of

functioning or warrant medical evaluation. The range of motor and sensory signs and

symptoms include motor weakness or paralysis, abnormal movement, swallowing symptoms,

speech symptoms, attacks or seizures, anaesthesia or sensory loss, or mixed symptoms. CD

is a chronic and debilitating disorder (Gelauff, et al., 2014). Maladaptive personality types

and a history of childhood abuse may be associated with a diagnosis of CD (American

Psychiatric Association, 2013). Psychological trauma is often associated with the onset of

CD, but this is not always necessary and not required to diagnose the disorder (Stone, et al.,

2011). CD symptoms historically have been ascribed to "hysteria". Recently other terms

including functional, psychogenic, and medically unexplained have been ascribed to CD.

This range of terms is in large part a consequence of the etiological and subsequent

nosological uncertainty associated with CD and related symptoms (Edwards, et al., 2014;

Fahn & Olanow, 2014). It is presumed that the symptoms are not intentionally produced or

feigned. For example, patients suffering from a CD tremor are not voluntarily producing this

tremor, and patients suffering from CD paralysis are not able to voluntarily move the affected

limb, despite the absence of any neurological lesion. The corollary is that subjective

awareness to control or initiate specific movement is impaired in CD patients, indicative of a

marked alteration in the sense of agency.

CD and hypnosis have been linked for many years. Hysteria and hypnosis have been studied

together since the mid-1800s when it was recognised that hypnosis could induce and treat

symptoms of hysteria (Bramwell, 1906). Early psychoanalysts, including Janet and Freud, used hypnosis to reveal "forgotten" early life distressing or disturbing experiences in their patients with hysteria that were thought to be responsible for the symptoms (Bliss, 1984). Furthermore, self-hypnosis has been considered as the "prime mechanism" in many patients with hysteria (Bliss, 1984). CD symptoms and similar states induced with hypnotic suggestion, including the subjective experience of involuntariness, are phenomenologically similar. Dissociative mechanisms, in which relatively circumscribed aspects of cognition become separated from normal subjective awareness or voluntary control, have been proposed to underlie symptoms of CD and responses to suggestions given under hypnosis with a body of research supporting a link between the two (Bell, et al., 2011; Oakley, 1999). Despite the apparent link, few objective studies have examined mechanisms likely shared between CD and hypnosis.

Kinaesthetic motor imagery (MI) ability may underlie the relationship between hypnosis and CD with motor symptoms (motor CD). Kinaesthetic MI involves the mental simulation of an action by using a strategy that results in the proprioceptive consequences of the action being felt in the absence of any overt movement (Jeannerod, 1994). Hypnosis involves imagining some state of affairs and then experiencing that state. It is reasonable to suspect there may be a relationship between one's imagery ability and their susceptibility to hypnosis.

How might hypnotic susceptibility, CD and kinaesthetic MI be related? First, studies that have investigated the relationship between susceptibility to hypnosis and mental imagery ability will be discussed. Second, mechanisms underlying kinaesthetic MI will be discussed

1 and related to studies of mental imagery ability, imagery related movement suppression and

2 hypnotic susceptibility. Imagery related mechanisms of hypnotic responding will then be

3 examined in the context of current explanatory theories of hypnosis. Studies investigating

4 links between CD and hypnosis, and MI in CD will be reviewed. Finally, a hypothetical role

5 of kinaesthetic MI mechanisms in CD with motor symptoms and hypnotic susceptibility will

6 be proposed to explain the relationship between these three entities.

7

8 2.   Hypnotic susceptibility and imagery abilities

9

10 Hypnotic susceptibility, the responsiveness of a person to suggestions while hypnotised, can

11 be assessed by administering a hypnotic induction and then a predetermined set of motor or

12 cognitive/perceptual suggestions. The number of items responded to either behaviourally or

13 subjectively is summed to form a susceptibility score. Many hypnotic rating scales have been

14 developed with various clinical and research purposes in mind. Research using these scales

15 has shown that individuals vary in their susceptibility to hypnosis (Barnier & McConkey,

16 2004), and their susceptibility remains stable over time (Piccione, et al., 1989). Despite these

17 findings, very few robust correlates of hypnotic susceptibility have been identified. Those

18 that have been identified account for only a small proportion of variability observed in the

19 general population (Kirsch & Council, 1992). One of the earliest identified and best

20 predictors of hypnotic susceptibility is the response of an individual to the same suggestions

21 outside of hypnosis (Barber & Glass, 1962; Braffman & Kirsch, 1999; Hilgard & Tart, 1966;

22 Weitzenhoffer & Sjoberg, 1961). This finding indicates some intrinsic ability to respond to

23 suggestions in general contributes to hypnotic susceptibility.

1

2 Suggestions to imagine and then experience a particular state of affairs are a key feature of

3 hypnosis. As noted by Kirsch (1997), hypnotic suggestions involve the "request that the

4 person imagine a counterfactual state of affairs…and experience the world and the self as if

5 this state of affairs were true". For example, in Item 2 of the Stanford Hypnotic

6 Susceptibility Scale: Form C (SHSS:C), the hypnotised participant is asked to extend their

7 arms ahead of themselves with their palms facing each other close together. They are then

8 asked "to imagine a force acting on your hands to push them apart, as though one hand were

9 repelling the other. You are thinking of your hands being forced apart and they begin to

10 move apart… separating…" (Weitzenhoffer & Hilgard, 1962). The requirement that the

11 hypnotised person imagine the suggested state before that state is experienced indicates that

12 imagery may play a critical role in the experience of hypnotic suggestions.

13

14 The idea that imagination has a role in the generation of hypnotic experiences is not new.

15 The "Report of the commissioners charged by the king with the examination of animal

16 magnetism", published in 1784, was initiated by the French Academy of Science and the

17 Paris Faculty of Medicine to investigate the claims of Franz Mesmer regarding animal

18 magnetism and its use in treating various conditions (Franklin, et al., 2002). No evidence of

19 "animal magnetism" was identified by the report, which concluded that imagination played a

20 major role in hypnotic responses. Since this hypothetical role of imagery in hypnotic

21 responding was first put forward, many studies have attempted to elaborate the nature of the

22 relationship between imagery and hypnosis.

23

1   2.1.   Hypnotic susceptibility and subjective measures of imagery

2   [Place Table 1 here]

3   Self-rating scales of imagery ability permit an investigation of the relationship between

4   imaginative abilities and hypnotic susceptibility. Participants rate their ability to imagine

5   particular situations in a specified sensory modality.

6   Insert Table 1 about here

7   Table 1 summarises the findings of studies that used self-rating scales of imagery ability to

8   assess the relationship between imagery abilities and hypnotic susceptibility. Despite being

9   designed to measure kinaesthetic MI abilities, the Vividness of Motor Imagery Questionnaire

10   (VMIQ) is more likely to measure visual imagery abilities (Campos & Perez, 1990).

11   Although Farthing, et al. (1983) specifically included the mental imagery of personal actions

12   questionnaire (MIQ:PA) to assess the relationship between kinaesthetic MI abilities and

13   hypnotic susceptibility, fewer than half of the participants reported a consistent feeling of

14   movement in response to the MI suggestions. Therefore, neither the VMIQ nor the MIQ:PA

15   are able to effectively measure kinaesthetic imagery ability, but assess visual imagery

16   abilities, similar to the other imagery scales listed above. Overall, it can be concluded that

17   imagery abilities rated by existing scales have a weak, if any, relationship to hypnotic

18   susceptibility.

19

20   There are two broad reasons why no clear association has been found between imagery

21   abilities and hypnotic susceptibility. First, there may be no association between imagery

22   abilities and hypnotic susceptibility. Imaginative suggestions include instructions to imagine

23   some counterfactual state of affairs and then experience the world as if that state of affairs is

1 real. Factors that distinguish individuals of high and low hypnotic susceptibility may not

2 relate to their imaginative abilities per se, but rather affect their propensity to experience the

3 suggestions as real (Kirsch & Council, 1992; Kirsch & Lynn, 1999). It may be that

4 imaginative processes do not mediate hypnotic responses at all as is contended for example in

5 neodissociative theories (Bowers, 1992). Second, the self-report measures of imagery ability

6 may not tap into a putative imagery ability that contributes to hypnotic susceptibility. The

7 findings that imagery is a multifaceted phenomenon and self-report scales do not correlate

8 with many of these facets or objective measures of imagery ability supports the second

9 possibility (Lebon, et al., 2012a; Lequerica, et al., 2002). Therefore a relationship between

10 imagery abilities and hypnotic susceptibility may not be observable using only self-reported

11 measures of imagery ability.

12

13 2.2.   Hypnotic susceptibility and objective measures of imagery

14

15 Objective assessments of imagery ability may reveal associations between imagery and

16 hypnotic susceptibility. In an early study, McBain (1954) instructed blindfolded participants

17 to use their "preferred" index finger to trace a path that had been cut into a panel of wood and

18 then attempt to retrace this path on a flat panel by recalling the "feel" of the earlier task. The

19 recall was of the kinaesthetic sensations generated by the earlier task and therefore a measure

20 of kinaesthetic imagery ability. Performance on the kinaesthetic imagery task correlated

21 weakly but positively with hypnotic susceptibility.

22

1   Williamson and colleagues (2002) measured perceived effort, force, forearm

2   electromyography (EMG), blood pressure and heart rate during a sustained grip task, and MI

3   of the same task in hypnotised participants of high ("highs") and low ("lows") hypnotic

4   susceptibility. In highs blood pressure, heart rate and perceived effort increased significantly

5   through the task, mimicking the changes observed in both groups during movement

6   execution. This effect was not seen in lows. Although the hypnotic state may be a

7   confounding factor the observation that subjective and behavioural responses of individuals

8   to suggestions with and without a hypnotic induction are very similar (Braffman & Kirsch,

9   1999) indicate that this is unlikely to be the case. The assertion that highs had greater MI

10   abilities than lows is supported by the observation that changes in autonomic activity during

11   kinaesthetic MI correlate with imagery abilities (Lebon, et al., 2012a).

12

13   Roelofs and colleagues (2002a) measured the time taken for participants of high and low

14   hypnotic susceptibility to carry out implicit and explicit MI tasks following the suggestion of

15   right arm paralysis given while hypnotised. The implicit imagery task required recognising

16   the handedness of line drawings of left or right hands presented at varying degrees of

17   rotation. No difference in performance was seen in the implicit task between highs and lows.

18   The explicit task required the participant to mentally rotate their left or right hand to the

19   orientation of the presented hand images. In the explicit task, highs reported an inability to

20   imagine paralysed (right) hand rotation on 11.5% of trials, compared with 3.5% of trials for

21   lows. In highs mental rotation took significantly longer for their paralysed (right) hand

22   compared to their normal (left) hand and to either hand of lows. Mental rotation times were

23   not altered between hand images in lows. A difference in underlying MI ability may have

24   been a factor. A greater ability of highs to imagine weakness and subsequently suppress

1 movement would result in either a distortion of self-representations or the failure to generate

2 an efference copy, which is theoretically required to generate the "feeling" of movement in

3 kinaesthetic MI (Tian & Poeppel, 2012).

4

5 An association between imagery and hypnotic susceptibility is not a consistent finding, with

6 no association found when imagery abilities were measured using four computer-based

7 imagery tasks (Kogon, et al., 1998). However, all these tasks were strongly visually based

8 with little or no MI component. Overall, these findings are consistent with the assertion that

9 a relationship between hypnotic susceptibility and MI exists when assessed using objective

10 tests, and that an association between hypnotic susceptibility and imagery ability may be

11 present in some imagery domains but not others.

12

13 Further findings indicate that imagery abilities may contribute to the ability of an individual

14 to experience suggestions both in and out of hypnosis. A person given a suggestion to

15 imagine and then experience some state may respond in one of two ways (Comey & Kirsch,

16 1999). For example, in response to Item 1 of the SHSS:C the hypnotised person may imagine

17 that they are holding something heavy and their hand is being pressed down, or they may

18 imagine the desired end-state, i.e., that their hand has become heavy and that it is dropping.

19 The intentional use of this end-state imagery is commonly reported by hypnotised individuals

20 (Comey & Kirsch, 1999). The ability to experience suggestions in the non-hypnotised state

21 has been shown consistently to be a robust predictor of the ability to experience these

22 suggestions in the hypnotised state (Barber & Glass, 1962; Braffman & Kirsch, 1999; Hilgard

23 & Tart, 1966; Weitzenhoffer & Sjoberg, 1961). The behavioural responses and subjective

1 experiences of participants are very similar between the non-hypnotised and hypnotised states

2 (Braffman & Kirsch, 1999). Overall, experimental findings indicate good MI ability may be

3 associated with high levels of hypnotic susceptibility when assessed using objective

4 measures, and that MI ability may mediate responses to imaginative suggestions both in and

5 out of hypnosis. On the strength of this observation we next consider how MI ability may

6 contribute to hypnotic susceptibility.

7

8 3.   Kinaesthetic Motor Imagery and Hypnotic Susceptibility

9

10 MI is the mental simulation of an action without corresponding motor output (Lebon, et al.,

11 2012a). MI and movement preparation and execution are associated with the activation of

12 similar brain areas, including the primary motor cortex (M1), supplementary motor area

13 (SMA) and premotor areas, and changes in electroencephalogram (EEG) activity (see

14 Jeannerod, 1994; Neuper & Pfurtscheller, 2010; Stinear, 2010 for review). These common

15 areas of activation support the neural simulation of action theory of MI, which states that

16 overt action and covert action (imagery) only differ by whether or not movement occurs

17 (Jeannerod, 2001). It follows that MI ability may be defined as the extent to which

18 mechanisms modulated during movement are also modulated during imagery of the

19 movement (e.g., Stinear, et al., 2006).

20

21 MI may involve a visual or kinaesthetic strategy. Visual MI involves an individual "seeing"

22 themselves perform a movement, whereas with kinaesthetic MI the proprioceptive

23 consequences of the movement are "felt" (Jeannerod, 1994; Milton, et al., 2008).

1   Kinaesthetic and not visual MI results in temporally modulated and muscle specific

2   corticomotor (CM) excitation (Stinear, et al., 2006). Neural patterns of activation differ

3   between kinaesthetic and visual MI when individuals imagine a prescribed series of finger

4   movements. Both result in the activation of the primary motor cortex, premotor cortex, basal

5   ganglia, cerebellum and SMA. However, kinaesthetic MI results in the exclusive activation

6   of the putamen, caudate nucleus and cerebellum, whereas visual imagery results in the

7   exclusive activation of occipital areas (Guillot, et al., 2009). Overall, the evidence indicates

8   that kinaesthetic and visual MI involve different neural pathways, and kinaesthetic MI results

9   in greater activation of motor related pathways than visual MI.

10

11   There is mounting evidence the alterations in autonomic nervous system (ANS) function that

12   occur in preparation for and during physical activity are modulated during MI in a similar

13   manner (see Collet & Guillot, 2010 for review; Decety, et al., 1991; Wang & Morgan, 1992).

14   Physiological parameters modulated by MI, chronometric measures, and MI self-rating

15   scales have been used to construct a measure of MI ability called the MI Index (MII) (Collet,

16   et al., 2011). In a study designed to assess the validity of the MII, good imagers as defined

17   by higher scores on the MII demonstrated changes in CM excitability in a muscle specific

18   and temporally modulated manner during kinaesthetic MI (Lebon, et al., 2012a). In contrast,

19   poor imagers demonstrated temporal but not muscle specific modulation of CM activity.

20   Changes in measures of ANS activity indicated greater arousal and concentration in good

21   imagers compared with poor imagers. Although individual scores on the MIQ-R (Hall &

22   Martin, 1997) and self-reports of imagery quality are used to generate the MII score, neither

23   correlated with any of the physiological measures of imagery ability. Overall, the findings

24   support the validity of the ANS components of the MII as a measure of kinaesthetic MI

1   ability and reinforce the assertion that self-report measures of MI ability do not assess

2   objective MI ability.

3

4   The MI tasks described above are explicit imagery tasks, in which the participant was

5   instructed to create a mental image of the motor task. Implicit imagery is thought to be

6   utilised in mental rotation tasks (Cooper & Shepard, 1975; Shepard & Metzler, 1971).

7   Mental rotation tasks involve identifying the handedness of an object such as an image of a

8   hand or a geometric design presented at varying degrees of rotation. Although there is no

9   explicit instruction to use mental imagery, task completion is thought to involve the implicit

10   use of imagery. Rotation tasks that involve images of hands activate motor brain areas

11   supporting the hypothesis that a MI strategy was utilised in this condition (Kosslyn, et al.,

12   1998). Rotation tasks involving geometric designs can activate visual or motor brain areas,

13   depending on the recent experience of participants (see Kosslyn, et al., 2010 for extended

14   discussion). Although implicit rotation of hand images involves motor related pathways, the

15   neural mechanism(s) appears to differ from that involved in kinaesthetic MI. A conditioning

16   transcranial magnetic stimulation (TMS) pulse to the right inferior parietal lobe has different

17   effects on subsequent left M1 excitability depending on whether participants are at rest or

18   engaged in an implicit or a kinaesthetic MI task, and the timing of the conditioning stimulus

19   (Lebon, et al., 2012b).   These different effects are consistent with a hypothesised inhibitory

20   pathway between the right inferior parietal lobe and left M1 that plays a role in preventing

21   movement during MI and is independent of MI ability. Overall, the findings indicate that

22   implicit and explicit MI activate distinctly different pathways.

23

1 3.1.   Kinaesthetic MI ability, motor suppression imagery and hypnotic susceptibility

2

3 MI appears to be multidimensional, with individual differences evident in subjective and

4 objective components, including modality (kinaesthetic vs visual) and the task (implicit vs

5 explicit) employed. The findings of a relationship between kinaesthetic MI ability and

6 hypnotic susceptibility (McBain, 1954) and relationships between the degree of modulation

7 of the ANS during MI with modulation of CM excitability (Lebon, et al., 2012a) and with

8 hypnotic susceptibility (Williamson, et al., 2002), indicate that a positive association may

9 exist between kinaesthetic MI abilities and hypnotic susceptibility. The finding that the

10 ability to experience imaginative suggestions outside of hypnosis is a good predictor of

11 hypnotic susceptibility indicates that kinaesthetic MI abilities may be related to a more

12 general imaginative suggestibility (Braffman & Kirsch, 1999). This relationship between

13 hypnotic susceptibility and kinaesthetic MI ability may be masked if the nature of the MI task

14 is not carefully and clearly specified.

15

16 A rudimentary mechanism of hypnotic responding to suggestions of movement, termed direct

17 motor suggestions (Woody, et al., 2005), readily follows from these observations.

18 Individuals who are good kinaesthetic imagers also show increased M1 activity during

19 kinaesthetic MI. This subthreshold activity for actual movement in an individual primed to

20 expect a response, and coupled with the instruction to experience the suggested state as if it

21 was real, may result in the occurrence of the suggested movement with the associated loss of

22 sense of agency. This is a process very similar to that proposed by Arnold (1946). It is less

23 clear how kinaesthetic MI and suggestions to imagine that one is unable to move, termed

1 motor challenge suggestions (Woody, et al., 2005), can be linked. Examples of motor

2 challenge suggestions include: part of one's body is rigid or restrained in some way and one

3 is not able to move against this, or that a limb is heavy or weak and cannot be moved

4 (Weitzenhoffer & Hilgard, 1962). It might be that the suggestion to experience some state,

5 such as a rigid arm that cannot be bent, results in the occurrence of that state by increased

6 activity in antagonist muscles, e.g., by increasing activity in the triceps brachii and opposing

7 flexion of the elbow by the biceps brachii. This increased muscle activity then acts in

8 opposition to attempts to counter the suggestion, e.g., to bend a rigid arm. Such a mechanism

9 is supported by the finding that the majority of hypnotised participants who were unable to

10 bend their arm in response to the suggestion of arm rigidity showed co-contraction of their

11 biceps and triceps as measured by surface EMG (Winkel, et al., 2006). If this mechanism is

12 correct then it might be expected that susceptibility to motor suggestions is associated with

13 good MI abilities.

14

15 Not all experimental data can be explained by this hypothesised kinaesthetic MI mechanism

16 for motor challenge suggestions. Individuals who successfully respond to a suggestion of

17 immobility do not necessarily activate both agonist and antagonistic muscles despite self-

18 reported effort to bend their arm. These individuals may experience a motor response

19 internally, with no observable movement, interpreting the instruction as another instruction to

20 experience rather than attempt to conduct the movement (Winkel, et al., 2006). The

21 diminished agonist activity renders a kinaesthetic MI mechanism to explain the limb

22 immobility redundant.

23

1   An alternative possibility consistent with the explanation offered by Winkel, et al. (2006) is

2   that participants who counter the suggestion but do not exhibit co-contraction of muscles

3   engage in what has been termed "negative imagery" (Sohn, et al., 2003), or motor

4   suppression imagery (MSI). This strategy could be applied to suggestions in which the

5   subject is told that a limb is heavy or weak and they are unable to move it. Participants

6   instructed to imagine inhibiting a TMS-induced hand muscle activation "by trying to do more

7   relaxation" displayed reduced motor evoked potential (MEP) amplitudes compared to a rest

8   condition. This suppression of CM excitability was unable to be explained by a change in the

9   amount of intracortical inhibition and facilitation, or excitability of motor axons at the

10 periphery (Sohn, et al., 2003). Reduced MEP amplitudes were also evident when participants

11 were instructed to mentally inhibit, in contrast to facilitate, a TMS evoked wrist flexion

12 (Bonnard, et al., 2009). In contrast with what Sohn, et al. (2003) reported, intracortical

13 inhibition assessed by TMS and EEG was associated with a decrease in CM excitability

14 (Bonnard, et al., 2009). This finding is consistent with the hypothesis that when participants

15 prepare to resist a TMS evoked muscle activation, the anticipatory processes cause a decrease

16 in the excitability by increasing inhibitory processes due to increased gamma-aminobutyric

17 acid activity at the cortical level. Although the findings of Sohn, et al. (2003) and Bonnard,

18 et al. (2009) cannot be directly extended to support the assertion that MSI is responsible for

19 suppression of movement in hypnosis, they do show that a centrally generated movement

20 (elicited by TMS over M1) can be intentionally suppressed. It is proposed that a MSI

21 mechanism becomes activated for a specific movement following a suggestion of paralysis or

22 rigidity. This MSI mechanism then inhibits any attempt at movement, resulting in no

23 activation of the agonist muscle.

24

1 MSI appears to differ phenomenologically from the inhibition of a prepared motor response

2 that occurs, for example, in a Go-NoGo task. With MSI the intention of the individual is to

3 actively resist a TMS-generated movement prior to the occurrence of the movement. In

4 contrast, in a Go-NoGo task the movement is prepared with the intention to release it on a go

5 signal. The inhibition occurs only if the instruction is given to withhold the response. The

6 extent to which the mechanisms underlying MSI may differ from those underlying the

7 inhibition seen with the instruction to withhold a response during a Go-NoGo task has not

8 been investigated.

9

10 MSI and kinaesthetic MI might involve common mechanisms. Kinaesthetic MI appears to

11 involve at least two processes, the activation of motor related neural mechanisms and the

12 inhibition of motor output at the level of M1. The mechanisms underlying the observed

13 inhibition of motor output with MI are unknown, but could include indirect inputs from

14 parietal lobe to M1 (Lebon, et al., 2012b). The finding that parietal lobe damage can lead to a

15 failure of suppression of imagined movements is consistent with the role of the parietal lobe

16 in suppressing imagined movements (Schwoebel, et al., 2002). Although speculative, the

17 processes that inhibit motor output during MI might also be active with MSI during

18 movement execution, suppressing the movement at the level of M1.

19   [Insert Figure 1 about here]

20 Figure 1 illustrates a preliminary model integrating KMI and MSI mechanisms to explain

21 responses to direct motor and motor challenge suggestions given under hypnosis. The

22 observation that M1 excitability is increased at rest after a direct motor suggestion is given

23 under hypnosis is consistent with M1 receiving less inhibitory drive (Takarada & Nozaki,

1   2014). The model is consistent with discrepancy-attribution (Barnier, et al., 2008) and

2   response set theories (Lynn, et al., 2008). According to these theories the altered sense of

3   agency that is a hallmark of hypnotic responding is due either to the ease with which a

4   response is generated relative to the expected effort required (a response that requires much

5   less effort than is expected is not attributed to the self), or the response expectancies of the

6   individual, respectively. The model in figure 1 is also consistent with a model proposed to

7   underlie CD and hypnotic responding that involves dissociative changes in monitoring of

8   initiation and control of movement (Bell, et al., 2011). Importantly, the illustrated model

9   generates testable hypotheses that may allow the elucidation of the mechanisms underlying

10   hypnotic responses to motor suggestions. These include that the mechanism/s underlying one

11   phenomena will also underlie the others. For example, the model predicts that the cortical

12   inhibitory mechanisms that underlie the inhibition of movement during KMI also operate

13   during MSI and motor challenge suggestions. It predicts that the inferior parietal lobe is

14   involved in modulating this inhibition in all of these phenomena. It also predicts that

15   hypnosis will enhance the temporal and spatial modulation of the M1 seen with KMI. This

16   may result in activation of the alpha motor neuron pool not usually seen with KMI.

17   Perturbation of inferior parietal lobe functioning using brain stimulation techniques is

18   predicted to enhance or inhibit this effect. All these hypotheses are yet to be investigated.

19

20   3.2.   MI ability and theories of hypnosis

21

22   Theories of hypnosis have focused on explaining the subjective sense that the self is not the

23   origin of the response to hypnotic suggestions and, to a lesser degree, to the changes in the

19

1   experience of reality felt by hypnotised individuals (Barnier, et al., 2008). Four prominent

2   theories of hypnosis prevail: Neodissociation (Woody & Sadler, 2008), cold control (Barnier,

3   et al., 2008), discrepancy-attribution (Barnier, et al., 2008), and response expectancy (Lynn,

4   et al., 2008). All are grounded in broader psychological theory and experimental data, and

5   have been developed with the aim of not only explaining experiences in hypnosis but also

6   aligning with more general models of human experience and behaviour. An extensive

7   literature elaborates and compares these theories and a detailed discussion is beyond the

8   scope of this review. These four theories invoke some separation between processes that

9   initiate motor or cognitive responses and those that monitor these responses. However, they

10 differ in which processes are affected and how they are altered by imaginative suggestions.

11 Two of these – discrepancy-attribution and response set theories – allow for a role of MI in

12 influencing susceptibility. In contrast, cold control theory and neodissociation theories do not.

13

14 For a number of years a debate has continued regarding the presence of possible hypnotic

15 susceptibility subskills (Woody & Barnier, 2008; Woody, et al., 2005). Until 2005, factor

16 analytic studies that investigated whether these subskills existed identified three factors, or

17 clusters of suggestion subtypes, to which participants responded. These were direct motor

18 suggestions, challenge motor suggestions, and cognitive-perceptual suggestions. However,

19 the statistical methods used in these studies failed to adequately address the confounding

20 effect of suggestion difficulty. This may have led to the spurious identification of the three

21 factors, with the level of difficulty of each suggestion type rather than the type of suggestion

22 per se resulting in the clusters. A factor analysis of the responses of 616 participants who

23 completed both the SHSS:C and the Harvard Group Scale of Hypnotic Susceptibility: Form

24 A (Shor & Orne, 1962) was carried out to address this methodological problem. This

analysis found that the hypnotic responses of participants mapped onto four factors that

correlated moderately well with each other: direct motor, motor challenge, perceptual-

cognitive and posthypnotic suggestion (Woody, et al., 2005). Woody, et al. (2005)

interpreted their findings as support for their hypothesis that a general hypnotisability factor

influences the susceptibility of an individual and that subskills related to the four factors

dictate which specific suggestions an individual will respond. In the context of this review,

this interpretation raises the possibility that response to direct motor and motor challenge

suggestions may be related to kinaesthetic MI and MSI abilities or subskills respectively.

4.   Conversion Disorder, hypnotic susceptibility and MI abilities

Evidence supports a positive relationship between hypnotic susceptibility and the

development of CD. Patients diagnosed with pseudo epileptic seizures, a type of CD, showed

greater levels of hypnotic susceptibility than either patients diagnosed with epileptic seizures

or healthy controls (Kuyk, et al., 1999). A group of 50 CD patients scored significantly

higher on the SHSS:C than a group of age and gender similar controls suffering from other

non-psychotic psychiatric disorders and a group of age, gender and education level similar

healthy individuals (Roelofs, et al., 2002b). The number of pseudo neurological symptoms a

patient suffered correlated modestly with their SHSS:C score. These data supported the

hypothesis that patients suffering from CD would show higher levels of hypnotic

susceptibility than patients with mood disorders, and that a relation between hypnotic

susceptibility and severity of conversion symptoms existed. A methodological shortcoming

of both studies was the possible influence of demand characteristics, i.e., changes in the

1 performance of participants in an experiment after they have formed a belief of what the

2 investigators expect to find. These may have influenced the relationship between

3 susceptibility and conversion symptoms. A point not made by Roelofs, et al. (2002b) was

4 that hypnotic susceptibility differences between the CD, mood disorder and normal groups

5 were small and placed all groups at the lower end of hypnotic susceptibility on the SHSS:C

6 (Roelofs, et al., 2002b). If hypnotic susceptibility did have a significant role in the

7 development of CD then patients with CD would be expected to score at the upper end of the

8 SHSS:C range, not in the range observed by Roelofs, et al. (2002b).

9

10 An association between hypnotic susceptibility and CD is not always evident. Moene, et al.

11 (2001) assessed hypnotic susceptibility in 97 psychiatric inpatients and outpatients with

12 diagnoses of either CD or Somatization Disorder motor type (American Psychiatric

13 Association, 1987). Although they excluded patients suffering from "a major affective

14 disorder…or other severe psychiatric diagnosis…" the sample still showed a high rate of

15 comorbid psychiatric disorders. Overall, the patient group was no more susceptible to

16 hypnosis than healthy controls.

17

18 Of the three studies concerning CD and hypnotic susceptibility, two support a link between

19 the level of hypnotic susceptibility and CD (Kuyk, et al., 1999; Roelofs, et al., 2002b),

20 whereas the other does not (Moene, et al., 2001). However, even when an association

21 between hypnotic susceptibility and CD is demonstrated, the positive relationship appears to

22 be small. The case-control nature of the studies is a shortcoming. This constraint is difficult

23 to overcome given that CD is relatively uncommon (Stone, et al., 2009). A very large cohort

1   of individuals of known hypnotic susceptibility would need to be followed for a considerable

2   period of time to observe CD cases that might develop before a confident statement regarding

3   the nature of a relationship between the two could be made. Another issue is the possibility

4   that CD does not represent a single disease entity but rather a group of disorders with

5   different underlying pathophysiological bases and risk factors. For example, an inherent

6   neurological trait that may predispose an individual to develop CD paralysis may not be a

7   predisposing factor in CD gait disturbance. The three studies that assessed hypnotic

8   susceptibility in CD patients examined patients suffering from different CD subtypes,

9   pseudoseizures (Kuyk, et al., 1999) and a range of motor and sensory CD (Roelofs, et al.,

10 2002b), or included patients suffering from Somatization Disorder (Moene, et al., 2001). If a

11 study population is made up of CD patients of all symptom types then this heterogeneity may

12 mask an association between an individual symptom type, such as motor weakness, and

13 hypnotic susceptibility. The inclusion of patients suffering from disorders other than CD may

14 further increase the heterogeneity of the study population and lessen any association.

15

16 4.1.   Hypnotically induced paresis and motor Conversion Disorder

17

18 A number of imaging studies have compared processes related to motor CD, specifically

19 motor weakness or paralysis, and hypnotically induced weakness or paralysis. Many of the

20 studies have also compared these motor impairments with feigned weakness or paralysis.

21

22 Similar patterns of cortical activation were found with attempted movement of a lower limb

23 paralysed due to CD and lower limb paresis induced by hypnosis. Positron emission

1   tomography (PET) was used to assess cerebral blood flow in a woman suffering from a motor

2   CD characterised by left sided paralysis (Marshall, et al., 1997). Imaging revealed activation

3   of premotor and motor areas when she prepared to move the paralysed left leg, but no

4   activation of M1 with her attempts to move this leg. The activation of premotor and motor

5   areas during preparation to move was evidence against intentional feigning. There was also

6   significant activation of the right anterior cingulate cortex (ACC) and right OFC during

7   attempted movement of the paralysed leg, which was not present in any of the other

8   conditions. This was interpreted as evidence of inhibition of the primary motor cortex by

9   these prefrontal areas. A similar pattern of cerebral blood flow changes was detected in a

10   healthy 25 year old man with hypnotically induced left lower limb paralysis while he tried to

11   move his left leg (Halligan, et al., 2000). This included significantly increased blood flow in

12   the right OFC and ACC. Halligan, et al. (2000) proposed that these areas inhibited

13   movement of the left leg despite dorsolateral prefrontal cortex (DLPFC) activation and

14   downstream activation in the cerebellum, as suggested by Marshall, et al. (1997). Halligan,

15   et al. (2000) acknowledged the alternative explanation that the OFC and ACC may have been

16   involved in management of mental dissonance produced when suggestion of paralysis

17   conflicted with instruction to move the "paralysed" leg. Whatever the explanation, similar

18   areas of activation indicates that a common mechanism was involved in motor CD and

19   hypnotically induced paralysis.

20

21   Other evidence supports the role of inhibitory processes in preventing movement of a

22   hypnotically paralysed arm. fMRI was used to compare patterns of cortical activation in

23   individuals of high hypnotic susceptibility during normal upper limb movement and

24   attempted movement following hypnotically induced left arm weakness (Deeley, et al.,

1   2013). Both tasks were carried out while in the hypnotised state. Relative to the paretic state,

2   the normal state showed increased right M1, right primary somatosensory cortex, and left

3   cerebellum activity, consistent with the role of these regions in the completion of movement.

4   In contrast, activity in the right SMA and ACC was increased in the paretic state compared

5   with the normal state. An increase in SMA activity in the paretic state may indicate greater

6   effort to move in the paretic state, whereas greater ACC activity may be related to inhibition

7   of the intended movement.

8

9   No evidence of frontally mediated inhibition was found on functional magnetic resonance

10   imaging (fMRI) in a woman diagnosed with a psychogenic left arm paresis while she carried

11   out a Go-NoGo task (Cojan, et al., 2009a). A healthy control group also carried out the task

12   with normal movement as well as a subset of healthy participants who completed it with

13   instructions to feign weakness by acting "as if" they were suffering from motor weakness and

14   unable to move their fingers. Preparatory activity was evident in motor cortex for the patient,

15   controls, and feigners when preparing to move their weak hand. In addition, activation in the

16   ventromedial prefrontal cortex (VMPFC) and left OFC was evident in the CD patient, but not

17   controls and feigners. Failure of the patient to move the weakened hand on Go trials was

18   associated with activation of precuneus (bilaterally), left superior frontal gyrus and right

19   ventrolateral frontal gyrus, but not the dorsolateral inhibitory areas previously shown by

20   Marshall, et al. (1997). Inhibition of response during NoGo trials in controls and failed Go

21   trials in the participants feigning paralysis activated a different set of cortical areas. A

22   functional connectivity analysis of the fMRI data revealed that the right M1 of the patient

23   showed greater connectivity with their precuneus and VMPFC, areas previously shown to be

24   involved in self-related processing, during preparation and attempts to move their paralysed

1   left arm. Controls and feigners showed greater connectivity between the M1 and ipsilateral

2   sensorimotor regions. The authors concluded that symptoms of motor CD weakness were not

3   related to processes involved in conscious motor inhibition, such as those associated with

4   activation of the inferior frontal gyrus, but instead to disturbances in the processes involved

5   in accessing self-related representations and emotional regulation.

6

7   Hypnotically induced left hand paralysis resulted in a similar but not identical pattern of

8   cortical activation to that seen with CD left arm paralysis during attempted movement (Cojan,

9   et al., 2009b). While undergoing fMRI, healthy participants performed a similar Go-NoGo

10   task to that of Cojan, et al. (2009a) in a non-hypnotised state and with hypnotically induced

11   left hand paralysis. An additional non-hypnotised group instructed to feign left hand paralysis

12   was also included in the study. Preparing to move the paralysed hand resulted in activation

13   of M1, as well as increased precuneus and extrastriate activity not evident in the other

14   conditions. When movement of the hypnotically paralysed hand was signalled by the precue,

15   both Go and NoGo trials resulted in the same level of activation of the inferior frontal gyrus.

16   This area was also active in NoGo trials during normal movement and Go trials with feigned

17   weakness. This was taken as evidence to support the view that activation of the inferior

18   frontal gyrus was not due to inhibition during the hypnotised condition, but due to other

19   mechanisms such as increased self-monitoring. A functional connectivity analysis found that

20   in the non-hypnotised state the right M1 showed greater connectivity to the right dorsal

21   premotor cortex and left cerebellum, whereas in the hypnotised state the right M1 showed

22   greater connectivity to the left angular gyrus and left precuneus. Connectivity between the

23   left M1 and other brain regions did not significantly differ in the normal and hypnotic states.

24   Overall, the findings identified a partially shared mechanism underlying psychogenic motor

1   weakness and hypnotically induced paralysis, indicated by the precuneus activation in both

2   conditions. This may have been related to changes in internal self-representations. However,

3   the activation of the VMPFC seen with psychogenic weakness but not with the hypnotically

4   induced weakness, and the extrastriate and inferior frontal gyrus activation seen with the

5   hypnotically induced weakness but not with psychogenic weakness, indicated mechanisms

6   underlying the two states were not identical.

7

8   Further evidence supports different mechanisms underlying feigned paralysis and weakness

9   compared with motor CD or hypnotically induced paralysis or weakness. Ward, et al. (2003)

10   used PET to compare cerebral blood flow in participants with hypnotically induced left leg

11   paralysis and participants instructed that their left leg was "completely normal and able to

12   move" but to pretend it was paralysed with the intention of deceiving an observer. Compared

13   with feigning, the instruction to move the left leg in the hypnotically induced paralysis

14   condition was associated with right OFC, cerebellar, left putamen, and thalamus activation.

15   In contrast, the instruction to move during feigned paralysis was associated with left

16   ventrolateral prefrontal cortex, right superior temporal, and medial parietal activation.

17   Spence, et al. (2000) also used PET to show that arm weakness due to "hysteria" and arm

18   weakness feigned by "pretending" resulted in different patterns of cortical activation.

19   Activity in the left DLPFC was reduced in hysteria patients compared with feigners and

20   controls when they tried to move their weak arm, irrespective of the side impaired. In

21   contrast, activity in the right anterior prefrontal region was reduced in feigners compared with

22   patients and controls during movement of their "weak" limb. The diminished left DLPFC

23   activity, shown previously to be activated by internal generation of action, demonstrated that

24   hysterical motor symptoms involved dysfunction of higher components of volition. It is

27

1   important to note these clear and consistent differences in cortical activation patterns This

2   observation supports the assertion that CD patients and hypnotised participants are not

3   feigning, an important assertion given that deception is commonly employed by neurologists

4   in their explanatory models of CD (Kanaan, et al., 2009).

5

6   Overall, there is evidence that similar structures are activated during hypnotically induced

7   weakness or paralysis and motor CD weakness or paralysis. This evidence includes the

8   activation of the ACC and OFC with hypnotically or CD induced weakness or paralysis.

9   Furthermore, a similar pattern of functional connectivity has been observed between the M1

10 and precuneus in motor CD and hypnotically induced paralysis, which differs from that seen

11 in the normal state. However, changes in activation in numerous other brain areas have also

12 been observed across the studies. The divergent findings are likely due to methodological

13 differences between studies. Methodological differences include imaging modalities, tasks

14 employed, between or within subject comparisons, and normal movement being assessed in

15 participants in the hypnotised and non-hypnotised states. These divergent findings have not

16 prevented the emergences of two separate hypothetical mechanisms for hypnotically – or

17 motor CD – induced suppression of movement. The first is the top down active suppression

18 of cortical motor areas by prefrontal inhibitory areas, which occurs outside of conscious

19 awareness (Deeley, et al., 2013). The second is a less clearly defined alteration of self-

20 awareness mediated by disturbances of self-representations and emotional regulation

21 (Vuilleumier, 2014). To date, neither of these explanations can take precedence based on

22 previous imaging findings.

23

1   4.2.   Motor Imagery and motor Conversion Disorder

2

3   The evidence to support a role of kinaesthetic MI in CD is sparse and more difficult to

4   interpret than the evidence to support a role of kinaesthetic MI ability in hypnosis. Only a

5   small number of studies have examined imagery ability directly in patients suffering from

6   motor CD. Most studies investigating imagery in CD have used imagery as a tool to probe

7   mechanisms underlying impairments and often presume similar mechanisms underlie

8   different imagery tasks and strategies, rather than investigate imagery abilities per se.

9

10   Direct evidence of impaired MI abilities in people suffering from functional movement

11   disorders has been found (Liepert, et al., 2008, 2009, 2011). Liepert, et al. (2008) assessed

12   CM excitability with single- and paired-pulse TMS in four patients suffering from upper limb

13   motor CD weakness (three suffering from a flacid paresis and one suffering from a dystonia)

14   and eight age matched controls. TMS was delivered while participants imagined a tonic

15   adduction of their left and right index fingers. EMG was recorded from the first dorsal

16   interossei (FDI) muscles. No difference in neurophysiological measures, which included

17   motor thresholds, intracortical inhibition and facilitation, and central conduction times, was

18   evident at rest between CD patients and controls. In patients, MEP amplitudes recorded in

19   the affected hand during MI of the same hand were reduced compared with MEPs recorded in

20   the resting baseline condition. Furthermore, MEP amplitudes in the affected hand of patients

21   during MI of that hand were also reduced compared with MEP amplitudes during MI in the

22   control group. It is not clear what type of MI instruction was given to participants, but the

23   clear change in CM excitability during imagery indicates the use of a kinaesthetic MI

1   strategy. The findings indicated that CM excitability was suppressed during MI of an upper

2   limb affected by motor CD.

3

4   As well as the suppression of CM excitability during MI of the affected limb, CM

5   excitability during MI of the unaffected limb was reduced relative to controls in a larger

6   sample of patients suffering from upper limb CD (Liepert, et al., 2009). Liepert, et al. (2009)

7   used TMS to assess CM excitability during imagery of tonic index finger adduction in eight

8   patients with upper limb motor CD (five patients had weakness, three a spastic dystonia), and

9   a healthy control group. FDI MEP amplitudes were measured for both the affected and

10 unaffected hands in patients, and one hand in controls. As previously observed, no difference

11 was reported in resting motor thresholds between affected and unaffected hands of the

12 patients or controls. MI of the unaffected hand of patients and controls resulted in the

13 expected increase in MEP amplitude in the hand involved relative to rest. Of note, the

14 increase in MEP amplitude with MI seen in the unaffected hand of patients was less than the

15 increase seen in controls, suggesting that the "unaffected" hand may not have been

16 asymptomatic.

17

18 A similar pattern of suppression of CM excitability has been shown during MI of foot

19 movement in patients suffering from motor CD weakness of their lower limbs (Liepert, et al.,

20 2011). CM excitability was measured during MI of phasic and tonic ankle dorsiflexion in

21 patients suffering from motor CD with left, right or bilateral lower limb weakness, and in a

22 group of age-matched controls. Details of footedness were not provided. MEP amplitudes

23 were measured in the tibialis anterior muscle. There were no differences in resting motor

1   thresholds between the groups or limbs. Measures of intracortical inhibition and facilitation

2   did not show any difference between the groups or limbs during MI. The control group

3   exhibited the expected increase in MEP amplitude in the lower limbs involved in the MI task

4   relative to rest, as did patients suffering from unilateral symptoms during MI of their

5   unaffected limbs. These increases were of a similar magnitude. In contrast, the affected

6   lower limbs of patients showed a decrease in MEP amplitude during MI of that limb when

7   compared with rest and with controls engaged in MI. Overall, the reductions in CM

8   excitability during MI of affected limbs in patients suffering from upper and lower limb

9   symptoms were similar.

10

11   These findings (Liepert, et al., 2008, 2009, 2011) identify a clear reduction in CM excitability

12   from baseline during MI involving the affected limbs of motor CD patients, and that upper

13   limb motor CD patients may show impaired MI of their unaffected upper limb. The first two

14   studies suggest either a state or trait difference in MI abilities in the patient group compared

15   with the healthy control groups, given the finding of a smaller increase in CM excitability

16   with MI of the unaffected limb in patients compared with controls. The reason for the lack of

17   difference between the unaffected lower limbs of patients and controls is unclear, but may

18   relate to differences in how the upper and lower limbs are modulated during imagery. A trait

19   difference may have existed between the patient and control groups, but this cannot be

20   confidently asserted because the affected limb was only compared with the unaffected limb

21   of the same type and not the other limbs, e.g., a functionally weak leg was compared with an

22   unaffected leg, but MI abilities in the upper limbs were not assessed. It might be that the

23   impairment evident in the affected upper limb somehow spilled over to the opposite limb, but

24   not the whole body, and is therefore not able to be generalised. This could occur either via

some higher process influencing the M1 bilaterally or through the affected M1 directly

influencing ipsilateral movement via interhemispheric or descending projections. This effect

was seen in a study examining, among other measures, reaction times in patients suffering

from motor CD with upper limb weakness (Blakemore, et al., 2015). Patients showed longer

premotor (the central component of reaction time) and motor times (the peripheral component

of reaction time) in both their symptomatic and asymptomatic limbs compared to healthy

controls. Therefore, performance of the "asymptomatic" limb was also impaired in addition

to the "symptomatic" limb.

Individuals feigning weakness showed a similar pattern of impairment to motor CD patients

(Blakemore, et al., 2015). These participants were instructed "to imagine that their left arm,

hand and fingers had become so weak, such as following a severe injury to the limb, that their

muscles would be unable to exert a lot of force", or "to imagine that their left fingers were

moving against a resistance that, however hard they tried, they would find difficult". These

are MI based instructions. The behavioural performance of "symptomatic" hand of feigners

was very similar to the symptomatic hand of patients, with prolonged premotor and

movement times compared with controls. The "asymptomatic" hand of feigners also showed

longer premotor times than controls, but similar movement times. Therefore, participants

feigning weakness using a MI based strategy showed a similar pattern of behaviour and

neurophysiological response to patients suffering from motor CD weakness.

The findings of Liepert, et al. (2008, 2009, 2011) and Blakemore, et al. (2015) indicate that

imagery related movement suppression may be involved in motor CD. Normally a presumed

1 inhibitory mechanism prevents overt movement during kinaesthetic MI. In the proposed

2 scenario, greater and automatic activation of this mechanism during motor execution or

3 kinaesthetic MI in CD patients would lead to the observed motor impairment, as is

4 hypothesised to occur with imaginative suggestions of weakness, or suppression of MEP

5 amplitude with MSI.

6

7 Although MI abilities were not directly assessed, the findings of Roelofs, et al. (2001)

8 provide some evidence of a role of MSI in motor CD weakness. Performance of patients

9 suffering from paralysis or paresis of one or more limbs and healthy age-matched controls

10 was assessed on a choice reaction time task, a letter rotation task, and explicit and implicit MI

11 tasks. Patients struggled to complete the explicit rotation tasks, showing an inability to

12 imagine rotation in 51.3% of foot movement trials and 8.9% of hand movement trials

13 (compared with 0% and 0.5% respectively in healthy controls). Overall, patients were slower

14 on all tasks, which was taken relate to general psychomotor slowing. Mental rotation was

15 also significantly slower in the patient group. This slowing of mental rotation was most

16 evident in the explicit hand imagery task and greatest in the affected limb. The dissociation

17 between explicit and implicit imagery performance was taken to show that impairment in MI

18 was greatest when imagery was intentionally generated, in line with the initial hypothesis of

19 the authors that explicit MI would be more impaired than implicit MI in patients with CD.

20 However, this interpretation presumed that explicit and implicit imagery tasks rely on similar

21 mechanisms with the only difference being that explicit imagery requires these mechanisms

22 to be intentionally activated and implicit imagery does not. This may not be the case, as

23 shown by Lebon, et al. (2012b). An alternative explanation for the differences observed by

24 Roelofs, et al. (2001) is that a MI related suppression mechanism was overactive in the

1   patient group during the explicit imagery task, contributing to their poor performance. This

2   suppression mechanism was not active during the implicit imagery task and subsequently had

3   no effect on performance.

4

5   MI has been used to provide evidence that motor dysfunction in motor CD is linked to

6   enhanced self-monitoring and not inhibition of the motor system. de Lange, et al. (2007)

7   used fMRI to investigate eight patients suffering from CD paralysis of an upper limb to

8   measure changes in cortical blood flow while participants carried out an implicit MI task. An

9   implicit MI task was used to control for processes such as altered sensory feedback and

10   enhanced monitoring that may have occurred with a failed attempt at actual movement.

11   Consistent with a previous study (Roelofs, et al., 2001), there was no difference in

12   behavioural performance measures and activation levels of motor related areas between the

13   unaffected and affected hands. However, MI of the affected hand was also associated with

14   activation of the ventromedial prefrontal and superior temporal cortices. These differences in

15   activation were due to a failure to de-activate these regions during imagery. The findings

16   supported the hypothesis that functional motor paralysis is associated with heightened self-

17   monitoring during actions of the affected arm, and that these cortical activations were not due

18   to movement inhibition as previously suggested by Marshall, et al. (1997) and Halligan, et al.

19   (2000).

20

21   Further evidence supports the hypothesis that motor CD is associated with heightened self-

22   monitoring. de Lange, et al. (2008) compared performance of patients diagnosed with CD

23   paralysis of an upper limb in implicit and explicit MI tasks while undergoing fMRI. The

description of the explicit task indicates it did not involve kinaesthetic MI, but rather

involved visual MI (in contrast to Roelofs, et al., 2001). The investigators hypothesised that

if the altered pattern of activity between the affected and unaffected sides seen with implicit

imagery in their earlier study (de Lange, et al., 2007) was due to increased self-monitoring,

then this would be abolished when self-monitoring was induced with explicit imagery. The

cortical activation differences seen in the implicit task were not present in the explicit task,

supporting their hypothesis. However, this assertion relies on the premise that implicit and

explicit MI and visual and kinaesthetic imagery share similar underlying mechanisms, which,

as already discussed, is not the case.

Overall these studies provide evidence to support the hypotheses that MI abilities or MSI may

contribute to motor CD. Roelofs, et al. (2001) and the series of experiments by Liepert, et al.

(2008, 2009, 2011) show that MI is clearly disrupted in motor CD with weakness, and that

this disruption extends beyond the affected or symptomatic limb. However, they do not

provide evidence that informs whether this disruption to MI is a consequence of impaired

movement planning or generation, or whether MI related mechanisms lead to the impairment

in movement planning or generation and observed behavioural impairment. The finding that

similar behavioural performances are observed in patients with motor CD and MI induced

weakness supports, albeit weakly, a causal relationship between CD and MI mechanisms.

Taken together, there is evidence to indicate common mechanisms underlie hypnotic

responding, hypnotically induced weakness or paralysis, and motor CD. This evidence

includes findings of greater hypnotic susceptibility in patients suffering from CD relative to

patients suffering from other conditions, and the activation of similar brain areas with

hypnotically induced and CD weakness or paralysis. There is also clear evidence of

disrupted MI in patients suffering from CD paralysis or paresis. Whether this disruption

results in or is due to CD motor symptoms is unclear. However, it is interesting to speculate

that given the potential role of kinaesthetic MI abilities and MSI in hypnotic responding to

motor suggestions, and the possibility of shared mechanisms underlying hypnotic responding

and CD, kinaesthetic MI and MSI, and motor CD symptoms may be related. It is proposed

that kinaesthetic MI and MSI also generate symptoms of motor CD in the same way they

generate hypnotic responses following direct motor and motor challenge suggestions, as

described in figure 1. This account of motor CD makes no presumptions regarding the

upstream factors that may trigger the activation of these imagery mechanisms. Such an

account is consistent with the proposed role of some unconscious maladaptive belief or

"idea"' that an individual adopts in response to an affective or stress-related state that results

in CD motor symptoms (Halligan, 2011). The effect of this maladaptive idea is shaped by the

context of the individual's own and wider societal beliefs and practices regarding illness. In

this formulation, the maladaptive idea affects voluntary motor mechanisms of the individual

outside of their conscious awareness, resulting in the motor symptom. Kinaesthetic MI and

MSI mechanisms provide a neuronal substrate on which a maladaptive idea may act, possibly

through dissociative changes in the executive control and monitoring of movement as is also

hypothesized to occur in hypnotic responding (Bell, et al., 2011).   Based on the model, it

may be hypothesized that the cortical inhibitory mechanisms that underlie the inhibition of

movement during kinaesthetic MI will also inhibit attempted movement during motor CD

paralysis or paresis, and that these inhibitory mechanisms are the same as those operating

during MSI. If these inhibitory mechanisms are found to be the same then perturbation of the

inferior parietal lobe functioning using brain stimulation techniques would be expected to

1   transiently enhance or inhibit motor CD symptoms in the same manner as kinaesthetic MI

2   and MSI performance. These hypotheses are yet to be investigated.

3

4   5.   Summary

5

6   Despite many years of research, hypnosis and the subjective alterations in experience it

7   generates in many individuals remains poorly understood. This lack of understanding

8   extends to questions concerning the stability and variability in hypnotic susceptibility seen

9   from individual to individual, and how these relate to susceptibility to imaginative

10   suggestions outside of hypnosis. Although hypnotic susceptibility and mental imagery

11   abilities have been linked and investigated for many years, no or only a weak association

12   between the two has been found when investigated using subjective measures of mental

13   imagery abilities. When objective measures of imagery abilities are included an association

14   between hypnotic susceptibility and mental imagery abilities is strengthened.

15

16   The nature of kinaesthetic MI and its link to motor related neural mechanisms has recently

17   been characterised. Kinaesthetic MI ability may be assessed by measuring changes in CM

18   excitability and ANS parameters during MI. It is presumed that the closer these changes

19   match with those seen during movement execution the better the imagery abilities of an

20   individual. MSI, the mental suppression of movement, may be a related MI ability, but the

21   nature and relevance of this construct needs to be investigated and validated. Theoretically,

22   both phenomena may contribute to the subjective experiences and behaviours observed with

23   direct and challenge motor suggestions.

1

2   Current evidence supports a role of kinaesthetic MI and MSI in the pathophysiology of motor

3   CD weakness and paralysis. Evidence supports an association between hypnotic

4   susceptibility and CD, and common neurological mechanisms underlying hypnotically

5   induced and motor CD weakness or paralysis. Furthermore, differences in performance of

6   kinaesthetic MI tasks have been observed in patients suffering from motor CD relative to

7   healthy controls. Accordingly, both kinaesthetic MI and MSI may contribute to the

8   subjective experiences and behaviours observed in motor CD.

9

10 It is important to investigate these possible relationships because the answers may provide a

11 significant advance in our understanding of CD, a chronic and disabling disorder, and

12 advance our understanding of the intriguing behaviours and subjective experiences associated

13 with hypnosis and the underlying sense of agency driving behaviour.

1 Table 1. The relationship between imagery abilities and hypnotic susceptibility.

Imagery Rating Scale	Imagery Modality Assessed	Hypnotic Susceptibility Rating Scale	Relationship with Hypnotic Susceptibility
Vividness of Visual Imagery Questionnaire (Marks, 1973)	Visual imagery	Harvard Group Scale of Hypnotic Susceptibility Form A (HGSHS:A) (Shor & Orne, 1962)	Correlation with susceptibility score, Pearson's r = 0.08 (Glisky, et al., 1995)
Vividness of Movement Imagery Questionnaire (Isaac, et al., 1986)	Kinaesthetic motor imagery	HGSHS:A	No correlation with susceptibility score (Glisky, et al., 1995)
Test of Visual Imagery Control (Gordon, 1949)	Visual imagery	HGSHS:A	No correlation with susceptibility score (Glisky, et al., 1995)
Shortened form of the Betts' Questionnaire Upon Mental Imagery (Sheehan, 1967)	Visual, auditory, cutaneous, kinaesthetic, gustatory, olfactory, organic	SHSS:C	No correlation with susceptibility scores (Perry, 1973)
Betts' Questionnaire Upon Mental Imagery (Betts, 1909)	Visual, auditory, cutaneous, kinaesthetic, gustatory, olfactory, organic	SHSS:C	No difference in imagery scores between groups of high and low susceptibility (Carli, et al., 2007)
Vividness and Control of Imagery Scale (Coe, et al., 1980)	Visual imagery	Modified SHSS:C	Correlation with susceptibility scores, Pearson's r = 0.33 (Coe, et al., 1980)
Mental imagery of visual scenes (Farthing, et al., 1983)	Visual imagery	HGSHS:A	Correlation with susceptibility score, Pearson's r = 0.32 (Farthing, et al., 1983)

|			
Mental imagery of personal actions questionnaire (Farthing, et al., 1983)	Kinaesthetic motor imagery	HGSHS:A	Correlation with susceptibility score, Pearson's r = 0.24 (Farthing, et al., 1983)
Visual imagery scale (Palmer & Field, 1968)	Visual imagery	Stanford Hypnotic Susceptibility Scale Form A (Weitzenhoffer & Hilgard, 1959)	Correlation with susceptibility score, Pearson's r = 0.274 (Palmer & Field, 1968)

1
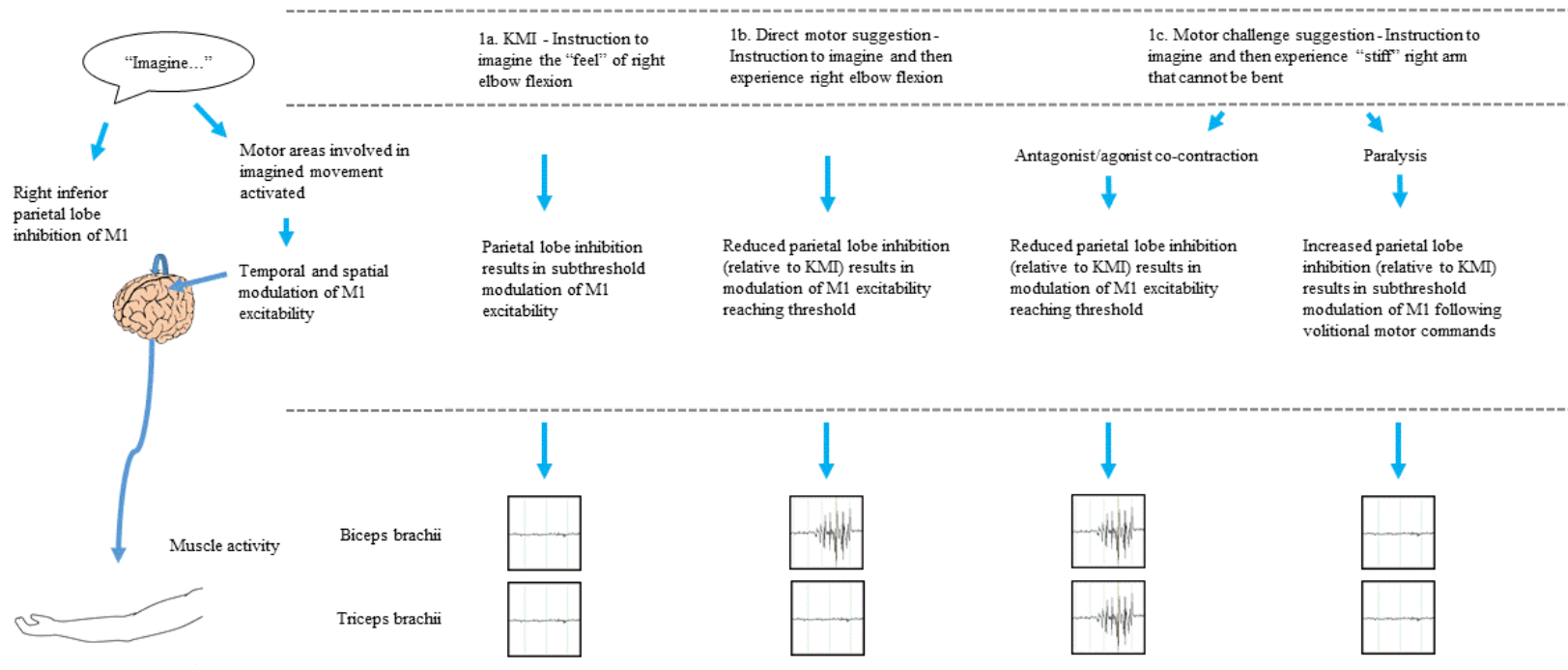
Figure 1. Two column figure.

1

1   Figure 1: Neural processes underlying different types of motor imagery. 1a. Kinaesthetic MI (KMI).   Contralateral inferior parietal lobe

2   inhibition results in subthreshold modulation of M1 excitation, preventing the upstream motor pathways from generating movement. 1b. Direct

3   motor suggestion reduces parietal lobe inhibition of the contralateral M1, resulting in threshold modulation of M1 excitability and subsequent

4   "spontaneous" occurrence of the imagined movement. 1c. Motor challenge suggestion. Participants use one of two strategies, antagonist/agonist

5   co-contraction or paralysis. Use of an antagonist/agonist co-contraction strategy activates both muscle groups in the same manner as a direct

6   motor suggestion, rendering the arm rigid. Use of a paralysis strategy increases parietal lobe inhibition of M1, or Motor Suppression Imagery,

7   preventing upstream volitional motor commands from generating movement.

8

1   References

2   American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders* (3
3   ed, revised ed.). Washington DC.: American Psychiatric Press
4   American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5
5   ed.). Washington, DC.: American Psychiatric Association.
6   Arnold, M. B. (1946). On the mechanism of suggestion and hypnosis. *The Journal of Abnormal &*
7   *Social Psychology, 41*, 107-128.
8   Barber, T. X., & Glass, L. B. (1962). Significant factors in hypnotic behavior. *The Journal of Abnormal*
9   *& Social Psychology, 64*, 222-228.
10   Barnier, A. J., Dienes, Z., & Mitchell, C. J. (2008). How hypnosis happens: new cognitive theories of
11   hypnotic responding. In M. R. Nash & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis.*
12   *Theory, Research and Practice* (pp. 142-177). Oxford, United Kingdom.: Oxford University
13   Press.
14   Barnier, A. J., & McConkey, K. M. (2004). Defining and identifying the highly hypnotizable person. In
15   M. Heap, R. J. Brown & D. A. Oakley (Eds.), *The Highly Hypnotizable Person: Theoretical,*
16   *experimental and clinical issues* (pp. 30-60). East Sussex, United Kingdom: Brunner-
17   Routledge.
18   Bell, V., Oakley, D. A., Halligan, P. W., & Deeley, Q. (2011). Dissociation in hysteria and hypnosis:
19   evidence from cognitive neuroscience. *Journal of Neurology, Neurosurgery & Psychiatry, 82*,
20   332-339.
21   Betts, G. H. (1909). The distribution and functions of mental imagery. *Teachers College Contributions*
22   *to Education, 26*.
23   Blakemore, R. L., Hyland, B. I., Hammond-Tooke, G. D., & Anson, J. G. (2015). Deficit in late-stage
24   contingent negative variation provides evidence for disrupted movement preparation in
25   patients with conversion paresis. *Biological Psychology, 109*, 73-85.
26   Bliss, E. L. (1984). Hysteria and hypnosis. *The Journal of Nervous and Mental Disease, 172*, 203-206.
27   Bonnard, M., Spieser, L., Meziane, H. B., De Graaf, J. B., & Pailhous, J. (2009). Prior intention can
28   locally tune inhibitory processes in the primary motor cortex: direct evidence from
29   combined TMS-EEG. *European Journal of Neuroscience, 30*, 913-923.
30   Bowers, K. S. (1992). Imagination and Dissociation in Hypnotic Responding. *International Journal of*
31   *Clinical and Experimental Hypnosis, 40*, 253-275.
32   Braffman, W., & Kirsch, I. (1999). Imaginative suggestibility and hypnotizability: an emperical
33   analysis. *Journal of Personality and Social Psychology, 77*, 578-587.
34   Bramwell, J. M. (1906). *Hypnotism. Its History, Practice and Theory* (2 ed.). London: De La More
35   Press.
36   Campos, A., & Perez, M. J. (1990). A factor analytic study of two measures of mental imagery.
37   *Perceptual and Motor Skills, 71*, 995-1001.
38   Carli, G., Cavallaro, E., & Santarcangelo, E. L. (2007). Hypnotizability and imagery modality
39   preference: do highs and lows live in the same world? *Contemporary Hypnosis, 24*, 64-75.
40   Coe, W. C., St Jean, R. L., & Burger, J. M. (1980). Hypnosis and the enhancement of visual imagery.
41   *International Journal of Clinical and Experimental Hypnosis, 28*, 225-243.
42   Cojan, Y., Waber, L., Carruzzo, A., & Vuilleumier, P. (2009a). Motor inhibition in hysterical conversion
43   paralysis. *NeuroImage, 47*, 1026-1037.
44   Cojan, Y., Waber, L., Schwartz, S., Rossier, L., Forster, A., & Vuilleumier, P. (2009b). The brain under
45   self-control: Modulation of inhibitory and monitoring cortical networks during hypnotic
46   paralysis. *Neuron, 62*, 862-875.
47   Collet, C., & Guillot, A. (2010). Autonomic nervous system activies during imagined movements. In A.
48   Guillot & C. Collet (Eds.), *The Neurophysiological Foundations of Motor Imagery* (pp. 95-108).
49   Oxford, United Kingdom: Oxford University Press.

Collet, C., Guillot, A., Lebon, F., MacIntyre, T., & Moran, A. (2011). Measuring motor imagery using psychometric, behavioral, and psychophysiological tools. *Exercise and Sport Sciences Reviews, 39*, 85-92.

Comey, G., & Kirsch, I. (1999). Intentional and spontaneous imagery in hypnosis: The phenomenology of hypnotic responding. *International Journal of Clinical and Experimental Hypnosis, 47*, 65-85.

Cooper, L. A., & Shepard, R. N. (1975). Mental transformations in the identification of left and right hands. *The Journal of Experimental Psychology: Human Perception and Performance, 104*, 48-56.

de Lange, F. P., Roelofs, K., & Toni, I. (2007). Increased self-monitoring during imagined movements in conversion paralysis. *Neuropsychologia, 45*, 2051-2058.

de Lange, F. P., Roelofs, K., & Toni, I. (2008). Motor imagery: a window into the mechanisms and alterations of the motor system. *Cortex, 44*, 494-506.

Decety, J., Jeannerod, M., Germain, M., & Pastene, J. (1991). Vegetative response during imagined movement is proportional to mental effort. *Behavioural Brain Research, 42*, 1-5.

Deeley, Q., Oakley, D. A., Toone, B., Bell, V., Walsh, E., Marquand, A. F., Giampietro, V., Brammer, M. J., Williams, S. C. R., Mehta, M. A., & Halligan, P. W. (2013). The functional anatomy of suggested limb paralysis. *Cortex, 49*, 411-422.

Edwards, M. J., Stone, J., & Lang, A. E. (2014). From psychogenic movement disorder to functional movement disorder: It's time to change the name. *Movement Disorders, 29*, 849-852.

Fahn, S., & Olanow, C. W. (2014). "Psychogenic Movement Disorders": They Are What They Are. *Movement Disorders, 29*, 853-856.

Farthing, G. W., Venturino, M., & Brown, S. W. (1983). Relationship between two different types of imagery vividness questionnaire items and three Hypnotic Susceptibility Scale factors: a brief communication. *The International Journal of Clinical and Experimental Hypnosis, 31*, 8-13.

Franklin, B., Majault, Roy, L., Sallin, Bailly, J.-S., D'Arcet, Bory, D., Guillotin, J.-I., & Lavoisier, A. (2002). Report of the commissioners charged by the king with the examination of animal magnetism. *International Journal of Clinical and Experimental Hypnosis, 50*, 332-363.

Gallagher, S. (2000). Philosophical conceptions of the self: implications for cognitive science. *Trends in Cognitive Sciences, 4*, 14-21.

Gelauff, J., Stone, J., Edwards, M., & Carson, A. (2014). The prognosis of functional (psychogenic) motor symptoms: a systematic review. *Journal of Neurology, Neurosurgery & Psychiatry, 85*, 220-226.

Glisky, M. L., Tataryn, D. J., & Kihlstrom, J. F. (1995). Hypnotizability and Mental Imagery. *International Journal of Clinical and Experimental Hypnosis, 43*, 34-54.

Gordon, R. (1949). An investigation into some of the factors that favour the formation of stereotyped images. *The British Journal of Psychology. General Section, 39*, 156-167.

Guillot, A., Collet, C., Nguyen, V. A., Malouin, F., Richards, C., & Doyon, J. (2009). Brain activity during visual versus kinesthetic imagery: an fMRI study. *Human Brain Mapping, 30*, 2157-2172.

Hall, C. R., & Martin, K. A. (1997). Measuring movement imagery abilities: a revision of the movement imagery questionnaire. *Journal of Mental Imagery, 21*, 143-154.

Halligan, P. W. (2011). Psychogenic movement disorders: illness in search of disease? In M. Hallett, A. E. Lang, J. Jankovic, S. Fahn, P. W. Halligan, V. Voon & C. R. Cloninger (Eds.), *Psychogenic Movement Disorders and Other Conversion Disorders* (pp. 120-133). Cambridge, UK: Cambridge University Press.

Halligan, P. W., Athwal, B. S., Oakley, D. A., & Frackowiak, R. S. J. (2000). Imaging hypnotic paralysis: Implications for conversion hysteria. *The Lancet, 355*, 986-987.

Hilgard, E. R., & Tart, C. T. (1966). Responsiveness to suggestions following waking and imagination instructions and following induction of hypnosis. *Journal of Abnormal Psychology, 71*, 196-208.

Isaac, A., Marks, D. F., & Russell, D. G. (1986). An instrument for assessing imagery of movement: The Vividness of Movement Imagery Questionnare (VMIQ). *Journal of Mental Imagery, 10*, 23-30.

Jeannerod, M. (1994). The representing brain: Neural correlates of motor intention and imagery. *Behavioral and Brain Sciences, 17*, 187-245.

Jeannerod, M. (2001). Neural Simulation of Action: A Unifying Mechanism for Motor Cognition. *NeuroImage, 14*, S103-S109.

Kanaan, R., Armstrong, D., Barnes, P., & Wessely, S. (2009). In the psychiatrist's chair: how neurologists understand conversion disorder. *Brain, 132*, 2889-2896.

Kirsch, I. (1997). Suggestibility or hypnosis: what do our scales really measure? *The International Journal of Clinical and Experimental Hypnosis, 45*, 212-225.

Kirsch, I., & Council, J. R. (1992). Situational and Personality Correlates of Hypnotic Responsiveness. In E. Fromm & M. R. Nash (Eds.), *Contemporary Hypnosis Research*. New York, New York, United States of America: The Guilford Press.

Kirsch, I., & Lynn, S. J. (1999). Automaticity in Clinical Psychology. *American Psychologist, 54*, 504-515.

Kirsch, I., Silva, C. E., Carone, J. E., Johnston, J. D., & Simon, B. (1989). The surreptitious observation design: an experimental paradigm for distinguishing artifact from essence in hypnosis. *Journal of Abnormal Psychology, 98*, 132-136.

Kogon, M. M., Jasiukaitis, P., Berardi, A., Gupta, M., Kosslyn, S. M., & Spiegel, D. (1998). Imagery and hypnotizability revisited. *The International Journal of Clinical and Experimental Hypnosis, 46*, 363-370.

Kosslyn, S. M., DiGirolamo, G. J., Thompson, W. L., & Alpert, N. M. (1998). Mental rotation of objects versus hands: neural mechanisms revealed by positron emission tomography. *Psychophysiology, 35*, 151-161.

Kosslyn, S. M., Ganis, G., & Thompson, W. L. (2010). Multimodal images in the brain. In A. Guillot & C. Collet (Eds.), *The Neurophysiological Foundations of Mental and Motor Imagery* (pp. 3-16). Oxford, United Kingdom: Oxford University Press.

Kuyk, J., Spinhoven, P., & van Dyck, R. (1999). Hypnotic recall: a positive criterion in the differential diagnosis between epileptic and pseudoepileptic seizures. *Epilepsia, 40*, 485-491.

Lebon, F., Byblow, W. D., Collet, C., Guillot, A., & Stinear, C. M. (2012a). The modulation of motor cortex excitability during motor imagery depends on imagery quality. *European Journal of Neuroscience, 35*, 323-331.

Lebon, F., Lotze, M., Stinear, C. M., & Byblow, W. D. (2012b). Task-dependent interaction between parietal and contralateral primary motor cortex during explicit versus implicit motor imagery. *PLoS One, 7*, e37850.

Lequerica, A., Rapport, L., Axelrod, B. N., Telmet, K., & Whitman, R. D. (2002). Subjective and objective assessment methods of mental imagery control: construct validation of self-report measures. *Journal of Clinical and Experimental Neuropsychology, 24*, 1103-1116.

Liepert, J., Hassa, T., Tuscher, O., & Schmidt, R. (2008). Electrophysiological correlates of motor conversion disorder. *Movement Disorders, 23*, 2171-2176.

Liepert, J., Hassa, T., Tuscher, O., & Schmidt, R. (2009). Abnormal motor excitability in patients with psychogenic paresis. A TMS study. *Journal of Neurology, 256*, 121-126.

Liepert, J., Hassa, T., Tuscher, O., & Schmidt, R. (2011). Motor excitability during movement imagination and movement observation in psychogenic lower limb paresis. *Journal of Psychosomatic Research, 70*, 59-65.

Lynn, S. J., Kirsch, I., & Hallquist, M. N. (2008). Social cognitive theories of hypnosis. In M. R. Nash & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis. Theory, Research and Practice* (pp. 111-139). Oxford, United Kingdom.: Oxford University Press.

Marks, D. F. (1973). Visual imagery differences in the recall of pictures. *British Journal of Psychology, 64*, 17.

Marshall, J. C., Halligan, P. W., Fink, G. R., Wade, D. T., & Frackowiak, R. S. (1997). The functional anatomy of a hysterical paralysis. *Cognition, 64*, B1-8.

McBain, W. N. (1954). Imagery and suggestibility: A test of the Arnold hypothesis. *Journal of Abnormal Psychology, 49*, 36-44.

Milton, J., Small, S. L., & Solodkin, A. (2008). Imaging motor imagery: methodological issues related to expertise. *Methods, 45*, 336-341.

Moene, F. C., Spinhoven, P., Hoogduin, K., Sandyck, P., & Roelofs, K. (2001). Hypnotizability, dissociation and trauma in patients with a conversion disorder: an exploratory study. *Clinical Psychology & Psychotherapy, 8*, 400-410.

Neuper, C., & Pfurtscheller, G. (2010). Electroencephalographic characteristics during motor imagery. In A. Guillot & C. Collet (Eds.), *The Neurophysiological Foundations of Mental and Motor Imagery* (pp. 65-81). Oxford, United Kingdom: Oxford University Press.

Oakley, D. A. (1999). Hypnosis and Conversion Hysteria: A Unifying Model. *Cognitive Neuropsychiatry, 4*, 243-265.

Palmer, R. D., & Field, P. B. (1968). Visual imagery and susceptibility to hypnosis. *Journal of Consulting and Clinical Psychology, 32*, 456-461.

Perry, C. (1973). Imagery, fantasy, and hypnotic susceptibility: A multidimensional approach. *Journal of Personality and Social Psychology, 26*, 217-221.

Piccione, C., Hilgard, E. R., & Zimbardo, P. G. (1989). On the degree of stability of measured hypnotizability over a 25-year period. *Journal of Personality and Social Psychology, 56*, 289-295.

Roelofs, K., Hoogduin, K. A., & Keijsers, G. P. (2002a). Motor imagery during hypnotic arm paralysis in high and low hypnotizable subjects. *International Journal of Clinical and Experimental Hypnosis, 50*, 51-66.

Roelofs, K., Hoogduin, K. A., Keijsers, G. P., Naring, G. W. B., Moene, F. C., & Sandijck, P. (2002b). Hypnotic susceptibility in patients with Conversion Disorder. *Journal of Abnormal Psychology, 111*, 390-395.

Roelofs, K., Näring, G. W. B., Keijsers, G. P. J., Hoogduin, C. A. L., Van Galen, G. P., & Maris, E. (2001). Motor imagery in conversion paralysis. *Cognitive Neuropsychiatry, 6*, 21-40.

Schwoebel, J., Boronat, C. B., & Branch Coslett, H. (2002). The man who executed "imagined" movements: evidence for dissociable components of the body schema. *Brain and Cognition, 50*, 1-16.

Sheehan, P. W. (1967). A shortened form of Betts' questionnaire upon mental imagery. *Journal of Clinical Psychology, 23*, 386-389.

Shepard, R. N., & Metzler, J. (1971). Mental Rotation of Three-Dimensional Objects. *Science, 171*, 701-703.

Shor, R. E., & Orne, E. C. (1962). Harvard Group Scale of Hypnotic Susceptibility: Form A. In. Palo Alto, California: Consulting Psychologists Press.

Sohn, Y. H., Dang, N., & Hallett, M. (2003). Suppression of Corticospinal Excitability During Negative Motor Imagery. *Journal of Neurophysiology, 90*, 2303-2309.

Spence, S. A., Crimlisk, H. L., Cope, H., Ron, M. A., & Grasby, P. M. (2000). Discrete neurophysiological correlates in prefrontal cortex during hysterical and feigned disorder of movement. *Lancet, 355*, 1243-1244.

Stinear, C. M. (2010). Corticospinal facilitation during motor imagery. In A. Guillot & C. Collet (Eds.), *The Neurophysiological Foundations of Mental and Motor Imagery* (pp. 47-61). Oxford, United Kingdom: Oxford University Press.

Stinear, C. M., Byblow, W. D., Steyvers, M., Levin, O., & Swinnen, S. P. (2006). Kinesthetic, but not visual, motor imagery modulates corticomotor excitability. *Experimental Brain Research, 168*, 157-164.

Stone, J., Carson, A., Duncan, R., Coleman, R., Roberts, R., Warlow, C., Hibberd, C., Murray, G., Cull, R., Pelosi, A., Cavanagh, J., Matthews, K., Goldbeck, R., Smyth, R., Walker, J., MacMahon, A. D., & Sharpe, M. (2009). Symptoms 'unexplained by organic disease' in 1144 new neurology out-patients: how often does the diagnosis change at follow-up? *Brain, 132*, 2878-2888.

Stone, J., LaFrance, W. C., Brown, R. J., Spiegel, D., Levenson, J. L., & Sharpe, M. (2011). Conversion Disorder: Current problems and potential solutions for DSM-5. *Journal of Psychosomatic Research, 71*, 369-376.

Takarada, Y., & Nozaki, D. (2014). Hypnotic suggestion alters the state of the motor cortex. *Neurosci Res, 85*, 28-32.

Tian, X., & Poeppel, D. (2012). Mental imagery of speech: linking motor and perceptual systems through internal simulation and estimation. *Frontiers in Human Neuroscience, 6*, 314.

Vuilleumier, P. (2014). Brian circuits implicated in psychogenic paralysis in conversion disorders and hypnosis. *Neurophysiologie Clinique/Clinical Neurophysiology, 44*, 323-337.

Wang, Y., & Morgan, W. P. (1992). The effect of imagery perspectives on the psychophysiological responses to imagined exercise. *Behavioural Brain Research, 52*, 167-174.

Ward, N. S., Oakley, D. A., Frackowiak, R. S. J., & Halligan, P. W. (2003). Differential brain activations during intentionally simulated and subjectively experienced paralysis. *Cognitive Neuropsychiatry, 8*, 295-312.

Weitzenhoffer, A. M., & Hilgard, E. R. (1959). Stanford Hypnotic Susceptibility Scale, Forms A and B. In. Palo Alto, CA: Consulting Psychologists Press.

Weitzenhoffer, A. M., & Hilgard, E. R. (1962). Stanford Hypnotic Susceptibility Scale: Form C. In. Palo Alto, California: Consulting Psychologists Press.

Weitzenhoffer, A. M., & Sjoberg, B. M., Jr. (1961). Suggestibility with and without "induction of hypnosis". *The Journal of Nervous and Mental Disease, 132*, 204-220.

Williamson, J. W., McColl, R., Mathews, D., Mitchell, J. H., Raven, P. B., & Morgan, W. P. (2002). Brain activation by central command during actual and imagined handgrip under hypnosis. *Journal of Applied Physiology, 92*, 1317-1324.

Winkel, J. D., Younger, J. W., Tomcik, N., Borckardt, J. J., & Nash, M. R. (2006). Anatomy of a hypnotic response: Self-report estimates, actual behavior, and physiological response to the hypnotic suggestion for arm rigidity. *The International Journal of Clinical and Experimental Hypnosis, 54*, 186-205.

Woody, E. Z., & Barnier, A. J. (2008). Hypnosis scales for the twenty-first century: what do we need and how should we use them. In M. R. Nash & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis. Theory, Research and Practice* (First ed., pp. 255-281). Oxford, United Kingdom.: Oxford University Press.

Woody, E. Z., Barnier, A. J., & McConkey, K. M. (2005). Multiple hypnotizabilities: differentiating the building blocks of hypnotic response. *Psychological Assessment, 17*, 200-211.

Woody, E. Z., & Sadler, P. (2008). Dissociation theories of hypnosis. In M. R. Nash & A. J. Barnier (Eds.), *The Oxford Handbook of Hypnosis. Theory, Research and Practice* (pp. 81-110). Oxford, United Kingdom.: Oxford University Press.

Zamansky, H. S., & Ruehle, B. L. (1995). Making hypnosis happen: The involuntariness of the hypnotic experience. *International Journal of Clinical and Experimental Hypnosis, 43*, 386-398.