Contemporary Hypnosis
22(4): 184–192 (2005)
DOI: 10.1002/ch.12

FUNCTIONAL CORRELATES OF CONVERSION AND HYPNOTIC PARALYSIS: A NEUROPHYSIOLOGICAL HYPOTHESIS

Felipe A. Tallabs

Centro de Rehabilitación y Educación Especial (CREE) department of neuropsychology, Monterrey, Mexico

Abstract

Since conversion disorder was thoroughly described by Pierre Janet psychiatric findings have traditionally been the only available source of contribution to the clarification of the phenomena. Some recent cognitive approaches though, have tried to explain conversion paralysis in terms of a disconnection phenomenon supposedly causative of degeneration in motor information processes. These approaches though relevant are purely theoretical, and only in recent years has neurological research provided sufficient evidence to commence developing a neuropsychological theory of conversion paralysis. Defined cortical regions like the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC) have been found to be related to conversion and hypnotic paralysis, while on the other hand, psychological evidence linking hypnotizability and conversion has also emerged. This paper attempts to relate some neurological and psychological evidence in a first step towards a general theory of conversion paralysis. Copyright © 2005 British Society of Experimental & Clinical Hypnosis. Published by John Wiley & Sons, Ltd.

Key words: anterior cingulate cortex, conversion paralysis, hypnotic paralysis, orbitofrontal cortex

Introduction

Conversion disorder has travelled through time as a riddle, possibly behind the phenomena of miraculous healing. It has always wandered on a thin line between psychiatry and neurology. The classical term 'Hysteria' made reference to a plethora of unclassified conditions that are acknowledged today as dissociative, somatoform, conversion, borderline and post-traumatic stress disorder.

It was Pierre Janet who in 1906 masterfully described hysteria in a series of lectures later published as *The Major Symptoms of Hysteria*(1907). Janet gathered these apparently unconnected conditions under a single rubric. He speculated based on experimentation with hypnosis and personal observation, that they shared a fundamental mechanism: dissociation. He also believed that hysterical conditions developed out of traumatic experiences that caused autohypnotic phenomena, ultimately responsible for hysteria (Butler, Duran, Jasiukaitis, Koopman and Spiegel, 1996). Contemporary theorists agree that conversion may result from spontaneous self-hypnosis involving a dissociation of sensory or motor function as a reaction to trauma or prolonged stress (Hilgard, 1977; Bliss, 1984; Kihlstrom, 1992; Oakley, 1999).

Butler et al. (1996: 45) described hypnotizability as a 'psychological predisposition or vulnerability (which may be biological in origin) to dissociative states under traumatic or stressful environmental conditions'. Evidence of the relationship between severe traumatic events and development of dissociative symptomatology is considerable (Spiegel, 1984, 1988; Frankel, 1990; Marmar, Weiss, Schlenger, Fairbank, Jorda, Kulka and Hough, 1994 ; Koopman, Classen, Cardena and Spiegel, 1995). However, little empirical evidence existed that could relate hypnotic susceptibility to conversion (Frischholz, Lipman, Braun and Sachs, 1992), until Roelofs, Hoogduin, Keijsers, Naring, Moene and Sandijck (2002a) provided hard evidence of this by comparing several conversion patients with control subjects on measures of hypnotic susceptibility, cognitive dissociation, and somatoform dissociation. Conversion patients were significantly more responsive to hypnotic suggestions than controls, however, this important result awaits independent replication.

Hypnosis can be explained as a controlled and structured dissociation (Kaplan, 1985) with a state of excessive focal concentration and relative suspension of peripheral awareness (absorption) and suspension of critical contextual evaluation (suggestibility) (Spiegel and Cardena, 1990). Absorption is a state of highly focused attention with a total involvement in a single dimension of experience, like perception, memory or ideation (Tellegen and Atkinson, 1974; Spiegel, 1992). This focalization excludes other experiences that should normally be present in conscious awareness. Janet described this as a 'retraction in the field of consciousness' (1907). This retraction requires the relegation of material to the periphery of consciousness where it no longer impinges on awareness. This aspect of hypnosis is considered a dissociation of content (Spiegel, 1990; Butler et al., 1996). Suggestibility is an increased responsiveness to environmental cues (real or perceived) and is a main characteristic of hypnosis, and is thought to result from the heightened focal awareness through absorption. A person under instructions of hypnosis 'is fully absorbed in only one or two aspects of awareness, and therefore is less likely to critically judge or evaluate the meaning of the experience' (Spiegel, 1992: 23). The narrowing of attention results in a diminution of higher order critical capacities; therefore a proneness to be influenced by suggestion develops.

Recent approaches to conversion paralysis

Conversion paralysis can be described as the inability to move a limb by volition in the absence of any neurological condition or physical causes. Instead psychological trauma and/or prolonged exposure to stressful situations are linked to the onset of the symptoms (Roelofs, van Galen, Keijsers and Hoogduin, 2002b). Theorists like Kihlstrom consider conversion paralysis as a dissociation between lower-level and higher-level information processes. According to this author the explicit or higher-level intentional information processes are impaired, whereas the implicit or automatic processes remain intact (Kihlstrom, 1992). Similar dissociation processes can be observed in hypnotically induced paralysis (Roelofs, Hoogduin and Keijsers 2002c). Shallice and Burgess (1998) proposed the 'supervisory attentional system' (SAS) to describe a high-level action control device based in the frontal cortex that is concerned with monitoring activity and modulating behavior when automatic routines are insufficient, i.e. in novel situations. According to their model, well learned behaviour is controlled by low-level action mechanisms, which can be activated by environmental stimuli, but if the situation involves tasks that require conscious awareness the SAS can override this automatic process. Conversion paralysis has been observed to affect patients when explicit tasks involving consciousness are attempted (Roelofs et al. 2002b), providing evidence of high level control processing, like the SAS, becoming dissociated from low-level action control.

Published by John Wiley & Sons, Ltd

Oakley (1999) has proposed a unifying model of conversion paralysis and hypnotically induced paralysis. According to this theory, hypnotic and conversion paralysis 'involves the inhibition of motor and sensory functioning on a high cognitive level of information processing'. The inhibition in hypnosis is a result of heterosuggestion, while in conversion it is thought to result from autosuggestion. This link between hypnotic and conversion paralysis is consistent with findings of similar activity in specific brain areas involved in both cases. Two recent brain-mapping studies show that the same brain structures are involved in conversion paralysis (Marshall, Halligan, Fink, Wade and Frackowiak, 1997) and hypnotically suggested paralysis (Halligan, Atwal, Oakley and Frackowiak 2000). In both studies the right primary motor cortex experienced a decrease in activity while the right anterior cingulate cortex (ACC) and the right orbitofrontal cortex (OFC) showed increased activity. In both cases conscious (willed) attempts to move the paralyzed (left) leg triggered paralysis, while unconscious attempts, i.e. during sleep, were successful. Although there are marked similarities between hypnotic and conversion paralysis, there is a fundamental difference. During hypnosis the subject is able to end the experience at any time. It is voluntary. While in conversion the onset of the symptoms is completely involuntary and always surrounded by difficult emotional circumstances.

Functional aspects of ACC

Apparently the ACC and the OFC develop some sort of functional inhibition of voluntary actions in the affected limb. Electrical stimulation studies of these areas have provided evidence related to motor inhibition of spontaneous movements (the so-called arrest reaction; Kaada, 1960; Lüders, Dinner, Morris, Wyllie and Comair, 1995). Devinsky, Morrell and Vogt (1995) have also implicated these regions in action, emotion and motor inhibition. The question that remains is what the role of these regions during hypnotic and conversion paralysis is. Some believe that the ACC is the disconnecting element of premotor and prefrontal cortex from primary motor cortex (Marshall, Halligan, Fink, Wade and Frackowiak, 1997), while the OFC is the distal source of unconscious inhibition (Fuster, 1981). However, there is no current description of how these areas could be implicated.

The ACC has been related to attention demanding tasks (Posner, Petersen, Fox and Raichle, 1988; Pardo, Pardo, Janer and Raichle, 1990) and more specifically it has been found to be involved in selective attentional processes. Using an oddball paradigm, Kropotov, Näätänen, Sevostianov, Alho, Reinikainen and Kropotova (1995) experimented with patients in two conditions: selective attention (attending deviant tones by pressing a button) and passive attention (read a book and ignore the tones). A strong auditory event related potential (ERP) complex was observed in ACC during response to deviant stimuli, while in passive attention no such activation was observed. This area has also been found to be active during pain perception. Kropotov, Crawford and Polyakov (1997) recording somatosensory event related potential (SERPs) with intracranial electrodes, found that hypnotizable subjects show activity in the ACC during hypnotically induced analgesia. This is consistent with findings using CO^2 laser heat pulses to elicit painful laser evoked potentials (LEPs) over the skin (Tarkka and Treede, 1993). This study reported evidence of ACC activity during painful stimuli, also consistent with positron emission tomography (PET) studies that implicated the involvement of ACC in painful stimulation (Jones, Brown, Friston, Qi and Frackowiak, 1991; Talbot, Marret, Evans, Meyer, Bushnell and Duncan, 1991 ; Casey, Minoshima, Berger, Koeppe, Morrow and Frey,

Published by John Wiley & Sons, Ltd

1994 ; Coghill, Talbot, Evans, Meyer, Gjedde, Bushnell and Duncan, 1994; Derbyshire, Jones, Devani, Friston, Feinmann, Harris, Pearce, Watson and Frackowiak, 1994). Devinsky et al. (1995) have suggested that the ACC could generate an affective component of pain. This is consistent with studies of patients with ablations to the ACC who reported sensory perception of pain with a lack of discomfort or distress (Foltz and White, 1968; Ballantine, Levy, Dagi and Giriunas, 1975). Kropotov et al. (1997: 4) also suggested that the ACC could be an area that 'organizes responses to noxious stimuli and is impacted by strategies for relieving pain'. Integrating the evidence presented above we can see that the ACC is an area related to active selective attention as well as to processing pain, and as we will see next, they could represent interconnected processes during conversion paralysis. The ACC has also been strongly associated with conflict resolution (Botvinick, Cohen and Carter, 2004), which offers another possible perspective on conversion paralysis, one not undertaken here.

The only characteristic of conversion paralysis that is not shared by hypnotic paralysis is a traumatic event and/or a prolonged exposure to a stressful situation. This could also indicate that during conversion paralysis stress monopolizes attention, driving it to become focused and transformed into a stressful situation, and an analogue to an hypnotic suggestion (focused attention as explained at the end of the introduction is a fundamental characteristic of hypnosis) thus disregarding peripheral awareness, and causing dissociative motor dysfunction, i.e. hemiparalysis. Following this line of thought, it is likely that as with pain, stress is tagged or classified by the ACC as a noxious stimulus. It is then feasible that the capacity of the ACC to respond to pain is dependent on ability to emotionally value stimuli as stressful, whether these stimuli are external (physical) or internal (psychological). Once a stimulus is tagged as stressful, a proportional level of absorption (high level of focused attention), depending on hypnotizability, is set. Empirical evidence regarding this process is awaited. It is then the control over the intensity of emotion attached to attention what gives the ACC all its 'power'.

To understand how the ACC manipulation of attentional intensity becomes a key element in conversion and hypnotic paralysis, certain aspects about the function of the ACC are required. The ACC is active in everyday life when we need, for example, controlled, distributed attention, such as listening to someone's voice while driving a car. According to Ratey (2001) the (ACC) tags incoming information with ratings on which items should be mulled over and which forgotten, allocating frontal lobe resources accordingly.

The ACC seems to coordinate the level of arousal, emotion and motivation feeding the attentional system through its extensive neural connections to regions involved in attention throughout the brain, and the key to this widespread power of coordination is the ability of the ACC to regulate its own dopamine levels, which enhance the reactivity of neural connections (Ratey, 2001).

There are two elements of attention strongly influenced by ACC; the first is coordination, which as explained above, depends on neural connections to brain regions that integrate attention. The second is intensity of attention; emotional tagging accomplishes this:

> The primary emotional signal the ACC receives comes from the amygdala, at the core of the limbic system, which influences attention by assigning emotional significance to incoming information. Even before a sensory perception has reached the frontal lobes, where it enters conscious awareness and undergoes fine categorization, the amygdala has already branded it with a raw emotional valence somewhere along a continuum from mildly interesting to 'Oh my God'. (Ratey 2001: 121)

The dependency of the attentional system on emotion and motivation is controlled by ACC. Through its dopamine connections, it receives afferent information from the basal ganglia and the ventral pallidum concerning conditioned incentives and their amplification by dopamine (LeDoux, 2002). It also has efferent connections with the accumbens (Groenewegen, Wright and Uylings, 1997). Everitt and Robbins (1999) demonstrated that connections between the amygdala and accumbens are the key to the ability to motivate new learning through conditioned incentives. It is likely then that the ACC controls learning of emotions: first it modulates the amygdala through the accumbens, then this information is paired to the general state of arousal received from the tegmentum (LeDoux, 2002) and stored in episodic memory. In this fashion a certain experience is registered as painful or pleasurable, and a person learns to associate positive or negative emotions with an event.

Conversion and hypnotic paralysis

The only characteristic related to the ACC that is evidently not consistent throughout the population is hypnotizability. This is most probably due to a hypersensibility of certain people in ACC regions processing emotional information under autosuggestion or hetero-suggestion. This hypersensibility to suggestive information triggers a disconnection of the affective subdivision (ACad) of the ACC from the cognitive subdivision (ACcd). These two subdivisions described accurately by Bush, Luu and Posner (2000) are found bilaterally but apparently the cognitive division is left side dominant while the affective division is right side dominant. Once a right side affective disconnection is activated (by inhibiting its ipsilateral cognitive counterpart), the left side becomes inactive; there is evidence of this disconnection during hypnotic suggestion (for example, see Maquet, Faymonville, Deguelde, Delfiore, Franck, Luxen and Lamy 1999). Neurophysiological studies carried by Gruzelier (1998) provided first evidence that a disconnection between cognitive and affective subdivisions in ACC is an important element for hypnosis. Also Corrigan, (2002: 12) explains: ‘The unilateral right-sided activation of BA24 or BA32 (ACC affective areas) may represent or initiate a disconnection of ACC function so that the reciprocal relationship between ACcd and ACad is disrupted during hypnotic induction’.

We have mentioned earlier that the ACC as well as the orbitofrontal cortex (OFC) are involved in both conversion and hypnotic paralysis, and that both areas are considered to be fundamental during motor inhibition. Bilateral activation of ACC and OFC has been observed during the observation of images with affectively neutral content (Corrigan, 2002) compared with right OFC and right ACC (also right amygdala) activity during the provocation of symptoms by emotionally loaded script driven imagery in post-traumatic stress disorder (PTSD) patients, for example (Rauch, Van der Kolk, Fisler, Alpert, Orr, Savage, Fischman, Jenike and Pitman, 1996). It is speculated that the ACC could be a mediator of the transfer of emotional to episodic memory due to connections to the amygdala (Rauch, Van der Kolk, Fisler et al. 1996). Similar activation of ACC-OFC can then be observed during three different conditions: PTSD, conversion, and hypnotic induction. It is thus very possible that the OFC is also divided in emotional and cognitive subdivisions and that such divisions can be coherently connected to the ACC's emotional and cognitive subdivisions, and would explain the ipsilateral destabilization experienced by both areas synchronically during PTSD, conversion and hypnosis. Orne (1959) defined suggestibility as an increased responsiveness to social (environmental) cues, real or perceived. Kolb and Whishaw (1998) found that animals with damage to the OFC

Published by John Wiley & Sons, Ltd

failed to respond correctly to environmental cues, see also Damasio (1999). This strongly indicates that OFC is indeed an area that functionally correlates with suggestibility. Further research on the mechanisms of OFC is required to elucidate the exact range of its functional characteristics.

To summarize then, focusing of attention (absorption) and the resulting lack of contextual evaluation (suggestibility) are a physiological result of right-sided unilateral activation of ACC and OFC respectively, thus causing a lack of cognitive processing of emotions (Corrigan, 2002). As a result of this, motivation for motor response is affected. During hypnotic paralysis, although there is no traumatic situation, the therapist induces absorption of attention by reducing environmental stimuli through a process of relaxation and stimuli reduction. They then induce a paralysis suggestion by providing specific paralysis cues to impair motor motivational information. This process is a result of an induced suggestion instead of a traumatic autosuggestion but it also leads to cognitively deficient processing of motor information for the duration of the hypnotic session. In conversion paralysis the process is centralized around a traumatic situation. During trauma, noradrenergic potentiation (long-term potentiation, LTP) enhances ACC-amygdala connections in the right emotional subdivision Acad. This, in turn, impacts cognitive processing of ACcd. Following the theoretical formulation of the OFC as divided and synchronized to the ACC presented above, a traumatic situation will also disconnect the OFC's cognitive division (OFCcd) from the affective subdivision (OFCad). Continuing this line of thought it is logical to conclude that the OFC right affective subdivision is also an element of the conversion paralysis circuit and that the right unilateral activation of OFCad is a functional correlate of an emotionally charged autosuggestion during conversion.

Conclusion

The mentioned studies by Marshall et al. (1997) and Halligan, Atwal, Oakley and Frackowiak (2000) have provided definite evidence of the relationship of the ACC - OFC in cognitive and emotional processing of motor information. It is thus hypothesized that in hypnotizable subjects the amygdala-ACC-OFC connections are enhanced by a trauma-induced LTP in the feedback circuit between the affective subdivisions of ACC and OFC and the amigdala. Such enhancement could certainly obstruct effective functioning of the cognitive subdivisions, leading to a dysfunctional motor processing that finally provokes the onset of symptoms of conversion paralysis.

In general, disconnection theories like Kihlstrom's (1992) and Shallice and Burgess's (1998) propose models in which higher-level intentional information and lower-level automatic processes are disconnected during psychogenic paralysis. However, from a neurological perspective, the puzzle is still incomplete. Vuilleumier, Chicherio, Assal, Schwartz, Slosman and Landis (2001) studied low-level brain areas involved in conversion paralysis, and found irregular hypoactivity in areas such as the thalamus and basal ganglia contralateral to the area of deficit (paralyzed limb), and they remark that higher-level OFC and ACC activity might influence motor function through their inputs into basal ganglia and thalamic circuits: 'abnormal striatal and thalamic activity might represent downstream effects due to primary dysfunction in orbitofrontal, cingulate or prefrontal cortex, allowing for the actual implementation of motor inhibition associated with conversion symptoms' (Vuilleumier, Chcherio, Assal et al. 2001: 1085). There is still no physiological evidence of disconnection between low and high brain areas during conversion or hypnotic paralysis, although as presented in this paper, there is evidence of

Published by John Wiley & Sons, Ltd

disconnection between cognitive and affective subdivisions in high areas, specifically ACC and OFC. It is likely, however, that such disconnection disables higher-level cognitive-intentional processes, thus failing to provide adequate information to low-level brain areas that handle automatic processes.

References:

Ballantine HR, Levy BS, Dagi TF, Giriunas JB (1975) Cingulotomy for psychiatric illness: report of 13 years of experience. In: WH Sweet, JG Obrator, JG Martin-Rodriguez (eds) Neurosurgical treatment in psychiatry, pain and epilepsy. Baltimore, MD: University Park Press, 333–53.

Bliss EL (1984) Hysteria and hypnosis. Journal of Nervous and Mental Disease 172: 203–6.

Botvinick MM, Cohen JD, Carter CS (2004) Conflict monitoring and anterior cingulate cortex: an update. Trends in Cognitive Science 12: 539–46.

Bush G, Luu P, Posner MI (2000) Cognitive and emotional influences in anterior cingulate cortex. Trends in Cognitive Neuroscience 4: 215–22.

Butler L, Duran R, Jasiukaitis P, Koopman C, Spiegel D (1996) Hypnotizability and traumatic experience. The American Journal of Psychiatry 153: 42–70.

Casey KL, Minoshima S, Berger KL, Koeppe RA, Morrow TJ, Frey KA (1994) Positron emission tomographic analysis of cerebral structures activated specifically by repetitive noxious heat stimuli. Journal of Neurophysiology 71: 802–7.

Coghill RC, Talbot JD, Evans AC, Meyer E, Gjedde A, Bushnell MC, Duncan GH (1994) Distributed processing of pain and vibration by the human brain. Journal of Neuroscience 14: 4095–108.

Corrigan FM (2002) Mindfulness, dissociation, EMDR and the anterior cingulate cortex: a hypothesis. Contemporary Hypnosis 19: 8–17.

Damasio AR (1999) The Feeling of What Happens. New York, NY: Harcourt Brace.

Derbyshire SWG, Jones AKP, Devani P, Friston KJ, Feinmann C, Harris M, Pearce S, Watson JDG, Frackowiak RSJ (1994) Cerebral response to pain measured by positron emission tomography. Journal of Neurology, Neurosurgery and Psychiatry 57: 1166–72.

Devinsky O, Morrell MJ, Vogt BA (1995) Contributions of cingulate cortex to behavior. Brain 118: 279–306.

Everitt BJ, Robbins T (1992) Amigdala-ventral striatal interactions and reward-related processes. In: JP Aggleton (ed.) The Amigdala: Neurobiological Aspects of Emotion, Memory and Mental Disfunction. New York: Wiley-Liss, 401–29.

Frankel FH (1990) Hypnotizability and dissociation. American Journal of Psychiatry 147: 823–9.

Frischholz EJ, Lipman LS, Braun BG, Sachs RG (1992) Psychopathology, hypnotizability and dissociation. American Journal of Psychiatry 149: 1521–5.

Foltz EL, White LE (1968) The role of rostral cingulotomy in pain relief. International Journal of Neurology 6: 353–73.

Fuster JM (1981) Prefrontal cortex in motor control. In: JM Brookhart, VB Mountcastle, VB Brooks (eds) Handbook of Physiology. Bethesda, MD: American Physiological Society, section 1.

Groenewegen HJ, Wright CI, Uylings HB (1997) The anatomical relationship of the prefrontal cortex with limbic structures and basal ganglia. Journal of Psychopharmacology 11: 99–106.

Gruzelier J, (1998) A working model of the neurophysiology of hypnosis: a review of evidence. Contemporary Hypnosis 15: 3–21.

Halligan PW, Atwal BS, Oakley DA, Frackowiak RS (2000) Imaging hypnotic paralysis: implications for conversion Hysteria. Lancet 355: 986–7.

Hilgard ER (1977) Divided Consciousness: Multiple Controls in Human Thought and Action. New York: Wiley.

Janet P (1907) The Major Symptoms of Hysteria. London: MacMillan.

Jones AKP, Brown WD, Friston KJ, Qi LY, Frackowiak SJ (1991) Cortical and subcortical localization of response in pain in man using positron emission tomography. Proceedings of the

Published by John Wiley & Sons, Ltd

Royal Society London, Series B, Biological Sciences, 1355–7.
Kaada BR (1960) Cingulate, posterior orbital, anterior insular and temporal pole cortex. In: J Field (ed.) Handbook of Physiology. Baltimore, MD: Williams and Wilkins, vol 2.
Kaplan HI (1985) Dissociative disorders. In: HI Kaplan, BJ Sadocks (eds) Comprehensive Textbook of Psychiatry. Baltimore, MD: Williams and Wilkins, 942–57.
Kihlstrom JF (1992) Dissociation and dissociations: a comment on consciousness and cognition. Consciousness and Cognition 1: 47–53.
Kolb B, Whishaw IQ (1998) Fundamentals of Human Neuropsychology. New York: WH Freeman and Company.
Koopman C, Classen C, Cardena E, Spiegel D (1995) When disaster strikes, acute stress disorder may follow. Journal of Trauma and Stress 8: 29–46.
Kropotov JD, Näätänen R, Sevostianov AV, Alho K, Reinikainen K, Kropotova OV (1995) Mismatch negativity to auditory stimulus change recorded from the human temporal cortex. Psychophysiology 32: 418–22.
Kropotov JD, Crawford HJ, Polyakov YI (1997) Somatosensory event-related potential changes to painful stimuli during hypnotic analgesia: anterior cingulate and anterior temporal cortex intracranial recordings. International Journal of Psychophysiology 27: 1–8.
LeDoux J (2002) Synaptic Self. New York, NY: Penguin Putman.
Lüders HO, Dinner DS, Morris HH, Wyllie E, Comair YG (1995) Cortical electrical stimulation in humans: the negative motor areas. Advances in Neurology 67: 115–19.
Maquet P, Faymonville ME, Deguelde CH, Delfiore G, Franck G, Luxen A, Lamy M (1999) Functional neuroanatomy of hypnotic state. Biological Psychiatry 45: 327–33.
Marmar CR, Weiss DS, Schlenger WE, Fairbank JA, Jorda BK, Kulka RA, Hough RL (1994) Peritraumatic dissociation and posttraumatic stress in male Vietnam theatre veterans. American Journal of Psychiatry 151: 907–9.
Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RS (1997) The functional anatomy of hysterical paralysis. Cognition 64: B1–B8.
Oakley DA (1999) Hypnosis and conversion hysteria: a unifying model. Cognitive Neuropsychiatry 4: 243–65.
Orne MT (1959) The nature of hypnosis: artifact and essence. Journal of Abnormal Social Psychology 58: 277–99.
Pardo J, Pardo P, Janer K, Raichle M (1990) The anterior cingulate cortex mediates processing selection in stroop attentional conflict paradigm. Proceedings of the National Academy of Science , USA 87: 256–9.
Posner MI, Petersen SE, Fox PT, Raichle ME (1988) Localization of cognitive operations in the human brain. Science 240: 1627–31.
Ratey J (2001) A User's Guide to the Brain. New York, NY: Pantheon Books.
Rauch SL, Van der Kolk BA, Fisler RE, Alpert NM, Orr SP, Savage CR, Fischman AJ, Jenike MA, Pitman RK (1996) A symptom provocation study of post-traumatic stress disorder using positron emission tomography and script-drive imagery. Archives of General Psychiatry 53: 380–7.
Roelofs K, Hoogduin KAL, Keijsers GPJ, Naring G, Moene F, Sandijck P (2002a) Hypnotic susceptibility in patients with conversion disorder. Journal of Abnormal Psychology 111(2): 390–5.
Roelofs K, van Galen GP, Keijsers GPJ, Hoogduin KAL (2002b) Motor initiation and execution in patients with conversion paralysis. Acta Psychologica 110: 21–34.
Roelofs K, Hoogduin KAL, Keijsers GPJ (2002c) Motor imagery during hypnotic arm paralysis in high and low hypnotizable subjects. The International Journal of Clinical and Experimental Hypnosis 50(1): 51–66.
Shallice T, Burgess P (1998) The domain of supervisory processes and the temporal organization of behavior. In: A Roberts and T Robbins (eds) The Prefrontal Cortex: Executive and Cognitive Functions. Oxford: Oxford University Press, 22–35.
Spiegel D (1984) Multiple personality as a post-traumatic stress disorder. Psychiatric Clinics of North America 7: 101–10.

Published by John Wiley & Sons, Ltd

Spiegel D, Hunt T, Dondershine HE (1988) Dissociation and hypnotizability in posttraumatic stress disorder. American Journal of Psychiatry 145: 301–05.

Spiegel D (1990) Hypnosis, dissociation and trauma: hidden and overt observers in repression and dissociations. In: JL Singer (ed.) Repression and Dissociation: Implications for Personality Theory, Psychopathology, and Health. Chicago, IL: University of Chicago Press, 121–42.

Spiegel D, Cardena E (1990) New uses of hypnosis in the treatment of post-traumatic stress disorder. Journal of Clinical Psychiatry (October supplement): 39–43.

Spiegel D (1992) The use of hypnosis in the treatment of PTSD. Psychiatry Medicine 10: 21–30.

Tarkka IM, Treede RD (1993) Equivalent electrical source analysis of pain related somatosensory evoked potentials elicited by CO2 Laser. Journal of Clinical Neurophysiology 10: 513–19.

Talbot JD, Marret S, Evans AC, Meyer E, Bushnell MC, Duncan GH (1991) Multiple representations of pain in human cerebral cortex. Science 251: 1355–7.

Tellegen A, Atkinson G (1974) Openness to absorbing and self-altering experience ('absorption'), a trait related to hypnotic susceptibility. Journal of Abnormal Psychology 83: 268–77.

Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T (2001) Functional neuroanatomical correlates of hysterical sensorimotor loss. Brain (A Journal of Neurology) 124: 1077–90.

Address for correspondence:
Felipe A. Tallabs
Jose Revueltas # 2748
Col. Alta Vista sur
Monterrey NL
Mexico
Cp:64740
Email: felipetallabs@msn.com

Published by John Wiley & Sons, Ltd