*New Zealand Journal of Psychology, 1988, 17, 24-31*

# Onset of Motor Neglect Following a Right Parietal Infarct and its Recovery Consequent on the Removal of a Right Frontal Meningioma*

Jenni A Ogden
Department of Psychology, University of Auckland

A patient with a meningioma of the right frontal lobe demonstrated only mild clumsiness of the left limbs on admission to hospital. A carotid angiogram performed prior to surgery caused an embolic occlusion of the right carotid artery, resulting in a right temporoparietal infarct. When the subsequent hemiparesis resolved the patient demonstrated a profound motor neglect of her left limbs, and left visuospatial neglect. Following the removal of the frontal meningioma 19 days after the occlusion there was an immediate and complete resolution of the motor neglect, while the visuospatial neglect continued to resolve slowly. A possible explanation is that motor neglect is the consequence of a unilateral motor programming and initiation deficit superimposed on a unilateral neglect disorder.

A patient with a lesion in one hemisphere of the brain may fail to report, respond or orient to stimuli (Heilman, 1979) or mental images (Bisiach & Luzzatti, 1978) in the side of space opposite to the lesion. This disorder is termed unilateral spatial neglect or hemineglect. It is not the result of a sensory deficit, but is a higher cognitive disorder. Spatial hemineglect can occur in any modality, although the majority of cases reported in the literature are of visuospatial hemineglect.

One of the less common disorders usually considered to fall within the broad classification of unilateral spatial neglect, is that of neglect of the contralesional half of the body (Laplane & Degos, 1983). While it appears that the more severe symptoms of unilateral body neglect follow lesions of the right hemisphere, commonly the parietal lobe (Cutting, 1978; Hecaen, 1968), there is no convincing evidence that milder symptoms of body neglect follow right-hemispheric lesions any more frequently than they follow left-hemispheric lesions (Cutting, 1978; Weinstein & Friedland, 1977). This parallels the finding that visuospatial neglect on a battery of drawing tasks is equally frequent following right- and left-hemispheric lesions in patients with acute lesions, although the neglect tends to be more severe after right-hemispheric damage (Ogden, 1985a, 1987). The more dramatic symptoms of body neglect are usually observed only in patients with acute lesions, and the symptoms spontaneously resolve as the brain stabilizes. This observation also holds for visuospatial neglect (Blumer & Benson, 1975; Gainotti, 1968).

Unilateral neglect of the body can take a number of forms, and it is not clear whether these are all consequences of the same underlying deficit, or are in fact independent disorders (Friedland & Weinstein, 1977; Heilman & Watson, 1977). Similarly, it is not clear whether neglect of the body is a form of unilateral spatial neglect, and therefore a consequence of the same deficit that underlies the more commonly observed visuospatial neglect (Denes, Semenza, Stoppa & Lis, 1982). Certainly, patients who demonstrate neglect of the contralesional half of the body almost always demonstrate severe visuospatial neglect also (Frederiks, 1969), although the reverse is not true.

A relatively uncommon form of body neglect is hemisomatagnosia. Patients with this disorder almost invariably have a hemiparesis or a hemianaesthesia, and their body neglect is expressed by their ap-

*This study was supported by the Medical Research Council of New Zealand. Grant 84/20. I would like to thank Dr J. Simcock of Auckland Hospital for permission to investigate the patient, and I am grateful to the patient, MB, for her cooperation throughout the investigation. Address for reprints: Dr J. A. Ogden, Department of Psychology, University of Auckland, Private Bag, Auckland, New Zealand.

parent lack of interest in their contralateral body half and their unwillingness to incorporate it into their activities. For example, they might not use or dress their contralateral limbs, or they might shave or apply makeup to the ipsilateral half of their faces only.

Anosognosia (Babinski, 1914, 1918; Waldenstrom, 1939), or the unawareness, denial or underestimation of illness, can occur in cases of hemiplegia. For example, when asked to describe what is wrong with the paralysed arm, the patient with anosognosia may say that the arm is a little stiff, but it will recover with a short rest. Frederiks (1969) and Cutting (1978) divided anosognosia for hemiplegia into explicit denial of hemiplegia (anosognosia) and other anosognosic phenomena such as misoplegia or hatred of the limb (Critchley, 1974), and personification or giving the limb a nickname (Juba, 1949).

A third form of body neglect has been called motor aspontaneity (De Renzi, 1982), unilateral hypokinesia (Heilman, Bowers, Coslett, Whelan, & Watson, 1985; Valenstein & Heilman, 1981), or motor neglect (Castaigne, Laplane, & Degos, 1970, 1972; Laplane & Degos, 1983). Patients with motor neglect may be fully aware of both sides of their bodies but do not spontaneously use the contralesional limbs even although these limbs are not paralyzed (although they may be weak). This disorder has the appearance of hemiplegia, and is probably often mistaken as such, but normal or near-normal strength and dexterity can be proven by prompting the patient to extraordinary effort. Laplane and Degos (1983) isolated a number of cases of 'pure' motor neglect in patients without hemiplegia or sensory deficits. The majority of these patients had left or right frontal lesions, presumably because patients with parietal lesions resulting in sensory deficits were excluded. The motor neglect either resolved spontaneously over a few days or weeks, or was masked by the onset of a hemiplegia in patients with progressive lesions.

The following case is instructive because of the unusual circumstances preceding the instantaneous recovery of a body neglect disorder that took the form of a severe motor neglect accompanied by anosognosic phenomena.

## Case History

MB, a 60 year old right-handed caucasian woman who worked as a receptionist in a medical practice, was admitted to the hospital neurology ward in order to ascertain the cause of a one month history of gradually worsening clumsiness and weakness of her left hand and leg. On neurological examination she was alert, oriented and cooperative. She was non-hypertensive, her fundi were normal and she had no sensory deficits. Her only symptom was a mild weakness of the left hand and forearm and the left leg. When asked to draw a 3-dimensional cube, a five-pointed star and a clock face, she did so without difficulty and with no signs of visuospatial problems or left-sided neglect. Computerized tomography (CT) of her brain revealed a meningioma (later confirmed after surgical removal) situated high in the right frontal lobe. Some local mass effect was evident. Over the next seven days, M.B.s condition remained unchanged and the weakness of her left limbs did not worsen.

Prior to scheduling surgery a right carotid angiogram was performed. The initial injection demonstrated normal filling of the branches of the internal carotid artery on the right side, but a subsequent injection into the right common carotid artery was followed by an immediate change in the patient's level of consciousness and she was unable to be roused for 2-3 minutes and then slowly regained consciousness. The angiography films showed an embolic occlusion of the right carotid artery, probably caused by a clot that had formed on the end of the catheter after the initial injection and before the final diagnostic injection. A CT brain scan taken one week later showed an infarct involving the posterior aspect of the right middle cerebral artery territory in the temporoparietal region.

Immediately following the embolic occlusion, MB had a flaccid left hemiplegia and demonstrated marked left-sided hemisomatognosia. She sat with her head turned towards her right shoulder and demonstrated a lack of awareness of the left side

of her body, leaving her left limbs uncovered and lying in awkward positions. Over the next seven days her hemiplegia resolved to a mild weakness of her left arm and leg, and when encouraged she could at times demonstrate reasonable movement in both left limbs. Her hemisomatagnosia had resolved to the stage where her visual fields could be tested by confrontation, and she had a left homonymous hemianopia.

MB was also at this time given four paper and pencil tests to assess visuospatial neglect. When she was asked to cross 40 lines spread apparently randomly over a page, (Albert, 1973) she crossed only the ten lines on the extreme right of the page, and when asked to check that she had crossed them all, said there were no more to cross. She also copied only the right side of a Necker cube and five-pointed star. Her copy of a line drawing of a scene also demonstrated severe neglect (see Figures 1A and 1B). When asked to draw from memory a plan of the living room in her home, the right side of her drawing was full of intricate detail (and correct as compared with her brother's plan of the same room), but the left side was completely missing (c.f., Bisiach, Capitani, Luzzati & Perani, 1981; Bisiach & Luzzatti, 1978). At this stage MB was alert, oriented and co-operative, and expressed a keen desire to recover. Therefore, her neglect disorder was explained to her and she was given daily exercises to help her to become more aware of the left side of space and her left limbs. Figure 1C demonstrates some improvement of left visuospatial neglect on copying tasks eight days later.

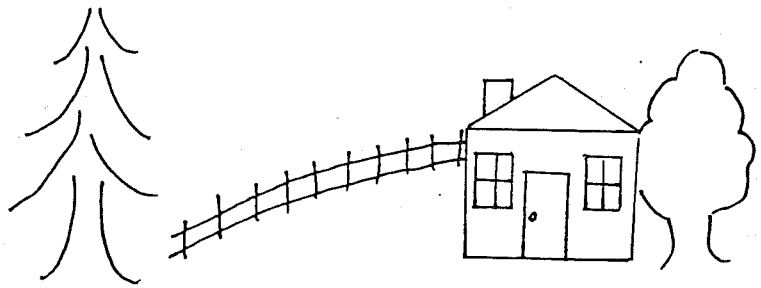
As her hemiplegia resolved her hemisomatagnosia became less marked. She would turn her head to the left when asked, and became aware of the left side of her body. At this point various anosognosic phenomena became apparent. She made comments about her left arm such as "I am tired of giving this bed and board" and "When I get this home I will have something to say to it" (misoplegia), and she referred to her arm as "that piece of wood" or "my wooden arm" (personification). She also said that the arm felt separate from her.

However, the most dramatic symptom to appear as the hemiplegia resolved was a severe motor neglect. Even although she appeared highly motivated to move her left limbs, she only managed to do so with extreme effort and verbal self-instruction. Under these conditions she sometimes demonstrated near normal strength and movement. In spite of her apparent understanding of the way in which she 'neglected' or 'did not want' to move her limbs even although they were no longer paralyzed, (i.e., she succinctly explained her neglect symptoms, both visuospatial and motor, to her visitors), she was, as she said, still unable to do anything about it.

Eight days after her embolic occlusion, in order to assess whether she had suffered a generalized mental deterioration that might in part be the cause of her neglect (Battersby, Bender, Pollack, & Kahn, 1956), she was given the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955). Her Verbal IQ was 131 and her Performance IQ, 109. Clearly her verbal abilities were excellent, and her scores on the performance subtests reflected her visuospatial neglect, and visuoperceptual and visuocontructional problems.

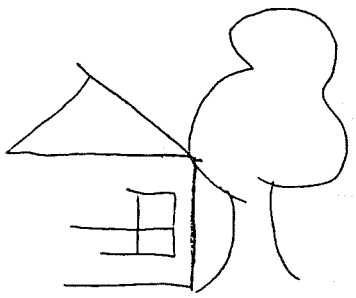
When surgery to remove the meningioma in her right frontal lobe was finally scheduled 19 days after her embolic occlusion, although she had improved, she still demonstrated visuospatial neglect (e.g., she missed six lines on the left side of the page on the crossing lines test) and her motor neglect was still severe, with minimal if any improvement. When asked, she usually managed to wriggle the fingers of her left hand with concentrated effort, and raise her left leg one inch off the floor when sitting.

Surgery to remove the meningioma was successful and uneventful. Later that same day I went to see her on her return to the ward. She greeted me by raising both arms from the bed and grasping my hand firmly in both of hers. She was clearly aware of the marked improvement in voluntary movements of her left arm, and commented that the removal of the tumour had cured her hemiplegia. From that day on she demonstrated no signs of motor neglect or anosognosic phenomena, and was able to walk with a stick, and perform all tasks nor-



COPY THIS DRAWING IN THE SPACE BELOW

A



B 18/7/84

C 26/7/84

*Figure 1.* 'A' is the 'scene' test of neglect. 'B' and 'C' are M.B.'s attempts to copy the drawing following her right temporoparietal infarction. Drawing 'B' is her copy after seven days, and drawing 'C' her copy after 15 days.

mally with her left arm and hand. Her left homonymous hemianopia had not resolved.

Although she had shown only minimal, if any, resolution of her motor neglect in the days preceding surgery in spite of daily physical therapy sessions aimed at helping her move her left limbs, she had shown a steady improvement on visuospatial drawing tasks with daily practice. This rate of improvement in visuospatial neglect did not appear to accelerate immediately following her surgery, but rather continued to improve at the same rate so that five days after surgery her neglect of drawings was restricted to the occasional small detail on the left side.

## Discussion

This case study is interesting because of the onset of visuospatial neglect, anosognosic phenomena and motor neglect fol-

lowing a right parietotemporal infarct, and the immediate and complete resolution of the motor neglect and anosognosic phenomena following the removal of a right frontal meningioma that had not initially been associated with the neglect symptoms.

One possible explanation for the onset and recovery of MB's motor neglect is that the combined effects of pressure from the frontal meningioma plus some transient ischaemia of Brodmann's area 6 (the premotor cortex and the supplementary motor area involved in the programming of movements) following the embolic occlusion of the right carotid artery, could have resulted in a motor neglect of the left limbs (Laplane & Degos, 1983; Meador, Watson, Bowers, & Heilman, 1986). The severe left visuospatial neglect could have been a result of the right posterior parietotemporal infarct, and quite independent of the motor neglect. The immediate recovery of the motor neglect would therefore be dependent on the release of pressure on area 6 following the removal of the meningioma.

This explanation poses some problems. It requires that any transient ischaemia occurring at the time of the carotid occlusion extends anteriorly as far as area 6, and is still causing a dysfunction of motor programming but no longer causing hemiplegia 19 days after the occlusion when the meningioma was removed. The CT scan taken seven days following the occlusion does not support such an anterior extension of the infarct, although ischaemia not visible on CT scan may occur outside the area of infarction.

There is another possible explanation for the onset and unusual recovery of MB's motor neglect that does not rely on a hypothetical ischaemia of area 6 that was still causing motor neglect but not hemiplegia 19 days after the embolic occlusion. This explanation calls on the combination of two separate deficits, a mild motor programming dysfunction and a left spatial neglect, that in concert result in a motor neglect of the left limbs. There is ample evidence that unilateral spatial neglect can follow lesions of the frontal lobe and thalamus (Damasio, Damasio, & Chui, 1980; Heilman & Valenstein, 1972; Meador et al., 1986; Motomura, Yamadori, Mori, Ogura, Sakai, & Sawada, 1986; Ogden, 1985a; Watson & Heilman, 1979), as well as lesions of the posterior parietal cortex (Heilman & Watson, 1977; Ogden, 1985a). One function of Brodmann's area 6 is the planning and programming of movements, and area 8 (the frontal eyefields) and the surrounding cortical areas are involved in the initiation and inhibition of exploratory and attentive movements, particularly in the contralateral hemispace (Mesulam, 1981).

It has been postulated that the dorsolateral portion of the inferior parietal lobe may be the site where an elaborate sensory representation of extrapersonal space is built up, partly on the grounds that sensory information arrives in this area only after it has been processed first in unimodal sensory cortex and then in polymodal cortex (Mesulam, 1981). Further support for this comes from experiments in which patients with right parietal lesions and unilateral visuospatial neglect were shown to neglect the contralesional halves of their mental representations (Bisiach et al., 1978, 1979, 1981; Ogden, 1985b).

To explain the different neglect disorders, Mesulam (1981) has proposed a cortical network that incorporates all the areas associated with neglect in humans and animals. The posterior parietal cortex provides a sensory representation of space, the frontal cortex provides the motor programmes for exploring space, the limbic system (cingulate gyrus) provides information about the motivational significance of stimuli in the spatial environment, and a reticular formation component regulates the level of arousal and vigilance. He postulated that the form of neglect may depend on where this cortical network is lesioned, and the severity of neglect may depend on the number and combination of different areas in the cortical network that are damaged.

In the case of MB, while the meningioma in her frontal lobe did not result in unilateral spatial neglect, it did have some mass effect, and her symptoms of increasing clumsiness and mild weakness of the left limbs suggest that there was some minimal dysfunction of the motor areas, probably

including areas 4, 6, and 8. The acute embolic occlusion that she subsequently suffered resulted in an infarct of the posterior parietotemporal area and not surprisingly caused a severe left-sided hemisomatagnosia and visuospatial neglect. As the hemiplegia resolved over the next seven days, presumably as a result of the resolution of oedema or ischaemia surrounding the infarct, MB's hemisomatagnosia also resolved, unmasking a motor neglect disorder.

On the assumption that the posterior parietal infarct damaged the site of sensory representation of contralateral space in the right hemisphere, it seems likely that the visuospatial neglect was entirely a consequence of the infarct. However, the motor neglect may be dependent on the combination of a dysfunction of areas 6 and 8 (as a result of the frontal meningioma) and the posterior parietal lesion. That is, in MB's case, the mild motor programming deficit (clumsiness) caused by the pressure of the meningioma on areas 6 and 8, only became profound (i.e., developed into 'motor neglect') when it was superimposed on the severe unilateral visuospatial deficit caused by the infarction of the posterior parietal cortex. From the patient's point of view, if the limbs are in the neglected extrapersonal hemispace, it may be much more difficult to overcome deficits in the motor programming and initiation of movement of those limbs and 'command' them to move.

When the meningioma was successfully removed, this removed the pressure on areas 6 and 8, rendering the motor programmes for the left limbs and the ability to initiate movement fully functional again. MB's motor neglect resolved instantaneously, but her visuospatial neglect continued to resolve more slowly because it was entirely the result of the acute posterior parietal infarct. MB no longer demonstrated anosognosic phenomena, presumably because these were dependent on her perception of her limbs as being paralyzed, feeling strange, and as if they did not belong to her. If this latter explanation is correct, it suggests that motor neglect is not strictly a form of unilateral spatial neglect, but is a unilateral motor programming and initiation deficit that is exacerbated by a concurrent unilateral spatial neglect affecting the same side of the body (or the hemispace) it is in. In some cases, the motor neglect may be caused by a relatively mild motor programming and initiation deficit in combination with a severe unilateral spatial neglect (as in MB's case), or it may be caused by a severe motor programming and initiation deficit and a relatively mild unilateral spatial neglect, as in some of the cases described by Laplane and Degos (1983).

How then does this hypothesis account for patients who demonstrate motor neglect following a single focal lesion of the frontal or parietal lobe? Patients with frontal lesions and motor neglect are not difficult to explain as frontal lesions in different patients can result independently in motor programming and initiation deficits (Hecaen & Albert, 1975; Luria, 1966; Meador et al., 1986) and in contralateral spatial neglect. Patients with parietal lesions and motor neglect are more difficult to explain because while lesions in this area commonly result in hemispatial neglect this area is not directly involved in the initiation of movement and motor programming. However, there are rich reciprocal neural connections between the frontal eye fields and the dorsolateral portion of the inferior parietal lobe, at least in the monkey (Pandya & Kuypers, 1969; Petras, 1971). It could be that in patients with posterior parietal lesions and motor neglect these neural pathways are damaged. In Laplane and Degos' (1983) group of patients with motor neglect, the majority of patients had either metastases or glioblastomas, thereby making it difficult to specify the boundaries of the lesions. The four patients with lesions in the parietal or temporal areas all had glioblastomas. While only seven of their twenty patients demonstrated visuospatial neglect and in a further three patients visuospatial neglect was queried, they did not specify how visuospatial neglect was assessed. It is possible that a more sensitive test or battery of tests may have identified a mild visuospatial neglect disorder in more of their patients (Ogden, 1985a, 1985b).

To conclude, this case study suggests that two separate cerebral lesions may in combination result in motor neglect, and it is pos-

tulated that a frontal lesion could result in a motor programming and initiation deficit of the contralateral limbs, and that this may develop into a more profound motor neglect when superimposed upon a unilateral spatial neglect resulting from an acute parietal lesion.

## References


Albert, M. L. (1973). A simple test of visual neglect. *Neurology, 23*, 658-664.

Babinski, J. (1914). Contribution a l'etude des troubles mentaux dans l'hemiplegie organique cerebrale (anosognosie). *Revue Neurologique, 27*, 845-848.

Babinski, J. (1918). Anosognosie. *Revue Neurologique, 31*, 365-367.

Battersby, W. S., Bender, M. B., Pollack, M., & Kahn, R. L. (1956). Unilateral 'spatial agnosia' (inattention). *Brain 79*, 68-93.

Bisiach, E., Capitani, E., Luzzatti, C., & Perani, D. (1981). Brain and conscious representation of outside reality. *Neuropsychologia, 19*, 543-551.

Bisiach, E., & Luzzatti, C. (1978). Unilateral neglect of representational space. *Cortex, 14*, 129-133.

Bisiach, E. Luzzatti, C., & Perani, D. (1979). Unilateral neglect, representational schema and consciousness. *Brain, 102*, 609-618.

Blumer, D., & Benson, D. F. (1975). Personality changes with frontal and temporal lobe lesions. In D. F. Benson and D. Blumer (Eds.), *Psychiatric Aspects of Neurologic Disease*. (pp. 151-170). New York: Grune and Stratton.

Castaigne, P., Laplane, D., & Degos, J. D. (1970). Trois cas de negligence motrice par lesion retrorolandique. *Revue Neurologique, 122*, 233-242.

Castaigne, P., Laplane, D., & Degos, J. D. (1972). Trois cas de negligence motrice par lesion frontale prerolandique. *Revue Neurologique, 126*, 5-15.

Critchley, M. (1974). Misoplegia or hatred of hemiplegia. *Mt. Sinai Journal of Medicine N.Y., 41*, 82-87.

Cutting, J. (1978). Study of anosognosia. *Journal of Neurology, Neurosurgery, and Psychiatry, 41*, 548-555.

Damasio, A. R., Damasio, H., & Chui, H. C. (1980). Neglect following damage to frontal lobe or basal ganglia. *Neuropsychologia, 18*, 123-132.

Denes, G., Semenza, C., Stoppa, E., & Lis, A. (1982). Unilateral spatial neglect and recovery from hemiplegia. A followup study. *Brain, 105*, 543-552.

De Renzi, E. (1982), *Disorders of Space Exploration and Cognition*, New York: Wiley and Sons.

Frederiks, J. A. M. (1969). Disorders of the body schema. In P. J. Vinken & G. W. Bruyn (Eds.), *Handbook of Clinical Neurology*. (Vol. 4.). North Holland: Elsevier Science Publishers.

Friedland, R. P., & Weinstein, E. A. (1977). Hemiinattention and hemisphere specialization: Introduction and historical review. In E. A. Weinstein and R. P. Friedland (Eds.), *Hemi-Inattention and Hemisphere Specialization: Advances in Neurology* (Vol. 18). (pp. 1-31). New York: Raven Press.

Gainotti, G. (1968). Les manifestations de negligence et d'inattention pour l'hemispace. *Cortex, 4*, 64-91.

Hecaen, H. (1968). Essai d'interpretation des asomatagnosies en pathologie corticale. In M. M. Velasco-Suarez and F. Escobedo (Eds.), *Lobulo Parietal* (pp. 141-156). Mexico: Instituto National de Neurologie.

Hecaen, H. & Albert, M. (1975). Disorders of mental functioning related to frontal lobe pathology. In D. F. Benson & D. Blumer (Eds.), *Psychiatric Aspects of Neurologic Disease* (pp. 137-149). New York: Grune and Stratton.

Heilman, K. M. (1979). Neglect and related disorders. In K. M. Heilman and E. Valenstein (Eds.), *Clinical Neuropsychology* (pp. 268-307). Oxford: Oxford University Press.

Heilman, K. M., Bowers, D., Coslett, H. B., Whelan, H., & Watson, R. T. (1985). Directional hypokinesia: Prolonged reaction times for leftward movements in patients with right hemisphere lesions and neglect. *Neurology, 35*, 855-859.

Heilman, K. M., & Valenstein, E. (1972). Frontal lobe neglect in man. *Neurology, 22*, 660-664.

Heilman, K. M. & Watson, R. T. (1977) Mechanisms underlying the unilateral neglect syndrome. In E. A. Weinstein and R. P. Friedland (Eds.), *Hemi-Inattention and Hemisphere Specialization. Advances in Neurology* (Vol. 18). (pp.92-106). New York: Raven Press.

Juba, M. (1949). Beitrag zur Struktur der ein-und doppelseitigen Korpesschemastorungen. *Monatsschrift fur Psychiatrie und Neurologie, 118*, 11-29.

Laplane, D., & Degos, J. D. (1983). Motor neglect. *Journal of Neurology, Neurosurgery and Psychiatry, 46*, 152-158.

Luria, A. R. (1966). *The Higher Cortical Function in Man*. New York: Basic Books.

Meador, K. J., Watson, R. T., Bowers, D., & Heilman, K. M. (1986). Hypometria with hemispatial and limb motor neglect. *Brain, 109*, 293-305.

Mesulam, M. M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10*, 309-325.

Motomura, N., Yamadori, A., Mori, E., Ogura, J., Sakai, T., & Sawada, T. (1986) Unilateral spatial neglect due to hemorrhage in the thalamic region. *Acta Neurologica Scandinavica, 74*, 190-194.

Ogden, J. A. (1985a). Anterior-posterior interhemispheric differences in the loci of lesions producing visual hemineglect. *Brain and Cognition, 4*, 59-75.

Ogden, J. A. (1985b). Contralesional neglect of constructed visual images in right and left brain-damaged patients. *Neuropsychologia, 23*, 273-277.

Ogden, J. A. (1987). The 'neglected' left hemisphere and its contribution to visuospatial neglect. In M. Jeannerod (Ed.), *Neurophysiological and Neuropsychological Aspects of Spatial Neglect* (pp.

215-233). North Holland: Elsevier Science Publishers.

Pandya, D. M., & Kuypers, H. G. J. M. (1969). Cortico-cortical connections in the rhesus monkey. *Brain Research, 13*, 13-56.

Petras, J. M. Connections of the parietal lobe. (1971). *Journal of Psychiatric Research,* , 189-201.

Valenstein, E., & Heilman, K. M. (1981). Unilateral hypokinesia and motor extinction. *Neurology, 31*, 445-448.

Waldenstrom, J. (1939). On anosognosia. *Acta Psychiatrica, 14*, 215-220.

Watson, R. T., & Heilman, K. M. (1979). Thalamic neglect. *Neurology, 29*, 690-694.

Wechsler, D. (1955). *Wechsler Adult Intelligence Scale. Manual.* New York: Psychological Corporation.

Weinstein, E. A., & Friedland, R. P. (1977). Behavioural disorder associated with hemi-inattention. In E. A. Weinstein and R. P. Friedland (Eds.), *Hemi-inattention and Hemisphere Specialization* (pp. 51-62). New York: Raven Press.