Posterior Cerebral Artery (PCA) Infarcts and Dreaming: A Neuropsychological Study

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DECLARATION

Compulsory Declaration: This work has not been previously submitted in whole, or in part for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation for the work, or works, of other people has been attributed, and has been cited and referenced.

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ABSTRACT

Recent case reports have shown that global loss of dreaming can result from medial occipito-temporal lesions. These findings have cast doubt on Solms's reformulation of Charcot-Wilbrand Syndrome (CWS) into two distinct disorders of dreaming, and caused substantial confusion in dream research as far as the neurological correlates of dreaming are concerned. This study attempted to confirm these case reports and determine whether there were any characteristics unique to the lesions among patients who had lost the ability to dream following damage to medial occipito-temporal cortex. Nine participants (three non-dreamers and six dreamers) who had suffered non-hemorrhagic infarction in the territory of the posterior cerebral artery were recruited in this study. Case histories and neuroradiological data were used to compare the lesion sites of non-dreamers with dreamers. It was confirmed that complete loss of dreaming could result from lesions in medial occipito-temporal cortex. It was found that non-dreamers always suffered bilateral cortical damage as opposed to dreamers who all suffered unilateral damage. The lesions in the non-dreamers tended to be more posterior than the dreamers. It was further speculated that concomitant damage to the thalamus or parietal areas played a role in the causation of heteromodal loss of dreaming. The implications of these findings were discussed in relation to CWS, Solms's dream system, and dream-function research. Finally, future directions were considered.

Keywords: non-dreamers; cessation of dreaming; dream loss; neural correlates of dreaming; medial occipito-temporal lesions; posterior cerebral artery infarction

CHAPTER 1: LITERATURE REVIEW

Although sleep and dreams have been the subject of contemplation and debate since antiquity, the more formal scientific study of these phenomena began in the latter half of the 19th century (Bassetti, Bischof, & Valko, 2005). The initial attempts to study and quantify dream content, by researchers like Aflred Maury, Hervey Saint-Denis, and Mary Whiton Calkins, went practically unnoticed during the first half of the 20th century, probably owing to the dominance of behaviourism, and psychoanalytic theory at the time. Specifically, after Freud (1900) published his psychoanalytic masterwork, "The Interpretation of Dreams", studies focusing on the manifest content of dreams were disregarded because of his preoccupation with the latent content of dreams, i.e. the "true" thought process obscured by the dream. Moreover, behaviourism would influence the study of dreaming by its assertion that only observable behaviours were of scientific value, and its consequent repudiation of mental experiences such as dreams (Desseilles, Dang-Vu, Sterpenich, & Schwartz, 2011).

However, a burgeoning interest in sleep and dreaming was ignited by the discovery of REM sleep (Aserinsky & Kleitman, 1953). REM sleep was found to be characterised by bursts of rapid eye movement, and loss of muscle tone with a paradoxically high level of cortical activation similar to that of waking. When further investigation revealed that 80% of awakenings from REM sleep resulted in positive dream reports, and that only 10% of awakenings from non-REM sleep resulted in such dream reports, REM sleep and dreaming were conflated as one and the same process (Dement & Kleitman, 1957).

Since this discovery was believed to represent a neurophysiological marker for dreaming, a flood of theories concerned with the function of dreams followed. Arguably the most influential of these theories, Hobson's activation-synthesis hypothesis, championed the notion that dreams serve no function at all, being mere epiphenomena of REM sleep itself (Hobson & McCarley, 1977). This theory, dependant on the conflation of REM sleep and dreaming, proposed that the cholinergic pontine brainstem mechanisms that drive REM sleep generate a surge of activation, which is then passively synthesised in the forebrain and organised to form the meaningless representations that we experience as dreams (Hobson, Pace-Schott, & Stickgold, 2000). Like many theories on the function of dreaming, activation-synthesis is centrally dependant on a precise understanding of the underlying neurological correlates of dreaming. However, the specific brain sites necessary for dreaming to occur are still a topic of controversy, and the exact posterior cortical regions in particular have not yet been adequately identified. Conveniently, activation synthesis is able to side-step these issues by equating the neurological correlates of REM sleep with those of dreaming. However, by collating multiple lines of evidence showing that dreaming and REM sleep are doubly

dissociable (Foulkes, 1961; Foulkes & Vogel, 1965; Jus et al., 1973; Solms, 1997), Solms (2000) demonstrated that dreaming and REM sleep are controlled by different brain mechanisms. Therefore, a better account of the known mechanisms of dreaming, and by extension the neurological correlates of those mechanisms, is crucial to any theory postulating a function for dreams. Such an account requires an understanding of the evolution of the controversial and non-controversial topics, from history to modernity, in our current knowledge of the neurological correlates of dreaming.

Classic cases: Charcot (1883) and Wilbrand (1887)

The first case illustrating the profound effects that neurological disease can have on dreaming was presented by Jean-Martin Charcot in 1883. The salient feature of the presentation of his patient (Monsieur X.) was a near-total loss of the ability to consciously conjure up visual mental imagery (*irreminiscence*), a deficit which the patient himself described as an absolute loss of the "power of mental vision" (Charcot, 1883, cited in Solms, 1997, p. 4). Importantly, Monsieur X's inability to mentally visualise objects while awake was associated with a similar deficit of visual imagery in his dreams. That is, while he retained the ability to dream, his dreams were comprised "simply of speech" (and presumably other non-visual material), and possessed no visual component at all (Solms, 1997, p.5). Unfortunately, Charcot's case was never brought to autopsy, which prevents any localising analyses from being made[1].

Four years later, Hermann Wilbrand (1887) described a similar case of a patient, Fräulein G., who after suddenly losing consciousness and collapsing, awoke in a state of "fevered agitation" characterised by extraordinary visual deficits (Solms, Kaplan-Solms, & Brown, 1996, p. 84). Although she was regarded as blind by all those around her, the patient was aware that she was not completely blind, being able to see the features of a table with a table-cloth on it in her room. She was unable to recognise her physician (*prosopagnosia*), frequently confused people, animals, and objects, and often became disoriented in familiar surroundings as a result (*topographical agnosia*). Interestingly, she was still able to vividly visualise the streets and buildings of her city of residence (Solms, 1997). Of significance is the manner in which her ability to dream was affected, in that whereas "before her illness she dreamt a great deal in pictorial images, now she dreams almost not at all anymore" (Solms et al., 1996, p. 85). Wilbrand later brought the case to autopsy (Figure 1) where bilateral infarction, involving the occipito-temporal region was found (Solms, 1997).

[1] There are those who argue that Monsieur X's symptoms had functional rather than neurological origins (Zago et al., 2011). However, the current consensus is that the clinical evidence suggests a cerebrovascular etiology (Solms, 1997).

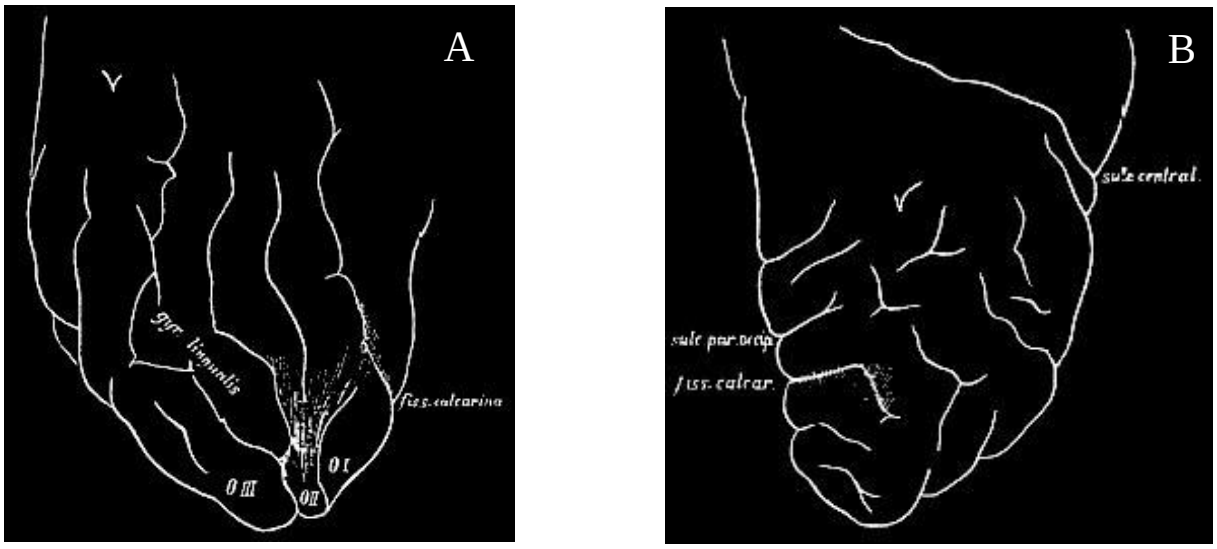


Figure 1. Autospy findings in Wilbrand's case, Fräulein G. A) Ventral surface of the right occipital lobe; B) dorsal surface of the right occipital lobe. Adapted from Solms et al., 1996, p. 99.

Charcot-Wilbrand Syndrome

Charcot's case was significant because he had presented the first evidence that dreams could be selectively impacted by neurological disease. Wilbrand's case was equally important because it was the first to illustrate that complete cessation of dreaming could result from neurological disease. Moreover, Wilbrand's and Charcot's cases were grouped together in the classical literature to form the nosological category known as Charcot-Wilbrand syndrome (CWS). The most universally used definition of this syndrome was put forth by Critchley (1953), who defined it as a condition "whereby a patient loses the power to conjure up visual images or memories, and furthermore, ceases to dream during his sleeping hours" (p.311). More recently, CWS was defined as "the association of loss of the ability to conjure up visual images or memories and the loss of dreaming...[indicating] a lesion in the acute phases affecting the posterior regions" (Murri, Arrena, Siciliano, Mazzotta, & Murarorio, 1984, p.185). The most recent definition of the syndrome describes the association of dream loss with visual irreminiscence, prosopagnosia and topographical agnosia (Bischof & Bassetti, 2004).

From the definitions above, it is obvious that CWS involves some form of dreaming deficit associated with a more fundamental deficit of visual imagery in general. Indeed, the fundamental symptom of this syndrome according to Critchley (1953) was deficient revisualisation (*visual irreminiscence*), while cessation of dreaming (or alteration of the vivid visual component of dreams), prosopagnosia, and topographical agnosia or amnesia were more or less necessary consequences of this primary deficit (Solms, 1997). However, in a more careful reading of the original literature, Solms and colleagues (1996) demonstrated the

inconsistencies between Charcot's and Wilbrand's cases, exposing flaws in the nosographic concept, CWS. For instance, while Wilbrand described several examples of topographical and other forms of misrecognition and misperception, he repeatedly made reference to his patient's intact ability to conjure up vivid visual imagery. Thus although visual irreminiscence is seen as the cardinal symptom of CWS, Fräulein G. had no such deficit, whereas Charcot's patient clearly did (see above). Moreover, the conventional definition of CWS held that the secondary deficit, cessation of dreaming, was a consequence of the primary deficit of visual imagery. However, Wilbrand's patient very definitely had the secondary deficit as described above, but very definitely *did not* have the primary deficit upon which the secondary deficit is supposedly consequent (Solms et al., 1996). The further observation that two fundamentally different descriptions of this secondary deficit of the syndrome were essentially confounded[2], led Solms (1997) to challenge its conventional characterization.

Solms's Reconsideration of Charcot-Wilbrand Syndrome

Solms (1997) challenged CWS by way of a clinicoanatomical study of 361 patients suffering from various neurological illnesses of varying localising potential. Beginning with generally overlooked complaints by patients that their dreams were altered in specific ways by their illness, he used clinical bedside interviews, CT-brain, and MRI-brain scanning to establish whether such complaints displayed any degree of uniformity, and whether any of them could be correlated with specific lesion sites. Central to the question of the validity of CWS, he hypothesised that the two characterisations of the secondary deficit of CWS, i.e. global cessation of dreaming and loss of visual dream-imagery, could be accounted for by two discrete lesions in two distinct regions of the brain.

To this end, Solms (1997) identified just two cases (1.1%) with abnormalities of visual dream-imagery, or Charcot's type of dreaming deficit. In fact, only one of those cases experienced a *complete* loss of visual dream-imagery, whereas the second experienced a loss of only the kinematic aspect of visual dream-imagery. Thus, based on radiological, surgical, and clinical data available for those cases, and in keeping with the previously published cases of this dreaming deficit, Solms posited a syndrome of non-visual dreaming, which was further differentiated into subcategories based on which aspects of visual dream-imagery were affected. A full discussion of these variants of nonvisual dreaming is not necessary here. Of relevance is Solms's finding that *complete cessation of visual dream-imagery was invariably associated with bilateral medial occipito-temporal lesions* (Figure 2A). Moreover,

[2] Charcot's was a case specifically of *cessation of the visual component of dreams*, leaving dreaming per se intact, while Wilbrand's was a case of *global cessation of dreaming*.

this syndrome was found to be typically associated with irreminiscence and visual short-term memory deficit, and to commonly co-occur with prosopagnosia, and some form of visual agnosia, topographical agnosia, and/or topographical amnesia (Solms, 1997).

In contrast to this rare deficit of visual dream-imagery, Solms (1997) identified a total of 112 (34.9%) cases with absolute cessation of dreaming, or Wilbrand's type of dreaming deficit. Thus another syndrome, one of total cessation of dreaming, could be posited. Curiously, two distinct lesion sites were found to be associated with this syndrome:

Deep bifrontal lesions (Figure 2B): *Deep bilateral damage to the ventro-mesial frontal white matter, immediately surrounding the anterior horns of the lateral ventricles, caused loss of dreaming*, which typically co-occurred with adynamia, disinhibition, and perseveration. Panksepp (1985; 1998) had previously designated these dopaminergic, mesocortical-mesolimbic pathways as the curiosity-interest-expectancy command systems of the brain, and found that these regions instigate goal-seeking behaviours and an organism's appetitive interactions with the world. This, together with the observation that chemical excitation of these circuits stimulates excessive, uncommonly frequent and vivid dreaming, and the fact the damage to these circuits causes total cessation of dreaming, led Solms (1997) to implicate these regions in the instigation of dreams. Solms's conclusions have since been supported by various lines of functional imaging (Braun et al., 1998; Maquet et al., 1996; Nofzinger, Mintun, Wiseman, Kupfer, & Moore, 1997), psychosurgical (Frank, 1946), neurophysiological (Dahan et al., 2007; Léna et al., 2005), and psychopharmacological (Hartmann et al., 1980; Sharf, Moskovitz, Lupton, & Klawans, 1978; Sandyk, 1997) evidence.

Posterior cortical lesions (Figure 2C): Complete cessation of dreaming was more commonly associated with posterior than anterior cortical lesions in Solms's (1997) series. *Damage to the parieto-occipito-temporal junction* (PTO), *i.e. the lateral surface of the inferior parietal lobule, and specifically the supramarginal gyrus, was implicated in cessation of dreaming*. No relationship was found between loss of dreaming and the laterality of the lesion. However, in cases with left unilateral damage, loss of dreaming co-occurred with visuospatial short-term memory deficit and elements of Gerstmann's syndrome. In cases with unilateral right damage, various apractagnosic signs and symptoms were seen, but only visuospatial short-term memory deficits significantly correlated with cessation of dreaming. Importantly, neither irreminiscence (the primary symptom of CWS) nor any other element of the classical CWS significantly correlated with complete loss of dreaming, as opposed to the two cases with cessation of visual dream-imagery where irreminiscence was present in both.

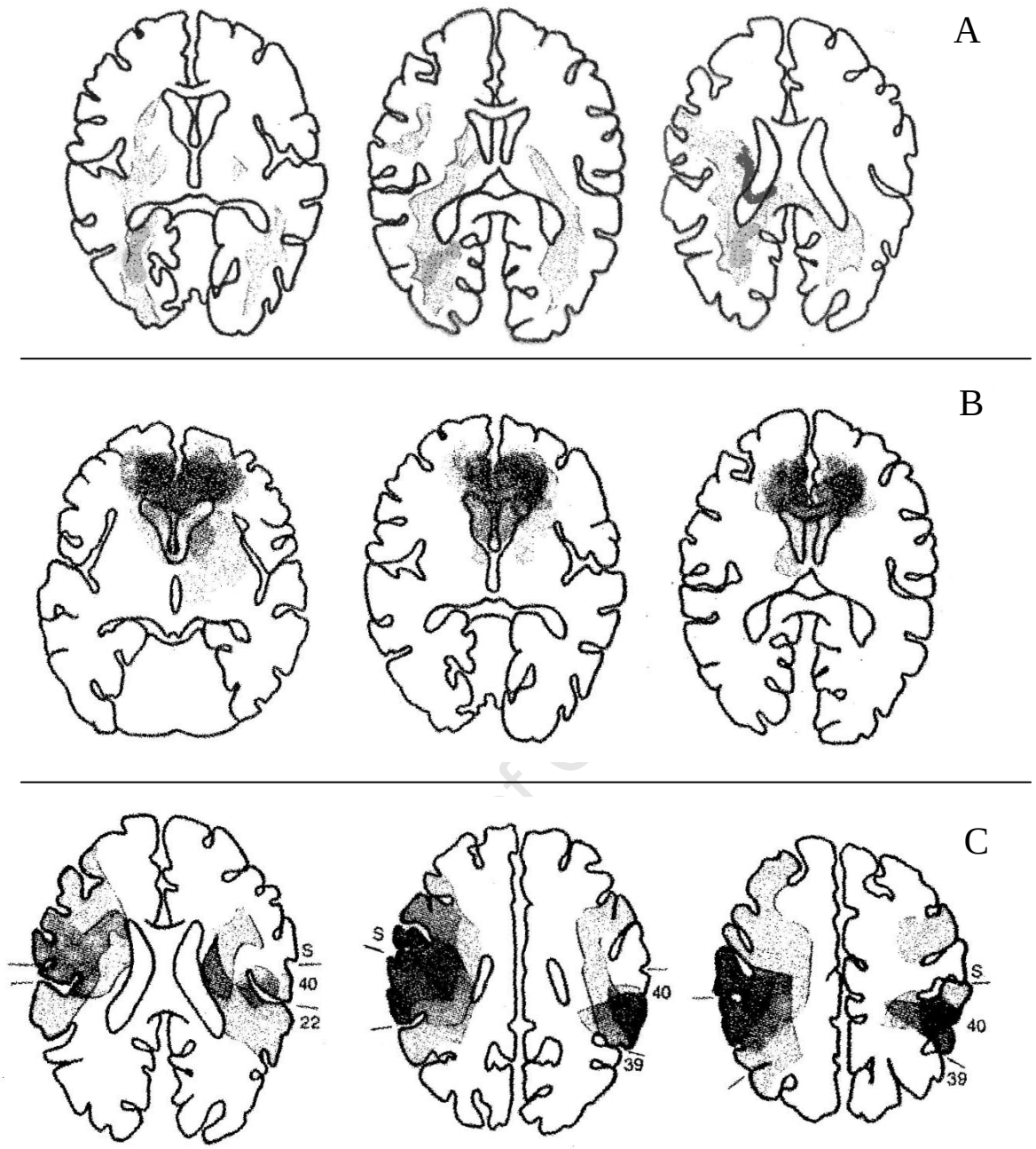


Figure 2. A) Overlap of two cases with cessation of visual dream-imagery; B) Combined facsimile of nine cases with global cessation of dreaming caused by deep frontal lesions; C) Combined facsimile of six cases with global cessation of dreaming caused by thrombotic infarction of the parietal lobe. Adapted from Solms, 1997, pp. 94-146.

Solms's Dream System

Solms (1997) had thus presented persuasive evidence that CWS was indeed a nosological amalgam. It was in fact, a hybrid of two distinct syndromes (although they overlap to some degree), each with its own pathological anatomical correlates, and each with its own neurobehavioural signs and symptoms:

(1.) Cessation of visual dream-imagery ("*Charcot's anoneira*" in Solms's nomenclature, p. 235) is invariably associated with irreminiscence, visuospatial short-term memory deficit, and to a lesser extent prosopagnosia, visual agnosia, topographical agnosia, and/or topographical amnesia, indicating a lesion involving the medial occipito-temporal regions bilaterally.

(2.) Total cessation of dreaming ("*Wilbrand's anoneira*" in Solms's nomenclature, p. 235) is typically associated with visuospatial short-term memory deficit when posterior cortical regions are involved, elements of Gerstmann's syndrome in unilateral left hemisphere lesions, and elements of right-hemisphere syndrome in unilateral right hemisphere lesions, indicating a lesion involving the inferior parietal lobe (specifically Brodmann's areas 39 and 40) of either hemisphere.

Solms's conclusions appear theoretically sound, since the lesion sites associated with either syndrome correspond with functional divisions of the intact, awake brain (Devinsky & D'Esposito, 2004; Mesulam, 1985). That is, Charcot's anoneira is a modality-specific disorder of dreaming, with a lesion in modality-specific association cortex, and Wilbrand's anoneira is a heteromodal disorder of dreaming affecting all modalities, with a lesion in higher-order heteromodal association cortex. The close proximity of these unimodal and heteromodal association cortices could easily explain the degree of overlap that may exist, in terms of the associated neurobehavioural deficits (Solms, 1997).

Solms (1997) integrated these observations with the then available literature concerning mental imagery, to provide a theoretical model of the normal dream process. He suggested that dreams occur when an arousing stimulus activates the dopaminergic circuitry in the mesocortical-mesolimbic pathway of the ventro-mesial frontal lobes. This activation is then backwardly projected onto posterior cortical regions, where "various perceptual and mnestic (occipito-temporo-parietal) mechanisms transform it symbolically and represent it concretely in the form of a visuospatial hallucination" (Solms, 1997, p. 246). These claims were widely accepted, but were also met with varying degrees of opposition from the dream science community, some of which posed fundamental challenges to the theory.

PET studies challenge Solms's Dream System

Solms's (1997) model was challenged on a fundamental level when a number of functional imaging studies that were published almost simultaneously with his, consistently reported deactivation of heteromodal association cortex, including the inferior parietal lobule, during REM sleep (Braun et al., 1997; Maquet et al., 1996; Nofzinger et al., 1997; Yu, 2001). Although these studies supported the assertion that deep bifrontal regions were critical to the dream process, consistently showing strong activation of limbic and paralimbic structures,

including ventral striatum, amygdaloid complex, hippocampal formation, and anterior cingulate cortex, and also supported the assertion that unimodal occipito-temporal structures were likewise critical, the inferior parietal lobule (IPL), including the supramarginal gyrus, was shown to be *deactivated* with equal consistency. Thus one of the posterior brain regions to which Solms (1997) had attributed important functions in dream processes were shown to be inactive during REM sleep, even when awakenings were performed to obtain dream reports (Maquet et al., 1996). It was thus implausible that a lesion in heteromodal association cortex could cause complete cessation of dreaming.

In order to resolve the disparities between Solms's (1997) clinicoanatomical study, and the PET studies at that time, Yu (2001) reanalysed an exhaustive body of literature in which precise anatomical data were provided. This included all of the case reports published prior to Solms's (1997) study, all of the available CT and MRI scans of the cases studied by Solms, and all of the available PET data published in the literature. He was able to chart the Brodmann's areas (BA) in many of these cases, and thus determined the extent of the involvement of inferior parietal cortex (BA 39), including the supramarginal gyrus (BA 40), in cases reporting complete loss of dreaming. He found that, out of the 61 cases published prior to Solms's study, only one case had a lesion circumscribed to BA 40, while the most common site involved was the temporo-occipital region (BA 18, BA 19, & BA 37; 16.4%). Investigation of Solms's (1997) original series revealed that 60% of his cases had lesions involving BA 40, but the same frequency of lesion was found in the superior temporal gyrus (BA 22). More specifically, of the 21 patients with BA 40 lesions, 76% also had lesions in BA 22, 48% also had lesions involving the temporo-occipital junction (BA 37), 38% also had lesions involving the thalamus, and 24% also had lesions involving V3 of the occipital lobe (BA 19)[3]. In short, Yu (2001) found that 90.5% of Solms's (1997) cases with lesions in BA 40 had concomitant lesions in the temporo-occipital junction and thalamus.

Since the PET studies had consistently shown deactivation of BA 39 and BA 40, but normal activation of BA 22, BA 37, and BA 19, Yu (2001) concluded that the loss of dreaming in Solms's series could be attributed to the occitpito-temporal lesions. A simple examination of Brodmann's areas in relation to the templates of Damasio and Damasio (1989) shows that Yu (2001) had identified medial occipito-temporal cortex as the critical brain regions in the dreaming process (Figure 3). The divergence between Solms's conclusions and the PET studies had thus apparently been resolved.

[3] The areas reported by Yu (2001) were not all the areas of damage identified in the literature he reviewed, but only those of importance to the controversy he sought to resolve. Likewise, I have not mentioned each and every area that Yu (2001) reported, merely those relevant to the current topic of discussion.

However, two recently published case studies have cast doubt on both Solms's (1997) and Yu's (2001) conclusions.

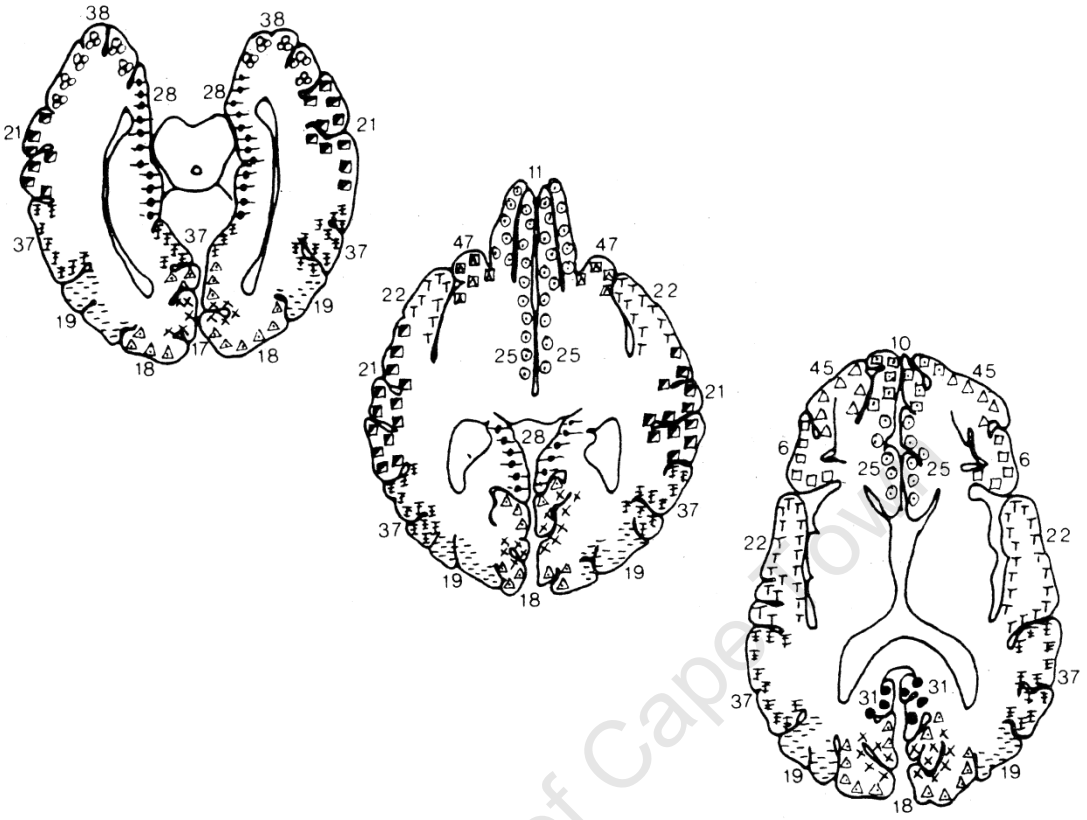


Figure 3. Cytoarchitectonic templates showing Brodmann's areas. Adapted from Damasio & Damasio, 1989, p. 185.

Cessation of Dreaming with Medial Occipito-Temporal Lesions

Bischof and Bassetti (2004) reported a case of a 73-year-old, right-handed woman who presented with complete loss of dreaming after non-hemorrhagic infarction in the territory of the posterior cerebral arteries (PCA). Brain MRI revealed an acute ischemic infarction of both deep occipital lobes, including the right lingual gyrus, and of the right posterolateral thalamus (Figure 4). In other words, their patient presented with *Wilbrand's anoneira* following a lesion in *medial* occipital cortex, extending into the occipito-temporal region, or unimodal visual association cortex. Although this is the precise location that Solms (1997) associated with Charcot's anoneira, the authors found no evidence of non-visual dreaming, or irreminiscence, prosopagnosia, or topographical agnosia/amnesia. These authors suggested that deep bilateral occipital damage, including the right lingual gyrus, might represent the minimum lesion extension necessary to produce CWS.

Soon after, the case of a 24-year-old man, who experienced total cessation of dreaming following a unilateral left occipito-temporal hematoma from a cerebral arteriovenous malformation (AVM), was reported (Poza & Martí-Massó, 2006). The authors

argued that a unilateral left occipito-temporal lesion was sufficient to cause complete cessation of dreaming. However, AVMs are nonlocalizable due to their widespread hemodynamic effects, and hemorrhagic infarctions often cause remote effects (Damasio & Damasio, 1989; Solms, 1997). Indeed the neuropsychological deficits reported by the authors (lack of initiative, memory impairment, and visual deficits) are suggestive of diffuse pathology possibly even affecting frontal regions, in addition to the occipito-temporal cortex. Thus localizing conclusions are not justified in this case.

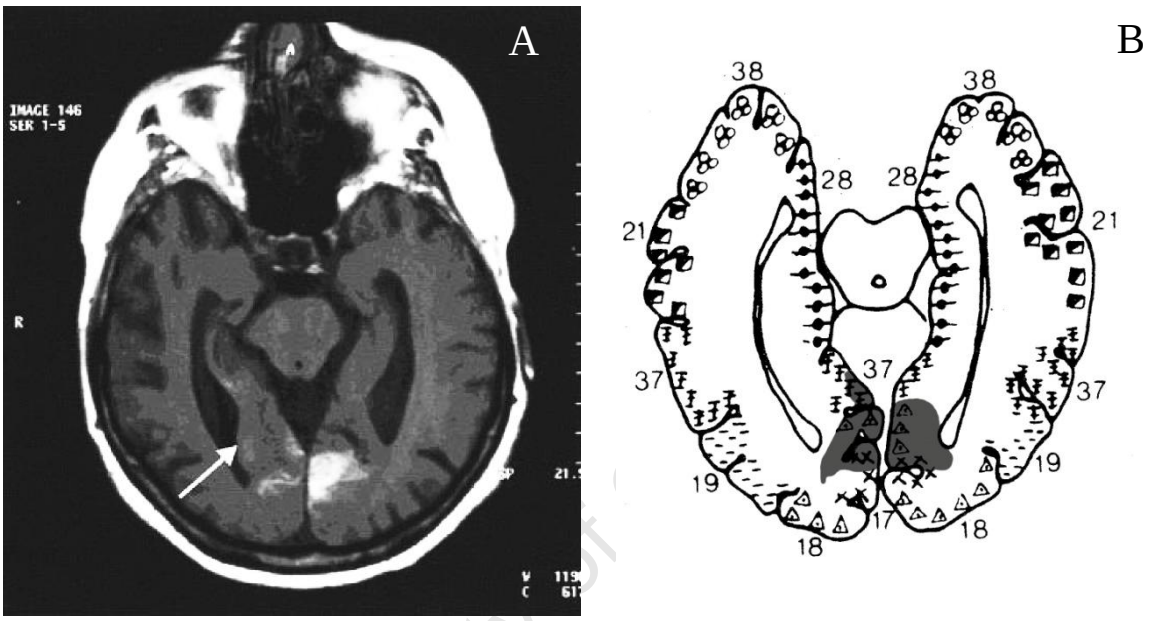


Figure 4. A) Original MRI-brain of Bischof and Bassetti's (2004) case. B) Brodmann's template showing lesion site in A.

On the other hand, the pathology in Bischof & Bassetti's (2004) case, i.e. non-hemorrhagic infarction, provides "the best specimen for neuroanatomical investigation and correlation with neuropsychological findings" (Damasio & Damasio, 1989, p. 95). Investigation of their patient's MRI-brain reveals that the lesion involved BA 37 (medially), BA 17, BA 18, and BA 19 (Figure 4). These sites are consistent with the bilateral medial occipito-temporal lesions that Solms (1997) had correlated with cessation of *visual* dream-imagery. They are, however, inconsistent with Solms (1997) who identified *lateral* posterior cortex as the lesion site correlated with complete cessation of dreaming. Bischof & Bassetti's case was unambiguously one of complete cessation of dreaming due to medial occipito-temporal infarction:

> "In conclusion, our case demonstrates the existence of *total dream loss* [italics added] as a distinct neuropsychological dysfunction after deep bilateral occipital lobe damage, in the absence of REM sleep changes."
>
> (Bischof & Bassetti, 2004, p. 586).

Thus Solms's careful differentiation of the occipital and parietal types of CWS appeared to have been undone.

A Return to Classics: Wilbrand (1887) Reconsidered

Bischof & Bassetti's (2004) case casts doubt on Solms's (1997) reformulation of Charcot-Wilbrand Syndrome. Specifically, it calls into question his assertion that nonvisual dreaming and complete cessation of dreaming are "independent entities *from both the clinical and anatomical points of view* [italics added]" (Solms, 1997, p. 91). At the very least, it presents a serious challenge to his attribution of modality-specific deficits of dreaming to lesions in modality-specific association cortex, and his attribution of heteromodal cessation of dreaming to lesions in heteromodal association cortex.

These challenges are greatly compounded when a reinvestigation of Wilbrand's case report is carried out. The following is an extract from an addendum that Wilbrand made in 1892 to his original publication:

> "*Autopsy report. Right hemisphere*: The lobus fusiformis has subsided deeply to form a limp, membranous sack extending to the pole of the occipital lobe. From above the entire occipital lobe appears to have subsided, with somewhat narrow convolutions but a surface that is nowhere softened. The posterior half of the cuneus is greatly reduced and softened. Its tip is contiguous with the focus in the fusiform lobe observed on the inferior surface. There is a slight change in the cortex of the fissura calcarina; *the precuneus is normal, as is the lateral surface of the occipital lobe and all parietal convolutions* [italics added].
>
> *Left hemisphere*: In the white radiation of the second occipital convolution, and a few millimetres underneath the grey cortex, in the depths of the fissure separating the first and second occipital convolutions, there is a small cavity that adjoins a softened zone at its frontal end (old focus). This softened zone gradually merges anteriorly into a fresh soft focus which has destroyed completely the central white matter of the hemisphere."
>
> (Translation by Solms et al., 1996, p. 98)

From the autopsy report above, it is clear that Fräulein G. suffered bilateral damage. Importantly, the report shows that the damage was to the *medial* surfaces of the occipito-temporal region, leaving the lateral surfaces intact (italics). The prototypical case of heteromodal cessation of dreaming, upon which Solms (1997) had founded his reformulation of CWS, was thus a case of bilateral damage involving unimodal association cortex (See Figure 1). Solms et al. (1996) and Solms (1997) may be forgiven for this oversight, in that their challenge to the CWS concept was wholly based on the individual case descriptions of the dreaming deficit, and the concomitant neuropsychological deficits respectively described by Charcot and Wilbrand. As mentioned earlier, Charcot's case never came to autopsy, making precise comparisons of Monsieur X.'s lesion with that of Fräulein G. impossible. And

Fräulein G. suffered a second stroke (in the left hemisphere) which obscures the status of the left parietal lobe at the time of the first stroke – the one that caused her loss of dreaming. Furthermore, Wilbrand's case of heteromodal cessation of dreaming was never confirmed by REM sleep awakenings. This applies also to all of Solms's (1997) cases. This makes Bischof & Bassetti's (2004) report the most empirically detailed case-study to date of loss of dreaming with posterior cortical lesions. However, one case does not a syndrome make.

From the above reviewed literature it is therefore apparent that the neurological correlates of global cessation of dreaming have not yet been adequately defined. Research is needed in order to establish whether medial occipito-temporal lesions do indeed regularly lead to complete loss of dreaming. That is, it should be established that Bischof & Bassetti's (2004) case is not unique. Furthermore, it is necessary to elucidate the exact nature of the lesion in such cases, if such an elucidation can be made. Such research would require precise localisation of the lesions in question and verification of the dreaming deficit by performing REM sleep awakenings in the controlled environment of a sleep lab. Only in this way can a clearer understanding of the mechanism of heteromodal loss of dreaming with damage to modality-specific cortex be achieved.

CHAPTER 2: RATIONALE FOR RESEARCH, AIMS, AND HYPOTHESES

The literature reviewed above indicates significant confusion regarding the neurological correlates of complete cessation of dreaming. Such confusion leaves a gap in our understanding of the mechanism of this disorder and therefore of dreaming itself. No theory of dreaming can be complete until all the critical brain regions responsible for dreaming have been established and validated through systematic study. No theory of the function of dreaming can be free of uncertainty when the systems, mechanisms, and individual brain regions upon which such theories are based, are themselves the topic of controversy.

This study aimed to bridge the gap in our current understanding of the mechanisms of complete loss of dreaming. In order to achieve this aim, this study sought to confirm whether heteromodal loss of dreaming can result from medial occipito-temporal lesions, and then to fully characterise the nature of the disorder and of such lesions provided that such confirmation was forthcoming. Therefore, this study adhered as close as possible to the methods of Bischof & Bassetti (2004), the most detailed study of heteromodal cessation of dreaming with medial occipito-temporal lesions to date. That is, patients with infarction in the territory of the PCA were subjected to REM sleep awakenings in order to determine whether or not they had the ability to dream. The lesions and neuropsychological status of patients who dreamt were then compared with patients who did not, in order to determine whether any characteristics were unique to either group.

In order to achieve these aims, the study described below tested the following hypotheses:

H_1: Complete cessation of dreaming can occur with lesions in medial occipito-temporal cortex (modality-specific association cortex).

H_2: The extent of the lesions in patients who cease to dream following medial occipito-temporal lesions will be different from that of lesions in patients who retain the ability to dream following medial occipito-temporal lesions.

CHAPTER 3: DESIGN AND METHODS

Design

A multi-case clinicoanatomical analysis was used to determine the neurological correlates of global cessation of dreaming in this study. The dependent variable measured was cessation of dreaming, while the independent variable was lesion location. Thus two groups were formed in order to compare the lesion locations of participants who ceased to dream following their stroke (quasi-experimental group), with those who did not (control group). Data were collected for both groups, while each participant was analysed as a separate case study in order to determine the relationship between their respective lesions, cessation of dreaming, and any other neuropsychological signs and symptoms that were found.

Setting

Arrangements were made to conduct this study in the sleep laboratory at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town. Further arrangements were made to conduct neuropsychological examinations for each participant at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town and at the neurology ward at Groote Schuur Hospital, Cape Town.

Participants

All participants were selected by referral from neurological specialists, working at Gatesville Medical Centre (Athlone) and Tygerberg Hospital (Belville). The neurologists were instructed to refer only those patients whom they suspected – on the basis of initial bedside questioning – might have lost the ability to dream following their stroke. Nine of the referred patients were asked to participate, while four were excluded as their neuropathology did not match the requirements for inclusion in this study. All participants had suffered non-hemorrhagic thrombotic infarctions in the posterior regions of the brain, corresponding with either a unilateral or bilateral posterior cerebral artery (PCA) territory infarction. The presence or absence of any dream reports from two REM sleep awakenings and two morning interviews following each night in a sleep laboratory was used to divide the referred patients into two groups.

Non-dreamers (Quasi-experimental group)

Non-dreaming participants consisted of neurological patients who had suffered a single thrombotic infarction in the PCA territory. Thrombotic strokes are ideal for lesion studies since they result in focal and circumscribed lesions, and create minimal confounding damage.

All lesions were thus focal and circumscribed. A strict inclusion criterion was that participants ceased to dream following their stroke. This was determined by participants' subjective accounts of cessation of dreaming, which was then verified in a sleep laboratory by awakening patients during REM sleep and asking them whether or not they had been dreaming. Exclusion criteria included the presence of any concurrent medical or sleep disorder that might have confounded the results, or the use of any medications that could have affected participants' sleep or dream recall.

Non-dreamers were selected by referral from neurological specialists at two separate institutions in Cape Town (Gatesville Medical Centre and Tygerberg Hospital). Due to this method of selection, sample size was dependent on the availability of patients with the correct neuropathology and corresponding lesions. It was predicted, based on the parietal cases studied by Solms (1997), that the occurrence of such patients would be extremely rare. Over a two-year period of searching for such patients, only three participants met the required inclusion criteria for the non-dreaming group, and were analysed in this study.

Dreamers (Control Group)

Dreaming participants again consisted of neurological patients who had suffered a single thrombotic infarction in the PCA territory. All lesions were focal and circumscribed.

Participants in the control group were from a similar age bracket to patients in the quasi-experimental group, which controlled for the effects that age has been shown to have on dream recall (Schredl, 2008). In addition, participants were of similar post-stroke chronicity. The strict inclusion criterion was that although the participants initially reported subjective loss of, or uncertainty regarding the presence of dreams following their stroke, at least one normal dream report was subsequently obtained. The preservation of dreaming was confirmed in the sleep laboratory by awakening participants during REM-sleep and asking them whether or not they had been dreaming. Exclusion criteria included the presence of any other medical or sleep disorder that might have confounded the results, or the use of any medications that could have affected participants' sleep or dream recall.

Participants were initially referred by neurological specialists on the suspicion that they may have lost the ability to dream following their stroke. However, after they reported dreams at REM sleep awakenings or morning interviews, they were designated as dreamers. Due to this method of selection, sample size was dependent on the availability of patients with the correct neuropathology and corresponding lesions. Six participants met the required inclusion criteria for the dreaming patients, and were analysed in this study.

Measures

Case History

The case histories for all participants were taken directly from their medical records and clinical interviews, with their consent. These included comprehensive details of their neuropathology, the onset, type, and severity of the cerebral accident, and the date of neurological assessment, as well as the written radiological reports detailing the observable lesion extent as imaged by their respective CT-brain and MRI-brain scans. In accordance with the APA guidelines for confidentiality and anonymity, any pertinent identifying information has been excluded before duplication of their case information herein (American Psychiatric Association, 2005). However, care was also taken to ensure that the relevant data were not distorted in any way.

Dream Recall

The presence, or lack thereof, of dreams since cerebral accident was initially identified by attending physicians. In clinical interviews, all participants were asked to provide subjective reports of whether they had noticed any change in their sleep and/or dreams since the occurrence of their stroke. Subjective reports of dream loss were verified by conducting semi-spontaneous nocturnal REM sleep awakenings, during the first of two nights in the sleep laboratory. Polysomnographic recordings were used to identify REM sleep in real-time by a registered sleep technologist. Participants were woken 10 minutes into the second REM period, and 15 minutes into the third REM period, or were interviewed after spontaneous awakenings during REM sleep and asked whether they had been dreaming and what was going through their mind just before waking up. Additionally, all participants were interviewed in the morning after each night in the sleep laboratory. These morning interviews consisted of brief questions regarding whether or not participants could remember any dreams other than those reported to the researcher during the REM sleep awakenings, and regarding the quality of their sleep during each preceding night.

Polysomnographic Measures

The PSG recordings were performed with a portable Alice © 5 *Respironics* polygraphic amplifier (Cape Sleep Centre, Gatesville Medical Centre, Cape Town). The American Association of Sleep Medicine (AASM) recommended recording montage was utilized in this study and included: electroencephalogram (EEG; 4 leads, 2 channels); electrooculogram (EOG; 2 channels); the submental electromyogram (EMG; chin and leg); as well as chest and abdominal strain gauges, snore microphone, positional marking and finger pulse oximetry.

Sleep stages were visually scored for 30-s epochs by a certified polysomnographic technologist based on AASM standard criteria (Hirshkowitz & Sharafkhaneh, 2009).

Neuropsychological Investigation

A number of neurocognitive tests were chosen for this study, focusing primarily on language, higher visual and spatial perception, visual and verbal short-term memory, and visual and audio-verbal long-term memory — In order to ensure that participants possessed the necessary neuropsychological abilities to recall and report their dreams. These tests were also used to assess for any other neurobehavioural signs and symptoms, in order to determine whether any relationships existed between these, the lesion site, and cessation of dreaming. The various neuropsychological subtests and scoring systems used are widely recognised and internationally established standard measures, and are used on a daily basis in the clinical practices of the neuropsychologists at Groote Schuur Hospital as part of the Groote Schuur Neuro-Cognitive Screening Battery (Mosdell, Balchin, & Ameen, 2010).

Language. Language ability was assessed using simple subtests for naming, comprehension, repetition, reading, and writing. A clinical judgment of the fluency of participants' language production (fluent or non-fluent) was made on the basis of their conversational abilities with the researcher. The Boston Naming Test (BNT) was used to assess confrontation naming (Kaplan, Goodglass & Weintraub, 2001). To assess repetition, participants were asked to repeat basic sounds, letters, words, and phrases, while reading was assessed by asking participants to read these linguistic components out loud. Similarly, writing was assessed by asking participants to write down these linguistic components. The aforementioned linguistic components, i.e. basic sounds, letters, words, and phrases were taken from the language items of Luria's Neuropsychological Investigation (LNI; Christensen, 1974).

Visuo-spatial perception. Visuo-spatial perception was assessed using selected subtests from the LNI, which included: 1) object recognition; 2) visual recognition of letters, words and phrases; 3) calculations; 4) colours and faces; and 5) language (Christensen, 1974). In addition, the BNT, which doubles as a language test, was also used for testing object recognition (Kaplan et al., 2001). Lastly, the line bisection test was used to test for the presence of visuo-spatial neglect (Schenkenberg, Bradford, & Ajax, 1980).

Constructional praxis. The Rey-Osterreith Complex Figure (ROCF) and the Wechsler Adult Intelligence Scale III (WAIS-III) Block Design subtest were used to assess perceptual organization and constructional praxis (Lezak, Howieson, Bigler, & Tranel, 2012).

Visual and verbal short-term memory. The Digit Span Test was selected from the Wechsler Memory Scale III (WMS-III), and used to assess audio-verbal short-term memory, and Corsi's Blocks were used to assess visual short-term memory (Lezak et al., 2012).

Visual and verbal long-term memory. Benton's Visual Retention Test (BVRT) was used to assess immediate visual recall (Lezak et al., 2012). The ROCF was also used to assess both immediate visual memory and long-term visual memory. The ROCF scoring system consisted of a copy trial, an immediate recall trial, and a delayed recall trial after approximately 30 minutes (Lezak et al., 2012). The Babcock Story was used for the assessment of long-term verbal memory (Babcock & Levy, 1930). In addition, drawings (with verbal description) of the South African flag and a canary, were included for the purpose of assessing the participant's ability to revisualise these objects from memory, without the aid of a copy (Lezak et al., 2012). Lastly, participants were asked to revisualise and verbally describe the interior layout of their own homes and the sleep laboratory, and verbally recount the experiential events surrounding the two nights in the sleep lab.

Procedure

All participants were subjected to an identical procedure. Informed consent from each participant was obtained before any data was collected. Participants were informed that the purpose of the study was to document the effects of cerebrovascular accidents (CVAs) on sleep and dreaming, in the hope of advancing our understanding of the function of dreaming. It was made clear to each participant that their participation was voluntary and that they could withdraw from the study at any time. Participants' medical files were analysed in order to determine the onset, type, and severity of cerebral accident and to provide detailed summaries of clinical and neurological symptoms. Age, sex, and time since stroke were also recorded. MRI-brain scans, CT-brain scans, and accompanying reports were further analysed to confirm details of the lesion site.

This study comprised three primary components: 1) assessment of dream experience, 2) neuropsychological assessment, and 3) analysis of brain lesions.

Assessment of dream experience

Following the initial bedside questioning, the sleep study took place over two consecutive nights at the Cape Sleep Centre, Gatesville Medical Centre, Cape Town. The first night functioned as the experimental night. The second night functioned as the experimental night for a different study (Cameron-Dow, 2012). Participants were restricted in the use of caffeine-containing liquids and other stimulants. During both nights, the participants were monitored by the principal researcher and a qualified sleep laboratory nurse, and the polysomnograhic recordings were monitored by a qualified sleep technologist.

First night. During the first night participants were connected to a polysomnograph and simply asked to sleep as they would normally at home. Nocturnal REM sleep interviews were conducted to confirm participants' subjective accounts of the presence, or lack thereof, of dreaming. These REM sleep awakenings were made 10 minutes into the second REM period, and 15 minutes into the third REM period. Alternatively, if the patient spontaneously woke during either one of these periods before the allotted time had passed, a dream report was obtained. The participants were awakened in accordance with the specific physiological variables of REM sleep present in the EEG recordings (Rechtschaffen & Kales, 1968). Additionally, participants were interviewed in the morning, and briefly asked if they could recall any other dreams, other than the ones recalled, or not recalled during the REM sleep awakenings.

Second night. Participants were once again connected to a polysomnograph and were asked to sleep as they normally would at home. Participants were not awaked by the researcher during the night. Detailed polysomnographics were recorded in order to measure the quality and quantity of sleep as part of a separate study testing a hypothesis regarding the functions of dreams (Cameron-Dow, 2012). However, in the morning participants were again briefly interviewed and asked if they could recall any dreams from the preceding night. Additionally, cessation of dreaming as opposed to lack of ability to remember dreams was confirmed by obtaining verbal episodic recall reports on the second morning of what had happened during the two preceding nights, from arrival at the sleep lab until falling asleep, during the lab awakenings, and in the morning before departing the sleep lab.

Thereafter, participants were thanked for their participation in the study and were compensated in accordance with the participation agreement (see below).

Neuropsychological assessment

Neuropsychological testing took place at the neurology ward at Groote Schuur Hospital. Assessment took place in a quiet, distraction-free room. Patients' subjective reports on dreaming and sleep since onset of stroke were recorded. A range of neuropsychological tests,

aimed at testing higher visual and spatial perception, visual and verbal short-term memory, and visual and verbal long-term memory were administered.

Analysis of brain lesions

All MRI-Brain and CT-Brain scans for participants were obtained from the existing clinical records. Since some participants had undergone multiple scans on different dates, the scan taken after their CVA on the date closest to and preceding that of the sleep laboratory dream study and neuropsychological investigations was chosen for analysis. A detailed report of the lesion site and extension corresponding to PCA infarction was provided with each scan by the radiological departments where they were performed. All lesions were then coded by Brodmann's areas, in accordance with the atlas of Damasio & Damasio (1989). A descriptive analysis was then carried out in order to determine if any lesion characteristics were unique to either group.

Data analysis

Each participant was analysed as a separate case study. Detailed information regarding neurological damage, and neuropsychological functioning, was analysed according to the dependent variable of presence, or lack thereof, of dreams. In addition, data collected for the non-dreaming patients was compared to data collected for the dreaming patients.

This study adopted a clinicoanatomical approach in the analysis of data. Each participant was analysed as a case study in comparison to matched controls. The MRI-Brain and CT-Brain scans, in combination with the neurology reports, were analysed to determine the precise location, laterality and acuteness of the lesions. These were then related to respective dreaming experience.

Ethical Considerations

This study adhered to the ethical guidelines for research with human subjects as specified by the Health Professions Council of South Africa (HPCSA), as well as the University of Cape Town (UCT) Codes for Research. Ethical approval was granted by the Psychology Department's Research Ethics Committee at UCT as well the Faculty of Health Sciences Research Ethics Committee at UCT. Data was only collected after ethical approval had been obtained. In addition, consent from the Cape Sleep Centre, Gatesville Medical Centre and the neurology department at Groote Schuur Hospital was obtained before their facilities were utilized.

Informed consent was obtained in writing from each participant before any data was collected (Appendix). The consent form outlined that participation was voluntary, that

participants were able to withdraw at any time without negative consequences for themselves, and that all data would be kept confidential and would only be used for research purposes. The consent form also obtained consent for participant's medical folders to be reviewed and for their medical information to be used confidentially.

There were no major risks associated with the administration of the study. However, if participants were uncomfortable at any time it was reiterated to them that they were able to withdraw from the study at any time. Participants were also made aware that there were no direct benefits for participating in this study other than the benefit of identification of sleep disorder if present.

Participants received a monetary compensation of R500 per night spent in the sleep laboratory (approximately £50 or $75).

CHAPTER 4: RESULTS

Participants

A total of 14 patients were referred for participation in this study. However, four patients were excluded as their pathology was deemed inappropriate for inclusion[4], leaving a total of nine patients who were asked to participate.

Non-dreamers

Three participants matched the inclusion criteria for non-dreamers. All three participants were female. The mean number of years of education for the non-dreamers was 10 years. The mean age for the non-dreamers was 60-years old. Mean chronicity (number of weeks between onset of stroke and the sleep lab assessment) was 85 weeks.

Dreamers

Six dreamers were matched with the three non-dreamers. Participants in this group consisted of two females, and four males. The mean number of years of education for the dreamers was 9 years. The mean age of dreamers was 56-years old. Mean chronicity was 57 weeks.

Table 1.

Demographic Characterstics of All Participants

Group	*Years of Education*	*Age*	*Chronicity* ¥
Non-dreamers (n=3)			
Case 1	9	61	232
Case 2	12	62	22
Case 3	10	56	1
Mean	*10*	*60*	*85*
Dreamers (n=6)			
Case 4	13	45	224
Case 5	10	60	76
Case 6	6	51	29
Case 7	11	67	9
Case 8	3	56	1
Case 9	12	54	1
Mean	*9*	*56*	*57*

¥ Number of weeks between onset of stroke and sleep lab assessment

[4] Two patients had suffered thrombotic infarction in the territory of the middle cerebral artery (MCA). One patient had suffered a haemorrhage in the right occipital lobe with vasogenic oedema and mass effect. One patient had suffered a small infarct in the posterior horn of the internal capsule, in the context of small vessel disease.

Case Studies

Non-dreamers

Case 1: Mrs PA

Date of birth: 01/07/1945
Date of CVA: 25/02/2006
Date of sleep study: 25/08/2010 and 26/08/2010
Chronicity: 232 weeks

History of Stroke

Mrs PA, a right handed female with nine years of formal education, presented to Gatesville Medical Centre, Cape Town, on 25 February 2006 with sudden onset blindness and severe headache accompanied by severe dizziness, and posterior neck pain.

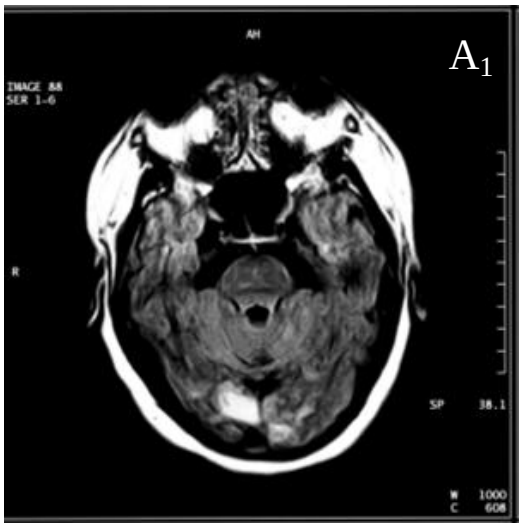
Initial work-up revealed hypothyroidism, micro voltages on ECG, and a thickened myocardium suggestive of an infiltrative process.

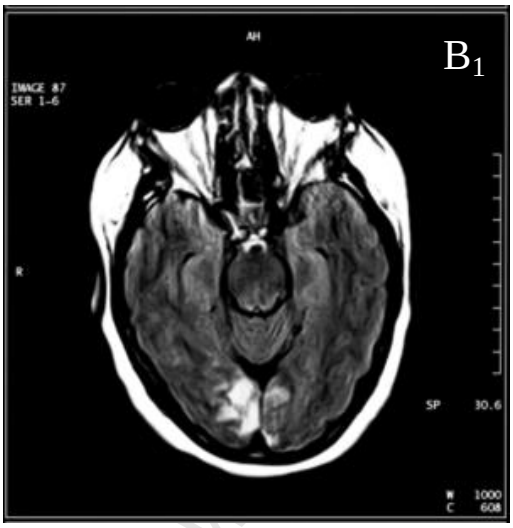
Neurological examination revealed an abnormal mental state characterized by disorientation to time, axial amnesia with forced confabulatory features, executive dysfunction characterized by difficulties with complexity and problem solving, and labile mood. Of note was the absence of visual agnosia or aphasia, and general judgement was preserved. Neurological examination further revealed cranial nerve dysfunction with a left superior quadrantanopia, large and small fibre sensory neuropathy, and a mild ataxic gait. No other focal localizing signs were noted. Mrs PA was also found to be photophobic, while blurred vision remained a consistent complaint. She emerged from her confusional state and regained the ability to lay down new memories approximately seven days after the initial onset of symptoms.

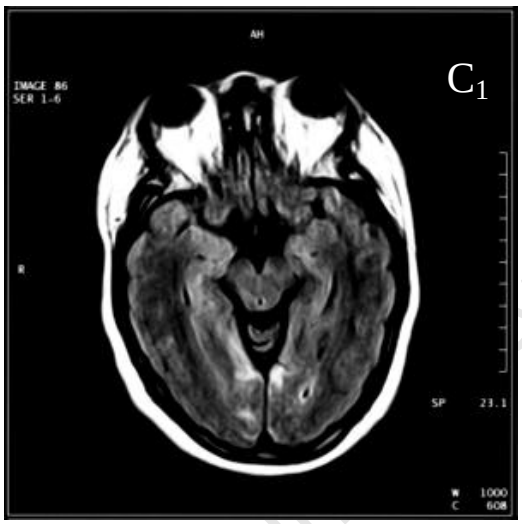
MRI-brain (Figure 5), taken 14 days after initial onset of symptoms, demonstrated acute infarcts in both occipital lobes (larger on the right) and the right cerebellar hemisphere. High signal was noted on T2 and FLAIR sequences in the thalami bilaterally. MRA revealed a tight basilar stenosis at the confluence of the proximal basilar and vertebral arteries. CT angiogram showed extracranial vasculature to be normal, with no features of dissection. Gradient echo sequences showed no evidence of haemorrhage.

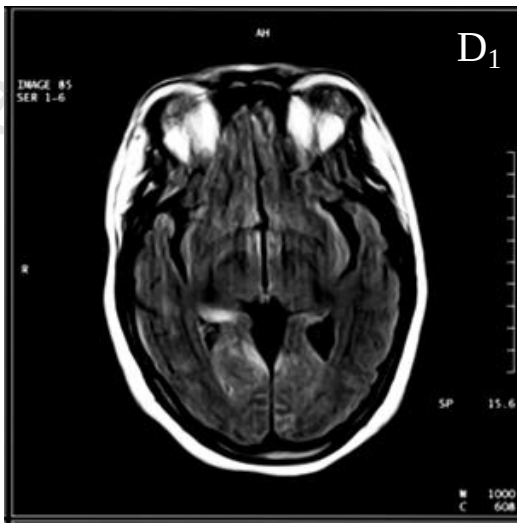
Neuroradiological Findings

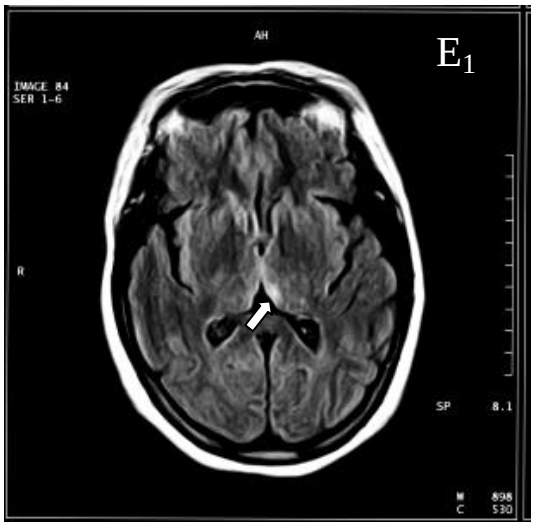
Figure 5. MRI-brain: Case 1











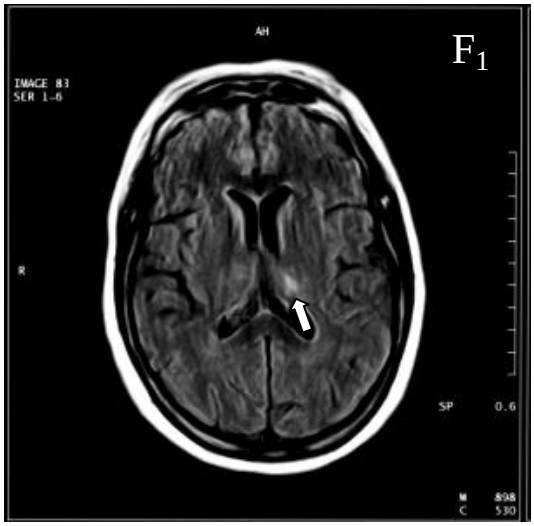
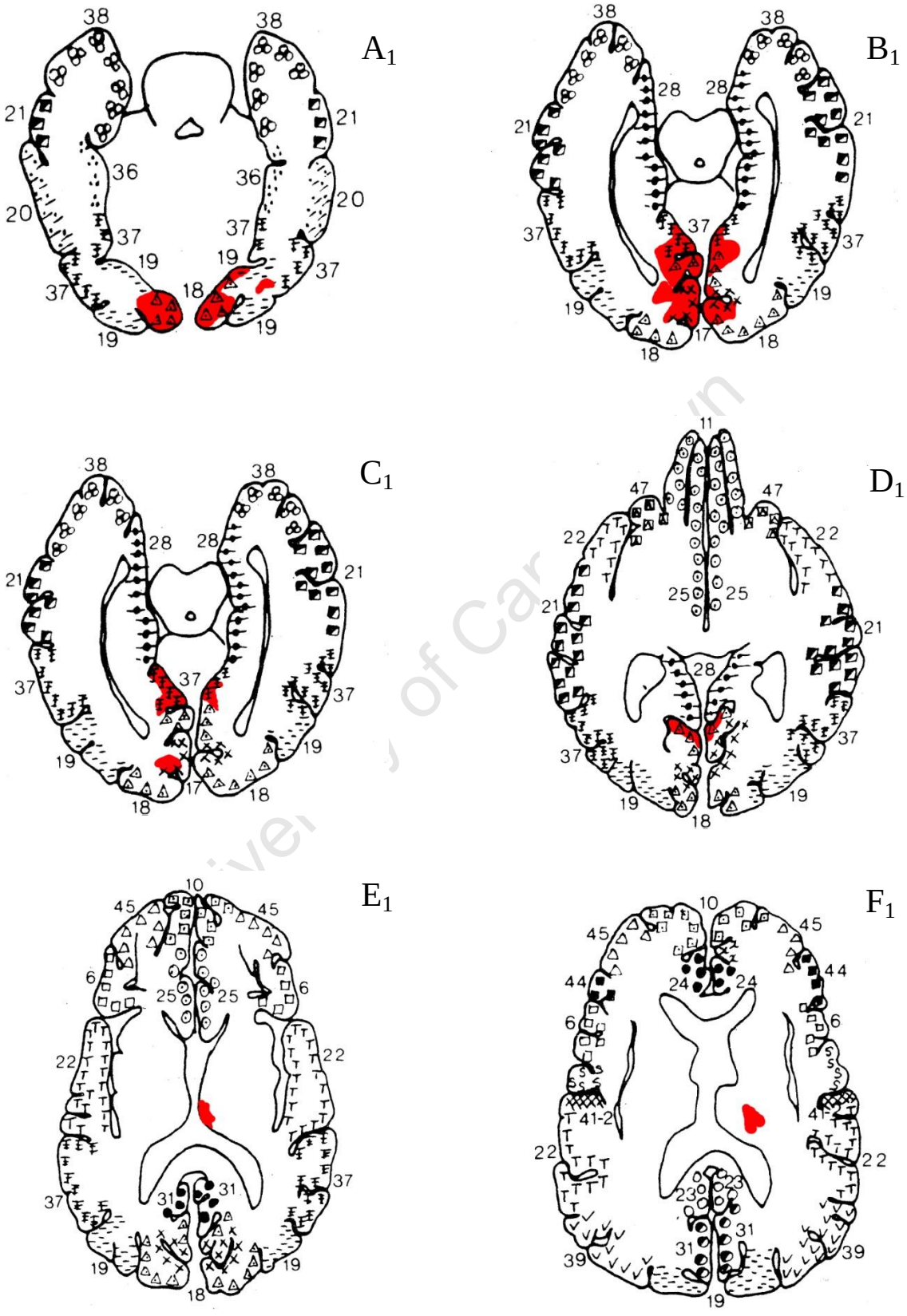


Figure 6. Brodmann templates depicting lesion sites: Case 1



The MRI-brain above clearly shows a bilateral lesion in the territory of the PCA, involving the medial occipito-temporal areas, including the left thalamus (white arrows – Figure 5, E_1 and F_1).

Depiction of the lesion on the cytoarchitectonic templates (Figure 6) indicates a lesion involving BA 17, BA 18, and BA 37 bilaterally, BA 19 on the left, as well as involving the left postero-lateral thalamus.

Table 2.

Case Summary: Case 1

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mrs PA	61	232 weeks	Bilateral	BA 17	BA 17
				BA 18	BA 18
				BA 19	
				BA 37	BA 37
				Thalamus	

Dream Recall

Mrs PA reported that she had not been able to dream since her stroke. This remained consistent over multiple clinical interviews. She reported that before her stroke she dreamt regularly, especially when she had worries, but that she had not dreamt at all since her stroke. This indicated that complete cessation of dreaming had occurred as a result of her stroke, and that dreaming had ceased for 4 years, 5 months since her stroke.

This was confirmed in the sleep laboratory by conducting nocturnal REM sleep interviews from unprompted awakenings during the first and second REM periods. During both interviews, Mrs PA reported that she had not been dreaming or experiencing any thoughts or mentations.

On a follow-up interview (4 years, 7 months post stroke; 1 month post sleep study), Mrs PA reported that she had experienced dream-like mentation two weeks prior, but was unsure of whether or not it constituted a dream. The following transcript indicates Mrs PA's recall of dream-like mentation:

"Mrs PA: I did have a dream but it wasn't really a dream.

Researcher: When did this happen?

Mrs PA: A couple of weeks ago. My aunt came to me in the dream because my brother-in-law is dying of cancer now.

Researcher: How do you know it was a dream?

Mrs PA: Because I saw her. To me it meant that she is coming to tell us that my brother-in-law, who is passing away, that his time is near. [This was explained further]

Researcher: Why do you say it's not actually a dream?

Mrs PA: Because the only thing I remember was seeing my aunt. Then I just had a feeling I had to go to my sister. [Explained what happened when she went to her sister]

Researcher: Did you just think about her, or feel her presence, or did you see her?

Mrs PA: No, I actually saw her.

Researcher: How much of her did you see?

Mrs PA: I just saw her face.

Researcher: What happened next?

Mrs PA: I didn't wake up. The next morning I just remembered that I saw her.

Researcher: So do you think it was a dream?

Mrs PA: No. It wasn't a dream.

Researcher: Please clarify.

Mrs PA: Because nothing happened in it. It was just her face. There was no story."

This possible hypnogogic hallucination indicated that Mrs PA may have begun to recover dream function at that stage.

Neuropsychological Assessment

Neuropsychological examination (Table 3) demonstrated that higher visuo-spatial perception was intact with normal performance on Luria's tests of higher visual perception and integration. Performance on the Boston Naming Test (BNT) was good.

Mild constructional apraxia was noted, with a poor copy of the Rey-Osterrieth Complex Figure (ROCF), and a loss of the overall gestalt. Performance on the WAIS-III Blocks was marginally better with 5 consecutive answers correct.

Performance on the Digit span test and Corsi's blocks showed normal visual and verbal short-term memory.

Tests of visual and verbal long-term memory showed a mild new learning deficit. However, revisualization of the sleep laboratory, the sleep technician, the South African flag,

a canary, and her husband's face were all excellent, showing revisualization to be entirely intact.

In particular, Mrs PA was able to recall the sleep laboratory procedure in accurate detail. The following transcript indicates Mrs PA's memory of the sleep laboratory procedure recorded one month after the sleep study:

> "I thought…I didn't absolutely hear him say Gatesville. That's why we came to Groote Schuur. We sat here 'til 8 O'clock at the reception area there (indicates). Then I said No…Nobody's phoning, so we went home. I got into bed. Then at 10 O'clock […husband interrupts] No it was before 10 O'clock because we got there at 10. Before 10 O'clock Brian called and said it's at Gatesville; we must come straightaway now. We can still make it. So I got up and got dressed and we went to Gatesville at 10 O'clock. Then Brian started preparing me for the sleep. He put all the gadgets on my face and my head and there was a monitor here (points to chest) and round my waist. {The monitor box was here (indicates left side)}. Then I was *supposed* to have gone to sleep. But I couldn't sleep. I usually sleep very quickly, but I just couldn't that night. Then just before Prof came to wake me up, I think I had just dozed off. I had just started to sleep that time when you came to wake me. You said I must go back to sleep, so I fell off back to sleep again. Then you woke me up again. Then I fell off to sleep again. Then I woke up at 6 O'clock when the sister came in. Then I went home."

In addition, Mrs PA was able to accurately revisualize the sleep laboratory room where the sleep study had taken place. The following transcript indicates Mrs PA's memory of the room in sleep laboratory recorded one month after the sleep study:

> "It was just one room…with a bed and a cupboard and a window, with blinds. There was a camera in there too. It was a small room, I don't know the measurements. It was beige. Outside the room was like a ward with beds. There were three that side and three this side; about six."

Therefore, neuropsychological assessment indicated that Mrs PA had mild constructional apraxia and a mild new learning deficit. In light of good performance on tests of visuo-spatial perception, short-term memory, and revisualization, as well as overall clinical impression, it was concluded that Mrs PA had the cognitive functions necessary for dream recall. In particular, accurate recall of the sleep laboratory procedure and room indicated that Mrs PA had the necessary cognitive capacity for dream recall.

Table 3.

Neuropsychological Assessment: Case 1

Test	*Score*
Luria's Visual Scenes	10/14
Boston Naming Test	57/60
Rey-Osterrieth Complex Figure	
Copy	25/36
Immediate	4.5/36
Delayed	-
WAIS-III Blocks (Scaled Score)	29/68 *(9)*
Digit Span	
Forwards	6
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	5
Babcock Story Recall	
Trial A	7/21
Trial B	8/21
Trial C	-
Benton's Visual Retention Test	6/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 2: Mrs MC

Date of birth: 10/01/1949
Date of CVA: 02/07/2011
Date of sleep study: 12/07/2011, 13/07/2011 and 29/09/2011
Chronicity: 22 weeks

History of Stroke

Mrs MC, a right handed female with 12 years of formal education, presented to Gatesville Medical Centre, Cape Town, on 7 July 2011 with a history of parasthesiae and numbness in her left upper limb, as well as an episode where she was dragging her left lower limb. Her family reported a six day history of confusion that had persisted and worsened over the following days. She also complained that vision had been affected.

This episode followed a motor vehicle accident three weeks prior, where she had injured her leg and fractured her patella. No head trauma was noted. After undergoing surgery on her leg, she developed severe hypertension and diabetes mellitus.

On initial neurological examination, she complained of a headache that seemed to emanate from the region of the left orbit and radiate to the vertex of the left head. The family further reported confusion and poor memory.

Further neurological examination revealed an abnormal mental state with disorientation to day and month, difficulties reading, and poor immediate recall. She displayed normal attention and no evidence of aphasia. Cranial nerve examination was normal. Power could not be tested in the right lower limb as the leg was bandaged and strapped, but power was normal in all other limbs. Sensation, coordination, gait, and fine motor movements were all normal.

MRI-brain (Figure 7), taken on the day of admission, demonstrated acute infarcts in the right basal ganglia, the right thalamus, and both occipital poles (worse on the right). MRA revealed atheromatous disease of the anterior and posterior intracranial circulation, with very tenuous vertebral arteries bilaterally, as well as stenosis in the basilar artery.

Neuroradiological Findings

*Figure 7***.** MRI-brain: Case 2

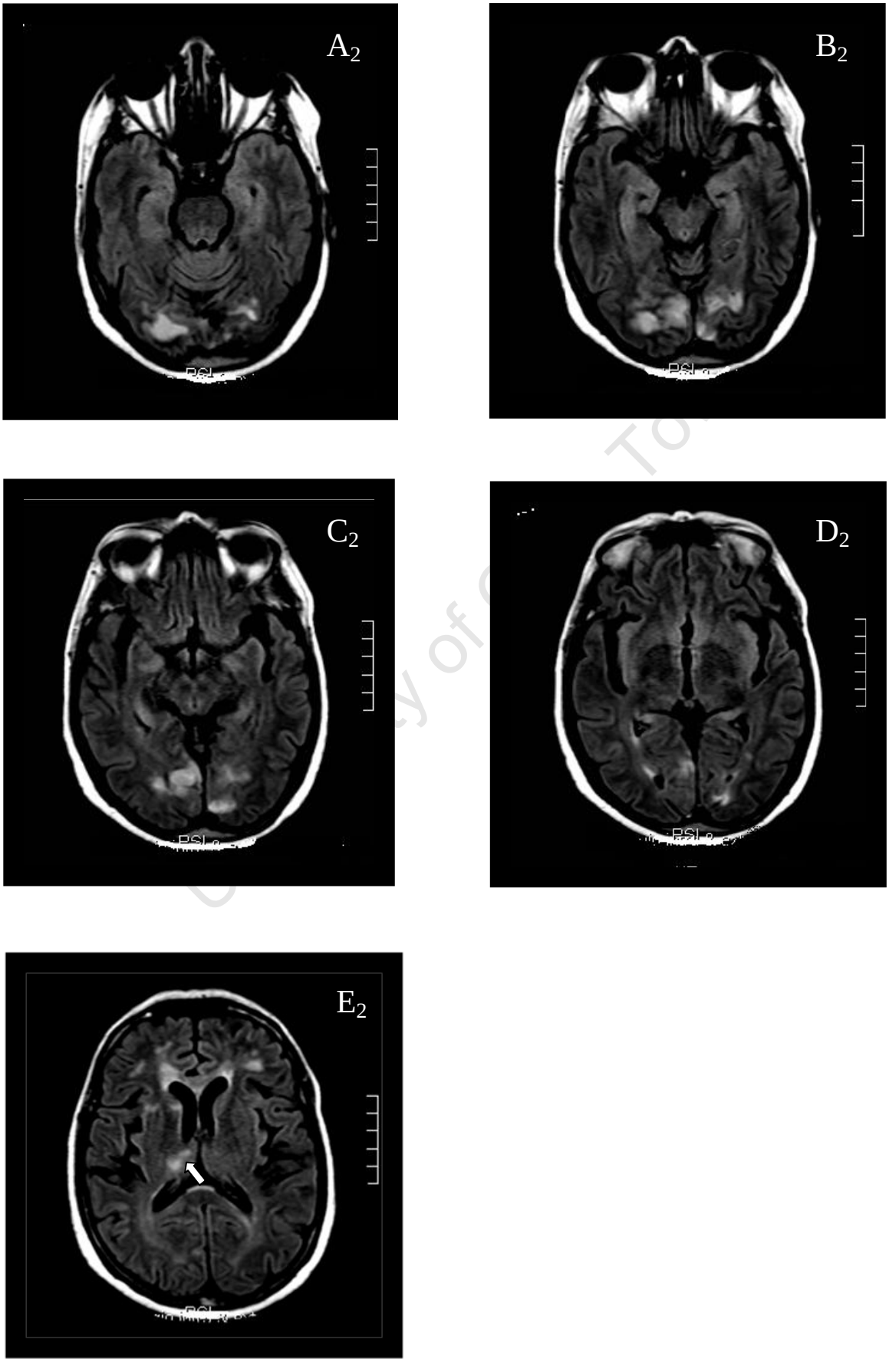
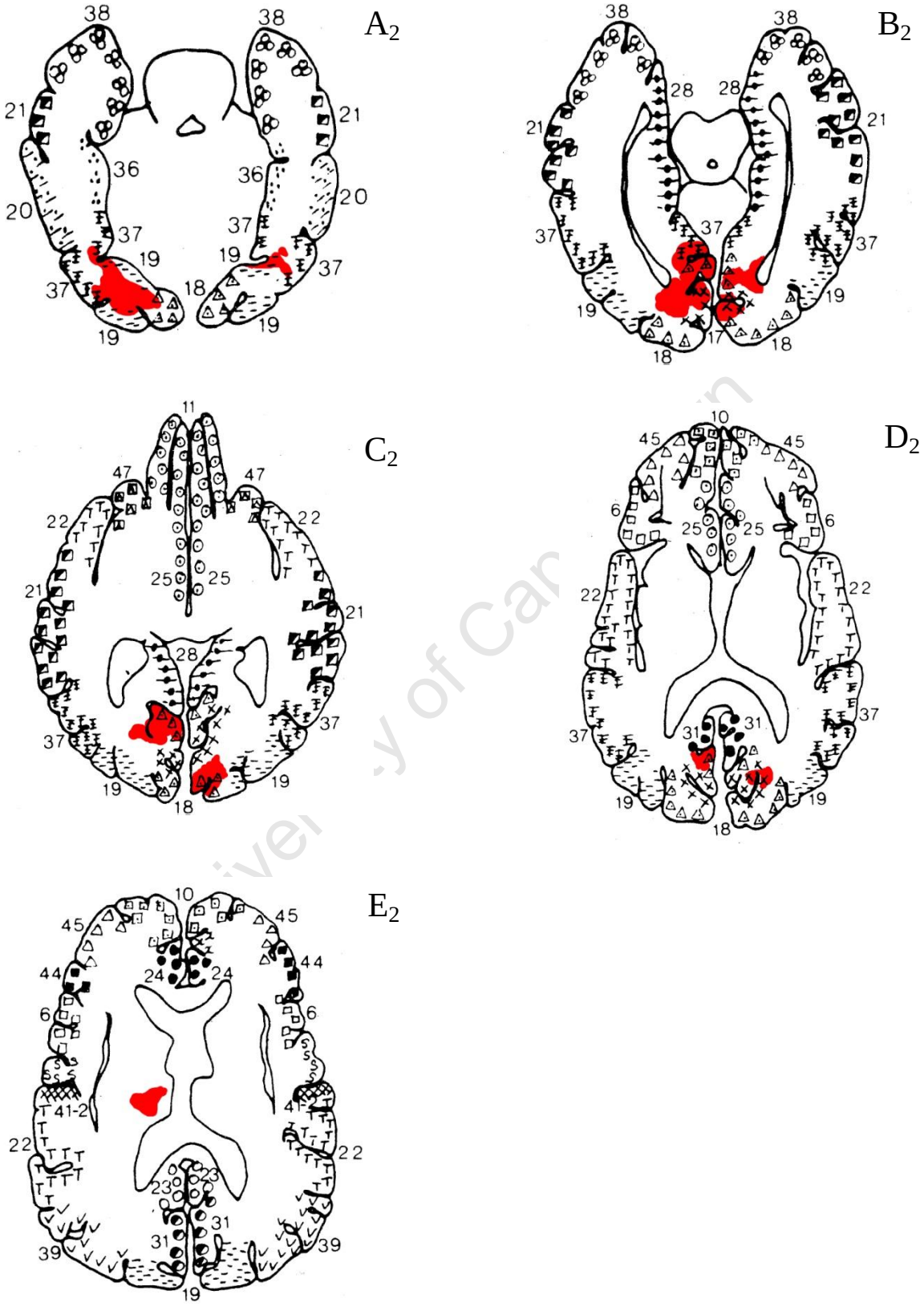


Figure 8. Brodmann templates depicting lesion sites: Case 2



The MRI-brain (Figure 7) above clearly shows a bilateral lesion in the territory of the PCA, involving the medial occipito-temporal areas, including the right thalamus (white arrow – Figure 7, E_2).

Depiction of the lesion on the cytoarchitectonic templates (Figure 8) indicates a lesion involving BA 36 and BA37 on the right, the right posterior thalamus, as well as BA 18 and BA 19 bilaterally.

Table 4.

Case Summary: Case 2

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mrs MC	62	22 weeks	Bilateral	BA 18	BA 18
				BA 19	BA 19
					BA 36
					BA 37
					Thalamus

Dream Recall

Mrs MC reported that she had not been able to remember any dreams since her stroke. This remained consistent over two clinical interviews, two months and three months post stroke. She reported that before her stroke she had dreamt frequently, indicating that complete cessation of dreaming had occurred as a result of her stroke.

This was confirmed in the sleep laboratory. Nocturnal REM sleep interviews could not be performed on the first night in the sleep laboratory as Mrs MC did not enter REM sleep on that night. However, lack of dream recall in the morning and from unprompted awakenings during the first and second night in the sleep laboratory supported that she was, in fact, not dreaming. In addition, a third sleep study night was specifically designed to confirm the lack of dreaming. Whilst a typical REM-NREM cycle was achieved on this third night, no positive dream reports were obtained.

Neuropsychological Assessment

Neuropsychological examination revealed palinopsia, complex visual hallucinations, tongue and lip anaesthesia, and photophobia, all of a paroxysmal nature.

Neuropsychological testing (Table 5) indicated that higher visuo-spatial perception was mildly impaired with very mild apperceptive visual agnosia evident on Luria's

Neuropsychological Investigation, and slight apperceptive errors on the BNT. Naming was shown to be intact.

Constructional praxis was shown to be intact with a good copy of the ROCF and adequate performance on the WAIS-III Blocks.

Performance on the Digit span test and Corsi's blocks showed normal visual and verbal short-term memory.

No visual and verbal long-term memory deficits were noted, and revisualization was intact. In particular, Mrs MC was able to accurately recount the sleep lab experience one month later, including details of the sleep laboratory nurse, the layout of the room, and the procedure of attaching the polysomnograph leads.

Therefore, neuropsychological assessment indicated that Mrs MC had a very mild apperceptive visual agnosia. Overall performance on all tests as well as clinical impression concluded that Mrs MC had intact functions necessary for dream recall. In particular, accurate recall of the sleep laboratory procedure and room indicated that Mrs MC had the necessary cognitive capacity for dream recall.

Table 5.

Neuropsychological Assessment: Case 2

Test	*Score*
Luria's Visual Scenes	11/14
Boston Naming Test	46/60
Rey-Osterrieth Complex Figure	
Copy	32/36
Immediate	15/36
Delayed	16/36
WAIS-III Blocks (Scaled Score)	20/68 *(7)*
Digit Span	
Forwards	5
Backwards	6
Corsi's Blocks	
Forwards	6
Backwards	4
Babcock Story Recall	
Trial A	7/21
Trial B	12/21
Trial C	-
Benton's Visual Retention Test	6/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 3: Mrs HS

Date of birth: 31/08/1956
Date of CVA: 27/10/2012
Date of sleep study: 05/11/2012 and 06/11/2012
Chronicity: 1 week

History of Stroke

Mrs HS, a right-handed female with ten years of formal education, presented to Gatesville Medical Centre, Cape Town, on 27 October 2012 with sudden onset of weakness in the right arm. She had a history of hypertension, poorly controlled diabetes mellitus, hypercholesterolemia, vertigo and dizziness, and chronic bilateral sensorineural hearing loss.

No abnormalities were detected on initial examination. Neurological examination, however, revealed a normal mental state, and the absence of aphasia was noted. Assessment of the fundi and visual fields indicated the presence of a right homonymous hemianopia, without any abnormalities of the cranial nerves. Examination of the motor system revealed normal tone, power, and generally reduced reflexes. Sensation was normal. Coordination was normal and there was no evidence of any cerebellar dysfunction. However, the gait was ataxic when she walked, and she struggled to tandem walk forwards and backwards. Fine motor movements were normal.

MRI-brain (Figure 9) performed two days after onset of symptoms indicated a lesion in the left occipital lobe. MRA-brain revealed partial occlusion of the left posterior cerebral artery.

Neuroradiological Findings

Figure 9. MRI-brain: Case 3

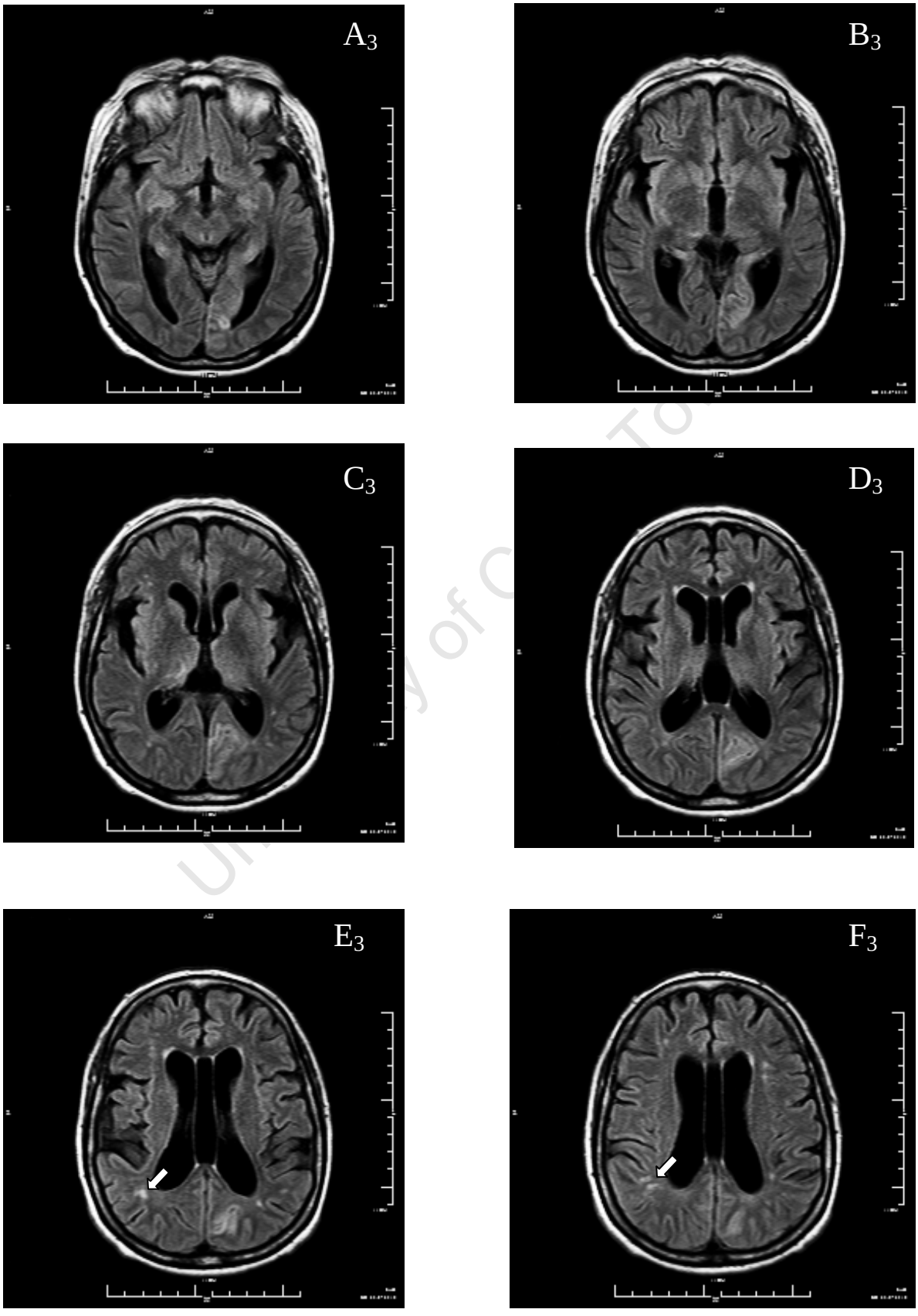
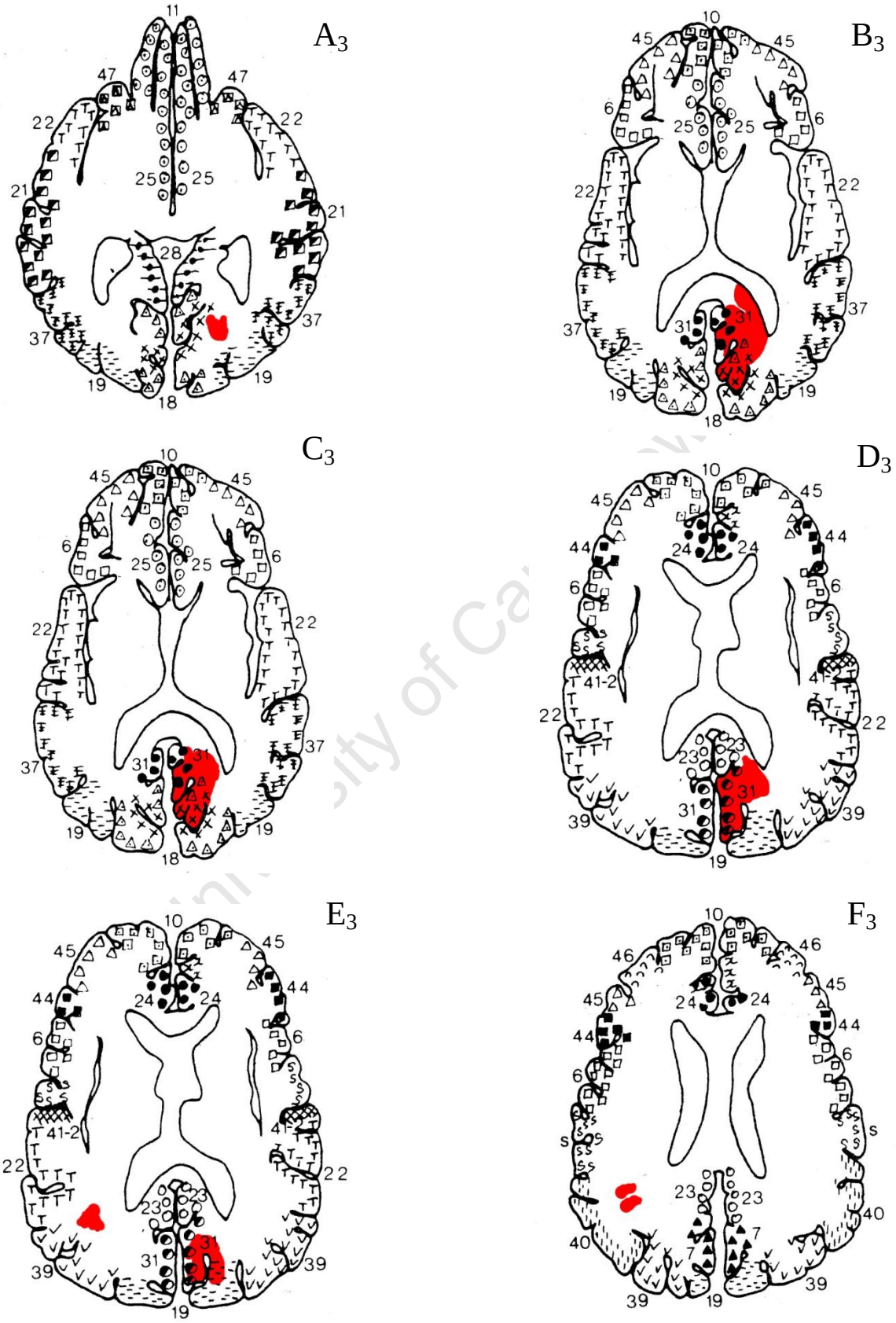


Figure 10. Brodmann templates depicting lesion sites: Case 3



The MRI-brain (Figure 9) above clearly shows a lesion in the territory of the left PCA, involving the medial occipito-temporal area. There is also a discrete hyperintensity in the parenchyma of the right parietal lobe (white arrows – Figure 9, E_3 and F_3). Thus, as with Case 1 and Case 2, the MRI shows bilateral damage.

Depiction of the lesion on the cytoarchitectonic templates (Figure 10) indicates a lesion involving BA 17, BA 18, and BA 19, with extension to BA 31 on the left. On the right, the lesion involves BA 39 and BA 40.

Notably, Mrs HS suffered another ischemic infarction one month after (20/11/2012) this first event. Another MRI performed one day after onset of symptoms revealed an acute right thalamic and prerolandic infarct.

Figure 11. MRI-brain and Brodmann template: Case 3's subsequent CVA

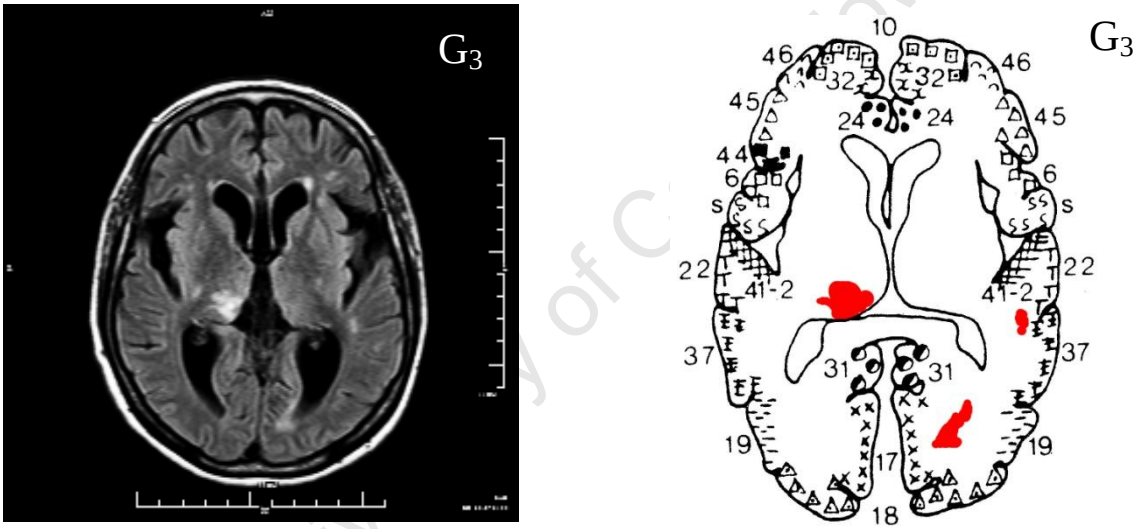


Table 6.

Case Summary: Case 3

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mrs HS	56	1 week	Bilateral	BA 17	
				BA 18	
				BA 19	
				BA 31	
					BA 39
					BA 40

Dream Recall

Mrs HS was assessed after her first stroke only. She reported that she could not remember having dreamt since her stroke. This was verified by REM sleep awakenings in the sleep lab. However, Mrs HS only entered one convincing period of REM during the first night, so only one awakening could be performed. Other evidence that she had lost the ability to dream came from the two morning interviews performed after each night. No dreams were reported at any of these three interviews.

At a clinical interview, two months post stroke, Mrs HS reported that she had begun to dream again two weeks prior. She was unable to recall the specific details of any one dream, but she was able to characterise her dreams. In particular she noted that whenever she dreamt, she dreamt about work, and about past events. She reported that her dreams never involved current events, but rather typically involved her previous occupation.

Neuropsychological Assessment

Neuropsychological testing (Table 7) was made difficult by Mrs HS's hearing difficulties. Nevertheless, higher visuo-spatial perception was intact as indicated by her performance on LNI and the BNT, notwithstanding the difficulties posed by her hemianopia.

Constructional praxis was preserved, as demonstrated by her copy of the ROCF and performance on the WAIS-III Blocks.

Performance on the Digit span test and Corsi's blocks showed normal verbal short-term memory, with a marginally impaired visual short-term memory. However, her performance on the BVRT was good indicating that her visual short-term capacity was intact.

Mrs HS's performance on the Babcock story recall was suggestive of severe learning deficit. However, interpreting the scores on this test in this way is potentially erroneous, in that it is likely that her profound hearing difficulties impacted on her performance. Visual long-term memory was poor as evidenced by her performance on the immediate and delayed recall trials of the ROCF test. However, revisualization of the sleep laboratory, and the procedure employed there, the South African flag, a canary, and the interior layout of her own home were all good, showing revisualisation and episodic memory to be essentially intact. The following is a transcript of Mrs HS's recall of the sleep laboratory room:

> "There was just a bed. The toilet facility was outside. I know the doctor asked me, and you asked me if I had dreamt anything. There was a door this side, facing that way [indicates correct position of the door]...the bed was this side [indicates correct position of the bed]. There was a trolley or table there on the side [indicates correct position of the wheel-table]"

Therefore, neuropsychological assessment indicated possible mild visual short-term memory deficit, and a new learning deficit. In light of good performance on tests of visuo-spatial perception, verbal short-term memory, and revisualization, however, as well as overall clinical impression, it was concluded that Mrs HS had the necessary cognitive capacity for dream recall.

Table 7.

Neuropsychological Assessment: Case 3

Test	*Score*
Luria's Visual Scenes	9/14
Boston Naming Test	34/60
Rey-Osterrieth Complex Figure	
Copy	30/36
Immediate	4/36
Delayed	4/36
WAIS-III Blocks (Scaled Score)	28/68 *(9)*
Digit Span	
Forwards	6
Backwards	4
Corsi's Blocks	
Forwards	4
Backwards	4
Babcock Story Recall	
Trial A	2/21
Trial B	5/21
Trial C	2/21
Benton's Visual Retention Test	5/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Dreamers

Case 4: Mrs TJ

Date of birth: 12/09/1965
Date of CVA: 21/03/2007
Date of sleep study: 20/07/2011 and 21/07/2011
Chronicity: 224 weeks

History of Stroke

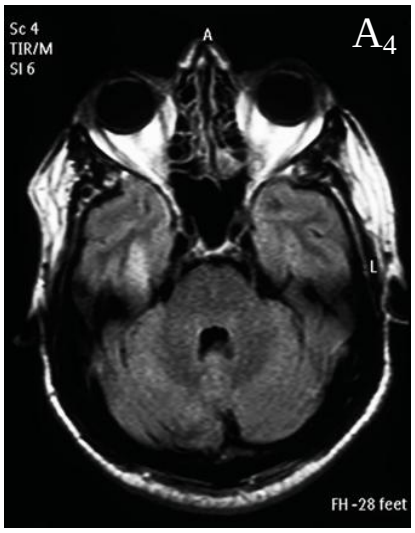
Mrs TJ, a right handed female with 13 years of formal education, presented to Gatesville Medical Centre, Cape Town, on 21 March 2007 with sudden severe headache, dizziness, vertigo, and light-headedness. She reported blurry vision in both eyes that had made reading difficult. Previous significant medical history included a possible panic attack in December 2006 where she had collapsed and lost consciousness for a few seconds as well as surgery to remove a left breast lump three years prior.

Neurological examination revealed a normal mental state. Cranial nerve examination showed a right homonymous hemianopia. Examination of the motor system showed normal power, tone, and reflexes. Sensation, coordination, gait, and fine motor movements were all normal. Subsequent neurological examination also documented a decline in memory function, and persistent difficulties with vision and balance.

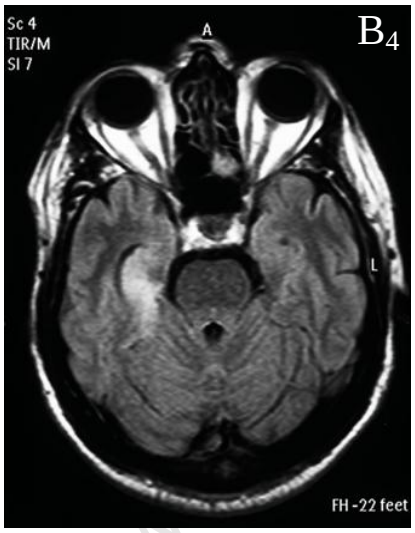
MRI-brain (Figure 12) revealed a unilateral infarct in the medial aspects of the right temporal and occipital lobe. MRA showed no evidence of intracranial tumor and no evidence of vertebral artery dissection. An aneurysm arising from the right carotid siphon lateral to the origin of the right ophthalmic artery was incidentally noted.

Neuroradiological Findings

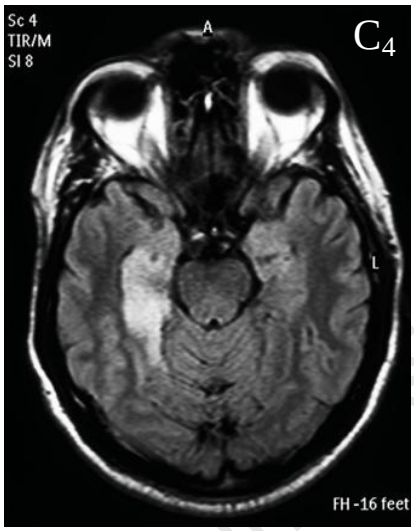
Figure 12. MRI-brain: Case 4



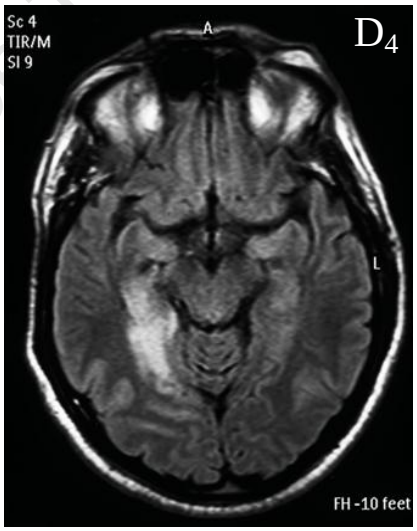
A_4



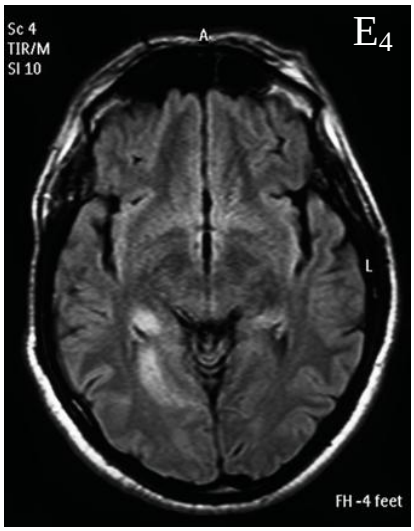
B_4



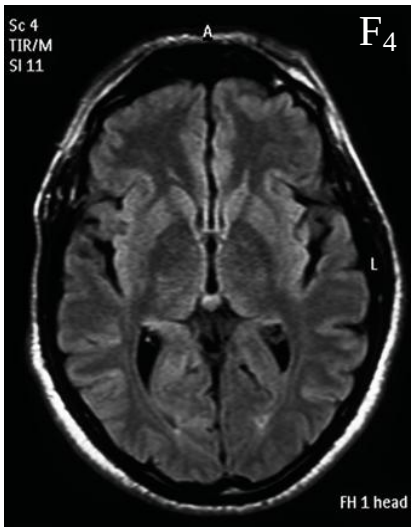
C_4



D_4

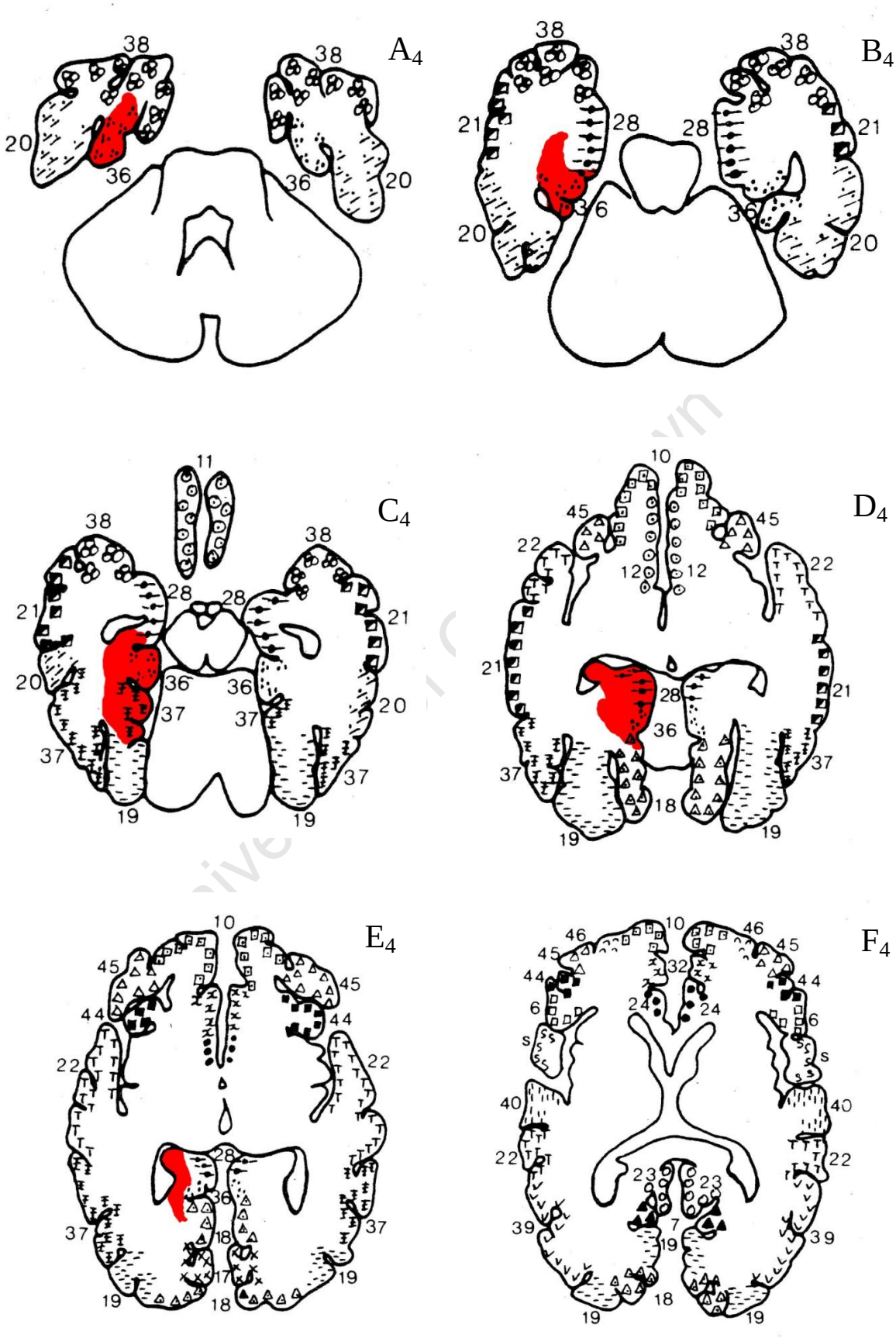


E_4



F_4

Figure 13. Brodmann templates depicting lesion sites: Case 4



The MRI-brain (Figure 12) above clearly shows a unilateral lesion in the territory of the right PCA, involving the medial occipito-temporal area. There are no lesions on the left. The thalamus is unaffected

Depiction of the lesion on the cytoarchitectonic templates (Figure 13) indicates a lesion involving BA 28, BA 36, and BA 37, in the right hemisphere.

Table 8.

Case Summary: Case 4

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mrs TJ	45	224 weeks	Right		BA 28
					BA 36
					BA 37

Dream Recall

Mrs TJ reported that she was a frequent dreamer, but that she was unsure if this had been affected by her stroke. Three nocturnal REM sleep interviews were performed during the first night in the sleep laboratory; one from a spontaneous awakening during the second REM period, as well as from waking the patient during the second and third REM periods. From these awakenings, two positive dream reports were obtained. The following is a transcript from the second awakening during the second REM period:

"Researcher: Mrs TJ? Hi, were you dreaming now?

Mrs PA: Yes, [yes]. I think so. I'm trying to remember now...The first one was...[yes] a person from work...he was talking to me about something, but now I can't remember this one. I know I was dreaming, [but] I just can't remember what it was.

Researcher: OK, that's fine. Go back to sleep."

Neuropsychological Assessment

On neuropsychological examination, Mrs TJ displayed a lower right quadrantanopia.

Neuropsychological assessment (Table 9) demonstrated that higher visuo-spatial perception was intact on LNI. However, performance was poor on the BNT, which, combined with the quadrantanopia, supported the difficulties that the patient had had with

reading. This did not, however, reflect an aphasic problem as the performance lacked language errors.

Constructional praxis was shown to be intact on the WAIS-III Blocks.

Performance on the Digit span test and Corsi's Blocks indicated mild impairment in visual and verbal short-term memory.

Intact verbal long-term memory was demonstrated by performance on the Babcock Story. However, visual short-term memory was demonstrated to be affected by poor performance on BVRT. Despite this, Mrs TJ was able to revisualize the South African flag, a canary, her own house, and the sleep laboratory in great visual-spatial detail. The following transcript indicated Mrs TJ's memory of the room in the sleep laboratory, recorded one month after the sleep study:

> "It was a tiny room. There was a window on the right, and there was glass. There was a cupboard on the left. The bed was in the centre. There was a little table".

Therefore, neuropsychological assessment revealed that Mrs TJ displayed mild difficulties with visuo-spatial perception and short-term memory. However, no severe deficits were noted. Based on the performance on testing, as well as clinical impression, it was concluded that Mrs TJ had intact functions necessary for dream recall.

Table 9.

Neuropsychological Assessment: Case 4

Test	*Score*
Luria's Visual Scenes	13/14
Boston Naming Test	31/60
Rey-Osterrieth Complex Figure	
Copy	-
Immediate	-
Delayed	-
WAIS-III Blocks (Scaled Score)	21/68 *(6)*
Digit Span	
Forwards	6
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	-
Babcock Story Recall	
Trial A	7/21
Trial B	9/21
Trial C	-
Benton's Visual Retention Test	2/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 5: Mrs CG

Date of birth: 19/06/1950
Date of CVA: 01/04/2011
Date of sleep study: 17/09/2012 and 18/09/2012
Chronicity: 76 weeks

History of Stroke

Mrs CG, a right-handed female with ten years of formal education, presented to Tygerberg Hospital on 14 February 2011, with a one month history of chronic headaches, not associated with nausea or vomiting, but associated with visual disturbances. These visual disturbances were in the form of light flashes in the right upper quadrant of her visual fields. Two weeks prior to admission, she developed acute onset confusion that improved within a few hours. This was followed by a loss of vision to the right.

On neurological examination she appeared in good general health. Assessment of her higher functions was normal, with no signs of meningeal irritation. All of her cranial nerves were assessed to be normal with exception of cranial nerve II, which showed a right-sided homonymous hemianopia.

Motor systems, sensory systems, and fine-motor skills were all assessed to be normal.

MRI-brain (Figure 14) performed on the day of admission showed a subacute infarction of the left medial occipital lobe unilaterally, extending to the tip of the visual cortex in the left posterior cerebral artery distribution. The thalamus was unaffected.

Neuroradiological Findings

Figure 14. MRI-brain: Case 5

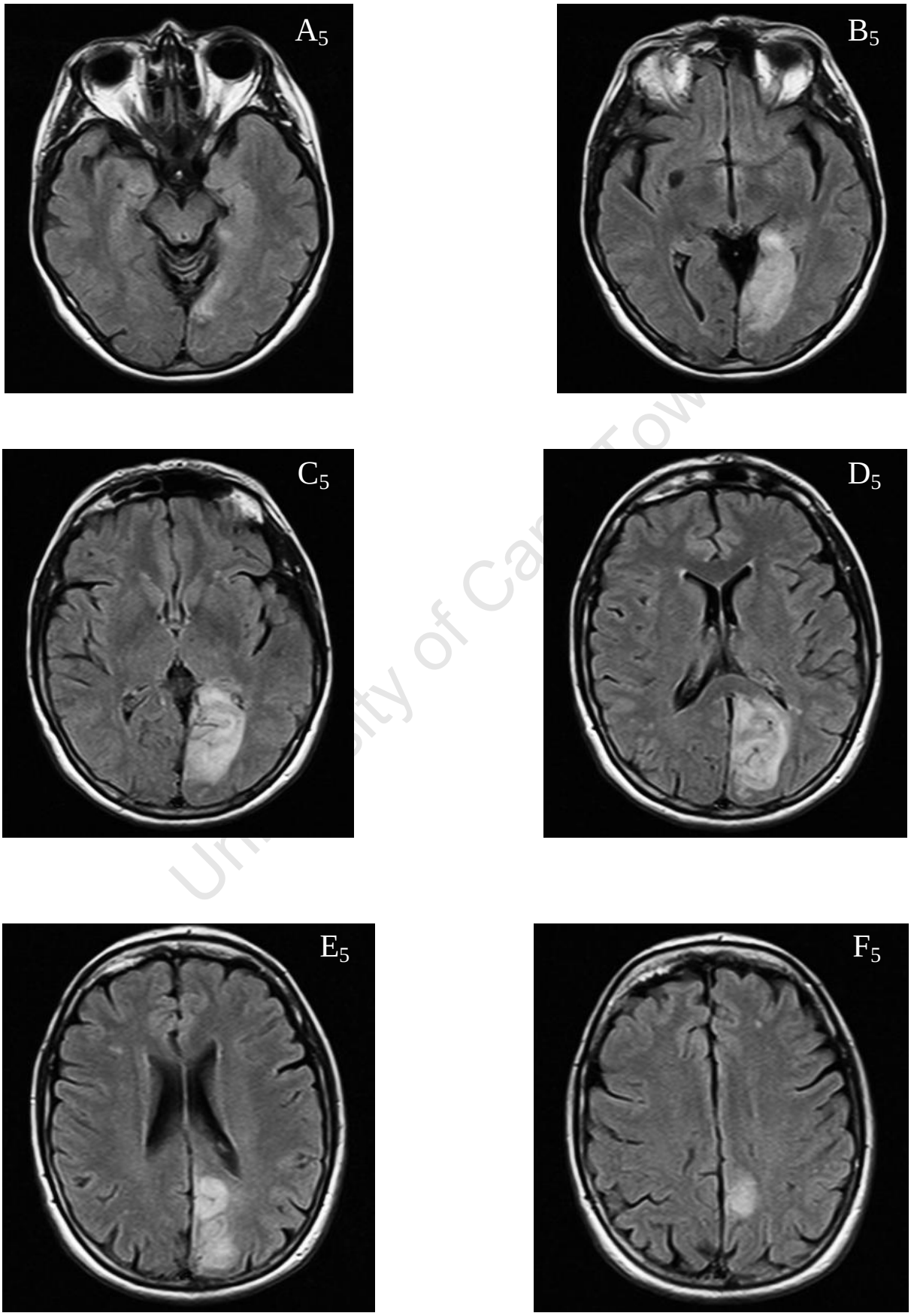
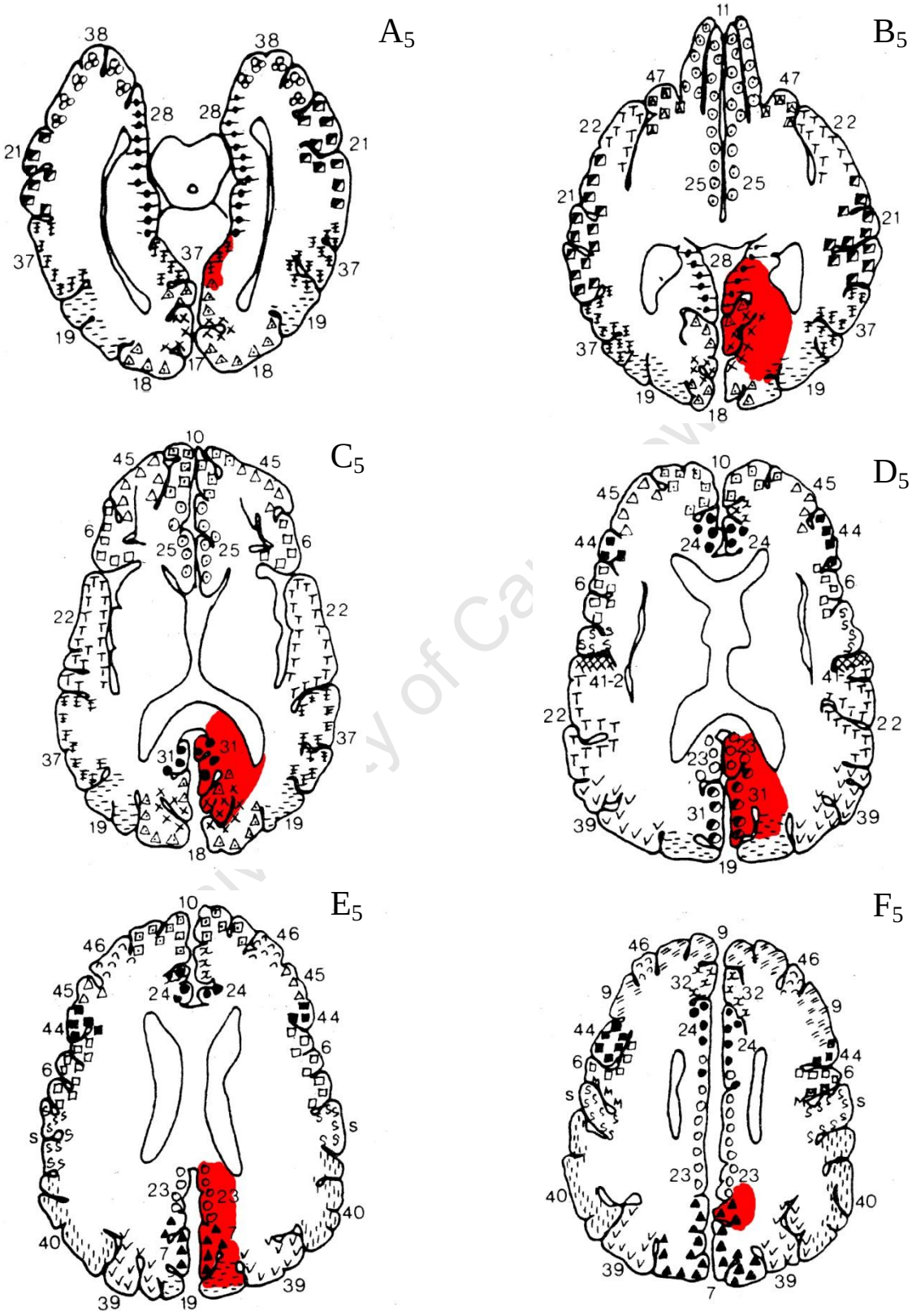


Figure 15. Brodmann templates depicting lesion sites: Case 5



The MRI-brain (Figure 14) above clearly shows a large unilateral lesion in the territory of the left PCA, involving the medial occipital lobe. There are no lesions on the right. The thalamus is unaffected.

Depiction of the lesion on the cytoarchitectonic templates (Figure 15) indicates a lesion involving BA 7, BA 17, BA 18, BA 19, BA 23, BA 28, BA 36, and BA 37, all in the left hemisphere.

Table 10.

Case Summary: Case 5

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mrs CG	60	76 weeks	Left	BA 7	
				BA 17	
				BA 18	
				BA 19	
				BA 23	
				BA 28	
				BA 36	
				BA 37	

Dream Recall

At initial interviewing Mrs CG reported that she was not sure as to the effect her stroke had on her ability to dream, if any. Two separate REM sleep awakenings were performed during the second and third REM periods. At both awakenings, a positive dream report was obtained.

On clinical interviewing, one month after the sleep lab assessment, Mrs CG reported that she had been dreaming, but thought that these were not as frequent as before.

Neuropsychological Assessment

Neuropsychological assessment (Table 11) demonstrated that higher visuo-spatial perception was intact on LNI.

Constructional praxis was shown to be intact, with an excellent copy of the ROCF, and good performance on the WAIS-III Blocks.

Performance on the Digit span test and Corsi's Blocks indicated mild impairment in visual and verbal short-term memory.

Intact verbal long-term memory was demonstrated by performance on the Babcock Story. Her performances on the recall trials of the ROCF, and the BVRT were good, indicating that visual long-term memory was intact. Additionally, Mrs CG was able to revisualize the South African flag, a canary, her own house, and the sleep laboratory in great visual-spatial detail. The following transcript indicated Mrs CG's memory of her experience of the sleep laboratory, recorded one month after the sleep study:

> "I was taken to a room, and given a meal. Then I went to the toilet. I got into my pyjamas, and had a cup of tea. I sat on the bed and waited for the nurse to put all these things on my head. Then I fell asleep. I was woken up in the night and asked if I was dreaming. I can still remember the dream. I was having an argument with my mother. Then I fell asleep again. Then I had to go to the toilet in the night and they had to disconnect all the wires, and I had to go with it in my hand".

Therefore, neuropsychological assessment revealed that Mrs CG displayed mild difficulties with short-term memory. No severe deficits were noted. Based on her performance on testing, as well as clinical impression, it was concluded that Mrs CG had intact functions necessary for dream recall.

Table 11.

Neuropsychological Assessment: Case 5

Test	*Score*
Luria's Visual Scenes	14/14
Boston Naming Test	58/60
Rey-Osterrieth Complex Figure	
Copy	34/36
Immediate	16/36
Delayed	14/36
WAIS-III Blocks (Scaled Score)	37/68 *(11)*
Digit Span	
Forwards	6
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	4
Babcock Story Recall	
Trial A	11/21
Trial B	15/21
Trial C	13/21
Benton's Visual Retention Test	6/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 6: Mr SR

Date of birth: 09/05/1960
Date of CVA: 23/12/2010
Date of sleep study: 27/06/2011 and 28/06/2011
Chronicity: 29 weeks

History of Stroke

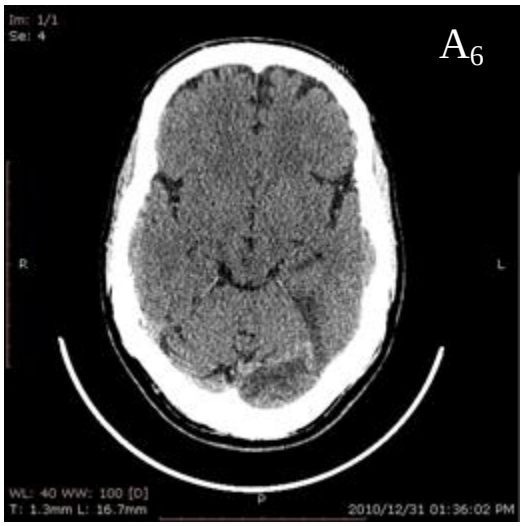
Mr SR, a right handed male with six years of formal education, presented to Kuils River Hospital, Cape Town, on 23 December 2010 with severe headache in the left frontal area and complaints of forgetfulness. He had an extensive medical history of rheumatic fever, mitral stenosis and hypertension. In addition, he was on prophylactic warfarin treatment due to atrial fibrillation.

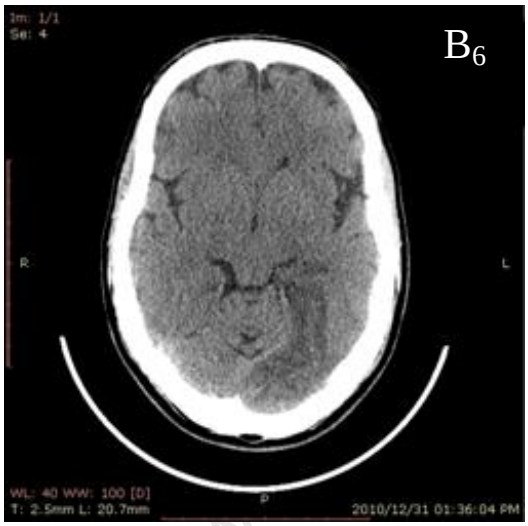
CT-brain (Figure 16), taken eight days after initial presentation, revealed a left PCA territory infarction, most likely of subacute duration. The infarction involved the occipital lobe on the left-hand side towards its inferior portion, with no evidence of the temporal lobes being affected. Mild localized brain swelling was noted, while there was no significant mass effect and no evidence of haemorrhagic transformation. No significant calcifications of the terminal portions of the vertebral arteries or of the basilar artery were seen.

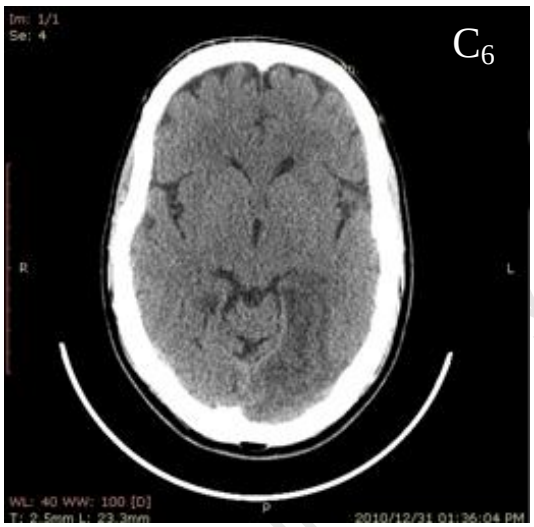
Subsequent neurological examination at Gatesville Medical Centre, Cape Town, revealed persistent headache, blurred vision and a poor cognitive state characterized by forgetfulness and personality changes.

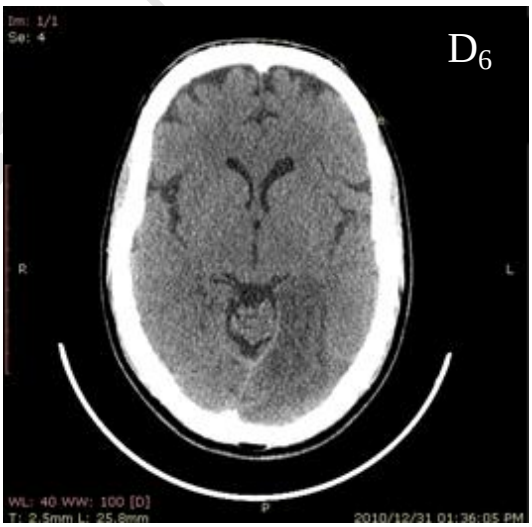
Neuroradiological Findings

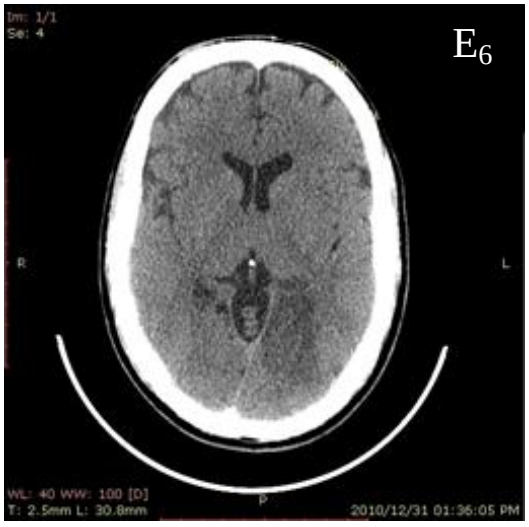
Figure 16. CT-brain: Case 6











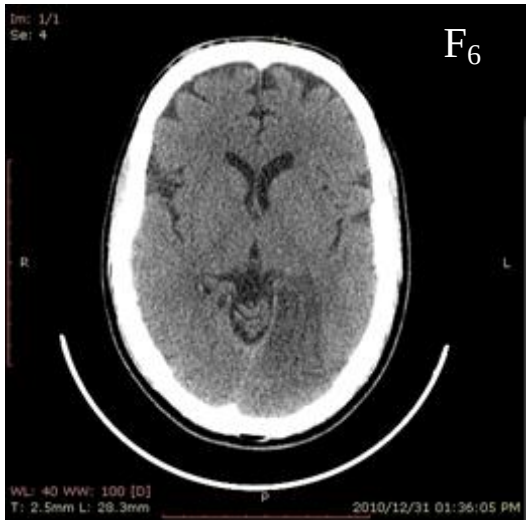
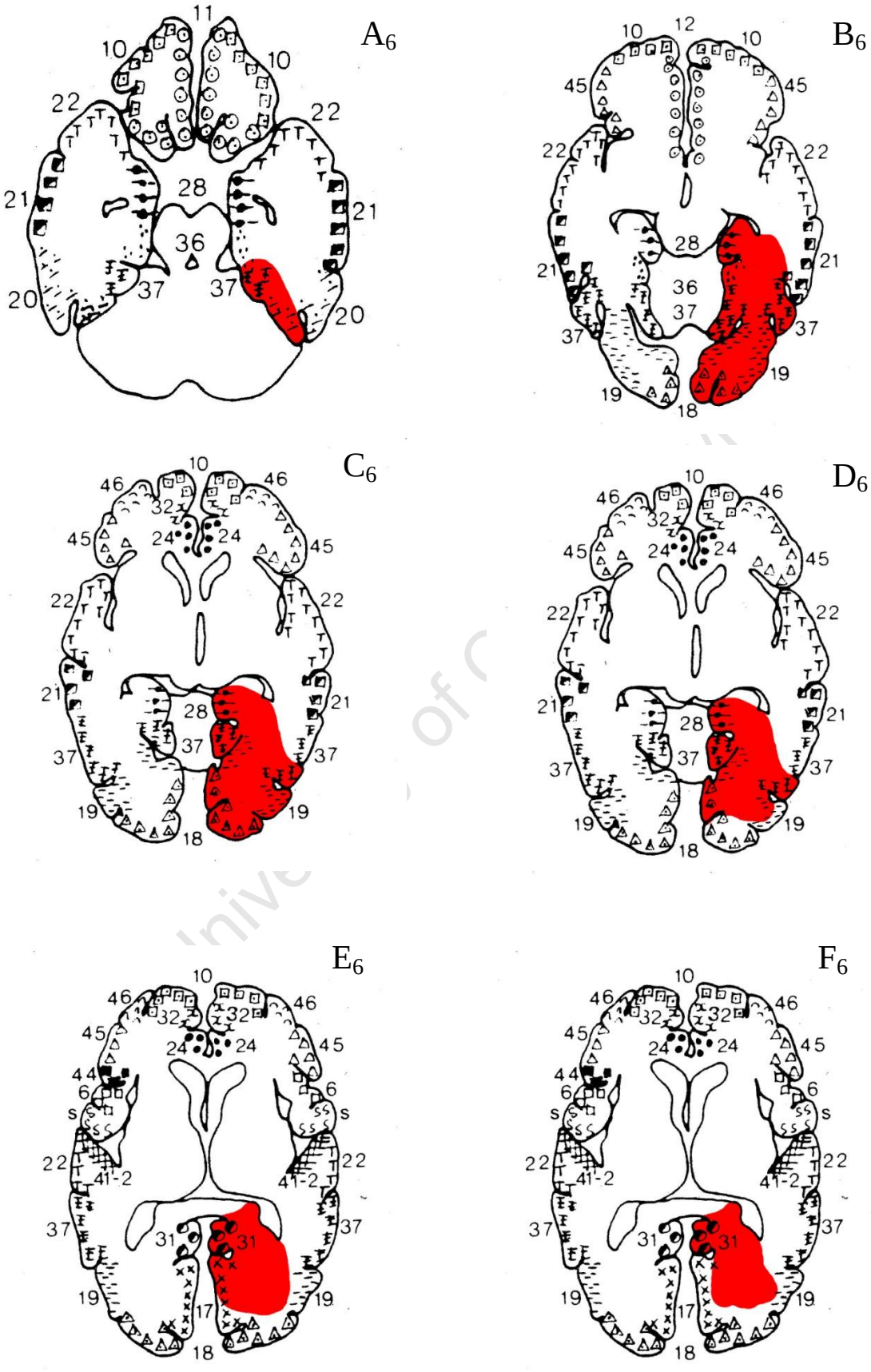


Figure 17. Brodmann templates depicting lesion sites: Case 6



The CT-brain (Figure 16) above clearly shows a large unilateral lesion in the territory of the left PCA, involving the medial occipital area. There are no lesions on the right. The thalamus is unaffected.

Depiction of the lesion on the cytoarchitectonic templates (Figure 17) indicates a lesion involving BA 17, BA 18, BA 19, BA 20, BA 28, BA 31, BA 36, and BA 37, all in the left hemisphere.

Table 12.

Case Summary: Case 6

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mr SR	51	29 weeks	Left	BA 17	
				BA 18	
				BA 19	
				BA 20	
				BA 28	
				BA 31	
				BA 36	
				BA 37	

Dream Recall

Mr SR reported that he dreamt regularly before his stroke. He was uncertain as to the effect his stroke may have had on his dreams. Nocturnal REM sleep interviews were performed during the first night in the sleep laboratory during the second and third REM periods. A positive dream report was elicited from the second awakening. In addition, Mr SR reported in the morning that he could remember dreaming a few times during the night.

Neuropsychological Assessment

Neuropsychological examination (Table 13) indicated that higher visuo-spatial perception was intact, as indicated by his performance on LNI. Performance on the BNT was adequate, and did not display any naming deficits.

Performance on the ROCF and WAIS-III Blocks indicated that constructional praxis was intact.

Verbal and visual short-term memory were within normal range on the Digit span test and Corsi’s blocks.

Testing of long-term memory showed that Mr SR did not display any major verbal or visual memory deficits, while immediate recall of the ROCF was poor. However, revisualization of the South African flag, a canary, the patient's own house, and the sleep laboratory was good. In particular, Mr SR was able to accurately recall the room in the sleep laboratory two months after the sleep study, including details of the layout of the room and of the polysomnograph leads.

Therefore, neuropsychological assessment did not reveal any major cognitive deficits. Based on overall performance on testing as well as clinical impression, it was concluded that Mr SR had intact functions necessary for dream recall.

Table 13.

Neuropsychological Assessment: Case 6

Test	*Score*
Luria's Visual Scenes	11/14
Boston Naming Test	33/60
Rey-Osterrieth Complex Figure	
Copy	27/36
Immediate	6/36
Delayed	-
WAIS-III Blocks (Scaled Score)	16/68 *(5)*
Digit Span	
Forwards	5
Backwards	3
Corsi's Blocks	
Forwards	5
Backwards	4
Babcock Story Recall	
Trial A	4/21
Trial B	7/21
Trial C	-
Benton's Visual Retention Test	3/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 7: Mr BD

Date of birth: 30/08/1943
Date of CVA: 11/02/2010
Date of sleep study: 18/04/2011 and 19/04/2011
Chronicity: 9 weeks

History of Stroke

Mr BD, a right handed male with 11 years of formal education, presented to Gatesville Medical Centre, Cape Town, on 11 February 2010 with an acute history of imbalance, slurred speech, severe vertigo, following a possible loss of consciousness. Mr BD had an extensive medical history of hypertension, diabetes, and ischaemic heart disease for which he had had coronary artery bypass surgery.

Subsequent neurological examination also reported topographical disorientation and forgetfulness while driving.

MRI-brain (Figure 18), taken 5 days after initial presentation, revealed an abrupt occlusion of the right PCA at the junctions of the P1 and P2 segments. T2, FLAIR, and DWI showed hyperintense signal change in the right uncus, parahippocampal gyrus, and hippocampus. No significant brain swelling was noted, and no evidence of haemorrhage was found.

Neuroradiological Findings

Figure 18. MRI-brain: Case 7

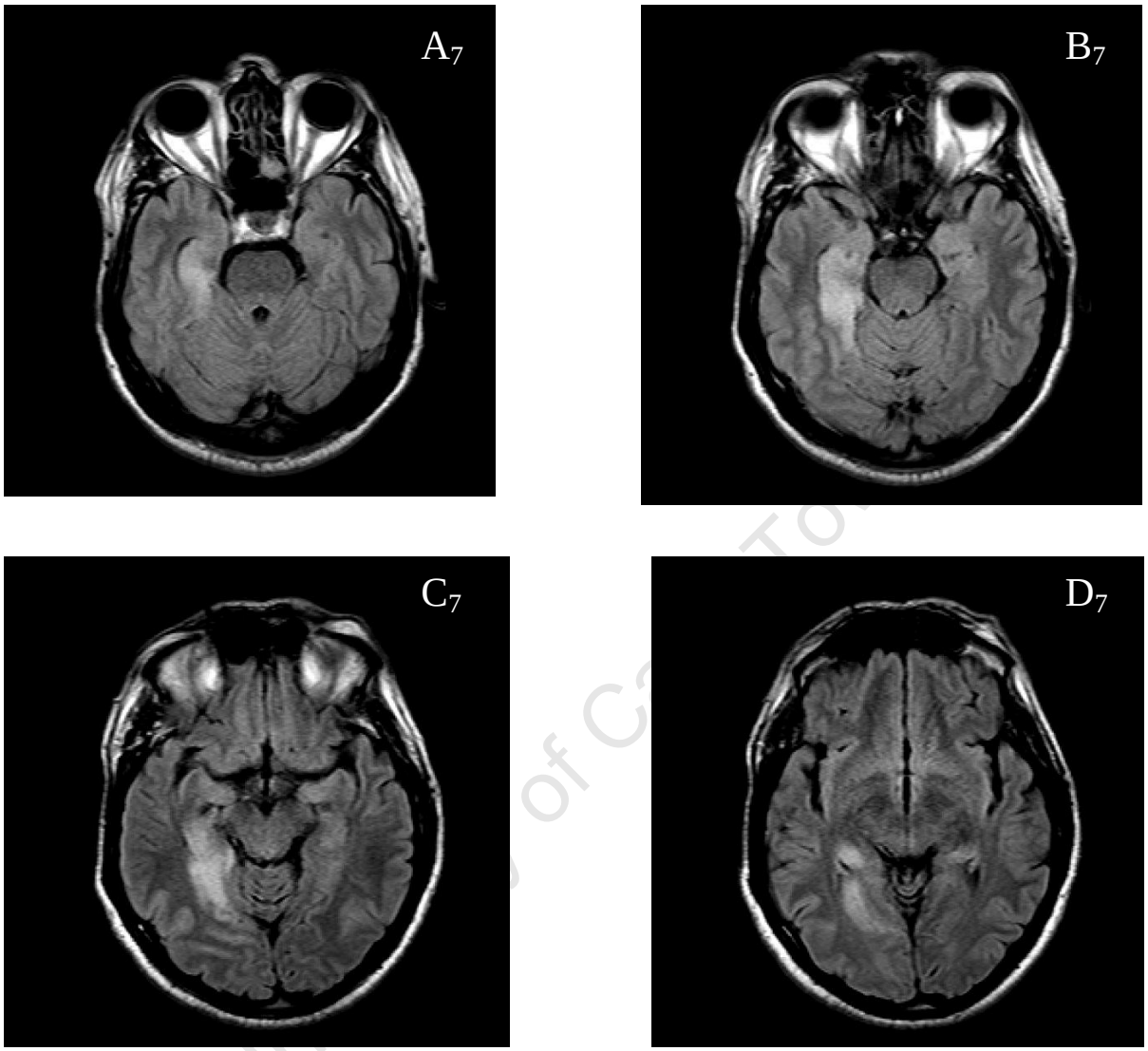
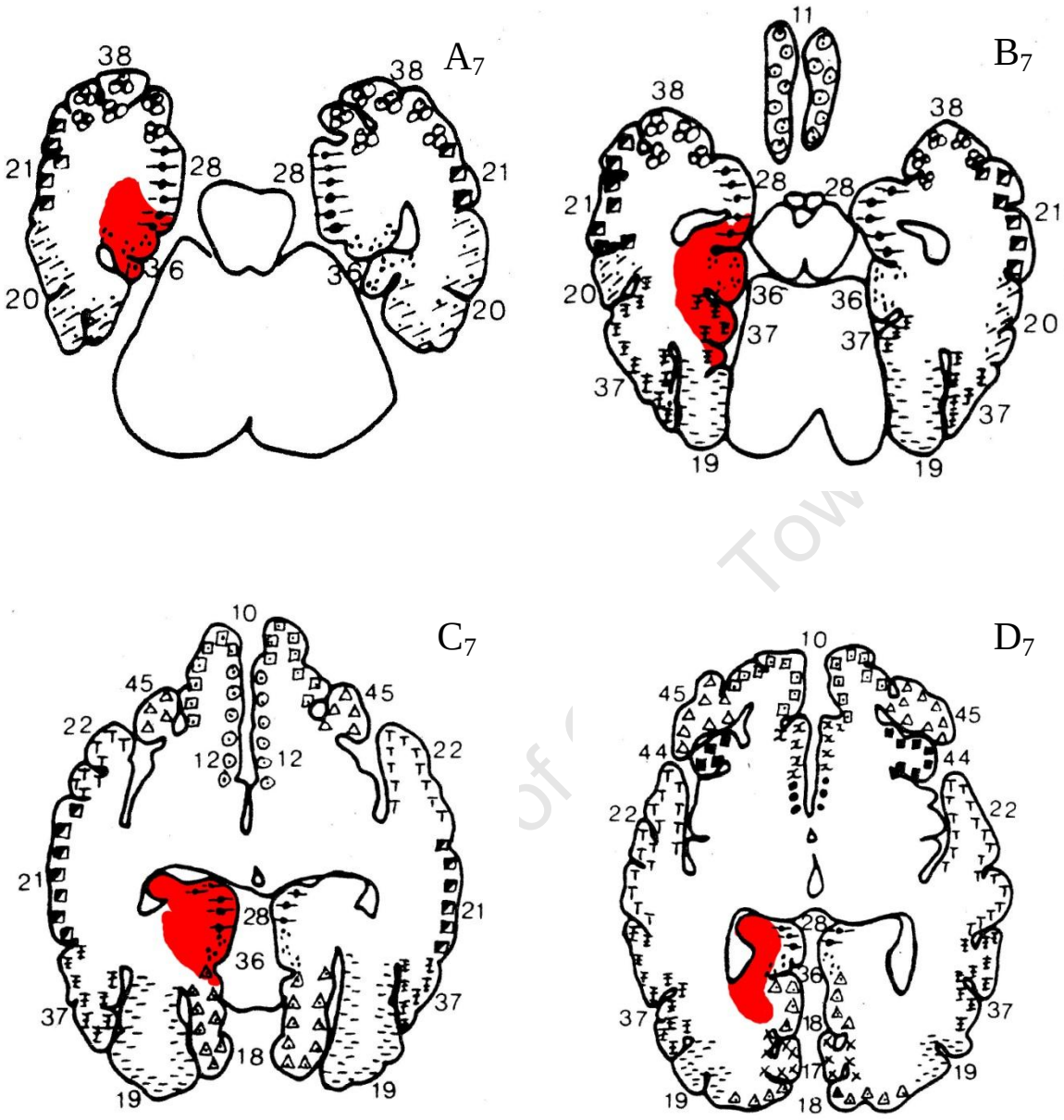


Figure 19. Brodmann templates depicting lesion sites: Case 7



The MRI-brain (Figure 18) above clearly shows a unilateral lesion in the territory of the right PCA, involving the medial occipito-temporal area. There are no lesions on the left. The thalamus is unaffected.

Depiction of the lesion on the cytoarchitectonic templates (Figure 19) indicates a lesion involving BA 28, BA 36, and BA 37, in the right hemisphere.

Table 14.

Case Summary: Case 7

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mr BD	67	9 weeks	Right		BA 28
					BA 36
					BA 37

Dream Recall

Mr BD reported that he had slept well since his stroke and did not feel that his sleep had been affected in any way. He reported that he dreamt regularly, and that this had not been affected by his stroke. He was able to recall a particularly vivid dream that had occurred one month prior to clinical interviewing and was able to recall descriptive details of characters in the dream. He was also able to recall a dream he had experienced the night prior to clinical interviewing, as indicated by the following transcript:

> "Last night, I dreamt that intruders tried to enter our home. I was helpless to do anything about it. Finally, they couldn't enter. I saw the intruder climbing over the wall, and entering the yard".

During the first night in the sleep laboratory, nocturnal REM sleep interviews were conducted by waking Mr BD during the second and third REM periods. Both awakenings elicited positive dream reports. In addition, Mr BD was able to recall one of those dreams in the morning, as well as an additional dream that he had during the night.

Neuropsychological Assessment

Neuropsychological examination (Table 15) indicated that higher visuo-spatial perception on LNI was intact, except for some face recognition difficulties. Place recognition difficulties

were also noted, but these were explained by the fact that the patient had topographical amnesia.

Constructional praxis was preserved, demonstrated by a very good copy of the ROCF.

Verbal and visual short-term memory were intact.

Performance on the Babcock Story indicated that verbal long-term memory was preserved. Poor immediate recall on the ROCF indicated a mild visual long-term memory deficit. However, Mr BD was able to revisualize the South African flag and the plan of his house in visual-spatial detail.

Therefore, neuropsychological assessment revealed no major cognitive deficits, except for a potential mild visual long-term memory deficit. Based on the overall performance on testing as well as clinical impression, it was concluded that Mr BD had intact functions necessary for dream recall.

Table 15.

Neuropsychological Assessment: Case 7

Test	*Score*
Luria's Visual Scenes	-
Boston Naming Test	-
Rey-Osterrieth Complex Figure	
Copy	32/36
Immediate	9/36
Delayed	-
WAIS-III Blocks	-
Digit Span	
Forwards	5
Backwards	-
Corsi's Blocks	
Forwards	-
Backwards	-
Babcock Story Recall	
Trial A	8/21
Trial B	12/21
Trial C	-
Benton's Visual Retention Test	-
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 8: Mr JS

Date of birth: 05/08/1956
Date of CVA: 25/07/2012
Date of sleep study: 30/07/2012 and 31/07/2012
Chronicity: 1 week

History of Stroke

Mr JS, a right-handed male with three years of formal education, presented to Gatesville Medical Centre, Cape Town, on 25 July 2012, having suddenly collapsed. Additionally he complained of sudden onset nausea and vomiting, together with an occipital headache. He was markedly drowsy. There was no significant past medical, surgical, or allergy history apart from old surgery to the right knee.

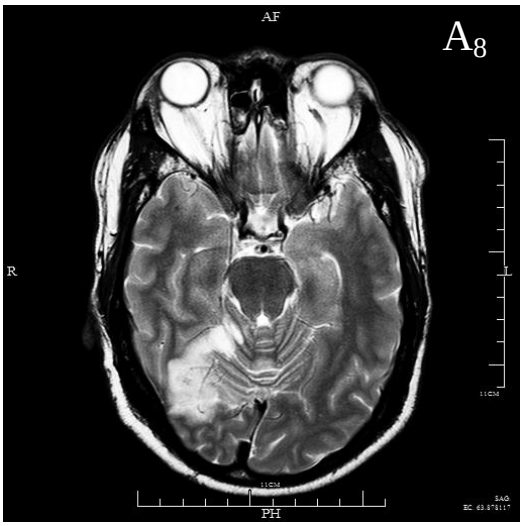
On examination he was alert, awake, and oriented. His mental state was assessed to be normal, except for his marked drowsiness. Examination of the cranial nerves, including the fundi and visual fields revealed a left homonymous hemianopia.

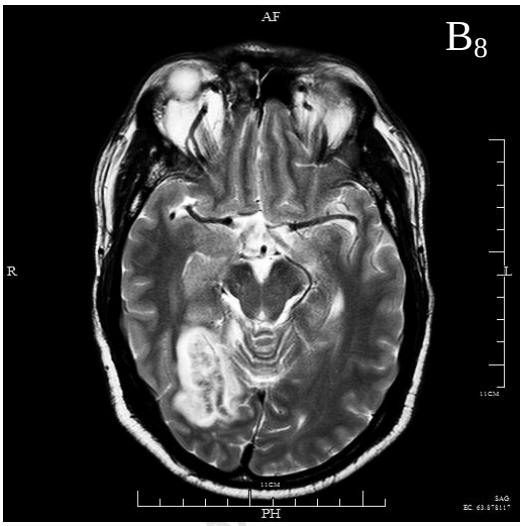
An assessment of the motor system, sensation, and coordination revealed that they were all normal. However, his gait was ataxic. There was no other evidence of cerebellar dysfunction, and fine motor movements were all normal.

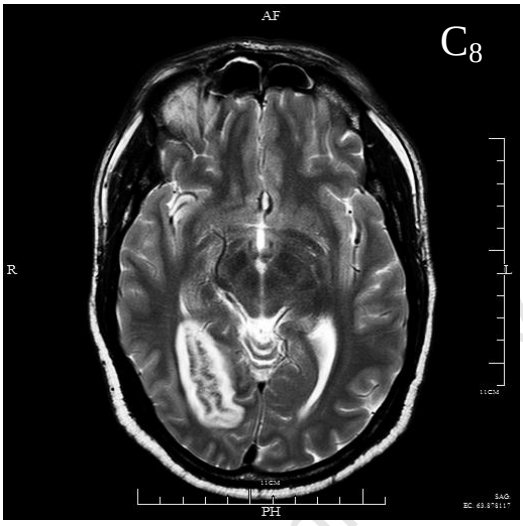
An MRI-brain (Figure 20) performed the day after admission showed a subacute right superior cerebellar and medial occipito-temporal with suspected cortical micro haemorrhage and surrounding vasogenic oedema. CT-angiography suggested focal dissection of the left vertebral artery superior to the arch of C1.

Neuroradiological Findings

Figure 20. MRI-brain: Case 8







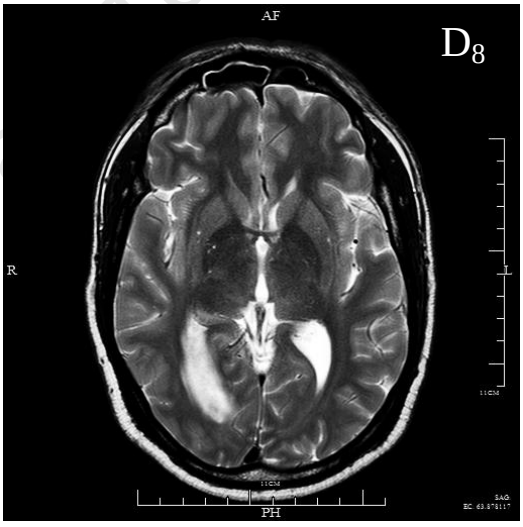
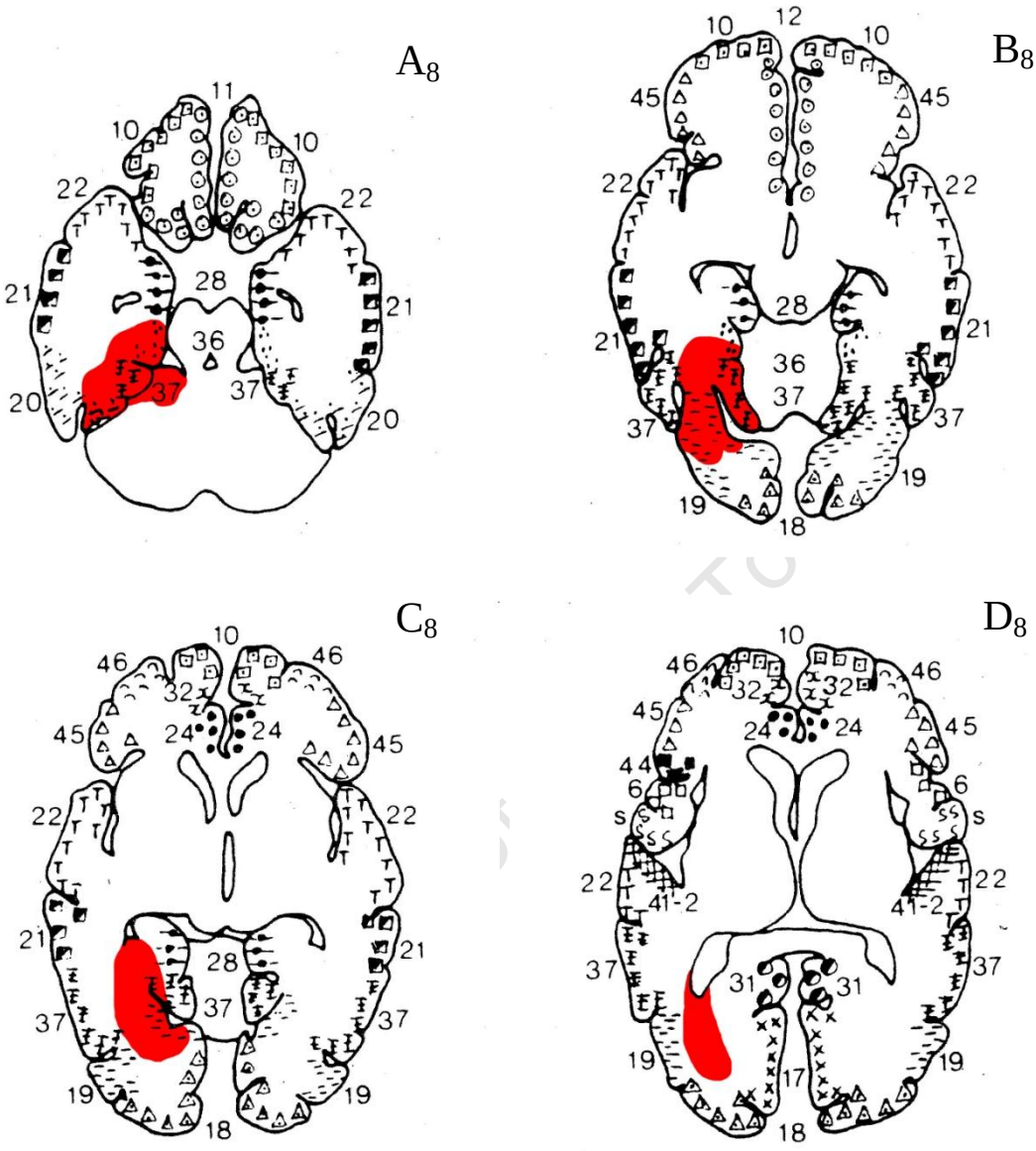


Figure 21. Brodmann templates depicting lesion sites: Case 8



The MRI-brain (Figure 20) above clearly shows a unilateral lesion in the medial aspects of the right occipito-temporal area. There is also a lesion in the right cerebellum. There are no lesions on the left. The thalamus is unaffected.

Depiction of the lesion on the cytoarchitectonic templates (Figure 21) indicates a lesion involving BA 19, BA 36, and BA 37, in the right hemisphere.

Table 16.

Case Summary: Case 8

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mr JS	56	1 week	Right		BA 19
					BA 36
					BA 37

Dream Recall

Mr JS reported that he did not know if his stroke had affected his ability to dream. This was determined over two awakenings in the sleep lab during the second and third periods of REM sleep. One positive dream report was obtained from these awakenings. At the morning interview, although he did not report any other dreams, he was able to further clarify the dream that he had reported at the second awakening.

At a clinical interview, one month after the sleep lab assessment, he reported that he had been dreaming. He could not give specific details on any one dream at that time.

Neuropsychological Assessment

Neuropsychological examination (Table 17) indicated that higher visuo-spatial perception was intact as indicated by his performance on LNI. His performance on the BNT may have indicated a language deficit. However, Mr JS had only achieved a grade 3 level of education. Thus incorrect, or non-responses on the BNT were most likely owing to his poor education. Indeed, much of his neuropsychological performance may have been affected by his poor level of education.

Constructional praxis was preserved, demonstrated by a good copy of the ROCF.

A moderate impairment in verbal and visual short-term memory was indicated by his performance on the Digit span test, and Corsi's blocks.

Performance on the Babcock Story indicated that verbal long-term memory was poor. His performance on the BVRT was suggestive of a deficit in visual short-term memory.

However, competent immediate and delayed recall of the ROCF indicated an intact visual long-term memory. Additionally, Mr JS was able to adequately revisualize the South African flag, a canary, and the plan of his house.

Therefore, neuropsychological assessment revealed potential deficits in verbal and visual short-term memory, naming, and verbal long-term memory. However, it is likely that his poor level of education impacted on his performance on these respective tasks. Thus based on his performance overall, and based on clinical impression, and the fact that he reported a dream on awakenings, and at the clinical interview, it was concluded that Mr JS possessed the necessary neuropsychological functions for dream recall.

Table 17.

Neuropsychological Assessment: Case 8

Test	*Score*
Luria's Visual Scenes	13/14
Boston Naming Test	20/60
Rey-Osterrieth Complex Figure	
Copy	29/36
Immediate	14/36
Delayed	15/36
WAIS-III Blocks (Scaled Score)	13/68 *(5)*
Digit Span	
Forwards	4
Backwards	4
Corsi's Blocks	
Forwards	6
Backwards	4
Babcock Story Recall	
Trial A	4/21
Trial B	5/21
Trial C	3/21
Benton's Visual Retention Test	0/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

Case 9: Mr RS

Date of birth: 04/02/1958
Date of CVA: 17/03/2012
Date of sleep study: 26/03/2012 and 27/03/2012
Chronicity: 1 week

History of Stroke

Mr RS, a right-handed male with 12 years of formal education, presented to Gatesville Medical Centre, Cape Town, on 17 March 2012, with sudden onset of weakness, and sensory dyesthesiae involving the right face, arm, and leg.

On neurological examination his mental state was assessed to be normal and he was not aphasic. Assessment of the cranial nerves, including the fundi and visual fields revealed a right upper motor neuron 7th nerve lesion as well as right homonymous hemianopia.

Power was reduced on the right hand side of the body, and was worse proximally in both the arm and the leg. Pinprick and joint position sense was reduced over the right side of the body. Coordination was abnormal on the right, and he required help to stand or walk. Gait was assessed to be normal, while fine motor movements were clearly reduced on the right.

An MRI-brain (Figure 22) performed five days after admission showed multifocal sub acute infarcts involving the left thalamus, extending into the posterior limb of the left internal capsule, left medial temporal lobe, left medial occipital lobe, and left posterior parietal lobe. MR angiogram performed five days after admission revealed an occlusion of the proximal left posterior cerebral artery.

Neuroradiological Findings

Figure 22. MRI-brain: Case 9

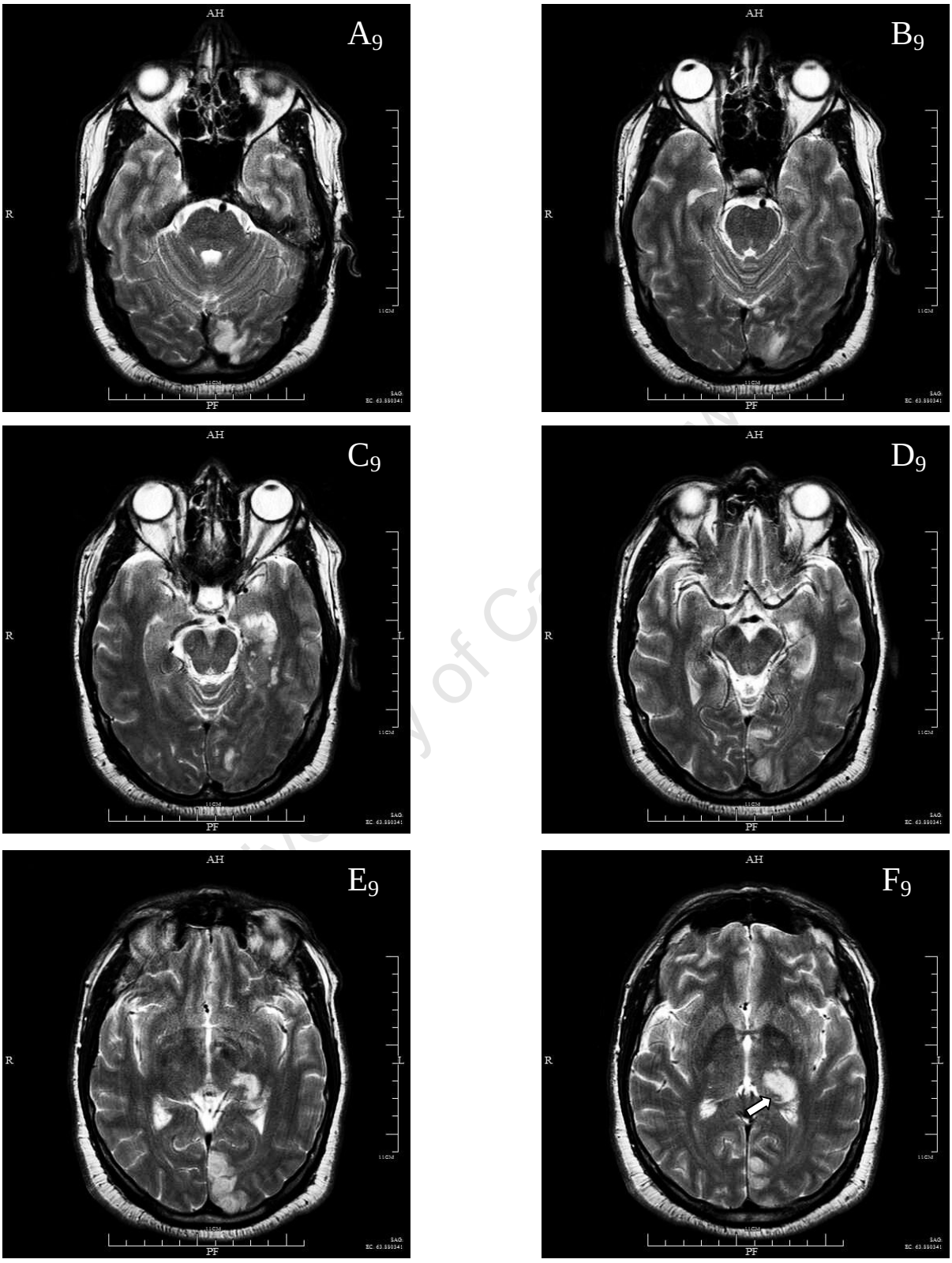
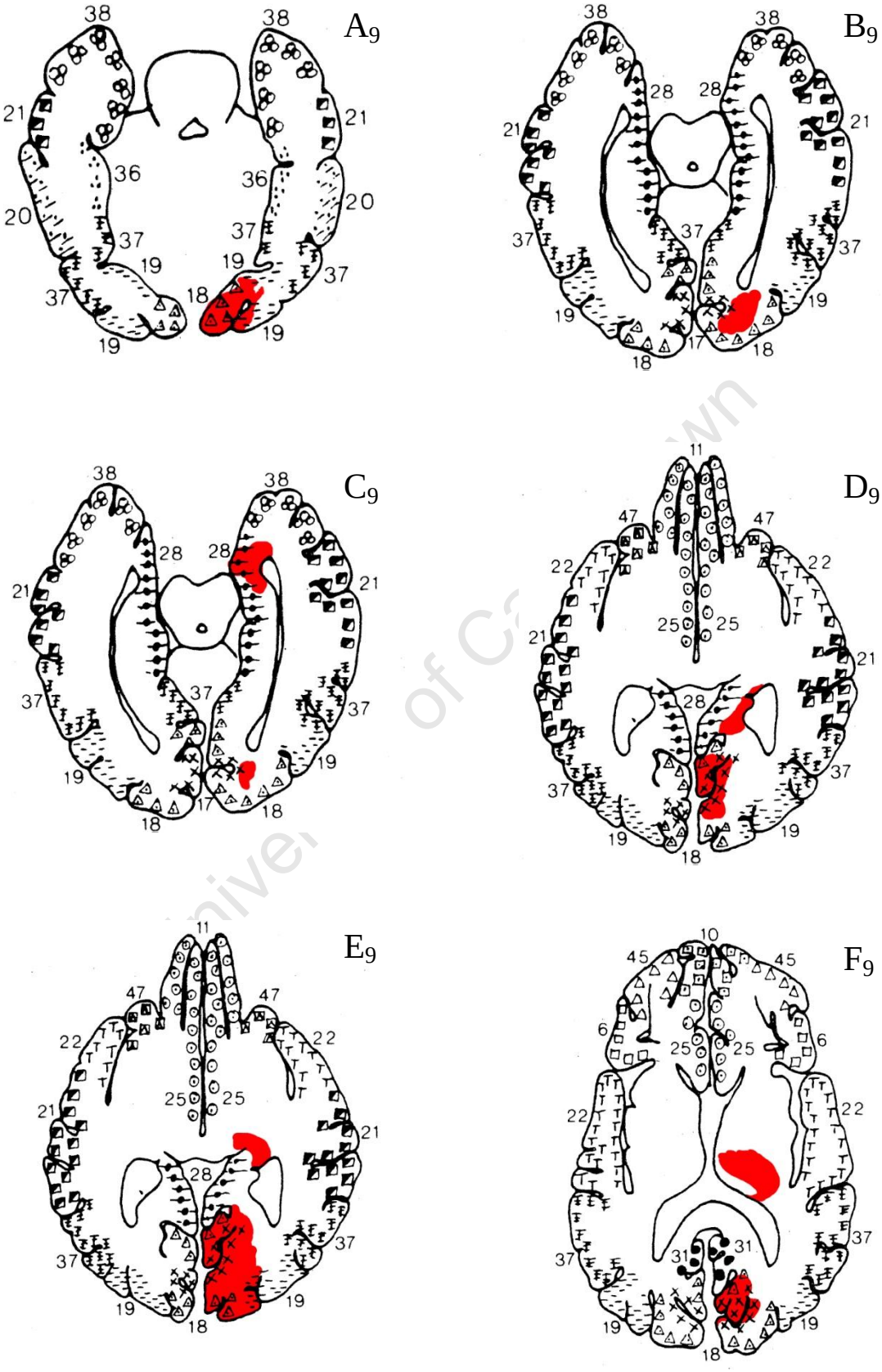


Figure 23. Brodmann templates depicting lesion sites: Case 9



The MRI-brain (Figure 22) above clearly shows multiple lesions in the left posterior cerebral artery territory. Notably, there are lesions in the left medial occipito-temporal areas, and in the left thalamus (white arrow – Figure 22, F_9). There are no lesions on the right.

Depiction of the lesion on the cytoarchitectonic templates (Figure 23) indicates a lesion involving BA 17, BA 18, BA 19, BA 28, BA 37, and the thalamus, all in the left hemisphere.

Table 18.

Case Summary: Case 9

Name	*Age*	*Chronicity*	*Laterality*	*Brodmann's Area of Lesion*	
				Hemisphere	
				Left	Right
Mr RS	54	1 week	Left	BA 17	
				BA 18	
				BA 19	
				BA 36	
				BA 37	
				Thalamus	

Dream Recall

Mr RS reported that he was unsure as to the effect his stroke may have had on his ability to dream. Three REM sleep awakenings were performed during the second, third, and fourth REM periods. At all three awakenings, positive dream reports were obtained. At morning interviewing, Mr RS was able to remember the dreams that he reported during the night.

At a clinical interview two months after the sleep lab assessment, Mr RS reported that he had been dreaming. However, he was unable to give any details regarding a specific dream.

Neuropsychological Assessment

Neuropsychological examination (Table 19) indicated that higher visuo-spatial perception was intact as indicated by his performance on LNI.

Constructional praxis was preserved, demonstrated by a good copy of the ROCF.

His performance on the Digit span test, and Corsi's blocks was within normal limits.

Performance on the Babcock Story indicated that verbal long-term memory was intact. His performance on the recall trials of ROCF indicated an intact visual long-term

memory. Additionally, Mr RS was able to revisualize the South African flag, a canary, the plan of his house, and the sleep lab in detail.

Therefore, no outstanding deficits were noted on neuropsychological assessment. Based on his performances at clinical interviewing, sleep lab assessment, and overall clinical impression, Mr RS was judged to have the necessary cognitive capacity for dream recall.

Table 19.

Neuropsychological Assessment: Case 9

Test	*Score*
Luria's Visual Scenes	14/14
Boston Naming Test	51.5/60
Rey-Osterrieth Complex Figure	
Copy	33/36
Immediate	13/36
Delayed	10/36
WAIS-III Blocks (Scaled Score)	20/68 *(6)*
Digit Span	
Forwards	5
Backwards	6
Corsi's Blocks	
Forwards	6
Backwards	5
Babcock Story Recall	
Trial A	12/21
Trial B	15/21
Trial C	13/21
Benton's Visual Retention Test	5/7
Revisualization	
South African Flag	normal
Canary	normal
Sleep Laboratory	normal

CHAPTER 5: BETWEEN-GROUPS COMPARISON

Neuroradiological Findings

Importantly, all of the cases with cessation of dreaming suffered bilateral damage, as opposed to the dreamers, who all suffered unilateral damage. This more obvious difference between the lesion sites of the two groups is observed from a comparison of the individual scans and templates (Figure 5-23).

Figures 24 and 25 show the coronal view of the most common lesion sites (darker areas) in both groups. A comparison of the these figures shows that *the lesions in the non-dreaming group were more posterior*, involving BA 18, BA 19, and to a less extent BA 37, than *lesions in the dreaming group, which were more anterior*, involving BA 28, BA 36, and BA 37. Furthermore, *the thalamus appears to have been a more common lesion site in the non-dreamers* (2/3 cases) than in the dreamers (1/6 cases).

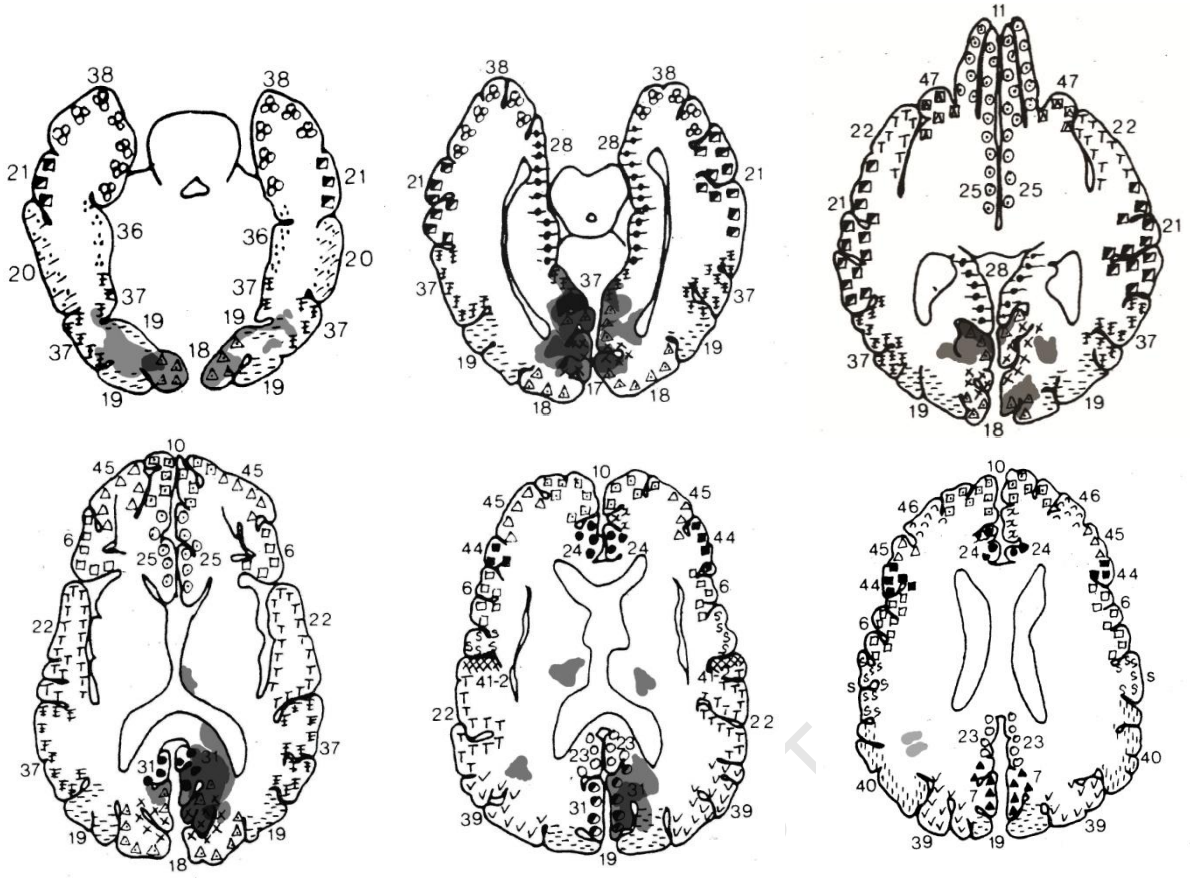


Figure 24. Combined template of the three non-dreamers (coronal view). All lesions were bilateral.

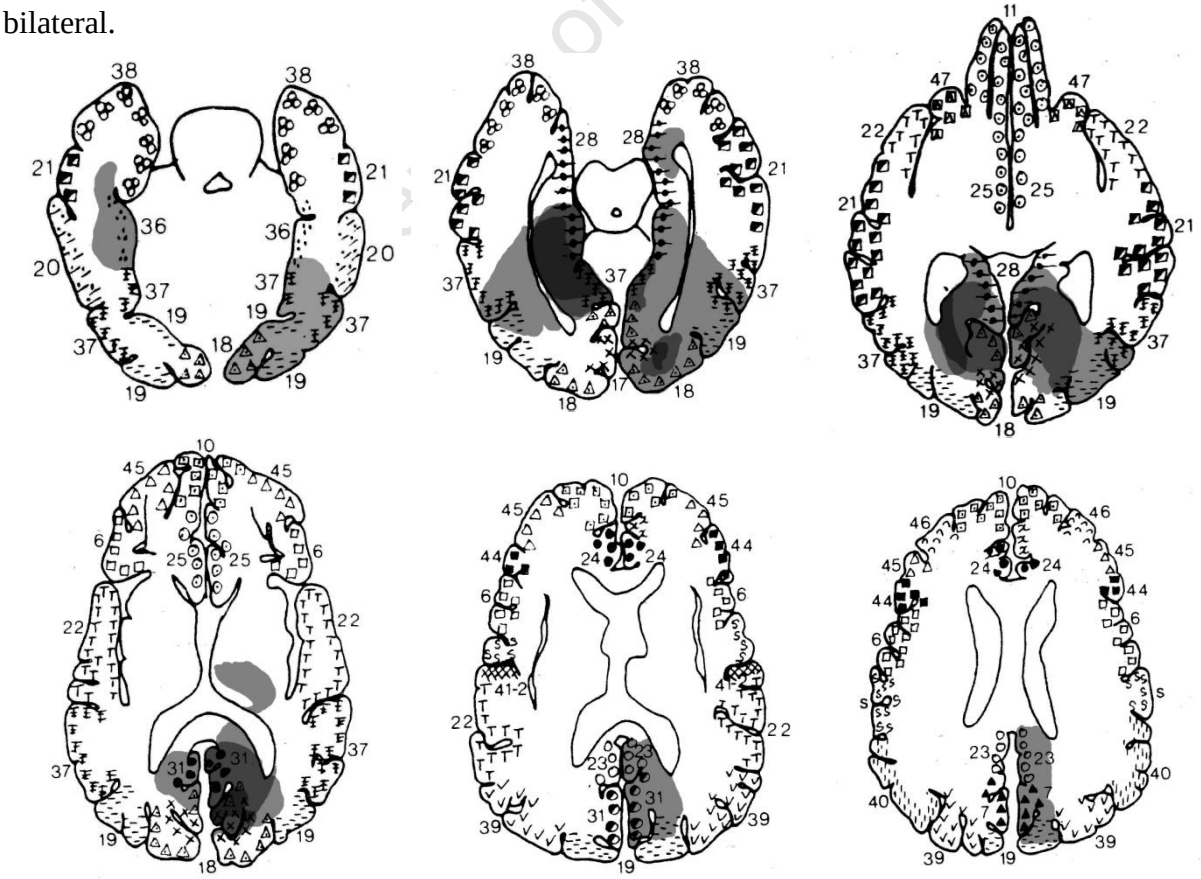


Figure 25. Combined template of the six dreamers (coronal view). All lesions were unliteral.

The anterior/posterior difference is easier to see on a medial view (Figure 26). This shows the medial view of the combined templates of the three non-dreamers for comparison with figure 27, which shows the combined templates of the six dreamers.

Figure 26. Combined template of left and right lesions in the three non-dreamers (medial view).

Figure 27. Combined template of left and right lesions in the six dreamers (medial view).

The comparison of figure 26 with figure 27 shows that in contrast to the lesions of the non-dreamers, which were generally confined to the occipital lobes, the lesions for the dreamers extended more anteriorly and inferiorly toward the medial temporal lobes and parahippocampal areas.

Table 20 shows the distribution of lesion sites for all cases, coded by Brodmann's areas for comparison.

Table 20.
Lesion sites of all cases

	Non-dreamers						*Dreamers*											
	Case 1		*Case 2*		*Case 3*		*Case 4*		*Case 5*		*Case 6*		*Case 7*		*Case 8*		*Case 9*	
Site	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*	*Left*	*Right*
BA 7									x									
BA 17	x	x			x				x		x						x	
BA 18	x	x	x	x	x				x		x						x	
BA 19	x		x	x	x				x		x					x	x	
BA 20											x							
BA 23									x									
BA 28								x	x		x			x				
BA 31					x						x							
BA 36				x				x	x		x			x		x	x	
BA 37	x	x		x				x	x		x			x		x	x	
BA 39						x												
BA 40						x												
Thalamus	x			x													x	

From an analysis of table 20, it was possible to chart the percentage frequency of lesion of the individual Brodmann's areas for comparison between the two groups.

Table 21.
Percentage frequency of lesion site

Site	*Non-dreamers* (*n* =3)	*Dreamers* (*n* =6)
BA 7	0%	16.67%
BA 17	66.67%	50%
BA 18	100%	50%
BA 19	100%	66.67%
BA 20	0%	16.67%
BA 23	0%	16.67%
BA 28	0%	66.67%
BA 31	33.33%	16.67%
BA 36	33.33%	100%
BA 37	66.67%	100%
BA 39	33.33%	0%
BA 40	33.33%	0%
Thalamus	66.67%	16.67%

Table 21 lists each lesion site and provides percentage means for determining the most frequently infarcted areas in the three non-dreamers and the six dreamers. Tables 20 and 21 are summarised in figure 28.

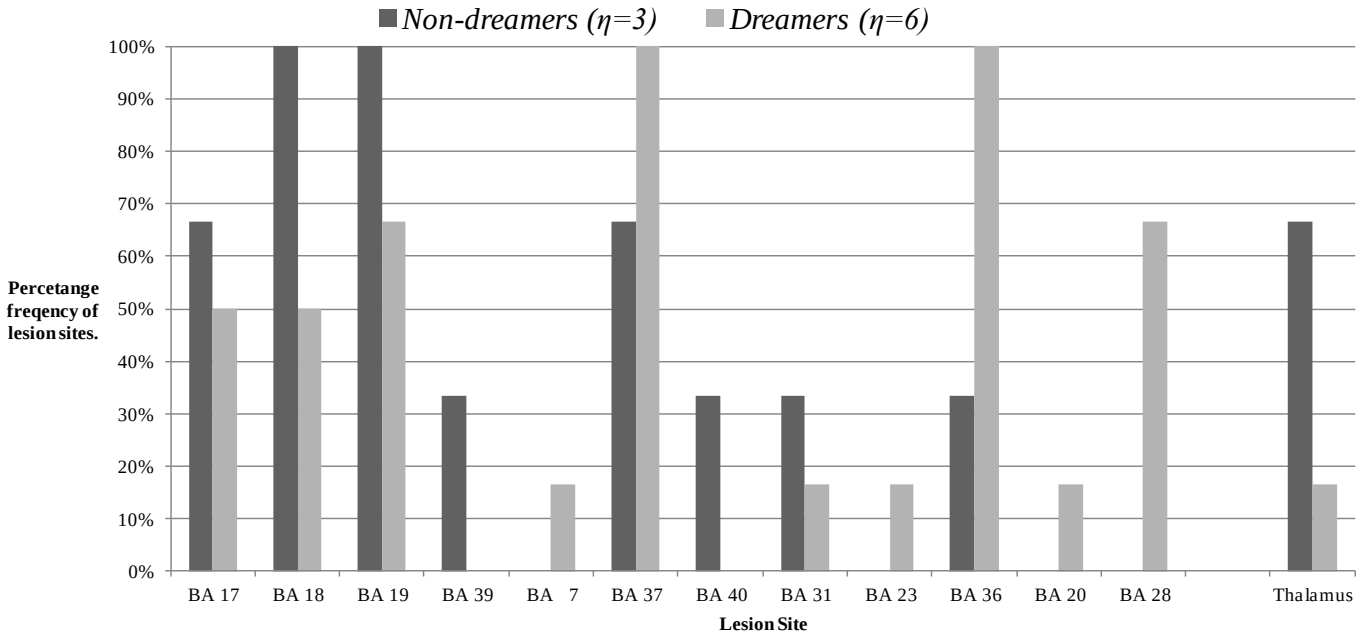


Figure 28. Graphical representation of lesion distribution among the three non-dreamers and the six dreamers.

Neuropsychological Findings

The neuropsychological test scores for all cases are tabulated below (Table 22).

Table 22.
Neuropsychological Assessment: All Cases

		Group								
		Non-dreamers			Dreamers					
Test	*Maximum Score*	*Case 1*	*Case 2*	*Case 3*	*Case 4*	*Case 5*	*Case 6*	*Case 7*	*Case 8*	*Case 9*
Luria's Visual Scenes	*14*	10	11	9	13	14	11	-	13	14
Boston Naming Test	*60*	57	46	34	31	58	33	-	20	51.5
Rey-Osterrieth Complex Figure	*36*									
Copy		25	32	30	-	34	27	32	29	33
Immediate		4.5	15	4	-	16	6	9	14	13
Delayed		-	16	4	-	14	-	-	15	10
WAIS-III Blocks	*68*	29	20	28	21	37	16	-	13	20
Digit Span										
Forwards		6	5	6	6	6	5	5	4	5
Backwards		3	6	4	3	3	3	-	4	6
Corsi's Blocks										
Forwards		5	6	4	5	5	5	-	6	6
Backwards		5	4	4	-	4	4	-	4	5
Babcock Story Recall	*21*									
Trial A		7	7	2	7	11	4	8	4	12
Trial B		8	12	5	9	15	7	12	5	15
Trial C		-	-	2	-	13	-	-	3	13
Benton's Visual Retention Test	*7*	6	6	5	2	6	3	-	0	5
Revisualization										
South African Flag		normal	normal	normal	normal	normal	normal	normal	normal	normal
Canary		normal	normal	normal	normal	normal	normal	normal	normal	normal
Sleep Laboratory		normal	normal	normal	normal	normal	normal	normal	normal	normal

Where possible, scores for each group were combined and means were calculated. Table 23 shows these mean scores for comparison. The mean values expressed in Table 23 were calculated from of the available scores for the non-dreaming and dreaming cases respectively.

Table 23.
Neuropsychological Test Means

Test	*Maximum Score*	*All Cases*	*Non-dreamers*	*Dreamers*
Luria's Visual Scenes	*14*	11.88	10	13
Boston Naming Test	*60*	41.31	45.67	38.7
Rey-Osterrieth Complex Figure	*36*			
Copy		30.5	29	31
Immediate		10.19	7.83	11.6
Delayed		9.8	10	13
WAIS-III Blocks	*68*	23	25.67	21.4
Digit Span				
Forwards		5.33	5.67	5.17
Backwards		4	4.33	3.8
Corsi's Blocks				
Forwards		5.25	5	5.4
Backwards		4.29	4.33	4.25
Babcock Story Recall	*21*			
Trial A		6.89	5.33	7.67
Trial B		9.78	8.33	10.5
Trial C		7.75	-	9.67
Benton's Visual Retention Test	*7*	4.13	5.67	3.2

A comparison of the neuropsychological test means reveals no striking differences between the two groups. However, non-dreamers performed somewhat better on the BNT and on the BVRT. This may indicate that memory-loss was greater for the dreaming cases, than for the non-dreaming cases. However, the scores from the Babcock story recall and from the recall trials of the ROCF show the opposite trend, i.e. dreamers performed somewhat better on these memory tests than non-dreamers. Of note here is the overall poor performance of non-dreaming Case 3, Mrs HS, whose scores no doubt impacted on the means for the non-dreaming group. The means were highly sensitive to fluctuations in individual case performances due to the small sample sizes of each group; an effect which was increased in the non-dreaming group. Statistical analyses, which could shed light on any potential differences between the two groups, are meaningless in such small sample sizes.

CHAPTER 6: DISCUSSION

This study addressed an area of controversy in our current knowledge of the neurological basis of cessation of dreaming. Specifically, this study first aimed to confirm a recent case report by Bischof and Bassetti (2004), which showed that heteromodal (complete) cessation of dreaming could occur as a result of a lesion in medial occipito-temporal cortex. Lesions in this brain region - unimodal association cortex - had previously been associated with unimodal loss of visual dream-imagery in Solms's (1997) reformulation of the classic Charcot-Wilbrand Syndrome (CWS). Thus the aim of this study was to confirm that heteromodal cessation of dreaming could indeed result from lesions in medial occipito-temporal cortex, by testing the ability to dream of nine posterior cerebral artery stroke patients over two consecutive nights in a sleep laboratory.

Cessation of Dreaming with Medial Occipito-Temporal Lesions

Our first hypothesis (p. 13) is confirmed. Three cases with medial occipito-temporal lesions (Case 1, 2, and 3) failed to report any dreams from a total of five REM sleep awakenings, and six morning interviews. It could be argued that Case 3 may have in fact been a dreamer, since only one REM sleep awakening was performed in this case, leaving the possibility that a positive dream report could have been obtained in a subsequent awakening. However, REM sleep awakenings were supplemented with morning interviews after each night in the sleep lab, and no positive dream reports were obtained from Case 3 at either morning interview or at follow-up clinical interview. Murri, Massetani, Siciliano, and Giovanditti (1985) tested dream recall from non-REM and REM sleep awakenings in 19 brain-injured patients. Importantly, the authors supplemented these awakenings with morning dream diaries. Significant agreement was found between the results obtained using morning diaries and the awakenings. It was thus concluded that the compilation of a diary on morning awakening appeared sufficiently reliable to reveal the presence or absence of dream recall in patients with focal cerebral lesions in the acute phase of the disease. Thus the fact that no positive dream reports were obtained from Case 3 (one week post-CVA) on REM awakening, or at the morning interviews, in addition to the fact that she reported a complete cessation of dreams at all clinical interviews, provides sufficient evidence that she was in fact a non-dreamer.

A corollary of the findings in this study is now apparent. That is, the case of Bischof and Bassetti (2004) is not unique; this study has reported three additional cases of complete cessation of dreaming with similar pathology, studied with similarly empirical techniques. Specifically, all four cases – that of Bischof and Bassetti, Mrs PA, Mrs MC, and Mrs HS –

had thrombotic infarction in the territory of the PCA, with lesions in medial occipito-temporal cortex. All four cases were awakened during REM sleep in order to confirm that they had completely lost the ability to dream. Additionally, all four cases underwent neuropsychological testing in order to confirm that none of these patients displayed any evidence of a neurocognitive deficit that would have undermined their ability to recall their dreams if they were in fact having any. Although Mrs PA and Mrs HS performed poorly on the Babcock story recall test and on the recall trials of the ROCF, both were able to revisualise the sleep lab, and both were able to describe the experiential events that took place at the sleep lab in narrative and visuo-spatial detail. Furthermore, a comparison of the means between the non-dreamers and the dreamers, revealed no striking differences in the scores on tests of memory. The non-dreamers were, overall, thus no worse off in terms of memory functioning than dreamers. Moreover, the concern that cessation of dreaming in brain-injured patients is in fact a failure to remember the dreams they are actually having, has been addressed by Yu (2006). He compared the memory functions of seven dreamers with that of a single non-dreamer and found that the non-dreamer was no more impaired in memory than the dreamers. Importantly, he found that even patients with profound memory deficits due to infarctions in posterior brain regions were still able to report their dream experiences, and concluded that dream cessation did not appear to be a secondary symptom of memory dysfunction. It is therefore unlikely that in the present study, Mrs PA and Mrs HS were dreaming, but failed to report any dreams because of a failure to recall them due to memory dysfunction. This is consistent with the findings of Solms (1997).

Implications for dream-function research

Consistent with Bischof and Bassetti (2004), Case 1, 2, and 3 provide further evidence that REM sleep and dreaming are dissociable states. That is, all three non-dreamers in this study were polysomnographically monitored, and a registered sleep-laboratory technician confirmed the presence of REM sleep based on the AASM criteria. Solms (2000) argued that REM sleep and dreaming are doubly dissociable states, i.e. that dreaming can occur without REM sleep, and that REM sleep can occur without dreaming. Although most dream researchers readily accepted the first claim, that dreaming could occur without REM sleep, some argued that no evidence had been presented in favour of the second claim that REM sleep could occur without dreaming (Coenen, 2000). However, despite the presence of normal REM sleep, all three non-dreamers in this study were not able to report a single dream, even when awakened from REM sleep. Case 1, 2, and 3, and that of Bischof and Bassetti (2004), thus provide further empirical evidence that REM sleep can indeed occur without dreaming. Consequently, these four cases provide further support for Solms's (2000)

argument that dreaming is controlled by forebrain mechanisms independent from the brainstem mechanisms that control REM sleep.

These findings carry significance for greater scientific questions regarding the function of dreaming. Specifically, by demonstrating that complete cessation of dreaming can occur in the presence of REM sleep, theories regarding the function of dreaming, which are founded on the erroneous conflation of REM sleep and dreaming, such as the activation-synthesis hypothesis (Hobson & McCarley, 1977), are thoroughly discredited. The necessity to separate scientific study of the function of dreaming from theories of the function of REM sleep is once again emphasised.

Implications for Charcot-Wilbrand Syndrome

A further finding from the neuropsychological testing of the non-dreamers in this study is that none of these cases showed any evidence of the neurocognitive deficits that are classically associated with CWS. All three cases were able to accurately revisualise the sleep lab room, and the interior layout of their homes. Thus, while complete cessation of dreaming had been confirmed in all three cases, none of them displayed evidence of the cardinal symptom of CWS, i.e. visual irreminiscence. In addition, no evidence of prosopagnosia or topographical agnosia or amnesia could be elicited on neuropsychological testing. Taken together with the case of Bischof and Bassetti (2004), these three cases provide strong evidence that total cessation of dreaming is a discrete neuropsychological symptom following damage to posterior cortex.

In light of Solms's (1997) challenges to the CWS concept, the finding that cessation of dreaming can occur without the neuropsychological deficits that are classically associated with CWS, is perhaps unsurprising. Solms et al. (1996) had already pointed out that Wilbrand's Fräulein G. experienced complete cessation of dreaming without suffering from the primary deficit (visual irreminiscence) upon which this secondary deficit was supposedly consequent. The same applied to the many cases of global cessation of dreaming that Solms reported 1997 study. However, at first glance, the localisation of the lesion in Case 1, 2, and 3 casts doubt on Solms's (1997) reorganisation of CWS into two distinct sub-syndromes, with two distinct pathological anatomical correlates. A clearer understanding of the implications for this can be gained from consideration of this study's second hypothesis.

Lesion Characteristics in Medial Occipito-Temporal Cessation of Dreaming

Comparison of the lesion sites between non-dreamers and dreamers revealed that all the non-dreamers suffered *bilateral* damage, while all the dreamers suffered *unilateral* damage. Case 1, and 2, suffered bilateral damage involving the medial occipito-temporal regions, while

Case 3 had bilateral damage of the left medial occipital lobe, and right inferior parietal lobule (IPL). The significance of this lesion in the IPL is questionable in view of the PET studies discussed earlier. Numerous imaging studies have consistently found significant deactivation of the IPL during REM sleep (Braun et al., 1997; Maquet et al., 1996; Nofzinger et al., 1997).

However, recent advances in neuroimaging methods have allowed for a higher temporal resolution than that obtained with PET or conventional fMRI. These new methods of measuring brain activation across the sleep-wake cycle have led some researchers to reconsider the findings from PET studies of old. For instance, using magnetoencephalography, one study found significant increases of slow wave and gamma band activity in the left dorsolateral prefrontal cortex (DLPFC) and left dorsomedial prefrontal cortex during REM sleep when compared with wakefulness (Ioannides, Kostoupolos, Liu, & Fenwick, 2009). Based on these findings, Kubota et al. (2011) monitored changes in the haemoglobin concentration and tissue oxygenation index in bilateral DLPFC throughout the REM sleep period. These researchers found that significant activation of DLPFC coincided with the appearance of the first REM just after the onset of REM sleep. They concluded that DLPFC, a region that had previously been shown to be deactivated during REM sleep in older PET studies, could play a role in cognitive processes during REM sleep. No mention of the IPL was made in these recent sleep-imaging studies. Nevertheless, the fact that some heteromodal association areas (DLPFC) have now been shown to be intermittently activated in REM sleep, leaves open the possibility that other heteromodal association areas, such as IPL, could show the same intermittent pattern of activation. Further research would therefore be needed before any conclusion regarding Case 3's 'parietal' lesion can be made.

Nevertheless, based on the older PET studies, Yu (2001) dismissed the role of inferior parietal cortex (BA 39 & BA 40), highlighting the importance of BA 19, BA 22, and BA 37 in dream formation. Highly consistent with Yu's (2001) conclusions, and with the lesion sites in Bischof and Bassetti's (2004) case (BA 17, BA 18, BA 19, & BA 37), Mrs PA suffered bilateral damage to BA 17, BA 18, and BA 37, as well as BA 19 and the thalamus on the left, while Mrs MC suffered bilateral damage to BA 18, and BA 19, as well as BA 36, BA 37, and the thalamus on the right. Mrs HS suffered similar damage to these areas (BA 17, BA 18, BA 19, & BA 31) albeit unilaterally. Therefore all three cases experienced heteromodal cessation of dreaming with lesions in unimodal association cortex, the area that Solms (1997) had associated with unimodal cessation of visual dream-imagery. It is important to note here that in his original series, Solms in fact identified four patients with bilateral medial occipito-temporal lesions who experienced heteromodal cessation of dreaming. Unfortunately, in all four cases, the pathological process did not lend itself to precise anatomical localisation,

ranging from multiple, diffusely distributed cysts, to large bilateral subdural hematoma, multiple lacunar infarcts, and haemorrhage secondary to trauma. This may have been why Solms did not take these cases as evidence of bilateral medial occipito-temporal lesions leading to heteromodal cessation of dreaming.

In the present study, two cases (Case 1, and 2) with precise radiological documentation of the lesion sites had the exact lesion that Solms associated with unimodal cessation of visual dream-imagery, i.e. *bilateral* damage to medial occipito-temporal cortex. Yet these cases indisputably experienced complete cessation of dreaming. Furthermore, six cases with unilateral thrombotic infarctions in PCA territory and precisely localised, focal lesions in medial occipito-temporal cortex were reported in this study. Importantly, these cases showed no evidence that their dreams had been affected by their stroke, in either the heteromodal or unimodal sense. When these cases (notwithstanding the case of Mrs HS) are considered together with Bischof and Bassetti's (2004) case, and Wilbrand's prototypical case, it appears that a tentative conclusion can be drawn *that bilateral medial occipito-temporal lesions can be associated with heteromodal cessation of dreaming*, as well as with cessation of visual dream-imagery. The two putative subtypes of the Charcot-Wilbrand Syndrome therefore collapse (at least partially) back into a single syndrome.

Of course, as Solms (1997) argued, heteromodal cessation of dreaming necessarily implies the cessation of visual dream-imagery, but what can be said of the lesion site responsible for cessation of visual dream-imagery in isolation? The fact that no cases of this latter dreaming deficit were reported in the present study is either a testament to its rarity[5], or evidence of the fact that medial occipito-temporal lesions are not *necessarily* associated with it. In fact, of the 12 cases that Solms (1997) reviewed reporting this type of dreaming deficit, only one case (that of Grünstein, 1924) was clinically judged to have suffered a thrombotic infarction in the left *lateral* occipital lobe. All other cases, including the two cases reported by Solms (1997), himself had suffered pathological processes of less localising quality, including AVM, carbon monoxide poisoning, Alzheimer's disease, and trauma. It is also of paramount importance to note that the rare clinical symptom of irreminiscence was observed in all these cases, but not in any of the present cases, and also not in Bischof and Bassetti's or Wilbrand's cases. Further research is thus necessary before a clearer account of the neurological and neuropsychological correlates of unimodal cessation of visual dream-imagery can be obtained. Such research would need to employ precise localising techniques in cases with precisely localisable pathology (such as thrombotic infarction), while thoroughly documenting the nature of the dreaming deficit.

[5] Solms identified an incidence of 1.1% in his series of 361 patients.

The thalamus and cessation of dreaming with medial occipito-temporal lesions

In addition to the clear pattern of bilateral damage in non-dreamers with medial occipito-temporal lesions, comparison of the lesion sites between the two groups in the present study showed that the thalamus was more often damaged in non-dreamers than in dreamers. Two out of three cases (Case 1 and 2) with complete cessation of dreaming had lesions in the thalamus. Importantly, the thalamus was also damaged in the case of Bischof and Bassetti (2004), while involvement of the thalamus is indeterminable in Wilbrand's case, since he seems to have examined only the cortex at autopsy, and no mention is made of the state of the subcortical structures. In vivo imaging techniques had of course not yet been invented in Wilbrand's time. However, an appraisal of the autopsy report (p. 11 above) suggests that the thalamus or at least the connections to it may indeed have been damaged. Specifically mentioned is "a fresh soft focus which has destroyed completely the central white matter of the [left] hemisphere" (Wilbrand, 1892, cited in Solms et al., 1996, p. 98). This suggests that the status of subcortical structures in this hemisphere consequent to the first of Fraulein G.'s two strokes, at least would have been difficult to determine. Therefore, a lesion of the thalamus in this case cannot be completely discounted.

Two further cases reported in the present study require consideration before any conclusions can be drawn regarding the role of the thalamus in medial occipito-temporal cessation of dreaming. First, the case of Mrs HS (Case 3) is a case of heteromodal loss of dreaming, with a lesion involving medial occipito-temporal cortex, yet without involvement of the thalamus. Second is the case of Mr RS (Case 9) who displayed no discernible evidence of a change in dream quantity or quality, yet his lesion is almost identical to Mrs HS. Moreover, these two cases are comparable in terms of chronicity, in so far as both cases underwent the sleep lab assessment within one week post stroke. Therefore a dissociation between heteromodal loss of dreaming and thalamic lesions seems to exist between these two cases. That is, *heteromodal loss of dreaming can occur* with medial occipito-temporal lesions *without involvement of the thalamus*, and medial occipito-temporal lesions *with involvement of the thalamus can occur without heteromodal loss of dreaming*.

However, three caveats demand attention here: first, Mrs HS presented with a history of sudden onset of bilateral hearing loss, which was followed by intermittent bouts of vertigo and dizziness. These latter symptoms may have been the result of chronic hypoperfusion to the posterior regions as a result of some vascular process, a possibility made more likely by the presence of long-standing risk factors. Second, Mrs HS indeed suffered another CVA one month after the CVA for which she was referred to this study, which resulted in damage to the right thalamus. This provides further evidence that at the time of the sleep lab assessment, she may have been suffering from an ongoing pathological vascular process in the territory of

the PCA. Third, while Mrs HS did have evidence of bilateral damage, the lesion on the right was more in the region of the parietal lobe white-matter (heteromodal association), than medial occipito-temporal cortex. Crucially, the postero-lateral thalamus specifically projects to parietal heteromodal association cortex (Walsh & Darby, 1999). Case 1 and case 2, both had lesions involving the postero-lateral aspects of the thalamus (though in Case 2 the damage was not confined to this thalamic region). Moreover, the case of Bischof and Bassetti (2004) also had a lesion involving the postero-lateral thalamus, although they did not consider this a critical lesion site in their case. Therefore all three cases of heteromodal cessation of dreaming with bilateral medial occipito-temporal lesions, had lesions involving the postero-lateral thalamus, while the only case that did not (Case 3), had a lesion in the precise brain region to which this area of the thalamus projects.

Neuropsychological testing of the functions related to the postero-lateral thalamus, and to the heteromodal association cortex to which it projects, would have been useful in determining if the neurocognitive deficits expected with lesions to these areas were actually present. Unfortunately in the present study, specific neuropsychological assessment of parietal lobe functions were not carried out because the possible involvement of this region was an unexpected finding, mostly due to the fact that only cases with focal PCA lesions were studied.

The thalamus and preservation of dreaming

The case of Mr RS can now be considered in view of the above mentioned caveats. Specifically, although he suffered damage to the thalamus and the medial occipito-temporal region, this latter damage was lateralised to the left. While there is potential for this to weaken arguments that a *thalamic lesion* is necessarily associated with medial occipito-temporal cessation of dreaming, it shifts our attention to the argument that *bilateral damage* in the territory of the PCA is necessary for heteromodal cessation of dreaming.

All cases reported to date of global cessation of dreaming following PCA strokes, including the cases of Wilbrand and Bischof and Bassetti as well as our own three cases, had bilateral occipito-temoral lesions.

A critical role for thalamic damage in cases of heteromodal loss of dreaming with bilateral damage in the territory of the PCA nevertheless remains a possibility. Clearly, further research studying additional cases with global loss of dreaming in PCA territory infarctions is needed in order to gain a clearer understanding of the potential role of the thalamus in such cases.

Negative Cases

In addition to the three new cases of complete cessation of dreaming, the present study reported six cases with preservation of dreaming. All six cases suffered unilateral damage to medial occipito-temporal cortex, and a total of 11 dream reports were obtained from 13 REM awakenings and 12 morning interviews. These cases support the argument that *bilateral damage* is a necessary pathological anatomical correlate of heteromodal loss of dreaming in PCA strokes. The argument by Poza and Massó (2006) that a unilateral left occipito-temporal lesion is sufficient to cause total dream loss is not supported, and the loss of dreaming in their case must be attributed to the non-localisable character of the lesion (congenital AVM with haemorrhage). Even in our Cases 5 and 6 where extensive unilateral lesions in medial occipito-temporal cortex were seen, these participants provided normal dream reports on REM sleep awakening.

Neuropsychological testing of the dreamers indicated that loss of dreaming was not secondary to an aphasic deficit, notwithstanding ongoing claims to this effect (Dumont, Braun, & Guimond, 2007; See Solms, 1997 for review). Although no case in the present study was clinically judged to have aphasia, dreamers performed somewhat worse on the BNT than non-dreamers. Unfortunately, statistical analyses could not be performed in order to test for significant differences between the two groups on this, or any other neuropsychological test mean.

An expected difference would be that since the lesion site in cases with preservation of dreaming is generally located more anteriorly (involving more medial temporal and parahippocampal structures), these cases would perform worse on tests of memory. However, in the present study the dreamers appeared, if anything, to perform slightly better on tests of memory than the non-dreamers. However, the mean scores for non-dreamers were surely impacted by the poor performance on these tests by Case 3, Mrs HS. The small sample sizes in both groups make them particularly susceptible to outliers. Further research with a larger sample is thus necessary in order to fully evaluate for potential differences in the neuropsychological performances of dreamers when compared with non-dreamers. At present all that can be said with confidence is that there is no gross neuropsychological difference between dreamers and non-dreamers with PCA stroke, notwithstanding the fairly robust pathological anatomical differences between the two groups. That is, the non-dreamers are distinguished from dreamers by a) bilateral lesions, b) thalamic or parietal involvement, and c) a more posterior (occipital) than anterior (temporal) location of the lesion.

Implications for Solms's Dream System

As mentioned earlier, the medial occipito-temporal lesion sites associated with heteromodal loss of dreaming in this study were associated only with a particular unimodal loss of visual dream-imagery in Solms's (1997) study. On the basis of the findings in his study, Solms therefore theorised that medial occipito-temporal structures contribute "a factor of visual representation to the overall process of dreaming" (p. 239). He further argued that the critical components of the cortical visual system for the representation of dreams may include V3, V3A, and V4, and exclude V5 and V6. Together with V2, these areas are often referred to as extrastriate cortex, correspond with BA 18 (V2), and BA 19 (V3-5), and form the visual association area of the occipital lobe (Hubel, 1995).

In the present study, a high frequency of BA 17, BA 18, and BA 19 lesions was observed in patients with global cessation of dreaming. The role of BA 17 lesions may be questionable in so far as patients with lesions in this area have been shown to have vivid visual dreams (Brown, 1972; Grünstein, 1924; Solms, 1997). Based on these observations, Solms (1997) noted an apparent lack of any contribution by primary visual cortex (V1 – striate cortex) to the normal dream process. This led him to conclude that the visual imagery of normal dreaming is internally generated. That is, Solms concluded that dreams are generated by a process of backward projection. These conclusions were subsequently supported in a PET study measuring cerebral activity to determine the interrelationships within visual cortices and their projections during REM sleep (Braun et al., 1998). The authors found selective activation of extrastriate visual cortices and attenuation of activity in primary visual cortex. They further reported a significant correlation between increased cerebral blood flow in extrastriate areas and decreased blood flow in primary visual cortex. Moreover, these researchers found extrastriate activity with concomitant limbic and paralimbic activity, but with marked reduction of activity in frontal association areas. Thus, consistent with Solms's (1997) conclusions, they suggested that the "visual association cortices and their paralimbic projections may operate as a closed system" during REM sleep, "dissociated from the regions at either end of the visual hierarchy that mediate interactions with the external world" (Braun et al., 1998, p. 91).

Thus it would appear that (bilateral) damage to the medial occipital lobes (BA 18 & BA 19) represented the minimum lesion extension necessary to cause global cessation of dreaming in this study. These findings are almost identical to those of Bischof and Bassetti (2004) who argued that deep bilateral occipital damage, including the right lingual gyrus, might represent the minimum lesion extension necessary to produce CWS. However, if the factor of visual representability is placed at the terminal end of the dream-generation process, as it is in Solms's dream system, then it is difficult to explain how damage to unimodal visual

association cortices can result in global loss of dreaming. However, Bischof and Bassetti (2004) overlooked the lesion in the postero-lateral thalamus – an area certainly damaged in two, and possibly pathologically affected in the third, of three non-dreamers in this study. Moreover, Case 3 suffered a lesion in the white matter adjacent to the precise area that the postero-lateral thalamus projects to – the inferior parietal lobule – which is the area that Solms associated with heteromodal cessation of dreaming. Therefore, to conclude that the case of Bischof and Bassetti (2004) or the similar cases reported in this study *definitively* refute Solms's (1997) model of the normal dream process would be premature. That is, although the role of the thalamic lesions cannot be confirmed, it cannot be disregarded. Further research is needed in order to determine the role, if any, of the concomitant thalamic (i.e. 'parietal') lesions in heteromodal cessation of dreaming with medial occipito-temporal infarcts.

Notwithstanding these caveats, the findings of the present study, combined with those of Wilbrand (1887) and Bischof and Bassetti (2004), strongly suggest that global loss of dreaming can occur with unimodal visual cortical lesions. Unless further evidence is brought to light, which supports the possibility that all such cases suffer concomitant thalamic and/or parietal lesions, Solms's (1997) division of the CWS into two distinct clinicoanatomical subtypes must be abandoned.

Limitations and Future Directions

An obvious limitation of this study is the small samples size of the non-dreaming group. An attempt was made to address the predicted rarity of the experimental group – participants with heteromodal cessation of dreaming due to PCA territory infarction – by recruiting patients from two large and busy medical institutions in Cape Town, South Africa. However, PCA territory infarctions are relatively rare, when compared to middle cerebral artery territory (MCA) infarctions, accounting for only nine percent of all strokes (Bogousslavsky, Van Melle, & Regli, 1988). In addition, heteromodal cessation of dreaming as a result of non-hemorrhagic PCA strokes has been shown to be exceedingly rare, with just two cases reported in the previous literature (Bischof & Bassetti, 2004; Wilbrand, 1887). Thus in future research, it may be necessary to recruit considerably more participants with the appropriate pathological anatomy.

A further limitation of this study is a direct consequence of the first limitation. That is, no meaningful statistical analyses could be carried out, as a result of the small sample sizes. This prevented evaluation for potentially meaningful differences in the neuropsychological profiles between the non-dreamers and dreamers in this study. Until such time as future research, with sample sizes large enough to perform statistical analyses of these differences is

undertaken, nothing conclusive can be said about the differences in neuropsychological performances between dreamers and non-dreamers. Moreover, the potential role of the thalamic lesions on the parietal functioning of non-dreamers as compared with dreamers must be addressed directly.

Another limitation of this study consequent on the small sample size of the non-dreaming group is the method of lesion analyses. Specifically, this study used three of the traditional methods of displaying and mapping the lesions, viz. 1) representative slices from each participant's MRI/CT scans were provided with 2) depiction of each participant's lesion on standard templates that corresponded with the MRI/CT slices provided, and 3) written descriptions of the lesions were given to ensure that the lesion sites were comprehensively defined.

However, these methods of presenting anatomical data are no longer the standard, and each method has its own drawbacks and shortcomings (for a full review of these see Rorden & Brett, 2000). There are now advanced, freely available computer programs that can be used to more accurately display and compare neuroanatomical data. Such programs provide automated methods for displaying the MRI scans of different patients in the same stereotaxic space, which allows for direct comparison of lesion location and volume (Rorden & Brett, 2000). Akin to functional imaging, the relationship between tissue damage and behaviour can then be analysed on a voxel-by-voxel basis (Bates et al., 2003). Many neuropsychological studies have now been carried out using these methods of spatial normalisation, statistical parametric mapping, and voxel based lesion-deficit mapping (Baldo, Schwartz, Wilkins, & Dronkers, 2006; Chen & Herskovits, 2010; Martinaud et al., 2012; Seghier, Ramlackhansingh, Crinion, Leff, & Price, 2008; Verdon, Schwartz, Lovblad, Hauert, & Vuilleumier, 2010). Various techniques for increasing the power, accuracy, and ease of these methods are being made available (Chen & Herskovits, 2010; Seghier et al., 2008). However, these methods are based on statistical and mathematical procedures that require the appropriate sample sizes. Thus future research with a larger group of PCA stroke cases would be able to make use of these modern methods of lesion mapping and comparison.

Indeed, a study of medial occipito-temporal lobe damaged cases with a sample size large enough to perform statistical analyses would allow for comparison with cases similar to Solms's (1997) cases of lateral occipito-temporo-parietal damage. This could aid in the formation of a complete map of the system of anatomical structures necessary for dreaming. Once all the areas of the brain critical for dreaming have been determined, a new evidence-based theory of the mechanism of the normal dream process can be developed.

Conclusion

Both of the hypotheses tested in this study were confirmed. Bilateral damage to the unimodal cortex of the medial occipito-temporal region as a result of non-hemorrhagic infarction in the territory of the PCA can result in heteromodal cessation of dreaming. However, concomitant damage to thalamic and/or parietal structures was noted in all three cases. This leaves room for doubt. The three cases of heteromodal cessation of dreaming from PCA territory infarcts reported in this study represent the third, fourth, and fifth cases of such description reported to date. In at least one of the two previous cases, the thalamus was also involved, possible in both. The implications of the findings in this study were discussed, and the limitations were described particularly in relation to future research.

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APPENDIX

Informed Consent Form

Title of research study: Posterior Cerebral Artery (PCA) Infarcts and Dreaming: A Neuropsychological Study

Name of principal researcher: Gavin Marchbank
Department/research group address: Psychology Department
Faculty of Humanities
University of Cape Town
Telephone: 021 650 3435
Email: mrcgav002@myuct.ac.za

Name of participant:

You are invited to take part in a research study from the Department of Psychology at the University of Cape Town in order to see whether your stroke has had an effect on your dreams. Your participation is completely voluntary.

Participant's involvement:
What's involved: Your medical folders will be examined and their information may be used. However, complete confidentiality will be maintained and your name will not be used.
The study will involve two consecutive nights in a sleep laboratory. You will be connected to a polysomnograph which is a simple device that involves small pads being placed on different parts of your body. You will be asked to sleep as you would normally at home. During the first night, you will be awakened twice by the researcher and asked whether you were dreaming. During both nights, your sleep cycles will be recorded.

Risks: There are no risks associated with this study, except the minor risk of falling out of bed. However, if you feel uncomfortable at any time you may withdraw from the study without any negative consequences for yourself. All data will be kept confidential and will only be used for research purposes.

Benefits: There are no direct benefits for participating in this study except for the possibility of detecting any sleep disorders that you may have.

Payment: As you would be giving up a considerable amount of your time, you will be paid R500 for each night that you complete in the sleep laboratory. Thus, if you complete the full two nights of the study you will receive R1000.

Please sign if you have read all the information and you agree to take part in the study.

Name of Participant: _____

Signature of Participant: _____

Date: _____

Name of principal researcher: _____

Signature of principal researcher: _____

Date: _____