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Mnemonic Monitoring in Anosognosia for Memory Loss

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ABSTRACT

Objective: Anosognosia, or unawareness, for memory loss has been proposed to underlie cognitive functions such as memory and executive function. However, there is an inconsistent association between these constructs. Recent studies have shown that compromise ongoing self-monitoring of one's memory associates with anosognosia for memory loss. Yet to date it is unclear which memory monitoring mechanisms are impaired in these patients. In this study, we examined the extent to which *temporal monitoring* or *orbitofrontal reality filtering* (e.g., ability to monitor the temporal relevance of a memory) and *source monitoring* (e.g., the ability to distinguish which memories stem from internal as opposed to external sources) are associated with awareness of memory deficits.

Methods: A total of 35 patients (M=69 years; M=14 years of education) with memory difficulties following a stroke were recruited from outpatient clinics. Patients were assessed with measures of self-awareness of memory difficulties, cognitive abilities and two experimental paradigms assessing source and temporal monitoring.

Results and conclusion: Results showed that patients unaware of their memory difficulties were more likely to externalize the source of their memories. Specifically, those unaware of their deficits were more likely to assign an external source to memories that were internally produced (e.g., imagined). No differences were observed in relation to temporal monitoring between patients aware and unaware of their deficits. This study informs current theoretical models of self-awareness of memory loss. Future studies should attempt to replicate these findings and explore different memory monitoring mechanisms in relation to anosognosia for memory loss.

Key words: Anosognosia, unawareness, memory, reality filtering, monitoring,

1. Introduction

Anosognosia for memory loss, or unawareness of memory loss, is a complex construct that refers to the lack of awareness of memory deficits due to brain injury or degeneration (Agnew &

Morris, 1998; Mograbi & Morris, 2018). Several studies have investigated lack of awareness for motor, sensory and cognitive disorders leading to a growing belief that this syndrome is a multifaceted phenomenon that can be caused by the disruption of different cognitive mechanisms (e.g. Marcel et al., 2014; Cocchini & Della Sala, 2010) and anatomical networks (Moro et al., 2016; Pacella et al., 2019). Not surprisingly, different mechanisms can underlie anosognosia for memory impairment (e.g., Cosentino, 2014; Mograbi & Morris, 2013). Although different subtypes of anosognosia for memory impairment may exist (Agnew & Morris, 1998), recent studies showed that the ability to track and control consciously certain elements of the ongoing memory experience, may be crucial for the emergence of awareness of memory deficits (Chapman et al., 2018; Cosentino, Metcalfe, Butterfield, & Stern, 2007; Cosentino et al., 2016). Specifically, these studies demonstrated that deficient online monitoring of one's ongoing memory performance, also referred to as local (lower) levels of awareness (Clare, Markova, Roth, & Morris, 2011), is associated with compromised global (higher) levels of awareness or anosognosia for memory loss.

Deficits in monitoring memory performance can be contextualized as deficits in a mnemonic comparator as described in the Conscious Awareness Model (CAM) (Agnew & Morris, 1998; Morris & Mograbi, 2013). This comparator is hypothesized to detect ongoing memory errors by comparing them to the expected performance based on previous experiences. It is unclear, however, what specific mnemonic monitoring disruptions are critical for the breakdown of awareness. In an attempt to elucidate the elements that play a key role in supporting global memory awareness, this study examined two specific mnemonic monitoring mechanisms that could lead to an impairment of a mnemonic comparator as described in the CAM: a failure of *temporal*

*monitoring (*Schnider & Ptak, 1999), and a failure of *source monitoring* (Johnson & Raye, 1981, 2000).

Temporal monitoring or *reality filtering* refers to the ability to monitor the temporal relevance of a memory, that is, its timing in relation to other memories and its relevance to the present moment or the 'now' (Schnider & Ptak, 1999; Schnider, von Däniken, & Gutbrod, 1996; Nahum et al., 2012; Liverani et al., 2015). This monitoring process has been shown to rely on the orbitofrontal cortex (see Schnider, 2013 for a revision). Failure in temporal monitoring or reality filtering, demonstrated repeatedly in patients with confabulatory phenomena and disorientation, has been proposed as the mechanism by which these patients are unable to determine what information is relevant to the present moment resulting in a profound confusion of what is currently relevant (Cocchini, Lello, McIntosh, & Della Sala, 2014; Dalla Barba & La Corte, 2015; Gilboa et al., 2006; Nahum et al., 2012; Schnider, 2008, 2013; Schnider, Ptak, von Daniken, & Remonda, 2000; Schnider et al., 1996). Within the context of self-awareness of memory performance, the ability to know if a memory relates to the past or the present would appear key for patients to learn that their memory is not what it used to be.

Source monitoring or reality monitoring on the other hand, refers to a source attribution process by which memories that stem from an internal source can be distinguished from an external source (i.e., imagined vs. seen; Johnson, 1991; Johnson & Raye, 1981, 2000). Failures in this monitoring ability have been associated with memory and perceptual disturbances in patients with mental disorders and acquired brain injury (Brébion et al., 2000; Cocchini et al., 2014; Johnson, 1991; Radaelli, Benedetti, Cavallaro, Colombo, & Smeraldi, 2013; Turner & Coltheart, 2010). Further, within anosognosia for motor deficits, researchers such as Venneri and Shanks (2004) hypothesized that deficits in source or reality monitoring can underlie anosognosic patients'

inability to assess the veracity of their beliefs. Other groups (Fotopoulou et al., 2008; Jenkinson, Edelstyn, Drakeford, & Ellis, 2009; Jenkinson & Fotopoulou, 2010; Saj, Vocat, & Vuilleumier, 2014) have since assessed monitoring deficits through experimental paradigms and showed that patients anosognosic for motor deficits had difficulties distinguishing between self-generated information (imagined actions) versus externally experienced (seen actions). Similar to patients with anosognosia for hemiplegia, patients with anosognosia for memory loss might have difficulties discerning the expected (i.e., internal source) outcome for performance from their actual performance (i.e. external source), on a given memory task, evidencing a bias toward "externalizing" internal sources.

This study investigated the evidence for the role of two memory monitoring abilities, temporal monitoring and source monitoring, in producing anosognosia. We hypothesized that if the results supported impairment in temporal monitoring, patients unaware of their memory deficits may experience difficulties in becoming aware of their deficits as due to an inability to distinguish the accurate temporal relevance of a memory. That is, they have greater difficulties identifying temporally relevant items to the now) and suppressing the proactive interference of previous items (e.g., filtering out temporally irrelevant items) compared with patients aware of their impairment. On the other hand, if results supported a source monitoring deficit, anosognosics will show difficulty in monitoring internally vs externally generated memories, and we would expect higher error rates when discriminating between these compared to those who are aware of the memory deficits.

2. Methods

2.1. Participants

Forty-one patients were recruited from stroke outpatient clinics at St. George's NHS hospital, London, U.K. and at Columbia University Medical Center, New York, USA. Patients were recruited consecutively at their routine stroke follow up visits. All patients were referred by a consultant neurologist as having suffered a stroke. Once consented participants underwent a screening for memory impairment, those who showed memory impairment on initial screening with a story immediate and delayed recall test of the BCoS Brain Behavior Analysis test (Humphreys, Bickerton, Samson, & Riddoch, 2012) and performed below 5th percentile in age corrected scores were then enrolled for the full study. All participants provided full consent and procedures were approved by the NHS research ethical board in UK and the Institutional Review Board at Columbia University Medical Center in the US. Out of these patients, 4 did not have an informant to describe the patient's performance and anosognosia, and 2 did not have a stroke confirmed by their radiological report, resulting in a final sample of 35 patients.

The 35 patients included in this study were on average 69 (*SD* = 13) years old, had 14 (*SD* = 3.7) years of education, and 40% (*n* = 14) were women. Most (71.4%, *n* = 25) self-identified as White American or British, 20% (*n* = 7) as Black American or British, and 8.6% (*n* = 3) as South Asian. Most patients 94 % (*n* = 33) were right-handed. All patients had suffered from a stroke, with 3 patients having had several strokes ranging from 2 to 3 strokes in total. Mean time since first ever stroke was 42 months (*SD* = 69). Lesion location, as described in radiological and clinical reports, is provided in Table 1. Lesions described by radiologists include both the acute clinical stroke that prompted clinical evaluation and incidental subclinical lesions. A total of 45 informants

(10 patients had two informants) were on average 48 years old (*SD* = 17) and had 14 mean years of education (*SD* = 3).

Table 1. Lesion description of 35 patients with memory difficulties following stroke

Nature of lesion	Unilateral Left Hemisphere	Unilateral Right Hemisphere	Bilateral	**Total**
Haemorrhagic	1	1	3	**5**
Ischemic	15	7	8	**30**
Total	**16**	**8**	**11**	**35**

Number of patients with lesions encompassing left, right or both (bilateral) hemispheres. For further description of please refer to supplementary Table S5.

2.2. Measures

2.2.1. Cognition and anosognosia

With regard to cognition, patients were assessed with measures of long term memory (The Rivermead Behavioural Memory Test – 2, RBMT – 2) (Wilson, Cockburn, & Baddeley, 2003) and short term verbal and visuospatial memory (Randolph, 2012; Wechsler, 1997). Language, executive functions and attention were assessed with the BCoS Brain Behavior Analysis test (Humphreys, Bickerton, Samson, & Riddoch, 2012).

Everyday memory functioning awareness (anosognosia) was measured through the Visual Analogue Scale for memory impairment (VATAmem; Chapman et al., 2019). The VATAmem consists of 15 questions exploring everyday memory and 4 check questions to control reliability of participants' responses. The 15 memory-related items explore two different dimensions of memory: prospective and retrospective memory. Prospective memory questions refer to those

activities in which an individual needs to remember an intention for a future action (e.g., remembering to make a phone call later). Retrospective items examine memory for activities in which an individual needs to recall past learned information (e.g., remembering that they have already told a person a story). All items are balanced across items referring to *self versus environmentally cued* activities; that is, those in which an individual relies on internal cues to remember information (e.g., remembering appointments without the help of a calendar) versus remembering information when cued by something in the environment (e.g., remembering to give something to someone when you see them). All items are also balanced across short versus long term memory (i.e., memory for information that was just learned versus information that had been learned before). Each item is illustrated by a vignette and participants are asked to rate their current difficulty in performing each task by means of a 4-point visual-analogue scale (0= "no problem"; 3= "problem"; see Chapman et al., 2019 for example). The final overall score could range from 0 to 45.

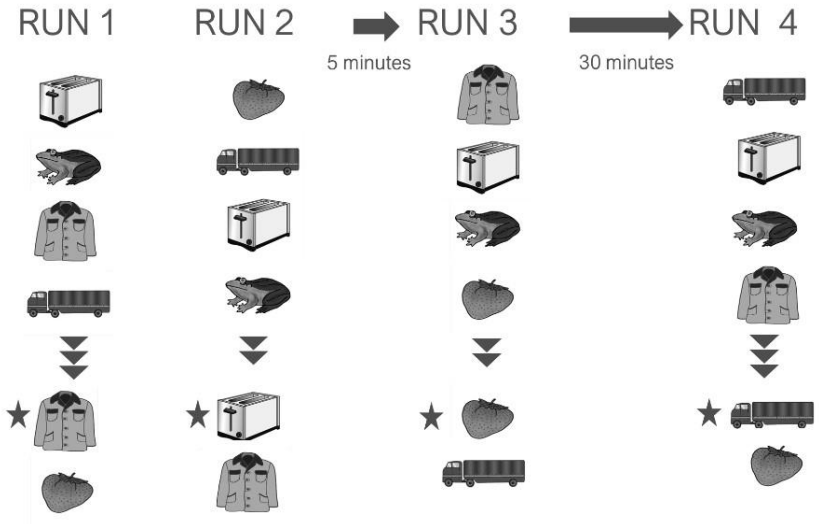
The VATAmem provides a continuous discrepancy score between informant and participant that can range from -45 (complete disagreement where the patient is underconfident of their abilities) to +45 (complete disagreement where the patient is overconfident or unaware of their difficulties). A discrepancy score of zero indicates a complete agreement between patient and informant, and therefore perfect awareness. A discrepancy score over +10.5 suggests that patients show a significant lack of awareness and this performance is considered to represent anosognosia (Chapman et al., 2019).

2.2.2. Monitoring measures

2.2.2.1.Temporal monitoring task

Temporal monitoring or reality filtering of memories was explored through a Continuous Recognition Test (CRT). Specifically, a version from Schnider and Ptak (1999) test, adapted by Cocchini et al. (2014), was used in this study. This test measures the ability to distinguish between information obtained at different moments in time, specifically which information is relevant to the 'now'. Four runs were displayed to the participant with different intervals between the runs varying from zero to 30 minutes see Figure 1). The first and second runs were presented immediately after each other, the third run after a five minute delay and the fourth run was presented 30 minutes after the third run. In every run, 80 stimuli, composed of coloured Snodgrass pictures (Rossion & Pourtois, 2004) were presented. The same stimuli were used for all runs, however, five randomly selected items randomly recurred six times in each run resulting in a possible total of 30 hits (i.e., I have seen this item before in this run) and 50 true negatives (i.e., I have not seen this item before in this run).

Figure 1. Depiction of the CRT four runs.



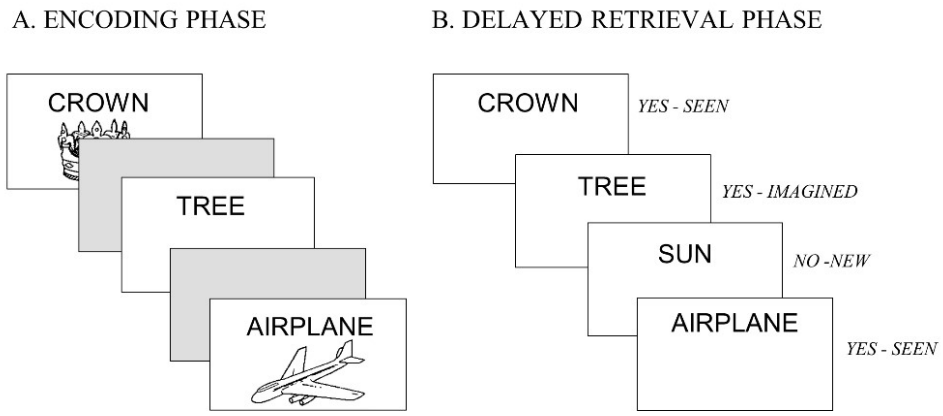
The participant must identify the item (here indicated with a *) that has already been presented within that run.

Patients were explicitly instructed to focus on the ongoing run only and to ignore stimuli presented in previous runs. Following, they were presented with the pictures one at a time and instructed to identify those that were recurrent in the ongoing run (Figure 1). All runs were preceded by four practice items with different pictures. The practice session was repeated until instruction comprehension was ensured. According to Schnider and Ptak (1999), if patients exhibit a temporal monitoring deficit, they will experience incremental false alarms across runs as they will experience difficulties discerning which item they previously saw in other runs from the ones that are currently relevant. This difficulty is interpreted as a failure to monitor the temporal relevance of newly learned information also known as a failure in reality filtering (Schnider & Ptak, 1999).

2.2.2.2. Source of memory task

An adapted and modified version of the Henkel et al. (1998) source monitoring task was used to measure the ability to distinguish between internally and externally generated memories (Cocchini et al., 2014). In the encoding phase (Figure 2A), patients are presented with 30 object words of which half were accompanied by a Snodgrass picture of the object (Snodgrass & Vanderwart, 1980). When the picture was not shown beneath the word, patients were explicitly instructed to create an image of the object in their head. Furthermore, patients were encouraged to focus on the object characteristics and their appearance by estimating how long it would take to draw the perceived or imagined object (Henkel et al., 1998). Snodgrass pictures used for this test were not used in the previous temporal monitoring task.

Figure 2. Depiction of the Source of memory task during the encoding (A) and delayed retrieval (B) phases.



A. Encoding phase: patients see or imagine (word only stimuli) common objects. B. Retrieval phase: Patients choose: i) Yes-seen (if they saw the object); ii) Yes-imagined (if they imagined the object); or iii) No-new if they neither saw nor imagined the object (i.e., new stimuli).

The encoding phase was preceded by a practice run of four words (two with and two without object pictures). After a 15-minute delay, a surprise recognition test was conducted in which patients were presented with 45 object words (Figure 2B). Of these words, 15 were new, 15 had been presented in the encoding phase with a picture and 15 had been presented in the encoding phase without a picture. Patients were then asked to indicate for each word whether it was a seen, imagined or new picture. Written instructions were provided during the recognition test listing these three options. The stimuli were presented in pseudorandom order.

2.2.2.3. Outcome measures

For both the temporal and the source of memory monitoring measures, we calculated the total Hit and False Alarm rates. To further examine how patients responded to the items, Pr (discrimination accuracy) and Br (response bias) were calculated following the two factor theory of memory which is recommended when the number of targets and distractors are uneven and to account for ceiling or floor effects (Brazzelli, Cocchini, Della Sala, & Spinnler, 1994; Corwin, 1994; Jenkinson et al., 2009). Pr represents how well the patient can distinguish between targets and distractors. Br represents the tendency with which the patient responds (i.e., more conservative or more liberal). These were calculated as follows:

Pr = (total hits + 0.5 / total targets + 1) - (total false alarms + 0.5 / total distractors +1)

Br = (total false alarms + .05 / total distractors + 1) / (1 – Pr)

Three additional scores were calculated for the source monitoring data following Jenkinson et al. (2009). A Source Proportion (SP) score was developed to capture patients' ability to monitor if the information learned was imagined (internal source) or seen (external source). To examine if there were different biases in source categories, an Externalization Bias (EB) and an Internalization Bias (IB) were also derived. These scores were calculated as follows:

SP = total correct source assignation / total hits

EB = $\sum$ erroneous external source assignation

IB = $\sum$ erroneous internal source assignation

2.2.3. Statistical analyses

Analyses were conducted in SPSS v.25.0. Descriptive analyses were conducted to examine the distribution of demographics and cognitive abilities in the sample. Fisher's exact test was conducted to examine differences in cognitive impairment across awareness status (e.g., aware vs. unaware). As all patients had impaired memory abilities, the severity of the memory impairment was compared using the Mantel-Haenszel test of trend. Two-way mixed repeated measures non parametric models were used to examine outcome measures (hits, False alarms, Pr, and Br) from the temporal monitoring task as assumptions for repeated measures mixed ANOVAs were not met (Noguchi, Gel, Brunner, & Konietschke, 2012). For these analyses, the R-package 'nparLD' using ANOVA-type statistics (ATS) was used (Brunner, Domhof, & Langer, 2002; Noguchi et al., 2012). Four separate models were run to consider hits, false alarms, Pr and Br as dependent repeated measures. Independent variables in each of these models included Group (aware versus unaware status), time (indicator of each of the 4 runs/trials of the CRT task) and an interaction between group and time. With regards to the outcome measures (Pr, Br, and source proportion) of the source of memory monitoring task, independent sample t tests and Mann Whitney U tests were conducted. Further, as parametric assumptions were violated, Spearman correlational analyses were conducted to examine if reductions in awareness were linearly associated with increased errors in source monitoring abilities. Finally, supplementary analyses were conducted in a subsample of cases matched on memory impairment scores in an attempt to examine possible bias from group differences. All supplementary analyses were non-parametric due to the limited sample size.

3. Results

3.1. Cognitive measures and anosognosia

All patients had impaired memory performance (i.e., BCoS episodic memory story immediate and delayed). Patients' performance on the RBMT-2 showed that 37% (*n* = 13) of the patients were classified as having severe memory problems, 37% (*n* = 13) as having moderate memory impairment, and 26% (*n* = 9) as having mild memory impairment.

According to the VATAmem cutoffs (see Chapman et al., 2019), 13 (37%) patients were deemed as unaware of their memory deficits, 1 patient showed severe unawareness (3%), 5 (14%) moderate and 7 (20%) mild unawareness. Impairment in cognitive measures was examined as a function of awareness to explore whether some cognitive abilities were selectively impaired, or more severely impaired, in those considered unaware versus those considered aware by the VATAmem. Results showed that anosognosic patients were more likely to be more severely impaired in memory abilities. No other differences were observed (see Table 2).

With regard to demographics and clinical outcomes in relation to anosognosia, no associations were observed in relation to age ($r = 0.20$, $p = 0.10$; $d = 0.41$) or education ($r = 0.26$, $p = 0.14$, $d = 0.54$). However, males tended to be marginally less aware of their memory deficits ($M = 7.40$, $SD = 9.98$) than females ($M = -2.00$, $SD = 17.02$) ($t = -2.03$, $p = 0.051$; $d = 0.67$). There was no significant difference between aware and unaware with regard to their lesion location ($\chi 2\ (2) = 2.38$, $p = 0.30$).

Table 2. Frequency of impaired performance in patients unaware and aware of their memory difficulties.

Cognitive performance impairment	**Aware** *n/N (%)*	**Unaware** *n/N (%)*	**Sig. Two tailed**	**Effect size** *Odds Ratio*
Long term memory – RBMT				
Mild impairment	**7/22 (32%)**	**2/13 (15%)**		2.07
Moderate impairment	**10/22 (45%)**	**3/13 (23%)**	**0.047***	1.97
Severe impairment	**5/22 (23%)**	**8/13 (62%)**		0.37
Short term memory– Verbal	2/22 (9%)	0/13 (0%)	0.39	2.36+
Short term memory – Visuospatial	5/17 (30%)	2/11 (18%)	0.42	1.62
Executive function	7/22 (32%)	7/13 (54%)	0.18	0.59
Attention	7/21 (33%)	6/11 (55%)	0.22	0.61
Language	9/21 (43%)	2/13 (15%)	0.14	2.79

*The Mantel-Haenszel test of trend showed a statistical linear association between unawareness and severity of memory impairment (χ^2 (1) = 3.94, p = .047). Significant results are bolded. +Odd ratios were calculated correcting the frequency of 0 impaired patients to 0.5 to enable and reduce bias of the ratio.

3.2. Temporal monitoring task

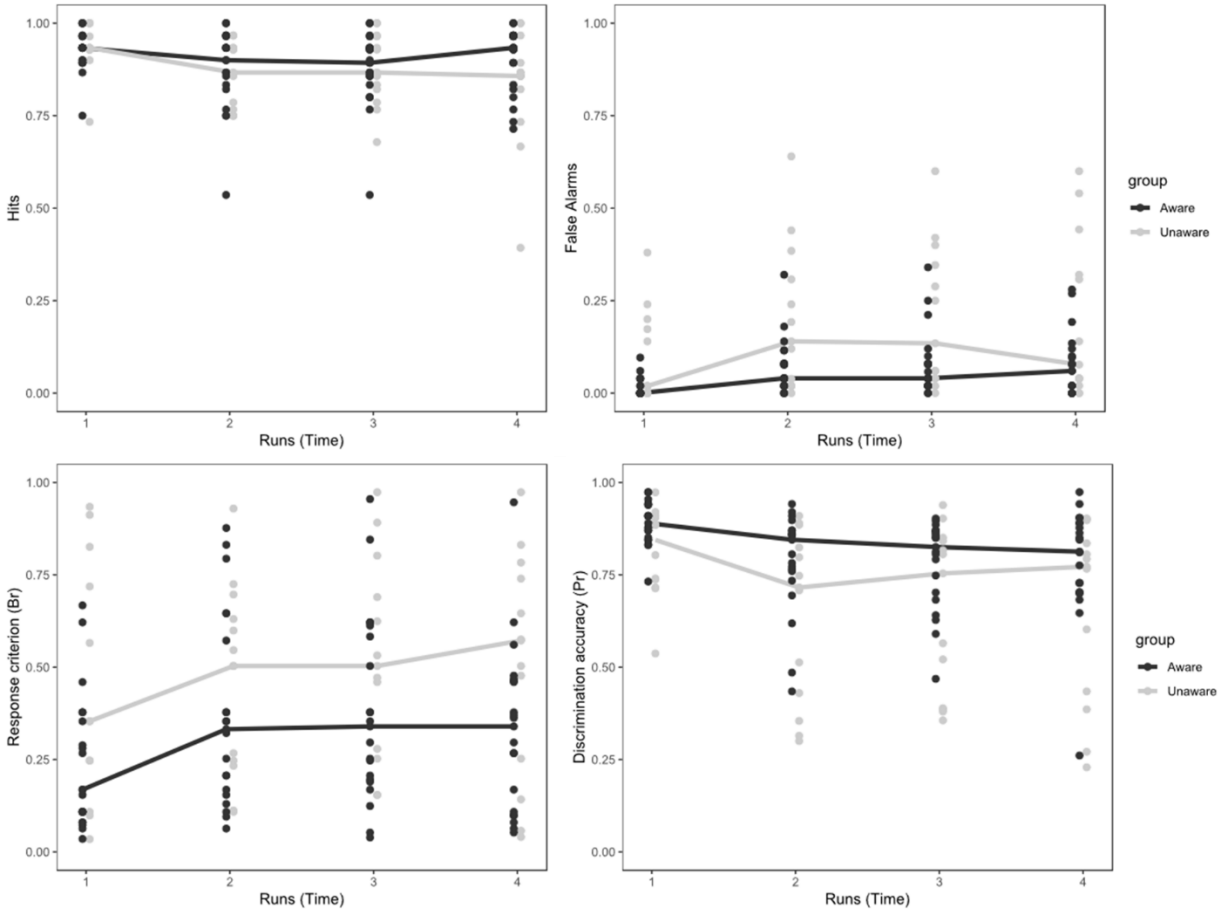
Performance on the temporal monitoring task was analyzed in relation to awareness. Total hits and false alarms, for aware and unaware cases, can be observed in Figure 3. A non-parametric equivalent of a mixed repeated ANOVA was conducted to examine differences between awareness groups and time (i.e., runs). As reported in Table 3, we found a significant effect of time (p = 0.002), but no significant differences in overall hit rates between aware (mean rank = 71.25) and unaware patients (mean rank = 64.06; p = 0.47), nor a significant interaction effect between awareness and time (p = 0.23). With regard to false alarms rates, a significant effect of time (p

<.001) was observed and a significant difference between groups, with unaware patients (mean rank = 82.26) showing increased false alarms in relation to aware patients (mean rank = 59.98; p = 0.04). There was no interaction between awareness and time (p = 0.96).

An absolute difference was also observed between patient groups in overall discrimination accuracy (Pr), with unaware patients having lower Pr (mean rank = 56.71) than aware patients Pr (mean rank = 75.80, p = 0.08; See Table 3), but this difference was not significant. Further, although a significant effect of time was observed (p < .001), there was no interaction between awareness and time (p = 0.91). Finally, with regard to response bias (Br) although unaware patients showed increased Br (mean rank = 80.81) compared to aware patients (mean rank = 60.88) this difference was not significant (p = 0.07). While there was a significant effect of time (p = 0.02) there was no significant interaction between group and time (p = 0.90) (see Figure 3).

Supplementary analyses were conducted in order to explore possible bias from group differences in long term memory performance. These analyses were run in a subsample of cases (n = 22) matched by age, education, gender and memory performance (see Supplementary section Table S2). The pattern of results remained the same across all analyses (see Supplementary analyses section).

Figure 3. Results of CRT task across aware and unaware participants.



From left top Hits, false alarms across aware and unaware groups. From left bottom to Response criterion (Br) and recognition accuracy (Pr).

Table 3. Results from non-parametric repeated mixed models of outcome measures from the temporal monitoring task (hits, false alarm, source proportion (Pr) and response bias (Br).

Temporal monitoring task	**Group Effect (aware/unaware)**			**Time effect (runs)**			**Interaction (awareness x time)**		
	F	*p*	*Effect size*	*F*	*p*	*Effect size*	*F*	*p*	*Effect size*
HIT rates	0.51	0.47	0.12	**5.12**	**0.002**	**0.55**	1.23	0.29	0.37
FA rates	**4.06**	**0.04**	**0.34**	**14.03**	**<0.001**	**1.01**	0.09	0.96	0.09
Pr	3.05	0.08	0.30	**13.77**	**<0.001**	**0.90**	0.12	0.91	0.11
Br	3.22	0.07	0.30	**3.74**	**0.02**	**0.46**	0.15	0.90	0.14

Significant results are bolded. Effect sizes can be interpreted similarly to Cohen's d.

3.3. Source of memory task

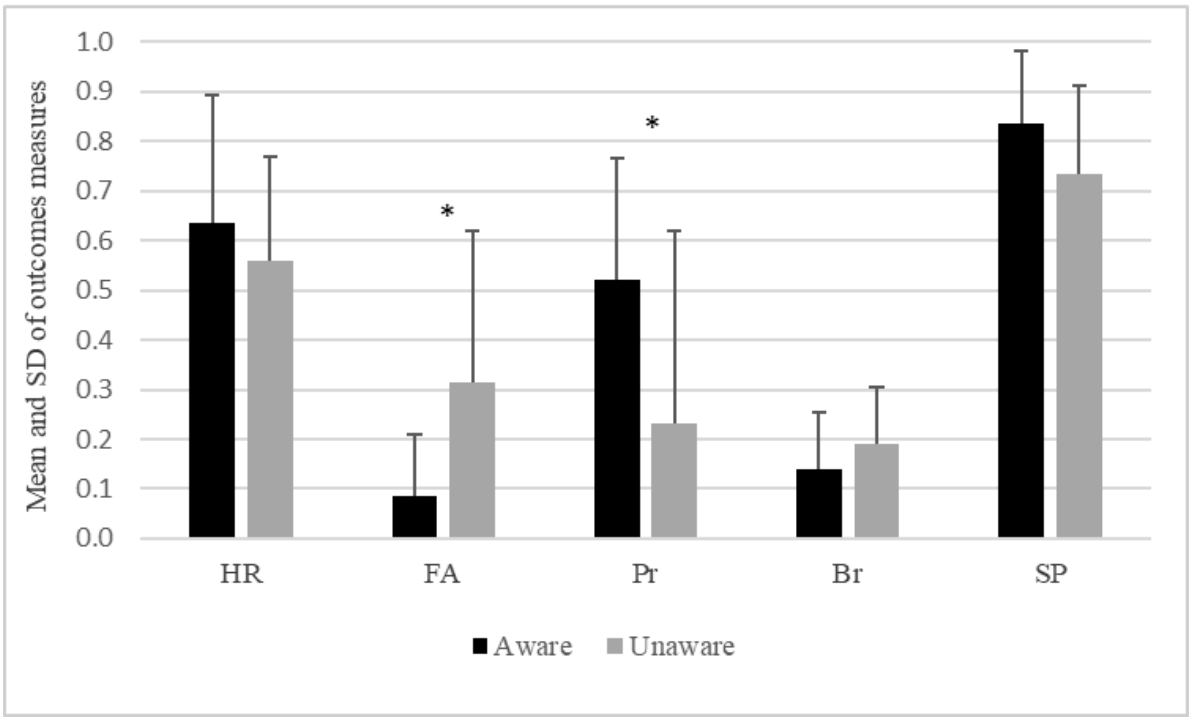
Results of the source of memory task were also analyzed in relation to awareness and are illustrated in Table 4 and figure 4. Overall hits were not significantly different between those unaware and aware of their deficits (t (33) = 0.90, p = 0.38). However, unaware patients made significantly more false alarms than the aware patients (U = 82.0, p = 0.04). Analysis of response bias (Br) revealed that those unaware of their memory deficit did not differ from those aware in their response criterion (t (33) = -1.25, p = 0.22). Finally, with regard to discrimination accuracy (Pr), unaware patients showed worse discrimination accuracy than aware patients (t (33) = 2.70, p = 0.01) (see Table 4).

Table 4. Results of outcome measures from the temporal monitoring task (hits, false alarm, source proportion (Pr) and response bias (Br).

Source monitoring task	**Group Effect (aware/unaware)**			
	*Mean (SD)/Mdn. (IQR)^* Aware	*Mean (SD)/Mdn. (IQR)^* Unaware	*Sig. Two sided p*	*Effect size Cohen's d/r^*
HIT rates	0.63 (0.26)	0.56 (0.21)	0.38	0.30
FA rates	**0.07 (0.13)^**	**0.13 (0.63)^**	**0.03**	**0.36^**
Pr	**0.52 (0.25)**	**0.23 (0.39)**	**0.01**	**0.89**
Br	0.14 (0.12)	0.19 (0.12)	0.21	0.42
Source Proportion	0.84 (0.14)	0.74 (0.18)	0.07	0.84

^ Non parametric Mann Whitney U test conducted, median and interquartile ranges (IQR) reported as descriptives and r as effect size. Significant results (p<0.05) bolded.

Figure 4. Results of source monitoring task across aware and unaware groups.



From left to right mean and standard deviations of Hit Rate (HR) False Alarms (FA), discrimination accuracy (Pr), response bias (Br) and Source Proportion (SP) in patients aware versus unaware of their memory deficits on the Source of monitoring task.

* Significant differences (p <0.05).

With regard to patients ability to monitor the source of the information (i.e., internal versus external information), there were no significant group differences in discerning internal versus external sources (t (33) = 1.87, p = 0.07) (see Table 4). Spearman correlations were then conducted to examine a potential association between the direction of a source bias (i.e., internalization or externalization bias) in relation to awareness. Results showed a significant correlation between unawareness and externalization bias (r = 0.44, p = 0.01; d = 0.98) but not internalization (r = 0.14, p = 0.42; d = 0.28).

We further explored the nature of the externalization bias and its relationship with awareness. In particular, we queried whether this bias was more likely to be triggered by imagined

information (i.e., 'imagined targets reported as seen') or by unfamiliar information (i.e., 'new targets reported as seen'). Results showed that unawareness was significantly correlated (r = 0.44, p = 0.01; d = 0.98) with externalization of imagined targets but not unfamiliar targets (r = 0.28; p = 0.11; d = 0.58).

As in the temporal monitoring task, we ran additional analyses on subsamples matching aware and unaware groups according to age, education, gender and memory performance (see Supplementary Table S1). Results did not differ from those of the original sample.

4. Discussion

This study's main goal was to assess, contextualized within the proposal of the CAM model (Agnew & Morris, 1998), the type of mnemonic monitoring failures observed in patients unaware of their memory difficulties. We examined two main monitoring processes, temporal monitoring and source monitoring (Johnson & Raye, 1981; Schnider, 2008). Findings from this study support an association between specific mnemonic monitoring failures and anosognosia for memory loss.

Thirty-four percent of patients were unaware of their memory difficulties. With regard to demographics, no association was found between factors such as age or education and unawareness of memory loss. Interestingly, males tended to overestimate their abilities, although this difference was marginally significant; supplementary analyses revealed no differences in the main findings when subjects were matched in gender. Overall, patients unaware of their memory difficulties were more likely to have impaired global cognition and memory than those aware of their difficulties. Some other studies have found similar trends (Levine et al., 1991; Vocat et al., 2010), suggesting that anosognosia may be linked to a general cognitive impairment that prevents

patients from becoming aware of a deficit, and therefore may be an epiphenomenon of other disorders (Agnew & Morris, 1998; Cocchini, Beschin, & Della Sala, 2012; Davies, Davies, & Coltheart, 2005; Levine, 1990). In the case of memory deficits, it has been posited that patients with poor memory abilities are less able to remember their mistakes and thus less likely to become (Mograbi, Brown, & Morris, 2009) or remain (Cocchini, Beschin, & Della Sala, 2002) aware. However, it is not always the case that anosognosia relates to more severe global cognitive and/or memory impairment (e.g., Derouesne et al., 1999; Migliorelli et al., 1995; Reed, Jagust, & Coulter, 1993), and it has been hypothesized that specific impairments in self-evaluative or memory monitoring processes may be the key mechanism by which awareness becomes impaired (see Cosentino, 2014). Indeed, our findings support the idea that anosognosia arises as the result of impairments to specific mnemonic monitoring failures rather than memory loss or cognitive impairment more globally.

Examination of temporal monitoring revealed no significant difference in the ability to monitor the temporal relevance of stimuli as a function of anosognosia. That is the pattern of false alarms over the course of the test trials was similar, with both groups experiencing more difficulty in Run 2 than Run 1 as expected (Figure 3), and false alarms plateauing by run 3. By run 4, false alarms *decreased* in the unaware group. As described in the methods section, patients with temporal monitoring deficits should show incremental difficulties across runs as they cannot rely on the temporal characteristics of the information learned (Schnider, 2008). This pattern of results thus appears not to support a temporal monitoring deterioration as a contributor to anosognosia, as errors should increase over time as it become more difficult to discern which stimuli are temporally relevant. Further, awareness groups showed no differences in response criterion.

It should be noted, however, that as compared to the aware group, the unaware group evidenced an overall higher number of false alarms than aware patients across all trials, potentially reflecting a deficit in more general cognitive abilities such as inhibition. Impairments in inhibition are well known to produce difficulties in suppressing proactive interference from recently learned information and thus could explain these results (Anderson, 2003; Anderson, Bjork, & Bjork, 2000; Anderson & Levy, 2007; Storm & White, 2010). Moreover, the decreased false alarms in Run 4 among unaware individuals may also reflect a more basic memory impairment (i.e., information from previous runs is forgotten and thus loses its interfering valence). The overall increased false alarms in the anosognosic group could be indicative of inhibitory deficits that could suggest executive dysfunction in these patients. Although aware and unaware participants on this study groups were not significantly different on the BCoS measure of executive function, our study did not include further executive function measures that assessed abstraction or inhibitory abilities which could have explained differences between aware and unaware participants. If an executive dysfunction was present in these patients this could lead to what is referred to as *executive anosognosia*, a type of anosognosia defined in the CAM model in which monitoring impairments reflect executive dysfunction (Agnew & Morris, 1998). Indeed, previous studies have shown the importance of the recruitment of monitoring abilities underlying executive function in the production of false memories (e.g., Ciaramelli, Ghetti Frattarelli & Ladavas, 2006). Regions known to be crucial for executive function such as the ventromedial frontal cortex have been shown to play an important role in monitoring the accuracy of memories, how memories are updated and reconstructed (Fletcher & Henson, 2001; Gilboa & Moscovitch, 2002; Moscovitch & Winocur, 2002; Rugg et al., 2002; Gilboa et al., 2009) Moreover, monitoring abilities of relevant and accurate memories are the foundation of autobiographical memory which represents memories

specific to the self and is the foundation for self-awareness (Morris & Mograbi, 2013). Future studies should apply carefully selected executive function measures in patients with variable levels of awareness to further characterize if executive dysfunction can underlie disordered awareness of memory loss.

With regard to source monitoring, results from this study support an association between anosognosia for memory loss and the ability to distinguish between internally versus externally generated information. In theory, patients with anosognosia for memory loss might have difficulties discerning *the expected* outcome for performance (i.e., internal source) from their *actual* performance (i.e. external source) on a memory task, evidencing a bias toward "externalizing" internally generated information. The pattern of results observed in the current study supports this possibility, and corroborates impairments in source monitoring that have been observed in patients suffering from anosognosia of motor deficits (see Jenkinson et al., 2009; Saj et al., 2014; Venneri & Shanks, 2004). Specifically, on the current source monitoring task, aware and unaware patients did not differ in total hits (i.e., recognizing a word that was learned), but anosognosic patients made more errors in identifying the course of the word (i.e., knowing whether it was seen or imagined). Moreover, these patients misidentified internal stimuli as external (i.e., externalization bias) with higher frequency than aware patients, similar to that shown by Jenkinson et al. (2009).

It has been suggested that in the context of psychiatric disorders, external misattributions serve to protect the ego by avoiding negative connotations associated with the self (Langdon, Corner, McLaren, Ward, & Coltheart, 2006). However, in such a scenario, one would expect a similar degree of 'externalization' for both imagined and new information; on the contrary, the externalization bias in the current study was mainly triggered by imagined targets. From a different

perspective, Garrison, Bond, Gibbard, Johnson, and Simons (2016) suggested that individuals may use a perceptual threshold to determine if a memory stems from an internal or external source (see also Bentall & Slade, 1985). In line with such a threshold theory, memories with lower perceptual richness would be identified as external whereas internally sourced memories would have higher perceptual richness. This approach would suggest that anosognosia in part reflects a deficiency in experiencing the perceptual richness that is necessary to attribute memories to an internal source which could be partly mediated by the degree of memory impairment in patients. Future studies may investigate the nature of this bias in patients with anosognosia as it may allow examination of both cognitive and motivational accounts of anosognosia for memory loss.

This study has some limitations that should be considered when interpreting results. The relatively small sample and the stroke onset variability could have influenced results of this study by limiting our power to detect more subtle differences between aware and unaware patients. Future studies should examine these mechanisms with carefully selected groups at various time points after stroke. Further, more statistical analyses were conducted for the source monitoring than for temporal monitoring assessments without statistical adjustment. Secondly, patients with severe aphasia were excluded from this study, potentially biasing our results. Third, time of stroke onset was variable across participants which may have affected their current state of awareness and thus influence the results of this study. Future studies should examine these mechanisms with carefully selected groups at various time points after stroke. Fourth, although we did not find any significant differences with regard to stroke lesions in this study, a more comprehensive examination of anatomical correlates could have provided interesting results. Indeed, Pacella et al. (2019) recently examined anatomical correlates of anosognosia for motor deficits in stroke patients through experimental voxel symptom mapping techniques such as VLSM (Pacella et al., 2019).

Their results suggested that anosognosia for motor deficits could be explained as a "tripartite disconnection syndrome" which can arise from impaired connections or damage to three main systems (i.e., premotor loop, limbic system and ventral attentional network). These results highlight the need for more refined techniques when examining anatomical correlates for anosognosia. Future research should implement these lesion mapping techniques in addition to monitoring mechanisms in patients with anosognosia for memory loss after stroke to further characterize these potential underlying mechanisms of anosognosia for memory loss. Moreover, it would be interesting to investigate whether the anosognosics' tendency to attribute stimuli internally generated to external sources may also be part of a more profound personality mechanism aiming to 'project' on external stimuli (or other persons) aspects of their own condition in addition to the cognitive monitoring failure..

To conclude, results from this study showed that patients with anosognosia for memory loss have impaired source or reality monitoring. Further, this impairment seems to be driven by an externalization bias by which internal sources are misattributed to external sources. In contrast, we found no support for a temporal monitoring deficit in patients unaware of their memory deficits. As discussed above, monitoring deficits can be contextualized within the Conscious Awareness Model (CAM) as specific to the mnemonic monitoring comparator. Results from this study suggest that deficits in this mnemonic monitoring comparator are not widespread to all types of memory monitoring (see also Bouzerda-Wahlen et al 2015), and that source monitoring may uniquely contribute to unawareness of memory loss. Future studies should examine this possible contribution in other etiologies of memory loss such as Alzheimer's disease. Further, as highlighted in the discussion more careful examination of cognition, specifically executive functions, can also help discern how these mechanisms can give rise to anosognosia for memory

loss and if they truly are a unique contributing factor to anosognosia for memory loss. Further monitoring mechanisms should be examined in relation to anosognosia to fully understand the extent to which deficits in the mnemonic comparator are unique to source monitoring.

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Supplementary analyses

Due to significant differences found across aware and unaware patient long-term memory, supplementary analyses were conducted to explore if results would remain in a subsample of participants matched on age, education and long-term memory performance. Propensity score matching led to a sample of 10 unaware patients and 12 aware patients. Neither demographics nor cognitive abilities including long term memory were significantly different across the groups (see Supplementary Table S1 and Table S2).

With regard to the temporal monitoring, results largely remained and no significant interaction between awareness status and time were observed with regard to temporal monitoring outcomes (see Supplementary Table S3). With regard to source monitoring outcomes, as in the larger sample, no differences were observed between aware and unaware (see Supplementary Table S4). Results showed a significant correlation between unawareness and externalization bias (r = .48, p = .02; d = 1.09) but not internalization (r = .15, p = .52; d = 0.30).

Supplementary Table S1. Demographics profiles of a sub-group of aware and unaware patients matched on demographic variables and memory impairment.

Cognitive profile of matched cases	**Unaware (n=10)**	**Aware (n=12)**	**p**
Demographics			
Age	68.00 (14.35)	64.83 (10.16)	.55
Education	14.10 (3.21)	14.91 (3.48)	.76
Sex (female)	3/10	5/12	.45

Supplementary Table S2. Performance on cognitive tasks by matched sub-groups.

Cognitive performance impairment	**Aware** *n/N (%)*	**Unaware** *n/N (%)*	**Sig. Two tailed**
Long term memory – RBMT			
Mild impairment	3/12 (25%)	2/10 (20%)	
Moderate impairment	6/12 (50%)	3/10 (30%)	0.37
Severe impairment	3/12 (25%)	5/10 (50%)	
Short term memory– Verbal	0/12 (0%)	0/10 (0%)	-
Short term memory – Visuospatial	2/7 (30%)	0/8 (0%)	0.20
Executive function	5/12 (42%)	4/10 (40%)	0.64
Attention	3/12 (25%)	4/10 (40%)	0.32
Language	6/12 (50%)	2/10 (20%)	0.16

Supplementary Table S3. Performance on Temporal Monitoring task in demographically and cognitively matched subgroup of patients aware and unaware of their memory difficulties.

Temporal monitoring task on matched cases	Group Effect (aware/unaware)		Time effect (runs)		Interaction (awareness x time)	
	F	*p*	*F*	*p*	*F*	*p*
HIT rates	1.43	0.23	2.54	0.06	1.51	0.21
FA rates	0.36	0.55	15.07	<0.001	0.34	0.79
Pr	0.53	0.47	9.94	<0.001	0.22	0.83
Br	0.06	0.80	5.27	0.002	0.16	0.89

Supplementary Table S4. Performance on Source of memory task task in demographically and cognitively matched subgroup of patients aware and unaware of their memory difficulties.

Source monitoring on matched cases	**Unaware (n=10)**	**Aware (n=12)**	
	Mean rank (IQR)	*Mean rank (IQR)*	*p*
Pr	9.55 (13.75)	13.12 (9.13)	0.20
Br	12.45 (9.50)	10.71 (13.25)	0.54
Source Proportion	9.65 (13.38)	13.04 (11.00)	0.23

Supplementary Table S5. Description of patients' stroke lesion.

Patient N.	Lesion description
1	Left parietal lobe infarct
2	Infarction involving cuneous of the right occipital lobe and isthmus of the cingulate gyrus.
3	Right thalamus infarct extending into the posterior limb of the right internal capsule
4	Right frontal lobe, anterior left temporal anterior left frontal lobe infarcts
5	Left basal ganglia infarct
6	Right cerebellum lobe infarct
7	Left cerebellum infarct
8	Left basal ganglia infarct extending to the corona radiata
9	Right posterior cerebral artery territory infarct
10	Mid brain, pons, superior vermis and right cerebellum; Left frontal, parietal and temporal lobe infarcts
11	Left putamen infarct
12	Basilar territory bilateral pontine infarct
13	Left cerebellar infarct
14	Posterior left frontal lobe and left parietal lobe infarct
15	Left frontal lobe infarct involving the caudate head with compression of the frontal horn of the left lateral ventricle
16	Aneurysm, subarachnoid hemorrhage
17	Aneurysm, subarachnoid hemorrhage
18	Left frontal lobe infarct
19	Right occipital lobe infarct, small old infarct in bilateral cerebellum and bilateral basal ganglia and thalamus
20	Right corpus callosum, bilateral basal ganglion, inferior cerebellum and left pons infarcts
21	Left middle cerebral branch infarction affecting the motor strip
22	Small chronic infarcts within the bilateral medial thalami on the right extending into the middle cerebral peduncle (bilateral thalamic and midbrain infarcts)
23	Aneurysm (right middle cerebral artery) subarachnoid hemorrhage
24	Right posterior corona radiata; mild cerebral volume loss and microvascular ischemic changes
25	Right midbrain, temporal and right cerebellar infarct
26	Left middle cerebral artery territory infarct
27	Left pontine infarct
28	Caudate nucleus infarct involving the left corona radiate and external capsule
29	Right middle cerebral artery territory involving the right corona radiata, frontal operculum, anterior insula, basal ganglia. Bilateral cerebellum
30	Left medial temporal lobe, left occipital and left thalamus infarct
31	Intracerebral bilateral hemorrhage
32	Right thalamic intracerebral infarct
33	Left frontal and left parietal lobes infarct including the left precentral sulcus, and a small focus in the head of the left caudate nucleus. Few foci of cortical ischemia in the lateral and anterior left frontal lobe.
34	Right frontal and parietal lobes infarct. Restricted diffusion in the underlying white matter. Old infarct in the left frontal operculum
35	Left posterior frontal and temporoparietal infarct