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# Time perception impairment following thalamic stroke: A case study

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## ABSTRACT

Impaired time perception is considered to be a relatively unusual and poorly understood consequence of brain injury. This paper presents a case study of altered time perception in JB, a 50-year-old woman who in 2011 had a small thalamic stroke affecting the right anteromedian region. We report on her subjective experience and present results from studies of retrospective timing (i.e., estimating how much time has passed and the clock time) and prospective timing (i.e., producing and reproducing intervals). The results showed that relative to neurologically healthy and brain-injured controls, JB had impaired retrospective timing and impaired prospective time reproduction. However, her prospective time production did not differ significantly from either of the control groups. We interpret this to mean that JB's essential timing functions are intact, and that rather, her time perception impairment stems from a problem in anterograde memory for time intervals. Further, we argue that unlike other cognitive domains, time perception alteration is neither anticipated nor evaluated in most patients, yet these impairments can have a remarkably serious impact on daily life. We encourage further investigation of this topic.

KEYWORDS: Thalamus, Stroke, Time

## Introduction

Thalamic strokes are relatively common. Approximately 10% of strokes are thought to affect this region (Milandre, Brosset, Botti, & Khalil, 1994). However, the cognitive consequences of thalamic stroke are poorly understood. As a result, patients may experience a range of unfamiliar symptoms such as difficulties with attention, aspects of learning and memory, language, and emotional processing that can impact significantly on their ability to participate in daily living activities. Altered perception of time is one such symptom that functionally impacts episodic and prospective memory as well as initiation and monitoring of goal directed behaviour (e.g., Hynes, Fish, & Manly, 2014). Although standardised tests exist for many cognitive symptoms associated with stroke, tools for the clinical assessment of time perception are limited.

An important part of the rehabilitation process is developing a shared understanding between the patient and the rehabilitation team of the symptoms experienced, their relationship to the injury, and their functional consequences. This understanding facilitates the design and implementation of compensatory strategies aiming to maximise social participation and facilitates emotional adjustment (Wilson, Gracey, Evans, & Bateman, 2009).

Here we present the case of JB, who suffered a right thalamic stroke and consequent significant disruption to time perception; we include her assessment, formulation and rehabilitation. We hope to raise awareness of time perception difficulties in clients with thalamic strokes and to suggest ideas for clinical assessment.

## **Thalamus**

The word thalamus derives from a Greek word meaning “meeting place” or “inner chamber” (Campbell, 2013). The thalamus sits near the centre of the brain, just above the brain stem, between the cerebral cortex and the midbrain. It is composed of nuclei organised within two lobes, joined at the midline, each roughly the size and shape of a walnut.

There are both afferent and efferent connections to and from the thalamus, respectively, often with both tracts coming from and going to the same structure. Each nucleus relates to a specific cortical area and relays messages from lower centres or from other cortical areas. All senses except olfaction travel via the thalamus to the cerebral cortex. It has hence traditionally been described as the “gateway” to the cortex. However, the thalamus is not a simple relay station; new evidence indicates that the thalamus can dynamically alter the information relayed. Murray Sherman and Guillery (2013) suggested it is better conceptualised as a gate that can be open, shut, or somewhere in between, hence more of a filter than a gate.

The literature on neural connectivity emphasises that the role of individual areas cannot be understood without an appreciation of their roles within wider networks (Bilder, 2011). Given that the thalamus is so heavily interconnected with both cortical and sub-cortical areas, the importance of understanding the thalamus and its functional connections is obvious.

## **Cognitive and sensory consequences of thalamic lesions**

Several researchers have described the cognitive and sensory consequences of damage to the thalamus. Graff-Radford, Damasio, Yamada, Eslinger, and Damasio (1985) studied a group of 25 people with thalamic infarcts and identified differential profiles of cognitive impairment dependent on the territory affected. Cases with posterolateral infarcts ( $n = 9$ ) showed sensory impairment in various modalities and only mild neuropsychological impairment (e.g., reduced visual recall and verbal fluency), and only when posterior cerebral artery infarcts had caused broader damage. Cases with anterolateral thalamic infarcts ( $n = 5$ ) showed far fewer sensory impairments and far greater cognitive impairment. Specifically, 5/5 had impaired face perception, 4/4 tested showed impaired visual memory, 4/5 were disoriented in time, and 3/5 were impaired on tests of language and verbal memory.

Cases with medial thalamic infarcts ( $n = 3$ ) had no sensory deficits and showed intact language, but were impaired on visual and verbal memory and visual perception tests; 2/3 showed temporal disorientation. Of cases with lateral thalamic and posterior internal capsule infarcts ( $n = 8$ ), all eight had a hemiparesis, and there were few sensory deficits. Cognitive deficits were, however, common. All seven tested showed impaired visual memory, 6/8 showed impaired verbal memory, 5/8 were dysarthric, 3/8 had impaired language, and 2/8 showed impaired visual perception. A more recent group study identified deficits in verbal learning, maze learning, and verbal fluency at the group level relative to controls. There was also evidence of delayed recognition of happiness and reduced ability to mentalise (Wilkos, Brown, Slawinska, & Kucharska, 2015). In addition, there is evidence of material specificity in the memory impairment of people with unilateral thalamic lesions (Carota,

Neufeld, & Calabrese, 2015), and autobiographical memory impairment has been noted in a case of bilateral paramedian thalamic infarction (Hodges & McCarthy, 1993).

Van der Werf et al. (2003) note that, although memory has received the greatest attention due to striking cases of diencephalic amnesia, thalamic damage can be associated with severe deficits in executive functioning and attention. For example, they found executive impairments in patients with lesions that included the medial dorsal nucleus, midline nuclei and/or intralaminar nuclei, and deficits in reduced complex attention following damage to the intralaminar nuclei (Van der Werf et al., 2003). Temporal disorientation has been described as a rare consequence of thalamic stroke, with an estimated prevalence of 4% within this patient group (Kumral, Gulluoglu, & Dramali, 2007).

## **Time processing**

All behaviour occurs in time, whether at the micro level of neurons and networks or at the macro level of actions, perceptions, and thoughts (Brown, 2005). Temporal phenomena are, therefore, a core element of virtually all psychological processes. This echoes William James' view that time perception is central to human experience; indeed, he devoted an entire chapter of his 1890 book, *The Principles of Psychology*, to this topic (Hancock & Block, 2012). However, the study of subjective time perception receded dramatically from then until the 1960s before making a comeback in recent years (see Block & Zakay, 2001, for a review). Significant advances have been made in understanding how the brain processes time and the development of rigorous methods of laboratory-based experiments.

## **Brain structures involved in processing time**

A range of structures has consistently been related to time processing, such as the prefrontal cortex, supplementary motor area, anterior cingulate gyrus, parietal lobes and basal ganglia (see Grondin, 2010; Harrington, Haaland, & Knight, 1998, for a review). These structures appear to underpin the perception of time but also other cognitive functions particularly attentional and executive functions (Mioni, Grondin, & Stablum, 2014). A few reports of patients with thalamic lesions have documented disorientation in time (Kumral et al., 2007; Lee, Chu, Kim, & Roh, 2010; Spiegel, Wycis, Orchinik, & Freed, 1956). In most cases "limbic thalamic nuclei", defined by reciprocal connections with limbic structures (see Schmahmann, 2003, were involved, such as the mediodorsal nucleus and the anteromedial nucleus. As the hippocampal diencephalic circuitry, often regarded as the "extended hippocampal system" (Aggleton & Brown, 1999), is critical for the formation of new memories, it is likely that the limbic thalamic nuclei have a role in forming memories that represent intervals of time. In these previous reports, disorientation in time was established on the basis of inability to estimate the exact clock time, date, season and length of time spent in a session (Kumral et al., 2007; Lee et al., 2010; Spiegel et al., 1956). However, these methods do not allow us to determine whether the time processing impairments observed were related to the process of storing time intervals or to another process within a cognitive model of time processing.

## **Cognitive model of time processing**

In the field of time perception, the dominant view is that temporal judgments are based on a single internal clock (see Grondin, 2010, for a review of this and alternative viewpoints). The Scalar Expectancy Theory (SET) (Gibbon, Church, & Meck, 1984) is probably the most influential model of time processing (Wearden, 2003). SET states that temporal judgments are underpinned by three

processing stages: the clock, memory, and decision stages. The essential feature of the clock stage of the model is a pacemaker. This emits pulses that pass through a switch and are stored in an accumulator or “cognitive counter”. The number of pulses stored in the accumulator determines the perceived length of an interval. One influential model by Zakay and Block (1995, 1996, 1997) introduced an “attentional-gate” into the SET model, between the pacemaker and the accumulator. This highlights the important role of attention in time perception, suggesting that if more attention is allocated, the gate opens wider and more pulses are sent to subsequent components. Thus, attending to time is necessary for pulses to be transmitted to the accumulator. The switch is either fully opened or fully closed. When an individual perceives a stimulus signalling the beginning of an interval, the switch is opened, the counter is set at zero, and the flow of pulses can be counted. When an individual perceives a stimulus signalling the end of an interval, the switch is closed, which prevents more pulses from being counted, at which point the count of pulses is transferred to working memory. The memory stage consists of working memory, in which the output from the accumulator is stored, and reference memory, in which the pulses accumulated on previous occasions are stored. The decision component involves comparison of the contents of working memory with standards stored in reference memory.

Block and Zakay (1997) have suggested that this model provides an accurate account of prospective timing, where participants are aware that they will be making time-related judgments, whereas somewhat different cognitive processes are involved in retrospective judgments, where no such prior warning is given. Retrospective timing likely also depends on the retrieval of contextual information associated with the event at the time of encoding and is less directly influenced by attentional factors. The contextual-change hypothesis proposes that the amount of contextual changes (e.g., setting, mood, or type of processing) stored in memory and available for retrieval determines the remembered duration of the time period (Zakay & Block, 1997). Disturbance of any stage of the SET model may disrupt temporal processing and may do so in different ways.

### **Time processing in clinical neuropsychology**

The models described previously predict that time processing may be disrupted by brain injury in numerous ways, depending on the structure(s) and the cognitive function(s) affected. This could include direct disruption at e.g., the pacemaker/accumulator level and indirect disruption as a result of impairments of the related cognitive functions of attention, working memory, reference memory, etc. However, we know relatively little about the nature and specificity of time processing deficits after brain injury.

Many methods are available to formally assess time processing abilities. The most widely used method of retrospective timing is orientation to time, which is generally considered to be underpinned by episodic memory processes (Grondin, 2010). This can be assessed by asking the participant the day of the week, date, month, and year. It is important to note, however, that some patients may have an accurate sense of time, yet be unable to remember the exact date. Indeed, Wilson, Cockburn, Baddeley, and Hiorns (1989) found that only 88% of neurologically healthy controls were able to report the date accurately. Conversely, some patients in structured rehabilitation settings may pass tests of orientation but not have a functioning sense of time (Brown, 1990). One method that overcomes some of these limitations is asking participants how long they have been in the testing room during assessment (McFie, 1960).

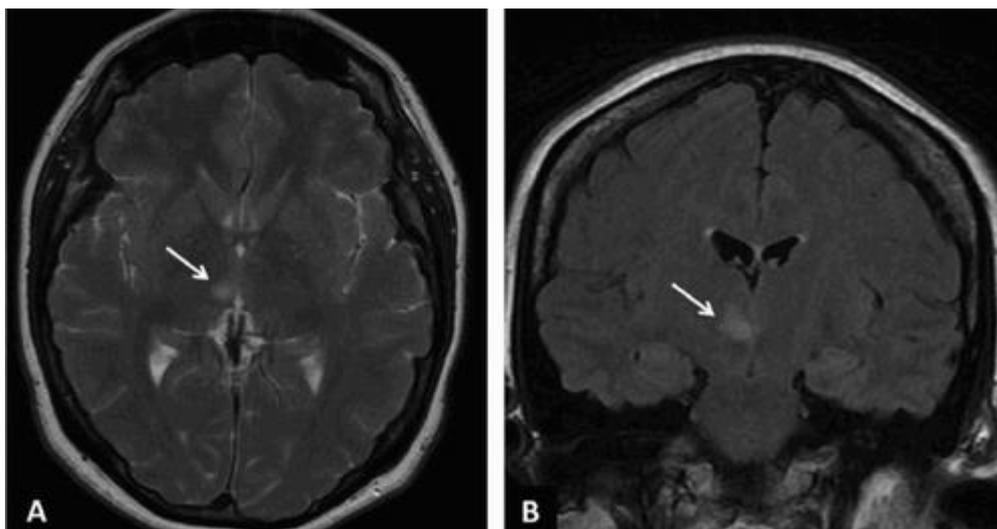
The majority of work in experimental psychology and psychophysics on time processing has employed methods of prospective timing. Typically, the time intervals used in these tasks is much shorter than those in retrospective timing tasks, often a number of seconds, and the majority of the literature on the cognitive processing of time is based on these methods. A variety of prospective

timing paradigms has been used (see Grondin, 2010 for a review) but none of these tests have been standardised for clinical use.

## Case report

JB was a physically active, high functioning 50-year-old woman who sustained a small anteromedian<sup>1</sup>. The terminology used varies according to which classification system is used. Using Carrera et al.'s (2004) classification, the lesion is localised as "anteromedian", whereas using Schmammann's (2003) classification, it is "paramedian". View all notes right thalamic infarct two years prior to enrolment in a holistic neuropsychological rehabilitation programme. A consultant neuroradiologist reviewed the MRI scans taken in August 2011, shown here in Figure 1. The infarct initially left her with mild left sided weakness, left upper quadrant visual field loss and altered sensation in the left side of her body. When assessed for the programme, she noted difficulty with multi-tasking ("I always did fifteen things at once, juggled things – this is the one that makes me feel the most useless"), poor learning and memory, trouble processing information, and difficulties with initiating and carrying through activities, as well as poor problem-solving ("I will wind myself into a frenzy ... I'm not so good at thinking in an abstract or structured way"). She reported being confused in unfamiliar places, and although she could drive, she noted difficulty in finding her way to new destinations. In addition, she reported impaired time perception, which left her without temporal anchors and unable to anticipate events ("I have no idea when Christmas will be here"), to sense where in the day she was (not knowing whether to eat breakfast or dinner), or to decide when to stop an activity (once, her husband had to stop her from raking leaves after three hours), among many other problems. As a consequence, she experienced significant fatigue and poor sleep, her mood was low, she no longer looked forward to anything, and she found the future to be frustrating and exhausting. Her inability to perform previous roles as mother, wife and worker to her own high standards took a big toll on her sense of self-worth and her identity. Her distress was complicated by her sense of feeling misunderstood by others.

Figure 1. JB's magnetic resonance imaging scan.



## Neuropsychological assessment

Neuropsychological assessments were conducted while JB attended the Oliver Zangwill Centre for Neuropsychological Rehabilitation. Relevant results are presented in Table 1.

Table 1. JB's performance on standardised neuropsychological tests

	Score	Scaled score
Optimum functioning		
WTAR	48	Standard score = 117
General		
WAIS-IV Matrix Reasoning	22	14
WAIS-IV Block Design	45	11
WAIS-IV Vocabulary	52	14
WAIS-IV Similarities	29	11
Verbal memory		
RBMT-3 Story – Immediate Recall	6	7
RBMT-3 Story – Delayed Recall	5	7
RBMT-3 First and Second Names – Delayed Recall	2	5
RMT Words	22	<2
Non-verbal memory		
RBMT-3 Picture Recognition – Delayed Recognition	6	1
RBMT-3 Face Recognition – Delayed Recognition	8	4
RMT Faces	23	<2
Learning		
RBMT-3 Novel Task – Immediate Recall	20	4
RBMT-3 Novel Task – Delayed Recall	8	5
Prospective memory		
RBMT-3 Messages – Immediate Recall	2	3
RBMT-3 Messages – Delayed Recall	2	2
RBMT-3 Belongings – Delayed Recall	2	3
RBMT-3 Appointments – Delayed Recall	2	7
Working memory		
WMS-III Digit Span forwards	6	–
WMS-III Digit Span backwards	4	–
WMS-III Digit Span total	10	5
WMS-III Spatial Span forwards	4	4
WMS-III Spatial Span backwards	5	8
WMS-III Spatial Span total	9	4
RBMT-3 Route – Immediate Recall	8	5
Speed of processing		
D-KEFS TMT Number Sequencing	78 <sup>r</sup>	1
D-KEFS TMT Letter Sequencing	90 <sup>r</sup>	1
D-KEFS TMT Motor Speed	92 <sup>r</sup>	1
Attention		
TEA Map Search (number circled in one minute)	10	<1
TEA Map Search (number circled in two minutes)	20	2
TEA Elevator Counting	7	Normal
TEA Elevator Counting with Distraction	2	5
TEA Visual Elevator (Accuracy)	7	8
TEA Visual Elevator (Timing)	11.9	<1
D-KEFS TMT Visual Scanning (none missed just slow)	39 <sup>r</sup>	3
Executive functioning		
D-KEFS TMT Number-Letter Switching speed	203 <sup>r</sup>	1
D-KEFS TMT Number-Letter Switching set loss errors	3	3rd percentile
BADS Key Search (lost marks for speed + minor others)	13	Profile score: 2
BADS Zoo Map (lost marks for speed + minor others)	15	Profile score: 2

Bold indicates performance within the impaired or borderline range, relative to published norms. WTAR= Wechsler Test of Adult Reading (Holdnack, 2001), WAIS-IV= Wechsler Adult Intelligence Scale (Wechsler, 2008), RBMT-3= Rivermead Behavioural Memory Test – Third Edition (Wilson et al., 2008), RMT= Recognition Memory Test (Warrington, 1984), WMS-III = Wechsler Memory Scale – 3rd Edition (Wechsler, 1997), TEA= Test of Everyday Attention (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994), DKEFS= Delis-Kaplan Executive Function System (Delis, Kaplan, & Kramer, 2001), BADS= Behavioural Assessment of the Dysexecutive Syndrome (Wilson et al., 1996).

Table 1. JB's performance on standardised neuropsychological tests.

JB's premorbid level of intellectual functioning was estimated to be in the high average range based on her high level of academic and vocational achievement (she achieved straight A-grades, undergraduate and postgraduate university degrees, and ran a successful business) as well as performance on the Wechsler Test of Adult Reading (Holdnack, 2001). On tests of verbal comprehension and perceptual reasoning, her scores fell across the average and superior ranges. However, her scores were lower than expected on tests of attention, verbal and non-verbal memory

and prospective memory, falling across the impaired and borderline ranges. Her performance was within the borderline range on tests of learning and verbal and visuospatial working memory and was within the impaired range on measures of processing speed. On tests of executive functioning her performance was severely affected by her slowed speed of processing. Although she made minor errors on subtests of the *Behavioural Assessment of the Dysexecutive Syndrome* (Wilson, Alderman, Burgess, Emslie, & Evans, 1996), she was also penalised for spending too long on individual items. However, she appeared to also have some executive difficulties that cannot be attributed to speed or temporal disorientation. On a test of switching between sequencing numbers and letters she made a number of errors, resulting in the accuracy of her performance falling within the impaired range.

## **Assessment of JB's time processing abilities**

### **Experiment 1: Retrospective timing**

When tested during her rehabilitation, JB was consistently disorientated to the exact date, season, clock time, and time spent in session. For example, she estimated that she had been in a session for five minutes on three separate occasions, when in fact it had been 45, 30, and 20 minutes. These sessions were unstructured sessions, including a clinical interview, group session and home visit, respectively. We soon learned that "5 minutes" was JB's default response. To investigate whether her estimates were indeed abnormal, we conducted a formal test.

#### **Method**

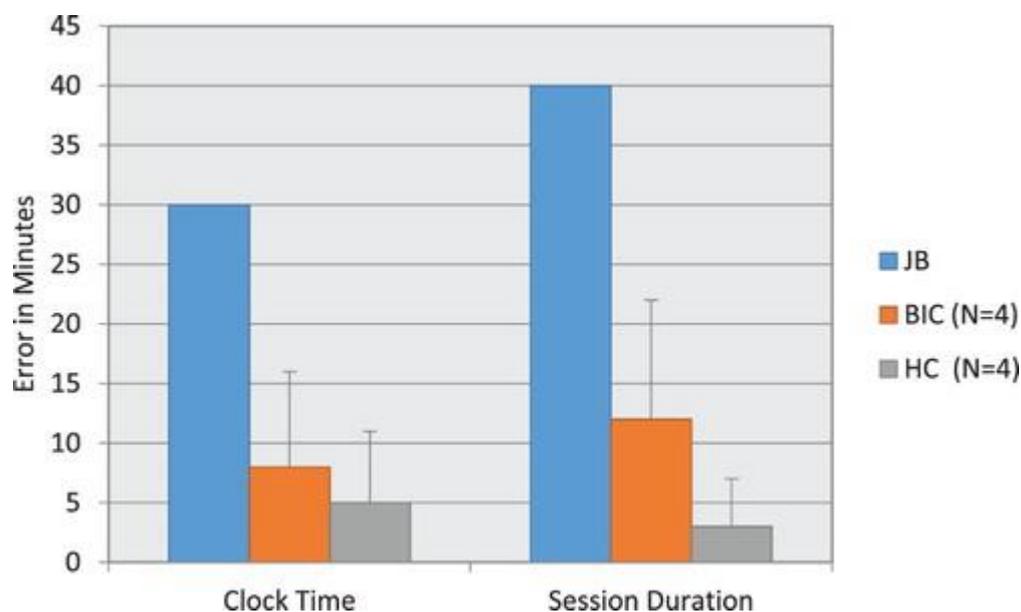
JB and two small control groups were administered tests of retrospective timing, requiring them to estimate both session duration ("how long has this session lasted?") and clock time ("what is the exact time?"). Because the structure of events that fill an interval is known to influence estimations of duration (Boltz, 2005), these tests were administered to JB and all control participants between two tests of prospective timing (Experiment 2). The dependent variable was the total error of estimations from target time (irrespective of under or overestimations). One control group consisted of four neurologically healthy women of a similar age and background to JB. To investigate whether JB's performance was simply a result of brain injury, rather than injury to the thalamus in particular, she was compared to another small control group of four women who had acquired a brain injury and who were of a similar age and background. In this group, the aetiology of each participant's brain injury was subarachnoid haemorrhage, herpes simplex viral encephalitis, traumatic brain injury, and thalamic stroke.

Comparisons were made using the computer programme SINGLIMS (Crawford & Garthwaite, 2002).

#### **Results**

Results are presented in Figure 2. JB's ability to estimate the clock time was significantly worse than that of healthy participants (SINGLIMS  $< .05$ ,  $Z_{CC} = 4.17$ ) and those with brain injury (SINGLIMS  $< .05$ ,  $Z_{CC} = 2.75$ ). Her estimation of the duration of a session was also significantly less accurate than brain injured (SINGLIMS  $< .05$ ,  $Z_{CC} = 2.80$ ) and healthy controls (SINGLIMS  $< .05$ ,  $Z_{CC} = 9.25$ ).

Figure 2. JB's ability to estimate clock time and session duration compared with that of control subjects with and without a brain injury. BIC = brain injury controls, HC = healthy controls. Error bars represent one standard deviation.



#### Comment

When tested formally, JB appeared to have significant difficulties estimating the duration of the session and clock time. After being in the session for 45 minutes, she estimated that she had been there for five minutes and estimated the clock time as if she had only been there for 15 minutes. These findings are consistent with previous reports of disorientation in time following thalamic stroke, which have also been established on the basis of judgments of retrospective timing (Kumral et al., 2007; Lee et al., 2010; Spiegel et al., 1956).

#### Experiment 2: Prospective timing

Previous reports of patients disorientated in time following thalamic lesions are an important indicator that the thalamus is involved in time processing in general, but not in any component/components of the time processing system in particular. As the limbic thalamic nuclei are involved in the formation of new memories, it seems likely that the anterior thalamus contributes towards encoding of time intervals.

#### Method

JB was assessed on two tests of prospective timing. The first required participants to reproduce an interval of time demonstrated by an experimenter and so was dependent on the ability to make a comparison with a representation of the target interval stored in reference memory. The second task required participants to produce an interval that had been specified verbally, which involves relating clock time to subjective time and is therefore less reliant on episodic memory (Shaw & Aggleton, 1994). Thus, although both tasks are reliant on comparing the current interval to a standard stored in reference memory, the former is more reliant on anterograde memory, as an estimated time interval has to be encoded, stored in short-term memory, and compared with stored

“samples” retrieved from reference memory. JB’s performance was compared to that of the same two groups of control participants used in the retrospective timing experiment, who underwent the same procedure.

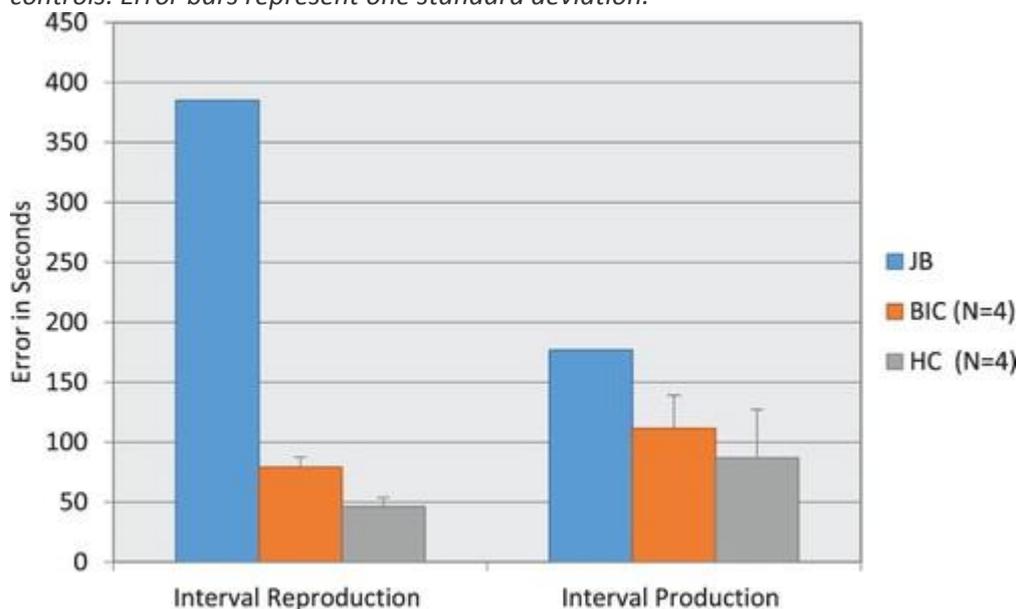
In an interval reproduction task, the experimenter first demonstrated an interval by starting the stopwatch and stopping it again after a set interval. The same stopwatch was then given to the participants and reversed so that the screen could not be seen. Participants were then asked to replicate the interval and then return the stopwatch to the experimenter to view the screen. The intervals of 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, and 60 seconds were used, so that there were 12 trials in total. The order in which these intervals were presented was randomised and presented to all participants in the same fixed order. To prevent participants from counting, all intervals were filled by asking participants to read stories aloud. In each new interval, a new story was presented, which differed in font type and size, so that the number of lines could not be used to estimate interval length. Participants were monitored to ensure that they were not counting by other means, such as by tapping their feet.

All participants were then administered an interval production task. This was the same as the interval reproduction task except the target interval was not demonstrated by the experimenter but was instead given verbally, e.g., “45 seconds”. In this task the same intervals were used but presented in a different fixed randomised order. Different stories were used to fill intervals. In both tasks, the dependent variable was the total error of estimations from target intervals, irrespective of under or overestimations, measured in seconds

## Results

As can be seen in Figure 3, JB had considerable difficulty with the interval reproduction task. On this task her performance was found to be significantly less accurate than that of healthy (SINGLIMS  $< .05$ ,  $Z_{CC} = 43.88$ ) and brain injured (SINGLIMS  $p < .05$ ,  $Z_{CC} = 36.84$ ) controls. However, on the interval production task, her performance did not significantly differ to that of healthy (SINGLIMS  $> .05$ ,  $Z_{CC} = 2.23$ ) and brain injured controls (SINGLIMS  $> .05$ ,  $Z_{CC} = 2.36$ ).

Figure 3. JB’s performance on interval reproduction and interval production tasks, compared with that of control subjects with and without a brain injury. BIC = brain injury controls, HC = healthy controls. Error bars represent one standard deviation.



On the interval production task, the length of the interval to be produced showed a significant positive correlation with estimation error in the healthy control group,  $r_s = .390, p < .01$ , the brain injured control group,  $r_s = .517, p < .001$ , and JB,  $r_s = .683, p < .01$ . On the interval reproduction task, however, the length of the interval demonstrated by the experimenter was correlated with estimation error in the healthy,  $r_s = .582, p < .001$ , and brain injured,  $r_s = .707, p < .001$ , control groups but not JB,  $r_s = .210, p = .26$ .

#### Comment

As JB was found to have great difficulty with the interval reproduction task but not with the interval production task, it is unlikely that her performance can be attributed to the clock mechanism, as any alterations at this level would affect both tasks. As the interval reproduction task requires time intervals to be encoded, stored in short-term memory, and compared with "samples" retrieved from reference memory (Shaw & Aggleton, 1994), it seems likely that JB's difficulties can be attributed to the memory stage in the theoretical model.

#### Discussion

##### Summary and interpretation of findings

We have presented results from two time perception experiments comparing case JB with neurologically healthy and brain-injured control groups. Experiment 1 demonstrated that JB had impaired retrospective timing for the actual time and for durations experienced. Experiment 2 showed that JB had impaired prospective timing for an interval reproduction task, but intact prospective timing for an interval production task. As her performance on interval production was close to that of healthy controls, this showed that JB does not have an absolute and generalised impairment of time perception; rather, there is a more specific deficit. We think that this dissociation is evidence that the clock mechanisms are intact, and that her impairment cannot be attributed to a disconnection between the clock mechanism and other stages in the SET model, as this would impair her performance on both interval reproduction and production tasks. Thus, her impairment must be related to a different stage in the model.

This raises the question of which process(es) are actually deficient. Both production and reproduction tasks require participants to respond by generating an interval, which requires intact functioning of the switch, accumulator and, in Zakay and Block's (1995, 1996, 1997) model, attentional gate. Thus, her impairment is unlikely to be located at any of these stages. Given the prospective time reproduction and retrospective estimation impairments, we think it likely that it is JB's *anterograde memory for time intervals* that is affected. Although both interval production and reproduction tasks require comparison with some standard stored in reference memory, the former involves "translating" clock time to subjective time (Shaw & Aggleton, 1994), whereas the latter requires participants to make a comparison with the stored representation of the sample interval which must be encoded, stored and retrieved from reference memory (Block & Zakay, 2006). Therefore, an impairment in anterograde memory for time intervals would account for the difference in performance between the two tasks, as well as her performance estimating both clock time and session duration. This would be the case regardless of whether the SET (Gibbon et al., 1984) or the attentional-gate (Zakay & Block, 1995, 1996, 1997) model is used as the theoretical framework, given that the stage at which they differ does not appear to be the locus of JB's impairment.

In support of this, although both control groups showed greater estimation error on trials where the interval to be produced or reproduced was longer, JB's estimation error was only significantly correlated with interval length in the interval production task. We interpret this to be consistent with our interpretation that JB has an impairment affecting her anterograde memory for time intervals, as the length of the interval that she produced in the interval reproduction task was unrelated to the length of the interval that was demonstrated, suggesting that the demonstrated interval had not been encoded, stored or retrieved successfully.

### **Relation to previous literature**

In addition to the impairment of time perception, JB's abilities in the domains of attention, executive function, and memory were reduced relative to the expected premorbid level. In the study by Graff-Radford et al. (1985), participants with anterolateral and medial thalamic lesions showed few sensory impairments, but pronounced cognitive deficits. JB's visual field and tactile sensation in the left side of her body were affected by the stroke and she also reported failing to notice internal sensations including hunger, thirst, pain, and fatigue until very high levels were reached. However, her significant difficulty with self-monitoring may have also played a part in her failure to recognise sensory experiences. We therefore assume she has a sensory processing dysfunction as a consequence of interacting cognitive and sensory impairments. This is consistent with the literature.

The attentional gate model of time processing informed the experiments reported here, and the studies in the existing literature were used to develop the experimental procedures and to understand the interactions between "clock" functions and other cognitive functions such as attention and memory. The dissociation we observed between performance on the interval reproduction and production tasks informs our understanding of JB's impairment, in the sense that the impairment seems to stem from the memory stage of the SET model.

As discussed, the cognitive processes involved in the perception of time appear to be underpinned by brain areas involved in other cognitive functions. For example, in a review of the literature on traumatic brain injury, Mioni et al. (2014) argued that the stage at which patients were impaired within the SET model was related to their more general cognitive impairments e.g., attention. It may be that there is a pattern between the stage at which time processing is impaired and the degree to which other cognitive abilities are affected following thalamic lesions; as different thalamic nuclei are thought to be associated with different cognitive functions (Schmahmann, 2003). That is, it may be that lesions to different thalamic nuclei may result in impairments in different temporal processes. The current case supports this to some extent, as JB's lesion was to the limbic thalamus, thought to be critical for the formation of new memories. JB showed difficulties that appeared to affect her anterograde memory for time intervals, in the context of general impairments in anterograde memory. However, it is important to note that she also had impairments in other cognitive domains, so the relationship between her time perception impairment and other cognitive difficulties is not entirely straightforward.

### **Suggestions for further research/implications**

Given that disruption of time processing by brain injury can have striking consequences, affecting a variety of basic everyday activities such as crossing a road safely, planning an activity, and monitoring task performance, we consider it important that clinical neuropsychological tools are developed that enable a greater understanding of time processing after brain injury and hence inform rehabilitation strategies. We also think that it will be important to assess perception of longer time periods, as impairments of this ability have consequences for thinking about the future and

anticipating risks and rewards and also impact emotional experience including mood, anxiety, and frustration.

Where rehabilitation for time processing has been discussed in the literature, it has typically been considered in the context of a more general state of disorientation e.g., to person, place, time, and circumstance, immediately following trauma to the brain (Sohlberg & Mateer, 2001). To our knowledge, only two attempts have been made to retrain altered time processing following brain injury. Covre, Ford, Bueno, and Bateman (2010) and Hynes et al. (2014) both attempted to train AD, a 45-year-old male with impaired time perception following an extensive right middle cerebral artery territory infarct. to estimate time intervals. Although training improved his time production performance, improvements did not generalise to daily life. However, anecdotal evidence reported by Covre et al. (2010) suggested that AD benefitted from compensatory strategies. For example, he timed himself completing several daily tasks and was able to refer back to this record to help him to decide whether he had enough time to do tasks. A similar approach is used by JB; she compensated for her altered time perception by drawing on her preserved intellectual abilities to plan her time, for example, by programming an oven timer to alert her to when food is ready. Thus, there is very little research into methods of rehabilitation for altered time perception but anecdotal accounts suggest that compensatory strategies may be a promising avenue for intervention.

### **Limitations**

Although neuropsychological findings are useful, it is important to note the limitations of such evidence. For example, Liebermann, Ploner, Kraft, Kopp, and Ostendorf (2013) note that thalamic lesions rarely leave extrathalamic brain regions unscathed (Carrera, Michel, & Bogousslavsky, 2004). Furthermore, as the thalamic nuclei are small, clinical imaging protocols are often insufficient to localise damage, and thalamic strokes typically affect several thalamic nuclei simultaneously because of their vascular supply (Percheron, 1973; Schmahmann, 2003). JB appears to have a small lesion, with sparing of other brain regions. However, it remains difficult to entirely localise the lesion, and to establish its impact at the network level.

We included two relatively small control groups in this study. However, this is not unusual as Crawford and Howell (1998) note that the sample size of control groups for single-case studies in neuropsychology is frequently less than five. They argue that this will often be the case when the theoretical questions posed cannot be addressed using existing instruments. This is particularly pertinent in the domain of time processing, for which no standardised testing batteries exist.

We also need to recognise that though these results are informative, they do not address the full extent of JB's time perception difficulties or the psychological consequences of these for her. These experiments formed only a part of her holistic neuropsychological rehabilitation programme.

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### **Disclosure statement**

No potential conflict of interest was reported by the authors.

## Notes

1. The terminology used varies according to which classification system is used. Using Carrera et al.'s (2004) classification, the lesion is localised as "anteromedian", whereas using Schmahmann's (2003) classification, it is "paramedian".

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