

## **Marcel Proust: Nascent Neurology of Memory and Time in *À la recherche du temps perdu***

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Marcel Proust (1871-1922) met a number of neurologists in connection with his treatment for asthma. And his great work, *À la recherche du temps perdu*, published 1913-1927, and first translated into English by C.K. Scott Moncrief, 1922-1931 as *Remembrance of things past* – later changed to *In search of lost time*, is widely regarded as one of the great novels of the 20<sup>th</sup> century. Aside from its literary value, the work is also important to the history of neurology because of its author's views of memory and time.

Key words: Marcel Proust, Joseph Babinski; *À la recherche du temps perdu*; *In Search of Lost Time*; neurology of memory, neurology of time

### **Prologue**

A monograph published in 2009 (Oxford University Press), *Joseph Babinski: A Biography*, comments on Marcel Proust.<sup>1</sup> And readers may wonder why a biography of Joseph Babinski (1857-1932) should include such a reference. However, the neurologist and novelist were acquainted and their connection can be related to the early history of the neurology of memory and time.

Proust is first mentioned in Chapter 1 of the Babinski biography: “In French literature, the greatest name of this period was certainly Marcel Proust (1871-1922), who wrote at the end of the nineteenth century and the first twenty years of the twentieth, the last parts of his most famous novel, *À la recherche du temps perdu* (Remembrance of things past) being published after his death in 1925. His chronic illnesses were responsible for frequent visits to doctors, particularly neurologists, one of whom was Babinski.” He is further mentioned in Chapter 8, describing Babinski when he was Head of a Department at La Pitié.

Babinski first treated the novelist's mother for hemiplegia with aphasia. And helped the novelist himself, tormented by a speech disturbance, by ruling out the possibility of aphasia in convincing him of his ability to pronounce complex words and phrases such as *constantinopolitain* and *artilleur de l'artillerie*. He also advised Proust to stop

taking sleeping pills. Babinski was also called in by Proust's younger brother to attend the novelist when he was dying.

Aside from Babinski, Proust also met other neurologists including Édouard Brissaud (1852-1909), who co-founded the journal *Revue Neurologique* in 1893, Jules Dejerine (1849-1917), Paul Sollier (1861-1933) and Jules Cotard (1840-1889).

The medical link was asthma and his wider family.

## **I. Proust's family and relations (Fig. 1)<sup>2-4</sup>**

Marcel Proust belonged to a family of four, of which two were physicians (his father and younger brother). His father, Adrien Proust (1834-1903), celebrated for his treatment of cholera, and later a Professor of Hygiene at the University of Paris, was acquainted with Jean-Martin Charcot (1825-1893), the founder of modern neurology. Adrien Proust wrote on cerebral infarction aphasia and asthma (his son's affliction) and contributed to the establishment of the International Office of Public Hygiene – later the World Health Organization (WHO). It was Marcel Proust's younger brother by two years, Robert Proust (1873-1935), a well-known urologist, who introduced him to Babinski.

Marcel Proust is said to have been closer to his mother than his father, and her aphasia and hemiplegia before death, greatly alarmed him. He was also related through his cousin to Henri Bergson (1859-1941) whose works, in addition to the medical library of his father, were freely available to him.

## **II. Proust's illnesses<sup>2-4</sup> and health problems**

Proust was a lifelong sufferer with asthma, starting at the age of nine and sleep disturbance. He also abused medications.

Figure 2 shows two snapshots retrieved from the internet using recently discovered footage of Proust (<https://youtu.be/51COHIgjbYU>).<sup>4</sup> Although these images are unclear, Bogousslavsky suggests that Proust's physique, with thoracic enlargement, suggests chronic respiratory disorder.<sup>2</sup>

In the latter half of the nineteenth century, the Parisian medical establishment regarded asthma as a neurological disease: a neurasthenic phenotype: a concept covered by psychosomatic disorders in modern terms. Furthermore, asthma has similarities with epilepsy because of its paroxysmal onset and possible, continuous attacks. Given our current interest in intestinal flora in association with Parkinson's disease and multiple sclerosis, plus diversity in the etiology of migraine (associated with the neural, vascular, and immune systems), asthma as it was seen at the time may not have been viewed amiss. Proust also owed his acquaintance with prominent neurologists through his family members and to the notion that his illness was a nervous one. These connections informed the writing of his masterpiece.

## **III. *In Search of Lost Time***

Marcel Proust's magnum opus, a masterpiece of French literature, has a number of

Japanese translations. And 2021, marking the 150th anniversary of his birth, has seen numerous related events and publications. In Japan, at least two books have been published on how to appreciate the novel<sup>3,4</sup>. The publication of such books of commentary indicate its intricacies and great length: in Japanese it runs to seven volumes and approximately 10,000 pages. It also has about 500 characters.

Moreover, a large number of reviews and articles have been published worldwide, some of which point to Proust's medical knowledge, particularly of neurology, in association with his own and his family's health problems.<sup>2,5</sup> Bogousslavsky draws attention to Proust's detailed knowledge of the neurological mechanism of memory and to his strong interest in time. In his novel, Proust is reported to have used memory-related terms (such as: remembering, forgetting, and recall) 1,210 times. And memory is referred to every 2.6 pages.<sup>5</sup> Yoshikawa outlines Proust's linkage of time and memory as follows.<sup>3</sup>

“Because I had had what is called ‘the time’ to forget them, by its fragmentary, irregular interpolation in my memory ... confused, destroyed my sense of distances in time, contracted in one place, extended in another.” Time is also a function of memory and forgetting (p116).

The so-called Proust effect (olfactory episodic memory) referred to in neurological studies,<sup>6</sup> is named after the following passages in the novel<sup>7</sup>:

I raised to my lips a spoonful of the tea in which I had soaked a morsel of the cake [madeleine]. No sooner had the warm liquid, and the crumbs with it, touched my palate than a shudder ran through my whole body, and I stopped, intent upon the extraordinary changes that were taking place.... And suddenly the memory returns.... The sight of the little madeleine had recalled nothing to my mind before I tasted it (pp.116-120).

This is an exquisite, literary account of the potential of olfaction (tea) and gustation (madeleine) to stimulate involuntary, emotional memory.

#### **IV. Neurology of memory**

During Proust's life, how was amnesia diagnosed and memory understood?

Sollier, who is thought to have shared the idea of involuntary memories with Proust, published two books on amnesia (Fig. 3). That published in 1892, written we believe under the direction of his teacher, Charcot, thus suggests that it shadows Charcot's notion of amnesia.<sup>8</sup> It clearly differentiates between anterograde and retrograde amnesia. And a revised edition of 1901 indicates its popular success. The second book, published in 1900,<sup>9</sup> contained diagrams associating involuntary memories with emotion and emotional factors.<sup>5</sup> Furthermore, Sollier also published a book on emotions.<sup>10</sup> Bogousslavsky *et al.* regard Sollier as the first clinical neuropsychologist<sup>5</sup> because his interests combined neurology and psychiatry. He was also concerned with memory, mental retardation, emotion and hysteria, and designed an assessment along the lines of an IQ test.

Proust spent six weeks in Sollier's sanatorium (1905-1906) while writing his novel,

and his potential influence on him is the subject of two studies.<sup>2,5</sup>

However, Sollier's pioneering role in the neuroscience of cognition and action involving memory, receives scant attention – partly because his works and materials were destroyed by fire in a bombing raid during the Second World War (they were stored in a hospital close to a Renault car factory).

<https://docplayer.net/10117291-Marcel-proust-and-paul-sollier-the-involuntary-memory-connection.html>

The well-known, longitudinal study of H.M. is of particular importance to subsequent progress in the neurology of memory.<sup>11</sup> H.M. had both medial temporal lobes including hippocampi surgically removed for treatment of epilepsy (1953, when 27 years old). Thereafter he served as a test subject for over 55 years until his death in 2008 at the age of 82. H.M. is particularly important in that he revealed the central role of the hippocampus in the memory.

The H.M. findings also impacted neurology in Japan resulting in reports of amnesia, as I recall, in the 1980s. And it was around that time that I and my colleagues started studying cases of amnesia with a focus on its symptomatology and underlying lesions – later publishing some of our results<sup>12</sup>. In this paper we attempted to understand impairment of episodic memory as a disruption of age awareness in terms of temporal cognition. When we compiled our findings on the limbic system, in 1993, we systematically described amnesia via the following three approaches - based upon contemporary notions<sup>13</sup>: (1) in terms of direction: that is, anterograde or retrograde; (2) in terms of storage: that is, immediate, recent or remote memory – neurologically speaking: short-term memory and long-term memory – psychologically speaking and (3) according to content: declarative (episodic and semantic) or procedural memory. Nowadays these classifications are common knowledge and used in general clinical practice. Furthermore, the neuropsychological approach – based on these classifications – has become essential to medical services for the explosive growth in number of dementia patients – particularly in Japan.<sup>14</sup>

A fresh glance at these classifications reveals the importance of the temporal aspect in memory taxonomy. We automatically try to grasp memory from the temporal aspect of past, present, and future. Conversely, we believe cases are pigeonholed into a rigidly classified amnesia framework when symptoms reported by patients are better understood as time-related symptoms.

## V. Neurology of time

While the neurology of memory has made remarkable progress since the time of Proust, and thanks to H.M., understanding of the neurology of time has only just begun. Although, in Japan, Koshika *et al.*, for example, published clinical research as early as 1954<sup>15,16</sup> in which they adopted a psychiatric/ philosophical approach based on Bergson and Kant.

In his classic work, *The Parietal Lobes*,<sup>17</sup> MacDonald Critchley (1900-1997), while conceding that “pure temporal disorientation ... occurring independently of spatial

disorders, is a rarer phenomenon, for more often, the two are combined”, also tried to classify symptoms into five types. These were: (1) inability to judge the passage of time; (2) difficulty in estimating the approximate hour of the day without some clue (such as recalling the meal last partaken); (3) temporal disorientation; (4) altered temporal associations with simple habitual activities such as shaving or taking a bath, and (5) sensation that time passes far too quickly (Zeitraffer Effekt). This classification, probably based on clinical observation of his own patients with parietal lobe symptoms, is simple but informative, and the fifth type is found in some cases of temporal lobe epilepsy.

In fact, researchers studying H.M.’s amnesia noticed that he showed abnormalities in estimating duration, and he was thus examined using a time-perception task.<sup>18</sup> This was very simple: he was presented by the examiner with time intervals from 1 to 300 seconds and asked to reproduce the same duration (the duration reproduction task). And while he accurately evaluated durations up to 20 seconds, he underestimated durations longer than 40 seconds. Thus, the hippocampus is probably involved in part of the time perception mechanism.

Clinical tests to evaluate time perception in detail are available but not many, and we believe we need in future to diversify assessment methods.<sup>19</sup> Of those assessments already available aside from time measurement (for example, stimulus presentation using a personal computer or stopwatch), hearing introspection for subjective symptoms is of particular importance to some cases of time perception impairment.<sup>19</sup> Alterations in time perception experienced by our patient with time agnosia (patient A) can be characterized via introspection of symptoms as follows: (1) loss of sense of temporal distance (inability to grasp the order of events due to failure to establish a time axis); (2) loss of date awareness (perception of a date as a fact without meaning); (3) attenuation of emotional responses to the passage of time (inability to feel the sorrow of parting and pleasure of reunion); (4) inflexibility of scheduling.

Patient A had a lesion in the medial aspect of the left frontal and parietal lobes. Partly because of absence of dedicated sensory organs, time perception is likely to involve many brain regions. We speculate that, in addition to the hippocampus, the default mode network around the medial parietal region, the precuneus in particular, may play an important role in temporal cognition.<sup>20</sup>

Thus far, we have written reviews on impairment of temporal cognition<sup>21-23</sup>, and held a roundtable discussion<sup>24</sup> in which we established that while a sundial indicates a point in time, an hourglass and water clock measure its passage. This recognizes a distinction between two aspects of the same entity: punctuation of time and length of time. We combined these to systematize our current notion of the forms of temporal cognition (Table 1).<sup>22, 25</sup> A list of time-related symptoms partly based on our clinical cases is also presented in Table 2.<sup>12, 18, 21, 23, 36-30</sup> Furthermore, we studied autistic patients for their ability to measure time.<sup>31</sup> We also experimented with a stopwatch, training intervention with PD patients.<sup>32</sup> We were also able to demonstrate that patients with AD while unable to report episodic memories verbally, nevertheless retained them as indicated by responses to non-verbal stimuli.<sup>33</sup>

## Epilogue

The recent expansion of brain research has been sufficiently rapid enough to encompass matters previously reserved to philosophy and the arts, such as ethics and aesthetics, and we also conducted functional imaging studies in healthy individuals in an attempt to elucidate brain functions related to the Proust effect.<sup>34</sup>

Proust wrote *À la recherche du temps perdu* in the rich cultural atmosphere of Fin de Siècle Paris – a period that included the birth of modern neurology. And we believe that understanding of his work and of that period has still much to offer to contemporary neurology.

## Addendum

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## Figure legends

**Fig. 1** Proust's tombstone in Père Lachaise Cemetery, Paris. He is buried alongside his father and younger brother. Reproduced by courtesy of the photographer, Dr. Raita Kikuchi (Department of Neurology, Ushioda General Hospital)

**Fig. 2** Proust's snapshots taken from film footage. The man in the red circle (added by the author) is believed to be the novelist. Reproduced from the YouTube footage: *Marcel Proust Caught on Film, 1904* (<https://www.youtube.com/watch?v=51COHIgjbYU>).



**Fig. 3** Photographs of Sollier’s books on amnesia.  
 Left, Sollier P: *Les Troubles de la Mémoire*. Rueff, Paris, 1892;  
 Right, Sollier P: *Le Problème de la Mémoire*. Félix Alcan, Paris, 1900.

## Tables

**Table 1** Forms of temporal cognition

1. Time measurement\*<sup>1</sup>
  - (1) Anterograde
  - (2) Retrograde
2. Temporal orientation\*<sup>2</sup>
3. Age awareness\*<sup>3</sup>
4. Introspection into time flow\*<sup>4</sup>
5. Temporal order\*<sup>5</sup>

\*<sup>1</sup> Time measurement is evaluated by using a stopwatch and a personal computer. The duration reproduction task used in the H.M. case was a time measurement task.

\*<sup>2</sup> Orientation is evaluated by asking about the date (year, month, day, and day of the week), season, and time. This evaluation method is included in the Mini-Mental State Examination (MMSE) and the Revised Hasegawa Dementia Scale (HDS-R).

\*<sup>3</sup> Age awareness can be assessed during a medical interview, of which secondary analysis based on video recording is often helpful.

\*<sup>4</sup> Time flow perception is evaluated from the patient’s introspection.

\*<sup>5</sup> Useful information for temporal order testing has been provided by Damasio *et al.* (1985)<sup>25</sup> and other authors.

Reproduced with revisions from Sugimoto A, Futamura A, and Kawamura M: Brain circuitry of mental time—proposals from neurological practice. *Brain Medical* 26: 19-23, 2014

**Table 2** List of time-related symptoms (based on review of our cases)

Brain region	Symptom	Etiology
Orbitofrontal cortex	Age awareness impairment* <sup>1</sup>	Anti-NMDA receptor encephalitis, <sup>26</sup> herpes virus encephalitis <sup>12</sup>
	Temporal order abnormality	Rupture of the anterior cerebral artery <sup>25</sup>
Hippocampus-basal ganglia	Time measurement impairment* <sup>2</sup>	Bilateral hippocampectomy (H.M. case) <sup>18</sup>
		Parkinson's disease <sup>27</sup>

		Alzheimer's disease <sup>27,28</sup>
Anterior cingulate gyrus, precuneus, and retrosplenial regions		
	Time flow abnormality* <sup>3</sup>	Left anterior cerebral artery infarction <sup>29</sup>
	Temporal disorientation* <sup>4</sup>	Alzheimer's disease <sup>30</sup>

- \*<sup>1</sup> In those cases with encephalitis, memories of the past intruded into experience of the present, thus impairing age awareness, and the patients confabulated time travel to the past.<sup>12,26</sup>
- \*<sup>2</sup> Patients with Parkinson's disease had shortened mental length by one second.<sup>27,28</sup>
- \*<sup>3</sup> Patient A, who was experiencing time flow abnormality, had to use a timetable to maintain a normal social life.<sup>21,23,29</sup>
- \*<sup>4</sup> In Alzheimer's disease, damage to the precuneus caused disorientation, resulting in alterations to the mental axis of past, present, and future.<sup>30</sup>