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Neurological Disorders in Famous Artists

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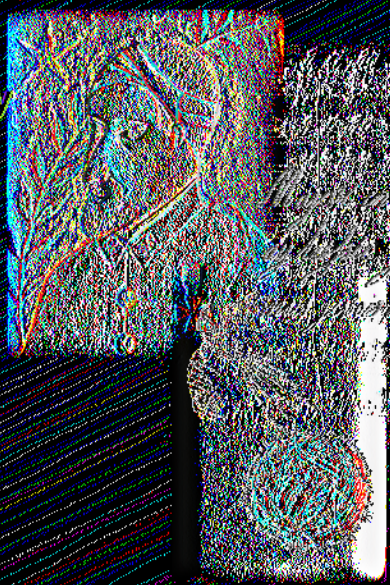

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Neurological Disorders in Famous Artists

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Contents

Preface

Art, creativity and neurology may seem widely different topics at first sight. However, it is obvious that art originates in the brain. Therefore, disorders of the nervous system often influence the preparation and development of artistic activities because they may alter motor, sensory, and above all, cognitive, functions.

The creativity of painters, musicians, writers, poets, and other artists is the magnified expression of one of the 'highest' brain functions in the human being, and it is understandable that brain, spinal cord, or peripheral nerve disease may have subtle or overt influences on artistic production. While every clinician has indeed observed instances of altered or modified creative abilities in individual patients with acute or chronic neurological disease, one of the best ways to address the problem probably is to have a closer look at famous artists, whose activity and production has changed (or has failed to do so) after the development of a lesion or dysfunction of the nervous system.

In the following chapters, the impact of various neurological diseases such as stroke, epilepsy, brain trauma, dementia, and other problems in famous artists (writers, philosophers, painters, and composers) is presented through the lens of changes in their behavior and in their production. Some 'cases' such as Ravel or van Gogh are already famous, but the nature of their disease has remained somewhat controversial. Other artists with a neurological disorder have been much less well studied, including Apollinaire, Daudet, Gernez, Haydn, or Kant, to quote only a few.

There is no such a thing as a 'creativity center' in the brain, and the production of an art work obviously requires a 'global' brain functioning, about which we are still lacking much knowledge. However, it is also striking to observe how a localized damage to the brain or other nervous structures has led

to subtle or dramatic changes in creativity and artistic production in many famous artists. Between ‘neurology of history’ and ‘history of neurology’, the study of how a neurological disorder can alter productivity in recognized artists and other creative people is a largely unexplored field. We hope that this book will represent a useful contribution to this domain.

Julien Bogousslavsky
François Boller

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Guillaume Apollinaire, the Lover Assassinated

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Abstract

The nature and the neurological consequences of the shell wound to the head of the famous French poet Guillaume Apollinaire, on March 17, 1916, during World War I, remains unclear. However, his contemporaries and biographers have been unanimous in stating that this event affected a major shift in his life. His personality and behavior changed dramatically, and his affective relationships were deeply modified, the most significant example being his rapid disinterest for his fiancée Madeleine, to whom he had written passionate letters nearly every day before the trauma. The study of Apollinaire's letters, scarce medical reports, available memories from friends, and Apollinaire's helmet allow one to better understand the type of brain lesion he had and why neurobehavioral dysfunction developed. While an 'intracranial abscess' had been emphasized initially, clinical manifestations, free interval, no infectious problems, and quick resolution of hemiparesis and seizures after a burr hole was performed strongly suggest a chronic subdural hematoma in the right temporal region. Irritability, susceptibility, emotional intolerance, affect flattening, anxiety, and personality change fit perfectly with the diagnosis of right temporal lobe dysfunction involving the lateral-basal area. Sparing of the mesial-temporal, parietal, and frontal regions explains the absence of significant memory, cognitive, and executive impairment, without disturbing his creative skills as a poet and art writer. This cognitive-affective dissociation secondary to isolated right temporal post-traumatic lesion allows one to delineate the 'deep sorrow' of Apollinaire during the two and a half years before he died of Spanish influenza in 1918.

Guillaume Apollinaire (Wilhelm Apollinaris de Kostrowitsky, 1880–1918), one of the greatest French poets of all time, was wounded in the head during World War I, after which his personality and behavior changed dramatically, until his premature death from Spanish influenza 2 years later. While the nature and the neurological consequences of the wound remain partially unknown, Apollinaire's contemporaries have been unanimous in stating that this event was

a major shift in his life. In particular, his affective relationships were deeply modified, the most significant example being his rapid disinterest for his fiancée Madeleine Pagès, to whom he had written passionate letters nearly every day before the head trauma. The study of Apollinaire's own letters, scarce medical reports, available memories from friends, and his preserved helmet allow to better understand which type of brain lesion he had and why neurobehavioral changes developed [Bogousslavsky, 2003].

Apollinaire in 1916

At age 36, Apollinaire already was a well-established writer and art chronicles journalist, who had been one of the first to defend cubism, as an admirer and friend of Picasso, Derain, Braque, and others. Shortly after the outbreak of war, Apollinaire, who was of Russian nationality through his mother's side, joined the French army as a voluntary soldier in December, 1914. Initially, he had been drafted into the artillery, but he was moved to the infantry, as he wanted to be involved in the front line of battle. He was promoted to the rank of under-lieutenant in November 1915 and, on March 14 1916, he received the documents informing him that his application to get his French nationality had been accepted. On the same day, he wrote a long letter to the famous poet Max Jacob and another one to his fiancée Madeleine, which looked somewhat premonitory: 'I give you anything I own in case I die, this is my will'. On March 17, in the trench 'au bois des buttes' in the Champagne countryside, while reading the literary magazine *Le Mercure de France*, he was shot in the head during a bout of enemy fire, while wearing his helmet. He did not lose consciousness, as he stated later to André Billy: 'I thought I had not been hit, when all of a sudden I started to bleed'. Indeed, the wound initially seemed of little gravity, as he wrote the following day to Madeleine: 'I was wounded in the head yesterday by a shell which penetrated my helmet. In fact, the helmet saved my life. I am well taken care of, and apparently this will not be severe'.

The Wound

I have been able to trace Apollinaire's helmet which hung at a hook for over 70 years in his apartment, 202, boulevard Saint-Germain in Paris, preserved as everything else by his young widow after he died in November, 1918. Together with part of Apollinaire's library, the helmet was subsequently acquired by the Bibliothèque Historique de la Ville de Paris, where I could examine and study it (fig. 1). There is a triangular ($2 \times 1.2 \times 1.2$ cm) hole just above the right

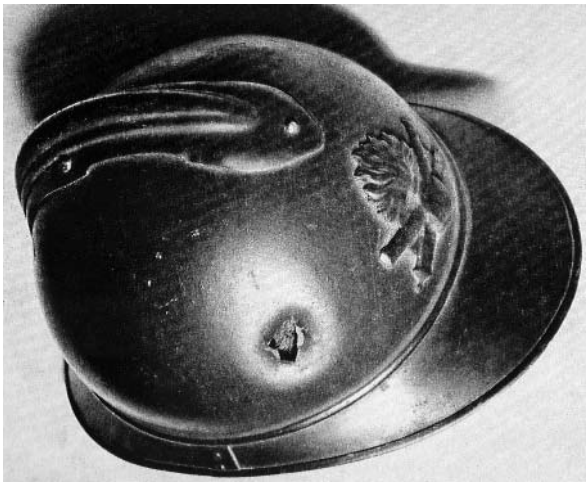

Fig. 1. Apollinaire's helmet (Bogousslavsky, 2003).

temporal region, the shape of which suggests an anterior to posterior, downward, trajectory of the shell. This is confirmed by the hole in the leather piece covering the inside of the helmet: this hole, which is smaller than the first one (1 cm), is located 2.2 cm behind the perforated metal. Moreover, examination of the leather bandage (worn by Apollinaire when recovering), which has also been preserved, shows that the center of the protecting pad is located 3.3 cm away from the point of entry into the helmet, backwards and downwards. These data allow one to reconstruct the potential point of impact on a standard skull and brain (fig. 2) [Bogousslavsky, 2003].

The First Days after the Wound

Three days after the wound, Apollinaire was transferred to Château-Thierry, and subsequently to the Val-de-Grâce in Paris. We are lucky that the doctor who took care of him knew more about neurology than the usual army physician, since she was the wife of the renowned neurologist Jean Nageotte, who had worked with Babinski and Dejerine. Herself a pediatrician, she left the best available description of Apollinaire's developing brain dysfunction. Her notes already mention the absence of any initial neurological event, with 'no brain accident, loss of consciousness, vomiting, visual or auditory disturbance, gait or speech disorder' until the last days of March, when Apollinaire developed headaches, fatigue, and dizziness. She also emphasized his 'neglected' aspect at that time. During the first days of May, while recovering at the Italian Hospital,

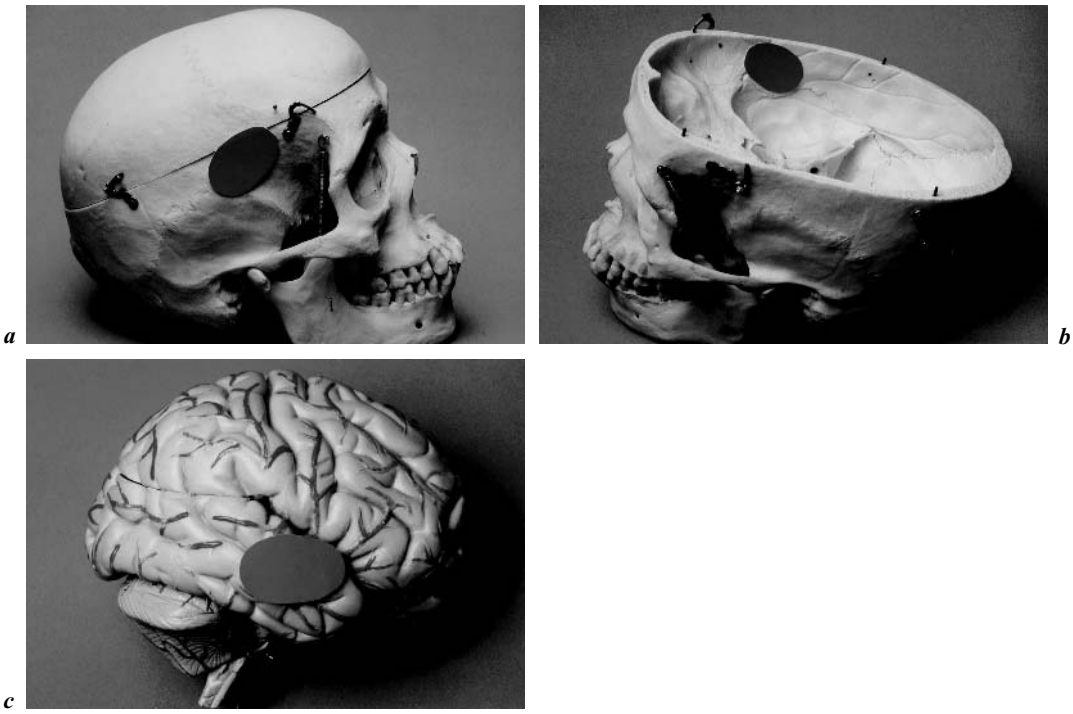

Fig. 2. Skull (***a***, ***b***) and brain (***c***) impact of the shell, reconstructed (Bogousslavsky, 2003).

Apollinaire developed increasing dizziness and had a sudden loss of consciousness in the street, suggesting a seizure, which was followed by a fluctuating hemiparesis mainly involving the left arm. Apollinaire was trepanated on May 9 by Dr. Baudet at the Villa Molière, which was affiliated to the Val-de-Grâce. An 'intracranial abscess' was reported, but no medical documents are available.

A Probable Chronic Subdural Hematoma

Retrospectively, it seems highly unlikely that Apollinaire had an intracranial abscess. The fluctuating clinical course, with a 6-week free interval followed by a complete resolution of the neurological manifestations (hemiparesis, seizures) after surgery, with no major infectious features, as well as uncomplicated survival without antibiotherapy, are much more compatible with a subdural hematoma over the right convexity. This is also suggested by the trivial-looking initial trauma, during which the helmet and skull seem to have absorbed most of

the kinetic energy of the projectile, with only a superficial, non deeply penetrating, wound. A last clue for a subdural hematoma is the report by the attending nurse of an immediate improvement of the hemiparesis during the operation: 'As soon as the skull was opened… the fingers of the paretic hand started to move slightly, which was followed by some movement in the leg'.

While the hemiparesis completely resolved, the personality and behavioral changes shown by Apollinaire persisted, which suggests a permanent damage to the lateral-temporal part of the right temporal lobe, due either to compression sequelae or to associated contusional damage.

Apollinaire's Affective and Emotional Disorders

In contrast with his own initial minimalization of the head wound, the first observable consequence was Apollinaire's new, dramatic, intolerance to emotional stimuli. This was particularly obvious in his correspondence with his fiancée Madeleine. While his previous letters, which he signed 'Gui', had been passionate, he changed to report mainly casual facts and ordered her 'not to irritate him' in either writing to him or coming to see him: 'Do not come, it would give me too much emotion. Do not write sad letters to me either, it terrifies me'; or: 'I am not anymore what I was in any matter, and would I listen to myself, I would become a priest'… 'I have become very emotional'… 'Do not send anybody, because visits from people I do not know frighten me'… 'It gives me too much emotion when I do not know'. This emotional dysregulation also appeared in letters to other persons, such as his war godmother: 'I am still very nervous, excessively irritable, and it seems that it will take more than one year for me to recover from this major trauma, which nearly killed me'.

Apollinaire's behavior also changed dramatically, the most spectacular modification probably being his rapid disinterest in his fiancée Madeleine, to whom he completely stopped writing within 4 months, without having seen her again after he was wounded (he had last seen her in Oran, Algeria, early January, 1916, i.e. 3 months before being wounded, and never saw her again). He interrupted any contact with her without any clear explanation to anybody, displaying an indifference, which was a great shock to many of Apollinaire's friends, who did not recognize his personality and behavior from before the trauma. There are several anecdotes, which emphasize Apollinaire's changes in emotional behavior and affective reactions: he was reported as having become irritable, susceptible, anxious (fig. 3), and with unpredictable emotional bursts, which contrasted with a loss of his sense of humor. Some degree of emotional flattening had also been noted rather early after the trauma, such as his striking absence of emotional manifestation when the doctors and nurses, themselves

Fig. 3. Portraits of Apollinaire after the traumatism, showing anxious and concerned expressions (***a*** by Jean Hugo and Jean Cocteau, ***b*** by André Rouveyre).

quite moved by the circumstances, came to his room to proceed with the trepanation on May 9, 1916.

Since Apollinaire died from Spanish flu less than 3 years after he was wounded, definitive conclusions on long-term changes in his behavior remain speculative. While he never mentioned Madeleine again, shortly before his death he married Jacqueline, an acquaintance who had taken care of him on several occasions. However, this relationship was characterized by its quietness, with none of the passionate features which had been so prominent with Madeleine, as well as with his previous girlfriends.

Emotional-Cognitive Dissociation with Spared Creativity

While brain parenchymal dysfunction was initially reflected by hemiparesis and seizures, no significant loss in cognitive and executive abilities was ever noted by Apollinaire's contemporaries. In the very same letters to Madeleine in which he was urging her not to provide him with any emotional stimulation, he was able to make a very detailed list of personal items, which he wanted her to send him back. Actually, his memory and cognition were never reported to be

altered. He also quickly went back to literary writing, both for poetry and art commentaries. He even introduced events of his head trauma in manuscripts, such as 'The Poet Assassinated' or 'Calligrammes' [Apollinaire, 1977, 1991, 1993]. He referred to a 'deep sorrow', and a 'secret unhappiness' with which he acknowledged he now had to cope and he could not control. Overall, while his lyrism did not decline, his general tone became darker, more defiant, and nostalgic, referring to 'something lost'. These elements underline a marked dissociation between Apollinaire's emotional-behavioral changes and the absence of impaired cognition and executive functions, including creativity. They also emphasize emotional dysregulation and modified (if not decreased) emotion-to-cognition coupling, rather than just a simple loss or flattening of emotional life.

Apollinaire's Right Temporal Syndrome

Apollinaire's emotional changes, including irritability, anxiety, defiance, intolerance to emotional stimuli, and sadness without true depression, fit very well with the rarely reported syndrome of lateral temporal dysfunction in the nondominant hemisphere. Indeed, right lateral temporal lobe lesions have been associated with such changes, in connexion with modification of personality, and affective behavior [Annoni et al. 2001; Bakchine 2001; Bogousslavsky, 1991; Bogousslavsky and Cummings, 2001]. This is in sharp contrast with mesial temporal damage, which is associated with memory impairment, and visual field defects and agnosia, due to involvement of the hippocampal and parahippocampal-fusiform gyri. In Apollinaire's case, the observed emotional and behavioral changes, together with spared cognitive and executive function, point to a limited lesion in the lateral part of the right temporal lobe, located immediately under the impact on the skull (fig. 2), together with full integrity of the mesial temporal, frontal, occipital parietal regions. This would be compatible with either direct concussion or compression sequelae from the subdural hematoma. A very similar, although non-traumatic, case was that of George Gershwin, who had undergone prolonged psychotherapy sessions for personality and emotional changes, while he shortly died thereafter, from a right temporal malignant glioma [Bogousslavsky, 2003; Ruiz and Montañés, p. 172–178].

It is interesting to emphasize that in the case of Apollinaire as well, the organic brain causality of his modified emotional behavior was not recognized by scholars, who paradoxically preferred to underscore a psychological shock associated with war experience, despite the head trauma [Apollinaire, 1977, 1991, 1993]. Given the purely emotional – behavioral nature of the clinical manifestations associated with right lateral temporal damage, it is likely that part of

the rarity of this syndrome is due to its lack of recognition, and mis-attribution to psychodynamic factors without organic cerebral dysfunction.

The lost love of Gui and Madeleine may be one of the best and purest historical examples of a dissociated emotional-cognitive/executive impairment from a focal brain lesion. The 'Poet Assassinated' indeed also was the 'Lover Assassinated'.

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Guy de Maupassant and Friedrich Nietzsche

A Comparison of Two Cases of 19th-Century General Paresis

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Abstract

Two late-19th-century writers provide parallel cases of syphilis, illustrating the progression of disease from infection to the final dementia of paresis. Guy de Maupassant (1850–1893) and Friedrich Nietzsche (1844–1900) each left their jobs when their health declined. Each spent a decade traveling in search of relief from the agonies of progressing syphilis, writing under adverse conditions of ill health. Each was institutionalized with a diagnosis of General Paralysis of the Insane after a sudden breakdown.

Paresis

The well-known syphilologist Joseph Earle Moore estimated that, in the days before penicillin, only about 5% of syphilitics progressed to the form of tertiary neurosyphilis known as paresis, General Paralysis of the Insane, or *Dementia paralytica* [Moore, 1943]. And yet, how many people today think of syphilis when it was epidemic only as it affected its victims in their demented last years? Two 19th century writers afflicted with paresis – Guy de Maupassant (1850– 1893) and Friedrich Nietzsche (1844–1900) – provide us with an illustration of the disease as it progressed from infection in youth, through years of excruciating pain and relapsing illness, to the final dementia. At the same time, we see the exhilaration that often marked an inspired period of creativity before the final break-down.

Infection

Syphilis is a sexually transmitted disease that begins with a chancre at the point of infection, usually the genitals, followed a few weeks after by a fever, rash, and severe malaise. It is usually infectious for the first 2 years, rarely after 5. In the 19th century, syphilis was a shameful secret, its name rarely spoken in polite society.

Infection – Maupassant

Guy de Maupassant violated the convention of secrecy with a notorious show of bravado. In a letter to a friend, he revealed that he had been infected with syphilis at age 20 by a ravishing boating companion but did not learn the diagnosis and the reason for his on-going ill health until years later.

'I hadn't been expecting that, I can tell you; I was very upset, but at length I said "What's the remedy?" "Mercury and potassium iodide", he replied. I went to see another Sawbones, who made the same diagnosis, adding that it was an "old syphilis, dating back 6 or 7 years"… In short, for 5 weeks I have been taking four centigrammes of mercury and thirty-five centigrammes of potassium iodide a day, and I felt very well on it. Soon mercury will be my staple diet. My hair is beginning to grow again… the hair on my arse is sprouting… I've got the pox! at last! the real thing! not the contemptible clap, not the ecclesiastical crystalline, not the bourgeois coxcombs or the leguminous cauliflowers – no – no, the great pox, the one which François I died of. The majestic pox, pure and simple; the elegant syphilis… I've got the pox… and I am proud of it, by thunder, and to hell with the bourgeoisie. Allelujah, I've got the pox, so I don't have to worry about catching it any more, and I screw the street whores and trollops, and afterwards I say to them "I've got the pox". They are afraid and I just laugh' [Quétel, 1990].

Infection – Nietzsche

Friedrich Nietzsche's early infection is not as clearly documented. In the clinical records at the asylum, he said that he had been 'twice infected' in 1866, which fits with rumors that he was treated for a syphilitic infection by two Leipzig doctors in 1867. (The second infection may have been another venereal disease, perhaps gonorrhea, as he told one of his doctors, Otto Eiser.) The examination at the asylum in Jena revealed a scar on his penis, a possible indicator of a prior syphilitic chancre. Nietzsche's sexual life remains a mystery, and hypotheses that he was infected by a woman in a brothel in Cologne or by a man in a brothel in Genoa are unsubstantiated.

Progressing Illness

In the years after infection, the syphilitic was often plagued by such a wide variety of illnesses in many parts of the body that the disease was named 'The Great Imitator.' Both Maupassant and Nietzsche had years of relapsing illnesses indicative of a severe progressing syphilis.

Progressing Illness – Maupassant

After 8 years as a civil servant with the Ministry of Public Instruction, Maupassant's poor health forced him to apply for a leave of absence (with pay) to recover in the spas of Switzerland from what he called a nervous malady. Over the next ten years Maupassant agreed to follow carefully the orders of numerous doctors (he referred to them as princes of the medical sciences) despite his profound skepticism regarding their abilities to alleviate his suffering.

Biographer Robert Sherard speculated that great quantities of mercury ('barometer syrup') had left Maupassant anemic and overly sensitive to cold. He abandoned vapor baths taken for limb pain because he feared an apoplectic stroke. When cold weather made everything worse, he yearned for tropical heat. He went from place to place seeking relief from his agonies, carrying with him a suitcase containing 'a cornucopia' of drugs of dubious benefit. Goncourt noted in his journal that Maupassant was haunted by fear of death and moved constantly on land and sea to escape from this fixed idea.

Maupassant described chronic headache, one of the most debilitating symptoms of progressing syphilis: 'The dreadful pain racks in a way no torture could equal, shatters the head, drives one crazy, bewilders the ideas, and scatters the memory like dust before the wind' [Williams, 1982].

By 1880 he was nearly blind in his right eye. An ophthalmologist found paralysis in the accommodation of the right eye and an oculist concluded from a dilated pupil that 'the mischief lay behind the eyes', recalling later: 'This disorder, apparently insignificant, caused me nevertheless to foresee… the lamentable end which awaited (10 years later) the young writer formerly so vigorous and so valiant' [Sherard, p. 208].

In 10 years of freedom from regular employment Maupassant was able to write six novels, three hundred stories, and three plays as well as travel books and poetry. Robert Sherard suggested that his gloomy wretchedness translated into his books as unvarying pessimism. One of his short stories, 'Bed Number 29', confronts syphilis. A dashing captain in the Franco-Prussian War (remarkably like Maupassant) returned home to find his lover Irma wasting away in a syphilis ward. When he leaned to kiss her forehead, 'He believed he detected an odor of putrefaction, of contaminated flesh, in this corridor full of girls tainted

with this ignoble, terrible malady' [Maupassant, 1955]. Before she dies, Irma gloats over having infected as many invading Prussians as possible.

Progressing Illness – Nietzsche

Like Maupassant, Nietzsche was forced to leave his job as his health declined. A year's paid sick leave from the University of Basel where he was professor of Classical Philology was extended to a permanent leave of absence. And like Maupassant, he spent the decade before his final breakdown traveling from place to place looking for relief from headaches, nervous complaints, limb pain, and depression. He tried the waters of St. Moritz as a remedy against what he called 'a deeply entrenched nervous illness'.

To his friend Franz Overbeck Nietzsche wrote:

'I am desperate. Pain is vanquishing my life and my will. What months, what a summer I have had! My physical agonies were as many and various as the changes I have seen in the sky. In every cloud there is some form of electric charge which grips me suddenly and reduces me to complete misery. Five times I have called for Doctor Death, and yesterday I hoped it was the end – in vain. Where is there on earth that perpetual serene sky, which is my sky? [Middleton, 1969a]'

He complained of an unmovable black melancholy and extreme weariness.

Nietzsche's voluminous correspondence during those years describes recurring attacks that left him shattered and exhausted, often only able to steal minutes or quarters of hours of 'brain energy' to write. 'The stomach would no longer be subdued even by the most absurdly rigorous diet', he complained. 'Recurrent headaches of the most violent sort, lasting for several days. Vomiting that lasted for hours even when I had eaten nothing. In short the machine looked as if it wanted to break down and I will not deny that I have several times wished that this could be the end' [Middleton, 1969b].

An ophthalmologist found bilateral inflammation of the inner layers of the eyes and diagnosed chorioretinitis, which, after iritis, is the most frequent syphilitic affection of the eye. An examination by another doctor yielded a further pessimistic opinion: Nietzsche must curtail reading and writing for several years, avoid bright light, wear blue sunglasses, avoid spicy foods, coffee, and heavy wine, and not exert himself mentally or physically. He had to place his paper two inches from his eyes in order to write. His life, he wrote, was a fearful burden:

'I would have long thrown it over if I had not been making the most instructive tests and experiments on mental and moral questions in precisely this condition of suffering and almost complete renunciation… On the whole I am happier than ever before. And yet, continual pain; for many hours of the day a feeling closely akin to sea-sickness, a semi-paralysis which makes it difficult to speak, alternating with furious attacks' [Middleton, 1969c].

Paretic Prodrome

Syphilologists looked for brief episodes of uncharacteristic behavior, grandiosity, hints of impeding madness, and euphoria alternating with suicidal depression in the years before the final breakdown – a time known as the paretic prodrome, or warning period. Both Maupassant and Nietzsche experienced intimations of madness while remaining enormously productive.

Prodrome – Maupassant

Maupassant's friend Frank Harris wrote:

'Three or four years before the end, Maupassant knew that the path of self-indulgence for him led directly to madness and untimely death… fits of partial blindness, then acute neuralgic pains and periods of sleeplessness, while his writing showed terrible fears… Then came desperate long-continued depression broken by occasional exaltations and excitements… and always, always, the indescribable mental agony' [Critchley, 1979].

Madame de Maupassant thought she saw the first signs of her son's madness in passages of *Sur l'eau,* a story written in 1888. Several of Maupassant's friends reported that he had begun to talk rather wildly. When the editor of *La Nouvelle Revue* told him he was talking like a madman, he answered: 'My brother, you know, is already mad; yes, mad. Didn't you know that he is no longer at Antibes, but in a private asylum? When will my turn come?' [Sherard, p. 353].

Maupassant described his optimism while planning *L'angélus*, which he anticipated would be his best work: 'I feel admirably fit to write this book. I have it all perfectly in my head. It was all thought out with an astonishing facility. It will be the crowning of my literary career' [Sherard, p. 375]. He wrote one story of 14,000 words in 4 days without a single correction: it 'was there, complete, erect within my mind' [Sherard, p. 360]. He had the manuscript copied so that he could save the perfect original.

Sherard agreed with a popular idea of the 19th century that syphilis could take genius to new heights, when he reflected that Maupassant's literary leap to fame in Paris in 1880 might have been the result a tremendous stimulation of the brain cells from this disease. The brains of syphilitics, he wrote, are for a time before the late stage 'capable of extraordinary production of far higher merit than they would ever have been capable of without this inoculation' [Sherard, p. 235].

Right before his breakdown Maupassant lamented: 'There are whole days on which I feel I am done for, finished, blind, my brain used up and yet still alive… I have not a single idea that is consecutive to the one before it. I forget words, names of everything, and my hallucinations and my pains tear me to pieces' [Sherard, p. 378]. He imagined that the salt baths he had been giving his nostrils had started a salty fermentation in his brain and that the dissolved brain

was flowing back through his nose. In Paris he announced that he had been made a count and insisted on being so addressed. In literary circles it was agreed that Maupassant had lost his mind.

Prodrome – Nietzsche

Sigmund Freud wrote of Nietzsche's extraordinary achievement in the period before the breakdown. On 28 October 1908, the Vienna Psychoanalytic Society devoted the evening to Nietzsche's posthumously published *Ecce Homo*. According to Freud:

'Nietzsche was a paretic. The euphoria is beautifully developed, and so on, and so on. However, this would oversimplify the problem. It is very doubtful whether paresis can be held responsible for the contents of *Ecce Homo*. In cases in which paresis struck at men of great genius, extraordinary accomplishments were achieved until a short time before the outbreak of illness (Maupassant). The indication that this work of Nietzsche is fully valid and to be taken seriously is in the preservation of mastery of form' [Nunberg and Federn, 1967].

Illness became his fate, Freud said.

'The degree of introspection achieved by Nietzsche had never been achieved by anyone, nor is it likely ever to be reached again. The most essential factor must still be added: the role that paresis played in Nietzsche's life. It is the loosening process resulting from paresis that gave him the capacity for the quite extraordinary achievement of seeing through all layers and recognizing the instincts at the very base. In that way, he placed his paretic disposition at the service of science' [Minutes, vol. 2, pp. 31–32].

Nietzsche described this euphoria and competence – what Thomas Mann called his soaring intellect blasted with ecstasy – to a friend on 18 December 1888, about 2 weeks before his breakdown:

'Never before have I known anything remotely like these months from the beginning of September until now. The most amazing tasks as easy as a game; my health, like the weather, coming up every day with boundless brilliance and certainty. I cannot tell you how much has been finished – *everything.* The world will be standing on its head for the next few years: since the old God has abdicated, *I* shall rule the world from now on' [Middleton, 1969d].

Breakdown

The syphilitic often experiences a sudden breakdown that marks a turning point from sanity to madness. Often periods of apparent sanity alternate with dementia after the collapse.

Breakdown – Maupassant

Maupassant shot himself, though the wound was not serious, and then slashed his throat. When his servant discovered him, he confessed: 'You see, François, what I have done. I have cut my throat. It's a case of sheer madness' [Sherard, p. 382]. A doctor sewed him up and packed him into a straight jacket. Waking from unconsciousness for a day, Maupassant announced that he must go to the frontier: war had been declared. On January 6, he was taken to Paris, still in restraints, and placed in the celebrated asylum of Dr. Blanche in Passy.

Breakdown – Nietzsche

In January 1889, Nietzsche broke down in the town square of Turin. After his landlord rescued him, Nietzsche stayed up late into the night banging on the piano and shouting. He composed a number of mad postcards to his friends, one of which summoned Franz Overbeck on a rescue mission. Nietzsche broke into tears and embraced his friend. Then he began to rant, uttering 'bits and pieces from the world of ideas in which he has been living, and also in short sentences, in an indescribably muffled tone, sublime, wonderfully clairvoyant. Unspeakably horrible things would be audible, about himself as the successor of the dead God, the whole thing punctuated, as it were, on the piano, whereupon more convulsions and outbursts would follow' [Middleton, 1969e]. Overbeck wondered if it would have been kinder to take his friend's life. Instead he tricked him into a train ride that ended at the nerve clinic of Dr. Wille, an expert on General Paralysis of the Insane. The sign-in sheet recorded: Friedrich Nietzsche, Professor at Basel at age of 23. 1866. *Syphilit. Infect*.

Asylum

Death usually follows within a few months or years (stationary paresis), although in slowly deteriorative types (galloping paresis), the patient may live 30 years or more.

Asylum – Maupassant

Although at times Maupassant seemed rational in the asylum, delighting his visitors with amusing stories, he also sometimes hallucinated and had to be restrained. A daily record kept by his doctor documents a rapid decline. Maupassant began to write of his huge fortunes of gold nuggets and buried treasure. He bragged of being the wealthy younger son of the Virgin Mary, expected twigs to sprout into baby Maupassants, and kept his urine because it contained diamonds and jewels. He howled and licked the walls of his cell.

‘You haven’t seen my thoughts anywhere, have you?’ he worried when they seemed to flee from his brain, but then he was cheery again when they showed up in the form of butterflies colored by mood-black for sadness, pink for good cheer.

At the end the powerful Maupassant had to be kept in restraints. His last words were reported to be ‘des ténèbres, des ténèbres’ – darkness, darkness.

Asylum – Nietzsche

When Nietzsche’s friends first visited him at the asylum, he seemed so sane that they thought he must be faking dementia. At other times, he was agitated and incoherent, screaming, drinking his urine, and smearing feces on the wall. He experienced delusions and auditory hallucinations. By 1895 he was showing signs of physical paralysis.

Nietzsche was eventually released into the care of his mother, who watched over him until she died in 1897; from then until his death, his sister, Elisabeth took charge.

Nietzsche died of a stroke on 25 August 1900. Elisabeth did not permit an autopsy; at that time what she called the ‘disgusting suspicion’ of syphilis had not yet been divulged.

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The One-Man Band of Pain

Alphonse Daudet and His Painful Experience of Tabes dorsalis

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Abstract

Like many of his contemporaries, Daudet suffered an early syphilis that accompanied his adult carrier, eventually extending into a disabling and painful neurological syndrome of locomotor ataxia. We provide here a quick picture of Daudet's life and work, his relationships with the high-society of late 19th century Paris, including Jean-Martin Charcot of whom he was the patient. We then proceed to describe Daudet's condition, with the available treatments of his time and their – largely ineffective – effects. We conclude with reflections on the experience of disease and pain on the author's work and life. Special attention is paid to a posthumous and unachieved work called *La Doulou*, a grim and most valuable first-person perspective document on the personal experience of unbearable pain.

Pain, you must be everything for me. Let me find in you all those foreign lands you will not let me visit. Be my philosophy, be my science [Daudet, 2002, p. 42][1].

…from the day that Pain entered my life [Daudet, 2002, p. 13].

Alphonse Daudet is classic reading in French-speaking school programs. Usually associated to his contemporary *naturalists*, he provided a realistically raw picture of 19th century Parisian life through his novels, chronicles, souvenirs, theatricals and musicals. However, he is mostly renowned for his marvelously witty and nostalgic depictions of his native sunny Provence in his many short stories. As this volume is devoted to the experience of neurological illness

[1] Except for the admirable, and only, English translation of La Doulou by Julian Barnes [2002], including its introduction and notes, all quotations in this chapter are translated by the authors from the French.

by famous artists, readers of *Les Lettres de mon Moulin, Les Contes du Lundi, Les Aventures Prodigieuses de Tartarin de Tarascon* or *Le Petit Chose* might be surprised to learn that their author suffered at an early age from a syphilis that deteriorated into a severely painful and disabling *tabes dorsalis*. Indeed, Daudet's work is most frequently qualified as being charming, light-hearted, sunny, funny, *Provencal*, etc. Less known is a posthumous work called *La Doulou* ('The pain'), which is none else than a *pain diary*. It is a terrifying work, dry, cold, helpless. And it is one of the most valuable literary documents on the personal experience of disease and pain. Perhaps more than any other artist included in the present collection, Alphonse Daudet spoke explicitly and uncompromisingly about his physical ailments. Was Daudet by any extent, as the custom goes in the Midi, exaggerating his symptoms for literary purposes? This is unlikely. The contrary seems more plausible, as we know from the numerous biographical writings of his relatives, including his own sons Léon and Lucien, Alphonse Daudet may well indeed have tried to *naturalize*, to describe as externally as possible, so as to dissociate from it, what was in fact an insufferable physical distress impossible to transcribe as such in words. What's more, in many places we can read his wish to not impose his sufferings to his fellows and readers, so he transformed complaints in sharp descriptions and striking metaphors. Thus, in this chapter, we don't have to unearth hidden or dubious 'evidence' from here and there to reassess our subject in hindsight, or even speculate on the proper impact of Daudet's disease on his work. We quite simply only have to take a look at his writings themselves, for they speak directly to the clinician's ears and to the heart of anyone.

We begin with a rapid tour of Daudet's biography and work, including a few words about his early friendship with the founder of modern neurology, Jean-Martin Charcot – of whom he was the patient. We then proceed to describe Daudet's condition and the symptoms he presented, and also the colorful treatments of his time. We conclude with reflections on the experience of disease and pain in the author's work and life, paying special attention to a posthumous and unachieved work called *La Doulou*, a grim and most valuable first-person perspective document on the personal experience of unbearable pain.

Alphonse Daudet: His Life, His Work and His Time

'I remember when I first met M. Alphonse Daudet. That was a long time ago, about 10 years back … He was handsome, subtly and nervously handsome, like an Arab horse, with an abundant mane, a silky barb, split like a two-headed fork, a big eye, thin nose, an amorous mouth; and on top of all this, a ray of light, a breath of tender sensuality that I can't quite figure, which drowned his

Fig. 1. Alphonse Daudet as a young adult. Source: Taken from Jouveau M.-T. (1980). Alphonse Daudet – Frédéric Mistral: La Provence et le Félibrige. Nîmes, Bene.

whole face in a smile at once witty and sensuous…' (fig. 1). This is Emile Zola's description of Daudet in his 1881 book *Les Romanciers Naturalistes* [cited in Daudet, 1997/1872, p. 170]. Of short stature, endowed with an irresistible smile, he was the beloved dreamy meridional darling of such famous figures as Dickens, James (who translated him into English), the Goncourt brothers, Mistral, Charcot and many others who literally adopted their 'petit Daudet' at the first encounter. Of course, ladies didn't ignore him either.

According to Bonduelle [1993], we know everything of his life. We have a quasi complete picture of it from multiple sources, including from many documents written by himself (*Trente ans de Paris, Souvenirs d'un homme de lettres, Lettres familiales, Notes sur la vie, La Doulou*, etc.) and his sons Léon and Lucien. Like many of his contemporaries, Alphonse Daudet is literally everywhere in the *Journal* of the Goncourt brothers. It was a time when everyone wrote about oneself and everybody else, a time when there was an abundance of diaries and *Mémoires*, just like now, but in that era people could actually *write*. Paris was a little place, the playground of virulent polemists, of which Léon, Alphonse's first child, would become a major controversial figure, the spin-head of the nascent *pre*-fascistic *Action Française*, among other little glorious etiquettes. Alphonse himself is part of the game, as he never was reluctant to ferociously describe his contemporaries – although with various disguises and tricks – in his works. This brought him much trouble, including incredibly violent critics and even various duels. To give a quick taste of the complexities of the character, and the harshness

of that era's public 'debates', we found particularly telling that Alphonse has been, at the same time, labelled with disgusted contempt as a 'Jew' by Octave Mirbeau on the one hand, while on the other hand being considered in republican circles as an anti-Semite for his acquaintance with Edouard Drumont, author of *La France juive*. It surely was a time of intellectual fury, the soil on which *l'affaire Dreyfus* would arise, and some would even say far worse later events.

Biographical Sketch

He was born in Nîmes (southern France), on the 13th May 1840, the 6th child of a bourgeois family. Alphonse will then be brought up by a peasant from Bezouce, near Nîmes. His schooling will be somewhat hazardous. Ruined by a father with poor talent for business, the family leaves to Lyon. There he goes to the Lycée Ampère, but seems mostly interested, already, by his precocious discovery of the bohemian life-style. Virginity abandons him at 12, as will in 1857 the idea of obtaining his baccalaureate. That year is full of upheavals for Alphonse. His elder brother Henri dies, he goes back to Provence for a job of school prefect in Alès – he will later write of his teenage miseries with the snobbish pupils that gave him a hard time in *Le Petit Chose*, a touching collection of memories –, is fired after 6 months, and decides to rejoin his brother Ernest in Paris in the explicit goal of becoming a writer. Lyon will quickly be forgotten, but his native Provence will always stay in his heart and work.

On a freezing November day in 1857, at age 17, pockets filled with 'quarante sous', he is greeted by his brother at the Gare de Lyon. Ernest was at the time a journalist for *Le Spectateur*, and also a fine connoisseur of the bohemian and fashionable capital. It wasn't long before Alphonse was introduced to the vibrating underworld of the Ville Lumière. Ernest and him share a room in the animated Grand Hôtel du Sénat, their young neighbor is Léon Gambetta, who will later proclaim the third Republic in 1870 and, incidentally, introduce Alphonse to Jean-Martin Charcot. Those were financially hard times for the brothers, but Alphonse is determined to become a poet and confident that better days are coming. Much later, he will write that 'literature was the unique goal of [my] life, and [I] know of no one else that started a career in the same state of absolute bareness as [mine]' [quoted in Caracalla, 2003]. In the carefree atmosphere of Parisian cafés, cabarets and bistros, he meets the very finest grain of local characters: Verlaine – *absinthe*-minded as ever –, Mallarmé, Baudelaire, D'Aurevilly, the contemporary Parnassists, etc.

And ladies, too. Marie Rieu, a famous model of her time, becomes his official maîtresse. *Official*, but by no means *only*. Indeed, Daudet's sexual needs are known to be untimely, others shall say excessive. In the words of neurologist Macdonald Critchley: 'Daudet – despite his exquisite sensitivity and his warm domesticity – had been an incorrigible sensualist since the age of 12' [Critchley,

1969]. And in those of British author Julian Barnes: '[H]e had always been "a real villain" in matters of sex…he slept with many of his friend's mistresses; about ten times a year he felt the need for the sort of "ordure" he could not ask his wife to permit… In 1884 he had an operation for a hydrocele. Having a grossly swollen testicle painfully drained… would probably make most men sleep in their trousers for weeks; Daudet's reaction was to go straight out in search of sex. In 1889 he reported to Edmond de Goncourt a dream in which he was caught up in the Last Judgment and defending himself against a sentence of 3,500 years in hell for "the crime of sensuality" ' [Daudet, 2002, p viii].

At this time, Ernest had to leave Paris for a new job. Daudet is left all by himself, pockets empty. Nevertheless, he manages to get his poems published, a collection named *Les Amoureuses* dedicated to Marie Rieu. This early work came to the ears of Impératrice Eugénie and Princesse Mathilde thanks to a *lectrice de la cour* (young ladies were appointed to select and read aloud from the literary releases of the time). Ironically, it was precisely from that reader who introduced Daudet into the highest spheres of intellectual circles and thus put an end to his material worries, that Daudet got the pox! He was only seventeen. After a short detour in le Midi, were he met with his life-long friend Mistral and the provincial poets of the félibre tradition, he is appointed as third secretary by the Duc de Morny thanks to the encouragements of the princess. More than a secretary he becomes a friend and a literary aide, although Morny, desperately would implore him on a daily basis to cut his long hair once and for all.

The disease showed its first signs early on, as will be developed below. But Daudet's career was launched by then. He travels to Corsica and Algeria, from where he would come back with many striking images and anecdotes that will fill the adventures of Tartarin de Tarascon published in 1872, his own pathetic Quixote. Meanwhile, he is busy with his poems, chronicles and theatrical plays, and in 1865 he meets the woman he would marry two years later, Julia Allard (fig. 2). Of course, in exact bohemian spirit, this couldn't happen without *chien vert* – Marie Rieu's curious nickname – making an extravagant scene in which she literally rolled at Daudet's feet!

According to Charles Mantoux [1941], Julia saved Alphonse from a dreadful life of careless debauchery and idleness. She became a precious collaborator to his work and a stabilizing element as his disease took hold of him progressively. With her affectionate guidance, success would not be long to come. The *crème de la société* is invited at the Daudet's on a weekly basis (*les jeudis des Daudet*). Many persons were to be found both there and at Charcot's Tuesday evenings. Daudet's Thursdays and Charcot's Tuesdays went somehow hand in hand and couldn't be missed by any Who's Who wannabe. This brings us to say a few words about this friendship, which gave to historians of neurology the best intimate picture of the great man of La Salpêtrière.

Fig. 2. Painting of Julia Allard by R. de los Rios (1894). Taken from Jouveau [1980].

Charcot and Daudet

We don't know exactly how and when Alphonse Daudet and Jean-Martin Charcot first met. According to Bonduelle [1993], it was Léon Gambetta that probably played a role in their meeting, as he knew both separately. The first record of their simultaneous presence is the 11th April 1882 entry in the Goncourt brothers' *Journal*. A frequent visitor of Daudet Tuesdays, Charcot was usually seated by Léon Daudet between Edmond de Goncourt and Emile Zola. It is precisely Léon that will become Charcot's best memorialist, through his many memoirs [Bonduelle, 1993, 1999]. An early friend of Charcot's children Jean and Jeanne, Léon spent at their house, almost next to that of the Daudet's, 'the golden years of his childhood' [quoted by Bonduelle, 1993]. Léon was to marry in 1891 Jeanne Hugo, sister of his long-time friend Georges Hugo – both grandchildren of Victor Hugo and adopted by the great writer since their father's precocious accidental death – instead of Charcot's daughter, as the neurologist so profoundly wished for very long. Maybe this is part of the reason why Léon had to abandon his medical career, following his definite failure at the internship exams of 1890. It appears that at the time Léon was very busy *not* studying, but flirting with Hugo's grand-daughter, and that Charcot had more than his word to say concerning the student's notations… Anyway, 5 years later Jeanne and Léon divorced. She remarried to Jean Charcot, whom by the way succeeded at the very same internship exams that Léon failed, but he never was to practice since he preferred the cold air of open seas and became a famous maritime explorer, who lived up to experience every captain's fantasy, namely to sink with his boat. To stretch it further, we will only add that in 1903 Léon

remarried to Marthe Allard, whose father was Julia Allard's brother, and mother was Alphonse Daudet's sister…

As we know, Léon Daudet wholeheartedly took advantage of the abrupt ending of his medical career to become that notorious right-wing polemist who could rip apart any prestigious career in a few lines. He always expressed sincere respect and fascination for the neurologist, whom he called 'the extraordinary classificator of the most arduous problems', a significant privilege under Léon's acerb pen. It is through his eyes that we know something altogether from the man behind the professor. He certainly was a despotic and arrogant figure, but also a shy man, a loyal friend and an erudite of the letters [Bonduelle et al., 1996; Daudet, 1915, 1939/1897, 1940]. When Daudet's health began to seriously deteriorate, it was Charcot – together with Potain, professor at Necker and *la Charité* and also a close friend of the Daudet's – that came to his bed to watch after him. Léon recounts: 'Thus these two augurs were at my dad's bedside. I was alone with them, my mother having gone. Charcot first took a look and my anguish was terrible. But he stood up and uttered out: "Nothing serious" and turning to Potain, whom was about to imitate him, "Unnecessary my dear friend, it is plain to see". Then Potain, in a breath: "Nevertheless, I prefer…". He made himself sure, then, turning towards me, his hand on my shoulder: "That's right… no serious lesion". Nevertheless, it was decided that my father would go to the waters of Allevard, in Savoie' [Daudet, 1940, p. 113].

What was the kind of friendship that united Daudet the writer and Charcot the professor? It was mutual admiration, *without* the worries of envy and competition. They had in common their fantastic observational skills. 'A fine mind which has no disdain for a writer. His style of observation: many analogies with my own, I think' [Daudet, 2002, p. 19]. French neurology acute memorialist Michel Bonduelle likes to quote this anecdote from Léon Daudet: 'There was between the rue de Bellechasse, where we lived, and the vast garden of the hotel occupied by Charcot, a blacksmith. The great scholar and the great writer often discussed which of the two would hear the last hammer bang. It turned out it was my father, although Charcot was pretty sure it would be himself' [Daudet, 1940, chap VII]. Of course, Charcot was as delighted with Daudet's works as the latter was fascinated by the great clinician. He was often a spectator of the fashionable *Leçons du mardi*, and upon receiving a printed edition of those teachings, he thanked his prestigious friend like this: 'Limpidity, solidity, concision and those broad strokes *à la* Tacite, which are those of a poet as much as of an observer, that's what gripped me in your book, which I read in fever and pain. But you know that inside me, until now, the literary hack and the dreamer are stronger than the tabetic. Thank you dear Master friend, for having thought of your old patient…' [quoted in Bonduelle, 1993, p. 1643].

Daudet's friendship with Charcot also reflected in his work. He wrote *A la Salpétrière*, a 'short study' were he describes the symptoms of hysteria, then a most fashionable disorder [Mantoux, 1941]. On many occasions, Charcot suggested to Daudet that he exploit some of his clinical observations or other, 'Daudet, you should relate this, I took some notes, I'll give you the details later…' [Daudet, 1940, p. 113].

Finally, circumstances coalesced to put an end to the Daudet-Charcot relationships, not the least notable being his son's eviction from the internship exams and the barbaric – and useless – treatment imposed by Charcot (the terrible Seyre's suspension, see below). However, it was primarily Julia that broke off communication, as she never could suffer the coldness and lack of tact of the man. It is true that he was the archetype of the 'medical power' denounced later by Léon in his *Les Morticoles*, a clinician more concerned with disease labels and prestige than by the patients themselves. However, Alphonse and Léon didn't bear a grudge on Charcot (though the younger Lucien passionately hated the man).

Work and Style

Daudet's contributions are multiple in form and content. He wrote poetry, prose, plays, musicals, novels, tales, souvenirs, chronicles and serials for newspapers, and even his notes on pain were published, although posthumously. He wrote about humbles and riches (*Le Nabab*, 1877; *Les Rois en exil*, 1879), beauty and misery [*Le Petit Chose*, 1868; *Les Lettres de mon Moulin*, 1869), true love and debauchery (*Les Amoureuses*, 1858; *Sapho*, 1884 – clearly devoted to his torrid affair with Marie Rieu), contemporary habits (*Fromont jeune et Risler aîné*, 1874), literary ambition (*L'Immortel*, 1888), religious fanatism (*L'Evangéliste* 1883, dedicated to Charcot), luck and the lack of it (*Jack*, 1876), politics of the day (*Numa Roumestan*, 1881), etc. And of course, about pain in *La Doulou*, published posthumously in 1930.

For a time, he was courted to enter the *Académie Française*. But he rejected the opportunity altogether on many occasions, for instance in this short notice he asked *Le Figaro* to publish on his behalf: 'Please do me the favor to insert this in one of your news items: I am not applying, I never applied, and I will never apply for the Académie. Yours, truly, Alphonse Daudet. (Paris, 31 octobre 1884)' [in Benoit-Guyod, 1947, p. 205]. Daudet's characters were colorful and grandiose, often composites of celebrities of the time or inspired by the numerous people he met on each of his travels. In his pages, they became the heroico-burlesque Tartarin, Mr. Seguin and his goat, the Curé de Cucugnan, etc. However, far from the comical and light-hearted tone for which he is famous, his fascination for the morbid constantly appears in his work. Mantoux [1941] draws an impressive list of sick children, disfigured men, lunatics, depressives, idiots, hysterics, etc.

Daudet himself is described as 'obsessed by the thought of death'. Indeed, there is not a single book of him without at least one of the characters dying. He once told his friend Antoine Albalat: 'The idea of death has poisoned my existence. Every time I move into a new apartment, my first thought is: how will they get the coffin out? They'll have to pass here, turn there…' [quoted in Mantoux, 1941, p. 54]. But Daudet's real talent was precisely to convey a myriad of affective nuances through the experiences of death and suffering.

His style was a unique mix of two almost antithetic schools, naturalism and the *méridional* spirit embodied by the *félibrige*. Naturalism refers to a quasi-philosophical approach that is based on the absolute diktat of *reality*. It is rooted in the nascent scientific materialism of the 19th century and proposes a raw and uncompromising depiction of things *as they are*. Of course, it is Emile Zola's work that first comes to mind, with his epic and rigorous observations of characters subjected to the implacable determinisms of society and heredity. That determinism is everywhere in Daudet's writings, and it is his acerb and laser-like ability to observe and dissect his fellow contemporaries that makes him a worthy representative of the naturalists (alongside with Flaubert, Maupassant, Huysmans, etc.). He described himself as a 'marvelous feeling machine', a skill he owned, according to his son Lucien, to his exceptional myopia that enhanced his hearing and smelling. In the words of the latter: 'He felt and listened to a landscape as much as he looked at it, or rather his eyes, his nostrils and his ears were accustomed, from his very primary perceptions of things, to unite in mutual aid' [Lucien Daudet, 1941, in Daudet 1997/1872, preface, p. 17]. Naturalism implied raw revelations about taboo topics, a process from which Daudet wouldn't flinch 'for he thought that cruelty was the paroxysm of truth' [Mantoux, 1941, p. 40].

However, the effervescence of pre-industrial Paris was not everything to Daudet. He is indeed remembered above all for his exotic depictions of his native Provence. He was very close to his friend Frédéric Mistral, founder in 1854 of the *félibrige*, a literary association which still exists 'devoted to favor, organize, safeguard and promote the langue d'Oc and the cultural specificity of South of France' (quoted from the *félibrige* official website: http://www.felibrige.com). An excellent entertainer, Daudet took full advantage of his southern sensibility frequently inspired by his many visits to his fellow *félibres* [Jouveau, 1980]. Bypassing strict naturalist standards, he loved to twist reality, play with counter-factuals and evoke dreamy metaphors. His writings are often said to be light-hearted, charming, precise, exact, luminous, easy, rhythmic, melodic, sometimes nervous, and febrile.

We find it ironic that Daudet's reputation is today that of a sober, well-behaved and harmless author, an entertaining and gentle storyteller that can be fed to little schoolboys eyes shut. Yet death, suffering, pain and betrayal are all

around in his tales and novels, and few figures of the time were spared his wit and irony. He did not back away from the preposterous duels in which he got involved, and several times took great risks with his career, for instance making fun of the catholic church, his fellow *provençaux*, and even the prestigious *Académie Française*, the accession of which remained for him forever closed after his sarcastic novel *L'Immortel*. More to the point, *La Doulou* definitely vindicates him as a genuine explorer of the deepest human torments and certainly would make any ex-schoolboy reconsider his former opinion of the 'charming' Nîmois.

Daudet's Syphilis and Tabes dorsalis

As strange as it may seem, despite the omnipresence of syphilis in the social landscape of the time, the word itself rarely appears as such in the literature [Wald-Lasowsky, 1982]. It is altogether absent from Daudet's work for instance, and it doesn't even appear in *La Doulou*. One commentator wrote: 'Barely suggested here and there, it is the great absentee of the text, as if the disease withdrew into the indivisible core of pain, black violence and only certainty' [Solal, 2002]. If there was a taboo on the word, such was not the case with the idea, as it was omnipresent in late 19th century literature. It even came to be fashionable in intellectual circles to be afflicted by it. Barnes [2002, p. 85] recalls the famous anecdote of Maupassant howling triumphantly when he was definitely diagnosed with it in 1877: 'My hair is beginning to grow again and the hair on my arse is sprouting. I've got the pox! At last! Not the contemptible clap… no, no, the great pox, the one François Ier died of. The majestic pox… and I'm proud of it, by thunder. I don't have to worry about catching it any more, and I screw the street whores and trollops and afterwards say to them, "I've got the pox"!' [see also Hayden, p. 9–16]. In brief, syphilis was proof of virility, originality, an unmistakable sign of creativity, the true seal of the artist, in short, the real thing.

Syphilis Then and Now

As late as 1937, it was considered that about 10% of Americans were at risk to be infected with syphilis in their lifetime [Golden et al., 2003]. What happened then, of course, was the introduction of penicillin in the 1940s which almost eradicated this medical and social catastrophe. Infection rates declined dramatically during the rest of the century, only to slightly re-emerge with the new millennium.

It is a complex disease. Not anymore an etiological mystery since the discovery of the bacteria *Treponema pallidum* by Schaudinn and Hoffmann in 1905, but still a clinical puzzle to practitioners of many medical disciplines.

Indeed, syphilis as been dubbed 'the great simulator', it can disguise itself in innumerable differential diagnoses liable to mislead any clinician from the dermatologist to the neuropsychologist. Its other strategy is to hide and wait. It is such a Machiavellian disease that some even argue that the incriminated agent rapidly evolved, upon his appearance in 16th century Europe, *precisely* to produce milder forms in early stages, which would in turn favor its spreading in the population [Knell, 2004].

Syphilis is a progressive disease par excellence and its manifestations present themselves through three stages, although only a meagre 10% of patients with first stage syphilis end up developing a full-blown third stage [Janier, 2004]. Unfortunately for him, Daudet did with his *progressive locomotor ataxia* (Duchenne de Boulogne's term for Romberg's *tabes dorsalis*). This is one of the many clinical appearances of third stage syphilis, which is almost always synonymous with neuropsyphilis. Meningitis and the ever terrifying dementia called general paresis are other little enviable presentations. That classification of syphilis in three stages, by the way, was first established by Philippe Ricord at the beginning of the 19th century [Janier, 2004]. He was the one who first diagnosed Daudet's disease, possibly in 1861. Another simpler distinction is made between early and late syphilis, distinguishing the severe manifestations of advanced neurosyphilis from the mildest manifestations of the years immediately following the inoculation of the bacteria. Nowadays, diagnosis is based on the careful study of the cerebrospinal fluid and *T. pallidum* serology. Brain imaging is useful in advanced cases to look for vascular lesions and the presence of gummas in the parenchyma, but mostly to exclude other diagnoses [Conde-Sendín et al., 2002]. All this was of course unavailable in Daudet's times, so Ricord and all others had to rely solely on clinical observations. The course of the disease is as unpredictable as is its clinical variety. In Daudet's case, as much as 20 years separated is its infection from his first severe tabetic manifestations. But as we will show, even though his last 10 years were the most excruciating, the disease was in one form or the other always present in his writing career and life as a family man. The dreadful spirochete *T. pallidum* can attack the central nervous system very early on. Indeed, in 25–40% of untreated cases the invasion occurs within 3–18 months of the infection [Silberstein et al., 2002]. Most often, this results in asymptomatic meningitis, but it may be relevant in Daudet's case for he presented painful fits, sleep disturbances and mood lability well before reaching his full-blown tabes, which was diagnosed by Charcot only in 1885.

Daudet's Early Syphilis

As we briefly hinted before, he caught it at 17 from a *lectrice de la cour*. The first manifestations of early syphilis were examined by Doctor Philippe

Ricord, one of Morny's medical advisors and a founder of syphilography. The specialist was absolutely positive in his statement: it is syphilis. Alphonse confessed to his friend Edmond de Goncourt: 'I caught the pox with a lady from the top drawer, a terrible pox with buboes and all, and I gave it to my mistress' [quoted in Caracalla, 2003, p. 92]. He was by then a new member of the prestigious French literary syphilitics club, right in the top five with Baudelaire, Maupassant, Flaubert and Jules de Goncourt [Daudet, 2002]. However, Daudet was not as proud as Maupassant, he considered his ailment as a form of penance. Anatole France reported that shortly before he died, Daudet declared: 'I am justly punished for having loved life too much' [Daudet, 2002, p. 85].

He was immediately treated with mercury, a substance used since the XVI century for the treatment of syphilis [Hediguer, 1985]. The disease mostly lay dormant for a time, and Barnes [2002, p. viii] notes that '[he] worked, published, became famous, married…, had three children. He also continued an active, carefree, careless sex life'. However, before the full emergence of tabes, we know that Daudet had various unpleasant disturbances, which he reports in hindsight in *La Doulou*: 'Warning signs going back a long way. Strange aches; great flames of pain furrowing my body, cutting it to pieces, lighting it up… A burning feeling in the eyes. The hideous pain from light reflected in a window. Also, from that time onwards, pins and needles in the feet, burning feelings, hyper-sensitivity… Hyper-sensitivity of the skin, loss of sleep, the coughing up blood… The first moves of an illness that's sounding me out, choosing its ground. One moment it's my eyes; floating specks; double vision; then objects appear cut in two, the page of a book, the letters of a word only half read, sliced as if by a billhook; cut by a scimitar' [Daudet, 2002, pp. 6–7]. It is difficult to precisely date these disorders, but other sources, like the Goncourt brother's *Journal*, show the ineluctable progression of the disease and the intermittent acute crisis, like those of hemoptysis that almost killed him on a few occasions [Benoit-Guyod, 1947; Mantoux, 1941]. Nevertheless, it is about Daudet's debilitating late syphilis that we have the most information, perhaps too much information we might say.

Daudet's Tabes dorsalis (Progressive Locomotor Ataxia)

As every psychiatrist knows, the clinical picture of general paralysis is at the very heart of modern neuropsychiatric reasoning, or better stated of 'the organic model of mental illness' [Collée and Quétel, 1994]. That a bacterial infection could produce disorders of the soul was indeed a revolutionary advance in medical thought. Tabes also has its own history, not devoid of controversy either. Moritz Romberg first described the condition, but didn't mention syphilis as a possible etiology. In his 1840 textbook, he did, however, insist on a past history of excessive drinking and sexual activity as being frequent

Fig. 3. Charcot presenting a case of locomotor ataxia (circa 1875–1880). From Gasser J, Burns SB: Photographie et Médecine 1840–1880, Lausanne, Institut universitaire d'histoire de la médecine et de la santé publique, 1991. With kind permission.

among victims. It is of course the long lapse of time since the inoculation of the spirochete to the various signs of spinal undermining that made it difficult to postulate, and prove, a causal link. Indeed, tabes dorsalis (also called simply tabes, the Latin word for consumption) is the very later form of appearance of third stage syphilis, usually 18–25 years post-infection. Duchenne de Boulogne, Charcot's master before he became the *Imperator* of neurology, was the first to have an intuition of the causal relation between pox and tabes (which he named progressive locomotor ataxia). This was in 1858, and it wasn't until 1875 that Jean-Alfred Fournier proposed his strong hypothesis, based on careful clinical and epidemiological studies. Thanks to his work and insight, syphilitic infection was soon widely accepted as the etiology of tabes dorsalis [Nitrini, 2000]. Well, not by everyone though. The causal link wasn't acknowledged by Charcot (fig. 3) or by Gilles de la Tourette at the time. Interestingly, as Daudet was followed by both Fournier and Charcot, the writer was inclined, though secretly – so as not to hurt the feelings of his long-time friend – to favor Fournier's opinion.

As Daudet very well knew, tabes is characterized by painful fits in the lower limbs that are distributed in multiple dorsal roots territories. Visceral disorders also sometimes manifest as abdominal pains. Next may come the loss of deep sensitivity, with painful paresthesias, areflexia, incontinence and, of course, locomotor ataxia. Pathology is multiple: atrophy of the dorsal columns of the spinal cord, local inflammation of the meninges and large arrays of

metabolic abnormalities, leading to a diminution of neurons in selected areas of the nervous system [Conde-Sendín et al., 2002]. Ache definitively entered Daudet's life in 1879. He was 39 and was to suffer incessantly and cruelly until his death 18 years later. An entry in Goncourt's *Journal* from 1879 reads: 'It was the last Sunday at Flaubert's. Daudet appeared for a short moment. His entry was that of an anxious sick man questioning the faces around him. He sat down. I was struck by his hands, the paleness of wax' [quoted by Mantoux, 1941, p. 28]. It was in 1878 that a terrible crisis of hemoptysis marked the beginning of the end: 'All of a sudden, at the heart of the book [he was then writing *Les Rois en exil*], in the effervescence of those cruel hours that are the best of my life, sudden interruption, cracking of the overworked machine. It began while working, by sums of minutes, dozes like those of birds, a trembling of handwriting, a languor interrupting the page, disquieting, invincible… Then, bluntly, without pain, a violent hemoptysis woke me up, the mouth acrid and bloody. I thought it was the end' [cited in Mantoux, 1941, p. 27]. Doctor Potain sent him for the first time to the spas of Allevard, a thermal station located in the Alps between Grenoble and Champéry, to cure what he then believed were rheumatisms. It helped to momentarily alleviate his pains, but somehow he knew that nothing would ever be the same as before. He wrote in his memoirs: 'I could feel it, something was definitively broken inside of me; from now on, I would not be able to treat my body like an old rag anymore, deprive it from movement and air, extend evenings until the morning in order to bring it to the fever of beautiful literary inspirations' [quoted in Mantoux, 1941, p. 27]. In *La Doulou*, he remembers the very first alert: 'Memory of my first visit to Dr. Guyon in the rue Ville-l'Evêque. He probed me: some tenseness in the bladder, the prostate a little sensitive. In a word, nothing. But that *nothing* was the start of *everything*: the Invasion' [Daudet, 2002, p. 6].

He will henceforth remain intensely focused on observing the changes inflicted by his affliction. At the beginning of 1880, it becomes clear that his symptoms far exceed those of rheumatisms. Potain suggests trying another thermal station, and sends him during the summer of 1882 to the spas of Néris, in Auvergne. He will return there in 1884, but to absolutely no curative effect (though during this period he managed to write *L'Evangéliste* and *Sapho* – two remarked novels at their publication – and to travel in Switzerland). One day, upon his return to his country house at Champrosay, he realizes to his astonishment that he can't run anymore. The pains in the legs are already unbearable, as we know from his notes for *La Doulou*. He decides to consult Charcot, not without apprehension: 'Conversations with Charcot. For a long time I refused to talk to him: I was scared of the exchange we would have. Knowing what he'd say to me. I told him, "I've been saving you up for last".' [Daudet, 2002, p. 19]. Léon Daudet relates a discussion with his father right after that encounter: 'The

sufferings of my father kept increasing and as he complained incessantly to Charcot, the latter told him: "Come talk to me about it. You will take your time. I'll spend an entire day with you". I was very anxious of that consultation… I thought it was something serious. [My father told me]: "So there it is: Charcot told me the truth, which I also sensed. But do not worry. I am affected with tabes, in an indisputable way, classic tabes. (He examined my reflexes with his little hammer, and with such a dexterity!). But it is a kind of slow tabes. I can go like this until ninety, which gives me a reasonable margin of time. Pains are located in the legs and the belt, sometimes in the bladder. Walking is faulty. I have a bit of *steppage* [a word referring to the characteristic ample forward steps of the tabetics, which alleviate their pains]. It is very possible that I'll just stay in the present state. A regression of symptoms is also very possible. There are frequent cases". I was stunned, but tried not to show it.' Alphonse Daudet went on: 'As a treatment, he suggested the spas of Lamalou in the first line. Then gold chloride (*chlorure d'or*), which gives results. Against acute flashes of pain, morphine, but keeping it below a certain dose and switching times, so as to not become an addict. "In that case, Léon will give you the injections and I'll talk to him. Don't do that by yourself (…) Don't get yourself too worked up. It was better to warn you, I did it". Come give me a hug… and not a word to your mother' [Daudet , 1940, p. 212]. Not a very useful advice, since Charcot will secretly tell her the grimmer truth, in his characteristic blank fashion [Clébert, 1988]. Indeed, a few months later, all signs of hope vanish in Alphonse's mind: 'Long conversation with Charcot. It's just as I thought. I've got it for life. The news didn't deal me the blow I would have expected' [Daudet, 2002, p. 23]. So Charcot finally declared him lost. Daudet is 45 years old at that time, he will not make it up to 90 but still will live, or survive, 12 more years in increasing pain, and he *did* eventually become a morphine addict.

A Litany of Symptoms

Before pain set itself up as a chronic ailment, Daudet was already marked in his flesh by various problems. We already quoted the note where he depicts his 'warning signs'; to these we can add the following: 'gastric and laryngeal crises, bone problems, rectal and urinary crises' [Daudet, 2002:83]. However, the most colorful symptoms are those he meticulously describes in *La Doulou*, the manifold manifestations of pain.

Excruciating pain is all over *La Doulou*. In fact, the reader of this little collection of notes – this diary of pain – from the beginning gets the disturbing impression that pain is treated as the central character. Indeed, Daudet manages to give it a life of its own, as if it was taking control of its victim's superior writing abilities. Indeed, the epigraph he chose for his project was '*Dictante dolore*' (Latin for 'with pain dictating'). Pain is described sometimes as an entity – 'the

most despotic and possessive of Imperial hostesses' [Daudet, 2002, p. 42], sometimes as an activity – 'What are you doing at the moment? – I'm in pain' [Daudet, 2002, p. 3]. Subtle 'Varieties of pain' fill the pages, often in powerful metaphors. It is a swarming, a burn, an invasion, an infiltration, a prison, an armor, a breastplate, even a crucifixion. It takes the form of 'an impish little bird hopping higher and thither, pursued by the stab of my needle; over all my limbs, then right in my joints'. Unfortunately, 'the injection misses its target, then misses again, and the pain is sharper every time' [Daudet, 2002, p. 28]. Some notes are amusing comparisons, like: 'Spasms in the right foot, with pains shooting all the way up my sides. I feel like a one-man band, tugging on all his strings and playing all his instruments at once… This is me: the one-man band of pain' [p. 26]. Other depictions are more shivering: 'Sometimes, on the sole of the foot, an incision, a thin one, hair-thin. Or a penknife stabbing away beneath the big toenail… Rats gnawing at the toes with very sharp teeth. And amid all these woes, the sense of a rocket climbing, climbing up into your skull, and then exploding there at the climax to the show' [Daudet, 2002, p. 21]. Several notes are plain descriptions, but still manage to convey a powerful sense of the pathetic: 'Intolerable pains in the heel, which only calm down when I move my leg. I spend hours, sometimes half the night, with my heel clasped in my hand' [Daudet, 2002, p. 21]. And so on. There's a deep insight that is conveyed by Daudet's various descriptions. For him, there's '[n]o general theory about pain. Each patient discovers his own, and the nature of pain varies, like a singer's voice, according to the acoustics of the hall' [Daudet, 2002, p. 15].

But pain was by far not the only inconvenience of Daudet's disease. As the name implies, progressive locomotor ataxia involves a severe disturbance of gait. This worry also appears constantly in his notes: 'Torture walking back from the baths via the Champs-Elysées' [Daudet, 2002, p. 4], 'concentration on walking straight' [Daudet, 2002, p. 5], 'a change in my condition: walking badly. Not being able to walk at all' [Daudet, 2002, p. 41], 'No strength anymore. On the Boulevard Saint-Germain a carriage nearly runs me down, and I react like a berserk marionette' [Daudet, 2002, p. 9], 'Return to childhood. To reach that distant chair, to cross that waxed corridor, requires as much effort and ingenuity as Stanley deploys in the African jungle' [Daudet, 2002, p. 47]. He courageously insisted on walking despite its horrible difficulties, and developed interesting strategies to do so, based on visual feedback information: 'I walk with more confidence when I can see my own shadow, just as I walk better when someone is alongside me' [Daudet, 2002, p. 16]. Some notes allude to a loss of the feeling of ownership of his own legs, akin to what we know as asomatognosia: 'Sense of losing control of a leg, of it slipping away from you, like something inanimate. Sometimes an involuntary *jeté*' [Daudet, 2002, p. 22]. Sense of posture was sometimes altogether absent: 'you close your eyes and chasms

open to right and left. Five-minute cat-naps filled with harrowing nightmares: skidding and sliding, crashing down, vertigo, the abyss' [Daudet, 2002, p. 19]. Indeed, sleep was almost impossible without morphine, as we will illustrate in the next section on treatments.

Worrisomely for an author, writing became an insuperable task: 'The change in my handwriting' [Daudet, 2002, p. 28], with an embarrassing episode in 1885: 'At times, my hand trembles so much that it's impossible for me to write, especially if I'm standing up. (Signing the register at Victor Hugo's funeral. People all around, watching me – dreadful…)' [Daudet, 2002, p. 16]. In his last years, he was compelled to give dictation to his relatives. Weight loss and apparent ageing were spectacular: 'In my cubicle at the shower-baths, in front of the mirror: what emaciation! I've suddenly turned into a funny little old man. I've vaulted from 45 to 65. Twenty years I haven't experienced' [Daudet, 2002, p. 3], 'Crossing the road: terrifying. Eyes don't work anymore, can't run, often can't even hurry. I have the terrors of an octogenarian' [Daudet, 2002, p. 10].

We also noticed a few signs suggesting disorders of higher functions, although they very likely were due to the powerful analgesics he took. These notes seem to bypass the poetic to enter the space of confusion, indeed, derealization, depersonalization and disorientation are apparent in a few places. For instance: 'Effect of morphine. Wake up in the night, with nothing beyond a mere sense of existing. But the place, the time, and any personal sense of self, are completely lost. Not a single idea. Sense of *extraordinary* moral blindness' [Daudet, 2002, p. 43]. He recounts a strange episode: 'Last night, in my study, around 10 o'clock, I had a couple of minutes of pure anguish. I was fairly calm, writing an unimportant letter. A servant came in and put a book or something on the table. I raised my head, and from that moment I lost all sense of everything for 2 or 3 minutes. I must have looked completely stupid, because the servant, taking my blank face as a question, explained what he'd come for. I didn't understand his words and no longer remember them. What was horrible was that I didn't recognize my own study: I knew that's where I was, but had lost all sense of it as a place. I had to get up and find my bearings, running my hand along the bookcase and the doors and saying to myself, 'That's where he came in'. Gradually, my brain began to work again, my faculties returned. A kind of hypnotic effect, compounded by fatigue' [Daudet, 2002, pp. 27–28]. That was not the first time he experienced such derealization, or *jamais-vu*, though. He immediately added: 'This morning, hurrying to write all this down, I remembered being in a cab a couple of years ago: I shut my eyes for a few moments, and when I opened them I found myself on the lamplit *quais* of a Paris I simply couldn't identify. I ended up leaning right out of the cab door, staring at the river and a row of grey houses opposite. I was bathed in a sweat of fear. Then, as we came to a bridge, I suddenly recognized the Palais de Justice and the Quai des

Orfèvres, and the bad dream faded away' [Daudet, 2002, p. 28]. Subtle executive difficulties also are apparent, as in this statement: 'Very happy with the state of my brain. Full of ideas, and the phrases come fairly easily, but it seems to me that coordination is now more difficult' [Daudet, 2002, p. 17]. And after speaking with a patient at Lamalou (the spa where he was regularly sent by Charcot): 'How I understand what he said to me yesterday: "Pain stops me from thinking".' [Daudet, 2002, p. 68]. At other times, thought was agitated: 'It's truly horrible. The only thing the will has no effect on is the perpetual motion of the brain. It would be so good just to be able to stop, but no, day and night the spider goes on spinning; a few hours' respite can be gained only through doses of chloral' [Daudet, 2002, p. 23]. At Lamalou, in the company of other patients, Daudet makes an amusing remark on memory and language disturbances: 'No one remembers anyone's name; brains are racked all the time; there are great holes in the conversation. It took ten of us to come up with the word *industrial*' [Daudet, 2002, p. 56]. And also very simply: 'Memory. Feebleness. Ephemerality of my impressions: smoke against a wall' [Daudet, 2002, p. 11].

A Litany of Treatments

La Doulou is also a fascinating document in its depiction of the treatments then available and their diverse (and adverse) effects. Daudet was followed by about the finest medical authorities of his time: Marchal de Calvi, Guyon, Potain, Charcot, Privat, Ricord, Keller, Fournier,… None of them could help him, though. Even on his deathbed, Gilles de la Tourette and Potain brutally tried to reanimate him during several hours, to no other effect than to fill his family and friends with revulsion. From the moment he was diagnosed with syphilis by the pioneer of syphilography Dr. Philippe Ricord, he was immediately prescribed mercury, the usual treatment for that disease for several centuries. Such was its popularity that a joke arose from it, it went like this: 'spending one night with Venus and the rest of your life with Mercury' [Daudet, 2002, pp. 85–86]. Other substances frequently alluded to in *La Doulou* include: laudanum, chloral, bromide, antipyrine, acetanilide, and of course morphine. These were either given in conjunction or in alternation. Barnes [in Daudet, 2002, p. ix] also mentions two bizarre treatments that Daudet tried: David Gruby's esoteric diet, which apparently was a less enviable solution than death, and Brown-Séquard's injections of an 'elixir extracted from guinea pigs' (which Zola tried – to no effect – with the purpose of increasing his sexual powers…). Daudet really did try everything. He even wondered if '[he] shouldn't apply for a course of Pasteur's inoculations: the strong analogy between [his] extreme bouts of pain, [his] furious shaking and writhing, [his] drowning-man contortions, and a fit of rabies' having struck him [Daudet, 2002, p. 17]. All these treatments had cruel effects on Daudet's organism and mind. For instance, he

notes: ‘The side effects of bromide decrease in terms of depression and memory loss. Unfortunately, its curative powers also decrease’ [Daudet, 2002, p. 18], ‘Effect of chloral on the skin: thick patches like make-up’ [Daudet, 2002, p. 18].

In the end, only morphine gave him a rest, despite the nausea and delirious states it induced. ‘Morphine gives you wakeful nights in which you are gently rocked in a heavenly manner’ [Daudet, 2002, p. 18]. Daudet’s relationship with morphine was ambivalent, it was a benediction and a torture at once. ‘Morphine. The irreplaceable anaesthetic. The imbecilic rages it stirs up’ [Daudet, 2002, p. 39], ‘Morphine. Its effects on me. The attacks of nausea are getting worse’ [Daudet, 2002, p. 16]. But it allowed him to sleep comfortably, and to detach his mind from his aching body: ‘Morphine nights: effect of chloral. Erebus, thick black waves, and then sleeping on the edge of life, the void beneath. As delightful as slipping into a warm bath! You feel yourself being taken hold of, enfolded. Pains in the morning; a feeling you’ve been bitten all over; but your mind is clear, perhaps even sharper – or simply rested’ [Daudet, 2002, pp. 18–19]. Julian Barnes adds there an interesting note: ‘In 1895, Daudet answered a survey of the famous by a certain Dr. Lacassagne; for the previous 5–6 years, he said, he had slept only with the help of narcotics, as a consequence of which he had lost all capacity to dream’. In his last weeks, looking back at his life he said to his son Léon about morphine: ‘I am aware of its inconveniences and hazards, but all in all, it helped me to live and without it I do not know what I would have become’ [quoted in Daudet, 1940, p. 254]. Indeed, such were his sufferings that Daudet soon became a morphine addict, something he felt sorry and shameful about. At some moment or other, he was given several times a day his shots of morphine by his son Léon, his wife or, mostly, his father-in-law, himself an opiate addict. It is the latter’s ‘little house’ that is often referred to in *La Doulou*, as in this note: ‘The little house in the rue***. I dream about it. For a long while I fight the temptation. Then I go. Immediate relief. Sweetness. The garden. A blackbird singing. Leg cut off. No pain. Horrors’ [Daudet, 2002, p. 9]. He went there until Julia’s father died in March 1889: ‘If I were to write in praise of morphine, I’d talk about the little house in the rue***. Well that’s all over now. My old companion, who used to give me injections, is dead. (…) My poor friend. It’s all over now’ [Daudet, 2002, p. 40]. One can discern a hint of envy in that last comment.

Despite the lull offered transiently by morphine, his pains kept increasing. Charcot decided to try a method he just introduced in France from Russia: the so-called Seyres’s suspension (fig. 4). Many commentators have described this barbaric treatment, and again they are Daudet’s words that brilliantly catch the imagination: ‘Suspension. Seyre’s apparatus. The hanging up of poor ataxics, which takes place at Keller’s in the evening, is a grim business. The Russian

Fig. 4. Seyre's traction, a cruel treatment imported by Charcot from Russia. It was later introduced in the USA by Weir Mitchell. Taken from Bonduelle M [1993]. Original in L'Illustration, 23rd March, 1889.

they hang up in a seated position. Two brothers; the little dark one writhing away. I am suspended in the air for four minutes, the last two solely by my jaw. Pain in the teeth. Then, as they let me down and unharness me, a terrible pain in my back and the nape of my neck, as if all the marrow was melting: it forces me to crouch down on all fours and then very slowly stand up again while – as it seems to me – the stretched marrow finds its rightful place again'. And the implacable conclusion: 'No observable benefit' [Daudet, 2002, p. 30]. Keller was a hydrotherapist to whom Charcot used to send many of his patients for showers, baths and suspensions. Those took place 'in a dark corner of the baths, after everyone had left', and were sometimes operated by Gilles de la Tourette, one of Charcot's most brilliant disciples. Daudet underwent 13 such suspensions, and had eventually to stop when he suffered a serious crisis of hemoptysis in February 1889. That was the beginning of the end of the friendship between Daudet's and Charcot's families.

Since the mid-1880s, Daudet had frequented a thermal establishment at least once a year. As was the case with his other treatments, when one spa didn't contribute any benefit, his doctors simply sent him to another. He was first sent to the waters of Allevard, then Néris, and finally, from 1885 to 1893, to Lamalou-les-Bains, which he mostly wrote about in *La Doulou* (fig. 5).

As we now know, none of these 'treatments' ever came close to a solution. Syphilitic degradation remained incurable until the introduction of penicillin in

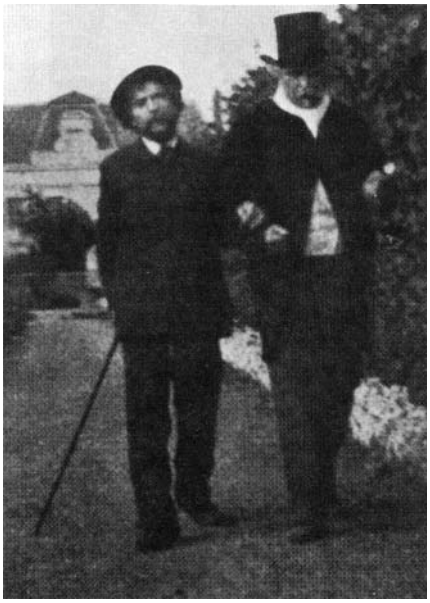

Fig. 5. Alphonse Daudet and Edmond de Goncourt at Lamalou-les-Bains. From Critchley [1969].

the 1940s, and from the very beginning of his discipline the neurological clinician gained the reputation of being a brilliant classifier conspicuously unable to heal whatever problem he was presented with. In the words of Léon Daudet [1940, p. 226]: 'I wondered why a man like Charcot never tried to heal. It was very well to describe lateral amyotrophic sclerosis. But it would have been even better to rescue all poor humanity from its claws. I also wondered why those who send their patients to the spas never investigated the origins of their certainly mysterious, but definite virtues. For there were cases of recovery thanks to those waters, and many of them. They acknowledged their happy results, but went no further. They left them with their secrets.'

The One-Man Band of Pain

It would be tempting to draw a chronological parallel between Daudet's works and the course of his illness. Indeed, it is true that his earlier works – *Les Lettres de mon Moulin, Le Petit Chose,* and *Tartarin* – might be more lighthearted than his latter productions. However, such a meticulous approach may be misguided, since it appears that suffering and writing are interlinked since the very beginning of Daudet's career as a man of letters. This at least is the central thesis of the French literary scholar Charles Mantoux in his book *Alphonse*

Daudet et la souffrance humaine [1941]. We quite agree with his choice of considering the largest issue of distress (*souffrance*) rather than solely physical pain per se (*douleur*). Suffering translates into innumerable nuances under Daudet's pen, thus building the very essence of the human condition into his writings. The work of the artist is to dissect human nature and restore its pieces into living images. Therefore, resorting to one's own distress is not an opportunity, but a necessity. Mantoux thus claims that Daudet's work and life are on the whole plagued with suffering. He calls him 'the painter of human suffering', and writes: 'An unhappy youth; the experience of life acquired through marriage and war; the always alert anguish of the creator, all this was enough to make Daudet a mature and conscious man, apt to understand and paint all of human suffering. However, one last ordeal was still missing, cruel and capital: that of physical pain, which added to this physiognomy – already so lively and tormented – the pity, the sweetness and the nobleness of heroism'. Summarizing his life with a poignant shortcut, Daudet said to his son (Lucien): 'In the first half of my existence, I have known misery; in the second one, pain' [Mantoux, 1941, pp. 26–27].

Of course, there is nothing original in saying that a writer's personal problems have an influence on his work, the contrary would be quite astonishing. However, it seems clear that no exact prediction can be drawn from a particular experience of pain and misery to a particular type of creativity. As Julian Barnes puts it [Daudet, 2002, pp. v–vi]: 'The prospect of dying may, or may not, concentrate the mind and encourage a final truthfulness; may or may not include the useful *aide-mémoire* of your life passing before your eyes; but it is unlikely to make you a better writer. Modest or jaunty, wise or vainglorious, literary or journalistic, you will write no better, no worse. And your literary temperament may, or may not prove suited to this new thematic challenge'. Léon Daudet, however, had another opinion: 'Nervous illness raises to the power of two – squares, as the algebraists put it – both the qualities and faults of those it touches. It sharpens them like pencils, as my father used to put it. The miser becomes a hyper-miser, the jealous man surpasses Othello, the lover turns frenetic. On the other hand, noble, generous, disinterested souls acquire, in the face of incessant pain, a strengthened sense of altruism; an almost saintly goodness blossoms forth. Such was the case with Alphonse Daudet' [Léon Daudet, 1940; quoted in Daudet, 2002, p. 16]. Author and academician Jules Lemaître thought that suffering had shrunken and dried out Daudet's style. Again, such was not the opinion of Léon: 'His thinking became purified, like that of Pascal, by the courage to endure pain. Then he reached serenity, a terrestrial serenity, through pity. Great pain leads to either meanness and belligerence, or pity. He chose the second way' [Daudet, 1940, p. 238]. As for Marcel Proust, then a young protégé of Alphonse and a close friend of Lucien Daudet,

he wrote: ‘I saw this handsome invalid beautified by suffering, the poet whose approach turned pain into poetry, as iron is magnetized when brought near a magnet’ [quoted in Critchley, 1969].

In his concise style, Alphonse Daudet simply put it this way: ‘Pain leads to moral and intellectual growth. But only up to a certain point’ [Daudet, 2002, p. 43]. That is to say, too much pain, too much distress or too much misery won’t enhance anybody’s talent. Skills have somehow to be there in the first place. Nevertheless, Daudet remained humble in front of his task, indeed, he wasn’t convinced at all that his writings on his own terrible condition could satisfyingly convey his personal experience: ‘Are words actually any use to describe what pain (or passion, for that matter) really feels like? Words only come when everything is over, when things have calmed down. They refer only to memory, and are either powerless or untruthful’ [Daudet, 2002, p. 15]. He once confessed to his son Léon: ‘It is bitter, this disproportion between what my pen determines and what my mind has conceived. I feel the suffering of the inexpressible’ [quoted in Mantoux, 1941, p. 24].

He constantly fought to keep a normal social life and to go on with his work despite his torments. As we see it, this involved a twofold effort. First he had to be constantly focusing his mind even for the simplest things, a tiresome exertion that allowed him not to give up altogether and, mostly, not to scare his relatives and be too much of a millstone for them. ‘Even the simplest and most natural of actions requires an effort of will: walking, standing up, sitting down, staying upright, taking your hat off or putting it back on. It’s truly horrible’ [Daudet, 2002, p. 22]. That attitude of consciously controlling his actions and thoughts included the careful observation of his own condition: ‘I can date each moment of my pain’ [Daudet, 2002, p. 23]. The second kind of effort he had to make was more natural to him, it consisted, in his words, in dissociating his ‘second Me’, which he also called the ‘observing machine’, from himself. Of course, it would be purely speculative to assert that Alphonse Daudet showed signs of dissociation in the strict psychiatric meaning, however, as many writers, he permanently kept an eye on himself as if from the outside, and qualified this skill by saying he was ‘homo duplex’: ‘I’ve often thought about this dreadful duality. This terrible second Me is always there, sitting in a chair watching, while the first Me stands up, performs actions, lives, suffers, struggles away. This second Me that I’ve never been able to get drunk, or make cry, or put to sleep. And how much he sees into things! And how he mocks!’ [Daudet, 2002, p. xiii]. And remembering the death of his brother Henri when he was 16, Daudet wrote: ‘My first Me was in tears, but my second Me was thinking, What a terrific cry! It would be really good in the theatre!’ [Daudet, 2002, p. xiii]. Maybe this long-standing attitude of ‘duplicating’ himself, at first mainly on intellectual grounds, eventually helped him to detach himself from his aching body. This can also be seen in the way he

wrote sometimes about pain, as if it were an entity separated from him, with a life of its own. As his life had been reduced to the acting and experiencing of his pain, it is possible that in the long run he managed to somehow dissociate from his insufferable body. Indeed, recent research shows that dissociation is not an uncommon reaction to chronic pain, a kind of spontaneous equivalent to what happens with dissociative analgesic-hypnosis [Merskey, 1993; Morse and Mitcham, 1998; Fishbain et al., 2001].

La Doulou: Pain, Life and Art

We will now take a closer look at that most valuable document which already guided us so much in this chapter. In the 50 riveting pages of *La Doulou*, Daudet paints with sharp uncompromising strokes a raw picture of his experience of pain. It is not really a book, rather a collection of notes he took from 1885 to 1993 for a project that remained unclear in his mind. A distant and vaguely stated goal was to describe 'the sexual desires and longings for death that illness provokes' [Daudet, 2002, p. xi], which would make the notes akin to a confession. Of course, the *immediate* purpose of taking these notes was cathartic, a way to try coping with unimaginable conditions of living. But it is apparent that Daudet wanted to construct a consistent narrative out of these notes, in his usual auto-fictional approach. Barnes [in Daudet, 2002, p. xi] quotes an interesting exchange with Edmond de Goncourt that took place in 1888: '[I]t begins like this. The terrace of the hotel at Lamalou. Someone says, "He's dead!" Then a character sketch of myself, done by myself. Then the dead man's servant slips his notebook into my hand. You see, like that, it's not me. I'm not even married in the book, and that will give me a chapter to make the comparison between suffering in the midst of a family and suffering alone. This notebook allows me a fragmented form, so that I can talk about everything, without the need for transition'. Indeed, Julian Barnes remarks that 'notes seem an appropriate form in which to deal with one's dying' [Daudet, 2002, p. xiv]. Critchley [1969] said of *La Doulou* that 'the writing is of a superlative order', which is probably unique for a plain notebook. Léon Daudet called it 'a terrible and implacable breviary'. Although the exact nature of the project was unclear from the beginning, the title was certain. *La Doulou* is Provencal for 'pain'. The word seems to sing with the crickets and to smell of lavender, it completely de-dramatizes the topic. The word also carries Daudet back to his childhood and innocence, a 'regression' which gives to some notes a deep sense of nostalgia. At first difficult to enter in, *La Doulou* progressively takes shape, becomes more consistent. Central ideas that would have constituted the heart of a fiction begin to arise page after page. The notebook was only published in 1930 by his widow Julia Allard, and the original manuscript is currently nowhere to be found, probably scattered around. It was not his last

work though, for he continued to write or dictate in his little home study until his last breath.

We already described the colorful symptoms and treatments Alphonse Daudet went through with many quotations from *La Doulou*. Now we will deal with the existential experience of pain as it is so brilliantly revealed in numerous notes, showing that pain in itself isn't, by far, the only topic dealt with in them. Daudet manages in very few words to extend his main topic into its many ramifications, which we will tentatively enumerate below.

The experience of pain evoked contrasting impressions, behaviors and social consequences in his life. As an immediate result, he felt deeply alone. Not being able to communicate his sufferings with words, compelled to immobility by locomotor ataxia, and not willing to impose his torments on his relatives, he felt confined to loneliness. In several places, he even uses the metaphor of imprisonment to describe his condition. This forced loneliness was not akin to the usual withdrawal of many artists, it was accompanied by distress and anxiety: 'Dread. Anguish in my heart. Since I've been left alone with pain, the life I have known has been so harsh' [Daudet, 2002, p. 48].

Along with loneliness came fear in its manifold manifestations: 'Very strange, the fear that pain inspires nowadays – or rather, this pain of mine. It's bearable, and yet *I cannot bear it*. It's sheer dread: and my resort to anesthetics is like a cry for help' [Daudet, 2002, p. 9], 'Bad night, woken with a jolt at three; no actual pain, but highly strung and in fear of pain' [Daudet, 2002, p. 8]. Most notable was his fear of degradation in the form of dementia: 'Since learning that I've got it forever – and my God, what a short "forever" that is going to be – I've readjusted myself and started taking these notes. I'm making them by dipping the point of a nail in my own blood and scratching on the walls of my *carcere duro*. All I ask is not to have to change cell, not to have to descend into an *in pace*, down there where everything's black, and thought no longer exists' [Daudet, 2002, p. 24]. That was a real concern for him. Here's the entry in Edmond de Goncourt's *Journal* for July 14, 1890, cited and translated by Barnes [in Daudet, 2002, p. 61]: 'Poor Daudet, who is haunted by an *idée fixe*: the fear of degradation, and the physical shame which paralysis entails. And when you try to reassure him, he tells you that he has studied the progression of his disease among his fellow-sufferers at Lamalou: he knows what will happen to him next year, and what will happen to him the year after'. Indeed, many of his fellow tabetics at spas ended with blindness, dementia or aphasia. Daudet was spared those tragedies, but not the angst of it.

Despair, death and suicide are explicit in many notes: 'My friends, the ship is sinking, I'm going down, holed below the water-line… Beginning of the end' [Daudet, 2002, p. 7], 'The end is near' [Daudet, 2002, p. 46], 'It's all going… Darkness is gathering me into its arms. Farewell wife, children, family, the

things of my heart… Farewell me, cherished me, now so hazy, so indistinct…' [Daudet, 2002, p. 31], 'I've passed the stage where illness brings any advantage, or helps you understand things; also the stage where it sours your life, puts a harshness in your voice, makes every cogwheel shriek. Now there's only a hard, stagnant, painful torpor, and an indifference to everything. Nada!… Nada!…' [Daudet, 2002, pp. 64–65]. We know from Goncourt that the only thing that held him back from suicide was his family: 'Daudet confides in me that 3 or 4 years ago his wife, having clearly seen into his heart and read the desire to make an end of it by suicide, forestalled his confession, and made such an eloquent plea for him to live for her sake and that of the children, that he renounced his intention of killing himself' [Goncourt, December 1, 1893, quoted in Daudet, 2002, p. 10]. In *La Doulou*, a short and implacable note states: 'Musing on suicide'; however, he immediately adds: 'One doesn't have the right' [Daudet, 2002, p. 10].

On the other hand, Daudet very often showed signs of hope and expressed many reasons and ways to fight for life. His deep sense of empathy certainly was one of these ways: 'My existence is effectively over: I live only through the novel – that's to say, through the lives of others' [Daudet, 2002, p. 48], 'We also inflict wounds, wounds to the pride of those who love us' [Daudet, 2002, p. 48], 'Pride in not imposing on others the bad moods and the somber injustices of my suffering' [Daudet, 2002, p. 47]. He once confessed to his friend and secretary André Ebner: 'Suffering is nothing, it's all a matter of preventing those you love from suffering…' [quoted in Daudet, 2002, p. x]. The same Ebner recounts a telling anecdote of Daudet's behavior towards his relatives: 'His last secretary, André Ebner, remembered Daudet sitting with a friend one morning, eyes closed, barely able to speak, martyred by pain. The door-knob gently turned, but before Mme Daudet could enter, her husband was on his feet, the color back in his cheeks, laughter in his eye, his voice filled with reassurance about his condition. When the door closed again, Daudet collapsed back into his chair' [Daudet, 2002, p. x]. The sick Daudet also felt angry to be so helpless as a spouse and father. In 1890, Julia fell seriously sick. He recounted his feelings this way: 'Painful hours spent at Julia's bedside… Fury at finding myself such a wreck, and too weak to nurse her. But my ability to feel sympathy and tenderness for others is still well alive, as is my capacity for emotional suffering, for emotional torment… And I'm glad of that, despite the terrible pains that returned today' [Daudet, 2002, p. 25]. Young Marcel Proust was also impressed by Daudet's attitude: '[T]his poet detached from himself and entirely devoted to us all, absorbed in *my* future and the future of other friends, smiting us and glorifying happiness and love' [Proust, quoted in Critchley, 1969, p. 211]. Daudet himself said it all in this poignant note: 'I only know one thing, and that is to shout to my children, "Long live Life!" But it's so hard to do, while I am ripped

apart by pain' [Daudet, 2002, p. 49]. Besides his family, Daudet also expressed empathy for those he called his 'doubles in pain' (Julian Barnes prefers 'Doppelgangers' to translate *sosie*). Thus, in *La Doulou*'s pages, one can read his concern for other literary syphilitics like Baudelaire, Heine, Léopardi and of course Jules de Goncourt, along with the unknown curists he met at thermal establishments. One of those worth mentioning was a Russian traindriver who suddenly became aphasic and started to speak a strange and unintelligible language. A young staff member of Spanish descent recognized the patois from the Balearic Islands, which turned out to be the first language the Russian ever heard as he had been entrusted to a nurse from there until he was five!

But empathy wasn't enough, he had to struggle vigorously to cope with his condition: 'Pain took its time to settle in me, and I fought hard against it, as much as I could', he was quoted by his son Léon, who added: 'My father, in speaking like that, was saying the plain truth, and anybody who reads *La Doulou* carefully will know so. From the day his sufferings became more acute and frequent, from the day walking began to alter, he required to his will an additional effort towards his work and his family life, which thus became, amidst his many works, his daily masterwork, a masterwork of love and perseverance' [Daudet, 1940, p. 235].

Time and nostalgia also appear as a watermark throughout *La Doulou*. Daudet was extremely attentive to the changes slowly taking place. This showed in his remarks on how places and things didn't appear the same as his illness progressed: 'Coming back again and again to the same place, like the wall you stood against as a child and on which they marked your height. A quantifiable change every time. But whereas the marks on the wall always demonstrated growth, now there is only regression and diminution' [Daudet, 2002, p. 67], 'The torment of coming back to the same spot again: I used to do that… I used to be able to do this… Well, now I can't anymore' [Daudet, 2002, p. 73]. He never got used to his condition in the long run: 'Pain is always new to the sufferer, but loses its originality for those around him. Everyone will get used to it except me' [Daudet, 2002, p. 19]. However, some notes may convey a sense of resignation, if not routine: 'Nothing but terror and despair at first; then, gradually, the mind, like the body, adjusts to this appalling condition' [Daudet, 2002, p. 48]. And also: 'What we want diminishes to fill the smaller space available. Today, I don't even want to get better – just to keep on at the same level. If they'd told me as much a year ago!' [Daudet, 2002, pp. 17–18].

Another personal way to cope with his condition was through escapism. He retained until the very end his powerful imagination and his passion for writing that allowed him to flee from his dolorous body. Also, reading Montaigne and accounts of explorers like Stanley – who he met in London in 1895 – were for him delightful diversions.

'Life Consists of Antagonisms': To Conclude

In one of his lapidary notes, Daudet scribbled 'Life consists of antagonisms' [Daudet, 2002, p. 49]. We found it particularly telling, although we can't be quite sure what he really intended to say or if he was alluding precisely to something. Taking a close look at Daudet's life and work, one is indeed struck by his many ambivalences and sheer contradictions. Consider for example the following: from a young age until the very peak of his career, he experienced the contrast between his native Provence and Paris; there he met Marie Rieu and *la bohème*, contracted the pox and lived in misery, until he married his loving Julia Allard and became a successful author and a devoted family man; his style was in the line of the naturalists but he managed to lighten it by incorporating his southern and fantasist streak; he described an inner double, a severe and mocking second 'me' that constantly kept watching him and his work; and, of course, his painful tabes dorsalis didn't quite fit his charming face and witty spirit. Instead of withdrawing in his 'armor of pain', Daudet fought courageously to spare his relatives from his distress and, with a little help from opiates, to carry on his work, the observation and satire of his fellow contemporaries.

We will conclude by quoting Charles Mantoux [1941], whom we think captured well the intricacies of Daudet's destiny with the sufferings he cruelly underwent: 'In Daudet, the larger audience obstinately only saw the author of *Tartarin* and *Les Contes du Lundi*. However, another wholly different aspect of Daudet, without doubt the greater, is barely known: it is that of the deep and solemn writer, to whom no human problem remained unknown, who observed life at length and who, from this contemplation, came out bitter and thoughtful. Indeed, a work solely comical cannot possibly be true and lasting. The spectacle of life is saddening and tragic. Anatole France said "In the middle of the eternal illusion we are shrouded in, only one thing is for sure: it is suffering. It is the cornerstone of life". Daudet showed us every aspect of pain and suffering in his work, and he could only succeed for he sustained them in his life'.

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Gustave Flaubert's Hidden Sickness

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Abstract

Many papers, thesis or books have been written on Flaubert's sickness. They speculate on the 'nerve sickness' which the author of *Madame Bovary* suffered. During his life and many years after his death, the diagnosis of 'neurosis' and even more of hysteria have been suggested. Nevertheless by reading attentively his prolific correspondence or *Les Souvenirs Littéraires*, written by his friend Maxime du Camp, the diagnosis of epileptic seizures is obvious. This has been perfectly demonstrated in a paper from Henri and Yvette Gastaut. Flaubert had presented at least two types of seizures: partial seizures – some with elementary visual symptoms and others with more elaborate symptoms – and generalized convulsive seizures. The identified causes of an occipital epilepsy are multiple and two hypotheses had been proposed by Gastaut and remain the most convincing: (1) an arteriovenous malformation which can explain the evolution in the recurrence of the seizures – frequent seizures at the beginning in relation with a bleeding of the malformation, then decreasing of the frequency of the seizures during the calm period – and the circumstances of death if we follow Flaubert's doctor who evoked a 'cerebral congestion'; (2) an occipitotemporal cerebral atrophy, the origin of which remains obscure but could explain that Flaubert, in his infancy, had some reading difficulties. The most amazing is the absence of mention of the illness in Flaubert's work. This absence which differentiates Flaubert from Dostoevsky, Daudet and Proust throws new light on the relation between illness and literary production.

Many papers, theses, and books have been written about Flaubert's illness and have speculated as to the nature of the seizures that the French novelist had during his lifetime. The Gastauts' paper, published in 1982, remains, in our opinion, the best treatment of this subject.

The phenomenology of the seizures is clearly described in the letters of Gustave Flaubert, and Maxime du Camp gives an excellent description of his friend's seizures 'which he frequently witnessed, distraught and powerless to intervene'.

The epileptic nature of the seizures is not in dispute. At least two types of seizures can be identified: (1) simple partial seizures with visual hallucinations evolving sometimes into complex partial seizures with psychosensorial features, and (2) convulsive generalized seizures.

Hypotheses regarding the etiology of the seizures can only be speculative; however, the diagnosis of 'neurosis' suspected by Dumesnil and portrayed as a form of hysteria in J.-P. Sartre's famous book on Flaubert, *L'Idiot de la famille,* can be dismissed.

'This illness has destroyed his life; it has forced him to isolate himself and has rendered him almost savage' wrote Maxime du Camp. Regardless, we know that his illness did not keep Flaubert from becoming one of the most prolific and the most widely-read novelists in the French literature, even though his friend wrote: 'if it were not for the nervous ailment that plagues him, he would have been a man of genius'.

History

According to all of the historical accounts (Bruneau, Dumesnil, Sartre and Gastaut), Flaubert had his first seizure in January 1844 – January 2nd according to Bruneau – and between January 20th and 25th according to Sartre. Maxime Du Camp, however, wrote in *Les Souvenirs Littéraires*: 'In October 1843, he had gone to Pont Audemer; his brother Achille went to find him there. They left together in a carriage driven by Gustave himself. In the vicinity of Bourg Achard, just as another carriage approached them from the left Gustave was thrown down and fell. His brother bled him right there on the spot, hoping without truly believing it that he had witnessed an isolated incident. Other episodes followed, and he went on to have four seizures over the next 3 weeks'. Based on these written accounts one may reasonably conclude that Gustave Flaubert had experienced a first convulsive seizure. The recurrence of the attacks tends to confirm this hypothesis. This first seizure threw Flaubert's family – particularly his father who was a surgeon at Hotel Dieu Hospital in Rouen and his brother who was also a doctor – into a state of turmoil: 'Flaubert's father was beside himself'. He resorted to various therapeutic means that were as helpful as they were harmful: 'One day after he had tried unsuccessfully to bleed Gustave, he applied boiling water to Gustave's hand thus causing a painful second degree burn'.

Flaubert mentions this first seizure in a letter to his friend Ernest Chevalier (Feb. 1884):

'You should know, dear friend, that I had a cerebral congestion, a kind of attack of apoplexy on a small scale, accompanied by nervous symptoms which I continue to display because it's good form to do so. I very nearly popped off in the midst of my family. They bled me in three different places at once until finally I opened my eyes.'

For at least the next 2 years, the seizures occurred frequently. Their semiology – simple partial seizures evolving into complex partial seizures with automatisms and some more elaborated hallucinatory features – were reported by Flaubert himself as well as by Maxime Du Camp:

'Very often, distraught and powerless to intervene, I would witness his seizures which were indeed terrifying to see. They always occurred in the same way and were preceded by the same features. Abruptly, without any reason, Gustave would raise his head and become very pale; that was when he was feeling his aura, this mysterious sensation that he would have in his face as though a ghost had just walked through him. His eyes would be full of anxiety and he would hunch his shoulders in a distressing and discouraging movement. Then he would say: 'I have a flame in my left eye' and a few seconds later: 'I have a flame in my right eye; everything is tinged with gold'. This peculiar state would last for several minutes. During this time, it seemed that he thought this was simply a warning and that it would go no further. Then his face would become even paler and it was obvious that he was extremely upset. He would walk to his bed quickly and lie down, dejected. As he was lying there, ominously, as though in a coffin, he would start to shout, 'I am holding the reins. Here is the carriage. I hear the bells. Ha! I see the inn's lights'. Then he would moan. That heartrending sound still rings in my ears. Then the convulsion would take him over. This paroxysm was invariably followed by a deep sleep and a stiffness that could last for several days' [*Souvenirs Littéraires*, p. 200].

The visual seizures are eloquently described by Flaubert in his correspondence:

'You speak about the hallucinations you have suffered. Be careful. First you feel them in your head, and you think that you are about to go mad. Then you are mad and you know it. You feel your soul leaving you, and with all your strength you try to stop it. Death must be like this, when we are aware of that it is happening' [letter to Louise Colet, January 15th, 1847].

'How can I be unfeeling, at the contrary, when I had, tonight, once more during a full half an hour, candles which were dancing in my eyes and which prevent me from seeing' [letter to Louise Colet, November 7th, 1847].

'As for your faithful servant, he is better, without being truly well. Not a day goes by that I don't see, every once in a while, what looks like a tangle of

filaments, or a burst of fireworks, passing before my eyes. This lasts for varying amounts of time. Still, my last big attack was less severe than the others' [letter to Ernest Chevalier, June 7th, 1844].

At the beginning of his illness, the seizures seemed to be relatively frequent, as Flaubert wrote, in January 1845, to Ernest Chevalier: 'as for my health, it is getting better; but the recovery process takes so long in these wretched nervous diseases that it is hard to tell (I am getting better)'. During a trip to Switzerland and Italy Flaubert had two more seizures: 'If I am getting better, I am not doing so very quickly'. During a trip with his friend Maxime du Camp, in Brittany, Flaubert had another seizure: 'The beginning of our trip was troubled: on the fourth day, while we were in Tours, Flaubert had a nervous attack. I called Doctor Bretonneau. This seizure was the only one to cast a shadow on our trip which we continued as soon as Flaubert was rested' [*Souvenirs Littéraires*, 251].

After 1846, the convulsive seizures were rare: 'But I will continue to live as I always have, suffering from nerves, this portal between the soul and the body, through which I keep trying to force too many things' [letter to Louise Colet, December 1847].

After this, Flaubert rarely ever mentioned his illness, except in a long letter to Taine. He lived cloistered in his house in Croisset, with his mother, his niece and his maidservant. He wrote *Madame Bovary*, *Salammbô* and *L'Education sentimentale*. He rarely traveled but wrote thousands of letters. Was he cured? This is what he seemed to imply in a letter to Miss Leroyer de Chantepie [May 18th, 1857]: 'You ask me how I recovered from my nervous hallucinations that I suffered previously? By two means: first, by studying them scientifically and making myself aware of them, and secondly by dint of will power. I would often feel the madness descending on me. My mind would become a whirl of thoughts and images, and my consciousness, my essence, would flounder, like a ship in a storm. But I held onto my sanity and played with the madness as Mithridate played with poisons. My reason, although battered and worn, prevailed.' Maxime du Camp, however, leads us to understand that the seizures recurred frequently: 'The torments with which he was plagued lent a dreadful intensity to the illness he had during his youth. The seizures occurred frequently and, at his age, they tended to be followed by a congestion' [*Souvenirs Littéraires,* p. 620].

The circumstances surrounding his death remain a mystery. Did he die during or after a seizure or from a 'cerebral congestion' as Doctor Tourneux' words would have us believe.

Tourneux was called by Felicie, Flaubert's maidservant. Dumesnil, who always has refuted the diagnosis of epilepsy, reports his words: 'I arrived at Flaubert's home to find him lying on an ottoman in his library, which was in

perfect order. I examined him. His face was flushed and he appeared not to be breathing although his heart was still beating weakly. There was no foaming at the mouth or evidence of convulsions. I am quite sure that there was no reason to believe that he had just suffered and epileptic attack.'

The descriptions of Flaubert's last moments, as they are reported by Maxime du Camp, who was not in Croisset at that time, are quite different and provide unequivocal evidence of an epileptic seizure: 'On Saturday May 8th, 1880, in the morning, he had a nervous attack which he tried to ward off by inhaling ether. When he recovered, the yellow visual disturbance that he called the 'golden vision' persisted. The head bothered him and his face was flushed blood red. He groped his way to the sofa and lay down on his back. Some rales could be heard coming from his chest. He breathing was labored and he tried to speak. In the midst of the darkness that enveloped him, he realized without doubt that his final moment was approaching. He shouted twice "Hallot! Hallot". His mouth twisted in a convulsive movement, and he expired' [*Souvenirs Littéraires*, p. 621].

The Epileptic Nature of Flaubert's Nerve Diseases

Although the epileptic nature of Flaubert's episodes is indisputable, two authors questioned this diagnosis: (1) Dumesnil, first in his thesis in 1905 and later on in 1947 spoke about 'nervous troubles', which he thought were a form of 'neurosis', and analyzed them in the context of many contemporary works on hysteria. He rejected the diagnosis of epilepsy because Flaubert never reported the various features of 'essential' epilepsy, particularly tongue biting and loss of continence. (2) Jean-Paul Sartre, in *L'Idiot de la famille*, analyzed in an astounding and forceful psycho-analytical language, the first seizure and considered all subsequent seizures as the beginning of a 'neurosis' brought on by the hatred between the father and brother. He wrote: 'Flaubert's illness is more a matter of hysteria than of epilepsy that has so often besieged him'. The great philosopher's utter ignorance of epilepsy is remarkable, 'We now understand that some epilepsies can be explained by hysteria'. Henri and Yvette Gastaut in their outstanding paper, provide a detailed refutation of this notion.

Flaubert had at least two types of seizures: (1) partial seizures – some with elementary visual symptoms and others with more elaborate symptomatology, and (2) generalized convulsive seizures.

Partial seizures with visual symptoms arise from an epileptogenic zone within the occipital lobe, particularly the primary visual cortex in the case of hallucinations or visual association areas in case of illusions [Bien et al., 2000]. Considering the seizure semiology that has been reported, it can be established

that the discharges most likely occured in the left striatal area. Apart from these brief episodes, Flaubert had longer seizures more elaborate hallucinatory features. These were sometimes associated with language deficits, fear or imminent sensations of death. These seizures suggest a propagation of the discharges into the ipsilateral temporal structures. This interpretation is based on the accounts he relayed in his letters.

'My nervous attacks merely mark moments when, without my being able to do anything about it, ideas and images begin to fade. At such times it is as though the psychic element leaps over and beyond me, and self-awareness disappears along with all sense of being alive. I am sure I know what it is to die. I have often distinctly felt my soul escaping, as one feels blood flowing from the incision when one is being bled' [letter to Louise Colet, July 6th, 1852].

'If being sensitive were sufficient qualification for being a poet, I would be better than Shakespeare or even Homer, who, I take it, was far from being what one would consider an overly nervous/susceptible/sensitive man. Such comparisons are sacrilegious. I am qualified to speak of such things, however, for I have been known to hear what people were saying in hushed voices behind closed doors thirty paces away: I have watched the viscera quiver beneath my skin; and sometimes, within the space of a single second, I have been aware of a thousand thoughts, images and associations of all kinds lighting up my brain like blazing fireworks' [letter to Louise Colet, December 27th, 1852].

'Often I felt that I was going mad. It was in my poor brain a whirl of ideas and images where it seemed to me that my conscience and my *self* sunk like a ship under a storm' [letter to Miss Leroyer de Chantepie, May 18th, 1857].

'My nerve disorder has been the foam of these little intellectual pranks. Each attack was a sort of bleeding of innervation. It was seminal discharges from the vivid faculty of my brain, one hundred thousands images blowing up at the same time, like fireworks. It was an excruciating feeling as though my body and soul were torn asunder. Each time, I was sure that I had died but I was always conscious even though I could not speak. Then my soul would withdraw into itself, as a hedgehog which would hurt itself with his own spines' [letter to Louise Colet, July 7th, 1853].

The possibility of an ophthalmic migraine was suggested by Bretonneau when Flaubert had a seizure in Tours, in 1847, but this hypothesis can be excluded on pure clinical grounds. In migraine, the hallucinations are usually black and white and are followed by constrictive cephalalgia. Flaubert, however, only once reported this kind of pain. 'I have this afternoon an excruciating migraine.'

We will not go back over the long discussion provided by Henri et Yvette Gastaut on the theory of 'neurosis' set forth by Dumesnil and of hysteria elaborated by Sartre: 'Dumesnil considers Flaubert as an hysteric who had hysterical

seizures. Sartre, on the other hand, considered him as an hysteric who had hysterical seizures but who may also have had epileptic seizures.'

The treatments prescribed to and taken by Flaubert are much more consistent with treatment for epilepsy than for neurosis: quinine, bromides, purges with castor oil plant, plant infusions, drainages: 'Yes my old friend, I have a séton (drainage tube) which flows and irritates me. I must keep my neck rigid, and I am so bothered by it that I have sweats. I am purged, I am bled, leeches are placed on my skin, good food and wine are forbidden me; I am like a dead man' [letter to Alfred le Poitevin, February 9th, 1844].

Etiological Hypothesizes

There are several possible known causes of occipital epilepsy [Taylor et al., 2003] and the following discussion can only be speculative and conjectural. Some causes can be easily discounted based on the historical accounts. These include idiopathic occipital epilepsies, tumors, infectious diseases, and brain trauma.

Both a posterior reversible leukoencephalopathy secondary to arterial hypertension and a non-ketotic hyperglycemia seem unlikely [Harden, 1991] as there is no evidence that Flaubert ever had any of the other symptoms or complications of hypertension or diabetes.

A cortical malformation or a mitochondrial disorder (e.g. MERFF or MELAS) are usually expressed by severe epilepsy with frequent seizures and associated with features that are not evident in the available accounts.

Finally, the two possibilities suspected by Gastaut are the most convincing: (1) an arterial or arteriovenous malformation (fig. 1) which would explain the fluctuation in the recurrence of the seizures – frequent seizures at the beginning in relation with a leakage of the malformation, then decreasing frequency of seizures during the quiescent period. This would also be consistent with the accounts of his death is we listen to Flaubert's doctor who referred to a 'cerebral congestion'. (2) An occipito-temporal cerebral atrophy, the origin of which remains obscure but could explain why Flaubert, as a child, had trouble learning to read.

The Role of Flaubert's Illness in His Life and His Works

It is clear that throughout Flaubert's life, he and his family hid his epilepsy from others. Only his friend Maxime du Camp revealed the secret. When Flaubert speaks about his attacks, he calls it 'nerve sickness' or uses other euphemisms.

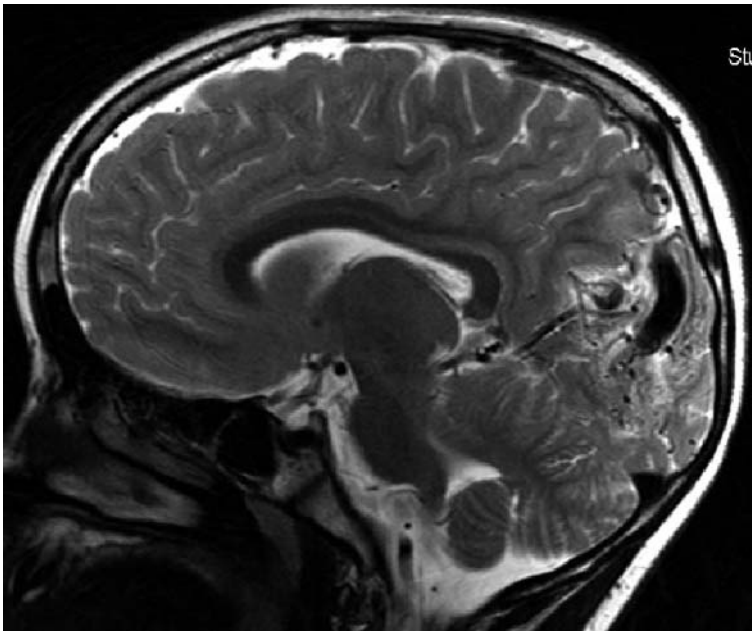

Fig. 1. An occipital arteriovenous malformation which could explain Gustave Flaubert's epilepsy.

For example, in a letter sent to Feydeau, October 1858, he wrote: 'I have the dark sickness. I had it once before as a youth. It lasted for 18 months and I very nearly died from it. It went away then, it will go away this time. We must have faith'. He referred to epilepsy only twice in his correspondence but surprisingly not in reference to his own illness: 'It is impossible to continue a correspondence which becomes epileptic [to Louise Colet, 1846]; 'Actually, Paris is completely epileptic' [to George Sand, 1871]. In Flaubert's writings, epilepsy is completely sublimated. Unlike Dostoevsky, Flaubert never used it in his works although he had planned to write a novel about his illness, *La Spirale,* which was never finished or published: 'My nervous sickness has been helpful to me in that it converted all those feelings into physical symptoms and left me with a cooler head. Furthermore it acquainted me with peculiar psychological phenomena that no one has ever envisioned, or rather that no one has ever experienced. Some day, I will have my revenge, in a book (that metaphysical novel with spirits about which I spoke to you). But that subject frightens me, speaking from the medical point of view. I must wait until I'm sufficiently distant from such feelings to be capable of using them factitiously as symbols, projections, without danger to myself or the book'.

Above all, Flaubert's epilepsy was kept a secret and even stigmatized by his family which included two physicians, his father and his brother: 'When I arrived in Rouen, Flaubert's father was laboring under the burden of his distress. The evidence of this could be plainly seen in the expressions on his face. One could see the humiliation and despair. He was resigned in the face of a force he was unable to control. For all of his scientific knowledge, he was powerless, and as a father who loved his son, he suffered all the more' [*Souvenirs Littéraires,* p. 220]. As Daniel Oster remarked, 'Flaubert's illness without being a secret, was treated as though it were one. Like something that is valued only because it is forbidden.'

Flaubert's epilepsy certainly influenced his decision to abandon his studies of law and reinforced his literary bent. After his first seizure, he entrusted Croisset to publish his works:

'My illness has benefited me in that I am allowed to spend my time as I like, a great thing in life. For me, I can imagine nothing in the world preferable to a cozy, well-heated room with one's favorite books and all the leisure time one could desire' [letter to Emmanuel Vasse, January, 1845].

'These nervous diseases are contagious anyway and maybe I have needed a robust constitution in order to resist the pounding of my nerves on the drumskin of my mind. For myself, I have an outlet (as one says in medicine). The paper is here and I am relieved. But the dankness/darkness of my spirits can escape and, in the end, do me harm' [letter to Louise Colet, June 1st, 1853].

'Once again so alone! I am so sick that I find a way to get better. Long ago, I learned not to ask for anything more. After all, what do I truly need if not my freedom and leisure to do as I please? I have deliberately deprived myself of so many things that I now feel rich in the midst of my destitution' [letter to Alfred le Poitevin, July, 1845].

It is obvious that Flaubert's seizures did not cause any cognitive impairments or 'mental deficiency'. His literary work is testimony to this fact that someone with epilepsy can live with his disease, even if he keeps it hidden, and can become one of the most widely read writers of the 19th-century French literature. We must re-emphasize the striking lack of any reference to his illness in Flaubert's work. This absence distinguishes him from writers such as Dostoevsky [Sutterman, 1993], Daudet [Dieguez and Bogousslavsky, p. 17–45] as well as Proust and forces us to reconsider the relationship between illness and literary creativity. Even while absent from his writings, his illness most certainly had an influence on Flaubert's identity as a writer, the feeing of exclusion, his solitary life, etc. Even if we hesitate to attribute a cause-effect relationship between 'illness' and 'genius' it is obvious that his affliction played a decisive role in creating the conditions under which Flaubert created his masterpieces.

Short Biography on Flaubert

1821: Born December 12.
1832: Schooling at the Rouen College.
1841: Begins his studies in Law in Paris.
1843: Begins the first chapter of *La première Education sentimentale*. Meets Maxime Du Camp with whom Flaubert had a tumultuous friendship.
1844: Suffers his first epileptic seizure. The whole family goes to live in Croisset. Flaubert abandons his Law studies.
1846: In January, Flaubert's father dies. In March his sister, Caroline, dies, leaving behind a daughter also named Caroline. In July, Flaubert meets Louise Colet, who will be his love for a period of 3 years.
1847: Takes a 3-month trip with Maxime du Camp in Anjou, Brittany and Normandy.
1849–1851: Long journey to the Middle East with Maxime du Camp.
1851: September, begins the novel '*Madame Bovary*'.
1857: Trial and acquittal for '*Madame Bovary*'.
1862: April, ended '*Salammbô*'.
1869: May, ended '*L'Education sentimentale*'.
1870: Croisset is occupied by the Prussians, Flaubert and his mother in Rouen.
1872: Ended the last version of '*La Tentation de saint Antoine*', published in 1874.
1875: Begins the publication of his last novel '*Bouvard et Pécuchet*'.
1875–1876: Writing '*Les trois contes*'.
1880: Dies on May, 8th.

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Edgar Allan Poe: Substance Abuse versus Epilepsy

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Abstract

Edgar Allan Poe was by all accounts one of the most talented of American storytellers, with many of his tales based on characters with episodic unconsciousness, confusion, and paranoia. Similar episodic behavior is well known to have taken place in Poe's life as well, and is generally attributed to alcohol or drug abuse. There is considerable evidence, however, that Poe suffered not from simple substance abuse but from complex partial seizures, possibly followed by prolonged confusion and postictal psychosis. Complex partial seizures were not well described in Poe's time, which could explain a misdiagnosis. Alternatively, he may have suffered from complex partial epilepsy which was complicated or caused by substance abuse. In any case, his personal experiences clearly influenced many of his major works. He represents a fascinating case of a neurological condition in a talented individual, who was then able to use his experiences to give deeper meaning and poignancy to his art.

Although Edgar Allan Poe (fig. 1) is one of the most famous and celebrated of American storytellers, misfortune, alcohol use, and drug abuse plagued his life. These were believed to account for his episodic alterations in consciousness and intermittent paranoid behavior. Descriptions of these spells, however, are also compatible with complex partial seizures; a diagnosis which was not considered in his time, perhaps because epilepsy was still a mysterious and poorly understood condition. Personal experiences probably influenced Poe to feature characters with intermittent lapses of awareness and alterations in behavior in a number of his works. He shows a poignant perspective on the blurring of reality and dream, of consciousness and unconsciousness, and of life and death through these experiences of his characters.

This review will first highlight the events in Poe's life, particularly descriptions of his episodic alterations in consciousness. Second, descriptions from

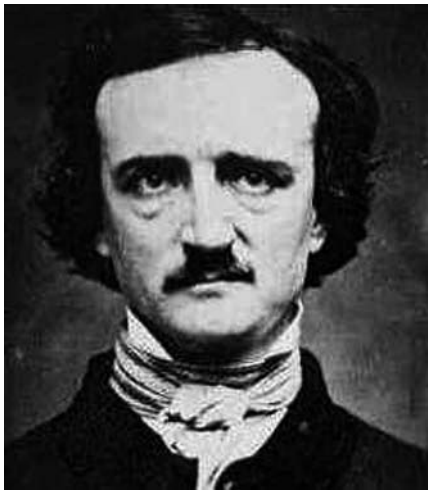

Fig. 1. Edgar Allan Poe. Photograph of a daguerreotype by W.S. Hartshorn, 1848. Library of Congress, Prints and Photographs Division [LC-USZ62-10610].

many of his works suggest experiences compatible with epilepsy. Finally, this information will be tied together in a discussion of whether he actually suffered from epilepsy.

Historical Background of Poe's Life

Edgar Poe was born in Boston on July 19, 1809 of two actors: David Poe and Eliza Arnold. His parents both died before he was 3 years old, and Edgar was adopted by a well-to-do, childless couple, John and Frances Allan; their surname became his middle name. His younger sister, Rosalie, was mentally retarded, while his older brother William drank heavily and died at a young age [Cumston, 1909]. Alcoholism may have begun at a young age for Edgar and his siblings, as their Irish nursemaid regularly gave them bread soaked in gin, and sometimes laudanum, in order to quiet them [Meyers, 1991]. In grade school and high school Poe excelled in academics and athletics, and in 1826 he enrolled in the University of Virginia but was forced to leave shortly afterward because of charges of gambling and unpaid debts to a tavern keeper. He enlisted in the military, and within 19 months achieved the highest rank attainable by an enlisted man: sergeant major. He was appointed to the US Military Academy in West Point, New York, initially doing well; however, he became despondent and irritable, and he was dishonorably discharged in March 1831 for failing to report for morning muster [Cumston, 1909].

Poe moved in with his aunt and her daughter, Virginia. In 1836 he married Virginia, then aged 13 [Knapp, 1984] (marriage between first cousins was not prohibited at the time). He took a job in Baltimore as an editor, but was forced to resign this position, too, allegedly due to alcoholism. He was a brilliant critic, and worked at several other magazines where he was very productive for a time, especially *Graham's Magazine*, which allowed him to finally pay his debts. His reported abuse of alcohol and opium worsened after the death of his wife in 1847 [Meyers, 1992]. By 1849, Poe was again engaged to be married and made an effort to remain sober. Upon returning to Baltimore from New York, Poe had a single drink. Soon after, he was found unconscious on a street and was taken to a hospital. After a brief lucid period, during which he complained of a headache, he became restless and delusional, lapsed into a coma, and died. His physician, J.J. Moran, wrote 'When brought to the hospital he was unconscious of his condition – who brought him or with whom he had been associating… To this state succeeded tremor of the limbs … and vacant converse with spectral and imaginary objects on the walls…his answers were incoherent and unsatisfactory… I found him in a violent delirium… until three on Sunday morning. At this time… he became quiet, and seemed to rest for a short time; then gently moving his head, he said "Lord help my poor soul!" and expired' [Harrison, 1970]. Speculations concerning the causes of his death have included encephalitis, delirium tremens, pneumonia [Scarlett, 1978], rabies [Benitez, 1996], head trauma [Patterson, 1992], and diabetic coma [Hill, 1968].

Evidence for Epilepsy

There is no question that Poe suffered from episodes of altered behavior, becoming frequent at about age 20 but probably beginning earlier. Some descriptions clearly imply binge drinking: 'He drank to excess, became coarse and vulgar, fell into fits of the deepest gloom… followed by several days of sickness' [Robertson, 1921]. John Robertson, a physician who extensively studied his medical history in the early 20th century, describes 'seizures' from which Poe tried to escape with alcohol or opium. 'When these seizures pass and the patient recovers, there may be… complete loss of memory. During the attack there is usually loss of self-control and an abnormal ideation' [Robertson, 1921]. Despite these descriptions, however, it is difficult to explain most of his episodes based on substance abuse. The quantities he consumed frequently seemed too small even for intoxication, and the binges too short to have resulted in withdrawal symptoms. Friends suggested that 'If he took but one glass of weak wine, or beer, or cider, the Rubicon of the cup had been passed with him, and it always ended in excess and sickness'. Perhaps the best description of his

paroxysmal behavioral changes came from another friend: 'I,… while walking with Poe, and feeling thirsty, pressed him to take… a glass of ale with me. Almost instantly a great change came over him. Previously engaged in an indescribably eloquent conversation he became as if paralyzed, and, with compressed lips and fixed glassy eyes, returned, without uttering a word, to the house which we were renting. For hours the strange spell hung over him. He seemed a changed being, as if stricken by some peculiar phase of insanity' [Robertson, 1921]. This sudden behavioral change is entirely compatible with a complex partial seizure.

Further supporting that these could have been epilepsy, spells were known to occur without any alcohol. 'Occasionally, even without any stimulant, there may develop an abnormal mental condition, the so-called change in personality which we so freely discuss without any real knowledge as to how it does occur' [Robertson, 1921]. Dr. Robertson believed that Poe's condition was not epilepsy because he did not completely lose consciousness and never suffered from a grand mal seizure; however it is now well known that complex partial seizures are quite common, and many of these patients never have grand mal seizures.

The semiology of Poe's spells, including staring, compressed lips, personality changes, mutism or speech changes, and amnesia is suggestive of complex partial seizures. The quiet confusion and behavioral changes described are most consistent with temporal lobe onset seizures. Alcohol-sensitive epilepsy has been reported commonly although never rigorously proved [Hauser, 1988]. It seems clear, however, that Poe's behavioral alterations were not simply due to intoxication as they also occurred without alcohol, although complex partial seizures do not usually persist for hours or even days, as described in Poe's case. Nonetheless, 'twilight states' or complex partial status epilepticus might have been responsible. Alcoholism could also have led to head injuries (particularly temporal lobe contusion) that subsequently resulted in post-traumatic seizures [Hauser, 1988].

Besides these attacks, Poe was known to have intermittent paranoia and even psychotic behavior. He had described Longfellow as one of the greatest American poets, then repeatedly accused him of plagiarism. In 1849, shortly before his death, Poe visited the office of John Sartain, an editor, and informed him that he had heard people on the train plotting to kill him. Poe even requested a razor to shave his whiskers so that he wouldn't be recognized. He talked incessantly and hallucinated a blessed feminine presence speaking to him. Similar episodes of hallucinosis during the last few months of his life have been attributed to delirium tremens even though they seemed to follow binge, rather than chronic, bouts of drinking. More likely, given his probable seizures, these could have arisen from postictal psychosis. This well known condition

typically begins 24–48 hours following a seizure or cluster of seizures, and can last for weeks. During this time patients can be actively hallucinating, and suffer from delusions and paranoid ideation.

Poe's death could also have been related to seizures. Mrs. Marie Louise Shew, the daughter of a physician who attended Poe and his wife during the latter's illness, wrote that Poe had a lesion on one side of his brain which was responsible for bouts of 'insanity' whenever he took alcohol or drugs. Tuberculosis was prevalent at the time, and in fact caused the deaths of Poe's mother and wife. Poe's subacute deterioration could have been caused by tuberculous meningitis or an expanding brain lesion, either of which could have resulted in frequent or prolonged complex partial seizures and subsequent death.

Evidence for Poe's Possible Seizure Disorder from His Writings

Episodic alterations of consciousness appear frequently in Poe's prose and poetry. A review of these works further suggests that Poe had personal experience with a seizure disorder. In *The Premature Burial*, Poe states that the author of this tale is subject to attacks of 'catalepsy':

'Sometimes, without any apparent cause, I sank, little by little, into a condition of hemi-syncope, or half swoon; and, in this condition, without pain, without ability to stir, or, strictly speaking, to think,… I remained, until the crisis of the disease restored me, suddenly, to perfect sensation. At other times I was quickly and impetuously smitten. I grew sick, and numb, and chilly, and dizzy, and so fell prostrate at once…' [Poe, 1992].

The horror of this tale is awakening in belief that he is buried alive. In his terror he screams, and is brought to his senses by companions. He finally realizes that he is in the tiny berth of a small sloop, not a casket.

These episodes are difficult to diagnose. Their gradual onset, very long duration, and absence of movement argue against epilepsy. It may be that in this example, Poe is giving an exaggerated version of his own spells with speculation about what transpired while he was unconscious. The character in this story is terrified of his lapses of consciousness, as are many patients with epilepsy. In the end, however, he chooses to live life fully, and with this change in mindset his disease actually improves.

The Pit and the Pendulum relates the horrors of a man tortured by the Spanish Inquisition. The beginning, however, offers a description that sounds very much like an epileptic seizure seen through the eyes of a literary genius:

'…then, all at once, there came a most deadly nausea over my spirit, and I felt every fiber in my frame thrill as if I had touched the wire of a galvanic battery, while the angel forms became meaningless specters… the blackness of

darkness supervened; all sensations appeared swallowed up in a mad rushing descent as of the soul into Hades. Then silence, and stillness, and night were the universe' [Poe, 1992].

The narrator then speaks as one who has had such experiences before, implying that this 'swoon' was not due entirely to *The Inquisition*:

'He who has (never) swooned, is not he who finds strange palaces and wildly familiar faces in coals that glow; is not he who beholds floating in mid-air the sad perfume of some novel flower; is not he whose brain grows bewildered with the meaning of some musical cadence which has never before arrested his attention' [Poe, 1992].

He seems to indicate some deeper beauty and insight which comes with these events, as have some artists with epilepsy who describe feelings and images from their seizures in their expressions including Dostoevsky [Clarke, 1915; Gastaut, 1984] and van Gogh [Monroe, 1978].

In *Berenice*, both the author and his cousin suffer from '…a species of epilepsy not infrequently terminating in trance itself'. Later in the tale, Berenice was 'seized with epilepsy in the early morning, and now, at the closing of the night the grave was ready for its tenant' [Poe, 1992]. In the ultimate tragedy of this grisly tale, the author finds himself suddenly overcome with horror, believing that he has been dreaming and has done something which he can't remember. He then discovers that he is covered in blood, and that he has dismembered his cousin and buried her alive. It is the only story in which Poe uses the term epilepsy. Again, the story may reflect some of Poe's own fears about his disease, which are shared by may persons with epilepsy: What happens during the time he is unaware? Is he capable of doing something horrible, unthinkable? Could he be buried alive while in a deep, postictal state?

In *The Sphinx*, the narrator relates a vision of huge dimensions consistent with the macropsia seen in temporal lobe seizures. In *Eleonora* Poe talks of those 'who dream by day' and suggests this brings insight. In *The Narrative of Arthur Gordon Pym of Nantucket* there are a number of episodes of prolonged altered consciousness. One could be interpreted as a generalized tonic-clonic seizure:

'…suddenly, a loud and long scream or yell, as if from the throats of a thousand demons… without having once raised my eyes to learn the source of my alarm, I tumbled headlong and insensible upon the body of my fallen companion' [Poe, 1992].

Conclusions

During his lifetime, Edgar Allan Poe suffered from repeated, prolonged attacks of altered behavior. The differential diagnosis for such episodic behavior

includes substance abuse, syncope, schizophrenia, complex partial seizures, and perhaps more unusual illnesses such as porphyria or idiopathic recurring stupor.

Poe's intermittent alterations of behavior have been attributed to alcohol and opium abuse. There is, however, clear evidence that similar or identical episodes occurred with minimal or no alcohol intake. This suggests a chronic, recurrent, underlying condition that was exacerbated by alcohol. The prolonged attacks are not compatible with syncope, and the episodic nature of the symptoms is not characteristic of most psychiatric illnesses. His lifelong pattern of productivity interrupted by periods of confusion and despair could certainly represent an alcoholic tendency. Most likely, Poe suffered from complex partial seizures, perhaps complicated by prolonged postictal states, including postictal psychosis, and episodes of complex partial status epilepticus. This condition could certainly be exacerbated by alcohol, even in small amounts.

Poe's death at a relatively young age is also a mystery. He clearly did not die of prolonged generalized seizure activity. If, in fact, he suffered from bouts of complex partial status epilepticus this condition is known to have a high mortality, which is dependent on the underlying condition [Krumholtz et al., 1995]. He could also have died from a condition known as sudden unexplained death in epilepsy, which does occur in the absence of generalized tonic-clonic seizures [Shorvon, 1997] although his last days seem to have contained a worsening condition rather than an abrupt death. Because no autopsy was performed, he may have suffered from a progressive lesion of the central nervous system, one manifestation of which was seizures.

Poe's works describe a number of characters with episodic altered consciousness and behavior, which probably reflects some of his own experiences. Some of these also have characteristics of epilepsy, including epigastric auras, staring, and macropsia.

Because there is only indirect information, we can never know for certain whether he suffered from epilepsy. His physicians at the time should not be faulted in any case; complex partial seizures were initially described in the mid-19th century by John Hughlings Jackson [1889] and not widely accepted until sometime after. Seizures were thought to occur only with complete loss of consciousness. If he did suffer from epilepsy, this may unfortunately have accounted for many of the descriptions of intoxication, and his continued reputation as an abuser of drugs and alcohol. While we hope that many of these prejudices and misinterpretations are improved today, there are still many epilepsy patients who are misdiagnosed with substance abuse and psychiatric disease. Whether or not he actually had epilepsy, his work reflects many of the experiences, fears, and hopes of patients with the disease. Whatever his own disease, it contributed heavily to his creative genius, and Poe was able to overcome its

limitations and use his experiences to become one of the most celebrated American storyteller.

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Dostoevsky and Epilepsy: An Attempt to Look Through the Frame

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Abstract

Fyodor Mihailovich Dostoevsky, one of the greatest novelists of all times, had a unique ability to depict the social and moral conditions of 19th-century Russia, and anticipated with his writings many philosophical and scientific developments, such as existentialism and psychoanalysis. The study of auto- and hetero-biographic reports, as well as of his literary production, suggests that he suffered from epilepsy since his mid-20s. Most of his seizures were described as generalized convulsive; however, many features, such as the presence of an 'ecstatic' aura, a pallor preceding the generalization, and especially a postictal dysphasia, point to a dominant (mesio-) temporal lobe origin. Although Dostoevsky in his late years complained of progressive memory impairment, he was able to write with his usual cleverness until the end of his life, when he succumbed to a chronic lung disorder. From an epileptological point of view, this uncommon relatively benign evolution is noteworthy, and it offers an insight to the natural course of this illness. A natural course that is mostly unknown in our times, as the availability of antiepileptic drugs and surgery has biased our knowledge. The relationship between epilepsy and Dostoevsky's production is twofold. Firstly, as he had a tendency towards autobiographical descriptions not only of facts but also of thoughts, his novels represent a valuable source for the understanding of his illness. Secondly, his works contribute greatly to the de-stigmatization of patients with epilepsy.

'…ma le fotografie chiudono il visibile in un rettangolo. Il visibile senza cornice è sempre un'altra cosa.'

('…but photographs constrict the visible in a rectangle. The visible without frame is always something else.')

Antonio Tabucchi
Notturno Indiano, 1984

The work of Dostoevsky, a novelist, journalist and short-story writer, is considered to be one the most impressive, not only among the Russian, but definitely also within the world literature. His powerful and finely tuned depictions of the human condition in all its variety, and his profound philosophical, psychological and religious insights of human nature are characteristic for his style. His novels anticipated and influenced several developments of the 20th-century ways of thinking, including psychoanalysis and existentialism. The writer's own troubled life, combined with a particularly fruitful background, enabled him to reach the sublimation of literary work.

Since his young years, Dostoevsky had recurrent seizures, thus fulfilling the definition of epilepsy. What type of seizures did he suffer from? And what influence did his illness exert upon his literary production?

If a modern Physician would be requested to find an answer to these questions, he would take the history, perform a physical examination of the Patient, order some ancillary tests, then formulate a differential diagnosis and, if necessary, prescribe a treatment. As the Physician is working more than a century after Dostoevsky's death, he only has to deal with 'second-hand' history taking. Furthermore, the irreversible passage of time prevents him from asking his Patient anything.

Our Physician has to keep his objectivity, although his work will be dominated by the 'problem of the frame': he only enjoys a limited view over the reality of the past (fig. 1).

Let's follow him during his attempt.

Personal Medical History

Fyodor Mihailovich Dostoevsky was born in Moscow in 1821, at the hospital where his father worked as a medical doctor. The Patient was the second of seven siblings [Siegel and Dorn, 2001]. At the age of 7, he had an episode of auditory hallucination (a frightened scream), possibly related to a mysterious upset event that happened in his family [Alajouanine, 1963]. In 1837, his mother died; in the same year, he suffered from some type of 'nervous breakdown' [Vallery-Radot, 1956], and he had a transient aphonia, probably of functional origin [Voskuil, 1983]. Two years thereafter, his father passed away.

After completing his training as naval engineer in St. Petersburg, the Patient began to work for the Ministry of War (1843). During that period, he began to focus on literature. These years were characterized by a difficult financial situation, further complicated by his passion for gambling [Siegel and Dorn, 2001].

According to most sources, the Patient experienced his first epileptic attack, a 'violent seizure' during a funeral, in 1846, possibly after an illness

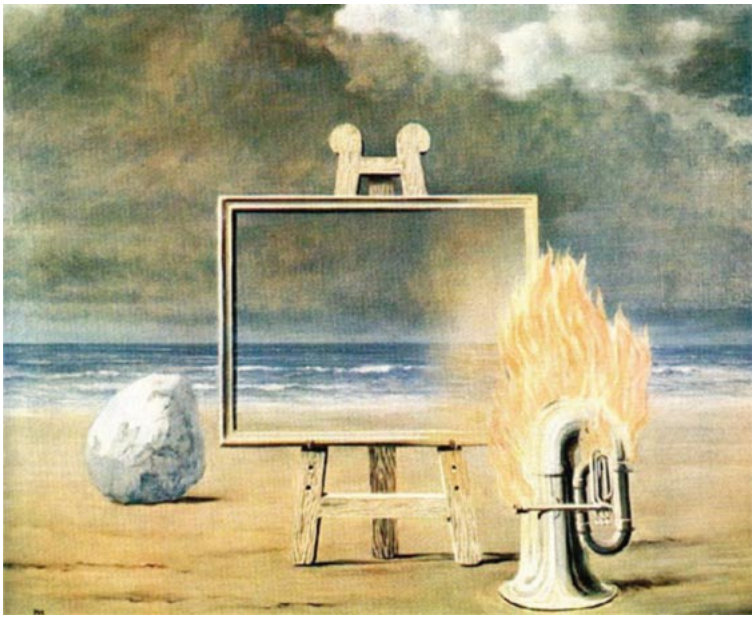

Fig. 1. René Magritte's *The Fair Captive* exemplifies this problem: only the marina, but not the stone, is seen through the frame. Copyright © ProLitteris, 2005, CH-8033 Zürich.

with 'an irritation of the entire nervous system, … the ailment attacked the heart…' [Voskuil, 1983]. In the same year, at a party, his face 'changed queerly, and a frightened look came into his eyes. A few minutes passed, and then in a hollow voice he asked: "Where am I?".' He went out of the room, but then he was found shaking [Voskuil, 1983]. Dr. Janowsky, a friend of his, described him as 'below medium height, broad boned…' (fig. 2), with an irregular pulse, and traces of ancient rickets and tuberculosis. This doctor mentioned further generalized attacks, sometimes heralded by an expression of terror [Voskuil, 1983]. In spite of these reports, the seizures of that period have been described as relatively mild [Alajouanine, 1963; Siegel and Dorn, 2001].

In 1849, Dostoevsky was found guilty of conspiracy with a revolutionary movement. At the beginning of the imprisonment, he wrote that his 'nerves were breaking down' [Vallery-Radot, 1956; Mayer, 1992]. He was sentenced to death, but at the very moment of the execution, the penalty was commuted to deportation to Siberia. This episode will mark our young Patient for his life. Those long years allowed him close contact with people of the most varied social conditions, and thus represented a key experience for his forthcoming literary work [Vallery-Jadot, 1956; Siegel and Dorn, 2001]. During that extremely difficult period he had many generalized convulsions. Although these have been regarded as the beginning of

Fig. 2. A rare photograph of F. M. Dostoevsky at the age of 40. Reproduced with kind permission of Aleksey Astrahantsev, www.dostoevsky.df.ru.

grand-mal seizures [Siegel and Dorn, 2001], they most likely represented an increase in frequency. In a letter to his brother Andrej, he mentioned his recurrent seizures as well as attacks of stabbing headache (1854) [Vallery-Jadot, 1956]. A Military Doctor, Major Ermakov, wrote in 1857 that the Patient had '… a quite weak constitution. In 1850 he experienced his first epileptic seizure, with shouting, memory impairment, muscle twitches, foam on his lips, difficulty of breathing, and rapid pulse. The attack lasted for 15 minutes, followed by the habitual exhaustion. In 1853 he had another attack, and since then, they recur regularly at the end of each month…' [Voskuil, 1983; Siegel and Dorn, 2001]. At the end of his prison sentence, still in Siberia, he married Maria Isayeva in 1856. During their honeymoon, he had two generalized seizures, probably triggered by sleep deprivation and champagne; he was told for the first time that he suffered from epilepsy [Voskuil, 1983]. At that time, he recognized that his recurring epileptic attacks 'cloud my memory … I fear they are leading me to dementia…' [Vallery-Jadot, 1956]. Although the Patient experienced most of his seizures during the night breaking his sleep [Gastaut, 1978], several were witnessed when he was awake.

One of his intimate friends, Strakhov, described: *'*…normally the attacks came once a month, sometimes more often …I experienced one of his attacks, I think, in 1865… we had a lively talk, … he began to be agitated, … excited…he opened his mouth to say something, I looked at him with close attention. Suddenly a strange, long, aimless scream came out of his open mouth and he fell to the floor, unconscious… his body twitched and his mouth became foamy… Dostoevsky told me that just before the seizures he used to become ecstatic…*'* [Siegel and Dorn, 2001]. Another friend, Mrs. Kovaleskaya, recalled that Dostoevsky compared his ecstatic aura, a feeling of absolute harmony and happiness, to Mahomet's vision of Paradise: 'I don't know if this felicity lasts for seconds, hours or months, but … for all the joys that life may bring I would not exchange this one!' [Alajouanine, 1963]. Prince Myskin (*The Idiot*) will make a remarkably similar statement. Further descriptions of the aura were related by Baron Wrangel [Voskuin, 1983]. The Patient also reported a sensation of bilateral twitching and 'itching in the hands' at the beginning [Gastaut, 1984; Siegel and Dorn, 2001], and speech impairment after the seizures: 'It was a long time before I could speak', and 'When writing, I still made mistakes with the words' [Alajouanine, 1963]. His first wife wrote about another seizure: '… suddenly he turned pale, lurched on the divan and began to lean over to my side…suddenly came a fearful cry…', followed by convulsions and postictal confusion [Voskuil, 1983].

After having resumed his artistic activity, the Patient was allowed to come back to St. Petersburg in 1859. As our Physician has noticed, the frequency of his seizures clearly worsened during and after his Siberian period [Alajouanine, 1963; Siegel and Dorn, 2001].

In 1862, Dostoevsky undertook his first journey across Europe; during those weeks he discovered the 'corrupted' nature of Western Europeans, leading to a reappraisal of the 'Russian spirit'. He began a second trip to Western Europe in 1863, where his gambling mania abruptly (but transiently) ended the relationship with his beloved Apolinarya Suslova. The following year brought two tragedies: the death of his wife (tuberculosis) and of his beloved brother Mikhail. Later, a third European journey ended again with a clash with Mrs. Suslova [Siegel and Dorn, 2001].

Forced into it by his financial situation, Dostoevsky accepted a tough editorial contract, compelling him to write a novel in a very short time. The stenographer Anna Snitkina helped him finish the work (*The Gambler*), which shares with many other novels a strong autobiographic trait. She married him at the beginning of 1867. A new peregrination through Europe was initiated shortly thereafter. In Basel he saw the Holbein painting of the Deposition, which will be mystically depicted in *The Idiot*: his wife wrote that in the museum he got a 'scared expression on his face, as I saw so often during the first minute of his

epileptic attacks', but fortunately it did not end up with a generalization [Schischkin, 2003]. However, in Geneva he experienced a prolonged convulsion as his wife started to go into labor, followed by a long postictal period [Mayer, 1992; Schischkin 2003]. His beloved daughter Sonja was born safely shortly thereafter, but she died tragically at the age of 3 months of an acute illness [Mayer, 1992].

Back to Russia, Dostoevsky was experiencing the exacerbating effects of stress on his seizures [Voskuil, 1983]. He published two masterpieces: *The Idiot* and *The Possessed*. Thereafter, he continued to write regularly, even if during his 50s he began to suffer from increasing breathing difficulties [Siegel and Dorn, 2001], for which the Patient underwent several cures in Bad Ems [Voskuin, 1983]. It seems that then his seizures tended to recur less often [Voskuin, 1983; Siegel and Dorn, 2001]; however, he complained in 1870, after one episode that: '…it is four days now, and my head is still not fresh…impossible to think or to work' [Gastaut, 1978], as well as of psychic depression lasting several days [Voskuin, 1983]. Noteworthy, he also reported significant memory impairment [Voskuin, 1983]. In his last years he used to take opium solutions, but this apparently was not intended to cure his epilepsy, for which, besides some bloodletting by Dr. Janowsky, he did not receive any specific treatment [Voskuin, 1983].

One year after completion of his last great work, *The Brothers Karamazov* (1880), he succumbed to his lung disorder, probably emphysema or tuberculosis [Mayer, 1992; Siegel and Dorn, 2001]. Remarkably, his epileptic seizures did not play a role in the etiology of his death.

Family History

The father had a tendency towards alcoholism, as did many members of his family, and he was described as a severe, pedantic, and stingy man. Early reports claimed that he was murdered by his servants [Vallery-Radot, 1956]. However, newer interpretations point to a hemorrhagic stroke [Voskuil, 1983]. The mother died of tuberculosis [Siegel and Dorn, 2001]. One of his sons died in status epilepticus during an acute illness at the age of three [Siegel and Dorn, 2001].

Physical Examination

Apart from the vague descriptions by Drs. Janowsky and Ermakow, our Physician did not find any pertinent medical records.

Complementary Examinations

Due to an insurmountable chronological problem, the Patient was unfortunately not able to show up at the right time for the scheduled EEG and MRI, or to have a blood screening.

Discussion

Our Physician will summarize the facts.

In contrast to Gastaut [1978], he does not find any evidence of a family history of epilepsy. In particular, the fatal status epilepticus of Dostoevsky's son was most likely symptomatic of an infectious illness [Voskuil, 1983]. The Physician also assumes that the birth and early development of the Patient were normal. The episode of verbal hallucination during childhood has been thought to represent the first partial seizure [Vallery-Radot, 1956]; however, our Physician tends to downplay it, as this appears to be isolated, and the Patient never reported acoustic sensations before his convulsions.

In his early years, Dostoevsky's transient aphonia and 'nervous breakdowns' suggest a neurotic trait [Voskuil, 1983], although the possibility of some ill-defined auras (simple partial seizures) cannot be ruled out with certainty. These elements, together with his putative animosity towards his father, induced Freud to interpret the parricide of *The Brothers Karamazov* as expression of an Oedipus complex; furthermore, the Austrian psychoanalyst regarded his seizures as psychogenic, since Dostoevsky was thought to have experienced his first convulsion at the moment when he was told of his father's death [Gastaut, 1978; Siegel and Dorn, 2001].

Our Physician's sources, nevertheless, clearly describe that the first documented seizure occurred in 1846, at the age of 25, thus 7 years after that episode. Several descriptions by his friends and relatives leave no doubt that Dostoevsky had 'organic' generalized seizures, with tachycardia, jerking of all extremities, long postictal periods, and triggering by stress, alcohol and sleep deprivation. This semiology, the notion of epilepsy of Dostoevsky's son (as we have seen, probably an incorrect assumption), the nocturnal occurrence of most episodes, the preceding bilateral twitches, the absence of a major psychiatric or neurological disease, and the questioning about the real existence of the ecstatic aura, led Gastaut [1978] to postulate an idiopathic generalized epilepsy (IGE), as 'petit-mal – grand-mal'.

However, other data need to be carefully evaluated.

Our Physician points to the fact that although arrest of speech and of activity may be seen in absences, they are well known in partial complex seizures.

Furthermore, nocturnal seizures are characteristic not only of IGE or frontal lobe epilepsy, but are also reported in temporal lobe epilepsy (TLE) [Bernasconi et al., 1998]. The postictal depression described by the Patient is interesting: this may be interpreted as a Todd's phenomenon [Stagno, 2001], but even if studies in stroke patients suggest a lateralization towards the (damaged) dominant hemisphere [Carota et al., 2001], the localizing value in epileptic patients is unclear. Headache is not localizing [So, 2001], apart from childhood benign epilepsy, and it is unclear whether the Patient suffered from peri-ictal or interictal pain; in the latter case, a migraine (which is somewhat linked to epilepsy), or a more common tension headache might be postulated. Dostoevsky's 'peculiar' ecstatic aura, although far less frequent than feelings of fear, has been convincingly described in patients with partial epilepsy [Cirignotta et al., 1980], and is thought to be encountered in about 2% of patients undergoing presurgical workup; it points to a mesiotemporal origin, is non-lateralizing [Stefan et al., 2004], and may suggest amygdalar involvement. It remains unclear whether the patient also experienced frightening auras, as a facial expression of 'fear' could simply reflect astonishment during the ecstatic feeling. Tingling over the midline or the entire body is recognized as a prodrome of TLE [Wieser, 2004], as generalized tremor [Gastaut, 1984], and autonomic dysfunction [Voskuil, 1983; Wieser, 2004], the latter possibly involving spread to the insula. The postictal aphasia represents the most important localizing feature, pointing towards the frontotemporal dominant hemisphere [Wieser, 2004]. Dostoevsky's complaints of worsening memory are well compatible with a chronic TLE, especially after 30 years of illness [Wieser, 2004]; his higher level of intelligence and education probably account for the fact that this aspect did not impair the Patient to the point of hindering his literary activity [Oyegbile et al., 2004]. Finally, one issue is clear for our Physician: the natural history of TLE is basically unknown, since this disorder (especially in association with hippocampal sclerosis) has been described only in the era of antiepileptic treatments. However, 'benign' courses clearly exist [Wieser, 2004], as is suggested by an increasing, but then decreasing seizure frequency throughout the life of Dostoevsky. Our Physician is also thinking of the fact that the Patient did not seem to suffer from hyposexuality, a relatively common issue in (treated) patients with TLE [Gastaut, 1984].

Dostoevsky was an excellent observer not only of his surrounding word, but also of himself. In his works he depicted his experiences, thoughts, beliefs, as well as his illness. As our Physician believes that the Patient is talking to us through his novels, his literary descriptions may further help to understand his disorder.

There are six epileptic characters in his novels [Siegel and Dorn, 2001]. The most famous is Prince Myskin, *The Idiot* (1868), considered the interpreter

of Dostoevsky's thoughts [Alajouanine, 1963]. As his 'inventor', he has ecstatic auras, and in the middle of the novel (part 2, chap. 5) possibly a long-lasting twilight state, characterized by a sort of 'forced thinking', as he recurrently feels a particular unpleasant thought, which precedes a (saving) generalized seizure. Forced thinking may be related to auras arising from the dominant frontal or temporal lobe, the former associated with a more verbal, the latter, as in Myskin, with a more emotional feeling [Mendez et al., 1996].

In the early work, *The Lodging Woman* (1847) the old Murin, an evil person, in whom the seizure inducing property of alcohol consumption is described, has an attack at the very moment he's attempting to murder Ordynov, an artist experiencing ecstatic feelings. This suggests that the patient already had those auras at the beginning of his illness. In *The Insulted and Injured* (1861), Dostoevsky describes in detail a generalized seizure and the postictal period of the peaceful girl Nelly. In *The Possessed* (1872) Kirillov, an atheistic mystic, also experiences ecstatic prodromes. Finally, in *The Brothers Karamazov*, Sverdyakov, the parricide, suffers from epilepsy, he constructs his alibi mimicking a status epilepticus, but his seizures worsen after the murder, and, seeing the Devil in his hallucinations, he hangs himself.

Conclusion

What type of seizures, respectively of epilepsy, did Dostoevsky suffer from? And how did his illness act on his literary production?

The Patient's disorder has been analyzed by several prominent epileptologists, before our Physician. Some concluded that Dostoevsky had IGE [Gastaut, 1978], but this does not account for the auras and the other ictal and postictal localizing signs. Others suggested a double diagnosis: partial epilepsy coexisting with IGE [Gastaut, 1984]. This combination is very uncommon, occurring in less than 1% of patients in an epilepsy clinic [Nicolson et al., 2004], and the likelihood for significant cognitive impairment would be greater than with just one sort of epilepsy. A double pathology consisting of epilepsy and 'pseudoseizures' has also been proposed [Dekkers and van Domburg, 2000]. This is surely not rare, and statistically the Patient would have had some chance for this combination. However, although the Patient might have suffered, at least in his young years, from some psychic instability, our Physician does not find positive evidence for non-epileptic seizures in the analyzed material.

Finally, partial epilepsy has been postulated [Voskuil, 1983; Siegel and Dorn, 2001]. Our Physician agrees with this interpretation, as many features of the Patient's history point to a focal onset: the aura, the prolonged twilight state possibly associated with forced thinking, the postictal dysphasia, and the

progressive memory impairment. Moreover, these elements suggest a dominant temporal lobe origin of the seizures. Thus, our Physician concludes that Dostoevsky probably suffered from TLE (most likely left mesiotemporal), with complex-partial and secondary generalized seizures, with a relatively benign course, possibly triggered by an intercurrent illness in 1846.

The significance of epilepsy in Dostoevsky's production cannot be emphasized enough. The nature of his auras is likely to have corroborated his mysticism. Besides giving us many detailed descriptions of epileptic characters, he showed that people with epilepsy should not be judged according to their disorder, but according to their nature. And, often, Dostoevsky's epileptics are found to be of pure, noble, and almost 'metaphysic' soul.

With the hope not to having missed too many elements outside of the frame (fig. 1), our Physician wants to conclude underlying that the personality of Dostoevsky and his work may be interpreted, from an epileptological point of view, as:

'…an extraordinary defense in favor of the countless persons who have been submitted to the unacceptable prejudice that they are destined to intellectual decline because of the repetition of their seizures and of those exceptional epileptic persons whose genius has erroneously been considered as a byproduct of their disease' [Gastaut, 1984].

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Immanuel Kant: Evolution from a Personality 'Disorder' to a Dementia

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Abstract

The philosopher Immanuel (or Emmanuel) Kant is felt by many to be one of the most influential thinkers of the modern age. For example, in his monumental work the *Kritik der reinen Vernunft* (Critique of Pure Reason) he showed how one can use the mind's faculties of knowledge to determine the limits of these very faculties. His life is of interest to neurologists for several reasons. He had a peculiar personality, he suffered from headache and he died with dementia. Kant was a man of legendary calm and regularity. For instance, his morning walks occurred always at the same time and it has been stated that people could set their watch when seeing him go by. He always followed the same itinerary and even walked the same number of minutiously counted steps. It is felt, however, that he did not fit the criteria of an obsessive-compulsive disorder. He suffered from headaches which were probably a true migraine. It has long been thought that a compulsive personality is often found in migraine sufferers. Finally, in his last years, Kant showed clear symptoms of dementia. Various etiologies have been considered such as vascular dementia or a slow growing tumor such as a frontal meningioma. Because he showed marked fluctuation of his cognitive symptoms, reported hallucinations and experienced repeated falls, we propose that Kant was affected by Lewy body dementia.

Immanuel (or Emmanuel) Kant (1724–1804) is considered by many as the greatest philosopher of the modern age. This is because his way of thinking about the world – or rather his way of thinking about our understanding of the world – is very compatible with the data originating from modern physics and psychology. As an example, Albert Einstein said that Kant's '*Critique of Pure Reason*' influenced him when he developed his famous relativity theory [Wood, 2003].

In addition to the considerable importance of Kant's work for philosophy and epistemiology, his life is of interest to neurologists for several reasons. He had a peculiar personality, he suffered from headache and he died with dementia.

Biography

The following data derive from the Literary Encyclopedia [Wood, 2003].

Immanuel Kant was born 22 April 1724 in the East Prussian town of Königsberg (now Kaliningrad, Russia), where his grandfather had emigrated from Scotland. At that time, it was a very cosmopolitan city. Kant's parents were devout Lutherans, of modest means. Kant grew up with his brother, an older sister, and two younger sisters in a working-class district on the outskirts of Königsberg, among laborers, small shopkeepers, and craftsmen like his father.

In 1734 the family's pastor, Franz Albert Schultz, who was also the principal at the Pietist secondary school in Königsberg, the *Collegium Fredericianum*, noticed young Immanuel's intelligence and persuaded his parents to allow him to attend. Here the future philosopher spent 8 years learning theology, Hebrew, Greek, Latin, mathematics and French. During that time, in 1737, his mother died. The boy excelled in his studies – especially Greek and Latin. However, even at the age of 13, Kant showed signs of his later legendary steadiness; he persisted in his studies and eventually graduated second in his class. In 1740, aged only 16, he enrolled at the University of Königsberg. Here he encountered gifted professors who introduced the young scholar to the worlds of philosophical thought and scientific inquiry. Over the next 7 years Kant not only deepened his study of mathematics, he also developed a lasting fascination for the methodologies of various sciences. In 1746 his father died, and Kant found that he could not afford to complete his education. On his own now, he acquired a position as tutor and was able to earn a living in this way for several years. While instructing the privileged children of wealthy Königsberg families, the young scholar also devoted much of his free time to independent study and work on his dissertation. In 1755, with the help of a friend, Kant was able to resume his studies in earnest. He promptly completed his dissertation, successfully defended it, and received the position of *Privatdozent.*

This position, which he held for the next fifteen years, was roughly the equivalent of an adjunct-assistant professor: it not only carried little prestige, but more seriously, it came with no salary. Consequently, Kant was forced to survive on meager fees paid to him by students. Eventually he secured a position as an assistant librarian, and worked a few hours each week in the library at

the royal castle. With this modest income, and by teaching up to 28 hours a week, Kant was able to afford a small room and frugal comforts. After lecturing on geography, mineralogy, physics, pedagogy, anthropology and philosophy, the young man enjoyed the newspaper over a cup of coffee. He relaxed by playing billiards or cards, and occasionally had a drink or two with friends. Kant returned in the evening to his one-room apartment, his table, chair, bed, and a few choice books. The only decor on his walls was a silhouette of the Swiss-French political theorist, Jean-Jacques Rousseau.

His thinking during these early years was influenced by the provocative ideas of Rousseau and the rationalism of Leibniz. But he was also deeply impressed by the achievements of the scientist and Biblical scholar Sir Isaac Newton whose works were just being introduced to the University of Königsberg. Kant published several books and numerous essays during this period on metaphysics, morality, aesthetics, logic, and various scientific topics, including astronomy. In 1755 he published the *Allgemeine Naturgeschichte und Theorie des Himmels* (Universal Natural History and Theory of the Heavens), which contains his vortex theory of the origin of the universe. Kant employed Newtonian principles to argue that a rotating nebula best explained the origin of the universe, and his theory was very influential. Pierre-Simon de Laplace further developed Kant's ideas in 1796, and this more refined model is now known as the Kant-Laplace theory.

In 1764 Kant published his *Untersuchung über die Deutlichkeit der Grundsätze der natürlichen Theologie und der Moral* (Inquiry into the Distinctness of the Principles of Natural Theology and Morals) in which he argues that the discourse of mathematics is grounded in pure reason and, consequently, is disconnected from the practical complexities of human moral existence. The work reveals that Kant realized the limits of the rationalist enterprise: in particular, he began to sense the inadequacies of the logical demonstrations carried out by such rationalists as Wolff who assumed that proving a proposition false necessarily implied that the contradictory must be true. Kant's misgivings indicate that, even at this early stage in his development, he was already moving toward a dialectical understanding of truth, what would later become his method of reasoning by antinomies.

Kant became a very effective teacher. He was a popular professor with students, not only because his lively teaching method incorporated provocative ideas, but also because of his humor. His effective teaching and steadily growing reputation as a writer eventually attracted many students to Königsberg. In 1770 (when Kant was 46), his alma mater admitted him as a full member of the faculty: he was promoted to professor of logic and metaphysics. Finally free of material preoccupations, he continued to teach at the University of Königsberg for the next 27 years. In 1788, he became Dean of the University.

At the age of 57, after 11 years of thought and reflection during which the book was often announced and often delayed, Kant completed his greatest work, his monumental first critique, the *Kritik der reinen Vernunft* (Critique of Pure Reason) [1781, 1787]. In this work, Kant attempts to use the mind's faculties of knowledge to determine the limits of these very faculties. His project is dialectical and subtle; on the one hand it makes pure reason the subject of critique, and on the other hand it employs pure reason in order to develop the inquiry. Kant's pivotal insight is that the mind plays an active role in structuring reality: the mind gives objects structure insofar as they must conform to the structure of the mind in order to be perceived in the first place. Kant rejects both the dogmatic metaphysics of the rationalists (Descartes, Spinoza, Leibnitz) and the skepticism of the empiricists (Locke, Berkeley, Hume), and employs a transcendental method of analyzing judgments and methodologies. To determine the 'transcendental' dimension of propositions, in Kant's sense, is to conduct an inquiry into how knowledge is to be possible a priori: he argues that the mind is devoid of content until interaction with the things themselves actuates its a priori structures. But these formal relations are analogous to templates for conjoining concepts into judgments. Kant does not argue that they are ready-made judgments like the allegedly 'innate' ideas of the rationalists; rather he suggests that the forms of our judgment are conditioned by the way in which our mind perceives our experience (what he calls our 'Forms of Intuition'). In short, Kant argues that knowledge of the things *in themselves* is unattainable, only knowledge of the *appearances* of things is possible. Through our senses, and the intellectual concepts (such as causality) that we apply to the information gathered by our senses, we never arrive at knowledge of '*Das Ding an sich*' (The Thing-in-Itself). We can conceive of 'reality-in-itself' but it is strictly unknowable, a 'noumenon'. What we know are 'phenomena', things shown (from Plato and the Greek *phainein*, 'to show'). We can, for example, know that we perceive time as passing and objects as distributed in space, but we cannot know that time and space actually exist independently of the qualities they are given in human perception: 'here' and 'there', 'then' and 'now', are imputed to matter because that is how the human mind organizes experience.

In contrast to his life which was so simple and regular (see below), his thought was revolutionary. He was a lifelong admirer of the ideas of Rousseau, and Kant's own thinking on the social and categorical nature of morality was progressive for its time. He was a true Enlightenment thinker, not only in his respect for Rousseau, but also in his admiration for the accomplishments of Sir Isaac Newton. Perhaps Kant's greatest achievement was to give rigorous arguments to the effect that purely abstract speculation is not only mistaken but even dangerous. But in his last years this generally modest man overcame his

humble Pietist origins by persistently daring to express his deep distrust of dogmatic and unverifiable religious doctrines.

Personality

Immanuel Kant was a man of legendary calm and regularity. He never married; moreover, he refused to leave the province of his birth. He never traveled more than fifty miles from home, even to visit the Baltic Sea, though it is a one-hour trip from Königsberg. Once his reputation was established, he refused many higher-paying job offers, which would have entailed leaving his hometown. Kant spent his evenings reading and writing, and went to bed at 10 o'clock. His bedroom was never heated, no matter how cold.

He always woke up at 5:00 AM. He was well known to citizens of his native city for the repetitious precision of his morning walks. They occurred always at the same time and it has been stated that people could set their watch when seeing him go by. He always followed the same itinerary and even walked the same number of minutiously counted steps. Only two events allegedly disrupted this routine: the publication of Jean-Jacques Rousseau's *Contrat social* in 1762 and the announcement of the victory of Valmy which saved the fledgling French Republic on September 20, 1792. Other segments of his life showed the same characteristic. For instance, he instructed his servants never to rearrange his furniture in any way, his meals, whether private or within the context of sumptuous receptions (which he often organized) were regulated down to the most minute details. Any departure from this pattern triggered violent reactions on his part.

Does this pattern of life qualify as an obsessive-compulsive disorder (OCD)? Let us remind the reader that, according to DSM IV [American Psychiatric Association, 1994], the diagnostic criteria for this disorder include *obsessions* as defined by recurrent and persistent thoughts, impulses, or images that are experienced as intrusive and inappropriate and that cause marked anxiety or distress. They also include c*ompulsions* as defined by repetitive behaviors (e.g. hand washing, ordering, checking) or mental acts (e.g. praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly. At some point during the course of the disorder, the person must recognize that the obsessions or compulsions are excessive or unreasonable. Finally, the obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships. OCD is specifically associated with a disorder of the orbitofrontal-subcortical

circuits and is rarely observed with lesions outside these circuits [McPherson and Cummings, 2002].

Obviously Kant's personality did not quite fit all these criteria: he did not have true obsessions as defined above. To our knowledge, he did not recognize his compulsive behavior as a product of his mind nor did he think that it was excessive or unreasonable. He is not known to have had any anxiety about it. Above all, it cannot be stated that it disrupted or interfered with his ability to function, at least not until the last few years of his life.

Headache

Quoting directly from a recent publication [Podoll et al., 2000], Kant suffered, since his 40s, from a migraine with aura which showed a significant exacerbation in his seventies, apparently coinciding with the onset of symptoms of dementia. Recorded symptoms of Kant's migraine include recurrent scintillating scotomas, one episode of diplopia, two episodes of complete amaurosis and frequent headaches described as oppressions of the head. The said symptoms of Kant's migraine can be traced not only in his letters and in accounts of his contemporary biographers, but also in the philosopher's published work.

It has long been thought that migraine occurs mostly in people who fit a specific psychological profile. Migraine sufferers were said to be neurotic, obsessive, compulsive, rigid, and to suffer from repressed hostility [Wolff, 1937]. This is a rather controversial issue. There are some recent papers that still claim such an association. A link between obsessive-compulsive disorder and migraine has been found in Tourette's syndrome [Kwak et al., 2003]. A French epidemiological study also suggests an association between obsessive-compulsive disorder and migraine [Guillem et al., 1999]. An Italian study has shown that patients with 'chronic daily headache' have significant psychiatric comorbidity; however, OCD was found in only 1.4% of patients with migraine [Verri et al., 1998]. In general, it must be said that research done in the last 15 years tends to show that migraine sufferers do not seem to have psychological profiles different from anyone else.

Dementia

In his last years, Kant showed clear symptoms of dementia. They have intrigued researchers and philosophers for many years [De Quincey, 1827]. As is almost always the case, it is difficult to state when they started. It has been claimed that subtle changes in cognitive abilities occurred around the age of

fifty, thirty years before his death [Marchand, 1997]. As mentioned above, other sources indicate an onset around the age of seventy. He gave his last lectures in 1796, at the age of 72 [Fellin and Ble, 1997]. It is only later, however, that his dementia became obvious. In the Fall of 1803 he was said to have marked memory loss and even to have had a marked language impairment for an entire day. Some time before, he had started showing a marked restlessness, always wanting to leave as soon as he had arrived in some new place. His obsessive behaviors turned to rituals. For example, he would endlessly button and unbutton his clothes.

As the disorder progressed, he could no longer sign his name or eat properly and he had to be fed. He exhibited frontal symptoms such as social disinhibitions, stereotypies and affective disinterest. His mental status showed marked fluctuations ranging from near mutism and inability to converse about his own affairs, being able at other times to provide very adequate answers to philosophical questions. Later on, he could no longer recognize his family and friends 'mistaking them for strangers'. He had an abnormal gait with poor truncal balance and he experienced frequent falls. He also complained of nocturnal hallucinations.

Conclusion

Much has been written and postulated concerning Kant's disease and the etiology of his dementia. Syphilis, so common in those years, is always a possibility, but none of the other signs associated with general paresis seem to have been present. The possibility of a frontal brain tumor has been raised [Marchand, 1997]. Arguments in favor of a tumor, possibly a meningioma, include an anosmia and several episodes of loss of consciousness compatible with late-onset epileptic fits. In addition, ophthalmological findings included transient visual obscurations and a progressive loss of vision, particularly affecting his left eye. This, however, may well have been due to a cataract. The lack of focal signs and the fact that he was not incontinent militate against a tumoral etiology.

Vascular dementia (VaD) is also a plausible hypothesis [Nores, 2000], even though, once again, no focal signs have been reported and there is no evidence that Kant had systemic manifestations of vascular disease. Another paper has dismissed the possibility of either an infection, a tumor or VaD being responsible and has suggested that he suffered of Alzheimer's disease [Fellin and Ble, 1997].

We would like to propose that Kant was affected by Lewy body dementia. Many reported symptoms fit the guidelines for that diagnosis [McKeith et al.,

1996; Binetti et al., 2001]. He showed marked fluctuation of his cognitive symptoms. He reported hallucinations and experienced repeated falls. On the other hand, 'instrumental' disorders such as aphasia and apraxia, typical of Alzheimer's disease, do not appear to have been prominent. As for his life-long personality, it is unlikely to have been an early symptom of his dementia. Rather, we suggest that it gave a peculiar coloration to his final clinical picture.

Did anyone openly talk to Kant about his condition? We do not know. One paradox of the situation is that current thinking about disclosure of the diagnosis of dementia reflects Kant's duty-based moral theory, which demands an absolute honesty on the part of the clinician [Monaghan and Begley, 2004]. We can only hope that those who surrounded Kant in his last years did follow this rule.

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Valery Larbaud

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Abstract

Valery Larbaud was a well-known and influential writer of the first part of the 20th century. Because he traveled extensively, he is considered as the founder of a movement, that of the cosmopolitan writers. He is also known for having been one of the translators of James Joyce's *Ulysses*. In 1935, he was affected by a stroke which left him with a severe aphasia and a right hemiplegia, which he survived for 22 years. His aphasia produced a short-lasting period of mutism. The aphasia later evolved towards a Broca-type disorder with a marked telegraphic style. However, the most striking aspect of his aphasia was a phase in which he could only produce a single recurring utterance. The utterance Larbaud produced was 'Bonsoir, les choses d'ici-bas'. This utterance has a literary flavor, which is difficult to translate (in his article published in *Brain*, Alajouanine left it in French). It roughly translates as 'Farewell, material things from this earth'. Stereotypes are not uncommon in aphasic patients. However, such an elaborate stereotype is a very rare, perhaps unique occurrence. Even though Larbaud's memory and intellectual abilities were preserved, he never produced any artistic work. Alajouanine concluded 'If aphasia destroyed literary language in the writer, if it stopped sound expression in the musician, it has left untouched plastic or figurated realizations'.

One hot afternoon of August 1935, a middle-aged man walking in the garden adjacent to his Latin Quarter apartment was struck by a cerebrovascular accident. It left him with a hemiplegia and an aphasia. So far, nothing could be more trivial. Yet several elements make this case quite exceptional: they include the man, the place where the stroke occurred, the syndrome that followed and the way it was recorded.

The Man

The man was Valery Larbaud. He is not nearly as well known today as he was in the 1930s and in subsequent years. He was the son of a pharmacist who had

made a fortune in exploiting the waters of Saint-Yorre and other resources of the Vichy area and who is said to have 'invented' the concept of bottling mineral water and distributing it commercially. Valery Larbaud soon jettisoned commerce, trade and profits in favor of two passions: writing and traveling. Of course others had done this before him, Robert Louis Stevenson comes to mind for instance. However, for France, it was sufficiently original for him to be considered one of the creators of a movement, that of the 'cosmopolitan writers'. The movement was followed in France by a series of authors such as Blaise Cendrars, Paul Morand and Paul Eluard. While combining traveling and writing, he learned at least six languages. Through translations and critical writings, he became a literary intermediary between France and the rest of the world, particularly England (and, to a lesser extent, the United States) and Spanish-speaking countries, with special emphasis on Latin America. He thus 'introduced' to the French and Western European literary scene great figures like Italo Svevo, James Joyce, Walt Whitman, Samuel Butler, Ramon Gomez de la Serna and Jorge Luis Borges. Even though he was not politically committed, he signed a celebrated pamphlet protesting the deportation of Miguel de Unamuno by the Spanish dictator Primo de Rivera.

Notable among his works is the creation of a totally fictitious character by the name of Archibald Olson Barnabooth, a young billionaire born in Chile (or Peru?) but originally from New York. Young Barnabooth goes around the world writing poems, tales and a 'Journal Intime' of which Valery Larbaud publishes the 'complete and definitive' edition which is in part an autobiography in disguise. In so doing, he preceded authors like Fernando Pessoa who distinguished 'pseudonym works' in which the author writes in person, from 'heteronymous work' where the author is outside his own characters.

This A.O. Barnabooth story was a great success. It received praises from the critics, made him known to the public and was followed by other novels and poems. What made Larbaud stand out were his style of writing, the things and places he wrote about and his personality, as well as the people he frequented.

Style

Cocteau has said that style is the art of saying complicated things in a simple way. Simplicity and clarity are the hallmarks of Larbaud's style. This is particularly remarkable since many writers of that period made a point of expressing themselves in a pompous and pretentious style that comes close to gibberish. He said once that he was writing to give pleasure to his readers. He certainly succeeded.

Subjects

He often wrote about contemporary means of transportation such as steamers and luxury trains. When he started writing, traveling was reserved to

the elite, but in the fifties, when mass tourism appeared or was about to appear, the subjects of his works was a reason for his success. His translations were also an important reason of his popularity. That of Joyce's *Ulysses* is particularly interesting because it was performed in close collaboration with the author; the style of the French version reflects some of Larbaud himself.

Personality

To the outside world, he appeared happy, gregarious and available. However, in contrast to his prose, so open and full of apparent simplicity, he was rather reserved and even introverted, perhaps in part as a result of a solitary childhood. This may explain his isolation-seeking behavior in the last part of his life. He was also a collector: he always wanted to know more places, more countries and more people especially women, without being a womanizer. He was an avid collector of tin soldiers. If it is true, as Freud says, that every collector is a substitute for a Don Juan Tenorio, Larbaud sublimated his passion well by being surrounded by the splendid and colorful uniforms of his tin soldiers [Mousli, 1998].

Friends

He was a close friend of many writers of his generation including, in addition to James Joyce and Sylvia Beach, André Gide, Samuel Beckett, Saint-John Perse, future Nobel laureate, and Jean Paulhan, Director of the Nouvelle Revue Française from 1923 until his death in 1968. Other friends included the painter Marie Laurencin and the publisher Gaston Gallimard who after his death gathered many of his works in the prestigious *Pleïade* collection. Among his friends was also a then young neurologist, Théophile Alajouanine who was to play a crucial role in the story.

Just before he died in 1957, France decided to have its most prominent writers presented at the World Fair of Brussels that was to take place in 1958. Valery Larbaud was chosen to be amongst the ten most significant ones.

The Place

The scene of the stroke was a section of Paris where scholarship and creativity have prospered since the Middle Age. It saw the foundation of the Paris University, la Sorbonne. The name comes from its founder, Robert de Sorbon, chaplain and confessor of Saint Louis, King of France. The University saw the light in the 12th century and consisted of the corporative organization of Paris masters and scholars. Originally installed in the Ile de la Cité, it soon moved to what was to become the 'Latin Quarter' (because Latin was the lingua franca

spoken by the many scholars who met there from all over Europe), where theology, law, medicine and art were taught outside to young pupils. The Sorbonne College, founded in 1253, was one of the numerous colleges that housed students on the hillsides of the Montagne Sainte-Geneviève.

The Latin Quarter coincides more or less with today's 'cinquième arrondisement'. One of its epicenters has traditionally been Place de la Contrescarpe at the top of the Montagne Sainte-Geneviève. Larbaud's dwelling was located nearby at 71, rue Cardinal-Lemoine. James Joyce invited by Larbaud had lived in that apartment for several months in 1921. He wrote at least one episode of *Ulysses* there. Ernest Hemingway had lived just across the street at number 74. Countless other great writers have lived in the area, close to Place de la Contrescarpe or at least hanged out in the local taverns. They run from François Villon, Rabelais and Ronsard to René Descartes and Blaise Pascal, Bernardin de Saint-Pierre, the naturalist Georges Buffon and, closer to us, Paul Fort, Paul Verlaine who died in rue Descartes and Emile Zola. Another friend, Sylvia Beach owner of the bookstore, Shakespeare and Company, was also nearby. It was Sylvia Beach who first agreed to publish James Joyce's *Ulysses*. This was a period of extraordinary censorship and the English-speaking world had refused to print what they perceived as pornographic filth.

Larbaud liked the countryside, but he hated his native Vichy, even though it did not yet have the sinister connotation it later acquired when Pétain and his infamous clique moved to Vichy and stayed there for 4 long years. When he was not traveling all around Europe, Larbaud felt very much at home in the Latin Quarter which he knew since his childhood, having attended the Lycée Henri IV and later the Sorbonne. In a poem about rue Soufflot, another landmark of the Latin Quarter, he wrote:

'Au fond, malgré la mer
Et tant de courses, nous ne sommes jamais sorti
D'ici et toute notre vie aura été
Un petit voyage en rond et en zigzag dans Paris'

(freely translated as: In the end, despite the ocean and so much running, we never left. Our entire life has been nothing but a small trip inside Paris).

The Syndrome

The stroke was followed by a severe right hemiplegia and a global aphasia. The hemiplegia remained more or less stable throughout the subsequent course, but the aphasia evolved over the years. He survived no less than 22 years after his stroke and never regained sufficient ability to write or dictate anything. The exact chronology of the evolution of the aphasic syndrome is not entirely clear,

but it went from mutism (probably short-lived as is frequently the case in this type of situation) to severely reduced language in the last years of his life. The most striking aspect of his aphasia was a phase in which he could only produce a single recurring utterance. In other words, a stereotype.

Stereotypes are not rare in aphasia. The 'modern' history of aphasia starts with the patient Leborgne described by Broca and who could only say one utterance which gave him the nickname by which he is best known: Tan-tan. Another famous example is Charles Baudelaire. In 1866, aged only 45, the author of *Les Fleurs du Mal* had a stroke. Little is known about the episode except that Baudelaire's verbal output became limited to a swear word 'cré nom' (more or less corresponding to 'damn'). Baudelaire never produced anything any more after the stroke. He died a year later.

Critchley discusses these recurring utterances (which he classifies among 'Iterations of written and spoken speech: verbal tics') and mentions a patient who could only say 'on the booze' [Critchley, 1970]. An American patient of mine had a convenient stereotype: whatever the question, he would reply 'Forget, forget'. The utterance Larbaud produced was 'Bonsoir, les choses d'ici-bas'. This utterance has a literary flavor, which is difficult to translate (in the article published in *Brain,* Alajouanine left it in French). It roughly corresponds to 'Farewell, material things from this earth'.

In a book about Larbaud written many years after the writer's death, Alajouanine [Alajouanine, 1973] indicates that the sentence 'Bonsoir, les choses d'ici-bas' was pronounced perfectly, in rapid and seemingly involuntary fashion. It was a true automatic discharge, sometimes a spontaneous verbalization, sometimes an attempt to reply to a question. It also replaced greetings such as *bonjour* or *au revoir*. It was pronounced with an absent-minded intonation that made it sound like a psalmody. It was sometimes accompanied by a hint of laugh or giggle, as if wanting to show the he had some awareness of the incongruity of his language production. Alajouanine adds that he had never witnessed such an elaborate stereotype. Neither have I. The archives of the Neurology Department of Milan University confirm that such an elaborate stereotype is a very rare, perhaps unique occurrence [Basso, pers. commun., 2004].

It has been said [Jackson, 1878] that stereotypes are the expression of the patient's thoughts at the time of the stroke, as if it were a stillborn proposition Alajouanine, however, thought that it was not the last sentence uttered or thought by Larbaud, but, rather, that it reflected the sense of disaster felt after the fatal stroke. It was 'a literary expression encasing its pathetic accent in the forced world of a leitmotiv' (une expression littéraire enfermant son accent pathétique dans le monde obligé d'un leitmotiv).

Based on his observation of many aphasic patients, Alajouanine has written extensively on stereotypes and has emphasized their automatic nature,

especially as they begin to appear. Alajouanine [Alajouanine, 1968] and later Brown [Brown, 1972] distinguished four stages over which the stereotypes evolve. First there is a stage of modification in which, through intonational adjustments, the stereotype acquires the ability to express a wide variety of emotions as well as denial or agreement. Then there is a stage of checking the utterance which signals the beginning awareness of the speech defect. This is followed by a period of fluctuation in which other expressions, stereotyped or not, come to accompany the original utterance, now no longer unique. Finally, there is abolition of the stereotype with gradual return of typical Broca-like speech. There is now slowness, laboriousness, lack of spontaneity and critical self-awareness.

Valery Larbaud did precisely go through these stages. The first changes did not modify the utterance, but the rhythm of the output, then its intonation. Sometimes he made the end inaudible, sometimes precipitated it as if to get rid of it. He later developed agrammatism with a telegraphic style. For instance, trying to describe his youth, he would say *'Jeunesse toujours merveilleuse'* (youth always marvelous). To describe a pleasant evening he said *'aujourd'hui bonne soirée, parler littérature'* (today good evening, speak literature).

Years before, Pierre Marie had written a series of much publicized articles in which he argued that all aphasic patients were demented. Alajouanine who never lost an occasion of contradicting his teacher on this point insists that Larbaud had kept a normal intelligence. As an example, he tells us that he was still able to judge his own previous work. While helping to put together a revised edition containing his own work, Gaston Gallimard was embarrassed because he felt that one particular piece was not up to par with the rest. To Gallimard's surprise and relief, Larbaud spontaneously pointed to that very piece saying '*pas bon*' (not good) showing that he wanted it taken out. Other examples show that his memory was intact despite his difficult verbal output.

This polyglot could still follow conversations on Spanish or English, but when attempting to speak English, it appeared that he had lost his previous nearly perfect accent, an instance of acquired dysprosody.

Alajouanine also comments on the way Larbaud reacted to his own stroke. At the beginning he was not very affected, probably because he did not realize the meaning of his own leitmotiv and the effect on his entourage of this 'lugubrious distress signal'. He was socially very nice and expressed naive joy at simple pleasures such as a cigarette or a wheelchair drive in the nearby public gardens. He accepted and went willingly through aphasia training as if it was a challenge and a game. This was not the case for his hemiplegia and its treatment. After a while, he refused physical rehabilitation which had to be discontinued altogether. After some years, however, he went back to his usual reserved attitude, often staying mute for long periods listening to the radio or reading

a dictionary. He often refused to see visitors. However, his last word, just before dying, was a simple 'merci' addressed to Maria-Angela Nebbia who had been his companion for 35 years, and to his doctor.

We have no element allowing us to speculate on the etiology of the stroke. Larbaud had been in 'poor health' all his life, but it was mainly a matter of flu-like episodes, severe colds or even pneumonias. Could he have had syphilis? After all, during his youth he was a frequent visitor of the Parisian 'maisons de rendez-vous'. We do not know whether he had any other risk factor such as diabetes or arterial hypertension.

The Recording

In an article written in French, but later translated into English and published in *Brain*, Alajouanine [1948] presents the case of Valery Larbaud within the framework of a reflection on the effect of aphasia on artistic realization. In addition to our writer, he presents the case of Maurice Ravel (who he had also examined personally) and of a painter whom he does not name because he was still alive at the time of the *Brain* article. The painter's production had continued after his stroke and Alajouanine concludes 'If aphasia destroyed literary language in the writer, if it stopped sound expression in the musician, it has left untouched plastic or figurated realizations'.

We have fortunately been able to trace the identity of the painter which is the subject of the next chapter.

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Alajouanine's Painter: Paul-Elie Gernez

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Abstract

In a famous paper published in 1948, the French neurologist Théophile Alajouanine discussed the influence of aphasia on artistic realization. He used as examples three artists he had seen personally. They included the musician Maurice Ravel, the writer Valery Larbaud and a painter whose name was not mentioned. We have since found the identity of the painter. We therefore present for the first time, with the permission of his family, the works of Paul-Elie Gernez (1888–1948) before and after his stroke. We confirm that aphasia did not really interfere with this painter's ability to produce works of art. However, we have reasons to believe that there was a change in his style which may have become less poetic, as if his ability to 'invent' had decreased and he had experienced some loss of spontaneity. This and other published cases strongly suggest that the effect of cerebral lesions is different in some artists than in non-artists, perhaps because of an expanded cortical representation, secondary to their lifelong formal training.

Cerebral lesions tend to impair several aspects of drawing aptitudes in untrained persons. They include constructional abilities [Grossi and Trojano, 2001] and, after aphasia, the capacity of associating color and objects [De Renzi and Spinnler, 1967]. Several reports suggest that cerebral damage, particularly aphasia, does not always produce a major impairment in the artistic abilities of trained painters [Chatterjee, 2004]. The French neurologist Théophile Alajouanine reported such an instance [Alajouanine, 1948], but did not disclose the name of the artist. The purpose of this report is to present for the first time, with the permission of his family, the works of Paul-Elie Gernez (1888–1948) before and after his stroke.

Paul-Elie Gernez was a prominent member of the 'French contemporary school' (fig. 1). Paintings by Gernez can be seen in several museums, particularly

Fig. 1. Paul-Elie Gernez. The date of the picture is uncertain. Thought to have been taken 'around 1940'.

the Eugène Boudin museum in Honfleur (Normandy) where he spent most of his life. His work is still very much appreciated and his paintings are often successfully proposed at art auctions. A monograph provides examples of his work [Rey, 1947].

He was born in a small village near Valencienne (Northern France) from a rather humble family. There is no evidence that his parents or other members of the family had any particular artistic talent. He started his professional career as an apprentice at the workshop of a house painter who soon noticed his artistic aptitude and made him follow courses at the Valencienne Academy. In 1911, aged only 23, because of a vacancy at the Honfleur 'collège' (high school) he applied for and obtained the post of 'Professeur de dessin', a position that only paid 90 francs a month, but which left him plenty of free time. He then started to produce and sell paintings, so that after 3 years he was able to leave his teaching post and become financially independent, thanks to his art. Because of a frail body build, he was fortunately considered unfit for military service during World War I.

In terms of style, he started with naturalistic paintings and drawings mainly composing still-life's, nudes, boats and landscapes. He experimented with pointillism and later on, towards 1920, he was inspired by cubism. We found no trace of paintings from that period, but we are told that from this experience, he retained a way to depict his subjects in a solid fashion and to

Fig. 2. Nu et coquillages, 1931.

give a frame to his compositions which, at the time of his maturity 'are tinged with poetry, intimacy and sensuality, striking for their originality and for the poetic vision they express' [Bergeret-Gourbin, 2004]. His manner of observing light and sea reminds one of the style of Jongkind. Alajouanine goes so far as to say that when looking at Gernez' paintings, one feels the sensuous poetry of Baudelaire and the polyphonic music of Debussy. Figure 2 shows a work by Gernez painted in 1931.

In 1940, at age 52, Paul Gernez was suddenly affected by aphasia after two short and transient aphasic spells. The only clinical details available are those provided by Alajouanine [1948] who knew the painter very well, although it is unclear whether their acquaintance preceded or followed the stroke. Alajouanine wrote that the aphasia was of the Wernicke type without any phonetic trouble and without any right-sided hemiplegia, but with a 'slight hemianopic defect'. He also had a transient apraxia. His spoken language was severely impaired with anomia and a marked agraphia. His comprehension was 'relatively good', although Alajouanine states that this was largely due to global intuition of the overall meaning of the conversation (we can assume that he did not formally test it).

He showed no evidence of intellectual deterioration, but he showed an accentuation of his premorbid introverted personality. He was quite aware of his deficit. In replying to a person who was congratulating him for his continuing artistic activity, he once said: 'There are two men inside me, the one who paints, who is normal while he is painting, and the other one who is in a vague state, who is lost, who does not stick to life. I don't express myself well. One of the men is in perfect touch with life and with reality. The other one is lost in an abstract world. When I am painting I am outside of my life; my way of seeing things is even more intense than before; I find everything again; I am a whole man. Even my right hand that seems alien to me, I do not notice it when I am painting. There are two men, the one who grasps reality in order to paint, the other one, the fool, who cannot manage words any more.' He also became more irritable and was often quite depressed.

He resumed painting after his stroke as soon as his apraxia improved and he continued to paint up to the time of his death 8 years later. Alajouanine stated that Gernez' artistic production was just as perfect and that he did not experience any changes in his artistic skills or style. He added that 'connoisseurs did believe that he had found a more intense and acute expression'.

The monograph on Gernez [Rey, 1947] shows some paintings dating from before and after the stroke. In addition, thanks to the kind cooperation of his family, we have been able to inspect additional paintings, also dating from before and after 1940. We agree with Alajouanine that no error in form, expression or color interpretation can be found in the works painted after the stroke. However, his style seems to show a change with a tendency to produce more concrete and realistic paintings (fig. 3). The almost oniric poetry found in some of his previous paintings (fig. 2) is apparently no longer found in works painted after the stroke.

Discussion

Aphasia did not really interfere with this painter's ability to produce works of art. We have reasons to believe that there may have been a change in his style which may have become less poetic, as if his ability to 'invent' had decreased and he had experienced some loss of spontaneity. Two caveats need to be mentioned. First of all, we were not able to inspect the entire production of the painter after the stroke and he may have produced works that were just as full of poetry and originality as some of his works painted before the stroke. In addition, most artists tend to change style as life progresses and it is practically impossible to state that any modification of his style was due to the stroke and its ensuing symptoms. However, we found nothing to justify Alajouanine's statement that Gernez' style showed more 'intensity'.

Fig. 3. Fleurs, 1941.

As mentioned in the introduction, there is a contrast between the effect of cerebral damage, particularly aphasia, on visual, visuospatial and drawing capacities, according to whether the patient is professionally untrained or trained. In untrained persons, one finds that drawing, either spontaneous or tested formally [Benson and Barton, 1970; Benton, 1974] is often impaired. First of all, drawing and related abilities may be impaired by constructional apraxia [Kleist, 1934] and unilateral spatial neglect, especially following lesions of the right hemisphere and, to a lesser extent, of the left hemisphere [Grossi and Trojano, 2001]. Lesions of the left hemisphere can also be accompanied by various disorders affecting color such as color aphasia (difficulty in naming and pointing to colors in the absence of aphasia in other domains) [Gelb and Goldstein, 1924] or color amnesia (inability to recall the color of an object) [Lewandowsky, 1908]. In addition, aphasic patients are known to often have difficulties in associating color and objects even when no overt verbalization is required. For instance, De Renzi and Spinnler [1967] demonstrated a severe deficit in color tasks involving language. In addition more than half of their aphasic population showed a marked

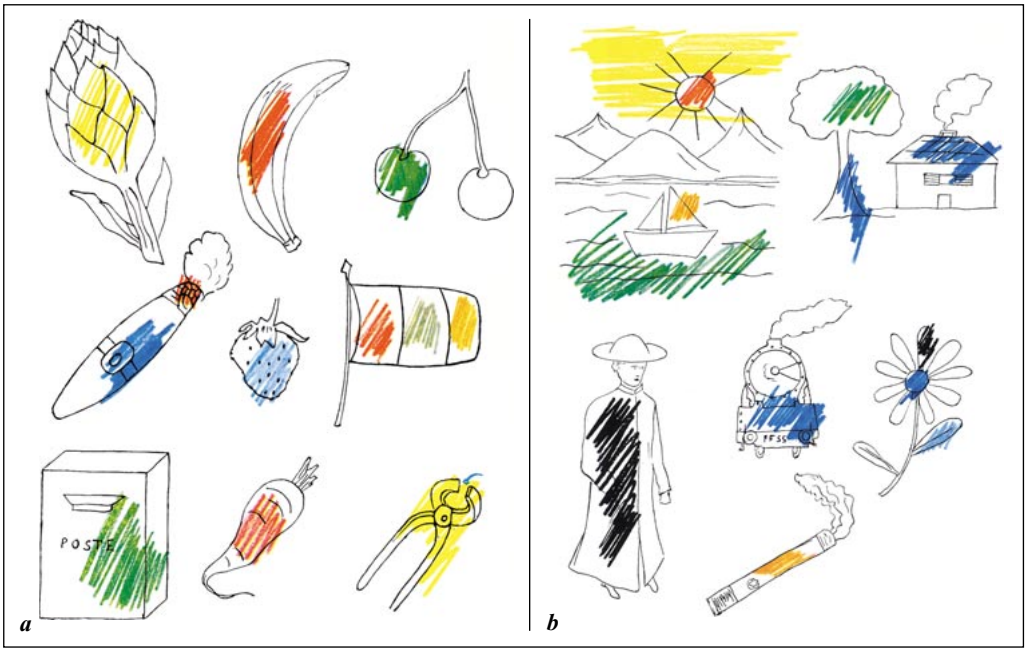

Fig. 4a, b. Sample coloring of drawings by an aphasic patient. Reproduced from De Renzi E, Spinnler H: Impaired performance on color tasks in patients with hemisphere damage. Cortex 1967;3:194–217, with permission.

deficit in coloring objects (fig. 4a, b). The deficit was correlated with severity of aphasia and with presence of a right hemianopsia. There is some evidence that left hemisphere lesions also impair imaging [Pachalska et al., 2001] and even ability to recall dreams [Pachalska, 2001].

In professionally trained persons, the effect of stroke and aphasia is quite variable. Striking examples of stylistic changes are found in the case of Federico Fellini, not a professional painter of course, but someone very much used to expressing himself with drawings. Following a right hemisphere stroke, his cartoons showed clear evidence of left side neglect while fully retaining their humoristic impact [Cantagallo and Della Sala, 1998]. A Polish painter described by Kaczmarek [1991], following an aphasia, was still able to produce skilled charcoal drawings, but lost his previous ability to paint highly symbolic paintings. He was quite aware of his deficit and complained that his mind was blank and he could not return to his pre-morbid style of symbolic painting.

There have been several other instances where painters continued to work successfully after an aphasia. Alajouanine cites the example of Daniel Vierge,

a draftsman who, despite his aphasia, continued to produce illustrations [Bonvicini, 1926]. The Bulgarian painter Zlatio Boiyadjiev has been studied in detail [Zaimov et al., 1969]. Boiyadjiev's pre-aphasic style was natural and pictorial and he used mostly earthy tones. Following a left hemisphere stroke, critics said that a new painter was born. His paintings became richer, more colorful, with fluid energetic lines demonstrating great vigor and inventiveness. Much of the imagery was bizarre and fantastical. Zaimov thought that the left hemisphere lesion had produced a liberation of his creative possibilities. Jason Brown has commented on the case and speculated that this kind of painting might reflect the right hemisphere's looser sense of semantic boundaries [Brown, 1977]. This is reminiscent of the hypotheses concerning Ravel's latest works [Amaducci et al., 2002].

In discussing Gernez' preserved painting abilities, Alajouanine hypothesized that aphasia might deeply alter the work of abstract painters, since they produce paintings that are not based on predominant sensory elements but on 'abstract figuration that is true ideograms'. Actually, Mazzucchi et al. [1994] have studied abstract painters who became aphasic. Their style was altered, but they continued to paint. For instance Afro Basaldella (1912–1976) reverted to 'neo-cubism' 2 years after a stroke that had left him with a profound aphasia [Mazzucchi et al., 1994].

As mentioned in the chapter on Larbaud, Alajouanine lost no occasion to stress that aphasic patients, particularly the ones he described in his article [Alajouanine, 1948], have not lost their intelligence, thus contradicting Pierre Marie's idea on the subject. On this basis, one might expect that dementia should profoundly alter a painter's artistic abilities. This was apparently the case for Willem De Kooning who was diagnosed as having Alzheimer's disease and whose later work deteriorated [Espinel, 1996]. There have however been instances where people with dementia continued to paint successfully [Fornazzari, in press] or even showed apparent 'improvement' [Miller et al., 1998; Thomas Anterion et al., 2002].

Is preserved artistic abilities after aphasia specific to painting? Alajouanine seemed to think so, since of the three aphasic artists he described, only Gernez was able to continue to produce works of art. Valery Larbaud (this book, pp. 85–91) and Maurice Ravel (this book, pp. 132–140) affected, respectively, by Broca and Wernicke's aphasia lost their ability to produce art. However, there have been instances of preserved abilities in areas other than painting. For instance, Tzortzis and her colleagues described a professional musician who was still able to make a living with his work despite being affected by a rather severe primary progressive aphasia [Tzortzis et al., 2000]. These cases strongly suggest that the effect of cerebral lesions is different in some artists than in non-artists, perhaps because of an expanded cortical representation, secondary to their

lifelong formal training. Another artist, Carolus Horn, also had Alzheimer's disease but continued to produce drawings and paintings until he died, albeit with changes in his style. The case is described in detail in the next chapter.

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Carolus Horn – When the Images in the Brain Decay

Evidence of Backward-Development of Visual and Cognitive Functions in Alzheimer's Disease

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Abstract

Here we analyze the artwork of Carolus Horn, a famous German artist. Despite developing Alzheimer's dementia (AD), he continued to produce drawings and paintings until he died. There are impressive changes in spatial relations, in the preference of colors, in the size of objects and other aspects of his paintings. The most prominent change is the loss of 3-dimensionality, followed by a continuous simplification and finally a decay of all objects and structures. We point to the relation between these changes in his artwork and the course of neuropsychological and neuropathological processes in AD.

Alzheimer's disease (AD) is the most common cause of dementia. It leads to severe impairments of memory and other cognitive abilities such as semantic and executive functions. AD also leads to widespread impairments in the visual domain, such as visual attention, motion detection, depth perception, angle discrimination, color discrimination, and visuoconstructional abilities [Rizzo et al., 2000; Mendola et al., 1995; Mendez et al., 1990; Ska et al., 1990].

One of the hallmarks of AD is a specific pathway of neuropathological progression – as indicated by accumulation of neurofibrillary tangles (NFT): Neuronal and synaptic degeneration begins regularly in limbic areas (transentorhinal cortex/hippocampal formation) and spreads to neocortical temporal, parietal and frontal association cortices. Primary sensory and motor cortices are

affected only in the end stages of the disease [Braak and Braak, 1991]. This specific pathway degeneration is assumed to be related to a different vulnerability to neuropathological changes across the cerebrum. Furthermore, [Braak and Braak, 1996] suggested that this path of degeneration reflects – at least at the neocortical level – a backward way of the cerebral ontogenetic myelinization: The cortical networks that have matured last (e.g. higher multimodal association areas) will be the first to undergo degeneration and the structures that developed first (primary sensory and motor areas) are most resistant against AD-related changes. In many respects the AD patient can be described as regressing step by step from the cognitive abilities of a mature adult to those of an infant.

From this perspective of neurodegenerative retrogression, we want to elucidate the artwork of Carolus Horn (C.H.), who lived from 1921–1992 [Maurer and Prvulovic, 2004]. C.H. was a famous German graphic artist and illustrator, who actively worked from the 1950s to the 1970s. He designed numerous advertisements for cars, clothes, and many other articles for internationally renowned brandnames. Beginning in the early 1980s, however, he showed slowly but steadily increasing signs of AD such as loss of memory, spatial disorientation, agnosia, and aphasia. Nevertheless, he continued to paint every day, even during the late stages of the disease, until he died. Therefore, his artwork presents an excellent chance to gather insights into the underlying changes of the visual world in AD. However, since almost all (healthy) artists show an evolution in their painting style and technique over time, it would be speculative to interpret the changes in the paintings of C.H. as a result of underlying AD-related pathology. On the other hand, specific impairments of visual functions in AD patients are known by independent neuropsychological testing. It would thus be of interest not only to verify whether the paintings of C.H. reflect some of the AD-related visual impairments, but also to elucidate whether subsequent paintings, made in the early, moderate and late stages of the disease, show any signs of a 'retrogression' or 'backward development' of visual functions. The paintings of C.H. are unique in that he depicted the same buildings and scenes, often from the same viewing point, at various stages of his career and thus allow for a quantitative analysis of disease-related changes.

Analysis of Series of Drawings of C.H.

Confusion of Spatial Relations

In the course of drawing development, the ability to depict geometrically correct spatial relations develops last. In turn, in AD patients, this aspect is one of the first drawing aspects to disappear.

Fig. 1. Painting of the bridge 'Eiserner Steg' in Frankfurt am Main. Horizontal line in dots. For an exact explanation, see text.

Figure 1 shows a painting of the bridge 'Eiserner Steg', which was created by C.H. during an advanced stage of the disease:

The vanishing points of the two men in blue (green vanishing lines) are far above the horizon while the vanishing points of the catwalk (black vanishing lines) are significantly below the horizon (dashed black line). To depict accurately the perspective of the scene, all vanishing points should meet the horizontal line. In contrast, the different objects in figure 1 have their own perspective, which leads to an impression of spatial and perspective inconsistency: The two men look like they are rather 'hanging' on a wall than standing on a catwalk. Moreover, the size of the windows of both ships do not decrease the further away they are from the viewer. Since reduction of texture size is one of the key elements for the spatial interpretation of two-dimensional images, the lack of this distance-dependent size reduction contributes to the significant spatial confusions within the image.

Mendez et al. [1996] have demonstrated that depth perception is impaired in AD: AD patients do have difficulties to interpret monocular spatial cues such as texture size and the reduction of object size according to vanishing lines.

Fig. 2. Ornament-like small geometric figures are an essential part of C.H.'s paintings in the later stages of the disease.

Furthermore, basic perceptual functions such as the discrimination of line orientation or angle size are affected in AD [Ska et al., 1990]. It is very likely that these basal perceptual deficits contribute significantly to the inability of C.H. to depict spatial relations in the right manner.

Effect of 'Small Objects'

An obvious characteristic of C.H.'s paintings in the later stages of the disease is the tendency to include ornament-like small geometric figures (fig. 2), which had never appeared in C.H.'s artwork before. C.H.'s latest drawings are

dominated by objects that are characterized by their relatively small viewing angle. A possible explanation for this phenomenon can be the impairment of spatial frequency contrast sensitivity in AD. The contrast sensitivity denotes the minimal contrast that is needed for a person to resolve white/black boundaries in a visual stimulus. The rough shape of objects is predominantly comprised by low-frequency contrast, while finer details contain high-frequency information. Several studies [e.g. Cronin-Golomb et al., 1991] have found that AD patients suffer from deficient contrast sensitivity (CS), especially in the lower-frequency range, which is crucial for successful object and face recognition. A recent study by Cronin-Golomb et al. [2000] demonstrated that the face-recognition rate is substantially enhanced in AD when the size of the presented face stimuli is scaled down. By this, the lower-frequency content of the stimuli is pushed up into a range, which provides better contrast sensitivity and thus object recognition in AD. It thus can be hypothesized that C.H. downsized the objects in his late drawings in order to compensate for the increasing deficits of low-frequency contrast sensitivity. From a developmental perspective, there is neurophysiological evidence that CS at high spatial frequencies almost completely matures during the first 3 years of life, while the CS in the low frequency range matures more gradually until the age of 9 [Adams and Courage, 2002].

The Uniformity of Faces and Bodies

While in the earlier stages of the disease sometimes confusions occurred about the age and the species of the faces, C.H.'s later drawings show a dramatic reduction of the complexity of faces.

This reduction of details goes in line with a loss of ability to discriminate between gender, age and any individual characteristics. Like children in the preschematic stage, C.H. increasingly uses two-dimensional primitive shapes (e.g. modified circles) for heads and bodies, which become indistinguishable from each other (fig. 3):

This uniformity of faces and bodies comes along with the patient's reduced ability to recognize familiar faces and subjects [e.g. Giannakopolous et al., 2000] and to differentiate between them, which, in turn, is assumed to reflect the patient's severely impaired access to semantic memory. AD patients in the late stages are increasingly unable to recognize even their relatives, which additionally strains interpersonal relationships.

The 'Color-Shift'

One of the most impressive changes of C.H.'s paintings over time concerns the different preferences in the use of colors. Before the onset of disease,

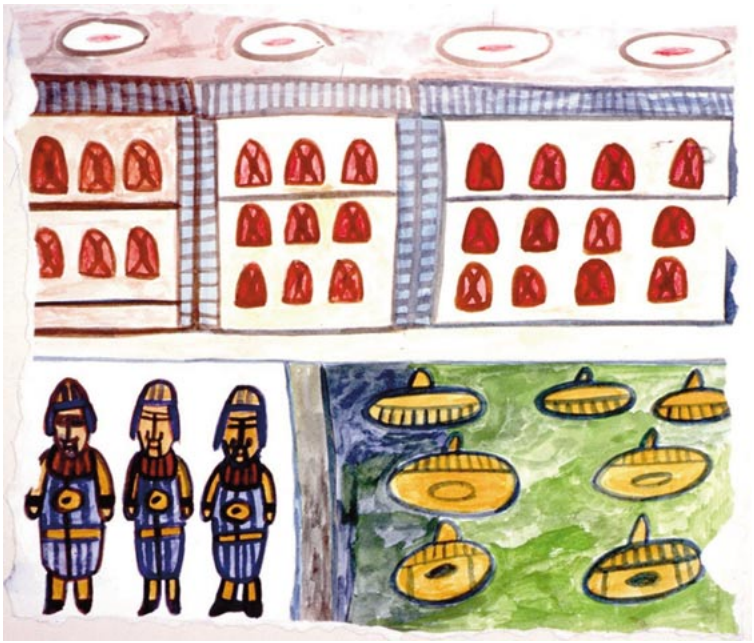

Fig. 3. This picture shows a reduction of detail. C.H. increasingly uses two-dimensional primitive shapes (e.g. modified circles) for heads and bodies.

C.H.'s portraits and landscapes didn't show any preference to a specific color or even color range. Interestingly, with the beginning of dementia symptoms, C.H.'s paintings show increasingly large amounts of dark colors, especially from the blue range (fig. 4a).

Early stages of AD are often characterized by symptoms of depression [Heun et al., 2002]. However, it is unclear whether the comorbidity of depression and AD is due to a common neurobiological mechanism or rather to the patient's reaction to his cognitive decay. The 'darkening' of C.H.'s paintings in the early stages of the disease may reflect a depressive and dysphoric mood of the artist. However, with further progression of the disease, the paintings become more and more bright and C.H. shows a clear tendency towards colors from the yellow-red range (fig. 4b).

Wijk et al. [1999] have found that AD patients do suffer from selective deficits of color discrimination in the blue-green range, while the ability to distinguish color tones in the yellow and red range is preserved in AD. Thus, the preferential use of yellow and red in C.H.'s late paintings might be related to a differential impairment of color vision in AD.

Fig. 4. Changes over time affect the different preferences in the use of colors. With the beginning of dementia symptoms, the paintings show increasingly large amounts of dark colors, especially from the blue range (***a***). In advanced stages of the disease bright colors from the yellow-red range are preferably used (***b***).

Furthermore, there is much less shading and mixing of colors, by which the amount of uniformly colored, 'solid' and flat surfaces increases in the late stages. In parallel, while the shading of surfaces is reduced, the outlining of objects and surfaces becomes more and more prominent. These changes are similar to some features of comic strips, which are commonly characterized by a reduction of detail. The solid color fill of surfaces without or with only small amounts of shading reflects this departure from pictorial realism. The outlining of geometric shapes compensates for the lack of realism and helps to easier identify the objects. By that the attentional demands are reduced and it is easier to realize the pictorial content of the painting, which is an important aspect of AD as it may result from restrictions of visual attentional capacity in AD [Rizzo et al., 2001].

The 'Scribbling Stage'

C.H.'s latest drawings (made several months before his death) are characterized by scribblings (fig. 5):

While in the beginning of the 'scribbling stage', the scribblings seem to follow some kind of spatial organization, the very latest drawings are void of any spatial or object-like features. This latest stage of drawing 'backward development' coincides with the latest stage of the disease, shortly before death. Stereotype, iterative movements do commonly occur in the late stages of AD. It may be assumed that C.H.'s scribbling is the consequence of an extensive decay of not only visuoperceptual and executive ('the person cannot draw what it sees')

Fig. 5. Drawings (made several months before his death) are characterized by scribblings.

functions but also of the patient's knowledge ('the person does not know what to draw'). C.H.'s scribbling finally reflects the patient's complete inability to access the knowledge about objects and their visual representations. That is why C.H. is not able to depict even primitive symbols of what he intended to draw.

Conclusion

The analysis of series of paintings of C.H. revealed significant changes across paintings, which have been created during different stages of the disease.

As one of the most important changes, the drawings from the moderate stage of disease show the beginning confusion of perspective and spatial relations [Kirk and Kertesz, 1991]. It is likely that these changes can be attributed to a decline of visuospatial processing and spatial perception in AD. Depth perception, which is highly impaired in AD patients, depends (among others) on the ability to properly discriminate angle sizes and line orientation, which is also disturbed in AD [Ska et al., 1990]. Recent neuroimaging studies revealed that visuospatial processing crucially depends on parietal cortex activation [Sack et al., 2002], which, in turn, is impaired in patients with already mild-to-moderate AD [Prvulovic et al., 2002].

Further on in the course of the disease, C.H.'s paintings reveal a shift in his colors of preference from the blue-green range to the yellow-red range, and a loss of shading. Both changes might originate in color-processing deficits in AD, as it has been shown that AD patients do not only have difficulties to name mixed colors (shading) but also have difficulties to discriminate colors in the blue-green range [Wijk et al., 1999]. Moreover, the loss of shading goes inline with a further decline of the spatial properties of the paintings, because distance-dependent texture shading and size adaptation are additional important pictorial hints for the spatial interpretation. Color-processing, including color perception and color discrimination are closely related to the 'color-processing center' in the inferior temporal cortex [Bartels and Zeki, 2000]. The inferior temporal cortex has been shown to be affected by accumulation of neurofibrillary tangles (NFT) in the course of AD [Braak and Braak, 1991].

The late paintings of C.H. show very coarsely painted objects with severely confused spatial relations and almost no three-dimensional perspective. Finally, the very last drawings by C.H. are scribbling without any objects, spatial organization and meaning. These late drawings seem to reflect the loss of the knowledge not only of *how* to draw the visual world, but also of the visual world itself. Evidence for this view comes from combined neuropathological and neuropsychological studies, which have consistently shown that structures of the occipital lobe are amongst the last to be affected by AD, and that the involvement of visual cortex is closely related with visual agnosia [Giannakopoulos, 2000] and the inability to copy even simple two-dimensional shapes [Nielsen et al., 1996]. It thus seems likely that the changes in the very

last drawings of C.H. are related to the progression of neuropathological changes in the last spared cortical areas: the early visual cortices, whose involvement may ultimately result in a large decay of basic visual functions in the AD patient.

In some aspects, the retrogression of C.H.'s drawings seems to follow an opposite way of the drawing development in children with the very latest drawings of C.H. appearing similar to the very first drawings in children (scribbling). It should, however, be considered that this parallelism remains speculative and that in the course of AD factors come into play such as psychotic phenomena (visual hallucinations, delusional misperceptions) and mood disturbances (periods of depression), which additionally affect and alter visual experience.

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Major Depression and Stroke in Caspar David Friedrich

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Abstract

Caspar David Friedrich, nowadays recognized as the most important German Romantic painter, stands out against his contemporary artists by the radical dramaturgy and the 'disturbing spiritualism' of his paintings. Being a loner, he embodies melancholy not only in his works, but also as an artist. Art scientists have related to losses in early childhood and depressive conditions for the interpretation of his work without examining in depths the specific features of his disease and their impact on the artistic performance and works. Having applied diagnostic criteria for psychiatric disorders to his letters and publications, to statements of his contemporaries and to his art we propose that he had suffered from a recurrent major depression before he was struck by a stroke when he was 61 years of age. Depressive episodes occurred in 1799, between 1803 and 1805, around 1813/14, again 1816 and between 1824 and 1826. They were associated with diminished or altered creative powers as can be derived by the chosen techniques and motives. In 1835, a subcortical infarction led to a right-sided paralysis causing severe difficulties for the painting in oils. As a consequence, Friedrich only produced one large-scale oil painting which has been characterized as his 'artistic testament'. Rather, he returned to the less strenuous and demanding sepias and water colors. Most probably, he also suffered a poststroke depression as reported by contemporaries. This is also illustrated by an accumulation of death symbols in his late work, which has been neglected by the Friedrich research.

Caspar David Friedrich is nowadays recognized as the quintessential German Romantic painter. His artistic works deal with the core themes of Romanticism, in particular with the transience of human existence, the redemptive powers of nature and man at the mercy of the elements as well as the ambivalence between fear of and longing for death. Among his best-known

masterpieces are *Tetschener Altar* (1807/8 Albertinum, Staatliche Kunstsammlungen Dresden), *Monk by the Sea* (1808/9 Nationalgalerie Berlin), *Abbey in the Oaks* (1809/10, Nationalgalerie Berlin), *Wanderer Looking over a Sea of Fog* (ca. 1817, Kunsthalle Hamburg), and *Chalk Cliffs on Rügen* (1818/19, Oskar Reinhart Foundation Winterthur). Although these paintings mark the period of his greatest fame in his lifetime (between 1808 and 1820), the artist had been productive for over 40 years. However, his creativity had been substantially reduced by several depressive episodes as well as a stroke in 1835 resulting in periods of rest which sum up to almost 10 years. During his depressions, he favored particular motives and preferred techniques requiring only little manual efforts, e.g. sepia, watercolors and pencil drawings. He completely lacked the power for painting in oils. Particularly in his last five years after the stroke, his drawings were filled with death symbols: vultures sitting on spades, eerie owls and godforsaken graveyards and ruins. Art-scientific interpretations [Koerner, 1990; Börsch-Supan et al., 1973] have often referred to Friedrich's affected states of mind and his lifelong debt feelings at the tragic death of his favorite brother (cf. biography); however, it has not been analyzed if he might have suffered from a neuropsychiatric disorder influencing his artistic work and performance. Particularly, his last productions originating from the time after his stroke have remained unknown until today. Before elaborating on these aspects in more detail, it might be useful to give a brief biographical account of his life.

Biography

On the 5th September 1774, Caspar David Friedrich was born as son of the candle-maker Adolf Gottlieb (1730–1809) and his wife Sophie Dorothea (1747–1781) in the Swedish Pomeranian town Greifswald, located at the Baltic Sea, where he received a Spartan Protestant upbringing. His mother died in 1781 when he was 6 years old, a sister 1 year later and another sister 10 years later. However, the most tragic event in his childhood was the loss of his favorite brother Johann Christoffer in December 1787: While skating Caspar David fell through the ice of a frozen lake and nearly perished. In the effort to save his brother, Johann Christoffer drowned and lost his life. In 1794, Friedrich began to study at the Academy of Art in Copenhagen, where he stayed until 1798. Afterwards he moved to Dresden, at that time one of the art capital of Europe. In 1799, he participated in the Dresden art exhibition for the first time. He further specialized in sepia and watercolors and painted in oils only since 1807/8. His reputation as exceptional landscape painter grew steadily: In 1810, the Prussian crown prince purchased two major paintings and Friedrich

became a member of the Academy of Art in Berlin in 1811. Five years later he became a member of the Academy in Dresden and in 1820, Tsar Nicholas I purchased several paintings while visiting the artist's studio. At that time, his financial situation made him able to start a family. In 1818, he married the much younger Caroline Bommer (1793–1847) and she gave birth to 3 children. His reputation as an artist began to sink in 1820, and although he was appointed as professor at the Dresden Academy in 1824, he was not given a teaching assignment and never got the post he had hoped for. Friedrich's fame vanished rapidly, and he was almost completely forgotten when he deceased on May 7, 1840.

The Major Depression

The melancholic trait of Friedrich's character has often been described by his contemporaries, e.g. by the artists F.A. von Klinkowström and W. von Kügelgen [Runge, 1840/1965; von Kügelgen, 1870/1985], and his friend, the physician C.G. Carus (1789–1869), even diagnosed some kind of a 'brain disease' as a long-term aftermath of his childhood tragedies [Carus, 1865–66/1966; cf. also his biography]. However, a more detailed analysis of his presumed affective disorder has not yet been achieved, although some typical depressive symptoms such as feelings of guilt have often been integrated in the art-scientific interpretations of his art [Körner, 1990; Börsch-Supan et al., 1973]. Applying diagnostic criteria for psychiatric disorders as defined by the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition [DSM-IV; American Psychiatric Association, 1994] to his letters and publications, to statements of his contemporaries and to his art might be a new and fruitful approach to arrive at a formal diagnosis and to elucidate his illness.

Our main thesis is that Friedrich suffered from a recurrent major depressive disorder (MDD). He was struck by the first episode when he was 25 years old and most probably he experienced five depressive episodes until his stroke in 1835. A letter to J.L.G. Lund, his friend from the Academy of Art in Copenhagen, gives a detailed account of his first depressive episode in 1799 with the symptoms of lassitude, hypersomnia, loss of interest, social isolation and the psychodynamically interesting feature of anger directed inwards [Börsch-Supan et al., 1973]. Between 1803 and 1805, a 2nd depressive episode with feelings of disappointment and anger culminated in a suicide attempt as testified by the physician Carus; it is supposed that he grew his beard to hide the resulting scar from view [Carus, 1865–66/1966]. Around 1813/14, Friedrich became depressed again, possibly triggered by the French occupation of Dresden. In a deeply moving letter to Lund, written in 1816, Friedrich impressively described a depressive syndrome with feelings of emptiness, a severely

diminished interest in activities, loss of energy, deficits in concentrating and indecisiveness [Börsch-Supan et al., 1973]. Between 1824 and 1826, another depressive episode struck the artist. For example, the art critic Quandt reported that Friedrich was 'indisposed' most of the time and 'unable to work' due to a severe loss of vitality [Börsch-Supan et al., 1973].

From a medical point of view, in addition to the mere symptoms there is further evidence supporting our hypothesis. (1) Epidemiological findings strongly suggest certain characteristics of major depressive disorder (MDD). The mean age of onset is in the late twenties and the average length of episodes ranges between 6 and 12 months. At least half of all patients with a MDD develop a recurrent form and the mean interval between episodes ranges between 3 and 10 years [Akiskal, 2000]. The mean number of episodes over a lifetime is five to six. Thus, from an epidemiological point of view the proposed course of Friedrich's depression can be considered as almost classical. (2) Psychosocial stress, e.g. the loss of a close relative, is important in the development of affective disorders. Although its precise role (e.g. as causal or as risk-enhancing factor) is much debated, there is no doubt that stressful events in childhood are closely associated with MDD in later life, particularly parental loss before adolescence [Gabbard, 2000]. Considering that his mother died when Friedrich was 6 years old and that he lost his favorite brother under tragic circumstances (cf. his biography), it seems plausible that he was at great risk for the development of an affective disorder. (3) Premorbid personality factors also play a crucial role in the pathogenesis of MDD. A specifically predisposing personality constellation was described as typus melancholicus with the core properties of orderliness, conscientiousness, norm orientation and intolerance of ambiguity [Tellenbach, 1961]. All of these features were characteristic for Friedrich as reported by himself, his family and contemporaries. (4) Psychodynamic theories emphasize as one key point in the development of depression that feelings of anger are directed inward at the self resulting in profound self-depreciation [Gabbard, 2000]. In some of his letters written in the depressive episodes, the artist vividly describes becoming so angry at times that he immediately fell ill [Börsch-Supan et al., 1973].

The proposed course of his depression corresponds well with Friedrich's artistic work. At the time of the manifestation of his depression, he was engaged with drawings reflecting the topics of death and transience. For example, the drawings *Farewell Scene at the Beach* (1799) and *Family in Front of an Open Coffin* (1799) deal with human tragedies belonging to Friedrich's daily life. In 1804, during his 2nd depressive episode, he participated in the Dresden art exhibition with one picture that made his contemporaries shudder: The sepia *My Funeral* (has gone lost) might be interpreted both as an anticipation of his suicide attempt and as a reflection of the Romantic death longing. Banished from life, but unable to die, Friedrich existed as 'wanderer' at the edge of the abyss.

Fig. 1. Caspar David Friedrich *Skeletons in a Dripstone Cave* ('Skelette in der Tropfsteinhöhle'), ca. 1826, Sepia, Pencil, 18.8 × 27.5 cm. Hamburg, Kunsthalle. Bildarchiv Preussischer Kulturbesitz, Berlin, with permission.

After years of success as painter since 1808, a 3rd and 4th depressive episode around 1813/14 and 1816 were associated with a deep decrease in his productivity. For example, for the year 1813, there are only one drawing and two watercolors recorded [Börsch-Supan et al., 1973]. In his next depressive episode, presumably manifest since 1824, Friedrich again was rather unproductive. His friend Carus wrote in 1824 that the artist had not sold a single large-scale painting, and the art critic Quandt reported for the same year that the artist only finished a series of 30 small views of the Baltic island Rügen [Börsch-Supan et al., 1973]. In the period following, Friedrich mainly produced drawings predominantly relating to the central theme of death, e.g. the sepia *Skeletons in a Dripstone Cave* (fig. 1), which can be viewed as a shocking illustration of death longing.

The Stroke

On the 26th of June in 1835, Friedrich suffered a stroke resulting in right-sided hemiplegia [Carus, 1840]. Initially, the severity of the symptoms forced

Fig. 2. Caroline Bardua *Portrait C.D. Friedrich*. 1840, Oil on Canvass. Dessau, Anhaltinische Gemäldegalerie, with permission.

him to stay in bed, but already some weeks later he travelled to Teplice, Bohemia, for a rehabilitation cure. Neither disturbances of cortical functions, e.g. aphasia or other neuropsychological deficits, nor any sensory loss have been reported. Therefore, it can be assumed that it was a left-sided subcortical infarction, possibly a lacunar stroke. However, these etiological considerations remain speculative. Friedrich never fully recovered, and the last portrait of the artist (fig. 2), painted by Caroline Bardua (1781–1864) a few months before his death, reveals a central paralysis of the 7th cranial nerve.

Already during his rehabilitation cure, the artist began with pencil studies. Having a presentiment of his near death, Friedrich built up all his strength to accomplish an unusual painting with his partially paralyzed hand. The painting *Seashore by Moonlight* (fig. 3) dating from 1835/36 is not only his last work in

Fig. 3. Caspar David Friedrich *Seashore by Moonlight* ('Meeresufer im Mondschein'). 1835/36, Oil on Canvass, 134 × 169.2 cm. Hamburg, Kunsthalle. Bildarchiv Preussischer Kulturbesitz, Berlin, with permission.

oil, it is also striking due to its large format, the insecure brush guidance and the missing glazes for the depth effect. It has been characterized as the 'artistic testament' of the old painter [Börsch-Supan, 1992].

In the years following his stroke, it is most likely that Friedrich developed a post-stroke depression [Robinson, 2003]. For example, the Russian poet A. Zhukovsky having visited the artist characterized him as a 'sad ruin' [Hinz, 1968]. He was only able to finish small-scaled pieces. He chose techniques and materials requiring only little manual efforts. After his stroke, he accomplished almost 80 sepias and watercolors. They frequently represent allegories of death which work in their directness threateningly, e.g. *Coffin at Grave* (ca. 1836), *Landscape with Grave, Coffin and Owl* (ca. 1836/37), *Little Screech-Owl at Grave* (ca. 1838) and *Owl in Gothic Window* dating from 1836 (fig. 4). This accumulation of death symbols in Friedrich's late work might be interpreted as his very personal way to deal with his imminent death, but can also be conceived as a reflection of his post-stroke depression.

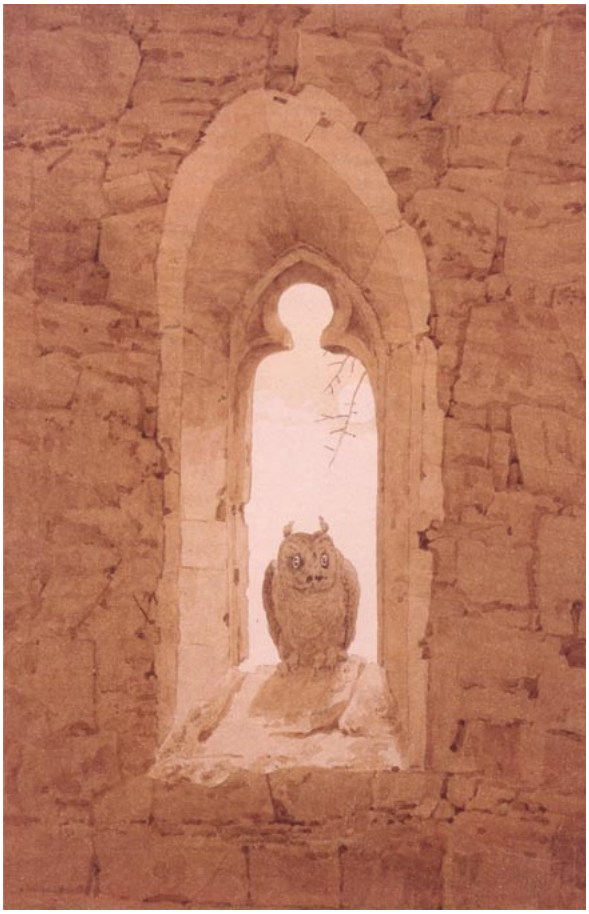

Fig. 4. Caspar David Friedrich *Owl in Gothic Window* ('Eule in gotischem Fenster'). 1836, Sepia, Pencil, 37.5 × 25.6 cm. St. Petersburg, Eremitage, with permission.

Concluding Considerations

Friedrich never tried to gain distance from his own mental and emotional condition. Rather, he resolved them in his art. 'A painting is not to be invented, but is to be felt' was one of his maxims [Hinz, 1968]. While his friend Carus partly engaged in painting and music for therapeutic reasons, he did not show this reserved attitude towards his artistic subject. He tried to give his mental and emotional affliction an artistic form, but often for the price of suffering and inner struggles.

Friedrich's works including his less known productions need to be conceived in the contemporary context of Romanticism. His fate belongs to the 'psychography of an epoch' [Hofmann, 2000] and is representative of a whole generation of Romantic intellectuals, whose longing for freedom and infinity appeared only attainable in the next world. The distinctive manner, in which he transformed Romantic ideas into the fine arts, and the radical dramaturgy of his landscape paintings, which also reflect inner conditions of man, is his very personal and exceptional achievement. They can also be understood from the point of view of his recurrent depression and stroke and his way to cope with his diseases. None of the Romantic artists was as able as Friedrich to deal with the Romantic themes of death longing, human transience and infinity in this totality.

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Understanding Van Gogh's Night: Bipolar Disorder[1]

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Abstract

Vincent Van Gogh (1853–1890) imparted in his art a deep essence of life, and in such a unique way that many would say it is possible to experience it vicariously by looking at his paintings even once. In 10 years, while exerting mental and physical efforts that may well have contributed to his premature death, he produced an impressive number of masterpieces. However, the specific neurological disorder Van Gogh suffered and how this may have influenced his art is still not clear. The combination of his eccentric personality, irascible temper, unstable moods and prolific creativity, makes the understanding of his illness a very complex endeavor and therefore poses a great challenge to those who investigate the relationships between the 'artistic mind', the brain and illness. In fact, most of the diagnoses (nearly 30) proposed for Van Gogh, during the last century, are not based on medical evidence but are ascertainable from analyses of his paintings and biographical data. Although no definitive diagnosis can be made based on such evidence, we conclude that according to DSM-IV criteria and findings extrapolated from his letters, Van Gogh is most likely to have suffered a bipolar disorder, affective or schizoaffective, which caused his death by suicide.

Although medico-historical data is not sufficient to advance a definite diagnosis, we will present some arguments that favor a bipolar disorder (affective or schizoaffective) as the principal illness of Van Gogh.

Van Gogh wrote on a fairly consistent basis to his brother Theo since 1870 to 1890 with an interruption in the correspondence that occurred between

[1] The title plays on a quote of Shakespeare: *Macbeth: What is the night? Lady Macbeth: Almost at odds with morning, which is which* [Macbeth, Act iii, sc. 4, I.126].

Table 1. Bipolar affective disorder: adapted DSM-IV criteria

Criteria for manic episode

Distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week

During the period of mood disturbance, three (or more) of the following symptoms have persisted and have been present to a significant degree

1. Inflated self-esteem or grandiosity
2. Decreased need for sleep
3. More talkative than usual or pressure to keep talking
4. Flight of ideas or subjective experience that thoughts are racing
5. Distractibility (i.e. attention too easily drawn to unimportant or irrelevant external stimuli)
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation
7. Excessive involvement in pleasurable activities that have a high potential for painful consequences

Criteria for depressive episode

Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) or (2)

1. Depressed mood
2. Markedly diminished interest or pleasure
3. Significant weight loss when not dieting or weight gain or decrease or increase in appetite nearly every day
4. Insomnia or hypersomnia
5. Psychomotor agitation or retardation
6. Fatigue or loss of energy nearly every day
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

Corollary: The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

February 1886 and October 1888, when he lived in Paris. His letters reveal all the symptoms of the bipolar disorder (table 1) although it remains difficult to reconstruct the exact duration of depressive and manic phases.

Before 1886, Van Gogh presented major and minor depressive episodes alternating with hypomanic or manic phases, often with rapid switching. Durable depressive episodes occurred in London after a love rejection, when he

was expelled from the church, and at the moment of his separation from the prostitute Sien and her son. Durable hypomanic or manic phases coincided with his beginnings as an evangelist, as well as an artist.

During the years spent in Paris (1886–1888), he abused alcohol, drank a lot of absinth and manifested anxiety, irritability, hostility, eccentricity and several somatic symptoms. In Arles (1888) he experienced anxiety, melancholia, remorse, insomnia and physical exhaustion, which he related to his insalubrious behavior. On Christmas Eve in 1888, during what was probably his first psychotic crisis (of which he remained amnesic) he cut off part of his left ear. After two brief psychotic crises he voluntarily entered the Saint-Rémy asylum in May 1889. In Saint-Rémy, during the following year, he experienced severe depressive episodes and three psychotic crises, of which at least two were concomitant with his temporary visits in Arles. The last of these crises, characterized by religious and paranoid delusions and auditory hallucinations, persisted over 3 months (February–April 1890) and left some vivid memories. Discharged from the asylum in May 1890 as being cured, he moved to Auvers-sur-Oise, where he manifested rapid switching between manic and depressive symptoms. On July 27, he shot himself in the chest and subsequently died two days later, July 29, 1890.

Van Gogh had a family history of mental illness. Of his four siblings, his brother Theo suffered from depressive episodes, his sister Wilhelmina lived for 30 years in a mental asylum and his brother Cornelius most likely committed suicide. A high rate of the disease among first-degree relatives is characteristic of a bipolar disorder and suggests the role of genetic mechanisms. There is no mendelian pattern, however, and polygenic inheritance is suggested.

There is also growing evidence that bipolar illness and schizophrenia syndromes share similar genetic mechanisms predisposing one to psychosis [Kendler et al., 1993; Detera-Wadleigh et al., 1999; Wildenauer et al., 1999; Berrettine, 2001].

A large majority of bipolar patients turn to substance abuse. Van Gogh was a heavy smoker, had an alcohol-absinth addiction and had probably consumed terpenes and camphor which are constituents of paints. We note that in the case of Van Gogh, manic and depressive symptoms are already identifiable many years before periods of substance abuse, an argument that suggests the abuse as a co-morbidity of the affective symptomatology rather than its cause.

Alcohol and other stimulants may help bipolar individuals in attenuating the severity of depression or in sustaining the increased arousal of manic phases, but they also have severe mood destabilizing effects. During the period of his confinement in the Saint-Rémy hospital, all psychotic crises likely occurred when he left the hospital to go to Arles. Once there, it is almost certain that he consumed alcohol and/or absinth to excess.

In his letters he frequently expressed strong anxiety as well as fears of poverty, disease, failure at his work and premature death.

Bipolar individuals may use sleep deprivation to reverse depressive symptoms, but lack of sleep, when sustained over several days, has the potential to precipitate mania. Van Gogh used to paint until late into the night without taking time to recover through rest on the following day. In his letters he often complained about the exhaustion provoked by this pattern of behavior.

Substance abuse, anxiety disorders and sleep problems are all known and frequent co-morbidities of bipolar disorders.

The severity of mania and depression may vary during the course of the disease. This variability explains the difficulty of diagnosis and related controversies in Van Gogh's case, as well as in general in bipolar patients. Nowadays, diagnosis is often missed or delayed in about 70% of individuals affected by a bipolar disorder [Hirschfeld et al., 2003]. The difficulty in identifying hypomanic symptoms is probably one of the reasons for this. Hypomania describes a manic state that is less severe, does not produce psychotic symptoms and does not lead to major impairment in social or occupational function. Cycles of full-manic and depressive episodes correspond to the bipolar I disorder (BPI), whereas cycles of hypomanic and depressive episodes occur with the bipolar II disorder (BPII).

This subdivision of the bipolar disorder into BPI and BPII forms is not yet supported by specific pathogenetic mechanisms but has clinical relevance because it allows the identification of 'soft' bipolars who may be at risk of severe complications.

Severe and frequent full-manic states would have been so destructive to the artistic expression to preclude the realization of the aesthetic goals Van Gogh so coherently articulated in the letters to his brother Theo. Van Gogh completely achieved in his art. It is thus reasonable to conclude that besides the depressive episodes, Van Gogh most likely initially experienced not full mania, but rather hypomanic phases.

Only in the 2 years prior to his death, Van Gogh's depressive and hypomanic symptoms, aggravated by alcohol/absinth abuse, seem to progess to psychotic crisis.

Arriving at the correct definition of hypomania is probably a key diagnostic issue in the case of Van Gogh. DSM-IV criteria of hypomania require a distinct period of persistently elevated, expansive or irritable mood lasting at least 4 days. Other authors propose a period of 2 rather than 4 days [Akiskal et al., 2000]. Overactivity, however, is the core symptom of hypomania and Van Gogh's functioning in daily life amply demonstrates the existence of that form of hyperactivity that the term hypomania is designed to capture. In Van Gogh's case, the overactivity was manifested not only in the impressive quantity of art

that he produced in a relatively short period of time, but also in the style and incessant rhythm of letters to Theo. In those letters overactivity and hypomania can be found in the emotional euphoria expressed therein, the irrepressible proliferation of ideas they contain, the verbosity they exhibit and the impulse to keep talking. It is also interesting to note that Van Gogh's production of paintings seems to have accelerated in periods of major light exposure such as springtime and summer [Stucke, 2005] and the connection between hypomania and exposure to light is a known phenomenon of the bipolar disorder.

In his last 2 years of life, Van Gogh experienced psychotic crises, accompanied by delusions and prevalent auditory hallucinations. The differential diagnosis of psychosis includes primary conditions such as schizophrenia and psychotic mood disorders (including bipolar disorder or schizoaffective disorder bipolar type), as well as secondary disorders due to medical conditions (such as epilepsy, porphyry, etc.) or substance abuse.

Based on our reading of Van Gogh's letters, we believe, however, that there is reason to prefer the hypothesis that Van Gogh's psychotic crisis were most likely the result of affective episodes (depression and mania) probably exacerbated by the influence of alcohol/absinth abuse. Our main argument that favors the diagnosis of an affective disorder over schizophrenia is that Van Gogh's letters, even those written many years before his later psychotic episodes and serious substance abuse, consistently express depressive and hypomanic themes. The DSM-IV dimensional/categorical classification acknowledges the possibility of coexistence of mood symptoms (either mania or depression) and psychosis (with either positive or negative symptoms) in mood disorders as well as in schizoaffective syndromes. For example, the 3-months crisis (February–April 1890), for this duration (superior than 2 weeks), according to DSM-IV criteria, may correspond to the category of a schizoaffective disorder bipolar type. For the diagnosis of a bipolar schizoaffective disorder it is necessary that, during some crisis, the psychotic symptoms be not related to prominent (depressed or manic) mood symptoms. We do not have sufficient historical evidence to define exhaustively the character of Van Gogh's crisis. However, a diagnosis of schizoaffective disorder bipolar type is likely because several arguments suggest that Van Gogh's psychotic crises occurred during bipolar mood episodes (affective or schizoaffective) rather than in pure schizophrenia.

In comparison to bipolar disorder, schizophrenia is a more severe chronic disorder with an earlier, more insidious onset [Fein and McGrath, 1990] and a poorer prognosis [Tsuang and Dempsey, 1979]. Van Gogh's depressive episodes lack negative symptoms of schizophrenia such as anergia, amotivation and catatonia. Moderate-to-severe negative symptoms are more common in schizophrenia than in schizoaffective disorder or affective psychosis, and may tend to persist over time [Fennig et al., 1994]. Van Gogh's letters suggest little if any

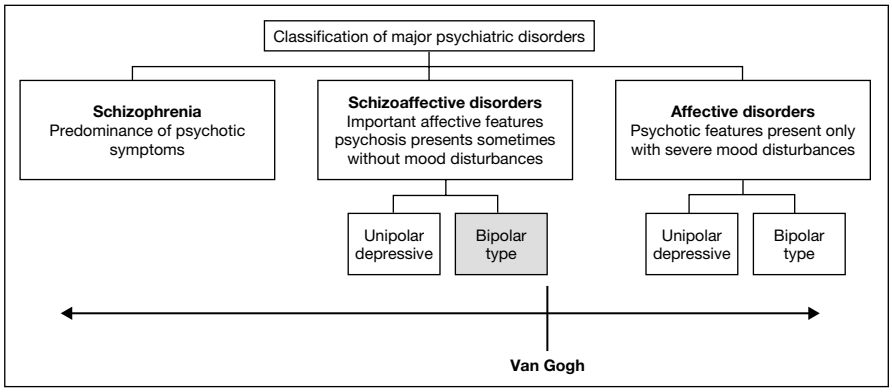

Fig. 1. Current classification of major psychiatric disorders, running from pure schizophrenia at one end, to pure affective disorder at the other end of the spectrum. Van Gogh's symptomatology was closer to the affective side of the spectrum.

evidence of thought patterns or verbal disorganizations characteristic of schizophrenia. Furthermore, individuals affected by schizophrenia generally do not have a history of previous depression or mania. The historical-medical evidence at our disposal do not permit a definite diagnosis of Van Gogh's psychotic crisis according to DSM-IV criteria. However, the artistic production and the many letters written by Van Gogh do not lead us to regard psychosis but rather the bipolar affective mood as the core symptom of Van Gogh's disease. Indeed psychosis is central to schizophrenia. Anyway, the psychiatric literature suggests that there is a continuum between affective disorders and schizophrenia, and that schizoaffective disorders (sharing more similarities to affective disorder than to schizophrenia) may be a link between them [Marneros, 2003; Marneros et al., 2004] (fig. 1).

The other possible diagnosis for Van Gogh's crisis is a temporal lobe epilepsy undertaking the forms of ictal or interictal psychosis and interictal dyspohria [Blumer, 2002]. Several elements suggest this diagnosis but they also are subject to criticism. The first issue is that Van Gogh manifested the typical traits of an epileptic personality (viscous traits, episodic irritability contrasting with an otherwise hypersocial disposition, hyposexuality, switching phases of excitation and depression, hyperreligiosity, verbosity), a condition also defined as Geschwind syndrome [Waxman and Geschwind, 1975]. Geschwind syndrome has been related to interictal subclinical epileptic discharges. However, traits of the Geschwind syndrome substantially overlap with the profile of bipolar individuals. Another argument is the possibility that Van Gogh had hippocampal sclerosis and manifested temporal epilepsy late in his life only because of the epileptogenic

properties of absinth. However, there is no founded evidence of perinatal traumas or developmental disorders suggesting the presence of mesial temporal pathologies such as sclerosis, hamartomas and gangliogliomas. Moreover, abrupt onset and residual amnesia are strong arguments for epileptic crises. Nevertheless, although the diagnosis of epilepsy was formulated long ago by Dr. Rey in Saint-Rémy, and not discarded until very recently, no valid clinical description has survived for posterity. For most of Van Gogh's crises, how they begin and subsequently terminate is not sufficiently known. Epileptic partial myoclonus, automatisms, sensations of déjà and jamais vu, dreaming states, depersonalization, derealization, autoscopy, Todd phenomena, unresponsiveness and other clinical signs or symptoms associated with complex partial seizures are missing. A generalization in 'grand mal' convulsive crises never occurred even if some rare episodes of fainting are reported. The long duration of crises in Van Gogh's case (generally several days but sometimes also several months) and incomplete postictal amnesia is at least suspected to have a non-epileptic origin (even if they did not exclude it). The long periods of remission from crises due to Van Gogh's abstinence from alcohol/absinth would indicate moreover a non-severe form of temporal lobe epilepsy, which is difficult to reconcile with behavioral changes related to interictal, postictal or ictal discharges.

Patients with temporal lobe epilepsy are more prone to epileptic psychosis which may have a clinical profile similar to schizophrenia [Sachdev, 1998] and have a higher rate of suicide [Fukuchi et al., 2002] than patients with other epilepsies.

However, the greatest difficulty in accepting the alternative hypotheses of epileptic psychosis and interictal dysphoria is due to the absence, in the case of Van Gogh, of significant clinical evidence for epileptic crises. Description or observation of crises still remain the more reliable diagnostic tools in epilepsy and according to such tools, evidence of epilepsy is absent in the case of Van Gogh.

There are no sufficient clinical data to consider the possibility of psychosis related to other medical conditions such as porphyria. Van Gogh himself never reported discolored urine. His frequent stomachaches, relieved by regular meals and forced periods of abstinence from alcohol, are more a reminder of the more common alcohol gastritis or gastric ulceration rather than acute intermittent porphyria, which is a very rare disease. Similar consideration, that is the absence of clear diagnostic elements, applies to Menière's disease or other diagnosis (neurosyphilis, digitalis toxicity, lead poisoning and so on).

Although manic episodes are the distinguishing feature of the bipolar (affective or schizoaffective) disorder, it appears that both major and minor forms of depression or dysthymia are often the dominant mood disturbance and that much of the functional impairment associated with bipolar disorders results from that [Angst et al., 2003].

In his letters Van Gogh very often expressed depressive themes: the misery of homelessness; the futility of his efforts to share affection with a warm-hearted woman; the hopelessness of achieving his professional goals or his goals as a person; and his fears of premature death. In his life, as in his paintings, he seems to have been reaching, searching for melancholy and compelled toward it. He demonstrated a great deal of interest in the poor and particularly precarious lifestyle of Borinage miners who lived lives infused with darkness, illness and proximity to death. He felt as though he were one of them. He involved in a sad and despondent relationship with a prostitute (Sien). The sorrow he experienced in that relationship is portrayed in several drawings. He described himself as melancholic and wrote 'I prefer feeling my sorrow, sorrow is better than laughter'. He said 'The sadness will live forever'.

Several of Van Gogh's paintings seem direct attempts to portray, capture and communicate sadness. In other portraits depression is depicted with more subtlety (see *Portrait of Doctor Gachet*, Auvers-sur-Oise, June, 1890). In the Saint-Rémy asylum, phases of inertia and depression between psychotic crises, inspired several paintings reproducing landscapes, wheat fields, cypresses and olive trees (see *Wheat Field with Cypresses*, Saint-Rémy, September, 1889).

Suicide attempts are more common among individuals suffering from bipolar disorder rather than from those with unipolar depression [Mitchell et al., 2004]. Between 10% and 19% of individuals with bipolar disorder attempt suicide, and 80% of suicidal actions of bipolar individuals occur during a depressed phase of the illness. Van Gogh committed suicide at the age of 37 during a depressive crisis following a manic-psychotic phase.

The transition between the two phases of his disease may be perceived in the *Wheat Field with Crows*, one of the last if not the last painting he completed before his death.

The stormy black sky and the turbulent field are reminiscent of depression and mania. The crows, flying toward or away from Van Gogh may symbolize either danger or hopelessness. The paths may represent Van Gogh's past history but also the alternative directions he could undertake at that difficult point in his life. With such an unreliable sense of direction, it would be difficult to find one's footing.

In a letter dated about 10 days before his suicide, he wrote: '*they are vast fields of wheat under troubled skies, and I did not need to go out of my way to try to express sadness and extreme loneliness... I almost think that these canvases will tell you what I cannot say in words, the health and restorative forces*'.

In Auvers-sur-Oise he felt the psychological absence of his brother Theo. Later, in fact, Van Gogh died in Theo's arms saying: 'I would like to go [on?] like this'. After Vincent's death Theo wrote to their mother: 'Life weighed so heavy upon him'.

Bipolar disorder induces significant disability in terms either of loss of hours of work or disrupted social relationships. Van Gogh failed at every attempt he made to work inside a structured organization. He failed as art vendor, teacher, predicator, and fellow in religious and artistic academies. He was irritable and intolerant of authority. Despite his strong passion for art, he also failed as a painter within the artistic community. He had disrupted relationships with his father and other family members. His pressing money requests, conveyed with lack of courtesy, put at risk even Theo's patience and understanding. He was not able to dress himself in proper clothes and shoes. He neglected his body, looked emaciated and toothless, and was unkempt, unshaven and verbally abusive. Despite his good intentions, his love for his cousin Kee expressed itself in tasteless and persecutory behavior toward her. Faced with Vincent's requests for love she simply stated: 'never, no never'. Even though he could not provide for them he assumed financial responsibility for a prostitute (Sien) and her two sons. He had troubles with neighbors, vendors and creditors in almost every place he lived. People with bipolar disorder have a 15 times more likely chance to be in the lowest income-earning categories [Kessler et al., 1997]. Theo guaranteed Van Gogh's rent. Van Gogh sold only one of his paintings during his life.

We will never know the effect that modern pharmacological treatments (antidepressants, lithium, valproate, carbamazepine, lamotrigine, olanzapine) and psychotherapies (psycho-educational and interpersonal and social rhythm therapy) available today might have had on Van Gogh's life and art. The artistic creation is a mysterious and individual process that is inseparable from personal experiences and we are inclined to believe and accept that the disease and its related artistic dimensions co-existed in Van Gogh. Through his paintings Van Gogh wanted to express authentic forces that deeply live in nature. These forces were symbolized not only in skies, stars, trees, flowers, portraits, but also in buildings, chairs, and rooms. By such symbolism he used the force of colors to enhance emotions. The experience of manic and depressive states may have guided him into a deeper insight of nature's spiritualism. In every period of his life, he probably used the processes of artistic creation to convert his suffering into positive paths [Stuke, 2005].

Starry Night, perhaps his most famous painting, may be an example of the therapeutic dimension of his art.

Starry Night was painted from memory while Vincent was in the asylum at Saint-Rémy in the period between severe psychotic crises. *Starry Night* evokes feelings of peaceful reconciliation between ancestral unknown forces (as in a turbulent mind) and the laws and the endless cycles of nature.

A close reading of Van Gogh's letters leads us to conclude that Van Gogh searched for an art able to provide access to both the artist and his audience to

a universal healing dimension where the suffering of the materialistic world ceases. In such an artistic-metaphysical pilgrimage he felt close to the miner, the peasants and the potato eaters who, like him, during the experience of poverty and illness remain in contact with nature and thus stay pure. His illness probably enhanced in Van Gogh this instinctive ability to feel the nature in humans and even in non-living elements. His creative talent allowed sublimation, in the Freudian sense, of the experience of suffering [Stuke, 2005].

Although there is evidence that people with bipolar disorder are more creative when effectively treated than when they are not treated, and only the early phases of mania appear to contribute to creativity [Schou, 1979], Van Gogh's creativity remained exceptional until the end. The most convincing perspective for Van Gogh is that he used to work heavily, cleverly and passionately for a resuscitating and sublime art. Beyond any theory on his illness, his paintings are certainly the result of incommensurable creativity and artistic skill. It must be borne in mind that these qualities were possessed by a man who had to cope with an undiagnosed disease, which caused his premature death by suicide.

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The Terminal Illness and Last Compositions of Maurice Ravel

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Abstract

Maurice Ravel (1875–1937) suffered from a presenile, progressive neurological disorder which annihilated his creativity in his final years. He died after craniotomy but only speculative retrospective diagnoses are possible since autopsy was not obtained. Nevertheless, it is likely that Ravel's illness belongs to the so-called Pick complex, which includes frontotemporal dementia, primary progressive aphasia and corticobasal degeneration. There are no valid arguments for an alleged influence of this disease on the style of Ravel's last compositions.

Maurice Ravel (1875–1937) lost his creativity by a debilitating progressive neurological disorder in the last years of his life when he was regarded as the greatest French composer since Claude Debussy and, in his own words, had 'still so much to say' [Jourdan-Morhange, 1945].

The History of Ravel's Decline

In 1927, Hélène Jourdan-Morhange worried about Ravel's complaints of 'great tiredness', 'cerebral anemia' and 'amnesia' after the completion of his *Sonate for Violin and Piano* [Jourdan-Morhange, 1945]. But Ravel often had such symptoms. Already in 1912, he had written to Ralph Vaughan-Williams: 'The various works which I had taken up to performances last season, especially *Daphnis and Chloë*, have left me in a pitiful state. I had to go to the country, to withhold a beginning neurasthenia' [Ravel, 1989]. In 1927, Ravel went to Dr. Pasteur Vallery-Radot, a famous Parisian internist, who similarly advised a rest for 1 year.

However, in the spring of 1928 he made a triumphant American tour of 4 months' duration and, on his return, composed the *Boléro*.

Between 1929 and 1931 Ravel worked simultaneously on two concertos for piano and orchestra: he interrupted a concerto in G major when Paul Wittgenstein, an Austrian pianist who had lost his arm during World War I, commissioned a concerto for the left hand, which he completed in 9 months before resuming the G major concerto [Marnat, 1986]. On November 20, 1931, he wrote to Henri Rabaud: 'Kindly excuse me regarding the Osiris competition: my concerto is finished, as far from being myself, I would risk falling asleep at the first candidate. I have been ordered complete rest, and am being treated with injections of serum' [Ravel, 1989].

The world première of the *Concerto for the Left Hand* by Wittgenstein on January 5, 1932 at Vienna took place in Ravel's absence. But the première of the Concerto in G major was given in Paris on January 14, 1932, Ravel conducting and Marguerite Long playing the solo part. Both made thereafter a European tour which lasted until April. In the summer Ravel received a commission for the film *Don Quichotte* and began to compose *Don Quichotte à Dulcinée*, a cycle of three songs for voice and piano [Marnat, 1986].

On the night of October 9, 1932, Ravel was injured in a taxi accident in Paris – assessed by Dr. Abel Desjardins [Mercier, 1991] – that, on his own confession 3 months later to Manuel de Falla, 'was not so serious: chest bruises and some facial cuts. Yet, I was incapable of doing anything, except sleep and eat' [Ravel, 1989]. On January 17, 1933, he nevertheless was able to conduct the Parisian première of his *Concerto for the Left Hand* with Wittgenstein as soloist. But shortly thereafter, he merely attended another performance in Monte Carlo and he had to decline an invitation for a Russian tour.

In June 1933, while on holiday at Saint-Jean-de-Luz, Ravel wanted to demonstrate to Marie Gaudin how to skim a pebble on the sea, but struck her on the face instead. A few days later he had to be brought in from the sea as, experiencing uncoordinated movements, he found himself suddenly incapable of swimming [Marnat, 1986]. On August 2, 1933, he wrote from Le Touquet, where he stayed in the villa of friends, to Gaudin: 'I feel more and more dull, Vallery-Radot: blood-pressure: rather weak. Blood test: urea high enough to alarm the doctor: it is back to normal but the [cerebral] anemia persists. Medication: a quantity of pills which I cannot oversee. After a month all the troubles were over' [Ravel, 1989]. Indeed, in November 1933 he conducted his *Boléro* and Concerto in G major in Paris but it was to be his last public performance. On February 6th, 1934, Ravel finally agreed to be treated in the clinic 'Mon Repos' near Vevey, but he did not improve since he had to use a dictionary in order to write a hardly decipherable letter to Maurice Delage on March 22, 1934 [Marnat, 1986].

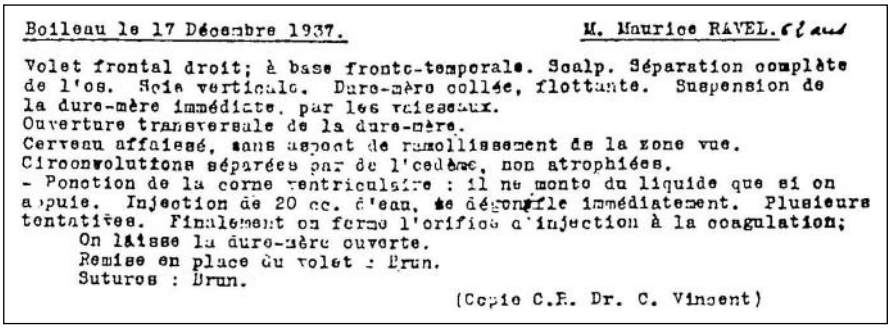
After Ravel's return, Manuel Rosenthal, a favorite pupil, noted his lack of control of movements which missed their mark, his defective eye movements which caused reading problems, and his speech difficulties which hampered communication. In order to assure the musical world that Ravel was still creative, Rosenthal then proposed to orchestrate *Don Quichotte à Dulcinée* and did so under his supervision [Rosenthal, 1995]. At the recording session of the work, in November 1934, Ravel 'made several remarks about wrong notes, tempi and dynamics, both for the voice and the instruments' [Orenstein, 1991].

On February 15, 1935, Ravel and the sculptor Léon Leyritz set out on a trip to Spain and Marocco. In August 1935, they again travelled to Spain. In April-May 1936, Ravel took a cure in the Institute at Lausanne where, according to the pension's housekeeper, he 'still [had] troubles to find proper names but set at ease he narrated his voyages or anecdotes on artists' [Ravel, 1989].

During 1936–1937, Ravel was eventually cared for by his housekeeper Mrs. Revelot at his house in Montfort-l'Amaury. Either alone or with Hélène Jourdan-Morhange, he made extensive walking tours in the nearby forest of Rambouillet, knowing every path and clearing, and pointing out every tree and bird [Colette et al., 1939]. At concerts his apathetic behavior and blank expression was 'frightened' [Demuth, 1947], 'he did not try anymore to speak, and yet, sitting between us, he gave the impression of somebody who is at risk of disintegrating' [Colette et al., 1939], but sometimes a growing irascibility also became apparent [Rosenthal, 1995]. Colette recalls that Ravel, 'grasped the teeth of his fork' at a dinner, looking at her with great despair [Colette et al., 1939] and Jacques de Zogheb noted that Ravel did not succeed to open the front door of his house [Jourdan-Morhange, 1945]. Yet, he showed insight, lamenting to Colette: 'it's tragic what comes over me', and to de Zogheb: 'Why does it comes over me, why?'

The Exertions of Ravel's Neurologists

According to Théophile Alajouanine, a famous Parisian neurologist, Ravel had 'a Wernicke aphasia of moderate intensity, without any trace of paralysis, without hemianopia (sic), but with an ideomotor apractic (sic) component' [Alajouanine, 1948]. In fact, in February 1936, Alajouanine noted [Mercier, 1991] a diffuse but moderate disturbance of speech and writing; the understanding of speech was generally better preserved than speaking and writing abilities; especially writing was seriously disturbed, in part due to the apraxia. He also noticed a dissociation between the impossibility of musical expression – both in writing and playing – and the still relatively well-preserved musical thinking. Ravel recognized not only melodies but also the precise tempo and the precise



Boileau le 17 Décembre 1937. M. Maurice RAVEL. 62 ans

Volet frontal droit; à base fronto-temporale. Scalp. Séparation complète de l'os. Scie verticale. Dure-mère collée, flottante. Suspension de la dure-mère immédiate, par les vaisseaux.
Ouverture transversale de la dure-mère.
Cerveau affaissé, sans aspect de ramollissement de la zone vue.
Circonvolutions séparées par de l'œdème, non atrophiées.
- Ponction de la corne ventriculaire : il ne monte du liquide que si on appuie. Injection de 20 cc. d'eau, se dégonfle immédiatement. Plusieurs tentatives. Finalement on ferme l'orifice d'injection à la coagulation;
On laisse la dure-mère ouverte.
Remise en place du volet : Brun.
Sutures : Brun.

(Copie C.R. Dr. C. Vincent)

Fig. 1. Craniotomy protocol by Clovis Vincent, December 17, 1937. With permission of Dr. Michèle Kujas, Service d'Histologie, Groupe Hospitalier Pitié-Salpêtrière, Paris.

pitch; in musical dictation, however, he made many mistakes, possibly due to a naming disturbance, given that his ability to sing previously played notes was still fairly good. Playing at sight at the piano was pitiful because, besides reading difficulties, he had problems to find the exact keys, although playing scales and his own compositions went somewhat more smoothly. Writing music was very difficult on dictation or when he tried to transcribe a composition, but was easier from his head. Singing melodies from his own compositions was still possible; moreover, he said that he heard melodies being sung in his head.

Urged on by Ravel's friends, two pioneering French neurosurgeons, first Thierry de Martel and subsequently Clovis Vincent, carried out numerous examinations, e.g. pneumoencephalography [Rosenthal, 1995]. Unfortunately, these documents have been lost, but Vincent's surgical protocol came to light in 1988 [Henson, 1988]. Vincent finally decided to perform a craniotomy and, on December 17, 1937, found, after a 'Right frontal bone flap; with fronto-temporal basis' a 'Slack brain, without actual softening in the area displayed. Gyri separated by edema, but not atrophied'. He tried to 'inflate' the lateral ventricle with 20 cm^3 water but did not succeed [Baeck, 1996] (fig. 1).

The Nature of Ravel's Illness

In January 1934, Dr. Vallery-Radot wrote: 'I have made various examinations to ensure that I did not miss a single lesion. There is none' [Ravel, 1989]. Consequently, the possibility of a systemic, vascular or infectious disease must be low.

Traumatic sequelae such as normal pressure hydrocephalus, whiplash [Otte et al., 2003], chronic subdural hematoma [Mahieux and Laurent, 1988] also

are unlikely given Ravel's focal symptoms, Desjardin's certificate, Alajouanine's examination and Vincent's protocol. Ravel neither complained of stiff neck, dizziness or headache nor did lose consciousness; his contemporaries mentioned neither gait disturbances nor incontinence; and craniotomy showed no signs of intracranial hypertension.

Nevertheless, the role of the taxi accident as a revealing factor of an underlying neurodegenerative disease cannot completely be ruled out [Kerner, 1975; Shafer, 1987]. Alajouanine's opinion was: 'The cause, though indefinite, belongs to the group of cerebral atrophies, there being a bilateral ventricular enlargement; but it is quite different from Pick's disease' [Alajouanine, 1948]. Alzheimer's disease [Dalessio, 1984] is hardly consistent with Ravel's relatively preserved memory, visuospatial skills and insight. However, the nosology of the so-called non-Alzheimer neurodegenerative disorders has much expanded since Alajouanine's days. Anatomopathological entities such as Pick's disease, corticobasal degeneration, progressive supranuclear paralysis and clinical syndromes such as frontotemporal dementia, nonfluent progressive aphasia and semantic dementia are now defined.

The frontal variant of frontotemporal dementia is ruled out because Ravel's striking apraxia is an exclusion criterion [Neary et al., 1998] and Alajouanine stated that Ravel's disease 'spares [his] personality' [Alajouanine, 1948]. Primary progressive aphasia (PPA) could be a diagnostic hypothesis [Henson, 1988]; but Alajouanine noted merely an 'aphasia of moderate intensity' opposed to an 'important apraxic (sic) component' [Alajouanine, 1948]. Ravel's dysexecutive syndrome with apraxia and aphasia also suggest corticobasal degeneration (CBGD) [Baeck, 1996]; however, asymmetric extrapyramidal symptoms have not been reported, since Dr. Michaud, who treated Ravel in 'Mon Repos', noted merely 'stiffness'.

Finally, might Ravel's disease have been hereditary? [Alonso and Pascuzzi, 1999]. Indeed, he worried in his last years that he would end as his father. But the mental deterioration of Ravel's father in his last two years – he died in 1908 at the age of 76 – probably had a cerebro-vascular origin as Ravel wrote on 28 July 1906: 'He was struck down by a cerebral hemorrhage … It happened during his sleep' [Ravel, 1989]. Nevertheless, it is noteworthy that, after the discovery of hereditary frontotemporal dementias (FLD) linked to markers on chromosome 17q21–22, a connection with PPA and CBGD has been suggested [Bird et al., 1997].

After all, instead of unravelling all neurodegenerative possibilities, it might be easier to conclude that Ravel's disease belonged to what Kertesz has called Pick complex, which includes FLD, PPA and CBGD [Kertesz et al., 1994]. Although the term itself has its opponents, an overlap is indeed accepted between clinical symptoms, macroscopic features of focal atrophy and underlying neuropathological changes [Rossor, 2001].

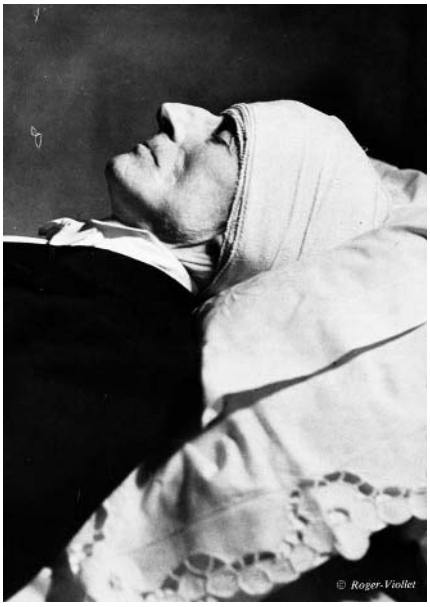


Fig. 2. Ravel on his deathbed, December 28, 1937. With permission, Presseagentur Dukas, Zürich.

Yet, there will never be absolute certainty since – after the trepanation from which Ravel died 10 days later, on the 28th December 1937 – no autopsy was performed (fig. 2).

The Last Compositions of Ravel

The relentless repetitions of the theme in *Boléro* have been interpreted as signs of disease [Cybulska, 1997; Kerner, 1975]. However, in Ravel's own words, *Boléro* 'is an experiment in a very special and limited direction […] consisting wholly of orchestral tissue without music – of one long, very gradual crescendo' [Calvocoressi, 1931]. Consequently, his later works do not have the *Boléro* characteristics anymore.

Considering that Ravel used in his *Concerto for the Left Hand* (fig. 3) a different style and structure than in the G major concerto, some authors have propagated the interesting idea that the *Concerto for the Left Hand* is 'music originating predominantly from the right hemisphere'. Acknowledging that the right hemisphere is activated, above all, by the processing of timbres whereas rhythm is associated mainly to the left, these authors claim that Ravel adopted the alternative use of different timbres to avoid the difficulty of elaborating a complex structured theme [Amaducci et al., 2002]. But there is properly neither

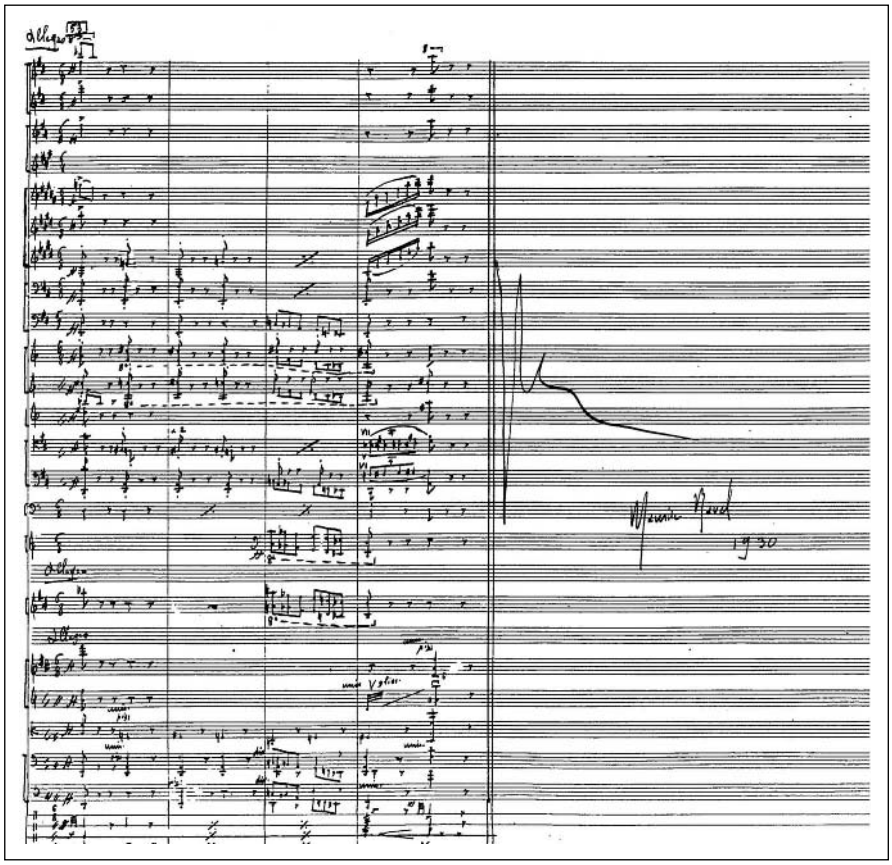


Fig. 3. Last page of *Pianoconcerto for the Left Hand,* dated 1930 and signed Maurice Ravel. With permission, The Pierpont Morgan Library, New York, Deposit Robert Owen Lehman.

more timbre nor less rhythm in the *Concerto for the Left Hand* than in the G major concerto.

Moreover, throughout his career, Ravel was fond of creating contrasting works. 'Planning the two piano concertos simultaneously was an interesting experience' he wrote [Calvocoressi, 1931]. Quoting Blom and Calvacoressi, the above-mentioned authors argue that the G major concerto is in fact the belated materialization of a plan that ever since his youth he had kept in the back of his mind. But at most, all he possibly used are sketches from *Zaspiak-Bat*, a short theme suggesting a Basque folk melody in the first movement of the G major concerto [Orenstein, 1991].

Of course, the *Concerto for the Left Hand* has a peculiar style, but this style was conditioned by Wittgenstein's commission and not by Ravel's disease. Ravel found it 'essential', as he wrote himself, 'to give the impression of a texture no thinner than that of a part written for both hands. For the same reason, I resorted to a style that is much nearer to that of the more solemn kind of traditional concerto' [Calvocoressi, 1931].

Finally, Ravel's pianoconcertos are not his last compositions. If the *Concerto for the Left Hand* should show features of a latent left hemisphere disease, then *Don Quichotte à Dulcinée*, written during 1932–1933, should show these presumed features even more. But they have neither melodic fragmentation, nor harmonic or rhythmic inconsistencies. Yet, the defenders of the hypothesis that disease influenced Ravel's last works quote musicologist Chalupt who suggested that *Don Quichotte* has been written 'by a friendly hand'. In fact, Ravel composed and completed – on April 6, 1933 he wrote to David Diamond: 'I have just finished *Don Quichotte à Dulcinée*' [Ravel, 1989] – these songs for voice and piano, they are thus genuine Ravel. And Rosenthal's orchestration, which was done in 1934 under his supervision, does not change anything of their musical content [Rosenthal, 1995].

Ravel still had plans for a ballet, *Morgiane,* of which only a few sketches remain, and for an opera, *Jeanne d'Arc* but admitted to Valentine Hugo in November 1933: 'I'll never be able to do my *Jeanne d'Arc*; that opera is there in my head but I'll never write it, it's finished, I can't write my music any more' [Hugo, 1952]. In July 1937, he said to Jourdan-Morhange, sobbing: 'I still have so much music in my head, I've said nothing, I still have so much to say' [Jourdan-Morhange, 1945]. His illness had annihilated his creativity.

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The Decay and Death of Modest Musorgsky

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Abstract

Modest Musorgsky (1839–1881) is the most original composer of a group known to musical history as 'The Mighty Handful'. The last years of his life are traditionally regarded as a period of physical and mental decay. However, outbursts of genius still occurred. He died at the age of forty-two from the consequences of chronic alcoholism, leaving his last two opera's, *Khovanshchina* and *The Fair at Sorochintsy*, unfinished.

Modest Musorgsky (1839–1881) wrote in his autobiographical sketch that he 'does not belong to any of the existing musical groups, neither by the character of his compositions, nor according to his views on music' [Orlova, 1983]. He was indeed the most idiosyncratic Russian composer of the 19th century. Of course, neurologists will focus on the last years of his life to determine the cause of his premature death at the age of forty-two and to find out if there might have been a decline in his creativity. But it will be unavoidable to trace his whole career first in order to comprehend his dramatic end.

An Unstable Life

Musorgsky was born in the village of Karevo, Pskov Province, on March 9, 1839 to 'an ancient Russian family' [Orlova, 1983]. As the second surviving son (two sons had died in infancy) of a landowner in tsarist Russia, he was destined to a military career. He thus attended the Cadet school of the Imperial Guards from 1852 to 1856 and, in 1856, enlistened in the Preobrazhensky Guards as an officer. However, aspiring to fulfill his musical ambitions, he resigned from the army on May 1, 1858.

In his autobiographical sketch, Musorgky singled out that at the age of 7 he 'played short compositions by Liszt' and at the age of 9 'the great concerto by Field before a large gathering in his parent's house' [Orlova, 1983]. At 13, he had already composed a piano piece which his father paid for to be published. But it was the acquaintance with Mily Balakirev, whom he met through a fellow-officer of the Guards, which proved to be decisive for his musical future. For several years, he joined the Stasov-Balakirev circle to which also belonged the military doctor Alexander Borodin, the army engineer César Cui and the naval cadet Nikolai Rimsky-Korsakov (the 'Five' amateur-musicians, dubbed 'The Mighty Handful' by their mentor the art critic Vladimir Stasov, all became famous composers). Musorgsky recalls that Balakirev's lessons 'covered the entire history of music through a strict systematic analysis' playing together on two pianos 'in chronological order examples drawn from the fundamental works of European composers' [Orlova, 1983]. Yet, a formal training in harmony, orchestration or composition was out of the question.

Shortly after leaving the army, Musorgsky suffered from 'a nervous ailment' for which he sought relief in a spa [Orlova, 1983]. During the summer of 1859 he was 'oppressed with a terrible illness […]. This was mysticism, mixed with cynical thoughts about the deity' [Orlova, 1983]. He recovered but ever since, his life was unstable. He never married nor founded a family of his own and lacked a permanent address for most of the rest of his life. His brother Filaret reports: 'During the years 1858–1863, he lived with different members of his family, until 1862 he lived with mother and me [their father had died in 1853], and in the latter part of 1862 and 1863, with me and my wife' [Musorgski, 1991].

The liberation of the serfs, decreed in February 1861, brought Musorgsky's family into financial difficulties. Consequently, at the end of 1863, Modest had to accept a commission as clerk in the engineering department of the Ministry of Communications. From 1864 on, he lived in a commune with five comrades. But after the death of his beloved mother in 1865 he fell seriously ill, suffering from a 'frightening disease (delirium tremens)', as his brother Filaret called it, adding that 'my wife made Modest leave the artel (workers' cooperative) and brought him home (against his will, at first)' [Musorgski, 1991]. He remained in his brother's household until the end of 1868.

In January 1869, Musorgsky resumed his civil employment at St. Petersburg, now in the forestry department of the Ministry of State Property. In search of a surrogate family, he was offered hospitality from the autumn of 1868 to May 1871 in the home of the Oposchinins where he began to compose *Boris Godunov*, based on Alexander Pushkin's play. However, in February 1871, the first version of this opera was rejected by the committee of the Mariinsky Theatre. Sharing a room from September 1871 to June 1872 with Rimsky-Korsakov until the

latter's marriage [Rimsky-Korsakov, 1914], Musorgsky wrote a second, completely recasted version of *Boris Godunov*, which was refused again. Eventually, a modification of this second version was created at the Mariinsky Theatre on February 8, 1874.

Living alone in St. Petersburg from mid-1872 until the end of 1874, Musorgsky began in 1872 at Stasov's instigation the complex elaboration of a new opera, *Khovanshchina*. Since the summer of 1874 he was also involved in another project, the comic opera *The Sorochintsy Fair* based on a tale by Nikolai Gogol. In 1875, he shared rooms with Count Arseny Golenishchev-Kutuzov, whose poems he had already set to music in the song cycle *Sunless* (1874) [Golenishchev-Kutuzov, 1991]. The *Songs and Dances of Death* were then composed, again on verses of the Count, who, however, moved at the end of July 1875. Musorgsky thereafter settled on Vasilyevsky Island in the household of Pavel Naumov, a retired marine officer and theater lover.

Unfortunately, Musorgsky's nightly stays in the public houses of St. Petersburg (especially in Maly Jaroslavez) began to exceed normal proportions. His lifestyle became rather irregular and careless. In March 1978, the death of his friend, the bass singer Osip Petrov, provoked a protracted drinking bout [Emerson, 1999]. Apollon Maykov 'was astonished, calling on Musorgsky one day. It was 2 o'clock in the afternoon' to 'found him in evening dress, asleep in an easy chair. On a table stood several bottles. There was nothing else in the room' [Orlova, 1983]. And Nikolai Lavrov recalls that 'there was (in a manner of speaking) a sort of incoherence plus a red face and grayish nose that at first glance revealed the typical alcoholic. His face was always somewhat swollen' [Orlova, 1983].

In the summer of 1878, Musorgsky was taken under the wing of the famous singer Darja Leonova, with whom he made a concert tour from June to October 1879 in South-Russia and in whose music school at St. Petersburg he worked afterwards as pianist-accompanist. On January 1, 1880, he was dismissed from the Department of Government Control, an event which prompted Stasov to ask Balakirev desperately for help because 'He's [Musorgsky] falling apart; since 1 January he's been without a job and without means of support! Now he'll start drinking even harder!' [Orlova, 1983]. His friends then decided to support him financially on the condition that he complete *Khovanshchina* and *The Fair at Sorochintsy.*

Loss of Creativity?

Stasov has divided Musorgsky's creative activity into three periods: 'a period of musical study, a period of full flowering of musical personality and creative

independence, and a period of incipient diminution and weakening' [Taruskin, 1993]. During the first period (1852–1864), Musorgsky composed piano works, songs, incidental music for *Oedipus in Athens* and some pieces for an unfinished opera *Salammbô* based on Flaubert's novel. His most famous works, the symphonic poem for orchestra *Night on the Bare Mountain*, the opera *Boris Godunov*, the piano suite *Pictures from an Exhibition*, and three song cycles: *The Nursery*, *Sunless* and *Songs and Dances of Death*, belong to the second period (1865–1875). The difficult elaboration of two unfinished operas, *Khovanshchina*, already begun in 1872, and *The Fair at Sorochintsy* characterizes the last period (1875–1880).

Golenishchev-Kutuzov on the contrary considered what Stasov had called Mussorgsky's 'decadent' period as 'the beginning of a new and fruitful period of creativity' [Golenishchev-Kutuzov, 1991]. He points rightly to an aesthetic change in Musorgsky's last works, stressing a growing importance of 'beautiful sounds' in contradistinction to the 'realistic' predominance of the former period. Thus, *Khovanshchina* eventually turned out to be quite different from the musical folk drama that Stasov had in mind when he suggested the subject to Musorgsky in 1872.

However, Richard Taruskin argues that the opinions of both Stasov and Golenischev might have been biased by the respective periods during which they exerted the greatest influence on the composer [Taruskin, 1993]. Moreover, neither Stasov nor Golenishchev being musicians, can be regarded as the most qualified judges of Musorgsky's scores.

Nevertheless, the composer and music editor Vyacheslav Karatygin also states that the year 1875 marks the chronological limit of the development of Musorgsky's talent and that 'afterwards, a decline set in, especially in the matter of quantity. Musorgsky composes slower, with lesser cohesion, in a more simple notation although outbursts of genius still occur until his last illness' [Karatygin, 1995]. Indeed, overwhelmed by the subject of *Khovanshchina*, Musorgsky continuously added new plots and discarded already composed music but he apparently lacked the necessary alertness and concentration to realize a coherent whole. The first act of *Khovanshchina* was finished in a rough state in the summer of 1875; the last act was begun in 1880 but remained unfinished. In the other three acts too, much is left incomplete. There also is much superfluous material that either had to be developed or rejected. Finally, Musorgsky's autograph consists of a vocal score with piano but, with the exception of some pieces, the music is not orchestrated [Lamm, 1995]. Consequently, in order to perform *Khovanshchina*, the music had to be completed, rewritten and orchestrated by Nikolai Rimsky-Korsakov in 1883. Maurice Ravel and Igor Stravinsky made a new orchestration in 1913, followed in their turn by Boris Asafyev in 1931 and Dimitri Shostakovich in 1959.

The same problems apply to *The Fair at Sorochintsy* which Musorgsky began in 1875. Only half of this opera in three acts is composed and the existing material (the fair scene, the dumka of Grizko, the song of Chiwrja, the scene with Chiwrja and Ivanovitch, the dumka of Parassja and the hopak) still shows many gaps [Karatygin, 1995]. The work was first completed in 1916 by César Cui who had to compose many extensions of his own, later by Nikolai Tsherepnin (1923) and by Vissarion Shebalin (1931).

In the end of the 1870s, Musorgsky still intended to write another opera *Pugachevshchina*, based on *The Captain's Daughter* by Pushkin, but he did not materialize this project.

A Dramatic Death

Darya Leonova recalls that on February 11, 1881 Musorgsky rushed unannounced into her apartment 'in a most nervous, irritable state and [said] that he had nowhere to go, he was left walking the streets, and that he had no resources at all and [thus] no way out of this situation' [Orlova, 1983]. Leonova let him in to regain his composure and both attended that evening a gathering at General Sokhansky's whose daughter was studying in Leonova's classes. Musorgsky accompanied her on the piano but, after singing, lost consciousness: 'It seemed he had suffered a stroke' but 'when it was time to leave, Musorgsky was completely recovered and on his feet'. Arriving at Leonova's apartment, he begged for permission to stay 'pleading in a kind of nervous, fearful condition' [Orlova, 1983]. Of course, Leonova consented and he slept the whole night in a chair.

The next morning, Musorgsky 'responded that he felt fine. With these words he turned to his right and suddenly fell full length [on the floor]. By evening there had been two more such attacks' [Orlova, 1983]. Alerted by Leonova, Musorgsky's friends pleaded with Dr. Lev Bertenson, appealing to his admiration for musicians and writers, to treat the sick composer in a hospital. Bertenson was then junior staff physician at two hospitals, the Rozhdestvensky City Hospital for common laborers, with general wards only, and the Nikolaev Military Hospital for soldiers and officers. However, the head physician of the Military Hospital only agreed to Musorgsky's admission on the condition that he should be registered as Bertenson's 'hired civilian servant' [Bertenson, 1991]. In 1922, forty years after the events, Bertensov claimed that his patient was placed 'in the quietest and the most isolated part of the hospital, in a spacious, sunny room with a high ceiling, furnished with the necessary (although not stylish) furniture', that he received the best treatment by 'two nurses […], hospital attendants, and a doctor's assistant', and that in addition to an officer's

Fig. 1. Portrait of Musorgsky (1881) by Ilya Repin. Tretyakov Gallery, Moscow, with kind permission.

ration, he 'received many different dishes in generous amounts from close friends' [Bertenson, 1991]. He thus blamed Mikhail Ivanov for having written in 1909 that 'the wretched surroundings [of Musorgsky's hospital stay] wrung one's heart' [Ivanov, 1991].

In a letter to Balakirev, dated February 15, 1881, three days after Musorgsky's admission, Stasov reports: 'The doctors are saying that he didn't have strokes but the beginnings of epilepsy […]. He looks as if nothing happened – now – he knows everybody, but he talks the devil knows what sort of nonsense and tells countless fantastic stories. They also say that, besides the epilepsy and strokes, he is also somewhat mad' [Orlova, 1983]. And the memoirs of Rimsky-Korsakov are concordant: 'Happy over our visit, he sometimes talked with us completely normally; but suddenly, he would pass into a mad delirium' [Orlova, 1983].

Nevertheless, a significant improvement occurred. In March, Repin portrayed Musorgsky in four sittings (fig. 1) and later recalled that 'He was living under a strict regimen of sobriety, and he was in a particularly healthy and sober condition' [Orlova, 1983]. Unfortunately, Musorgsky's condition worsened suddenly, due in Ivanov's words, to 'his own gross imprudence' [Ivanov, 1991]. Ivanov does not specify in his obituary of Musorgsky what this imprudence was, but an entry for March 17, 1881 in the diary of Ilya Tyumenev reveals what happened: 'His brother came and brought money [in view of the anniversary of Musorgsky's birthday which was erroneously, even in his autobiographical sketch, dated on 16 March]. Although the guard was strictly forbidden from

obtaining alcohol for the sick, Modest Musorgsky, driven to extremes by his disease, offered the guard a commission of 25 roubles, and that seduced him; he took the money and brought alcohol. For this money, the miserable patient bought himself a premature death' [Orlova, 1983]. Ivanov reports that 'recovery was no longer a possibility. His arms and legs were paralyzed […] Paralysis was gradually taking hold of his respiratory organs: he was able to breathe only with difficulty and constantly complained about the lack of breath. However, up until the last minute he was mentally alert' [Orlova, 1983]. Musorgsky passed away on March 16, 1881 at 5 o'clock in the morning.

According to Ivanov, Musorgsky's 'complicated' illness included such diagnoses as 'liver disorder, fatty tissue in his heart, and inflammation of the spinal cord' [Orlova, 1983]. He probably had made inquiries of Vasily Afanasiev, medical examiner of Nikolaevsky Hospital, who had performed an autopsy (the composer's name does not appear, however, in the table-book, which mentions in March 1881 only one man who died of alcoholism) [Orlova, 1983].

The Consequences of Chronic Alcoholism

Stasov was outraged about Ivanov's assertion in his obituary of Musorgsky that [Musorgsky's] enemy was that sad propensity which has ruined so many gifted Russian people. This fatal passion gripped him powerfully, it sent him to the grave, along with his artistic endeavors' [Orlova, 1983]. In his own obituary notice of Musorgsky, he mentions more decent causes of death: 'paralysis of the heart and of the spinal cord' [Emerson, 1999]. Nevertheless, he too knew the truth, having reported to Balakirev on February 13, 1881 the opinion of Musorgsky's physician that 'he might be able to live a year or more, but he could also die right now, at once; it is impossible to guess, especially if he were to drink again' but adding significantly 'Just imagine, yesterday at Leonova's apartment […] in the interval between the second and the third strokes, he was already asking for wine!' [Orlova, 1983].

Moreover, testimonies of other contemporaries and biographical essays of later musicologists, all agree that Musorgsky's chronic alcoholism was the cause of his physical and mental decline.

Alluding to the Naumovs with whom Musorgsky stayed from 1875 to 1878, Sinaida Savjolova argues that 'He [Musorgsky] contacted people who stimulated and favored this fateful passion, which already awoke in the time of his military service' [Savjolova, 1995].

In his recollections, Nikolai Kompaneiskii deplores that 'From day to day [since the stage performances of *Boris Godunov*] the influence of alcohol showed an increasing effect on the alertness of his spirit and on his potentiality to cope

with the huge nervous stress without which neither inspiration nor creativity are possible. The clouding of his mind and the weakening of his organism led to a stagnation of his creativity' [Kompaneiskii , 1995].

Karatygin in his turn is convinced that 'Alcoholism, this fatal vice to which Musorgsky gave in evermore and which since the late seventies not only severely damaged his general health condition but also weakened the musical logic of his compositions, hindered the completion of *Khovanshchina*'. And Musorgsky's intemperance has led, in his opinion, to a further disablement of his extremely sensitive psychic nature [Karatygin, 1995].

However, Karatygin's conclusion might be reversed. In an interesting and well documented paper, Lerner refers to the events of Musorgsky's life and concludes that his alcoholism was rather secondary in nature, considering that 'intemperance often accompanies mental disorders' and that 'chronic alcoholism is likely to conceal a disguised mental disorder that is somewhat alleviated by drinking'. According to him, Musorgsky suffered from a schizoaffective disorder, mixed type [Lerner, 1998].

Lerner's argument expands thus Musorgsky's pathography but anyway, primary or secondary, the neurological evidence of alcoholism remains [Victor, 1990]. Apparently, Musorgsky had withdrawal seizures (rum fits) followed by delirium tremens: the first event necessitated his removal from the commune in 1865, the second occurred in 1878 and the last, repetitive fits compelled to hospital treatment in 1881. Of course, a differential diagnosis of this last attack has to be made with a stroke, a not so infrequent cause of symptomatic seizures [Burn et al., 1990]. As we learn from Stasov's letter, even Musorgsky's physicians have taken this into consideration. But the antecedents, the absence of hemiparesis and the initial improvement during the hospitalization speak against this eventuality. Moreover, the additional diagnosis of 'liver disorder, fatty tissue in his heart and inflammation of the spinal cord' reported by Ivanov, points to alcoholic cirrhosis, cardiomyopathy and polyneuritis. Finally, Stasov's letter mentioning 'he talks the devil knows what sort of nonsense and tells countless fantastic stories. They also say […] he is also somewhat mad' suggests clearly Korsakov's psychosis. The ultimate cause of Musorgsky's death, occurring after what Ivanov has called 'a gross imprudence', i.e. a drinking bout, was thus in all probability a Wernicke-Korsakov syndrome. And it can be regarded as a remarkable coincidence that in the same year of Musorgsky's death (1881), Wernicke published the syndrome of acute hemorrhagic polioencephalitis superior [Wernicke, 1881], whereas Korsakov's description of the syndrome that carries his name appeared in 1887 [Korsakov, 1887].

Although Musorgsky thus died from the consequences of severe chronic alcoholism, Leont'ev might nevertheless have the last word: 'It had been said that his death was the result of his excessive passion for alcohol. What nonsense!

What blasphemy! Even if there is some physiological truth in that statement, he mainly fell victim to the burden of fulfillments that exceed the human potential to which he was condemned by his genius, and… to poverty' [Leont'ev, 1991].

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Georg Friedrich Händel's Strokes

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Abstract

Georg Friedrich Händel was a musical giant and probably the first composer to be the manager and promoter of his own works. The story of his life and his illnesses is full of myths, invented and embellished by various biographies. Existing pathographies written by authors from various specialties suggested him having suffered from psychiatric diseases like cyclothymia or mania and rheumatologic disorders like arthritis, while others tended to interpret his recurrent palsies as typical sequelae of ischemic strokes. During his last years of life, Händel was struck with blindness, which in his era had been interpreted as being due to cataracts. This led to three 'coucher' operations, all of them without any lasting effect. Although a definite diagnosis cannot be inferred from the original sources, the most plausible explanation for Händel's palsies and visual impairment may be based on one single context, i.e. cerebrovascular disease. The possible differential diagnosis will be discussed in this paper.

Notes about Biography and Character

Händel was born in 1685 in Halle an der Saale, the son of a 62-year-old surgeon and a 33-year-old priest's daughter. He received his musical education in Halle and Hamburg. The medical history of his parents is remarkable for cerebrovascular disease in his mother and grandmother: Händel's mother died at the age of 79 years in 1730. Händel had visited her for the last time in June 1729, when she allegedly recovered from a stroke 'paralyzing the whole of her right side and her tongue' [Chrysander, 1858, 1860, 1867]. Also Händel's grandmother Dorothea Taust is said to have died from a stroke [Flesch and Baselt, 1978–1986]. He travelled to Italy in 1706 and returned from there as a celebrity in 1710. Known as 'il caro Sassone', he was an excellent virtuoso on the harpsichord and was already renowned as a composer in the musical world

Fig. 1. Georg Friedrich Händel. Engraving after the portrait by Sir Thomas Hudson (1749). With permission of the Staats- und Universitätsbibliothek Hamburg, Germany.

of his time. He got an appointment to become the 'Kapellmeister' at the court of the 'Kurfürst' in Hannover, who ordered Händel to move to London and to work as a composer in England, which he rarely left until his death in 1759.

Händel is described as an intelligent and well-educated man, who was able to talk and to write in German (with a Saxonian accent), English, French and Italian [Deutsch, 1955]. He was said to be quite handsome as a young man, which is the reason for rumors concerning romantic affairs with young ladies of the aristocracy. Portraits of Händel as an elderly man, however, show a rather corpulent man [Rackwitz, 1986] (fig. 1), being reported to consume incredible quantities of food as well as being partial to Port and Madeira [Coxe, 1799]. As a matter of fact, Händel became easily irritable and was said to be hot-tempered in his function as a conductor and concert manager. Some reports exist of eruptions of anger, curses and verbal 'thunder and lightning' even on stage [Deutsch, 1955; Keynes, 1980; Mainwaring, 1760]. One day he had a discussion with an Italian singer, Ms. Cuzzoni, who refused to sing the aria 'falsa immagine' from Händel's opera 'Ottone', ending with the words '*Oh! Madame, je sais bien que vous êtes une véritable Diablesse, mais je vous ferai savoir,*

moi, que je suis Belzebu, le Chef des Diables.' (Oh, lady, I know well, that you are a true devil, but I will show you that I am Beelzebub, the chief of the devils). He finally lifted her up from the floor and threatened to throw her out of the window [Mainwaring, 1760].

What Neurological Disorder Did Händel Suffer From?

There are two main features deriving from a closer look onto Händel's illnesses: progressive relapsing and finally progressive visual loss, first described in 1751 and recurrent palsies dating back to 1737, 1743 and 1745.

A first mention of this symptom can be read in the London *Daily Post* on April 30, 1737, when Händel was 52 years old: 'Mr. Handel who has been some time indisposed with the rheumatism, is in […] way of recovery'. On May 14, 1737, the London *Evening Post* reports: 'The ingenious Mr. Handel is very much indispos'd, and it's thought with a Paraletick Disorder, he having at present no Use of his Right Hand, which, if he don't regain, the Publick will be depriv'd of his fine Compositions' [Deutsch, 1955]. We get closer to the symptoms of his disease by reading a letter of James Harris to the Earl of Shaftesbury dated May 5, 1737: 'Y^{r}Lord^{p}^{s} information concerning M^{r} Handels Disorder was y^{e} first I received – I can assure Y^{r} Lord^{p} it gave me no Small Concern- when y^{e} Fate of Harmony depends upon a Single Life, the Lovers of Harmony may be well allowed to be Sollicitous. I heartily regrett y^{e} thought of losing any of y^{e} executive part of his meritt, but this I can gladly compound for, when we are assured of the Inventive, for tis this which properly constitutes y^{e} Artist, & Separates Him from y^{e} Multitude. It is certainly an Evidence of great Strength of Constitution to be so Soon getting rid of So great a Shock. A weaker Body would perhaps have hardly born y^{e} Violence of Medicines, w$^{ch.}$ operate So quickly' [Deutsch, 1955]. The Earl of Shaftesbury made the following remark in his 'memoirs of Händel': 'And when the heats of summer 1737 came on, the disorder seemed at times to affect his understandings […] the palsy took entirely away the use of 4 fingers of his right hand and totally disabled him from playing.' In Mainwaring's biography we find additional information to this point: 'The observation that misfortunes rarely come alone, was verified in Handel. His fortune was not more impaired than his health and his understanding. His right-arm was become useless to him, from a stroke of the palsy; and how greatly his senses were disordered at intervals, for a long time, appeared from an hundred of instances, which are better forgotten than recorded. The most violent deviations from reason are usually seen when the strongest faculties happen to be thrown out of course' [Mainwaring, 1760]. Until then he recovered fairly rapidly from the palsy but there is no doubt, that

the concomitant circumstances, i.e. intermittent states of mental confusion which might even have been aphasia interpreted as confusion over a longer period of time, were a big shock for his friends and the public, worrying about Händel's future as a composer and virtuoso. As mentioned by Frosch [1989] Händel obviously sought help at this time, because there is a brief remark in Händel's hand at the bottom of a manuscript dating from the year 1737: 'Mr. Duval medecin in Poland St.'. There is no question about the fact that Händel finally made a reasonable recovery 6 months after his stroke, after receiving famous cure treatment while he was in Aix-la-Chapelle (today Aachen) for 6 weeks, staying for longer periods in the hot waters than usual [Deutsch, 1955]. This cure is mentioned in Stefan Zweig's novel based on Mainwaring's biography, culminating to the point when Händel is sitting down at the local organ improvising at full strength, to thank God for the wonderful cure [Zweig, 1929]. Although fully recovered before the end of the cure, Händel stayed for at least 6 weeks, which was according to Mainwaring 'the shortest period usually allotted for bad cases'. The London *Daily Post* announced Händel's return on November 7: '[Mr. Handel is back from Aix-la Chapelle] greatly recovered in his health' [Deutsch, 1955].

In 1742, an interesting remark can be found in the composer's own hand concerning a duet entitled 'Troppo cruda': '*Dieses ist so verwirrt geschrieben, wie mein Kopf ist, habe niehmanden es abzuschreiben verdammen wollen*' (This is as confusingly written, as my head feels, and I didn't want to make somebody copy it) [Leichtentritt, 1924]. Obviously, Händel's criticism regarding his composing performance at that point is not shared by contemporary artists since the duet 'Troppo cruda' has been recorded several times on compact disc by various artists.

A brief recurrence of similar symptoms may have occurred in 1742, when Händel traveled to Ireland. Discovered only in 1985, English oboist Simpson remarks: 'When Handel was in Dublin, he was attacked by another Paraletic stroke, while he was at dinner with my father Dubourg. it was violent and universal. it hap'ned luckily, that doctors Barry and Quin, & Mr. Nichols, Surgeon General, were present.… by violent bleedings & other evacuations, & by the immediate assistance he receiv'd he was soon perfectly recovered, & had never any return of it, tho' very apprehensive' [Mann, 1985]. To comment on this contemporary report, one can only speculate on the nature of the 'Paraletic stroke'. Most probably bleedings and other evacuations refer to therapeutic interventions like blood-letting by the accompanying doctors, which might have been useful through a reduction of hematocrit.

In 1743, Händel had had a further serious impairment of his health, preventing him from taking part at the oratorio concerts. This was the reason for a visit in Tunbridge Wells or Cheltenham for a cure, both being preferred localities for Händel's cures. In a letter written by Horace Walpole to Horace Mann

dating from May 4, 1743 it says: 'We are likely at last to have no Opera next year: Handel has a palsy, and can't compose' [Deutsch, 1955]. At this point of Händel's disease, Mainwaring comments briefly 'some return of his paralytic disorder' [Mainwaring, 1760], while valuable additional information can be drawn from a letter by Charles Jennens to Edward Holdsworth (April 29, 1743): 'I hear Handel has a return of his Paralytick Disorder, which affects his Head and Speech. He talks of spending a year abroad, so that we exspect no Musick next year…' [Flesch and Baselt, 1978–1986] Indeed, Händel's creativity may have been disturbed only until mid-June of the same year when he worked at 'Semele'. When Jennens adresses himself to Holdsworth again on September 15 we learn that: '[…] Handel is perfectly recovered and has composed a new Te Deum' [Flesch and Baselt, 1978–1986]. Interestingly, on the occasion of this brief illness, there can be found the first clues to an impairment of Händel's speech. This is also the point, where a rheumatic disorder or peripheral nerve lesion can definitely be ruled out. The defective state of his speech may be interpreted either as a dysphasic or dysarthric component. The latter being seen quite frequently together with isolated motor impairment of the upper extremity as the dysarthria-clumsy-hand syndrome due to a lacunar infarction in the basal ganglia or brain stem, respectively. There is no exact comment on this second palsy at this point, especially on any leg involvement, but with an impairment of his speech at the same time the palsy may well have affected his right arm, this being a possible reason for Händel's inability to compose for a short period of time [Deutsch, 1955].

A new breakdown occurred in summer 1745. Händel had made plans to visit the Harris family, but hesitated to leave his home because of his state of health (Malmesbury papers I,3; August 25, 1745). On October 24, Lord Shaftesbury states in a letter to his cousin James Harris: 'Poor Handel looks something better. I hope he will entirely recover in due time, though he has been a good deal disordered in his head' [Deutsch, 1955]. Again we find proof of Händel's behavioral disorders occurring in phases of his 'paraletick disorder'.

Händel's Visual Impairment

Sometime in 1750, Händel lost his sight. Again, this process was disabling the great composer not at once but in recurrent 'strokes'. Händel personally noted in the score of 'Jephta': '*Biss hierher komen den 13. Febr. 1751 verhindert worden wegen relaxation des gesichts meines linken auges so relaxt*' (got as far as this on Wednesday 13th February 1751, unable to go on owing to weakening of the sight in my left eye, fig. 2) [Deutsch, 1955]. Interestingly enough, it is Händel's left eye which gives indication of start of the 'eye' problems, and,

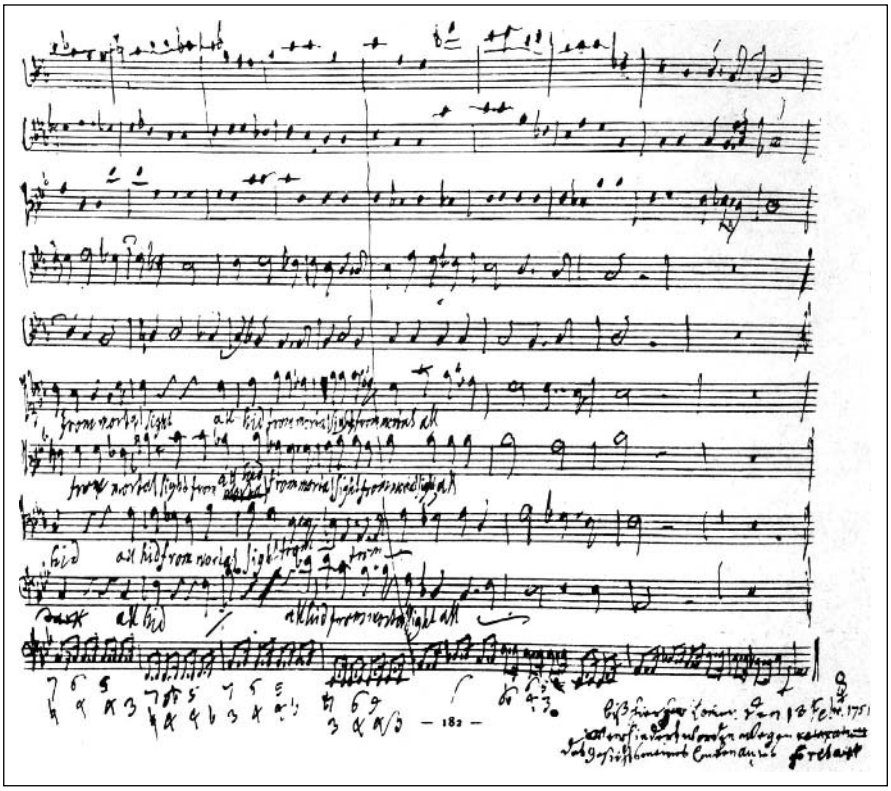

Fig. 2. Händel's notice in the score of 'Jephta': '*Biss hierher komen den 13. Febr. 1751 verhindert worden wegen relaxation des gesichts meines linken auges*' (Got as far as this on Wednesday 13th February 1751, unable to go on owing to weakening of the sight in my left eye). With permission of The British Library.

moreover, it is noticeable that, in this state of mind, Händel writes in German. Ten days later he states: '*den 23. dieses etwas besser worden wird angegangen*'. (Saturday the 23rd of this month a little better, started working again) [Deutsch, 1955]. Sir Edward Turner wrote to Sanderson Miller on March 14, 1751: 'Noble Handel hath lost an eye, but I have the Rapture to say that St. Cecilia makes no complaint of any Defect in his Fingers' [Deutsch, 1955]. This is an interesting remark, since the author seems to be aware of a possible occurrence of blindness together with motor symptoms. March 13, 1751, James Harris received a letter from the Countess of Shaftesbury: '…I went last Friday to 'Alexander's Feast'; but it was such a melancholy pleasure, as drew tears of sorrow to see the great though unhappy Handel, dejected, wan, and dark, sitting by,

not playing on the harpsichord…' [Deutsch, 1955]. And – strange enough for a blindness caused allegedly by cataracts, as was thought to be the case in Händel – the genius was able to play the harpsichord again as soon as the following month of April. He went to Cheltenham again until June 1751 (as reported in the General Advertiser on June 15, 1751) [Deutsch, 1955]. After his return, Händel consulted Samuel Sharp, surgeon to Guy's hospital, to undergo eye surgery. Sharp diagnosed 'gutta serena' (drop serene) which corresponds to the modern term of amaurosis, and which was then diagnosed in cases of 'an abolition of the sight without any apparent cause or fault in the eyes'. On August 17, 1752, we find the following note in the same journal: 'We hear that George Friderick Handel, Esq; the celebrated Composer of Musick was seized a few days ago with a paralytic Disorder in his Head, which has deprived him from sight' [Deutsch, 1955]. On November 4, Händel had been couched again, this time by the Princesses' of Wales doctor William Bromfield. 'Yesterday, George-Frederick Handel, Esq; was couch'd by William Bromfield, Esq; Surgeon to her Royal Highness the Princess of Wales, when it was thought there was all imaginable Hopes of Success by the Operation, which must give the greatest Pleasure to all Lovers of Musick' (General Advertiser, November 4, 1752) [Deutsch, 1955]. A short time later we find a notice in the Cambridge Chronicle (January 13, 1753): 'Mr. Handel has so much recovered his sight that he is able to go abroad', meaning that Händel was able to go out of doors [Deutsch, 1955]. Only 2 weeks later the public was confronted with the following news: 'Mr. Handel has at length, unhappily, quite lost his sight. Upon his being couch'd some time since, he saw so well, that his friends flattered themselves his sight was restored for a continuance; but a few days have entirely put an end to their hope' [Deutsch, 1955]. From 1753 on his visual impairment forced Händel to dictate his compositions to Christopher Smith. However, until his last days, there are some documents existing with remarks in his own handwriting, e.g. small corrections made in his testament. There was even a third eye operation by the 'Chevalier' John Taylor in Tunbridge Wells in 1758 (see London Chronicle, August 24, 1758) [Deutsch, 1955]. Taylor was the same man, who twice couched Johann Sebastian Bach without lasting success in 1750, and may actually have induced an iatrogenic wound infection leading to Bach's death in July 1750.

Which Medical Evidence Is There to Support These Diagnoses?

Händel's eye problems should not be interpreted without taking his cerebrovascular disorder into account. To come as close as possible to a diagnosis, we have to discuss the differential diagnosis of cerebrovascular disease possibly

linked to repeated visual impairment followed by blindness. Knowing that Händel suffered from recurrent palsies exclusively on his right side, together with speech impairment occurring in stroke-like episodes one must consider left hemisphere embolic ischemic events, most possibly due to left internal carotid disease [Hennerici and Daffertshofer, 1995; Szabo et al., 2001]. The most exact clinical description given by The Earl of Shaftesbury who reports a 'palsy [that] took entirely away the use of 4 fingers of his right hand and totally disabled him from playing' occurred in 1737. This is worth a closer look, because a clinical pattern like this may well be caused by a small emboligenic stroke in the cortical hand knob, as depicted and published in the Magnetic Resonance Imaging era, and which has usually a rather benign course [Back and Mrowka, 2001; Gass et al., 2001].

Partial or monocular loss of vision in carotid disease, usually occurring ipsilaterally, results in most cases from a central retinal artery occlusion or from one or more branch occlusions. Other discussed causes of monocular visual loss in patients with carotid artery disease include ischemic optic neuropathy (rarely simultaneous episodes of monocular visual loss and hemodynamic cerebral infarctions are described as optico-cerebral syndrome), venous stasis retinopathy, and the ocular ischemic syndrome with its sequelae [Bogousslavsky et al., 1987; Chawluk et al., 1988; Goodwin et al., 1987; The Amaurosis fugax Study Group, 1990].

Retinal stroke from central artery occlusion or branch artery occlusion presents clinically with acute loss of visual acuity (Händel: 'relaxation' of his eye), visual field, or both. Visual loss is usually unilateral, but it may be bilateral and simultaneous, if there is bilateral carotid disease. Some patients experience transient visual loss (amaurosis fugax) before persistent visual loss occurs [Hennerici and Daffertshofer, 1995; Szabo et al., 2001]. The most common risk factor is systemic hypertension, others include cardiac disease, diabetes mellitus and a history of cigarette smoking and alcohol abuse. As far as we can determine, this is probably very close to Händel's risk factor profile.

The onset of ischemic optic neuropathy, which is the most likely diagnosis and may involve both anterior and posterior – retrobulbar – optic pathways is usually acute. It often occurs in patients with severe carotid artery disease and may be associated with repeat ischemic cerebrovascular events prior to the blockage. There is usually painless loss of visual acuity, there may be an altitudinal, arcuate, or less common, a central field defect. Visual function stabilizes within several days to weeks, although it may improve or worsen in a stuttering fashion. Even slowly progressive ischemic optic neuropathy has been described in patients with carotid occlusive disease, sometimes due to additional venous stasis retinopathy and chronic ocular ischemia [Hennerici and Daffertshofer, 2001].

Taking into account the long course of Händel's disease with possible stroke recurrences in 1737, 1743 and 1745 and the visual problems only starting in 1751, the differential diagnosis of Händel's palsies must include lacunar strokes which show often complete recovery in function and do have recurrences with long symptom-free intervals. A recent study has shown a proportionally benign course of lacunar stroke with survival rates similar to those of the general population for the first 5 years following the first lacunar stroke with recurrent lacunar stroke occurring in 23.5% of the patients, corresponding to an annual risk of 2.4% [Staaf et al., 2001]. The suitable lacunar syndrome in Händel's case according to the classical papers may have been dysarthria clumsy hand syndrome, a 'pure motor' stroke sparing the leg [Fisher, 1967]. Since there is no single reference quoting pain as a symptom during the episodes of palsy, the repeatedly proposed diagnoses of muscular disorder, arthritis, peripheral neuropathy [Frosch, 1989], radiculopathy, brachialgia [Franken, 1997] or muscular rheumatism [Keynes, 1980] are highly unlikely. The authors preferring such diagnoses commonly seek to rule out cerebrovascular disease and argue that recurrent strokes are unlikely in Händel because of missing sequelae, obvious progression or progressive mental deterioration [Keynes, 1980]. However, all of these are certainly not obligatory for cerebrovascular disorders as mentioned above.

To summarize these pathophysiological remarks, we conclude that Händel, a man with a probable wide profile of risk factors including systemic hypertension, smoking and most probably hyperlipidemia, had recurrent palsies of his right side (following the available sources involving uniformly his upper extremity), partly with simultaneous speech impairment, either dysarthia as a component of the dysarthria-clumsy-hand-syndrome or dysphasia as a result of embolic middle cerebral artery stroke, which may have been interpreted as confusion. He may well have had a severe stenosis of his left carotid artery with recurrent embolism to the left hemisphere. Since for a certain period of time only his left eye was affected by visual loss, the suspected left carotid artery stenosis may also have been the source for embolic events within the left retina.

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The Subcortical Vascular Encephalopathy of Joseph Haydn – Pathographic Illustration of the Syndrome

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Abstract

When Joseph Haydn died in 1809, the 77-year-old man was the most famous composer of his time. With increasing age, he complained of progressive forgetfulness preventing him from composing for about the last 8 years of his life. He spent his days increasingly immobilized and inactive, suffering from a disabling gait disturbance. Still, most biographers quote the diagnosis of the composer's final illness and a reason for his death as diffuse atherosclerosis and congestive heart failure (CHF). A more sophisticated pathography, however, can be referred to detailed analysis of documents and sources, which lead to a diagnosis of subcortical vascular encephalopathy (SVE). SVE is caused by progressive cerebral microangiopathy presenting with two main types of pathological changes: recurrent lacunar strokes and widespread diffuse white matter changes. Recently, research criteria have been proposed for subcortical vascular dementia, which is a misnomer for the syndrome, since important and predominant features as mood changes, urinary symptoms, and in particular a characteristic gait disturbance are not incorporated in this term while dementia is only mild and occurs later in the course. All of these manifestations, which probably result from ischemic interruption of parallel circuits from the prefrontal cortex to the basal ganglia and corresponding thalamocortical connections can be found in the famous composer. Haydn was severely disabled by the symptoms of SVE for several years and often reported difficulties in finishing his last oratorio *Die Jahreszeiten*. Subsequently, the disease prevented him from composing another large oratorio which had already been drafted. Finally, the progress of SVE stopped his long career as a composer at the age of 73 years.

When in 1791 two of the major musical geniuses of all time, Haydn and Mozart, met in London Haydn's biographers, Griesinger and Dies both comment

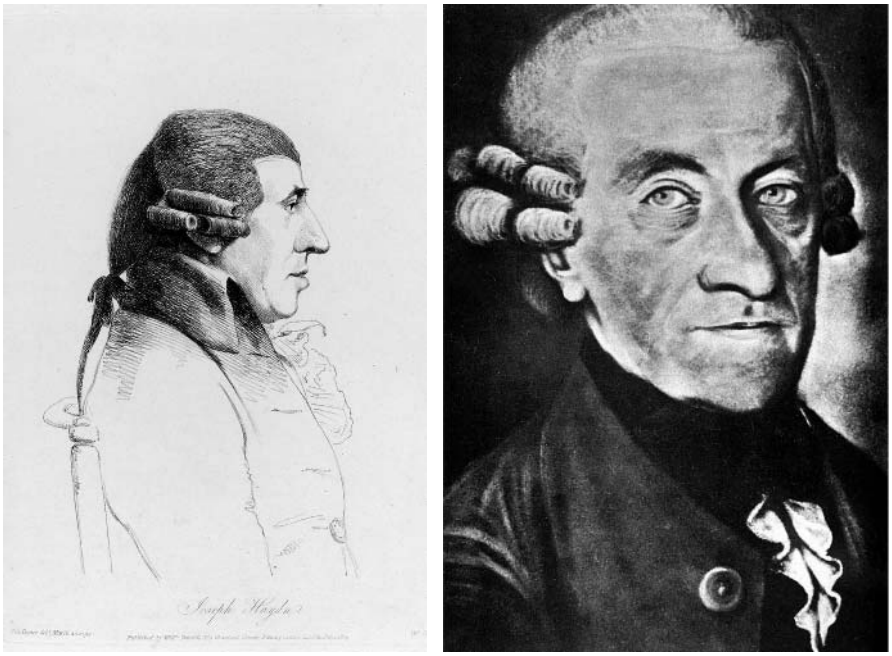

Fig. 1. a Pencil drawing by George Dance, dated March 1794. Haydn himself stated that this drawing resembled him the most. 'His nose of a hawk (he suffered from a nasal polyp which undoubtedly enlarged this feature) as well as the other parts of his face were pox-scarred' [Dies, 1810]. Courtesy National Portrait Gallery, London, UK. ***b*** Unsigned pastel, portrait showing Haydn in his last years, whose authenticity has been doubted by Somfai [1966], shows a toothless old man, the cheeks markedly colored, which has been taken as a sign of congestive heart failure by Franken [1976]. Courtesy Verlag Bärenreiter, Kassel, Germany.

on the emotional moment of Haydn's departure when Mozart is reported to say: 'we're going to say farewell for the last time in our lives' [Dies, 1810; Griesinger, 1810, 1987; Landon, 1988]. At that time, Mozart was 34 years old and might not have anticipated himself as the one to die only a few months later. 58-year-old Joseph Haydn (fig. 1a), however, became a famous man in London and survived his younger colleague by more than 18 years.

Until today, it has been suspected that the 78-year-old Haydn died of generalized atherosclerosis and weakness (fig. 1b) [Carpani, 1812; Dies, 1810; Franken, 1976; Griesinger, 1810, 1987; Landon, 1976–1980; Neumayr, 1989; Pohl, 1875–1927]. However, a more sophisticated study of the few authentic sources and of Haydn's early biographies led us to construct a more differentiated pathography based on more recent insight into a common disorder of the elderly termed subcortical vascular encephalopathy (SVE) [Bäzner and Hennerici, 1997].

Table 1. Etiologies, brain changes and clinical syndrome of subcortical vascular 'dementia'

Etiology

Primary vascular mechanism

Small vessel disease: Obliteration and occlusion, increased resistance, decreased autoregulation, endothelial changes, blood-brain-barrier and -carrier changes, perivascular changes

Brain changes

Primary type

Ischemic WMLs: Araiosis, état crible, demyelination, axonal loss, changes in oligodendrocytes and glial cells, incomplete infarcts

Lacunar infarcts

Primary location

WMLs: Extending periventricular and deep WMLs affecting especially the genu or anterior limb of the internal capsule, anterior corona radiata and anterior centrum semiovale

Lacunes: Lacunes in the caudate, globus pallidus, thalamus, internal capsule, corona radiata, frontal white matter

Clinical syndrome

1. Early presence of a gait disturbance (small-step, 'magnetic' or apraxic-ataxic gait)
2. History of unsteadiness and frequent, unprovoked falls with clinical signs of postural disturbance
3. Episodes of mild upper motor neuron involvement such as drift, reflex asymmetry, incoordination
4. Behavioral and psychological symptoms such as depression, personality change, emotional incontinence, psychomotor retardation
5. Early urinary frequency, urgency, and other urinary symptoms not explained by urologic disease
6. Dysarthria, dysphagia, extrapyramidal signs (hypokinesia, rigidity)
7. Cognitive syndrome interfering with complex (executive) social activities not due to physical effects of cerebrovascular disease alone
   a) Dysexecutive syndrome: Impairment in goal formulation, initiation, planning, organizing, sequencing, executing, set shifting and maintenance, abstracting
   b) Memory deficit (may be mild): Impaired recall, relative intact recognition, less severe forgetting, benefit from cues
8. Distinct occurrence of lacunar syndromes, often missing during the course of the disease

What Neurological Disorders Did Haydn Suffer From? (tables 1, 2)

Gait Disturbance

Haydn's gait disturbance became obvious early during his disease. His last appearance as a conductor took place on December 26, 1803 in Vienna [Landon, 1976–1980]. On June 16, 1805, Pleyel noted: 'we found him very weak, his face

Table 2. Symptoms reported in Haydn's biographies

Clinical syndrome	1799	1800	1801	1802	1803	1804*	1805	1806	1807	1808	1809
1. Gait disturbance					6		1,6		1,2,4, 5,8	1,2, 4–7	1,2, 4,6
2. Unsteadiness, dizziness						3				6	6
3. Hand incoordination							4,7,8	1,2			6,7
4. a) Behavior, b) Mood, c) Lability of affect	b)3	a)3			b)3		a),c)6		b),c)6	c)1,2, 4,6	c)1
5. Urinary symptoms					none reported						
6. Hypokinesia, rigidity										6	
7. Memory, cognition							2,6	2		2,6	
8. 'Rheumatic nerve fever'		3			8		2	2			

*Stopped composing [Griesinger, 1987]. 1 = Griesinger, 1810; 2 = Dies, 1810; 3 = Griesinger, 1987; 4 = Carpani, 1812; 5 = Pohl, 1875–1927; 6 = Landon, 1976–1980; 7 = Franken, 1976; 8 = Bartha, 1965.

hasn't probably changed a lot, but he can hardly walk [Landon, 1976–1980]. Dies reports from his visit on April 15, 1805: 'After his usual nap he is getting completely dressed again, and climbs down the stairs with pain and misery'. On March 15, 1806: 'Haydn just barely got up, tripped towards the writing table, took his visiting card…'. On August 17, 1806: 'Haydn stood up, and we walked slowly back and forth in his room' [Dies, 1810]. In 1807 and 1808, Haydn hoped to get some relief from his gait disturbance, when he let himself be carried to the monastery of the Servites in a Vienna suburb, where he visited the chapel of St. Peregrinus, who was the saint of people suffering from leg diseases [Franken, 1976; Pohl, 1875–1927]. The last public appearance of the composer took place on the occasion of a performance of Haydn's oratorio *The Creation* on 1808: the Prince of Esterházy sent his carriage and his retinue and Haydn drove slowly to the university. Haydn was carried to the concert hall in an armchair (fig. 2) [Carpani, 1812; Dies, 1810; Griesinger, 1810]. During the intermission, the old man was found to become too emotional, so it was decided to carry him back home. 'Two strong men took the armchair he was sitting in and amidst the greetings, cheers and applause of the audience in the concert hall, the master of melodious music approached the steps [Carpani, 1812]. On September 8, 1808, Iffland paid a visit to Haydn: 'his house-maid said that Haydn was just coming back from the garden with his servant. However, his gait was somewhat slow, so that we had to be patient. […] He made a movement as if he wanted to get up. His servant assisted him, and he approached, his hands held over his eyes, taking short steps, whereby, although willing to go fast, he

⇑ ↑

Fig. 2. Miniature by Balthasar Wigand on the top of a small wooden box displaying the scene in the concert hall of the old university in Vienna, on the occasion of Haydn's last public appearance for a concert of his oratorio *The Creation*. In the center of the painting Haydn is seated in an armchair (↑) with friends and admirers around him. On the left side, in a dark robe, Beethoven can be seen (⇑). Courtesy Österreichische Nationalbibliothek, Vienna, Austria.

shuffled across the floor' [Landon, 1976–1980]. Finally in November 1808, Reichardt describes Haydn on the occasion of his visit: 'He was sitting very stiff and rigid, moved close to the table, both hands on the table, not dissimilar to a living waxwork' [Landon, 1976–1980].

Dyspraxia and Incoordination

As a mediator between Haydn's editors and the composer himself, Griesinger disburdened Haydn, who started to have trouble writing letters. Indeed, his handwriting became more and more illegible, so that he had to actually stop writing letters in his own hand in 1805 [Bartha, 1965; Franken, 1976]. However, the last handwritten remark in his diary, although quite shaky, is still legible, dating back to 1809, only a few months before his death. It says 'Today I sold my beautiful pianoforte for 200 fl., Jos. Haydn in the 78th year of life' [Landon, 1976–1980]. Haydn's doctor suggested that he buy a piano which was easier to play already in 1802 for his compositions, because his old pianoforte became too stressful for him [Dies, 1810; Griesinger, 1810], which seems rather a precaution regarding a presumed source of frustration, since Haydn still

was able to compose at that time. Griesinger in a letter on January 8, 1804: 'Haydn complained of the negative influence of the humid weather on his health: if he worked for only half an hour, he would become dizzy. He would have to take care of himself, if not he might suffer a stroke at the piano and so on' [Griesinger, 1987]. During a conversation with Haydn on August 17, 1806, Dies recorded: 'On my question: "How long haven't you touched your pianoforte?", he sat down, started to improvise slowly, missed the keys dilettantishly, looked at me meanwhile, corrected his mistakes and continued to fail in correcting. "Oh", he said after a minute (the performance didn't last longer than that), "you can see yourself that it doesn't work anymore" ' [Dies, 1810].

Behavioral Changes, Lability of Affect and Depression

Often, tendencies to avoid public life, loss of interest, spontaneity, personality and creativity can be detected in the original sources. Griesinger in a letter on November 11, 1800: 'Haydn is not moving to the town this winter as previously, but is staying in his house in one of the suburbs. There he can stay without being disturbed' [Griesinger, 1987]. Camille Pleyel noted on June 16, 1805: 'We found him sitting with a rosary in his hands and I believe he spends the whole day in pray' [Landon, 1976–1980]. As a farewell to the composer, Breitkopf and Härtel added the beginning of a late Haydn choral where it says: 'gone is all my strength, old and weak am I', to the printed scores of his incomplete last string quartet [Op. 103]. Again Iffland added valuable information in 1808: 'Haydn said: "I hold my prayers in the free today. I cannot do otherwise." There he folded his eyes to start crying. […] "I'm well, quite well!", he said. "But now I cannot do otherwise; whenever I am pleased about something, I feel like crying. I don't want to, but I can't help it" ' [Landon, 1976–1980].

Cognition: Dysexecutive Syndrome, Memory Deficit – Vascular Dementia

On July 12, 1799 Haydn wrote in a letter to his publisher: '…because of my impeding old age, and declining mental capacity, I can only fulfill the smallest part of my business… some days my weak memory and the deterioration of my nerves bring me down so much… that many days afterwards I am lost for ideas…' [Griesinger, 1987]. Remarkably, this is Haydn's very subjective impression, obviously written in a state of bad mood. Dies informs the reader of his biography in 1806 that '(Haydn) seems to take delight in picturing his own state of health worse than it really is' [Dies, 1810]. Objective remarks

concerning a decline in his memory from his biographers are dated much later. Haydn himself attributed his decreasing productivity to the overwhelming burden he had put on himself with the composition of his oratorio *Die Jahreszeiten* (The Seasons), which was his last big opus published in 1801. Haydn himself was not completely satisfied by his composition and in his own judgment, as to musical quality, *The Seasons* ranged far behind *The Creation*. In 1804, he retired from his position of Kapellmeister to the Prince of Esterházy. On February 28, 1804 he wrote to Esterházy: 'I heartily regret that I cannot have the pleasure of conducting my little work for the last time' [Bartha, 1965]. Meanwhile, it became impossible for Haydn to compose because it took him an enormous effort to produce ideas and write them down. Griesinger wrote to Breitkopf and Härtel on August 22, 1804: 'Haydn has completely stopped working due to his health problems, and a quartet, of which two pieces are already completed, is now his baby, which he is caring for and, with a big effort, is sometimes spending a quarter of an hour on' [Griesinger, 1987]. Dies found on April 15, 1805: 'He is aware of his weak spirit. He cannot think, not feel, not write, not listen to music' [Dies, 1810]. On August 17, 1806: 'I found Haydn surprisingly weak. He looked jaundiced and complained of headache, deafness, forgetfulness and other ailments' [Dies, 1810]. Haydn on the same day: 'Often people come to visit me, but talking, even giving only short answers leads to such a confusion that, in the end, I don't know where I am, and I'm longing to rest' [Dies, 1810]. Haydn to Griesinger on September 3, 1807: 'I never thought that a human being could sink down like this, as I feel now. My memory is gone, sometimes I still have some good ideas at the piano, but I could cry, as I am not capable any more of repeating them or writing them down' [Griesinger, 1810]. Only in 1808, from Iffland's report, one can diagnose an obvious memory problem with perseveration in his words and thoughts. Haydn's words spoken on the occasion of Iffland's visit in 1808 have been recorded as follows: '*The Seasons* exhausted me completely…. For whole days I struggled with one single idea and then, then – no, you just won't believe how much trouble I had … My *Creation* has brought in more than 2000 Taler for the poor in Berlin.' At this point he was lying back in his chair and burst into tears of joy. 'For the poor! My work has given a good day to the poor! This is marvelous, this is consolatory!' After a while he sat up again and spoke somewhat gloomily: 'It is over now! I'm not working any more. But,' gazing friendly at everybody in the room, 'it went quite well, didn't it? How much did *The Creation* bring in to the poor again? Take note of it!' [Landon, 1976–1980]. And finally Dies added on April, 1808: 'Haydn's extraordinary exhaustion doesn't allow him to think of the past. Even the present was not clear to him. His memory was completely blunted, but this was not going as far to call his weakness childish' [Dies, 1810].

Stroke Episodes?

It cannot be proven whether Haydn suffered from distinct stroke-like episodes nor can clear lacunar syndromes be diagnosed from the biographical data. However, there is a mention of 'rheumatic head fever' in 1800 (a common and unspecific diagnosis at that time), preventing him from attending a performance of his oratorio *The Creation* [Dies, 1810]. In 1801, at the age of 69, he decided to make his will. In a letter to his brother Michael on January 22, 1803, Haydn mentioned: 'I have lacked my previous good health for such a long time… due to a continuing nerve weakness, I have been totally incapable of undertaking anything for 5 months' [Bartha, 1965]. On August 20, 1803 Haydn replied to a letter: 'Härtel (Haydn's editor) is wealthy and my illness costs me a lot, since the doctor comes to see me twice daily' [Griesinger, 1987]. Indeed, Prince Esterházy, Haydn's former employer for many years, was liable for his doctors bills and costs for medication in his last years of life. It remains fairly unclear, which exact symptoms Haydn suffered during these episodes which recurred in 1805 and 1806. Maybe these episodes have been associated with a certain headache, 'continuing nerve weakness' may either be taken as a correlate of paresis or may well be Haydn's term for a depressive episode. Griesinger reports on June 18, 1803: 'Haydn was totally inactive for some months and didn't feel like doing anything (which could be taken as symptom of depression); now he's strengthened by the good season and the use of the baths (this has been a common medical treatment for sequelae of stroke) and he admitted to be able to "improvise a little again"' [Griesinger, 1987].

Haydn's Final Days

On May 10, 1809, when the French army approached the borders of Vienna, Haydn's servants came into his bedroom to get him up and ready for the day, when 4 cannon balls detonated very close to the building. According to Dies, Haydn was absolutely horrified by the first detonation, and a heavy trembling and shaking took over his body, and without the help of his servants standing by he would have collapsed without a doubt, and with the following three detonations his convulsive trembling got even worse. However, Haydn then gathered all his courage together and screamed in an unnatural and horrendous sounding voice: 'Children, don't be afraid. Where Haydn is, nothing can happen to you!' [Dies, 1810] He was taken back to bed, and his physical weakness worsened from that moment on. On May 26, he made his servants carry him to the small piano in his living room, where he was able to play his beloved *Kaiserlied* (Emperor's song) for the last time. In the evening he complained of

a headache and chills. His doctors were called, but they couldn't help any more. Haydn died in the early hours of the morning on May 31, 1809.

Which Medical Evidence Is There to Support the Diagnosis of SVE?

During his last 10 years of life, Joseph Haydn suffered from an illness that included symptoms of progressive gait disturbance leading to final inability to walk but with preserved motor skills of his arms, i.e. moderate upper limb dyspraxia, lability of affect, depression, and finally dementia, all representing cardinal features of SVE. This clinical entity develops in the consequence of severe cerebral microangiopathy which affects the small cerebral arteries and arterioles and leads to two main pathological manifestations with predominance of either one or combined mixture: (1) recurrent lacunar strokes leading to the so-called lacunar state, and (2) a more diffuse and widespread degeneration of cerebral white matter on the basis of chronic hypoperfusion and incomplete ischemia, mostly in arteriolar watershed zones close to the lateral ventricles. Following the original description of Pierre Marie [1901] many reports since have stressed the particular manifestation of frontal lobe territories and lead to the assumption of critical involvement of parallel circuits from the prefrontal cortex to the basal ganglia and corresponding thalamocortical connections [Cummings, 1994; Fisher, 1965]. Early onset is often characterized by postural imbalance and progressive disturbance of locomotion [Bäzner et al., 2000] and mood, often in the absence of distinct lacunar syndromes when white matter lesions became evident on magnetic resonance imaging (MRI; fig. 3) [Gass, 1998]. Excellent recent publications propose a set of clinical criteria which will serve as a basis for clinical and pharmacological studies [Erkinjuntti et al., 2000; Román et al., 2002] (tables 1, 2), although they have exclusively referred to the misnomer of subcortical vascular dementia, a term rather unsuitable for the complex syndrome. This is important, because frank dementia frequently becomes evident only late in the course of the disease, whereas features like mild personality changes with lack of initiative and psychomotor slowing with gait and postural disturbance are present in early stages and even predict the occurrence of non-Alzheimer dementia [Bäzner et al., 2000; Verghese et al., 2002] (fig. 4).

Haydn is an excellent example of the classical course of SVE: his gait disturbance, with small shuffling steps, the need of assistance to go from his bed to a chair, and his lack of postural control with acoustic stimuli reflects the typical gait disorder in SVE. He had to be carried to his small piano but was still able to play. Later, he stopped writing letters, but was still able to note short handwritten documents throughout the last year of his life. His inability

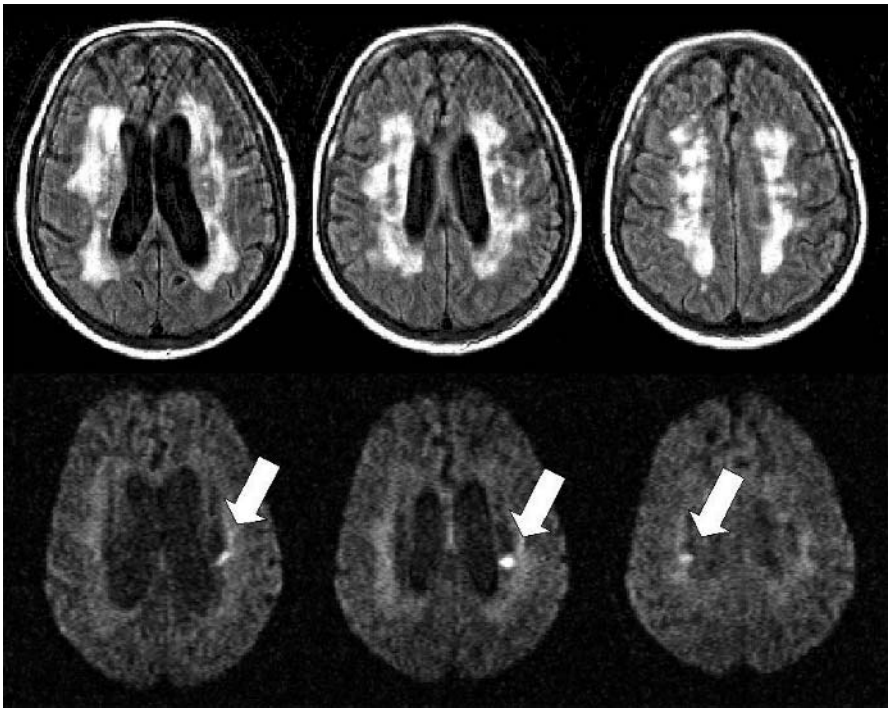

Fig. 3. Cranial MRI of a subject with severe subcortical vascular encephalopathy (SVE). Diffuse periventricular white matter lesions are demonstrated in the fluid attenuated inversion recovery (FLAIR) sequences (above). The natural history of the progressive disease is illustrated in several acute bihemispheric lesions (arrows), which are visualized through diffusion weighted imaging (DWI; below).

to improvise or to compose on the piano may be explained by a progressive dysexecutive syndrome and change in personality. In addition, a tendency to perseveration and a marked lability of affect can be demonstrated. He also reportedly suffered from short episodes of depressed mood. As to Haydn's risk factors, frequent leg swelling and shortness of breath point to congestive heart failure (CHF). CHF is a frequent consequence of long-lasting arterial hypertension and concomitant small vessel disease in the heart. Also his frequent headaches and dizziness may be interpreted as being due to high blood pressure. However, medical procedures to diagnose risk factors like hypertension hadn't reached a clinically useful level in Haydn's time, and general treatment of risk factors for vascular disease was not available. This is in contrast to the situation in the new millennium where the early symptoms of SVE should already provoke antihypertensive therapeutic consequences even in the

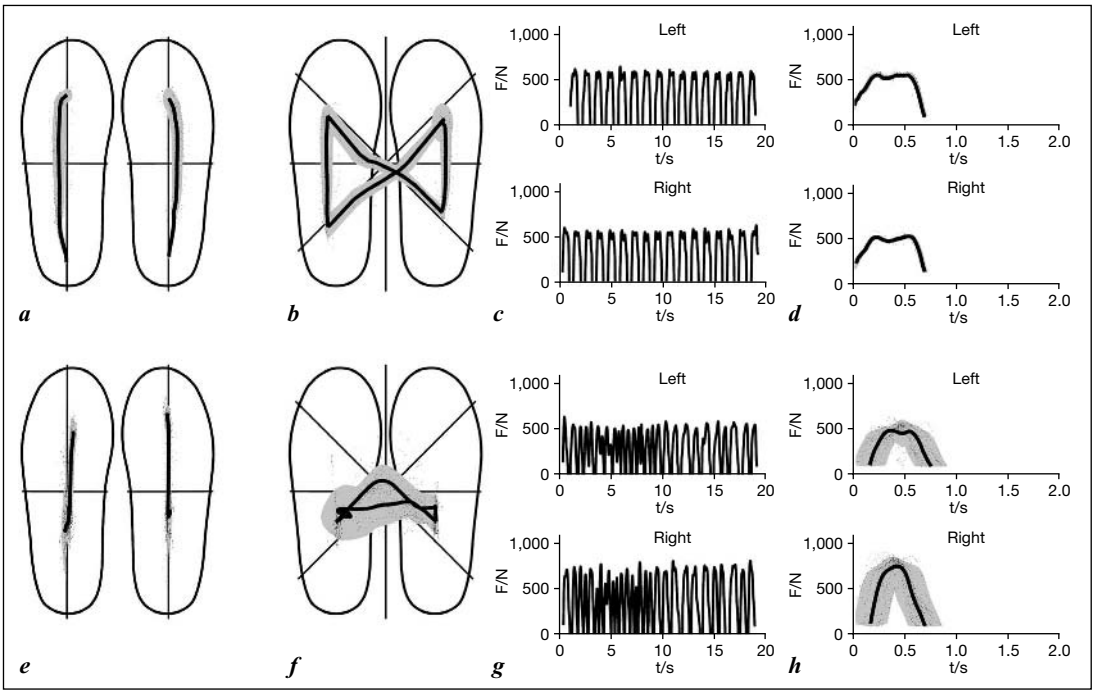

Fig. 4. Example of quantitative computerized gait analyses (ComputerDynoGraphy, Infotronic, The Netherlands) in a patient with subcortical vascular encephalopathy (SVE). Data are recorded with a 5-year interval. The first gait examination (***a–d***) shows mild gait unsteadiness with a large variability of gait lines (***a***) and cyclograms (***b***). Modulation of ground reaction forces (***c***) and superimposed ground reaction forces (***d***) is severely diminished. Five years later (***e–h***), the patient displays severe postural control failure with highly irregular gait lines (***e***) and cyclograms (***f***). In addition, marked locomotion failure with breakdown of regular gait rhythm (***g***), and a highly irregular modulation of ground reaction forces (***h***) can be noticed.

absence of hypertensive blood pressure values. Although the spectrum of therapeutics is gradually growing with the introduction of NMDA antagonists [Bäzner et al., 2000; Orgogozo et al., 2002] and cholinesterase inhibitors [Erkinjuntti et al., 2002; Kumar et al., 2000; Pratt and Perdomo, 2002], they are mainly being tested for the dementia aspect and interventional studies for other key features of the disease are still lacking. Future treatment studies might include innovative regimens such as the 'polypill' [Wald and Law, 2003] including beta-blocking agents, ACE inhibitors, and diuretics. SVE might be an excellent target for all of these drugs.

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Music and the Brain: Gershwin and Shebalin

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Abstract

Studies on the relation between music and the brain have generated heterogeneous results influenced by the location of the cerebral injury and by the degree of previous musical knowledge. Some reports have shown that musical impairment is invariably associated with aphasia (aphasia with amusia), but others have demonstrated that language impairment does not entail disruption of musical skills (aphasia without amusia or amusia without aphasia). The complex relations between music and the brain and music and language are illustrated by the cases of 2 famous musicians: Vissarion Shebalin, with a left temporo-parietal lesion, severe aphasia and an amazing conservation of his musical production, and George Gershwin, affected by a temporal right hemisphere cerebral tumor that only produced apraxia in its last days. Both musicians, despite their cerebral lesions, preserved their highly qualified musical competence. In the case of Vissarion Shebalin, his remarkable preservation of receptive and expressive aspects of music, despite a profound aphasia, demonstrates the independence of cerebral processing of language and music and the probable correlation of the sparing of these abilities, with a talent developed early in childhood and a repetitive and formal training that generates an expanded cortical representation. In the case of George Gershwin, a highly trained artist, the preservation of his left hemisphere musical representation may explain his preserved musical competence.

Music, defined as the art of transferring sounds into notes and rhythms to obtain a desired pattern or effect, is tied to language in different forms such as recitatives, songs, poetry, daily language inflections or musical notes [Epstein, 1988].

In general, musical functions require three fundamental capacities: The sense of sound (intensity, duration, timbre and height), the rhythm sense and the

capacity to transfer the musical perception to an emotional or intellectual content. To perceive music is a very complex cognitive process based on diverse forms of categorizing the stimuli. Cerebral injuries sometimes lead to surprising dissociations of these musical abilities.

Clues as to how the brain processes music come from clinical case reports of patients with altered musical function, from neuropsychological testing of either normal or brain-damaged subjects, and, more recently, from studies using functional imaging (positron emission tomography (PET) and functional magnetic resonance imaging (fMRI)) which demonstrate that different components of music, e.g. pitch, timbre, duration, loudness, rhythm, are most likely processed through separate circuits in the brain [Blood et al., 1999; Botez, 1994; Liegeois-Chauvel et al., 1998; Schuppert et al., 2000].

Amusia is an acquired impairment of musical processing. As with aphasia – a comparable impairment of language – the problem can be expressive, receptive, or both. The studies on the relation between music and language have generated heterogeneous results influenced by the type or location of the cerebral injury and by the degree of previous musical competence.

The evaluation of musical competence is very complex. Botez [1994] proposes a battery that includes 48 subtests that include the receptive component (tonal elements, melody, rhythm), the lexical element (musical note naming), the expressive component (vocal emissions, song), and instrumental tests of musical note writing and musical memory. The cases described with amusia did not always have an exhaustive evaluation of these capacities in order to classify their impairments.

The existence of differences in the degree of hemispheric dominance for music between musicians and non-musicians is considered to be clearly established today.

In people without any musical knowledge, the right hemisphere injuries lead to difficulties in sonorous recognition (contour, height, global organization), whereas injuries of the left hemisphere lead to alterations in the temporary and sequential organization of the information (rate) [Blood et al., 1999; Peretz, 1990, 2002; Platel et al,, 1997; Schuppert et al., 2000]. In musicians, studies have demonstrated that musical processing predominates in the left hemisphere. Nevertheless, dissociations and associations in verbal and musical processing are very variable: musical impairment in recognition of height, but preserved recognition of tones [Mazzuchi et al., 1982], loss of the capacity to use a musical instrument (instrument amusia or instrument apraxia) but preserved recognition of melodies, aphasia with amusia, and aphasia without amusia [Botez, 1994; Tzortzis, 2000] have all been described.

The complex relations between music and the brain and music and language are illustrated by the cases of 2 famous musicians: Vissarion Shebalin,

with a left temporo-parietal lesion, severe aphasia and an amazing conservation of his musical competence, and George Gershwin, affected by a temporal right hemisphere cerebral tumor, which only in its last days produced apraxia as evidenced when trying to play the piano.

An important approach to the study of music and the brain is the 1965 article 'Aphasia in a Composer' written by the famous neuropsychologist Alexander Luria. This investigator had the opportunity to observe Professor Vissarion Shebalin for a period of 3 years; after a left vascular injury, Vissarion Shebalin presented with profound aphasia but his musical abilities were preserved. He continued his creative work and executed numerous musical compositions, several times with great success [Luria et al., 1965].

Vissarion Yakovlevitch Shebalin was born in Omsk, Siberia, on June 11, 1902. He initiated his studies in the conservatory of Omsk in 1921 and in the following year went to the conservatory of Moscow, under the direction of Nicolai Myaskovsky, where he graduated in 1928. After his graduation, he was named Professor in the Conservatory. He obtained the Chair of the Department of Composition in 1941 and in 1942 was Director of the Conservatory. Some important Russian composers were his students: Denisov, Gubaidulina and Khachaturian. At age 26, he had already composed several quartets, romances and compositions for the pianoforte. During his school years he composed his first symphony. During the following years, he composed several symphonies and one of its operas was presented at the Bolshoi Theater. In 1948, he was accused of being too 'formalist'.

On December 14, 1953, he had a cerebrovascular accident with hypoesthesia and paresia of the right hand and the face and severe language impairment. Some weeks later, he improved and could return to his work.

During the following 6 years, he worked actively on his compositions and as Director of the Conservatory of Moscow, but on October 9, 1959, he had a second vascular accident. He presented a right hemiparesia and profound aphasia. He slowly recovered right-side activity but he continued to have spasticity and hypoesthesia. Three years later, he had an epileptic seizure and on April 30, 1963, a third vascular episode associated with cardiac infarct leading to his death on May 29, 1963.

At post-mortem, the brain displayed hemorrhagic edema of the left hemisphere in the temporary and parietal regions with a left temporoparietal hemorrhagic cyst.

After the second episode, the patient could no longer understand language nor could he speak. After some weeks, he still could not articulate words successfully. He could not repeat phonemes, had abundant paraphasias in his spontaneous language and attempts at formulating phrases were unfruitful. Neuropsychological tests and therapies were implemented for the next 2 years.

A noticeable deterioration of the phonemic organization and alienation of the meaning of words were observed. Naming was imperfect and his clinical profile was a sensory aphasia with some kinesthetic deficiencies in the motor organization of language.

Later, he could emit separated sounds but he was incapable of discriminating phonemes, could not repeat phrases and produced paraphasias. Reading was preserved and to a certain extent, writing. In the third year, there was no great progress in his condition, but he continued with his work as a composer. He worked with his students listening to his compositions and analyzing and correcting them. He finished compositions that he had begun before falling ill and he created other new compositions, which musicians have considered to be of the same quality as those before the neurological injury.

During his disease, he produced: *Sonata for violoncello and pianoforte*, three *Moldavian choirs*, *Quartet No. 8*, *My Father* (8 songs, prize winner), the *Earth of Moldovia* (three songs), the *5th symphony*, *Quartet No. 9*, *For My Grandsons* (4 choirs), *In the Middle of the Forest* (7 choirs), and *Sonatina op. 60*. He also reviewed several suites and concerts. Some of his works were interpreted in his presence on October 9, 1962. Shostakovitch said: The 5th symphony of Shebalin is a shining work of elevated emotions, optimism and vitality. This symphony, composed during his disease, is the creation of a great teacher. Kherennikov affirmed: We can only envy the shining creative activity of this man who, in spite of his disease, created the 5th symphony, full of youthful feelings and wonderful melodies.

The case of Vissarion Shebalin, of his remarkable preservation of receptive and expressive aspects of music, despite a profound aphasia, demonstrates the independence of cerebral processing of language and music and the probable correlation of the sparing of these abilities, with a talent developed early in childhood and a repetitive and formal training that generates an expanded cortical representation, as has been proposed by other authors [Tzortzis et al., 2000].

Another exceptional musician was George Gershwin, who in his short life produced very important musical pieces for the North-American musical art. His grandfather, of Russian origin, was a famous gunsmith and inventor, which somehow explains Gershwin's inherited flair for geniality. His father decided to migrate to the United States, in love with Rose Bruskin who he later married, and settled in New York, where he changed his name, Gershovitz, to Gershwin. Of that union were born Israel (Ira) and Jacob, who later changed his name to George Gershwin. Ira worked with George in the creation of many songs and was close to him throughout his artistic career.

While his father dedicated himself to the hotel profession, George, with great talent and influenced by Maxie Rosenzweig, who was himself a violin virtuoso from his 8th year on, dedicated his life to music. He learned to play the

piano following his brother Ira and, under the direction of Professor Ambitzer, he refined his technique and began to attend concerts. He made his debut on March 21, 1914 playing a tango that he had composed. At age 15, he left school to work as a pianist.

In 1924, he writes *Rhapsody in Blue* in a few weeks and this work immediately becomes a success. In 1927, he writes *An American in Paris* and his first presentation is on December 13th at the Carnegie Hall, interpreted by the Philharmonic Society Orchestra and conducted by Walter Damrosch. In 1931, he writes his second rhapsody; in 1932, his first musical film *Crazy Girl* and after a brief vacation in Cuba creates the *Cuban Overture*, an orchestral rumba. Acclaimed by the public, his production includes many styles that range from orchestral music, opera (*Porgy and Bess*) and Jazz. Some of his songs have been immortalized by A. Johnson. He was also a wonderful dancer, to the point of teaching some steps to the great Fred Astaire. Apparently persecuted by his fame and his impossible love for Paulette Goddard, Charles Chaplin's wife, he suffered a severe depression which lasted several months.

Hypochondria, spastic colitis and anxiety were some of the problems that deviated his attention. On February 11, 1937, the philharmonic orchestra of Los Angeles, directed by Alexander Smallens, interpreted the *Concerto for Piano in F Major* and, while playing the piano, Gershwin had an absence which lasted 20 seconds, in which he omitted several compasses; however, although it went unnoticed to the public, the composer mentioned the episode of a 'blackout' to Dr. Zilboorg, a psychiatrist, and specified that the episodes were preceded by an uncinated crisis which is characterized by the unpleasant smell of 'burned rubber'. Dr. Zilboorg suspected an organic origin and asked for a medical evaluation. A psychosomatic problem with 'defense mechanisms' was diagnosed. Shortly after, while in a barber's shop, he presented a new episode of loss of consciousness that lasted a few seconds.

At the beginning of 1937, he began to complain of migraine, which was attributed to his working too hard. Dr. Ernest Simmel and Dr. Gabriel Segall also studied the patient, but only psychosomatic diagnoses were made. He continued having uncinated crises and they appeared in diverse circumstances, both while playing the piano or while playing tennis. Dr. Segall recommended a lumbar puncture, but it was not accepted by Gershwin who hoped that his symptoms would soon disappear.

In 1937, he was hospitalized in the 'Cedars of Lebanon' in Los Angeles to investigate the worsening of his migraines, and the appearance of nausea, dizziness and olfactory hallucinations. His relatives noted important behavioral changes, such as using a powdered chocolate bar as an ointment on his body or the occasion when he pretended to open the door of his car and tried to throw out the driver. He complained of motor limitations of his right hand and of

motor incoordination. Radiology of the skull, EKG and a Wasserman test were made with negative results. A diagnosis of 'hysteria' was proposed [Teive et al., 2002].

Progressively, he complained of hyposmia, persistent migraine, restlessness and new complex partial seizures. On July 8, 1937, when he tried to interpret a piano piece for his doctor, it was impossible for him to work the keyboard. On the following day, he was found unconscious and was hospitalized again.

He was examined by Dr. Carl Rand, who wrote that the patient did not react to painful stimuli, had small and unequal pupils, and no voluntary movements. He presented with slight right hemiparesis, papilledema with retinal hemorrhages, and normal blood pressure without a stiff neck. His condition deteriorated and, on July 10, a tentative diagnosis of brain tumor was made. Dr. Harvey Cushing, a famous neurosurgeon was called, and he recommended a consultation with Dr. Walter Dandy, but he was on a cruise in Chesapeake Bay. Finally, at 9 p.m. of the 10th of July, Dr. Rand and Dr. Howard Naffziger trepanated him in order to make a ventriculography at which they found a cerebral hernia caused by a right hemispheric temporal tumor. The nodule was removed, the wall of the cyst was cauterized and the dura mater was closed. The procedure lasted 5 hours, but the clinical situation worsened and the patient died 5 hours after the operation.

The lesion was described as spongioblastoma multiforme, which is known today as multiform glioblastoma. Based on the clinical and pathological re-evaluation, Dr. Gregory Sloop of the University of Louisiana now proposes the diagnosis of pilocystic astrocytoma, with a history of 3 years of evolution, solid and cystic injury with intracranial hypertension and delayed diagnosis in a severely deteriorated patient.

The uncinated and hypogastric crises in an adult, the absences that later confirm the clinical signs of complex partial seizures and the progressive intracranial hypertension and neurological deterioration were clinical signs of a temporal mesial lesion and late uncal herniation.

The radiological studies showed a large-size right temporal tumor and, in spite of the operation, the patient could not survive the malignant glioma.

During the evolution of his disease, his musical productivity was great. It was at the pinnacle of his career when he began to suffer progressive migraine, apathy, indifference and affective symptoms that were erroneously interpreted as a 'neurotic protest to the artificial world that surrounded him'. Only 4 days before his death did he have difficulty coordinating the movements he needed to play the piano [Laws, 1997; Ljunggren, 1982, 1991; Schoemberg, 1980; Sloop, 2001].

In the case of George Gershwin, a highly trained artist, the preservation of his left hemisphere musical representation may explain his preserved musical competence.

The musical genius observed in these 2 patients with structural and functional brain alterations raise a great challenge to the understanding of the nervous system and its role in music and artistic creativity. To listen to the *Rhapsody in Blue* of Gershwin or the *5th Symphony* of Shebalin, generate different aesthetic sensations. The motivation to create each one of the compositions, the musical style, the complexity of each one, its intrinsic beauty and the different sensations that they generate are, without doubt, deep reasons that activate or modify neural circuits, and put into operation the wonderful machinery of the human brain.

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Robert Schumann's Focal Dystonia

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Abstract

Robert Schumann is one of the most prominent composers of the early romantic period. He was born in Zwickau, Saxony, in 1810. Early in his adolescence, he displayed extraordinary skills in piano playing and attempted to become a concert pianist. After an initial success, increasing technical difficulties hampered pianistic progress in the years 1831 and 1832. Finally, he developed a task-specific loss of voluntary control of the middle finger in his right hand. By means of a finger-stretching device, Schumann tried to improve the situation. In parallel, he composed the Toccata, Op. 7, a piano work which allowed high level virtuoso performance without the use of the middle finger of the right hand. However, from 1833, he almost completely gave up playing piano literature, but continued to improvise. Robert Schumann was suffering from a focal, task specific dystonia of the right hand, also referred to as pianist's cramp. This disorder is characterized by a painless loss of skilled motor control in a task specific context. The neurobiological origin is seen in maladaptive plasticity of neuronal networks with blurring of afferent and efferent receptive fields of adjacent finger representations in the cerebral cortex and the basal ganglia. The general basis of such a blurring may consist in a deficient lateral inhibition of synaptic pathways. Risk factors for developing musician's dystonia are male gender, extensive cumulative practice time, extreme motor workload concerning the temporal and spatial quality of the affected movements and personality traits such as proneness to anxiety and perfectionism. All these factors can be demonstrated in Robert Schumann's early life.

Introduction and Biographical Sketch

Robert Schumann is known to us as one of the most creative and fruitful composers of the romantic period in the first half of the 19th century. However, in his younger years, Robert Schumann had the ambition to become a concert pianist excelling the most prominent virtuosos of his time, 'playing warmer

than Moscheles and greater than Hummel', as announced by his piano teacher Friedrich Wieck in a letter to Robert Schumann's mother. His piano playing career was short. Which circumstances led to the termination of his career as a pianist? This biographic study concentrates on the years 1829–1833 and on the development of his neurological disorder, a loss of fine motor control in his right hand that took place during this time. It is based on a thorough review of his medical history using predominantly contemporary sources, especially Robert Schumann's diaries and letters. The psychological crises of that time and his later developing psychiatric disorder will not be included into this case report. They have been reviewed in the past by several biographers [Ostwald, 1985; Lederman, 1999; Steinberg, 1999].

Robert Schumann was born on June 6, 1810 in Zwickau as the fifth child of the bookseller, author, and publisher, Friedrich Schumann. At the age of 7, Robert received his first piano lessons and father Schumann purchased a highly valuable grand piano and allowed his son to play for him daily after lunch. After only a few years, his first piano teacher, Baccalaureate Kuntsch declared himself incapable of giving Robert further lessons, so that Schumann, with regard to his pianistic education, was left to his own means as of 1825. In 1826, his father died and Schumann agreed to his mother's strong recommendation to pursue a career as a law student in Leipzig, despite his preference for piano playing. The great pianistic skill and excellent sight-reading alongside considerable technical shortcomings were noticed by the prominent piano teacher Friedrich Wieck, who, beginning on August 1, 1828, gave lessons to the fledgling law student.

In 1829, Schumann transferred to the University of Heidelberg to continue his studies of jurisprudence with the highly regarded expert Anton Justus Thibaut, who also happened to be an enthusiastic amateur musician. Schumann used the many occasions of performing in private salons and increased his piano practice up to 7 hours daily, neglecting his law courses. Undeniably, the progress desired at the time was not always attainable: '*2 hours finger exercises – Toccata 10 times – finger exercises 6 times – variations 20 times myself – and it still didn't work with the Alexander Variations in the evening – frustration over this – extreme frustration (January 4, 1830)*' [The texts written in italics are diary entrees. They are quoted according to Robert Schumann, Tagebücher, in Eismann, R, Stroemfeld/RoterStern Verlag 1971, Band 1–5]. However, three weeks later, Schumann did play the same Alexander Variations, – an extremely difficult virtuoso piece of Ignaz Moscheles, – with considerable effect. This was his first and only public appearance on the piano. In a famous letter to his mother, dated July 1830 he finally stated his intention to pursue a pianist's career and urged his mother to write to Friedrich Wieck, asking him to accept Schumann as a piano student.

The Return to Leipzig

He returned to Leipzig for a second time in order to continue his studies and from the very beginning, he encountered many more problems. Robert was under enormous pressure to succeed. In order to pacify his mother, he had agreed to a six month trial period, after which Wieck would decide whether or not Robert was suited to a career as a pianist. Financial worries forced him to write ever more desperate letters of request to his home. He finally moved into a small apartment in the house of his piano teacher Friedrich Wieck. The spatial closeness to this personality likely caused a change in the perception of his teacher. Schumann discovered, much to his disappointment, that Wieck was less interested in his training than that of his daughter Clara. It remains unclear, whether or not Wieck, as early as the end of 1830, had noticed the physical limitations and especially the diminution of Robert Schumann's fine motor skills, and due to this, changed his prognosis.

In the diary entries, beginning in the spring of 1831, there are a noticeably frequent number of entries concerning playing technique, hand position and relaxation. It is a mix of frustration mingled with the occasional, ever diminishing experiences of success at the piano. The month of May 1831 seems to be a key month in this regard, in which the technical playing difficulties are revealed:

May 12th: *Much piano played, Field's Rondeau, Moscheles' third etude, my middle movement – very relaxed in the etudes.*

May 13th: *Got up early – My soberness is rewarded; played very well – soft, pearl-like tune and fantasy.*

May 14th: *Piano playing yesterday was quite satisfactory and made progress. Should Wieck be right about my studies?*

May 25th: *– Piano bad – the Moscheles Etude timid and unsure – Where does that come from? Been playing on it for fourteen days, attentively and persistently studied.*

July 5th: *The Chopin is going excellently; today is the fifth day on which I have studied four hours each day. If only there will be no relapses! Protect me, my genius and never disappoint me!*

July 9th: *My dear Robert, don't lose your courage if it is not flowing and going so well, like in the last eight days; practice patience, lift your fingers quietly, hold your hand still and play slowly: and everything must come back together.*

July 13th: *The piano didn't want to work yesterday; it was as if someone was holding me by the arm.*

July 21st: *It has been sincerely miserable on the piano the past several days; I cried yesterday with rage.*

August 14th: *Now I want to proceed in such a manner with my quiet art: since I know where it is, it must also be reachable; if only I had no fingers and could play with my heart for others!*

Schumann worked on a change in his hand position, which temporarily brings an improvement in his attacks:

October 13th: *It is going quite well with the piano, excellently in the past few days. The flexibility is shocking and the tunes are flowing and progressing as in old times…. I am holding my wrist a little higher, approximately like the Belleville (famous female piano virtuoso), even though the graceful, wavelike line is missing*.

While Clara Wieck and her father were on a concert tour in Paris from September 1831 until April 1832, Robert developed an apparatus to improve the strength of his middle finger, and questionably that of his index finger as well. The doctor and friend Dr. M. Reuter remembered 10 years later in a letter from 1841 [Rothe, 2002]:

He mentioned first in his youth that the pointer and middle fingers had noticeably less strength and flexibility than the other fingers. The long term usage of a machine, with which the aforementioned fingers were pulled closer to the back of the hand, led to a state similar to laming, to the degree that they first, only had a weak sense of feeling and secondly, with regards to movement, could no longer be controlled by will.

Schumann christened this device 'Cigar mechanics'. Later he revealed to friends that he had constructed a sling that was attached to the piano, which pulled the middle finger upwards and held it there. The other fingers remained moveable at the keyboard.

May 7, 1832: *It is going pretty good with the third finger through the* '*cigar mechanic*'.

Now and then, Robert became hopeful:

May 11, 1832: *Yesterday I composed and played; the new method is the only one that works; it is the one.*

But also this attempt at self-therapy does not help for long:

22. 5. 1832: *– the third finger seems really irreparable.*

June 14, 1832: *The third finger is completely stiff.*

After this entry, there are no more clues to be found about Robert's hand ailment in his diaries. The subject seems to be closed. Apparently, Robert had finally given up the plan to become one of the greatest piano virtuosos of the time. This relieving decision was certainly made easier through the first successes of his compositional work – he had switched from one life's goal to another. He formulates this in a letter to his mother, just two years after giving up his piano studies [Schumann, 1898, p. 176]:

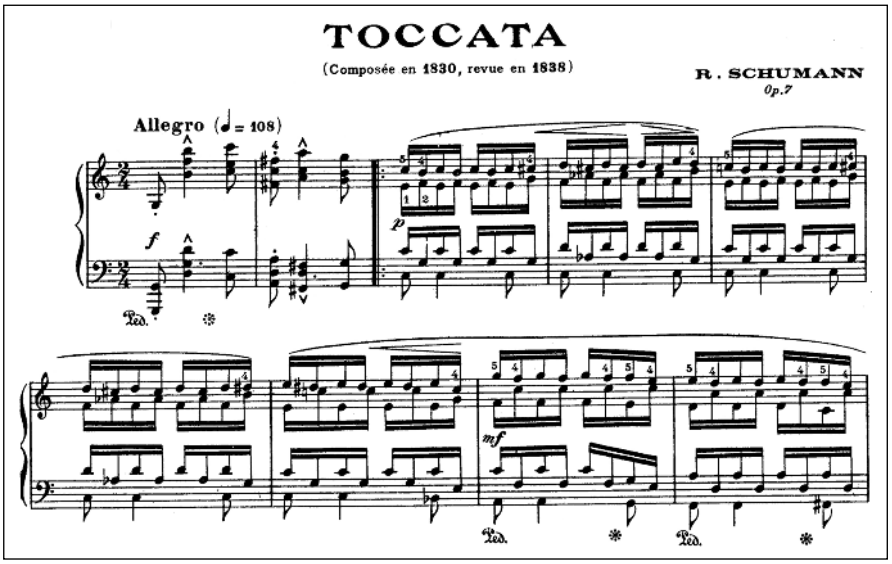

Fig. 1. Beginning of the Toccata Op. 7. This extremely difficult piece can be played without the middle finger of the right hand. Verlag Eduard Hallberger, Stuttgart, 1890; in: Hallbergers Prachtausgaben der Romantiker.

... Do not worry about the finger! I can still compose, and I would hardly be any happier as a travelling virtuoso – for that, I was spoiled at home. It doesn't bother me when I improvise.

Even my old courage to improvise in front of people came back.

A special work for piano accompanies the difficult years from 1829–1833 – and was quite possibly inspired by the movement disorder. This is the 'study in double notes', the later Toccata Op. 7. In the figuration of the right hand, the middle finger can be largely left out (fig. 1). The third passages starting in measure 32 are somewhat uncomfortable, but by alternating the ring and index fingers, the upper voice is quite playable. It is not a far reach to say that this is an attempt to find a creative solution to the movement disorder through avoiding the use of the middle finger. Schumann drafted the piece as an etude and was proud of the horrendous difficulties, which the Toccata offered. He wrote to his mother on July 2, 1834 [Schumann, 1898, p. 240]: *Take the attached piece (the Toccata) as proof of my continuing efforts. Anybody in Zwickau will hardly ever make it*.

The Toccata Op. 7 is probably the most original of Schumann's early compositions. It is closest to the romantic, demonizing style of Liszt and Paganini. The piece had an enormous impact on the contemporaries. In a review of one of Clara Wieck's piano evening on November 9, 1834 [Boetticher, 1984] it was

said: '*The last piece made a wonderful impression, a Toccata from Schumann – the work is a mold of originality and novelty, and in spite of its strict style, it worked with a deeply gripping magic on all the listeners.*'

Medical Treatment

Even though Schumann had inwardly distanced himself from virtuosity, he still tried, from the spring of 1832 to the summer of 1833, to improve his situation through medical measures. The treatment included rest, diet, electricity, bathing the hand in animal blood, and homeopathy. All new and expensive treatments of the day that were available were – unsuccessfully – utilized [Neumayr, 1989]. Little noticed is, however, that Schumann did not completely become silent at the piano. Improvisation still belonged to his daily musical activities. Even in the spring and summer of 1832, in the time of the critical worsening of his movement disorder (June 6, 1832: '*the finger is completely stiff*'), Robert tells about incredible experiences at the piano:

May 29, 1832: *....as I came home, close to nine o'clock, I sat down at the piano and it seemed to me that utter flowers and gods were coming out of my fingers, such was the stream of thoughts.*

July 4, 1832:*...How long and overflowing did I fantasize yesterday.*

It is surely the freedom of the choice of musical means, which made it possible for him to compensate for his movement problems while improvising, similar to the case of the Toccata. Furthermore it can be assumed that the degree of the fine motor skill disturbance varied. Schumann still played piano literature, mostly to get to know particular works. He preferred to perform his own, in part very demanding pieces in small private contexts. Hironymus Truhn describes Schumann's piano playing in the year 1837 as quite impressive [Jansen, 1883]:

'*He moved his fingers with an almost frightening speed, as if ants were crawling around on the piano; he played his own things – I honestly never heard anything else from him – with only very little accentuation, but with lots of usage of both pedals. In the last instance, one naturally must not find a lacking of taste; he only played with as much pedal as needed to help a not so grand piano*'.

Diagnosis

There has been much speculation about the causes of Robert Schumann's hand problems. Even the finger affected was under discussion. In a letter to Agnes Carus in September of 1830, Schumann reveals pain in the ring finger of

the right hand in the winter of 1829/1830 [Schumann, 1898]. In the retrospective report of Robert's friend Dr. Moritz Reuter, the index and middle fingers are discussed [Rothe, 2002]. A loss of control in the middle finger of the right hand is described in the diaries beginning in May of 1832. On the whole, these precise and repeated diary entries from the critical time period around 1832 speak clearly for an isolated and painless loss of control in the middle finger of the right hand. It is noticeable that the extent of the pain symptoms was overestimated by many biographers. Indications of pain in the hand or arm existed only after an arm injury in December 1828. These were sufficiently explained by a fall after too much wine and did not really affect Schumann after the fact [Altenmüller, 2004]. Several diagnoses are discussed in the literature. Edler [1982/2002] diagnosed a 'complicated, seeping tendinitis followed by stiffening', Fahrer [1992] an 'augmentation of the extensor tendon of the middle finger through the cigar mechanics', Franken [1997] the 'result of an arsenic treatment in 1831'. None of these three diagnoses can be applied. A complicated tendinitis would have caused a great amount of pain if it had occurred at close to the same time as the movement disorder. Such pain was not mentioned in the diaries or letters. An augmentation of the extensor tendon due to the 'cigar mechanics' can be eliminated because the causality was reversed: the stretching mechanism was supposed to better the movement disorder. The completely uncharacteristic symptoms speak against the diagnosis as a result of an arsenic treatment for syphilis. Arsenic poisoning leads to pain, stomach and intestinal cramps, and numbness and paralysis of the feet and hands. A paralysis would also have affected the hand outside of piano playing. Neither an arsenic treatment nor any symptoms of poisoning were mentioned in the diaries or letters in May of 1831, the assumed time frame of infection and potential therapy. After sexual contact with his lover Christel (Charitas), Schumann reports in his diary on May 12, 1831 about a wound on his penis, which could be considered a syphilis infection. It is, however, atypical that the wound hurt, and that treatment with quicksilver or arsenic was not recommended, but instead baths with narcissus water, which was supposed to reduce inflammation. Schumann especially, who tended to be a hypochondriac, would most likely have mentioned a dangerous arsenic therapy and the resulting side effects in his diary.

Robert Schumann suffered from a task-specific focal dystonia, a musician's cramp. It is the only diagnosis, which can sufficiently explain all of the symptoms and the progress of the sickness. Already Merriman et al. [1986] suspected a focal dystonia as the underlying pathology; however, without providing supporting empirical evidence in detail. Merriman's viewpoint was adopted by Lederman [1999]. The entries from his diaries, the attempts to compensate the disorder by composing piano music extremely well suited for the disorder in question and the circumstances under which the illness broke out, do not leave

Fig. 2. Presumed hand position of Robert Schumann's dystonic right hand when playing the first bars of the Toccata. Note the middle finger in hyperflexion.

any doubt concerning this diagnosis. Beyond that, according to new epidemiological studies, Robert Schumann had the typical profile of an 'at-risk patient' [Altenmüller, 2003]. In summary, Schumann's medical history in the years 1829–1832 is the first convincing, documented case of this illness.

The musician's cramp is a neurological disorder characterized by the loss of fine motor control of long practiced skilled movements during instrumental playing. It is related to writer's cramp, but seems to be more frequent in the population at risk. On average, 1 of every 100 musicians in Germany will develop musicians' cramp. Men are affected approximately six times more frequently than women. The movement disorder is usually task specific limited to instrumental playing and does not extend over to other movements. There are no indications that Schumann's writing skills were affected, even though his editorial and compositional work required many hours of writing each day over a long period of time. There was no pain or deformation associated with the loss of control. Probably beginning in May 1831, Schumann's middle finger involuntarily drew itself in. In a possibly unconscious attempt, Robert Schumann composed the final version of the Toccata, Op. 7. Figure 2 shows just such a hand position with a curling middle finger during the playing of the first measures of the Toccata.

Although the neurobiological origins of this disorder are not yet completely clarified, it is probable that musicians' dystonia is in most cases due to

dysfunctional (or maladaptive) brain plasticity. Support for this theory comes from a MEG study performed in musicians with focal dystonia. Compared to healthy musicians, the dystonics showed a fusion of the digital representations in the somatosensory cortex, reflected in the decreased distance between the representation of the index finger and the little finger when compared to healthy control musicians [for a review, see Münte et al., 2002]. Such a blurring of receptive fields of the digits may well result in a loss of control, since skilled motor actions are necessarily bound to intact somatosensory feedback input. The pathological mechanism of the blurring may be based on an impaired lateral inhibition of adjacent neuronal networks processing afferent information from single fingers. With a corresponding genetic predisposition, this disintegration can be accelerated by excessive and intensive practicing. The emotions play an important role, since the fixation of incorrect motor programs happens especially intensely under the influence of anxiety and stress hormones. Musicians with focal dystonia very often suffer from anxiety disorders and from perfectionistic tendencies [Jabusch et al., 2004]. These personality traits are present long before the beginning of the sickness and can also be recognized in Robert Schumann. According to newer epidemiological studies, the high-risk group is considered to be young to middle-aged men who play classical music, whereby guitarists and pianists are affected most often. Within the instrumental groups, those who spent the longest time practicing their instrument on average during their studies were most at risk. Practice time, professional position, and personality traits stand in close mutual connection [Jabusch et al., 2005].

The therapy for focal dystonia remains problematic to this day. In Robert Schumann's case, the result of the treatment with the 'cigar mechanics' and the other therapy attempts is clear – a career as a pianist remained unattainable for him. On the whole though, his sickness must be seen as a mild and non-progressive form since he was still quite able to play piano. The improvisation at the piano must have been a return to the roots of music for him, connected to an inner freedom from fear, doubt, and from the extreme pressure of pianistic perfection. The energetic diversion of his creative potential to composition shows the tremendous will for creation, which had already set him apart as an adolescent. For us, Schumann's decision to follow a career as a composer was a blessing, because it allowed his creative talent be developed to masterful perfection.

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