Title
Without Thinking: An Ethnography of the Diagnosis of Dementia in an American Clinic and in a French Clinic

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Without Thinking:
An Ethnography of the Diagnosis of Dementia in an American Clinic
and in a French Clinic.

by
Laurence Anne Tessier

A dissertation submitted in partial satisfaction of the
requirements for the degree of
Doctor of Philosophy
in
Anthropology
in the
Graduate Division
of the
University of California, Berkeley

Committee in charge:
Professor Xin Liu, Chair
Professor Charles Hirschkind
Professor Barry Stroud

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Abstract

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This dissertation describes the building of knowledge about dementia in two renowned clinics in the world of neuroscience: one situated in America, the other in France. How do these teams of neuroscientists distinguish the demented person from the reasonable person?

Drawing on 18 months of ethnographic research, I analyze how the understanding of frontotemporal dementia (FTD), a neurodegenerative disease that is defined in opposition to Alzheimer’s disease (AD), allows neuroscience to explore, from the destruction of our brain, our emotional and social being. Well equipped with contemporary and American cerebral theories on “empathy” and “sociality”, I then examine how teams of neuroscientists diagnose the pathology of the social and emotional being in an individual patient. How do they reach a diagnosis in a context at times marked by uncertainty? I lay out these uncertainties that point to the weakness of an exclusive positive definition of knowledge and I study how medical expertise can be conceived as connoisseurship, bringing to light the “feelings” and “tastes” of these doctors for the disease as a decisive tool for the making of a diagnosis. I conclude on the differences I observed in the understanding of FTD in the French clinic versus in the American clinic.
A ma grand-mère, Geneviève Bernad (1921-2007)

A mon père, Paul Tessier (1917-2008)
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Alzheimer’s disease or Alzheimer disease (AD) is the most common form of dementia. It was first described in 1906 by the German psychiatrist and neuropathologist Aloïs Alzheimer. The most common early symptom is short-term memory loss: difficulty in remembering recent events. It then worsens with symptoms such as confusion, language troubles, aggression and long-term memory loss. In the end, bodily functions are lost, ultimately leading to death. There is no cure for Alzheimer disease. The diagnosis is usually made through symptoms history, brain scans, neuropsychological test and sometimes analysis of biomarkers. Yet, examination of brain tissues through autopsy is required for a conclusive diagnosis. At the autopsy, plaques composed of amyloid-beta and tangles composed of tau protein are observed in the brain tissues through the microscope. Alzheimer disease is a neurodegenerative disease predicted to affect one in 85 individuals globally by 2050.

Amyloid-beta is a protein involved in the neuropathology of Alzheimer disease: it is the main component of the amyloid plaques found in the brains of Alzheimer patients. Amyloid-beta can be detected today during the life of the patient through analysis of the level of this protein in the spinal fluid of the patient, or through cerebral imaging PiB Scan.

Amyotrophic lateral sclerosis (ALS) or Lou Gheric disease, or Maladie de Charcot is a progressive neurodegenerative disease that affects motor neurons, which reach from the brain to the spinal chord and from the spinal chord to the muscles throughout the body. The progressive degeneration of motor neurons leads to their death, the ability for the brain to initiate and control muscles movements is lost, patient in the later stages become fully paralyzed; this eventually leads to death. There is no cure for ALS.

Aricept is the trade name for the molecule Donepezil, an acetylcholinesterase inhibitor. Its main therapeutic use is in the palliative treatment of Alzheimer disease. No definitive proof shows that its use alters the progression of Alzheimer disease. In 2012, a Cochrane review yet showed that acetylcholinesterase inhibitors were “efficacious for mild to moderate Alzheimer’s disease”.

(an) Attending neurologist supervises the activities of the team, he or she supervises the making of the diagnosis.
(a) **Biomarker** refers to the biological indicator of some biological state or condition. Two biomarkers are used for the diagnosis of dementia: the amyloid-beta protein and the protein tau, they are found in the spinal fluid of the patient, a sample of which is taken through a lumbar puncture. A lower level than the norm for the protein beta-amyloid and combined with a higher level than the norm for the protein tau, signify that the person is likely to have Alzheimer disease.

**Dementia** is a broad category of brain diseases that causes long term loss of the ability to think and reason clearly and that is severe enough to affect a person’s daily functioning. From Latin, *demens*, prefix *de*: privation (removal, separation) and *mens*: mind.

**Frontotemporal dementia (FTD)** is a neurodegenerative disease characterized by a predominant neuronal loss in the frontal and the temporal part of the brain. It was first described by the Czech neurologist and psychiatrist, Arnold Pick; it was first called Pick’s disease. Today FTD is subdivided in three “variants”: the behavioral variant, the semantic variant and the non-fluent variant. The first variant, bv-FTD, is characterized by changes in social behavior and conduct, with loss of social awareness and poor impulse control. The semantic variant is characterized by a loss of words understanding although speech remains fluent. The non-fluent variant is characterized by progressive difficulties in speech production, although the understanding of words, at least at the beginning of the disease, is fully preserved. FTD is the second more common form of dementia after Alzheimer’s. FTD is a lethal neurodegenerative disease without cure.

**Lewy Body dementia (LBD)** is a type of dementia closely associated with Parkinson’s disease. It is characterized by the presence of Lewy Bodies, clumps of alpha synuclein and ubiquitin protein in neurons, detectable at the autopsy of the brain. Principal symptoms are fluctuating cognition with great variation in attention and alertness, and recurrent visual hallucinations. Parkinson’s symptoms are sometimes associated. LBD is lethal and there is no cure for LBD.

**Magnetic Resonance Imaging (MRI)** is a medical imaging technique used in radiology to investigate the anatomy and physiology of the body (including the brain). Functional MRI (fMRI) measures brain activity by detecting associated changes in blood flow.

(a) **Neurodegenerative disease** is the umbrella term for the progressive loss of structure and function of neurons. The different modalities that this process of demolition of the brain can take are described through the clinical and neuropathological presentations of Alzheimer’s disease, Frontotemporal dementia, Huntington’s disease, Lewy Body dementia, Parkinson’s disease, Amyotrophic lateral sclerosis, Cortico basal degeneration, Progressive supranuclear palsy...
(a) **Neuropsychologist** aims at understanding how brains structures relate to behavior and thinking. Neuropsychologists usually hold a Ph.D in Psychology and they test cognitive and emotional capacities of the patients with neuropsychological tests.

(a) **Neurology Fellow** is in a period of medical training in neurology after having completed his or her residency.

**Parkinson’s disease** is a neurodegenerative disorder induced by a degeneration (unknown cause) of neuronal cells producing a neurotransmitter dopamine. This degeneration causes troubles such as shaking, rigidity, slowness of movement and difficulty with walking and gait, as well as depression and cognitive troubles. The treatment is based on a dopamine precursor, the Levodopa, or on dopamine agonists.

**Tau** is a protein, it stabilizes microtubules and is abundant in neurons. Hyperphosphorylation of tau proteins can result in the self-assembly of tangles of paired helical filaments and straight filaments which are involved (but it is unclear how) in the pathogenesis of Alzheimer disease and frontotemporal dementia. These dementias (to which needs to be added Cortico basal degeneration and Progressive supranuclear palsy) in which tau proteins accumulate are called tauopathies.
INTRODUCTION

It is early morning on the 15th floor of a grand hotel in the city. On this sunny morning, which makes the whole city shine, we are not quite at a conference, we're at a consortium: scientists came from all over the world to convince some American philanthropists to finance their research on a protein called “tau”. Tau is involved in the neuropathology of dementia, including Alzheimer’s disease and frontotemporal dementia.1 I met one of these scientists, a neurologist, the evening before at a diner. Now, while drinking a coffee and eating a croissant, the neurologist asks me what I have been doing at the Memory clinic these last years. I tell him, again, as I had said the previous evening, that I have been studying the work of the medical team, the ways they make a diagnosis of dementia. Dr. Martin who works at the Memory clinic, passes by; he stops and shakes hands with the neurologist.

The neurologist (to Dr. Martin). – I finally figured what she was doing: she is watching us and then she is going to write about us...(Big laugh.) And now I realize we have to be nice to her because she...

Dr Martin (smiling). – You know, no matter how nice we are to her... we are going to come out looking foolish...

Me (to Dr. Martin). – Why do you say this?

Dr. Martin (wooly). – No ... What we do...

Dr. Martin is interrupted by a strident hiss. Dr. Daniel, the chief of the Memory clinic, is whistling with his fingers in his mouth. People stop talking. Breakfast is over. Everybody makes its way to its seats. Dr. Martin goes to his chair around the giant table and begins to introduce the consortium on tau.

We can never be certain of how stories start. This one, we might think, began with my arrival at the Memory clinic, or perhaps started the week just before when a sociologist who was already doing an inquiry there, offered to introduce me to the chief of the clinic, Dr. Daniel. But the real beginning of this story might be different: if I hadn’t met the sociologist in the first place (we didn’t meet by chance) the story couldn’t even have started to take shape. Maybe the story

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1 Tau protein aggregates pathologically in the brain. In Alzheimer’s disease, the protein is deposited within neurons and aggregates in the form of neurofibrillary tangles. It is unclear whether these tangles are causing the disease or are its consequence. Tau’s pathological aggregation is found in numerous neurodegenerative diseases referred, as a result, as “tauopathies”: frontotemporal dementia, cortico basal degeneration, progressive supranuclear palsy, chronic traumatic encephalopathy and others.
commenced one year before, some years into the PhD in anthropology, when I found out that I
wanted to study “memory” and “dementia”. Or even before, when I decided to go to Berkeley to
do a PhD in anthropology. After I left Paris for San Francisco, my father told my mother that the
reason I went to Berkeley was to do some “tourism”. I’ll take my father’s words as the
indisputable beginning of this story. After two years of tourism at Berkeley, I decided to do more
tourism into the world of dementia. I first went into a dementia facility in a home in the United
States. I tried to learn “how to speak dementia” as one staff member encouraged me to do. After
five months I was disheartened, I wasn’t making any progress in my understanding of this
unknown language. I thought that instead of studying the diseased, I would study the ones who
were studying the diseased, the ones who understood their actions, their values, their beliefs, etc.,
the ones who were making knowledge about dementia, the ones who diagnose it.

In 1999, one year before being awarded the Nobel Prize for his work on procedural
memory, Eric Kandel wrote: “The mind will be to the twenty first century what the gene was to
the twentieth century.” Later on, he develops this neurological ambition for the mind along the
lines of others neuroscience works as “delineating the biological basis for the various unconscious
mental processes”. In the context of the tremendous expansion of neuroscience over the last 30
years, I wondered: what are demented patients teaching neurologists about the mind and the
brain? One could have told me that doctors are not philosophers, that they are only here to treat,
but the problem is that there is no treatment for dementia; at the time I am writing it is still an
incurable disease. Then, what is medicine doing with demented patients? Medicine must be
thinking about dementia, but what and how do they think about it? With this question I met the
sociologist, a friend of one of my professors of anthropology, who was already working with Dr.
Daniel. This is how I entered this clinic, that I choose to name the Memory clinic, situated
somewhere in the United States.

I met Dr. Daniel, we talked about dentistry and anthropology, about Alzheimer’s disease
and frontotemporal dementia. After thirty minutes the meeting was over, Daniel had other

2 “The common behaviour of mankind is the system of reference by means of which we interpret an unknown
language”, writes Wittgenstein in §206 of the Philosophical Investigations. My problem in understanding what was
going on in this home (or any other) was that I perceived many behaviors that didn’t seem to have anything in
common between each other; there seemed to be not one single language but as many as there were people.
3 To cite just a few: Jean-Pierre Changeux, Neuronal Man, The Biology of Mind, New York, Pantheon books, 1985
1990.
5 As it is traditionally the case in anthropology, the American clinic as well as the French clinic, which I present later,
have both been kept anonymous, as well as the places where they are situated. The anonymity was mandatory for
this research to receive its IRB approval. Yet, because these two clinics are well known on the international scientific
scene, it is not impossible with the aid of a basic inquiry to find out about their real names. Yet, to confuse a bit the
detective, when I make references to articles published by the neuroscientists who work in these clinics, I do not cite
them in the footnotes but I reference them in the bibliography at the end of the dissertation. The staff and the
patients are presented under pseudonyms. I kept the ways in which the staff addressed each other: the chiefs and
some of the doctors are addressed by their last name, hence I gave them a fictive last name, whereas the rest of the
team who is addressed by their first name, ended up with a fictive first name. Usually each member of the two teams
has one pseudonym and thus is represented by one character, but I sometimes gave the same pseudonym to different
persons to avoid too many characters and in order to ease the reading.
appointments waiting for him; when I left his office, the chief had granted me full access to the life of his clinic, I could go anywhere in the clinic and come whenever I wanted. The ease with which Daniel opened his door to me — and before me to the sociologist who introduced us — was in keeping with the pluridisciplinary policy operated at the University of R. This medical university, which hosts the Memory clinic, hosts also a program in medical anthropology of which one of the foundational claims is precisely to allow a (exterior) look onto medical practices.

I started coming to the Memory clinic in September 2010 and I left the clinic in December 2011. During that time, I was at the clinic three to four days a week, I didn’t wear a white coat; no one at the clinic does. I had the chance to be present at about 150 consultations of patients who were, or were not, part of a research program at the Memory clinic. I always introduced myself as an anthropologist to the patients. If I was introduced by some staff member, it was not as a colleague or as a student, but always as a, or as “the” anthropologist. I explained to the patients that I was studying their doctors and asked for their oral consent for staying and taking notes on the consultation.

A regular consultation unfolds in the following way: first the patient and his caregiver meet a neurology fellow for a long interview (one to two hours). The patient then meets the neuropsychologist (and many more people if the patient is part of a research program). While the patient is doing the neuropsychological test, the “caregiver” (whoever is accompanying the patient) is having a private chat with a nurse: in the absence of the patient, the caregiver has now a chance to say whatever he or she couldn’t say to the fellow neurologist in front of the patient. After that, the fellow, the neuropsychologist, the nurse and every other person that the patient encountered, meet with the “attending doctor” to discuss the “case”; this “case-conference” begins without the patient. At the end of the discussion the team often speculates about a diagnosis. Then the patient and his or her caregiver meet the whole team, questions are asked again, some tests are repeated, and the attending doctor gives the diagnosis. The patient then leaves, sometimes the team stays to discuss for a little longer.

Besides my presence at the different stages of these consultations, I was present at the weekly conferences listening to guests presenting their research to the clinic, the “journal club” where the team discusses scientific articles, talks and lectures given by the staff to the general public, fundraising events, a consortium, the clinic’s annual retreat as well as parties at the clinic. I interviewed all the clinicians (except for one neurologist, Albert, who repeatedly refused to meet me in private) and met with some of them at the occasion of a drink, a dinner, a lunch or a walk. Several months after I began the fieldwork, I asked Daniel if I could spend a moment alone with the patients of his research program: because these patients stay several days at the hospital, it was possible to find some time for a discussion, unlike with patients who came to the clinic only for the day. Daniel agreed. I entered the busy schedule of these patients under the event: “Breakfast with Laurence”. One morning of the week, I had a coffee and talked with the patient and his or her caregiver, in their hospital room. We usually had met before at the occasion of the interview by the fellow neurologist. I never systematized these interviews, our discussion depended on the questions raised earlier with the fellow, it depended on what they had to tell me, it depended on me feeling legitimate in soliciting them in these difficult moments. We talked about their life with and without their troubles, with or without a diagnosis, with their expectations, without treatment.
The Memory clinic, aside from a sociologist and some anthropologists (another one came after me), also welcomes artists in residence, and after I had left the clinic, a philosopher began collaborating with some of its members. Unlike this philosopher I was not collaborating on a project, my presence at the clinic wasn’t justified by my usefulness, I hadn’t been solicited and certainly not for my skills in anthropology. Unlike this philosopher, I thus started my inquiry in the position of a mere observer, free from any duties with respect to the medical team; I didn’t have to worry about being totally absorbed by the structure and by the others. Yet, an observation that would be completely exterior would only amount to imagining the perceptions, thoughts and feelings of the others and would run the risk of completely missing the actual meaning of the practices. Nevertheless, me occupying this other status of the anthropologist referred to as “the actor” or the “participant” surely wasn’t immediate. At the beginning of my fieldwork, Daniel’s co-workers didn’t exactly share the spontaneous openness of their chief: some remarked to me at several occasions (when for example I asked about a schedule, when I wanted to be present in a case-conference, etc.) that Daniel is too well known for saying yes” to “everybody” — or to anybody. They had questions and reservations about my presence at the clinic, which must have appeared shady to many. They “realized”, like the neurologist I met at the consortium, that I was there to observe, and potentially to criticize. As the neurologist exclaimed while eating a croissant, this meant that I needed to be handled carefully, especially nicely, so as the critiques wouldn’t come out too harshly. Going to the clinic almost every day of every week for more than a year has surely instituted another relationship between me and the team than the relationship one can have with a United Nations’ inspector. I have indeed shared a (form of) life with the team of the Memory clinic. I tried to be up to date about what was going on in the clinic, I asked questions, I asked for explanations, I was surprised and sometimes this lead to comments, I learned about the way of doing a diagnosis, I participated in it — I, too, “bet” on the diagnosis —, in brief I slowly learned to know my “field”: one possible root of this strange word “fieldwork” analyzes Jean Bazin, brings its meaning close to a “field of operations or maneuvers, or even of the battlefield”. At the end of my fieldwork I was told several times that I was part of the team, but how was I part of it? I was never considered as a neurologist, or a neuropsychologist or any other staff member, so in which capacity did I “participate”? What was (were) my role(s)?

6 As Jeanne Favret-Saada analyzes in Désorceler, Paris, Editions de l’Olivier, 2009, about the position of the “actor” occupied by the anthropologist: if this status is not balanced by this other status, the one of the observer, the unlikely “fusion” with the other that might result from it, would only affect the ethnographer and wouldn’t throw light on the affects of the other.

7 No doubt that until the end of my fieldwork, I assumed the position of the observer, an essential position to preserve a distance, essential in order to be able to describe anything. Me occupying this position was accompanied with questions until my departure of the clinic: What did I take into notes? What will I write? “Did we do good?” I was asked the last day. Yet this questioning was not only assorted with mistrust. The day I left the Memory clinic, in December 2011, some members of the team opened a box with a big cake, there was even some applause. I was touched. While we were eating the cake and listening to music in the big office where the neuropsychologists and the fellows worked together, Jack, a fellow, said: “We need a staff anthropologist!” Why not appointing me when I’ll come back from France: “I could give them some feedback”. The others at the small party agreed with Jack. Thus, observing doesn’t only lead to censorship, but also to its opposite: having an exterior point of view on clinical practices can be an added value; a form of reflexivity that the observed can lay hold of.

In March 2011, seven months after I had arrived at the Memory clinic, I entered the big room on the 8th floor where case-conferences usually took place. Some members of the team were already sat around the table waiting for Daniel to start the discussion about the patient. Judith, a genetic counselor, who is also Daniel’s right-hand man, had entered the room right before me; she had made her way to the corner of the table far from the door, where I usually sat. When I came in, she was still standing but about to sit. She was laughing with Olivia, a neuropsychologist. When Judith saw me entering, she apologized: “Oh! I was going to take your seat”, she said. I answered that it didn’t matter for me to have a specific seat, and that actually I will take “hers” (the one close to the door, vacant in front of me, where she usually sat). I pulled out the seat from under the table. But Judith insisted: she walked towards me while saying that really, no, no, I should sit where I usually do. I am polite, I complied: I went to the other side of the table, and sat where I usually did. In front of me, on the other side of the table, Olivia was smiling; Olivia told me that she and Judith were wondering what it felt like to take the seat of the anthropologist. This episode obviously shows that I was considered as an observer and that Judith and Olivia wondered what I was observing. A position though, that wasn’t fully dismembered from others since Judith was curious to occupy it —even if symbolically. In the end, she decided, with a strange insistence if we think that what was at stake were simple seats, that our positions were not exchangeable; Judith, one of the mainstays of the Memory clinic, didn’t want to swap her role for mine.

Six months after the episode of the chair, I was in a case-conference, with the sociologist who introduced me to Daniel. The fellow started presenting the case to the attending doctor but suddenly coup de théâtre: the fellow presenting was not the one who saw the patient; he was covering for his friend who couldn’t make it to the conference. The attending doctor was furious: “So actually it isn’t your patient? This is unacceptable”, he said, icily. To calm his anger and as to obey to the proverbial It doesn’t do to wash one’s dirty linen in public, the attending doctor decided to expel the “public” from the room: guests, medical students, the sociologist and I, had to leave. After I left the room I met Judith in the corridor, she asked me what I was doing there, she thought I would be at the conference. I explained what happened. She exclaimed: “But you are part of the team, you’re not an observer!” She then went knocking on the closed door of the conference room to attempt to let me in, but also got thrown out by the attending.

Between these two moments, when Judith told me I was part of the team, that is, not an observer, and before when she insisted that I keep my observer seat, six months had passed, during which I may have played different roles. My progressive “participation” in the life of this clinic has surely depended on time but also has depended on the positions that people at the Memory clinic gave me, it has depended, as Jeanne Favret-Saada has terrifically shown in her Deadly Words10, on the kinds of knowledge that people granted me. If I was quite content (and surprised) when I was told I was part of the team, it nevertheless wasn’t an achievement: the roles that I played are the ones that people gave me to play. I followed. I was there. If “being there,”

9 The distinction between “observer” and “part of the team” was hers. I didn’t mention the word “observer” when I recounted the story to Judith; I didn’t say for instance that the attending expelled all the “observers”.

10 See Jeanne Favret-Saada, Deadly Words: Witchcraft in the Bocage, Cambridge University Press, 2009. At the beginning of her inquiry on witchcraft in Mayenne, Jeanne Favret-Saada asks questions, none of which help her progressing on her understanding of witchcraft. It is only when she starts occupying the position of the unwitcher, because Jean Babin and his wife granted her this knowledge, and because she accepted to occupy the role they her to occupy, that she could progress on the treatment of her object of inquiry.
Clifford Geertz writes, is what sustains the legitimacy of the anthropologist, then, with respect to the body of this dissertation, to what I have learned and now recount from the time I spent at the Memory clinic and then in the French clinic, it doesn’t seem superfluous to attempt to clarify who I was, there.

Who I was, this knowledge that people granted me depended on what people wanted to see me doing—which often depended on their own questioning about their work. What were these roles they wanted to see me playing, where did they want to take my inquiry and did I follow their lead?

Michael, a fellow neurologist, always presented my project (to the patients, to a new fellow, to a guest) as focusing on the ways “doctors took care of their patients”; Michael never gave up on this humanitarian, so to speak, orientation of my project, even though I repeatedly reminded him that I was focusing on the ways neurologists make knowledge and not on the empathy at stake in the relation doctor-patient. I listened to Michael’s worries, which not only bore on the relation he had with his patients but also, on the one he had with his chief, but I cannot really say that the role of the confidante that Michael gave me, impacted my inquiry.

After a while of my presence at the clinic, Olivia, a neuropsychologist, told me how interesting it must be to study people (her and others) at the Memory clinic. “Why?” I asked. Because, she said, I couldn’t have missed the dynamic between two different “clans” at the clinic: the one of the neuropsychologists and the one of the fellow neurologists; how the latter looked down on the work of the former, how the neuropsychological test occupies only a supporting role in the making of the diagnosis compared to the powerful role of the presentation of the patient by the neurologist. I hadn’t missed the surprising minor part that seemed indeed to be granted to the multitude of tests that Olivia, Emily and the other neuropsychologists spend time giving to the patient, and further grading and interpreting. My inquiry on the diagnosis (chapter four) does justice to Olivia’s remark (I allude to these tests), yet I didn’t write about the sociological dynamic Olivia was hoping to read about.

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11 See Clifford Geertz, *Works and Lives, The Anthropologist as Author*, Stanford University Press, 1989, p 4: “The ability of anthropologists to get us to take what they say seriously has less to do with either a factual look or an air of conceptual elegance than it has with their capacity to convince us that what they say is a result of their having actually penetrated (or, if you prefer, been penetrated by) another form of life, of having, one way or another, truly ‘been there.’ And that, persuading us that this offstage miracle has occurred, is where the writing comes in.”

12 Even though Michael’s lack of confidence shows how hospital services (American or French) are responsible for the deep regression into childhood of their fellows and residents. Michael and the other fellows (and the ‘interne’ in France) appeared to be constantly looking for any piece of recognition from their chiefs as if their lives were suspended to their critique.

13 Other members of the team had other expectations about what I would write. Here are two more examples of where some members wanted to take my research. The nurse Beth, as well as the other nurse and the social worker of the clinic, asked me repeatedly if I would write about the ways the team “spoke about the patients” (in their absence). I don’t know if they were hoping that I would write about it or that I wouldn’t. At several occasions, they apologized silently to me, with an embarrassed smile, in the middle of a conference when the team was laughing about a patient. One other example: when I went to interview the only psychiatrist of the clinic, and told her that I was trying to understand how the team succeeded in making a diagnosis, she reinterpreted my project as an observation of how the doctors at the clinic make diagnoses differently one from another; she indeed had a distinctive approach to her patients than her neurologists’ colleagues. Even though I told her I was not precisely working in establishing different clinical styles, she then at several occasions told me how fascinating it must be to
The seat I occupied in front of Daniel, who is the most present figure in my dissertation, has, I think, influenced quite a bit what I then wrote. In front of Daniel, I occupied the position of the “psychologist”—or perhaps even, of the “psychoanalyst”. Each time Daniel asked me to give my opinion about what the diagnosis could be, he would always give me the choice between a “psychiatric” or a “neurological” cause. He would rarely present the question in that way to a fellow, or to a guest neurologist. This role led to discussions, to which some members of team sometimes also participated, about the place of a patient’s life—his or her “milieu” (familial, social, etc., in brief relational, reorganized by the disease)—in a neurological diagnosis. These discussions solicited by my role, directed my inquiry in two directions: understanding the way behavioral neurology—Daniel’s discipline—perceives the patient (chapter one), and understanding how the neurologist used his “feelings” to “penetrate” the patient, in order to make a diagnosis (chapter four). The psychoanalyst, for Daniel (as well as for the vast majority of neurologists I met), embodies irrationality and obscurantism. I sometimes saw this role of mine as a proof that the chief of the Memory clinic didn’t think that I could understand the complexity of neurology. Yet once, after a quite turbulent discussion about a patient’s case between Emily a neuropsychologist, Daniel and myself, Emily told me spontaneously that if Daniel “poked” me it is because he “respected my opinion”. Emily herself seemed to have begun to question her chief’s diagnoses, and she told me she felt good because she “stood up for herself more”. When I came back from my fieldwork in France, Emily had left the Memory clinic; I had the impression she had been fired. Perhaps the role that I occupied in front of Daniel couldn’t be played by someone else.

In December 2011, I left the Memory clinic and I went to France to do comparative fieldwork at the Alzheimer clinic. I stayed in the French clinic until July 2012, so seven months. I was introduced to the chief of the Alzheimer clinic, Dr. Vincent, by a famous French neurobiologist, friends with one no less famous French surgeon who is part of my family.

I chose to do this comparison at the Alzheimer clinic because these institutions share similar features, yet they are not totally alike. The Alzheimer clinic is to France what the Memory clinic is to the United States: one of the biggest clinics of the country for diagnosing and researching dementia. Patients travel from all over the country to be diagnosed at the French clinic, as they do at the American clinic, because: “It’s the best”. The Alzheimer clinic has research programs, like the Memory clinic and most of the clinicians who work in these clinics are also involved in research. This wasn’t guaranteed: the Alzheimer clinic is one of the few clinics in France which, inspired by the American model, has integrated research and clinic together at the heart of the public hospital. In France, this is a quite recent endeavor, as Jean-Paul Gaudillière has shown. Nevertheless, research at the Alzheimer clinic operates on a much smaller scale than at

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14 But it is a lot smaller: the French clinic employs one-third the number of people the American clinic employs today.

15 The reform in the US is due to Abraham Flexner whose report, ordered by the Carnegie foundation, was in 1910, at the origin of the union of teaching, medicine and research at the heart of the hospital (see Jean-François Picard, “Poussé scientifique ou demande de médecins : la recherche médicale en France de l’Institut national d’hygiène à l’Inserm”, Sciences Sociales et Santé, 10(4), 47-106, 1992). This reform will wait for the post second-war years to radically change the French hospital. Jean-Paul Gaudillière develops that the advent of this biomedical model in France provoked “an inversion of the center of gravity in the system”: the biology lab replaced the hospital service as
the Memory clinic. When Dr. Vincent visited the American clinic at the end of July 2012, he
told me how “extraordinary and magical” he found the place. The chief of the French clinic
exclaimed: “Dr. Daniel has 90 persons who are working on frontotemporal dementia: the whole
department of research in neurology in our hospital! How are we going to make that happen?”
Vincent asked me. The French neurologist realized that his financial means were not comparable
to the ones of the American leader. I was having dinner with Daniel, Vincent and the
neurologist who opened this introduction: while drinking some pinot noir, Daniel and the
neurologist were telling us how difficult it was to gather 80 million dollars in order to hire a new
psychiatrist and to create his new research program. Vincent looked confused: “You mean, he
[the psychiatrist] is asking for what?” Daniel repeated the number: banal in the world of research
explained the neurologist, who told us that his research program on imaging and dementia
received 140 million. Vincent laughed: “I have three million... a little bit more!” Daniel eased the
atmosphere: “That’s good, you’ll pull it off!” he said to reassure Vincent.

The reason I went to the Alzheimer clinic in France was not to know how different modes
of financing, industries and administrative regulations, contribute to shape medical practices. I
went to France mainly to have another point of view on the American clinic. In that respect I
could have gone anywhere; as long as I had found a comparable place, I could have stayed in
America. I chose France, not only because I speak French but also because I had another question.
I was wondering if the psychodynamic or psychoanalytic model that seemed still more resonant
in France than in the United States (versus a model more oriented towards biology in the United
States) had any impact on the understanding of the troubles, and the diagnosis of the patients
with dementia. If Alzheimer’s disease, frontotemporal dementia and other dementias have

16 Frontotemporal dementia is one type of dementia that fascinates Daniel, as we will see.
17 Both the American and the French hospital are considered “public”: both are financed by the state. In reality, the
American clinic is mostly funded by private funds and grants won from the NSF. If the French clinic tries to bring in
some private funds, philanthropy being not much encouraged by the French government (compared to the
American government especially through the lowering of taxes), the Alzheimer clinic ends up being financed mostly
by the state.
18 Indeed a research on PubMed shows that articles, letters and editorials about the relations between neurology and
psychiatry, have exploded since the middle of the 1990s and that the ones who argue for the “breaking down” of the
“barriers” (Kandel and Squire, “Neuroscience: Breaking Down Scientific Barriers to the Study of Brain and Mind”.
Neurology and Psychiatry”, Neurology, 40 (2), 388, 1990) that would pass “through the brain” to “integrate”
(Martin, “The Integration of Neurology, Psychiatry, and Neuroscience in the 21st Century”, American Journal of
Psychiatry, 159 (5), 695-704, 2002) psychiatry and neurology are almost exclusively written by American authors. This
“programme fort” of neurosciences, as Alain Ehrenberg has called it (in *Le sujet cérébral*, Revue Esprit, Novembre
2004, p 132) aims at treating psychopathology in neuropathological terms. Ehrenberg observes that besides the
scientific literature arguing for the fusion of psychiatry with neurology, the National Association for the Mentally Ill
defends since the 1970’s an understanding of mental diseases as cerebral diseases, an orientation promoted by the
American institutional system which unlike the French one, “encourages” a “materialist” reading of mental diseases.
This reading is justified in the United States by the argument that the mind-body dualism authorized by the
separation of psychiatry from neurology is a source of “stigmatization of mentally diseased” that in the end leads to a
univocally a biological cause they are mainly diagnosed on psychic troubles thus could different traditions in psychiatry and neurology change the ways the neurologists understood these diseases? The first day I arrived in the service of the Alzheimer clinic, I was not disappointed: there were some psychologists (and not only neuropsychologists), who were seeing and reporting on each patient; I had never seen a psychologist at the Memory clinic. Yet, their role in the making of the diagnosis quickly appeared to me as being quite minor. There were differences though in the way one French neurologist, Elise, understood the symptoms of her patients: not necessarily as the phenomena caused by a neurological lesion but also as the signs of a mental disease with a neurological dysfunction. Elise insists on the relevance of a psychological understanding of her (neurological) patients. But Elise is quite unique at the Alzheimer clinic, and broadly speaking she is an incomparable neurologist. If the comparison basically failed on the ground of the hypothetic influence of two national traditions in psychiatry and neurology, being at the French clinic allowed me to understand what had appeared to me unusual or disturbing in the American clinic. Somehow the comparison with the Alzheimer clinic revealed and at the same time made commensurable what was disconcerting in the medical practices at the Memory clinic. I develop how in what follows.

At the French clinic, unlike at the American clinic, I was wearing a white coat on which I pinned a badge with my name, like everyone else in the clinic. I introduced myself to patients as a sociologist (‘anthropologist’ raised too many questions) and asked for their oral consent to stay at their consultation. When staff members introduced me to the patients it was often as a medical student, as a collaborator, or “my young assistant” as Vincent said. I was present at around 60 consultations. Like at the Memory clinic, a regular consultation brought together the resident, the patient and the person who came with him or her – usually called “the wife”, “the husband”, etc., more than “the helper”: the French equivalent for “caregiver”. Compared to the fellow who was doing this interview at the Memory clinic, a resident has, in general, less experience and the interview was much shorter than at the Memory clinic. The patient also met the neuropsychologist; the test took usually longer than in the American clinic and the testers had much more initiative: they were able to vary the tests if they thought this would bring new light on the diagnosis. The person who accompanied the patient was not necessarily heard privately by a staff member, it happened, though not always. Two other staff members listened to the patient’s story: a psychologist and a social worker. The case-conference that followed with the attending doctor was generally much quicker than in the US — half an hour maximum when in the US it was two hours. Besides these regular consultations, I went to the consultations held by Pierre, a neurologist who worked in another hospital, but came one afternoon every week at the clinic.

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19 Nicolas, the resident in neurology who was at the Alzheimer clinic during most of the time I was there (from January to May 2012) had just left medical school, thus he had no experience.

20 The fellow at the Memory clinic interviews one to (more rarely) two patients in half a day. The resident at the Alzheimer clinic interviews five patients in half a day and also at the same time, performs lumbar punctures, fills patients files, answers to questions the staff asks (or shouts) at him or her, supervises and chat with medical students, makes phone calls to the imaging service, to the patients’ doctors, etc.

21 Psychologists are absent in the American clinic. There is one social worker employed part-time at the Memory clinic; her primary role -- when she has a chance to see patients -- is the same as the nurse’s: she listens to the “caregiver”. But she also has the mission to find financial aids for the patients. Of course her range of action is much more limited than the French social worker who operates in a “health care system” still worthy the name.
Alzheimer clinic to see “complex cases”. I went to the private consultations of Dr. Vincent and of other neurologists, François and Elise. I interviewed and discussed over lunch with some clinicians. I went to several scientific conferences held by or with the neurologists of the Alzheimer clinic.

Very quickly, the first day I met Vincent actually, I knew about the political dissensions that opposed the Alzheimer clinic to the rest of the neurology department. Maybe as a result, Vincent saw me as a sort of auditor, imagining that the comparison with the American clinic would diagnose some of the failures of his service, and will help to make it more efficient and rentable for the department. Vincent was wrong in believing this, because the Memory clinic functions on a model of care that is not at all rentable for the American university that hosts it; a model that would be rejected today by Vincent and his hospital administration. I actually met the administration of this French hospital at the traditional “galette des rois”, I introduced myself to the chief of the neurology department, openly hostile to Vincent. The chief firmly grabbed my neck (as if I was his patient, to immobilize me) and spoke in my ear: “An anthropologist at the Alzheimer clinic, perfect! I think this is exactly what we need: you’ll report to me about what’s going on there before you leave.” Contrarily to how it started at the Memory clinic, Vincent awaited my exterior (and American) point of view in order to evaluate the medical practices in his clinic. For the chief of the department, it even would have been desirable that my “expertise” helped him censor Vincent’s clinic (I never went to report to the chief). When I gave a presentation of my project to the French team, I clearly said that I was studying, and not evaluating, clinical practices. Yet, the mere fact that I was “comparing” the French clinic with another clinic, whose international reputation moreover was firmly established, meant that my inquiry would potentially assess or rank the French practices against the American practices. Although the Americans never asked me about the French, the French often asked me about the Americans. It is from the perspective of this French fieldwork that the “Americans” started to appear to me as an “other”. Jean Bazin writes, quoting Bakhtine, that a “culture” can be “revealed” —and thus produced by the ethnographer— only through an “essential exotopia”. Bazin also quotes Boones: “A culture can materialize only in counterdistinction to another culture.” This is exactly how the French fieldwork operated: with the displacement it created, it “materialized” —in writing— the American practices as different.

At the Alzheimer clinic I had one privileged interlocutor: Elise, a neurologist. I liked our discussions, without hurrying and with many silences. These silences punctuated her interviews with her patients too, they never carried discomfort, they were somehow therapeutic, inhabited by

22 Vincent, himself, and the “surveillante en chef” of the Alzheimer clinic told me about these antagonisms.
23 If the Memory clinic flourishes the way it does it is only because Dr. Daniel and his team are able to raise funds, especially from wealthy habitants of the city or philanthropic patients, and are very talented at winning grants. But the model of care of the Memory clinic is exactly the model the French clinic cannot give to itself.
24 At Epiphany, each department of this hospital (as well as many French people) gathers its staff to share a “galette des rois”, a round and flat pastry filled with almond paste. Inside there is a “fève” hidden, a small thing or figure. The one who finds the fève in his or her portion becomes the king or the queen.
25 At the time I was doing my fieldwork at the Memory clinic, the Americans didn’t know I would do a comparison, I didn’t know it myself. But even afterwards, when I came back from France and spent a little time again at the Memory clinic, the Americans didn’t ask me about what had been going on in the French clinic.
27 Ibid. p 428. Quoting J.A. Boone, Other Tribes, Other Scribes, Cambridge, Cambridge University Press, 1982, p. IX.
her consideration for the answer she was thinking carefully about before giving it to her patient, or to me. I had discussions with other neurologists and some neuropsychologists, but overall, I felt that I was not addressing a “team”. I could have had this impression because precisely I wasn’t part of it. Nevertheless, my impression was confirmed by a neurologist who worked in these two clinics—previously in the American and now in the French—and was also based on several occurrences. There was a turnover of the staff (just before I arrived one neuropsychologist had left, and two neurologists left while I was there) and “leaving the Alzheimer clinic” was also a common matter of discussion among the staff. Case conferences rarely succeeded in gathering all the people together from the beginning until the end: often, once one had made his or her report to the attending neurologist, he or she left the room to go work on something else. It thus became difficult for me to understand what was going on in this clinic as the project of a “whole”. Also, from the first day I arrived at the Alzheimer clinic, I was reminded that the hospital is a big enterprise. Something that I knew from my previous work (four years as a dentist in the Emergency Room of the same hospital) but that I had forgotten during my years of tourism away from the French hospital. Vincent had already told me about the conflicts he had with his administration and how medicine is also thought in terms of personnel. Besides, I heard every week about hours or days of work to make up for, transportations and child daycare, tasks that were not assigned to the right staff member, lack of personnel, and lassitude. I was back “home”, and in front of what Antoine Sénanque28 calls this “distinctive discordance” of medical practices: between the impression that medicine is a job like any other and what is “behind”: suffering, death and experiences dissociated from the everyday life. This discordance undoubtedly existed also in the American clinic but somehow it was in the background. Thus, my feeling of familiarity at the Alzheimer clinic along with my vague impression that I was observing two different styles of medical practices—to borrow a perilous comparison with cinema: on the one hand “French realism29” and on the other hand American science-fiction30—revealed to me what was so peculiarly, so disturbingly proper to the Memory clinic. I organized the dissertation around these unfamiliar and troubling aspects; the emphasis through the dissertation is thus on the American clinic.

The first two chapters revolve around a talk Daniel gave about his research on frontotemporal dementia, his specialty. It is a form of answer to the question I was asking myself when I left the old people’s home: what our contemporary neurologists understand about dementia? Because of their expertise in this strange disease31, I mainly focus on the understanding of FTD at the American clinic.

The first chapter aims at situating this knowledge in relation to the patient first, and second in relation to two disciplines: neurology and psychiatry. It begins with a presentation of Daniel and Vincent’s field of expertise: behavioral neurology. Through the brief history of this

28 Antoine Sénanque is a French neurologist who writes under a pseudonym about his experience of “medicine, this shit” echoing Louis-Ferdinand Céline. In Antoine Sénanque, Blouse, Paris, Grasset, 2004, p 177.
29 The paragon of French realism in cinema is Maurice Pialat (L’Enfance Nue, 1969, Sous le soleil de Satan, 1987). His style has sometimes been rerouted by other film-makers towards the mere expression of boredom; as the filmmaker Arnold Pasquier illustrates it: a child who cannot go to school because she has measles becomes the principal subject of this “kind” of movie (personal communication).
31 What is strange in this disease is first and foremost what the American team says about it.
field, we begin to understand what allows the behavioral neurologist to have a distinctive access to the patient and broader, to the human. The chapter follows the development of Daniel’s talk: how is frontotemporal dementia (FTD) understood in relation to the well known Alzheimer’s disease (AD), and how the revival of the diagnosis of FTD participates not only in the making of knowledge in the field of neurology, but more broadly to the understanding of mental diseases. Following this situating chapter, the second chapter focuses more on the emergence of the diagnostic entity, FTD, and on the development of knowledge about the “social brain” that preceded and accompanied it. Through the writings of neurologists over one and a half centuries I tell how knowledge about the “social brain” progressively took shape through the study of the impairment of the frontal lobes, how did the neuroscientists understand what was impaired (reason and then emotion), and how these scientists understood this elusive concept: the “social”. The endeavor is not history, but a comparison of different explanatory models of how the brain is involved in our social being. I finish the chapter describing how FTD helps to specify what defines our sociality and further our humanity, according to the American team.

It seems to me, that the French and the American clinics have been differently influenced by these models and writings, and thus understand quite (but not totally) differently, how this “fellow circuitry” —as Daniel would say— is impaired in FTD. If the understanding of FTD by the French team is discrete through these two first chapters, the conclusion of the dissertation provides a more proper comparison of how FTD is understood in the French and the American clinic. Thus contrarily to the body of the dissertation in which the fieldwork in the French clinic is used mainly as a spotlight in the background that illuminates the uniqueness of the work done in the American clinic, in the conclusion the different knowledge produced about FTD in both clinics are considered for themselves. I’ll wait until the conclusion to make this comparison because it benefits from the descriptions of the clinical practices from chapter three and four. The aim of this comparative conclusion is certainly not to use the mediation of “mentalities” or “culture” to go from the particular to the general –culture is a concept, Jean Bazin has analyzed, that only pretends to “explain” these differences—but rather and only to describe a variation of medical practices. This is an important word of caution: if I said that the French fieldwork helped me see the unfamiliarity and the troubling aspects of the American practices, it was only the point of departure of the investigation but not its ending point. In the end, these practices only appear as different. We will read in chapter four, how the chief of the French clinic actually expresses nostalgia for clinical practices that were his before, and that are today (without him knowing it) the ones I actually observed at the American clinic; the differences observed are thus only, as Bazin writes: “The realized variants of the same universe of the possible”.

Despite the “total” knowledge –biological and social—of the human that the neuroscientists have build from the cerebral destruction, when time comes to diagnose an

32 Neurologists often talk about circuits or networks in the brain: the “consciousness” circuitry, the “default mode network”, the “salience network”, etc. The “fellow circuitry” is one of Daniel’s inventions, for now this term has stayed between the walls of the Memory clinic.

33 Jean Bazin, in Des clous dans la Joconde, analyzed that the “ethnological paradigm” declares that culture is a “cause” of differences. This “ethnological hypothesis” allows the anthropologist to “explain” a behavior by saying that it is “Californian”, “French”, “Chinese”, etc. Bazin criticizes the explicative range of an observation that is first and foremost reflexive; the anthropologist needs to face this fact: “culture” is first what we don’t understand in the other.

individual with dementia, some uncertainties arise. Chapters three and four examine how the knowledge about dementia, is exercised in front of an individual patient, but also how, this individual enriches knowledge. In chapter three, I describe the uncertainty that sometimes surrounds the diagnosis of neurodegenerative diseases. As the neurologist Antoine Sénanque reflects on his particular medical specialty: “In neurology, we don’t know the cause of the diseases, and we don’t know how to heal them.” Margaret Lock has recently engaged with the ethical and epistemological problems posed by this uncertainty around the diagnosis of Alzheimer’s disease. I describe some of these uncertainties around the diagnosis of neurodegenerative diseases, but differently than Lock, it is primarily done from the actual clinical practices and not through what the scientists say about them. The description of these uncertainties examines the ethical and epistemological troubles that the neurologists (and we) face, and serves as a springboard to land in chapter four, which aims at describing how the clinical team succeeds in making a diagnosis despite these uncertainties. Perhaps because of these uncertainties actually, the neurologist uses his or her flair, a clinical sense, a feeling, an impression to arrive at diagnosis: tools which are for the clinician, as reliable as the technological tools, and maybe more. “Medicine is not a science”: anyone who isn’t sick is ready to accept this verdict; this fourth chapter describes, how and why we say that medicine is not a science. Or, as Steve Shapin recently wished for: “How [in science] taste judgments come to be formed, discussed and sometimes shared” so as one could learn more about the “subjectivity” at work in science. The reading of this outline might give the impression that I am dissociating research or theory (chapter one and two), from clinic or practices (chapter three and four). As we will see, this frontier isn’t real: the knowledge exposed by Daniel and his team about FTD informs directly the impressions of the team, and those contribute to the building of knowledge.

This work is a description of the building of knowledge about dementia from the variable standpoints of the scientists who make this knowledge. As such the form I gave to this ethnography is inspired by the way Georges Canguilhem characterized knowledge: like life perhaps, knowledge is to be found in human mistakes as well as in human “wandering.” Thus this work, chapter after chapter, describes the different points of view on the disease (ethical, historical, neuropathological, social, philosophical, clinical, psychological, cultural, emotional, and

35 In Blouse, p 263.
36 See Margaret Lock, The Alzheimer Conundrum, Entanglements of Dementia and Aging. Princeton University Press, 2013. Lock examines the recent efforts of neuroscientists to diagnose Alzheimer’s disease earlier and earlier, long before the first symptoms appear. The ethics of such a project are in question through the text (since there is no cure for Alzheimer’s nor an efficacious treatment to slow down the disease), but it is mainly on the epistemological ground that Lock challenges this enterprise. She examines the debates in the “Alzheimer’s world” around the definition of Alzheimer’s disease, debates sustained by the persistent uncertainties in the diagnosis of this disease: spreading from the difficult concept of “normal aging”, to the still unknown cause for Alzheimer’s, to the impossibility to ascertain the diagnosis other than retroactively, by post mortem autopsy of the brain. Against these uncertainties, neuroscientists have aimed at a better standardization of the diagnosis with the help of recent technological innovations. Lock details greatly these new tools and her analysis underlines the naive belief that with the advent of technologies, conceptual problems and uncertainty would disappear.
37 Steve Shapin in “The sciences of subjectivity”, Social Studies of Science, 42 (2) 170-184, 2012, p177, deplores the lack of ethnographical treatment of the subjectivity at stake in science.
38 “L’homme se trompe parce qu’il ne sait pas où se mettre” (“Man makes mistakes because he doesn’t know where to stand”, my translation) wrote Georges Canguilhem in “Nouvelle connaissance de la vie”, in Etudes d’histoire de la philosophie des sciences concernant les vivants et la vie. Paris: Vrin, 1968. p. 364. Where do the ones who make knowledge about dementia do stand then? This is the question I am trying to answer.
of course cerebral) adopted by these doctors in the pursuit of the understanding of dementia. If I used much the verb “to describe” in this introduction, it is in the hope that this work will actively succeed in describing how the neuroscientists that I saw acting for two years, understand dementia —what they think and what they believe dementia is—, how they distinguish the demented person from the reasonable person, how they make a diagnosis of dementia. I learned from the anthropologist Jean Bazin and from the philosopher Peter Winch, both inspired by Ludwig Wittgenstein, that what ought to be done in order to understand who we are, is to understand how we do. For that no interpretation is required⁴⁹, nor an explanation. This is what Wittgenstein tells us:

“I think one reason why the attempt to find an explanation is wrong is that we have only to put together in the right way what we know, without adding anything, and the satisfaction we are trying to get from the explanation comes of itself.”⁴¹

We can only describe and say, human life is like that.”⁴¹

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⁴⁹ Jean Bazin, in Des clous dans la Joconde and Peter Winch in the little book that he wrote in 1958, The Idea of a Social Science and its Relation to Philosophy, both hold against Max Weber that to understand the meaning of an action, we cannot start by observing humans as a naturalist would: isolating their gestures and then interpret their meaning in order to try to understand the sense of what they are doing. An anthropologist, or a sociologist, observes that human beings behave not in any which way and also differently: what lacks is not the meaning or the sense of what they are doing as if the inquirer was in front of an indecipherable message, what lacks is the capacity of describing what they do. Bazin writes: “I don’t begin to observe the movement of the eyelid [reference to Geertz’s foundational example of the twitch or the wink] or the gesture of raising the rifle [reference to Weber’s example in Economy and Society] to then attempt to understand the meaning of these behaviors. If to wink or to shoot a condemned person pertain to an ensemble of actions that are familiar to me, I know right away what they are doing, I don’t need to proceed to an interpretation”. In Des clous dans la Joconde, p 450, my translation.


⁴¹ Ibid. p 3e. Emphasis in the text.
CHAPTER 1

THE SCIENCE OF FRONTOTEMPORAL DEMENTIA

“Emotion is more the soul
Memory is more the brain”

Olivia, a neuropsychologist at the Memory Clinic.

Sunday, Michael came for dinner at my house. He is the only neurologist that I still see since I left the Memory Clinic eight months ago. He told me that Dr. Daniel was going to be honored this week for his work at the hospital of R.

Whilst I was in France doing comparative fieldwork, the Memory Clinic moved to a more spacious and airier part of the town. Some of the staff at the clinic feel a little bit isolated here. The new site that will soon welcome half of the departments of the hospital of R. is still partially under construction. The part that is now completed is crossed by large white paths bordered with growing palm trees. Michael and I walk towards the new buildings, the heads of the palm trees stir in the wind. The breeze is constant here, he says. The sun is hot; it feels as though we are in the South.

The ceremony takes place in a big amphitheater with comfortable beige seats covered with felt. The chief of the neurology department at the hospital of R. is honored to introduce and to decorate Daniel today. He tells us that they first met in 1998. He and Stanley Prusiner42 went to find Daniel in his “trailer”: “Blown away by the structure of the clinical service” that Daniel had developed, they poached him from the “trailer”, gave him a professorship and Daniel founded the Memory Clinic. Since ’98, Daniel and his clinic have come a long way:

“Daniel is an amazing team builder and educator. Today he has 22 faculty, way more than a hundred people, more grad support than any of us could ever wish for, and the Memory Clinic is certainly in the clinical neurosciences, the place where ambitious young people would dream once to train.”

42 Stanley Prusiner received the Nobel Prize in Physiology or Medicine in 1997 for his research on prion.
When Daniel created the Memory Clinic, he was surrounded by five people. Fourteen years later, in 2012, there are 116 employees showing their I.D. every morning to the concierge posted in the imposing entrance in front of the big staircase in wood. They then walk up to one of the three floors that lead to the offices, conference rooms and labs of the brand new Memory Clinic. Olivia, a neuropsychologist at the Memory Clinic, once told me: “Daniel made the clinic of our dreams”. “It’s fabulous!” the French neurologist Dr. Vincent exclaimed, when he came to visit the new building made of glass walls. The chief neurologist of R. describes Daniel as a team builder and “a very good guy: loyal to his kids, to his wife, to his parents, loyal to his colleagues, the kind of person you walk towards when you see him down the hall.” It is, however, not only for his kindness and his entrepreneurship that Daniel is distinguished today; it is also as a scientist that the prestigious medical university of R. is proud to count him among its most glorious members. “Daniel’s science” is a science of frontotemporal dementia, a cerebral disease that affects the frontal lobes, the ones that “make humans human” or as Oliver Sacks would put it, without which “civilization would never have arisen”. The problems raised by this disease helped the Memory Clinic grow during all these years, and will continue to make it flourish if the growing interest for the frontal lobes and for frontotemporal dementia in contemporary neuroscience is sustained.

In the dark amphitheater, the chief neurologist standing in front of the microphone remembers:

“When those of us with grey hair were in medical school, the dementias were problems that had slightly different manifestations but we thought were all the same, even though we knew since a hundred years that the tissues looked a little different when you looked closely, but nobody had really pulled that together in a useful way. This is really what Daniel did using as a model a disease that we now call frontotemporal dementia. That’s a very common cause of dementia, not as common as Alzheimer’s disease... but a problem in which the degeneration begins not in the memory centers but in the emotional core of our nervous system, in the anterior part of our frontal lobes and of our temporal lobes. And Daniel laid out the clinical symptoms and signs and behavioral disturbances that may not read in an IQ test but that are socially

43 The new institute in France devoted to the brain, the ICM (Institut du cerveau et de la moëlle) is also made of glass walls. The architects were hoping that the enlightened corridor that runs along those glass walls would be a welcoming place for the scientists to “share” or “to discuss informally” about science. These informal discussions would be the springboard for creative science. In the end, the scientists stay in their rooms (which ended up to be without windows because all of them are in the corridor) because they have work to do and do not hang out in the corridors. If informal conversations happen it is mainly outside in front of the building, when people get out to smoke a cigarette, as the immunologist who works in a lab in front of the smoking area, once told me.


46 On PubMed, when entering frontotemporal dementia, the website mentions six articles on the subject during the year 1975. In 1995 the number is multiplied by six: 36 articles during that year. Six years after, in 2001, the 1995’s number is multiplied by five: 177 articles and the research on frontotemporal dementia took off... Exactly when Daniel created the Memory Clinic. There is a steady growth during the first 10 years of the 21st century, reaching 400 articles in 2010 (multiplied by 3 in 10 years). 2013 is the best year: 590 articles, and 2014 is promising: in April there are already 232 articles on that subject.
devastating: loss of empathy, apathy, loss of normal inhibition, loss of ability to read a crowd... And then he developed ways to actually distinguish this presentation from Alzheimer’s disease.

As it is typical of Daniel, he was able to find the very best people to attack this problem and as it is typical of Daniel, he was able to excite them for this problem. We are rewarding Daniel today for the magnificent work he has made in helping us to distinguish the dementias one from the other, telling us what pathways are involved in different people and in the larger population. And, as we are moving in the neurosciences to an era of therapy, understanding that not all people have the same pathways of genes and proteins involved is obviously essential... And of all Daniel’s contributions, I think this is the most lasting and important for society. So, Daniel, congratulations well deserved.”

The neurologist with grey hair leaves his spot on the stage to the chief of the Memory Clinic. Everyone applauds. Daniel, still troubled by a bad flu he caught during a trip to England, introduces himself in a hoarse voice: “Simon Daniel, I am a behavioral neurologist”.

1. A Story of Behavioral Neurology’s Birth and Thrive.

During the two years I spent at the Memory Clinic, Daniel, whenever came his turn to introduce himself in front of the neurologists, neuropsychologists, nurses, social workers who have worked with him for years, in front of this core team to which was sometimes added a resident or a visitor, Daniel inevitably repeated: “Simon Daniel, behavioral neurologist.”

What is behavioral neurology? How to describe this field to which Daniel is so proud to belong? Neurology is the discipline that studies the nervous system and particularly the brain. Why and when was the word “behavior” chosen to define a sub-specialty of neurology?

The origin of this label is, in the opinions of many (Benson; Heilman, Damasio and Boller; Boller and Daniel himself), imputable to Norman Geschwind, a famous neurologist from the East coast of the U.S. Daniel met Geschwind only once: when he was applying to a residency program with him, which he unfortunately didn’t get. But of course, he heard a lot about Geschwind. For Daniel, his main merit is that he created a “field” that he baptized “behavioral neurology” in the 1970’s. The making of this new field owes much, paradoxically maybe, to Geschwind’s curiosity for the past. His “obsessive” curiosity, Daniel once told me, got Geschwind buried in the old European neuroscientific literature:

“Like Dejerine, Alzheimer, Pick... and he sort of framed it (the disparate work of these neurologists) as a field. He took the sum of the behavioral things that

[ Footnote references are not visible in the image. ]
they did and re-taught the anatomy that people had forgotten. He just started to read the old papers, you know Geschwind read French and German, and he re-described ‘alexia without agraphia.’ He didn’t do so many new things himself, and some of the things he did were wrong, but he recognized that there was a story that had been told that had been forgotten. Is there a word for that, someone who... (I suggest: rediscover?) Yeah, you know that’s not easy to go back and to understand that these papers were significant papers that had been forgotten, this is really important, he was kind of a genius you know.”

Was Norman Geschwind indeed a genius? Did he “change the face of neurology” or was he “just a good scholar” who revived old ideas at a fortuitous time? These are questions that Frank Benson, who was one of Geschwind’s students and who was also Daniel’s mentor, asked himself51. With resort to Thomas Kuhn’s discussion of scientific revolution and paradigm change, Benson concludes that Geschwind’s work was revolutionary in the sense that he succeeded in establishing a new paradigm, but that we should not give Geschwind “sole credit for an idea discussed over a century”52.

This idea “revived” by Geschwind, which will lead to a “scientific revolution” and to the birth of a new field called behavioral neurology53, is set out in two major articles of Frank Benson’s master that were published in Brain in 1965: “Disconnection Syndromes in Animals and Man”54 (DSAM). The “idea” revived by Geschwind is “disconnection.” These pioneering and impressive articles (each is 60 pages), became increasingly important over the years: “DSAM” was cited 100 times in the 60’s, 380 times in the 70’s and 600 times in the 80’s: “once every five days between 1980 and 1985.55” The papers, and the theory of disconnection that they outline served as manifestos for what would become behavioral neurology, even though the word “behavioral neurology” does not yet appear in these papers. In the following paragraphs, I explain what this theory of disconnection is and what in this theory is considered as a rupture according to many neurologists. I then explain what I think constitutes the real change in the “paradigm shift” initiated by Geschwind and how this change framed our contemporary behavioral neurology.

1.1 Geschwind’s Disconnection theory: homecoming to Localizationism, splitting from Holism.

When reaching the end of the second part of “Disconnection Syndromes in Animals and Man,” it is quite surprising to read that Geschwind takes up the “philosophical” impact of his work. In his conclusion, Geschwind distinguishes four “philosophical implications” for his theory of disconnection. The first implication is the only one that the behavioral neurologists and

52 Ibid., p 866.
53 Ibid.
54 Norman Geschwind, “Disconnection Syndrome in Animals and Man”, Brain, 88, 237-294 (Part I) and 585-644 (Part II), 1965. I refer to this article as DSAM in the next footnotes.
amateur historians of their own discipline have remembered: the effect of the work of their master in the break with a “holist conception of man”; the regnant conception during the first half of the 20th century that – to the opinion of Daniel and many other neurologists-- negated the work of the localizationists of the 19th century.

The “principle” of the “whole man” as Geschwind calls it is, in the opinion of other neurologists (Benson, Daniel, Whitacker, Price), the principle that dominated neurology almost exclusively during the first half of the 20th century. Holism in neurology is associated with the names of Pierre Marie in France (1906), Henry Head in England (1926) and especially Kurt Goldstein in Germany, who eventually immigrated to the USA in 1935. All of these authors, writes Geschwind, stress: “The importance of thinking of the patient as a whole, seeing his responses as those of an integrated unitary structure, even in the face of damage.”

During the first half of the 20th century, neurology, under the hold of holist’s ideas, would have been dominated by a critique of the localizationist heritage from the masters of the 19th century. Geschwind explains that, as a result of his own training in this holist tradition, he was:

“Overwhelmingly skeptic towards the view that there were highly characteristic aphasic syndromes associated with different lesions in the brain. Even more forcefully I had accepted the view that any attempt at “explaining” the syndromes on the basis of anatomy was a futile endeavor.”

Geschwind was not only skeptical of the correspondence between a cerebral lesion and a type of aphasia. Holism left him “troubled” and “perplexed” by the “general rejection of the anatomical approaches”, “by the fact that people who had left their mark so indelibly in neurology, such as Wernicke, Bastian, Dejerine, Charcot, and many others, could apparently have shown what was asserted to be the sheerest naiveté and incompetence in the area of higher functions.”

The presumption of this historical injustice urged Geschwind to read the work of the localizationists of the 19th century in their original texts (Geschwind indeed spoke fluent French and German) “rather than (by) reading the interpretations of later hostile authors.”

His rereading inspired the two articles “Disconnection Syndromes in Animals and Man”. With the works of Broca, Wernicke, Liepmann and especially Dejerine on aphasia, bolstered by his own studies of aphasic patients, Geschwind reflects on the “disconnection” between different cerebral areas and the area of speech as a possible explanatory notion for different types of aphasias. Geschwind revives this notion of disconnection, already presupposed by Dejerine in 1892, expanding it with new cases and new types of aphasias. Such an extension begins to create

56 DSAM, p 637.
57 Benson, 1993.
58 Private communication.
61 DSAM, p. 637.
63 Ibid.
what Benson will later qualify as a “paradigm” (“the disconnection paradigm”). The disconnection between the cerebral “area” devoted to reading and the area of speech, explains why, for example, Dejerine’s patient was able to write but incapable of reading and even incapable of reading what he just wrote. It is the same problem for a patient who cannot identify a glass of water and a minute later picks it up and drinks from it: such a patient embodies Ryle’s separation between knowing that (retrieving facts about how a bike works) and knowing how (riding a bike). Likewise for another of Geschwind’s patients who cannot tell the color of the apple that Geschwind is holding in front of him but succeeds the test of sorting things by color. For Geschwind, all of these patients who are not otherwise aphasic are in fact not capable of literally “saying” what word they are reading, or what object they are looking at, or what color they are seeing. They cannot say it because of a disconnection between the area of speech and a specific area for “reading” or “looking at objects” or “perceiving colors”.

All these patients “fail to give a verbal account of what is going on in their visual receptive regions and yet are able to respond nonverbally to a nonverbal stimulus”, describes Geschwind. When he was caught in the holistic point of view, Geschwind couldn’t answer the question: “If he can speak normally and he knows what he is holding in his hand why can’t he tell you?” It is only when he accepted to “regard the patient as made of connected parts rather than as an indissoluble whole”, that he could consider that “that part of the patient which could speak (in the left hemisphere) was not the same part of the patient which “knew” (non verbally, in the right hemisphere) what was in the left hand.”

This cerebral explanation of aphasia leads to the “philosophical” (and anthropological) implication; true for any human being. From the disconnection theory that explains best the cases of aphasia described by Geschwind, the neurologist infers that it is necessary to put in question the term “the patient,” which is “misleading.” Geschwind fleshes out his philosophical argument with an example borrowed from urology. In front of an incontinent patient (incontinence due to a transection of the spinal chord), if the patient urinates we would say: “the patient urinated.” It “means,” however, that the patient did so “involuntarily” because we know that it is not the “patient” who actually urinated (even if we say it) but “the patient’s spinal cord (who) urinated” (even if we don’t say it). If the concept of disconnection at lower levels is acceptable, pursues Geschwind, it might well be more difficult to accept it for these “highly organized activities” that take place in the brain. We must become accustomed, Geschwind tells his reader, to “not considering animals or humans as a unit but as a union of loosely joined wholes.” It is furthermore important to acknowledge that Geschwind says this is true “even in the normal person.”

The extreme materialism of our language obliges us to embrace the idea that we are “one” and not a free union of joined wholes. But this is not all. It is worth noting that Geschwind

66 Dejerine’s patient could write (spontaneously or to dictation) but could not read what he had written. He could read tactiley though, with his fingers tracing over the outline of letters. From the autopsy, Dejerine postulated that the lesion disconnected the visual input in the preserved left visual field from the visual memory centers for words (preserved too).
68 DSAM, p. 637.
69 Vincent Descombes makes an enlightening point on the classical opposition between materialism and spiritualism. Spiritualism upholds that mental capacities must be ascribed to an immaterial part of the person: the
also says that these wholes can be emancipated from the influence of our will. This reflection goes on about the necessity to re-evaluate the idea of a unity of consciousness: are there multiple consciousesses? Or shall we attribute consciousness to the only cerebral hemisphere that “can speak”: the left hemisphere? Geschwind does not settle that question. Thus, the disconnection theory that was first used to describe certain aphasic cases is then generalized to all of us, to the normal person: we are all disconnected and moreover we are all disconnected from ourselves.

By re-reading the works of the neurologists of the 19th century, Geschwind indeed broke with holism, which was, if we believe Benson and others, the dominant paradigm at that time in the United States. Geschwind named his theory disconnectionism (instead of connectionism) and by doing so he reasserted his opposition to holism. Indeed, there could be a danger in naming a theory after the pathological cases it describes: the theory would be feeble in that it would not allow a generalization to the normal. Geschwind freed himself from this problem. He

soul. Materialism defends that all the parts of the person are material. For the materialist the subject of thought is physical, for the spiritualist it is immaterial. Vincent Descombes judges that this distinction does not reflect the actual (and the remote one as the one of the 19th century) distinction in the domain that he calls the philosophy of mind. Descombes argues that during the development of the cogito in the Meditations, the thinking subject is obviously not material; indeed the answer to the question ‘who thinks’ is spiritualist. Thus, the thinking subject is not René Descartes but an immaterial part of this individual: his soul. If to be materialist consists in reversing this thesis, a materialist would thus say that it is Descartes, his physical person, this piece of mater named Descartes, who is actually thinking. But this is not the materialist thesis. The materialist answer to the question ‘Who thinks?’ says that it is the material mind of René Descartes: his brain, who thinks. In this answer, the materialist thus defined, is willing to give an answer upon the problem of the relationship between the body and the mind, as such his position accepts the terms of the problem. Indeed he accepts to answer by the affirmative to the first question posed by dualism which is: ‘Does it make sense to distinguish the subject of physical actions from the subject of mental operations?, If both the ‘spiritualist’ and the materialist answer ‘yes’ to that question, they are not in agreement to the answer required by the second step of the questioning which tries to identify the nature of those subjects: for the former there is a dualism in term of the kind of substance involved (the body and the soul), when the materialist formulates a monism regarding the nature of the substances (but again, distinguishing the material system of the mental device, the brain, from the material system of the external behavior: the body). In La Denrée Mentale, Paris, Les Editions de Minuit, 1995, p 99-101. According to that illuminating distinction, our use of the term “patient” -- criticized by Geschwind— is, properly speaking, materialist.

70 Geschwind’s theory is not only based on a simple opposition to holism. I am stressing this aspect because it is in term of paradigm change and rupture that the neurologists-historians talk and write about Geschwind’s theory but his disconnectionism is also based on a scientific consensus. In the 1950’s, the works of some neurosurgeons already supported the idea that a trouble could be explained not by the dysfunction of cerebral centers but by the dysfunction of the connection between these centers. For instance the work of Sperry and Myers (Myers & Sperry, 1953; Myers, 1955, Sperry, Stamm, & Miner, 1956) on split brains in cats concluded that the cat brain had the capacity to act as two separate brains if their corpus callosum (that connects the two hemispheres) was split. The work on split brains is carried on in humans in the following decade. Sperry and Gazzaniga start in the 1960’s a series of experiences - that will be worth a Nobel Prize for Sperry in 1980 - on epileptic patients who had had a previous callosotomy (a section of the corpus callosum) as a treatment for their epilepsy (this procedure is still practiced today). They concluded (Gazzaniga, & Bogen, 1969, Sperry, 1970 and see Terrence Deacon’s synthesis: “Holism and Associationism in Neuropsychology: An Anatomical Synthesis” in E. Perecman (Ed.), Integrating Theory and Practice in Clinical Neuropsychology. Hillsdale, NJ: Erlbaum, 1-47) that these humans, like their cats, tend to have two independent brains and even two independent consciousesses. Aside from these works in neurosurgery, we can speculate about the influential role of cybernetics in Geschwind theories. What is at play in the aphasias described by Geschwind is a disease of communication. Thus Geschwind seems to renew the classical neurology by giving to it the scientific flavour of his time; a renewal that understands the brain as a “network”, as it is still understood today.
said that because of the influence holism had on him, it prevented him to see what was the case: that the brain of his patients was “disconnected.” He then uses “the patient” as a concept to generalize--a “patient” who is potentially any of us--on the basis that if holism was wrong about his patients and if language was misleading for any patient, they were also wrong and misleading for any human and animal.

1.2. The paradigm at work in Geschwind’s disconnectionism.

What presupposition delimits “holism” in neurology, which Geschwind is challenging? If I could circumscribe holism I would understand better what delimits the field of the opposing paradigm that will soon take over: the not yet called behavioral neurology.

Following Geschwind’s argument in the DSAM article, and in line with what neurologist-historians say (except for Cubelli71), holism’s principal presupposition is to exclude the idea that certain parts of the brain are causally involved in the trouble of a patient. The holist paradigm is defined by the impossibility of the neurologist-holist seeing a trouble in relation to a local lesion. It would thus be anti-localizationist: anti-previous paradigm. And yet, it is not at all certain that holism states that it is impossible to understand a trouble in relation to a local lesion or to a specific topography of the brain.

As Geschwind himself acknowledges elsewhere72, Kurt Goldstein discussed the question of localization up to his late writings, and “his contribution as a localizer in the classical sense is in fact highly significant although rarely taught.”73 Cubelli also, stresses the localizationist heritage in Goldstein’s theories and even generalizes its influence to the theories of Marie and Head.74 As Forest explains in his Histoire des aphasies75, Goldstein argues that the brain in its entirety is concerned by a local lesion, whatever and wherever the lesion is. The existence of the lesion is not denied. Goldstein does not refuse to distinguish functions (or functional vocations) for different regions of the brain. However the questions that Goldstein is mainly interested in are: what are the (multiple) implications of a lesion in different behavioral spheres? What are the conditions for the organism in its entirety to accomplish a function? Goldstein does not question the existence of different structures and functions in the brain, but for Goldstein the question is elsewhere: to understand the meaning of the pathology for the organism in its totality. To give one example of this meaning, Benton states that one of Goldstein’s “most valuable contributions” was to see that some symptoms could be defensive reactions that protect the patient from failure, from confusion and loss of self-esteem. Thus “apathy”, “rigidity” and “lack of concern” could be a way for the person to “cope with disabilities and avoid painful awareness of mental incompetence.”76

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72 “Evidently Goldstein did not reject his predecessors as much as many other people thought he had” in Geschwind, “The Paradoxical Position of Kurt Goldstein in the History of Aphasia”, p 223.
73 Ibid., p 224.
74 Head, for instance with his localization of four types of aphasias and his notion of scheme would come near to the notion of center (in Cubelli, 2005, p. 273)
As Forest very well sums up, Goldstein presupposes the validity of classic neurology but unlike the neurologists-localizationists of the 19th century, he examines the question of localization from “the standpoint of doing and the standpoint of meaning” and not anymore from a “standpoint of having.” Without getting into the details of the nevertheless fascinating studies of Goldstein, it might be possible to understand what “the standpoint of meaning” as opposed to the one of “having” means, through the philosophical analysis of the normal and the pathological of Georges Canguilhem.

Canguilhem actually relies a great deal on Goldstein’s works to defend his analysis of the pathological against a theory, like Broussais’, which views the difference between physiological and pathological states in exclusively quantitative terms. If the aphasis patients studied by Goldstein do present a reduction of their lexicon, it is not the only way to describe their pathology.

“One must refrain from thinking that the different attitudes possible in a sick person merely represent a kind of residue of normal behavior, what survived destruction. The attitudes which have survived in the sick person never turn up in that form in a normal subject, not even in the inferior stages of its ontogenesis or phylogenesis, as it is all too frequently admitted. Disease has given them particular forms and they cannot be understood well unless the morbid state is taken into account.”

Leaning on Goldstein’s observations, Canguilhem warns us that if disease rightly “represents a change in what remains” it is not true to think with John Hughlings Jackson that it “creates nothing”; “for the individual, disease is a new life characterized by new physiological constants and new mechanisms.” The trouble does not only amount to a gap with a norm but reveals a new norm, or a new relation with the previous norm. Thus in the aphasic’s discourse some strategies or prejudices command the choice of certain linguistic forms by the avoidance of others. Reciprocally if we take, as Canguilhem does, language as a “milieu,” the speaker becoming aphasic sees the relation he entertained with his linguistic milieu transformed in a way that is constrained by the nature of such a milieu. Thus “agrammatism” does not exist. Instead there are forms of agrammatism that are the ways in which all the languages of the world define, through their distortion, the type of relation the aphasic entertains with them.

It does seem that the rupture between Goldstein and Geschwind, between holism and the disconnection theory (or the not yet born behavioral neurology), hadn’t much to do with the acknowledgement of cerebral localizations. It seems it had to do, rather, with a standpoint on disease. For Goldstein, the relations between the disease and the individual are central. Holism sees the disease in its relationships with the individual taken in its full dimension: physiological, psychological and social; “total” said Marcel Mauss (Mauss actually saw in Henry Head’s theories the validation of his own). Geschwind takes an opposite standpoint: the use of the expression

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77 “Du point de vue du faire et du point de vue du sens et non du point de vue de l’avoir.”
79 Canguilhem, Ibid., p 188.
“the patient” is put in question. The diseased, the normal individual and the chimpanzee cannot be “one.” In the end, the disease rules, and talks for, this fragmented individual and their relationship is broken. The rupture with holism is not directed to Geschwind’s “rediscovery” of the topography of the cerebral matter but in the liberation of this topos from the patient. In that line of thought, Geschwind also writes that the patient’s introspection “may be an extremely ineffective way of obtaining information about the patient’s experience.” Look for instance at this patient, who misidentified an object (the glass of water), and later “excused himself by saying that his eyes had deceived him at first.” This might have led the neurologist to think that his patient may have had “some sensory illusions of some type.” At the occasion of another exercise the same patient will, just as incorrectly, blame his poor memory. However concerning this patient, says Geschwind, the misidentification was only a “misnaming.” What Geschwind calls the “confabulatory answers” of this patient (an “eye problem” or a “memory problem”) are thus of no help to the neurologist. Indeed, if a part of the brain is disconnected from the speech area how would it be possible for the speech area to give an account of what goes on in the disconnected part of the brain? The idea of a disconnection between two regions of the brain allows Geschwind not only to independently study the functions of these two regions but also to state that these functions are somehow freed from the individual. The disconnection between two cerebral regions assumes the disconnection with the self. It thus becomes possible to study the neurological disease without the patient.

1.3. The cycles of Behavioral Neurology: birth, thrive, and reign?

I underlined that Geschwind’s take on disease in relation to the individual actually instituted the real rupture with holism. However, we have seen that Benson (and Benson’s student, Daniel) saw the novelty of the theory somewhere else. Benson, who was one of Geschwind’s students, actually spent a great deal of time in trying to formulate what this novelty (or paradigm change) was about. He analyzed that Geschwind’s renewal of classical neurology offered a middle way between localizationism and holism. His renewal shared in localizationism, insofar as the brain is, like in the good old times of Gall’s phrenology, understood along a topography: “Each separate region carries out specific functions,” the left hemisphere knows verbally when the right knows non verbally. It partook of holism insofar as humans are made of “loosely joined wholes.” “Complex behaviors”, says Benson without telling us what he means by “complex,” are a combination of these functions. Finally, there is a specific novelty to

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81 Geschwind, DSAM, p 638.
82 Geschwind, DSAM, p 594.
84 Franz Joseph Gall established in 1810 a list of twenty-seven moral and intellectual faculties proper to man and assigned to each of those a specific cerebral localization. The map was established through ‘cranioscopy’. Gall postulated that the skull was a true representation of the brain, and through the study of the bumps of the skulls of criminals, mentally ill patients and famous men, drew the topography of these faculties. Benson actually thought “that behavioral neurology as an entity starts with Gall’s pronouncements on phrenology”. Gall’s phrenology was clearly an early personality theory and in the way it “categorizes human behaviors as independent psychological functions”, it is for Benson a method that inaugurated the field of behavioral neurology.
disconnectionism: the emphasis on the understanding of the functioning of the brain in terms of a “network.”

For Daniel, Geschwind’s talent lied in his ability to give birth to a field through his archival work. This field took the name “behavioral neurology” at the congress of the American Academy of Neurology (AAN) in 1972. This field nevertheless existed under another name before that time: the neurologists who gathered under the same banner as Geschwind (in the panels of the AAN for instance) recognized themselves under the name “neuropsychology.” The necessity to rename this banner did not concern European neurologists (Teuber, Hecaen, Lhermitte): at that time only the American neurologists felt this need. From what the neurologist François Boller told me, the American neurologists wanted to part with this term because it maintained neurology in a tradition that was too “psychological,” since it overlooked the question of cerebral localisation. To be affiliated with such a tradition seemed to have become untenable for this group of neurologists (Boller, Damasio, Heilman and Benson; all students of Geschwind, and of course Geschwind himself) who were following “Geschwind’s faith that anatomy had to play a central role.” The term “psycho” was thus abandoned and replaced by the term “behavioral”. It is however worth noting that the domain of neuropsychology didn’t disappear in the U.S.: today it gathers psychologists who have an interest in neuroscience; these neuropsychologists give neuropsychological tests to the patients at the Memory Clinic and at the Alzheimer Clinic. However neuropsychologists are not neurologists; even if their role in the diagnosis is crucial they do not make the diagnosis.

Christian Derouesné explained to me that the term behavioral was chosen because of its ability to describe what manifests outside. Unlike the unobservable that took place in the “mind,” Behavioral neurology was thus chosen because this point of view subscribes to Behaviorism. The term was also selected because in the American context of the 1970’s, one marked by the unprecedented success of psychoanalysis, this neurology was (and is) strongly opposed to the discipline that studies mental life as that which is plotting inside. Behavioral neurology couldn’t be better suited to ban the word psycho.

The term behavioral is thus first carried by a theoretical principle: the expulsion from neurological understanding of whatever can relate symptoms to a biography, to a personality, to the relations the patient entertains with his entourage in the experience of the disease, in brief to a subjectivity. Behavioral neurology wants to understand the disease without the patient. The living is treated as inert. In this respect behavioral neurology owes much to Norman Geschwind who already supported this idea in his disconnectionism theory. From this perspective, the idea that the disease can disrupt the relationships that the patient entertains with himself and the world is untenable: all the symptoms are signs of a neurological dysfunction explained by the cerebral lesions and not, as Derouesné views it, the signs of a “mental disease with a neurological dysfunction.” Christian Derouesné and one neurologist of the French clinic, Elise, are the rare neurologists I know to uphold this contrary point of view.

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85 See Boller, “An International Figure”, in Schatchter, 1997.
86 It might be different today in France: since the end of the 1990’s the domain of “neurologie comportementale” exists in France.
87 Personal communication.
88 Benton, Exploring the History of Neuropsychology, p
89 Personal communication.
Unlike Derouesné and Elise, Daniel views the patient exclusively “phenomenologically”. We will read more about it in chapter 4, but for now this is how Daniel explained to me what phenomenology is:

“What we see, just describing carefully what we see, and taking nothing for granted other than what we see... Nothing happens except the behavior, not the environment, not the family... I think the family has a certain influence but the phenomenology is stronger than the family. Behaviors so strongly reflect the brain and specific circuits... If you really capture what the behavior is I think you will understand from where it comes from in the brain. I think that’s the key, phenomenology is the key for all neurological disease.”

We might understand better what are the peculiarities of this “phenomenological” method when we will see the chief of the Memory Clinic at work. For now, I understand that phenomenology, for Daniel, is the method whose paradigm is the exclusion of whatever the behavioral neurologist decides is not ‘there’: relationships (familial or others) for instance. Thus the method recommends to focus on the “behavior”, and at the same time to forget about what one knows about the biography of the person; the focus on the (outside) phenomenon, subsequently reveals what is inside: like a scan, phenomenology uncovers the brain circuits.

On the other hand, the term behavioral does not only refer to a method in order to apprehend the patient, it refers also to specific symptoms presented by the patient, to a distinctive kind of patient. I read on the web site of the Society for Behavioral and Cognitive Neurology that symptoms exhibited by patients studied by behavioral neurologists are those of “abnormal behaviors that were associated with subcortical diseases.” Daniel says he “loves understanding how the brain influences these bad behaviors.” What counts as a “bad behavior”? Aphasia, which was Geschwind’s favorite domain of study, would not be considered today as a “bad behavior.” Neither would memory loss. Language and memory are capacities appended to the term “cognition.” “Bad behaviors” designate disorders tied to passions and affect, traditionally reserved to the “psy” sciences.

Being attentive to the phenomenon and only to the phenomenon is yet only one step into the characterization of the disease. This close attention to the phenomenon accompanied with a forgetting of whatever is not the phenomenon, seem in a way close to Husserl’s phenomenology. Yet the method here recommended by Daniel is the first step of a process, which is directed towards a cerebral explanation of the numerous phenomena that constitutes a neurodegenerative disease. Then when Daniel is looking at the phenomenon, isn’t he looking at it already with the point of view of the neurologist? If that is true, he would be looking at the phenomenon from an outsider point of view and he wouldn’t be adopting a sort of internal point of view of the phenomenon itself; a point of view that he seems to recommend. Obviously, this point needs much more discussion then what I offer in this footnote; this is only a direction for a potential discussion. Etienne Souriau has precisely criticized phenomenology (as a philosophy) David Lapoujade explains to us, because the phenomenological perspective, he says, is always the one of a consciousness and never the one of the phenomenon itself. See David Lapoujade “Etienne Souriau. Une philosophie des existences moindres” in Philosophie des possessions, Dider Debase (ed.). Monts: Les presses du réel, 2011, p 177. Thus how could we actually seize things from the inside? For Souriau, to perceive is not to observe from the exterior, it is to the contrary to “sympathise” or to “participate” in order to get the point of view of the phenomenon. (Ibid.). So does Daniel “sympathize”, (and how?) with the phenomena, e.g. with the symptoms? Only a description of Daniel at work in the making of a diagnosis can attempt an answer to this strange question, a description that I will try to give in chapter 4.

90 History of SBCN: http://www.the-sbcn.org/1743.cfm
Considering this distinctive kind of patient studied by the behavioral neurologists, I thought that Geschwind, who was mainly interested in aphasic patients, might not be thought about as the first “behavioral neurologist.” Once while I was having a conversation with Daniel, I asked the chief of the Memory clinic about the legitimacy of Geschwind’s paternity on behavioral neurology if we consider this new focus of the field on the psy sciences.

Daniel (to me). – Yeah, you’re right... Geschwind was practicing a very constrained form of behavioral neurology and all the people that followed him, I mean Antonio Damasio, Marsel Mesulam, were strictly localizationist... But I liked his student Benson... Frank wasn’t that way. I mean, Frank was not as original but Frank was fun, and he was interested in psychiatry, real psychiatry and real behavior... For Geschwind, it was all one lesion: one cognitive syndrome, and Frank was more broadly interested in the way people behave. He watched the psychiatrists describe the behavior...

Me. – But Frank wanted to localize these behaviors in the brain, in the end ... Or no?

Daniel. – Oh yes, yes he did. That’s what I loved. He was just more charismatic and fun.

When behavioral neurology seized (after psychiatry and geriatrics) the field of dementia, they found the perfect field for the cerebral knowledge of “bad” or “funny” behaviors to thrive. Neurodegenerative diseases, like Alzheimer’s or frontotemporal dementia combine cognitive and psychiatric symptoms to an indisputable biological basis. For Martin, who thinks about the Integration of Neurology, Psychiatry and Neurosciences in the 21st century92, Alzheimer’s disease defined some new frontiers. Indeed, Augusta D93., presented first to Aloïs Alzheimer with psychiatric symptoms: “She believed one intended to murder her, she was delirious, she moved objects from one place to the other94.”

Today, Alzheimer’s disease is essentially diagnosed through cognitive symptoms. Whereas Frontotemporal dementia (and Lewy Body dementia95) is mainly diagnosed through the presence of these “funny” “behavioral” symptoms --this is not only a metaphor for weird, these behaviors, as we will see, actually arouse laughter. Laughter of the team aids in making a diagnosis, there are a sign that the patient the team is speaking about has FTD; Daniel says it: “so the rule of thumb: if you hear a patient’s story and you are giggling it is usually FTD”. When Frank Benson thought about “the past, present and future” of behavioral neurology96, he asked himself if “Behavioral Neurology is either a new medical/academic discipline undergoing an explosive growth or a far

93 Augusta D. is the first patient of a long series, to be diagnosed with the not yet named “Alzheimer’s disease” by Aloïs Alzheimer.
94 Ibid., p 698.
95 Other neurodegenerative diseases than Alzheimer’s and frontotemporal dementia are diagnosed by this team of clinicians. Because they are more rarely diagnosed I don’t mention them in the main text: Amyotrophic Lateral Sclerosis, Progressive Supranuclear Palsy and Cortico Basal Degeneration are diagnosed equally on behavioral, cognitive and motor symptoms when Parkinson’s disease is essentially diagnosed on motor symptoms.
older, almost venerable, medical/academic activity that is enjoying a hearty rejuvenation.” Discipline or activity? New or venerably old? The question of the historical and institutional status of behavioral neurology will be left without an answer, Benson doesn’t conclude; what counts after all is its flourishing; either in the form of an “explosive growth” or of a “hearty rejuvenation.” Indeed, since Geschwind’s intervention at the AAN congress in 1972, the field of behavioral neurology keeps thriving. After its first separation from the psy sciences, behavioral neurology moved towards the absorption of what was guarded by psychiatry. If Alzheimer’s disease targets today memory – “the brain” stated Olivia, the neuropsychologist, in introduction to this chapter -- frontotemporal dementia allows behavioral neurologists to wonder where to localize “emotion”, “insight”, “caring”, and even “humanity” -- “the soul” -- in the topography of the brain. Thus behavioral neurology can now easily leap from dementia to autism, schizophrenia, bipolar disease and sociopathy. As Daniel will later conclude his talk, his work on frontotemporal dementia will permit the science of neurology to fill in the “great divide”:

“More and more we will think of psychiatric disorder in the way we think of frontotemporal dementia. Whether the brain circuits or the genes cause that dysfunction, if we start to frame psychiatric diseases in the way FTD has been framed, we will have a huge understanding of them. This is the beginning of a breakdown [in the distinction] between psychiatry and neurology.”

Like Daniel, many neurologists (especially in the U.S.) see this “great divide” (Price, Adams and Coyle97) between psychiatry and neurology as “artificial” and like Daniel, they wish that the “barriers” (Kandel and Squire98) or “the wall” (Baker99) might soon “breakdown” in favor of a “bridge” (Daroff100) or an “integration” (Eisenberg101, Martin102) between neurology and psychiatry.

2. “What If It’s Not Alzheimer’s?”: FTD and AD in a Paradigmatic Relationship.


In the dark amphitheater, Daniel talks about his work with this affability that seems so natural to him. I never knew Daniel other than pleasant. I never saw him edgy or irritable, his smooth voice never betrayed him. His team respects him tremendously. Nicole, a neuropsychologist, praises his “wisdom”, many of his colleagues and students find him “so intuitive” and Leticia, enthusiastic neurologist visiting from Italy, didn’t hesitate one second to call him “a genius”. Respect sometimes turns into fear; Michael, who is sitting next to me, is regularly worried of being rejected by “the man” who is, for him, a “kingmaker”. During his talk, Daniel is careful to sprinkle the presentation of his work with the names of all of his collaborators. Michael’s name hasn’t been mentioned yet; I know Michael is eagerly waiting. Daniel’s talk takes us back to the beginning of the 1970’s in the United States. Daniel, back then, was a young resident and had just picked out neurology as a medical specialty. Daniel remembers these years as the “dark ages of dementia”. Before 1970 he says, there were two rare neurodegenerative diseases “that I was told I would never see: Alzheimer’s disease and Pick’s disease”. Both were surrounded by the same marginal attention in the medical world. But quite suddenly, in the middle of the 70’s, everybody focused on Alzheimer’s disease. Not only at the expenses of Pick’s disease, as Daniel underlines, but quite simply the interest for Alzheimer’s grew in the field of neurological sciences and managed to emerge as a topic, a term, and problem of public and medical concern. The history of this sudden enthusiasm for Alzheimer’s disease, a disease that was previously limited to a few specialists and totally ignored by the lay public, is due, Daniel tells us, to two papers. The first is a “real scientific paper”: in a nursing home in England, it was shown that the brains of old people who died of dementia were affected by the pathology described by Aloïs Alzheimer in 1911: the amyloid plaques. The other paper is a “sales paper” from one of Daniel’s friends: Bob Katzman. In 1976, Robert Katzman established in a short editorial that

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104 The paradigmatic (or “associative” for Saussure) relationship between elements of language involves processes of selection and substitution: in building a sentence it thus involves choosing a sign among others in the code with which it entertains relationships of similarity: from synonymy to antinomy. The relation between Alzheimer’s disease and frontotemporal dementia is antonymous.


106 Jesse Ballanger, *Self, Senility and Alzheimer’s disease in Modern America: A History*, John Hopkins University Press, 2006. In his historiography of Alzheimer’s, Ballanger writes that the publication of studies led by Martin Roth and co. in Newcastle (UK) correlated the number of neuritic plaques in patient’s brain to the score to cognitive test obtained by those patients.


Alzheimer’s disease was the 4th or 5th leading cause of death in the United States and he speculated about the enormous financial loss that awaited the Americans if nothing was done. Daniel, in his version of the story, insists that Katzman, “in order to get more money”, needed to “have a name” for this disease. And this name was Alzheimer’s and “almost instantaneously all dementia was Alzheimer’s”. Katzman’s call was heard: his resounding article launched an exponential growth of the budgets allocated to Alzheimer’s research within a few years and mobilized the media who made this disease the major public health problem of which we are all now aware. As for Pick’s disease, it fell in the limbos of neurology, the students passing on to each other this “old myth” of a bad omen: “Don’t pick Pick’s”. “Don’t pick it because it is so rare... And a very simplified version of Alzheimer’s emerged that required memory as a primary symptom”, continues Daniel.

Yet, dementia is not reducible to a memory problem, neither to the pathology revealed by Alois Alzheimer: the amyloid plaques. Few neurologists kept that intellectual doubt alive and one of these “very few people who knew better” was Daniel’s mentor, Frank Benson:

“Frank, could tell me: this patient has Pick’s disease and that patient has Alzheimer’s disease”, at a time when no one believed you could diagnose Pick’s disease.”

In his history of Pick’s disease, Derouesné is in agreement with Daniel: Pick’s disease was unknown to the American neurologists in the 70’s and even in the 80’s. Frank Benson was maybe one of the only neurologists at that time in the United States to differentiate clinically Pick’s from Alzheimer’s. For Derouesné, it is not before the 1990’s, that American neurologists began to make the distinction between these two diseases. Their late interest is surprising says Derouesné, because some French psychiatrists, followed by some Swiss neurologists, some Swedish and finally some British, had all written about Pick’s before the 1990’s: how could the Americans had ignored their research? If it is understandable that the articles published by the French psychiatrists in French as soon as 1958, were not read by the Americans, how to understand their indifference towards the Swedish’s articles published in English? Derouesné suggests an explanation: the late interest of the American neurologists in FTD is due to the “traditional independence of the psychiatric American culture compared to the European culture”. As if the troubles experienced by the patients now classified FTD and taken care of by the neurologists, were previously monopolized by some American psychiatrists reticent to recognize in these troubles the causal effect of a neurodegenerative disease, readily identifying them, as Daniel often complains, with schizophrenia or depression.


112 Derouesné’s explanation could be valid: I have heard Daniel complaining many times about the work and mistakes of American psychiatrists confined in their obscurantist bubble, ignorant of the sciences of the brain. Yet, I have heard the same critic from Pierre, a French neurologist. So I am not too sure about the differences Derouesné is underlying between so-called “European culture” and the “American culture”. How to explain otherwise the delayed interest in FTD from the Americans? Differently from Derouesné, I would speculate on the very strong interest of
Be that as it may, it is to the Swedish group of Lund that Pick’s disease owed its metamorphosis into “Fronto Lobe Dementia”. Gustafson et alia proposed this term in 1975 in order to differentiate a frontal dementia histologically different from Pick’s disease. The Lund group then joined a research group from Manchester in 1994 and they defined the first clinical and neuropathological criteria of what is still today called Frontotemporal dementia. Under this umbrella term, they described three clinical forms. These forms slightly changed names between 1994 and today. In 2014, they are: (1) the behavioral variant, it is the most frequent (two third of the patients); (2) the semantic variant and (3) the non-fluent variant. The last two variants affect language. The first one affects behaviors. The behavioral variant (bv-FTD) is the one whose symptomatology is really new; it is also the one that fascinates Daniel. Bv-FTD affects predominantly the frontal lobe. The non-fluent variant and the semantic variant affect the frontal and temporal lobes respectively, their symptomatologies are not exactly new: their symptoms resemble the aphasias described by Broca in 1861 and by Wernicke in 1871.

Histologically, what was true for Pick’s disease is still true for FTD: the lesions are different from the ones observed for Alzheimer’s disease. As Derouesné summarizes: FTD--like Pick’s disease before it--is defined as *what it is not*: Alzheimer’s. What it is, though, is more difficult to say.

Histologically, it is a very complex nomenclature that keeps changing almost every year. Today there are three big groups of histological lesions: the first one is described by an abnormal accumulation of the tau protein (taupathies)--Pick’s disease that is defined by the presence of Pick’s inclusions pertains to that group, the second one is described by intraneuronal inclusions with ubiquitin protein, and the third group is described by a frontotemporal degeneration without any specific anomalies, neither of the tau nor of the ubiquitin proteins. There is no correspondence between the “phenotype” (the symptoms) and the histopathology. And the Americans neurologists in Alzheimer’s disease, possibly obscuring their view of other kind of dementia. This explanation could be supported by Daniel who always complained that Alzheimer’s diagnosis is overly given at the expense of FTD diagnosis. Daniel actually translated the articles of Brion, Delay and Escourolle mentioned in the previous footnote and uses it as the basis for a critic of the American myopia at FTD when the French were already in 1958 discerning the differences between the two diseases. The seduction exercised by Alzheimer’s disease in the United States, at the expense of any other kind of dementia (not only FTD) is formulated here in a quite polemical way by a French doctor: « La maladie d’Alzheimer, vaste synthèse des Américains pieusement suivie par quelques dévots, est une mystification, une blague, peut-être une erreur. Elle englobe tout. Il n’y a plus de démence sénile, de démence alcoolique, épileptique, etc., de démences vésaniques [survenant au terme d’une psychose] répertoriées pendant des demi-siècles par d’éminents clinicians. On distinguait autrefois parmi ces diverses démences deux formes plus rares, la maladie d’Alzheimer et la maladie de Pick. Elles se distinguiaient par des signes cliniques, la forme évolutive et l’anatomopathologie […]. Notre Société devrait consacrer une séance à la maladie d’Alzheimer où l’on pourrait rétablir et même magnifier les acquisitions de la clinique passée. » quoted by Fabrice Gzil , in “Problèmes philosophiques soulevés par la maladie d’Alzheimer”, PhD dissertation, Université Paris I 2007, p. 9.

113 In the *motor aphasia* described by Paul Broca, the expressive function is lost resulting in an inability to speak, however the patient understands the meaning of speech. This aphasia is very similar to what one of the language presentation of the frontotemporal dementias called the “fluent variant”.

114 In the *sensory aphasia* or word-deafness described by Carl Wernicke, the expressive functions are perfect; the patient can speak, read and write but cannot understand the sense of what he says, read, etc. This aphasia presents very much like the second language type of frontotemporal dementia called the “semantic variant”.

situation is even more complex when considering the genetic forms of FTD that were discovered these last years.

Clinically, the chief of the Memory clinic defines FTD behavioral variant in opposition to Alzheimer’s disease. Alzheimer’s patients, he says, “have an early and profound difficulty in retaining new information”, they have “spatial difficulties”, whereas “memory loss is less prominent in most patients with FTD” and “spatial difficulties are rare”. Alzheimer’s patients “tend to maintain socially appropriate behavior”, whereas FTD patients do not “preserve their social graces”. As AD progresses, it is well possible that patients “act inappropriately in financial and other situations requiring judgment”, but “this is primarily related to their cognitive problems rather than to lack of concern for social norms”. If Alzheimer causes apathy it is because of the situation: “apathy is likely to occur in situations that are confusing or overwhelming”, whereas apathy for FTD patients is constitutional: it “is more pervasive” and “more often reflects an indifference about others”. In short, “cognitive” problems could summarize the whole AD symptomatology, whereas affective troubles occupy the foreground of FTD symptoms. This division is grossly inscribed in the topography of the brain: Alzheimer’s affects the back of the brain and FTD, the front.

2.2. Dementia now: the youth, American football and war.

Daniel, 30 years after taking up the torch lit by Frank Benson, preaches to his followers of the Memory Clinic that “frontotemporal dementia is a major common disorder”.

First, because “if you are young, frontotemporal dementia is not rare”: patients under 65 years old become demented from AD as much as from FTD; there would even be more patients under 60 years-old affected by FTD than by AD. Besides, autopsies have shown that 50% to 60% of the brains of people diagnosed with AD show an overload in TDP-43 protein (a component of ubiquitin). And, Daniel goes on, TDP-43 is “one of the neuropathological hallmarks of frontotemporal dementia”: the question of a misdiagnosis of more than half of the Alzheimer's patients is open.

After having pointed to the possibility of a wide scale epidemic of this as yet little known form of dementia, Daniel goes on to broaden the scope of his socio-medical diagnosis even further: to violence on the football grounds and on the war fronts in Iraq and Afghanistan. In the last two years, Daniel often talks about NFL football players and war veterans because of “the relationship between head trauma and the two major proteins found in the brain of frontotemporal dementia patients: Tau and TDP-43”. Daniel’s observation about the possible relationships between football, war and FTD is however exclusively limited to the talks that he gives; none of the 700 hundred peer-reviewed scientific articles that he has (co-) authored speculate about these relationships. His interest for the subject seems to owe much to his encounter with the neuropathologist Bennet Omalu who was invited in 2010 to present his work at the weekly grand rounds of the Memory Clinic. That Friday morning, on the big screen opened out on the wall, the forensic pathologist shows us close-up shots of the faces of the football players stretched down on his table. Omalu indeed said in an interview to PBS that he

always looks at the face of the dead before he’ll open their skull. We watch their swollen eyes, their distended faces and their strong and large shoulders. In the brains of these football players, Omalu identified a new cerebral disease that he baptized Chronic Traumatic Encephalopathy (CTE). His first case was Mike Webster, a football player who died of a heart attack when he was 50, and “a drug addict suffering from severe depression, living like a vagabond, having lost all his money”. After Mike, Omalu carried out an autopsy of the brains of the players Andre Waters and Terry Long117 – both of whom had committed suicide, Terry Long by drinking anti-freeze. Omalu also investigated the bodies of Justin Strzelczyk who died from a collision with a truck when he was driving against the flow of traffic at 90 miles an hour, and Tom McHale who died of overdose. In their brains, Omalu found a tauopathy: the tau protein aggregates creating neurofibrillary tangles with a specific topography and an abundance that doesn’t look like Alzheimer’s disease. And even if these players had memory problems, their other symptoms wouldn’t make one think about Alzheimer’s: depression, addiction to drugs for some of the players, “being a bad person” and the inability “to maintain meaningful relationships with those whom he loved” would make one think more about FTD. This is why that morning, Omalu, in a loud but unsteady voice, claimed to the audience of the Memory Clinic that: “CTE is hot”. Indeed CTE seems highly significant for the clinic specialized in a dementia whose symptoms and pathology, whose “theme” says Daniel, “is definitely frontotemporal”.

At the end of his talk, this native from Nigeria shared with us his fatigue from academia. He sees himself as a victim à la Semmelweis120: while Omalu works for truth that “shall get you free”, his science also questions “the American way of life”. This attack was found intolerable by the NFL and MTBI committee, they riposted asking that his first paper on Mike be recalled accusing him of fraud and they “insinuated that I was not practicing medicine, but that I was practicing voodoo”. During the PBS interview, Omalu is in fact a strong critic of football’s violence, but there is more to that than the simple expression of an opinion. Omalu’s way to present the cerebral disease that he discovered in these “victims of football” links, in a vicious circle, the internal and cerebral to the external and social. “CTE also damages the part of your brain that is responsible for your emotions, for your mood. Every domain of human functioning is compromised by continuing, repeated insults, physical insults. And NFL players will tell you that they know that because they inflict injury and cause harm to other players. They will tell you that.” The physical violence of American football provokes cerebral damages; what is hit though by CTE is this part of the brain responsible for emotions. Now, the insensitivity and the coldness

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118 The wife of Mike Webster said to Dr Omalu.


120 Ignaz Semmelweis, a Hungarian obstetrician born in 1818, demonstrated that puerperal fewer was infectious and contagious and could drastically be reduced by appropriate washing of the hands. He made this discovery after realizing that interns in obstetrics who just had performed autopsies on cadavers would directly go deliver babies without washing their hands. The simple treatment that he proposed, washing hands, was yet in conflict with the scientific opinions of his time; he was dismissed from his hospital and ostracized from the medical community. Semmelweis battled all his life to make the truth shine, but failed and died mad (interned in an asylum) of his failure. His story and the reasons of his failure are beautifully told by Louis Ferdinand Céline, *Semmelweis*, Paris, Gallimard, 2009.
that spread into their brains freed the violence from the football players: the violence of the “American way of life” neurologically reproduces itself.

If Daniel’s cautiousness maybe prevents him from publishing on such a political subject, he communicates more and more on CTE. The recent suicides of the players Ray Easterling, Dave Duerson et Junior Seau who shot himself in the heart “so science could study his brain” summarizes Daniel, might have something to do with it; maybe Omalu is right that “all of the NFL players have CTE”. To these football’s victims, continues Daniel, one must add war’s victims: “these NFL football players and war veterans suffer a progressive neurodegenerative disease called Chronic Traumatic Encephalopathy; it is very very similar to FTD and we see the Tau protein aggregating in the areas of the brain that play a role in the modulation of fear, anxiety, inhibition and drive.” Omalu, actually carried out an autopsy on a veteran of 18 years old, previously diagnosed with Post Traumatic Stress Disorder (PTSD): he found the same cerebral anomalies as for the football players. Thus, Daniel and Omalu are not only talking about victims of head trauma, rather of trauma victims tout court. Daniels asks: “with more than 21000 people coming back from Afghanistan and Iraq with mild traumatic brain injury: how common are these tau conditions going to be?” Extending the cases to PTSD, he wonders: “and these PTSDs at the Veteran’s Hospital progressively getting worse, I mean, is that a neurodegenerative disease? I think this is an incredibly important question for psychiatry…” And Daniel concludes on the relationship between dementia and war: “This is another story so strongly linked to FTD that it has became hard to think of FTD without thinking of this military crisis that we are facing as well.”

2.3. Social norms and values at work in the knowledge upon frontotemporal dementia.

From Daniel’s presentation, we have learned that frontotemporal dementia affects youth, whereas Alzheimer’s disease (at least in general) combines with senility. The widening of FTD’s targets to war veterans and NFL football players, pairs this disease with men whereas Alzheimer’s disease was at its birth at least, paired with women. More significantly in Daniel’s talk, FTD is married to violence.

The relationship between FTD and violence was already questioned in an article published by Daniel in 1997. This article studied 22 patients and compared the “violent, socially disruptive and antisocial behavior” of FTD patients in contrast to the “socially pleasant” person with AD (Alzheimer’s disease). The article supports the hypothesis that the “aberrant behaviors” observed in FTD patients, are caused by a “common neuroanatomical substrate”: the frontal and anterior temporal lobes dysfunction “spared in early AD”. The article ends on the legal reach of this observation: in the context of the “explosion of knowledge regarding the influence of the brain upon behavior”, could we establish a causal relation between crime (in general, not only (petty) crimes committed by people with FTD) and a cerebral area? With some recent work showing a

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121 Daniel actually mixes up Dave Duerson and Junior Seau, both shot themselves in the heart but only Dave left a note (a text message) saying that he wanted to leave his brain to science (though he didn’t specify that he aimed at the heart to save the brain), Junior didn’t leave any suicidal note.

“strong anatomical basis for murder” correlating with marked frontal (64.5%) and temporal deficit (29%)”, what can the study of FTD behaviors and cerebral deficit teach us about criminality and the brain? The authors end on a speculative note: “Is it possible that the relationship between basal-frontal dysfunction and impulsive crime is more widespread and significant than has been considered? What is the association of theft, violence and aberrant sexual behaviors with temporal lesions?”

In this article, only one FTD case is described at full length: compared to the other patients this man gathers the maximum “criminal” behaviors. He stole (like five other FTD patients): “After being placed on a diet, he was caught stealing chocolate at a local convenient store.” He had an “indecent exposure” (like one other FTD patient): “He masturbated watching pornography in his living room with the curtains open visible to neighbors.” He made sexual comments/advances (like four others): “He stared at women and children and was sometimes reprimanded for his behavior.” He committed one physical assault (like four others): “One day he hit his wife.” He was involved in a hit and run accident (like two others): “He became careless in driving and ran red lights.” What he didn’t do that one person diagnosed with FTD did, is displaying “unethical job conducts”, and he did not “urinate publicly” (unlike one other person).

The goal of the article is first to show that FTD patients display criminal behaviors and that Alzheimer’s patients don’t. The reflection does not stay confined to the comparison between the symptomatology of these two diseases. It also aims to broaden out to crimes like “murder” and their possible anatomical basis. However, from stealing chocolate, staring at women and even hitting (with no other precision) one’s wife, there is a stretch to an act of murder. Yet, from the descriptions of this man (he “stared at children”, “pornography”), from the crimes qualifications (“indecent exposure”, “sexual advance”, etc.) that seem to come out from a police’s report, from its conclusions about the possibility of “localizing” criminal behavior in the brain, from this whole article I get a strange impression of discomfort.

I had a comparable impression from Daniel’s speech that day he was honored for his career. An impression that was perhaps due to the images of violence that permeated his speech: the violence for instance, done to these football players crushed by the American way of life. The image of Justin: one day a famous player, and the next descending into dementia, until his last night: driving frantically against traffic on the highway. We will see his face one last time framed by the camera of the neuro-pathologist who is about to open his skull. It is also the racist violence towards Omalu the pathologist, who struggles against the multi-billion dollar institution to make the truth come out from the brains of the players lying on his table. It is the image of the veterans, alcoholics and insomniacs, who battled for America but who, when back to their country, found the same destiny as some FTD gene carriers described by Daniel: “They start to show addictive behaviors, they start to become outliers in families... they become black sheep”. And it is the violence that FTD patients, football players and veterans inflict to themselves and back to others. I find the intensity in Daniel’s discourse disconcerting. None of the clinicians of the French clinic would have dared to make such demagogic relationships between war, violence and neurological diseases.

There is in Daniel’s speech a good amount of fantasy because Daniel seeks this atmosphere of violence more in images than in scientific facts: when Daniel talks about the relationships between FTD, CTE and PTSD he begins to ask cautiously: “How common are these tau
conditions going to be?” Yet, his prudence becomes quickly unnecessary since he will affirm in the end that war is “so strongly linked to FTD”. Daniel produces, in his discourse and in his article, “the” sensational (effect). With the enlargement of the FTD field to the war and football fields, with the identification of FTD “crimes” with murder, the quantity of the tau victims increases drastically; as if the coldness that took over their brain could spread through the whole society. As if coldness and indifference, like Catherine Malabou –inspired by the work of the neurologist Antonio Damasio– argues, were a new pattern of our contemporary society.

“The” sensational effect is not limited to making FTD appear as a new epidemic and its symptomatology appear like a new mentality. The images of war, of the American football field, etc., which are all used by Daniel in his talk, tend to create an “essence” of FTD, and its essence is violence. The same effect, as well as affect, are apparent in the article: the rather imprecisely qualified acts that transformed the person discussed in the article into an FTD sufferer, are quickly absorbed by a police language and by the confusion of petty crimes (robbing chocolate) with murders. Because Daniel invokes the images of war and murder, Daniel overlooks the precise acts that created this “identity” of FTD. He works to create a community of “tau victims” who are defined by violence. Quite significantly, these veterans, NFL players, FTD sufferers (and murderers) are not only victims of (extreme) violence but are also, of course, its perpetrators. The work of production of this essence, from the slapdash classification of these persons, makes the FTD carriers appear as “inhumane”, “uncaring” and “sociopaths”; words that define them also (see next chapter). As we will see in the process of the diagnosis, this essence that defines FTD sufferers can function backwards: it is not the acts that create the essence, but the reverse: the actions of FTD bearers will be founded on this preliminary essence. What I find discomforting is how, through Daniel’s talk and article, surreptitiously, FTD sufferers have been withdrawn value.

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In this chapter, I situated Daniel’s science, behavioral neurology, in relation to the person, the patient, as well as to the field of neuroscience and even more broadly to the field of mental diseases.

From the work of neurologist Norman Geschwind, founder of behavioral neurology, we have understood that we “are” a brain, undermining the relational aspect of our lives. These neuroscientists only accept the materiality of our life. This is not the only possible interpretation though: as Janelle Taylor or Antoine Hennion and Pierre Vidal-Naquet have shown, even in dementia our brain is still related to other brains. Yet, in the understanding of these neuroscientists our cerebral life implies our relational (and moral) death. This is a legitimate interpretation: isn’t that true that this point of view–life under the sign of death—is a scientific

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123 Catherine Malabou’s argument in Les Nouveaux Blessés: de Freud à la neurologie, penser les traumatismes contemporains. Paris : Bayard, 2007, is based on the work of the neurobiologist Antonio Damasio who has worked extensively on the frontal lobes and the emotions.


125 Antoine Hennion et Pierre Vidal-Naquet, Une ethnographie de la relation d’aide : de la ruse à la fiction, ou comment concilier protection et autonomie. Treize récits de cas, Rapport de recherche pour la MIRé (Drees), 2012.
necessity (see Foucault’s “Open Up A Few Corpses”) that will make it possible to face and at the same time to legitimately treat these cerebral diseases?

We then read about the diseases that constitute the focus of behavioral neurology and that are gathered under the terrifying term ‘dementia’. Daniel’s talk showed us two faces that dementia could take: Alzheimer’s disease and frontotemporal dementia. We’ve notice how the later, Daniel’s specialty, was understood in opposition to the former. We’ve learned also how dementia (particularly FTD and to a lesser degree AD) is a cerebral condition whose clinical symptomatology could easily constitute the bridge between neurology and psychiatry, wished for by the supporters of the “strong program” in neuroscience, as Alain Ehrenberg has called it.

Ehrenberg has distinguished two kinds of “programs” in neurosciences: a “weak” one, [le programme faible] aims at, he writes: “Progressing in the treatment of neurological diseases (Alzheimer’s and Parkinson’s) and at discovering the neuropathological aspects of mental diseases, like schizophrenia. The “strong program” [le programme fort] identifies philosophically speaking, knowledge of the brain and knowledge of oneself, and believes it can fusion psychiatry with neurology”. The strong program will “in fine treat psychopathologies in neuropathological terms” and will construct a “neurobiologisation of personhood”.

Undoubtedly, Daniel as the honest heir of Norman Geschwind is “philosophically speaking” a partisan of the strong program. Yet, when we learn about the work of Daniel on FTD, we learn that the fusion between psychiatry and neurology needs not only to be considered as the movement of psychiatry towards neurology, as Ehrenberg considered it in his article. In this fusion what is at stake is not only to find the biological causes (through molecular biology and cerebral imaging) of autism and schizophrenia. The movement, which we saw in Daniel’s treatment of FTD’s symptomatology, goes also from neurology towards psychiatry. Daniel told us that FTD patients are as socially impaired (particularly because of their violence) as PTSD sufferers or schizophrenic patients are. It is also from the psychopathology of frontotemporal dementia that the “strong program” in neuroscience, which aims at a fusion between neurology with psychiatry, might have a chance to come into being. The strong program thus also starts with the peaceful work of neuroscientists on the “treatment of Alzheimer’s disease”, a program that appeared merely ‘weak’ to Ehrenberg. This is why neuroscience is not exterior to “mental health” as Ehrenberg writes, not only because it constitutes its “scientific and technological tip” but also because it is already essentially mental.

Some anthropologists such as Alain Ehrenberg, Sherill Mulhern and Allan Young have chosen troubles recognized with certainty as psychiatric (depression, personality disorder and

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128 Ibid. My translation.
129 Ibid. My translation.
130 This “philosophical stand” is enough in my opinion to make one neurologist pertain to one program or to the other. Elise, the French neurologist, I believe, is more a supporter of the weak program because Elise is, as Oliver Sacks that Ehrenberg cites in example (ibid. p144), a neurologist who is attentive to the psychological troubles of her (neurological) patients.
131 Ehrenberg, Le sujet cérébral, p147.
post traumatic stress disorder) to fruitfully study the relationships between the “mental, the cerebral and the social¹³⁵”. As we have noticed, neurological troubles --even if presented from the outset as biological troubles-- can also be an interesting and appropriate entry point.

Much more needs to be said about this relation between the social and the cerebral. This is the object of the next chapter titled “the social brain”. Again, here I reverse the path taken by Ehrenberg. The inquiry doesn’t question how the “strong program left the labs” and by which means “a naturalist language permeates our society¹³⁶” but rather how the social entered our brain.

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¹³⁵ Ehrenberg, Le sujet cérébral, p 130.
¹³⁶ Ibid. p 145.
CHAPTER TWO

THE SOCIAL BRAIN

“On peut tomber amoureux d’un schizophrène
Pas d’un DFT”

“You can fall in love with a schizophrenic
Not with a FTD”

Adrien, a psychologist at the Alzheimer’s clinic\textsuperscript{137}.

On the stage of the amphitheater, behind the microphone, Daniel now gets to the core of his talk: what frontotemporal dementia (and particularly its behavioral variant\textsuperscript{138}) taught him about human nature.

Frontotemporal behavior variant hits more the right side than the left, and the so-called non eloquent right hemisphere is very eloquent, it is just not eloquent for language, it is socially eloquent.

\textsuperscript{137} Adrien’s remark was made while the French team was discussing the case of a woman who had been diagnosed with schizophrenia many years ago. Recently though, the woman presented with different symptoms than before and the team was inquiring whether FTD was not adding to the old schizophrenia (this will, in the end, be their conclusion). The case was complex: some symptoms were very much in favor of FTD and others not at all: the team thus wondered if the new symptoms weren’t merely an aggravation of her schizophrenia, or to the contrary, if the woman would have been misdiagnosed with schizophrenia at first and instead had developed FTD over all these years. Without entering into the details of this case, I need to add that the husband of this woman was very much in love with his wife. Adrien’s remarked that this woman couldn’t possibly have been misdiagnosed with schizophrenia at first: if it were the case her husband would never had loved her (even though he still loved her and this didn’t prevent the team to diagnose her with current FTD).

\textsuperscript{138} We’ll remember of the three types of frontotemporal dementia: the behavioral variant which hits specifically the frontal lobe and our social abilities, and two others: the semantic and the non fluent variants which hit primarily language; semantic variant hits the temporal lobe and the non-fluent variant hits the frontal lobe.
If we know since Broca and Wernicke that “language” is “in” the left frontal and temporal lobes, we learn today that the “social” is “in” the right frontal lobe: “And when you lose this right frontal lobe you lose this unbelievably socially eloquent hemisphere”, Daniel explains. The mapping of the social brain is recent, according to Daniel: “When I started in this area, people didn’t have the foggiest idea about what the frontal and anterior temporal lobes did.” Thanks to the stimulating environment provided by R.--which made Daniel feel that he “was in medical school every day”--, he could clear up some of the mysteries of the frontal lobes: “The story of the frontal lobes is a R. story”, Daniel recapitulates.

The story of the frontal lobes is not only the story of Daniel at R. It started before Daniel came to R., even before he intended to do medicine. If we follow Benton, this history has two stages: before 1950 it was, what he calls, the “early story”; it was only after 1950 that the history of the frontal lobes properly started: “The advances in understanding, since 1950 (and particularly since 1970), are so radical and far reaching as to justify the designation of developments (upon the prefrontal region) up to 1950 as ‘early history’”, writes Benton.

The early history starts in 1848 according to Benton with the “case” of Phineas Gage. Since the story of this man came to crucially influence our contemporary neurosciences, especially Daniel’s and Damasio’s views on the frontal lobe, it seems worth the detour. After this detour and before getting into the ways Daniel characterizes our social and cerebral fragility, I will stop by some of the stages of these “advances in understanding” about the frontal lobe. By doing so, I do not aim to provide a detailed analysis of the literature on the frontal lobes, neither its history from 1848 up to today. Rather, with some examples taken from American and French scientific literature, I will try to grasp how some scientists with different methods, different descriptive terms and different explanatory concepts understood the frontal lobes. The choice of these scientists is mainly governed by the influence they seem to have on the views held today at the Memory clinic and at the Alzheimer clinic. I say they “seem”, because in the two clinics if I have heard at many occasions their names mentioned in conversations, no one clearly claimed to adhere to what these scientists wrote. In this brief inquiry, I thus aim to draw a sort of chart of the ways in which some neurologists, neurosurgeons and psychiatrists understand the function of the frontal lobes and how these ways influence one another until today. I will focus especially on the place of an idea that is well accepted today: that the frontal lobes authorize our social role. How, from 1848 until today, have scientists understood the attribution to the frontal lobes of our capacity to play our social role? When does a notion of the “social” appear in these writings? And how to understand what it means? If the word “social” per se does not appear, is there a close notion of what it means today, and what it did mean back then? An articulated theory of the social brain doesn’t appear before 1984 with François Lhermitte, a neurologist who influenced the view held at the Alzheimer clinic in France. I will end this first part by comparing Lhermitte’s theory about the frontal lobe and our sociality with Antonio Damasio’s theory (1994) that itself, greatly influences the views held at the Memory Clinic, and I will ask: how Lhermitte and Damasio study this social function? And, what theory do they propose to explain the social rupture caused by the pathology of the frontal lobes?

140 Ibid.
In the second part of this chapter, I will detail the ways in which Daniel exposes his understanding of the frontal lobe: first at the occasion of his talk --the one we have been following since the beginning of the first chapter--, and second at the occasion of the visit of a philosopher at the Memory clinic.


1.1 “Gage was no longer Gage.”(1848).

When Paul Broca\(^{141}\), in 1861, reflected on his discovery of language in the left frontal lobe, he was then arranging capacities situated in the region of the frontal lobes (judgment, reflection, comparison and abstraction) into a high-ranked category in contrast to the other lobes (occipital, parietal and temporal), to which would be designated the low-ranked capacities: passions and instincts. Broca never thought about the social as a capacity and even less about the possibility to localize it in the brain. According to a number of contemporary neuroscientists (Damasio\(^{142}\), Boller\(^{143}\), Dimitrov et al.\(^{144}\) however, the history of the frontal lobes starts around 1848: the “social” had already been spotted in the frontal lobe during Broca’s time. However, the event that dates the beginning of this history went unnoticed at that time. It is only today while neuroscientists are driven by a new interest for localizing the “social” in our brain, that they rediscover the testimonies of John Martyn Harlow\(^{145}\) concerning Phineas Gage; the emblematic case of the man who lost a part of his frontal lobes.

During the summer of 1848, after an explosion on the construction site of a railroad in New England, a tamping iron (named as such because it was used to tamp the powder before lighting up the fuse) ran through the head of the foreman called Phineas Gage. Not only did Phineas Gage survive the crowbar passing completely through his head, he was furthermore able to walk right after the accident. The reverend Joseph Freeman saw him arrive at the house where he would convalesce for three months: “in a cart, sitting up without aid, with his back against the fore broad. When we reach the quarters, he rose on his feet without aid, and walked quickly, though with an unsteady step, to the hind end of the cart, when two of his men came forward\(^{145}\).


and aided him out, and walked with him, supporting him to the house. An hour later, Dr. Harlow arrives at the house and met Gage for the first time, “his person and the bed on which he was laid, were literally one gore of blood.” He examines the wound and describes what seems to have been the trajectory of the iron tamping in his skull: it would have entered his face “under the left zygomatic bone, fracturing the floor of the orbit of the left eye”, it would have continued its trajectory upward and backward “fracturing the parietal and frontal bones extensively, breaking up considerable portion of the brain”, before emerging at the top of the skull and landing about 20 yards behind Gage. Harlow manages to stem the profuse hemorrhage and removes spiculae of bone that had taken “a portion of the brain which hung by a pedicle”. He closes the wound: “the larger pieces of bone replaced on the lacerated scalp was brought together as nearly as possible and retained by adhesive straps”. Harlow struggled against the infection of the wound for three months and Gage miraculously survived and lived for 12 years. But what were the consequences of this accident for Gage’s mind?

To this haunting question, the reports of that time give little answers. The details of Gage’s story were given only by four primary sources: by Gage’s doctor, Harlow, on two occasions: in 1848 right after the accident and then in 1868; by the surgeon Henry Bigelow in 1850; as well as by Jackson in 1849 and 1870.

Harlow’s first report mainly describes the physical impact of the accident on Gage but gives rare elements on its impact on Gage’s psyche. Harlow describes Gage before the accident as “a great favorite with his men” who had “temperate habits” and a “considerable energy of character”. Immediately after the accident, his “memory was perfect as ever” but Gage was “capricious and childish”, getting “uncontrollable”, for instance, in “his desire to return home”. When Harlow showed him money, he estimated it “inaccurately”. But when Gage was physically able to get to the store, he used money accurately to purchase things.

Jackson in 1849, found him “well in mind but memory seemed somewhat impaired.” His remark does not fit with Harlow’s previous observations of Gage’s memory. Bigelow in his report in 1850 writes that Gage “quite recovered in faculties of body and mind”. Bigelow’s observation does not fit with Harlow’s subsequent report.

Harlow’s second report does not come out before 1868. Gage had already died eight years before. We learn that after the accident, Gage didn’t get his job back as a foreman. Instead, he became an attraction at Barnum’s American museum in New York, and traveled to lecture and to exhibit himself. He then worked for 18 months in a livery stable before leaving for Chile where he

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149 Harlow, John Martyn (1868). "Recovery from the Passage of an Iron Bar through the Head", in Macmillan, An Odd Kind of Fame.
worked as a driver of stagecoaches. He stayed there until 1859, and then returned to his family in San Francisco where he died of seizures in 1860.

Harlow’s 1868 report is different from the one of 1848: Harlow describes the details of Gage’s personality and with the professional language of a psychologist. Before the accident, Gage is now described with a “nervo-bilious temperament” with an “iron will” and a “well-balanced mind”, “energetic and persistent” in his plans. His contractors considered him as “the most efficient foreman”. After the accident, they decided “the change in his mind (was) so marked that they could not give him his place again”. Harlow analyses that the “equilibrium between his intellectual faculties and animal propensities seems to have been destroyed”, he became “a child in his intellectual capacities” and he has “the animal passions of a strong man”. He is “fitful”, “irreverent” and “profane”, at times he gets “obstinate” yet “capricious” and “vacillating”; he forms many plans for the future and then abandons them “for others appearing more feasible”. Gage’s mother told Harlow that he used to entertain nephews and nieces (probably after he came back to his family in San Francisco) “with fabulous recitals of his wonderful feats, without any foundation except in his fancy”. She said also that he “conceived a great fondness for pets and souvenirs, especially for children, horses and dogs”. Finally, Harlow adds this crucial comment: “his mind was so radically changed, so decidedly that his friends and acquaintances said he was “no longer Gage”.”

What does Harlow mean when he writes that the “equilibrium between his [Gage’s] intellectual faculties and animal propensities seems to have been destroyed”? The terms “propensities” and “faculties” come straight out from Gall’s phrenology. In Gall’s treatise on phrenology, “propensities” pertain to the order of “feelings” and are divided in two “genus”: propensities shared by animals and humans and propensities proper to humans. “Faculties” are by definition “intellectual” and pertain to the second order: the one of “intellectual faculties”, exclusively human. Harlow cautiously tells us that the equilibrium between these two orders “seems” to have been destroyed. This disequilibrium seems to be caused by a fault of the intellectual order (“a child in his intellectual capacities”) more than by excess in the order of feelings (“animal passions of a strong man”). Indeed, Gage was already strong before, he already had an “iron will” and a “nervo-bilious temperament”: if his mind is not as “well-balanced” as before, this seems to be due to his regression into childhood (through his stubbornness, capriciousness and his incapacity to form plans for the future); a regression that affects his intellect, Harlow tells us. It seems that, for Harlow, it would be Gage’s intellectual faculties that were damaged by the accident. This interpretation is different from the one of contemporary neurosciences --Damasio’s especially--, which stresses the impairment of emotions and feelings as the consequence of a lesion of the frontal lobe.

Harlow doesn’t establish a connection between the cerebral lesion and a potential impairment of a “social” capacity; this term does not actually exist in Gall’s phrenology. However, the testimony: “He was no longer Gage”, which is not the one of the doctor but the one of Gage’s “friends and acquaintances”, condense the most profound change in personality assessed, indeed, by the social entourage. This sentence “Gage is no longer Gage” is from Harlow’s report the one that went down in history. Today, neuroscientists rely heavily on the judgment of the patient’s family (more rarely patient’s friends) to give this strange verdict: that people sick of FTD are persons who “are” not “themselves” anymore.
1.2. Lobotomy: Are the frontal lobes really necessary? (1871-1953)

Lobotomy of the frontal lobes begins at the end of the 19th century and will continue to be in fashion until the middle of the 20th century. The consequences of a lobotomy of the frontal lobe on the psyche of the lobotomized are variably appreciated depending on the observers. For some it is difficult to characterize its effects, for others the effects are clearly adverse and for a number of surgeons the effects are clearly beneficial for the lobotomized. It is not possible here to engage in a review of the literature about the effects of lobotomy153, but it is important to evoke the method that allowed knowledge of the frontal lobes to be built.

During the first half of the 20th century lobotomy became more and more fashionable. If we remember that Geschwind and his students saw this period as anti-localizationist, we have to point out that their opinion is in contradiction with historical facts. If there is one procedure in neurology that takes localization of cerebral function for granted it is indeed lobotomy. The first lobotomies154 on humans155 were carried on by Egas Moniz in 1935 to treat patients with schizophrenia and incurable depression. Moniz received the Nobel Prize in 1949 for his highly controversial psychosurgery. I here linger a little on a report made by Jean Lhermitte, neurologist of the well-respected hospital Salpêtrière in Paris, who reflects in 1953 on the last six years during which he practiced frontal lobotomy on 22 patients156. In its defense of lobotomy, Jean Lhermitte’s report provides a synthetic view on the outcomes and benefits of the procedure on the grounds of ethical and moral criteria, thus supplying my question with interesting facts. Except for two deaths (one was an elderly of 78 years old melancholic-obsessive whose life was hell anyways, the other a 50 years-old irreducible polytoxicomaniac), the improvement of symptoms with lobotomy of the frontal lobe was very encouraging. For instance, Jean Lhermitte reports the situation of a chronic melancholic who has enjoyed, thanks to the surgery, a “perfect equilibrium157” for three years158. The criticisms made of Egas Moniz on the adverse outcome of

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154 There were first lobotomies and then became leucotomies. Leucotomies are less “invasive” than lobotomies. Leucotomy aims at cutting some white matter in three places of the frontal lobe (different techniques can be used: for instance, Egas Moniz used first alcohol injection than an instrument called the leucotome), whereas lobotomy of the frontal lobe spreads the disconnection of white matter.
155 Before 1935, lobotomies were carried on animals. David Ferrier, a British neurosurgeon, practiced the first one on monkeys in 1870. After Ferrier, Leonardo Bianchi, an Italian neuropsychologist, practiced many lobotomies on monkeys and dogs, which sustains his influential work translated from Italian into English and French: La mécanique du cerveau et la fonction des lobes frontaux (traduit par MM. les docteurs André Collin & Sanguinetti ; préface de M. le Prof Charles Richet), Paris : L. Arnette, 1921. Bianchi concludes that the frontal lobe were, among many other faculties, responsible for “this feeling of affection for the others that we call sociability” (Ibid. p. 380). If love and friendship are feelings induced by the frontal lobes, Bianchi refuses to localize emotions in the frontal lobes (Ibid. p. 393). What is diminished in his lobotomized guinea pigs is the “power of intellectual sentiment”, but not “primitive emotions” (Ibid.). Much more needs to be said about Bianchi’s work and how it influenced current knowledge upon the frontal lobes, but I decided here to limit my expose to the knowledge built from experimental psychology on humans and not on animals.
157 Ibid. My translation.
his psychsurgery—“transformation of personality, bad tempers, aggressiveness, dissoluteness, passivity of mind and slowness of pragmatic activity” -- these criticisms do not reach Jean Lhermitte; the neurosurgeon finds they are not “based on anything or [are] hugely exaggerated”. Still, this list of consequences of frontal lobotomy is interesting for the question about the relation between the social and the frontal part of the brain: outcomes such as “dissoluteness”, “alteration of the ethical and affective sphere” and “perversion” seem to have a close meaning with what is characterized today [at the Memory clinic] as “inappropriate social behavior”. In the middle of the 20th century there are thus already some discussions about the relations between the frontal lobes and notions such as morals, ethics and affects. Since Lhermitte believes these outcomes are hugely exaggerated, we cannot much rely on him to exemplify what they mean. About “the alteration of the ethical and affective sphere”159, Lhermitte says he never observed it as a result of a frontal lobotomy. Quite the contrary. He gives the example of two nuns “obsessed and incapable of following the Rules of their Order, incapable to receive communion and to confess160” who, thanks to lobotomy, found again the faith and the religious fervor that they had before the psychosis. In Lhermitte’s view, the removal of the frontal lobe with lobotomy surgery is thus therapeutic, but before the surgery the two nun’s frontal lobes were actually responsible for an alteration of the “ethical sphere”. Lhermitte exemplifies what such a degeneracy means: for a nun, it is the impossibility to live accordingly to the ethical and moral rules imposed by her Order and affectively it means the loss of her fervor. The frontal lobe’s dysfunction is responsible for a loss of what one is, a nun, because one is not suited anymore to the social environment to which one chose to belong. This observation is close to what neuroscientists think today about some lesions of the frontal lobes. It is also close to the verdict given by Gage’s entourage quoted by Harlow: an alteration of the frontal lobe makes one no longer him or herself. At the Memory clinic warning signs of a latent frontotemporal dementia includes: job change, partner change, change of religious or political inclination. Yet, Lhermitte’s view is totally in opposition with today’s theories: for Jean Lhermitte it is actually a major lesion of the frontal lobes, in the form of a lobotomy, which brings back the affects and the ethics that were gone. Thus, if a dysfunction of the frontal was responsible for the troubles, the removal of the matter clears up the problem. Today this statement would be untenable, because the frontal lobe is seen as the container and the contents of our affective, social, ethical and moral dispositions: the more matter is missing the less inclined we are to live accordingly to these dispositions.

### 1.3. Escourolle, Brion, Delay: the first systematic studies of Pick’s disease (1958).

Shortly after Jean Lhermitte’s article, in 1958, Raymond Escourolle publishes his thesis on Pick’s disease161. Escourolle works under the supervision of Delay who will publish a book on

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158 Jean Lhermitte recounts also this quite extraordinary comment of a patient during his surgery: operated on without general anesthesia, this patient questioned during the surgery by Lhermitte, said that he felt relaxed right after Lhermitte sectioned the last frontal connection!

159 Ibid. my translation.

160 Ibid. my translation.

dementias with Brion in 1962\textsuperscript{162}. These psychiatrists of the Hospital Sainte-Anne in Paris, observe that the differences between Alzheimer’s and Pick’s disease faded to the point that, in the 1950’s in France, Pick’s disease was overly diagnosed compared to Alzheimer’s. Thus Delay advises the young Escourolle to study the specificity of Pick’s disease from 13 cases. Brion and Delay, ten years after Escourolle’s thesis, will publish their book in which they’ll synthesize Escourolle’s work and put it in perspective with other dementias. In this book, each chapter is dedicated to one dementia: Pick’s, Alzheimer’s and the third chapter on “senile dementias”. Indeed, Pick’s disease and Alzheimer’s disease are dementia of the young (the oldest person in their series of cases is 58 years old), thus they are not “senile dementias”. It is important to remember that in the 1950’s, scientists remembered from Alois Alzheimer’s\textsuperscript{163} work that his dementia (as well as Pick’s) were pre-senile dementias.

The comparison between AD and PiD made by the psychiatrists in 1958 is interesting because it shows a lot of similarities with the actual descriptions of AD and FTD. It is also interesting for the link it provides with Daniel’s work: Daniel actually went back to translate into English an article published by the French psychiatrists and used it as the basis for a critique of the American myopia towards FTD when the French were already in 1958 discerning the differences between Alzheimer’s and FTD.

The classificatory and comparative studies of Escourolle, Brion and Delay are the first of the kind. The authors observe that memory, language, praxia, gnosia and the “psychical and affective sphere” are affected differently in PiD and Alzheimer’s disease (AD) symptomatology. AD is characterized by memory troubles and problems of spatial and temporal orientation, that they do not observe in most of the patients with PiD (but in some). Language is affected in both diseases but the troubles are different. Patients with AD “distort words”\textsuperscript{164}, whereas patients with PiD “have stereotypies”. The later is exemplified by this woman who repeats tirelessly: “The jockeys are gone, they told me: Goodbye darling, Goodbye my love”, saying it switching from laughter to tears\textsuperscript{165}. In this register of repetition, patients with PiD also sing the same songs: for instance this woman who interrupts Escourolle’s exam to sing to him a verse of the Veuve Joyeuse or of the Rêve de Valse\textsuperscript{166}. On the other hand, the impairment of gnosia - the recognition of colors for instance- and praxia - the capacity to make a specific gesture, as for this woman who was incapable to complete the sign of the cross on demand: “She would bring a hand to her forehead and stopped with a sigh, embarrassed”\textsuperscript{167}, are typical of AD\textsuperscript{167}. Likewise for what the authors call “psychical troubles”: patients with AD can be “jealous, can develop a feeling of persecution and have a hostile attitude”\textsuperscript{168}, all of which contrast with the “indifference” of some patients with PiD. The authors actually differentiate between two modes of expression of PiD: the

\textsuperscript{163} For a discussion upon Alois Alzheimer’s work, see Fabrice Gzil’s PhD dissertation : “Problèmes philosophiques soulevés par la maladie d’Alzheimer”, Université Paris I, 2007.
\textsuperscript{164} “On va au commissiasa... au commissaire... quelque chose comme ça.” To say “commissariat” in Delay et Brion, 1962, p. 16.
\textsuperscript{165} “Les jockeys sont passés, ils m’ont dit: Adieu ma chérie, Adieu mon amour”, in Escourolle, p58.
\textsuperscript{166} In Escourolle, p. 28.
\textsuperscript{167} Delay et Brion, p. 16.
\textsuperscript{168} Delay et Brion, p. 14, p. 18.
indifferent mode and the jovial mode that they also call “moria”\textsuperscript{169}, from Latin morio: buffoon. People with moria sing, ramble and are inanely euphoric. Some patients with PiD present also a “flagging of the affectivity and of moral sense”\textsuperscript{170}. This “flagging of moral sense” along with a “deterioration of the ethical sphere”, remind us of the term “alteration of the ethical and affective sphere” used by Jean Lhermitte to describe the sudden lack of fervor of the nuns. Here, Escourrolle give us another example of what an alteration of the ethical sphere means: three cases (out of 13) are thus characterized because of their “erotic preoccupations\textsuperscript{171}”. “An erotic background appeared” in this woman for instance, who “made advances to her son-in-law and then to her own son\textsuperscript{172}”. A deterioration of the ethical sphere is equally diagnosed in this man, a film actor, who is to be found every night sitting in the front row of the orchestra at Mayol concert, where he (merely) contemplates naked dancers\textsuperscript{173}. The last case is Jeanne, who continuously repeats “A Monseigneur tout honneur”, says “erotic things [without more details] and sings silly songs\textsuperscript{174}”. By contrast, “to light a fire on one’s apartment’s floor” is not understood by the psychiatrists as a “damage to the ethical sphere”, but as a “damage of judgment\textsuperscript{175}”. “Dedicating one’s walks to follow funeral processions” is neither understood as a “flagging of moral sense” but as an “illogic act\textsuperscript{176}”. Today, this could have been understood as an affective trouble, for the taste of morbid or the pleasure to contemplate people in grief, suffering... To write a “rubber check” is understood as an “act of dementia\textsuperscript{177}”. And to not “worry about one’s familial life” manifests an “affective flagging\textsuperscript{178}” but not a “moral flagging” (as it might be understood today).

That which most resembles the “social” symptoms described by contemporary neurologists is the “ethical and affective sphere” appreciated by the three psychiatrists in the 1960’s. Their descriptions characterize the ethical or moral aspect of the PiD by a slightly overflowing eroticism; yet the moral symptoms of a lesion of the frontal lobe is limited to eroticism. Today, in the 2010’s, the symptomatology covered by “social troubles” encompasses eroticism (actually Daniel says that it takes more often the form of an hypo-eroticism than an hyper-eroticism) but is not limited to it; as we will see the moral degeneration of frontotemporal dementia patients circumscribes today a much richer symptomatology.

\textsuperscript{169} See Escourrolle, p 51. Some neurologists of the 19\textsuperscript{th} century already observed “moria” in patients with frontal lobe tumors. In 1888, Moritz Jastrowitz describes clinically a “specific form of dementia” in a patient with tumors of the frontal lobe. The symptom, an “oddly cheerful agitation”, is also described by the term “moria”. Hermann Oppenheim in 1890, refines Jastrowitz’s description, him too from patients with brain tumors: he observes that when tumors are localized on the right frontal lobe patients exhibit “childishness”. Yet, Oppenheim doesn’t agree with the description of moria (that he seems to understand as “silliness”): for him the attitude of his four patients is best described by an “addiction to trivial joking” on the sarcastic mode.

\textsuperscript{170} Delay et Brion, p. 31.
\textsuperscript{171} Ibid.
\textsuperscript{172} Escourrolle, p. 38.
\textsuperscript{173} Details of the case, in Delay et Brion, p. 77. Or in Escourrolle, p. 65.
\textsuperscript{174} Escourrolle, p. 73.
\textsuperscript{175} Delay et Brion, p. 31.
\textsuperscript{176} Ibid.
\textsuperscript{177} Ibid.
\textsuperscript{178} Ibid.
1.4. François Lhermitte: the frontal and the constraint of the social (1986).

François Lhermitte was Jean Lhermitte’s son; Jean Lhermitte wrote about the beneficial effects of lobotomy. Thirty years after his father, François was too, a neurologist, and held his office, like his father, at the hospital de la Pitié-Salpêtrière in Paris. François Lhermitte gave his name to the building that hosts the French clinic where I did my fieldwork. His name doesn’t only designate the building. François Lhermitte put his mark on the ways the French neurologists, in that clinic, understand today the symptoms of frontotemporal dementia. This is a good reason to study his work. But there is more. His two-part study published in 1986 called “Human autonomy and the frontal lobe\textsuperscript{179}”, is original in its style and in its thinking. I only consider the second part of the study entitled: “Patient Behavior in Complex and Social Situations: The “Environmental Dependency Syndrome”. In this article, Lhermitte develops a concept of the “social” –he actually uses the word- in relation with the notions of “free will” and “autonomy”. He reflects on what the frontal lobes teach us, from two patient cases, about our own capacity to be free and especially to be free from social constraints. This study is thus a view on the “social” from the field of neurology that could be compared with the views expressed by Damasio and Daniel on the same topic. However, I should make it clear that this comparison cannot coincide perfectly: the projects of these neurologists are slightly different. The part of the frontal lobe implicated in the social function in Lhermitte’s study is the dorso-lateral part, whereas Daniel and Damasio are especially interested in the orbital part of the lobe (also sometimes called the ventro-medial part), more in relation with “personality”, “consciousness” and “emotions”. Nevertheless, Lhermitte’s study shows us how a neurologist also a member of the Académie des sciences morales et politiques\textsuperscript{180}, thinks about the “social”; on these grounds, we can compare Daniel’s and Damasio’s ways of thinking about this elusive concept.

In this article, Lhermitte presents two patients: a man and a woman. They are not affected by a neurodegenerative disease, but were affected by a tumor that was removed by a lobectomy of the frontal lobe. After the surgery, Lhermitte observes how this woman and this man act in everyday life.

The man is 51 years old, an “engineer coming from a high socio-cultural background, well read, knowledgeable about art and keen hunter”. The woman is 52 years old, from “an average socio cultural background, who had confined herself to domestic work\textsuperscript{181}”. In the method section, Lhermitte warns the reader against what is going to follow: the experiments are “difficult” and to succeed one needs to have “established a close relationship\textsuperscript{182}” with the patients and their family so that the experiments don’t shock them. The unusually anthropological tone for a scientific article that seems to be set up in the method section is confirmed in the rest of the article. The author is present, in the first person, through the narratives of the experiments\textsuperscript{183}: “Patient 2 and


\textsuperscript{180} Established in 1795, this academy is the oldest institution concerned by human and social sciences. Its role is to describe the life of people in society in order to propose the best forms of government.

\textsuperscript{181} Ibid., p 336.

\textsuperscript{182} Ibid., p 335.

\textsuperscript{183} The anthropologist presents at the first person in its narrative is more an incongruity than a given in anthropology. The anthropologist is too often absent from the description he or she offers, observing his or her subjects as the entomologists observe insects, describing them from nowhere: the anthropologist is “a-topic” as
I sat in my office. I put some medical instruments on my desk.”...“In front of the audience, I presented Patient 1 with the insignia of “Office of the Order of Medical Merit. I embraced him in the traditional manner and then made a short speech”... “I showed her the syringe. She took it. When I took off my jacket and shirt, she picked up the needle (...) then bent down to my buttock to give the injection”. The doctor is also present in the surprising pictures that illustrate the article: François Lhermitte, in a white coat, takes a walk with his patient in the gardens of the Salpêtrière. In his office, still in his white coat, the patient takes his blood pressure, the doctor then sticks out his tong for her to examine his throat. The most curious photo is the one which illustrates the description that I just reproduced: Lhermitte, with his pants down to his shoes, his buttock naked, is turning his back to the patient who is armed with a syringe and ready to inject.

François Lhermitte observes his two patients in the midst of the action in different “social” situations, some of which might have required an elaborate composition: in a bedroom, in the doctor’s office, during a walk in the garden of the hospital, during a cocktail organized for an imaginary award, in a fictitious museum (in fact a well-decorated room of Lhermitte’s apartment) and in a gaming party where “20 elegantly dressed people” are playing chess, backgammon and roulette at ten in the morning. As in a script for a play, these scenes where the action takes place title the paragraphs that describe how the patients act. The theatrical effect is even more pronounced with this series of photos that catch this man and this woman in the middle of the action. The actions photographed are ordinary: “getting into a bed”, “choosing an object on a table”, “watering the flowers”, “hammering a nail into the wall”, “drying a plate” or “taking the change”. Yet, the photograph seizes also the subtlety of the actions: the man “refuses to use a set of woman cosmetics” placed on the table in front of him: in profile, his hands crossed on the table, the man sits stiffly, frowning angrily at Lhermitte who, we imagine, is sitting in front of him (“he glares crossly towards the examiner184”). We also see Lhermitte’s Parisian apartment transformed into a “museum” (to do so, it was enough for Lhermitte to pronounce the word “museum”): the man is planted in front of a painting, his arms are dangling by his side, he is “inspecting” the paintings hanging on the wall (“having heard the word “museum”, patient 1 methodically inspects all the objects in the entrance185”). In the following photo, pointing at a painting, his eyebrows raised, the man “makes some comments on a picture”.

Through the description of the actions of his patients in these fabricated situations, Lhermitte shows that both go along with the game with disconcerting naturalness. But this is precisely where the theatrical effect stops: these patients are obviously not playing. Of course, we may wonder: is it not possible that they knew that the situations were fabricated? Aren’t they actually making a fool of their doctor without letting him know that they know? From Lhermitte’s words, there is no room for doubt. On the contrary, Lhermitte readily analyzes that these two patients are caught up by the social environment he created for them and behave normally: as if they were truly in a museum, as if they were really playing in a gambling room, as if they could all of a sudden be honored and genuinely receive the insignia of the “Office of the Order of Medical Merit”, as if the roles could suddenly be reversed: that they could become the doctor of their own doctor and Lhermitte the patient of his patients.

Jeanne Favret-Saada argues in the fabulous Deadly Words (translated from French: Jeanne Favret-Saada, Les Mots, la Mort, les Sorts, Gallimard, 1985.)

184 Ibid. p 339.
185 Ibid. p 338.
One detailed example: Lhermitte is in his office with the woman: he suddenly gets up and leaves. Without a question and a hesitation, the woman follows him. The doctor gets in his car, she sits on the passenger seat. He drives her to the gardens of the hospital. She takes one pitcher of water from Lhermitte’s hands and she waters “painstakingly” all the flowers in the flowerbed. She then take the second pitcher of water from his hands and resumes “her labors with such care” that she waters “all the flowers right up to the end of the row”\(^\text{186}\). Then, they go to a square and pose “like tourists for the photographers”. And they leave as they came. During the 40 minutes of this scene, not a word was spoken. A few days after the outing in the garden, Lhermitte asked her about the event: the woman recalled and showed no surprise at all finding the outing normal even if Lhermitte “didn’t ask her to do anything”. The outing with the man went the same way, however Lhermitte changed the scenario slightly: when deciding to return to the office, they went to his car but Lhermitte got into the passenger seat and “the patient got into the driver’s seat without hesitation”. Both patients, when questioned, said that they “thought there were duties to be carried out and that they reacted in a natural way towards the environment”\(^\text{187}\).

I wonder: if Lhermitte had proposed to the woman, not to the man, the conductor seat (or “the insignia of medical merit”, that he actually proposed only to the man), would this woman, described by her doctor as modest, humble, especially preoccupied by domestic work, have seen these events as “duties”? Would she have behaved with the same naturalness as when she followed Lhermitte in the gardens and watered the flowers? Maybe not. Indeed Lhermitte noticed that despite the irresistible attraction the “duties” have on these patients, sometimes the patients resist them. Thus, when the man is placed in front of the set of cosmetics, he “was clearly offended and refused to use them”. Lhermitte analyzes that it is the “patient personality” that is expressed in this refusal. He sees it expressed too when the man, “from a high socio-cultural level”, was very interested by the paintings in the “museum”, and “made appropriate comments”. However, the woman, “from a humble background”, was mostly interested in the “curios” of the “museum” (a lion skin with the head decorates the floor of the apartment of the neurologist, a collection of collectible coins is exhibited on the mantelpiece) and also “derived great satisfaction from being photographed, as if it was a special privilege”.

The obviousness with which the patients were spontaneously involved in these games, as if it were the situations themselves that were soliciting them, is characterized by Lhermitte by the term “environmental dependency syndrome” (EDS). We learn what Lhermitte understands by “social” in the last paragraph of the article entitled EDS, Autonomy and the Frontal Lobe. What is social is what prevents autonomy: “For the patient, the social and physical environments issue the order to use them even though the patient “himself” or “herself” has neither the idea nor the intention to do so.\(^\text{188}\)” The social photographed by Lhermitte is the injunction of the museum for a person to behave like a spectator: to walk at a certain pace with the arms dangling by our body, to comment on the paintings, to stop in front of the mantelpiece to look at the collection of coins, to slowly walk around the lion skin.

Of course one first needs to believe one is in a museum to behave accordingly. In order to explain that his patients believe whatever he tells them, Lhermitte acknowledges that they must

\(^{186}\) Ibid., p 340
\(^{187}\) Ibid.
\(^{188}\) Ibid., p 342.
have lost some self-criticism\(^{189}\). In any case, what is troubled in his patients is not only their credulity that attests to their loss of self-criticism but also their way of being caught by the social, its rules and duties. For Lhermitte these patients are too bound to social situations, which in the end we all are: it is an illusion to think that we are free he concludes, to believe in freedom is to ignore that “each personality is dependent on a whole range of genetic and environmental factors and that these factors make him what he is. As a result, each individual subject does not possess true freedom\(^{190}\).”

Thus the social for Lhermitte seems to be what constrains us. But how do we know when we are constrained? It seems that we can give an answer to this question only a posteriori. Indeed Lhermitte makes a distinction between the “duties” that issue an “order” on which the patients act, and the ones to which they resist. Thus when the man refused to put on make-up, it was the sign of the expression of his individual personality says Lhermitte, which opposed to the social calling. But shouldn’t Lhermitte also take into consideration that this man was raised as a man, and men don’t put make-up on? Lhermitte also notes, in the museum, the basic attraction of the woman for the coins and the lion’s skin, and the educated taste of the gentleman for real art. Surprisingly, Lhermitte doesn’t explain this by the influence of a social milieu but again by their individual personality, even if he underlines himself the differences in the socio-cultural background of both of these patients. Thus the social, for Lhermitte, seems to be first and foremost what constrains us. The social is the name for these sceneries in his experiments in which the actors do what they are supposed to do. Because Lhermitte says that the social stops us from being autonomous, passivity in front of a situation is enough to characterize the situation as “social” when defiance, to the contrary, expresses individual autonomy, even if the defiance itself, in these cases, could have been explained by a “social” context.

Lhermitte explains that if his patients are too heavily constrained by the social situations they live in, it is due to the lesions in the dorso-lateral part of their frontal lobe. This explanation will be contrasted with more contemporary explanations (Damasio’s and Daniel’s) that see lesions of the frontal lobe as destroying our social inscription. Furthermore, compared to Damasio’s and Daniel’s works (and to the phrenological background that informs their views), Lhermitte doesn’t see the frontal lobe as a receptacle for a psychological function. The project of localizing autonomy — “a psychological function and not a physiological one” — in the frontal lobe, is meaningless says Lhermitte. All we can say is that the inferior part of the frontal lobe seems to “regulate” the EDS, by inhibiting the parietal lobe “which links the individual to the environment\(^{191}\).” In the case of damage to the dorso-lateral part of the frontal lobe, the parietal lobe is no longer inhibited and the person loses her autonomy and her free will. The frontal lobe is thus only a regulative element, and its lesion enslaves the individual to the duties of the social.

\(^{189}\) Even if this is true, Lhermitte fails to mention the special relationship he has with his patients: a medical relation that doesn’t only involve trust but also vulnerability and power that partly conditions the experimental situations. Lhermitte, the doctor, has a power of suggestion as much as his “museum”.

\(^{190}\) Ibid., p 343.

\(^{191}\) Lhermitte, Ibid., p 343.
1.5. Antonio Damasio and colleagues: emotion, the frontal and the social.

Antonio Damasio was a student of Norman Geschwind (as was Daniel’s professor: Frank Benson) and is today a famous neuroscientist of the west coast of the United States. He has published extensively: scientific articles but also books about the brain for the lay public. His later work is about the neural basis of consciousness but his publications in the 1980’s until the late 1990’s have focused on emotion. His *Descartes’ Error*\(^\text{192}\) published in 1994, is devoted to the role of emotion in rational behavior. He began to think about emotions in the 80’s and published several articles with his colleagues at that time. In what follows, I refer to Damasio’s book and two articles, one that he published with his wife Hanna Damasio and his colleagues in 1994\(^\text{193}\) and the other published in 1985 with Paul Elsinger\(^\text{194}\). His work on emotion and the frontal lobe as well as the neo-Darwinian theory that underlies it, are interesting because they were fully adopted (despite the antipathy Daniel has towards Damasio) at the Memory clinic.

The general aim of *Descartes’ Error* is to show with a few cases (Damasio’s patients and the mythic Phineas Gage) that emotion is essential to reason. How does Damasio describe the loss of emotion and the irrationality of these persons? How does he explain this loss neurologically? Before answering these questions, let’s say a word about the title of the book: Damasio argues that Descartes made a crucial error in thinking that emotion was insignificant for rational reasoning. Yet, Damasio’s statement is inappropriate: if Descartes thought that passion should serve reason, he didn’t claim that it was insignificant for reasoning\(^\text{195}\) as Damasio argues briefly in the third section of the book. As we will see there are other inappropriate comments in this book. The general impression at the reading of *Descartes’ Error* is that Damasio is engaging with the reader in a sort of monkey business: Damasio the thaumaturge shows us around the fabulous land of neurosciences and wishes that dazzled by the magic, the reader would merely accept his final argument (of three pages) that the discoveries of neurosciences surpassed the capacities of introspecting philosophers.

The emblematic case of Phineas Gage opens the book. Like his teacher, Geschwind, the student refreshes his memory with the work of 19\textsuperscript{th} century neurologists. Damasio “rediscovers” Gage and like Geschwind with Dejerine, Wernicke and Broca, this recovery doesn’t only add another case to the ones he studied himself but also supports his general argument. Gage’s story is particularly relevant to Damasio’s argument because of the verdict pronounced by Gage’s friends: “Gage was no longer Gage”. This little sentence that expresses the most radical change in personality bears on the testimony of the social entourage of the patient. This little sentence is of


\(^{195}\) (“(The error was) the abyssal separation between body and mind” in DE: 249). Yet, in *The Passions of the Soul* (1649) when Descartes writes: “The strongest souls belong to those in whom the will by nature can most easily conquer the passions and stop the bodily movements which accompany them”, Descartes argues that despite the fundamental role the passions play in rational thought, it is ultimately our ethical responsibility to achieve absolute control over them. He thus does not see passions as irrelevant or even inexistent for rational reasoning but claims that passions should serve the will.
great importance because while Damasio argues that reasoning and decision making are dependent on normal emotion, he specifies that this is true for one particular kind of rationality: one that is of use in “social life”. By contrast, for Damasio, there is another kind of reason that does well without emotion: one that doesn’t takes place in social life. We will see later how Damasio differentiates these two kinds of rationality and what it signifies for his understanding of the social and of the frontal lobes. Let’s linger first with the description that Damasio gives of Phineas Gage and of one of his patients, Elliot, “a modern Phineas Gage” because “Elliot was no longer Elliot”.

From Harlow to Damasio, there are some modifications in Gage’s story. As MacMillan well showed, these historical revisions are not peculiar to Damasio but to virtually anybody who wrote about Phineas Gage. What first strikes Damasio as impaired in Gage (in Descartes’ Error) is his capricious working life after the accident. He describes him as holding “no one job for long”, as someone who “moves around a lot” and “works at one place or another briefly”. However, from what Harlow reported, we know that Gage actually worked in the same farm in Chile for eight years. Damasio qualifies Gage’s departure to Chile “a theatrical coup” and doesn’t seem to believe Harlow’s report on the matter of what Gage did in Chile: “He may have worked on horse farms, and was a sometime stagecoach”. When Harlow only told us that Gage joined the Barnum’s museum, Damasio adds that he was showing his wound “vaingloriously” at Barnum’s. And about Gage in San Francisco, Damasio freely imagines him in the dive-bars of the city “drinking and brawling in questionable places”.

Damasio’s patient, Elliot, underwent a surgery for a tumor in the frontal lobe and as a result, explains the neurologist, resembled Gage in significant respects. Like Gage, Elliot’s memory, his language and his attention were intact. Like Gage, Elliot was “incapable of making the right decisions”. After his accident, he went back to work but only from time to time, he was mildly interested in what he was doing: “he might interrupt the activity he had engaged, to turn to something more captivating at that particular moment”, in the end he got fired, and engaged in an “ill-fated enterprise” with a “disputable character”; he then lost all his savings. He also got divorced. In brief, Damasio concludes that Elliot (and Phineas) had “fallen from social graces”, because of their inability to “reason and decide in ways conducive to the maintenance and betterment of himself and his family, no longer capable of succeeding as an independent human being”.

How to explain that both of these men had “fallen from social graces”? Damasio says he “was certain that in Elliot the defect was accompanied by a reduction in emotional activity and feeling”. Damasio speculates that Gage too was lacking emotions; he infers this from Gage

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196 DE : 34.
197 DE : 36.
199 DE : 9.
200 DE : 9.
201 In that line of thought: Damasio et al. (1994) write that Gage’s “respect for social conventions by which he once abided had vanished”.
202 DE : 36.
203 DE : 37.
204 DE : 38.
205 DE : 38.
“lacking the feeling of embarrassment, given his use of foul language and his parading for self-misery”\textsuperscript{206}. How does Damasio know that when Gage was exhibiting himself to get (the only) profits from his accident, he was not embarrassed? To infer such a thing, Damasio seems to take for granted that one would be so embarrassed to exhibit one’s self in a circus that this embarrassment would prevent one to do so, whatever is one’s situation. In any case, Damasio is certain that Elliot and Gage’s troubles involve an impairment of their emotions. Why? For Gage, this is speculation. For Elliot, Damasio uses his clinical intuition: if “at first glance there was nothing out of the ordinary about Elliot’s emotions”, looking at him more closely, Damasio realizes that Elliot was “always controlled”, not “facetious, just quietly humorous” and seemed “neither sad or joyful”. “He seemed to approach life on the same neutral tone”\textsuperscript{207}. Further, Damasio analyzes that Elliot “was not inhibiting the expression of internal emotional resonance... He simply did not have any turmoil or hush”\textsuperscript{208}. To summarize Elliot: he is a man able “to know but not to feel”\textsuperscript{209}.

Damasio’s hypothesis is that Elliot’s (and Gage’s) incapacity to feel is responsible for the important troubles of his social behavior. To test his hypothesis, Damasio first rules out a “fundamental intellectual deficit”: he subjects Elliot to the same kind of tests patients do at the Memory Clinic and at the Alzheimer Clinic. Some of those put to the test Elliot’s “executive function”. The executive function gathers psychological functions as vague and as dubious as “reasoning”, “task flexibility”, “problem solving”, “planning” and “execution”\textsuperscript{210}. One of the neuroanatomic substrates of the “executive function” is usually considered to be the frontal cortex, especially the dorso-lateral part of the frontal cortex. However, as some authors underline\textsuperscript{211}, some patients with a frontal lesion do not have an impaired executive function and vice versa. Be that as it may, Damasio’s patient performed well on those tests. Elliot was then subjected to another type of tests to evaluate his “knowledge concerning social behavior”\textsuperscript{212}. These kinds of tests offer some ethical dilemmas, usually in relation to money, for instance: Is one right or wrong to rob if one is lacking money? The correct answer, according to the test, is that it is ‘wrong’. Other kinds of dilemmas in relation to “moral values” or “moral judgments” are also tested: one person killed a man to survive on a desert island. Back to his normal life he has nightmares: he goes to the psychiatrist to seek treatment but the psychiatrist refuses to treat him, is the psychiatrist right or wrong? Wrong is the correct answer\textsuperscript{213}. Elliot has “excellent results”. Damasio concludes that if on the one hand: “It is clear that certain sectors of his brain still contained normal knowledge of facts, skills and principles of behaviors”\textsuperscript{214}, it seems on the other

\textsuperscript{206} DE: 51
\textsuperscript{207} DE: 44, 45.
\textsuperscript{208} DE: 44.
\textsuperscript{209} DE: 45.
\textsuperscript{212} DE: 46
\textsuperscript{213} These examples come from the article Damasio wrote with Eslinger about Eliott, “patient EVR”, in 1985.
\textsuperscript{214} Eslinger and Damasio, 1985, p1737.
hand that Elliot cannot apply what he knows in “real life situations”. So how to explain neurologically, Elliot (and Gage’s) “sociopathic” behavior, asks Damasio?

That explanation wasn’t ready in the article of 1985, it is only in 1994 that Damasio speculated on the impairment of emotions to explain Elliot troubles. The explanation is also mentioned briefly in the article published by Hanna Damasio et al. in 1994, which reconstructed the trajectory of the iron bar in Gage’s brain. H. Damasio and A. Damasio, in this 1994 article, observe that the iron tamping “must” (this is an hypothesis though) have damaged the ventral and medial sector of the right frontal lobe in Gage’s brain. Elliot scan’s too, showed that the right frontal lobe is more injured than the left, and that the lesion occupies the orbital and medial part of the frontal lobe. Damasio carries on his anatomical observation on other patients, most of them not his own: for that he goes back again into the past in Descartes’ Error and again makes some historical revisions. These mistakes, in Damasio’s too quick reading of Harlow’s report, in his misinterpretation of Descartes’ theory and now in his omission of some significant details in the study of Jacobsen and in the study of Hebb and Penfield, makes it look as if he was trying to force weight into his theory, which succeed in making it appear suspect. The conclusion is synthesized from those historical cases and from the observation made in his laboratory: the anatomical findings combined with the symptomatic inability of these patients to “make rational decisions in personal and social matters” and with the observation of their compromised “processing of emotion”, has led to the hypothesis that “emotion and its underlying neural machinery participate in decision making within the social domain and has raised the possibility that the participation depends on the ventromedial frontal region.”

215 Elliot is “sociopathic” on two occasions in the 1985’s article (p1734 and p1737), Gage’s behavior also fits with an “acquired sociopathy” (DE: 231).
217 Hebb and Penfield’s (1940) patient was the victim of an accident at 16 years old which damaged both of his frontal lobes, he developed during 10 years: “convulsive disorder, violent behavior and impairment of memory”. Damasio tells us about that patient that “his development in the field of social relations stopped and his social behavior deteriorated”. Damasio forgets to mention that Hebb did a subsequent surgery on his patient excising a large portion of both frontal region and that recovery following the operation was remarkable: “He was considered by his relatives and friends to have once again become his old self, i.e., before the head injury” (Benton quoting Hebb, in Benton, Arthur. Exploring the History of Neuropsychology, Selected Papers. Oxford USA: Oxford University Press, 2000, p 85)”. Also, Damasio mentions the experiences of Fulton and Jacobsen on two female chimpanzees Becky and Lucy. Fulton and Jacobsen observed that Becky who was loving and affectionate would fly into “temper tantrum” when she made errors on what was considered to be difficult experimental tasks. Lucy, on the other hand, was easy-going and not prone at all to temper tantrums. Both underwent lobotomy. After left prefrontal lobotomy, in front of a difficult task, Becky would still develop what Jacobsen called an “experimental neurosis”. Lucy’s nice character was equally unchanged. However, after right frontal lobotomy Becky no longer had “temper tantrums” and showed a remarkable equanimity. The total lobotomy of Lucy’s frontal lobes had the exact opposite effect. Damasio here does not make any distinction between the chimpanzees: both were “uneasy animals and were easily frustrated and malicious” (DE: 105); whereas Jacobsen described the temper tantrums for Becky but not Lucy. After the complete lobotomy, Damasio confuses the animals, Becky and Lucy both become “indifferent” and are not anymore “malicious”, they are “calm”, this is only true for Becky, Lucy is now the one having temper tantrums: reported by Benton 2000: 86.
Thus, rationality functions differently depending on where it takes place: rationality needs emotions when it bears on social and personal matters, and doesn’t function with emotion when it concerns “nonsocial” and “nonpersonal” matters. What is a nonsocial matter?

Nonsocial problems are the ones exemplified in the tests done in Damasio’s lab. The type of reason at work in that case, is of the type, Damasio tells us, that Howard Gardner called “mathematical intelligence”. This intelligence can be measured quantitatively. Damasio then exemplifies Gardner’s theory: people who demonstrate this kind of intelligence are the creative scientist or artist – “that we all know” - and “whose social sense is a disgrace, and who regularly harm themselves and others with their behavior”. In his efforts to exemplify the “mathematical intelligence”, Damasio in fact gives us examples of people impaired in the other kind of intelligence (as it seems: the social intelligence). Also we see that Damasio theory can apply to persons who don’t necessarily have a (known) neurological problem, but who behave like the artist and the scientists: as figures of the socially withdrawn. Because Damasio thinks he demonstrated that Elliot’s and Gage’s “sociopathic” behavior was caused by an impairment of a cerebral center involved in processing emotions, he thinks he also demonstrated that socially withdrawn people were necessarily emotionally impaired and could thus only express one type of intelligence. Yet, if being socially withdrawn necessarily equals emotionally withdrawn, I wonder about the example of the artist chosen by Damasio. This example is indeed surprising: the figure of the artist as someone whose emotions are impaired goes against common knowledge (and Damasio makes a point to use clichés) that artists are equipped with great emotional capacity.

On the other hand, how to characterize the other kind of intelligence and who is embodying it? The social is not reproducible in Damasio’s lab, thus the social is not measurable with instruments. “Reasoning and deciding in a personal and social space”, this is what Howard Gardner called the “social intelligence”. Again, there are persons Damasio tells us – that we also all know - “who are exceedingly clever in their social navigation, who have an unerring sense of how to seek advantage for themselves and for their group, but can be remarkably inept when trusted with a nonpersonal, nonsocial problem”. Unlike what he did when he exemplified mathematical intelligence, Damasio doesn’t give us any occupational example of social intelligence, indeed there is a sense of naturalness (it is actually an “unerring sense”) in the way these persons excel in seeking “advantage for themselves and their group”. As if those equipped with social intelligence would be positioned on the side of the natural, when those lacking it (even if equipped with mathematical intelligence) would then be located on the side of the monstrous.

In this exposé of types, it seems that we have actually diverged from Damasio’s theory about emotions as much as from Elliot’s case. In generalizing his theory, Damasio seems now much less interested in emotional incapacity as he is in social incapacity: the figure of the a-social or the “sociopath”, as he says, is the central pathological figure of Descartes’ Error, not the figure of the a-emotional. Probably because neurosciences experience difficulties in grasping a coherent view of what is an affective experience, when it is easier to offer demagogically the more

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219 DE: 169
220 DE: 169.
221 Or of the figure of the absent-minded professor who is “the benign variety of the latter type [the nonsocial type]”, see DE: 169.
222 DE: 169.
conventional term “sociopath”. Since the sociopath is central to Damasio’s theory, what is then the social in this theory?

We recall that the social was for Lhermitte essentially made of rules and duties that could deprive us from our (illusory) autonomy. Lhermitte’s patients with a dorso-lateral lesion of the frontal lobe – a lesion that can affect the mathematical but not the social intelligence for Damasio – ended being caught up by their social environment. In a way, Lhermitte’s patients had a social surplus. In Lhermitte’s theory, a social excess is pathological, for what he observes in his patients, but also for the generalization of his theory: social duties deprive his patients and to a lesser extent all of us, of our autonomy.

For Damasio, it is quite the contrary: what constitutes the pathology is the figure of the a-social who “regularly harm themselves and others” while the ones who have social intelligence are good (seeking advantages) for themselves and quite importantly, “for their group”; e.g. for their society. In this perspective, the pathological is the lack of a “social” capacity. In Damasio’s theory, there is neither aftertaste nor reserve in the way our environment deprives us from our free will; social rules and duties can only be beneficial. In a Darwinist perspective, he proposes a genesis of social conventions and ethical rules around the avoidance of pain: “It is likely that they [the rules and conventions] evolved as a means to cope with the suffering experienced by individuals223. These rules thus appeared in individuals who “were able to realize that their survival was threatened or that the quality of their post-survival life could be bettered”224. The social is reduced to a context governed by the necessity to adapt and is only understood as a context favorable to the reproduction and the survival of the individual. Damasio then links up pain to emotion and the cycle is now completed: suffering is associated with the emotions of pleasure and pain, thus it is those emotions that drive the elaboration of our ethical rules and social conventions. At the end of Descartes’ Error, the relation between emotion and reason is not only physiological, it is also phylogenetic: emotion is not only what allows us to follow social rules it is also what created them. Our reactions of pain to the milieu225 thus produce and explain the social226. The meaning of our actions is governed by the theory of evolution. In this perspective, Damasio sets out the goals – “to possess a home”, “to preserve our physical and mental health”, “to have a job, an income and to be socially recognized”– to accomplish in order to stay in tune with our emotional and rational nature.

As much as Damasio adheres to what he understands as the social world, Lhermitte resists to it. Lhermitte shows how the living is caught by the social and criticizes our appetite for freedom almost in a spirit of resistance. Damasio shows why the living should rightly follow social rules and duties, in a spirit of seriousness. Damasio’s psychology is able to justify everything in the name of this spirit of seriousness. These two different ethical attitudes seem to leave an impact on scientific knowledge. Somehow, the political correctness that in the end overcame Damasio’s theory made it appear dubious, whereas I read enthusiastically and was convinced by Lhermitte’s description of these two patients enslaved to certain social codes.

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223 DE : 261.
224 DE : 261.
225 But if this milieu pre-existed to the social milieu, this milieu is not the social itself, then what is it? It is unclear.
226 Damasio at different points of his argument also coherently pleads for a form of sociobiology.
1.6.  *A chart to conclude.*

As a conclusion to the first part of this second chapter, there is a little chart on the following page, to summarize the rough influences of the neuroscientists we read about, firstly on each other and secondly on the French (Alzheimer clinic) and the American (Memory clinic) clinic (see next page).
Phineas Gage (1823-1860)
Case study by John Martin Harlow (1868):
The frontal is a center for the “intellect”.

Lobotomy (1870-1953)
David Ferrier (1871), Leonardo Bianchi (1922): The frontal as a center for “attention” and “representations”.
Jean Lhermitte (1953): Lobotomy of the frontal lobes restores the “ethical and affective sphere”.

Escourolle, Brion, Delay (1958, 1962)
1st systematic distinction between Pick’s disease and Alzheimer’s disease.

François Lhermitte (1984)
The frontal (especially the dorso-lateral part) regulates (via the parietal lobe) our autonomy from the “social”. A lesion of the frontal lobe provokes a strong attachment to social rules.

Antonio Damasio (1994)
The frontal (especially the orbital part) is the center for our emotional and affective abilities. Lesion to the frontal produces sociopathy and a strong detachment from social rules.

The Memory Clinic, USA (2010-2012)
The Alzheimer clinic, France (2012)
2. The Behavioral Variant of Frontotemporal Dementia at the Memory Clinic.

We are now back in the amphitheater of the University of R., where the chief of the Memory clinic, Simon Daniel, finishes telling us what thirty years of work on patients with behavioral-variant frontotemporal dementia patients taught him. Daniel’s talk and the second part of the chapter more generally only deal with “behavioral variant FTD” patients (not the semantic and non-fluent variants). From now on, I will stop specifying “behavioral variant FTD” and I will simply call these patients, as Daniel does, “FTD”.

Simon Daniel, like Antonio Damasio, differentiates the troubles caused by a lesion of the dorso-lateral part of the frontal lobes and the ones caused by a lesion of the ventro-medial (or orbital) part of the frontal lobes. When a lesion affects the dorso-lateral part of the frontal, the troubles can be objectified with a series of neuropsychological tests that examine the so-called executive function. However, says Daniel: “Some patients don’t have any deficits in the dorso-lateral part of their [frontal] brain. As we studied gene carriers, we have learned that some of the early patients don’t have any deficits in executive control. So how do they present? Well, with a part of the brain involved in the regulation of emotions and behavior.”

The troubles that are of interest to Daniel are the ones that target “emotions” and “behavior”. We will see in the diagnosis process (in chapter 4) what precise actions or behaviors will be telling to affirm that one is affected by the behavioral-variant of frontotemporal dementia. For now, let’s see how Daniel decided to exemplify these troubles in front of his audience that day. Which symptoms did he choose that he thought would describe frontotemporal dementia best? How does Daniel understand the core of the troubles of FTD? After answering these questions with thus Daniel’s own presentation of his work, I will describe how a philosopher who began to collaborate with the team of the Memory clinic for a project on “love”, was told about by the team, understood and analyzed what is missing in persons affected by frontotemporal dementia.

2.1 Lack of punishment and emotional deficit.

The trouble that Daniel mentions first is what he calls “lack of punishment”. The notion of punishment, says Daniel, can be put to the test with the “chocolate paradigm”: the head wedged in a scan to perform a fMRI\textsuperscript{227}, someone is given chocolate.

Daniel. – After 6, 8... 10... 15 bars... it starts to become pretty aversive. What happens in normal people is that the lateral orbito frontal cortex begins to activate. This orbital cortex has different functions: there is a medial part that is reward-based and there is a lateral part - with the insula- that is involved with punishment: we think this part is over-hit. These people don’t feel punishment

\textsuperscript{227} fMRI (functional magnetic resonance imaging) links the activity (electrical and chemical) of the brain with a psychological state of the subject.
when they overeat, they don’t see punishment in the future when they do steal, they do things that most of us would avoid.”

Stealing thus is controlled by the same cerebral mechanisms as bulimia, because both events are understood as the transgression of something prohibited.

Daniel further illustrates what a brain lacking the notion of punishment would do with a video of one of his patient in a church.

Daniel. – Church is a great place to tape people with FTD (Laughs in the audience.) No! Let me say that again (The audience laughs even more.) It’s a place where there are rules that we rigidly adhere to.

The film starts: we see a man, the patient, sitting down on one of the benches. In the back of the church, someone is playing a joyful air on a piano. The patient suddenly leaves his seat and goes towards the line of communicants. He takes the hands of the people standing in the line, and shakes them with a smile. He is maybe confusing in the order of the ceremony, the moment for greetings with the solemn moment of the communion. His wife stands up and goes to get her husband. We hear her, commenting on the film over the same joyful music which is still playing in the back: she says that she avoids going to church with her husband on the first Sunday of the month; a day, apparently, when communion is especially busy.

In the audience some people can’t stop laughing.

Daniel. – So if you have a disorder associated with punishment and reward you may do things that are very, very grossly disturbing... that really grossly stand out for people who don’t have this disease. The minute the patient is in a social environment there is trouble.

FTD sufferers do not respect social rules: they steal, they eat too much; they don’t follow any kind of social ceremony (not only that of a church). Daniel explains what he seems to understand as a form of disobedience, by the lack of the notion of punishment and reward. We remember, that in Damasio’s view, social rules were created to maximize pleasure and to minimize suffering. In Daniel’s description of a patient affected by FTD as someone “in trouble” in a social environment and in Daniel’s explanation of it by a lack of the notions of punishment (suffering) and reward (pleasure), Daniel positions himself in the same line of thought as Damasio. The two neurologists also root our emotional capacity in the frontal lobes. The lack of emotion of FTD patients is actually the second characteristic of these patients that Daniel chooses to talk about.

Daniel. – This is a list of the emotional deficits that we see in FTD: cruelty to children, animals and to the elderly, lack of concern when the others are sad, rude comments to others, loss of respect for interpersonal space, disgusting behaviors - I think because of loss of disgust- and sometimes loss of pain. Isn’t that an unbelievable constellation of behaviors? In our society we would think that they are primarily psychiatric induced by an abnormal environment, and in this case they are so strongly driven by anatomy that there is almost nothing else as a cause.
Daniel illustrates the loss of emotions with a scene that takes place in a lab of experimental psychology. Patients who stay at the Memory clinic for a week are also part of a research program. One day of the program is scheduled for an excursion in the neighboring university at a lab specialized in the psychology of emotions. The stay at the lab begins at nine in the morning with a viewing of film extracts: the patient is asked to grade on a scale (from one to five) the intensity of the emotions felt (or actually played) by the actors, and the intensity of the emotions they felt in return watching the actors. During the viewing, the person is sitting in a sort of dentist’s chair, and wired with electrodes on her hand, her ear, her heart, in order to measure her heart beat, her pulse and her body temperature. After viewing around 20 film extracts (and looking at more than a hundred pictures), the emotions of the patients are then put to the test in “real” situations. One of these situations aims at reproducing a scene of disagreement between the patient and a family member; this experiment is called the “social interaction task”. The patient and the family member (spouse, parent or child, all of whom are called “caregivers”) are sitting one in front of another in a room staged like a living room: an old-fashioned wall paper, a dusty carpet, a coffee table, a TV on which someone put a vase with some plastic flowers. The patient is still tied up to her wires, sitting in front of his caregiver who is only wired with a microphone. The patient and his caregiver are asked to discuss a subject that provokes frequent stormy discussions. The conversation is viewed on TV screens from the neighboring room and is also audio and video recorded. Among the 600 scenes that were recorded in this lab, Daniel chose one that exemplifies “cruelty” of FTD patients: “So here is a patient and his mum, an incredible person, and they are talking about a conflict and the conflict is his cruelty to the family dog...” We get only the soundtrack: we hear the mother who is talking to her son, being careful of pronouncing clearly each of the words:

The mother. – When Max barks do you sometimes squeeze him? Or put him between your legs? And sometimes he squeals because you are hurting him. Are you doing that because you are angry with him or because you want him to stop?

The son. – Because I want him to stop.

The mother. – Do you think you could just talk to him? And not squeeze him so that he squeals? Do you think you could do that?

Silence.

The son. – I don’t know what you are talking about.

The mother. – Uh.

Daniel explains that Will (the son) has sincerely no idea of what his mother is talking about: “When we studied him in the lab, he could not detect any negative emotions”. Will couldn’t tell if Drew Barrymore was playing sadness, anger or joy. Will couldn’t tell if his mother was angry at him or if the dog was in pain. He didn’t know what she was talking about when she asked him to stop hurting the dog, he didn’t hear the anger behind his mother’s efforts to control her voice. He didn’t even seem to experience his own anger: he told his mother that he wasn’t angry at the dog; he just wanted the dog to stop barking. This is I think what Daniel’s interpretation would look like. We could also imagine that Will doesn’t know anymore how to “obey” his mother: being affected by a lesion of the frontal lobes he doesn’t understand the rules (established by his mother and by all the ones who protect the animals) and even less understands
the consequences of the transgression of these rules (since he lost the ‘notion of punishment’). We could also imagine that he actually understands what his mother is asking him but doesn’t know anymore how to comply: speaking to people is already difficult and speaking to dogs is not within everyone’s means. We could imagine different explanations of Will’s incapacity to follow his mother request. However, Daniel added that the lab’s tests concluded that Will could not detect any negative emotions. The same conclusion could be reached from Will’s everyday occupation: Daniel explains that Will, who was an artist, was very fond of representing (human) faces: “We learned that he was obsessed with relationships: he painted very odd pictures of people”. These faces that Will is painting, are odd, actually the famous psychologist Paul Ekman saw the paintings and Daniel tells us that he “said they didn’t show normal emotions”. Ekman, that Daniel admires a lot, is the most famous theorist (after Darwin) of the psychology of facial expressions. In the 1960’s, Ekman went to Papua New Guinea and through a comparative anthropology demonstrated that facial expressions were universal, hence were innate. So if Ekman said that the emotions painted by Will were abnormal it might be true that Will, through his incessant painting practice, was trying to seize a meaning that constantly slipped away: “Will lived with very distorted emotions”… and Daniel shows us some of the paintings: “And now he is drawing little bits of eyes, faces, and bicycles; and in the late stages he is down to single lines. He died with ALS and FTD. That was Will.”

In this brief presentation of the ideal-typical FTD patient, we may have noticed how Daniel and Damasio share a common understanding of the lesion of the frontal lobe as causing a lack (or even an absence) of emotion and compliance to social rules.

Daniel takes one step further concluding on the anatomy of the loss of “empathy”: “If you loose empathy, you are statistically very likely to show atrophy in the temporal pole, medio orbito frontal cortex, caudate, medio frontal cortex, all on the right, all critical circuits for relating to other people. The regions hit are on the right hemisphere and not on the left. It really strongly shows in the study of dementia that the misbehaviors, the behaviors that the caregivers consider bad, are driven very strongly by loss of function in very specific brain circuits.”

This notion of “empathy” defined as “the incapacity to relate to others” gathers in one go the deficiencies in our “social” and “emotional” capacities. But how to be sure that FTD bearers are not “relating to other anymore”? More generally, what can allow us to say that a person does not have empathy? This is precisely what a philosopher, new to the clinic, is going to attempt to understand. In the next part of this chapter, I first describe the interrogations phrased by a philosopher in front of the team of the Memory clinic about the capacity to “care” of FTD patients. Following this meeting, the philosopher will analyze and conclude on her question at the occasion of a presentation she made at a philosophy conference. I was unable to attend this conference but she kindly passed her paper on to me. This information is treated anonymously, without quoting or citing the philosopher, as is the rest of the fieldwork.

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228 ALS: Amyotrophic Lateral Sclerosis, also referred to as Motoneuron disease, Maladie de Charcot or Lou Gehrig’s disease, is a disease that is traditionally diagnosed on exclusive motor symptoms. However, since 2011 a unique gene (C9orf72) is considered as causing both ALS and FTD. Thus it is possible for someone to be sick of FTD and ALS, to the point that at the Memory clinic, some wonder if it is not the same disease.
We are now leaving the dark amphitheater and Daniel’s talk at the University of R. and slipping back in time to the philosopher’s encounter with the team of the Memory clinic, which took place around two months before Daniel’s award.

2.2 To care or not? The philosopher at the Memory clinic.

This Friday Morning, for the weekly grand rounds, the Memory Clinic is welcoming a philosopher. She will talk about demented patients’ capacity to “care.”

It’s been a long time since I last came to the Memory Clinic, I haven’t seen the team since almost a year except for Michael, the fellow, and Beth, the nurse, with whom I sometimes had dinner. When I received the email with the grand rounds’ title: The capacity to care of demented patients, I thought I would like to attend to this talk. I wondered why one would ask about the patient’s capacity to “care”, when a more pressing and unsolved question is to know how we, the non-demented, could care for these patients.

The philosopher is not thinking about dementia for the first time. She already thought about the concept of “care” for Alzheimer’s patients. Today she wants to clarify the concept with frontotemporal dementia’s symptomatology. For this inquiry, she naturally found her way to the world-renowned clinic on frontotemporal dementia (FTD). At first sight, she indeed seems to be at the Memory Clinic to do an inquiry: yesterday she was in the clinic and she interviewed a patient, today she’ll present her project to the neuroscientists, and in six months time she is planning to come back to gather more information. She is also collaborating on a project with Nicole, a neuropsychologist of the clinic. In the end, I don’t know if the philosopher met a lot of patients in the clinic, nor if she talked to many of them. I know that two years after this talk, during which she paved the way for her question, she wrote a paper, in which she tried to give an answer to her question. I know that between the talk that I heard and the paper that I read, the patient’s cases are the same. I know that except for a patient, a woman, that she met personally and whose case helps the generalization of her argument, the other cases are patients that she heard about from the neuroscientists or that she read about in their files.

The room is packed this morning but I found a seat in the back. Without departing from the Friday mornings’ Power Point practices, the philosopher starts with a reminder of her work on Alzheimer’s patients. Against Ronald Dworkin, she defended the thesis that Alzheimer’s patients were still capable of autonomy. First, she established that these patients were not only beings with “desires”. For a long time, she says, patients with Alzheimer’s disease are capable of “valuing” things. This observation allows the recognition that Alzheimer’s patients retain autonomy, until a more or less (unspecified) advanced stage of their disease. Indeed, unlike Dworkin who postulates that autonomy is to live accordingly to the kind of life we choose, the philosopher distinguishes two conditions in this proposition: the first condition for autonomy is to express one’s character in one’s life, the second is to be able to implement it. From her experiences with Alzheimer’s patients, the philosopher thinks that they are capable to meet the first condition but not the second. It is only with the help of the other(s), in that it will help them to implement their value.

229 I am using inverted commas because we don’t know yet what meaning she is going to give to this polysemic word: is it an activity (to take care) or a feeling (to care for), or is it both?
in their life, that these patients will be capable of living their life accordingly to their values. Therefore, her reflection on Alzheimer’s patients has a practical scope: she asks and answers how we can care for these patients. For FTD patients, she will ask if they retain the capacity to “care”, but that will be it. Probably because of the negative response she will give to the former question, she could only turn away from answering the practical question of care for these patients.

In keeping to the concept of value that the philosopher just talked about, she introduces the concept of “care”: “To value you at least need to care. So it is very hard to value without having a capacity to care. In my opinion. I am just stating. This is the way I see it.”

Alzheimer’s patients would clearly have kept this capacity to “care”. She demonstrates this with some excerpts from the film *Complaints of a Dutiful Daughter*. In this documentary, Deborah Hauffmann films her mother affected with Alzheimer’s disease. We see Deborah’s mother, Doris, sitting in an armchair, in a conversation with her daughter. We learned from a preceding extract that Doris doesn’t always recognize Deborah as her daughter. In this conversation, the mother says to her daughter that she likes her partner, a woman, who is there too, sitting on the coach. Deborah tells us that before her disease, her mother disapproved of her homosexuality (by refusing to acknowledge the partner), but after her disease even if Doris doesn’t always recognize Deborah as her daughter, she yet recognizes that this woman who is often with Deborah (she is also the director of the film), brings her happiness. The philosopher exclaims: “Look at these people: they seem to still deeply care about certain things. You have this woman, she cares about this person who comes around and doesn’t even understand that it’s her daughter...”

The choice of this example seems to root the meaning of care in a feeling; as someone in the audience, touched by the scene, comments about the mother: ‘She loves her’. It would thus be in the sense of to “care for” that the philosopher understands the persistence of care in Alzheimer patients. The team of the Memory Clinic is in full agreement with the persistence of this feeling in people diagnosed with Alzheimer’s disease. In that line of thinking, a neurologist intervenes: he raises his hand: “I couldn’t help with a comment... I try not too but...”

People laugh. People are especially in a good mood this morning. They participate a lot and ask their questions without waiting for the end of the expose. The philosopher interrupts her talk to listen and to answer the questions but in a quite unusual way for these conferences: she is not only discussing, sometimes she also corrects as if she were teaching (“Your view is more Dworkin’s view”, “Right, yeah, but there it is different”); it feels like we are in class.

The neurologist (*launching into the conversation*). – Several things come to mind with what you’ve shown: there is a very strong connection with the

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230 She states it for the audience of the MC, but she has developed elsewhere the difference between value and care: valuing would require a reflexive understanding of one’s own mental states by the correctness of one own’s belief: one has to value what one’s value. By contrast caring does not require endorsement of either belief or volition. For the philosopher since a very young child can subsumed the attitude of “caring”, caring presupposes neither motivational hierarchy nor evaluation.

231 The philosopher gives another example: a woman who doesn’t want to bath, but who cares about seeing her grandchildren. In this example too she stresses on a value, seeing the grandchildren, which implies love for others.
people around them in this kind of caring though and I can’t help think that
this is what drives our values, that a lot of what we think about as our value is
that we don’t want to hurt others, that we want to play well on the playground,
that is maybe the underpinning of our core value: we want to make the ones
around us happy.

The philosopher (*half-convinced*). – I agree with you that valuing is rooted in
caring, but you are talking of it as if it were very object specific: like care about
other people, but hate is a form of care in my view... and you can care about
your collections, not necessarily about people.

The philosopher then tells us more about her framework. One will be said to be “caring” if
his or her dispositions vis-à-vis the object of caring can satisfy four conditions.

First, one is *emotionally* attuned to the object of care (worry when O is in danger, happy
when O is flourishing, hope that things will go well for O...). O is not necessarily a person, or a
domestic animal; O can be a collection, or an activity. As strange as it sounds, it is possible
though to imagine one saying that one hopes everything goes well for “anthropology”.

Second, one has “cognitive dispositions” towards the object of care: for instance seeing a
“danger” hanging over O would be a reason to act.

Third, one has some *motivational* dispositions: promoting and protecting actively O.

Fourth, one *deliberates* in the consideration of the welfare of O, as it will affect other
domains of deliberation.

With this definition in hand, the philosopher asks about FTD patients: “Do they still have
the capacity to care?” Are they like Alzheimer’s patients, or is there something specific to FTD
that prevents the person who bears this disease from caring? She is specifically asking the team to
provide her some answers. She thus wants to take up her question with a neurological approach; if
she was teaching at the beginning of the talk, the neurologists have now a chance to teach her.

In order to present the dilemma, she takes the example of the patient she met yesterday at
the Memory Clinic: a woman, diagnosed with FTD, who used to be devoted to her family and
now doesn’t seem to care about them anymore. However, one thing that seems important to this
woman, is to eat out for every meal... The philosopher first asks the team: can we say about this
person that she doesn’t care about her family? And, second, can we say that she doesn’t care
about anything at all?

These questions are indeed asked in the negative form. I wonder if the philosopher has
already the sense of an answer. Yet, she seems cautious about giving a too quick response to her
questions: “I am just raising the question”, “I should be careful to how I phrase this dilemma”.
Certainly prudence is acceptable, because an answer to this dilemma is no trivial matter: it is
about still considering a person as a person. If the philosopher establishes that this woman sick of
FTD is not capable of caring, she explains that there is “no reason to put as much weight in what
they are [she is] saying as in what you are saying”. The philosopher showed that for Alzheimer’s
patients who retain their values, there was “something at the core we need to respect”, therefore
they “need help with translating their value and care into concrete decisions”. However, if FTD
patients do not care, if she establishes that what FTD patients say “is coming from an individual
who is not capable of valuing overall”, what will that mean about the “core” of this woman who bears FTD and what will be the consequence of that meaning for her need for help and respect?

What can the neuroscientists teach the philosopher about care and FTD this morning?

Daniel is sure of his answer: these patients do not care anymore. Without the slightest hesitation. Daniel, moreover, observes this “breakdown in caring” very early on. Thanks to the genetic forms of FTD, it is possible to observe a “breakdown in caring” and “how it threatens the family”, “30 years before you have a real dementia”. “Not caring” would thus be one of the first signs of FTD. The philosopher wonders how to separate in that case, between what Daniel would reconstruct a posteriori, after the diagnosis is made, and what one could actually observe from the attitude of someone still healthy. The philosopher ignores that Daniel allows himself to diagnose healthy gene carriers. Daniel, as we will see, uses his clinical intuition to diagnose people in perfect health but who unfortunately carry a monstrous gene. And this “deficit in caring” is, according to Daniel, one of the first prodromes of FTD. Thus “not caring” would actually constitute an essential root for diagnosing people with FTD. What if Daniel’s intuition could be replaced by a formal test that any clinician (especially one without intuition) could use? The collaboration between the philosopher and the team might offer this kind of perspective. The project of the philosopher takes another dimension: the stake is not only to give a philosophical analysis of the notion of care from her observations at the clinic, but also to give a framework to “test these kind of things”. The philosopher could give a practical scope to her thought. If for Alzheimer’s patients her reflection served the care for these people, for FTD patients the philosopher would serve the diagnosis: an odd way of “caring for” people affected by a disease that medicine cannot treat today as curable illness. But, as she cautions, the implementation of such a test is tricky because in the lab environment, the test would only capture something instantaneous, when “care” is actually a pattern of disposition that develops over time...

Nicole the neuropsychologist who collaborates on the project, thinks that the question goes beyond FTD’s symptomatology: it bears also on “autists and sociopaths”: “It’s like if you have someone who is incapable of caring for other people: are they still a formal agent? Should they be considered as a partner in the decision making process? And this is not only for FTD and its prodromes but it can be for the one who during his life long doesn’t care about people”.

As we have remarked with the work of Antonio Damasio, Nicole as well, seems to consider FTD and sociopathy as synonyms. Since I hesitate on the meaning of the word sociopath, I look on the Internet and I read the definition on Wikipedia:

“Psychopathy (or sociopathy) is traditionally defined as a personality disorder characterized by enduring antisocial behavior, diminished empathy and remorse, and disinhibited or bold behavior.”

Wikipedia’s definition of sociopathy is indeed very similar to the one given by the Memory Clinic on FTD. But here again, the terms “diminished empathy and remorse” do not seem to be applicable to other things than people. It doesn’t make sense to say that one has empathy for anthropology. Yet, the philosopher is really attached to her definition of care and reminds Nicole that in her framework, “care” is not a notion reducible to love of others. “OK” she says, “so it’s very important: I am not only talking about caring for people. So one possibility of what is going on with FTD is that the patients have very attenuated capacities to care about
people (if at all) but they might be able to care of other things and the fact, you remember I said that care was not object specific this is were it becomes very important... right?”

Albert, one of the most illustrious neurologists of the Memory Clinic and a potential successor for Daniel, tries to clarify the problem. Albert sees a crucial difference between care for people and care for something else than people. He gives us the example of one of his patients whom he describes as “caring only about old records”.232

Albert (after thinking). – So I feel he had the capacity in your model to really care about these records and yet his capacity to care for his family members and this long-term wellbeing of family and job and all these sort of things were clearly impaired and even in the very, very early stages. So I think that the capacity to care ... is sort of ... I mean... sort of segregated based on the nature of the object: maybe social and human objects have a different status than nonsocial objects... or other kind of objects...

The philosopher (conciliating). – And that would be differentiated in the brain?

Albert. – Differentiated in the brain so that a patient could actually come in with an object or a domain specific caring... Because I think this is what we really see in the earlier stage. Now, in the later stages, that same patient may evolve to loose non-social objects caring capacities and ... But I think that the early lesion impairs the social domain. It makes me think that in this [the philosopher’s] model of caring, the only thing that may be missing is the idea that it can be targetedly deployed to different kinds of objects...

The philosopher (enthusiastic). – Right, but... right... so... but let me just ask you... your sense of this patient that were somehow you know, “shallow” and ... (Carefully.) You know the kind of language that people... (Smiling and cheerful tone.) I heard used about these patients... that there was something sort of at the core human that was missing, or, in so far as he was...

Albert (irritated). – What’s the point of asking questions like that when we have to start to define what’s the core of human thing?

The philosopher. – Yeah...

Albert. – Then you really get to the end of this, and ... If I answer yes [unclear to what question], I feel I will be going circulate...

The philosopher. – Right.

Albert. – I mean, even if I believe that the core of humanity... (Laughing.) Is to be able to care about others.

The philosopher (not laughing). – Right, OK.

From this strange conversation, we learn that Albert, like Daniel, thinks that FTD patients loose their capacity to care. He differentiates two kinds of “target” for caring: people (“human”)

232 Note that Albert says to care about records, and not to care for records. Is there a different meaning in English? To care for is coming from a feeling, has this feeling vanished when one talks about caring about? Is Albert talking about the same thing as the philosopher?
or “social objects” and “non-social objects and other kind of things”. We remember that Antonio Damasio made a similar distinction. Albert’s patient who collects records has lost his capacity to care for his family but retained a capacity to care for old records: an example of a non-social object. Why is this distinction important to Albert, since in the end he thinks that his patient is loosing his capacity to care altogether? It seems from the conclusion of the conversation that Albert values a lot more the capacity to care for people than the other one, to the point were the first one literally defines what is a human being. The second one looks only tangentially to what humanity is. It is difficult though to follow the blurry conversation between Albert and the philosopher. The philosopher asks more or less implicitly a question that seems to be in keeping with one of her observation: the team of the Memory Clinic says –as I heard it often said- that FTD patients are “shallow” and that they lost their “humanity”. From her hesitations, from her smile and the cheerful tone she puts into her comments, she feels she tackles a delicate question; one that she actually does not have time to ask. Albert interrupts her and administers her a basic lesson in logic: the question (that he understood her asking) is circular. Listening to Albert’s final answer: I believe that the core of humanity is to be able to care about others, we can imagine that the mysterious question was: Is what you insist in saying, e.g. that caring really amount to caring for other humans, what you also define as the core of humanity? Yet, this question doesn’t seem circular to me, unless we take the answer as obvious. And it is not.

Nevertheless, not everyone at the Memory clinic is ready to conclude that FTD patients have lost their capacity to care. Oliver, a neurologist of the team, disputes Albert’s idea that we could know if these patients actually care or not.

Oliver (troubled). – How do you know these patients don’t care fully? If you ask that individual ‘Do you think it’s OK to spend 75% of the family money on old records?’... ‘You know, the family doesn’t have much money and cannot afford rent’... Many, many times the patients would say ‘No, it’s not OK’. So they could still express a value system, that actually is consistent with their value but they cannot longer act on these values. To me, they are like addicts.

Albert (smiling). – But this is just words... They don’t care fully.

Daniel (to Oliver). – It’s just words: the left hemisphere is very good at saying things, you know.

Oliver. – I don’t agree: there are things they wouldn’t do. If you ask them: ‘Should you murder people?’ They would say no and they don’t do it.

Daniel (smiling). – That requires drive...

Big laugh.

The philosopher (not laughing). – I don’t want to take a stand on this debate; I want to know from you... We talked about value but there is will. So you say they might have some value but not the will to carry it out? My intuition is that this kind of thing you are describing is more like addiction, this disorder of will.

Oliver. – But that’s what this is!
The philosopher. – Maybe but I am hearing something different over here! *(Pointing at Daniel and Albert seating close to one another).*

Oliver *(to the philosopher).* – They [the patients] are just missing information. Their emotions, which inform their decisions, are broken. Their sensation went away, they are missing vital information. Care or not is another problem.

The philosopher *(to Oliver).* – If you were dealing with an intact person who professed for instance how they love their children but never do the appropriate things: would you say they care?

Albert *(spiritedly).* – I would say they are sociopaths! *laughs.*

Oliver does not seem to think that the system for valuing is impaired for these patients, then he says that caring or not, one cannot say. For Oliver though, acknowledging that FTD patients have a trouble with their will, like in addiction, would be sufficient to explain what is going on. Moreover, Oliver acknowledges that these patients do say they care. Oliver agrees with Daniel and Albert (and Damasio) that the emotional system of FTD patients is broken. Then they disagree. For Oliver, this emotional impairment is to be put in relation with a disorder of the will (and the black boxes that come with it like “indifference” and “apathy”) that might explain the non-caring attitude of FTD patients. The role of will is also very important for the French neurologists to understand the impairment of FTD patients; we will see it in the conclusion. For Oliver, “not caring” seems to mainly be an impression one has about these patients but that is not rooted in any direct damage to their brain, however it would ensue from the impairment of their will and of their emotions.

For Daniel and Albert, if there is no doubt that patients have lost emotions and their will too (Daniel calls it ‘drive’), it is necessary to acknowledge that they have also lost ‘caring’ and that this loss does not ensue from the loss of emotion or will. Also, we shouldn’t trust what these patients say: the left brain is speaking not the “patient”, according to what Geschwind have taught us (see chapter 1). Therefore Daniel and Albert are seeing “caring” as a psychological notion lacking in FTD patients independently from any other trouble: lack of caring is created ex-nihilo by the disease. What is very importantly stated is that this loss of caring in FTD is analogous to what happens in sociopathy, which is understood as a loss of humanity. Considering this loss, the dismissal of what patients say is made easier. For Albert and Daniel, patients might say they care but they aren’t aware of what is going on with them. In this view, any psychiatric or behavioural troubles are created by the disease and cannot be the consequence of the adjustment to a cognitive impairment linked to the disease or to a relational trouble caused by the disease in the patient’s family.

How did the philosopher seize these different ideas about the capacity to care of FTD patients expressed that day by Oliver, Daniel and Albert? She came to the Memory Clinic to understand what neuroscientists had to say about this question, but they had contradictory views. What did the philosopher conclude from that discussion in the paper she presented two years after? I asked her that question and she kindly sent me her paper. I briefly describe her conclusions.

Overall the philosopher concludes that FTD patients have lost their capacity to care
altogether. She develops her argument in two steps: taking the example of the woman she already
mentioned in her talk, she’ll successively answer the questions: does this woman care about her
family (e.g. does she cares about people)? She’ll answer no. And, does this woman cares about
eating out at every meal (e.g. does she care about something else than people)? She’ll answer no.
With the analysis of the second question she refines her framework about care, and with her
answer to both questions she generalizes her conclusion to any patient with FTD. The philosopher
presented her conclusions in a philosophy conference in duo with Nicole the neuropsychologist.
Nicole’s presentation followed the philosopher’s and aimed at giving a neuroscientific
explanation to the conceptual work done by the philosopher. Hence these two women are
collaborating: the philosopher giving a conceptual validity to the neuroscientific project. I am
here only commenting on the conceptual work of the philosopher. There might be some
problems with her conclusions. I think she wants to settle an answer to her (and the
neuroscientists’) question too quickly.

To illustrate the dilemma: Is a FTD patient incapable of caring? The philosopher takes an
example of the woman that she discussed already in her talk. In the text she gives more details
about this patient. From these details I recognized this woman. We met a year before the
philosopher, when she came for the first time to the Memory Clinic. Back then, I remember that
the MRI left Daniel in no doubt that this woman was bearing the behavioral variant of FTD (bv-
FTD). This woman is young. She was 46 years old when I met her. Her troubles started when she
was 39. The philosopher calls her Karen. When I read the description I thought that Karen was
this woman I met, for two main reasons. Firstly, the description of this woman draws on the
report of her husband, which struck me at the time as peculiarly violent. Second, some of the facts
that he reported about his wife stuck in my mind because they were repeated over and over by the
team during the case conference.

The facts are the following: Karen, who was a stay-at-home mother, used to ‘be devoted’
to her husband and children. For the philosopher, there is no doubt that she was ‘caring’ for them:
the full set of the dispositional patterns (see her definition above) that characterize ‘caring’ were
focused on her husband, her daughter and her son.

According to her husband, however, it is clear that since she developed FTD, her wife
‘stopped caring’; I read in my notes that this expression was actually his. He also reported, the
philosopher tells us, that his wife was ‘emotionally flat’ and ‘does not know how to give and
receive affection’. In support of this verdict the team repeatedly mobilized a pair of events during
the case-conference, in which I participated: when Karen’s son was moving out to college, he
wanted a hug from his mother but Karen did not see any reason to get out of bed to say good-bye
and moreover to hug him. And, when her daughter asked her to accompany her to shop for a
prom dress, Karen refused to go.

I learn from the philosopher’s description that Karen lives now with her mother. When I
met her, Karen was living with her husband and children. She and her mother saw each other
quite a lot, which made the husband angry because the mother, he told us, is “probably the biggest
bitch I ever met in my life”. We don’t know what motivated the change of Karen’s living
situation. At that time though, the husband gave me the impression that living with his wife
became so unbearable that separation would be the logical step to avoid more familial drama. The
nurse, Beth, was very disconcerted by how he spoke of his wife. During the case-conference, this
is how I took into notes the way Beth laid out the situation:
Beth. – He [the husband] is kind of letting her [Karen] behind and the way he worked it out with his kids is against her: she is the problem. And so, she kind of retreats to her mother and to the things she kind of wants to do, because when she is in the family she is always messing up so she kind of goes elsewhere: she literally goes to her bedroom... (Sadly.) So I’m not sure what is going on. He is different from other caregivers... I guess I am more familiar with this kind of strategy where they gather the two worlds trying to take care of their partner and trying to take care of the kids, and I less commonly saw somebody who goes away with the kids, I think this is their strategy you know, because they make fun of her, they have little things to say on how she behaves...

In answering her first question: “Does Karen still cares about her family?” the philosopher relies exclusively on the husband’s report. She lays out her framework and concludes that Karen doesn’t care anymore: she has lost her emotional dispositions (“she is emotionally flat”, the husband said), her cognitive dispositions (she doesn’t call her children, and calls her husband only to “gossip”), her motivational dispositions (she didn’t want to take her daughter shopping for a prom dress), and her deliberative dispositions (she wants to go eat out every meal even though she is fully aware of how these excursions threaten the family finances). Yet, Karen told the philosopher that her husband and children are still important to her: what to make of that? asks the philosopher. Maybe to do justice to Oliver’s suggestions, the philosopher imagines that Karen would actually care about things even if she doesn’t give that impression. Thus, she imagines that one of the cognitive consequences of her disease would make her incapable of understanding the subtle social emotional need that supports, for example, the desire for a teenager to go shopping for a prom dress with her mother. However, she should understand some more raw emotional need (that the philosopher considers “context independent”) such as seeing her child hurt. We can wonder here what the philosopher would say about mammalians: when they nourish and protect their cubs do they “care” or not? Many would answer yes, not taking “care” only in the sense of an activity but also in the sense of a feeling. But even what cats and dogs could do, Karen cannot, and the philosopher bases her statement on what the husband speculated: that his wife would not be worried if she knew he was traveling in a place where a deadly storm raged: she might be very intrigued though by the information and might want to pass it on as a piece of “juicy gossip”. The philosopher then concludes that to hypothesize about a lack of caring coming from some cognitive impairment doesn’t do justice to the facts of FTD.

Yet, the conclusion is a bit quick since the philosopher doesn’t take into account the familial dynamic. What if the husband, as it looked, resented his wife to the point that he didn’t actually care for her anymore? Indeed one doesn’t need to be sick of FTD to dismiss his or her partner. Then what to make of his speculative statements about his wife’s capacity to ‘worry for him’? Besides, since the philosopher’s inquiry seems to examine a specific mode of being: ‘being someone who doesn’t care’, it seems important in order to grasp this mode of being, to take into account the context in which it takes place. The dismissal of the familial context and of the possibility that Karen reacts to this context does not seem to me to do any justice to Karen neither to a philosophical inquiry. Interestingly, the philosopher goes as far in her dismissal as to forget to ask the question: does Karen actually care for her mother?
This dismissal shows that the philosopher seized Albert and Daniel’s ideas about the capacity to care of FTD patients. I cannot explain why this idea (more than Oliver’s) is so attractive to the philosopher. I think though that before getting into a philosophical or an anthropological inquiry that targets people who are sick, one is responsible for trying to understand the peculiar way of thinking and being (so different than the norm) of these people. I think the philosopher didn’t assume fully what I take to be her responsibility to Karen.

The second part of the analysis tries to answer the question: does Karen care for anything at all (knowing that she doesn’t care for people)? Specifically: Does Karen care about ‘going out’? If she respects the framework she presented at her talk, the philosopher has to conclude that yes, Karen cares about going out. She gets emotional about it (prospect is exciting, hindrance is upsetting), she is cognitively engaged about it (the philosopher remarked that Karen was more attentive during the consultation when her husband complained about it), she is very motivated to eat out even if it is at a cost (upsetting the husband and risking her family’s finances), and eating out affects how she deliberates about other issues (even if she acquiesces about the need to curtail her expeditions, she forgets it as soon as the next opportunity presents). Moreover, this set of dispositions is lasting and persistent.

Yet the philosopher says she wants to resist the conclusion that FTD affects only one form of caring (about people) and spares another (caring about non-people). She says she wants to resist concluding this, because she trusts the intuition of the clinicians that suggest to her that FTD patients have lost their capacity to care altogether. The intuition of the clinicians is very important to the philosopher analysis, as much as that she will change her framework in order to prove this intuition right. Let’s look at the change she proposes.

In her old framework, she says we didn’t look carefully enough at the kind of activity people were engaging in, this is a mistake since some activities can qualify as a proper object of care, and others not. In order to avoid the confusion between desire and care, the activity that constitutes a proper object of care, must have a “reality” independent from anyone desire. This means that there are norms coming from this activity itself, to live up to. Alternatively, this means that some standards of flourishing or perfection are at work in the activity that constitutes a proper object of care. What are examples of activities with standards of flourishing/perfection or with norms to live up to? The philosopher gives some example: ‘Painting’ is one, ‘dancing’ is another; in both activities there are norms to live up to. Such activities are opposed to things like ‘taking drugs’ that don’t come with norms of when it is done well or badly. These definitions are confusing because the expression done well or badly conveys a quite different meaning than the expression to live up to: the second one seems closer to an ideal (like the idea of perfection) or to a utopia rather than what motivates the activity of playing piano for the majority of people. When

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233 Of course we know that here the philosopher is not thinking of Oliver’s intuition, which was precisely going in a different direction. The philosopher is thinking of Daniel’s and Albert’s intuition. But we might remember that if Albert said that patients with FTD would eventually lose the capacity to care altogether, he differentiated two stages depending on the progression of the disease. We remember that he said that the first stage is the loss of the capacity to care for human and social objects, and subsequently the loss will affect the capacity to care for non-social objects and other kind of things. Karen, if we admit she is at the first stage, could well still care about non-social objects, according to Albert. I don’t know why the philosopher does not take this part of the intuition into account. But two years passed by between the talk and the paper, and I wasn’t there, so maybe at the occasions of other discussions the intuition of the clinicians on that matter has changed.

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it seems quite difficult to say that the activity of taking drugs is one that has standards to live up to, I don’t agree that there are no standards of taking drugs well or badly. A heroin addict needs to prepare the shoot in a certain manner to avoid the overdose, with some standards of hygiene to avoid getting AIDS, in a certain place as not to be caught by the police, injecting it in a certain way and not anywhere on the body, etc.\(^\text{234}\) The philosopher takes the time to stress that what qualifies in her view as a proper object of care has nothing to do with the fact that the activity is considered good or bad by a certain society at a certain time. Thus, for her, being an assassin and being a Mafioso do qualify because these roles come with standards to do them well or badly. I don’t see why murdering people would get some norms and taking drugs wouldn’t. Both necessitate to be done well in order to not be caught nor killed.

With respect to Karen, the philosopher analyses the activity of going out with the new framework, and gives her answer right away: like with taking drugs there are no standards to live up to in the activity of eating out... But the philosopher feels she is going too fast: what if eating out masked another kind of motivation?\(^\text{235}\) Again, according to the husband, if Karen wants to go out to eat it is not for the taste of food (noble, to live up to) but for “entertainment”. Entertainment, because when Karen goes to the same five restaurants with her mother, she hopes that people will recognize them and will be willing to chitchat. But Karen is not seeking “friendships” -she does not care about people as we know already- all she wants is to “gossip” with them. The philosopher analyses that gossip does not qualify as a proper object of caring because it is not something that can be done well or badly. Is that true? I am not convinced that they are not bad or good ways to be gossiping: the notion of “juicy gossip” is not empty of meaning; an example of which -as the husband reminded us- is to know someone in an area devastated by a storm. Isn’t a juicy gossip a good gossip because it will capture the attention of the group of gossipers better than a boring gossip? Isn’t failing to capture the attention of the group of gossipers a way to fail in gossiping?

The philosopher will finally conclude that any FTD patients (at least any “behavioral variant FTD”) are incapable of caring. She extends her argument with other cases than Karen’s, also borrowed from tales of patients at the Memory clinic\(^\text{236}\): these patients, she concludes, do not

\(^{234}\) See Philip Bourgeois and Jeffrey Schonberg, Righteous Dopefiend, Berkeley, University of California Press, 2009, for a full description of heroin users’ practices. Also, Gilles Deleuze reflects in “B comme Boisson” (L’abecedaire, 2004) and in a course: “Cours Vincennes - St Denis : la Taverne - 24/02/1987” (source : webdeleuze.com), about the alcoholic as someone not only seeking for booze, but also seeking for a specific kind of alcohol (the taste is important), a specific environment made of sounds (clinking of glasses), seeking for company and conversations in cafes, a whole context that heals temporarily from solitude. There are thus other things to pay attention to in addiction then the mere desire for the drug. Deleuze goes as far as saying that it is a “practice” in a strict Aristotelian sense: you begin drinking so as to end perfectly: all drinks lead to the last drink.

\(^{235}\) As it will certainly prove to be the case: as long as one takes the time to ask and answer where Karen is going, to do what, with whom, in short as long as one gets interested in what she is actually doing rather than to catalog her way of being as meaningless.

\(^{236}\) For instance, this man diagnosed with FTD who “collected” plastic bottles: his only goal was to “collect bottles”: any bottle of any amount and he certainly didn’t do it with the idea of protecting the environment in mind. As well, many of these FTD patients who exercise at the gym “rigidly” every day at very specific hours; they are certainly not going to the gym for the sake of “health” the philosopher analyses, hence they don’t care about going to the gym. This last example strikes me as odd: what does it mean to do something for “health”? The philosopher says that there are standards about that, but she doesn’t give any example. Is it to go to the gym “to live better and longer”? Is it to go to the gym “to get protected from dementia” (one of the only treatment proposed at the Memory clinic to people
practice their activities with a standard of perfection in mind, hence they don’t care about what they are doing, hence they have lost the capacity to care.

The philosopher, at the end of her talk, attempts to define what constitutes an “obsession” (like Karen’s desire to “go out”) and differentiates it from “legitimate caring”: the presence of a standard of perfection is the criterion that she uses to differentiate legitimate caring from illegitimate caring, e.g., from obsession. Of course one problem with this analysis is that it is for the philosopher, and only for her, to tell us what is and what isn’t an activity with such standard, what is legitimate and what is not. But this difficulty does not appear to me as the biggest. If I find the philosopher’s attempt to define ‘obsession’ interesting because I do think it is of importance to understand the peculiar way of thinking and being of FTD patients (I am not denying that this way is different than other ways), I find very problematic her generalization from her observation of one activity (a specific activity someone is doing that she says does not qualify as “care”) to the whole capacity of these persons to actually “care”. The problem with her argument is not as much epistemological as it is ethical. When the philosopher infers from a specific action of a FTD patient that she has lost her capacity to care in anything that she is doing, she embraces the reductionism of the neuroscientists. I remember when I started my fieldwork at the Memory clinic I watched a video of Daniel (on you tube): I saw Daniel day dreaming in a bus, watching the streets of his favorite city passing by the window, he was reflecting on the relationships between neurosciences and philosophy: “I think the next philosophers, the philosophers of the 21st century, are going to be neuroscientists”, he said. We saw that this is already true: Damasio talking about Descartes’ Error, Daniel and Albert thinking that humanity is defined by empathy and caring; these are philosophical thesis more than scientific hypothesis. Then, are philosophers becoming neuroscientists too? At least, this is true for this philosopher who in her efforts to collaborate with Daniel, Nicole and Albert at the Memory clinic on Love and FTD, choose to embrace the neuroscientific’s method and paradigm.

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This chapter explored how a disease of the frontal lobes, due to a lesion caused by a tumor, a dramatic accident, the action of the surgeon in the form of a lobotomy, or a neurodegenerative disease, first called Pick’s disease and then FTD, has lead scientists to build over time the relationship between our brain and our sociality. Sociality is increasingly understood as tributary to our emotions, as favored by our empathy. This contemporary understanding has been also explored by Allan Young in The Social Brain and the Myth of Empathy, not from the study of a disease, but from two pervasive theories in contemporary neuroscience: the mirror neurons theory and neo-Darwinism. In the conclusion of The Social Brain, Young quotes an article fearing to get demented)? Some people do sport simply because they feel better afterwards, and the exercise does good only because it’s always redone. Actually, one may wonder: what is an “exercise”? Frederic Worms (in Revivre, Flammarion, Paris, 2012) asks this question about the remedies prescribed by Ancient philosophy, precisely the exercises. A practice he says, that has effects only through its repetition and doesn’t have an aim outside of itself, yet it can produce an “oeuvre”, but that is contains in its exercise. I wonder: if the philosopher were to include in her framework these “exercises” (prescribed, again, by philosophers) which are not done for the sake of performance but only for themselves; would they count as “legitimate” or “illegitimate caring”? 
published in *The New York Times* by David Brooks\(^{237}\), which is of interest to us. In *The New York Times*, one can read about a “revolution in consciousness” recently allowed by the neuroscientific understanding of the human. Brooks explains that the idea of consciousness inherited from the Enlightenment is now passé, instead we elevate: “Emotion over pure reason, social connections over individual choice, moral intuition over abstract logic, [and] perceptiveness over I.Q\(^{238}\).” Through the study of the knowledge of the frontal lobes we have observed the same “revolution”: over time a focus on emotion and social connection has developed in the world of brain science, today this understanding is fully established and even experimented with through the behaviors of FTD patients; at least this is the case in the American clinic. I will come back on the place of empathy in the diagnosis of FTD in both clinics in the conclusion of the dissertation.

The work done in this chapter leads me to conclude with Marc Augé\(^{239}\) that the term “medical anthropology” doesn’t have much relevance to anthropology, except for the administrative and financial interests that it may provide. Instead of trying to “build a new sub-discipline”\(^{240}\) (a sort of branch of medicine which aims at studying patient’s cultural conceptions in order to help the physicians) with boundaries that parcel out the field of anthropology, Augé favors over “medical anthropology”, the term “anthropology of disease [anthropologie de la maladie]” that takes ‘disease’ as an object of knowledge in a society -an object like any other- and which informs us about this society. The study of frontotemporal dementia revealed how neuroscientists who think they are philosophers and a philosopher who thinks she is a neuroscientist, have built and now disseminate, through conferences about FTD for the American lay public, through discussions with caregivers and scholarly conferences in philosophy, an understanding of the human in terms of empathy. Yet, an anthropology that focuses on “disease” instead of focusing on patient’s suffering --the kind of anthropology that I understand to be the proper object of study of medical anthropology-- doesn’t lead to evacuate pain from the framework. Because medicine is constituted by a patient, a doctor and a diagnosis, a treatment of the disease that focuses on doctors and diagnoses necessarily leaves space to the patient and her pain; which may not be central but which may come out through cracks\(^{241}\), as I hope we will see in the next two chapters.

Now, how do we go from the social to the individual? We’ve read about theories about humanity, sociality and empathy, yet we don’t know how the doctors diagnose the pathology of the social being in an individual patient. What happens to these interpretative schemes when they are facing a singularity? The next two chapters aim at describing how this knowledge is applied and also build, through the reality of the troubles and in the making of a diagnosis of dementia.

\(^{237}\) Columnist at the NYT and author of *The Social Animal: the Hidden Sources of Love, Character and Achievement*.


\(^{240}\) Ibid., p 82.

\(^{241}\) Thank you Lyle Fearnley for drawing my attention to this point.
The pianist is playing For Elise. The lady next to me is humming.
Jesus is talking to himself in Spanish.
The Russian lady with vaporous white hair speaks to the woman dressed in purple, they speak a lot.
The lady next to me stops humming and talks with Alex for two seconds.
I say Hello to Kim, she answers me with a smile.
There’s Beethoven.
Jesus whistles to call a nurse.
The lady next to me told me something then she went off. She seemed to have a cold.
I went to see Renée, she told me God bless papa and mama.
I speak with Alex, he is saying that he was a rabbi, he lights a candle for Chanukah, he says he is going to marry a Jewish woman and that he is from Berlin, he asks me if I am Jewish, he asks me N times if I am Jewish, he looks at my décolleté. Renée touches her breast. Jerome kisses my hand, the nurses laugh loud. They can’t stop laughing. Ruth asks the pianist to play a song, she feels sorry for him, she asks him if he gets paid. She doesn’t want to dance because she is not paid (she tells me about this woman Jane that she hates because she is stealing money from her everyday).
She tells me that many people here are sick including her (she says it twice) “They are sick in their brain, including me, if you had been through what I have been through these last ten days... I should write a book one day”. I propose to be her writer; she says she can only be a dancer. The pianist is done; he locks the piano and returns the keys with a quick dance. Alex is tired.
“I know a little bit about the state of the science of the brain and I know that no one knows much about that.”

A (demented) patient at the Memory Clinic.

“I mean, I think with all the lack of treatment that we deal with, the art of being a doctor is much more important where we work. Because it’s not like “OK, you’ve got that X cancer and we are going to plug you in with this protocol and we hope you don’t have that much side effects, and at least we know we did by numbers what seemed to be the most important thing for you”. We are in a much vaguer world here, and I bet you there is not a type that goes into this field; there is a type that goes into each field of medicine, but I bet you there is not one type that goes into the field of behavioral neurology. In some disciplines, I think there is a type, but not in this field: I think we are all different people.”

Dr. Blake to me, at the Memory clinic.
This chapter deals with some of the uncertainties that run through the practices of the teams of diagnosticians in the French and the American clinics. What has now become a whole chapter, I initially only intended it to be the introductory part of a chapter about the diagnosis, which is now chapter four. Writing about uncertainty was meant to be a sort of foil to introduce the need for a “clinical intuition” in situations where the diagnosis looked highly uncertain. I thus started to exemplify why some say that the science of the brain is uncertain, through a talk that the chief of the French clinic, Dr. Vincent, gave at the American clinic. Vincent’s talk stated how the diagnosis of Alzheimer’s disease has today become much more certain than it was, thanks to the improvement of new (and less recent) technologies. Yet, at the end of his talk, some American voices raised a few objections: the neuropathologist of the American clinic, Laura, underlined how the neuropathology of Alzheimer’s disease was still inconclusive in 30% of the cases and the chief of the Memory clinic, Dr. Daniel, presented an ambiguous case, for which Daniel’s clinical intuition proved wrong the supposed certitudes brought by the technological apparatus just praised by Vincent. Vincent concluded his talk admitting that no technologies could be compared to the clinician sense and experience. After I wrote this introductory story to what is now the next chapter, I thought that there was more to say about uncertainties than what Vincent’s talk and Laura and Daniel’s objections underlined. In the diagnosis of neurodegenerative diseases, there are interrogations even when things look certain. I had difficulties to find some cases to write the first section, which is about certitudes. There is also an unresolved ambiguity of the concept of a prodromal stage, or early stage, of dementia; this is the subject of the second section. There are situations of higher uncertainty than others: when the diagnosis involves the genetics of neurodegenerative diseases (and they are more and more involved); I give one example of that in the third section. Finally, the fourth section is about blurred situations where the limits between psychiatry, neurology and “normality” appear labile.

I do not intend to write a full inventory of the situations (that wouldn’t be complete anyways\textsuperscript{242}) that display uncertainty in the practice of the diagnosis of neurodegenerative diseases. I am here only wondering about some of the forms, nature and intensity that uncertainties can take. Needless to say, this chapter doesn’t call into question the existence of people called “demented” neither the reality of dementia facilities where Jesus, Ruth and Renée are --as some of us will surely be--, ending their lives. Writing about uncertainty in medicine also doesn’t amount to a denunciation of a knowledge that would be characterized by its irrationality; rather it is more to try to pose a question: how is medical work possible when doctors do not have the support of a stabilized knowledge? The actual body of knowledge on neurodegeneration (techniques in molecular biology and genetics, means to visualize the brain, etc.) is not absent from this chapter, yet it does not occupy a central position. The aim is not primarily to set out the state of our contemporary science of the brain, in which the neurologist would be the mediator between a general biological knowledge and an individual patient, but more to look at the central

\textsuperscript{242} In the diagnosis of a neurodegenerative disease, I can think of other moments that display uncertainty: for instance, when the doctors announce to the patient and his family that the patient has a brain disease, the family often asks the doctors to predict what will happen next and when it will; there is no way for the doctor to give them a sure prediction. How to answer then? Some stay elusive, some lie, other describe all the violence that is contained in the worst-case scenario. Another instance of uncertainty is sometimes raised by the patient, after being given the diagnosis (usually some time after): some patients I talked to ended up putting the diagnosis into question (and had reasonable arguments to do so).
position occupied by the neurologists when, in a certain situation of uncertainty, they will need to take (and sometimes to justify) a decision.

I. Certitudes.

Scene 1."MMS at three."

I am at Dr. Vincent’s private consultation at the Alzheimer Clinic in France. I am sitting by the window, facing the door and the large office of the chief of the Alzheimer clinic: a white and bright room on the ground floor of the building. Vincent is sitting at his desk style Louis XV making a phone call. He hangs up and calls the first patient of the morning. A woman and a man enter the office, they look in their mid-sixties\(^2\). They are accompanied by a younger and energetic woman; it’s their daughter. They take their places on the other side of the desk, in front of Vincent.

The daughter explains to Vincent that she wants power of attorney over her mother. Vincent is listening to her while reading the file of the mother, he mumbles: “Still living at home, MMS\(^3\) at three... MMS at three... OK...”

The mother. – What is this? MMS?

Vincent. – It’s a thermometer that allows us to measure your memory; it’s really low.

The mother. – And that’s not good?

Vincent (after a silence). – You know you have a disease?

The mother. – Yes, I know. It’s... pffff.... I know.

Vincent. – It affects memory.

The mother. – I know and we’re gonna get stuffed!

Vincent. – Why do you say that?

The mother. – I don’t know... We can’t do much about it...

Vincent (to the daughter and the husband). – She understands everything we say!

The mother (calm). – Yeah, I hear you...

\(^2\) I will learn later that the woman is actually 68 years old.

\(^3\) MMSE, here shortened to MMS, and sometimes abbreviated in the American clinic as Mini, stands for Mini Mental State Examination. This is the first test of a whole battery given to the patient. The score obtained to that test acts as a broad indicator of the capacities of the patient, especially memory capacities. Thirty is the maximum one can get.
Vincent (showing to the mother the picture of a bird). – How is that called Madam?

The mother. – It’s a... a... I see, I see...

The daughter (interrupting). – Her problem is that she is looking for words.

The mother (irritated). – Yes! Ah yes! This is so annoying245!

Vincent (calmly, to the mother). – Show me the window and then the door.

The mother laughs.

Vincent (same tone). – Show me the window and then the door.

The mother. – Well, the window... (She turns towards me and sees me:) Oh! Mademoiselle! (Laughing)... and ... (pointing at the door on the other side of the room) the door!

Vincent. – Very good!

The mother (offended). – Well! We are not moronic... well ... No! (Furious.) This... No! ... Or I... Or I...

Vincent. – Shush!

The discussion shifts then towards the question of the medications. The woman is already taking the medication Aricept; it thus seems that she was diagnosed with Alzheimer’s disease. Vincent decides to add antidepressants to the prescription, because he sees that “it is painful”. A long discussion follows about who is going to administer the antidepressant that needs to be taken at night time: the woman says she can take the meds herself, the husband says he can supervise his wife, the daughter trusts none of them and wants a nurse to come home to administer the meds, the parents refuse the presence of a nurse. For now they won: they’ll manage; Vincent allows for a trial period before he’ll reassess the situation. Vincent takes them back to the door, the woman turns towards me and smiles. Vincent closes the door.

Vincent (to me). – They are tiresome... (Low.) The husband is alcoholic... She is astonishing isn’t she?

Me. – And she has a diagnosis of Alzheimer’s disease? [The word has never been mentioned during the whole consultation].

Vincent. – Oh, Yes. This is certain, certain.

Vincent turns back to the door, opens it and calls in the next patient.

Vincent is certain that this woman has Alzheimer’s disease; I didn’t have time to ask him to explain to me why he is certain. According to the NIA-Alzheimer’s Association, one cannot be

245 An attempt to translate "ça m’énerme" instead of “ça m’énerve”. 
“certain” that someone has Alzheimer’s, frontotemporal dementia or any other neurodegenerative disease before the death of the patient. In theory, it is only at the moment of the autopsy of the person’s brain that the diagnosis can be confirmed with a maximum of certainty, through the analysis of certain molecular structures present inside the brain. This quantitative and qualitative analysis is in theory, the gold standard to determine if someone had a neurodegenerative disease and which one it was. Thus, the revised three-part report published in 2011 by the NIA-Alzheimer’s Association reasserts that the diagnosis of Alzheimer’s disease, such as it is recorded in charts during the patient’s life (or when it was not confirmed by autopsy), should be preceded by the qualifier “probable”.

However, as Vincent told us, it is possible to be certain that a person has Alzheimer’s before her death. Indeed in reality, very few autopsies are carried out, compared to the number of diagnoses of Alzheimer’s disease that are given every year in America or in France, and presented as “certain” to living patients and their family. What arguments support this certitude?

Actually, the certitude that Vincent displayed during his consultation is not limited to what he claims in his private practice. Vincent argues in the scientific literature and elsewhere that the “diagnosis can be established in vivo”: “no more reference to post-mortem is needed” thus the qualifier “probable” attached to the diagnosis becomes no longer necessary. It is today possible to diagnose Alzheimer’s “positively”, which means that Alzheimer’s is not anymore an “exclusionary diagnosis”, Vincent argues. When Alzheimer’s was (according to Vincent) an exclusionary diagnosis, the physician had to rule out every other possible cause for the symptoms before diagnosing Alzheimer’s. It was Alzheimer’s only if it was not another disease (depression, hypothyroidism, vitamin B12 deficiency, Parkinson’s disease, Huntington disease, stroke, a head trauma, etc.). What is the royal pathway today to access this disease positively, that didn’t exist before? Today, as before, the neurologist bases his diagnosis on his clinical expertise (this is a very important tool, we will see how in chapter four) and on the result of a neuropsychological test (of which the MMS is only a small sample). On top of these two fundamental tools, Vincent assesses the diagnosis with various biological markers: the MRI, the cerebrospinal fluid analysis, the


247 5.4 millions of Americans have Alzheimer’s disease in 2012 according to the ‘Facts and Figures’ of the Alzheimer’s Association (see http://www.alz.org/alzheimers_disease_facts_and_figures.asp). 860 000 French suffer from it according to the Ligue Européenne de la Maladie d’Alzheimer, but this number is underestimated: still according to the Ligue, only one patient upon two is correctly diagnosed. We don’t know what “incorrectly diagnosed” means.


249 This argument is one of Vincent’s main concerns: he talks about it in French newspapers, he teaches about it in his clinic to residents and medical students. He also developed this whole argument at the occasion of a conference he gave at the Memory clinic in July 2012, a conference where I was. I summarize here his argument based on his account during this conference and on Margaret Lock’s analysis of his articles (that I cannot cite directly in order to respect Vincent’s anonymity).

250 Fieldwork notes, Vincent’s conference at the Memory clinic in July 2012.

251 Idem.

252 Idem.

253 Magnetic Resonance Imaging: the most common scan to do for the patients who consult a neurologist in the American and French clinics. When the neurologist wonders about Alzheimer’s disease, he will look first at the form and volume of the hippocampi. The hippocampi are two structures (one on the right, one on the left) located in the
PiB\textsuperscript{255} scan or/and the presence of an autosomal dominant gene mutation for AD\textsuperscript{256}. In addition to the clinical diagnosis and the results of the neuropsychological test, a positive result on one (or more) of these markers (that Vincent calls “the biological footprints of the disease\textsuperscript{257}”) allows the neurologist to diagnose Alzheimer’s disease with certitude. This reasoning is formalized by what Vincent calls an “algorithm\textsuperscript{258}”. Among these biological tools, one cannot really say the MRI is a new tool\textsuperscript{259}. The relatively newer elements are the cerebrospinal fluid analysis and the PiB scan, we will see how they help the diagnosis in section two. The knowledge of the genetics of neurodegenerative diseases is growing; this is the main center of interest at the Memory clinic. We will see in section 3 of this chapter and in chapter 4 how the genes inform the diagnosis.

Going back to the consultation of the woman with Vincent, we learn that the result of her MMS was three: it is three points from zero, yes it is low. The maximum score is 30: below 27, neurologists begin to wonder if there is a “cognitive” problem and when a patient scores 23 it is already certain that there is a problem\textsuperscript{260}. We know that this woman has trouble to express herself: one possible clinical symptoms of AD. We don’t know what the “biological footprints of the disease” revealed, but they must have contributed to Vincent’s certitude. We know from the surprise expressed by Vincent (“She understands everything we say!” “She is astonishing”) that this woman should not be capable to understand everything. From these remarks, Vincent probably considers her very advanced in the disease. She shouldn’t even understand that she is sick\textsuperscript{261}, which she does (painfully). We know also that she takes the drug Aricept; this doesn’t tell us much. This drug is given to patients diagnosed with Alzheimer’s since the late 1980’s, and has been thought (and is sometimes still thought) to prevent momentarily this disease from progressing. I have witnessed several times the efficacy of that drug questioned by the very medio temporal lobe of the brain. They are thought to play a major role in memory (short term and long term memory) and in spatial navigation. In Alzheimer’s disease they are expected to be one of the first region of the brain that suffers damage. A way to appreciate their function is to measure their volume on a MRI. This measurement can be done at a quick glance or with the aid of a software. The smaller the volume, the poorer the memory.

\textsuperscript{255} The doctor punctures the cerebral spinal fluid in the marrow of the patient. The measurements of the levels of both the amyloid-beta and tau proteins indicate if the patient has Alzheimer’s or not: a low level of amyloid-beta combined with a high level of tau is believed to signify that the patient has AD.

\textsuperscript{256} This specific exam aims at showing the presence of a marker of Alzheimer’s disease in the brain of the patient: the amyloid-beta protein. A ligand (something that binds a protein) is injected to the patient. This ligand is a radioacative analog of thioflavin T that binds physiologically the protein beta-amyloid in the brain. This binding is visualized with the positron tomography scans. This specific exam - a PET scan with this ligand- is called a PiB scan for Pittsburgh compound B.


\textsuperscript{258} Fieldwork notes from Vincent’s conference at the Memory clinic in July 2012.

\textsuperscript{259} The first MRI scan on a human was made in 1977. MRI machines became commercially available in the 1980’s.

\textsuperscript{260} The web site of the Alzheimer’s association summarizes how the plethora of scientific articles interprets this pervasive test: “The maximum MMSE score is 30 points. A score of 20 to 24 suggests mild dementia, 13 to 20 suggests moderate dementia, and less than 12 indicates severe dementia. On average, the MMSE score of a person with Alzheimer’s declines about two to four points each year” (http://www.alz.org/alzheimers_disease_steps_to_diagnosis.asp).

\textsuperscript{261} Alzheimer’s patients like FTD patients, as Huntington patients, are considered anosognosic \textit{a priori}; they cannot be conscious of their physical and psychological symptoms.
neurologists who were prescribing it\textsuperscript{262}. Some neurologists think it has no effect. Others believe that this drug actually slows down Alzheimer’s disease. In any case, no neurologist would use the effect (or its absence) of this drug to confirm or deny a diagnosis of Alzheimer’s. This drug cannot put the diagnosis to the test. It is more generally impossible to test with a medication, a hypothesis of Alzheimer’s disease, frontotemporal dementia or any other neurodegenerative disease (with one exception\textsuperscript{263}). In the world of dementia, it is not possible to test a diagnosis hypothesis from the patient’s response to a treatment because there is no cure for these diseases.

From this, we learn one important thing about the medicine of neurodegenerative diseases: the objective that has been pursued for a thousand years by medicine, to cure, does not need to be actualized in order to build a rational medicine. To make a diagnosis, a very efficacious rationality (Vincent’s “algorithm”) is sufficient and does not need to be assisted by the power to heal.

\textit{Scene 2. “Mortified.”}

A man in his hospital bed is repeating endlessly the name of the hospital: “U.R. U.R. U.R. U.R....”

He has big eyebrows, he looks like Jacques Lacan. He is part of the research program at the Memory Clinic; he was diagnosed with FTD five years ago. He is 69. Michael is examining him for this yearly visit. The man came with his wife. After Michael and I introduced each other to the couple, the man pissed in his bed. He looked at me and stammered: “Mortified”, a nurse came in with gloves, he saw the gloves and said: “No, no, no, no”. Michael and I left the room while the bed sheets were getting changed.

When we come back, his wife tells us he is like a pet. While she says that, the man is looking at us all sat around his bed.

She’ll talk the whole time. He won’t answer the few questions that Michael will cautiously ask him. She summarizes how her husband got sick: things started seven years ago when he became very tired and had the feeling when walking that he was walking in the mud. At that time he became very demanding for sex (she rolls her eyes and smiles at me) and he started to get selfish and angry.

While she looks back on the first symptoms, she recalls that at first she was still able to “instruct” him about some things when he forgot how to do them. She remembers that even three years ago they were able to do things together. He

\textsuperscript{262} From the discussions I had with some neurologists who do not believe in the efficacy of Aricept but still prescribe it, I got that if it does not have a biological effect it still has a psychological effect: giving hope to the patient and his family and giving to the doctor the reassurance of being useful.

\textsuperscript{263} The exception is Parkinson’s disease: it is actually only after the treatment (L-dopamine) is successful in reversing the symptoms that the diagnosis of Parkinson’s disease can be firmly made. This reversal, however, is only for a certain period of time, called the ‘honey moon,’ because it does not last. Thus contrarily to Alzheimer’s and to the other neurodegenerative diseases, the diagnosis of Parkinson’s is given in a “definite” fashion by putting a medication to the test.
was still strong: he used to open cans for her. Today, she has to help him on the stairs so he doesn’t fall. Today, she says, “I am lucky if he knows who I am”. Michael asks her if she thinks he “understands”. She doesn’t think so. She started thinking he didn’t understand anymore when he stopped talking to her, three years ago. Yet, “he can still add numbers, he still enjoys puzzles, but not the kind that you and I would enjoy”; she shows us a child puzzle. And he “cares about music”. He takes heavy doses of sedative meds: Trazodone (antidepressant) and Risperidone (antipsychotic). Once, she tried half of a pill of the antipsychotic (he takes three of those every day) to see what it was like: it froze her mouth she tells me, she was unable to talk for hours. She wants to keep him “safe” though and as long as possible so he would be alive if a cure comes up; the meds help him stay safely in his bed.

Every piece of information has already been recorded during the previous years in the man’s chart, things seem to have been said over and over during these five years and nothing is really new or interesting to Michael. I got the same impression during the case-conference that followed Michael’s conversation with the wife.

Dr. Blake is the attending doctor for this case conference. As usual the team first gathers in the absence of the patient and of his caregiver so they can discuss freely about the patient. Actually, Blake tells us that he wants the couple out of the research program. In the first place, patients very far along in their disease do not teach them much anymore. Moreover the wife refused to promise to the Memory clinic the brain of her husband for autopsy. Blake justifies his wish to have the couple out from the program: the autopsy of the brain is a big contribution to the research; this man is obviously not contributing.

The case-conference will be short, no one is discussing the diagnosis: it is FTD.

Still, Blake wonders what is causing FTD in this man’s brain: what is the underlying pathology? Different proteins can cause this disease, so is it TDP-43 or tau264?

Blake. – Pathology is a 50-50 chances [tau or TDP]: I don’t know how to separate tau from TDP: Parkinson’s is tau, motoneurone265 is TDP, in an early paper the caudate atrophy was more associated with tau [the caudate of this man was seen atrophic on his brain scan]. But we can flip a coin…what did they say last year?

Michael. – They said tau first.

Blake. – If it is tau, it is Pick’s… I don’t think this could ever be AD, it is so frontal focal, but 10% of the people who have a behavior variant are AD. I

264 Tau and TDP43 (one component of ubiquitin protein) are two possible causes of different histopathological types of FTD. It is also possible to encounter FTD without encountering any specific histological anomalies in the brain, in those cases neither tau nor the ubiquitin proteins are damaged.

265 Blake is talking about motoneurone disease also called amyotrophic lateral sclerosis (ALS) or maladie de Charcot.
think it is Pick’s but I am never sure with behavior...

In this scene, the diagnosis was not in discussion: this man is affected by FTD (behavioral variant). What is discussed is the cause: What would Blake find in this man’s brain (if only this man could give his brain to the Memory clinic)? What is the protein causing these symptoms? What is the underlying neuropathology?

At the Memory clinic, this kind of guessing game is common. I never witnessed it in the French clinic. The French team guesses about diagnoses, this is sufficient. They do not speculate about the neuropathology. One reason might be that at the Alzheimer clinic, as François told me, “we have almost never confirmation [with the autopsy]” (despite it being a major research center in the country); there is not much point asking a question to which one wouldn’t be able to ever give an answer. At the Memory clinic, there is a “brain donation program”, and each patient who enters the research program is asked to sign a consent form to donate his or her brain to the clinic; some refuse and some accept.

The question asked by Dr. Blake is actually a hard one, worth the riddles of the Sphinx. It is a difficult question because the causal link between the symptoms and the cerebral proteins involved in the troubles has not yet been established. How is a particular protein (tau, TDP43, amyloid- beta, etc.) found in excess in the autopsied brain producing the symptoms? No one knows, yet.

This question is partly at the source heart of The Alzheimer’s Conundrum described by Margaret Lock. With regards to Alzheimer’s, two proteins are suspected to cause the disease, the amyloid-beta protein and the protein tau, because they are found in excess in the brain of patients diagnosed with Alzheimer’s disease. The problem is that one third of normal, non-demented people, show Alzheimer’s neuropathology post-mortem: a combination of amyloid plaques and neurofibrillary tangles. Some neurologists assume that if those people had lived longer they would have developed Alzheimer’s disease or another type of dementia. Other researchers argue that these structures formed essentially by the two proteins, amyloid-beta and tau, supposed to be the signifiers of Alzheimer’s disease, are maybe not that significant. In any case, it is unclear if these two proteins are the cause of the disease (if they cause the destruction of neurons), if they are its consequence (a way for the neuron to defend itself) or if they are neither of those. As Dr.

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266 I was told that 30 patients part of a research program that covers all France to which six French clinics participate are autopsied each year (at least in 2012). Around one third of this 30 patients come from the Alzheimer clinic: so ten diagnoses get confirmed each year by the autopsy. Compare to the Memory clinic where... This is why Vincent insists so much on the validity of the biomarkers, whereas Daniel is more reticent; for Vincent the biomarkers’ levels give the biological tangibility to the diagnoses made in his clinic.

267 One of the difficulties, as Margaret Lock summarizes it, is that Alzheimer’s disease pathology “has never been explained” Margaret Lock, The Alzheimer Conundrum, Entanglements of Dementia and Aging. Princeton University Press, 2013, p3.

268 Since the end of the 1980’s, the research for diagnosis and treating Alzheimer’s has mainly focused on the study of the amyloid-beta protein, without much result regarding the treatment. The believers in the causality of tau protein in Alzheimer’s disease, the “Tausists”, think the “Baptists” are loosing everyone’s time. Daniel is definitely a Tausist, whereas Vincent seems to me more like a Baptist.

269 One could also put forward the Louis Pasteur’s idea of a “terrain”: some people might be insensitive to the presence of these proteins. The role of the milieu in the susceptibility of people to present symptoms is assumed in the Alzheimer’s world by the concept of the “intellectual reserve”. An intellectual reserve (that concerns the more educated) is supposed to slow down the progression of the disease, for some it just the disease.
Blake would conclude: “I think we are very far away from treatment because we don’t know what the problem is. It’s probably not plaques and tangles, that make AD, it’s probably a response...”; this uncertainty indeed constitutes a serious obstacle for therapeutic interventions.

With regards to FTD, the problem gets worse. If Christian Derouesné questions the “uniqueness” of Alzheimer’s disease\textsuperscript{270}, he also reminds us\textsuperscript{271} that FTD is far more complex: no clear parallelism can be established between the “phenotypes” (behavioral variant, semantic variant or non-fluent variant) and the neuropathology (tau, TDP-43 or neither of those) and the genetic data (the number of genes susceptible to cause FTD increased recently and complicate the problem\textsuperscript{272}).

How does Blake think he may have an answer to the question he asked himself? He cannot know, but again this kind of bet is very frequent at the Memory clinic: last year the team who saw the man bet on tau. The team likes to test their guesses; it is not only to play with chance. With a little bit of luck it might be possible to build a new form of expertise even in the absence of any known relationship between the pathology and the symptomatology of FTD. Last year, they said tau first, TDP second. Presenting the bet in this manner, with an order, reconstructs what a future autopsy report would look like: interestingly the autopsy report never concludes the presence of one single protein but always to two (or more), in order of frequency. Blake says: “If it is tau, it is Pick’s\textsuperscript{273}”: this is how Pick’s disease is identified and defined: by the observation of intraneuronal inclusions tau reactive called the Pick’s bodies\textsuperscript{274}. But just when the diagnosis looked certain, thinking about the autopsy results allows Blake to express a doubt: “I don’t think it could ever be AD”. He argues that: “It is so frontal focal”, in line with what Arnold Pick described\textsuperscript{275} and after him Delay, Brion and Escourolle\textsuperscript{276}: the lesions of frontotemporal dementia are confined (‘focal’) and especially localized in the frontal lobe: FTD is what is not AD, and as a result one should find no lesions of the AD type at the brain’s autopsy. Yet, Blake explains that 10% of people diagnosed with the behavioral variant FTD have lesions of the AD type and in fact


\textsuperscript{272} The genetic forms of FTD are believed today to count for 30 to 50% of all the cases. Mutations have been found on chromosome 17 (several mutations involving the gene MAPT but also the progranulin gene), chromosomes 3 and 9 (very recently, a mutation of one gene on K9 are suspected to cause FTD and/or ALS or Parkinson’s disease). Some mutations like the one of the gene MAPT can be responsible for autosomal dominant forms or just be a risk factor for FTD. Moreover, the relationships between the mutation and the phenotype is very variable (in Derouesné Ibid.)

\textsuperscript{273} There are other FTD associated with anomalies of the tau protein: FTD caused by a mutation on chromosome 17 (FTD-17), and FTD’s associated with two other neurological diseases: Cortico Basal Degeneration (CBD) and Progressive Supranuclear Palsy (PSP). So tau doesn’t necessarily means Pick’s, but Blake is here reasoning from what he knows of the man: it cannot be linked to K17, neither CBD neither PSP: there is only Pick’s disease left if the disease is linked to tau.

\textsuperscript{274} Pick’s disease became today a subcategory of FTD, which diagnosis can only be done when the histological exam shows these famous Pick’s bodies. It was not the case before the 1980’s: before the Swedish modified the classification Pick’s disease was also a clinical diagnosis. FTD replaced Pick’s in the realm of the clinic.

\textsuperscript{275} The description of the circumscribed character of the lesions is Arnold Pick’s.

\textsuperscript{276} See Chapter 1.
“are AD” (remember the neuropathology is the gold standard). This seems to suffice to challenge, for a moment at least, his clinical impression: “I am never sure with behavior”, he said. Yet, after having met the man and his wife, Blake concludes: “I think it’s Pick’s disease…fairly advanced... With a MMS of 12! ... How bad MMS is to detect FTD!”

**Scene 3. “Ninety-nine years old.”**

Back to Vincent’s private consultation. An old woman comes in, alone. She is walking slowly, dragging her feet.

Vincent *(watching the old woman).* – Why do you shuffle like that?

The old woman. – Because it’s age.

Vincent *(reading her file).* – 95 years old, indeed...

The old woman *(distractedly).* – I don’t know...

Vincent. – You were born in 1916, so you’re going to turn 95.

The old woman *(uninterested).* – Well, yes.

The old woman has white hair with purple highlights, she wears a green coat and a royal blue dress. Her two hands are resting on Vincent’s desk, her purse is tied around her neck.

Vincent *(smiling at her).* – It’s been 15 years that we know each other; you were a kid at the time! *(Reading her file.)* Under Aricept... O.K. ... I’m going to do a little MMS, to kind of see where we are.

Vincent begins with the first question of the MMS: the date. The old woman doesn’t know the year, she knows the day and the month. She doesn’t know the region of France we are in. Vincent says “hat, lemon, car”. And asks her to repeat the words right away. She repeats hat and lemon but not car. Vincent doesn’t bother having her memorize and having her repeat the words after the traditional one-minute delay. He moves on to the next task. She copies the drawing, she follows well Vincent’s instructions (take this piece of paper with your right hand, fold it and place it on the floor), she writes a sentence that Vincent reads aloud: “Time strides along towards autumn”. She gets 11 points.

Vincent. – Very good and you made very nice drawings. How is life? Everything well?

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277 This kind of surprise goes both way. The Primary Progressive Aphasia (APP) of the Logopenic type, is seen as one manifestation of Alzheimer’s disease. At the autopsy though if 54% of the lesions belong to the Alzheimer’s neuropathology, 23% belongs to the FTD neuropathology, and the remaining 23% are described as “various lesions”. In Harris JM, Gall C, Thompson J, Richardson AMT, Neary D, du Plessis D, et al. Classification and pathology of primary progressive aphasia. Neurology 2013; 81: 1832-9.

278 This observation is pervasive, everyone at the Memory clinic complains about the usefulness of the MMS to diagnose FTD patients.
The old woman. – It’s a banal life.
Vincent. – Banal...?
The old woman. – We are many.
Vincent. – What?
The old woman. – We are many.
Vincent (after thinking). – Mmmm, you are in a home.
The old woman. – Yes, in a home.
Vincent. – It’s nice?
The old woman. – It could be if people were well behaved.
Vincent. – The staff?
The old woman. – Oh no, the staff is very nice.
Vincent. – This is good.
The old woman. – No, the people who live there... (With contempt.) They are a little...
Vincent. – They come to your room?
The old woman. – Oh no!
Vincent. – So what’s going on?
The old woman. – They talk very loud...
Vincent. – There might be some nice people too; did you make any friends?
Vincent. – Are you unhappy there?
The old woman. – Oh no! Not at all!
Vincent. – You are happy to be there?
The old woman (wearily). – Well, when my husband died there was no other solution.
Vincent. – How is the mood?
The old woman. – Bad.
Vincent. – Why?
The old woman. – Because I cannot go out, because people are uninteresting, nothing interests them.
Vincent. – OK, we’re going to write to your GP.
Vincent dictates a letter for the doctor: he mentions the score to the MMS, calls attention to a “deterioration of intellectual and cognitive efficiency” and suggests antidepressants.
Vincent. – OK, very good, it was nice to see you again.

Vincent escorts the old woman back to the door.

After he closed the door, I asked Vincent if he had diagnosed the old woman with Alzheimer’s. Impatient to pursue his consultation, he answered me shortly: “What’s the point of doing a diagnosis at 95 or 90 years old? All this problematic becomes irrelevant.” I cautiously asked him to explain. He said: “When people are very old it’s not interesting: we make a diagnosis of dementia but not necessarily of Alzheimer’s; the older one gets, the more difficult it becomes.” Actually, Vincent’s statement goes against what the layman may think, but even to what a medical student may think. Nicolas the resident at the Alzheimer clinic once asked a medical student: “What is Alzheimer’s for you?” The medical student without thinking said: “For me it’s a dementia mega associated with old age.” “No”, corrected Nicolas: “It’s a cortical neurodegenerative disease.”

In the chart of the old woman, Vincent wrote “demented”. The term “dementia” is unspecific regarding the cause of the trouble. “Dementia”, of course, can be caused by Alzheimer’s disease, but also by any other neurodegenerative diseases or by diabetes or by a stroke or even by depression. Old age dooms the neurologist to be unspecific, this is how the “very old” becomes “uninteresting”: by challenging the clinical expertise of the clinician. Vincent is not the only one to be defied by aging. A psychiatrist interviewed by Margaret Lock reports to the anthropologist his puzzle in front of people older than 80: “It is impossible to be precise when making a diagnosis” because of the multiplicity of factors at play. Like Vincent he hides in the ambiguity: his “most common diagnosis for people over 80 is ’mixed dementia’”. Why is it more difficult to diagnose “the very old” than to diagnose the “young”? Because aging alters the “biological footprints of the disease”: with age it is not unusual to find atrophy in the brain; the MRI may look abnormal even if the person is quite “normal”. Second, as the neurologist confides to Lock, the neuropsychological test looses its specificity for the “very old”: “What is a normal baseline at this age [95 years old]... We really have little idea.”

The royal pathway that allowed the neurologist with the aid of his algorithm to access to the positivity of the disease becomes, in the case of the very old, a muddy road. There is a milieu—a “terrain” wrote Louis Pasteur-- that troubles Vincent’s rationality. There is indeed a parallelism between Vincent’s difficulties and Pasteur facing the question: “Why in contact with the same germs some fall sick and other don’t?” Pasteur then carefully distinguished the question of the germ and of its propagation and the question of the milieu. The problem here is a bit different: not to know why people get sick and other don’t, but if they are sick differently? Is Alzheimer’s disease actually the same disease when it targets the old compared to the young? This question

279 Tom Dening in The Alzheimer Conundrum p 58 and p 59.
280 Tom Dening mentions psycho-social factors, another neurologist (David Dunn) interviewed by the anthropologist mentions “diabetes and high blood pressure”, p 59.
281 Margaret Lock cited epidemiological research that showed that the brains of individuals over seventy-five are remarkably similar, whether or not these people were demented in life.
283 This is a question that provokes much debates, I won’t get into the details of this question but for a great analysis of this debate in the light of what Aloïs Alzheimer thought about this question I suggest the reading of Fabrice Gzil’s dissertation: “Problèmes philosophiques soulevés par la maladie d’Alzheimer”, Université Paris I, 2007.
doesn’t have a clear answer but could be considered by the neurologists as the entry point for an empirical exploration of the complexity of the human brain. Vincent seems to find little interest in exploring scientifically that question[^284^], but as we saw his disinterest doesn’t exclude caring for the old woman (for 15 years). He discussed with her, inquiring about her (banal) life, and instituted a sort of treatment: same as the younger woman who was diagnosed with AD with certainty, the old woman diagnosed with dementia is being prescribed an antidepressant (on top of the Aricept that she is already taking[^285^]). In terms of medication, it didn’t change anything to have a firm diagnosis of Alzheimer’s and a firm (even if imprecise diagnosis) of dementia.

If old people are of little interest to Vincent, the same is true at the Memory clinic. In both clinics, neurologists are more interested in “young” than in “old” people (let’s admit the ambiguity of these categories because this is how the neurologists talk). Pierre, a neurologist who comes every so often to see patients in the French clinic, explained to me that the disease appears purer in young people: unpolluted, he says, by the “crystallization of behaviors”. “Young”, however, doesn’t always describe a civil age, it seems sometimes to refer to the age of the disease: patients advanced in their disease – American neurologists say they have a “full blown Alzheimer’s (or FTD)”, French neurologists say “it” is “un Alzheimer bien cogné[^286^]”-- as were patients in Scene 1 and 2, are less interesting to the neurologists than patients who are new, unpolluted by the work of time, sort of virgins to the disease. Patients come every day to these clinics because they, or their general practitioner, are worried that they might have this widespread disease --it is an “epidemic[^287^]”-- called Alzheimer’s, to which they hope these teams of experts have a solution. The Memory clinic and the Alzheimer clinic also implements numerous research programs which aim to find a cure for these various diseases and thus recruits patients early into their illness and even sometimes recruits asymptomatic patients. I did not see so often in these clinics many patients that are said to be those “dead without cadaver” like Lula, the wife of Serge Rezvani[^288^], or like this man who looked like Jacques Lacan, whose “neuronal souls[^289^]” have inexorably been destroyed by these brain diseases. The vast majority of the patients I encountered in the French and the American clinics, live a pretty normal life and the work of these experts, in most cases, aims at spotting subtle symptoms that are expressed in the “prodromal[^290^] stage” of these diseases.

To describe this prodromal stage, a word was invented in 2001: Mild Cognitive Impairment (MCI). In the next paragraph, I describe the uncertainty that emerges with the conceptual problems raised by MCI.

[^284^]: Still, during this follow-up, Vincent mobilized a technological tool-- a “little MMS” to “kind of see where we are”[^284^]-- to test her “intellect”, to assess her “degradation” and the evolution of the “dementia”.

[^285^]: Even if she was not formally diagnosed with Alzheimer’s.

[^286^]: “Un Alzheimer bien cogné”. Cogné is here used as an adjective, which is not proper French. This made-up adjective derives from the verb “cogner”, that can mean to knock (a screw into the wall), or by extension to bang with violence (one’s head often). It seems that it is more in the second sense that it is employed here: “A well banged Alzheimer’s”...

[^287^]: The web site of the Alzheimer Association mentions these “quick facts”: “Every 67 seconds someone in the united States develops Alzheimer’s”, “More than 5 million Americans are living with the disease”, “Women are the epicenter of the Alzheimer’s epidemic”. At http://www.alz.org/alzheimers_disease_facts_and_figures.asp


[^289^]: See Serge Rezvani, L’éclipse.

[^290^]: Prodrome, from Greek pro: before, dromos: act of running, is the premonitory symptom of a disease.

Scene 4. “To be continued...”

At the Alzheimer’s clinic, François, the neurologist, is going over the files of the patients seen this morning by Nicolas the resident, the neuropsychologists and the psychologists. In order to make a diagnosis, François is gathering all the information got by those who saw the patients. It is noon at the Alzheimer’s clinic; it is time for the synthèse.

The case of a woman is discussed, people found her a little bit depressed but the psychologist didn’t have time to see her yet, so no firm diagnosis of depression has been made. Nicolas, the resident, is quickly making his report to François. Cécile, the neuropsychologist who tested the woman, is already gone so Nicolas is speaking in her place.

Nicolas. – MMS at 29. It is mostly a dysexecutive syndrome with the BREF291 at 12. That being said I thought that she was rambling on...

François. – The problem is that you can’t say “amnestic MCI” because the dysexecutive syndrome is too important.

Nicolas. – “Multiple domain MCI” then...?

François. – Fine. And a MRI? Is there any?

Nicolas. – Yes.

Nicolas opens up the MRI on the computer, François and Nicolas are looking at the screen.

François. – There is a discrete atrophy. For 73 years old it’s really moderate, not major... So in total: “Multiple domain MCI” in a depressive context? (Looking at the psychologist) You’ll tell us... right? (The psychologist nods.) OK... To be continued.

Nicolas. – Yeah.

These last twenty years, diagnosing earlier has been made the priority for research on Alzheimer’s disease and on neurodegenerative diseases as a whole. For one fundamental reason: if there is any hope for cure(s), they will not be able to reverse the damage already done by these diseases in the brain. The objective is to diagnose Alzheimer’s, frontotemporal dementia, and all other sort of neurodegenerative diseases, as early as possible and ideally before any symptoms.

291 Batterie Rapide d’Efficience Frontale: Rapid Battery of Frontal Efficiency (my translation), this ensemble of tests is not used at the Memory Clinic. As we might remember from chapter two a frontal impairment (especially the impairment of the dorso-lateral part of the frontal lobe) could be revealed by this kind of tests which investigate our executive capacities.
show. Studying patients earlier also allows for a better understanding of the specificity of the symptoms, indeed all these neurodegenerative diseases lead in the end to ‘dementia’. In the end, all patients appear the same to neurologists: demented; at that stage there is no way to know what the patient is really dying from. Thus when treatments will be available for these diseases, neurologists will ideally have 1. Learned how to detect subtle symptoms and diagnose correctly each of these neurodegenerative diseases in their earliest stage and therefore 2. Give the accurate drug to treat the disease (each disease will get a different treatment) to sane patients but who surely are on their way to dementia.

How would it be possible to diagnose Alzheimer’s earlier than when the patient has symptoms of dementia? The hypothesis is that before the person shows any symptoms, the disease is already there, “working” in the brain. Daniel and Vincent assure us that the disease is at work 20 to 30 years before the symptoms finally show. This hypothesis is valid for Alzheimer’s disease, for frontotemporal dementia, and every other neurodegenerative disease.

Since 2001, the idea of a ‘prodromal stage of dementia’ has been formally recognized as ‘MCI’: Mild Cognitive Impairment. MCI diagnosis was created for patients with memory complaints, which are considered as on the road to Alzheimer’s disease. Today, as we saw in Scene 4, MCI can encompass any kind of trouble, not necessarily a memory trouble; now MCI is a stage that can precede any (or every) type of dementia.

MCI “diagnosis” is meant to describe a sort of intermediary between “dementia” (a diagnosis of Alzheimer’s disease for instance) and “normal aging”. “Neither normal, neither demented” is one of the criteria of the consensus report of 2004. There are numerous conceptual problems posed by this diagnosis. One of them is actually its poor capacity in predicting the evolution of patients; so this concept does not fill very well its ‘role’ as a prodrome. Depending on the studies, one-third to two thirds of the patients who bear this diagnosis will actually convert to Alzheimer’s disease in the year, without being possible to ascertain who will convert before they do. The remainder of the patients will keep the same status quo for years, or will actually reverse to normality; there are several examples of those in one of the research program at the Memory Clinic. Hence, the fateful question: is MCI a pre-dementia stage for Alzheimer’s disease? Or is it only a risk factor for Alzheimer’s disease? The response to this question is unsettled. If it is a risk factor, it seems then that it cannot be considered as a diagnosis (about a present state), but more as some piece of information about the future.

In scene 4, at the Alzheimer clinic, the vagueness of the conclusion seems enough to disqualify MCI as a diagnosis. In this scene, MCI is fully unspecific: the neuropsychological test showed some trouble in the executive domain but not in the memory domain, yet Nicolas found that the woman was “rambling on”: MCI will be “multi-domain”. MCI can go along with depression; it is not even possible to differentiate between both (whereas depression is a differential diagnosis for Alzheimer’s disease). Finally, “diagnosing MCI” is an activity that doesn’t seem to grip the doctors. Yet from this scene, it seems impossible to know how the

292 Field notes from the Vincent’s conference in July 2012.
294 Responses vary according to the one who answers the question; Margaret Lock in The Alzheimer’s Conundrum documents their variety from interviews she did with different neurologists.
doctors considered the term MCI for their patient --as a risk factor or as a diagnosis--, because we don’t know how they think MCI will affect the life of their patient: will knowing she has multiple-domain MCI transform her life, or will this information leave her as indifferent as her doctors? The way to know would have been to follow Nicolas and François when they went to talk to their patient to deliver her their conclusions, but I wasn’t with them at that time. The next two scenes, though, show that MCI can be considered as a diagnosis, even if it is not presented to the patient as such. The next two scenes, confront the conceptual problems posed by MCI with decisions doctors need to take: How the neurologists tackle the uncertainty that comes with MCI? What kind of decision do they take when they diagnose patients with such an elusive concept?

**Scene 5. “MCI-ish”**

We are at the Memory Clinic. The medical team is talking of a patient who came this morning with her son. The patient is an old lady from New York. She is 92 years old. Dr. Martin is the attending doctor leading the discussion.

Dr Martin. – She sounds like MCI-ish... MCI is such a difficult concept. The net positive to our field is for people you don’t want to label as having Alzheimer’s. If our society was better at making judgment... when you label someone as having AD it changes their life dramatically, but MCI does not that much, but concept wise...

John (the fellow interrupts to give his sentiment). – What bugs me is the heterogeneity...

Dr Martin. – John Morris does not believe there is something like MCI. He says that it is for those who cannot decide who has AD; so a slowly progressive decline he calls it AD even if they don’t meet the criteria for dementia.

Dr Martin acknowledges the complexity of the concept. Is he also slightly envious of the eminent neurologist John Morris, who does not “believe” in MCI? Dr Morris does not use the word MCI at all, but the term “early Alzheimer’s”. For his part, Martin analyses that there are needs for such a concept, even if they are more social than scientific: Martin sees MCI as a euphemism for Alzheimer’s whose effect on the beholders, in our current society, might be too violent. Scientifically, however, even if Dr Martin does not elaborate, essentially he seems to contemplate the vagueness of the concept -“MCI-ish”- while John thinks that MCI is unspecific: too “heterogeneous”; MCI leaves Martin and John perplexed. Their irresolution is apparent in the way Dr Martin delivers his conclusion to his patient.

Later Dr Martin meets the old lady and her son. After testing her with questions about the latest news (a volcano erupted in Iceland) and the name of the actual president of the USA, Dr Martin gives his diagnosis.

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295 On this issue, see John Morris in a lively conversation with Margaret Lock in *The Alzheimer’s Conundrum* (p 92).
Dr Martin (to the old lady). – So you told me your doctor said you had Alzheimer’s, I think we would call it MCI, which will almost always but not always lead to Alzheimer’s. It’s an intermediate zone, you fit more into this category. We are convinced that you have a problem, not only of getting older, there is a problem, it’s a serious problem, it’s not fine because we call it MCI and in my opinion the likelihood is pretty good that it will lead to Alzheimer’s. So the problem is memory but other things look in pretty good shape: you can take care of yourself - we rely on the information you and your son are telling us- and this is the chief distinction between MCI and Alzheimer’s.

Little silence. Dr Martin looks at the old lady and at her son, both remain quiet.

Dr Martin. – So I want to stress that your MCI will lead to Alzheimer’s.

The old lady. – If I live until then: I’m 92.

A silence.

Among the information Dr Martin delivered to his patient, the uncertainty carried by the diagnostic category MCI is high: “In my opinion the likelihood is pretty good”, “an intermediate zone”, “almost always but not always”, but it is at odds with what Dr Martin wants to convey: the certitude that the old lady will eventually have Alzheimer’s disease: “It’s a serious problem, it’s not fine”, “we are convinced”, and the final conclusion: “I want to stress that your MCI will lead to Alzheimer’s”... When? “When” is not something that Dr Martin can predict precisely. Samuel Beckett, in his Proust, writes about the future: “As long as the future cannot be situated with precision, as long as a date hasn’t been assigned, the event to come remains indistinct, we cannot perceive the consequences.” For the old lady, Martin’s prediction is as blurred, as abstract as the eventuality of her own death. The prediction pertains to “the water of the future, lifeless, pale, monochrome (…) It appears to us exempt from the bitterness tied to fatality.” The situation is very different for Dr. Martin. Dr. Martin first zigzags between the idea that MCI is a risk factor for AD and the idea suggested by John Morris: that MCI is AD. In the end, when Martin delivers his fateful prediction, he embraces Morris’ idea. Martin must think the old lady has Alzheimer’s already; the event Alzheimer’s disease, for Martin, belongs to the present. When Martin stresses that the old lady will end up with Alzheimer’s, he is not only giving her a piece of information (a risk) about her future, he is making a diagnosis (about her present state). MCI is not a diagnosis; Alzheimer’s is a diagnosis. Indeed there is a nuance between both: according to the current definition, MCI does not guarantee Alzheimer’s. MCI doesn’t guarantee anything. I don’t know on which scientific ground Martin bases the equation MCI=Alzheimer’s: from his previous conversation with John, we know that nothing was settled. It seems that Martin made up his mind and chose Morris’ camp while he was talking to the old lady. This prediction is for Martin as

297 Ibid., p 25.
298 The discrepancy between the vague prediction of Dr Martin and the response of the old lady, as if they referred to two temporalities that did not meet, is also described by Aude Beliard in her thesis. Beliard examines how the neurologists inquire about the appearance of the symptoms of Alzheimer’s disease: during this inquiry she remarks that patient’s time and doctor’s time do not necessarily coincide. See Aude Beliard, “Des familles bouleversées. Aux prises avec le registre diagnostic Alzheimer”. PhD dissertation, Université Paris Vincennes Saint Denis, 2010.
colored and agitated as the “water of the past” that Beckett contrasts with the water of the future. As a result it might transform decisively the future of the old lady.

Dr Martin. – So the question is what is going to happen... I would be concerned by your ability to stay on your own. It would be reasonable to think about the next step: right now you are independent but it would be good to think about assisted living...

The old lady. – A woman is coming a few times a week...

Dr Martin. – To be honest for the moment it could work but for how long? In other words, what is gonna happen if no one can keep an eye on you, if you get into trouble?

The old lady (tense). – But I have a home down there and you want me to leave that?

Dr Martin. – What do you think of this business of assisted living?

The old lady (coldly). – I don’t like it, it’s not home like, it’s an institution.

The son. – These places can be nice, I was visiting those people, there is a continuum of care...

The old lady (sights, tired). – I should let it go and let someone do all of it.

A silence.

The son (to Dr Martin). – And what about the drugs she is taking? [The old lady takes Aricept].

Martin. – We have a diagnosis: we think she should stay on these drugs; they are fine. But (to the son:) is she taking them? That’s something you should keep an eye on.

The son. – She’s good [at taking them].

The old lady. – Those medications don’t do that much, they don’t do that much good...

The son. – Who knows if they do anything...

Dr Martin. – Who knows if they do good, but you know the hope is more that they stabilize...

The old lady (a little bit disgusted). – The prediction about the future is as vague and as negative [as before the visit to the Memory Clinic].

Dr Martin (swiftly and prophetic). – Let’s do an MRI! Sometimes looking at the scan changes our opinion but I suspect it won’t.

The son (laughing). – Maybe you’ll find a cockroach!

The old lady is not laughing.
Scene 6. The struggle.

It is Friday afternoon at the Memory Clinic, the team just discussed the case of a woman, Mrs. Y, who is here in the context of a banal consultation (like the old lady she isn’t part of a research program): she is worried that her memory is “getting worse”. Oliver is the attending physician and after discussion with the fellow and the neuropsychologist, he diagnoses her MCI. The woman and her husband are not here yet and Oliver is in discussion with himself so to speak— it is a soliloquy— and tries to decide if he should prescribe the woman the drug Aricept.

Olivier (to himself and to the team). – So first we gave Aricept like water and over time we realized it had some cardio effects; in the clinical trials the MCI had a slighter higher death rate... So everybody got a little nervous. On the other hand she has MCI but we think she has AD... MCI is a very human way to say you have a brain problem... That said these are the very people who were in the trial and Aricept did not improve them. How do you deal with that? So, I struggle a little bit: there is a medicine and it is effective for AD but not for MCI. Does Dr Daniel diagnose them with AD? He would say: “early AD”.

The efficiency of the drug Aricept for improving memory is controversial. Some neurologists say it has no effect, neither for MCI patients nor for AD patients: Daniel at the Memory Clinic, Pierre at the Alzheimer’s clinic, something that was suggested too by Martin in Scene 5: Martin didn’t have much to oppose, other than faith, when the old lady and her son disqualified the drug. On the other side, some neurologists say it improves memory, at least for a while: that day Oliver seemed to believe so, François, a neurologist at the Alzheimer’s clinic, believes it too299. In his soliloquy, Oliver didn’t question only the efficiency of the drug for MCI patients, he also underlined some side effects of Aricept. Oliver’s problem appears to be the following: studies have shown that Aricept has a beneficial effect only for people with AD but not for those diagnosed MCI, since it also has a detrimental effect, is it worth prescribing it for MCI patients? Obviously the answer is no. But what to do for this woman that Oliver diagnoses MCI but who is actually “thought” to have AD? MCI diagnosis, as Oliver develops, is only the euphemism for AD diagnosis. It is the nice and human way to say that there is a “brain problem”. It seems that for Oliver, like for Martin in the previous scene, MCI means —nicely—AD; isn’t Daniel himself, the chief of the Memory clinic, calling MCI “early AD”?

So, is there really a problem? Oliver could announce to the woman she has “MCI” (even if he thinks she has early AD) and would prescribe her the drug he prescribes to AD patients (it doesn’t matter that this drug is detrimental for patients diagnosed with MCI since Oliver thinks she doesn’t have MCI). Yet, Oliver seems to be bothered by the discrepancy between what he

299 In 2011, the Haute Autorité de Santé in France, re-evaluated the benefit of Aricept and of two other medications (Exelon and Reminyl) given to treat Alzheimer’s symptomatology The HAS concluded that these medications had a « weak therapeutic interest ».
believes to be the case and what the studies have shown: for those people labelled MCI, Aricept didn’t work as well (or at all) as for people labelled AD. Then, in those studies, there must be a difference --maybe a biological one, maybe a psychological one-- between MCI and AD. According to these studies, MCI is not necessarily AD. So is there a difference between MCI and AD, or are MCI and AD the same? As such, the question will be left unsolved. Yet, Oliver needs to take a decision for his patient.

The woman, Mrs. Y came with her husband. They are seated close to Oliver, at the end of the table.

Oliver (to Mrs. Y). – What makes you think your memory is worse today?

Mrs. Y (anxious). – When I want to say something and cannot say it. I hope I’ll never get it [AD].

Oliver. – What about you Mr. Y?

Mr Y (reassuring). – No, I think it is a normal process of aging.

Mrs. Y looks in panic.

Oliver (after a silence). – OK... (To Mrs. Y:) So you think it is getting worse and that tells us something; over the years we learned that patients who developed AD had [first] memory complains. So we moved from reassuring everybody like “don’t worry about AD, AD is only when you have [actual] memory problems” to “mmmm, maybe yes maybe not”... So now let me talk about this term dementia that you mentioned [Mrs. Y actually didn’t say that word, she said “it”]: that whole thing about dementia doesn’t apply to you at least now. The question is: will you get there? And there is no way to answer. Only time will tell. In the literature a term appeared: MCI... and this is how I think of you; there is something to worry about but only time will tell. Exercise is very important in this context, we don’t know what it does but we do think from animals’ studies that it is good for the brain. Aricept keeps your memory a little better than what it would be if not taking medicine: in a year you would still be able to do these things [that she does well today]... but I cannot be sure of that... It is probably a good idea [to take it] but it’s up to you.

Mrs. Y says she doesn’t like to take medicine in general.

Mr Y. – If you were her would you be on that medicine?

Oliver. – I think so, yeah.

A silence.

Oliver. – I propose to start with half dose.

Mrs. Y. – And when you stop the medication it doesn’t do anything to your brain?

Oliver. – No. It’s your decision, it’s up to you, but I think it’s worth it.... OK, so do you want to give it a try?
In the end, Oliver acted as if Mrs. Y had AD, and prescribed her some Aricept, yet he suggested her to participate to a research program on MCI, to learn more about her.

Oliver explained to the couple what he learned over the years: people like Mrs. Y who came to see him with mere memory complaints, could end up having Alzheimer’s. Memory complaints might be a prodrome for the disease and the diagnosis ‘MCI’ captures this eventuality. Oliver expresses also a number of uncertainties. He cannot be sure Mrs. Y will end up with Alzheimer’s: only Time knows. He suggests a drug, Aricept. When Mr. Y asks Oliver to put himself in Mrs. Y’s shoes and asks: “Would you take it?” Oliver is positive. Yet he is not sure the drug will have preserved Mrs. Y’s memory in one year’s time, or that it will eclipse her complaints. Moreover, Oliver concludes that medicine doesn’t know much about MCI and tries to recruit the couple into a research program at the Memory clinic. The couple will enroll into the program. I cannot be sure of why Mrs. Y and Mr. Y accepted to be part of it because I didn’t ask them, but it seems to me that when one faces so many questions one may be tempted to learn more; participating in the research program might be seen as a good mean. But will the MRI and the tests done in the research program bring Mrs. And Mr. Y some answers?

The tests done in the research program are neuropsychological tests, MRIs —they will be repeated every six months to see if Mrs. Y stays level, declines or gets better (it happens)— and tests that measure the levels of some biomarkers. These later tests are of two kinds: the analysis of biomarkers (amyloid-beta and tau proteins) found in the cerebrospinal fluid (CSF) of the patient and the PiB scan\textsuperscript{300} that visualize the deposition of the amyloid-beta protein into the brain. At the Alzheimer’s clinic, Dr. Vincent strongly advocates\textsuperscript{301} for the analysis of the biomarkers found in the CSF to diagnose patients with early AD, and thus to differentiate them from patients with a mere MCI\textsuperscript{302}, or with a depression, or with frontotemporal dementia etc.\textsuperscript{303} Everyday Dr Vincent

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\textsuperscript{300} The fixation of a ligand to the amyloid-beta protein is visualized on the PET scan of the patient: this ensemble is called a PiB scan.

\textsuperscript{301} Vincent advocates in the scientific literature and in front of the whole neurology department of his hospital.

\textsuperscript{302} Note that if the analysis of the biomarkers might “convert” some ‘MCI’ patients into ‘Alzheimer’s’ patients, it is not a solution to the conceptual problem posed by the diagnosis MCI. The question of the status of MCI —a risk factor for AD or early AD—is left unsolved by the result of the analysis of the biomarkers. A positive result means, for Vincent, that the patient has AD. But a negative result, for a patient with a dubious neuropsychological test doesn’t prevent the clinician to label the patient MCI.
prescribes this test to some of his patients; at the Alzheimer clinic, the analysis of the CSF is considered by many neurologists as the new gold standard. By contrast, at the Memory clinic, this test is rarely done in the context of everyday consultations\textsuperscript{304}, but is frequently done, in conjunction with the PiB scan, in the context of a research program\textsuperscript{305}; in the end though, in the American clinic, the CSF analysis doesn’t participate as much as in the French clinic in the diagnostic decision\textsuperscript{306}.

I had once a discussion about the utility of the biomarkers analysis in the CSF and the certitude it brought into the diagnosis, with Elise. Elise is a neurologist at the Alzheimer clinic. She is interested in thinking about what she is doing, and interested in telling it to me. She talks patiently, with a lot of pauses. That day, Elise just saw a patient whose CSF analysis was negative. She was relieved: “Luckily” she said, “it was negative... I was worried that it would come back half significant”. I asked Elise about the accuracy of the test, I was thinking of the risk of false positives. As we know, 30% of normal non-demented people show AD post-mortem pathology, the test measures the same proteins that are identified at the autopsy: tau and beta-amyloid proteins. Elise told me:

“Technically, this exam has a good specificity, I would say around 90%: it’s close to 100%... but we are not there yet. And what do we do when only one biomarker comes back positive and not both? We flounder a bit. And the more we search the less we find, this can deprive us from our clinical intuition. But, my colleague Mona, if you ask her, she’ll tell you that biology prevails over the clinic... It’s interesting: it means that for her, biology cannot lie. Biology doesn’t lie, but how do we interpret it? How do we interpret this level that we measure?”

I told Elise that indeed compared to the American clinic, I had the impression that the French diagnosticians trusted the biology over the clinic, whereas at the Memory clinic in the case of an ambiguous patient, the clinicians would have trusted their clinical intuition over the biological results... Elise was surprised. She thought the Americans were much more “formal”, she said. This idea, sometimes came out as a critique on the part of the French neurologists I met, who when they asked me to tell them about the differences between their practices and the American practices, without giving me the time to answer, assumed that the Americans were mediocre.

\textsuperscript{303} For Vincent, the analysis of the biomarkers combine with the clinical work, the neuropsychological test and the MRI is the real and new gold standard for the diagnosis of Alzheimer’s disease; this ensemble replaces the autopsy.

\textsuperscript{304} The American neurologists use a lot less the analysis of the CSF in the context of everyday consultations than the French neurologists. The cost of this biological analysis is taken care of by the national health system in France; it is free of charge for French patients. It is not the case in the US where it costs at least a thousand dollars to the patient. Thus in the American clinic, this analysis is reserved to patients who are, either willing to pay that price (and the neurologists provide no incentive for doing so) or enrolled in a research program. If patients are enrolled in a research program, the cost is being taken care of by the program, however for reliability questions (which is an important problem) the CSF of the patient is being sent to a lab situated at the other side of the country and the result will come back to the Memory clinic... only two years after that.

\textsuperscript{305} The PiB scan is not practiced at the Alzheimer clinic because this exam demands the presence of a cyclotron at proximity, and there is none in the vicinity of the French clinic.

\textsuperscript{306} As I explained in footnote 55, the American team look for the biomarkers in the CSF but mainly in the context of research program. Yet, since they get the results two years after sending the CSF, this result doesn’t participate much to the making of the diagnosis. This is not true for the PiB scan, they get the results almost immediately.
clinicians (but good biologists), while the French were good clinicians but couldn’t compete with the Americans on the ground of lab research.

I insisted: I told Elise that I was surprised to notice how frequently the neurologists in the French clinic were prescribing the biomarkers analysis in the CSF, how significant it was for them to make a diagnosis. Elise agreed that it was very pervasive at the Alzheimer clinic, although she wasn’t the one to use it the most:

“Yes here, I think that we do it a lot. I still don’t know what to think about it in the context of individual practice. It is very satisfying when everything matches up, we feel almighty, we do a great and clean work, that’s what we wanted, isn’t that what we are looking for? I mean who will that help...? That’s the question I ask myself, is it for us? Or does it get the patient any further? It’s more for research purposes... Yes... in a way... yes, we need to be fair: it’s to build some coherent data. It’s good at the same time because we make our expertise, the more results we have the more we know if they are reliable or not, the more we learn how to interpret them... and since we are an ‘expert center’... If we don’t do that who will? At the same time, in the clinic we are always towed by research but if we are trying to be honest: does that help the patient? There is no treatment. For the very young forms, I don’t doubt about the usefulness: I think we need to be very rigorous so we don’t miss an inflammatory disease. But for those who have an MRI and a neuropsychological test that kind of speak for themselves... before we were pleased with that, and we waited: ‘On laissait le temps au temps’.”

Doctors acquired knowledge with that test: it allows them to detect people with Alzheimer’s. Time used to have that knowledge, now it is medicine. This is a good thing for research, but is it good for the patient? This is the painful problem that Elise wonders about, since there is no cure. Beyond this detecting limit, medicine is no longer competent. Doctors are not omnipotent, that’s a given, but what is knowledge, in medicine, without the power to cure? In the next scene and paragraph, Daniel, the chief of the Memory clinic faces the same nagging question, but this time with an even more powerful biological tool: the genetics. The genetics of neurodegenerative disease constitutes the most interesting biological matter to Daniel. People who have or had a demented parent for whom a gene is the principal suspect of the disease come more and more often with their whole family to meet the chief of the Memory clinic. These families, usually asymptomatic persons, will go through the traditional neuropsychological tests, MRIs, PiB scan, they will meet --like every other patient—first a fellow and then they will meet Daniel. Then they will get an extra test: a blood sample to look for the mutated gene.

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307 Baptiste Moutaud in his dissertation, reports the same idea from the French neurologists he worked with. In “C’est un problème neurologique ou psychiatrique?” Paris Descartes, 2009, p 77.

308 For instance, the neurologist shouldn’t confuse Alzheimer’s disease with Multiple sclerosis.

309 A possible translation could be: “We let time take its course.”
Scene 7. High uncertainty.

Jack. – Lucia really doesn’t have... I mean both of them don’t have a lot of symptoms. I mean both of them were really trying hard to tell me a lot of things to make sure I was not missing anything, but nothing they told me qualifies as a symptom.

Daniel (to Jack). – She [Lucia] would not be here if it was not for the fact that we are doing a study on gene carriers?

Jack. – Probably.

Daniel (to Olivia). – Do you agree with that?

Olivia (authoritatively). – Yes, absolutely.

We are at the Memory clinic in a conference room, the one that is looking onto the stadium. It is around one p.m., the stadium is nearly empty; high-school kids haven’t arrived yet, neither the late afternoon runners. Jack, the fellow, is presenting two cases to the chief of the Memory Clinic: a sister and her brother. The room is full of visitors newly arrived from Spain, Brazil and China. The regulars of the Memory clinic are here as well: the chief Daniel, the nurse Beth, two neuropsychologists, Olivia and Emily who respectively tested the sister and the brother, and Kim the genetic counselor.

Kim is here because the sister and the brother had a sibling, Aaron, who was diagnosed with AD; he died of it when he was 69. After his death, the team at the Memory clinic found a mutation in one of Aaron’s gene: the APP (Amyloid Beta Precursor Protein) gene; a mutation that might have caused him this deadly disease. Aaron’s brother José and Aaron’s sister Lucia who are now in their sixties, have no symptoms of AD whatsoever but they want to know if they carry the same gene as their brother. But things are not that simple. The mutation in the gene APP found on Aaron’s 21st chromosome seems to bring more problems than answers. At one point, Kim explains why:

Kim. – So there was a variant found in Aaron: it’s like ... it’s a glutamate to glutamine change and it’s in the region where most deleterious mutations on APP are found. Although this particular variant has never been reported before. (Low voice.) So that actually has been an issue... with this family. So that variant was found in their brother and at some point I think there was either a miscommunication or a misunderstanding with the family because I think the family walked away thinking that... that was definitely disease causing... I mean you know in all likeliness it probably is, but unfortunately it has never been reported before so there is absolutely no data... I think Dr. Martin spoke with them [the siblings], and they really want to know if they do have it...

310 Glutamate and Glutamine are two different amino acids.
Someone sighs.

Kim. – And so Martin basically told them we’re still... (Articulating each word.) JUST NOT SURE...

Daniel (confident). – The pathology [found in Aaron’s brain at the autopsy] was sort of also family looking: there was a big chunk of amyloid ramping in the cortex and ... I think it’s a mutation.

Olivia. – Mmmmm.

Kim (preoccupied). – I mean... Martin seems... was hesitant to... firmly condemn on the fact that it is pathogenic and I think they [the siblings] were somewhat frustrated with that...

Daniel. – Well, it would be nice if we could take a stand on this for them...

Beth. – Yeah, I know.

Daniel (animatedly). – Because John Morris in the DIAN studies has been following people longitudinally and they are going to choose a drug to treat gene carriers with and I think in this case it would be nice if they could be included in the DIAN.

A little silence.

A resident – How often your guys have brand new unsure clinical significance mutations...?

A silence. Daniel is looking at last year reports. A chair cracks.

Kim (mumbling). – Sss...o... (To Daniel.) Do you want to comment on that ...? (Shyly, voice almost inaudible.) Daniel...

Daniel (to the resident). – Say the question again...

The resident. – I mean, I was just wondering, if you had this type of thing happened before where you see new mutations that have not been clearly documented?

Daniel (circumspect). – Mmmmm ... I don’t know... I am not sure Martin and I disagree but Aaron’s pathology really looked like APP, you know: MA-SS-ive amyloid chunks, and I don’t think this is a benign variant: I mean we looked for it in 5000 controls...

The visitor from Spain (excited). – Yeah? 5000 subjects...

Daniel (with delight). – Yeah. I think this is why we are here: to find new genes and you know, I think there are a lot more equivocal genes than... We find all the time variants... But I think, we’ve done good diligence around this one: we’ve looked at the healthy controls and not only that but the pathology in the brother was what made us start to think about APP... so you know, so it was really... I think this is real mutation.

The DIAN (Dominantly Inherited Alzheimer Network), a study conducted by the
eminent John Morris, is designed for children of a parent who carried a mutated gene causing dominantly inherited Alzheimer’s disease\textsuperscript{311}. Did Aaron carry such a gene? No one has reported a case with the mutation found in Aaron’s genotype before; Aaron, for now, is the only one. Is this glutamine to glutamate change a “silence” mutation with no effect on its bearer? Or was a new mutation causing Alzheimer’s disease discovered at the Memory clinic? If the mutation has never been reported before it is not possible to be sure it is actually deleterious. Some mutations are not deleterious; Aaron could have carried it with no consequence for him. Search for this specific mutation was carried on in 5000 healthy “controls”, and was never found. This is the available data: 5000 people unaffected by Alzheimer’s (“controls”) do not carry this mutation. But without more “cases” (affected people) one cannot know for sure if this mutation is correlated to a higher likelihood to develop Alzheimer’s disease.

Dr. Martin thought he couldn’t take a stand on the question: he explained to the siblings that nothing is sure. In any case, the siblings want to know if they carry that gene. If they learn they do carry that gene, how are they going to live with an uncertainty specially created for them by medicine? This question is never clearly asked by anyone in the room but seems to lie behind the little silences and hesitations breaking in Kim’s sentences. Moreover, how is medical work and more specifically how is Daniel’s work, possible on such an unsteady knowledge?

Daniel only mildly agrees with Martin’s decision to maintain the family in this high uncertainty situation: Daniel thinks it would be “nice” to “take a stand”. Also, Daniel seems to think, unlike Martin, that the researchers at the Memory Clinic have been conscientious enough (testing 5000 controls for Aaron’s mutation) and got very close to the evidence that the mutation is deleterious. But there is more: there is the autopsy of Aaron’s brain that showed a “sort of family looking” pattern, the amyloid protein aggregated in a way that is “sort of” typical of genes carriers. From the autopsy then, Daniel concluded: “This is real mutation”. This is the provisory conclusion for now.

But a question remains: are the siblings carrying this mutation? For now, in the conference room no one knows their gene status except Kim. It thus comes to Jack, and to the neuropsychologists Olivia and Emily, to give their impression of the siblings. Jack said at the beginning of this scene, that neither Lucia nor José have anything that “qualifies as a symptom”. Olivia, who tested Lucia, confirmed to Daniel that the woman wouldn’t be there if no one had told her about the potential deadly gene of her brother. Yet, when comes Olivia’s turn to report on Lucia’s tests, the impression we got at the opening of the conference is going to change slightly.

Olivia tells us that Lucia’s tests were good except for her ‘visual memory’\textsuperscript{312}, which was “impaired”. Pressed by Daniel to guess if Lucia is a gene carrier, Olivia answers: “I think something was wrong...” As for Daniel, he says he is “worried”. Before hearing Lucia’s results to the tests, Daniel “was 50/50” that she was a gene carrier. After Olivia’s report, he’d “bumped it up to 2/3 likely

\textsuperscript{311} Persons included in the study can be asymptomatic or not, can carry themselves the dreadful gene or not. Dominantly inherited means that it is sufficient to have one allele of the mutated gene to be sick. Morris’ research is an ”observational study” as well as clinical trials to test three kinds of drugs.

\textsuperscript{312} Visual memory is tested by asking the person to copy a fairly complex geometrical figure and to remember it. The person will then have to redraw the figure from memory and finally to recognize the figure among four similar ones. Lucia didn’t succeed much at copying the figure, neither at drawing back from memory and she didn’t recognize it.
that she is a gene carrier”, based on her results to the test (especially her drawing and her memory of it).

Emily, who tested José, begins her report by asking if anyone of us got, like she did: “A weird vibe from him...I don’t know, he was very friendly with me”, she says. A visitor present in the conference room --a woman too--, who met José, had the same impression of “overfriendliness”. Asked by Daniel to specify what José exactly told her, Emily’s answer is quite disappointing: “Nothing inappropriate, he just wanted to hear about my training... and that kind of stuff”. According to the results to the tests, Emily tells us that José was better in “visual memory than his sis” but “executive functions were less than I would want”, “memory was low... a little low for his age”, and “he had trouble understanding the instructions”. Daniel presses Emily to divine.

Daniel. – What’s your likelihood that he is a gene carrier?

Emily (after a silence). – I think he is a gene carrier.

Daniel. – 90%?

Emily (clapping vigorously the pile of tests against the table). – 95%!

Olivia. – WOW!

Daniel, unlike what he did for Lucia, doesn’t give his impression about José. He is waiting to see the scans that “might show some atrophy”. But José’s scan doesn’t show much, it is “so subtle” says Daniel, that “it is on the edge of me calling it normal”. At first, Lucia’s scan looks “worse than her brother”, then Daniel finds that “they are kind of like twins” so much so that Jack, for a moment, cannot distinguish one from the other: “You are sure it’s her scan?” he wonders. It is hers indeed; Daniel gives us his impressions about Lucia’s scan: “not so much atrophy”, “maybe a little frontal” and “a little posterior I guess...” Daniel shows to Olivia the back of Lucia’s brain on the scan: “Maybe that’s her ‘visual test’...?” Without waiting for her answer, Daniel says to himself: “Yeah I guess there is atrophy back there, I’ll accept that... I think she is a gene carrier”. Daniel, then, consolidates his verdict by a strange observation.

Daniel. – It’s funny somebody... You know if you are a FTD gene carrier you are less likely to participate in research... But somebody told me that, Oh! (Looking at Olivia: you were telling me, that John Ringman who got a lot of presenilin mutations in Mexico [these genetic mutations are responsible for AD], in these families if you participate in research, you know, pre-symptomatically, you’re much more likely to be a gene carrier...

313 Lucia’s weakness lied in her inability to copy properly a figure and to, a fortiori, remember it correctly. This weakness is supposed to be associated with a lack of function of the posterior brain: the occipital cortex (grossly) involved with vision. The hippocampi involved in memory and spatial navigation are also classic candidates for the topography of Alzheimer’s disease but Lucia’s hippocampi are, according to Daniel, “kind of spared”.

314 John Ringman is a neurologist leading a study on gene carriers of the presenilin mutation --a gene predisposing for Alzheimer’s disease- a mutation apparently quite prevalent in Mexico.
The visitor from Spain (laughing). – Oh...yeah? A priori...?

Daniel (joyful). – Yeah (Laughing and crackling.) Yeah... So I guess maybe it wouldn’t be a coincidence that we have two patients...

A small silence.

Kim (softly). – We’ve had her [Lucia’s] blood for some time now...

The whole conversation happened as if the mutation found in Aaron’s gene was actually causing the brain disease; if there was an uncertainty on this matter at one point, it has now become obsolete.

Emily risked a 95% chance for José to be affected. Daniel, himself, risked numbers only on Lucia: from one chance upon two, to two chances upon three, until he simply came to think that she “is a gene carrier”. Still, numerous hesitations lay against these affirmations and their assessments: “Maybe... I guess”, “On the edge of normal”, “I’ll accept that” as if Daniel, here, was negotiating with someone else other than himself about the “atrophy back there”.

Daniel then, remembers what Olivia told him at one point: in John Ringman’s study on children of parents affected with AD due to the mutation of the gene “presenilin”, children carrying the presenilin gene (without knowing it and asymptomatic) were more likely to participate in the study than the ones not carrying the gene (without knowing it either and as much asymptomatic)... Daniel infers from this observation that Lucia and José, since they are participating in Daniel’s study, are likely to be “patients”, e.g. to carry the gene. The neurologist visiting from Spain catches the strangeness of this observation: Yes! At the Memory clinic it might be possible to make a diagnosis “a priori”. The clinical exam, the tests, the MRI, the discussion, the whole diagnosis apparatus, even the genetic test, can be invalidated by Daniel answering this simple, unsophisticated question: a priori, is someone sitting in my office yes or no?

In John Ringman’s study though, people are carrying a certain gene (presenilin) whereas José and Lucia are supposed to carry another one (APP). Still the observation is valid for both genes: the main thing is that those two different genes predispose people to the same disease: Alzheimer’s. Participating in a research program on AD genes is thus becoming a symptom of AD. Unlike, “participating in research program on FTD genes”: not a symptom of FTD (but not willing to participate to the same program is a symptom of FTD). Here, the very possibility to contrast AD with FTD, --because FTD and AD are, as I developed in chapter one, two diseases opposed in every manner--, was significant in elevating “participating in research” to the rank of a symptom of Alzheimer’s disease.

In a situation characterized by its very high uncertainty -- patient don’t know if they carry the gene, doctors neither, no one knows for sure if carrying the gene is actually a problem, in the case it’s a problem there is no remedy to it--., there are ways for neurologists to carry on with their work. The first step, we’ve seen it, is to deny the dilemma: Daniel found that Aaron’s autopsy was telling enough: from the “chunks of amyloid” in Aaron’s brain, Daniel could tell he was a gene carrier. The autopsy, for Daniel, settled what Dr. Martin considered as shaky: the mutation is now considered deleterious.

The second step, as it came to light with Daniel’s last observation, is to use the uncertainty of the situation to build certainty. If Lucia and José are here today it is because medicine created
questions for them. Daniel said it at the beginning of the conference: Lucia wouldn’t be here if it wasn’t to participate in Daniel’s research. She is here because there is a question, raised by medicine, unanswered by medicine, and to which she wants an answer. It is possible that Lucia hesitated before coming to the Memory clinic. It is possible that she talked about the genetic test with her brother José, and her other siblings (they are eight siblings). It is possible that she finally decided to come to the Memory clinic because she had a reason to come: it is possible the reason was that she wanted to know she didn’t carry the gene\textsuperscript{315}. This is how A.R, after she learned her mother had Huntington disease\textsuperscript{316}, explained her decision to do the test. A.R. writes:

I wanted to know to not be sick. To repel medicine and the disease from me. I was sure I wasn’t carrying it, I was not saying it aloud, but I was at the same time afraid and confident: I don’t have this thing. If I had it, I would have felt it, I’ll feel it. But feel what, this I didn’t know yet. I also knew that as long as I wasn’t doing the test, I would be haunted by a doubt, like a house can be haunted by a very burdensome phantom that never cease to upset night and day\textsuperscript{317}.

I cannot know what Lucia thought before she decided to come to the Memory clinic. What I know is that Daniel sees Lucia’s desire to know as a manifestation of her genetic make-up. This way to approach Lucia’s desire, flouts any kind of agency on the part of the patient. The fact that Lucia is there, reveals that this gene is in her. Backwards, it means that it is because she has the gene that she came: it is the gene who is the agent. Daniel is the worthy heir of Norman Geschwind, the father of behavioral neurology and phenomenology: for Geschwind, the left brain of the patient was speaking in her place, fifty years later for Daniel, the gene drives the patient to the neurologist’s office. And if the patient has a gene that causes AD she’ll show up\textsuperscript{318}. Yet, genes have their peculiar way to oblige the patient: some attract the patient to the doctor, some repel the patient from the doctor. This typology is, unsurprisingly, not reported in the scientific literature; when I asked Daniel to tell me more about it, he just said he based it on experience. In any case, in Daniel’s experience people carrying an AD gene want to know they do, but people carrying a FTD gene don’t want to know they do. FTD genes push patients away from medicine. Is it because, since the disease is already at work (in an asymptomatic patient), is it because this FTD gene is already causing a lack of drive, a lack of concern and a lack of care? It is difficult to give to this typology more rationality than it can offer: indeed how to understand the opposite attitude of the ones not carrying the genes\textsuperscript{319}(and yet so similar in every genetic respect)?

\footnotesize{\textsuperscript{315} In my opinion, it is frequent that people (potential gene carriers or not) who come to the French and the American clinics do come because they want to know that they are not sick.} 
\footnotesize{\textsuperscript{316} Huntington disease is a neurodegenerative disease, fatal and incurable. It is also a monogenetic disease with a complete penetrance: having the gene guarantees that one will develop the disease, without knowing how and when (according to statistics between 35 and 50 years old).} 
\footnotesize{\textsuperscript{318} People don’t go to the doctor randomly: this echoes the way patients are diagnosed MCI, partly on their results to the neuropsychological test, partly on the basis of their complaints: being present in the neurologist’s office is always suspicious.} 
\footnotesize{\textsuperscript{319} People not carrying an AD gene don’t want to know they don’t but people not carrying an FTD gene want to know they don’t.}
If I don’t see the rationality of this typology, I can see some of its effects. One of these is to create certainty: as Daniel concluded the last conversation, it’s maybe “not a coincidence” if these two persons are here: maybe these persons are actual “patients”. There might be more to this strange typology than to fabricate certitudes with respect to the diagnosis. It seems to me that it also allows the doctor to save himself the question: am I responsible for what promises to be a quite depressing situation for the patient? This genetic testing pertains to the domain of medical research. As Daniel explained to the resident, this is their job to find new genes: this is why “we are here”. As such the experimental situation and the unavoidable uncertainty that comes with it are legitimized by the research enterprise. Yet, at one point it is not anymore a test and a gene that will encounter one another, but a doctor who will have an answer to give to a patient coming with questions. Giving to the gene the agency of bringing in the patient to do the test, or not, makes it look as if there was something (but what?) irresistibly attracting or repulsive between the test and the gene. As if the gene behaved like a diving rod towards the test. As if there were no patient with questions, as if there were no doctor who proposed the test, as if there were only a natural and evident relationship between the test and the gene, with which humans had little to do.

Nevertheless, the scene is not finished: we left the last conversation when Kim cautiously (and quite awkwardly) said that the team had been in possession of Lucia blood for a while... Why did she say that? Jack who “feels out of the loop” asked her what she meant. Kim answers:

Kim (hesitant). – Because we... (To Daniel:) can I say Lucia’s [gene] status? Is that...?
Daniel. – Sure.
Kim. – I mean... (In a breath:) she doesn’t have the mutation.
Daniel (flat voice). – She doesn’t have the mutation?
Kim. – No.
Daniel (half-detached). – OK, good...
Silence.
Daniel. – Does she know she doesn’t carry it?
Kim. – No, no, no. (Big sight of relief.) She doesn’t know.
Daniel. – And what about José, does he know?
Kim. – Neither of them knows.
Daniel. – And do we know what he is?
Kim. – No because he is being seen for the first time.
Daniel. – Mmmm.
Kim. – But, I think there was a question about whether or not... (Extremely cautious.) Lucia is truly... normal...? I don’t know.  

Silence.

Jack (uncertain). – She doesn’t have that mutation yet... we think that there is something going on with her...? Right? So...

Daniel (confident). – I think she was much more equivocal [than her brother].

Jack. – Yeah.

Daniel. – I mean... we are talking about a visual memory test on her that was a standard deviation below [the norm].

Olivia (reflecting). – I think she is not a very spatially gifted person but maybe she has always been that way...?

Jack. – Well, she was a flight attendant: you’d think that flight attendants would...[Jack implies that flight attendants should be spatially gifted.]

Olivia (incredulous). – So! They wouldn’t bump into people...?

Laugh.

Jack (awkward). – No I mean... but yeah: you have to walk the cart, cabinets, know where the things are located, take them out... I don’t know.

Daniel (joyful). – Some do... some don’t! They come in all shape and forms... they really do.

Laugh. Then, a long silence. Daniel is looking at Lucia’s old scan because Lucia asked it to be compared with the new scan. After a while, Daniel concludes: “Not much”.

Daniel (concerned). – So I don’t know... this is going to be very awkward but I really don’t know what to do here. One of them probably is the mutation carrier, the other one isn’t... (Wearily.) There is no treatment for either one of them. What am I gonna do? (Heavy sigh.) What am I gonna tell these poor people?

Silence.

Daniel (gloomily). – Wow... I don’t know what to do.

Someone sneezes.

Some people. – Bless you!

Beth (comforting, to Daniel). – I think she [Lucia] is gonna come and roll a lot of questions that she wants to ask...

Daniel. – OK...OK.

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320 Lucia was seen previously at the Memory Clinic, this is why her blood was drawn: Kim is talking about the impression of the neurologists (presumably Dr. Martin) who saw her that last time.
Jack (*cheerful*). – So we let her lead the session!

*Laughs.*

Daniel (*lightened*). – OK, let’s go ahead...

Olivia. – What if she says: “I wanna know”?

Kim. – We did tell her that if down the road we firmly, firmly, believe that what we found in her brother is pathogenic; then yes we’ll bring them in and ... (*Almost whispering*) they can find out. They may ask again though: they have been asking everyone...

Emily (*prophetic*). – Even if they, or she is not a carrier, there maybe still something about... I don’t know, this family has so much other stuff going on...

Daniel (*to Emily*). – So you think she might be early Alzheimer due to...non genetic... She is at the age right?

Emily (*cautious*). – I don’t know... she is at the age... I don’t know how statistically likely... I don’t know.

Daniel. – I don’t think she is quite MCI is she?

Olivia. – No because she doesn’t have complaints about...

Daniel. – She doesn’t have complaints, yeah... (*Deflated.*) O.K ... I don’t know...

*Silence.*

Daniel. – She is easier, I think.

Olivia. – If she had complaints plus this testing, I would think she is MCI.

*Silence.*

Beth. – Do we want to introduce the idea of being seen separately?

Jack. – Yes, I think we can say hi to both of them and then maybe ask like one to wait and then bring the other.

Daniel (*deflated again*). – Yeah... OK.

Beth. – I think maybe, because they are so sensitive about this whole family issue, we’re going to have the core team only.

Daniel (*to those who are not ‘core’*). – Yeah, you’re the lucky ones who get to go!

*Big laughs.*

Jack. – Am I core?

*Enormous laughs.*
People move their chairs, we make our way towards the door; only Daniel, Olivia, Emily, Jack and Beth stay. The rest of us, the non core, leave\textsuperscript{321}.

In the last part of this scene, doubt, indecision, hesitation, possibly and maybe are injected into the discussion of the team at the Memory clinic. Something like a stop, a limit appeared after Kim’s \textit{coup de théâtre}: Lucia isn’t carrying the gene. Beyond this limit, Daniel is destabilized. Lucia was yes, now Lucia is no. Is it because the test said no that Daniel is troubled? It is possible. Yet, even if Lucia doesn’t carry the gene, the team doesn’t quite reject the possibility she has AD. Olivia zigzags between normalizing Lucia’s troubles (“Maybe she has always been that way”) and justifying the possibility that she might be on the road to AD (“If she had complaints plus this testing, I would think she is MCI”). Emily puts forward that Lucia might have “something” (“There maybe still something about...”) but then retreats (I don’t know...I don’t know... I don’t know). Daniel doesn’t seem ready to abandon his belief that Lucia will be sick, of AD (“She was much more equivocal than her brother”, “She is easier\textsuperscript{322}”). Yet, Daniel’s reassuring belief flickers faced with the depressing and corrosive perspective of the encounter with his patients (“What am I gonna tell these poor people”, “I don’t know what to do”, “There is no treatment for either of them\textsuperscript{323}”). Daniel’s trouble is the obvious way out of a situation of high uncertainty: how to predict the likelihood that Lucia and José will have (and if they do when will that be?), or not have, a deadly and incurable neurodegenerative disease in the next years to come, without being certain of anything?

4. Certitudes that fails.

Some patients, in the French and American clinics are diagnosed with a neurodegenerative disease, which, as the name indicates, will involve a life turned towards “degeneration”, yet these patients do not degenerate. These patients were diagnosed with AD, more often with FTD, but they evolve in a way that does not obey to the general laws of these diseases\textsuperscript{324}. As if they were not sick. These patients are rare: this kind of uncertainty does not make up the everyday of the neurologists. At the Alzheimer clinic, I only met one person in that case, whom I tell about it in scene 8. At the Memory clinic it happened more frequently than in the French clinic, this is maybe why these patients are referred to by a medical term made just for them: “phenocopy”; a term I never heard at the Alzheimer clinic. How in both clinics, the doctors explain the absence of degeneration in someone who is supposed to degenerate? If these examples

\textsuperscript{321} That day I wasn’t core. I came into that case-conference because Jack told me about it over lunch, just before it started. I hadn’t met Lucia and José before hand, unlike most conferences I assisted to, so I could not prevailed that I had met the patients to constitute myself as core. I wasn’t there for the rest of the conference; I don’t know how Daniel managed his uneasiness in front of Lucia and José.

\textsuperscript{322} She is easier should mean that Lucia is easier because she doesn’t have AD. It should mean that, since Daniel knows now that she doesn’t carry the gene, it’s easy, she has a diagnosis: she doesn’t have it, she is normal. Yet, Daniel said previously she was much more equivocal than her brother, he said also that there is no treatment for either of them: meaning that both of them are sick. It appears to me that the reason why Lucia is easier, is because Daniel has a firmer diagnosis on her, and that this diagnosis is actually AD.

\textsuperscript{323} This last one is a weird remark, is Daniel saying that both siblings will have Alzheimer’s since both need a treatment? Lucia, will be sick but not because of a gene, José will be sick because of the gene.

\textsuperscript{324} They are getting older though, they do like all of us, but aging does not necessarily means “degeneration”.
are marginal (to my knowledge: one case at the Alzheimer clinic, five cases at the Memory clinic), they nevertheless cause a trouble. A trouble and interrogations, that reveal a limit beyond which the model in use in neurology—every psychic manifestation has a neurological cause—becomes more labile.

**Scene 8. A somatoform disorder.**

In France at the Alzheimer’s clinic it is time for the *synthèse*: it is mid-day, the psychologists, neuropsychologists and Nicolas, the resident, are gathering in the small room full of sun to discuss with Elise, the neurologist in chief, about the patients they saw this morning. In two hours, up to five cases will be discussed. People come and go into the room. Nicolas is on a run; several times he quickly leaves the room to look for a file or a pen he forgot next door, banging energetically the door each time. The neuropsychologists get out once their have done their report, sometimes before the end of the discussion about the patient they saw, always before the end of the *synthèse*. They leave their chair, or their stool, to another neuropsychologist who saw another patient. I remember the atmosphere at the Memory clinic, where no one moves during the two-hours discussion dedicated to one patient, as sacred in comparison.

Cécile, the neuropsychologist is telling us about a woman diagnosed with Alzheimer’s disease one year ago at the clinic. This woman, Madam B., is 68 years old. She came this morning for her follow-up visit and she did some tests. Madam B. scored “extremely bad” on all the tests, Cécile tells me. Yet, she took the metro alone and arrived at the hospital on time and with no problem, she lives by herself, shops and prepares her meals for herself.

Cécile (to the team). – There was a very important dissociation between the results on the test that were very bad and her life... Did she do it intentionally?

Another psychologist. – Madam B. was working in the human sciences.... she was a psychologist...

Cécile. – Which means that she is more apt to malinger than someone else...

Elise, the neurologist (to Cécile). – Does she give you the feeling of someone who has AD?

Cécile. – No she doesn’t have this feeling.

Elise. – She doesn’t have this “anxious perplexity”... (*After a silence.*) Is that a somatoform disorder?

Cécile. – No idea...

Elise. – Is she aware of the diagnosis?

Cécile. – She didn’t say the word with me... She came a little bit like, as if nothing was happening: she has projects, she wants to do pottery...
Was Madam B. malingering? Cécile, interested to know more, will try to meet with her again. She’ll phone her in the afternoon to give her an appointment in order to do more testing. Madam B. doesn’t pick up the phone, Cécile leaves a message. Cécile’s idea is to give Madam B a special test to detect if she was malingering or not. I asked Cécile to let me know when she’ll meet Madam B., but unfortunately she never called Cécile back.

Elise doesn’t imagine that Madam B. was pretending (“malingering” or lying) to be sick, instead she first tries to sort out if Madam B. is actually sick, of AD or of some other disease. Since the results to the neuropsychological test are confusing, Elise questions Cécile’s feeling towards the patient: does Madam B. give the feeling of someone who has AD, a sort of anxious perplexity? Cécile immediately catches what Elise is talking about, and says Madam B. didn’t give her this kind of feeling. Elise suggests that Madam B. could have another disease than Alzheimer’s: a “somatoform disorder”.

A “somatoform disorder” is a diagnostic category (of the DSM IV) for troubles that are called “psychogenic” or also “functional” or “hysterical”. The term psychogenic refers to the psychological or a psychiatric origin of the trouble: the patient doesn’t have a neurological trouble but somatizes a psychic event and develops motor and/or cognitive symptoms that are very close to troubles with a neurological cause. Yet, the neurologists find no organic cause. I have seen at different occasions, patients diagnosed with a somatoform disorder at the Alzheimer clinic; Pierre, a distinguished colleague of Dr. Vincent, says it is indeed “very frequent”. Now is a somatoform disorder a matter for neurology or psychiatry? Is the trouble caused by the (Freudian) unconscious or by a neurological mechanism still unknown? At another occasion, the French team gathered to discuss about one of Dr. Vincent’s patients, the debate that followed may help to specify how the neurologists could answer the questions asked above.

Today, Dr. Vincent gathered the whole team to discuss about one of his patients whom he cannot diagnose precisely. It’s been 10 years now that this patient has trouble walking, falls, and sometimes half-consciously bangs his face on the floor so much that he bleeds. He lost his job and spends most of his days watching TV. For 10 years he complains that no one gave him a firm diagnosis other than “it’s psychiatric”, which he doesn’t accept as a diagnosis. He ended up coming to see Dr Vincent in the hope that the famous doctor will

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325 These kind of tests try to make very easy questions look very difficult, so if the persons fails those more than actual harder questions, he or she could be identified as a malingering.
326 I saw Madam B. again in a patient group. At the Alzheimer clinic some patients diagnosed with AD meet every month with two neuropsychologists. During these sessions, they answer some questions about French geography and history as if they were in high school again. Elise plans to change the ethos of this group: Elise would like that patients have a chance to talk about their experience of the disease, about their life with Alzheimer’s. Instead, for now, the persons participating to the group meetings are asked to give the names of the French presidents since 1936, when they got elected, and what outstanding things they did. Madam B. showed up late at this reunion. The way she answered precisely to any kind of questions, contrasted strangely with the general hesitation and silence of the group before her arrival.
327 I develop in chapter four, the importance of this feeling for the diagnosis, so I won’t elaborate on it here.
328 There is usually a motor component to the trouble. However, Madam B., who is basically only forgetting, doesn’t have any added motor symptoms (like fainting, falling, shaking), but as Elise is suggesting a somatoform disorder can also present as a purely cognitive trouble.
settle the question. After a careful examination and a long interrogation of the patient, Dr. Vincent asked the audience to raise hands if they thought the patient had a “functional” disorder. The whole room raised hands. Elise, slightly annoyed by the way the team jumped so easily to a conclusion, reacted: “We need arguments to say it’s psychiatric: psychiatry is not neurology by default”. Vincent argued: “The semiology is fully functional! He doesn’t have one shaking bit that could be neurological, his collapses are not neurological collapses: nothing here places us into the organicity... But we are lucky, we have a specialist with us: Pierre what do you think?” Pierre is a neurologist who works in another hospital but comes every week at the Alzheimer clinic to see difficult cases; Vincent holds Pierre in high esteem. Pierre analyzed: “I agree that he has a semiology that is not neurological and if he doesn’t have a neurological semiology he doesn’t have a neurological disease, but as you [Elise] say, what is the psychiatric disease? But it’s always like that no? ... Yeah, we are so ignorant of the psychopathology of these patients”. Yet, Dr. Vincent remarks that in any case his patient doesn’t want to hear he has a psychiatric trouble: “That’s what he reproaches medicine for, you know he has a very subtle analysis of the approach at the hospital: it’s because we don’t have any neurological argument that we consider it psychiatric... It gives him the impression that no one takes his disease seriously, plus it’s stigmatizing. Yet, he has a mutilation behavior... (Vincent is thinking) and I don’t see by which mechanism...”

The symptoms of Vincent’s patient, seem to fall into the category “functional” (or “psychiatric” without more precision), but we see that this category -- “functional” or “somatoform”-- occupies a blurry zone: it is not neurological but no one can really say it is psychiatric either. What is it then? Moreover what to do? Pierre finds a way out: “There is a way out: whatever the symptoms are we can say that they are not caused by the Holy Spirit [le Saint Esprit] but by the body. And we’ll prescribe him some TMS, for these patients it sometimes works: it’s amazing all the symptoms vanish. And it’s interesting for us: it means that the pathology had an organic substratum...”

Pierre’s solution is indeed a way out of a sort of taxonomic absurdity: the somatoform disorder posed a problem to the neurologists because if it is identified as the matter of psychiatry, then this kind of trouble would not be the matter of neurology anymore. Pierre here reestablishes a balance, a somatoform or a psychogene disorder might involve “the psychopathology of the patients”, but its cause is organic. The irony of Pierre’s remark puts a stop to the previous indecision regarding the origin of the trouble: in this affair, the “Holy Spirit” is cleared, unlike the body which appears as the principal suspect. In French, “esprit” (“spirit”) means also “mind”: if Vincent’s patient is not a puppet in God’s hands, he is neither the toy of his unconscious; any

329 The idea that a psychiatric disease is more stigmatizing than a neurological disease is pervasive: a psychiatric disease is associated with madness when a neurological disease doesn’t carry, according to neurologists, the same stigma.

330 Transcranial Magnetic Stimulation is the heir of electric shock treatment: it consists in stimulating some cortical zones with electric impulsions produced by coils placed on the skull of the patient.
reference to the mind (l’esprit) is dismissed by Pierre’s remark. The responsible agent is the “body”: there is a neuronal basis to every psychic trouble; this is the neurological paradigm\textsuperscript{331}. The TMS will be the principal witness in the affair: if the stimulation of the brain via an electric shock on the patient’s skull, heals the patient, it means a neuronal mechanism reacted positively to the application of a electric force.

Daniel’s view on hysteria shares some similarities with Pierre’s. Like Pierre, Daniel thinks that hysteria \textit{is not} the matter of psychiatry; the chief of the Memory clinic, explained to me that: “Healthy brain don’t do that [being suddenly paralyzed or aphasic]: you have to be neurologically impaired to have hysteria”. But since Daniel also sees that “the image doesn’t show anything and everything is neurologically normal”, how is it that hysteria be the matter of neurology? Unlike what Pierre could have said, Daniel didn’t answer me that it will be a neurological disease one day, once someone will have explained how. Daniel recalled discussing with some of his patients who had been diagnosed “hysterical” – but not by him-- and concluded: “People don’t like my opinion about hysteria... but the difference between factitious and hysteria depends on how the doctor likes the patient.” Does he think these patients were faking their symptoms? “I think so,” said Daniel, “maybe not at the beginning, you know people who gets this are so annoying, they are so exasperating: part of this is that they are annoying before they get the conversion disorder\textsuperscript{332}; they have a funny personality to begin with, it doesn’t happen to you or me, they are annoying because they have this sort of brain dysfunction.” Daniel’s explanations seem contradictory: hysteria would be caused by a neurological dysfunction yet the symptoms –that define hysteria—would be faked; so they wouldn’t be neurological (neither psychiatric). Yet, this explanation is quite reasonable if we consider its potential in justifying the uneasiness that the neurologist feels in front of these patients. These “hysterical” patients contracted a strange neurological disease that seems to target neurons involved in “being annoying and exasperating to medicine”, which leads them to fake their symptoms to irritate their doctors with their unclassifiable disease.

\textit{Scene 9. “Munchausen”... “Scary.”}

Mr. P had been enrolled in the research program for three years. He came for the first time to the Memory Clinic in 2008 and Daniel diagnosed him with FTD. He came back in 2009 for his yearly visit, Oliver confirmed it: it is FTD. He is back again at the end of 2010. But this time the conference --to which I attend-- is tinged with suspicion: towards the sincerity of Mr. P, towards the accuracy of the diagnosis and to a lesser degree, towards the good faith of Mrs. P.

At the end of 2010, I had just started the fieldwork and I was not quite familiarized with the schedule of the Memory Clinic: I was not yet attending the clinical exam that preceded the case conference nor did I have breakfast with patients. Therefore I had never met Mr. P before that conference, but because I was so impressed by what was said, I went to look at Mr. P’s

\textsuperscript{331} This paradigm, for the most radicals of the Alzheimer clinic, amounts at finding the neurological basis of the (Freudian) unconscious. One of the close collaborators of Pierre, at the Alzheimer clinic, already published two books upon this kind of quest. This neurological paradigm is not shared by Elise, she is the only neurologist I know who doesn’t accept the neuronal explanation for every kind of psychic trouble.

\textsuperscript{332} Conversion disorder is another name for hysteria.
medical files; what follows is based on my reading of these files and on the notes I took at Mr. P’s case-conference in 2010.

This year Mr. P’s case is presented by the fellow Ellen. Dr. Blake is the attending doctor. Mr. P is 65. He is a physician (from now on I will call him Dr. P). Ellen restates briefly some of the events that were consistently reported by his wife at each of their visits since 2008: “A few years ago, Dr. P just arrived at a party and stated to a whole group of people, “I’m leaving right now”. The whole room was shocked when he said it.” More recently he “took an iPhone from a child in a restaurant”. He also confided to his daughter lately: “I want more sex”.

In 2009 he began to “exhibit some new behaviors”. In the 2009 report, I read: “these disinhibited behaviors have become significantly more severe”. “In public, he might shout “make way” when he wants people to get out of his while he is walking”. Recently, in a plane, he engaged in conversation with his neighbor, a physician who was sitting across the aisle from him and at “one point for no apparent reason he shouted to this doctor: “I love little kids so much that people think I’m a deviant,” in such a way that everybody in the airplane could hear".

The 2009 report concludes that these “disinhibited behavioral changes” and “problems with social interactions” have led to “believe that Dr. P meets criteria for possible frontotemporal dementia.”

Ellen pursues the story: since his visit to the Memory Clinic the previous year, Dr. P moved into an assisted living facility; he seems to be more “relaxed” there and he shows a “good empathy for people” who live with him in this facility. He is “helpful with people with gait problems” and the residents “like to be around him”.

Blake. – This doesn’t sound very FTD to me.

Ellen. – Yeah, he did seem more pathological in the first years... Since last year he doesn’t seem to have changed and this year the tests were better; he was better in everything.

Blake. – What have we told them?

333 The report specifies “because the patient is very attracted to electronics”.
334 This sentence written in the report by the fellow is not written in reported speech, the fellow is not reporting the wife’s discourse, it is thus his own words (or he doesn’t make the distinction between his words and Mrs. P.’s words) even though it seems doubtful that everybody in the plane could hear, unless of course it is a very small plane, which is not mentioned.
Ellen. – FTD. His wife is convinced that he doesn’t have empathy. She made the diagnosis of FTD herself in 2004. He had a CT scan in 2007 that showed some frontal atrophy.

Blake. – It sounds like phenocopy.

I am hearing this word for the first time.

Me (to Blake). – What is phenocopy?

Blake. – They look like FTD but they do not progress.

Me. – Ever?

Blake. – Very little, slowly.

A phenocopy is here understood as an entity that imitates the disease but is not the disease: it is a copy of it. The term comes from the field of evolutionary genetics. It was coined by Jean Piaget to describe the assimilation of the environment into the genome. It designates a phenomenon in experimental genetics: the apparition of new mutations, not due to heredity, responsible for new characters. For instance, the Himalayan rabbits who, raised in a moderate temperature, are white in color but when raised in a cold temperature, become black, resembling the Black rabbits: the Himalayan rabbits are hence said to be a phenocopy of the Black rabbits. Piaget’s explanation was that the environment would produce these modifications into the DNA. This Lamarckian justification has been recently criticized. In any case, the term phenocopy is now used in the field of neuroscience - this is not a terminology proper to the Memory clinic - to describe a disease that looks like FTD but is not quite FTD. In comparison to the original definition, the actual one doesn’t presuppose a reference to genetics. The parallelism with the original term though, is conserved in so far as the genetic means more generally the biology: FTD is psychic disease with a biological cause, FTD phenocopy copies the psychic symptoms of FTD but what causes it?

John Hodges published on the concept: as Dr. Blake just explained, Hodges writes that bv-FTD phenocopies have the symptoms of bv-FTD “without actively progressing to frank dementia”, for Hodges it means that there is not “an underlying neurodegenerative condition”. In his “Cambridge sample”, Hodges reports that two FTD-phenocopy patients have came to autopsy and did not show frontotemporal lobar degeneration. Other studies, Hodges complains, did not verify their cases neuropathologically. The article concludes that these patients are nevertheless

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335 The 2008’s report actually mentions that when Dr. P saw his doctor in 2007 prior coming to the memory clinic he said to his physician that “he believed that his family was against him and they thought he was demented.”


338 Kipps, Hodges and Hornberger, “Nonprogressive behavioural frontotemporal dementia: recent developments and clinical implications of the ‘bvFTD phenocopy syndrome’”, Current Opinion in Neurology 2010, 23:628–632, p628. Also, in his article Hodges underlines that the MRI doesn’t need to show atrophy in the frontal lobes (or anywhere else in the brain). One can be diagnosed with FTD without one principal suspect of this evidence-based neurology: the image of the brain.
not normal but may have some “neuropsychiatric” conditions like a “decompensated personality disorder or autism spectrum disorders such as Asperger's syndrome\textsuperscript{339}. What causes these diseases? Is it psychiatric or neurological? Hodges says it is “neuropsychiatric”; like Vincent’s patient diagnosed with a “functional” disease, or the woman diagnosed AD but who could have a “somatoform” disorder, the taxonomy of these diseases is unclear and their entity ambiguous.

Dr. P and Mrs. P enter the room. Dr. P is smiling. He sits next to Dr. Blake, comfortable and relaxed with his knees going sideways. Dr. P makes jokes. People around the table look at each other half-sorry, half-dismayed, no one is saying anything. His wife sits two chairs apart from her husband, keeping some distance. Dr. Blake asks Dr. P about his medical specialty. Dr P talks about what he did, articles he published, when and why he retired (essentially because of back pain), he then talks about his life in the facility.

Dr. P. – She (pointing at Mrs. P:) wants me to stay in an ill birdcage and go “cot-cot-cot” \textit{(He imitates a chicken with his elbows.)} I am in prison right now. I live in a dementia facility and if I get out of there it might be for two hours and this makes me a very boring boy...

Dr. Blake. – Yeah it’s frustrating, we’ll see with the social worker before you go if they can arrange activities for you.

Dr. P. – What kind of activities? They are 90 years old people!

Dr Blake suggests swimming, which would be beneficial to Dr. P’s back pain. Blake then changes subject.

Dr Blake. – You did very well on the test today... you are falling in the slow progression group. Whatever is going on in your brain, it does not seem to have worsened a lot...

Dr P. – Where would I fit in?

Blake. – In the 10% or so of people who do not progress... but you are more the exception.

Mrs. Blake suggests that her husband’s life in the “safe environment” provided by the facility has something to do with these results. They talk again about swimming and about the possibility for his wife to pick him up at the facility to take him to the swimming pool... She promises she'll do it. Then, they leave. Dr. Blake has something to do and leaves very soon after the couple.

I stay with Ellen, Beth the nurse and Olivia the neuropsychologist. There is a strangely electric atmosphere in the room.

Olivia is the first one to speak. Almost whispering she says: “Münchhausen...” I look at her, she looks at me smiling and mysterious, and tells me with the same tone: “Scary...” If Dr. P has Münchhausen syndrome it means that he simulated intentionally FTD in order to get attention, care, compassion, etc. In

\textsuperscript{339} Ibid. p 632.
Munchhausen’s syndrome, the patient knows he is not sick but pretends he is. In front of me, Ellen is not smiling and finds Dr. P “very manipulative; a trait confirmed by his wife”. I ask Ellen why she would think such thing. She answers me: “Why choosing the precise moment of the conference to say all of these things?” e.g. why accusing his wife in public for dooming him to a boring life in the facility? What Ellen finds especially manipulative is Dr. P’s choice to speak in front of everyone at the conference as opposed to speaking to Ellen alone in the privacy of the clinical exam that preceded the conference. Beth, the nurse, jumps in the conversation wondering clearly if Dr. P is “demented or not”. This is the first time this question is asked openly. Immediately after though, she attempts to excuse a possible misdiagnose saying that when she saw him last year he “was a lot worse” and that it was “justified to put him in the facility” at that time. Ellen assures Beth, that “he is impaired cognitively whether it’s a brain disease or his meds [that he takes for back pain]”.

I ask Ellen: “In the case you would consider him not demented, how would you make the difference between someone manipulative and someone... (I am searching for words, Beth finishes my sentence for me:) trying to meet his needs?” Just when Beth finishes her sentence, Mrs. P suddenly bursts her head into the room. She stays at the doorstep and asks: “Did he progress in the disease or is he better?”... Ellen answers her reluctantly: “It is hard to know”. Mrs. P turns around and declares: “This is a comfortable answer for me”; she is already gone. Ellen looks at us irritated: “If another person than Daniel or Oliver [both diagnosed Dr. P the precedent years] tells her it is not FTD, she is not going to understand”. Ellen says a bit disgusted: “We rely so much on caregivers for this diagnosis, the day we will have biomarkers we won’t be so dependant anymore”. She then leaves with Olivia. I take the elevator with Beth, we talk about the possibility that Mrs. P wanted to estrange her husband form Beth, and from society in general, Beth sighs: “and we are part of it”.

The 2010 report made by Ellen will conclude that Dr. P “has not worsened over the past year and performs better on some tests” and “that it is not typical in frontotemporal dementia to see improvement from one year to the next” nor it is typical “to have such little deterioration over a ten-year time”. Ellen will not go as far as to report a possible misdiagnosis. She only signals in her report that Dr. P seems to fit in a “subset of patients diagnosed FTD at the Memory clinic who do not progress”.

The “subset” Ellen is alluding to, is the group of patients at the Memory clinic designated by the elusive diagnosis of “phenocopy”. From Dr. P’s story, I wonder if a phenocopy is, in truth, 

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340 The difference with hysteria is that in the later the patient doesn’t know she is not sick. At least if we don’t believe Daniel.
341 During all the time I spent at the Memory Clinic I never witnessed a family member or a patient coming back, especially after a while, to the conference room. This is exceptional.
“normal”? Olivia didn’t believe so, Olivia thought Dr. P had Munchausen’s syndrome: FTD was a misdiagnosis due to Dr. P’s pathological talent to play his symptoms. Ellen’s reading of the case was more ambiguous than Olivia’s. Ellen, on the one hand, seemed to think, like Olivia, that Dr. P is an excellent liar (“very manipulative”), but on the other hand she believed he (his brain) “is impaired”. It seemed to me that, for Ellen, a diagnosis of FTD and the capacity to “manipulate” were not exclusive. Yet, if Dr. P was not considered neurologically (demented) or psychiatrically (Munchausen) sick a priori, how to distinguish between a manipulative attitude and a legitimate attitude (trying to get out of what he called a “prison”)? I won’t have the answer to my question because Mrs. P’s sudden entrance on the scene captivated everybody’s attention (including mine, I didn’t take any notes). Reassured by Ellen’s verdict, Mrs. P leaves. Yet, it is now for Ellen to be worried by her subjection to the information given by Mrs. P: she wishes for new biomarkers to diagnose FTD that will free neurologists from caregivers. Ellen doesn’t say that there was a misdiagnosis but her remark seems to imply it. The only person who said clearly that a mistake had been made was Beth. After this conference, maybe two or three days after, I asked Olivia if she still thought it was a Munchausen’s syndrome, she mumbled me a sort of yes, and I understood she didn’t want to elaborate more on the subject. I didn’t hear about Dr. P again. I myself forgot about this story, maybe because of its violence and maybe also because the sentence pronounced by Beth in the elevator, “we are part of it”, didn’t spare my responsibility. A long time after this, Daniel gave a talk in the psychiatry department and came back on three kinds of “mistakes” made by his team about FTD diagnosis. Here I only develop on the first kind, which when I was listening to Daniel suddenly reminded me of Dr. P.

Daniel tells this audience of psychiatrists that four patients at the Memory clinic were called FTD phenocopies when they should actually have been called “FTD by proxy”. Daniel made the expression up from the (real) syndrome called “Munchausen by proxy”. By “FTD by proxy” Daniel means that the one who is faking FTD is not the patient but his or her spouse and does it by proxy: in a manner that the symptoms are transferred on the patient. For mysterious reasons that Daniel didn’t explain during his talk, the neurologists get caught by the spouse and diagnose the patient with FTD (they sometimes don’t even realize the imposture and subsequently diagnose the patient (like Dr. P) with Munchausen’s syndrome (like Olivia)). The moral is, Daniel complains, that neurologists depends so much on the words of the “informant”; a nagging problem indeed for the neurologists as much as for the anthropologists.

How do the neurologists rectify their mistake though after the diagnosis of FTD has radically transformed the life of a patient? Is it possible to go backwards once such a diagnosis has been made? In Dr. P’s case, as far as I know, it had been impossible. I once randomly met Dr. P’s wife at the occasion of a “course” on FTD organized by the University of R. to inform the lay public

342 This word takes meaning only in relation to the term “pathological” (e.g. Munchausen, FTD, etc.). Of course Dr. P, like most of us, has never been normal.
343 It is considered in the French clinic that a high level of the protein tau, with a normal level of amyloid-beta, might be indicative of FTD. In the American clinic, as I said this dosage is more rarely done, and second such a result is not considered meaningful.
344 Munchausen by proxy usually applies to parents who fabricate diseases for their kids. The mobile is the same: to attract attention on the parents. Some extreme cases of child murder have been explained by a syndrome of Munchausen by proxy on the part of the mother.
345 I am planning to inquire about the ways in which the speech of the other during the hospital consultations constructs the identity of the patient. It is the project of a post-doc that will start in January 2015.
about this disease. I went to talk to her after the conference, I asked her how her husband was, good she said. I asked her if he was still living in the dementia facility, yes she said.

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Faced with the uncertainty that sometimes surrounds the diagnosis of dementia, we have noticed that neurologists may engage more technological explorations and may, as well, mobilize their affects, in order to (maybe) arrive at a diagnosis. On the one hand, neurologists can prescribe a new MRI to a patient whose diagnosis isn’t clear (scene 5), wait for the result of the DNA analysis (scene 7), recommend a new kind of neuropsychological test (scene 8) to determine if a patient is lying or telling the truth, and wish for more research on biomarkers (scene 9). And on the other hand, neurologists express themselves with terms that Vololona Rabeharisoa has also heard in multidisciplinary consultations on autism (in which uncertainty is pervasive), a language that she describes as designating “what affects the senses of the observer”. We remarked also in this hesitant context that the language used by the neurologists sometimes spoke directly to their senses (“it sounds like”, “it doesn’t sound very FTD”, “does she gave you the feeling of AD”, “a weird vibe”), expressed beliefs transmitted by a sort of confidential knowledge (“it is funny...somebody told me”, “I think, he would say: ‘early AD’”), or was stated through idioms (“Only time will tell”, “On laissait le temps au temps”); a language, Rabeharisoa summarizes, that “doesn’t engage the physician in a work of rationalization of her sensations, nor in a categorisation of the patient”. In the following chapter, I inquire into the senses and the feelings of these doctors revealed by this situation of uncertainty: how do they participate in the making of knowledge? The endeavour focuses on the participation of feelings in the diagnosis, yet the reader shouldn’t imagine that the medicine that we will observe in action at the American clinic would only be legitimized by the exercise of intuition, impression and feelings. The medicine practiced today in the American clinic is also --as we already read in this chapter-- the matter of statistics, genetics, molecular biology and brain imaging.

Inquiries about “subjectivity in science” have been too rarely undertaken Steve Shapin deplores, therefore the historian wishes for ethnographies that “would look a lot like those produced by laboratories studies of science, concerned with how fact and theory judgements come to be formed, discussed and sometimes shared”, except that they would focus on “taste judgements”. How to tackle “taste judgements”? Antoine Hennion may have answered: “with pragmatism”. Without reservation I’ll follow the lead of this sociologist who studies the taste of

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347 Ibid. My translation.
348 The endeavor focuses on the participation of these feelings in the making of a diagnosis. Yet, I do not want the reader to conclude that this medicine that we will observe in action, would only be the matter of intuition, impression and feelings. As we will see, this medicine practiced today in the American clinic is also --as we already read in this chapter-- the matter of statistics, genetics, molecular biology and brain imaging.
350 Ibid., p 177.
wine connoisseurs and music lovers. To study taste, Hennion writes, we have to start acknowledging that taste is not an “attribute of things and people”\(^{351}\); a property that would be determined a priori. To the contrary taste is “an activity” and it is through an activity (listening to music, drinking wine and even diagnosing neurodegenerative diseases, as we will see) that taste and things appear together: “inextricably”, writes Hennion “in” the objects tasted and “in” the sensibility of the taster\(^{352}\). Thus, “taste” is a “word of the in-between \(\text{entre-deux}\)\(^{353}\), and its study brings us at the heart of pragmatics: “Yes things have an effect – if one gives it to them\(^{354}\).” Hence taste ought not to be studied like natural science does: by attributing an effect to things only – taste is there understood through chemical, physiological and neurological analysis; it doesn’t matter that we find something ‘good’ or not. Neither should it be studied as sociology does: withdrawing from things any effect --in showing how taste is socially constructed, sociology explains our relation to things with determinants such as “categories, institutions, authority of the leaders, imitations of the others\(^{355}\), etc., and transforms “things into signs without body\(^{356}\), and as a result sociology neglects “the uncertainty of sensations, the operations to render one sensitive to the things searched for and to feel oneself feel\(^{357}\)”.

As we will read in chapter four, neurologists do speak about a taste or a smell for the disease, when they talk about their clinical sense. As Henri Bergson so well defended William James’ pragmatism: to commit to a pragmatic analysis means to treat our experience of facts and things as it is in reality: flowing. Far from, Bergson, writes, the dry universe of elements well arranged and well shaped, not only in relation to each other but also “coordinated to the Whole\(^{358}\).” It could mean to not treat clinical sense as the mere effect of social determinants (clinical authorities for instance), or as the fantasy that ‘disease have taste’ (a belief that the sociologist would be in an unique position to unveil) but rather to describe, slowly, how these tastes, feelings and flavors for the disease, emerge, happen, develop, often from a situation of uncertainty, how they are discussed, shared with others and supported by others, how they are put to the test by scientific technologies, and how in the end they participate in the making of knowledge about cerebral diseases.

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\(^{353}\) Ibid. My translation.

\(^{354}\) Ibid. My translation.


\(^{358}\) Henri Bergson, Sur le pragmatisme de William James, Paris: PUF, 2011, p. 3.
THE TEST

April 2012.

It’s morning at the Alzheimer clinic. A woman and a man are seated on the chairs of the waiting room. The woman is looking at the paintings on the walls.

The woman (to the man). – I would like one to put it in my kitchen.

No answer.

The woman (looking at the man). – Don’t be scared!

The man. – No.

The woman (encouraging). – You’ll have to answer five or six questions and that will be it.

February 2012

Are you still working?
No.

How old are you?
Sixty-two... or sixty-four.

Maybe a little more?
Seventy?

You’re going to be soon sixty-eight. Do you have trouble with numbers?

Sometimes. The dates are the worst.

Which year are we?
2000.

What day?

More like at the end of the month. The end of February. I think it’s Wednesday.

Which season?

Soon it will be spring; it’s winter.

Can you do this addition?

No I can’t.
But you didn’t learn how to count in school?
In school I wasn’t sick.
What’s the name of this object?
It’s a watch.
Ball, key, lemon. Can you repeat these words?
Ball... ball... I don’t know. I don’t remember anything, it’s like it shut down, it’s zero because...I...

June 2012
Seven minus six?
One.
Four minus two?
Two.
Eight minus seven?
It’s... uhm, it’s one.
Thirty-five minus eleven?
Don’t know... Twenty-six maybe?
Seven plus six?
Eleven. It’s eleven.
Four plus two?
Six.
Eight plus seven?
I don’t know, I can’t do it anymore.
Seven by six?
Forty-two.
Eight by seven?
Fifty-two maybe? I don’t know.
Five by four?
Twenty.

January 2011
Put the watch on the other side of the pen and turn the book.
Can you repeat?
...
I know you can’t repeat. I guess it’s your profession.
Try to do it and then I’ll repeat.

March 2012.
What do you see on this image?
There is something like an umbrella, yes, it seems so, I see the hand of the umbrella but I don’t see the umbrella, I only see the hand.
Good, you see the details.
I don’t see the point of this test.
And here what do you see?
There is a little girl, and here it’s the mother. The mother has knocked over some water, on the floor, I don’t really know what’s going on, here it’s the mother, there is a liquid, I don’t know what it is, I don’t know if she broke something, oh yes something, she knocked over... I don’t know, I don’t see.

December 2010
Can you tell me what lights up the room?
This light.
Show me the floor and then the ceiling.
Do you want me to name all the room?
Show me how you wave good-bye with your hand.
Is there a methodology?
Show me how you lace your shoes.
No.
Show me how you saw a log.
I don’t saw it when I approach a log.
Imagine the carpenter.
How can that be good? How can that be helpful?
I don’t know. Maybe you’re right.
February 2012.

What work were you doing?
I was in chemistry, I was a chemical engineer.

What is the date?
Indisputably it’s winter. We are currently in February.

It’s possible. Can you say more?
Well, we are at the end of the month of February. I take this opportunity to tell you that I’ve lost quite a bit of my memory. When I have my memory with me it’s fine, but sometimes it leaves me. What can I do? I’m not going to change my brain.

Ball, key, lemon. Can you repeat those words?
There was ball, lemon... ball... lemon...Wait...

It was key.
Yes: key. But I couldn’t repeat it.

Now you are going to subtract 7 from 100 and then 7 from the result you’ve got and so on.

OK. 100 minus 7 it’s 93... And then 93 minus 7 it’s 86... No? You see I have hesitations, there is no doubt about that, on many occasions I remarked that I had this kind of problem.

OK. Do you remember those words I told you before?
There was ball, lemon... There was a third one that bothers me, I mean: “bother”, it doesn’t stop me from sleeping.

February 2012

Tell me as many animals as you can.
Dog, cat, mouse, wolf... what else? These are the most frequent.

Come on, Sir! Concentrate!

Canaries, crows, eagles, fishes... a carp, why not. No, I don’t see anymore. Swallows.

Very good. Now tell me as many words as you can, starting with the letter P.

Prune, Pear, Peach... Wow! It came like a puff!

March 2011

Let me do things to test your memory: what’s going on in the news these days?
The war in Iraq, the election...

What election?
The city mayor.
Who is the governor?
Oh God, I don’t know.
You heard the news this morning?
Yes, there is a volcano erupting in Island.
Who is the president?
Roosevelt...... I’m kidding!!
Who is the president?
...
He is black.
Oh! That should ring a bell.
Barack ...
Obama! Barack Obama!
What’s that?
Your shirt.
And that?
The collar.
And those?
Your shoes.
That?
The sole.
That?
The hill.
Good.
Good? It’s kindergarten.

November 2011
Do you feel your life is empty right now?
Well, yes.
Are you in good spirit most of the time?
No.
Are you helpless?
Yes.
Happy most of the time?
No.
Do you feel full of energy?
No.

April 2011
Apple, anaconda, anybody, automobile, let’s see.... Shit! Come on mind! Catch up with me.

March 2011
Repeat this sentence: “No ifs and or buts.”
No if but but no if.
Mamama
Mamama
Snowmen.
No men.

The mini mental state examination, the Montreal cognitive assessment, le test de Grober et Buschke, the verbal fluency test, the categorical verbal fluency test, the California verbal learning test, the Boston naming test, les cinq mots de Dubois, the Wisconsin modified test, the calculations, the Peabody picture vocabulary test, faux pas recognition test, modified trails testing, the copy and recall of the modified Rey-Osterrieth figure, visual object and space perception battery, abstract reasoning and proverbs, face perception and affect naming, the geriatric depressive scale, the scores, repetitions and rules violation, one or two or three standard deviations below and above the mean, uncorrected and self-corrected errors, a score significantly below average, the percentile rank.

François, a French neurologist, once said to me: “Do not forget that the neuropsychological profile allows you to understand the troubles but it doesn’t determine the etiology”. 
CHAPTER FOUR

THE DIAGNOSIS

A doctor consulted in a case like this must be more than just well versed. In the face of symptoms which may be those of three or four different illnesses, the thing that enables him to decide which of them he is most likely to be dealing with, behind appearances which are very similar, is ultimately his flair, the sharpness of his eye.

Marcel Proust, In the shadow of young girls in flower\textsuperscript{359}.

Studies haven’t shown anything about the matter, but studies never demonstrate the consultation’s impressions: the only reliable tools of the doctor.

Antoine Sénanque, Blouse\textsuperscript{360}.

1. Smell and Nostalgia.

Dr. Vincent’s car slows down while I am walking by the endocrinology building of the hospital where the Alzheimer clinic is. This is where I am spending most of my days right now. Vincent proposes to take me back home. Of course, I accept. We drive through the paths, the courtyards and around the chapel of this vast French hospital commissioned by Louis XIV. These days, the buildings are bordered with fresh flowers renewed each month by the gardeners of the


\textsuperscript{360} Antoine Sénanque is a neurologist who writes under a pseudonym. In Blouse, p 20. My translation.
hospital; February is blossoming in yellow. We leave behind us the statue of the alienist Philippe Pinel posted at the entrance of the hospital.

In the comfort and the silence of the car, our discussion bears on the way of doing a diagnosis in behavioral neurology, what is so peculiar about it? How these neurologists search for the etiology of the troubles: the disease? There are two ways of doing it, explains Vincent. The first is the one that he imposes today in his clinical center: it consists in “framing” the patient, in that case the use of the term “diagnosis” is almost usurped “because we cannot ask questions”. That way of doing which comes down to labeling patients (“étiqueter”, as I hear it often said in the service) bores him. What fascinates him is this other way that belongs to an epoch “when he had time”. “It is something that is quite original, you will realize it, and that is pretty much French... Here, we are not trying to categorize but to observe, we have a mission which is exactly to make science progress and the thing is that the patient, we will interrogate him as a dog would... you see... would sniff, trying to find, to understand”.

I laugh a bit because sniffing is not that specific to the French school. When I started my fieldwork in the American Clinic, I was surprised by the vocabulary of “taste”, or “smell”, used by the medical team to express their diagnostic impression; I would hear such things:

“What do you think, personality disorder?”
“A little bit of that yeah, but mainly an AD flavor.”

Or, it is the taste of a part of the brain that was appreciated:
“Yeah, so Emily is giving us, maybe, a right temporal flavor.”

Or, the taste of a cerebral protein:
“It doesn’t have a FUS\textsuperscript{361} flavor to her [the patient is a woman]”.

Smell also was invited into the reflection:
“I think it is important to see that he has something that smells like Capgras\textsuperscript{362}.”

If these flavors were not robust enough, if the diseases could not be clearly classified, Olivia a neuropsychologist, told me the diseases were “vanilla”, a sort of in between, lighter and subtler.

But sometimes there were no flavor, no smell, no feeling, nothing:

\textsuperscript{361} FUS is a gene that codes for a protein named FUS/TLS (Fused in Sarcoma/Translocated in Sarcoma). A mutation in this gene has been found associated with the occurrence of Amyotrophic Lateral Sclerosis and FTD.

\textsuperscript{362} Capgras is a syndrome named after Joseph Capgras (again --like Alzheimer’s, Pick’s, Huntington’s, Parkinson’s etc.-- an incurable disease that bears someone’s name) which principal symptom is the delusion that the spouse, the parent, the brother, the friend, etc. of the diseased, has been replaced by an identical looking impostor.
“I don’t have a feeling that tells me that he is FTD.”

I say to Vincent that he will hear about tastes and smells too, when he will come to the United States to visit the Memory clinic lead by Dr. Daniel. Actually, Vincent finds Daniel to be “an atypical [with respect to the American neurological tradition]\(^363\) and a real clinician, an authentic clinician”. Then, the real clinician models his attitude upon the one of the hunting dog; the hunting dog tracks the game, what is the real clinician tailing? Vincent answers me, while driving along the river:

This affair of smell, I think is the heart of our work, because first we are not researchers, that is not my thing at all, what interests me is the integration, it’s the man, it’s the integration of the whole. It’s to shell him, it’s to smell him... Sniff... It’s to understand what there is behind... We can’t understand the psychology, we are not analysts, but when there is a behavior problem we have today the keys to understand it... Pierre does it very well, that said he is doing it little systematically, apparatus after apparatus, without playing enough. This is something I find extremely interesting, this is clinical research properly speaking and I will find some time to do it when I will be retired.

Dr. Vincent differentiates two ways for making a diagnosis in his discipline. Everyday, in his clinic he practices the first way; it is boring but he is bounded to it by the hospital administration and its efficiency policy. It consists in making a diagnosis as fast and as accurately as possible. In order to diagnose quickly (from 30 minutes to half a day) and accurately, neurologists have at their disposal different technological means: the scores on a battery of neuropsychological tests, the assessment of cerebral atrophy with various techniques of brain imaging, to these two classic bedrocks of the diagnosis are sometimes also added: the measurement of specific proteins in the cerebral spinal fluid of the patient and (if the involvement of a gene is considered) a genetic test. The results of these psychological, biological and imaging tests are integrated in what Vincent calls an “algorithm” that will give the likelihood that the patient has Alzheimer’s disease or another neurodegenerative disease. If the everyday of the neurologist is occupied by diagnosing that way, Vincent is nostalgic for a second way of doing; unfortunately he has no time for it anymore.

The words of smell and taste in circulation in the discourse of the French neurologist and in the American clinic, seem to speak of the existence of a “clinical sense”. Smell and taste, however are not literally senses associated with the forms that a classic contemporary clinical examination would take at the “bedside” of the patient. These days, medical students learn that the clinical examination takes four forms and involves three senses: inspection uses vision, auscultation uses hearing, palpation uses touch, percussion uses hearing. Smell and taste are senses that seem to pertain to the medical practices of the Middle Ages (tasting of urines was one of the supports for uromancy\(^364\)) and to the plays of Molière\(^365\), but since the birth of the anatomo-

\(^{363}\) The idea that French neurologists are far superior in the field of the “clinic” (as opposed to the one of the research) compared to their American colleagues, is an idea largely shared by the French neurologists that I interviewed.

\(^{364}\) By tasting urines and looking at their color, Middle Ages doctors predicted the future.
pathology method analyzed by Michel Foucault\textsuperscript{366}, they should have disappeared from the clinical exam. Yet, they haven’t fully disappear, at least smell didn’t. By smelling a diabetic foot, a good clinician still identifies today the presence of the bacteria staphylococcus pyogenes. In the French hospital where the Alzheimer clinic is situated, in the department of bacteriology (a remote building that has few relationships with the department of neurology), residents in bacteriology smell the effluvia of the bacteria growing in a Petri dish by fanning its lid. They hide when they do it, because they say they are not allowed do it anymore. By doing so, yet, they claim that they can identify each species of bacteria by their distinct smell. A popular anecdote is that the \textit{staphylococcus lugdunensis} smells like a typical sausage called rosette de Lyon. Even if there are today other means that are faster and more accurate (via the spectrophotometric analysis for instance) to arrive at the same diagnoses, the practice holds, as well as the nostalgia.

If it is self-evident that I never actually observed neurologists tasting or smelling the cerebral matter, it nevertheless should be asked what this choice of smell and taste as points of expression of a clinical sense, reveals about the practice of the diagnosis of neurodegenerative diseases today. The image used by Vincent, of the bloodhound running flat out, tracking the animal, evokes a capacity to reach the whole, the game, with almost nothing: a smell, an evanescent trace. This holistic way of reaching knowledge seems to go against the principle of atomization dear to the scientific method. Smell enables the clinician to perceive “the man”. In the same line of thought, I often heard fellows at the Memory clinic telling about “their gestalt” about such or such patient (“From gestalt: she is not normal”) or such and such disease (“AD! That’s gonna be my gestalt”). From what Vincent told us, sensuality and holism held by a creative and playful inquirer (Pierre lacks a bit of this creativity said Vincent) characterize this second mode of doing a diagnosis. And it’s the only “real” way to make a diagnosis said the neurologist.

This second manner to arrive at a diagnosis is the one of the doctor-clinician. In contrast to this figure clearly articulated by Vincent, the one of the doctor-researcher appears in counterpoint. This opposition between the clinic and the laboratory reveals a long-lasting tension due to the mission of scientization of the French hospital launched after the Second World War\textsuperscript{367}: following American and German models, research and clinic were progressively integrated together at the heart of the hospital; clinicians became lab researchers and conversely: “M.D, Ph.D”. This disruption introduced what Jean-Paul Gaudillière calls a “new center of gravity”\textsuperscript{368} in the production of medical knowledge, where the clinic lost its predominance to the benefit of the laboratory. Somehow, this trajectory of the French hospital over 60 years shows in Vincent’s nostalgia.

Nonetheless if these days, the movements between the laboratory and the clinic are dense, the distinction between clinicians and researchers has not yet become obsolete. Vincent is

\textsuperscript{365} Medical practices in the XVII century didn’t seem to progress (despite the fundamental discovery of the circulation of blood by William Harvey in 1628 and Molière ridiculed doctors and medicine in different plays (\textit{Le Médecin malgré lui, Le médecin volant, Tartuffe}). In \textit{Le médecin volant}, Sganarelle who is a fake doctor, needs at one point to proceed to the analysis of the urines of his patient, but for that he has to taste them as it used to be done at that time; the impostor tastes the urines, a way for Molière to mock this practice.

\textsuperscript{366} Michel Foucault, \textit{Naissance de la clinique}, Paris, PUF, 1963.


\textsuperscript{368} Ibid. p 9.
nostalgic, it is true, but for the clinician that he thinks he really is. He has not given up on being one again (when he will retire); this kind of practice still constitutes who he is. Moreover, from how I observed the American team making a diagnosis, it becomes difficult to affirm—at least in behavioral neurology—that “the clinical laboratories and imaging departments” have become today, as Rosenberg\textsuperscript{369} writes: “the ultimate arbiters for diagnosis”; it seems that the time has not yet come to announce the death of the clinic. If “holism” characterized biomedicine between 1920 and 1950\textsuperscript{370}, practiced as “an empirical art”\textsuperscript{371} as Christopher Lawrence writes, it is the case today that this notion is not only reserved for homeopathy, naturopathy and herbal medicine but has retained a meaning in mainstream medicine. Moreover, the assemblage of terms: “experience, holism, expert, Gestalt, affect, sensuality”, doesn’t only pertain to the mid 20\textsuperscript{th} century science identified by Lorraine Daston and Peter Galison as the epoch of “trained judgment”\textsuperscript{372}. How is this concretion of features translated in the ways the French and especially the American teams of diagnosticians, practice behavioral neurology? Before examining situated clinical practices, let’s consider another comparison used by Vincent that will tell us more and might set out the questions more clearly.

2. Experience and Art.

The month of February in France seems now far away; it is summer in America. Vincent took some holidays to visit the natural beauties of the United States with his family. Vincent had also planned a stop at the Memory clinic to meet Daniel and to give a talk about the usefulness of biomarkers for the diagnosis of Alzheimer’s disease. He is now standing in front of the staff, the neuropsychologists and the neurologists of the Memory clinic. He has just finished presenting his algorithm and the ways in which it renders the diagnosis of Alzheimer’s disease straightforward. It is now time for questions. The neuropathologist of the Memory clinic, Laura, who is in charge of the autopsies, has a question.

Laura. – I have a question... Thank you for your talk it was very nice. So, you’ve been involved in defining the new criteria for many years, the biomarkers are getting better and better... But from neuropathological work we know that about 30 percent of people we see, have pathological criteria for “Alzheimer’s severe stage” - so they are biomarker positive- but they don’t have

\textsuperscript{371} Lawrence actually opposed this art to the scientific reductionism associated with laboratory practices in “Still Incommunicable: Clinical Holists and Medical Knowledge in Interwar Britain”. In Lawrence and Weisz (ed.) Greater Than the Parts: Holism in Biomedicine 1920-1950. New York: Oxford University Press. 1998, p 96.
\textsuperscript{372} “Trained judgement” is a mode of their history of objectivity (in Lorraine Daston and Peter Gallison, Objectivity. Cambrige: MIT Press, 2007, Chapter 4). Like Foucault described modes of truth, Lorraine Daston and Peter Galison describe modes of objectivity.
any cognitive decline... So how do we decide if we should treat them or not [in the hypothetic case that a treatment would be available]?

Vincent (smiling, inspired). - I use to say that because of the biomarkers, we need to be better clinicians, because everybody will have access to the biomarkers and if we don’t control precisely the clinical phenotype we will make a lot of mistakes. So we need to be better clinicians, because of the biomarkers. And paradoxically, because of the biomarkers, we will have to teach to our students exactly what is the phenotype of Alzheimer’s disease. So for example: I am loosing every time my glasses everywhere, I am very you know... I have attention disorder... (Laughing).

Some laughter in the audience.

Vincent. - So if someone said I have Alzheimer’s disease I would say NO!

Bigger laughs.

Vincent (laughing). – No... (Serious again.) So we should be very prudential. Alzheimer’s disease is not... En français, on dirait: “Ce n’est pas n’importe quoi”: It’s not anything. It’s a very specific... I think we should work on the clinical phenotype because if we don’t do that... if we leave this [the biomarkers] to anybody... This is the reason why I want to keep this for research because if we leave this to any GP... (Vincent seizes an imaginary thing between his index and his thumb.) It’s so subtle... It is important to have our... (To Daniel.) YOUR expertise to say: “OK the biomarkers are positive, this fit well with the clinical condition, this is Alzheimer’s disease”, and only experienced people can do that.

Dr Daniel nods.

Alzheimer’s diagnosis, said Vincent is not anything and it is not for anyone. The biomarkers might become available for everybody (“any GP”), but as Laura pointed out, since 30% of people are biomarkers positive but don’t have the disease, there are risks for misdiagnoses. To avoid these mistakes, Vincent thinks the diagnosis of AD should be reserved for experienced neurologists: they know about the subtlety of a disease that cannot be reduced to the level of two biomarkers, neither to a vague symptom that can affect anyone at anytime: forgetting where one left one’s glasses. But, if these neurologists have experience, what are they the experts of?

After his talk, I saw Vincent alone in a room of the Memory clinic. He was waiting to be shown around. I asked him to tell me more about his expertise and about how it played in his knowledge of the disease. He answered me enthusiastically: “It’s an immediate impression, it’s amazing: the patient enters my office and Paf! Right away it evokes something... The diagnosis is made... Then, you are going to set off all the confirmation processes [imaging, neuropsychological and biological exams] and that will fit with the impression or not... But the truth is: it’s like visual recognition, like... I am very keen on modern art and it’s interesting: when you see a painting in a snap shot you identify the painter, it’s immediate, if you know a little bit about art you’d recognize immediately a Rothko or a Staël. With the patient it’s the same. We
might think that there is a very systematic approach, that we need to go step by step, unrolling everything but that’s not the case. Maybe what I am saying isn’t good, maybe it isn’t true ... (Louder.) IT IS TRUE but it’s maybe not... how to say.... politically correct. (After a silence.) There is a neurologist at the Alzheimer clinic, but I won’t tell you who that is, who is not a good clinician: when that person makes a diagnosis I usually consider that I need to make the opposite diagnosis... For the clinic, you need to have the sense.”

“Experience”, “subtlety”, “impression”, “immediate recognition”, “sense” again and “art”. Vincent, this time, didn’t compare the good, experienced and subtle clinician to a gun dog but to an art connoisseur. It is now an acquaintance with a form, a style or a gestalt, which allows the clinician to perceive, or to be impressed by, the disease behind the patient. Like the art lover who identifies the artist behind the art.

Vincent, as with the smell, repeated the holistic quality of the clinical sense, but he developed here one more quality of this sense: its immediacy. The clinician identifies the disease in a glance; a keenness to which Vincent opposes the slowness of the analysis. It is indeed a “process” of appointments, paperwork, movements to a place and to another, encounters with several people, time to do the exams and an even longer time to analyze, to read, to score and to interpret them. Among these exams: the “battery” of tests given by the neuropsychologist. We only had a little glimpse into the variety and the multiplicity of these tests right before the beginning of this chapter. Surprisingly at the Memory clinic as well as at the Alzheimer clinic, only a few features of this plethora of assessments will, at best, be taken into account by the neurologist at the moment of the diagnosis. At worst, the conclusion of the neuropsychologist will be courteously listened to but not heard. Unlike the neuropsychologist who needs to perfuse the patient with an IV of tests, unlike the inexperienced student who unrolls and details every steps of the diagnosis—but also unlike the unsubtle GP who might be quick but unrefined—, the experienced neurologist, who pertains to the elite of art lovers, glimpses at the patient still standing at the doorstep of his office and recognizes immediately the signature of such or such disease. Daniel would agree with that: he, who admired his mentor Franck Benson, pictured him to me as this kind of neurologist who “did a lot of looking, a lot with him was experience, he had seen everything, he had the gestalt for how things turned out”. The experienced neurologist is an expert in connoisseurship. This is not a politically correct thing to express, says Vincent, but only if we admit a rigorously quantifiable and positive definition of scientific knowledge.

Indeed, the comparison of the clinician to an art lover that Vincent deployed to describe this elusive clinical sense, somehow points at a “subjective” mode of knowledge. The historian Steven Shapin reminds us that taste and odor “have been widely accounted for the most private, arbitrary, and lest discussable of all subjective modes”. Now, when Vincent compared the clinician to an art connoisseur, our understanding of the clinical sense tends more and more towards a definition of taste as a liking, a fondness or a preference. Something that would be rooted in a “sentiment” or a “feeling” that has to do with me only: something subjective. This is one definition of taste. Another, the other, is “taste” as a sense that perceives a flavor or a savor:

373 Vincent is himself a connoisseur of modern art; he has actually decorated the Alzheimer clinic with the work of some famous French contemporary artists who are his “friends”, and who gave him the pieces suspended on the walls in the waiting room and the corridors of the French clinic.

like when one tastes a lemon, if asked what one tasted one would answer: “It’s a lemon”. Taste taken with this second meaning doesn’t seem to retain anything of the subjectivity that Steven Shapin reminded us of, especially in an era where the “objectification” of taste and smells has been launched by the agrifood sector through “sensorial analysis” and “calibration of stimuli”.

When Vincent said he was sniffing like a dog, tracking the disease like a game, I freely compared him to a gun-dog for whom we actually cannot say how much its “liking” or “preference” for the smell of the game intervenes—or if it intervened at all—in its capacity to reach it in the density of the woods. So how are we to understand Vincent’s clinical sense: as something objective, as something subjective, or as both?

David Hume in his essay *Of the Standard of Taste* has analyzed that if the proverbial *De gustibus* “has justly determined to be fruitless to dispute concerning tastes”, one must still acknowledge that there is a “species of common sense which opposes it, at least serves to modify and restrain it”. First, Hume observes that taste admits some “general principles of approbation” (Hume gives the example of Homer, Ariosto, John Milton and Joseph Addison) as well as general principles of “blame” (two poets get condemned by Hume: John Ogilby and John Bunyan). Second, some people are recognized as more competent judges than others, Hume fashions their “delicacy of taste” as an alliance of finesse—to allow nothing to escape—and of precision—to perceive every ingredient in the composition. Third, taste can be educated through a “practice” and “the frequent survey or contemplation of a particular species of beauty”. Thus if taste doesn’t lie entirely in the object, it doesn’t fully lie in the subject either. Hume, then, wonderfully illustrates how this dual property of taste allows for a privileged access to knowledge, with a story recounted by Sancho to Don Quixote. This story stages two parents of Sancho, known for their good judgment in wine. His two parents, tells Sancho, were “once told to give their opinion of a hogshead”, supposed to be excellent. The first one tasted it, thought about it and found it good, despite a slight taste of leather. The second one tasted it too, liked it too, if it were not, he said, for a taste of iron. Sancho’s parents were “both ridiculed for their judgment. But who laughed in the end?” asks Hume: once the hogshead was emptied they found at the bottom of the pitcher “an old key with a leathern thong tied to it”. Hume concludes that: “Though it be certain, that beauty and deformity, no more than sweet and bitter, are not qualities in objects, but belong entirely to the sentiment, internal or external; it must be allowed, that there are certain qualities in objects, which are fitted by nature to produce those particular feelings”. This story re-told by Hume brings to light the “feeling” of Sancho’s parents: the first one’s dislike a taste of leather in the wine, and the second one dislike of a taste of iron, were in accord with the qualities that pertained to the object lying at the bottom of the pitcher: the key. Of course, to perceive these qualities and to experience these feelings, one needs to first possess the delicacy of taste of Sancho’s parents, like Vincent said about the diagnosis of Alzheimer’s

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375 This is how Thomas Vanheebergen, a doctoral student who studies how these industries understand taste, presented to me his current Ph.D project.
377 Ibid. p. 214.
378 Ibid. p. 218.
379 Ibid. p. 221.
380 Ibid. p. 216.
381 Ibid. p. 217
382 Ibid.
disease: it is not “anything”, it calls for an expertise. Yet, it is an expertise close to connoisseurship: not fully reducible to an objective definition of science. In the rest of this chapter, I will thus envisage this clinical sense like connoisseurship, trying to get closer and closer to the participation of some “feelings” in the diagnosis, to understand better what these “sentiments” are about.

Two words of caution though: in my efforts to delimit the role of these “feelings” and “tastes” in the production of a diagnosis, I must be cautious of not hardening too much the following notions by delimitating them with oppositions: objectivity versus subjectivity, reductionism versus holism, mechanistic versus sensual, creative versus boring, etc. The risk is to maintain a false dualism between feeling and (objective) knowledge, when my intention is to study how these “feelings” actually participate to knowledge. Moreover, in practice, the division between two “ways” for making a diagnosis impersonated by the split neurologist described by Vincent at the beginning of this chapter—one time a doctor clinician, another time a doctor researcher-- is of course much more porous than the impression we might have had from what Vincent told us in the car. It is not the case that Daniel, Vincent, and anyone who participates to the making of a diagnosis, when they enter in the clinician modus operandi would abandon at the same time what seems to owe more to the laboratory than to sensuality and holism. Already in Vincent’s last remarks, we well understood that to arrive at a diagnosis the neurologist will always go more or less down both roads; the clinician launches the exam process that allows to see if the clinical impression “fits” with the imaging, with the neuropsychological exam, with the levels of proteins in the spinal fluid, and with the DNA sequencing. Vincent explained that these tools validate his sense.

I will first develop, in the next two sections, how teams of diagnosticians at the Memory clinic actually combine their elusive clinical “sense” or “feeling” with the biological tools, to make a diagnosis. Two patients’ cases (Dr. K and Cheryl Joe) are developed in extenso, respectively in sections 3 and 4: this amount of detail and this slow progression seemed unavoidable in order to begin to understand how feelings progressively appear in the everyday practice of these teams, how they are formed, how they are discussed, and how they are shared.

3. Dr. K: “Actually I love tests.”

Dr K. is in his 70’s and was taught obstetrics in the prestigious University of R., where we are. This March morning, he returns to R. to ask for a second opinion: the one of the experts at the Memory Clinic. He came here alone, his wife was too sick to be there. John is the fellow examining him; John starts his interview: “So in general, Dr. K., in terms of cognitive things... where do you think you fall compared to the people of your age?” Dr K. replies: “Nowhere, I think I am better”. The physician explains why he is at the Memory clinic today: he has no intention to stop working but the director of the clinic in Santa Barbara, where he has practiced as an OBGYN for the last 30 years, forced him into early retirement. During these last two years, twice, Dr K. forgot sponges in the vagina of his patients, he shrugs: “Twice in two years, that’s

383 It is indeed in the American clinic, and not in the French clinic, that the “tasting” of brain diseases takes a salient place.
not unusual”. There is something else: during one busy night (six births), Dr. K. slightly dreamy, wasn’t paying attention to what the nurses were telling him about the patients; a distraction with no medical consequence but which raised suspicion. Actually, Dr K. says he feels a general climate of bad vibes around him and he thinks it arose from the complaints of three patients unhappy with their C-Sections: “In one year, two complaints is OK but three...” All of this led the director of Santa Barbara clinic to send Dr K. to a neuropsychologist in order to test his memory. Dr K. first forgot to go to the appointment. Finally, the test was done and the report that came back was alarming: the gynecologist would be “at the edge of MCI” (Mild Cognitive Impairment); if this verdict spares him for now from dementia it assures him a premature retirement. Until further notice, the director of the clinic forbade him from being on duty.

This Friday afternoon, at the Memory clinic, Dr Martin is the attending doctor running the discussion of Dr K. The team meets up at one p.m. to discuss about the case, while eating a salad prepared at home early in the morning or some chips bought on the sidewalk in front of the hospital. John the fellow and Brian the neuropsychologist who interviewed and tested the patient this morning, are going to set out his case for Dr Martin. The OBGYN, reminds Martin of the story of another OBGYN who ended up having FTD: “It was in the New York Times: he did a C-section and put his initials on the tummy of the patient!” Martin tells us, joyfully. John smiles and starts his presentation.

John. – The tricky thing is that his wife is quite ill, we have no corroboration yet and we have a patient who is over motivated to minimize everything... Anyhow, during the last three years, Dr K. noticed some problems like misplacing objects and sometimes he substitutes words without thinking, other than that he says that he is pretty functional. He seems mostly happy. Functionally, he is kind of responsible for the entire household.

Martin. – So he does manage finances... his wife is not going to be helpful, but I guess we are not going to be able to diagnose dementia in this guy, he would not be at this level otherwise he would not even come here; so the question is does he have MCI? And this is going to be difficult...

John. – He had 27 on the MMS done in Santa Barbara, he could not remember the three objects.

Martin. – It’s going to be very telling: the NeuropsyK [neurologist’s abbreviation for neuropsychological test] is going to be the most useful thing.

Brian (jumping in). – He interrupted a lot.

John. – He is a very talkative kind of guy...

Martin (cheerful). – He is a true doctor! Trying to get over! The anthropologist will take note about the alpha male... he is a silverback!\(^{384}\)

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\(^{384}\) A silverback is a gorilla typically more than 12 years of age and is named for the distinctive patch of silver hair on its back. The silverback is the center of the troop’s attention, making all the decisions, mediating conflicts, determining the movements of the group, leading the others to feeding sites and taking responsibility for the safety and well-being of the troop. Younger males subordinate to the silverback, known as blackbacks, may serve as backup
**I take notes.**

John pursues with the physical exam, specifies only what was concerning: it took a little bit more instruction than expected, especially the Luria sequencing.\(^{385}\)

Martin (*enthusiastic*). – This is tough! He reminds me this case of FTD a little: he was a professor, came to class, rambled, brings a video and plays the same video the next day; when the students complained he said that education is a lot about repetition! It is not uncommon for this kind of high functioning people to be able to cover up. You’ll find in all university departments these profs who are still around because they have people to cover for them: an administrator picks them up from work, brings their mail, they take a nap, it is fine if they don’t do anything, they are emeritus even if they have reached the stage where they are incompetent. But you have to talk to their assistant and not to their wife. So for this guy: to know what really is going on, talk with an administrator.

**Sequence 1: The informant.**

According to Dr Martin we will not be able to get an accurate idea of the state of Dr. K’s brain; at least not by having a conversation with the patient. As soon as John started to present his case, he warned us of a problem: Dr. K. came alone. Usually patients don’t come alone: they come with an “informant”. Usually, while the patient is doing the tests with the neuropsychologist, in another room the informant is having a private chat with a nurse or a social worker; in this confidential setting, the one who takes up the role of the informant—the spouse, the child, the parent, sometimes the friend—has the opportunity to speak freely of the patient. Yet, today there is only Dr. K., the informant did not come, and in general I can say that neurologists do not believe their patients are good informants. They have several reasons to think that. Here, as we know, Dr. K. wants to go back to work; thus he has an interest, as John said, in minimizing his troubles. But there is more: Dr. K is a “true doctor”. It apparently means, that Dr. K gets his way better than other patients. Doctors, says Dr. Martin, like professors, belong to a “high functioning” group; the group of the chiefs: if he were a gorilla Dr. K will be a silverback. It is thus difficult for John, who is also a doctor but a young one (if he were a gorilla John might only have been a blackback), to fail Dr. K, it might even have been difficult for John to run his interview properly. Because Dr. K is strong, he is not trustworthy. In any case, Martin seems to think that Dr. K’s wife wouldn’t be a good informant either, because she is already being cared for by her husband. Only one person could reliably inform us about the patient: someone who works as an assistant for Dr K. (a nurse, a secretary); someone who actually takes care of him and of his errands and who might cover his errors for him.

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\(^{385}\) The Luria manual sequencing task is named after his inventor, the famous psychologist Alexander Luria. In this test, the patient is asked to tap the table with a fist, open palm, and side of open hand and then to repeat the sequence as quickly as possible. It’s a difficult task that very few patients (demented or not) succeed in doing straight away.
In this case, with no proper witness available, Martin can only rely on the objectivity of the neuropsychological test, freed from the anthropological parameters that interfered with John’s exam.

Martin. – So Brian, describe him to us!

Brian. – He really had a hard time understanding the directions. He repeated the instructions a lot. There were only a few tasks for which he didn’t repeat what I just said... (To a pharmacist intern who was there during the test:) Right?

Pharmacist intern (cautiously). – Yeah, well... I could not tell if he was anxious about not doing something wrong or if he didn’t understand the directions...

Brian (coldly). – That’s fair but well... He got 30/30 at the MMS.

Martin. – Why don’t you tell us about the previous one [test in Santa Barbara]...Why they interpreted it the way they did?

Brian. – In January, he got basically 99% verbal, 99% intellectually, 98% executive...

Martin. – Except...?

Brian. – For memory, the scores were average\textsuperscript{386}; he was in the 50% percentile, loosing track of his thoughts. Verbal skills: 90%. Visual memory was not as good: in the 51% percentile. He recalled the history immediately. After a while he got only 24% of the history... though it is still in the norm...

Martin. – Their basis was his performance on memory compared to the rest.

Brian. – Yeah. I gave him the long form\textsuperscript{387}: he got 4, then 9, then 12, then 7, and then 12. All of those are in the normal range, when he recalled larger numbers of words he had more repetitions and when he scored 7 he forgot a category. After ten minutes, he recalled 8.

Martin. – That’s not really too bad...

Brian. – Yeah, that’s the right average for his age. Executive: a little bit slow for doing trails. Color naming: average. Inhibition: more than one standard deviation below. D words: 32. Semantic: 26. Copy was pretty good and the recall...

\textit{Brian shows us the drawing.}

\textsuperscript{386} Santa Barbara's evaluation « is in the normal limits » for all the capacities tested.

\textsuperscript{387} The neuropsychologist tells the patient a list of words (16 words for the "long form" or 9 words for the "short form") and then asks the patient to reconstruct this list from memory: there are five trials of this kind to test what is called the "free recall". After these five trials, the patient is given some cues to help him reconstruct the list; this task tests the "cued-recall". Finally, the neuropsychologist suggests some words and asks the patient to say if these words were on the initial list: this task tests "recognition". These three different tasks allow evaluating troubles linked to learning memory (the first task) and troubles linked to stocking and recollection (the second and third tasks). Finally after 20 minutes (during which the patient was doing other kind of tests), the patient is asked to recall the words of the list (long-delay free recall).
Martin. – WOW! Not bad!

Brian (incredulous). – A little bit above... (A bit disgusted.) All for visual memory... And abstract reasoning was fine, I guess... for the second proverb, he used the still water analogy and it’s not related.

Martin (sulks). – It’s kind of related... OK, like I said, there is no way we can diagnose dementia... I have a gut feeling this guy is on the edge of MCI. Actually MCI is too much ahead; it would be e-MCI, early MCI, a kind of MCI... So this does not make him MCI but at the worse he would hide with it.

John. – The report in Santa Barbara said he would be at the edge of MCI, the neuropsychologist did not say he needed to stop working, they wrote that when the stakes are high...

Martin. – I don’t know what to do...the only time he had a problem is when he left the sponges... Let’s look at the scan.

Martin, followed by John, goes to the back of the room to look at the MRI on the small screen of a computer. The intern in pharmacy stays behind them and tries to see. From where we are, we hear them comment but in such low voices that we don’t know what they are saying. Brian, bent over his tests, talks to himself: “They should have different standards for medical practice... I don’t know, there was something with him...”

Sequence 2: The test.

In Santa Barbara, Dr K.’s results on the test were unequal, his memory not as strong as his excellent achievements in the other domains of the test. At the Memory clinic, the test did not conclude such disequilibrium. On the one hand, some of the capacities tested in which memory is not supposed to play a direct role (the executive) were not amazing, and on the other hand Dr. K’s memory seemed good to Martin—or “not really too bad”. John was more severe: “It is all for visual memory”; Dr K. would absorb all his mnemonic capacity to remember drawings and none would be left for auditory memory. In any case, the results on the test didn’t settle the decision as it was hoped; Martin is left with “a gut feeling”, he says, “that this guy is on the edge of MCI”. Why, since the results were in “the normal limits”? For a physician, Dr K. should be better than he is, he should be perfect. As Brian remarked: a regular assessment with the neuropsychological test is not enough; to evaluate doctors different standards are needed. The neuropsychologist in Santa Barbara also had reservations for when “the stakes are high”: lives of babies and mothers are at risk.

It is out of the question for Dr. Martin to conclude dementia (he doesn’t even bother specifying the type of dementia it could be), but he also rules out the possibility that Dr. K. could be normal. There is only one left: MCI, but Martin is not ready to conclude MCI. Thus he looks for a middle way: the prodrome of the prodrome, e-MCI, early MCI, edge of MCI. Like the tester in Santa Barbara, Dr Martin does not want to rule out the prodrome but he does not want to accept it fully either.
Dr Martin, John and the intern come back and sit around the table.

Martin (perplexed). – I don’t think... if you believe hippocampal volume has any meaning, he has fat ones. I am just trying to decide how much we should equivocate... (To John:) what do you think?

John. – I don’t know if there is anything that we can say...

Martin. – I don’t think we should make a recommendation...He does not meet any criteria for dementia...and MCI? It is quite possible he had a cognitive decline from high level...The diagnostic impression is important to us but... (Firmly, to John.) You should clearly say that he does not meet any criteria for MCI or dementia and we cannot say if it represents any decline... In a way we are not qualified to make a decision to say if he can practice, we are like the DMV, the DMV evaluates. We are giving the second opinion, they can fight about it; they’ll get the lawyer. It’s their job not our job. Sooner or later you’ll be involved in a legal situation. Like a change of will: an elderly man who took up with a younger woman and rewrote his will. Frequently, we are asked to say if he was demented when he wrote his will and it’s impossible. Even with an AD diagnosis, we cannot prove that a person knew what he owned and who was his relative. (After a silence.) It is interesting how medicine and society are linked.

John. – Yeah, I want to start this study on the financial judgement of FTD and Alzheimer patients.

Martin. – But we don’t even know how normal people make financial judgments! ... (Laughing.) I hope we don’t talk to his wife and she says he puts dirty clothes on!

Sequence 3: The MRI.

After looking at the MRI Dr Martin’s assessment changed a bit. The MRI showed chubby hippocampi and the neurologist decided to not be equivocal. If Dr Martin says that it is not his job do decide if Dr K. has the right or not to practice Obstetrics, he knows that words like dementia, Alzheimer’s and MCI (and even edge of MCI) will decisively change the future of the gynecologist. He decides to avoid the word MCI, John will make a different report than the one done in Santa Barbara. It is impossible to affirm that the normality of the image decided the diagnosis (and the fate) of this man but we can reasonably assume it. However, at the end of the discussion, Martin joked that everything is still possible: Mrs. K. could reveal to the team that her husband has already reached an advanced stage of dementia.

388 The hippocampi are two structures (one on the right, one on the left) located in the medio temporal lobe of the brain. They are thought to play a major role in memory (short term and long term memory) and in spatial navigation. In Alzheimer’s disease they are expected to be one of the first region of the brain that suffers damage. A way to appreciate their function is to measure their volume on a MRI. This measurement can be done at a quick glance or with the aid of a software: the smaller the volume, the poorer the memory.
Later in the afternoon, Dr Martin and Dr K. meet for the first time. Mrs. K is there, she felt better. “How does she find her husband?” asks Martin.

Mrs. K. – He is crazy... (Catching herself.) No, I shouldn’t say that... He is crazy about his work: he loves to bring babies to the world.

She also says she finds him in a better shape since he stopped doing nightshifts; her husband was sleep deprived, this is how she explains his memory lapses. Dr. Martin asks her if she thinks that her husband’s memory has changed in the last year.

Mrs. K. – As far as getting older I would say yes... But I always thought he was spoiled: the nurses take care of him. There is more of filling in but I have not experienced him not remembering something.

Dr. Martin (to Dr. K.). – Do you go shopping without a list?

Dr. K. – I try to practice with six things.

Dr. Martin (to Dr. K.). – Can you tell me the three words that you learned this morning that were on the first list?

Silence.

Dr. K doesn’t remember any.

Dr Martin. – Mmmm, I would have thought you would have done better with those.

Dr. K. – Well, I have to come up with a strategy.

Dr. Martin. – Is that something you have done all your life: strategies?

Dr. K. – Gosh! That’s very hard to answer... I cannot come quite with a situation.

Dr. Martin. – The results of your testing show that there is nothing that is abnormal, the worse you do is average. That is not to say that there is no decline and your memory is more close to average than the rest of your tests. So the most I can say is that there was a drop but I can’t say that it is abnormal. I don’t know what I would do in your situation... There is a memory decline, but we are so far away from the early stages of AD.

Mrs. K. – The person who did the testing made the suggestion of him not working.

Dr Martin. – I don’t really feel confident to say if you should practice or not, what the neuropsychologist was reacting to is that your memory was lower than in other domains. We cannot really rule out that there is a decline in your memory....

Dr K. – But what does that mean? That I passed?

Dr Martin. – If we had decided you had AD, we would probably comment on it and say you shouldn’t practice.
Dr K. – And I would say too.

*Dr K. smiles. He stands up, his wife too.*

Dr K (joyous). – It was a very enjoyable experience, actually I love tests!

*Dr K. and Mrs. K. leave.*

*Dr Martin takes the way back to his lab. John and I are following him in the hallway.*

Dr. Martin (annoyed, to John). – His memory was not that good. He was effortful. If I had to bet, I would bet his memory is declining.

**Sequence 4: A bet.**

During his brief encounter with Dr Martin, Dr K. was asked to repeat the words of the morning’s list. The little memory exercise wasn’t a success: this afternoon the gynecologist could not reconstruct the list of three words (ball, flag, tree) of the MMS. To succeed in this kind of exercise, he explained, he would have needed a strategy. “Is that something you have done all your life: strategies?” The neurologist asking that question, suspects that Dr. K’s memory might not be working as naturally as before, that it needs tricks to function, some sorts of internal thought devices to compensate for recollections that don’t come spontaneously anymore. That Dr. K failed the little memory exercise obviously instilled a doubt in Dr. Martin’s mind. Enough doubt to reverse the meaning of the MRI and of the neuropsychological test? Despite the normalcy of Dr. K’s memory attested by these two central tools, Martin is told John that he would have “bet” that Dr K’s memory is “declining”. The decision to say that Dr. K wasn’t sick is now, not only questioned, but more categorically, is transformed into the certainty (it is worth a bet) that there is something. “Something” had already been perceived by Brian, and an impression (“the diagnostic impression” said Martin) had already been evoked during the conference, but the bet conveys a more powerful feeling than an impression. Martin didn’t tell us much, and certainly not something as precise as a diagnosis, he expressed a suspicion: his patient’s memory is “declining”—it is unclear to what extent. Dr. Martin left us in the corridor with the feeling that there is some hidden meaning that he perceived; yet it is unclear what meaning he perceived. And we won’t know more: Dr. K left the clinic joyous, he will be able to practice again what he loves doing, while Dr. Martin left the clinic annoyed, he felt something, he would have bet on it. Yet, unless Dr. K and Dr. Martin’s paths cross again, Dr. Martin will never know if he would have won his bet.


Cheryl Joe came to the hospital of R. in September 2011. When I entered her hospital room, she was sitting on her bed, a foot on the floor the other swinging. I asked her how she was, she said: “I feel strange”. The load of tests during her stay at the hospital tired her and, as she has already described to many of her doctors, she feels “spacy all the time”; it is a sort of lightness in
her brain, she says while brushing the back of her head. It is like being “cooped up”. It means: “Tight, a small space like a chicken”, her husband explained to me, sat on a chair next to her.

Cheryl Joe is a slim and tall woman with straight gray hair. She looks stoic. She doesn’t talk much because, she says, she doesn’t like to be the center of the attention. At the hospital, she says she felt like an animal in a zoo. When Mrs. Joe talks, she suspends her voice and her gestures as if her thoughts took the time to voice in the pacing down of her whole attitude. Neurologists say that she is slowed. Her late answers are always precise and sometimes they are curt. Her deadpan humor, condensed in a short line said with her thin and grave voice, strikes all the doctors of the clinic who examine her. Mr. Joe remembers that ever since he has known his wife, her dry humor has surprised many. Some do not understand it. Mr. Joe smiles: maybe the doctors of the Memory Clinic will impute it to her disease. He himself enjoys the jokes of his wife; he welcomes them with a big laugh that trembles all alone in the room.

Cheryl met her husband at a football game in college. He fell in love with her right away. He left her indifferent; she wasn’t particularly impressed. Persistent, Mr. Joe finally got Cheryl to agree to go on a date. They went to a bar, he was nervous; he kept bumming cigarettes from her. After a power cut in the bar, they went for a walk and then, she fell in love with him. They got married a long time after that. When John the fellow, asked Mrs. Joe what she still enjoyed doing today, she said: “Walking, swimming, being in love with my husband”. Mr. Joe too, from the way he listens to his wife, seems to love her. This visit to the Memory Clinic is their last attempt, says her husband, to understand why “a high functioning person, suddenly crashed in the space of one month”. “But, let’s not get depressed”, Mrs. Joe tells me, stoically. Thus, they try to live the way they want: “Doing a lot of cruises, seeing a lot of family... and that’s what we gonna keep doing because we don’t know what the future holds”. For now, they hope for a diagnosis after the team conference of this afternoon. Waiting for this conference makes Mrs. Joe nervous.

Patients like Mrs. Joe, who stay a few days and nights at the hospital, take part in a research program of the Memory clinic. All the tests, the imaging, and their stay at the hospital are financed by this research program. The team conference for these patients—supposed to be more complex cases than others—is often run by Dr Daniel, the chief of the Memory clinic. This research program is “Daniel’s baby” Bruno told me the first time I met him. Bruno is the research programmer who plans the stay of these patients. Several times during the day, he crosses the street between his office and the hospital to see its residents and to re-explain to them in minute detail, articulating each word, the program of the day already printed on a piece of paper. Bruno gets round any kind of incident: a fellow went home sick, the MRI machine broke down, the neuropsychologist is delayed, a patient makes a scene in the hospital... Sometimes, Bruno writes scientific articles. Right now, he is writing an article on three cases with initially unclassifiable symptoms, for which it was just discovered that they carry a gene casually called “the C9”\(^{389}\), that predisposes them to FTD or/and to ALS. The day before the publication of the article revealing the discovery of this gene, I met Bruno in the corridor: he told me solemnly that something

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\(^{389}\) C9, or C9ORF72, is a gene partly discovered at the Memory clinic and partly in another clinic in the US. It predisposes to FTD or ALS or FTD and ALS and sometimes to Parkinson’s disease. It’s penetrance isn’t full: sometimes carrying this gene doesn’t cause any of these diseases. See “Expanded GGGGCC hexanucleotide repeat in non-coding region of C9ORF72 causes chromosome 9p-linked frontotemporal dementia and amyotrophic lateral sclerosis”. Mariely DeJesus-Hernandez, Ian R. Mackenzie, [...], and Rosa Rademakers in *Neuron*, Oct 20, 2011; 72(2): 245–256.
decisive happened at the Memory clinic, but he didn’t want to say anything. He looked so moved that I guessed: the Nobel Prize for Dr Daniel? It wasn’t. Bruno didn’t reveal anything about the C9 either—Bruno takes publication’s secrets very seriously—but he told me gravely that later he would like to be able to work in a place like the Memory clinic, which provides the means to make such “discoveries”. This young man is 25 years old and is applying this year to medical school to become a neurologist or a neurosurgeon.

This September afternoon, in the room without windows on the 8th floor, the seats around the big oval table are all occupied except one. The one at the end of the table, close to the door; it’s the seat of Dr Daniel who always comes in last. Bruno is connecting the computer to the screen to have the MRI projected on the wall. John is displaying his notes in front of him. Emily the neuropsychologist finishes grading a pile of tests. Kim the geneticist is considering her family tree. Three other persons are waiting around the table with a pen and a notebook: James, a resident in psychiatry, Leticia, a resident in neurology and myself. Beth, the nurse, went home sick but she gave a report to Bruno. Two neurologists visiting the Memory clinic—Leo from Spain and Milton from Brazil—, and two students in medical school, bring chairs and make a half circle behind us. Dr Daniel comes in, smartly dressed in a white shirt and a grey linen jacket. He closes the door.

Daniel. – What have we got?
Bruno. – Ahhhhh, very tough case.
John. – Yeah, she is puzzling... Bruno might have something...
Daniel (to Bruno). – You think you’ve got the diagnosis?
Bruno (serious). – I have my hypothesis.
Daniel (kindly smiling). – All right.
John. – I think... well, we’ll see what you think, but certain things make her hardly fit in anything in particular... actually one question is whether this is neurodegenerative... So, this is Cheryl Joe, a 63 years old right-handed woman, with multiple eye problems. She also has a family history of ALS and Parkinson’s disease. She is here for a two year history of memory loss and functional decline. These changes were initially associated with anxiety and panic attacks, although over the past year or so the anxiety totally resolved... according to the husband.
Daniel (to John). – Trusting?
John. – Uhm... hard to say... Mmm, I would say yes but... there’re reasons to feel like her husband might not be the most perceptive.
Daniel (impassive). – Have we ever had a perceptive husband?
John (laughing). – I have seen more or less. But just from gestalt: she is described as being the normal one, behaviorally... and she is not normal.
Bruno (not laughing). – He is definitely an odd informant. When I was talking to them, first I thought he was the patient...
Someone laughs in the back.
Sequence 1: The informant.

Collecting information from the patient and from his or her relatives is a crucial step for the diagnosis. We saw previously how the unavailability of the right informant hindered Dr. Martin. Yet, one problem for the neurologist is to evaluate the credibility of this information. In that respect the patient is being set aside automatically. But unlike Dr K. who was considered untruthful because of his aptitude to disguise his symptoms and to manipulate John, the young neurologist, most patients likely to have frontotemporal dementia or Alzheimer’s disease, are believed to be unable to perceive clearly their difficulties. On the one hand, because of their cognitive troubles these patients’ words might not reflect reality, on the other hand it would be in the neurological nature of these diseases to cause “anosognosia”. Anosognosia: from the Ancient Greek: ἀ- a-, “without”, νόσος nosos, “disease” and γνώσις gnōsis, “knowledge”. Oliver, a neurologist at the Memory clinic, explains to the family of his patients that “their brains don’t have the ability to know what is the problem, you know but they don’t know”. Dementia deprives the demented from their means to know in general, but also from the possibility of knowing how the disease affects them in their own flesh and blood. A patient, who has (or might have) AD or FTD (but also ALS, Lewy Body dementia, Huntington disease, etc.) is considered a priori anosognosic: the person is assumed to be unconscious of the terrible psychological and physical symptoms that the disease produces in him or her. This conviction, leads the neurologist to trust someone else than the patient: the spouse, the child, the parent, the friend, whoever came with the patient, who will thus for the occasion endorse the role of the informant. The competence of the informant to recount reality the way it is, is—even if this is logically unverifiable—usually assumed. In that case, the informant is said to be “a good historian”. Sometimes, however, informants might be believed to be incapable of recounting the facts the way they are, because they are in love or because, to the contrary, they are indifferent to the patient, or because they somehow, give the impression of being “bad historians”, or because they are “odd”, like Mr. Joe.

Mr. Joe is so odd that he could be the patient: according to Bruno, the informant might be the demented one. For Daniel, Mr. Joe, because he is a “husband” (a man?), cannot be “perceptive”. Daniel and Bruno exaggerate, they are joking, but they emphasize a reality: the team assesses the informant’s personality to evaluate its capacity to set out another individuation. If the judgment is negative, like it is for Mr. Joe who is seen as odd and unperceptive, the informant runs the risk of being disqualified from his role as informant. This is what is going to happen later: some of Mr. Joe’s remarks will not be taken into account for the elaboration of the diagnosis.

Sequence 2: The intuition.

This problem is general to the medical profession, yet in other fields of medicine than neurology this questions bears more on the conformity of the patient to the treatment prescribed by the doctor than on the diagnosis. The problem of trust is probably less crucial to the diagnosis considering that in many of these other fields of medicine, there are trustful biological markers that allow bypassing the words of the patient in order to make a diagnosis.

John continues with his presentation of Mrs. Joe’s case.

John (recites, monotone). – So the onset of change was associated with her eye problems. In 2009, she had a corneal ulcer and at the same time the panic attacks started. When she healed the panic attacks continued. She was still working at that time. Her husband is a lawyer, he manages this successful law practice, and she basically was still working full time as the bookkeeper. Then, she had another corneal ulcer and during the evaluation she received this diagnosis: FUCHS\textsuperscript{392}... Something that causes a kind of very slow progressive visual loss, which in the end usually results in the need for corneal transplant...

The idea that she was going to have progressive visual loss really exacerbated her anxiety. I think the way they think about this is: they feel like that the eye problem triggered anxiety, which seemed to set off everything. It is sort of curious, because her father had ALS and her sister had Parkinson’s disease and both those cases seemed to be precipitated by some emotional stressors. That’s kind of how they understand it, as a separated process that set off...

Daniel looks at Bruno who is smiling.

Daniel (to Bruno). – I know what you think it is...

The case is very tough, sighted Bruno just after Daniel entered the room. This kind of observation that often launches these conferences creates an exciting atmosphere; a difficult case challenges the capacity to perceive and the possibility to conceive what is announced as unconceivable and unperceivable. John approaches this complexity blindly: “not sure if this is neurodegenerative” (does that mean he thinks it is psychiatric?); at lunchtime, one hour before the conference, John was telling me he had no idea of what the diagnosis could be. However, if John—a neurologist— is sure of nothing, Bruno who is not a neurologist knows what the diagnosis might be, even if it is only a hypothesis, as he responded humbly to Daniel. Daniel looked at Bruno silently smiling, and read Bruno’s mind: Daniel knows what Bruno knows. This complicity between the professor and the novice is real: in the end Bruno’s hypothesis will be proved to be the same as Daniel’s. It is often the case that right after the conference starts, Daniel gives the impression that he knows how it will finish. Dr. Blake once told me that Daniel’s perception is a form of “intuition”; Blake, enthusiast, described to me his chief during a typical case conference: “He asks five or six questions and he is done. He is the one [neurologist] who is the most fascinating to me, because he doesn’t take much time, he listens to a bit of the history [of the patient] and he is sorting it out from the first word the resident tells him”.

Nicole a neuropsychologist who also works at the Memory clinic, doesn’t say Daniel has intuition but “wisdom”, I asked her what she meant by that. She started to explain that Daniel’s wisdom owed much to his experience: “This patient A and this patient B and this patient C, they don’t behave all the same but as we stack them up in our experience, there are certain patterns that start to naturally extract themselves. The medio-frontal cortex does this; it’s just fascinating how this works! Overtime it’s the pattern that you see and not the details anymore and I think overtime

\textsuperscript{392} Fuchs’ dystrophy also known as Fuchs’ endothelial dystrophy, is a slowly progressing corneal dystrophy that usually affects both eyes.
the diagnosis is just about a certain amount of wisdom: it’s just like you realize that “that” symptom is important.” Yet, Nicole also says that it is not always a “good thing”: seeing the “pattern”, the “structure in the chaos” might take one away from very important details to which the “novice” is still attuned. Nicole concluded on a real lesson of wisdom: “There might be pieces of the puzzle that will come from the novice, and a good attending will listen... A good attending will listen.”

**Sequence 3: The symptoms.**

John pursues his presentation. Last year Mrs. Joe, because of her panic attacks, went to see a psychiatrist who gave her antidepressants. Then, Mrs. Joe started to forget. Once, she was at the hardware store and suddenly she didn’t know where she was. She forgot where she parked her car. She forgot the name of her psychiatrist. And she misplaced her glasses, John tells us. After a while, her psychiatrist thought whatever Mrs. Joe had, it didn’t look like a neurosis, schizophrenia or bipolar disease; moreover the antidepressants didn’t do anything to ease the anxiety of Mrs. Joe. The psychiatrist thought it might be neurological. Mrs. Joe went to see a neurologist. The neurologist gave her the little memory test, the MMS: she got 23. A score that we know is worrisome, for these competent medical authorities. Five days later she came back to see the neurologist to do more tests, but this time she got 30 on the MMS. The neurologist didn’t understand anything; he thought that maybe her patient had a “rapidly progressive dementia”. She did a full battery of tests. The exams came back negative: Mrs. Joe doesn’t have a rapidly progressing dementia. Today, one year and a little more after her first visit to the psychiatrist, Mrs. Joe’s anxiety has eased. But her whole life has changed. She stopped working. She stopped driving because she couldn’t remember where she parked the car, she doesn’t read books anymore, and she doesn’t go to her exercise class anymore because she doesn’t understand the instructions. She feels spacy all the time. She still swims often and she still does the laundry. And she does one new thing: she plays sudoku a lot.

Daniel. – Is that a compulsion?

John. – She says she feels that calming.

Daniel (*detached*). – I never played...

John (*smiling*). – Yeah I know... So one interesting stuff in term of executive: she says she can still cook but she has to do it all from recipe, she

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393 As the name indicates a rapidly progressive dementias progresses rapidly: over a few months, unlike Alzheimer’s disease or frontotemporal dementia which are usually considered as slow progressing. Examples of RPD are Jakob-Creutzfeldt disease, or dementias with neoplastic or toxic causes.

394 She had two MRIs, had a LP (lumbar puncture); the biomarkers are half conclusive (“sort of low A-beta”) she had a PET showing hypometabolism in the medio temporal, she had two EEG (Electroencephalogram): non specific, a CT scan of pelvis searching for tumor: no tumor.

395 The executive is a quite elusive capacity tested by the neuropsychological test. Here John seems to give to it its most obvious meaning rooted in the business world: it is the capacity to manage: to be an executive.
can’t do it from memory. But she’ll work out you know, equations: if you have a recipe that feeds eight and you got five people, she’ll work out for each ingredients how much to put in.

Daniel. – A little compulsive. Is this new?

John. – I don’t get the sense...

Daniel. – Math and cooking... that’s unusual.

John. – Yeah... But she seemed to be someone very organized: her husband said she worked out a whole computerized system for his businesses.

Daniel. – Not apathetic?

John. – She says she is less interested in doing things... (Little silence.) So I am going to try to get to the behavioral issue: the husband reports she is the stable one in the family. At least for me, he didn’t report any personality changes except you know the anxiety... no change in hygiene... In terms of eating, she lost weight last year but gained it all back, and... uhm... no change in sexual function... In term of motor, she has some Parkinsonism...

Daniel (eagerly). – Good! So should we bet on what this is?

Mrs. Joe was anxious. Was she angry? Was she sad? What she scared? Was she in pain? Because she felt she was getting old? Because of the ophthalmologist, who told her that she would lose her sight? Debating on the causes of anxiety is not interesting to Daniel; at the Memory clinic an exogenous and psychological cause to anxiety, like for instance the effect of the disclosure of a serious disease, is not acceptable. Only neurological and endogenous causes are acceptable. Michael once summarized this idea for me: “We often forget that mood is part of the brain”. Anxiety is caused by a neurological dysfunction: it is part of the presentation of Alzheimer’s disease, FTD, Parkinson’s disease, ALS, Huntington disease, etc396; it is a symptom of these diseases. Daniel wasn’t struck either by the fact that Mrs. Joe now forgets things. What is striking is this insignificant hobby: Mrs. Joe plays sudoku. Sudoku is the favorite game of patients who have FTD; this is well known at the Memory clinic. Is playing to this game enough to say that Mrs. Joe is compulsive397? And these equations that she mingles with her cooking... But John stresses that Mrs. Joe has always been that way, very organized, as the quality of her work testified. Mrs. Joe seemed organized and rigorous. Did the disease make her rigid and compulsive? Or, could that be that Mrs. Joe’s qualities get expressed in other domains than her work, now that she stays home all day? This is somehow what John suggests: Mrs. Joe is of a meticulous and scrupulous nature. John takes into account the character of Mrs. Joe as he figured it out during his conversation with the couple. If he had understood that Mrs. Joe used to be extravagant and scattered, he might have thought that her sudden taste for a redundant and patient activity such as sudoku, could be the imperceptible sign of an imminent metamorphosis. Behavioral neurologists thus need to turn back to the whole life of their patients in order to apprehend their true nature and ask themselves if any changes in their habits, in their tastes are coherent with that nature. The record of these small changes cannot be done without the help of the informant: Mr.

396 Therefore it cannot be its cause, like Mrs. and Mr. Joe might think it is as John told us before.
397 Compulsivity is defined by “performing an act persistently and repetitively”.
Joe described his wife as “the stable one” in the family. Except for her recent taste for sudoku, he didn’t mention anything else. At that point, Daniel has heard enough: he is ready to bet on the diagnosis.

**Sequence 4: The bet.**

### Long Silence.

Daniel. – Okay... Mmmm, Bruno can’t vote, he’s seen the image, John have you seen the image?

John. – I haven’t, but Bruno is very bad at reading the image and like, keeping it quiet... *(Looking at Bruno, smiling.)* We basically all said that if Bruno is ever in a Texas hold’em game we would all like to be in that game!

### Big laughs.

Daniel *(smiling).* – All right... *(Turning to me:)* Laurence, what do you want? Neurodegenerative? Psychiatric?

Me *(discountenanced).* – I don’t know... Because of the anxiety and the Parkinsonism ...maybe Parkinson’s398...?

Daniel *(impassive).* – OK. Leticia?

Leticia. – For me, most probable could be a Lewy Body399, for fluctuation, for anxiety, for motor symptoms and also for visuo-spatial problems, for all these things...

Milton -neurologist visiting from Brazil-(to Leticia). – But she has some behavioral features.

John *(to Milton).* – They are more from observation than from history though.

Milton. – Yeah, but apparently the husband is not a good historian, so it looks like something is wrong...

Daniel. – So is it neurodegenerative or psychiatric? *(To Milton.)* You have a choice, don’t you? If you had to bet what would you say this is gone be?

Milton *(with authority).* – Neurodegenerative.

Daniel. – Neurodegenerative which one?

Milton *(prophetic).* – Mmm... Bv-FTD400: especially with the family history. I don’t think we can ignore that.

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398 Parkinson’s disease versus Parkinsonism. Other than these two symptoms that fit with Parkinson’s disease, I “wanted” Parkinson’s disease because it is the only neurodegenerative disease for which a treatment exists. I wanted that an anti-parkinsonian be tried out on Mrs. Joe (that hasn’t been the case yet); I would like that Daniel tries out the medication that sometimes does miracles.

399 Lewy Bodies are abnormal aggregates of proteins that develop inside neurons in Lewy Body dementia and Parkinson’s dementia. Lewy Body dementia is usually diagnosed on the presence of Parkinsonism (but not Parkinson’s disease proper), fluctuating attention and alertness, and recurrent complex visual hallucinations.
Daniel. – Would you go as far as saying you know the gene?
Milton (smiling). – Not yet...
Daniel. – What about you Leo?
Leo (neurologist visiting from Spain). – I would say like Leticia: DLB... Maybe also some AD: with the low A-Beta...? (To John.) How low was it?
John. – They just said it is low and they didn’t give us references.
Daniel (to the student in psychiatry sitting next to Leo). – James?
James. – I can’t tie it together any better than Leticia, so...
Daniel (shaking his head). – I can’t get away from the family history, I’m sorry. I think Leticia framed it beautifully, I don’t think there is any doubt this has a lot of Lewy Body flavor, but with the father having ALS...
Leticia (incredulous). – Yeah, I know.
Bruno. – Can I say my thought prior looking at the image?
Silence. We are all looking at Bruno.
Bruno. – She is the identical doppelgänger of another patient: Lea Strong...
Comparing this nursing report (holding Beth’s report) to the one of Lea Strong, you could transplant them.

In the same way as in every conference he attends, Daniel sought our opinion by asking us what we “want”. He doesn’t ask us what we “think”. In Daniel’s question, there is an invitation to get in relation to the disease on the terrain of the desire. The unshakable desire to search for the meaning of the symptoms sustains these conferences; I realized how much this desire supported Daniel’s clinic as soon as I started my fieldwork in the French clinic.

The team’s quest for a diagnosis takes shape from a desire: does it mean that the endeavor is capricious and irrational? Are the diagnosticians rocked by their impenetrable preferences that can change and vary in many ways? Let’s already remark that during the search for a diagnosis, the team leans on specific tools, discusses, argues, takes the time to weigh the pros and cons; a clinical impression is not passively felt, neither arbitrarily thought, nor incapable of being shared. What seems surprising though is the possibility for these diagnostician teams to actually succeed in making a diagnosis in a field where the climate of uncertainty is rife. I think that Daniel overcomes this uncertainty, and thus overcomes the dullness in the decision-making that might result from it, by asking all of us to express a preference and to bet on it. In this search for meaning, that runs the risk of being a quite permanent state (indeed the diagnosis won’t be know before a long time), the bet, I think, supplements our desires of an engagement without which the “inexhaustibility of the desire” – that Clément Rosset sees as one essential condition to

400 BV-FTD, Behavioral Variant FTD, is one of the three forms of FTD. The two others are the semantic variant and the non-fluent variant: both of these hit ‘language’, each in their own way. Bv-FTD hits primarily “the behavior”.
401 Clément Rosset, Le réel: Traité de l'idiotie, Paris, Les Éditions de Minuit, 2004, p 62: “To be permanent—to have no reason to stop, whatever might happen—a research needs to satisfy to a double condition: to be tuned into an inexhaustible desire, and to be incapable to ever succeed”. (My translation.)
define the permanence of a quest—could wear out. Thus, while waiting to know what the diagnosis is, let’s bet on it.

When I started the fieldwork at the Memory clinic, this mania to bet on diagnoses stupefied me at first. I would have forgotten about my initial astonishment if one of my friends hadn’t refresh my memory while I was telling him, later, about the importance of this activity in the making of the diagnosis in the American clinic and its strange absence in the French clinic. Once, when I was wondering to Vincent about the exceptional occurrence of the bet at the Alzheimer clinic: he answered me while framing the bet on a diagnosis in the description of a larger “staging”, which characterizes for him the practice of the “real clinician”. A practice underlined by the appetite for the enigma that fills these large case-conferences that, much to his regret, Vincent organizes only exceptionally at the Alzheimer clinic.

Dr Vincent. – There are not a lot [of neurologists] to do it [the case conference] ... and for different reasons. First, you need to have this appetite and then there is the ethical argument.

Me. – What do you mean?

Dr Vincent. – It’s to say that the patient... We’re not in a cattle fair, we’re not in a circus, there is this idea...The idea of exhibiting the patient, doctors don’t talk about it, but the psychologists say it, they think this display... (Hesitant.) They are feeling awkward. But it’s (Stressing.) THEM, not the patients... Actually sometimes I speak about this with the patients and for them: not at all... (Stressing.) They UNDERSTAND that we do it. We are ambivalent in our approach. We do it of course to solve medical questions for the patient, we do it to progress in our general knowledge of these diseases and we do it also because we probably like this theater aspect, this staging [“mise en scène”]. Because there is a staging and people come to find that, there is a diagnostic game, there is an enigma... I didn’t do it much when you [me] were there but I used to make the patient leave and then I turned to everyone in the room and I make them vote, asking: Is it organic [neurological] or not [psychiatric]? It’s like a TV show...

As I said, I have only exceptionally witnessed this “mise en scène” at the Alzheimer’s clinic in France, whereas the “TV show” runs several times every day at the Memory Clinic. At first I couldn’t have agreed more with the ethical problem brought up by Vincent (in the psychologists’ name): betting on a diagnosis, that is to say on a decision that will transform someone’s life isn’t it to resort to tragedy in order to build up the hype, like in a TV show? This staging, aside from first provoking my indignation, then appeared to me also as a source of an important density of exchanges, which very efficaciously produces a discussion that will actually end in a diagnosis.

It is a discussion that leans on emotions: like in a casino before gambling on the black or the red, I feel my heart beating faster when it is my turn to bet on the diagnosis. Yet, it will be a long time before the verdict will be announced (at the autopsy of the patient’s brain), except—as we will see in section nine, at the end of this chapter— for the exceptional case of genetically determined dementia. A long time will usually go by between the bet and its outcome, long enough for everyone to forget who said what. Thus, one could have a nonchalant and detached
attitude about the whole thing. Yet, it is indisputable that at the very moment one bets, one is doing it with engagement. At the Memory clinic it is palpable that each of us, following our arrangement around the big table, engages a bit of his credibility, his honor, a bit of himself, when he plays this game. When I arrived in the French clinic, what struck me first was the dullness in decision-making; compared to the American clinic, the diagnosticians of the Alzheimer clinic appeared to me as analphabets in emotions. Moreover, in France, when discussions such as the one that we are witnessing exceptionally occurred, only Professors in neurology (and almost exclusively men), isolated from the crowd in a small circle, participated in the conversation. At the Memory clinic, neurologists—professors and fellows—, neuropsychologists, residents, visitors and guests bet on the diagnosis. All are invited, each—cornered by Daniel’s questioning—have the same duty to play; indeed the layman, the novice or the observer can hardly be excluded from a practice that appears like a game of chance. The democratic form of this practice is thus worth noticing, even if of course, in the end, the bet of the leader, Daniel, will speak with more authority than other bets (although we will see later that Leticia, the young neurologist who is now taking part in Mrs. Joe’s conference, didn’t give up on her bet, despite Daniel’s authority). This general invitation to bet produces an affirmation and also an exaggeration of opinions that asserts the decision. The practice of betting on a diagnosis underlines the uncertainty in which behavioral neurology evolves today—it is like a game of chance—, and as a result of this uncertainty the necessity to engage actively one’s decision. Against uncertainty, there is nothing like a good and robust bet. I think that as a result of this engagement, the team at the Memory clinic does actually make a diagnosis more easily and more frequently than the French team. This decision might be questioned afterwards, a diagnosis will not be necessarily given to the patient but one will emerge from the discussion; it is far from being the case in France.

Sequence 5: The twinship.

Daniel (to Bruno). – Mmmmm ... What Beth says in the report?

Bruno reads Beth’s report in one go. After the discussion Beth had with Mr. Joe, her informant, Beth wrote in her report that Mrs. Joe “compulsively plays Sudoku”, is “apathetic regarding the household”, that she does “some cooking but is very methodical about it: crossing each step while it is completed”. Mrs. Joe is also “methodically taking her eyes drops everyday”. Mr. Joe did say that his wife “cares much about her family but has much less initiative”, Bruno reads: “so more apathetic.” We might think, on the basis of this report, that Mr. Joe, when he talked to Beth about his wife in the intimate setting provided by her absence, used himself those terms: compulsion, methodical, apathy, all pathological, all pertaining to the pro language. It is not the case: as we will see later Mr. Joe doesn’t know what apathy means. These are adjectives chosen by Beth to characterize Mrs. Joe’s attitude from the descriptions given by Mr. Joe. This imposition achieved by Beth makes some FTD symptoms appear now clearly, when before they were veiled by John’s hesitant report. The nurse’s report, thanks to its command of a specific language, is a critical step in the revelation of the pathological. This pathology, concludes Daniel, is the one of “bv-FTD”: “We have apathy, we have compulsive repetitive motor (Sudoku,
crossing while cooking, taking eye drops). Though, some symptoms are lacking: “We don’t really have a good eating story and we don’t have loss of sympathy and empathy for people”...

A silence.

Daniel (after thinking). – But she is atypical. I cannot say that the genetics have not totally influenced the way I heard the story. (Pensive.) I keep trying to suppress it, but in my heart to heart I believe this is gonna be that chromosome 9 gene, which our patient Lea carries. I think the gene carriers are not so typical of FTD, but... you know... it’s a stretch.

A silence.

John (with animation). – So on exam: she is slowed and she has quite of very deliberate manners, and one thing that struck me is that she has a very quick kind of wicked sense of humor.

Daniel (intrigued). – Give me some examples.

John. – So one example was that her husband said that they went for a tour in France and she said: “We must have walked by some mad cows!” (Laughing.) Or when I asked her to give me her thumb for the exam, she said: “It is attached”.

Laughs.

Daniel. – So she plays with words a little bit...

James (to Daniel). – This is not very FTD...

Daniel. – I mean... Sometimes people in the humor area are overrepresented in FTD, and I think there were a number of humorous people who got FTD, not famous but you know... and specially right side cases [the lesion is on the right side of the brain] do a lot of punning and playing with words ... but it’s not quite that...

Bruno. – I have seen a few FTDeese in the research program who had a solid ironic sense of humor including Lea.

Daniel. – Yeah, so Lea had a real love for horror, she’d just watched horror movies all day long, and her dad died of something, probably ALS, and she had that kind of dark ironic humor, and she went very slowly and a lot people didn’t think she had FTD. I did. Anyway, she’s got this chromosome 9 gene... (To Bruno:) and she reminds you most this...

Bruno (with great authority). – Doppelgänger.

What Daniel knew that Bruno knew since the beginning, was that Mrs. Joe could be sick of FTD caused by a gene. It would be the same gene, the C9, that Lea Strong was recently discovered to carry. Often, the name of Lea Strong is mentioned during conferences and the

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402 When Mrs. Strong came for the first time to the Memory Clinic, she was 48. She first saw Dr Blake, who diagnosed her with bipolar disorder. Six months after she met Dr Daniel, who diagnosed her with FTD. But the years
ones who had the chance to meet her in person (I didn’t meet her) tell to the others, who are listening with an air half-fascinated half-amused, about her weird symptoms (her love for “horror”). It is common, as much in the American clinic as in the French clinic, that a patient resembles another patient. The analogy between these patients doesn’t bear on the particular expressions of certain symptoms: for instance watching horror movie, playing sudoku, putting eye drops in the eyes everyday. This analogy often, lives on the impression of the observer on these patients; it is a resemblance that strikes the observer and that cannot be really explained. Bruno didn’t explain why, and no one asked him to explain why, he had the impression, let’s even called it the certitude, that Lea Strong and Cheryl Joe were “doppelgängers” (from the German: double goers); it is sufficient that Bruno felt that Lea Strong, like a shadow of the evil twin\textsuperscript{403}, was walking hand in hand with Cheryl Joe.

Elise, the neurologist of the French clinic, once explained to me that neurodegenerative diseases “give a coloration to the personality”; for instance, she recognizes the same “sort of anxious perplexity” in Alzheimer’s patients. According to Elise, a disease gives to its bearers a sort of family resemblance; an air that the neurologist and even the novice—to the condition he has seen several patients already, like Bruno—might intuitively perceive.

Beth, the nurse of the Memory clinic is well known for her aptitude to diagnose Alzheimer’s disease in the eyes of people. She identifies their family resemblance by this “look” in their eyes... When pressed by Emily and I to say more, she added that: “It is a sort of lost look, like a befuddled, anxious, lost look, and in people who are pre-symptomatic [of Alzheimer’s], I think it is more sort of an anxious not exactly emptiness but a little bit of a ...”, she then stopped, apologized for being not scientific and concluded: “but people kind of have it.”

François, a neurologist in the French clinic, would agree with Beth. At the end of my fieldwork at the Alzheimer clinic, François told me: “In Alzheimer’s disease, the gaze gets a little bit lost, uncertain, looking for help... And you see it: they are lost, they turn to their family for help; you can feel it.” Like Beth, François doesn’t think this is science: “It’s more an intuition but today, really every diagnosis comes under evidence-based medicine, it means that we need a formal proof, the tests and the image. You cannot base a diagnosis upon an intuition: that’s a no no.” Yet, François thinks the lost look in the eyes of people with Alzheimer’s could one day be measured by scientific means: “It’s not pure science, but it could become: I wanted to do a project on that, with eye tracking, to see if we could objectify this gaze, it could become a new biomarker. But I have already so many projects to do.”

\textsuperscript{403} The word doppelgängers not only signifies twin, it also refers to the evil twin because these doubles “like to confuse their family members and confuse their human counterparts” (from http:// exploretheshadows.webs.com/creaturesmythslore.htm).
When Elise told me that neurodegenerative diseases gave a coloration to a personality, she then
hesitated: “Unless diseases develop on specific personalities...” she said, but decided against it: “I
don’t think so, I think it’s the disease”. Elise didn’t think the theory could go both ways. Daniel,
however, does believe in this symmetry: as he just told us, if people with FTD are quite humorous,
it is also true that a “number of people in the humor area got FTD”. For Daniel, not only these
diseases give a certain nuance to the personality, but also these diseases preferably affect certain
personalities.

Bruno’s certitude that Lea Strong and Cheryl Joe were “doppelgangers” doesn’t only lead to a
diagnosis, FTD, but also to what caused it: a gene. The idea that Mrs. Joe has FTD and that it is
genetically determined, justifies the “atypical” presentation of the disease: Mrs. Joe does not
present the classic range of symptoms of bv-FTD: she “cares”, she is “empathic”, her humor
appears strange (like her husband predicted) but “it is not quite that” (“that” is “punning”: the
typical humor of FTD bearers). Mrs. Joe is “atypical” just as Lea Strong is “atypical”; she also has
the same “dark ironic humor” as her twin. If Daniel tells us that his knowledge of the disease of
Mrs. Joe’s father, made him stray from clinical reasoning, that he should repress what is only
speculative and theoretical and should consider what is empirical and genuine (the symptoms),
this theory of twinship helps rationalize–with “atypia” conceived here as a symptom–what
Daniel’s “heart” believes to be true: that Mrs. Joe, like Lea Strong, carries a gene, that Mrs. Joe’s
father most likely carried, a gene that predisposes for FTD and ALS. With that diagnosis and its
etiology in hand, Daniel’s impression will now be confronted by two important tools: the
neuropsychological test and the MRI.

**Sequence 6: The test and the image.**

Emily, the neuropychologist who tested Mrs. Joe, surprised everyone when she announced that Mrs. Joe got 22 at the MMS –except Daniel who betted 23, everyone thought she would get a higher score. Emily also shared with us her “impression” about the patient, very different from Bruno’s: “Interpersonally... I thought she was odd, with a strong AD flavor... but it’s me, I could just have missed her”, she told us. After the detailed presentation of the scores Mrs. Joe obtained to the multiple tests, Emily summarized: “So the main thing I really saw was memory”.

Emily’s conclusion, combined with the motor problems remarked on by John during the physical exam (in particular cogwheeling, Hoffman’s sign and hyperreflexivity) allowed Leticia to argue confidently that Mrs. Joe has Lewy Body disease. Daniel listens, agrees that memory problems combined with motor problems could very likely point towards Lewy Body, but reasserts

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404 “Cogwheel” rigidity is described as such because of the ratchetlike interruptions of passive movement. Cogwheel rigidity is one of the criteria for the diagnosis of idiopathic Parkinson disease and is often an early sign of the disease.

405 Hoffman’s sign: The terminal phalanx of the middle finger is flicked downwards between the examiner’s finger and thumb: when the examiner lets go and in state of hyperreflexia (e.g. Hoffman is positive), the tips of the other fingers flex and thumb flexes and adducts. If it appears on one side only it can be an early sign of unilateral pyramidal tract disease.

406 Hyperreflexive: an excessive brisk reflex has a low threshold to elicit and the movement produced is sudden and short lived.
his belief that Mrs. Joe carries the gene, hence that she is sick with FTD: “because of the family history and also because of her behavior.” Emily, Leticia and I, press Daniel to explain: what behavior? Daniel answers, impatiently: “She has a very strange personality that your guys were really taken by... right? It was not subtle...it was...” and he firmly concludes: “Personality changes come from the structures in the brain that are involved with personality and Lewy Body doesn’t do that”.

Emily is blushing.

Emily (to Daniel). – How do we know her personality has changed? Nobody told me, the husband...

Daniel (coldly, to Emily). – Even if it hasn’t changed it doesn’t matter to me, it’s an unusual personality that’s enough for me.

Silence. Bruno proposes to look at the MRI. He opens his computer. The image of Mrs. Joe’s brain appears wide on the wall, in black, gray and white. Bruno turns the light out. The team looks at the MRI in a respectful silence. Daniel talks first.

Daniel. – Temporal... temporal and occipital...is where the atrophy is...

Silence.

Daniel. – Looks more Lewy Body than FTD. (A silence.) I still think it’s a gene carrier...

Enormous laughs in the dark.

Daniel (serious). – I mean: the gene carriers are a little funny... They sort of are not the classic FTD phenotype... Which is interesting.

No one laughs anymore.

Daniel (after thinking). – I don’t know what it is, I wish I could tell them what I think this is; I don’t think I can. Because of the family history of ALS, I want to see whether she carries that gene.

Kim signals that she collected the blood samples.

John (after a silence). – So on this sheet that they go home with.... what are we writing?

Daniel (pensive). – I don’t know, let’s talk about it, I’ll tell them I’m having a hard time with it.

The team at the Memory clinic, like the one in France, always reserves the image for the end of the discussion. Unlike in France, where the image of the brain (a scintigraphy\textsuperscript{407} as often as a

\textsuperscript{407} Scintigraphy is a form of diagnostic used in nuclear medicine: radioisotopes are injected by IV and its distribution, here in the brain, monitored with a gamma camera. Brain scintigraphy informs on the adequacy of blood flow to the cerebral cortex. The Memory clinic never asks patients to do this exam; they consider it not telling, useless.}
MRI) is usually held by the neurologist’s hand and examined like a radiography in the light of the day or of a neon, the American clinic owns a home cinema, the appearance of the MRI is awaited like the beginning of a movie and we all watch in an anxious silence the images of the brain following one another. This is a tense moment: les jeux sont faits; the team at the Memory clinic now expects the outcome.

Seeing Mrs. Joe’s brain, unlike hearing Emily’s test, powerfully challenged Daniel’s conviction that Mrs. Joe had FTD. He didn’t give up on his belief that she carried the gene though, but it’s palpable that he is more ready to admit Leticia’s diagnosis than he was before seeing the MRI. What will he tell to the couple now? What will Daniel think about Mrs. Joe now that he can finally meet her?

Bruno comes back with the couple; Mrs. and Mr. Joe come in.

**Sequence 7: The disclosure.**

\textit{Daniel stands up, introduces himself and shakes Mr. and Mrs. Joe’s hands.}

Daniel (charming). – Nice to meet you.

\textit{Daniel offers Mrs. Joe the seat next to his. Mr. Joe sits down on a chair next to his wife. Everybody is looking at them.}

Daniel (to Mrs. Joe). – So I would like you to tell me a little bit about your past...

The excitation of the team heated by the search for a diagnosis, fades slowly with the long quiet pauses and the sparse answers of Mrs. Joe. Her responses, disappointingly concise, force Daniel to constantly revive the conversation with a question that finally finds an answer in two words uttered without hesitation in a low and smooth tone. Mr. Joe is more talkative: he describes his wife as “very bright, reserved, not a big talker... She likes to make jokes, and always had!” What Mr. Joe finds really new is her habit of playing sudoku: she plays all day when one year before she had never played it in her life.

During the interview, Mrs. Joe makes some jokes. Mrs. Joe’s humor creates a diversion, as if she was trying to exit the path marked out by Daniel’s questions. Daniel ignores them or meets them with a dismayed “mmm”. During the physical exam that Daniel does roughly, testing only what he knows to be failing, he asks Mrs. Joe to tap her index against her thumb “real fast”, then to wave her hands as if she were playing marionettes. Mrs. Joe asks: “Got any music?” Without smiling, Daniel answers that yes, there is certainly music, but we are not playing right now. After these tests, Daniel turns his chair and faces the couple.

Daniel (formal, monotonous tone). – So I think the illness is unusual... The family history of ALS, worries me a little bit that this might be related to that... Only in the last month we’ve been able to figure out the genes that cause this: the gene we found recently is associated with frontotemporal dementia behavioral disorder and also ALS and sometimes Parkinsonism. But I wouldn’t
call the motor problems bad enough to be ALS. We talked about a Parkinson’s type of problem and I hope that’s what it is. We’ll get in touch with your local doctor to have him prescribe some Sinemet. I wouldn’t call the behavior bad enough to be frontotemporal dementia. The image doesn’t show a lot of frontal problem either, so it is not classical... although it does worry me. So I think the behavior change I heard is maybe a little bit of apathy and compulsive playing of Sudoku and these sorts of things...

Mr. Joe (vigorously). – Oh yeah! She was... she graduated at UCLA school of business, she worked in my office doing all the accounting and she quit that. (Bitterly.) And now she won’t even write a check at home!

Mrs. Joe (half-serious). – But I’ll write one right here!

No one laughs.

Daniel (to Mr. Joe). – So a lot of apathy.

Mr. Joe (doubtfully). – If that’s apathy...When you ask her, she’ll say that she is unsure of herself: that’s what you call apathy? The cognitive slowing is called apathy?

Daniel. – No. I think there is a little cognitive slowing but Boy! (To Mrs. Joe.) You are quick with your wit, that hasn’t slowed at all! But I think the lack of interest in things sounds like a change. (To Mr. Joe.) That, I call apathy. (To Mrs. Joe.) Do you agree?

Mrs. Joe. – With your definition? Yeah, I agree.

Daniel. – Do you think you are apathetic?

Mrs. Joe (to Daniel). – About some things... (Laughing lightly.) To the things that may be of interest to you...

Daniel (not laughing). – OK... Do you think you’ve become less interested in a broad range of things?

Mrs. Joe. – No.

Mr. Joe. – She has, I mean, she was very interested in politics, she was a member of all these political organizations... and now, not at all. She was a devout catholic, and now she doesn’t go to church.

Daniel (to Mrs. Joe). – Lost interest in church?

Mrs. Joe (discouraged). – Well... joining... the car...

Daniel. – If there were a car to drive you there, would you go?

Mrs. Joe. – Sure.

Mr. Joe (to his wife). – But you don’t like to see a lot of people...

Mrs. Joe (to her husband, piqued). – Says who?

Mr. Joe (to his wife). – You told me that!
Mrs. Joe (*calm*). – Well, I would like to go to the early service. (*To Daniel:* so yes I would go.

*Silence.*

Daniel (*moving on*). – We will probably get some information about the gene in six weeks time, would you like us to let you know if we find something that is relevant?

Kim (*interrupting, timidly*). – I am not sure our IRB, our ethics board, would allow the release of the results...

Daniel. – Yeah... that might be a problem. You know, it’s a very new discovery: we don’t quite understand it and some people might do very well with it. But as soon as this becomes an official test you will have a chance to do it.

Mr. Joe (*discontented*). – Well, you took the blood: it’s going be done.

Daniel (*nodding*). – Yeah. Any question for me?

Mrs. Joe. – I don’t think so.

Mr. Joe. – I have a couple.

Mrs. Joe (*teasing, to her husband*). – About the apathy?

Mr. Joe (*big laugh*). – Ah-Ah-ah! ... (*Serious again, to Daniel.*) So apathy is the cognitive slowing and the memory status is...?

Daniel. – We think it is poor.

Mr. Joe. – You think it’s *really* poor?

Daniel. – Not *really* poor but...

Mrs. Joe (*calmly, to her husband*). – Poor.

Daniel. – Fair to poor: not terrible. (*To Mrs. Joe*) I mean you remembered Bruno, lot of things you remember, it’s not like your memory is wiped out. But you’ve got some...

Mrs. Joe. – deficits.

Daniel. – Yeah, some deficits.

*A silence.*

Daniel. – I am going to give you my card, email me whenever you have a question.

Mr. Joe. – Thank you for your interest. So do you think she is a good candidate for your study? Where she went to school and where she is now... so rapidly...

Daniel. – I know: it’s pretty rapid isn’t it? (*To Mrs. Joe.*) What was the hardest test you went through?

Mrs. Joe (*after a silence*). – They were all hard.
Mr. Joe (anxious). – She gets her spinal tap tomorrow...

Daniel (comforting). – Often it’s not too hard: a little freezing and ... (To Mrs. Joe.) Easy for me to say, uh?

Mrs. Joe (to Daniel). – Well, it is reassuring to hear.

*They stand up, everyone says good-bye, they thank everyone. They leave the room.*

Daniel didn’t hide his doubts: he sincerely told the couple he was uncertain about the diagnosis. He finally decided to favor Leticia’s hypothesis: a “Parkinson’s type of problem”, although he didn’t mention the term Lewy Body. Daniel proposed to put this hypothesis to the test with the only treatment available in the therapeutics of neurodegenerative diseases and that will be effective only if Mrs. Joe actually has Parkinson’s disease. He also told the couple about the possibility that Mrs. Joe carried a gene that would predispose her to FTD-ALS. Daniel’s own enthusiasm about the recent discovery of the C9, seems to have rendered him myopic to the fact that it was indeed a new discovery, not “quite” understood. As Kim diplomatically slips into the conversation: this test is a matter of research. It is an experiment in which only the blood of the patients might be involved, not their soul. Mrs. Joe and her husband won’t know what the genetic test will reveal.

In front of Daniel, neither Mr. nor Mrs. Joe asked details about the diagnosis. What Mr. Joe was especially concerned about was his wife’s cognitive decline (the state of her memory, her “cognitive slowing” and her fall: “Where she is now.”). Mr. Joe didn’t know the meaning of the word apathy (it indeed belongs to the professional language) and seemed, so to speak, deaf to Daniel’s explanations: for Mr. Joe, the disaster does not lie in his wife’s lack of interest for certain things— that he otherwise acknowledges— but in her fall of status. To say the least, Mr. Joe experiences his wife’s trouble very differently than Daniel.

Unlike her husband, Mrs. Joe understands the meaning of the word apathy; she says she is in agreement with Daniel’s definition. She then ripostes with irony, to Daniel’s inquisitive questions. As we witnessed, there is a paradox in these encounters: a patient with a brain disease assumed to be “anosognosic” – i.e. assumed to be unconscious of what she called herself her “deficits”– is summoned to make her auto-analysis of aspects of her personality publicly. The patient always fails this test. Mrs. Joe refused Daniel’s analysis, she didn’t agree with the sentence: she is not apathetic; if only someone was willing to drive her to church she would go, especially early in the morning when there are less people. A silence followed and then Daniel moved on to a different subject. This abyssal indifference gives the radiography of the humiliating reality of this interview: the patient can say what she likes she will not be heard. James, the student in psychiatry, will tell me later, talking about this conference and about several others, that he felt the patient was considered as a bull to be sacrificed to the toreador, the neurologist, and – he didn’t say it but he could have– to us, spectators excited.

James’ impression that neurologist were “sadists” echoes Vincent’s preoccupation about the ethical dilemma raised by these conferences. James’ feeling that the conference is a “bull fight” he said, a combat that the patient will always loose and Vincent’s remark about the *mise en scène* at stake in the conference that reminded him of a “circus” or a “TV show”, are tributary to what just happened to Mrs. Joe: the patient, somehow, enacts its disease. The case-conference perceived as a TV show, as a circus, as a bullfight, could also remind us of a painting: *Une Leçon*
clinique à la Salpêtrière by André Brouillet (1887). The scene shows Jean-Marie Charcot giving a lesson on hysteria to an audience formed exclusively of men (neurologists but also the lay public) in front of which Charcot demonstrates his theories on a woman, the patient, half-bare and held unconscious in the arms of the neurologist Babinski. In these medico-artistic sets, the “metteur en scène”, the director, is the clinician (Charcot, Daniel, Vincent more rarely) giving to the public a feel, a taste or a flavor of the disease. The patient is an actor despite him or her self, who enacts the disease. And the fellow, the medical student, the rest of the team and I the anthropologist, form the public, a public on a quest for a truth. There are indeed, some similarities between the atmosphere of the TV show at the Memory Clinic and Charcot’s clinic that Bertrand Marquer portrays as fantastical and positivist, stretched between a fascination for the demented and the desire to understand.

Sequence 8: The diagnosis.

John left with the couple, the rest of us stayed around the table, in silence. Leticia speaks first.

Leticia (proudly). – Nothing frontotemporal dementia in my opinion.

Daniel. – Oh really?

Leticia (shaken). – No! Why? Do you think she is frontotemporal? I think she is, you know... she performed well with you, she is very appropriate, after one year of history if we think she has frontotemporal dementia: she doesn’t have any symptoms! After one year: she is critic...

Several persons talk at the same time.

Daniel (amused). – Even the image isn’t good for frontotemporal dementia. (To Milton.) What do you think it is?

Milton (impassive). – I still think the same thing.

Daniel (resolute). – I think it is frontotemporal dementia with ALS.

Leo (smiling). – I am changing my position: I think the interview...

Daniel (to Leo). –That too. Yeah...

Milton and Leo nod.

Leticia (disturbed). – But why doctor Daniel? Why? I never saw an ALS that slow: this is too strange.

Babinski was Charcot’s assistant, he found the sign that still bears his name, while feeling the legs, the feet and the toes of the paralyzed of the Salpetrière. Babinski sign allows differentiating between a disease of the body and a disease of the soul. By scratching the sole of the foot, the big toe flexes if the paralysis is due to hysteria and spreads out if the patient is really paraplegic.

Daniel (after a silence). – Here is why I think she is FTD: mostly cause of her father, you know you have to start to get into statistics: the disease happens to six persons out of a hundred thousand of people. So that part really bothers me. Also, I think she has a personality very ... ooodd to the point of pathological personality. She is pathologically cold, she doesn’t care about this disease, she is rather indifferent, she is happy, she sits around playing sudoku, she doesn’t ask about the effect on the children: all the emotions, all of the anterior insula stuff that we all do, HE [the husband] does it for her. She isn’t concerned about anything, she isn’t concerned about her LP [lumbar puncture], there is nothing about this you know: “You might have ALS”, the slightest... if you put her in the laboratory to measure her GSR [galvanic skin response] when I told her she might get this disease that killed her father, I don’t think she'll got a bullet: I think she is really emotio-nally blunt. And then, the punning, the joking... (Annoyed.) I mean she was more interested in thinking of making a joke than anything I said: that’s what her whole thought process was during all this. So I went from like pretty sure she was FTD-ALS on its way, to really sure. That’s me... (To Leticia.) How many Lewy Body patients have you seen that look like that?

Laughs.

Leticia (fiery, to Daniel). – How can you explain the memory deficit and the atrophy that we see on the MRI? She has temporal posterior atrophy on the left. How can you explain this if we think it is frontotemporal dementia?

Daniel (energetic). – This is the gene, this is the gene! It gets a lot of posterior. (To Bruno.) Right? That’s one of the conclusions in your paper? (Bruno nods.) They get a lot of posterior atrophy.

Me (to Daniel). – But then... what is the essence of FTD?

Daniel (smiling). – I know... I know... (Confident.) She is FTD.

Laughs.

Daniel. – The essence is there: the odd personality with all the jokes.

Me. – But, would you consider that she is not comfortable being with doctors and so that she jokes to ease her malaise? She has this response to you and to John too.

John. – Yeah, it was exactly the same.

Daniel. – But, you know me...I never...

Me (defeated). – Yes, I know...

Daniel. – I just look at the phenomenology, I just don’t listen to any explanations, cause I think they are more likely to mislead you. Here is somebody who got a profound denial of illness, her husband says that she sits like a lump on a log all day long in one of the most beautiful cities in the world and like play sudoku all day in the bedroom, and she says: “Well... you know...”
(Confidant.) This is not a full-blown FTD yet but probably will be in a year. And I don’t think it’s a full-blown ALS yet but it will be. That’s the beauty of these patients: you get to see them before the full-blown syndrome has emerged and then you can piece back what was salient and what wasn’t. I bet in six months she will be FTD and everyone will agree, and she will be ALS, that’s my guess.

John (pensive). – I was struck by the anxiety that sort of resolved by itself. I guess if she is a gene carrier... because she got very anxious, that was her first symptom...

Daniel (casual). – Oh, yeah...? I don’t see it now. She is flat like a pancake. (Joyfully.) OK! Good case! Thanks.

Everyone disperses.

We leave this conference with the desire to know if Daniel was right: we will have to wait for tomorrow. Our desire will be granted by the same dissatisfaction as with the previous conference with Dr. Martin. The search for a meaning and the affirmation that there is a meaning somewhere are, Clément Rosset writes: “underpinned by a knowledge that is always to become”410. This postponing authorizes Daniel’s messianism: in six months everything will be clearer for you (“she will be FTD”), trust me (“I bet”), you will see (“everyone will agree”).

In this final scene though, Leticia and I have learned that a diagnosis in neurology can be in contradiction with what the image of the brain shows. Leticia wondered about the following incongruity: how is it possible to make a diagnosis of frontotemporal dementia, a disease that hits principally the frontal and the anterior part of the temporal lobes, on a patient whose MRI does not show such geographical atrophy and –quite the opposite– whose posterior brain mainly, is atrophic? Daniel’s answer –with the gene everything is possible– flouts the presupposition that frames his discipline: the causal relation between, on the one hand the cerebral anatomy, and on the other hand the cognitive capacities or the behaviors. Yet, this is the way that brains destroyed by dementia, are meant to open this field of knowledge of madness and of what makes humans human. Now, if this paradigm is not in current use anymore, how is FTD still a neurological disease? How does the neurologist make a diagnosis of FTD? Daniel’s answer to my question (“what is the essence of FTD then?”) is a –quite imprecise– redefinition of the disease: “an odd personality and a taste for jokes”; the less we can say is that his definition is based on what he perceived from the moment he just spent with Mrs. Joe.

Daniel’s final diagnosis is not based on the scores on the neuropsychological test that he ignored, neither on the image of his patient’s brain which ended up being turned around (with the gene everything is possible: it is even possible to observe an inverted image of the disease). His diagnosis, Daniel told us, is primarily founded on statistics: ALS is a rare disease that Mrs. Joe’s father had, there must be a gene in the family that could explain that Mrs. Joe’s father had ALS, Mrs. Joe’s sister has Parkinson’s and that Mrs. Joe herself has now (and can only have) FTD. The father and his two daughters would show the three possible clinical expressions of the C9 gene. Statistics and genetics are surely the bedrocks of Daniel’s diagnosis. It is neither statistics nor

410 Clément Rosset, p 64.
genetics, however, which helped the chief of the Memory clinic to sketch out a portrait of his patient and that helped him express several judgments on Mrs. Joe.

If Daniel’s diagnosis is not only based on statistics, it is also based on... what? It seems to me that it was based on an impression, a feeling, alternatively on an affect. Daniel seemed impressed by the “lack of emotions” in his patient when he judged her “oooodd”. Daniel seemed affected by her “indifference” to judge her “pathologically cold”. Daniel seemed to feel that Mrs. Joe “cared” more about “making jokes”, than about the disclosure that she could have ALS. What characterizes these feelings? They can be fully shared: Milton, Bruno and Daniel shared the same one since the beginning, and Leo who first didn’t, in the end sided with them convinced by something (but he didn’t say what) during Mrs. Joe’s interview. Also, they can be discussed. Leticia didn’t have the same feelings towards Mrs. Joe as Daniel but she discussed hers (“she is very appropriate”, “she is critic”) and Daniel his (“she is emotionally blunt”, “she is pathologically cold”, “she is odd”, “she is flat” etc.). Yet, discussions about Daniel’s impressions have a limit: to my attempt to argue that Mrs. Joe’s humor was not a salient symptom of FTD, Daniel retorted with his “phenomenology”. His rough perception of the real versus my explanations about the real: double, triple... infinite; always missing the phenomenon itself. I speak too much, when the presence of the phenomenon seems to be almost silently felt. I’ll come back to Daniel’s phenomenology in section eight.

The story of Mrs. Joe’s diagnosis, made clear I think, that impressions, feelings, affects, sentiments, whatever we want to call them, participated in the making of her diagnosis: it was first through the impression that Mr. Joe wasn’t a good informant for his wife, it was then through Bruno’s impression that Mrs. Joe had a patient twin. It was in the end through Daniel’s portrayal of his patient with terms that were not neurological neither psychiatric but pertained to our common language, one we might hear sometimes at the occasion of a conversation in the bus or the metro, one that we use to express and share our negative impressions about people that we’ve just met. The long development of Mrs. Joe’s passage at the Memory clinic was I think necessary to understand how the realm of feelings participates in the diagnosis, as well as the bet, the desire to know, a fascination with a knowledge that is always postponed, the image and the test, the aura of the chief of the Memory clinic, a patient who responds and whose voice is not heard, the suffering inherent to a medical situation as well as the conflicts that sometimes come with it.

Now, and until the end of this chapter, I am going to focus more directly on this “feeling”, on what it is and what it produces. I finally decided to keep the term “feeling” instead of affect, impression or sentiment, because it is the one of the four terms that I realized was actually in use: in the 1920’s Eugène Minkowski wrote about the “diagnostique par sentiment” and Ludwig Binswanger about the “Gefühlsdiagnose” in the field of psychiatry, both best translated in English by “diagnosis by feeling”.

Also, Jack, a fellow at the Memory clinic, once explained to me that the diagnosis Daniel was making of the daughter of a patient was a “diagnosis by feeling” (I tell this story in section 7). I then asked Jack about this term that he had whispered in my ear: where did he hear about “diagnosis by feeling”? Is it something doctors speak about in closed circles? Jack answered that he was himself told about “diagnosis by feeling” by one of his professor when he was still in medical school. It was right after Jack had examined and interviewed a patient who “annoyed” him by asking him “many questions” as if she wanted “to test” him and his medical aptitudes;
Jack went to see his professor and told him how much he had been irritated by this patient. Jack’s professor advised him to make use of the “feelings” he had had against her (and other patients): they could actually be of great help to make a diagnosis. And he sent Jack to the library to look for a textbook called *Outpatient psychiatry*, in which he would find a chapter written by Edward Messner, MD: “Autognosis: diagnosis by the use of the self”—of great interest for Jack’s problem. Autognosis is a reflection on a form of countertransference. Messner advises the clinician about his or her “subjective responses” to the patients: especially useful for “the diagnosis”, for “therapeutic impasses” (in psychiatry), as well as for the clinician’s “personal growth”. These subjective responses can be common, e.g. many clinicians can be affected the same way by a disease: “For instance, many clinicians feel in the presence of borderline patients as if they are walking on eggs and that they may be verbally attacked as soon as they attempt to be helpful”. These responses can be idiosyncratic too (for example a “sense of comfort and relaxation” in front of borderline patients). “Common” or “idiosyncratic” both kinds of responses matter for the diagnosis: both are reliable, concludes the psychiatrist. In the end Edward Messner, despite the educational orientation of the textbook, doesn’t offer a chart to link subjective responses and diseases, but he gives at least a list of the “common subjective responses” so the clinician is on his guard when he feels: “the desire to rescue the patient from destructive family members, sexual excitement, fear of the patients verbal violence, annoyance, boredom, humiliation, the desire to humiliate, the sense of being manipulated, and the sense of feeling emotionally drained or exhausted.” Half a century before Messner, Minkowski and Binswanger had written about the idea of a diagnosis by feeling, with an orientation that was less practical (even if Messner in the end doesn’t offer many keys to his students-lectors): with an epistemological endeavor they situate the place of these “feelings” in the making of knowledge.

5. Eugène Minkowski: “Feeling is a tool for our knowledge”

This sentence concludes the first chapter of “La schizophrénie” a book that Eugène Minkowski, a French psychiatrist, published in 1927. Conscious that this chapter will not spare him “harsh critics”, Minkowski still develops a quite provocative take on the diagnosis of schizophrenia. In front of a patient, “How can we establish the schizoid character of his psychology?” asks the French psychiatrist. And he answers: “To perceive this notion we have an infallible instrument in us. It is our own affectivity, our own personality”.

In a similar vein as Vincent, the French psychiatrist distinguishes two ways for diagnosing schizophrenia. The first dissects and leads in a diagnosis by “reason”. However in front of a patient, the point of view of the impassive spectator who enumerates and classifies psychotic symptoms to reach a “so-called scientific diagnosis”, this point of view is not enough. In front of a patient the psychiatrist needs to “enter into the personality as a whole and to perceive it in a single act: by feeling”.

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413 Ibid. p 93.
414 Ibid. p 98.
The idea of a “diagnosis by feeling” was first (in 1924) put forward by Minkowski’s friend, the Swiss psychiatrist Ludwig Binswanger415. Again, faced with the schizophrenic patient, Binswanger criticizes the method akin to natural sciences: a dissection of the symptoms that are then reunited to form the clinical picture of schizophrenia. This way of doing is often not negotiable because most of the times the “striking symptoms” are not quite obvious. Nevertheless, we might get “the certitude that we are in front of schizophrenia”. How? “We will talk of a Gefühlsdiagnose416”, writes Binswanger.

What Binswanger calls diagnosis by feeling cannot be made from “little facts”: a detail, a face expression for instance. On the contrary, the Gefühlsdiagnose of schizophrenia is characterized by a perception of the “personality in its wholeness417”. This is the first point. It will become manifest to the psychiatrist, by a “sudden certainty”418 that this person, who he knows well, is actually schizophrenic. This certainty can be anchored in his perception of a “lack of affective contact419” or in the “distinctive expression of her gaze420”. This mere “affective barrier” will “strike” Binswanger each time he meets that person in a way that he is gripped by “a movement of interior retreat421”. Very importantly, this movement of retreat that affects Binswanger is not analogous to antipathy, he says. The diagnosis by feeling, if it does not call for a reasoning is nevertheless the diagnosis of a psychiatrist, moreover an experienced one (Binswanger said that it requires a lengthy apprenticeship), thus it should not be confused with a banal feeling of sympathy or antipathy that anyone can experience towards anyone. The second point is that this feeling is one that can be cultivated only by a professional; a powerful screen against love or hate. Binswanger makes it clear that it is not a “gratuitous supposition, without any appreciable reason, that can sometimes be true422”, it is not founded on nothing and it is not the mere result of chance. It is founded on experience. On that ground, Minkowski comments that the term “feeling” brings out too much the idea of a “subjectivity” of this practice. Instead of the original appellation of Binswanger, Minkowski likes better the formulation: “diagnosis by penetration423”.

Binswanger and Minkowski, like Vincent, opposed two ways of making a diagnosis. However, with the two psychiatrists it is not only two epistemological attitudes that are differentiated but also two mediations of the medical knowledge. Binswanger and Minkowski described a first epistemological attitude mediated by “reason”. More specifically it is a sort of analytical reasoning that “measures” the presence of elements, the symptoms, classify and organize them to form the clinical picture of schizophrenia. In the same way, as the objectification of dementia is mediated today by different instruments of measurement: brain imaging techniques, molecular biology techniques, neuropsychological tests, genetic testing, etc.

415 In L. Binswanger, “Welche Aufgaben ergeben sich für die Psychiatrie aus den Fortschritten der neueren Psychologie?” Zeits.f.d.ges.Neur.u.Psych., t.XCI, 1924. This article is translated by Minkowski in La schizophrénie.
416 Minkowski, p 96.
417 Ibid.
418 Ibid.
419 Ibid. p 97.
420 Ibid. Here the word “gaze” does not assume that vision is pathological; as Binswanger specifies an examination of the fundus of the eye will not reveal anything.
421 Ibid.
422 Ibid. p 96.
423 Ibid. p 95.
To this principle of atomization characteristic of the scientific method, Binswanger opposes an apprehension of the “personality in its wholeness” that allows the objectification of schizophrenia with a “sudden certainty”, not with a step-by-step method. Vincent would say: “integration of the whole” for an “immediate” recognition of the disease. Vincent however, did not tell us what kind of mediation this second way of doing a diagnosis would take. Binswanger and Minkowski suggest that the “feeling” of the psychiatrist towards the patient mediates this second way of reaching knowledge. Feeling is a form of “instrument” for Minkowski: the goal is still to “objectify” a trouble; calling it schizophrenia is already an objectification. Nevertheless, this feeling is not an instrument of “measurement” because it is not bounded to an epistemological method that dissects. This feeling is “in” the clinician, in his body, unlike medical reasoning that comes from an exterior, taught in medical school. The body of the clinician is the medium for reaching knowledge. Knowledge or “truth” can be reached, as Minkowski concludes quoting Blaise Pascal, with the aid of both “reason” and the “heart”.

In the following scenes that all happened at the Memory clinic, we will see how the team jokes about this feeling (section 6), how it actually develops in the diagnosis (section 7), what method and human quality forge it (section 8) and finally how it contributes to the making of knowledge (section 9). Since this “feeling” does not “measure” but grasps, since it is not machinery, in my attempt to figure out what it is, I will not consider it within my range to draw explicit rules that would characterize this mode of knowledge.

6. The Dog Sign.

It’s Wednesday at the Memory clinic. Usually on Wednesday’s afternoons, Daniel diagnoses patients enrolled in the research program on FTD. This afternoon’s case is a man. He came to the clinic with his wife and his dog; it’s a little black dog who never leaves him. It’s not any dog, it’s a service dog who preciously assists the man when he goes for a walk in the countryside: if the man gets, or if he feels he is getting lost, he just needs to ask the dog “Mummy?” and the dog takes him back to the house where the wife is usually waiting for them.

This man had had contradictory diagnoses: AD, FTD and amnestic MCI, none of which were made at the Memory clinic. The wife heard about this place of worldwide expertise when she was in a conference on FTD in Indianapolis. She was interested to know more about FTD after her husband got diagnosed with this disease; in Indianapolis she heard Daniel and Oliver talking about their research program at the Memory clinic: “They said they were looking for 250 FTD’s brains for the program: that’s why we ended up here. And also to learn more...” she told me.

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424 The goal is not to give a “phenomenological description” of the troubles freed from psychiatric theories in order to listen to a “singular expression”, as Binswanger will write in Basic Forms and the Realization of Human “Being-in-the-World”.

425 “Nous connaissons la vérité non seulement par la raison, mais encore par le coeur”. Blaise Pascal in Pensées, quoted by Minkowski, p 99.
The case-conference will start as soon as Daniel will enter the room. We (Michael, Olivia, Beth, some medical students and me) are seated around the table, waiting for him. He comes in, he closes the door: “What have we got?” he asks. Sometimes, Daniel sounds out his audience before the fellow (this afternoon it’s Michael) begins the formal presentation of the case. Michael tells Daniel about the contradictory diagnoses given to the man: AD, FTD and MCI. Olivia adds with an intense look in her blue eyes: “He had this posterior feel, he is very sweet”. Olivia didn’t say “memory deficit”, “language trouble”, “attention problem”, “impairment in visuospatial”; the neuropsychologist said “a feel”: sweet and posterior. Usually, when one is “sweet” one is assumed to have Alzheimer’s. “Posterior” characterizes the cerebral geography of Alzheimer’s. Michael also informs Daniel of the dog that might be there, in the conference room, with the patient when Daniel will deliver the diagnosis. Daniel doesn’t mind having a dog around. Daniel is smiling silently as if he was thinking about something amusing. Then, Daniel tells us about a woman, one of his patients: he diagnosed her with FTD and she had a dog.

Daniel. – The dog picked up on her strangeness and attacked her. I don’t think dogs would do that with AD patients. I would think that dogs like AD [patients] better than FTD [patients], even if you take away the aggression in FTD ... (Daniel looks at me:) Laurence and I should train dogs to diagnose AD ... (Laughing.) We would do better than the AD clinics in the US!

Big laughs.

Michael and Olivia suggest that we find a French word to give a name to that “dog sign”; French words always look better than English words they say, cracking up.

Michael (to me). – How do you say dog in French?

Me. – Chien.


Everyone is delighted.

Two months or so after this, the team is discussing about another patient, also a man. Michael is presenting the case to Dr. Blake, the attending doctor. Olivia is there, Beth too, Judith also. Judith is a genetic counselor, and the right-hand man of Dr. Daniel.

This patient was, too, diagnosed previously by another team in the country: the family said they were told he had FTD, but it is confusing because the patient was given Aricept by these doctors; a medication usually reserved for patients with AD. Michael presents the case, at one point Blake asks Michael if the patient is empathic, Michael answers without any hesitation that he is “adorable to others”. Yet, some facts are in favor of FTD: his brother has Attention deficit disorder (ADD) and his son was autistic and committed suicide.
Blake. – So there is some suicide... (Blake looks at the medical students and teaches:) There is a very high comorbid psychiatric family for patients with FTD.

Judith. – His mother at 92 was very obsessed with certain things. And she had two brothers who died. His grandfather [the mother’s father] died at 31 of “broken heart” (Judith mimic the inverted comas) because he had two sons who died...

Blake (to the medical students). – So, you see...

Judith. – Yeah you can definitely Snif something.

Michael (looking at me, smiling). – And he has a dog... That’s a sign Daniel is looking for (Michael points at me, with a smile of complicity:) it’s a sign of FTD.

Blake frowns, uncertain about what’s going on.

Olivia (smiling, to Blake). – It’s kind of a running joke...

Michael (to Olivia). – I don’t know... (Serious.) Dogs can tell you, dogs like people when they are nice with them.

The dog of the “dog sign” is not exactly Vincent’s dog: the bloodhound tracking the game, his nose digging the dead leaves, his sharp flair awakens in order to reach the game hiding in the woods. The dog of the “dog sign” is man’s best friend. The smell of Vincent’s dog perceived the game. The smell of the “dog sign” perceives love. Love and sweetness characterizes AD, but not FTD (strangeness characterizes FTD). AD patients are loving, FTD patients are strange; dog is man’s best friend, dog’s friends are man’s friends. Let’s have Daniel and I train dogs to better diagnose AD, and thus to better diagnose FTD! This running joke was made in my honor: it was four months after the beginning of my fieldwork at the Memory clinic, at that point I had no idea that I would one day write something about the “diagnosis by feeling”, I never heard or felt anything about it. I was wondering though, as I had told Daniel, how he diagnosed these diseases in situations where the uncertainty was high –like in this particular case: when several diagnoses had been given. The “dog sign” was a joke (of course) and maybe also a manner to invite me, the (French) laywoman, to participate in the making of the diagnosis. Nevertheless, Michael commented to Dr Blake that it is also true that dogs are sensitive to love, like people are; an idea that would have been appealing to Jeremy Bentham. Mostly known for his invention of the panopticon at the end of the XVIII century, Bentham has also proposed in Introduction to the Principles of Morals and Legislation a utilitarian moral and legal code. He noticed at the end of this book that non-human beings’ sensitivity had been too much neglected by jurists, he thus questioned the current classification of beings based on reason: what if one day we were to consider beings not on the ground of reason but on the ground of sensitivity?

What else is it that should trace the inseparable line? Is it the faculty of reason, or, perhaps, the faculty of discourse? But a full-grown horse or dog is beyond comparison a more rational, as well as a more conversable animal, than an infant of a day, or a week, or even a month, old. But suppose the case were

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426 One is maybe interested to know what were the diagnoses of the team on these two patients: the first man ended up being thought of as “malingering”, and the second one as “psychiatric-MCI”.

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otherwise, what would it avail? the question is not, Can they reason? nor, Can they talk? but, Can they suffer?427

7. “Diagnosis by Feeling.”

The first time I heard about “diagnosis by feeling” it was in the United States at the Memory Clinic. It was an afternoon in July: Jack, the fellow, murmured to me “diagnosis by feeling”, as a comment about the diagnosis his mentor, Dr. Daniel, just made of the daughter of a patient.

The patient and his daughter left the room. The team stays to discuss, as it is often the case, its last impressions. Daniel is sitting at the table with Jack the fellow, Beth the nurse and Emily the neuropsychologist. The room is small; the residents, the guests and I, are squeezed sitting on chairs and filing cabinets arranged around the core group. The patient who just left, is an 80 year old Ukrainian man who immigrated to America twenty years ago. The course of his disease is unexpectedly long, eighteen years from the apparition of the first symptoms. Because of this, Daniel suspects a genetic etiology. He is seen at the Memory clinic for the last ten years and was diagnosed with frontotemporal dementia. This diagnosis surprises Emily, who is seeing today this patient for the first time. She tells Beth: “He is so sweet, so cute...” Beth agrees and expresses her surprise as well: actually she “thought he had AD”. But “it” is FTD, or so it seems. Their exchange doesn’t surprise me: I am at the Memory clinic now since long enough; I have learned that AD and FTD are articulated by the team in a set of mutually constitutive oppositions. FTD patients are described as emotionally blunt, selfish and lacking empathy whereas AD patients are seen as overly sensitive, emotional and “sweet”. What will surprise me is Daniel’s remark about the daughter of this patient.

Daniel (after a silence). – I am worried about her [the daughter].

Emily (surprised). – Emotionally or degeneratively?

What Emily is asking is: [Whether Dr. Daniel is] worried about her, as a “caregiver” or as a potential “gene carrier”? As a daughter involved in an affective relationship with her father –whose relationship could possibly be damaged by his sickness– or as a daughter in a genetic relation to her father and who could have inherited of his probable (according to Daniel) deleterious gene?

Daniel. – Degeneratively... A funny nuance affect...

It is thus the brain matter that is here in question, not the relationship with her father.

Beth. – She is worried she is getting this.

Me (to Daniel). – What bothers you about her?

Daniel. – A bit of a stare...

Jack (softly to me). – Diagnosis by feeling...

Beth (to Daniel). – All the time I knew her she has been like that.

Daniel (hesitant). – I don’t know, I wouldn’t bet a lot of money on it...

The conversation continues with a question from a visitor about the correlation between the long course of the disease and a possible genetic cause. I am thinking about what Jack just whispered to me; I am wondering if the term “diagnosis by feeling” is common. Then, Daniel comes back to his impression about the daughter.

Daniel. – She had a funny look: she is caregiving but she has no emotion... It’s unsettling isn’t it? She makes you feel... She is different: compared to this other caregiver who writes books that are so emotional, it is almost unbearable, and who ended up on ABC news!! This one has no emotion.

A resident in neurology. – She is a little bit apathetic to the condition of her father.

Emily (risks an explanation). – It’s cultural, the slavics...

A resident in psychiatry nods to Emily’s remark.

A guest (refuting the culturalist argument). – Did she say she was in panic? She kind of said it but with no emotion...

Daniel. – Yes, she is flat, a little staring, a little bit odd.

I have to say that at that point of the conversation, I am totally unaware of what Daniel, the resident and the guest are finding odd in this woman’s attitude. From what I witnessed during her father’s consultation, she seemed to me calm, articulate and maybe somehow weary of being there. What I perceive as a control over some stressful circumstances (the consultation), Daniel perceives it as odd and unusual. So, I ask if I understood well that she was actually working as a medical provider of some sort. Beth tells me that, yes, she is a respiratory specialist for patients with Lou Gehrig’s disease. I am thinking that someone whose work addresses the suffering of patients affected by a neurological disease that evolves towards muscular paralysis (ending in the paralysis of respiratory muscles causing first dyspnea and then death by asphyxia), must be someone who has a close relation to suffering as well as to the whole medical stage.

Me (to Daniel). – Don’t you think this is part of it for someone who is used to take care of very sick people?

Daniel (to me). – You know me, I’m dumb, I am a phenomenologist, you can always explain something by a story... but phenomenology is much more
powerful than the explanation, in this sense psychoanalysis took us much more away from the explanation. Phenomenology is all we have: the way people behave in front of us; I don’t care if they’ve lost their parents in an accident when there were four, I don’t care if they’ve lost their two arms.

8. “I’m dumb, I am a phenomenologist.”

We remember that already when I tried to give a reason for Mrs. Joe displaying humor during the time Daniel examined her, Daniel didn’t take my observation into account and told me: “You know me...” He then resorted to phenomenology, like he just did to justify his feeling that Mrs. Joe’s humor was “odd”. Again, it indeed seems to me fair to call what Daniel expressed, like Jack did, “a feeling”: a feeling that there is a meaning to discover, a feeling that Daniel doesn’t bother otherwise to specify: she is “odd”, she has “a bit of a stare”, that’s it. This time, Daniel also told me that he was “dumb”. And at several other occasions, when I will attempt a discussion with him about his clinical feelings, Daniel will always give me this same curious answer “You know me... I am dumb”.

My first impression was that Daniel was ironical. Daniel does not actually think of himself as dumb, understood here as idiot, or he would not subsequently favor his method, phenomenology, over any other method, since this pretended stupidity should question his own knowledge. By describing himself as dumb he appears to be ironically self-deprecating, whose effect is a detachment from the conversation that I am trying to engage with him. About ironic detachment, Alasdair MacIntyre writes that it “involves a withdrawal from our common language, our shared judgments and thereby from the social relationships which presuppose the use of that language.”. However, after a time, I thought that this repetitive and particular disengagement from the “common language” that Daniel and I seemed to share in other numerous circumstances, might point to something else than to sidestepping.

Jeanne Favret-Saada at the beginning of her inquiry into witchcraft in Mayenne, realized that some of her questions were answered by silence, an answer like irony, which refuses the commitment to communication. In this silence she sees a sanction of the ethnographer, and learns from her faux pas the difficult practice of tact: precisely to avoid those questions. I did not take literally the lesson of Jeanne Favret-Saada; I persisted in asking Daniel to justify his epiphanies. I carried on trying to obtain explanations because I was, at that stage of my fieldwork, already “participating” in the game, as Kant writes, and not a mere spectator of it anymore: I was able to find that an action, as I take Daniel’s judgment to be, was poor or skilful, and to look for the reason for such a judgment. However, instead of maintaining that Daniel was ironically avoiding my questions, I realized that with the recurrent choice of the adjective “dumb” he was saying that he could not possibly give me another answer, that my questioning was leaving him genuinely speechless. We, anthropologists, ask for explanations for these actions that we find

obscure or unusual, but that people know how to do these actions does not entail that they can explain how they do them. Indeed, Daniel never accounted better for his feelings and judgments than with the resort to “phenomenology” and “dumbness”.

But, is it actually possible to better characterize this “feeling” and the phenomenological method that is associated with it? Binswanger wrote that feeling is an “instrument” but it is “in” the clinician, in his body, unlike medical reasoning that comes from an exterior, taught in medical school. The body of the clinician is here the medium for reaching knowledge. Nevertheless, if this feeling is an instrument it is not of “measurement” because it does not rely on an epistemological method that dissects. That I did not get more explanations for Daniel’s initial hesitant judgment (he started to back off) and then for Daniel’s certitude that the daughter of this patient had the same disease as her father, is really not surprising: if the “diagnosis by feeling” does not rely on an analytical method, it is probably refractory to the analysis. Or, as Christopher Lawrence would put it, it is “still incommunicable”. What we can still say is that, like most French and American neurologists, Daniel is not seduced by psychoanalysis. Daniel sees psychoanalysis as a method that searches for “explanations” backed up by prefabricated theories. By contrast phenomenology in Daniel’s view, appears as a grasp of what is there, in front of the neurologist. The feeling here involved seemed to be a negative one: it springs from a feeling of oddness, of strangeness, of alienation. The phenomenology that Daniel puts forward has in common with Binswanger’s, an absence of reference to taught concepts. It grasps an implicit. The implicit is “preconceptual” writes Gilles Deleuze; there is no need to define it with other concepts. And Deleuze articulates this term with a “conceptual persona” that he calls “the idiot”. “The idiot” analyzes Deleuze, is embodied by Descartes: when the philosopher says: “I think, therefore I am”, he uses presuppositions that belong to everyone... Everyone knows, more or less, what is “I” and “think” as opposed to the definitions required to clarify what is meant by the concepts “animal” and “reasonable”. Let’s take that for granted. Descartes the idiot, outlines a persona that is according to Deleuze, the antinomy of the professor, the scholar, the technician; the idiot is the layman and the private thinker. When Daniel says that he is dumb, it might in the end be neither an ironic nor a genuine excuse to my questioning, but the necessary quality of the good clinician in behavioral neurology, the anti-scholar, the anti-psychoanalyst, who thinks “by himself, by the natural light”. A “natural light” that I would have seen too, if only I were dumb enough. Which means: if only I could have perceived this reality in its roughness, in its uniqueness; actually the original definition of the word “idiot”, writes Clément Rosset in his Traité de l’idiotie. Idiôtès signified first “particular”, “unique”. Only afterwards the term acquired its actual definition: without intelligence. Thus according to this original definition, idiot is opposed to what is double, to a reality that appears in a mirror, writes Rosset: “Which replaces the

432 There is a crucial difference between Binswanger and Daniel. Daniel’s phenomenology takes the phenomenon back to a clinical picture (AD or FTD) and to a physico-chemical cerebral state (the overload of specific proteins in the brain). When what interests Binswanger is the phenomenon and only the phenomenon.
434 Deleuze. Ibid.
presence of things by their appearance in images\textsuperscript{436}. My contact with reality is not dumb because I perceive its image; Daniel perceives its roughness.

If we now begin to see that this clinical feel is akin to a method called phenomenology and depends on a clinician displaying dumbness, we might still be wondering how this feeling and these notions that accompany it, participate in the building of knowledge. What are the effects of the affects and of this dumb disposition upon the progress of diagnosing practices? The following story will tell us more.

9. “I’ve never been wrong.”

The man coming that day to the Memory Clinic is 48 years old, he has no symptoms of dementia whatsoever. He looks young with his wisps of hair falling on his forehead, he has a relaxed and casual attitude. He came to the clinic with his wife. His father was seen at the clinic before his death two years ago; he died of FTD and a gene was clearly identified as the cause of the disease. His father, remembers Daniel, was very abusive towards his children; he beat his kids... This evokes another family with a FTD gene for the chief of the Memory clinic “in which the phenomenon was incredible cruelty to children”. The man, who was an abused son, is here today because he wants to know if he carries the same gene as his father, or not. For the moment his genetic status is unknown, to him and to the team. I asked about the penetrance\textsuperscript{437} of the gene: Daniel and Judith, the genetic counselor, answered me in chorus, without hesitation: “100%”. It means that if the man has the copy (in this case one copy is sufficient) of the deleterious gene he will be sick\textsuperscript{438}.

We remember that already in chapter three, Daniel was wondering about the prodromes of Alzheimer’s disease for Lucia, the sister, and José, the brother, both still asymptomatic. Similarly here, what Daniel will be testing during the case-conference of this man is primarily the sharpness of his own clinical intuition in detecting FTD prodromes, these very subtle behavioral changes that announce a disease already progressing in the brain. Daniel’s perceptiveness for the presence of the mutated gene in this man’s DNA, will then be confirmed, or infirmed, by the result of the genetic test. These case-conferences are Daniel’s favorite: because the patient is asymptomatic the diagnosis is the most uncertain. The search, the feeling that there is a meaning becomes the most violent; as the chief of the Memory clinic told me once: “I love guessing”.

The case conference starts as soon as Daniel enters the room and takes his seat. As usual, the first part of the conference takes place without the patient and his wife. Michael, the fellow begins the presentation of the patient, Beth the nurse adds information she got from her interview with the man’s wife. Half an hour into the presentation of the patient by Michael, Daniel comments: “Nothing is going to dissuade me that he is not a gene carrier”. Several features in the description given by Michael and Beth “worry” Daniel.

\textsuperscript{436} Ibid. p 51.
\textsuperscript{437} Penetrance in genetics is the proportion of individuals carrying a particular variant of a gene (an allele) that also expresses an associated trait (the phenotype).
First, the wife said to Michael that her husband “stands up for himself more”; Daniel understands this as a shift of personality towards dominance. Cold versus Warm, Dominance versus Submissiveness, are “traits” analysed by Nicole a neuropsychologist of the Memory clinic specialized in social cognition. She places these traits at the four poles of a circle and the personality of each individual is understood in relation to these poles. Daniel explains Nicole’s theory while drawing the circle and its poles on the board: a personality shifting towards the North East cold-submissive position is a personality typical of FTD. “Being dominant” therefore, is not a symptom of FTD, yet a change towards a “cold” personality is. Daniel thus wonders: “Is he getting cold?” Neither Beth, nor Michael, have sufficient information to answer Daniel’s question. Second, the couple told Michael about a curious episode: two years ago when the patient was under a lot of stress, he began to feel that he “could not complete a yawn”, this sensation lasted for months, he tried some meditation techniques to “focus on his breathing” but without success; the sensation eventually passed when he stopped focusing on it. This bizarre episode led Daniel to deduce that this man is “compulsive” and to diagnose a “basal ganglia439 kind of symptom” typical of FTD. Third, the patient wants to quit his job and “endorses concentration problems” Michael reports; Daniel summarizes: “sounds he is in trouble at work”. Fourth, he smokes pot everyday: an “addictive” behavior that matches well with FTD’s symptomatology, although we will learn later that this behavior is not new; the man has been smoking pot since he is 16. Fifth, Beth tells us that the man remarried 10 years ago and that his (relatively) new wife is 13 years older than him: “Maybe he was looking for someone more mothering”, suggests Beth. Judith, the genetic counselor, asks candidly the chief of the Memory clinic: “This thing of having new partners, is that a symptom [of FTD]?” Daniel answers, only half joking: “Yeah, I’m suspicious of everything...” Yet, overall, Beth doesn’t add much: the wife told her that her husband is “doing very well, no memory problem, he just got depressed since he learned that his father had a gene”. And she didn’t mention to Beth the yawning episode. This led Beth to conclude that this woman is not “deeply psychological”, to which Michael adds that “she might be in denial”. Additionally, both found her to be “prickly”, “suspicious of medical approach” and “anti-western medicine”. Finally, the team as a whole found the man “anxious”, “intense” and “rigid”. Emily the neuropsychologist, gives an example of this rigidity: when he took the test he was asked at one point to “make a design” by connecting 4 dots out of 5, the patient complained: “I don’t know what you want: what by ‘design’ do you mean...?” Judith found that he had “a weird stare”, a weirdness that Michael attempts to specify: “A little stern, maybe”.

439 The basal ganglia is situated at the base of the forebrain; it contains several subcortical nuclei connected with the cortex, the thalamus and the brainstem; they have a function in the control of voluntary motor movements, emotions, cognition and eye movements. See Fix, James D. (2008). "Basal Ganglia and the Striatal Motor System". Neuroanatomy (Board Review Series) (4th ed.). Baltimore: Wulters Kluwer & Lippincott Wiliams & Wilkins. pp. 274–281.
The results on the neuropsychological test dispensed by Emily were generally very good (including the MMSE: he got 30), except for one test: uttering a certain number of words starting with the letter D. In one minute he came up with only nine words; Daniel exclaims “Oh...! Tss, pretty bad”. If the impression that this man has a gene predisposing him for FTD – which means that the man already appears as sick – is making its way, one event is going to change slightly the deal. The lights go out. The MRI appears wide on the wall. We look in silence at the sections of the man’s brain following one another; for a while we could only hear Daniel’s index finger scrolling on the mouse. Daniel for a moment wonders about the shape of the right temporal lobe and the insula; Michael doesn’t find the atrophy “striking”. After discussion with Michael, Daniel concludes that the MRI “is in the normal limits”. He tells us: “So it’s hard... should I bail?” No one answers.

Shortly after, the patient enters the room accompanied by his wife. After everyone around the table was introduced, the man reassures us: he has nothing against being watched by more than six pairs of eyes: he is “an open case”. Daniel starts the interview; he first wants to “hear a little bit” about the man “as a kid”. The man says he has “never been a social person”, he is shy he tells us: “Even if I don’t play shy”. Never been anxious as a child? Never. Nor as an adult: the anxiety attacks are recent, only since these last two years and a half. Daniel doesn’t ask for the reason of this anxiety. Smoking marijuana gives the man “a peaceful feeling”, but it is not new: he started smoking when he was sixteen. What about his strengths and weaknesses in the classroom? The man answers that he was a good student, he liked “artistic things” more than anything. Today, he works in a genetic company (“ironically” he comments) as a lab person, but is not satisfied by his job. He prefers landscaping, horticulture and climbing up trees: “an applied art that has practical implications...” What he wants and always wanted is to be a “free thinker”. Daniel now makes him wave his both hands like marionettes. The left hand is moving with slightly less ease than the right; Emily is giving Michael several glances charged with meaning. After Daniel completed his exam, it is time for the patient to ask his own questions. The man reiterates his desire to know if he carries the gene of his father or not.

Daniel (perplexed). – You would be one of the few people who decided pretty strongly you wanted to know... Do you have any mixed feelings about that? Sometimes we found that if you find out that you don’t carry the gene it is even more stressful.

The man and his wife (with one voice, dubious). – How so?

Daniel (with authority). – Guilt.

Judith (to the man). – You know, if the result was negative you could feel terrible.
The man (*firmly*). – You know I could test myself, with my company. Until now I have resisted because I’d rather do it through your program. But if you don’t want to do it, I will do it my way.

The wife (*to Daniel*). – I don’t understand why it is so odd that he wants to know, there is 50% chances that this is positive and ...

Daniel (*interrupting*). – I think you are perfectly rational... but in Huntington’s families, one patient out of five wants to know.

Judith. – In the Vancouver families with a progranulin\(^{440}\) mutation, no one wanted to know.

The wife (*irritated, to her husband*). – Why do they keep telling us that the others don’t want to know?

Daniel (*to the man*). – It seems even, that if you are a gene carrier, you’ll want to know less than the one who is not a gene carrier... So the fact that you want to know: let’s put that on the plus side.

Judith. – And there are benefits but risks too...

The wife (*exasperated*). – I don’t see what the risks are... Suicide? This is the only risk I can think of...

Daniel (*calmly*). – I would call that a “genetic worry”: I don’t want to do harm... I just don’t want to harm... But we would not mind setting it up, and I think you will enjoy Kim [the other genetic counselor], she is very intellectual.

In the end, Daniel and Judith commit to the desire of the man; he and his wife will leave the room thankful for their help. Daniel and Judith’s repeated attempts to discourage the desire of the patient and his wife to know the result of the genetic test, manifest their deep ambiguity towards the test. Unlike Mrs. Joe’s test not yet approved by the IRB, the man who accepted to have his blood drawn to get his DNA tested is legitimately in a position to know the result; moreover this is why he is here. Strangely, his decision destabilizes the chief of the Memory clinic: Daniel’s “love” for guessing just gave way to a “worry”; the man’s desire to be part of the game of divination seems to be tormenting. Daniel and Judith attempted to dissuade the patient to follow through with a particularly infantilizing argument: because other genetically susceptible persons by a majority refuse to know—yet, in the example of Huntington’s used by Daniel, the same persons do not do the test (e.g. they don’t have their blood drawn)—the man should blindly adopt the same attitude. Let the team divine in peace without aftertaste, without having to think about the person who might then have to manage a life, as A.R. writes about the test for Huntington’s disease: “with a genetically foretold disease \(^{441}\)”. The confrontation between the man, his wife and the team, has built up an opposition that it is important to bear in mind for what is going to follow. After closing the door, Daniel goes to the blackboard, draws two columns

\(^{440}\) A mutation in the progranulin gene causes FTD too.

\(^{441}\) That’s the expression used by A.R. writing about the test for Huntington’s disease. In *Manifeste de Dingdingdong*, p 75.
and casts out to the team: “What is against it [FTD]?” And he begins by answering his own question in the left column: “The fact that he is here, the fact that he wants to know” [his gene status].

Lucy (a medical student). – No clear symptoms.
Beth. – The scan looks pretty healthy.
Emily (changing the direction of the conversation). – He has a trouble with his left side...
Daniel. – I’ll come back to that... He’s got a good relationship, he is working, he is not apathetic, he is not euphoric and he has insight: people who carry the gene tend to be anti-research, but... (Daniel shifts to the right column and agitates his left hand like a drowsy puppet:) I really didn’t like this... And he can say only nine D words, he is rigid clinically and neurologically [his personality is rigid as well as his body].
Judith. – He does not blink.
Emily. – He is anxious.
Daniel. – He has a psychiatric symptom: marijuana.
Emily (to Daniel). – Why is that psychiatric? He has been doing it since he is 16...
Emily asks why does that count as a symptom of a (sudden) degeneration of his brain? He has always done it.
Beth (to Emily). – He is addicted!
Addiction is a symptom of FTD. This suffices. And it doesn’t really matter that the addiction is not recent since for Daniel, the disease is somehow at work very early in life, especially when a gene might be involved. Remember also, how for Daniel diseases do not only affect personalities in a certain way, but also certain personalities constitute a terrain for the development of specific diseases.
Daniel (adding to the right column:). – Change in job plan, he wants to do something creative, this is always something bad... [it is a change and moreover FTD can make patients “more creative”]. And this guy would put pressure on our team to know his gene status; he would rigidly pursue it, irrationally.
Very strangely, the desire for knowing which was at first counting “against” the chances that the man had the deleterious gene, is now revealing a rigidity and irrationality and counts “for” the chances that he is a gene carrier. This direction’s change seem to owe much to the dissension (and to the “feelings” that may have aroused) between the team and the couple about their desire to know the gene status.
After this inventory, Daniel proposes that each of us bet on the gene status of the man. Beth goes first.
Beth (betting). – 75% [ chances that the man carries the gene].
Judith. – 85%.
Emily. – 100%.
A guest. – 75%.
Daniel. – 95%.
The medical student, to Daniel’s question: “What do you want Lucy?” answers, troubled: “I don’t want him to be sick” and bets 0%.
Me. – 50%.
I explain that the probability that the man has the gene is grossly 50% and that I will go with chance alone. So I decided to trust luck and I refused to see that man as sick (and thus to predict that he is a gene carrier like most of the people did), or not sick (like Lucy). I did bet and in that sense I participated in the team’s “language game”, as Wittgenstein says. Yet, I refused to be in a position to divine about that man’s future. Chance will decide I said, not me. If I refused it is not because, like Lucy, I believe that my bet could have an actual impact on that man’s future. Indeed Lucy didn’t “want” the man to be sick and “chose” zero as if saying that the man will have FTD, would give him FTD. Surely, I didn’t think the man was currently sick: first because I didn’t agree that the man was “irrational” in pursuing his desire to know if he carried his father’s gene. Diagnosing him “irrational” and “rigid” are value judgments that act as reasons for considering him pathological already. The anthropologist needs to identify these facts of values and this supposes an understanding, but not necessarily an engagement for those values to which the anthropologist can even object to. Moreover, I didn’t “feel” anything. So I didn’t feel legitimized to bet on his future.

To my refusal to divine, to my trust of luck, Judith exclaimed: “But it’s got to be science!”

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442 This kind of clinical divination, this impression the neurologist has, that he or she can see the disease despite the absence of symptoms, is a practice certainly not specific to the Memory clinic. I never witnessed it at the Alzheimer clinic, but A.R., a woman diagnosed with Huntington’s disease, writes in an astounding way about the revelation of her gene status: “On the appointed day, I go for the third time to see the neurogeneticist. I came with Emmanuelle who had made the whole route by my side, playing at each appointment the role of my girlfriend (since they asked me to play a role, let’s do it and invent myself as homosexual), and by another of my great friends. Fortunately they are there because I can’t remember this moment of the disclosure, my memory refused to catch that moment, and my two friends from then on keep the recollection of this instant. I only remember that the neurologist told me it was bad and that she was surprised since, she said, her experience usually doesn’t need the test to discern/feel when one has the disease and that for me, she hadn’t actually “seen” anything as such. She delivers the sentence in the guise of the number of CAG: 44. There is no possible ambiguity. (...) I desire one thing only: to run away from this place but I need to first pretend listening to her recommendations about coming to see them again and being followed by their team of shrinks. I make the silent oath of never seeing her again. I close my ears and I try not to break down (my affects, my emotions, these are pieces of privacy that she doesn’t deserve). Five minutes later, in the corridor, I collapse shortly; I wait being outside of the hospital to scream.” A.R, Manifeste de Dingdingdong, p71, my translation.
Daniel (*correcting Judith*). – *It’s* experience, I’ve never been wrong with them [the patients].

Judith didn’t understand that I choose luck, over what “must” be the matter of “science”. But Daniel corrects the genetic counselor: “it” –but what is it? Is it a feeling?– is the matter of “experience” and not “science”. Nevertheless, Daniel’s experience is as true as science.

Emily, half humorously, tries to convince me that I should trust the team’s science by feeling: “And, you know, Beth can tell who has AD just by looking at them [the patients]!” Thus, there is no question about “it”: we are talking about feelings.

Beth turns towards me... I joke: “Don’t look at me...” But she is being reassuring: “You don’t have AD!”

The conference is over a little bit after that and everyone leaves.

Nevertheless, the story does not end there. Three weeks after, I was in another team conference, attended by Daniel who was again intuiting about the diagnosis of a woman. At one point of the discussion, Daniel mentions climbing trees as a prodrome for FTD. I am surprised but I don’t ask anything during the conference because I know that disclosing a gene status is forbidden, even if alluding to it doesn’t seem to be. Only after the conference is over, I meet Daniel in the lobby, Laura is there with him in front of the elevators. I ask more or less directly if his allusion during the conference meant that his intuition was right the other day. He was right indeed, and advises me to not bet anymore on this matter with him, although I can go betting safely with him on the psychological scores; at that he is bad.

From now on, at the occasion of several conferences, when the diagnosis is uncertain (as it is often), when a gene might be at stake in the cause the disease, I will hear this triad of prodromes (with some variations): “climbing trees” (or “trimming trees”), “married to an older woman” (but she needs to be much older, five years for instance as it was once discussed, would not be enough), “addicted to pot” (or “incapacity to say enough D words”), as the new epistemological ground for frontotemporal dementia’s diagnosis.
CONCLUSION

When we speak of dementia what are we speaking about? I tried to answer this question from the point of view of neuroscientists who talk about and who diagnose dementia in an American and in a French clinic. I first described the ethical contexts in which these behavioral neurologists build their knowledge: how is a patient with a brain disease --and even, how is anyone with a brain-- thought about in the paradigm of behavioral neurology? After this detour through the medical ethics of the discipline, I considered its situation in relation to neuroscience and to psychiatry. By the end of chapter 1, we will have learned how dementia (particularly FTD) is a cerebral condition whose clinical symptomatology could easily constitute a bridge between neurology and psychiatry. I then analyzed in chapter 2, what neuroscience has taught us over time about our social, emotional, rational and cerebral being with a (humble) history of the “social brain”. From the end of the 19th century until today, we observed how the understanding of our social being by neuroscientists who studied the “frontal lobes”, increasingly focused on our capacity to feel emotions and less on our capacity to reason. I then showed that despite the radicalism of the theories about human nature that one can hear in the American clinic and despite the mastery of a number of technologies that gives, in both clinics, access to our biological and cerebral nature, uncertainties and fragility in the diagnosis of dementia do remain. I finally described how, despite the vulnerability of their knowledge, neuroscientists succeeded in making a diagnosis, and particularly how, because of this climate of uncertainty, the feelings of the American team were as reliable a tool as the technology in order to arrive at a diagnosis. At the end of the last chapter, the quest for meaning and our desire to know were satisfied, the “real” was not anymore to “come” as Clément Rosset would say; the work could have ended with the end of the quest for the diagnosis. Yet, in this conclusion I would like to begin to clarify one point: what are the values of this science of dementia? Even if this question takes us back to the ethnography for an instant, it seems worth making that detour in order to ask what is (are) the project(s) of these science(s)?

We have noticed that facts and values were involved in the knowledge and the diagnosis of these brain diseases: MRI, autopsies reports, DNA analysis, memory tests, language tests and testing of emotions, meet with not only the pain and the anxiety of the patients, but also with some philosophies, doctors’ impressions and affects. This is not surprising; the intrication of facts and values is inevitable in biological sciences as Georges Canguilhem taught us. Unlike mechanics and physics, we cannot look at the phenomena of the living with a neutralized eye that wouldn’t take into account the fundamental polarity of facts and values. This polarity is

444 In “Qu’est-ce que la psychologie?” (1968).
inevitable and desirable: the sciences of the living are about life thus these sciences have to do
with what constitutes life. Hence, Canguilhem writes, that this science is not limited to the
“investigation of facts, to the exploration of a domain”\(^{445}\). But that “science more or less allows
itself these facts and thus takes over what we call its domain. (...) The object of science is not only
the specific domain of problems, of obstacles to solve, it is also the intention and the aim of the
subject of science, it is the specific project that constitutes a theoretical consciousness as such”\(^{446}\).
What then is the project of this science of dementia?

To answer this question, I do not aim to frame the project in terms of an ideology that I
would appreciate or criticize from a reality, or from a criterion of truth, which would be situated
outside of the domain of science. To answer this question, the comparison becomes an invaluable
tool: not only does it provide an answer from inside the field of neurosciences, it also helps
differentiate two projects. Indeed in these two clinics, the project that this science gave to itself
seems to be slightly different; the values that infuse the conceptions of the human and the
reading of the social being are, I think, different in the French and in the American clinic. I thus
describe briefly the projects of this science with a comparison of the knowledge made about
frontotemporal dementia in both clinics. It is indeed through the knowledge produced about this
disease that the differences stand out more radically. I start with the French clinic. Here the
treatment of the comparison is not the same as in the body of the dissertation; the French clinic is
considered for itself, and not only as a reference in order to elaborate on the particularities of the
American clinic.

What is unique about frontotemporal dementia? I asked Pierre. He said he has a theory on
that matter: he calls it “la frontalité”. There are degrees of frontality, Pierre said, which
characterize our autonomy towards the environment. Pierre constructed a noun (and a concept
maybe) from the adjective “frontal”: we can now speak of frontality like we speak of solidarity,
modernity or femininity.

Pierre comes every week to the Alzheimer clinic to see difficult patients’ cases. The
building that hosts the Alzheimer clinic is named after François Lhermitte, an eminent French
neurologist. I described some of his work on the frontal lobes in chapter two. Lhermitte’s patients
were not sick with FTD but they had a lesion of the frontal lobes; from his observations Lhermitte
built the concept of EDS: Environment Dependency Syndrome. For these patients, the
social and physical environment issue an “order” to use them, wrote Lhermitte in 1986, an order that is
so strong that it deprives them of their free will. Lhermitte died in 1998 but he left his mark in the
practices and the discourses of the neuroscientists who now pace up and down the corridors of the
Alzheimer clinic. Pierre is a central actor in this conclusion, because he is at the same time a
researcher (Dr. Vincent doesn’t have much time for research) and a renowned clinician; what
Pierre says and does, gives us at once an insight on the practices and the knowledge at the
Alzheimer clinic.

For Pierre, a high degree of frontality is physiological at certain times of life, he says. For
instance, all teenagers display a frontal syndrome: Pierre’s son who is sixteen years old, asked his
dad to buy him the same T-shirt as all of his friends—he is very conventional, comments Pierre--,

\(^{445}\) Georges Canguilhem, “Qu’est ce que la psychologie?”, in *Etudes d’histoire de la philosophie des sciences

\(^{446}\) Ibid.
if his dad tells him that he’ll have to wait until the end of the week (or worse, that he won’t buy him this T-shirt), it’s going to be very painful for him. I must be looking only half convinced, because Pierre adds: “Remember when you were 16, or when you were six, how difficult it was to wait for Christmas to get the gifts, today it’s easier to wait for Christmas: it’s not that you are less interested in gifts, it’s that you have another system which is a system of behavior control that helps to equilibrate all of this”.

At my age, the degree of frontality is generally low; I should be able to put things into perspective. But you see, Pierre observes, we are not equal: frontality is not only a question of age, it’s also a question of context and temperament. Sarkozy for instance (the ex-president of France who was at the time of this interview running for his second mandate) when he wants to say something in a debate, he has to say it: on TV the other day he was under the hold of emotions; his control system got dumbfounded. Sarkozy and Pierre’s son, teenagers in general, are frontal as well as very old people too. Yet, for the old, it’s not quite the same: there is more irritability, more impulsivity, less flexibility.

The frontal lobe is an instance of control. It controls our dependence from the emotional environment built up by a debate on TV, it soothes the unbearable wait for a gift or for someone, it frees us from the group – “groups of adolescents reinforce frontality in making each individual’s autonomy weaker than when they are alone”. The frontal controls our desire to fade away into our surroundings, our temptation to be what the others want us to be, the frontal tempers, moderates, conciliates; without such control, we are decidedly no more free --assuming that we ever were, Lhermitte would have said.

When Pierre tests a patient for a frontal syndrome, he offers his hands to the patient. Pierre holds out his two hands open in front of the person: the quintessence of the friendly gesture. Pierre maintains that the sane person won’t take his hands or if she does it won’t be without expressing some perplexity, patients with a frontal syndrome, including FTD, will take his hands. Pierre then repeats the gesture but now accompanies it with a warning: “Don’t take my hands.” The hands of FTD patients should be drawn towards Pierre’s. The neurologist will then crosses one of his hands on top of the other and he’ll offer his cross-palmed to the person; what seemed like a friendly gesture now looks like a trap. The residents and I observe Pierre tenaciously proposing his hands to the person in front of him, who understands the subterfuge and awkwardly tries to resist Pierre’s deceiving invitation.

Pierre is offering his hands for the third time to a woman, the patient.

The woman (to Pierre). – You want to hurt me.

Pierre (to the woman). – Why “hurt”?

The woman brings her hands nearer to Pierre’s.

Pierre (severely to the woman). – What did I say about the hands?

The woman. – That I ought not to take them.

447 “Frontalité” designates actually also an artistic point of view, which precisely is lacking of perspective or depth: like in Byzantine and Parthian arts, where characters are represented only in the same frontal plane.
Pierre (to the residents). – So I think we have... (The woman touches Pierre’s hands.) A strong element here...

The woman (taking her hands away). – That’s enough!

Pierre (to the residents). – We’ll retain the idea of a very progressive trouble... There is... (Turning to the woman who is looking at him.) We try to understand: if we want to treat your memory, the first step is to do a diagnosis. When we look carefully at your MRI we have the impression that there is something else than a normal aging of memory. This is why you’re here and maybe you’ll have to wait a bit before we can maybe tell you what you have.

The woman (to Pierre). – The answer is youth Doctor.

Offering hands is not part of the diagnostic practices at the American clinic. There, I never saw the neurologists oppressively holding out hands to their patients. Daniel was Frank Benson’s student and didn’t learn neurology by watching Francois Lhermitte testing people at the Alzheimer clinic. Note that there is a history that impacts the way neurologists make their clinical exam. This diagnostic test is central to Pierre’s exam (even if he uses others), and not to the chief of the Memory clinic, nor to his collaborators. Daniel however, might not disagree with the theory of frontality that Pierre just told us about.

First because Daniel says, like all the neuropsychologists at the Memory clinic, that FTD patients are “stimulus bound”; at least some of them. Daniel doesn’t explain frontal disorders by the scholarly notion of frontalité, but Daniel thinks that they are well captured by an image pulled out from the cartoon Beavis and Butt-head. The chief of the Memory clinic describes one episode of the series with enthusiasm (he is a “fan”, he has the “silver collection”): a dollar bill is floating from someone’s hand, Beavis and Butt-head are looking at it. One of the two guys imagines “this fantastic castle that he has bought with all these women, you know, this wild fantasy” and the other guy only sees a dollar bill in his thoughts, “there is nothing generative about him”. Both impersonate in their own ways two figures of FTD: the first is wildly “confabulating” and the second is “stimulus bound”. In the end, these two figures are not very different: one “generates” whereas the other doesn’t, but both are dumbfounded: one by his dreams, the other by reality.

Second, Daniel as Pierre compares his FTD patients with teenagers: “Like teenagers, they do not have the ability to project in the future, they don’t care about tomorrow. If they don’t pay the bill today, tomorrow the light will be turned off, but they don’t care. This inability to project into the future and emotionally feel what it will be like tomorrow when these bills aren’t paid: this is FTD.” FTD sufferers lack perspective for Daniel as for Pierre; they are baffled. Yet the reason for that might be a little different for the two neurologists. Daniel associated the inability to project in the future with a blunting of the emotions, Pierre didn’t -- on the contrary, in one example given by Pierre, Sarkozy was dumbfounded because he couldn’t extricate himself from his strong

448 Daniel recounts that Frank Benson was the first to identify “confabulation” as a symptom of FTD: the one who confabulates does not “censor”, Daniel explains: “Whatever pops into the idea bank is outputted in the speech without any censoring”.

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emotions. Daniel said that teenagers and FTD patients are lacking perspective because of the absence of an emotional feeling of what tomorrow will be like: the redundancy seems to express that if they are cognitively aware that tomorrow will go by without lights, they are not affectively aware of it. Something is lacking in the teenager’s understanding: able to picture without being affected, as if the teenager today would not empathise with the teenager of tomorrow.

In chapter two, I described at length how Antonio Damasio connected the destruction of the frontal lobe with our capacity to be social and emotional beings. Emotions are critical to our social brains in that it allows us to understand other brains; empathy is the medium—in understanding the thoughts, the feelings and the pain of others—of our social brain. We saw in chapter two how FTD sufferers teach the neuroscientists of the Memory clinic about emotional coldness, lack of empathy and of attachment by displaying cruelty (“to animals, children and the elderly” Daniel told us), by manifesting an absence of care or love towards others. We’ve seen that the description of their pathology by Daniel, Albert, Nicole and a philosopher who worked with these scientists, has resemblance with Damasio’s description of what a destruction of our frontal lobe does to our emotional and thus to our social being. FTD patients “are not able to read emotions” anymore, neither “to make decisions in social settings”, says Nicole. Empathy is the glue of the social. Even if Albert finds that there are “four or five flavors of FTD”, he sums up the blend with this dominant taste or “thread” as he says: “this loss of social and emotional connections” which involves a “particular set of brain structures: the orbito-frontal cortex”. This idea is central to the diagnosis of FTD at the Memory clinic, central too to the conclusion of the philosopher who collaborated with the American team: FTD patients fail to “care” (for the others) is the message she communicates in philosophy conferences. I could also speculate about the centrality of empathy in the attitudes that the neurologists, especially in the American clinic, embrace towards their patients: connecting with the disease, trying to “penetrate” as Minkowski

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449 The high degree of frontality of teenagers or of Nicolas Sarkozy, said Pierre, shows the lack of an instance of control that would have extricate them from the sideration of the moment. François Lhermitte (but not Pierre) gives the following neurological interpretation: the frontal lobe controls the parietal lobe, when the frontal is incapable of exercising its control over the parietal (because of lesion or a neurodegenerative disease) the parietal lobe is freed to merge the diseased into the present environment (see chapter 2).

450 Allan Young in his recent research on “The social brain and the myth of empathy”, states that even in the absence of a standard definition of empathy among social neuroscientists, there is a general understanding that “empathy” refers to a state that is shared by an observer and another individual, present or imagined. Empathic states include, singly or in combination, cognitive empathy, emotional empathy, and somatic empathy (mainly pain).” Science in Context 25(3), 401–424, 2012, p 405. Jean Decety, a researcher on the neuroscience of empathy, also a collaborator of Allan Young on the book Empathy, and an occasional lecturer at the Memory clinic would agree with the definition and with its imprecision. Jean Decety says that he actually “doesn’t care about having an accurate definition” of the notion. Thus empathy is: “a ‘concern’ or ‘caring’ for someone else”, is also “one’s emotion that matched other’s emotion” as well as “knowing what the other is feeling or thinking”: “empathy is all of that”. Jean Decety at the Friday’s morning conference at the Memory clinic.

451 Jean Decety told his surprise to the team of the Memory clinic when he realized that the system involved in these contradictory feeling (love and hate, empathy or absence of care) was the same: the psychopaths he tested with fMRI activated their insula even greatly than the normal people. Yet he found that the connection between amygdala and the orbitofrontal cortex must have been impaired in the psychopaths’ brains since those show a lack of response in the orbitofrontal cortex. Communication at the Memory clinic. For more details, see Jean Decety, Chenyi Chen, Carla Harenksi and Kent A. Kiehl, “An fMRI study of affective perspective taking in individuals with psychopathy: imagining another in pain does not evoke empathy.” Front Hum Neurosci. 2013; 7: 489.

452 Albert in a filmed interview with the philosopher.
The description of FTD sufferers as persons lacking emotions and empathy didn’t appear to me as central to the diagnosis in the French clinic as in the American clinic and I never heard this specific verdict about caring at the Alzheimer clinic. I once told Pierre who was precisely asking me about the differences I noticed between the two clinics, about the differential emphasis on empathy in the making of the diagnosis of FTD, he looked somewhere else and told me: “Empathy? No, we don’t do much research on that”. Of course, the French team shares similar knowledge about FTD with the American team: the notion that FTD patients “lack empathy” is known to the French team as it is to the world: since 2011, “loss of empathy and sympathy” is the third item on the list of the international criteria to diagnose behavioral variant FTD. When the French team discusses a patient case and share the impression that more tests are needed to settle a diagnosis of FTD, they say: “We need to look at the affective side”, “We need to examine again the emotional aspect”, “She maybe has an affective indifference”, etc. Thus, French patients are sometimes subjected to the “faux pas recognition test” which investigates their ability to read other minds and are asked, like in the American clinic, to recognize the emotions expressed by a set of faces. Yet, emotional coldness, absence of caring and failure to love are notions that didn’t appear to me as much scrutinized by the French team as by the American team. I suggest here two reasons that could explain my impression. One is that in the French team, the reading and the analysis of FTD’s symptoms is more through apathy than empathy, more as an impairment of action than of love. A second reason is that, “emotional coldness” is backed up in the American team, but not in the French team, by a theory of human nature. I briefly develop these two aspects below. Too briefly, but it is granted that these points aspire to further development.

Pierre’s domain of research is not empathy but apathy. Pierre is interested in apathy because, he says, it is “the most common behavioral trouble”: 100% of FTD patients, and 60% of AD patients are “apathetic”. It is also a common symptom in Parkinson’s disease, Huntington’s disease, schizophrenia and of course, depression. Pierre explains that both the

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453 Yolande Pijnenburg, “New Diagnostic Criteria for the Behavioural Variant of Frontotemporal Dementia”, European Neurological Review, 2011;6(4):234-237. The criteria “loss of sympathy and empathy” was introduced in 2011 for the diagnosis of bv-FTD by this international working group. The authors added this criterion as well as others to detail and specify the previous symptomatology (“impairment of social and interpersonal conduct”, “emotional blunting”) established by Neary et al. in 1998 (Neary D, Snowden JS, Gustafson L, et al., Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria, Neurology, 1998;51:1546–54.)

454 The faux pas recognition test that is given to patients at the Alzheimer clinic is inspired from tests developed by Simon Baron-Cohen, an authority on autism. These kinds of tests investigate the ability of people to read other minds, ability also called “Theory of Mind” (the work of Baron-Cohen is the paragon of this theory). People listen to a series of stories in which at one point someone said something that shouldn’t have been said (the faux pas), patients are then asked to answer a few questions that test their capacity in understanding the faux pas. At the American clinic, Theory of Mind is also tested but with tests directed towards the ability to detect sarcasms, bluff and irony, more than “faux pas”.

455 This is not to say, of course, that “French people” are not sensitive to this aspect of the pathology. To cite just one: Catherine Malabou, a French philosopher (also in residence at UC Berkeley for two years) in her inquiry about brain plasticity wrote an entire book about cerebral destruction (due to dementia and trauma): she refers to and fully embraces Damasio’s theory that demented patient are not themselves anymore because of their emotional coldness. In Les Nouveaux Blessés: de Freud à la neurologie, penser les traumatismes contemporains. Paris : Bayard, 2007.

456 Pierre at the Alzheimer clinic presenting his research on apathy to the team.
frontal lobe and the basal ganglia (the “slave” of the frontal) are implicated in apathy—which he defines as the “quantitative reduction of a goal-oriented action”\footnote{A definition that Pierre qualifies of “local opinion”, that Pierre sees as the major contribution of his work: not doing anything (apathy) is not necessarily due to a trouble of motivation. I here summarize very briefly what Pierre says about apathy, when this need to be scrutinized more closely, particularly Pierre is not at all definitive about his description. The pathology of action can be explained by four kind of troubles: a trouble of “intention”, “planning”, “execution” and/or “expectation”. Pierre develops that intention and expectation (or valorization) both involve “motivation”. Planning doesn’t involve motivation but reasoning. A trouble of the action can also be due to a trouble both systems. Pierre isn’t sue about “execution”: is it a third or a forth system?}. The frontal lobe has its share in two causal mechanisms of the pathology of the action, to each of them corresponds an anatomical region: planning the action is controlled by the dorsal part of the frontal (involved in “cold cognition”)–a lesion of this part of the brain impairs the “realization” of the action–whereas the orbital part of the frontal is involved in a “valorization” of the action–a lesion of the orbital part causes what Pierre understands as a “motivational” problem. What Pierre says about the orbital part is interesting because this is where Damasio, Daniel, Albert and Nicole localize empathy (Nicole localizes it in the right temporal lobe too). Pierre tells us that the orbital part of the frontal, in opposition to the dorsal part, is devoted to “warm cognition” \cite{cognition_chaude}: it “regulates our motivation, our emotions and social interactions”. “Emotion” and “social interactions” are also attributed to this cerebral region by the American neuroscientists. Yet, note that Pierre here used the term “cognition”, even if it is a warm one, to subsume our emotional and social capacities. Note also that Pierre didn’t mention empathy as a task of this part of the brain. In Pierre’s framework, motivation is determined by our capacity to “attributes a value” to an action; if one doesn’t “valorize” an action, one doesn’t act. We remember from chapter two, that the philosopher asked the team if FTD patients still could “care”, care being for her a variant of valuing. Pierre could have joined the debate that went on at the Memory clinic that day with the philosopher: Pierre says that valorization of the “action”, and also of the “context”, is “probably coded in the orbital part of the frontal lobe”. Yet Pierre didn’t mention anything about valuing “people”. We might remember, from the debate that took place between Oliver on the one hand and Albert and Daniel on the other, that Oliver refused to settle that FTD patients didn’t “care for others”, were “sociopaths” or “didn’t love”, but that Oliver understood their indifference as a problem of the “will”; “motivation” Pierre might have said. An understanding that seems very close to the orientation Pierre gave to his research: what the disease impairs is the capacity to value an action, not necessarily to value one’s kin or friends; Pierre, as Oliver, had nothing to say about that. None of the neuroscientists at the Memory clinic would disagree with Pierre that one of the symptoms of FTD is apathy. Yet, when Albert describes to the lay public\footnote{Albert talking about FTD in a talk for the lay public at the University of R.}, an FTD patient as someone who “refuses to pick up the child in school” or “shows disinterest for date nights” he doesn’t explain the ‘refusal’ or ‘disinterest’ in terms of an impairment of the will, but as a trouble of empathy: “They don’t show anymore an ability to connect to others, or to be concerned in others”, and he authenticates his reading with the testimony of one caregiver: “I don’t know what happened to our connection” she said, “this sense of being supported by my spouse, this is gone”. In the framework used by Nicole, Albert and Daniel, the idea of an apathetic love, a reduction of the actions that constitute “love” isn’t sufficient to explain this particular kind of indifference, the one that is directed towards other beings. A theory of action cannot fill in for this sense of being connected and supported. This sense belongs to the notion of empathy, a notion that exists for itself and that is individualized by a neurological center; Nicole says she
“believes” that “connecting to other people is intrinsically rewarding\textsuperscript{459}”. The individualization of empathy and of the cerebral network that necessarily underpins it, owes much to neo-Darwinism; we already saw in chapter two how Damasio backed up the relationship between sociality and empathy with evolutionary theory, I will now briefly refer to the work of Allan Young in The Social Brain and the Myth of Empathy to inquire further about the role of neo-Darwinism in an understanding of human nature in terms of empathy.

Allan Young tells us that since 60 years “human nature is increasingly understood with reference to empathy, brain science and evolutionary biology, rather than reason, and without recourse any longer to social anthropology\textsuperscript{460}”. Allan Young specifies that this is true in the “Anglophone world at least\textsuperscript{461}”. One aspects of what Young calls “the social brain puzzle” focuses on the following question: why did our brain, a very costly organ in terms of metabolism, continue to grow (it quadrupled) after our split from great apes six millions years ago, when “further growth was no longer adaptive?” Young continues: “This is the puzzle and its solution is a story about how brains adapted to other brains\textsuperscript{462}”. The solution that several scientists embrace, including the neuroscientists of the Memory clinic, is that this growth was devoted to a mind reading capacity\textsuperscript{463}. I never heard the French team expressing an opinion on what Allan Young has called the “back-story\textsuperscript{464} of the social brain”; I don’t know if the neuroscientists in the French clinic ignore this story but I know that they remained silent about it. On the contrary, Nicole’s lectures at the Memory clinic on the neuropathology of empathy is supported by our irrepressible desire to pass our genes on–our nature–which is the ultimate justification for the central place that empathy is given in our contemporary neuro-social lives: the brain allocates “more resources” to things that “happen between ourselves and others”, it is a matter of “survival”, it is of “security relevance”. Nicole exemplifies how “survival” is at stake: “If I insult my boss regularly I would lose my job and I would be unable to provide for my family, I could end up homeless. If I go out on a date and if I don’t know how to behave, if I am inappropriate, maybe I won’t get a second date and thus I will fail to pair bond and to reproduce\textsuperscript{465}”. FTD patients’ inappropriateness at a candlelit dinner or in the office is understood as a trouble of empathy, is explained by the cerebral lesions in their orbital part of their frontal lobe and is rooted in a neo-Darwinian “myth” as Young calls it. This “mythical” back-story gives empathy a central place in the endeavour of neuroscience about our neuro-social nature and reciprocally allows judgements to be made about our “humanity” from the study of FTD patients. Nicole says that neurologists and neuropsychologists are “experts in human nature”, that she always wanted “to understand what makes people human”. Her project, which is the one’s of neuroscience in general–some French neuroscientists too, think that it is possible to explain the human entirely from his brain\textsuperscript{466}–is

\textsuperscript{459} Nicole’s talk on empathy, at the Friday morning conference, at the Memory clinic.


\textsuperscript{461} Ibid.

\textsuperscript{462} Ibid., p 414.


\textsuperscript{464} “Back-story”, Young writes, because the neo-Darwinian story is “a fictive or a notional story”, that can only be inferred, Ibid., p 409.

\textsuperscript{465} Nicole lecturing on a Friday morning at the Memory clinic.

grandly facilitated at the Memory clinic by the mythical back-story provided by evolution theory. Nicole’s understanding of FTD is echoed by Daniel’s verdict on the diseased: someone who is “stripped” of her “humanness". It also fashioned the analysis of the philosopher: FTD patients irremediably lose their “capacity to care” and it resonates with Albert’s valuing the later capacity as “the core human". I didn’t notice that the neuroscientists in the French clinic were venturing about a theory of human nature neither from FTD patients nor from Alzheimer patients, though maybe they were, but not in front of me.

The difference that I noticed between the understanding of FTD at the Memory clinic versus at the Alzheimer clinic, seems to lead to the following observation: empathy and love are notions particularly valued in the American clinic, and the project that they develop from their knowledge of FTD, aims at defining human nature within the theoretical framework provided by evolutionary biology. This is not the case in the French clinic. “Love” is not a notion understood for itself, it seems more to pertain to a general framework that understands the pathology of affects (among others) in terms of a diminution of action. This theoretical framework might also influence the way the clinicians in the American clinic encounter the disease. As we’ve seen, at the Memory clinic, the team of neuroscientists draw on their feelings towards the person sat in front of them in order to experience the disease. The clinical practices at the American clinic more explicitly showed than at the French clinic, that the lack (FTD) or the surplus (AD) of emotions, care and love that are signs of these diseases, incarnate into and are experienced by the clinician when he or she makes a diagnosis.

See chapter two for more debates about what FTD reveals of our humanity.

See chapter two.

Is this understanding actually peculiar to the “Anglophone” neuroscientific world? I cannot tell from my observations, which are limited to two clinics, moreover the difference could be fully explained by Pierre and his colleagues’ disinterest for the subject of empathy; in another French clinic I could have observed something different. To answer to this question, I could inquire in other clinics (ideally but unlikely) or more simply I could begin with a comparison between the popularization of scientific work of French (for example: Jean-Pierre Changeux and his “physiology of truth” in L’Homme de vérité and more recently Du vrai, du beau, du bien: Une nouvelle approche neuronale, Odile Jacob, Paris, 2008 and Lionel Naccache for his work on the neuronal unconscious in Le Nouvel Inconscient Freud, le Christophe Colomb des neurosciences, Paris, Éditions Odile Jacob, 2006 and in Lionel Naccache, De quoi prenons-nous conscience?: Exercice de neuroscience-fictions, Paris, Éditions Manucius, 2013) and American neuroscientists (for example: Antonio Damasio who after Descartes’Error published Looking for Spinoza: Joy, Sorrow and the Feeling Brain, Harcourt, 2003. and Gerald Edelman in The Remembered present: A Biological Theory of Consciousness, New York, Basic Books, 1990) focusing the inquiry on the place and the treatment of empathy and neo-Darwinism.


