Narratives of Neurology
Exploring the Broken Brain Through Interview

by

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This Thesis is dedicated to my Noni
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Narrating Neurology

This thesis has, in reality, been nine years in the making. When I was thirteen years old, I discovered writing for the first time at a Young Writer’s Camp at Duke University, where, in the library, I combined simple rhyming words to craft my first poem that I felt proud of. I shared it with anyone who wanted to hear it, including my mother, who cried at its simple, cliché message, likely because it was something I had written and not because it was worthy of any actual tears. That poem came to me easily and smoothly, a misleading beginning for a complicated and turbulent practice.

From that initial poem, my writing became unpredictable. I experienced periods of intense creativity and ease, and then I inevitably fell into weeks and months where I wrote absolutely nothing, and forgot about the joy that came from being proud of something I had created all on my own.

It must have been during one of these times that I found neuroscience, which began for me as a simple thought exercise on the nature of being. Such imagining can lead a young learner in many directions - toward philosophy, or religion, perhaps – but instead, I found myself looking at mechanisms and biology, and finding all the wonder in the world the closer I looked at the basic functional units of life.

I turned writing even before I had the words to understand what I was experiencing. I must give it the credit it deserves as being my first love; it found me well before I stumbled upon science, or pictured myself happy as a doctor, and I suppose that after years of being a consuming force in my life, it will now have to take a back seat to those things. When I embarked upon this thesis, I intended to tell the stories of those who are most commonly misunderstood in our society - those with wounds that are usually invisible but profoundly deleterious. I wanted to explore the brain from the perspective of
a writer; I wanted to take it outside the laboratory and into the written world, to pull apart its beauty and complexities through people who live real lives and have real stories to tell. At first I tried to separate the person from their malady, to make it clear that it was possible to live a regular life and be a regular human being despite living with a broken brain.¹ I imagined my typical chapter going something like this: Meet [name]. [Name] has [insert disorder here]. But [name] also does [insert career/hobby/sport/club], and that makes [name] just like you and me, so we should not stigmatize [name’s] disorder.

I thought that if I presented my project in this way, where the illness was not the focus, I would have a much easier time finding people who would be willing to talk to me. But as I got further into the interview process, and was even approached by some people who wanted to be a part of my thesis, I found myself grasping a different truth. None of these people wanted to be separated from their disease. A couple of the interviewees were friends of mine, with whom I often interacted outside the context of my thesis, with whom I had plenty of other things to discuss, and all they wanted to talk about was living with their disorder. People wanted to tell me what it was like to have their brains. I was finally asking the questions that others in their lives were too afraid or too nervous or too hesitant to ask. Some questions were hard. Other questions were easy. And over the course of these interviews, not one of my questions went unanswered.

Through this I realized a seemingly obvious truth: A broken brain is not a broken ankle. There is no shame in a broken ankle, nor is there denial that the ankle is actually broken. Asking someone how they broke their ankle is not an awkward and

¹ I prefer referring to this range of neurological illness as different aspects of a “broken brain”. It makes sense for two reasons: One, because disorder, illness, and disease are all terms that only accurately describe some, but not all of the maladies I cover here; and two, the word “broken” is simple enough that it releases some of the stigma normally associated with these types of problems.
uncomfortable endeavor, and people do not hesitate to ask if the ankle feels all right. If someone is seen crutching down a hallway, bystanders do not avert their eyes and pretend not to notice; they ask if the injured person needs help carrying their books. Those unfortunate enough to break their ankle are not suddenly plagued with the monstrous task of explaining their cast in a way that will not result in people treating them differently. A broken brain is not a broken ankle.

In this thesis I cover a wide range of neurological illness. I expected certain illnesses, like anxiety and bipolar disorder, to be saturated with stigma. Others, like Alzheimer’s disease and multiple sclerosis, I expected to be relatively stigma-free. After all, we can see MS on an MRI and we can measure the levels of Alzheimer’s pathology in cerebrospinal fluid; there is no denying that these ailments exist. But the diagnosis did not matter. Nothing about the brain is free from stigma, it would seem. Despite their differences, everyone I interviewed was excited at the opportunity to talk about their illnesses with someone who was willing to listen.

This guided me to discover an additional reality that surrounds living with a broken brain: the necessity to narrate. To return to the broken ankle analogy for a moment, it is rare to find a sufferer from such a wound who feels a need to sit their loved ones down and explain what it is like to have a broken ankle. Only on the odd occasion will the ankle casualty find it hard to introduce themselves to new people, fearing that this freshly made acquaintance will find out about their injury through other means and hesitate. Conversely, these sorts of feelings are often on the mind of someone who suffers from a broken brain. More so than any bodily injury, a wounded brain brings with it the desire to narrate, to explain the daily struggle, and to ensure that friends, new and old, are comfortable with its presence.
I imagine that a few people reading this are scratching their heads. I can almost predict the puzzled thoughts. “But Taylor,” you might ask, “is it really all that bad? After all, this is the 21st century! Mental illness is hardly at the place it was 100 years ago.” To that, I agree. Things have changed. Epilepsy is no longer perceived as a body commandeered by demons,2 “insanity” is no longer a legitimate medical diagnosis, and institutionalization is no longer the first step taken with someone who is mentally ill. Despite these changes, mental illness still find itself in a well, surrounded by insurmountable walls of prejudice and stuck, in many instances, with a prehistoric interpretation in an otherwise modern world.

It is well established that mental illness suffers from intense stigmatization (Byrne 2000; Cheon and Chiao 2012; Corrigan and O'Shaughnessy 2007; Stier and Hinshaw 2007). Stigma affects mental illness across nations and cultures, “constituting a significant barrier to successful treatment, reducing key life opportunities, and predicting poor outcomes over and above the effects of mental illness per se” (Stier and Hinshaw 2007). In the stigma-heavy public sphere, parents have been blamed for causing their child’s mental illness; derogatory words like “retard,” “psycho,” and “crazy” are ubiquitous across cultures and age groups, and the media portrayal of mental illness tends to emphasize “violent or bizarre behavior and social incompetence” instead of truths (Corrigan and Miller 2004; Stier and Hinshaw 2007). The stigma surrounding a broken brain has been truthfully summarized as such: “If David’s body were hurting, people

2 This is true for most cultures. In some populations, for example the Hmong people of Laos, epilepsy is still believed to carry ethereal significance. In the Hmong culture, epilepsy is called qaug dab peg, which translates roughly to “the spirit catches you and you fall down.” Hmong epileptics are highly revered as shamans who can communicate with and journey into the spirit world (Fadiman 1998).
would send gifts, but because it is his mind that is hurting, they throw bricks” (Ben-Dor 2001).

One psychiatrist describes stigma as follows, highlighting the way intense misunderstanding can generate negative notions:

For me stigma means fear, resulting in a lack of confidence. Stigma is loss, resulting in unresolved mourning issues. Stigma is not having access to resources... Stigma is being invisible or being reviled, resulting in conflict. Stigma is lowered family esteem and intense shame, resulting in decreased self-worth. Stigma is secrecy... Stigma is anger, resulting in distance. Most importantly, stigma is hopelessness, resulting in helplessness.

(Byrne 2000)

Stigma as it relates to mental illness manifests itself in three different spheres: the personal sphere, the authority sphere, and the public sphere (Corrigan and O'Shaughnessy 2007). In the personal sphere, self-stigma perpetuates shame and the prejudice that individuals develop against themselves because they belong to a stigmatized group. This may prevent them from telling others about their illness and therefore lowers the chance that they will receive help. The authority sphere promotes structural stigma through policies, from private and government institutions alike, that limit opportunities for the mentally ill. Finally, in the public sphere, stigma propagates stereotypes, prejudice, and discrimination in the ways that the public reacts to the stigmatized group (Corrigan and O'Shaughnessy 2007). The latter is an important area for targeting efforts to reduce stigma across the board. In fact, in 2001, the World Health Organization stated in their World Health Report that the stigma and discrimination toward people with mental illness is the “single most important barrier to overcome in the community” (World Health Organization 2001).

An Internet video called “Minding the Elephant” depicts a man in an elephant costume wandering around various public places while statistics about mental illness fill
the space around him: “Less than half of those affected [with mental illness] will receive any kind of mental health service…Self and social stigma are all too present…The fear of disclosure amongst physicians, employers, and loved ones leads to ignored symptoms” (Reagan and Zash 2014). At the end of the two-minute video, the viewer is urged to “mind the elephant in the room” and end the stigma by talking about mental illness.

While working on this thesis, I talked with many different people, and so have learned firsthand that discussion is an important mediator for understanding. I certainly hope that more campaigns like this one will further the awareness of mental health stigma and the pain that it causes.

Each of the chapters of this thesis represents a different experience. Each experience carries its own burden, a burden different from any of the others. In order to protect the privacy of those who shared their burden, interviewees have been given a pseudonym. The first chapter, “My Brain is a Mess,” covers an interview with a Wesleyan student who suffers from multiple sclerosis and faced unfair treatment from the university because of this diagnosis. Despite resolving that issue, she is still challenged every day with questions of how to address (or not address) her chronic condition.

Next, “The Precipice” looks at how illness can beget illness. The individual in this story, another Wesleyan student, had two coexisting conditions – anxiety and an eating disorder - that fueled each other and led to emotional and physical damage. These two illnesses are both relatively common, but that does not save them from being condemned. In fact, anxiety and eating disorders may be among the more difficult psychological disorders to understand, as most people experience stress and monitor their diet. Because of this, it can be challenging for the healthy individual to ignore their own experiences when trying to understand these chronic conditions and how they develop.
The following chapter, “This is Your Brain on Trauma,” looks at the unexpected difficulty that can come from concussions. For this chapter, I conducted two interviews: first, with a college senior from the University of Colorado at Boulder who sustained multiple concussions over her life and is still living with the aftereffects; and second, with the CEO of a company whose head impact technology is being used to prevent sports concussions before they even happen. Both of these stories shed important light on the “concussion crisis” in American sports, most importantly the National Football League, and raise questions about the casual reaction to a potentially devastating injury.

“The Man with Two Faces” explores the difficulties of bipolar disorder by way of an interview with a middle-aged man haunted by the “what ifs” caused by his illness. We spoke over the course of one long car ride and in that time I came to realize that the greatest source of stigma in his life at this point, after close to twenty years of living with this disorder, came from within, which highlights the issue of self-stigma and self-acceptance that surrounds many of these maladies.

The final chapter, “The Dying Brain,” describes two of the most common causes of death in the United States: Alzheimer’s disease and metastatic cancer. For this piece I spent a day on the hospice ward of Middlesex Hospital and then, about a month later, interviewed the hospice physician who worked there. Though we talked little about the very real stigma that exists surrounding these degenerative conditions, I saw firsthand the burden a dying brain can be, and how heart-rending it is to lose someone to a disease that slowly takes the mind before it takes the body.

By compiling the stories in this order, I put forth a linear story, snapshot by snapshot, of how the brain matures. We see a young brain transform into an older brain, and then into a dying one, exposing different wounds that can plague it along the way.
I have taken up the challenge of narrating neurology with some trepidation. My goal for this thesis, so fluid and uncertain in the beginning, became clearer and clearer as I wrote. I strive to tell the tale of a broken brain, in many varied ways, so that the reader may understand it better. I aim to explore the brain the way a writer would. By writing in an accessible and interesting way, I can give neuroscience the recognition it deserves, and with my knowledge of neuroscience I can fuel my passion for writing. In this way, both of my passions find validation in each other.

This project has given me a new understanding of my own mental health and how I would like to incorporate written work into my future as a scientist and a doctor. I have learned that narration has value as a therapeutic tool, as an outlet for people who feel like they have no voice. Most importantly, these stories, told to me by wonderful people, have given me new insights with which to view the brain, healthy or otherwise. If my thesis motivates other people to look at the broken brain a little differently, even in the smallest of ways, then I will consider this endeavor a success.
References


When she was 18 and a freshman at Wesleyan University, Carolyn Lewis was diagnosed with the tragic and life-changing condition of needing to wear socks. It all started with a night of drinking and a terrible hangover. The hangover was the worst she had ever had: She was constantly dizzy and nauseated, and vomiting far too often and far longer than was normal. After two days of this illness, which clearly was not a hangover, she went to see a doctor, who told her that her vertigo was probably the result of an ear infection. When tests confirmed that this was not the case, she was told that it was probably a virus and sent home with instructions to return in two weeks if symptoms had not corrected themselves.

For two weeks she lay in bed in misery. She could not stand without the dizziness, she could not eat without vomiting, and she could not do anything about it except wait. And so she waited. She waited for as long as the doctor prescribed, and, as if on cue, by
the end of the second week her symptoms went away. Her two-week hangover appeared like another temporary illness bump on an otherwise smooth road of health.

Only a couple of weeks before starting the second semester of her freshman year, a new symptom reared its unassuming head. Her feet – on which just weeks earlier she had been barely able to stand due to dizziness – went numb. She thought it was strange at first, but a non-serious sort of strange, like when a cross-country runner feels an unexpected stab of chest pain. Strange, but overall not a big deal. She thought that maybe they had unexpectedly become hypothermic and were slowly freezing due to invisible frostbite, but after bathing them in hot water that she could hardly feel through the numbness, Carolyn ruled that this was unlikely.

As school got into full swing and Carolyn’s feet remained numb throughout the passing weeks, she finally decided that her condition was serious enough to merit a visit to her university’s health center. She told them that her feet were numb and they suggested that she get more exercise. She went for a run and her feet remained asleep. She went back and they postulated that her feet might be suffering from exposure to “fish toxins,” but Carolyn, as a vegetarian, did not eat fish. Still, the fish toxin hypothesis was a favorite. Finally, the health center suggested that her feet might be cold, as this was New England, after all, and this is when she received her earth-shattering diagnosis: needing to wear warmer socks.

But Carolyn knew her feet were not just cold, she had already tested that, and she was no longer satisfied with waiting for the health center to fumble around with their medical detective work, and so she took matters into her own hands.

Carolyn had not been idle following her two-week hangover and increasingly worrisome numbness. Armed with an Internet search engine and symptomatology
buzzwords, she had done her own sleuthing. The next time she returned to the health center, wearing thick, warm socks on her indifferent feet, she had her own demands.

“I want an MRI.”

“We don’t think you need an MRI. This is a pretty clear case of fish toxins.”

“But I don’t eat fish! I had sushi, like, one time last semester.”

“Yeah, like we said, it seems pretty clear. It’s definitely fish toxins, or maybe Lyme disease.”

“I would like an MRI. I am really concerned about this, and I think it’s a very serious issue.”

“Ok, we’ll give you an MRI, but this really isn’t necessary.”

When Carolyn had pushed the ‘enter’ button on her Internet search, WebMD, the site famous for making simple and non-threatening symptoms seem tragic and fatal, returned results for diseases that were tragic and fatal. One of them, multiple sclerosis (MS), seemed like the best fit for her experienced symptoms. The indications included numbness and tingling, dizziness, trouble moving, muscle stiffness, and weakness and fatigue (WebMD 2012). The only other diseases that come up when you punch in those symptoms are diabetes, pregnancy, and anxiety disorder (WebMD n.d.), which Carolyn could confidently rule out by herself.

Intrigued, she asked her father what he knew about the disease, which, it turned out, was absolutely nothing. But that did not stop him from voicing his opinion. “It’s something old people have, Carolyn,” he told her confidently.

The WebMD symptoms resonated in her mind. “Could I have it?”

“No, of course not,” he assured her, “didn’t you hear what I just said?”

But something about the description of the disease stuck with her, and, finally,
after her numb feet refused to return to normal and the number of misdiagnoses drew uncomfortably close to double digits, she decided to medically pursue it as a possibility. Getting an MRI, the Internet told her, was an important step toward ruling it out.

But when the MRI returned, it revealed white, ghost-like splotches that dotted her brain and spinal cord. Her brain had three, as did her cord, making for six marks in total. The doctor called them lesions, which meant exactly what Carolyn thought she had: multiple sclerosis. Despite the implications, she was relieved with the validation of her fears. Almost immediately after getting these results, she started to deal with the prejudice of mental illness. A close friend told her that they thought she wanted [her illness] to be something bad for the attention.

![Figure 1: These are two MRIs of MS patients that show a) lesions in the brain and b) a lesion in the spinal cord. (Gaillard 2014)](image)

Then, as if waiting for just that, the hangover-like symptoms returned, which, in her new MS-oriented jargon, Carolyn knew was called a “relapse.” But in practice, “relapse” was just a word used to describe constant dizziness and nausea, the inability to stand, and continual vomiting that lasted for a period of time measured in days.

As Carolyn contemplated her disease while bedridden because of it, the Wesleyan Health Center reeled at the newly established diagnosis. Faced with a student whose concerns and symptoms were named instead of mysterious, they found themselves unable
to provide any more help. Instead, they assured Carolyn that they were sorry about the diagnosis, but that she had suddenly transformed into a liability. They suspected she would have to leave Wesleyan.

Even before she had fully recovered, Carolyn sought support from her class dean, who promptly echoed the recommendation given from the Health Center, adding, “you should come back when you are feeling better.”

While clutching a trashcan and trying not to vomit, Carolyn responded, “I understand that this is a condition with no cure, and I don’t know what you mean by ‘get better.’ This is a progressive disease: It only gets worse. You can’t tell me to come back when I feel better, because then I’ll never come back.”

***

Carolyn and I sit across from each other in my bedroom. We are both sitting on different black chairs: mine is a desk chair, with armrests and wheeled feet and a tall, stiff back; hers is a padded bucket seat that sits low to the ground. My computer is on my lap – for the most part I just ignore it as it audio records our conversation, but occasionally, when Carolyn says the name of a drug or a person or an important date, I jot it down quickly in the notebook feature of Microsoft Word.

Carolyn is beautiful and confident, with a quirky personality and comedic mannerisms that make our cheerless conversation oddly upbeat. Her hair is a fiery red color, and hangs loosely in unkempt curls tossed to one side of her face. As if to offset the intensity of her hair color, her skin is delicately pale and her eyes are soft and Easter egg blue, but difficult to see behind her heavy, thick-rimmed glasses. About every fifteen minutes or so, these glasses slide all the way down to the tip of her nose and she pushes them back up again with the back of her hand. She is dressed comfortably in stylish but
loose-fitting clothes, and remains relaxed while we talk. Only a couple times during the conversation does her voice catch, and when it does she quickly gets it back under control.

Carolyn is a storyteller. She leads a student forum on trauma writing at Wesleyan and writes about her experiences dealing with multiple sclerosis on a blog. She is familiar with telling her story in a very particular way: Normally, she talks about the unjust treatment she received from Wesleyan upon discovering her disability. She was not accustomed to people (outside the anonymous bubble of the Internet) asking about the disease itself.

“So what exactly is multiple sclerosis?” I ask her.

“… It’s the immune system attacking the myelin in the central nervous system just causing these lesions - which are essentially holes in the myelin - which are mostly not fixable. It seems like in some cases the myelin can sort of regrow, but once you are older, scar tissue starts to form and the likelihood of it fixing itself really decreases.”

“Have any of your lesions gone away?”

“My lesions are still there. Some have healed a little bit, but they are still there, plus some new ones. My doctor won’t give me a count right now, I think because having a count would be depressing.”

“Do you like not knowing?”

Carolyn pauses and thinks about it. “I think I like to know the numbers of things.”

Her doctor tells her that the number of lesions is not necessarily important, but the size of them is. And though he will not tell her the number, he tells her that some of her lesions are big.

Multiple sclerosis, as Carolyn said, is an autoimmune disease. Like other autoimmune diseases, it is characterized by the body’s own immune system attacking
another part of the body. Depending on what that part is, the disease can be serious or mild. In Crohn’s Disease, the immune system attacks the gastrointestinal tract. In the condition of psoriasis, the immune system attacks the skin. In multiple sclerosis, the immune system attacks a specific cell in the central nervous system: the oligodendrocytes.

Oligodendrocytes are a type of glial cell, a nervous system cell found around and between neurons (Bear, Connors and Paradiso 2007). Back in the 1980s, what glial cells did was not well understood, which is what got them their name (glial means ‘glue’ in Greek), as they were thought to simply take up space and hold the brain together (Hamilton 2010). More recent research, however, has illuminated the many diverse functions of glial cells and proved that neurons would not be able to survive and thrive without the support they provide (Bear, Connors and Paradiso 2007). Einstein, for example, was shown to have an average number of neurons, but an above average number of oligodendrocytes and astrocytes (another type of glial cell) (Hamilton 2010), which suggests how important these support cells are for learning and information processing (Fields 2008).

Oligodendrocytes are strange looking, without a doubt. Like most cells, they have a double-layered membrane made out of phospholipids that separates the inside of the cell from the outside, called the phospholipid bilayer. Oligodendrocytes seek out the axonal processes of neurons (the long, skinny “wire” that sends electronic signals to other neurons), and extend tendrils of membrane to wrap around and around the axon like a fatty shawl. This protective layer of fat creates an insulating barrier and helps the axon propagate signals over long distances, like rubber around an electric wire. This insulating fat is called myelin, and in relation to the axon, it is called the myelin sheath.
If it were not for myelin, our axons would need to be magnitudes bigger than they are in order to generate the same speed of signal propagation. If this were the case, our heads, filled with brains built upon billions of axons, would be roughly the size of barn doors. Indeed, myelin is so ubiquitous that it can be seen at a macro level by the naked eye - from this perspective it is called the “white matter” of the brain (Bear, Connors and Paradiso 2007).

So in multiple sclerosis, when the myelin is damaged and destroyed, there is a range of symptoms, all stemming from the simple fact that neurons have lost their ability to propagate signals as quickly as they are supposed to. An area of significant damage to the myelin is what merits the title of “lesion.”

A relapse, Carolyn says, occurs when a lesion becomes larger or a new lesion appears. They can feel different, also, depending on where the lesion is in the brain. During her last relapse in February 2013, Carolyn experienced phantom chest pain, and all day she felt like she was having a heart attack.

Fortunately, she has not had a relapse for over a year. But there are daily
symptoms of her condition that require constant management.

“Almost every day I have a migraine,” says Carolyn when I ask her about the daily struggles, “I would say – I count it by month because I get my migraine medication in prescription packs per month – I would say I have a migraine at least 20 times every 30 days. It’s the worst. I’m also often nauseous, sometimes dizzy. If the temperature changes a lot, like if it gets warmer – which happened a lot last year, it went from 30 to 50 degrees overnight - that’s really bad. I wake up and can’t function. Because heat affects MS.”

“Do you like winter better than summer?” I ask her.

“Winter is better than summer, usually. I feel better in the winter. Although in the winter, inside spaces are heated a lot so then I’m usually pretty uncomfortable. I definitely like having control over my own temperature.”

She also struggles with her memory.

“My memory has gotten really, really bad, and I try not to talk about it too much, because I was talking to my mom about it the other day and I think it really scares her. I have to write down a lot of things. I wish I could have a normal person memory and remember conversations that I had or things that I did and not have to write everything down. My memory is a mess…”

She pauses and laughs, “I keep saying that. I say that things are a mess because it amuses me, because ‘a mess’ sounds like MS. It’s my go-to term for when things are bad.”

The severity of her symptoms tends to differ from day to day. Some days she is able to do certain things, and other days she cannot do those same things.

This simple fact makes dealing with her disability particularly hard. Not only does it make it difficult for Carolyn to make plans far in advance, but it also makes her disease almost impossible to explain. Other people, Carolyn says, have a really hard time
accepting the inconsistency.

“[People think that] because there is an inconsistency there must be a lie. That it couldn’t possibly be that some days I am fine and other days I am not. It has to be that I am not telling the truth about it. It is especially frustrating because what I always want to say is, ‘it’s really frustrating for me too - to not know every day - I can’t plan things!’ Say I want to go on a hike. I can’t say I want to go on a hike on Saturday because I don’t know if Saturday is a day that I will be able to go on a hike. And that is really frustrating.”

Carolyn pauses and looks down at her hands.

“People want other people to be a consistent way. And I think that’s why wheelchairs are easy. Like if my disease progresses and I end up in a wheelchair, people will be like, ‘we understand, you cannot walk.’ ”

“So are you saying that being in a wheelchair doesn’t scare you?” I ask.

She looks back up at my face. “Being in a wheelchair terrifies me. It’s the worst possible thing that happens. But I think people will understand it a lot more.”

***

When Carolyn was asked to leave school, she fought back, hard. She told her dean outright that she was going to stay. But the dean, operating under recommendations from the health center that she leave campus due to health issues, was strongly against the idea. She tried to hand Carolyn withdrawal papers, even after she said she was staying and asked for other options. Fortunately, both of Carolyn’s parents, who accompanied her to the dean’s office that day, work as professors at a different university, and knew the ins and outs of academia. Carolyn’s mother told the dean that what she was doing was illegal, that you could not tell someone with a disability to leave your educational institution.
When the word ‘disability’ was mentioned, her dean reluctantly backed off, but made sure to hand Carolyn the withdrawal paperwork as she left her office that day.

Despite this unpleasant interaction, Carolyn was allowed to stay. In the following weeks, a second opinion and a spinal tap (a procedure so excruciating that she blacked out due to the pain) confirmed the initial diagnosis.

Carolyn’s grades got very bad as she worked on managing her new disease: dealing with the intermittent relapses and frequent migraines made it hard to take tests, go to class, and keep up with homework. She filed for disability at Wesleyan and was approved to ask for extensions, reschedule exams, and miss classes in the event of a relapse, and to use a laptop in class, as muscle fatigue and numbness could affect her fingers and make it difficult to write. Despite this, keeping up was hard. Some professors were less permissive than others. Some did not voice disapproval outright, but made Carolyn feel uncomfortable about asking for her exceptions, even after they knew her situation. Though at times she wanted to drop out, Carolyn’s parents were adamant that she stay in school and do the best that she could, convinced that it was of the utmost importance that she get a college degree. Faced with their daughter’s illness, they found themselves constantly worried about her future, and realized that the best way to deal with whatever lay in store was to equip her with as many tools as possible.

While her parents preached the importance of education, Carolyn started regularly visiting a nearby neurologist. This doctor quickly proved to be an incredibly pessimistic presence in her life. She told Carolyn that she was not going to finish college. She told Carolyn that she would not have a normal life. She told Carolyn that she was going to be in a wheelchair, that she could never live in a small town, never go to the beach or take a hot shower.
But Carolyn, fresh from her similar negative experience with Wesleyan, was prepared to stick up for herself. Uninterested in paying a medical professional to tell her about all the things she could not do, she got a new doctor, a neurologist based out of Johns Hopkins in Baltimore, Maryland. Although the commute is difficult, he is an expert on multiple sclerosis, and Carolyn likes him much better.

What she did not like was the medication he put her on. Alas, while the depressed patient has dozens of drugs to choose from (Drugs.com 2014), the MS patient has but ten (National Multiple Sclerosis Society n.d.). Four of these drugs are interferon betas, a class of drugs that a recent study verified was ineffective at slowing the progression of MS (Shirani, et al. 2012). Another one of the drugs, Tysabri, a once a month infusion medication, was not recommended for people who had previously been exposed to John Cunningham’s (JC) virus, a common virus that can cross the blood-brain barrier and infect oligodendrocytes (Elphick, et al. 2004). Taking Tysabri while infected with JCV increases an individual’s likelihood of developing progressive multifocal leukoencephalopathy (PML), another demyelinating disease (like MS) (National Multiple Sclerosis Society n.d.). Studies suggest that 70-90% of humans are infected with this virus (Agostini, et al. 1997), and anyone who is not can easily become infected during the course of their MS treatment. Because of this, Tysabri could become significantly more risky almost instantly without anyone knowing it. For this reason, Carolyn refused to take it. Gilenya, another one of the drugs, was appealing due to its great success in clinical trials and because it is an easy, once-a-day pill, but it causes a significant number of problematic side effects, including liver problems, increased risk of infection, and heart failure. In fact, death had become such a common occurrence for new patients that it is now required for someone starting Gilenya to spend six hours after taking the first pill.
under a doctor’s surveillance and attached to an EKG in case their heart stops (National Multiple Sclerosis Society n.d.). Carolyn’s doctor refused to prescribe it. Another drug, Novantrone, was so cardiotoxic that it could only be taken for a maximum of three years, and patients had to have their heart tested prior to each dose. Furthermore, it was a known risk factor for leukemia (National Multiple Sclerosis Society n.d.). Because of this, Carolyn refused to take it. Aubagio, another oral medication, had only a few main side effects: notably, severe liver damage, renal failure, and severe skin rashes (National Multiple Sclerosis Society n.d.). It goes without saying that Carolyn refused to take this one as well.

This left her with only one drug to choose.¹ That drug was Copaxone, a medication suspected to work by blocking myelin-damaging immune cells, though the mechanism is not entirely understood. The drug took the form of a once daily injectable,² delivered to the subcutaneous layer of the skin, directly above the muscles and below the dermis. It hurt intensely, especially if injected incorrectly, which would often happen due to Carolyn’s lack of medical training. She injected herself every night before bed, and would spend the entire day dreading it. Sometimes she forgot about the nightly ritual, and would live a few hours in ignorant bliss before the thought would come crashing back sometime at dinner or in the early evening, and her good day would turn sour in an instant. The injections had to be rotated from day to day, moving from her legs, to her arms, to her hips, before making their way back to her legs again. The hip injections were the hardest: Unable to do them herself, she had to call upon a friend to do them for her, an intrusion and necessity that made her feel vulnerable and never ceased to bring the

¹The tenth drug on the list was not yet approved for use in the United States at the time Carolyn was beginning treatment.
² Recently, Copaxone was approved as a larger dose that only has to be taken every other day.
reality of the disorder crashing home. Her boyfriend, Matthew, did the hip injections for her. Then they broke up, but because he knew how to do it and she trusted him, he continued to help her with the hip injections: an awkward yet unavoidable situation.

To make matters worse, she did not feel like the drug was actually working. So Carolyn started skipping doses, putting off the pain and awkwardness as much as possible. It was during this time that Tecfidera, a drug originally used in Germany for the treatment of psoriasis, was approved by the FDA as a treatment for MS. Almost immediately, Carolyn’s doctor switched her to this twice-a-day capsule. It was by far superior to other MS treatments – not only was it easy to take, but unlike the other oral therapies, none of its side effects warned of possible death. Even better, the treatment actually worked for her. She stopped having relapses, and has not had one since starting the drug.

Tecfidera, like all MS medication, is not a cure for her disease. Instead, it simply slows the continual progression of damage. MS is not considered a fatal illness, but the risk of debilitation has focused research efforts on developing more treatment options as well as finding a cure.

Although Carolyn appreciated her new routine of twice-a-day pill instead of once-a-day injection, she still had difficulties with disease management. The most troublesome were the migraines. To cope with the frequent debilitating head pain, she was on four different medications. Topamax, which her doctor said should prevent the migraines, she took despite its seeming ineffectiveness. Aleve and Fioricet relieved the symptoms of inevitable migraines and were excellent at dulling pain, but did little to fully eliminate it. The best weapon in her arsenal was Zomig, a super-powered migraine crusher, but she was only given 18 of these per month. So while most 20-odd-year-olds learn the lessons of budgeting money, Carolyn worked at budgeting pain.
She looks out the window of my room, somewhere, a car has honked. It is getting dark. We have been talking for about forty minutes now, and still there is much to say.

I ask Carolyn if drinking had anything to do with the symptoms finally presenting themselves, as she had initially thought her MS was a bad hangover. She smiles at me impishly, like a wise grandmother refusing to be tricked by her rowdy grandchildren.

“For a while,” she says, “I was worried that I gave myself MS by drinking so much. But no, it was a coincidence.”

Although drinking is not a known risk factor for MS, the potential causative influences are various and still under debate. However, like many disorders of complex origin, multiple sclerosis is known to have both genetic and environmental risk factors (Ramagopalan, et al. 2010; Hedström, et al. 2010).

The first hint that the risk for multiple sclerosis was somehow genetically related came from observations of familial clustering - that is, people who have relatives with MS are at a higher risk for getting the disease themselves (The International Multiple Sclerosis Genetics Consortium & The Wellcome Trust Case Control Consortium 2 2011).

Some researchers proceed cautiously with this type of indirect data since increased family incidence does not necessarily correspond to genetic factors, as closely-related family members are more likely to share the same environment as well (Noseworthy 1999). However, there are certain populations that, as a whole, demonstrate a decreased prevalence of MS. African American men, for example, are 40% less likely to get MS than white men, and the disease is “virtually non-existent” in people of Chinese or Filipino heritage. This effect, says researchers, is “almost certainly genetically
determined” (Ramagopalan, et al. 2010, 728). Population-specific risk data is particularly interesting for groups that reside in temperate zones, as the incidence and prevalence of MS increases the further one moves from the equator. This persistent statistic may stem from another genetic cause – the fact that people of northern European origin are at the most risk for MS, and most of these individuals live far from the equator (Noseworthy 1999, 40, 41). Conversely, this equator-related risk factor could also come from an environmental cause: vitamin D deficiency, which is less likely to happen if you live in the sunny tropics (Ascherio, Munger and Simon 2010; Ramagopalan, et al. 2010). There have been a number of studies looking into the effects of vitamin D deficiency on MS risk, and the “epidemiological evidence of an increased MS risk among individuals with low vitamin D concentrations has achieved substantial strength” (Ascherio, Munger and Simon 2010).

However, genetics cannot be ignored. In fact, “the development of MS must start in individuals who are genetically susceptible” (Ramagopalan, et al. 2010, 727). According to one report, there are over 60 known risk loci (gene areas that correspond to an increased chance of getting MS), but the relative causative influence of these various loci remains unknown (The International Multiple Sclerosis Genetics Consortium & The Wellcome Trust Case Control Consortium 2 2011). Because there is no known genetic allele that will lead to MS 100% of the time, the current understanding is that multiple genes, interacting with each other, increase disease susceptibility (Noseworthy 1999). Genetically susceptible individuals, however, will not contract MS unless exposed to specific environmental factors (Ramagopalan, et al. 2010).

Smoking is a known risk factor, shown to increase disease susceptibility of genetically at-risk individuals (Hedström, et al. 2010). Other risk factors include vitamin
D deficiency, as noted earlier, and exposure to the Epstein-Barr virus (EBV), which many researchers believe is necessary to trigger the disease (Gilden 2005). EBV is a widespread virus from the herpes family, more commonly known for causing infectious mononucleosis, or mono. Markers for this virus are found in >99% of MS patients, compared with “approximately 94% in age-matched controls” (Ramagopalan, et al. 2010). To summarize, the current understanding of MS is as follows: “The risk of developing multiple sclerosis…is determined by a combination of genetic and environmental factors. The latter include Epstein-Barr virus (EBV) infection, cigarette smoking, and inadequate serum concentrations of vitamin D” (Ascherio, Munger and Simon 2010, 599).

Carolyn contracted mono when she was sixteen, and upon diagnosis with her MS, it was discovered that her body, though exposed to vitamin D on a daily basis, was unable to absorb it adequately. Though she has never had her genome sequenced, she more than likely has any number of the genetic alleles necessary to predispose for the disorder. As Carolyn says, she really “hit the jackpot.”

By the time she turned 18, the symptoms of MS had already set in, but they were so unassuming that it was hard to realize that anything was wrong. The summer before her freshman year, Carolyn went to the beach with her family for a week. She was exhausted the whole time, likely (though she did not know it at the time) due to the heat, and how sensitive MS is to temperature. After sleeping for most of the vacation and even getting in trouble for not spending time with her family, Carolyn chalked it up to “being really tired.”

During the whole first semester of college she was still really tired, and her sleep patterns changed because of that. She also became much more sensitive to alcohol.

“I used to drink a lot - shots were no issue - and then I couldn’t drink as much.
Two drinks and I was good to go. I felt sicker after drinking and hangovers came more easily. My body changed, like puberty, part II: the MS Edition. Kidding. That was a terrible joke.”

Carolyn looks down, smiling sheepishly. She is a comedian, a member of two comedy groups on campus, one of them a stand-up group. I ask her if she has ever thought about doing a stand-up routine about MS.

“Well, I feel like whenever I try to make a joke about MS, people are just like ‘are you OK?’ That’s why I haven’t done stand-up about it…I think it would be really cool if I could, I’d put together some really kick-ass jokes about it, but I don’t know… It would be amazing, it would be the best stand up ever, I would do crazy disease stand-up, but I am terrified of it. I feel like people would not laugh and people would not know how to react and it would seem too confessional, or like I was trying to make something funny that cannot be.”

Carolyn has faced a lot of misunderstanding due to her disorder. Disease management seemed to come unavoidably packaged with misperception management. She dealt with this misperception on almost every side of her life. Family members were insensitive: her aunt offered, upon her diagnosis, to make her house wheelchair accessible, which Carolyn interpreted as a pessimistic prediction of the worst. Other doctors she saw, physicians not related to her MS treatment, were incredulous about her diagnosis. They would ask questions like, “Are you sure you have MS? Who diagnosed you?”

But by far the worst discrimination came from complete strangers:

Today I was walking up to my class in PAC and I prefer to take the elevator because I do not like the stairs. Stairs are very difficult for me. It doesn’t look like stairs are difficult for me, but they are… sometimes. And someone broke the elevator in PAC, right before I got there, and I was walking up the stairs behind the person who broke the elevator in PAC and he was joking about it like, ‘Haha,
I’m an asshole,’ and his friend was like, ‘Well, it’s a good thing that no one with a disability has class today.’

And I was right behind them! And suddenly I felt invisible. I should have felt empowered that I didn’t look disabled, but instead I feel utterly erased. Like my problem is not an issue because you can’t see it and because I’m walking up the stairs and I’m trying not to look like I’m dying slowly, and I’m walking and doing it, and it’s like I have erased my own problem by putting a brave face on it and by allowing it be invisible.

(Lewis 2014)

All of this misinterpretation, stigma, and lack of sensitivity has made Carolyn wary about sharing her experiences. On the one hand, she feels that if she talks about her disorder, people will treat her differently because they do not understand it. On the other hand, the only way to get people to understand it is to talk about it.

“I’ve started hiding things more than I want to, not because I don’t trust people but because I feel like it hasn’t worked out well in the past. It’s not that I don’t trust specific people - I trust you - I trust that I can give you this information and you won’t treat me differently. But I’m also giving you all this information and with some people I don’t have that…they don’t have the time. And I don’t know if I want to take the time to talk about this for however long we’ve been talking about this, and explain it all and for them to have this deep understanding about what my life is, and then have them treat me however they would treat me because of it…”

What multiple sclerosis means, quite literally, is “many scars.” In the disease pathology, these scars develop at the location of lesions. But it seems that with any disease that progresses invisibly and is suffered in silence, “many scars” may take on a different significance.

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Last year, Carolyn’s doctor reclassified her MS from “mild” to “aggressive”
relapsing-remitting multiple sclerosis. Though the new categorization was not the brightest of news, Carolyn is grateful that her disease is still in the relapsing-remitting stage, and has not turned into progressive MS.

The relapsing-remitting form of the disorder is characterized by the occasional attack of a relapse followed by a period of relative normalcy during which the disease does not worsen (Fox 2014). In the progressive manifestation of MS there are no relapses, but there is a steady progression of disability that does not respond to treatment. Relapsing-remitting can become progressive MS at any point after diagnosis (Lublin and Reingold 1996). This switch is called secondary progressive MS, and it is essentially a more drastic form of relapsing-remitting MS with less recovery time (Fox 2014).

![Diagrams](image)

**Figure 3:** The different types of Multiple Sclerosis. Relapsing-remitting MS is the most common form the disorder (upper left corner) as the initial diagnosis for 85% of patients with MS. Primary progressive MS is more common in patients who are older at the time of diagnosis (lower left), and characterized by continued advancement of disability. Relapsing-remitting can become secondary progressive MS (upper right), which is characterized by a varied rate of disability marked by an occasional relapse. Progressive-relapsing MS (lower left) is the least common form of MS, effecting only 5% of people with MS (Fox 2014).

The locations of the lesions are also an important indicator of how well things will turn out. According to her doctor, Carolyn has a lesion delicately teetering at a “just made it” location: That is, if the lesion were in a slightly different place, she could already be paralyzed.
“Well, that’s good,” I say, because she sounds troubled.

“Yeah, but what if it happens next time? It could always happen next time.”

What she worries about the most is having children. Having children means going off medication. Medication has to stop not only during actual pregnancy, but also during the time spent trying to get pregnant. During that time, a relapse or worsening of condition is very possible. However, “in women with multiple sclerosis, the rate of relapse declines during pregnancy, especially in the third trimester” (Confavreux, et al. 1998, 285).

“Pregnancy,” Carolyn muses, “is this cool, blissful time when you are MS free.” But as soon as the baby has been delivered, the body snaps back to reality, and the risk of having a relapse “increases during the first three months post partum before returning to the prepregnancy rate” (Confavreux, et al. 1998, 285). The terror in this, without a doubt, is the possibility of having a child and being instantly non-functional – being paralyzed, or dizzy and vomiting for weeks.

The truth is, Carolyn has no idea what the future holds for her. Some people with MS have a few relapses and then... nothing. Some people do not have any relapses for twenty years, go off their medication for one second, and then suddenly, out of the blue, they have - in MS jargon - “the big one.” Paralysis. Wheelchair. Carolyn has not had a relapse in over a year. Should she breathe easy? Or should she wait?

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Carolyn has two tattoos. After dealing with doctors and needles that she did not choose (needles for blood tests, for the spinal tap, for IVs, and for the daily injections), she wanted the tattoos to signify needles that she chose. “Might as well have something
happen to my body that I chose,” she tells me.

The first tattoo says “vivir” (to live) in a thin scrawl behind her ear. The second, across the left side of her back, is a small verse from one of her favorite poems, Rimas by Gustavo Adolfo Béquer. It reads simply “cadencias que el aire dilata en las sombras,” which translates to “verses that the air prolongs in the shadows.” She was reading the poem in a class during the time she was getting diagnosed, and the beautiful writing stuck with her. It is the sort of writing that she wants to emulate with her own work. Carolyn has won literary prizes for her writing, and hopes to be published some day soon.

After our interview, she shared some of her work with me, and it made me cry, and laugh, and think about the nature of things. But what I thought about most, after we had talked, and after I had re-listened to our talk a dozen times, was the things that made her sad. Though I can imagine she has spent plenty of time crying or panicking or being angry about her diagnosis (and her writing suggested all of these things), during our interview her disease took on relatively little of that emotional burden. Yes, she worries about the future, and some of the outcomes scare her. But from what I saw, the sadness – the melancholy - was reserved for something different: misconception.

The Carolyn I interviewed was dealing with MS, but fighting a different battle. Every day she makes decisions based on what others might do or say or think. She is almost forced into medical leave for having a diagnosed disease; she stays silent about daily struggles to avoid perceptions of incapability; she feels erased because her disability is not obvious; her voice catches because no one believes her; she averts her gaze because a wheelchair is the smoothest path toward understanding.

This is the burden of the broken brain. No one questions the broken ankle, wrist, or nose. Even the broken heart is understood. But the brain, broken, with many scars, is
outside our powers of comprehension. The irony of this, an irony that should not be missed for the remaining pages of this thesis, is that only the brain can achieve such comprehension. But that same brain, capable as it may be of imagining the pain of a broken bone, torn muscle, or wounded skin, is impossibly lost when it comes to envisioning the pain of its own wounds.
References

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Lewis, Carolyn, interview by Taylor Goodstein. *My Brain is a Mess* (February 2014).


The Precipice

Precipice, from the French précipice meaning a “steep place, danger, or disaster” and from the Latin praecipitium meaning a “fall from a great height.”

From 1606-1678, it meant a “headlong fall or descent” (Oxford University Press 2014).

Laura S. Godson liked school; she had always liked school, until abruptly she hated it. She was in 10th grade when the change happened, when the love turned to hate, and it came as a surprise to both her and her family.

She did not know why she suddenly hated school. Her mother thought that she may have been stressed by the three AP courses that she was taking, but Laura was not convinced. After all, had not she always had a heavy course load? This much was certain: She hated it, she did not know why, and it upset her.

Every day, she went to school with a “very overwhelmed feeling,” just on the verge of tears, all the time, without any idea why. She would go out for a night with friends, have a great time, and then come home and cry for hours, still utterly at a loss for a source of her sadness. She was trapped inside cyclical thoughts: I’m upset; I do not know why, that makes me more upset. I’m upset; I do not know why…

Fortunately, Laura’s family was familiar with the importance of mental health. Her father is a psychiatrist, her brother and mother suffer from ADHD, and both sides of her family have a history of depression. Discussing mental health was an everyday topic for them. So instead of suffering in silent agony while her family scratched their heads, Laura’s feelings were acknowledged soon after they appeared. It was her father who reached out to her. “If there is anything you need to talk about, you can tell me,” he suggested.
Laura knew she needed help, and so she started seeing a psychiatrist. “It feels so good to be validated,” she says, “and it’s really important for people who have some sort of mental [illness].”

After spending time talking to the psychiatrist, Laura was diagnosed with generalized anxiety disorder.

Generalized anxiety disorder is defined in the DSM-IV-TR\(^1\) as excessive anxiety and worry that occurs “more-days-than-not” for at least six months, about any number of activities or events. The person must find it difficult to control the worry. This general anxiety is associated with other symptoms, such as feeling on edge, being easily fatigued, having difficulty concentrating, or being irritable. Finally, the symptoms of this disorder, in order to count as generalized anxiety, need to significantly impair functions of normal life (American Psychiatric Association 2000).

\(^1\) Diagnostic and Statistical Manual of Mental Disorders, 4\(^{th}\) Edition, Text Revision
For Laura, generalized anxiety disorder mostly manifested as uncontrollable, racing thoughts. She was confused, prone to depressed feelings, and constantly oscillating between “get up, you are fine” and “no, I am not.” She suffered from obsessions and the occasional panic attack. Panic attacks have been shown to further exaggerate generalized anxiety disorder, as apprehension about panic relapses causes “chronic anticipatory anxiety,” also known as interpanic anxiety (Klein 2002).

“I know I have a predisposition to anxiety,” Laura says, “and I obsess over things. Like, a lot my mental space can easily be taken up by something - and then anything else that happens is the straw that breaks the camel’s back.”

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Although modern anxiety disorders push people toward the breaking point, anxiety, in fact, developed as an evolutionary advantage. The ability to be anxious or stressed was a beneficial trait for species that were subject to a variety of natural and social dangers (Klein 2002). In other words, the gazelle constantly checking its surroundings - on its toes and ready for a quick getaway - is more likely to survive than its relaxed, lethargic conspecific. In the days before modern civilization, being anxious meant being ready. This readiness is achieved by an assortment of chemical and hormonal mechanisms called the “stress pathway,” which culminates in activating of the “fight or flight” response. In its normal state, fight or flight is activated by an external warning stimulus. This can be a loud sound, a rank smell, or visual confirmation of the danger.

Pretend you are a newly-evolved homo sapiens, searching for food in a forested environment. Suddenly, you spot a jaguar crouched in the bushes just a few
feet away, ready to pounce. There is no time to consciously process the danger for the necessary reaction, so most of the initial fight or flight happens at a subconscious level. The visual information is sent immediately to a subcortical structure in the brain called the amygdala, the hub for emotion, anxiety, and fight or flight (Sapolsky 2003; Pappas 2009). The amygdala quickly shoots hormone messengers to the brain stem and down to the adrenal glands above the kidneys, which immediately start pumping epinephrine (adrenaline) en masse throughout the blood stream. A body flooded with epinephrine is ready to bolt or battle: the heart races; pupils dilate to let in as much visual information as possible; blood flow moves toward the legs to enhance running; the palms become sweaty, ready to grasp a weapon; and all digestion stops (Bear, Connors and Paradiso 2007). But behind this initial burst of life-saving adrenaline is another hormone, activated at the same time but working at a much slower pace, called cortisol (Sapolsky 2003). Cortisol performs many of the same functions as adrenaline - it recruits energy for muscles, increases heart rate, and turns off nonessential functions - but more gradually, as a mechanism for maintaining fight or flight after the initial panic has gone away.

Cortisol is also capable of a feat beyond the abilities of adrenaline: it can reactivate the stress pathway in the brain without an initial trigger. Because of this, in cases of chronic anxiety like Post-Traumatic Stress Disorder (PTSD), high levels of cortisol are usually found in the sufferer’s bloodstream. This represents a deleterious positive feedback mechanism: as anxiety increases, the adrenal gland is signaled to produce more cortisol, that cortisol then reactivates stress pathways, and anxiety increases further (Sapolsky 2003).
For primates, however, regulating this alarm can be tricky, as an abstract thought or apprehension about a specific fear can be enough to trigger the full effect of the stress pathway. So, although anxiety is a necessary and vital response for staying alive, humans are capable of habitually *expecting* danger, which is the first step on the pathway to neurosis (Sapolsky 2003).

For therapeutic purposes, diagnosing someone with an anxiety disorder is only one step toward a solution. It is also necessary to determine where the anxiety originates. Because most fear processing happens at the subconscious level, the override signal needs to come from a strong conscious effort (Foa and Kozak 1986), which is impossible without knowing what environmental trigger sets off the subconscious cascade. Only after realizing what causes such mental stress can steps be taken to try and manage the disorder.² Laura’s anxiety was apparently likely instigated by her relationship with her father.

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Laura’s father had always been a difficult man to deal with, particularly because, to others, nothing seemed to be wrong. But trying to handle his personality, his quirks, and his actions for 17 years had left Laura so emotionally ‘wound-up’ that it started to spread into all aspects of her life.

“His situation is that he is brilliant but very socially awkward. You wouldn’t notice it unless you are really close to him, but there are some social situations where you are just… talking two different languages and you just can’t get him to… get it.”

² “Face your fear” phobia rehabilitation is based on this principle. The idea is that exposure to a fearful stimulus without consequence will overwrite the pre-existing anxiety (Foa and Kozak 1986). The first step to this type of rehabilitation is to recognize the fear itself so such steps can be taken.
Laura’s father has dealt with lifelong depression, but Laura thinks he might have another, undiagnosed mental disease, perhaps a high-functioning form of Asperger’s syndrome or social anxiety. Because such a condition existed without ever being acknowledged, all of the feelings that Laura had about her father were confusing. For years, she felt angry and frustrated with him, but she never understood why. They would fight constantly. Over time, she even had a hard time being in the same room with him. All of this culminated and eventually led to her disorder.

When Laura realized where her anxiety came from, she was relieved. She worked on her relationship with her dad, and was able to reach an understanding with him that helped alleviate her symptoms. At first, this was very difficult, and she has no memory of some initial conversations that she had with her father (another side effect of cortisol on a rampage) (Sapolsky 2003). For the rest of high school, she continued to work with her psychiatrist, and for that time, her apprehension was under control without the need for medication. Laura was especially excited to leave for college, a time when, able to maintain her relationship with her father on her own terms, she expected to have reduced anxiety.

She had an uneventful freshman year, but trouble began again when she was a sophomore. Laura began suffering from the symptoms she faced in high school. She became upset without knowing what was wrong; she felt stuck in a chaotic life over which she had no control.

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3 This avoidance behavior is typical in cases of learned fear. As anxiety will decrease when not in the presence of the fear stimulus, “escape behavior is reinforced by successful decrease in anxiety” (Klein 2002).
“So I developed an eating disorder. The eating disorder was a purging disorder - not anorexia or bulimia, because [I was] eating normal amounts of food and then throwing it up. I don’t even remember the first time I threw up because I was in such a fog at the time.”

The eating disorder felt new, but not completely surprising. Laura was overweight as a kid, and because of that, she had many body image issues. It did not help that her father, with his strange social ineptitudes, was particularly opinionated about food. Throughout her life, he made comments about what she was eating, what she should be eating, and what he would never eat. He never seemed to realize that these comments negatively affected Laura’s body perception, and led her to have a hypersensitive relationship with food. This, combined with her brain’s tendency to obsess over her worries, generated the purging disorder.

Eating disorders are known to be among the more deadly psychiatric disorders. Anorexia nervosa, in fact, is considered to be the most deadly mental illness (Arcelus, et al. 2011). This is particularly disturbing since eating disorders tend to affect younger populations, and are, in particular, significant causes of mortality in young women (NEDA n.d.).

The internal consequences of purging are various, and over time, can be deadly. Constant vomiting makes the body dehydrated and leads to an imbalance of electrolytes – soluble salts necessary for important body functions – which, in turn, can cause an irregular heartbeat. Eventually, electrolyte imbalance can contribute to heart failure. But purging can also cause more immediate problems: inflammation and
rupture of the esophagus from frequent vomiting, tooth decay from stomach acid, and development of peptic ulcers due to increased acid production (NEDA n.d.).

This eating disorder pushed Laura’s life into a strange, vacillating state, as her purging and anxiety had both a positive and negative effect on each other. For one thing, her anxiety tended to manifest as obsessions over stressful situations. This obsessive thinking perpetuated the eating disorder. Whenever she ate something, she would think about that thing for hours and hours afterwards, thinking so much she often made herself sick to her stomach. She worried about the future, constantly plagued by thoughts of “If I don’t throw up now, I’ll feel terrible later.” Her eating disorder was the source of her control, and therefore, her source of happiness, but it was also a huge stressor because she knew it was a bad coping mechanism. The eating disorder would stress her out, the stress would worsen her anxiety, her life would feel out of control, and she would regain control by continuing the eating disorder.

Laura’s later-stage anxiety is plagued by the characteristics of obsessive-compulsive disorder, which highlights how interconnected anxiety disorders can be. Her generalized anxiety was marked by anxiety-provoking obsessions and anxiety-reducing compulsions (Klein 2002). In fact, it is likely that this obsessive-compulsive behavior is what fueled Laura’s eating disorder in the first place, as controlling her food intake helped reduce the anxiety produced by her stressed, obsessing mind. Prior to the eating disorder, her generalized anxiety manifested itself as obsessions, pervasive apprehension, and being on edge, but demonstrated little in the way of relieving compulsions.
In order to maintain the highly delicate balance between her anxious obsessions and purging compulsions, Laura began hyper-organizing her day and school work.

“I was highly efficient and got all my work done and had everything planned out and scheduled - what am I going to do today, what am I going to eat today - But I was also very emotionally raw, and physically very fatigued and dehydrated.”

For the most part, the obsessive scheduling worked, and Laura was able to balance both her anxiety and purging disorder without being pushed over the edge. However, life is unpredictable, and occasionally Laura’s routine would be disrupted.

For example, she sprained an ankle during this time, which was terrible because she could not exercise, and exercise had become an important part of her daily routine.

“If something came along that broke up my routine, I would just cry and hyperventilate and that was it…”

One challenging aspect of the anxiety, the eating disorder, and the obsessive organization was their ability to generate only extremes of emotion. She was either perfectly happy because everything was going smoothly or desperately panicked because something had been interrupted.

“I would have a great day where I got everything done and felt really good… but then one day I slept through a quiz on accident and I had a breakdown. It’s such tenuous control. You feel like you are on top of everything, but it is just a precipice.”
Despite this, the thing that Laura found the strangest was how easily she could get away with these habits. Afraid of being stigmatized, she kept her purging a secret from everyone in her life, a feat that was easier than she expected.

“The craziest thing is the extent to which it didn’t really affect me. I went to all my classes, but I have thrown up in every bathroom on campus. Like, I would go to class, go to lunch, go to the bathroom, throw up, go back to lunch… it became a very routinized thing and no one knew about it.”

This continued for months. In order to explain her weight loss, she gave up cheese and potatoes for Lent. She ended a bad relationship, which was good, but it allowed her to become even more controlled during her day. Her life likely would have continued in the same way – highly organized routines with a loss of control always bubbling near the surface – if Laura had not gone home for spring break.

On the second to last day of spring break, she was running on the treadmill at home, and suddenly, she had a panic attack.

I got off the treadmill before it hit me. I could feel it coming on… I think realizing that this was my chance to tell someone about [my eating disorder] is what brought it on. This was my chance to say something, and it’s really hard to say something, because you don’t want to - it’s your source of control and happiness, but I subconsciously knew that I had to tell my parents - it was the second to last day of spring break - and that’s what preceded the panic attack and then the panic attack led to the conscious realization that I should tell them about the eating disorder.

(Godson 2013)

I asked Laura to explain what a panic attack was like:

“The panic attack has hyperventilation, and a mental fog, in that you aren’t having thoughts…not thinking, almost…. It’s like…um… a paralyzing... it feels like it’s not going to end because it’s like… your whole body… and, like… I couldn’t
string a sentence together while it was happening…and I couldn’t have any sort of logical thoughts.”

Her mom came home and found Laura in this state. The confession spilled out: “I haven’t been eating, I’ve been throwing up…” Laura told her mom everything.

The next day, she visited her psychiatrist, and was put on once-daily Lexapro, a selective serotonin reuptake inhibitor (SSRI) used specifically to treat anxiety disorder (Cerner Multum, Inc. 2013).

SSRIs are some of the most widely prescribed drugs in the United States, and provide effective therapeutic treatment for a wide variety of disorders, including depression, obsessive-compulsive disorder, panic disorder, and bulimia (Stahl 1998). They work by selectively inhibiting transporter proteins that reuptake serotonin into the presynaptic neuron (Nutt, et al. 1999). This effectively increases the amount of serotonin that remains in the synapse. More serotonin in the synapse means more activation of serotonin receptors, which exaggerates the normal function of serotonin in the human brain.

Figure 2: This simplified diagram illustrates the effect of an SSRI on a serotonin reuptake transporter. By blocking the reuptake of serotonin into the neuron, more serotonin remains in the synapse, and consequently, more serotonin receptors are activated.
Serotonin is an important neurotransmitter with a diverse number of functions. It “regulates behaviors such as mood, anxiety, sleep, sexual behavior, and eating behavior, as well as such functions as body temperature, appetite, gastrointestinal motility, and movements” (Stahl 1998). Because serotonin plays such an important role in mood, anxiety, sleep, and eating behavior, it is thought that in disorders such as depression, anxiety, and eating disorders, the amount of serotonin in the brain is reduced (Nutt, et al. 1999). By blocking the natural reuptake of serotonin into cells, more serotonin remains in the synapse, therefore compensating for the reduced total amount of serotonin.

After starting Lexapro, and getting over the initial side effects (mostly dry mouth and dizziness), Laura began to feel much better. “I know some people don’t like the thought of being medicated. For me, the medication was like… thank God!”

Laura is still on her medication, and planning to stay on it for the foreseeable future. She knows how tough college can be, and does not want to risk ending up in a dangerous place again like she was during her sophomore year. “I do think I have predispositions towards anxiety and body image stuff,” she says, and because of that, she knows she should be careful.

Laura’s story illustrates a dark period in her life that was relatively short and handled well by her family. So many other people who suffer from similar illnesses do not have the ability to admit it to themselves or their loved ones, and many who do do not receive the encouragement and help that Laura did.
“It’s really easy with some psychiatric illnesses to just pass them off and say, ‘man up,’ so I am really fortunate that my family is so in tune with ‘psych stuff’. It was relieving that [my dad] perceived it as a problem, too.”

Laura gives advice to many people who feel like they may be suffering from a mental illness: “It’s not taboo, it’s not weird - talk to someone. You hear it all the time, but you have to tell people that it’s not something you can just get over. That’s another feeling - that it’s never going to end - and I had lots of suicidal thoughts because I thought that I was never going to get over it.”

One of the biggest struggles with mental illness is the lack of physically observable symptoms. While someone with an eating disorder may experience weight loss, and someone with anxiety may have acute panic attacks, the basis of the disease is in the mind, and therefore invisible.

For years, it has been known that many mental illnesses, especially anxiety and eating disorders, are strongly influenced by external factors. Anxiety can arise from a specific environmental stressor, as in the case of PTSD, and eating disorders tend to predominantly affect young women dissatisfied with their bodies due to the portrayal of “the ideal body” in popular media⁴ (National Association of Anorexia Nervosa and Associated Disorders 2014). For years, the general opinion about people affected by these types of illnesses was that they were simply “too sensitive,” and their symptoms were abnormal and extreme responses to normal life experiences.

Knowing this, it took science a long time to begin investigating potential brain differences found in people with these disorders. After all, the conditions seemed so

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⁴ A body type that is found in only 5% of American women (National Association of Anorexia Nervosa and Associated Disorders 2014).
“mental,” so how could they possibly have physical manifestations in the brain? The answers may help understand why some people develop eating/anxiety disorders and others do not.

For example, when I was younger, I was exposed to the same media-driven “ideal body image” that Laura was. I even wanted, at times, to be anorexic or bulimic because I did not fit this ideal. However, I was never able to follow through with it. Try as I might, after a few hours I would eat food again, and no matter how much I wanted to, I could not get myself to throw up. My conscious desires were never able to overcome the subconscious mechanisms of satiating hunger and absorbing nutrients. So what is different between my brain and Laura’s? Did she have a structural or chemical difference that allowed her to control her eating in a way I never could?

Furthermore, I have been stressed about school and life, and my family situation is not perfect, yet I have never developed an anxiety disorder. Obviously, there have been times in my life when my cortisol levels ran high, yet this has never captured me in a never-ending stress loop. What makes one person develop severe anxiety and another person avoid it? Are there brain differences that make people more susceptible to cortisol?

Recently, research has finally started to answer these questions by looking inside the brain in comparison studies between ‘normal’ and ‘ill’ individuals.

A study conducted at the Stanford University School of Medicine found that neuronal connections from the amygdala – the aforementioned fight or flight hub - to other brain regions were disrupted in the brains of patients with generalized anxiety
disorder (Pappas 2009). It was found that the brains of patients with generalized anxiety had amygdalae that were less connected to the region of the brain responsible for determining the importance of stimuli (Pappas 2009). This suggests that the brains of people with generalized anxiety have a more difficult time distinguishing between rightly distressing circumstances and minor irritations, which is why they are prone to a general increase in worry.

Another study added further significance to brain differences by examining a different brain region, the ventral prefrontal cortex (vPFC), which is implicated in suppressing and regulating fear. In patients with a high level of anxiety, it was demonstrated that their fear-regulating vPFC was less active and only weakly sustained in response to a fear-generating situation as compared to patients with a low amount of anxiety, who demonstrated a highly active and strongly sustained vPFC response (Indovina et al. 2011). This suggests that people with anxiety disorders are affected more intensely by stressful or frightening situations than typical individuals.

For eating disorders, accumulating evidence supports similar physical bases for disease etiology. In a study examining anorexic, obese, and average individuals, it was found that anorexic patients had decreased responses from reward areas of the brain when looking at pictures of food. This could mean that those individuals are less likely to find eating pleasurable, and therefore have an easier time prolonging fasting periods (Nauert 2012).

Another brain area implicated in eating disorders is the primary gustatory cortex, the region responsible for taste processing and the reward associated with certain flavors. In a recent study, healthy women and women recovering from
different eating disorders were shown to have different responses in this area of the brain to the taste of sugar. Women recovering from anorexia had less activity than the healthy woman, and women recovering from bulimia had more activity than the healthy women. The researchers took this data as evidence that abnormal responses to sugar may predispose people to eating disorders (Firth 2014).

Perhaps more intriguing than differences in brain wiring, scientists have also found differences in the brain size of individuals who suffer from eating disorders. For example, one study demonstrated that girls with anorexia nervosa had a greater amount of orbitofrontal grey matter compared to the control group. This area of brain has been associated with sending signals of “stop eating” when satiated. Researchers think “a larger volume of this brain area may promote eating cessation, even before the individual has eaten enough” (University of Colorado Denver 2013).

However, researchers recognize there are limits to this information. The most important confounding factor is the inability to determine causality of brain differences for mental illnesses. That is, it is not known whether or not these brain disparities precede the development of mental illness or are generated by the presence of that illness (Pappas 2009). If structural and chemical changes arise because of a mental disorder, then we are still very much in the dark as to why some people develop these illnesses and others do not.

However, the fact that physical differences exist in the first place should motivate a revolution in the way mental disorders are viewed by society. Despite better understandings in many other medical fields, mental illness is still perceived as a sign of weakness (Byrne 2000). With such stigma attached, being anxious, unable to
eat, or prone to panic attacks are signs of a fragile mind, and therefore, the associated illnesses carry shameful undertones. But by studying the physical changes that the brain undergoes when afflicted by mental illness, experts provide a new way to conceptualize these disorders. If more people know about variations that exist in the mentally ill brain, then the mental illness becomes concrete, like a physical wound. Perceptions change.

Anxiety and eating disorders carry a particular burden of stigmatization. Being anxious or monitoring your diet are, after all, normal aspects of life. But when the normal mind is at its most stressed, the feeling ultimately goes away. Because of this, professionals and laypeople alike have a tendency to look at anxiety from the perspective of their own experience – as a fleeting, chaotic moment in a sea of calm, as worry controllable by deep breathing, as unease in the pit of your stomach that disappears once the source of the fear has left – instead of interpreting it as the chronic condition that it actually is. Try as she might, Laura could not restrain her worry. She felt overwhelmed, confused, sad, and out of control for most of the day during most days. Her brain’s natural stress response had been hijacked and turned up to a thousand, constantly in escape mode, constantly at full alert. All it takes is a small number of influences – an imagined foe, a cross-wired cortex – and the brain plunges into impairment, psychosis and obsession.

Eating disorders carry the same stigma. After all, we all choose what we eat. It seems so unlikely that food would exert such a pull over someone’s life, driving them toward emotional and physical collapse. But with the correct environmental stimuli –
body dissatisfaction, an ingrained “ideal”– and a brain with the tendency to hyper-control, and suddenly, an eating disorder can take hold.

Laura was fortunate enough to have her symptoms recognized, her feelings validated, and her voice heard. Because of actions she felt comfortable taking, her disorder is under control and she has learned to recognize her own warning signs in case she starts to slip again. Her story demonstrates the ideal scenario that transpires when someone is not burdened by self-stigma and is therefore able to speak up about the difficulties they are having. So many people living with mental illness go untreated and unrecognized, robbed of the normal life they could have without the burden of stigma (Reagan and Zash 2014).

Still others - who may look fine, seem fine, and act fine - are really on a precipice, balancing on the edge between wellness and illness. If the conditions are right, a slight push could make them fall abruptly into dysfunction. What waits at the bottom can be any number of maladies – perpetual anxiety, or continuous fasting, perhaps – that hold a firm grasp and prevent the fallen from climbing back to the top of the cliff. Once we accept the precipice, once we recognize its existence; it becomes easier to hear the cries for help, reach out and grab someone, and help them balance before they fall.
References


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This is Your Brain on Trauma

My mind slips. Thoughts get crossed. Cannot find my words. Major growth on the back of skull on lower left side. Feel really alone. Thinking of other NFL players with brain injuries. Sometimes, simple spelling becomes a chore, and my eyesight goes blurry ... I think something is seriously damaged in my brain, too. I cannot tell you how many times I saw stars in games, but I know there were many times that I would “wake up” well after a game, and we were all at dinner.

On the back: “Please, see that my brain is given to the NFL’s brain bank.”

- Dave Duerson’s suicide note

Part One: Living with Injury

Maggie McLaughlin grew up in a small town in Colorado. She has an older brother and two older sisters, and had she been a boy, she would have been named “Gunther.” Her parents met in college and are still happily married; they live in a single story red-roofed house on the edge of a mountain, the same house they have been living in since 1997. When she was in middle school, Maggie learned that her Scottish ancestors came to America because they were exiled from Scotland for being horse thieves. She learned that fact the same year she became middle school class president.

Maggie was always involved in sports; from an early age she dabbled in soccer, swam competitively, and became an excellent tennis player. In high school, she followed in her sisters’ footsteps and became a cheerleader, where, as a base, she was responsible for throwing and supporting the nimble girls designated as “flyers.” Maggie is very proud of never having dropped a flyer, a feat that she was only able to accomplish by occasionally putting herself in harm’s way.

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1 Dave Duerson (1960-2011) was an NFL football player who played as a safety for the Chicago Bears, New York Giants, and Phoenix Cardinals. His career in the NFL lasted for ten years, from 1983-1993. He committed suicide by shooting himself in the chest in order to preserve his brain (NPR Staff 2013).
Maggie currently attends the University of Colorado at Boulder, where she studies Speech, Language, and Hearing Sciences. At first glance, Maggie seems just like any other student. She belongs to a sorority, Alpha Phi; attends football games decked out in UC Buffalo spirit; and almost every single one of her Facebook photos is of her and a collection of friends, smiling toothy white smiles, peering around perfectly tamed locks, and linked together by arms around thin waistlines.

The first time you notice something different about Maggie might be if you set up a coffee date with her and she sets three reminders on her phone: one twelve hours before, the other two hours before, and finally the last ten minutes before the date. Or, maybe she is coming down the stairs and she trips a little, and you notice eight of her sorority sisters jump and reach toward her head. Or maybe, you and Maggie have decided to study for a class together, and she shows up with handwritten notes, typed notes, notecards, a study guide, and self-made quizzes. Maybe you see this as strange, or maybe you think she is just an over-dedicated student.

But even more likely, as she has gotten in the habit of doing, Maggie might just tell you what is wrong very shortly after you meet her.

“I have a Traumatic Brain Injury due to multiple concussions.”

“How many is multiple?”

“I had ten concussions from 2006-2012. Eleven, if you count a minor head bump from cheerleading, but I don’t normally count that one.”

Maggie’s first concussion came when she was young: In 7th grade she was elbowed in the forehead while playing dodgeball. To this day, she still has the marks of that injury in the form of a blood clot just under the skin of her forehead, a couple of
centimeters below the hairline above her right eye. For the rest of middle school, this clot was well-known and often sought out: When people would ask about it, she would take their fingers and pass it over the top of “the permanent bump” on her forehead, at which point they would pull back in astonishment. Everyone liked the bump, and whenever Maggie played the popular get-to-know-you-game, “Two Truths and a Lie,” one of her truths would always be “I have a permanent bump on my head.” Her closest friends used the bump as fodder for jokes, telling Maggie she was so clumsy that her head stayed perpetually bumped, and Maggie herself would use it as an excuse to get out of doing housework.

But then, Maggie’s concussions started piling up. After sustaining one concussion, her brain was more vulnerable and more likely to sustain another, even from a less serious impact to the head (Collins, et al. 2002; Guskiewicz, et al. 2003). It was as if the floodgates had opened, and over the next three years she had five concussions. Two of these concussions came from cheerleading, where, true to her goal not to drop anyone, she was constantly exposed to falling knees and elbows. Another two concussions came from playing “night games,” a series of tag-like activities played at night, which were hugely popular among her group of friends. She hit her head on a brick wall during the first night game, which resulted in a brief loss of consciousness, followed by a severe headache and vomiting. She went to the emergency room and was given a CT scan, which came back normal. The second time, while hiding under a car in a game of “Sardines,” she turned and hit her forehead against the exhaust pipe.

The fifth concussion came at the end of her sophomore year, and followed very shortly after the exhaust pipe incident. Maggie and her family were rafting with a guide
who turned out to be incompetent, driving the raft directly into a bridge pylon in the middle of the river, on which Maggie, of course, hit her head. She did not lose consciousness, but emerged from the river with two black eyes and a significant, persistent headache. This concussion and the exhaust pipe concussion were close enough together that they amplified each other; almost a month later she was still feeling the effects so severely that, during a tennis tournament, her headache intensified and she began vomiting heavily. At this point she went in for her first neurological evaluation, where it was recommended that she not play sports or exert herself. Her headaches resolved, but not for long (Mangeot 2012).

A few months later, her seventh concussion came from a familiar source: cheerleading. She lost consciousness, as she would each time she sustained a concussion from then on, and her headaches returned with new force, affecting her daily and lasting for several months. For her eighth, almost a year later, she was pulling into the intersection of a highway and got "T-boned" by a car running a red light at 55mph. But this time, a mere concussion was considered lucky, as having survived such an accident at all was a miracle. This concussion resulted in an ambulance trip to the emergency room, and Maggie found herself visiting the emergency room after each of her subsequent concussions.

Her ninth concussion came in April 2011, at the end of her freshman year of college. She was playing Powderpuff Football for a pep rally at her school. Maggie was unconscious for longer than ten minutes, she has no memory of either the injury or the trip to the ER, and her lifetime of head injury finally resulted in serious after-effects (Mangeot 2012).
Unbeknownst to her, Maggie started to suffer from Post-Concussion Syndrome (PCS).

“Anything I am told or have to remember, I have to have it written for me, verbally communicated to me, and then I have to set 2-3 reminders. For everything - studying, dinner dates, events - it all ends up on my phone in the calendar. Most information has to be processed a couple of different ways, up to 5 times.”

Maggie speaks in the present tense, because these memory deficits that she started to suffer from after her ninth concussion are still challenges for her, and likely will be for the rest of her life.

“Immediately following this concussion, I spent 15 hours sleeping in a 24 hour period. I would need eight to ten hours of sleep at night and three to four naps during the day in order to stay functioning.”

Maggie’s major sleep needs started while she was still in school. Her schoolwork suffered greatly during this period.

“Written essays were fine because I could go back and see what I was writing, but anything that was dependent on memory was completely… non-functional. I couldn’t remember or process 90% of the things that were told to me. So exams were impossible. I chalked it up to just taking hard classes, that I couldn’t remember the information because it was hard.”

Her memory problems and sloth-like sleeping habits were not the only symptoms Maggie dealt with. In the months leading up to her tenth concussion, she developed depression and became unable to communicate her feelings. Her grades deteriorated.
“I did really well my first semester in college, and then the spring of my ninth concussion I did really badly, and then the fall of my sophomore year I did even worse, and then in November when I got my tenth concussion, my parents just pulled me out of school.”

Maggie’s tenth concussion was devastating. Her PCS had been slowly developing over the succession of concussions, and all of them were cumulative (Gronwall and Wrightson 1975). Regardless of the force of the blow, her brain suffered more and the effects were worse with each one. The tenth concussion, by itself and with no other concussions to bolster it, was severe. But because it was preceded by an already-developed neurological impairment (PCS), it changed Maggie’s life forever.

She was walking to class one day when it was cold enough to put ice on the streets. Looking both ways, she stepped off the sidewalk to cross the road, slipped on ice, and fell backwards, hitting the back of her head directly on the curb. It is unknown if or for how long she was unconscious, because she does not remember falling. After the injury, she was able to walk to her grandmother’s house, and went to lie down after telling her grandmother what had happened. After a short time, her grandmother tried to wake her and was unable to do so (Mangeot 2012). She called an ambulance, and Maggie went to the ER, where she was kept overnight. She was sent home for a week to recover, and then she went back to school.

“It took two weeks before I realized it was impossible to salvage the semester.”

The effects of this concussion included an exaggerated version of the PCS she was already suffering from, marked by consistent headaches, extreme fatigue, focus problems, memory deficits, and depression, but it also came with new symptoms,
including anxiety, confusion, a sensitivity to bright light and loud sounds, ADHD, and mood swings (Mangeot 2012).

“I would be hanging out with my family, everything normal, and someone would say something and I would suddenly be so angry. I was so easily offended at the drop of a dime. It was really hard to deal with, and not who I am at all. It was really hard for me to stay balanced and control my moods.”

In January, her parents decided to seek serious medical help. The neurologist who examined her diagnosed her with “post-concussive syndrome, with worsening cognitive function” (Mangeot 2012). He sent her in for her first brain MRI. The results, when they returned, were shocking.

Maggie’s brain was huge from swelling, and she had developed a subdural hematoma (blood clot) above her occipital lobe in the back of her head. Either of these conditions, had they remained undiscovered, could easily have killed her. She was put on steroids to reduce the inflammation; the steroids made her so sick that she only took them for two weeks of the two months that were recommended. The doctor left her hematoma alone and monitored it closely, mostly because any meddling with her head could so easily have led to greater damage.

But these two characteristics of Maggie’s brain were commonplace compared to what else the scan revealed. Her cerebellum, the dense posterior component of the brain hugely important for motor coordination (Bear, Connors and Paradiso 2007), was puckered and squeezing out of the foramen magnum; a cone-shaped opening in the skull normally reserved for the spinal cord on its way to the rest of the body. Maggie’s neurologist quickly diagnosed this structural defect as a Type I Chiari malformation
(CM), a condition characterized by cerebellar tissue (called the tonsils) extending into the spinal canal due to an abnormally small or misshapen skull, which presses on the brain and forces it downward (Nishikawa, et al. 1997; Marin-Padilla and Marin-Padilla 1981).

![Figure 3](https://www.frontalcortex.com)

**Figure 3**: An MRI of a Type 1 Chiari Malformation. The cerebellar tonsils protrude into the foramen magnum (red circle) (Frontal Cortex 2008).

This malformation is a neurological condition completely unrelated to the lifetime of head injuries, or so her doctor says. Although Type I Chiari malformations can be caused by injury (NINDS 2013), Maggie’s neurologist thinks her CM was established during fetal development. In this case, the structural defects of the skull are caused by either genetic mutations or a lack of proper vitamins and nutrients (Columbia Neurosurgeons 2014). This type of Chiari, called primary or congenital CM, is much more common than one acquired through injury (NINDS 2013).

Maggie’s neurologist believed that the Chiari malformation existed unnoticed and unaffected all throughout her life. As her brain suffered impact after impact, the CM simply existed, neither harming nor exacerbating her growing PCS. But the final concussion was different – the area of impact was precisely located on the area of her
cerebellum that protruded into the foramen magnum. It was as if the curb had made direct contact with her brain, without a skull to protect and diffuse the impact in the slightest. As if the existing PCS were not enough, Maggie’s CM made her tenth concussion the worst by far, and is a huge contributor to both the continued severity of her post-concussion symptoms and her bleak chances of making a full recovery.

The general consensus on the prevalence of Type I Chiari malformations is uncertainty. Some Type 1 CMs are asymptomatic and usually diagnosed during the regular screening for some other disorder - in Maggie’s case, that disorder was multiple concussions (NINDS 2013). If it had not been for her concussions, it is likely that Maggie would have continued living her life with no knowledge that the CM existed. The malformation could have been detected later in life when it manifested itself as severe headaches, or she could have lived her whole life without ever noticing it.

But because of her PCS and the location of the tenth concussion, it is almost certain that the CM will cause problems as she gets older. At some point in her life, she will have to undergo brain surgery; part of the dura mater from the top of her head will be excised and inserted where her cerebellum protrudes, in order to form extra skull-like protection.

“On the day to day, it is really just an irritancy. If I get headaches it’s either there, at the back of my head, or on my forehead, where the blood clot is from my first concussion.”

After the Chiari Malformation was discovered, Maggie’s relationship with her brain changed drastically. Suddenly, there were three medical professionals constantly
checking up on her: a neurologist, a family doctor, and a neuropsychologist, with whom she began eight months of extensive neuropsychiatric therapy. This therapy entailed games you’d expect to find at an elementary school: word puzzles, memory games, balancing exercises, and color-coding, to name a few.

“On top of that, I had to go to normal therapy for the entirety of that year [I was out of school] for the mood swings and anxiety.”

Maggie was tested on several different measures of intelligence and concentration, including the Wechsler Adult Intelligence Scale, the Boston Naming Test, the Wisconsin Card Sorting Test, the Millon Clinical Multiaxial Inventory and the Verbal Fluency test. All these tests slowly began to put together a picture of what was happening inside Maggie’s brain. Her IQ, verbal comprehension, perceptual reasoning, and memory were all average. But her psychomotor speed, focused attention, and auditory working memory were all below average. Tests on her executive functioning revealed significant deficits in sustained attention and response inhibition (Mangeot 2012).

During her year of recovery, she got a job working at Starbucks, a move recommended by her therapist when, unable to get along with her family, she was told to find something to distract herself with. She started working at the coffee company in late January, but the first two months of work were very hard, and she was unable to work for any significant portion of the day. By mid-August, she was starting to feel better. Her symptoms were subsiding, and there was a visible change in how long she could work and how easily she could remember orders.

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2 Maggie gave me an evaluation written by her neurophysiologist, Dr. Mangeot, for the purpose of this thesis.
Needless to say, it was not feasible for Maggie to return to college until she got her brain up to a functioning level. The recovery took the entire year of 2012. During that year, her subdural hematoma resolved itself. She learned how to cope with her PCS. She went through head protection training for in the event of a fall, and finally, in January 2013, she was able to return to her classes.

But Maggie will never again be the student she used to be. Her neuropsychologist made the following recommendation about her return to school:

When Ms. McLaughlin does return to school she should always be seated near her instructor and preferably away from other potential distraction, to minimize any ongoing difficulties with attention. She should also take advantage of any group or “learning lab” time offered, so that she is not trying to study for long periods on her own. Ms. McLaughlin will also need to get in the habit of allowing ample time to complete assignments, so that she can work in short but frequent time periods, to maximize her attention. Ms. McLaughlin may also need to take in-class exams in a separate room, free from distractions.

(Mangeot 2012)

“Any disabilities you can file for at school, I have. I’ve had to reformat the way I study. I take written notes and then I type them on the computer. I write study guides and then I make practice quizzes for myself and I have to do readings one to two times. I’m doing well in school but it’s a pain in the butt.”

Maggie needs to take two medications: Adderall, a central nervous system stimulant used to help her ADHD, and Diamox, a drug that is typically used to reduce headaches, nausea, and dizziness associated with altitude sickness. She hates the Adderall because it makes her heart speed up, but she has to take it on regular school days to keep her anxiety and stress at bay and help her focus.

The PCS affects more than just the academic aspect of her college life. Maggie cannot drink alcohol or do drugs.
“I am allowed to drink, and I can do it to my own ability, but I found out very quickly after returning to college that I get really drunk really quickly, and it takes me about two days to recover from a night of drinking. I get really depressed and I can’t function even if I take medicine. I don’t drink to anywhere near the extent that someone our age likes to drink. Wine really hurts my head, as does really hard liquor. Sugary drinks are hard as well. Shots are really painful. I can drink beer, but I don’t like beer.” She pauses, looking to summarize everything she just said. “I don’t drink very often.”

Furthermore, Maggie gets dehydrated about three times as fast as the average woman her age, a condition that worsens with a hangover, or when she exercises.

“I am allowed to exercise, but no excessive exercise like running or biking. I can never again participate in winter sports. I can’t play doubles tennis, but singles tennis is fine as long as I don’t approach the net. I usually have a headache after workouts.”

The extra diligence required for her schoolwork and her social incapacities are not even the most challenging aspects of returning to college. In fact, what Maggie struggles with most is explaining her condition to people she meets.

“The problem with concussions that I have found is there’s no physical cast. People don’t see it and they don’t understand it. It’s hard to explain to someone who doesn’t know about the brain what’s going on… Half of the people I’m talking to understand what it is and are sympathetic, but half the people don’t get it. People treat it with a lot less… a lot more lightly than it should be… When I introduce myself and tell people that I have had ten concussions, they say, ‘oh, wow, you must have been a snowboarder!’ and treat it with a very happy-go lucky, knocked-your-noggin sort of
attitude, when really what they should be saying is ‘oh, wow, you’ve had a serious brain injury.’”

To decrease these flippant reactions, Maggie’s therapist has recommended that she refer to her PCS as a traumatic brain injury (TBI). By calling it a TBI, she removes the normalcy that most people associate with concussions, and better conveys how it truly affects her life. People take it more seriously, and it saves her a lot of grief.

This really, is the true pain of living with a TBI. “People don’t take it seriously and it’s under talked about. [But] a year of my life is gone from these things.”

The brain, as we often forget, is a soft organ (often described as having a consistency like gelatin) that is encased in a fluid-filled bony cavity (Concussion, Traumatic Brain Injury (TBI) 2012). The fluid, cerebrospinal fluid (CSF), plays a vital role in the daily life of the brain, keeping it nourished, protected, chemically stable, and buoyant. Indeed, without this fluid surrounding and buoying both the outside and inside (by filling cavities called ventricles) of our brains, our five-pound centers of intelligence would collapse under their own weight (Bear, Connors and Paradiso 2007).

When the brain is subject to a particularly large accelerating or decelerating force, the CSF is unable to provide enough protection, and the brain makes contact with the skull case (Concussion, Traumatic Brain Injury (TBI) 2012). The best-known type of brain-skull impact injury is referred to as “coup-countercoup.” In this scenario, the brain initially hits one side of the skull (coup) and then rebounds to make contact with the other side of the skull (countercoup) (Centre for Neuro Skills 2014).
Figure 4: Diagram depicting a sagittal (front-to-back) coup-countercoup injury. The countercoup impact is generally more damaging than the coup impact (Centre for Neuro Skills 2014).

Another type of force that is generated from a concussive impact is a fluid wave inside the ventricles of the brain, which can cause additional injury to the delicate tissue (McKee, et al. 2009).

One of the most damaging aspects of brain trauma is “shearing” force. A shearing force arises because, during either a brain-skull impact or a fluid wave, only a small amount of brain tissue comes into direct contact with the acting force. The un-contacted tissue stays still while the contacted tissue moves with the force, and this opposition generates shearing. This, in turn, leads to diffuse axonal injury, defined as damage to the delicate internal structure of the axon. Only in the most extreme cases do axons tear as a direct result of brain trauma. Normally, an axon undergoes specific changes (like mitochondrial swelling and disruption of microtubules) at the time of injury, and these changes can eventually lead to axotomy (axon tearing) up to 24 hours later (McKee, et al. 2009).

During an acute brain injury, neurons die from three main sources of trauma: from direct physical damage, like during a skull-brain impact; due to necrosis from excessive
and immediate release of excitatory neurotransmitters, such as glutamate; and with a delay, from apoptotic death cascades (which, for example, could be triggered by axotomy) (McKee, et al. 2009). Other factors that contribute to neuronal death are focal ischemia (a blockage of blood flow to a particular area), which kills neurons by suffocation; a breakdown of the blood-brain barrier, which leads to release of neurotoxic blood proteins - such as thrombin and fibrin (Bell, et al. 2012) - into the brain; and inflammation (McKee, et al. 2009).

Considering all of these different problems that arise from a concussion, it should come as no surprise that neuronal death has been shown to continue up to one year after injury, even after symptoms have all but disappeared (McKee, et al. 2009).

For Maggie and her ten concussions, that meant a very long recovery to get to where she is now, and the recovery process continues. She has not had a concussion since her tenth one in November 2011, which is exactly what her doctors prescribed. In December 2013, she tripped and fell on ice, but fortunately, thanks to her fall training, her thigh took the worst of it. Close calls like that one serve as constant reminders for Maggie that should she damage her head again, she would likely end up in a coma, brain dead, or dead.

But Maggie does not like to spend time mulling over what might happen. She likes to look at the positives, like how her naturally happy personality helped her

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3 To understand what happens here, imagine the neuron as a Best Buy on Black Friday. Employees (glutamate) go from door to door (glutamate receptors), opening them as they get there. With more employees, more doors are open. With more doors open, more and more people (positive ions) flood in, and, in excess, this can lead to irreparable damage to the contents and structure of the store. The same is true for a neuron with too much glutamate binding: damaged neurons release more glutamate, more glutamate means more glutamate binding, and that increased binding leads to damage – from an influx of positive charge - that can trigger cell death.
integrate back into society after recovering from her trauma. She has a lot of friends who understand what she is going through, and this support system means she does not require on-going therapy. She also has a lot of freedom to make choices about the drugs she is on. She does not want to be on Vicodin even though she could be, and she refuses to be on antidepressants even though she should be. She will get headaches once to twice a day, but normally she is able to tolerate them so she does not have to take any pain medication.

As for her future, Maggie is planning to go to nursing school after getting her masters in speech pathology. One day, she will need to get surgery for her Chiari malformation, but that day is a long way off. Right now she is focused on graduating, spending time with her friends, and keeping her head away from hard surfaces.

**Part Two: Preventative Medicine**

In October of 2013, when I was already well on my way with this thesis, a Frontline documentary was aired that stirred up much controversy in the news. Called *League of Denial*, the documentary was based on a book of the same name, published by investigative reporters/brothers Mark Fainaru-Wada and Steve Fainaru on the same day the documentary aired. They focused on concussions and brain injuries in the NFL, the long legal struggle that has been trailing the issue for years, and how the League did everything in its billion-dollar power to ignore the disturbing medical facts.

Their story begins in 2002 when Mike Webster, an ex-center for the Pittsburgh Steelers, died at the age of 50 after several years of declining cognitive abilities (Fainaru, et al. 2013). His brain was examined by a local neuropathologist, Bennet Omalu, who
discovered that Webster’s brain exhibited all the hallmarks of a dementia previously described in boxers as *dementia pugilistica* (boxer’s dementia), or “punch-drunk” syndrome. The disorder, renamed Chronic Traumatic Encephalopathy (CTE) in the 1960s, was defined as a degenerative brain disease resulting from repetitive traumatic brain injuries (Omalu, et al. 2011). Since 2005, another neuropathologist, Anne McKee, has examined 46 brains of deceased NFL football players, and found CTE in 45 of them (Fainaru, et al. 2013). One of these brains included Dave Duerson’s, whose final wishes were observed after his gruesome suicide; his brain was also found to have CTE (NPR Staff 2013).

As *League of Denial* gained popularity and the NFL’s “concussion crisis” sparked outrage, I managed to secure an interview with Rick G., a man involved closely with the dispute. “This is a highly charged political issue with lots of money at stake,” he tells me almost immediately, “And I’m right in the middle, so I will be pretty careful about what I say. But I’m happy to share with you the truth.”

A biomedical engineer and entrepreneur, Rick founded a product development company called Symbex, and in 2001, he developed a biofeedback system called Head Impact Telemetry (HIT).

“Back when we developed this,” he tells me, “no one had any idea how often and how hard people got hit. And not just in football, by the way. We [use the system] for hockey, soccer (we put it in headbands), boxing, skiing, and with the military, who also need to know when their soldiers get hit. I’m proud that the system has been used for a decade, and that 55-60 research papers have been published while using this technology. NIH research has shown that diagnosing concussions is a really hard thing to do, and that
it’s probably an art as much as a science. All we are trying to do is provide another tool in the arsenal of the clinician.”

His company currently holds a contract with Riddell, the sports equipment company best known for its football helmets (and for providing those helmets to the NFL). Riddell implants the HIT system in helmets in order to monitor head impacts in football, as well as other sports.

So how exactly does the HIT system work?

“We put little sensors inside the helmet of a football player,” Rick tells me. “The sensors are accelerometers - they measure the motion of the head - and every time an athlete gets hit while wearing the sensors, whether in practice or a game, we record the head acceleration, process it, and send it to a database for analysis later. What that lets us do is to measure what we call head impact exposure (HIE). HIE is a measure of how often, how hard and where on the head athletes get hit. Since 2003 we’ve measured well over 2 million head impacts in collegiate, high school, and youth football players, men and women’s/ boys’ and girls’ hockey players at different levels, and now we are just starting with the National Football League.”

For the last decade, the HIT system has been most popular for universities conducting research: Dartmouth, Brown and Virginia Tech are just three of the many places being funded to conduct research that uses data supplied by the HIT system. One of the research projects, for example, looks at how incidences of traumatic brain injury relates to head impact exposure. It may seem obvious that there would be a correlation between the two, but being able to measure an athlete’s HIE is so recent an advance that
medical staff are still learning how to use this information to contextualize symptoms, make diagnoses, and correlate neuroimaging results.

The database is a very important part of how the HIT system works, especially in order to relate HIE to real-life effects. When an athlete from one of the schools conducting research gets hit, the HIT system sends that information to the database. Later, when that athlete presents to medical staff with complaints of, say, dizziness or nausea, the staff can look at the information on that athlete that the HIT system collected at the time of injury. That school then has a separate database, one that combines HIE information, the athlete’s signs and symptoms, and observations made by medical staff. In this way, the clinical data is correlated with the mechanical information, so that the mechanical information can itself become a predictor of the likelihood of injury.

This research has helped determine an impact “threshold” and some universities (and even high school and youth programs) have adopted the HIT system in order to use this threshold as an injury prevention technique. “Our system measures the impact and if that impact is over a certain threshold, it sends a beep - an alert to the sideline staff. And that tells the sideline staff to go look at the athlete and do their medical protocol. So it doesn’t diagnose a concussion, it says ‘Hey! Something happened to this athlete, and it’s very unusual.’”

Another use for the HIT system, first pioneered by the University of North Carolina, is as a teaching tool. For many years, coaches encouraged football players to lower their heads to hit another player with the front/top area of their helmet.

The way the game was played up to a couple of years ago was that the players were more like… missiles launching at bodies. They are so strong; they just launch their body at someone else, and that was the way to tackle. You just use your head as a battering ram. And once we found that out, we knew we had to
change that. So the HIT system is one method of educating players to hit differently.

What we found is that if you see a guy who is hitting over and over again with the front of his helmet, getting hit there about 50-60 times a game, and he’s only been in for like 50 plays, or he’s getting 30 big hits a day – or even 20 – and you see that it’s always in the same location, you can bring that player in, show them their video, show them their head impact exposure, and teach them new ways of tackling and playing so that they don’t do that. It’s proven to be very effective.

(G. 2013)

The HIT system has been useful not only for research, threshold monitoring and education, but it has helped build better helmets, and has led to some changes seen in football, such as the new kickoff rules. Riddell, the only company that sells the HIT system, has been using the technology since 2004.

“Just this year [2013], we launched the second generation product, called Insite. It’s a cheaper version of the HIT system. The idea is that it can be used more extensively by youth and high school football teams. The HIT system costs anywhere from $400-$1000 per helmet, and Insite will only be $100 per helmet.”

Rick G. thinks that, to lower incidences of head injury, youth and high school leagues are much more important battlegrounds than the NFL. Not only does he believe that the HIT system should be geared more toward protecting younger players, but that the mindset surrounding head injuries in youth athletes needs to change.

“[When it comes to injury, the thing] everyone is always talking about is return to play. Everyone wants to know how soon that athlete can return to play because all they care about is winning. What we care about, especially with our kids, is about return to life. It’s about being able to go back to school, to interact with your family, to be in your community, to interact with boyfriends and girlfriends… all of that.”
I tell Rick about Maggie, and her current battle with TBI from multiple concussions, many of which resulted from her time as a cheerleader.

“You know what, when it comes to incidence, not prevalence...” Rick goes on to clarify that prevalence refers to the raw number that corresponds to a statistic, and incidence refers to that number normalized with exposure. Because there are many more high school football players (more exposure) in America than there are high school cheerleaders (less exposure), there is naturally a much higher prevalence of head injuries among football players. “Well, when it comes to incidence, cheerleading has the highest incidence of brain injury of any sport, even more than football.”

Cheerleading, he says, is as contact a sport as they come. The stunts that a typical cheerleading squad performs are dangerous for everyone involved: the flyers risk falling from several feet in the air onto a variety of surfaces – basketball courts, track, and packed dirt – and bases risk getting hit by misplaced elbows or knees.

“It’s a very severe problem that doesn’t get a lot of attention because there are so few cheerleaders compared to football players.”

He continues, highlighting why brain injuries in young people are such a problem. “All the science and neuroscience literature shows that if you sustain a brain injury, you are much more likely to sustain another one with a less significant blow. Once you get two or three, you are even more susceptible to the next one. So your friend, she will probably be injured by anything that hits her head for the rest of her life.”

Never having talked to Maggie, Rick G. understood her daily struggles due to head injury. But so many people do not have that understanding, and fail to recognize that concussions are a serious problem, especially for young athletes.
In April 2010, while the NFL was still fighting to keep the concussion crisis as hidden as possible, a 21-year-old Penn State football player named Owen Thomas hanged himself in his off-campus apartment. His friends and family described the time leading up to his suicide as an “uncharacteristic emotional collapse” (Schwarz 2010). Upon autopsy, it was discovered that Thomas had Chronic Traumatic Encephalopathy, the Alzheimer’s-like dementia disorder produced by head injury, previously discussed in cases like Dave Duerson’s and Mike Webster’s. The discovery that Thomas had the disease was a shock for two main reasons: First, it was unbelievable that someone so young had contracted a degenerative dementia disorder, and second, Thomas had never been diagnosed with a single concussion during his lifetime (Fainaru, et al. 2013). This information, combined with the undoubted presence of CTE pathology, pointed toward a new truth: Sub-concussive impacts produced from everyday practice of some sports were enough to distort the internal environment of the head enough to yield irreparable damage (Fainaru, et al. 2013). All of these cases, from boxers to NFL athletes, from college football players to cheerleaders, illustrate an inconvenient truth: The brain is far more susceptible to trauma than we care to admit.

But research has gone further than these attention-grabbing case studies. A 2013 questionnaire study aimed at improving head injury understanding looked at how well coaches recognized and managed concussions. The study highlighted important misconceptions about concussions, including beliefs that it was “easy to tell if a person had brain damage by the way a person looks or acts,” and that “head injury cannot cause

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4This point was also illustrated by the case of Jeffrey Astle, an English soccer player who was a prolific header of the ball. Astle died at the age of 59, after five years of cognitive decline, and was also found to have CTE (Spiotta, et al. 2011).
brain damage if the person is not knocked out” (Hossler, Phang and Passannante 2013).

This study demonstrated that there is still much for athletic coaches to learn when it comes to concussions. “Only 16.9% of coaches correctly described concussion as a chemical disruption altering brain nerve transmission. Furthermore, 16%... knew that adults recover more quickly from a concussion than teens, and slightly less than half, 42.5%, knew that the size of a teen’s skull…made them more vulnerable to whiplash-like effects” (Hossler, Phang and Passannante 2013).

A recent article published in *Neurology* looked at how long brain defects remain after a mild concussion. Using distinctive scans, the researchers demonstrated that abnormalities were still present four months post-injury, after symptoms have mostly disappeared (Ling, et al. 2013). These results provide support for altering “return to play” guidelines, a policy change that Rick G. thinks is vital for safeguarding young athletes’ futures. It is difficult to look at a case like Maggie’s and not wonder how things would be different had this modern understanding of concussions been in place during her time as an athlete. Would her cheerleading coach have asked her to leave the team? Would she have begun avoiding contact games earlier? These questions will remain unanswered, but with stories like hers, Rick G.’s technology, and work being done by healthcare professionals and researchers alike, the concussion mentality has slowly begun to change.

One can only hope that some day soon, the idea that an asymptomatic brain is a healthy brain will no longer exist. The idea that injury only exists if tangible has been a long-standing hindrance for neurology. Because of this belief, people seem to think that the only things that break are those that can be wrapped in a cast.
But there is no cast for the brain. There are surgeries that can be performed in extreme cases, steroids that reduce inflammation, anticoagulants to disrupt clots, and pain relievers to dull headaches. But there is no cast.

The brain heals itself silently, invisibly, as the surrounding body goes through motions and restores an outward sense of normalcy. Perhaps this lack of visible damage is a relief, an empowerment, or perhaps it is the body’s greatest betrayal.
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The Man with Two Faces

Branford, CT, 1996

Carl looks down at himself. He is missing both his shoes and his shirt. In his hand, he clutches a crumpled piece of unfamiliar paper. A distant memory tickles the back of his mind; he knows the paper is important, somehow. The memory grows stronger. This paper is magic. He knows it. It is the answer to something, maybe a riddle, maybe life itself. Carl realizes the burden he holds in between his trembling fingers. Someone has to know. He sees an auto repair shop not too far away and lurches toward it.

Inside, he is fortunate. He sees a familiar face, a friend’s brother. Carl rushes toward him and explains what he has discovered, but in his attempt to convey importance, he begins to raise his voice. Other people take notice. One mechanic sees him and gets angry, starts yelling at him. Suddenly, Carl is concerned.

This man wants to kill me. You can’t just wander into random places with the answer to life in your hand. Who knows what could happen.

In an attempt to protect his find, Carl goes into fight mode. He starts to yell back at the angry man. But suddenly, a new face enters the picture, a kind, trustworthy man. He tells Carl to leave. “Escape,” he says.

Carl knows the man wants to help him, and so he runs outside. Now all he wants to do is get home. He has the answer to life, and he is God. His vision zooms back and he looks down at the world from above, the whole world like a computer, with everyone moving around as a part of its system. He holds the paper tightly, and navigates his way toward his apartment the quickest way possible. He starts walking on the highway. All he is thinking about is the safety of home.
His time as God is short-lived. A police car pulls up. Carl recognizes the officer. Shirtless, shoeless, and with nowhere to run, he does not fight. At the station, he is placed in a holding cell next to a shadowy figure. He wonders what happens to people who think they can play God. Uneasy, he shouts at the figure.

“The devil doesn’t exist!” he cries. The form shifts in the dark. Carl has to show the thing how powerful he is. “I flushed him down the toilet! I flushed the devil down the toilet!” Suddenly, the wraith becomes a normal boy, a faceless teenager, who pulls up his shirtsleeve to reveal a burning world tattooed across his forearm. The world is engulfed in flame, crackling, ripping up earth across his flesh and searing itself into Carl’s mind. He has never seen anything so horrible, so terrifying.

Image Credit: “Many Faces” by Nina Allen, retrieved from photobucket (Allen n.d.).
Middletown, CT, 17 years later

The Star and Crescent Eating Club is at the height of its dinner rush hour. Waiters hustle in and out of the kitchen, carrying hot plates of Thai red curry and refilling water pitchers. I dodge away from a tray of salads and break through the tiny door leading into the kitchen. Inside, it smells wonderful. The head chef is giving directions to the lead sous chef. He demonstrates how much food he wants placed on each plate and reiterates how important it is to serve the food quickly, while it is still hot. He is the center of attention, pointing, nodding, and tenderly sprinkling basil onto plates of rice.

Off to one side and away from the action, another man stands thoughtfully looking down at his salad. He takes a bite, a big bite, and chews slowly. He is wearing old jeans and a jean jacket with faux fur lining, an outfit more suitable for the early 90s, perhaps. His face is adorned with a couple of day’s worth of stubble; a grey and white striped cap hides his bald scalp. As a 41-year-old on a liberal college campus, where everyone else is twenty and dressed like a hipster, he is noticeable only because of his age. He and the executive chef, Ryan, are the only real adults around.

I approach him.

“Hey Carl, “I say, “You ready to go?”

With a lopsided nod he says he is ready when I am. Carl waves at Ryan, who waves back, and we are out the back door.

“So,” I begin, “what are we doing today?”

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Carl and I are in his car. We are driving through Middletown, Connecticut in a rainstorm. Our conversation is gloomy, made gloomier by the drone of the windshield wipers and the patter of rain on asphalt.

I sit in the passenger seat. Nervously, I push my phone closer to Carl’s side of the car, hoping that its little microphone is strong enough to pick up our voices over the background sounds of weather and road.

Carl briefly tells me about his childhood. He describes himself as a sensitive child, an attribute likely related to the tense relationship he had with his father, who was constantly moody due to the irregular sleep pattern that came with his job as a foreman at a steel mill. He was impatient, prone to yelling, and young Carl was always scared of him.

One day, when he was 25 years old and had recently returned from a trip to Europe with his girlfriend, he was sitting in the bathtub in the upstairs bathroom of his parents’ house. After he had been in there for a while, his dad came in to check on him. Carl snapped. He accused his father of looking at him. Screaming, he ran downstairs, declaring that his father had sexually abused him. As if an overwhelming memory had suddenly come crashing back, he yelled out at his father, “I know what happened! I know what happened!” The outburst continued like this for several minutes. His parents, desperate and afraid, brought him to the Yale Psychiatric Institute (YPI), where he was diagnosed with bipolar disorder.

“Looking back,” Carl says, “I know it didn’t happen.”

But Carl’s life was not completely free of abuse. When he was around eight years old, a kid who lived in his neighborhood sexually abused him, building him go-carts to
keep him from telling anyone. Carl has a hard time remembering exactly what happened, but he can vaguely recall some details. These memories may have come to life at the moment Carl accused his father of abuse, a notion that emphasizes the intensity of his condition, as hallucinations are associated with more severe cases of bipolar disorder.

When he was initially diagnosed, he had a hard time believing anything he was told. “When they discharged me from YPI, I wouldn’t sign the papers,” he says, “they called me a ‘delusionary idealistic.’ That sounds like a good thing! No sooner had they discharged me and I wouldn’t take the medicine, I was back in the hospital.”

Carl is one of 5.7 million American adults who suffer from bipolar disorder (National Institute of Mental Health 2010). The DSM-IV-TR classifies bipolar disorder as a mood disorder, a title it shares with depression (American Psychiatric Association 2000). This modern understanding of bipolar disorder did not come about until the early 1900s, when a German psychiatrist named Emil Kraepelin spent time studying its untreated symptomology (Burton 2012). Before his studies, this disorder was known by one of two other names: “dual-form insanity” or “circular insanity,” depending on who was asked (Burton 2012). Thankfully, Kraepelin’s research led to a breakthrough in understanding that motivated a new way to describe the disorder, and the name was changed to “manic depression,” a term devised to embody the two main cycles that wreak havoc on a person’s consciousness: mania and depression. Attempts to decrease the stigma associated with mental illness required another name change, and the modern name for the syndrome, adopted in the 1980s, is used because the definition of the word “bipolar” - having two poles or extremities - succinctly and accurately captures the manifestation of the disorder (Burton 2012). A sufferer from bipolar disorder alternates
through the frenzied state of mania, sometimes prolonged by a persistent, elevated hypomania, and the lethargic state of depression. Carl’s illness is severe enough that at the height of his mania he sometimes wanders into episodes of psychosis, like the one that occurred in Branford in 1996.

Mania is well encapsulated by the persona of a millionaire playboy on his birthday. His mood is abnormally elevated or easily irritable, he has impossible reserves of energy, and is almost instantly aroused. He is outgoing and competitive. He has an extravagant style, he never needs to sleep, and his self-esteem is infinite. He is the life of the party, talking often and for long lengths of time - bouncing from idea to idea, jumping from thought to thought - his racing thoughts perfectly coupled with his physical hyperactivity. He indulges himself not only with material things but also with enjoyable behaviors, such as unprotected sex, hardly pausing to consider the consequences (American Psychiatric Association 2000).

If mania is a rich party animal, depression is an old man lost at sea. He is either unable to sleep due to the scorching sun, or sleeping constantly out of pure exhaustion. Even the simplest tasks, like drinking water and stretching his legs, are almost impossible. His whole body aches, and he is unable to concentrate. He can do nothing to change his situation, and so he feels helpless. He has no hope, and day after day of nothing but endless sea makes him doubt the worth of his own life (Smith, Saisan and Segal 2014).

Hypomania, which literally means “below mania,” is a state that is very similar to mania but at a lower intensity. It is in this state that creativity and productivity are often enhanced. It is distinguished from mania by its lesser impact on normal function and by
the absence of psychotic symptoms and grandiosity (American Psychiatric Association 2000).

I ask Carl how he would describe the disorder to someone who had never heard of it: “High lows and low lows, “ he says. “The manic side, I have it with psychotic episodes, where my mind literally plays tricks on me. Like accusing my father, first real example of some totally psychotic thought that had no basis in reality but in my own mind.”

Any type of stimulus can cause Carl to have a psychotic episode. “ Anything you are taking in, something somebody says, a look, something you see, a way you feel about something, you intake it like a computer and then the computer takes it and jumbles it all up and it’s no telling moment to moment what type of scenario you can create with it, and usually it’s pretty ugly.”

Carl and I are driving to his friend Richard’s house. They are in a band together, along with another man named Chuck, and Ryan, the head chef of the Star and Crescent and our mutual friend. Carl plays guitar in the band, a passion he has held onto since high school. Recently, he sold Richard his Honda, but he has yet to receive payment. Even more recently, he backed into someone’s car down in New Haven, and convinced the guy to let him pay for repairs instead of going through insurance. And so the wild goose chase commenced: Carl was driving to Richard’s to collect and then to New Haven to make good, all with me in tow.

Before Carl was diagnosed, he was interested in spending time with his friends and practicing his guitar. He played hockey during some of his teenage years, but decided to quit and focus on his music. After he graduated from high school, he went to music
school in California. There, he was transformed as a musician. He loved everything about the Golden State, and wanted to make his way in the music industry. His dreams were big and, to him, unstoppable.

But he ended up coming back to Connecticut shortly after music school to stick with a friend who was returning to the East Coast. “I had such a strong connection with him playing-wise that I decided to come back, too. I never wanted to. I was miserable.”

I ask him if he knows about the correlation between bipolar disorder and enhanced creativity. He says he does, and that the most creative time of his life was right around when he was getting diagnosed.

He describes the state of hypomania, the pleasurable heightened “stuff” you feel right before getting trapped in an episode of psychosis.

“You feel like you can do anything, you don’t need a lot of sleep. You spend money like it doesn’t exist. It’s like the world is your oyster. You don’t really have any concerns. It’s like you can do anything. It’s quite intoxicating. If you can maintain that you can be really productive.”

I ask him if he can maintain it, and he tells me it is a dangerous place for him to go.

Carl tells me about one of his more recent psychotic episodes. The medicine he takes gives him vivid dreams, and prior to this episode he had been having dreams about the world ending. During his psychosis, he remembered these dreams and he saw the earth plummeting through space. He truly believed the world was going to end, and so he took it upon himself to stop it.
Carl likes to collect things. When he is having an episode, he will rearrange them and put them in a certain order, giving major significance to where he might put something. During this specific episode, he looked around his apartment and realized that the only way to save the world was there, with him and his stuff. His arrangements had never been more important. He carefully collected items and placed them where they needed to be, turning a cup one quarter of an inch clockwise, moving a sweater half a foot to the left. Once everything was arranged where it needed to be, he went to his sink and turned the water on. With the water on, he knew that he could walk in a certain direction and he’d be able to manipulate time. Even though Carl believes these delusions so completely when he is having an episode, he is completely aware of how outlandish they are once he has returned to his normal state.

“It’s maddening, because you can’t stop it. I would stay up all night dealing with this stuff.”

What happens in the brain during a psychotic episode is still not specifically understood. There is an increase in activity; with bipolar disorder, it is suggested that there is greater increase in the left hemisphere of the brain relative to the right hemisphere (Lohr and Caligiuri 1997). There is a complex relationship between psychosis and the reward neurotransmitter, dopamine, and it is further postulated that a dysfunctional relationship between the neurotransmitter glutamate and one of its receptors is also important (Kapur, Mizrahi and Li 2005). Some theories make more inclusive claims, proposing that many psychotic symptoms arise with problems in ownership perception (Blakemore, et al. 2000). That is, internally generated voices, for example, are mislabeled by the psychotic mind as coming from the external world.
Psychotic hallucinations arise from aberrant activity in a sensory cortex: The brain fires as if it had received a signal from a sensory organ (like the eyes, ears, or nose), and is therefore able to generate novel pictures, sounds, or smells with absolutely no connection to the real world.

Carl takes two different medications to help his symptoms: Depakote and Lamictal. They are both used to treat epilepsy because of their anticonvulsant effects, but are equally useful in bipolar disorder management as mood stabilizers. Although bipolar disorder is classified as a mood disorder, both it and epilepsy can be understood as diseases of the extreme (hyperactivity that leads to seizures versus mood swings that lead to highs and lows); in this sense the drugs act to balance the brain.

A paper published in 2003 by the journal Psychiatry Research: Neuroimaging shed some light on how exactly mood stabilizing and antipsychotic medication worked inside a bipolar brain. In this study, twenty-four bipolar subjects and thirteen healthy comparison subjects were placed in a functional magnetic resonance imaging (fMRI) machine and their blood oxygen level dependent (BOLD) responses were measured as they were tested for manual reaction times (Caligiuri, et al. 2003). Because the brain, like all organs, needs oxygen to function, oxygenated blood will flow towards brain regions being used more intensely. In this way, the BOLD technique used by fMRI machines can show real-time changes that occur in the brain when a subject is given a specific assignment, such as a motor test. This experiment showed that bipolar subjects in both manic and depressed states exhibited abnormally elevated neuronal activity in numerous cortical and subcortical areas of the brain (Caligiuri, et al. 2003). However, bipolar subjects taking mood-stabilizing medication exhibited significantly lower brain activity
in these areas, specifically in the motor cortex (cortical), basal ganglia (subcortical), and thalamus (subcortical) (Caligiuri, et al. 2003). These findings suggest that antipsychotics, anticonvulsants, and mood-stabilizing medication work through a mechanism of suppressing specific cortical and subcortical functions (Caligiuri, et al. 2003).

Neurons in the brain communicate by way of different signaling molecules, more commonly known as neurotransmitters. When a drug works by suppressing specific brain functions, a very encompassing statement, it should be understood that this suppression, as well as any suppression or excitation, happens at the level of neurotransmitters. A neurotransmitter called GABA (short for gamma-aminobutyric acid) is widely regarded as one of the brain’s most important inhibitory neurotransmitters. If a neuron receives GABA from another neuron, it is as if that neuron is saying, “Stop! Do not fire any more signals.” As well as performing other functions, Depakote, one of the drugs that Carl takes, works specifically by enhancing the transmission of GABA (Rosenberg 2007). Lamictal, his other prescription, works in much the same way but by an entirely different mechanism. Instead of up-regulating GABA, studies support that Lamictal actually blocks sodium channels, which are absolutely necessary for firing action potentials (Rogawski and Lösch 2004). More action potentials equals more brain activity, so blocking these sodium channels quiets an overactive brain. Depakote and Lamictal are essentially two sides of the same inhibitory coin.

The point of this medicine is to act as a “buffer for his emotions” and keep Carl sandwiched somewhere in between depression and mania. He takes both drugs every night. “Usually I remember… I think I remember,” he muses.
Before finding his ideal cocktail, Carl went through a lot of different drugs, the first of which was lithium. Lithium, he says, was like putting glue into his system. He felt lethargic and slow, and had a hard time staying focused. He also tried Wellbutrin, a depression drug, but that did not work either. The medications for mental illnesses do not work across the board for everyone. Their effects vary depending on the person, and each drug requires a testing period, sometimes as long as six months, before it can be determined whether it works or not. The whole process is exhausting.

Carl starts driving with his knee on the steering wheel, pulling out an old CD case and browsing through the CDs. They are all burned discs, with one or two identifying words scribbled on them in blue. He does not find what he is looking for, puts his hands back on the wheel, and continues driving.

His phone rings. It is Ryan, calling to ask how he enjoyed the food from dinner that night. “It was delicious,” Carl says. “With peanuts and cilantro it would have been great. And it could have been a little hotter temperature-wise.” I hear Ryan through the phone, agreeing and thanking Carl for his input.

Carl went to culinary school in 2003, and met Ryan through a catering job a little while later. At the time, he was beginning to give up on his dreams of becoming a famous musician, and before getting into cooking he worked a number of part time jobs, never finding anything he loved as much as music.

Now, he works as a job coach for a direct service provider. He handles clients who have mental and/or health difficulties, and the majority of them are not capable of working. It is a lot like babysitting, he tells me. He makes $11.99 an hour, and he has been there for four years.
Despite the time spent at culinary school, Carl does not cook much any more, and has not since getting divorced five years ago.

Carl says that his marriage ended because it began for the wrong reasons. He had been with his girlfriend, Shannon, for seven years, and they were both in their early thirties. Carl was motivated to get married because of the safety that comes from starting a life with someone. In the end, he does not think they were right for each other.

Carl’s voice starts to trail off. “She probably wanted to save me, that sort of thing…”

When they got married, Carl thought it meant he would not have to take his medication anymore. He figured that settling down meant his brain would calm down, too. But he had an episode shortly after his marriage, ending his hope that it would solve his problems. Other than a couple of flings, he has been single since the divorce.

“I wasn’t really the marrying kind.”

* * *

The mania is much easier for Carl to talk about than the depression. More often than not, overwhelming sadness follows moments after the end of a psychotic episode. In trying to explain it, Carl’s thoughts and words stumble over each other.

Like if you come down from, cause you do, you retain things, it’s not like you totally forget the things you do, being totally absorbed in this thing that isn’t based in reality, when you come down – that’s why I think these illnesses have such stigma, like people are so sensitive about it. When your mind plays tricks on you, it’s not a humbling experience. It’s completely humiliating. To not, like…. it seems like normal people, no matter what goes wrong, at least they know who they are, have some sense about them. Granted, their life might be falling apart in front of them, or they might have to deal with all this stuff but they’re kinda, at least, in some sense, not completely gone. Maybe sometimes they wish they were, so they wouldn’t have to deal with it.
After you come back from an episode, you are able to realize what you just went through or what you just put people through or that it might take you months and months or years and years to get back to where you were. Like you might have lost a job. Every time you have one of these episodes there is no guarantee you are going to get back to where you were. You are going to decompensate. You may never get back. You could do damage, you know, in a variety of ways. They say the more episodes you have the harder it is to really… that’s the danger, you know, of having a bunch of episodes.

(Carl 2013)

The depression is crippling. It takes away the ability to function. Carl usually has a hard time remembering his depressive episodes. He describes it as being inside a cocoon. And when you finally emerge, you do so not as a butterfly, but as a confused caterpillar that does not remember why it went into a cocoon in the first place.

There is no definite, established cause of bipolar disorder. Unlike a bone, which will always break with a specific amount of perpendicular force, the factors that set the stage for mental illness are always shrouded in uncertainty. One study, published in 2004, examined gene expression in brains with bipolar disorder and discovered widespread dis-regulation of mitochondrial energy metabolism, suggesting that dysfunction in mitochondria, the energy-producing organelles found in almost every human cell, is correlated with bipolar disorder. This same study found 43 genes with decreased expression in subjects with bipolar disorder, implying an important genetic contribution to the illness (Konradi 2004). Another study, this one from 2003, looked at the role of oligodendrocytes (discussed earlier with regard to their breakdown in multiple sclerosis) in both bipolar disorder and schizophrenia. This study found that bipolar brains showed a “downregulation of key oligodendrocyte and myelination genes, including transcription factors that regulate these genes” (Tkachev, et al. 2003). Additional studies look at abnormalities in a number of different brain structures, including the basal ganglia and
the amygdala, the emotional hub of the subcortex important for “fight or flight,” and their roles in bipolar disorder (Strakowski, et al. 2012; Kempton, et al. 2008).

Without me realizing, we had exited the highway and were now turning left and right through various neighborhoods. After about five minutes, we reach a steep driveway and go up it. A man who I immediately assume is Richard pulls in just behind us and stops in front of a garage. It is no longer raining, but the area around the driveway is muddy and slippery. Richard is tall and attractive, charismatic and kind at the instant of our meeting. Carl pulls two amps out of the backseat of the car and puts them into Richard’s garage.

He looks down at his jeans and sees a small spot of mud on the front of his right pant leg. With a napkin from his jacket pocket, he bends down and compulsively wipes the mud away. In an exasperated tone, Richard asks him what he is doing, and then turns to me with a big smile.

“Why you hanging out with this kid?” he asks. “This kid is crazy.”

Carl looks down as if he is ashamed, but explains my presence without getting defensive. When we leave Richard’s (plus money, minus amps) and begin our drive to New Haven, he says nothing but good things about his friend.

Carl met Richard when he was going through his divorce, and after discovering that he was a singer, he started working on music with him. Richard has two kids, Shai and Lyric. Their names are tattooed across his forearms.

“I have a really nice network of friends,” Carl tells me, “but they got their own things going on. Families, you know? I drive around a lot. Sometimes, I’m sitting at home and I just have to drive. I get bored and lonely.”
Carl always wanted to have kids.

He asks me to move the passenger seat back. I oblige, commenting that I always have a hard time looking into the right lane with a passenger’s head in the way.

“That’s not it,” he says. “I just get nervous sitting so close to the windshield.”

I can see that he is starting to deflate. He talks with heaviness in his voice, a frown constantly on his face when he is silent. This trip is beginning to get exhausting for him. He has to stop at a gas station to fill up the car, which he does while lamenting not being able to spend his money on things other than someone else’s repairs. In the gas station he buys himself two yellow Vitamin Waters, and when we begin driving again he tells me that he was not even supposed to be in New Haven when the wreck happened – that he was just giving a friend a ride. Several times he expresses his frustration with having to make this drive and part with his money.

“You are definitely catching me at an interesting point: a transitional point. Some things are for better and some things are for worse. Like music, it brings out aspects of my personality that I miss in some ways – like the passion, and being creative, and getting stuff done. But working with other people and within time constraints and with other peoples’ strengths and weaknesses – it’s frustrating. I can do a lot of music in my house but I have trouble concentrating.”

Carl is currently living in an apartment that is adjacent to someone else’s house. He does not mind it, but sometimes feels like he is living in his parents’ basement.

“My family is very generous,” he tells me. “The money I make pays for my apartment and a few other bills, but all the other little luxury things I do I wouldn’t be able to do if it wasn’t for my family.”
Carl tells me that in the last month he had spent a lot of money on his credit card, and that it probably was not the best idea. “It’s embarrassing that I can’t support myself, but I get to do what I want. I had pretty much every opportunity in the world to go to school. But I was left to make my own decisions and I didn’t make good decisions. Whether it was because I wasn’t very bright, or had delusions to begin with…” He trails off.

Music had obviously always been a part of Carl’s life, but maybe it was not always a positive one. He tells me that as much as he loves his music, he worries about what it cost him in life. He thinks it may have ruined a lot of his relationships, maybe even with his ex-wife. And he certainly never expected to accomplish anything less in life than becoming a famous guitarist.

“You know people who say they don’t have any regrets?” He shakes his head and looks blankly down the highway. “That’s not me, that’s for sure. I have tons of regrets. When I was in YPI, they wanted me to take the medication, but I didn’t, and when I look back I wonder, wow, I wonder if I had taken the medication how different my life would have been.”

Carl’s life is full of questions like this. He will never know exactly what events were triggered by the bipolar disorder and which ones were his own volition. He gives me examples. His spending habits could be irresponsibility or could be a symptom of the mania. He had an episode that resulted in a friend of his getting divorced, as Carl told this friend that his wife was cheating on him. They do not blame him for what happened, but he is still haunted by the fact that his disorder set the wheels in motion to end their marriage. Along those same lines, could his own marriage have ended because of his
disorder? Carl, like everyone, has a “one that got away” but in his case, she left him shortly after their trip to Europe together, where, in a fit of mania, he made her leave and go back to the States. Finally, he never went to college; was that because of his personality, his love of music, his difficulty concentrating, or are all three of those simply side effects of living with bipolar disorder?

Everything Carl tells me about his life instills questions of “what if?” and as he talks, his frustration grows.

It’s very stressful for me right now. It’s too much. And knowing that I messed up a lot of things in my life, I’m getting to be in my early 40s, going to be 42 this year, I’m getting to that point where I feel like I’ve missed out on certain opportunities. Like when I was younger I always wanted a family. But now it’s like, would I even want to bring...you know, considering my unpredictability health-wise. You gotta start to consider things like that, too. What if I did have a child, do I have to worry about them having the same issues I have, some mental health issues, or do I have to worry about myself having an episode or not being able to be there? I don’t really consciously think of that at all, but it’s... considering the opportunities I had, and things I could have taken advantage of, and places my life could have been - my life could have been a lot worse - but it could have been a lot...better, you know?

(Carl 2013)

We sit in silence. “Going to California to make music was my dream.”

We arrive in New Haven and drop off the money. Carl is nothing but friendly and cordial to the owner of the dented car, but when we begin our drive back to Middletown, he looks defeated. I realize that he does not want to answer any more of my questions. We spend the rest of the drive making small talk: the conversation becoming increasingly one-sided as I talk about school and Carl grunts one-word responses.

We reach my house, and Carl pulls over to the side of the road. I thank him for telling me his story and start getting out of the car. He stops me.
“My band is performing at this bar in Watertown. It’s in a couple of weeks. You should come.”

I say that I will do my best, say goodbye, and close the door.

**Watertown, CT, 2 weeks later**

I enter out of the cold into the Red Door, a bar in Watertown, CT. It is a Thursday night. Carl and his band, Six Train Soldier, are getting ready to perform. I see Ryan at a table with someone I do not recognize. They are drinking beers and copying their set list onto printer paper with a red sharpie. I go and greet them, offering encouragement for the performance ahead. I am introduced to Chuck, the drummer of the band. Looking around the large, well-organized bar, I see Carl, buzzing with activity, walking to and from the stage to the bar, the sound box, the bathroom. He keeps his head down and does not interact with many people. He is wearing dark bell-bottom jeans, a white shirt, a black jacket, and a fedora. When he sees me, he comes over to greet me, but quickly rushes off to fiddle with the set-up on the stage.

Soon after, he returns, all business, and alerts the band that it is time for a sound check. They organize themselves on stage, Ryan on the left, Richard in the middle, Carl on the right, and Chuck, on the drums, in back. Almost immediately, Carl strums his chords a couple of times, plays a short little riff, and then wails on his guitar in a way worthy of an audience.

Once the sound check is done, it is not long before the band starts playing. Carl is focused on finding perfection, constantly adjusting his sound with a pedal and monitor at his feet, hearing flaws indiscernible to the untrained ear.
Seeing him on stage should have brought me joy. After all, there he was, 41 years old and able to do what he loved. But knowing what I knew it was impossible to fully embrace his music. He was talented, brilliantly so, but that did not surprise me. I asked myself one question repeatedly: “Who is Carl without bipolar disorder?”

On stage, he stands mostly still. His head is rocking back and forth, getting lost in the music, but his face displays no emotion. He is perfectly in his element, and yet, something about his body language seems tense and controlled. As the owner of a brain reigned by extremes, he seems to regard himself with wariness. He does not trust himself. He blames himself for his disappointments. I know that Carl is taking medicine, but on stage with his guitar, I do not see a man who is receiving treatment for his illness. Instead, I see a man who has been traumatized by his own mind, who is cautious about feeling his own emotions. Carl has never talked to a therapist about his brain. He understands his illness only in terms of the drugs he is supposed to take, and his regrets. So, he holds himself still. He tries not to think. As he plays his music, I feel like I can see Carl at his most vulnerable, just moments from being swept away, either upwards or downwards, into psychosis or into depression, but never into a state that is simply content.

When the song ends, and the audience starts to clap, I hold myself still, like Carl, and try not to think.
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The Dying Brain

The hospice ward of Middlesex Hospital is on the seventh floor. It is a long, stark hallway scattered with rooms, with a nurse’s station about three quarters of the way down on the left-hand side. A few lost wheelchairs are visible, hastily stored against walls or next to important-looking machines. On the door to every patient room is a sticker depicting the pedestrian crosswalk man, but he is tilting backwards at an impossible angle, arms raised above his head in obvious surrender, the words “Fall Risk” printed in large block letters underneath. Some rooms have other signs as well; signs that warn against airborne illnesses or alert caregivers about patient allergies, all of them printed on brightly colored paper and typed in bold font.

Hospice comes from the Latin word *hospit-em*, which means “to host” (Oxford University Press n.d.). The clients of this particular hospitality, however, are not your typical vacation guests. For one thing, most of them will probably never check out. So when the opening of elevator doors signified my arrival on the seventh floor of Middlesex, it was with great solemnity that I stepped onto the ward. Gingerly, I proceeded down to the nurse’s station, doing my best not to disturb the milling nurses or volunteers, and when I got there, I stammered out an introduction.

“Hello, I’m Taylor. I’m looking for Dr. Zermatt. Is he around?”

They pointed me in the direction of his office, where I found the white-haired man I had encountered at an early morning meeting to discuss cancer patient prognoses (tumor board, as it is called in medical circles). He had immediately piqued my interest as a hospice physician in a room full of oncologists. As the man who inherited their dying, his presence felt to me like a symbol of medicine’s ultimate failure and a statement of
inevitability. All the doctors at the meeting were calm and businesslike, a jarring juxtaposition to the projected MRI scans of bodies lit up by tumors. The discussions were never heated, no opinions were rudely shot down, and no one hesitated to admit it if they needed another doctor to provide a second opinion. For a room intrinsically besieged by malady, it was utterly serene.

At one point, the oncologists were discussing an elderly woman with a particularly vexing problem. She was found to have serious, late-stage liver cancer, but was surprisingly asymptomatic. The discussion was as follows: If she began treatment, the chemotherapy would drastically decrease her quality of life. Should her oncologist recommend treatment, to give her maybe a few extra, lower-quality months? Or should he recommend no treatment, and therefore a shorter life with less discomfort? It was for the second course that Dr. Zermatt advocated, arguing against the unnecessary pain. One might think that his time in hospice had made him used to death, which is why he favored the no-treatment option. But that could not be farther from the truth. Hospice is not a place where people go to die. Hospice is a place where people go to regain quality of life in the days before they die, so that they may die with dignity. Dr. Zermatt, a hospice physician, was a man fully invested in life. Understanding this, the oncologists nod their agreement, and it is decided that the woman will not be recommended for cancer treatment.

Dr. Zermatt is an older man, likely in his 60s. His hair and moustache are just as white as the doctor’s coat that he wears over a forest green button-down and multi-colored tie. He has large, thick glasses, and an approachable but professional demeanor. There are lines around his eyes that reveal both the satisfaction and the sadness that a
lifetime of caring for the sick will bring. He looks like he comes from Georgia, or Tennessee, or any one of those Southern states where people speak in a drawl and welcome you onto their porch for sweet tea.

He may look the part of a Southern gentleman, but when he talks, his speech is free of a Southern accent. He knew why I wanted to meet him, and quickly got down to business.

“Let me introduce you to one of our nurses,” he told me, and hurried off in a way that suggested I should follow. I did. We made our way back to the nurses’ station, where a tall, thin, middle-aged blonde woman stood, dressed head to toe in patterned scrubs, holding a pill cup in her hands.

“Taylor, this is Yvonna. Yvonna, Taylor is a Wesleyan student who is interested in what we do here. She’ll be sitting in on our department meeting later today. Until then, she is going to follow you around, OK?”

Yvonna seemed neither pleased nor irritated at the idea. She had a job to do, and she would do it regardless of tagalongs. We went into a patient’s room, and Yvonna set down her pill cup on a breakfast tray laden with pancakes, yogurt, and dark coffee.

She grabbed the metal fork off of the breakfast tray and started mashing the pancakes into a mush worthy of a newborn baby bird. The man for whom she was mashing was awake, alert, and rather perturbed. He accused Yvonna of being late with his breakfast, stating over and over that he “always had his coffee at seven.” He looked at me, stared at me for a long time without blinking, but did not seem to question my presence. Instead, he asked if I could send him home, and reiterated that he was used to drinking his coffee at seven. With the pancakes mashed, Yvonna firmly and loudly
encouraged the man to eat them, brandishing the fork like a syrupy saber, edging it closer and closer towards his pinched mouth. He could feed himself, he told her.

“All right, here’s the fork.” Yvonna placed it precariously close to the edge of the plate, minimizing the distance between the fork handle and his crooked, arthritic hand. She tucked a napkin into the front of his hospital gown as he reached for it, picked it up, and, like a child learning to coordinate its limbs, brought a little pancake mush up toward his mouth. While his jaw went through unnecessary chewing motions, his eyes wandered over to the window.

I had been in hospital rooms before, from time spent volunteering as a high school student, but I was on edge about my presence in the room. After all, I had never visited someone with a diagnosis, essentially, of “dying,” and placing myself so last minute into someone’s life felt somehow intrusive. The man, though, did not seem concerned by a new person being around. He did not really seem concerned about anything. I wondered if he had family coming to visit him, but, too terrified to say anything, I just stood awkwardly in a corner of his hospital room while his pancake mush got cold.

His eyes left the window and shot toward the clock on the wall.

“I always have my coffee at seven,” he reminded us.

The rest of the breakfast was a series of little battles between Yvonna and the man. She fixed his coffee, with milk but no sugar, and put a lid on it with a straw sticking through. He seemed unable to enjoy the coffee as much as he could have if he had gotten it at seven, but he drank it nonetheless. He made comments that only seemed loosely related to the present situation. Occasionally he asked if I could get him out of the hospital, but most of the time he talked and asked questions as if he was picking up in the
middle of a story, not realizing we had not been around for the beginning of it. He smiled and did not smile and his eyes seemed simultaneously vibrant and empty. When it was time for his medicine, he made a fuss but ultimately obliged, washing it down with a sip of coffee through the straw. While standing there, I could not fully grasp anything he said; it was both impossibly confusing and almost comprehensible, like reading a letter with all of the “a’s” and “the’s” removed.

When we finally left the room, I asked Yvonna what was wrong with him.

“He has COPD\(^1\) and congestive heart failure. But he is also a little confused.”

And that was that. Later that day, I would learn that “confused” is a term frequently heard on hospice wards, used to describe patients whose neurological processing has been compromised, but not severely enough to be qualified as dementia.

At the hospice department meeting, I sat on the periphery of a room filled with nurses and volunteers. Dr. Zermatt was both the only doctor and the only man in the room, a strange contrast with the male-dominated oncologist meeting. The group started the meeting with a prayer independent of any religion: no god was mentioned, and the prayer seemed geared toward the group itself, encouraging and motivating the caretakers to have strength and find peace in their work.

The meeting was an overview of all the patients currently in the hospice program at Middlesex. They were split into three categories: patients currently at the hospital, patients at home being cared for by volunteers and nurse visits, and patients who had died since the last meeting, a week before.

\(^1\) Chronic Obstructive Pulmonary Disease – a common cause of death in our country, characterized by low airflow into and out of the lungs. Subtypes of COPD include emphysema and chronic bronchitis (Zermatt 2013).
The deceased patients were discussed first. This discussion was different from any other discussion at any other department in the hospital: there was no mention of medicine, or diagnoses, or treatment, or money. Instead, the nurse closest to the patient would speak about the ease of the patient’s passing and how the family dealt with their grief. There would be a moment of silence, and then the next patient on the list would receive the same business-like eulogy.

When they started talking about the patients who were still alive, however, there was much more to discuss. The nurse would state the patient’s diagnosis, review the medicines they were taking, and summarize the most pertinent information relevant to their care. For patients at home, this would likely include how well their families took care of them. For patients in the hospital, they talked about sleeping habits, ability to eat and drink, and frequency of visitors. Often, the nurse would also speculate about how much time a patient had left, a number that was generally smaller for patients in the hospital compared to those who were not.

To help me keep up, I was given a piece of paper with all of the patients’ names and locations. Their diagnoses were omitted, so on the left-hand side, next to their names, I tried to jot down a couple of words describing the nurses’ summaries. By the end of the meeting, I noticed a pattern. Reading down the paper, the first three diagnoses I had written were: dementia, dementia, cancer. Then the next three: dementia, COPD & confused, Alzheimer’s. Three more after that: cancer, heart failure w/ confusion, dementia.

Almost every patient either had a dementia diagnosis or some other type of neurological impairment. Almost every one. The aging and dying brain was a very real
presence, and it would not be ignored. Until that point I had always considered death something that happens to the body, and while that does include the brain, it had always been something I had contemplated separately, high on its spinal pedestal, doing its best to hold the effects of aging at bay. For nearly a century, the average brain sends signals to keep the heart beating and the lungs breathing. Toward the end, it shoots motivational hormones at the expiring organs and floods the body with endorphins to combat the pain of dying. Then, finally, as the heart sputters out its final beat and the lungs exhale their final breath, the chaotic brain signals that had manifested as consciousness and living just…stop. The brain is silent. Its job is done. Somewhere well outside of its fluid-filled tomb, another brain will declare it dead.

But now I was faced with a seemingly obvious truth: far from ruling over all from well above the feeble body, the brain is a part of the body, and as the body begins to die, so too does the brain. And the diseases associated with a dying brain are far more numerous than I once realized.

**Part One: Dementia**

Death is a part of everyday life for Dr. Zermatt. Every day he says goodbye to patients he has cared for, and says hello to new patients for whom he will do the same. Of these patients, about one third are dying from metastatic cancer – the discussion topic for part two of this piece. Another third are dying from anything and everything else. The final third, however, are all dying of dementia.

“Dementia is their terminal illness; it is what qualifies a patient for hospice care. But it does not actually kill them, no; the complications from dementia are what
ultimately cause the patient’s death. Complications such as aspiration, pneumonia, urosepsis (a urinary tract infection that enters the bloodstream), a fall and a fractured hip...

**Dementia is defined by the DSM-IV as a loss of brain function that occurs with certain diseases. It affects memory, thinking, language, judgment, and behavior** (Alzheimer's Association 2013, 209). The criteria for dementia include a decline in memory and in at least one of the following cognitive abilities: “the ability to speak coherently or understand spoken or written language; the ability to recognize or identify objects; the ability to perform motor activities… and/or the ability to think abstractly, make sound judgments, and plan and carry out complex tasks” (Alzheimer's Association 2013, 209). Significantly, in order to qualify as dementia, these deficits must be severe enough to interfere with daily life (Alzheimer's Association 2013, 209-210). Cognitive impairment that does not meet these specific criteria is generally what merits a label of “confusion.” Dr. Zermatt quickly names a few diseases that lead to dementia: Vascular dementia, caused by strokes which create a problem in blood supply reaching the brain; Lewy-Body disease, the second most common type of dementia, which can mimic Parkinson’s in its symptomatology (Mayo Clinic Staff 2013); and Creutzfeldt-Jakob’s disease, famous because of its link to bovine spongiform encephalopathy, or mad cow disease (Evolutionary Analysis 2007). These are some of the better-known causes, but there are others, such as Lyme disease, vitamin deficiencies, and chronic alcoholism. (Also concussions and brain trauma, if we recall chronic traumatic encephalopathy as it was discussed in “This is Your Brain on Trauma.”)
And yet, all of the above-mentioned diseases account for less than half of known dementias. The most common type of dementia is Alzheimer’s disease, which research has determined as the diagnosis behind 70% of all dementia (Plassman, et al. 2007).

Figure 5: Overlap in symptoms and pathology of common dementias. Information for this chart came from Alzheimer’s Association Report (Alzheimer's Association 2013, 210).

According to Dr. Zermatt’s hospice breakdown, it would seem rational to hypothesize that 30% of our nation’s elderly suffer from dementia, which, when viewed through a narrow lens, is correct. For people who live to be 90 or older, 37% develop dementia (Plassman, et al. 2007, 127). But not everyone lives to be 90 years old, and dementia is a disease linked inexorably with age. Only 5% of individuals in their 70s develop any type of dementia, and of those in their 80s, 24% suffer from dementia (Plassman, et al. 2007, 127).

“[But] what’s the most rapidly growing segment of our population right now?” Dr. Zermatt asks, and then chips in before I can answer, “90-100 year olds. As the society ages and our average life expectancy gets older, we are going to see more and more…”
Death by dementia is a cruel fate. It is a slow death, sometimes lasting as long as fifteen years, usually lasting no more than seven. By the time friends and family realize what is happening, their loved one can be past the point of comprehending it. In this sense and this sense alone, the disease can be a comfort. To the sufferer, it is not painful. There is no acute awareness of their disintegration. But for the family, it is a nightmare. In the early days, before the disease is obvious, they must make sense of sudden personality changes: a proneness to anger, unexplainable confusion, a lack of interest in once beloved activities, and/or a tendency toward emotional outbursts (Nuland 1993, 93-95).

“Rage and improper emotion control are an aspect of Alzheimer’s,” Dr. Zermatt tells me. It is not a symptom people normally anticipate for a disease whose hallmark is forgetfulness, but this and other indicators are clear early signs of Alzheimer’s. “Patients in early Alzheimer’s stages can be overly sexual when they didn’t have a tendency toward that in the past, and some of them laugh inappropriately.”

Next, the trademark bouts of forgetfulness begin, and though the implications may be obvious, some people continue to deny it, and continue to hold out hope against the inevitable. But then the forgetfulness can become dangerous. Husbands who do not recognize their wives may take them for thieves and attack them (Nuland 1993). Many a demented individual has been found dead following a night exposed to the elements, unable to find their way home after blankly wandering away from their neighborhood.

As the forgetfulness sets in as a permanent state rather than as isolated incidents, the true pain of dementia begins. Mothers forget the names of their children. Men forget their wives after decades of marriage. The end-of-life care provided to dementia patients
by their families is among the most tragic – it is one thing to provide care to a loved one with whom you can share tender final moments, a completely different thing to provide care to a loved one who cannot acknowledge ever knowing you.

But still the incessant march of dementia is not over. In its final stages, the demented become completely unreachable, as if their brain, hermit-like, has shut down perception of the outside world. Lost inside of itself, the brain loses control of the body. Bowel movements and urination occur at random as the patient sits and stares vacantly, unable to grasp what has happened. They lose weight because they have forgotten how to eat. They become a husk. Sometimes, as described by Sherwin B. Nuland in his 1993 book, *How We Die*, patients exhibit excessive, obsessive, erratic behavior, right up until the very end:

And through it all [the patient] never stopped walking. He walked obsessively, constantly, every moment the ward personnel let him… Even when he was so weak that he could barely stand, somehow he found the strength to walk back and forth, back and forth, around the confines of the ward. When too exhausted to continue, he would stagger along until… the nurse grabbed his shoulders and eased him down into a chair, too winded and too weak to go farther.

Once seated, the frail body bent sideways because [the patient] hadn’t the strength to hold himself up any longer. The nurses had to tie him in lest he topple to the floor. And even then, his feet never stopped moving. Sitting there, unaware of the world around him, trussed into a chair by a sash around his waist, out of breath from the effects of his ceaseless effort, he would nevertheless keep moving his feet in a pathetic imitation of rapid walking. He was driven to do it, as if pursuing something he had lost forever. Or perhaps that wasn’t it at all. Perhaps something inside him knew the fate that awaits those who are in the terminal phases of Alzheimer’s disease, and he was running from it.

(Nuland 1993, 101-102)

Even as we are able to trace the course it will take from wellness to death, there is still much we do not know about dementia. Presently, there is not even a universal
method of laboratory diagnosis, i.e. a diagnosis based significantly on laboratory reports or test results (The American Heritage® Stedman's Medical Dictionary. n.d.).

“No specific blood tests,” says Dr. Zermatt, echoing that sentiment, “will say yes, you have dementia or no, you do not. Occasionally, a blood test will lead to an improvement…for example, they could have an underactive thyroid, and treatment of that will lead to an improvement of their dementia.”

“But that’s usually pretty rare, about one in 100,” he adds quickly.

Instead, dementia is a clinical diagnosis, obtained through careful interviews with the patient and their family. Dr. Zermatt typically performs what is called a Mini Mental Status Exam: a basic list of questions that illuminate how the patient processes information and how intact their short-term memory is.

An MRI can be a helpful tool for diagnosing vascular dementia by revealing small strokes that can go unnoticed, and a CAT or CT scan of the brain could help diagnose Alzheimer’s, but Dr. Zermatt usually does not depend on these imaging techniques unless the patient is relatively young. Looking at the brain of a patient with Alzheimer’s however, would usually reveal a significant amount of brain atrophy, with shrunken lobes and increased air and fluid beneath the skull.

![Image of brain scans](image_url)

**Figure 6:** “Alzheimer’s MRI” retrieved from Scitable (Nature Education n.d.).
But brain atrophy is not always directly correlated with Alzheimer’s. Says Dr. Zermatt: “There are some people who have a very normal-appearing brain and have dementia, and some people who have a very abnormal-appearing brain and function very well for a long period of time. “

As the hunt for a definitive laboratory diagnostic procedure continues, more and more is being learned about the mechanism of dementia, especially Alzheimer’s disease.

Alzheimer’s disease has small and humble beginnings. It starts, according to the most widely accepted hypothesis, with a single subunit of a single protein. The protein in question is called Amyloid Precursor Protein (APP), which is found embedded in the membrane of nearly every human cell (O'Brien 2011), projecting a long peptide chain into the extracellular space, like a great oar protruding from the hull of a Viking longboat. This APP is expressed at particularly high levels in the cells of the brain, where it is thought to be important for neuronal growth, synapse maintenance, and memory formation (Mileusnic and Rose 2010). In the membrane, APP is content; it does its protein job and lives its protein life. Nothing bad happens. The problem transpires when it is time for APP to be metabolized (broken down so new, freshly made APP can take its place). One of two things will happen. In the first scenario, two proteases\(^2\) (protein slicers and dicers) will approach APP in its membrane-bound home, and cut it twice, each protease making a single chop. The three pieces\(^3\) that were once APP drift off in different directions, a couple of them go and serve other functions, but ultimately they are degraded into their peptide components and recycled to make new protein. This pathway,

\(^2\) \(\alpha\)-secretase and \(\gamma\)-secretase

\(^3\) A p3 component, a soluble ectodomain (sAPP\(\alpha\)) and an intracellular C-terminal fragment (AICD)
called the non-amyloidogenic pathway, will not lead to Alzheimer’s disease (O'Brien 2011).

In the second scenario, proteases do not approach APP at the membrane. Instead, one of the most astounding capabilities of eukaryotic life transpires. The cell membrane, fluid and malleable as it is, starts to pucker. A great crater, with APP in the middle, forms from the outside and pushes inward. As the crater grows deeper, the walls start to close, forming a roof above the protein, enclosing it inside a bubble. In an instant, the APP is inside the cell. It has been absorbed through the process of endocytosis, and its new home (the bubble) is called an endosome (O'Brien 2011).

The proteases present in the endosome are different from the proteases at the cell surface. One of them, called β-secretase, cuts into the APP at the end of a region called the β-amyloid peptide (Aβ). The second protease, called γ-secretase, cuts at the beginning of the β-amyloid peptide. These two slices – harmless as they may seem – generate Aβ, a small peptide believed to be important for some aspects of cell health. But when overproduced, they can aggregate together to form extracellular plaques that decrease neuronal communication and block transmission of important nutrients. These plaques, called Amyloid β, senile, or neuritic plaques, are a key pathological feature of Alzheimer’s disease (O'Brien 2011).
Figure 7: This diagram depicts the Alzheimer's pathway for APP metabolism inside the endosome. As represented, the γ-secretase is capable of making one of two different cuts, yielding a peptide either 40 or 42 amino acids in length. Research supports that the Aβ42 is more dangerous than the Aβ40, capable of self-aggregation and recruitment of soluble peptides, as well as being acutely toxic to neurons. (O'Brien 2011; Price and Sisodia 1998; Ridge, Ebbert and Kauwe 2013).

But aggregating into plaques is not the only thing that the Aβ peptides do. They are also capable of indirectly adding phosphate groups to a microtubule-associated protein called tau. Microtubules are the highways of the cell, used by protein “trucks” to transport important materials from one location to another. These highways are of particular importance to neurons, which have microtubules stretching the length of their axons so the cell body can transport vesicles and other materials to the synapse. Tau promotes the construction and maintains structural stability of these highways (Iqbal, et al. 2009). When Aβ peptide stimulates the pathway that adds extra phosphate groups to tau, a process called hyperphosphorylation, tau undergoes a conformational change that leads to a new ability: for it to aggregate, much like Aβ, into deleterious filaments called neurofibrillary tangles. These tangles disrupt microtubule stability, making it almost impossible to send materials down to the synapse. The neuron starts to die. In fact, the number of tau tangles, not Amyloid β plaques, more directly correlates with the degree of dementia seen in a patient (Iqbal, et al. 2009).
If you were to take the brain of a deceased Alzheimer’s patient, fix it in formaldehyde, slice it into slivers thinner than a piece of paper, stain those slices with silver, and then look at this brain under a microscope, you would see brown splotches and black teardrops. This pathology, first seen by Alois Alzheimer in 1907, is Alzheimer’s disease (Gavett, Stern and McKee 2011). These plaques and tangles are the physical manifestation of the clinical symptoms.

![Image](image.png)

**Figure 8:** Amyloid β plaques and tau tangles (Rensberger 2010).

Over the years it has become more and more apparent that many diseases and disorders have a basis in our heredity, and Alzheimer’s is no exception. The allele (gene variant) of note is called APOE4 (apolipoprotein E4), and a copy of it significantly increases an individual’s chance of developing Alzheimer’s (Alzheimer’s Association 2013). In fact, genotypes that are heterozygous for APOE4 and its benign counterparts, APOE2 or APOE3, have three times the risk of developing the disease. For APOE4 homozygous individuals, the outcome is even bleaker, with a 12-fold increased risk (Bell, et al. 2012).

Recent research has shown that the primary function of the Apoe protein is to regulate cholesterol and other lipids in the body, as well as maintain the integrity of the blood-brain barrier (Bell, et al. 2012). Studies in mice have demonstrated that the high-
risk variant of the gene, APOE4, actually increases the chance that the blood-brain barrier will become damaged (Bell, et al. 2012). With a compromised barrier, the ability for the brain to distinguish between needed nutrients and harmful substances is significantly lowered. Further studies indicate that the APOE4 allele is linked directly to increased brain Aβ deposition, as E2 and E3 variants (but not E4) are capable of binding Aβ tightly and are therefore better at removing it from the brain (O'Brien 2011). Although its connection to amyloid β plaques and overall brain health is clear, is still not evident exactly how the APOE gene plays a role in the formation of Alzheimer’s disease.

To further complicate the issue, there are some experts who think that the pathology of Alzheimer’s does not start with Aβ at all. These scientists think that the disease begins with mitochondria, the adenosine triphosphate (ATP) synthesizing microorganisms within our cells, without which we would be unable to break down the glucose we eat into usable energy. These mitochondria undergo age-related changes that experts believe may propagate the formation of Amyloid β plaques (Kopeikina, et al. 2011; Seo, et al. 2011). Some even go so far to say that mitochondria, with their steady production of free radicals, are responsible for aging itself (Ridge, Ebbert and Kauwe 2013).

Despite the fact that we have many strong theories and support for various mechanisms of degenerative Alzheimer’s,² we have yet to develop anything that resembles treatment, let alone a cure. Current Alzheimer’s research is looking into numerous potential therapies, including inhibition of the Aβ forming pathway or

² For the purpose of this thesis, I speak only of Late Onset Alzheimer’s disease. Early Onset Alzheimer’s, a rare (accounting for either 0-1% or 6-7% of all dementia diagnoses) and almost entirely genetic disorder, follows a different mechanistic route (Ridge, Ebbert, & Kauwe, 2013).
mechanisms for clearing these pathologies once they have accumulated (Kitazawa, et al. 2011; Yao, et al. 2011).

One of the most important things to understand when talking about dementia is that dementia itself is not a disease. It is a symptom, an umbrella term to describe a variety of diseases and conditions that develop when the nerve cells of the brain start to die (Alzheimer's Association 2013). Dementia is the hallmark of a dying brain. Its implications are brutal and various, and not just limited to loss of memory. The entire brain carries the weight of dementia, which is what leads to the variety of other symptoms described in its definition.

As Alzheimer’s progresses, it brings with it a range of different experiences. “Sometimes,” says Dr. Zermatt, “Alzheimer’s patients continue to eat until they get close to dying, continue to say a few words, and continue to be very happy and content in the moment. Other times there is agitation, restlessness, and anxiety, and oftentimes I think that is somewhat dependent on how [their loved ones] react to them…”

“The patient,” he continues, “often doesn’t suffer as much as those around them; because they don’t realize it. They can be very happy in the moment, even a couple of weeks before they die. Not all of them are, but they can be. None of us want to be demented, I mean, we all want to live as fully as we can until the day we die, but one of the reasons we don’t want it is because most of us don’t want to be a burden on our kids and families and those who are left behind.”

The dementia patients that Dr. Zermatt most often sees are the ones he treats through hospice, and patients are admitted to hospice care based on their functionality, as opposed to their diagnosis. Because of this, his patients are almost always at the latest
stage in their disease: They are bedridden, incontinent (both bowel and urine), hardly able to speak, and have either forgotten how to swallow or can only swallow with difficulty. Dr. Zermatt has compared the end-of-life care for a dementia patient to taking care of an infant. The correlation is so strong that the analogy can be used as a prognostic tool: the more infant-like the patient, the more their dementia has progressed.

But despite this correlation, caring for a baby and caring for the elderly are perceived very differently. “As a society,” says Dr. Zermatt, “we are much more tuned to taking care of babies and the joy that goes along with that, and tend to be hands off toward the elderly and demented and not give them the same care that we give infants.”

It is a sad truth, one that plagues us more and more as parents live longer lives and their children live busier ones. Dr. Zermatt finds this to be one of the biggest tragedies of dementia, especially because he has seen how much good a loving family can do.

“If you’ve got a family that is really loving and caring and relaxed and not too anxious, that relaxation and calmness gets through to the patient and perhaps they are a little bit less agitated or angry than they would have been otherwise.”

Most often, Dr. Zermatt’s dementia patients die quietly and calmly, and their families usually have done a great deal of anticipatory grieving. This type of grieving is possible because of the normally slow progression of dementia. “This gives the family a lot of time to deal with the grief, to realize that the patient is dying, and to focus on keeping the patient comfortable.”

Usually, Dr. Zermatt reports, the families are relieved to see their loved one go. No one should be blamed for feeling that way; in fact, hearing this information inspires an insight about dementia usually reserved only for those who look in as it advances.
The saddest thing about the disorder is how it affects someone without them realizing. This, of course, means that there is no sadness felt by the demented, for how could they be sad if they are unaware of their condition? Only the clear of mind can truly bear witness to this death, a death made bitter because the dying leave slowly, over time, as they forget themselves.

Relief at someone’s death, as strange a concept as it may be, is a natural and expected emotion. People suffering from advanced dementia are no longer themselves, and the only remedy for this great loss is death, which, in this case, is perhaps a lesser evil. Dr. Zermatt has been in this business for so long that the concept of death as relief is not foreign to him. His job is a strange one, no doubt, especially because it seems perfectly at odds with the normal intentions of a doctor. From what I could tell, most doctors operate in denial of the inevitable, with every intention to fix what is broken, to cure what is sick, and to send their patients off towards blissful and healthy lifetimes.

Dr. Zermatt, however, works constantly within the glare of death. Every patient he cared for he did so with the knowledge that their bodies were not long for this world; that he was their last stop before an unknown eternity. He knew, without a doubt, that every single one of his patients would die. And yet, I could imagine there being a strange and elusive comfort in this profession. As a doctor for the living, you deal with grief, with pain, with destroyed dreams and ill-advised hope. As a doctor for the dying, you deal with relief.

“Relief” has roots in the French relever, which means “to lift up, to remove” (Oxford University Press 2014). The word “death,” however, means “the act or fact of

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Inspired by Dr. Rita Charon’s TEDx talk “Honoring the Stories of Illness.”
dying; the end of life; the permanent cessation of the vital functions of a person, animal, plant, or other organism” (Oxford University Press 2014).

In this sense, death is a ceasing to live, but relief is ease; it is a lifting; it is a simple disconnection from the body. I think, given these meanings, I prefer to think of all death, especially death of the human consciousness, as nothing short of relief.

**Part Two: Cancer**

Along with the people under hospice care dying from dementia, an equal number are dying from metastatic cancer (cancer cells that have traveled from the original primary site and started multiplying in other organs). Cancer remains one of the largest causes of death in America, with over 1.6 million dying from all cancers in 2012 (American Cancer Society 2013). The vast majority of these deaths occur in people over the age of 65, a statistic that reveals a characteristic of cancer that is much like dementia: If you live long enough, you dramatically increase the likelihood of developing it.

In his biography of cancer, *The Emperor of all Maladies*, Siddhartha Mukherjee describes cancer as follows:

Cancer, we now know, is a disease caused by the uncontrolled growth of a single cell. This growth is unleashed by mutations – changes in DNA that specifically affect genes that incite unlimited cell growth. In a normal cell, powerful genetic circuits regulate cell division and cell death. In a cancer cell, these circuits have been broken, unleashing a cell that cannot stop growing…Cancer cells can grow faster, adapt better. They are more perfect versions of ourselves.  
(Mukherjee 2010)

Only about 1.4% of deaths from cancer are due to brain and nervous system cancers. In general, brain cancer is much more rare than most other cancers. But nearly 15% of all cancer deaths in 2012 were a result of cancers of the respiratory system
The two do not seem at all correlated at first glance, but the lung and the brain are actually quite similar in their spongy, malleable texture. Due to these similarities, the development of cancer in the brain from cells that have traveled from other primary sites occurs most often with lung cancer. With close to 170,000 people developing cancerous brain metastases every year (American Cancer Society 2013), the prevalence of lung cancer cannot be ignored when considering the health of the nervous system. In fact, brain metastases are the most common form of brain tumor, and it is estimated that “there are more metastatic than primary malignant brain tumors per year” (American Brain Tumor Association 2014).

![MRI images of brain metastases](image)

**Figure 9:** “Multiple metastases to the brain from breast primary” (Elmasry 2009)

Dr. Zermatt tells me about a 52-year-old woman currently on the hospice unit. Last year, she was diagnosed with aggressive colon cancer. After unsuccessful treatment, the cancer spread to her liver. About a month ago, it made its way up to her brain.

“It means her time is very limited. Very, very limited. Usually there isn’t a lot of quality time left after that.”

Although brain metastasis from lung cancer is most common, it can occur with colon, breast, skin, and kidney cancers as well (National Brain Tumor Society n.d.). In fact, cancer from just about any primary site can lead to metastatic brain tumors, and 20-
40% of all other cancers will eventually develop into brain metastases (Glass-Macenka 2013).

The ability of cancer to metastasize is perhaps its most intimidating feature, and in late-stage cancers, it is almost a guarantee. In order to travel, “the tumor must erode through the wall of a blood vessel or lymph channel, and then some of its cells must become detached and pass into the flowing stream. Either individually or clumped into an embolus, the cells are then carried to some other tissue, where they implant and grow” (Nuland 1993, 215).

Forming a metastatic tumor is not an easy task for a few drifting cancer cells. If it were, cancer patients would be riddled with metastases as soon as their primary tumors became bulky enough to cast cells into the lymph or blood stream. However, the body has many defenses that prevent this from happening. A cancer cell could easily die on its voyage through the circulatory system, either by the sheer physical stress of tumbling through the pushing, pumping vasculature, or at the hands of ever-vigilant immune cells, for which a nomadic cancer cell is easy prey (Nuland 1993, 215-216).

Even on the off chance that a hardy cancer cell survives this perilous journey and settles in a distant organ, it is still not guaranteed success. To form a prosperous colony, it needs nutrients, and to get nutrition, the cancer must be capable of stimulating blood vessel growth, hijacking the body’s own nourishment system to sustain its own, malignant needs (Nuland 1993, 216).

Once it takes hold, a metastatic tumor interferes with the local functions of whatever organ or tissue in which it takes up residence. With enough metastases, these local interferences lead to full body malfunction and, ultimately, death.
When cancer metastasizes to the brain, rapidly multiplying tumor cells press into, irritate, and destroy normal brain tissue. The patient, who has already been dealing with the fatigue, pain, and nausea normally associated with cancer and chemotherapy, suddenly presents with new symptoms, including headaches, seizures, speech problems, weakness, blurred vision, numbness, difficulty moving, paralysis, and vomiting (National Brain Tumor Society n.d.).

Because brain tumors interfere with the function of our centers of intelligence, the symptoms of such tumors are not limited to physical manifestations. These metastases also lead to mental changes, including impairment of memory, reading abilities, and/or speech production (National Brain Tumor Society n.d.). “Brain metastases are sad,” confesses Dr. Zermatt, “Sometimes, it’s almost like the patient is having a stroke. They get a real weakness on one side of the body, they’ll get severe headaches, and they will become a different person from who they were before. There is a vagueness about somebody with brain metastases.” More often than not, a patient with brain metastases will present with any number of these symptoms, but about one-third of patients diagnosed with metastatic brain cancer have no symptoms at all (National Brain Tumor Society n.d.).

Despite the “death sentence” reaction that usually accompanies a diagnosis of cancer, the treatments available have actually helped extend lives far longer than had previously been possible. However, Dr. Zermatt tells me that treating most cancers is still a game of “how many years can I add to this person’s life?” as opposed to “how can I cure them forever?”
It is for this reason that seemingly cured cancers are given a status of “remission;” it forces both doctor and patient alike to remain vigilant and remember that cancer is nothing if not a sleeping monster. But despite this cautionary warning, it is hard not to be optimistic. After all, with increases in understanding and improvements in medicine, life expectancy continues to rise for even the most serious, active cancers (Zermatt 2013).

“Because of chemotherapy, and surgery, and radiation, and the other treatments that are out there, cancer is really becoming more of a chronic illness.”

The survivability of cancer has brought with it new, unpredictable problems, some directly dependent on patients living longer than they did before. One of the more common of these problems is a strange condition Dr. Zermatt referred to as “chemo brain.”

This condition shows up mostly in patients who are surviving with cancer and have undergone chemotherapy treatment. They report what physicians call “mild cognitive impairment” and laypeople call “cloudiness,” which includes symptoms such as memory lapses, difficulty concentrating, difficulty remembering details or common words, an inability to multi-task, and slower information processing (American Cancer Society 2012). Dr. Zermatt reports his patients have “a sense of vagueness” about them, and “difficulty with short term memory.”

“It’s usually not progressive,” he says, “thank goodness.”

The condition came as a surprise to many oncologists who initially believed that chemotherapy drugs could not pass through the blood-brain barrier. But with the high prevalence of cancer and the continued use of chemo, the effects have become impossible to ignore. Chemo brain manifests itself most often over the course of chemotherapy and
remains for a period of time after the treatment has stopped. Fortunately, for most people the effects are transient, but sometimes a patient experiences long-term mental changes. Though it is familiar to both patients and oncologists, studies to help explain chemo brain have only recently gained ground (American Cancer Society 2012).

Imaging tests have revealed that in some post-chemotherapy patients, certain brain areas have actually shrunk, including the areas that deal with memory, planning, initiating movement, monitoring thoughts and behavior, and inhibition. In general, these problems were more common with patients who received higher doses of chemo (American Cancer Society 2012).

Furthermore, MRI images of the brains of breast cancer survivors treated with chemotherapy revealed a lower resting brain activity when compared to those patients who had not received chemo. These global brain changes were still present in some women 5 or 10 years after their treatment ended. During a memory test, women with these changes needed to call upon and use larger areas of their brains to answer questions than women who had not gotten chemo (American Cancer Society 2012).

However, some studies counteract the idea that chemotherapy causes lasting brain changes. A small 2012 study examined women who had recently undergone surgery for breast cancer but had yet to start any chemo treatment. About 25% of them already showed a lack of proficiency with word skills, and 14% of them had problems with memory. So some of the problems being attributed to chemo brain could actually be due to another factor entirely, including the cancer itself, high stress levels, low blood count, or fatigue (American Cancer Society 2012).
Further inconclusive data includes the statistical estimates of how many people will get chemo brain along with their chemotherapy. One expert put the range at 15-70%, another attested that the maximum could be no more than 50% (American Cancer Society 2012).

Interestingly enough, there is some support for a genetic correlation with an increased likeliness to develop long-term chemo brain. The gene is a familiar one, APOE, the same one implicated for increased risk of developing Alzheimer’s. In fact, the same E4 variant is what puts post-chemotherapy cancer patients at higher risk for developing chemo brain (Ahles 2003).

Regardless, chemo brain only matters if the patient is going to live.

“If they are going downhill more rapidly, there would be other things that come to the surface that they would have to deal with first,” Dr. Zermatt tells me.

By other things, he means the pain, discomfort, and severely disrupted quality of life that come from aggressive metastatic cancer. Once enough metastases have taken hold, the patient only has a matter of time. At this point, hospice focuses on giving them a “good death,” one marked by “the most dignified exit possible by offering the dying person care, affection, and companionship; the truth about diagnosis; and the maximum personal control over the final phase of life” (Berk 2010, 505).

It is hard to say what will finally bring about a cancer patient’s death, because tumors do more than impede normal functions. Indeed, by cancer’s unchecked growth alone, “tubular organs are obstructed, metabolic processes are inhibited, blood vessels are eroded to cause minor and sometimes major bleeding, vital centers are destroyed, and delicate biochemical balances are deranged” (Nuland 1993, 217), but cancer does not
stop there. Because it consists of immortal, replicating cells, cancer is always hungry, and satiates this appetite at the expense of its host. The body grows weaker and weaker from malnutrition driven by the perpetual growth of malignancy. But it keeps fighting back. Perhaps in an attempt to starve the tumor, white blood cells send hormones to the brain’s feeding center, decreasing appetite. In order to hold off its own starvation, the body seeks out alternative energy sources, which it finds in the proteins of the muscle. The metabolism breaks these proteins down, and the muscles waste away. The patient loses weight rapidly (Nuland 1993, 218).

But cancer has a bigger arsenal, and well-established metastases are brutal and unstoppable. Tumors release chemicals that supress the immune system, an open invitation for infectious bacteria and viruses to colonize the susceptible body. Many cancer patients die from these secondary infections (Nuland 1993, 218).

Near the end, the patient is too weak to even cough. The cancer has seized control of all resources. Blood supply commandeered to nourish the metastases decreases the amount of blood circulating throughout the body, and organs start to fail from lack of oxygen. This causes many patients, especially older patients, to die from stroke or heart attack (Nuland 1993, 219).

A cancer patient, starved, oxygen-deprived, and ghost-like, may spend their final hours with deep and gurgling respirations, a sound so haunting that it has been christened a “death rattle” (Nuland 1993, 219). When the sound finally stops, signifying that the patient has died, those surrounding exhale with relief. The cancer, after what may have been months or years of greedy destruction, has finally lost, killed by its own ruthless consumption.
Part Three: At Death

Death proceeds through three stages. The first is the agonal phase, from the Greek word *agon*, which means “struggle.” The dying will gasp, and their muscles spasm, an indication that the heartbeat is shuddering and giving out. The second stage is clinical death. There is no heartbeat, breathing, or brain signaling, but the patient can be resuscitated. Finally, after a short time, they pass into biological death, the point of no return (Berk 2010).

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As I stand waiting for the elevator on the seventh floor of Middlesex Hospital, I think about my own brain, as I had many times before when I felt particularly philosophical. I imagine it sitting diligently inside my skull, memorizing to do lists that I give it and moving my muscles when I ask, recalling the names of familiar faces that I see and finding the appropriate words for emotions that I feel. The brain does so much so effortlessly that there is no easy way to imagine it failing, whether it be from either a build-up of plaques or an invasion of tumors. It is hard enough to imagine losing your memory, harder still to imagine losing your personality, and mostly impossible to imagine not being fully aware of any of it.

Imagine a healthy, functioning brain, the brain of someone who is not plagued with any of the fractures I have outlined in this thesis. That brain, as it dies, will become an embodiment of the malady, disease, disorder or condition it could have suffered from in its youth. The dying brain, like the bipolar brain, can be prone to great fits of mania or depression. It may suffer from absent-mindedness like its concussed counterpart; it loses
control of the body, slowly, like the late stages of multiple sclerosis. A brain, close to death, feels anxious and cannot eat.

We cannot forget that all of our brains are dying, and that mental disorders or neurological illnesses, if they can be accepted as insights into a dying brain, are really just insights into our own potential futures.

But as the elevator dings to indicate the end of my visit to hospice, these thoughts promptly drop from my mind, such forgetfulness a common side effect of living.
References


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Concluding Remarks

There are many ways to explore the broken brain. It can be studied in a lab, discussed in a doctor’s office, or read about in books. I selected interview as my method of exploration. By using this medium, I was able to hear first-hand accounts about living with mental illness. I met real people with real stories.

Looking back on the interviews and research I conducted, it is apparent that stigma is a pervasive force that affects the broken brain. Sometimes, overcoming this prejudice can be an even greater challenge than overcoming the illness itself. But by addressing stigma and writing to reveal the truths of neurological disorders, the stereotypes and silence that surround these ailments may slowly melt away.

People who suffer from chronic conditions, like Carolyn, can begin to address their illness without worrying about others feeling uncomfortable, treating them differently, or considering their diagnosis with skepticism. For someone like Maggie, who suffers from an injury that society perceives as “normal,” clarifying the realities of that injury will eliminate the dismissive reactions it garners. Situations of self-stigma, like Carl’s, will greatly benefit from more understanding; if the brain is treated just like any other organ, than when it breaks, sufferers will not feel blameworthy. If we can defuse stigma, we will create more stories like Laura’s, which end with successful treatment and healing.

Narrative, I have learned, has great value as a therapeutic tool. The desire to communicate arises alongside many of these injuries, and narration possesses enormous potential as an outlet for people who feel like they don’t have a voice. Understanding this
concept has influenced the way I want to practice medicine, and because of this project, I plan to employ narrative as a remedial tool in my future practice as a physician.

As promised, each chapter of this thesis told a different story. In “My Brain is a Mess,” Carolyn talked about learning to live with a chronic condition. She spoke about her greatest difficulties, her biggest worries, and many sources of stigma that she had to overcome. Much about multiple sclerosis is still a mystery, especially to those who study it, but that does not mean we can only consider it as a liability. It is, at least in part, a manageable condition, and Carolyn’s story emphasizes the need for comprehending it as such.

“The Precipice” examined anxiety and eating disorders, and how these common and often misunderstood illnesses are mendable given the proper environment. Laura emphasized the need for people who suffer from these types of disorders to seek help, dispensing with the common belief that feelings of anxiety or depression will “just go away” and that people who suffer from eating disorders can “just eat something.”

Maggie and Rick spoke about concussions in “This is your Brain on Trauma.” Presently, concussed athletes are only removed from play until their symptoms subside. Concussions are treated casually, like normal wear-and-tear. But, in light of current research and stories like Maggie’s, such practices must change. Even mild concussions should be treated as brain injuries that can result in lifelong brain changes and affect cognitive abilities. As Rick so aptly put it, “it’s about return to life,” not return to play.

“The Man with Two Faces” explored bipolar disorder and how unsettling it can be to live with a broken brain. Carl lives his life seemingly frozen in place, as if his disorder, too strong to suppress, has taken over. He has remained stagnant, without
genuine respite, shifting ceaselessly between two extremes. His story raises many questions about the possibility of healing after living with a confusing and difficult disorder for close to two decades. Is it possible for Carl to relax the grip of his illness? Will he ever find happiness, despite his regrets? Can he learn to forgive himself?

Finally, I concluded my exploration of the broken brain with “The Dying Brain.” After examining some of the maladies that can plague the brain as it lives, it seemed fitting to end with an examination of the deterioration that can cause its death. Whether the brain dies by Alzheimer’s disease, cancer, or some other means, it will eventually die. Dr. Zermatt and I discussed the burden a dying brain can be, and how it is sometimes difficult for the living to accept that burden. “No one wants to be demented,” he says, and it is easy to see why.

After all, we do not actually love with our hearts or make decisions with our gut. We do not believe things with our souls or feel apprehension in our stomachs. It is the brain from which these human qualities emanate. We do not pass judgments with our eyes, write poetry with our hands, or sing songs with our mouths: the body is just a vehicle through which the brain acts. Everything that has ever been invented was first an idea inside a brain. Every abstract notion – like destiny, or time – has arisen from the squishy, gelatin-like, fluid-covered organ in our skulls.

I suppose this assessment provides a reason for why the broken brain can be so terrifying and misunderstood. The brain, after all, did not evolve to look at itself. It evolved to interact with and react to the physical world. So, in order to comprehend the wounded brain, we must turn our sights inward, focus on the structure whose job is to focus on everything else, and request that it consider its own weaknesses.
This thesis, a narrative of neurology, endeavored to do just that. Conducting interviews, telling stories, and enhancing that narrative with current research has been my approach to clarifying neurology. I offer small insights into this substantial issue, and there is much work to be done if we strive to fully expose the brain and all its maladies. But with a collection of little lights, like mine, shining into the darkness, the broken brain can be illuminated, pulled from obscurity, and suffered in silence no more.