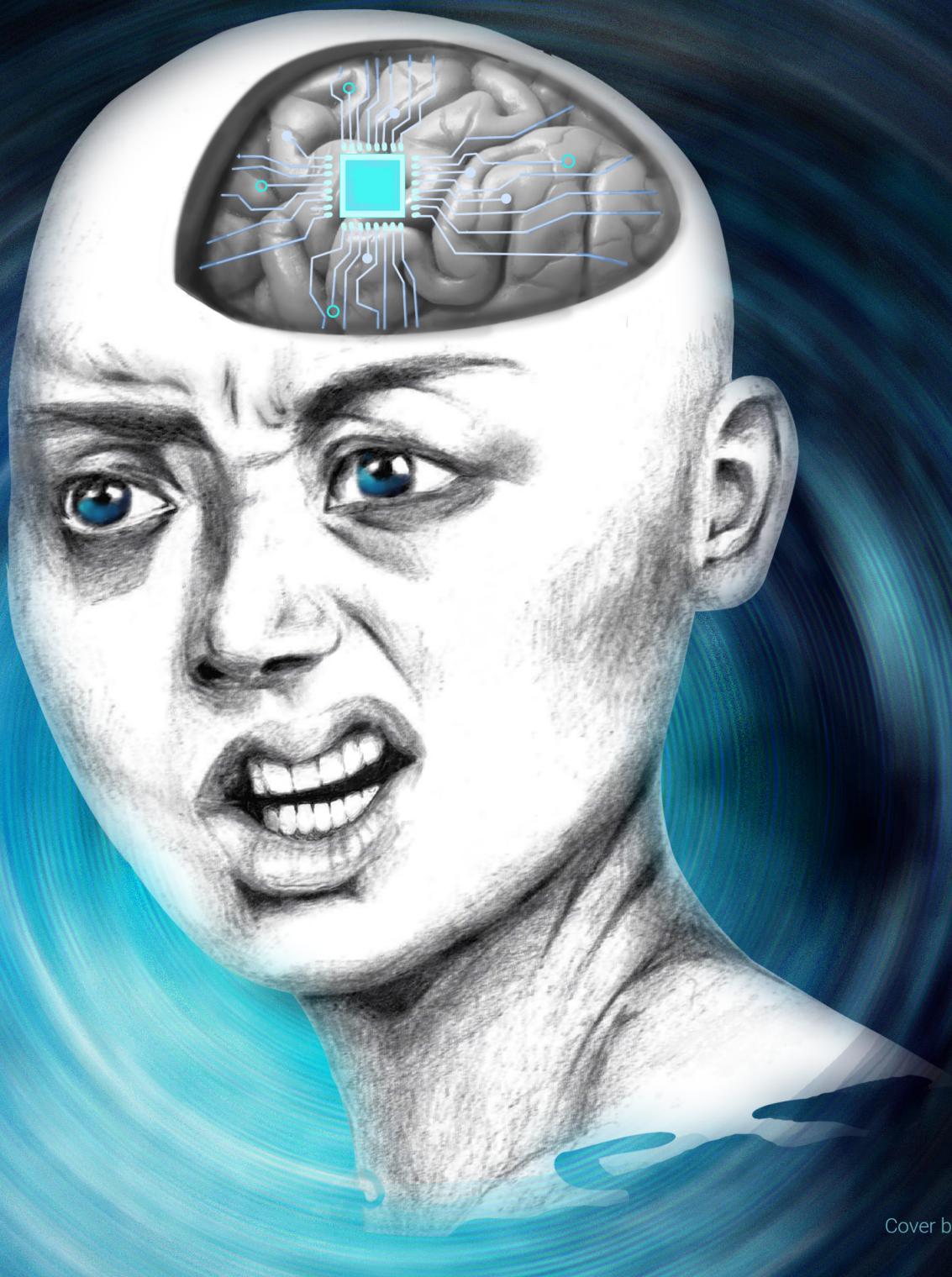
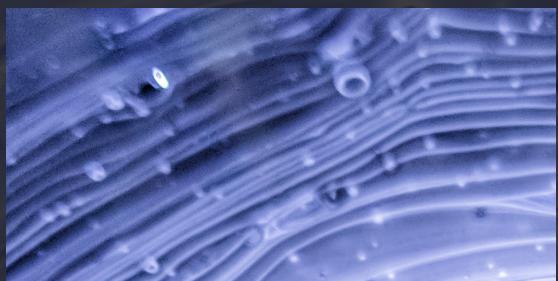
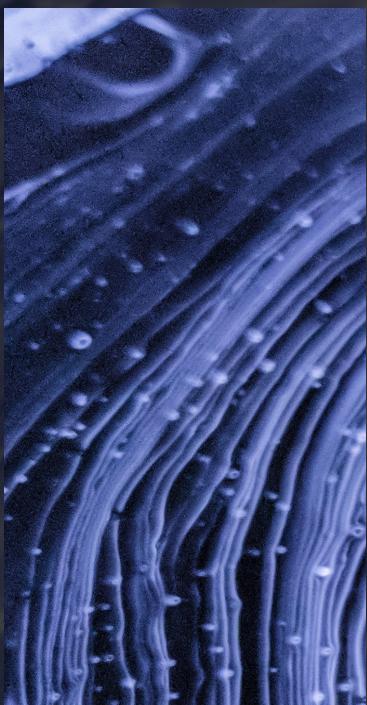


mind

Issue 8: Mindf*ck | Fall 2022



Cover by Hilary Lu



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Letter From the Editors

Dear Reader,

Fall 2022 could not have gotten off to a better start for Neurotech@Berkeley's Publications Division. After receiving nearly 40 applications to the division, we welcomed 12 talented new members to work on writing, podcasting, and design. In addition to finishing and publishing our Spring 2022 edition of Mind, with the help of our Operations Division, we crossed 14,000 followers on our Medium blog. Then it was time to pick a new theme for the Fall 2022 edition of Mind...

Mindf*ck was not the editors' first choice for the theme of this issue. In fact, it was not our second choice. Or third choice. Or fourth, or fifth, or six, or seventh— (you see where this is going). But, of course, when our writers voted, it was Mindf*ck that emerged on top of twenty other ideas. We were no doubt concerned. What would the rest of the club think? How would we come up with unique articles?

However, our concerns never materialized. In this issue you will find a myriad of insightful work stretching across all that neuroscience and technology has to offer, whether abstract or

concrete. Stories in this issue truly encapsulate what makes studying the brain such a mindf*ck. We hope these pieces will not only bend your mind, but pique your interest on subjects from the confounding dying brain to artificial intelligence systems just in need of a good nap.

It has been our pleasure to lead the Publications Division and a privilege to learn from our passionate members each week. We would like to take this time to truly thank all of them and you, the reader. Thank you for continuing to support our articles and goals to create an accessible, free space to teach and learn all there is to know about the brain, mind, and technology.

With that, we are proud to present Mind, Issue 8: Mindf*ck...

Sincerely,
Jacob Marks & Mary Shahinyan

Before You Go: The Cognitive

By: Catherine Duan

The Experience

It's 3 AM when you finally escape the agonizing confines of Moffitt Library, eyes bleary from hours of arduous study. Your mind is consumed as you cross the street, trepidation for your upcoming midterms fusing with tranquilizing exhaustion to produce that uniquely numbing headache that every Berkeley student knows when suddenly—

The night is split by a screeching sound, the blaring of a horn. Time slows as you briefly register the flickering of traffic lights, muffled shouts from the sidewalk, and at the edge of your field of vision, something you can't quite make out, something hurtling towards you at an impossible speed. The last thing you see is a leather glove, a face under a helmet, the shock in the man's eyes mirroring your own.

Everything fragments.
And then there is nothing.

When you next awake, it is to the rolling squeak of a stretcher as EMTs rush you towards the operating room, fluorescent lights glaring overhead. Echoing voices fade around you as your consciousness ebbs and flows.

You find yourself jerked out of your body, drifting through a dim tunnel towards an incandescent brightness, the kind that suffuses your entire being with an ineffable warmth and purpose. As scenes from your life flicker before your eyes, you're flooded with an incandescent sense of *light*—the sense of existential peace that can only result from the knowledge that *everything will be ok*.

You, like 4-15% of the population¹, have just encountered a near-death experience (NDE), a term first coined by psychiatrist Raymond Moody in 1975 as an umbrella term for the set of profound, psychological experiences commonly occurring in life-threatening conditions², particularly those involving severe neural or cardiac injury in which awareness should be impossible³. Your symptoms—out-of-body experiences, movement through a tunnel, terminal lucidity, and life-review⁴—have been observed numerous times in circumstances transcending individuals, cultures, religious affiliations, and historical eras.

Characterized by their transformative defiance of egotic and spatiotemporal barriers⁵, NDEs are unique among states of consciousness by virtue of their profound and lasting transformations of personality⁶, with many subjects reporting dramatically evolved outlooks on human relations, spirituality, life and death⁵. Even more striking is the occurrence of veridical NDEs, in which people reportedly out-of-body have observed events or gathered information that was later verified by others upon the patient's return to a conscious state⁷.

But just what makes these transcendental events possible? How can the labyrinthian network of the human brain, even on the brink of death, produce such electrifyingly ethereal experiences?

Neuroscience of a Dying Brain

The Explanations

The earliest known description of an NDE was recounted by Plato in his novel *The Republic*, written in 420 BC. In it, he tells the story of Er of Pamphylia, a soldier who reportedly dies in battle and descends to the underworld, returning to life with descriptions of the celestial spheres of the astral plane¹. Since that time, scientists and civilians alike have attempted to identify the origin of NDEs, with contributions deriving from neurology, medicine, psychiatry, parapsychology, anthropology, theology, and philosophy⁸, and explanations largely falling into one of three categories.

- 1) *Spiritual* theories assume that consciousness can become detached from the neural substrate of the brain and that the NDE may provide a glimpse of an afterlife⁹.
- 2) *Psychological* theories include the proposal that the NDE is a dissociative defense mechanism that occurs in times of extreme danger or, less plausibly, that the NDE reflects memories of being born⁹.
- 3) Finally, a wide range of *organic* theories of the NDE have been put forward including those based upon altered blood gas levels, endorphins and other neurotransmitters, and abnormal wave activity⁹.

i. Susan Blackmore's "Dying Brain Hypothesis"

In 1993, British parapsychologist Susan Blackmore published a novel, *Dying to Live: Science and the Near Death Experience*, setting forth what has since become known as the "Dying Brain Hypothesis"¹⁰. Essentially, Blackmore contended that all NDEs were purely artifacts of neurophysiological processes, the final visions produced by a massively disinhibited and dying brain¹¹. She defended her position with the use of two core pillars—the "Consistency Argument" and the "Hallucination Argument"—which argue that 1) the consistency of the NDE derives from the fact that everyone has a structurally similar brain that mediates the experience of dying in much the same way, and 2) all significant features of the NDE can occur in the absence of a near-death crisis, meaning that they could only be the result of neurophysiological processes, rather than of dualist or spiritual ideas¹¹. Since its proposition, the Dying Brain Hypothesis has drawn wide criticism from scholars maintaining that Blackmore's fiercely skeptical and materialist views are far too reductionist¹², and that her fragmentary models for the behavior of dying brains fail to explain many primary global characteristics of NDEs, such as veridical NDEs¹³. Thus, for near-death scholars today, its significance lies purely in the fact that it was the first widely-considered theory to include only naturalistic and neurological explanations for this enigmatic phenomenon.

ii. Altered Blood Gas Levels

Also in the realm of purely organic explanations, a number of theorists have considered the possible role of abnormal blood oxygen and CO₂ levels in NDEs, with particular attention given to the conditions of hypoxia/anoxia and hypercarbia⁹.

Cerebral hypoxia and anoxia are defined as the conditions in which oxygen supply to the brain is

(decreased, with many symptoms mirroring those of NDEs¹⁴. In 1997, researcher James Whinnery noted that there are indeed many similarities between NDEs and g-force induced loss of consciousness (G-LOC) episodes, which often occur in fighter pilots engaged in maneuvers that can result in a loss of adequate blood supply to the brain. Based on observations of almost 1000 episodes of G-LOC, he noted that such episodes often involved “tunnel vision, floating sensations, out-of-body experiences, vivid dreamlets of beautiful places, psychological alterations of euphoria and dissociation, inclusion of prior memories and thoughts, confabulation, and a strong urge to understand the experience¹⁵. ” Whinnery’s analysis launched cerebral anoxia to the forefront of NDE studies for a short time before subsequent studies revealed that many NDEs occur without either hypoxia or anoxia¹⁶, thereby discrediting his theories.

Additionally in 1997, psychiatrist Karl Jansen proposed hypercarbia (increased levels of CO₂ in the bloodstream) as another potential explanation for NDEs, noting shared symptoms such as out-of-body experiences, relived past memories, mystical experiences, and bright lights. He reasoned that hypercarbia was empirically shown to release a flood of glutamate, overwhelming N-methyl-D-aspartate (NMDA) receptors and resulting in neurotoxicity¹⁷. To this end, Zalika Klemenc-Ketis studied 52 cardiac arrest patients for NDEs, finding that those with higher arterial blood partial pressures of CO₂ experienced significantly more NDEs, and concluding that hypercarbia was, in fact, a reliable independent predictor of NDEs¹⁸.

iii. Neurochemicals

Based on such symptoms as euphoria and dissociation, many theorists have set forth a possible role for neurotransmitters—most frequently endorphins or other endogenous opioids released under stress—in the generation of NDEs¹⁹. In 1982, endocrinologist Daniel Carr noted that many characteristics of NDEs were synonymous with those of a “limbic lobe syndrome” associated with an excessive release of beta-endorphins and enkephalins²⁰, a suggestion that would later be corroborated by the more comprehensive neurobiological model put forward by Juan Saavedra-Aguilar and Juan Gomez-Jeria in 1989²¹. In both instances, researchers observed that endorphins are released under conditions of stress, and are correlated with a reduction in pain perception as well as a pleasant, even blissful, emotional state⁹. Consistent with the hypothesis that endorphin release may be possible for the euphoric state achieved through most NDEs are the 1983 findings of IR Judson and E. Wiltshaw, both of whom recorded occasional reports of pleasant NDEs changing into unpleasant “hellish” experiences upon administration of endorphin-blocking drugs such as naloxone²². Subsequent theorists, however, have noted that endorphin-associated pain relief and euphoria tend to last for several hours, whereas the peace and cessation of pain in NDEs are often only a few seconds¹⁹.

Since then, various models have implicated other neurotransmitters—serotonin, adrenaline, vasopressin, and glutamate—none of which have so far not been supported by any empirical data¹⁹. However, several more recent studies have confirmed a link between N-methyl-D-aspartate (NMDA) receptors and NDE occurrence. In 2019, researcher Charlotte Martial assessed the semantic similarity between 15,000 reports linked to the use of 165 psychoactive substances and 625 NDE narratives, finding that the NMDA receptor antagonist ketamine acted at sigma sites²³, blocking NMDA linked phencyclidine (PCP) receptors to reduce ischaemic damage and produce NDE-associated experiences²⁴. Her work suggests that ketamine could be used as a safe and reversible experimental model for NDE phenomenology, and supports the speculation that endogenous NMDA antagonists with neuroprotective properties may be released in the proximity of death²³.

iv. Oscillatory Gamma Waves

The final, and most recently advanced, major explanation for the occurrence of NDEs surrounds the abundance of oscillatory gamma waves occurring at the moment of brain death. In 2013, a team of researchers at the University of Michigan performed continuous electroencephalography (EEG) in rat models undergoing experimental cardiac arrest, analyzing changes in power density, directed connectivity, and cross-frequency-coupling. Within the first 30 seconds after cardiac arrest and preceding isoelectric EEG, they observed a transient surge of global and highly-coherent gamma oscillations exhibiting a striking increase in anterior-posterior-directed connectivity and tight phase-coupling to both theta and alpha waves²⁵. Seeing as gamma waves are strongly associated with waking consciousness, altered states of consciousness, and memory recall, researchers theorized that these synchronized neural oscillations in the gamma range might be correlated with the generation of NDEs²⁶.

Recently, similar results have been observed in a human model for the first time, further cementing the role of gamma waves in NDEs. In February 2022, an 87-year-old man was admitted to the hospital for epilepsy before undergoing a traumatic subdural hematoma, all of which was recorded on a continuous EEG. Researchers observed an increase of absolute power in gamma activity in the narrow and broad bands, combined with a decrease in theta power after suppression of bilateral hemispheric responses. While delta, beta, and alpha power all decreased, a higher percentage of relative gamma power was observed when compared to the interictal interval—a condition which revealed a state of *heightened consciousness* on the journey to *permanent unconsciousness*²⁷.

The End

Any close brush with death reminds us of the precariousness and fragility of life, stripping away the layers of psychological suppression that shield us from uncomfortable thoughts of existential oblivion. Although there exists no conclusive answer as to the mystery of NDEs, empirical perspectives on the nature of death are now being achieved for the first time, allowing us to gain profoundly significant insight as to the ageless mystery of human mortality and its ultimate

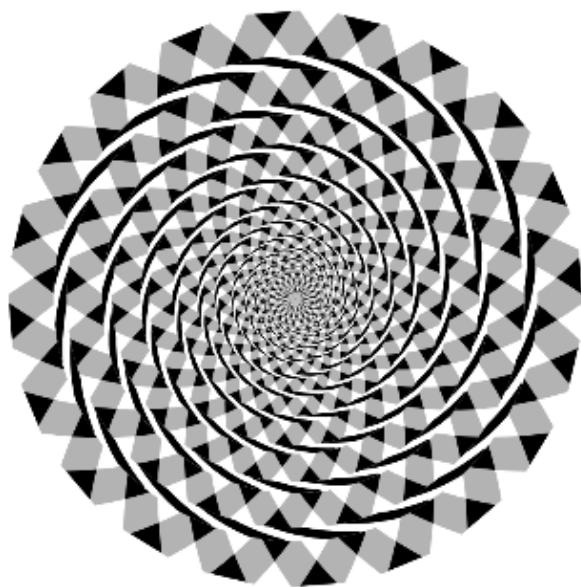
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Seeing Is Believing (*but is it really?*)

By: Parvathy Nair

Take a look at the image below (Fig. 1). What do you see? A majority of people interpret this image as a continuous spiral, looping from the outside of the image into its innermost point. If, however, you begin to trace the spiral with your finger from the outside to the center, it will soon become clear that the spiral never existed after all. The image is simply made up of several closed concentric circles layered over a checkered background. This false spiral is an example of an *optical illusion*, or an image or picture that our minds perceive differently than they really are. In order to understand why our brains sometimes fail to see the ‘whole picture’, we have to first understand how exactly we process visual information from the world around us.

Fig. 1



Shining A Spotlight: Visual Attention

At any given moment, our eyes receive a constant stream of light from our environment, reaching our visual processing centers only a tenth of a second later. The light is then converted into electrical impulses, which our brains then interpret. Similarly to how it may be difficult for *us* to multitask and concentrate on several pieces of information at once (while studying, for example) our brains are also unable to focus on every individual stimulus that enters our eyes. In order to simplify this chaotic and overwhelming barrage of information, our brains selectively concentrate on what is most ‘important’ in our environments.

Interestingly enough, this behavior process evolved from the dangerous lifestyles of our early ancestors. Early human beings had certain priorities: to hunt, to gather, and to not die while hunting and gathering. This meant that they had to focus most of their attention on specific parts of their environment: namely, predator and prey. Being able to selectively focus on the important stimuli while out in the natural world helped early humans survive encounters with these predators, and even avoid running into obstacles like trees¹.

As a result, our brains have adapted the ability to “shine a spotlight” on relevant stimuli, and simply filter out the rest. In regards to our sense of sight, this ability is called *visual attention*: it helps us bind together features like color and orientation to form the perception of complete objects in the environment². For example, when searching for a friend wearing a blue coat in a crowd, our attention is mainly drawn to blue objects, filtering out irrelevant people and colors³. While our selective attention may help us focus our awareness in everyday situations, it also means that we are not always able to consciously process certain elements of an image or scenario. Optical illusions take advantage of this behavior.

Illusions!

So, why exactly did you see a continuous spiral

when you looked at Figure 1? After tracing the pattern ourselves, we *know* that the pattern is simply made up of concentric circles (Fig. 2), but we still can't help but to see a spiral. This illusion, called *Frasier's Spiral*, tricks our minds into perceiving "phantom twists and deviations"⁴. We unconsciously process the tilted checkered pattern in the background as a continuous line, altering our perception of the concentric circles in the foreground and creating a false spiral. Our selective attention chooses to focus on the lines on the foreground, and does not consciously process the skewed background.

(Brief Warning: The following illusion has moving lines and bright colors)

Another optical illusion that takes advantage of our visual processing systems is the *psychedelic peripheral drift illusion*. If you take a look at Figure 3, you will notice that the lines in this pattern seem to ebb and pulse outwards and inwards—in reality, it is a fixed, stationary image. In fact, if you fixate on one specific point in the image, the lines should stop moving. Similarly, if you stare at a point to the left or right side of the two wheels in Figure 4, the wheels should begin rotating within your peripheral vision. Peripheral drift illusions work best when our gaze is directed to "different locations around the stimulus, a point outside the display is fixated and the observer blinks, or when the stimulus is sequentially displayed at different locations whilst the observer fixates one point"⁵. If you quickly shift your focus directly onto the image, the motion stops.

So, how are we seeing motion where it doesn't really exist? This effect is attributed to the way our brains perceive light and dark, as well as our eye movements. The presence of alternating light and dark components within the pattern tricks the motion sensitive neurons within the brain, making us perceive a stationary image as if it actually had moving parts to it. The illusion would work if it were in gray-scale as well, as the shades of light and dark in luminance are what confuse our minds, not the colors themselves¹.

Fig. 2

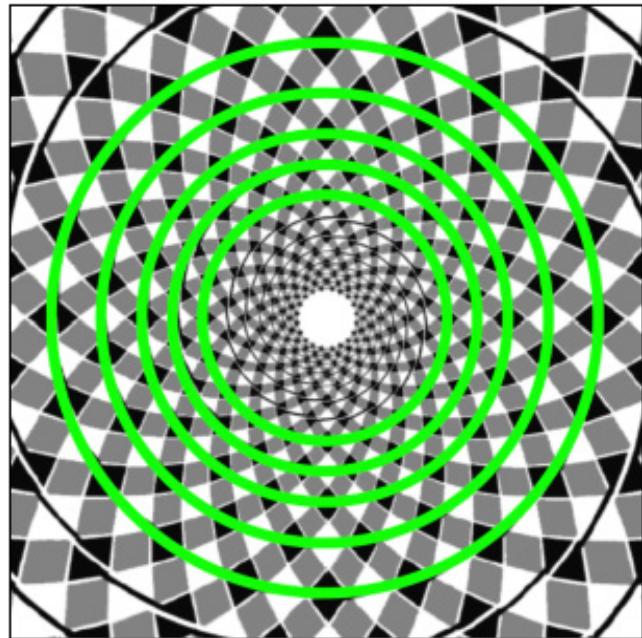


Fig. 3

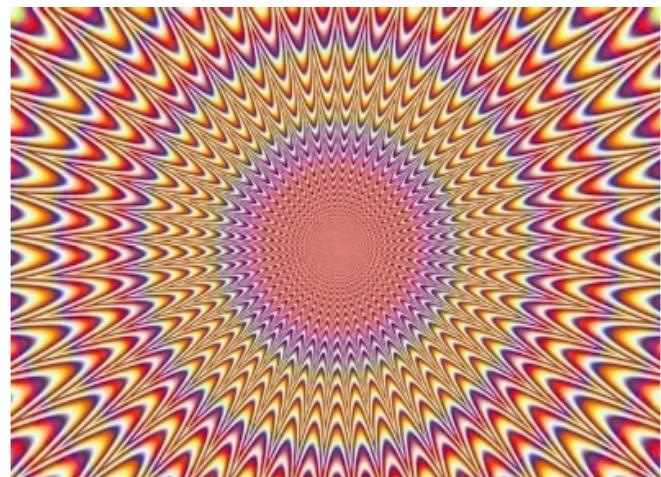


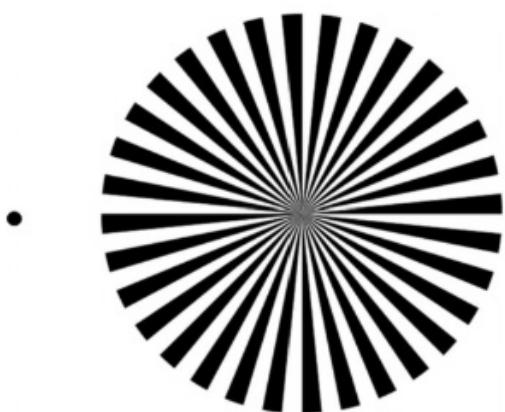
Fig. 4

Peering Into Our Own Minds

Our brains produce five separate types of brainwaves: delta, theta, alpha, beta, and gamma. Alpha waves most often occur when we are engaged in calming activities such as daydreaming, meditating, or practicing mindfulness. In 2013, neuroscientists Rodika Sokoliuk and Rufin VanRullen proposed that the pinwheel optical illusion (Fig. 5) actually reflected a viewer's own alpha waves. To understand what they mean, focus on a point a few inches away from the center of the pinwheel in Figure 5. Hopefully, you should notice the slight flickers emanating from the center of the wheel. According to the two neuroscientists, "The occipital alpha rhythm of the EEG was the only oscillation that showed a time course compatible with the reported illusion: when alpha amplitude was strong, the probability of reporting illusory flicker increased. The peak oscillatory frequency for these flicker-induced modulations was significantly correlated, on a subject-by-subject basis, with the individual alpha frequency measured during rest, in the absence of visual stimulation" ⁶.

Sokouliuk and Vanrullen claim that these flickers emanating from the illusion occur at the same frequency (about 10 Hz or 10 times a second) as the alpha waves our brains produce. Of course, the flickering we see in Figure 5 may be occurring due to the false motion effect, and the matching frequencies could just be a coincidence—however, it is an interesting idea to think about and perhaps research further in the future.

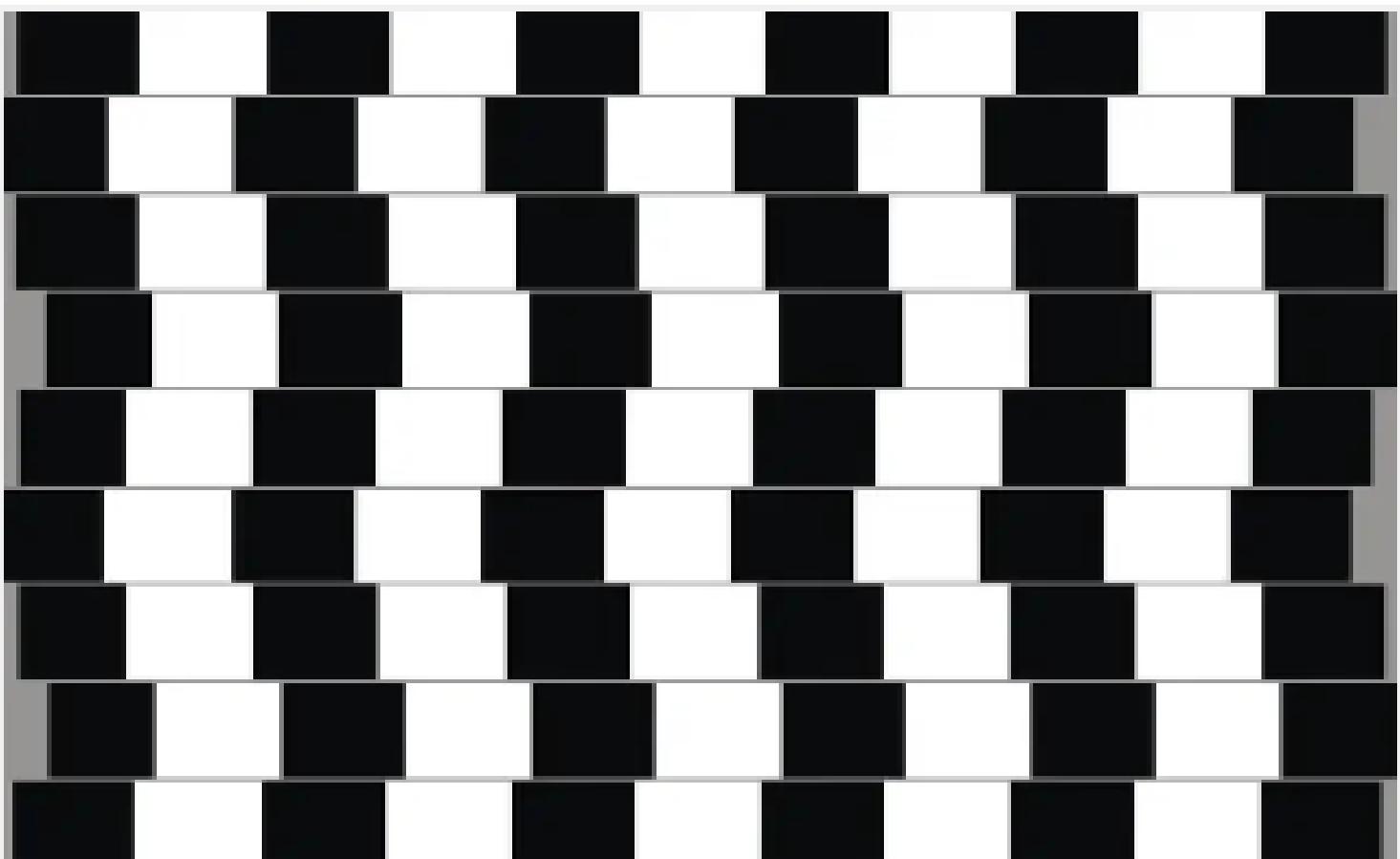
Fig. 5



EEG Time!

The link between optical illusions and specific brain waves was made again in 2016, when researchers used a 32 channel EEG apparatus to monitor the brain waves of participants in a study as they looked at various illusions. They experimented with four different types of stimuli: normal patterns that have no illusory stimulus, cognitive illusions, geometrical illusions, and physiological illusions. EEGs measure the five different types of brain waves discussed earlier: delta, theta, alpha, beta, and gamma. After the researchers calculated the "percentage bandwidth" of the biggest peak, they found that focusing on specific types of illusions generated different brain waves. For example, when looking at the cognitive optical illusions, the participants' brains produced more gamma waves, which are associated with higher brain functions like cognition and memory. When focusing on physiological and geometrical illusions, more beta waves—which are involved in conscious thought and logical thinking—were produced. In general, more alpha waves were produced when looking at all types of illusions. The researchers proposed that optical illusions "promote more coherent signal transmission in our brains, resulting in more intuition and imagination compared to stable, non-illusory images" ⁷. These findings may indicate that simply observing certain optical illusions can stimulate our minds and create new neural connections.

Optical illusions, while fun and interesting to experiment with, are also a good reminder that we can't always blindly trust what our minds perceive to be true. Remember, seeing isn't always believing!



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Your Inner Reptile: Mysterious, Misunderstood, and Confused

By: Shobhin Logani

Do you ever wonder what was going on in the minds of dinosaurs on the day their extinction began? Maybe it was immediate panic—suddenly prey stopped being chased and predators stopped hunting, every single one running for their lives. Perhaps for a brief moment, after the shaking and the boom of the meteor impact faded and before the orange haze of the apocalypse set in, every Cretaceous being looked up and had one powerfully primordial thought—we're f*****.

The dinosaurs may be extinct, but the universal, intrinsic feeling of terror is something that persists into the present—after all, it is well known that in any organism, fear, self-preservation, and a will to survive go hand-in-hand. Inside you, a tiny, frightened reptilian voice whispers for you to run when you are threatened, find light when it's dark, and curl up into a ball when you are scared.

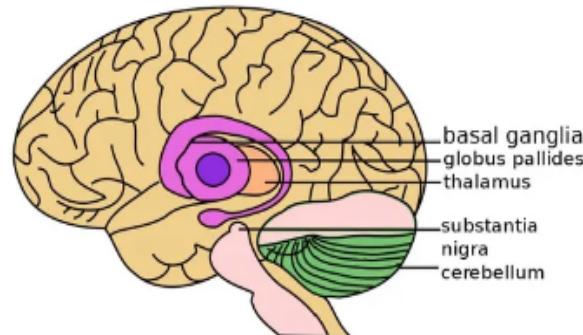
Welcome to the “lizard brain”.

A Scientific Introduction to Your Inner Reptile

That's not a made up term. The “lizard brain”, also known as the reptilian complex, is a term that combines concepts in evolutionary biology with neuroanatomy. It refers to the parts of your brain that we share with almost all tetrapods (4-legged animals), but also controls functions that are present in almost all living things in some form.

The basal ganglia—groups of neuron bundles that sit deep within the brain—refers to the parts of the brain that control function we share with all of species related to us, including 3 major structures: the brainstem, cerebellum, and midbrain¹. Through the basal ganglia, these structures maintain connections with every major area of the cortex while still being responsible for less human-specific tasks such as breathing, movement, and lower-level auditory processing. This gives an indication towards how, despite its relative simplicity, the “lizard brain” and its associated structures play a crucial role in supporting the higher level social, emotional, and logical functions that we, as humans, often use to separate ourselves from other species.

Basal Ganglia and Related Structures of the Brain



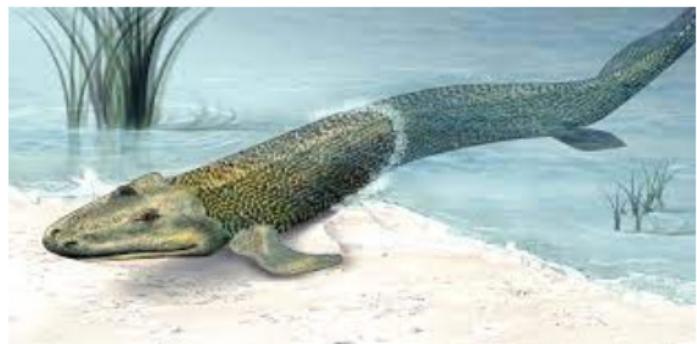
An anatomical overview of the basal ganglia, the subcortical areas in the lizard brain¹⁶

A Gift, From Fish to You

Most people are comfortable with the idea of humans sharing our most recent common ancestor with the other ape species, such as chimpanzees, gorillas, and bonobos. However, it can be much more difficult to grapple with the sheer magnitude of the time it took for our species to emerge from the complexity of life. This is where the ‘lizard’ in “lizard brain” comes in- it can be traced back to the very first fish-tetrapod hybrid organisms (affectionately called ‘fishopods’) that crossed the barrier from ocean to land, through the evolution of reptiles and the divergence of mammals, across millions of years of extinctions, Ice Ages, meteor strikes and volcanic apocalypses, all the way to the organ that is letting you read this article today. This is because, at its core, the brainstem enables you to react to the sensory input from the world around you- something that is so crucial to life that evidence of sensory structures and mechanisms can even be found in fossil records of the first multicellular life-1.6 billion years ago². It’s not called a “lizard brain” because it makes you think like a reptile- rather, it’s a modern-day evolutionary souvenir from when you were a reptile, and it shows how certain elements of natural selection can be conserved over vast amounts of time over many evolutionary changes.

This is why the ‘deep time’ component is important to the story of the “lizard brain”. Life first evolved on Earth about 4 billion years ago but our lineage diverged into bipedal (upright) organisms from chimpanzees only 7 million years ago³. From then, the oldest fossil of the *Homo* genus was dated to only about 3 million years ago, located in East Africa. This is one of the main pieces of evidence that supports the “Out-of-Africa” theory, stipulating that our genus evolved in Africa and dispersed to the rest of the continents over thousands of years⁴. One of the key things we were able to do as bipeds that other primates couldn’t was walk on two feet, and from there, the harsh pressures of savannah life away from the protection of the trees honed our evolution. Our sweat response to heat and the norepinephrine-mediated

flight or fight response that causes our pupil to dilate under threat are just two of the many unique traits that were strongly selected for in early human populations. Importantly, many of these traits are controlled in the brainstem, the cerebrum, and within the midbrain- in fact, your brainstem is the root of 10 of the 12 cranial nerves that control the sensation of stimuli in the environment through sight, touch, and taste. Therefore, many of the traits that made us successful in our new environment stemmed from advantageous rewiring of the neural circuitry and physical anatomy of the “lizard brain”. This gave us new and helpful functions to structures and physiological traits that may have been dormant in our bodies all the way from, at the earliest, our last reptilian common ancestors. This is known as “preadaptation” in evolutionary biology.



A rendition of Tiktaalik, the famous ‘missing link’ fossil between land and marine vertebrate.⁵

If the evolution of our species is just a millimeter on the ruler of the Earth’s history, then the transition into the modern world might as well be invisible. Although migration out of the cradle of Africa happened in waves, modern humans (*Homo sapiens*) only began leaving for other continents in significant numbers between 60,000 and 80,000 years ago⁶. Not only this, but widespread agriculture evolved 10,000 years ago. This is fundamental because the transition of human populations from hunter-gatherers to people living in specialized societies deeply affected how we interact with other humans. When not all of our energy was going towards food, the role of individuals in a society changed drastically as populations and civilizations got bigger. But, this also means that for the

vast majority of our history, humans lived very differently than we did today, and so adaptations that accumulated from living in small hunter-gatherer tribes for 2 million years (including evolutions from other, older members of the *Homo* genus and its predecessors) are still very much relevant today⁷. However, seeing as our species now lives in very different conditions today, has evolution had time to ‘catch up’? How has living with brains fine-tuned to Paleolithic life affected us today?

The Natural State

Adaptations to environmental pressures that are either abiotic, like geography or climate, or interspecific, like special breathing and cooling mechanisms to chase after prey or run from predators, are well-known to have shaped how our ‘lower’ brain structures work. However, it is the intraspecific (human to human) interactions early humans evolved that distinguishes our evolutionary process the most from other species. The role of the brainstem and other “lizard brain” structures in the development of our social and emotional regulation is a rapidly expanding research field.

Most neuroscientists used to think that most, if not all, brain functions related to emotional response, recognizing and speaking to other humans in different ways, and feeling anxiety, elation, or depression were limited to the cerebral cortex, the more complex part of your brain. This idea has truth to it: the majority of functions critical to modulating social behavior are areas of the medial prefrontal cortex, a part of your brain that is evolutionarily ‘newer’⁸. However, new studies show that the cerebellum and brainstem specifically have more prominent roles than once thought, not only in regulating basic physiology, but maybe also in the more basic social and emotional behaviors as well. This makes sense; brain function is very hierarchical and not always linear. Higher levels of function can stem from lower processes and vice versa.

One of the main things that the “lizard brain”, particularly the brainstem, does is define and

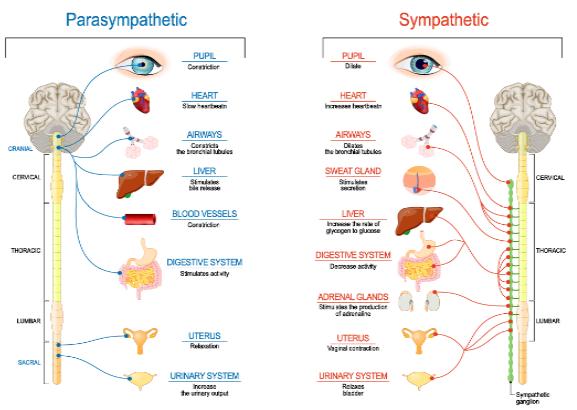
maintain our natural state (homeostasis): the set of internal and external conditions that our body considers “normal”, and more importantly, that we function most optimally in. While this includes physiological variables, like normal oxygen levels and heart rate, a relatively new idea in the field of neuroscience is a ‘normal’ state defined by variables of social interaction—how often you interact with people, and what kinds of interactions you have with them.

Now, let’s go back in time to the Paleolithic era—the 3 million years before the development of agriculture. *Homo sapiens*, along with other now extinct-species in the *Homo* genus, lived in small hunter-gatherer groups, in which survival was directly dependent on the strength and quality of your interpersonal interactions.

Almost all of your time, energy, and cognition was geared toward survival, specifically maintaining a constant flow of nutrition to maintain our large brains. If the hunt fails, your closeness with other members of your tribe determines whether they would be willing to share with you. Not only that, but the long term survival of your offspring meant trusting the others. For thousands upon thousands of years, this is exactly what your brain would become more fine tuned to do—create a ‘normal’ state that involved heavy and trust-based interactions with a small and close group of people, and having a defined role in your social space. This means that, despite the rapid changes in the life history of humans in the last 10,000 years, our bodies are still operating under the conditions of the 3 million million years below that. This is exactly where they start to run into problems when confronted with the modern world.

One of the main ways your “lizard brain” tries to maintain your normal state is through your parasympathetic and sympathetic nervous system, which controls the “flight or fight response”. The precise definition of this response is “... an automatic physiological reaction to an event that is perceived as stressful or frightening”⁹. These systems exemplify the old scientific adage, “every action has an equal and opposite reaction”. When something knocks

your normal state out of whack, it can cause, among other things, physiological and emotional ‘symptoms’ that we normally associate with panic and anxiety: a high heart rate, rapid breathing, blood vessel dilation and increased blood flow, and the rapid breakdown of glycogen into glucose give you a burst of energy.



Overview of the counter-acting functions of the sympathetic and parasympathetic nervous systems.¹⁰

Let's go back to the dinosaurs at the time of their extinction—the worldwide, existential panic that ensued (at least, it's easy to imagine that it did) when suddenly, the entire ecological world order at all scales was turned upside down by a cataclysmic event. Like the dinosaurs, we are currently the dominant and most widespread genus on the planet. We cannot define the ecosystems of the Earth without considering our impact. Can we think of a recent situation where a global event severely disrupted our normal state of interactions with other people? What would worldwide, existential panic look like for us?

The COVID-19 Pandemic as a quasi-Apocalypse (according to your inner reptile)

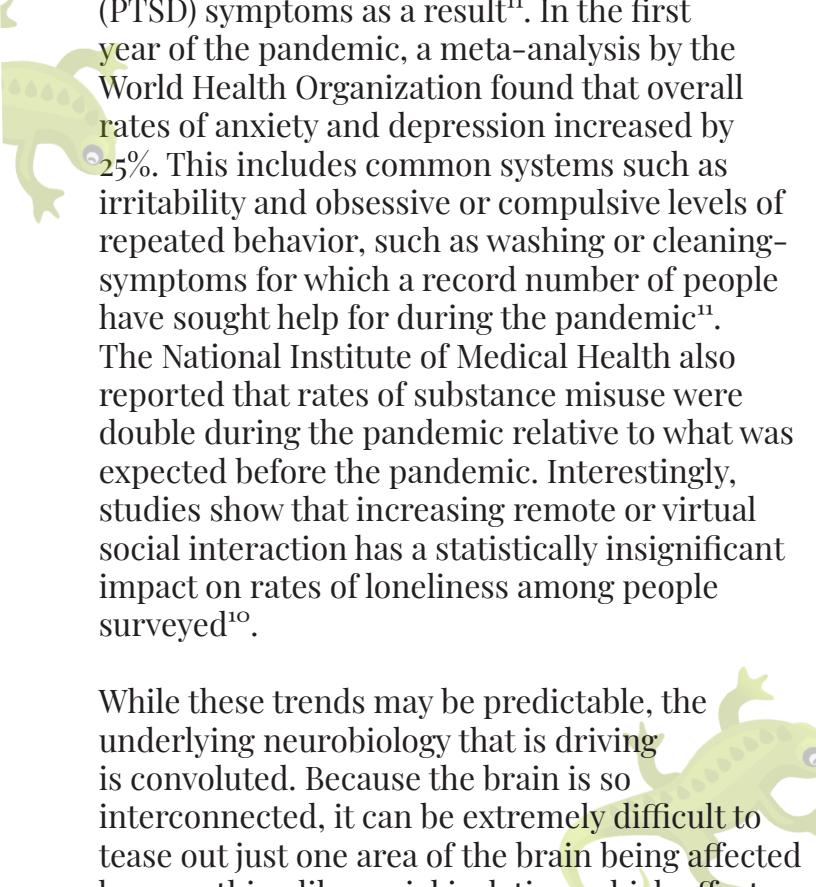
The COVID-19 pandemic, from a purely evolutionary history perspective, is unique. It is the only time in recent history where a truly global event altered the state of social interaction for the vast majority of people. Despite the variability in responses to the health crisis across different countries and communities, overall the world shifted to minimizing face-

to-face interaction as much as possible¹⁰. In the context of how our brain has adapted to survive, the pandemic represents a full deviation from the conditions it has come to expect as ‘normal’. Therefore, the COVID-19 pandemic, which was unprecedented in a number of ways, also provides possibly the only example of a worldwide phenomenon where we can look at what the outward response was to suddenly limited social interaction across populations and communities.

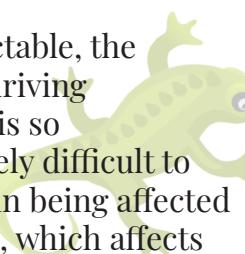
Thankfully, the pandemic has not been the human equivalent of the meteor strike that ended the dinosaurs. That being said, your brain doesn't necessarily ‘know’ that. The “lizard brain” within you is mainly responsible for fixed-action pattern responses: predictable cascades of induced responses to a given stimulus, usually from higher levels of the brain. Given that the parasympathetic and sympathetic nervous systems work with each other to maintain homeostasis, it becomes clear how your “lizard brain” responds when you no longer receive your normal amount of social stimulus. Specifically, the sympathetic nervous system works to create the observable responses of panic and stress when other parts of your brain are signaling that your normal state— in the case of the pandemic, your state of social interaction has suddenly shifted. Therefore, your “lizard brain” would absolutely consider the COVID-19 pandemic to be the “end times” in terms of maintaining homeostasis, given that the level of normal social interaction it is used to could not be attained. As recent evidence shows, this has significant implications for the neuroscience underlying the large-scale responses that we saw in large populations of people as social isolation decreased.

How Your “Lizard Brain” May Be Screwing You Over

The high level of social isolation as a result of stay-at-home orders during the pandemic created a kind of secondary “behavioral epidemic”, in various countries, with predictable increases in the level of mental health disorders such as anxiety, depression, and even the



recurrence of post-traumatic stress disorder (PTSD) symptoms as a result¹¹. In the first year of the pandemic, a meta-analysis by the World Health Organization found that overall rates of anxiety and depression increased by 25%. This includes common systems such as irritability and obsessive or compulsive levels of repeated behavior, such as washing or cleaning- symptoms for which a record number of people have sought help for during the pandemic¹¹. The National Institute of Medical Health also reported that rates of substance misuse were double during the pandemic relative to what was expected before the pandemic. Interestingly, studies show that increasing remote or virtual social interaction has a statistically insignificant impact on rates of loneliness among people surveyed¹⁰.



While these trends may be predictable, the underlying neurobiology that is driving is convoluted. Because the brain is so interconnected, it can be extremely difficult to tease out just one area of the brain being affected by something like social isolation, which affects areas of cognition and emotional regulation. However, in terms of the observable stress responses that result from isolation, a leading theory, contextualized by the environment in which our “lizard brain” evolved, is that stress responses are being activated by the brainstem, cerebellum, and midbrain as a ‘panic’ response to compensate for the lack of social stimulation experienced by people worldwide.

This can be seen by peering into the neurotransmitter systems that are activated within the “lizard brain” during social interaction. One such system is the ‘raw feels’ system, which is modulated primarily by endorphins that create senses of euphoria and content during positive social interactions. When endorphins are released within the brainstem, they cause the release of dopamine and serotonin, two chemicals that create the ‘reward system’ of your brain- they cause you to feel good in order to incentivize more behavior that, in a brain adapted for survival based on social interaction, it views as crucial.

However, when long periods of social isolation occur, your “lizard brain”, and this reward system in particular, can go haywire. Because the “lizard brain” mainly acts through fixed action-response patterns, it is not very good at responding to unique situations- and it is very persistent. When you know longer receive a homeostatic level of social interaction, the structures in your “lizard brain” will do whatever they can to compensate for this loss, including trying to replace the stimulation it lacks. This may explain the increased rate of substance use, as most stimulants act directly on the rewards system in the brainstem. The longer term problem is when your “lizard brain” creates a chronic state of panic in your, in direct response to a prolonged state of social isolation. Your sympathetic nervous system is responsible for maintaining homeostasis, and not receiving normal social stimulation causes it to put you in “flight” mode. When this is prolonged, it is easy to reason how these symptoms become the chronic symptoms of long term anxiety. The rapid increase in blood flow turns into hypertension. When glycogen gets broken down into glucose, the extra energy has nowhere to grow, and the glucose is converted into triglycerides that can raise cholesterol and cause weight gain over time. All of these symptoms can be directly tied back into your “lizard brain” trying, and failing, to get you back into social homeostasis in a world where it hasn’t evolved to perform. Experiencing these symptoms, and their root cause in a discrepancy between our brain’s evolved function and its current environment, is known as “evolutionary mismatch disease”¹².

Implications for Public Health and Neurotechnology: Vagus Nerve Stimulation

Clearly, the global long-term effects of the pandemic will include a battle with increasing rates of chronic mental health conditions such as anxiety and depression. Viewing these issues through the lens of the “lizard brain”, and its symptoms as evolutionary mismatch diseases, allows us to contextualize the way we respond to this “behavioral epidemic”. Importantly, it may now point to new, non-pharmacological

treatment options for those suffering the effects of prolonged social isolation as a result of the pandemic.

The vagus nerve is both the longest nerve in the body, and also the largest component of the parasympathetic nervous system. It is the part of the counteraction against the sympathetic nervous system, and induces the “rest and digest” response- downregulating the panic induced by an overactive sympathetic nervous system. While stimulating this nerve as a treatment for seizures has been around since the 1980s, new methods of stimulating the nerve as treatment for anxiety and depression- especially in people resistant to typical treatments- has become increasingly popular. There is an emerging trend in research to find new non-invasive ways to stimulate this nerve while accessing the nerve as close to the brainstem as possible. Stimulation via the outer ear is especially compelling, as the devices both interface externally with the brain and can be done with patients at home, rather than requiring patients to enter a clinical setting¹³.



An example of outer-ear stimulation of the vagus nerve¹³

However, as we confront the realities of global increases in mental health disorders, it is important to continue to frame responses through the evolutionary mismatch between our “lizard brain” and the modern world. The idea of the “lizard brain” should serve to destigmatize anxiety and depression disorders, and as a way to frame symptoms to patients and reduce feelings of self-blame. After all, while you can’t tell your inner reptile’s voice to stop stressing you out, you can understand that it may be just

as confused as you are.

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by
Ananya Chawla

Please Put Your AI to Bed!

What happens when machines don't work? "Just turn it off and on again!" So you do, and voila! There goes your tech - all ready to work again. If that still doesn't do the trick? Feel free to bring it to the nearest tech store for a check-up. Is it too messed up for an adjustment? Time for a replacement! This seems to be the standard procedure for dealing with technical difficulties, but what if your technology needed a nap instead? What if it had to rest peacefully and dream for a while?

While we tend to think of machines as metal, circuits, and code, scientists at Los Alamos National Laboratory have found that AI may need to undergo something that closely resembles sleep as we experience it.

How did they find this out? Do the machines snore?

An extensive amount of time and effort has gone into researching neural networks. Through their research, Los Alamos National Laboratory (LANL) was able to

figure out that AI may require sleep in order to increase stability¹.

At the beginning of the experiment, the AI was bombarded with a variety of photos that it was required to categorize based on what it discerned as separate categories². This is a sort of unsupervised dictionary training.

What does unsupervised dictionary learning look like?

It essentially shows a variety of items to be categorized without prior reference or knowledge of how they should be categorized¹. Imagine telling a child to organize a collection of household items. Even if they don't know what a spoon is, it is unlikely that they will categorize it in the same group as doors. The categorization of items that haven't been seen before is unsupervised dictionary learning.

LANL was initially studying how the systems would respond to learning, but after a certain period of time,

the systems became unstable and were apparently, “generating images that were analogous to hallucinations”³.

We can combine this information with Google’s research on AI dreams to better understand what that might mean. Part of that process of AI classifying images is tweaking the “noise” that AI receives from an image to better fit our label⁴. For example, you start off with something like this:



Image from Google Research

and optimize the learning process to produce an image that looks more similar to what we (researchers/humans) want the machine to output. Here is an example of AI attempting to display weights:



Image from Google Research

You can over stimulate AI (the way unsupervised learning does). While learning, LANL researchers found that the network’s “learned features” start off reason-

ably and then begin to become meaningless as the network becomes more and more unstable². Overinterpreted images can also be produced where a network is attempting to find the “already learned items” within any image even if those features are not there⁵. This overinterpretation can cause the network to hallucinate and produce images such as these:



(it kind of looks like what I see when I rub my eyes too hard).

Humans hallucinate in a very similar way when sleep deprived, but in order for us to calm down and stop hallucinating, we need to recover and sleep. Researchers weren’t sure exactly how to restore the neural networks’ hallucinations since they aren’t humans.

In an attempt to stabilize the neural network, a variety of things were tried until, eventually, the researchers began exposing the network to different frequencies. The frequencies that stabilized them the most were similar to the waves human brains experience when undergoing deep sleep. It was like a little nap for the machine! Afterward, stability was restored to the network. Additionally, this sleeplike state was required in order to encourage a neuron in the neural network to

to even begin to function¹.

Some researchers in Italy went one step further and were able to determine that different forms of ‘sleep’ for AI can help the networks in different ways⁸. Surely, you may have been exposed to the idea of sleep cycles in humans and how we alternate between varying forms of sleep in order to maintain balance. In a similar way, neural networks can imitate slow-wave sleep in order to “consolidate important memories” and mimic REM sleep that works to remove unimportant information from these systems⁸!

Essentially, in order to recover from hallucinations and optimize the learning process for a machine, we need to artificially simulate sleep in the neural networks!

Why can't you just turn it off and on? Or leave the computer alone? How is this different?

Sleep in neural networks is not an incident where the computer has paused all computation and action and is frozen. It is more similar to the human activity of sleep, where the body is still working but in a recovery stage where the neurons are stimulating activity, and they are not inactive. This is not the same as turning the device on and off. Turning a device off and on is like turning off the brain entirely and turning it back on. It is a form of death for the neural network, and once it is back on, the system will have forgotten the

learning and will undergo what is equivalent to memory loss³.

This experience of requiring a form of “sleep” is not something seen in all other AI networks. Instead, it is specific to neural networks or AI/machines that are used to understand biology and/or have “biologically realistic processors”¹. With AI that doesn’t have those characteristics, these problems do not arise because the algorithms and operations other AI must perform aren’t equal to the way that the neurons/the brain process those algorithms and operations.

Do I need to fluff some pillows for my computer now?

Neural networks seem to require sleep, so items may need to be developed to ensure that these networks can get their rest and maintain peak performance.

In fact, Intel is currently working on a chip⁵ that creates a process by which AI can sleep every once in a while⁶. This would be a big step towards a future where these neural networks are better and more safely integrated.

This study also raises some of the following questions:

- Do neural networks require a sleep cycle similar to humans? (somewhat answered by researchers in Italy)
- Would there be a required sleeping time and/or a need for consecutive hours of sleep, or can it be broken up?
- Are there effects of sleep deprivation on these networks?
 - If so, how do we manage expectations for the speed and precision of work that AI can do in these situations?

The framework of questions being researched are similar to those regarding sleep in humans. This then raises another question:

Are neural networks and the human brain similar enough to be studied together in some situations?

If the answer is yes, then we have stumbled upon an entirely new frontier.

Neural networks are inherently related to the brain in terms of structure, but the fact that this phenomenon of “requiring sleep” was not directly built into the processes of neural networks studied at LANL, means that simply creating biologically realistic processors can result in the creation of a “brain-like network”. Because

of this, perhaps the more we learn about the brain, the more we learn about neural networks... and the more we learn about neural networks, the more we could learn about the brain... and if these systems are similar enough - what does that mean for humans? Let's go deeper into this idea.

What Might This Mean for Humans Going Forward?

Thanks to the LANL study, we now have a very strong bridge between biological and mechanical processes. Since we have found that only biologically realistic processors have mimicked the human brain in this way, creating and studying AI becomes more tied to biology than it ever was before. Instead of using technology as an appendage or calculator, technology can be treated like a peer. In that case, perhaps AI that mimics the human brain somewhat can perhaps be used for extensive studying of the brain. It may also be used to research and attempt to understand biological systems even better with a mechanical model!

The mechanisms and effects of “sleep deprivation” on AI were noticeable, and through this, we now have an outlet through which sleep deprivation effects can be more directly observed while controlling other variables. In a similar way, if more thoroughly developed and modeled, perhaps there could be more in depth

RETHINKING CONSCIOUSNESS: THE POSSIBILITY OF MIND UPLOADING

BY SUHINA SHARMA

Would you ever digitally upload your brain?

At first glance, the thought may sound daunting, but imagine if your loved ones never had to say goodbye. Imagine if, even after you physically die, they could somehow extend a fragment of your consciousness into the future.

Would you do it now?

But what if it wasn't just a fragment? What if everything- your mind, memories, emotions, personality- that made

you, you were never lost, creating a whole other version of yourself living in a virtual, immortal world?

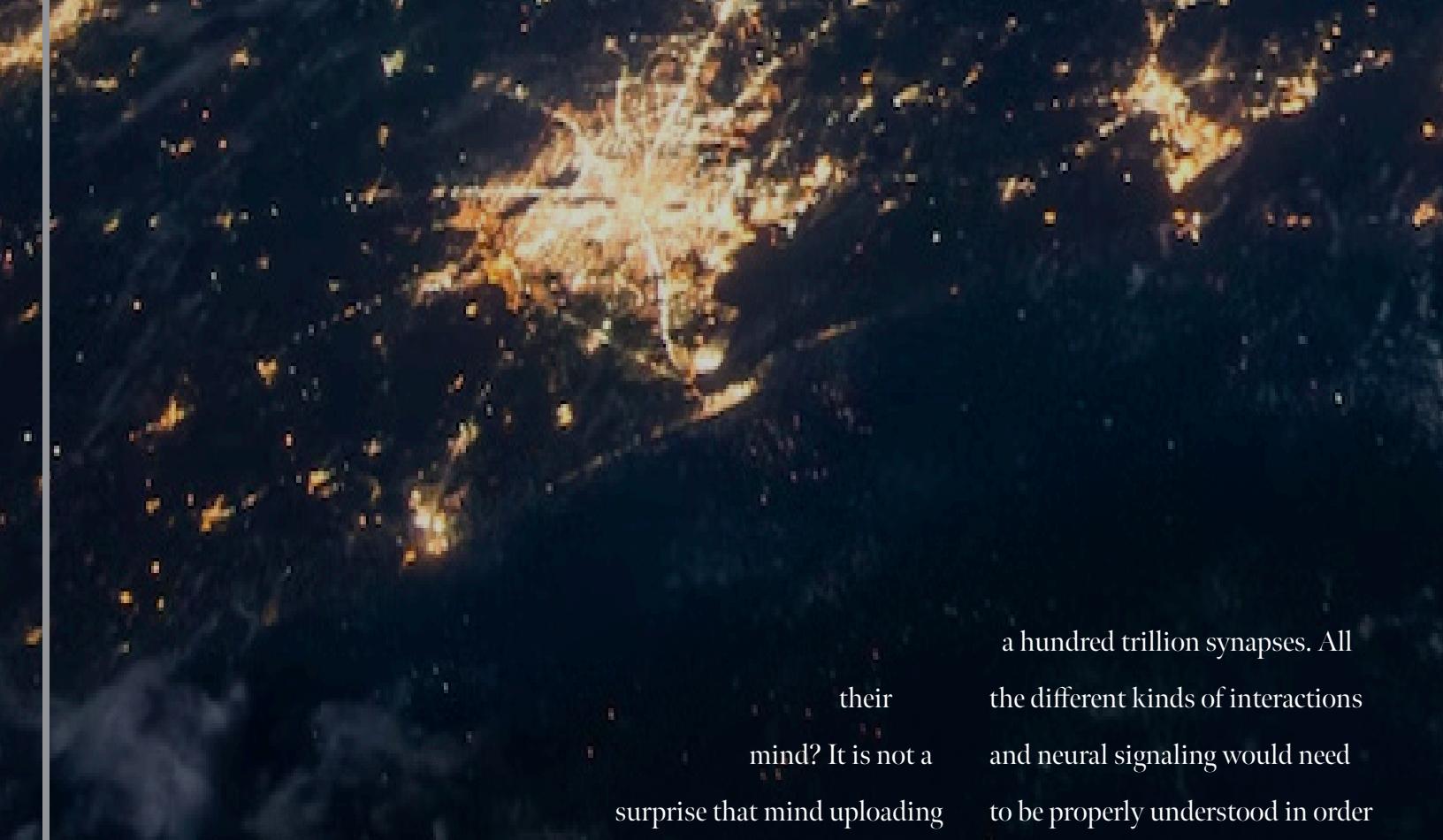
How about now?

This futuristic possibility of digital duplication of one's mental essence is called mind uploading. The science of the brain and of consciousness increasingly suggests that mind uploading is possible. While the actual implementation of this idea is likely to be far in our future, given how there is already a lot of interest and effort directed

towards this goal, mind uploading seems inevitable and it may creep up on us faster than we thought.

The Birth of Mind Uploading

The idea of mind uploading originated from taking a mouse brain and observing the area of the brain where memories were processed. With a silicon chip, researchers were able to duplicate functions of information storage in a mouse brain. This idea of replicating memory storage in a mouse



brain. This idea of replicating memory storage in a mouse brain sparked the idea of expanding this into replicating an entire human brain with similar technological methods.

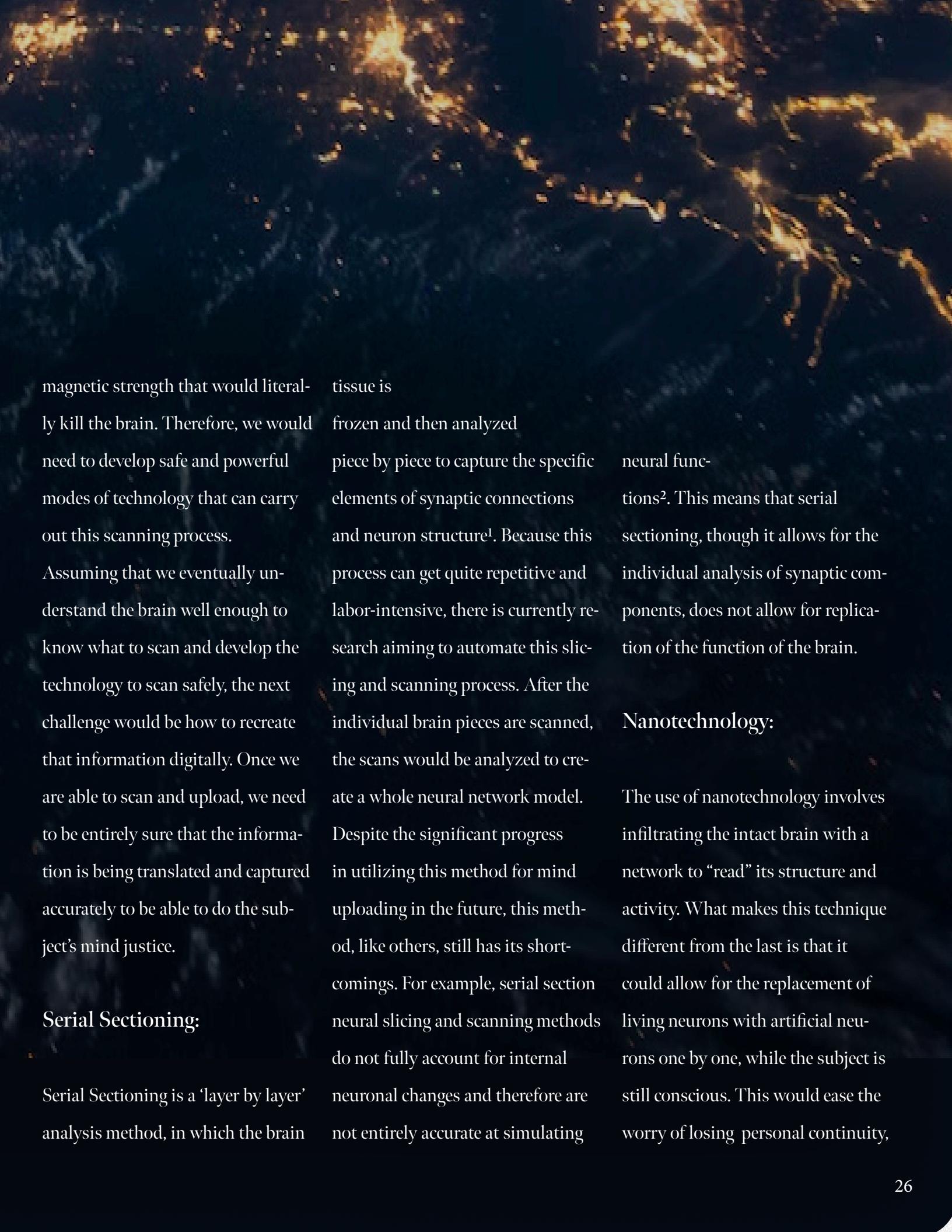
How Feasible is it? A Deep Dive into the Technological Mechanisms and Techniques

Mind uploading has powerful appeal, but what would it actually take to scan a person's brain and upload

their mind? It is not a surprise that mind uploading would require us to break our existing technological barriers, opening this sort of gateway and blurring the lines between the fragility of human consciousness and experience and this powerful emerging force of computing power and cryopreservation. The main challenge is scanning the brain in enough detail to capture the mind and perfectly recreate that detail artificially. The question is, where do we even begin the scanning process? The human brain contains a complex and intricate neural network containing approximately 86 billion neurons connected by at least

a hundred trillion synapses. All the different kinds of interactions and neural signaling would need to be properly understood in order to effectively and accurately map a person's mind. Unfortunately, our current understanding of the brain isn't solid enough to determine what we need to scan in order to replicate the intricacies of the mind.

Currently, we can accurately scan a living human brain with small resolution using our best non-invasive scanning method—magnetic resonance imaging (MRI). However, MRI runs on very strong magnetic fields. Scanning at the resolution required to determine the details of individual synapses would require a



magnetic strength that would literally kill the brain. Therefore, we would need to develop safe and powerful modes of technology that can carry out this scanning process.

Assuming that we eventually understand the brain well enough to know what to scan and develop the technology to scan safely, the next challenge would be how to recreate that information digitally. Once we are able to scan and upload, we need to be entirely sure that the information is being translated and captured accurately to be able to do the subject's mind justice.

Serial Sectioning:

Serial Sectioning is a 'layer by layer' analysis method, in which the brain

tissue is frozen and then analyzed piece by piece to capture the specific elements of synaptic connections and neuron structure¹. Because this process can get quite repetitive and labor-intensive, there is currently research aiming to automate this slicing and scanning process. After the individual brain pieces are scanned, the scans would be analyzed to create a whole neural network model. Despite the significant progress in utilizing this method for mind uploading in the future, this method, like others, still has its shortcomings. For example, serial section neural slicing and scanning methods do not fully account for internal neuronal changes and therefore are not entirely accurate at simulating

neural functions². This means that serial sectioning, though it allows for the individual analysis of synaptic components, does not allow for replication of the function of the brain.

Nanotechnology:

The use of nanotechnology involves infiltrating the intact brain with a network to "read" its structure and activity. What makes this technique different from the last is that it could allow for the replacement of living neurons with artificial neurons one by one, while the subject is still conscious. This would ease the worry of losing personal continuity,

as it provides a much more smooth transition from an organic to the digital brain than the earlier process. Though there is not as much research testing nanotechnology as of yet, it has picked up a lot of attention as a potential option as it allows for greater trust and surety from the person undergoing the mind uploading process.

Let's Get Ethical

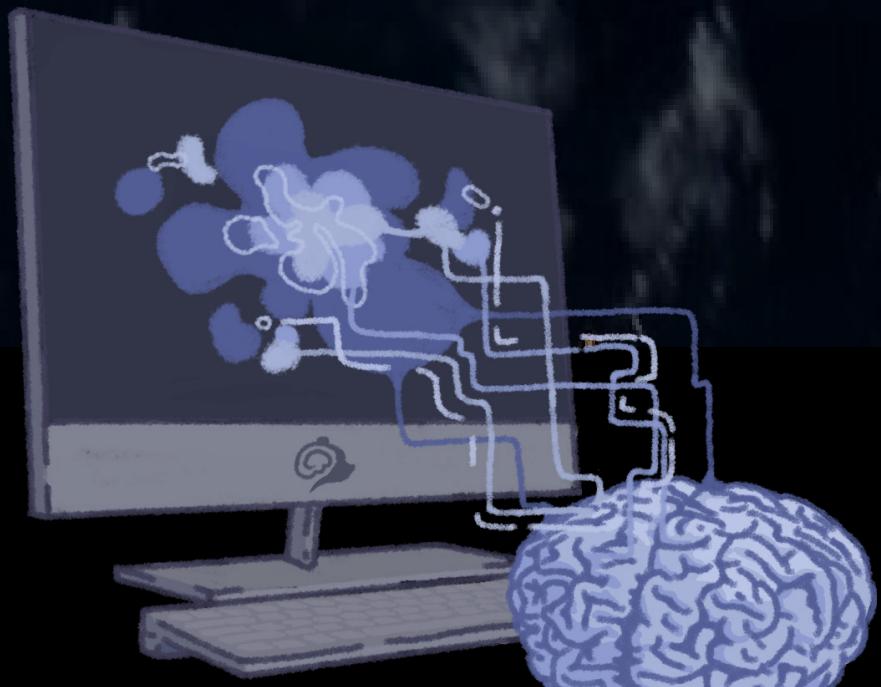
Why do we want to find a means of escaping death, and what are the implications if we do? The ethical implications of mind uploading is like peeling the layers of an onion—the factors to consider keep getting deeper. Before we start diving into the layers and layers of “what ifs”, let’s take a step back and look at the bigger picture. Let’s first consider who would have access to our mind uploads in the first place— not everybody, that’s for sure. Such technologies will most probably be affordable

to only the upper class. What does it mean for those who can’t afford it— will they feel behind that they can’t experience “immortality” while others can? It is no doubt a greater amount of inequality and uneven distribution of power will result from mind uploading. The question is, is that social stability worth sacrificing?

Now that we have looked at the bigger societal impacts, let’s narrow our focus to the individual authenticity concerns and balance of technological control. Though some of the methods discussed above offer a way mind uploading can occur while the subject is conscious, allowing for them to

ensure authenticity of their mind contents being uploaded, this is not always the case. If we weren’t conscious, how would we be able to verify our authenticity in our ‘virtual’ self, or how will we be able to control, at a technological level, who will access them later?

Of course, there are many profound effects for humanity that come with mind uploading as well. We could increase humans’ overall well-being, reprogramming the brain with neural features that correspond to high happiness. We could change the way we interact with people, forming deeper connections through informational and emotional exchange. However,



we should think about which of these brain modifications should be allowed before our definition of human changes completely. We could enhance the brain to increase memory capacity or mental calculations, but should we also allow deletions? What would it mean if we were able to remove memories, or even emotions? Tinkering with this ability would definitely screw over our core definition of what it means to be human.

of existing in a virtual world. When the time comes for you to choose how you want to be memorialized, what will you decide?

The big circulating idea, however, is that if this technology becomes available, it is unclear if people will prioritize their quantity of life or their quality of life. Is one necessarily better than the other? For someone whose life was cut short, it might not be a bad idea to be brought back in a virtual form than not at all and have some element of preservation. But for someone like myself, I am not sure I can compromise the most gratifying parts of my life for the sake

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The DSM, Unraveled

by Oce Bohra

The DSM & and its dubious definition of “normal”

The most interesting conspiracy theory I've ever read is that the DSM is purposefully eroding the definition of 'normal' in psychiatry so that the pharmaceutical industry can sell more pills to more people for more money. Frankly, I believe it.

The DSM (Diagnostic and Statistical Manual of Mental Disorders) is the Bible of psychiatry. It's published by the American Psychiatric Association as an authoritative guide on mental disorders. It's consulted by just about everyone - medical professionals, researchers, pharmaceutical and health insurance companies, policymakers, and drug regulation agencies within the US and abroad.

The book carries a lot of weight as it essentially determines who can get a diagnosis and who can get medicated. According to the current version of the DSM, almost 50% of all Americans are eligible for a mental disorder diagnosis at some point during their lifetime - a staggering statistic. This begs the question - why has mental illness become this prevalent? Is it because Americans are getting sicker? Have we become better at identifying mental disorders? Has our definition of good mental health become more narrow?

It's a bit of everything.

When the DSM-5 was released in 2013, it caused an uproar within the scientific community. The number of diagnoses in the manual skyrocketed from 106 diagnoses in the DSM-1 to a whopping 300 in the latest version. Mental health practitioners accused the DSM task force of creating "unsubstantiated and questionable disorder categories" with no scientific basis to

increase the audience for the pharmaceutical companies they were affiliated with.

Doctor Allen J Frances, who chaired the DSM-4 taskforce, called the release of the DSM-5 the "saddest moment in his 45-year career of practicing psychiatry." In an article published in Psychology Today, he described how these "fad diagnoses" could lead to people with normal levels of grief, temper, gluttony, and stress - among other experiences - being given inappropriate psychiatric treatment.

For example, the un-reproduced results of a single study led to the addition of Disruptive Mood Dysregulation Disorder (DMDD) in the DSM-5. The diagnosis frames repeated temper tantrums in children as a psychiatric disorder, which Frances worries will exacerbate the inappropriate use of medication in kids. Another new diagnosis is the Prolonged Grief Disorder, which will allow people who have grieved a death of a loved one for over a year to receive psychiatric treatment. Frances claims this will "medicalize necessary emotional reactions to the loss of a loved one." In cases such as this, mental health problems rooted in environmental or psychological issues have been reframed as biologically-based illnesses that should be solved through medication.

The Case of Binge Eating Disorder

Perhaps the most interesting case is that of the binge eating disorder (BED). The addition of binge eating disorder to the DSM-5 has been one of the most polarizing decisions in modern psychiatry.

Since BED was recognized as a formal diagnosis in 2013, it has become the most common eating disorder in America, affecting roughly 2.8 million Americans. BED is characterized by regularly

eating large amounts of food in one sitting and feeling out of control while eating. Binge eating disorder began as a theory in the 1950s when it described a combinatory pattern of extreme hunger, insomnia, and anorexia during periods of stress. In earlier DSMs, binge eating was listed as a symptom of bulimia nervosa, explains activist Sarah Fay in her article “The New DSM Is Coming and That Isn’t Good News.”

The criteria for BED is extremely loose. Patients have to identify with three of the five following criteria: “1. eating much more rapidly than normal; 2. eating until feeling uncomfortably full; 3. eating large amounts of food when not feeling physically hungry; 4. eating alone because of being embarrassed by how much one is eating; 5. feeling disgusted with oneself, depressed, or very guilty after overeating” (DSM 5).

The problem with these criteria, says Fay, is that there are no real parameters. There is no consensus on the definitions of “rapidly” or “large amount of food,” which makes the diagnosis fundamentally unreliable. There is no scientific evidence that points at biological reasons for overeating. In fact, 4 in 5 Americans admit to regularly overeating, making the diagnosis easily applicable to most of the population. Field trials show that two clinicians only agree on a BED diagnosis half of the time, which falls short of the 70% agreement needed for the diagnosis to be considered ‘satisfactory.’

So why exactly was binge eating made a disorder? Shire Pharmaceuticals developed Vyvanse, a highly addictive amphetamine that can suppress the appetite. When binge eating disorder was being considered as an official DSM diagnosis, they went on a two-part marketing offensive to legitimize BED to the public.

First, they poured millions into raising awareness of the disorder through online and print advertisements. They hired famous tennis player Monica Seles as a paid spokeswoman for the company, and she went on a media circuit to discuss her struggles with binge eating. They

funneled millions in donations into patient advocacy groups like the National Eating Disorder Association.

After getting the public’s attention, they began more directly marketing their drug, Vyvanse. They propagated the false claim that BED was due to chemical imbalances in the brain, which their drug could successfully resolve.

I have to emphasize – the biological reasoning for binge eating isn’t known at all. It is likely that binge eating is the result of a combination of genetics, cultural factors, and stress. The American food industry is heavily reliant on highly processed foods, and “junk food is designed to be binged on” (Fay). Fast food has been specifically engineered to be addictive, and corporations pour billions of dollars into advertising it. Overeating is also a very common side effect of stress. Vyvanse only ‘works’ because it is an amphetamine, and common side effects of most amphetamines are weight loss and appetite suppression.

While federal officials have reprimanded Shire for improper marketing, the company has faced little blowback. Vyvanse is Shire’s top selling product, racking almost \$1.4B in sales in 2014 alone.

This anecdote only scratches the surfaces of the pharmaceutical industry’s manipulation of American psychiatry. It shows how the false “chemical imbalance theory” can be weaponized to sell Americans unnecessary drugs. It shows how Big Pharma wants to sell drugs as a “quick fix” to larger societal problems. It reinforced the enormous amount of money companies stand to gain through overdiagnosis.

The perpetuation of the Chemical Imbalance Theory

In high school, I read a book called “An Anatomy of an Epidemic: Magic Bullets, Psychiatric Drugs, and the Astonishing Rise of Mental Illness in America” in which journalist Robert Whitaker investigates why the number of disabled

mentally ill in the United States has tripled over the past two decades.

In one chapter, he describes how Big Pharma perpetuated so-called “chemical imbalance theory.” The theory arose in the 1950s, when researchers attempted to define depression and other affective disorders as purely biologically-based illnesses that arose due to a chemical imbalance in the brain. An investigator at the National Institute of Medical Health (NIMH), Bernard Brodie, found that reserpine lowered levels of serotonin (a neurotransmitter in the brain) in rabbits and made them “apathetic.” He concluded that there could be a link between low serotonin levels and depression.

This, among other studies in the late 1900s, attempted to hammer in the idea that too much or too little of certain neurotransmitters in our brains were the root cause of mental illness. One theory argued that the depletion of monoamines (norepinephrine, serotonin, and dopamine) led to depression, while another theory put forth that schizophrenia was triggered by dysregulation of the dopamine system.

However, these theories had little solid scientific backing. One of the main studies that propelled the “chemical imbalance theory” into the public eye was conducted by Marie Asberg at the Karolinska Institute in Stockholm in 1975. She found that 20 of the 68 patients she tested suffered from low serotonin levels, and this subgroup had more suicidal tendencies than the rest of their cohort.

However, this theory wasn’t supported by most other literature during that period. In 1984, the NIMH found that serotonin levels varied in depressed patients. Countless other studies by universities like Stanford, University of Pennsylvania, and Southwest Medical Center in Dallas all concluded that the serotonin theory of depression was, for lack of a better term, hogwash.

Researchers found the same to be true of schizophrenia: there was no compelling evidence that psychosis was caused by an overactive

dopaminergic system (Malenka). “This idea of chemical imbalance has been useful—that you have a kind of chemical soup in which there may be too much dopamine or too little serotonin,” says Robert C. Malenka, a professor of Psychiatry and Behavioral Sciences at Stanford University, “but in truth, the ‘brain soup’ idea is no longer adequate.”

Even though the chemical imbalance theory had been resoundingly discredited by the academic community by the turn of the century, the pharmaceutical industry jumped on it.

In the 1980s, Eli Lilly developed Prozac, a selective serotonin reuptake inhibitor (SSRI), as the world’s first antidepressant. Prozac was soon hailed as a ‘wonder drug,’ and companies raced to follow Eli Lilly’s suit. SSRIs were developed as treatments for a number of depression and anxiety disorders, including post-traumatic stress disorder (PTSD) and obsessive-compulsive disorder (OCD). The “chemical imbalance theory” and pharmaceutical marketing campaigns have fueled each other. The theory became the cornerstone of marketing campaigns for SSRIs, and the PR that these antidepressants have done for this theory cannot be understated: a study conducted by University of Melbourne finds that over 80% of the Australian public believe that chemical imbalances are one of the causes of depression.

SSRIs are among the world’s most prescribed medicines; between 2015 and 2018, 13% of American adults were on antidepressants. While the drugs have helped millions of people ease symptoms of depression and anxiety, the mechanism behind them is still unknown - to scientists, companies, and the public. There is also controversy behind the topic of how much these improvements can be attributed to the antidepressants themselves; one large factor why antidepressants work could be attributed to the “placebo effect,” a non-pharmacological mechanism.

The placebo effect and long-term effects of antipsychotics

The placebo effect is when a person's health improves after taking a "fake" treatment (like a sugar pill instead of an antidepressant) that appears real. This effect is due to the person's belief in the efficacy of their treatment and their expectation of feeling better. In the 1990s, famous Harvard psychologist Irving Kirsch studied the placebo effect in the pharmaceutical field and found that a third of people taking placebo pills demonstrated significant improvement. The real kicker was that clinical trials showed that antidepressants worked only slightly better than placebos, and this improvement was not statistically significant. A recent study further delved into Kirsch's work. The FDA investigated all the placebo-controlled trials of antidepressants submitted to them - this spanned a total of 242 studies and over 73,000 participants. They found an average difference of only 1.75 points between the drug and placebo effect. A further breakdown of this data found that the placebo and the drug worked similarly among 85% of the participants, but the drug significantly outperformed the placebo in 15% of the participants (Simons 2022).

The conclusions from this research show that antidepressants can indeed be incredibly beneficial to some people struggling with depression, among other mental health concerns. However, considering antidepressants can't outperform placebos in most patients & antidepressants are saddled with a host of side effects, they shouldn't be used as a blanket treatment.

Long-term side effects of antidepressants are plentiful. In 2016, medical journal Patient Preference and Adherence surveyed people on long-term antidepressants and found that 72% reported sexual problems, 46% reported reduced positive feelings, 36% reported suicidal thoughts, and 43% reported feeling like they were addicted. In 2019, a study in the UK found that 56% of patients who stopped antidepressants suffered withdrawal symptoms for weeks to months, and 46% described these symptoms as severe. The researchers concluded withdrawal symptoms were a root cause of long-term antidepressants

use, as withdrawal was often confused with relapse.

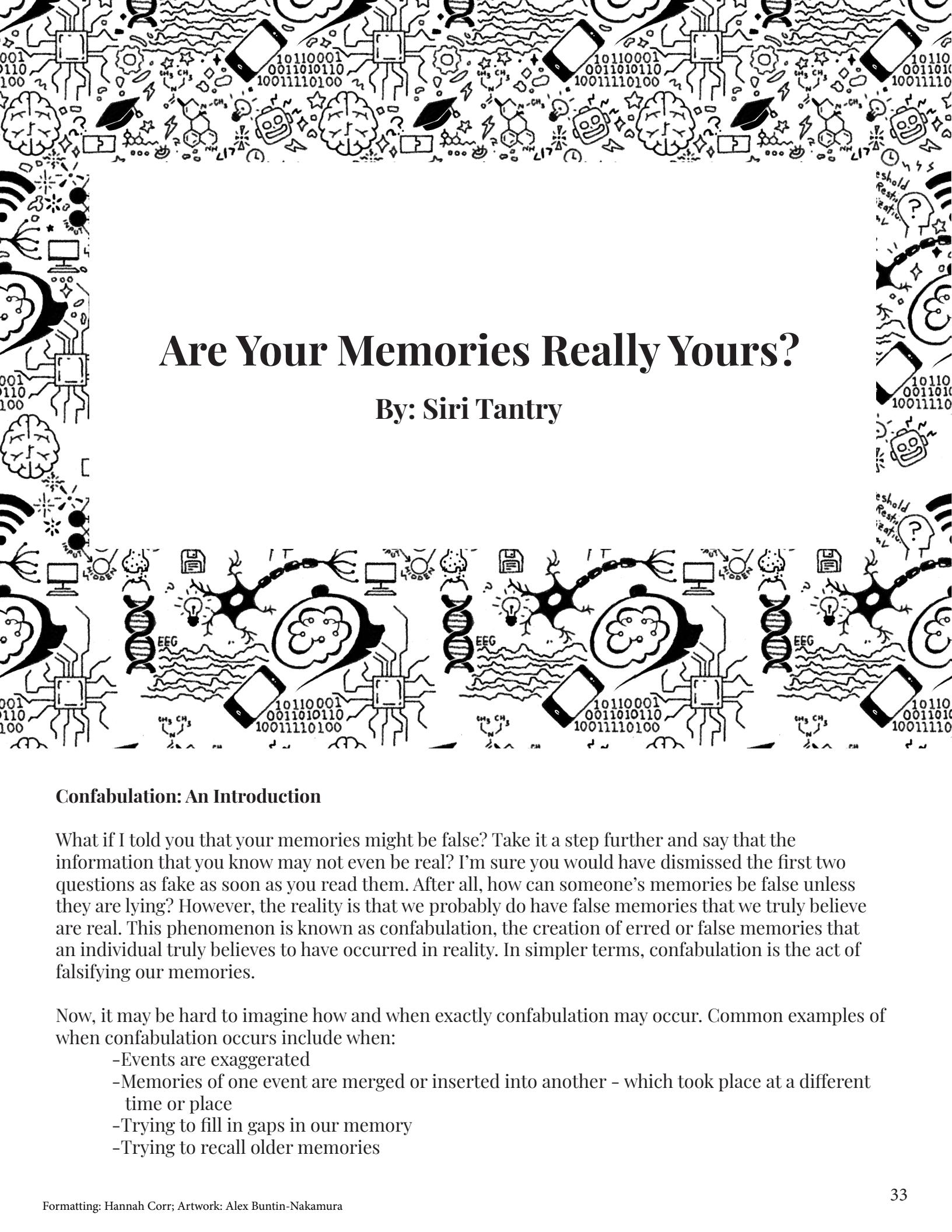
Several studies have also been conducted on how long-term use of antidepressants and antipsychotics (drugs used to treat schizophrenia) have affected patients. A 20-year longitudinal study by researchers at the University of Illinois found that while antipsychotics reduced psychosis for most patients in the short run, by the four-year and 20-year marks, patients with schizophrenia who weren't prescribed antipsychotics had significantly higher work functioning and fewer symptoms than those who were. Further studies by the team concluded the same.

Most of the studies that 'prove' the efficacy of these antidepressants and antipsychotics are funded by pharmaceutical companies.

While the beneficial effects of antidepressants cannot be understated, the lack of knowledge about their mechanisms enable the pharmaceutical industry to exploit the narrative and perpetuate a false 'chemical imbalance' theory. Over the past few decades, this theory has been used to explain most psychological phenomena - from grief, eating habits, to mood - and has been a huge part in the overmedication of today's generation. The propagation of this theory plays a huge role in the ever-expanding DSM and earns the pharmaceutical industry billions of dollars each year.

The pharmaceutical industry, like other medical industries, is ripe with corruption. This corruption is pervasive and insidious - it infiltrates the content of the DSM; it manipulates public knowledge about psychiatry; and it presents medication as the only viable solution for an ever-expanding list of diagnoses.

According to the World Health Organization, "childhood adversity and instability, abuse and neglect, socio-economic disadvantage, discrimination, bullying, rape, incarceration, war, substance misuse, and human rights violations all make far greater contributions to mental ill-health than heredity" (Wand 2018). As long as modern psychiatry boils mental health down to a biological diagnosis, the roots of people's problems will never be truly addressed.



Are Your Memories Really Yours?

By: Siri Tantry

Confabulation: An Introduction

What if I told you that your memories might be false? Take it a step further and say that the information that you know may not even be real? I'm sure you would have dismissed the first two questions as fake as soon as you read them. After all, how can someone's memories be false unless they are lying? However, the reality is that we probably do have false memories that we truly believe are real. This phenomenon is known as confabulation, the creation of erred or false memories that an individual truly believes to have occurred in reality. In simpler terms, confabulation is the act of falsifying our memories.

Now, it may be hard to imagine how and when exactly confabulation may occur. Common examples of when confabulation occurs include when:

- Events are exaggerated
- Memories of one event are merged or inserted into another - which took place at a different time or place
- Trying to fill in gaps in our memory
- Trying to recall older memories

For example, if an adult were to recall their third birthday party, there would be an extremely high chance that they would experience confabulation because of the time gap from retrieving that memory to recalling that exact memory. There will surely be some gaps that will take place in the memory, which our brain will try to fill in with other details that may not have even occurred. Additionally, it must be noted that confabulation, in its essence, is an extremely personal and unique occurrence that each individual is affected by. In other words, one person's experience with confabulation may not be the same as another's. We often cannot even correctly identify whether a memory is correctly identified as false or confabulated.

Moreover, it is important to consider that while anyone can be affected by confabulation, typically, those diagnosed with brain damage, Fetal Alcohol Spectrum Disorder, traumatic brain injury, and Wernicke-Korsakoff (a degenerative memory disorder that is associated with alcoholism) are more likely to experience the condition of confabulation.

Confabulation: Trying to Make Sense of It

When studying confabulation occurring in individuals who haven't been affected by the conditions listed above, it can be challenging to understand why it actually occurs. Obviously, no one wants to deliberately speak of their false memories or create their own memories (when they have no intention of lying). Given this, some explanations include overlearning and poor encoding. While it's no secret that our brain processes a large amount of information, there are times when there is too much information for the brain to process, overwhelming the brain – hence confabulation occurs. To further elaborate, certain pieces of information tend to emerge with greater importance in an individual's brain, causing a sort of overcrowding for the other details that are stored in the memory. So, if or when gaps do occur in the memory of a specific event, the information that stood out more can end up dominating the memory causing other facts and memories to be forced out.

Additionally, people also experience confabulation when poor encoding takes place. To expand further on the idea of poor encoding, often, information that helps create our memories may not truly and fully be stored in the brain, which can cause gaps in our memories to occur and also cause further confabulation. A common example of this would be when individuals are distracted. When we are not fully focused during a moment of an event, there are higher chances of them not paying attention to key details that would help build memories of the event. Thus, when recalling that memory where an individual did not truly pay attention to what was happening, they are more likely to misremember what occurred or create their own fillings of the gaps when recalling the memory.

Confabulation: Can it be Cured?

One may expect there to be a cure for confabulation, given all the information the scientific community has about it. Unfortunately, there is no set-in-stone cure to treat confabulation and prevent individuals from unintentionally creating false memories. However, problems and experiences associated with the phenomena of confabulation can be treated through psychotherapeutic and cognitive behavioral treatments. Specifically, the technique encourages people to question what exactly they remember and what they don't. Based on this information, the individual is encouraged to respond to questions that ask them to recall a specific part of the memory by saying they do not know or aren't sure rather than confabulating a response that may not have even existed. More specifically, research shows that neuropsychological treatment was supported for confabulation in people who experienced a brain injury. In a 2017 published clinical trial study, the experimental group of 20 confabulators received nine treatment sessions that consisted of some brief material that they had to learn and then recall both immediately and after a certain period of time. They then received feedback on their performance on and after recording and analyzing the pre-and post-treatment

measurements. The results showcased a “significant decrease in the confabulations that took place as well as a significant increase in correct responses in the experimental group” while the control group did not improve during the period. As seen from this study, though there isn’t a clear cure for confabulation, the research did provide valuable insight into the possibility and benefits of using neuropsychological treatment.

Confabulation: Where does Neurotechnology come in?

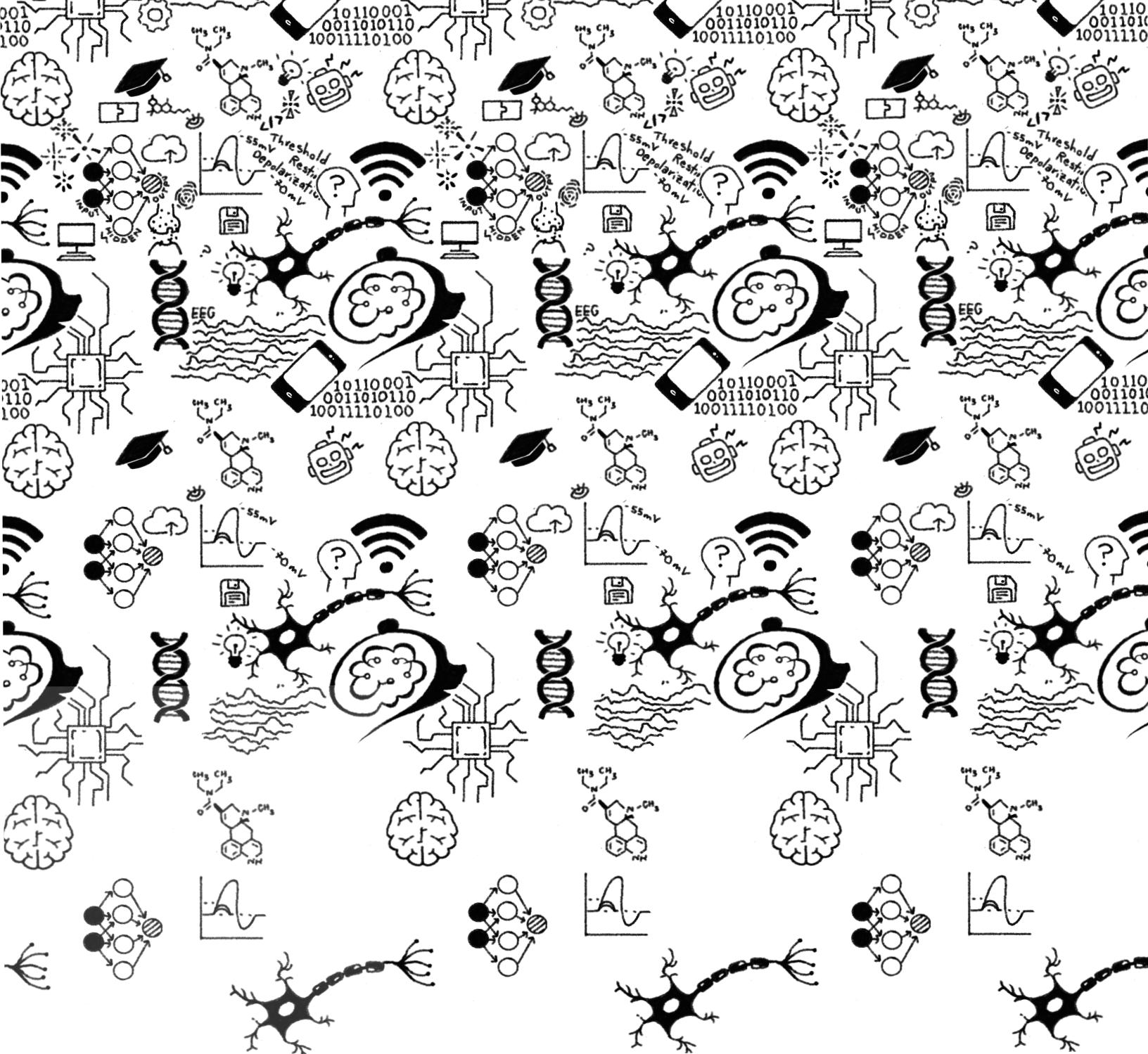
While the connection between neurotechnology and confabulations exists, it is still relatively new, and little research has been published for the public. However, there has been some research regarding technology and how it relates to confabulations. To engage in the idea of using neurotechnology to find a cure, it is important that we first understand how confabulation is explained with regards to technology. Essentially, experiences that alter our cognitive experiences (like confabulations) can be explained through a “reduced integration between the subjective and the objective components [that are] related to experience.” By using High-Density Electroencephalograms (EEG), alpha oscillations are markers for the two basic processes (subjective and objective components related to experience), which can then allow for the development of new precision treatments to increase the stimulation of the integration between the two processes. The integration can then increase while altered cognitive experiences such as confabulations decrease. Research and technology integration has already been underway; It can be seen through the relationship “between perceived sensory events and their subjective interpretations” at the clinical level in schizophrenia and the case of confabulations. Essentially, by using neurotechnology, confabulation can be decreased in the patient. Similarly, another technology, known as, Transcranial Magnetic Stimulation (TMS), was used to verify the connection between the subjective and objective components related to experience in terms of confabulation. The neural oscillations could now be directly controlled using TMS. Through the independent increase and decreased speed of the oscillations for the alpha wave, the accuracy and confidence a person has in their perception could be verified and proved that there was a correspondence. Through this technology and the results that the experiment generated, there may be a high possibility and hope that, alpha wave oscillation speed manipulation will be used to study confabulations and their cures in the future.

Confabulation: What Now?

With this new information about confabulations and the implications it has with treatment through technology, it can be seen that we may soon be seeing a unique and further productive manner of treating confabulations. While current technological research has been used mainly in helping treat schizophrenia, clinical trials will hopefully be on their way to help treat confabulations soon.

While not everyone is heavily affected by confabulations in their everyday lives, it is important to understand that they do exist in our lives; There is a very good chance that many memories of our lives may not be exactly as we recall them. Perhaps there have been a few details that we falsely created, and we have been sharing those erred memories with everyone around us. Additionally, understanding confabulations only helps to deepen our awareness and encourages an increase in our attention and awareness when being part of certain memories and events because who knows when our memory will fail us and start creating false memories. What if it’s happening to you right now?

Now, given your new understanding of confabulations, I want you to go back and reflect on your memories. At the beginning of this read, I asked you to think about a situation where your memories were false. Now I implore you, which of your memories are false? Are you able to correctly identify gaps in your own memory? Which memories have you confabulated? Which memories are truly yours?



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TWO HANDS, TWO MINDS, ONE PERSON

By Zane Ashkar



Imagine this: one day, you suffer from a stroke. You go to the hospital, just to find out not only did you suffer from a stroke, but you have a primary malignant brain tumor. But no worries, you're at one of the top neurosurgical medical facilities, so you're in good hands. Wrong. After some weeks of recovery, time off work, and many breakfasts in bed, you realize that both of your arms have a mind of their own. Eventually, when you're trying to eat your waffles, your other hand stops it from reaching your mouth. Trying to drink a glass of orange juice? Nope, you smack it out of your hand, and the glass breaks. Now you're asking yourself why you would do such a thing, but you can't figure it out.

This constant confusion and frustration becomes your daily life. You have two arms, each with a mind of their own. Alien Hand Syndrome. What a mindfuck, right?

Background on Alien Hand Syndrome

Alien Hand Syndrome (AHS) is a condition where a person is physiologically unable to control their hand movements. AHS is characterized as impulsive, involuntary movements of one's arms, and has no known cure. The first case was documented in 1908, when Dr. Kurt Goldstein was treating a woman who suffered from a stroke who developed what are now known symptoms of AHS. But it was not until 1972 when medical professionals began referring to this phenomenon as Alien Hand Syndrome.¹

The etiology of this syndrome is not entirely known, and patients who have AHS do not manifest with any concrete symptoms. However, most patients present with some form of autonomic neuropathy, which is classified as damage to nerves responsible for motor functions in the body. It's not genetic, and no certain injury will lead to this 100% of the time. Data from the

Mayo Clinic Medical Records Linkage System shows that patients with AHS resulted from a variety of different medical problems including cerebral infarction/hemorrhage, CBD (corticobasal degeneration), Alzheimer's Disease, Creutzfeldt-Jakob Disease, brain tumors, and stroke.² Patients with AHS also experience other neurological irregularities present in their speech, spastic motor movements, and in recollection of memory.

Different Manifestations of Alien Hand Syndrome and Their Diagnoses

Alien Hand Syndrome can present in patients in one of three ways: frontal AHS, callosal AHS, and posterior AHS.³

The frontal variant of AHS is the most common form of the syndrome, and is caused by damage to the anterior corpus callosum, the extremely dense bundle of myelinated nerve fibers that are responsible for the transmission of information and signals from each hemisphere of the brain to the other.⁴

A damaged corpus callosum can result from callosal infarction, stroke, neurosurgery, tumors, and more. A corpus callosotomy is a procedure performed on patients who suffer from seizures, done by cutting the connection between the two hemispheres. Although this procedure is considered a last resort method to control seizures, it is often offered to patients who have received other forms of treatment, but experienced no success in alleviating the seizures. Candidates for this surgery most commonly suffer from drop attacks, a type of seizure resulting in sudden falls, accompanied by loss of consciousness and abnormal muscle contractions. Unfortunately, the corpus callosotomy has serious side effects, including speech problems, stroke, and AHS.⁵

When damaged, cerebral hemispheres, each of which control one side of the body, cannot communicate with each other, which results in coordination problems. Patients who have frontal AHS present with lesions in the anterior corpus callosum, anterior cingulate gyrus, and medial prefrontal cortex of their dominant hemisphere, thus affecting their dominant hand. The lack of coordination in their dominant hand can be explained by the increased frequency of dominant limb spastic reflexes paired with non-dominant inhibition. The anterior cingulate gyrus is responsible for behavior regulation, attention allocation, and behavior in response to a known error in one's environment.⁶

Patients with the frontal variant are affected in their dominant hand and involuntarily reach and grasp for objects, perform groping movements, and tonic grasping.⁷

The callosal variant of AHS is caused by callosal lesions in the corpus callosum.

Patients who present with this form of AHS are characterized by having damage to the rostral fibers, the region of the corpus callosum responsible for transmission of information between the frontal lobes. Callosal AHS affects the patient's non-dominant hand, and causes intermanual conflict. Intermanual conflict is essentially when each of your hands has a different mind, resulting in them being unable to cooperate in order to complete a task.. Trying to drink a glass of water? Nope. You can't even open a door without the other hand inducing intermanual conflict.

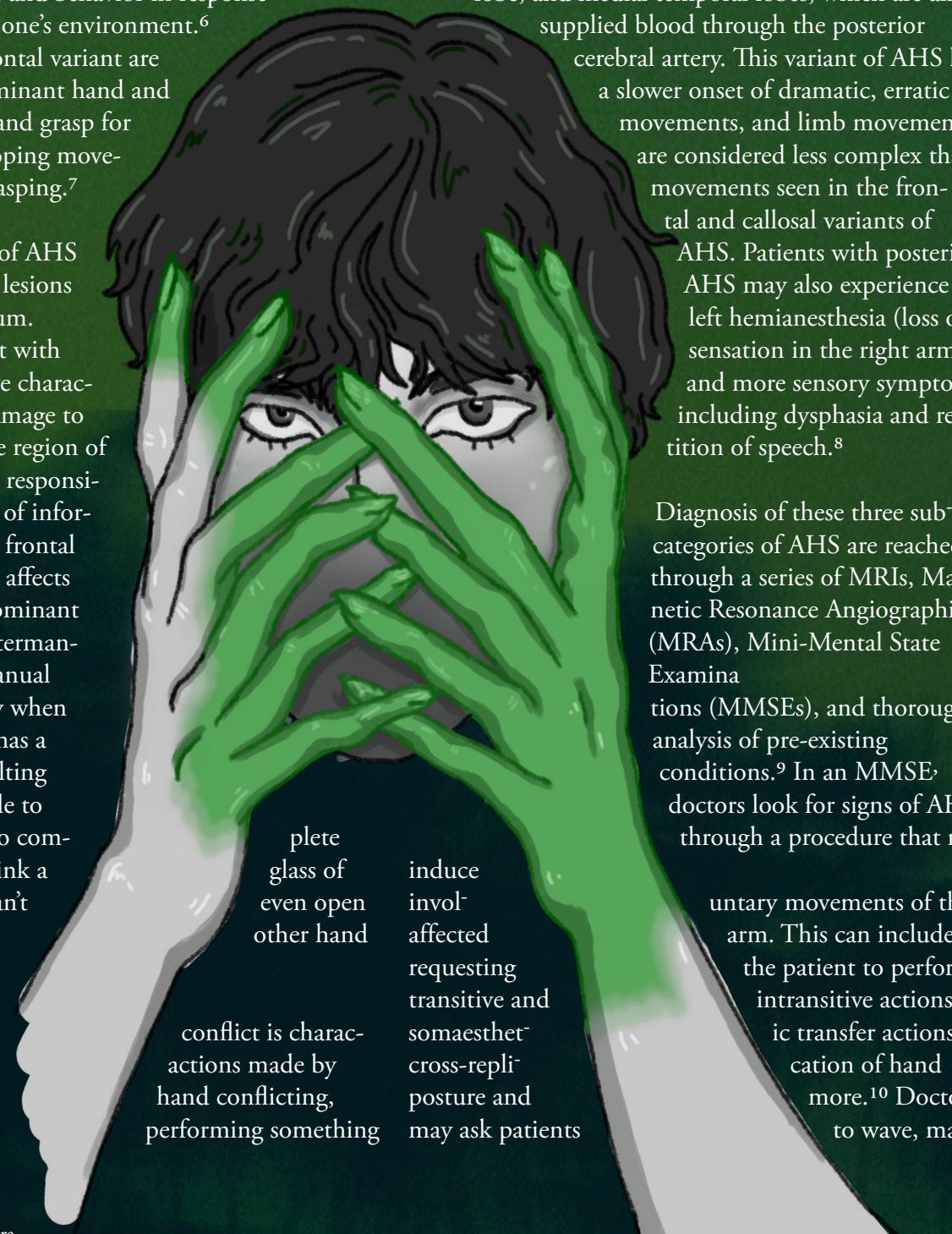
Intermanual conflict is characterized by motor actions made by the non-dominant hand conflicting, interrupting, or performing something

out of sync with the dominant hand. Patients also suffer from apraxia in the non-dominant hand, putting the two limbs even further into an "out of sync" state. Apraxia is a disorder causing patients to have decreased coordination, increasing difficulty in daily tasks, regardless of having the conscious desire to do so. Apraxia can manifest as speech or motor, called speech apraxia and ideomotor respectively.

Posterior AHS is caused by lesions in the cortical and subcortical structures, including the thalamus, parietal lobe, and medial temporal lobes, which are all supplied blood through the posterior cerebral artery. This variant of AHS has a slower onset of dramatic, erratic movements, and limb movements are considered less complex than movements seen in the frontal and callosal variants of AHS. Patients with posterior AHS may also experience left hemianesthesia (loss of sensation in the right arm), and more sensory symptoms including dysphasia and repetition of speech.⁸

Diagnosis of these three sub-categories of AHS are reached through a series of MRIs, Magnetic Resonance Angiographies (MRAs), Mini-Mental State Examinations (MMSEs), and thorough analysis of pre-existing conditions.⁹ In an MMSE doctors look for signs of AHS through a procedure that may

unitary movements of the arm. This can include the patient to perform intransitive actions, cross-representation of hand posture and more.¹⁰ Doctors may ask patients to wave, make



the same gesture with both hands simultaneously, name objects by touch, or demonstrate how to use a specific tool. These tests help to show the provider how much control a patient has over their supposedly affected limb, giving them a sense of their voluntary motor dysfunction. During this test, if the provider notices a patient performing repetitive mirrored, dyspraxic movements, or strong grasping there is a good chance the patient suffers from a form of AHS.¹¹

The Future of Alien Hand Syndrome

As of now, there is no cure, nor any therapies that are known to control the symptoms of AHS. One therapy that has been found to increase the quality of life of patients is behavioral modifications among their every day lives. Patients who distract their affected limb with an object or restrict it by putting it in their pocket or behind their back experienced a decrease in the frequency of involuntary movements.¹² This type of behavioral therapy can extend and be tailored to the different forms of AHS, including spatial recognition exercises and visual reinforcements.

So there it is. It's you against yourself for the rest of your life. You will forever be hindered in your ability to do most things in life. Living alone isn't a viable option anymore and neither is going out alone. The uncanny nature of this syndrome paired with the sheer possibilities that could be labeled as its concrete cause continues to baffle doctors, researchers, and scientists to this day.

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⁴ Harini Sarva, Andres Deik, and William Lawrence Severt. "Pathophysiology and Treatment of Alien Hand Syndrome" (England: Tremor and Other Hyperkinetic Movements, 2011)

⁵ Ragesh Panikkath et al. "The Alien Hand Syndrome" (USA: Baylor University Medical Center, 2014)

⁶ Harini Sarva, Andres Deik, and William Lawrence Severt. "Pathophysiology and Treatment of Alien Hand Syndrome" (England: Tremor and Other Hyperkinetic Movements, 2011)

⁷ Keith Joseph and Martin Rossor. "The Alien Limb" (London: Divisions of Behavioral Neurology and Movement Disorders, 2004)

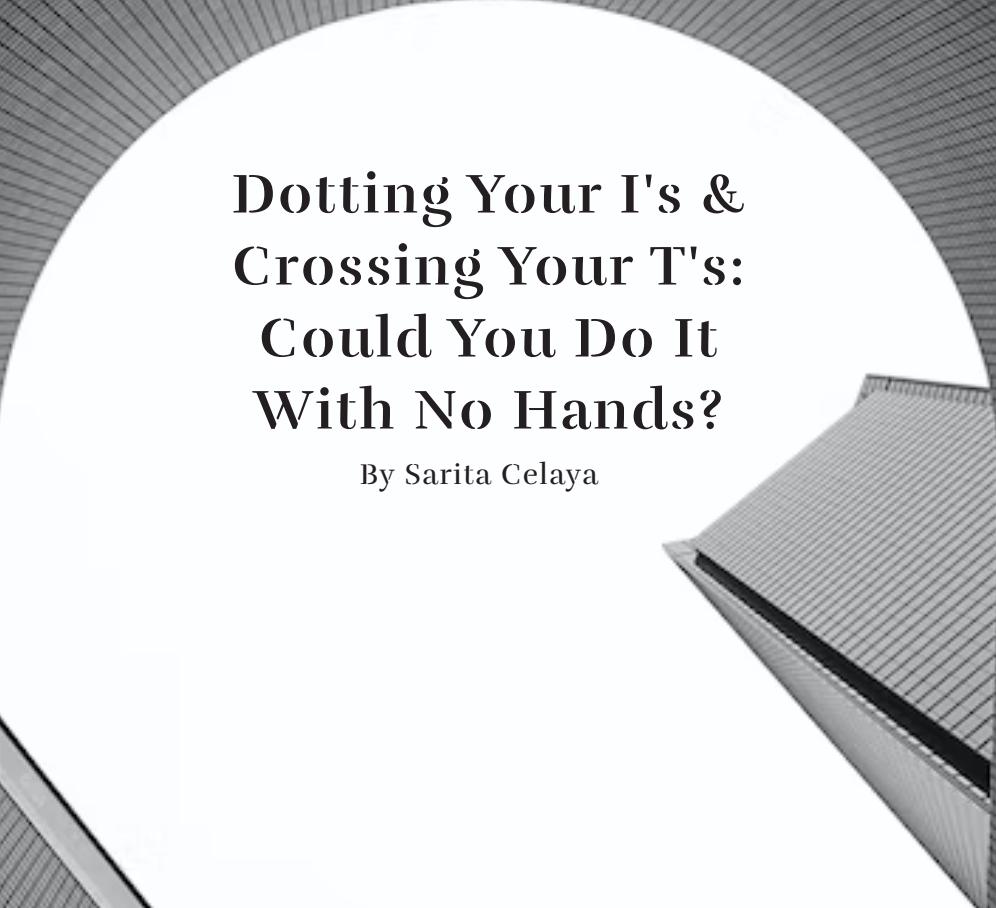
⁸ Gaurav Gheewala et al. "Posterior Alien Hand Syndrome from Acute Ischemic Left Parietal Lobe Infarction" (USA: Cureus, 2019)

⁹ Aakash Pradhan et al. "An Investigation on the Preconditions and Diagnosis Methods for Alien Hand Syndrome" (USA: Cureus, 2022)

¹⁰ Keith Joseph and Martin Rossor. "The Alien Limb" (London: Divisions of Behavioral Neurology and Movement Disorders, 2004)

¹¹ Aakash Pradhan et al. "An Investigation on the Preconditions and Diagnosis Methods for Alien Hand Syndrome" (USA: Cureus, 2022)

¹² Harini Sarva, Andres Deik, and William Lawrence Severt. "Pathophysiology and Treatment of Alien Hand Syndrome" (England: Tremor and Other Hyperkinetic Movements, 2011)



Dotting Your I's & Crossing Your T's: Could You Do It With No Hands?

By Sarita Celaya

Independence is not Just Physical

Independence is a powerful and essential component of human existence. On a very large scale, we might associate this concept with countries fighting against a tyrannical government, or perhaps we might think of civil rights movements and how humans will always resist and oppose oppression. Over the course of an individual's life independence can look like many things: getting your first job, renting an apartment, or buying a car. Whatever image this word might conjure up, every person can agree that to be independent is to feel free – emotions which are key to shaping the lives of individuals. So what happens when you lose that sense of independence? What can anyone do when their body will no longer cooperate and function as they command it to?

Now consider your average day: you wake up, use the restroom,

brush your teeth, get dressed, eat breakfast, and go to work. Throughout the day, you express your ideas and needs with friends, peers, family, or coworkers, and when you get home you wind down and rest. These actions, which many of us take for granted, are all forms of exercising independence. You are in control of your body, from your limbs to the words you speak. But for someone who has severe speech or motor function impairment, this kind of routine is not a reality.

Telekinetic Penmanship

Writing is one of the oldest forms of communication and human expression around. Created in Mesopotamia, now present-day Iraq, around 3200 BC¹ writing has blossomed into an essential motor skill. Writing is, in its traditional form, physical: pencils, pens, keyboards. When an individual loses their ability to write out their thoughts, especially when they cannot speak, they

lose a part of their independence. Their sense of self can become fragile, as their needs may not be met and they cannot express how they feel. However, a new technology may be able to change that.

Even after an individual's body becomes paralyzed, their brain can still produce signals of intent related to movement². Even if this individual has not been able to execute a motion for 20 years, their brain can still remember what it was like, and that is all that is needed for telekinetic penmanship. Brain-computer interfaces, or BCIs for short, are computer-based systems that take signals produced in the brain, process them, and translate them into commands, which are then sent to output devices³. New BCIs are now able to process these intent-to-move signals and translate them into commands such as handwriting or typing. This neurotechnology enables people to communicate who may be paralyzed or may suffer from a

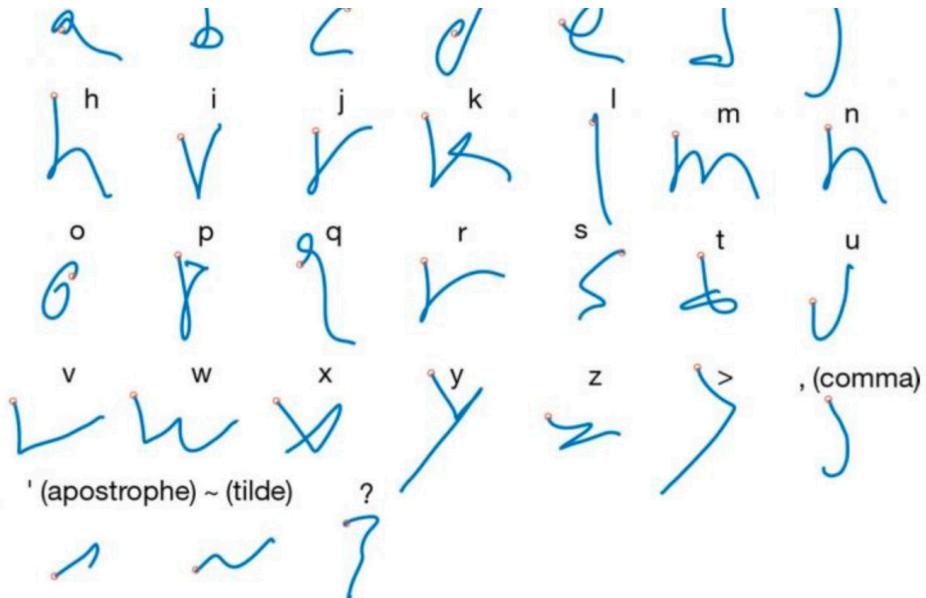
¹Wright, James. The Evolution of Writing. The University of Texas at Austin. International Encyclopedia of Social and Behavioral Sciences, Elsevier, 2014.

²O'Leary, Karen. Handwriting with a Brain Implant. Nature News. Nature Publishing Group, May 27, 2021.

speech or motor function impairment. Thus, someone can write using only thought.

How Does Hands-Off Handwriting Work?

This BCI uses small electrodes to record signals from the brain associated with handwriting and then processes them to create text on a screen. This is produced as it is being thought, mimicking how handwriting would physically play out in real time. The handwriting BCI functions based on an array of microelectrodes that take in signals from the precentral gyrus – your precentral gyrus is the part of your brain that is responsible for the signals associated with voluntary movement⁴. Using this technology, a study that was part of a BrainGate clinical trial found that a paralyzed participant was able to “write” by thought at a rate of 90 WPM⁵. The leaders of this clinical trial included doctors and professors from Brown University, Stanford University, and Howard Hughes Medical Institute. There are two ways that doctors are using this technology. In the first method, the participant imagines they are clicking keys on a keyboard, and the sensor records which keys they are clicking. In the second method, the participant concentrates on imagining physically writing the letters of the alphabet, and the process repeats.



6

(Above is an example of a patient's recreated handwriting using the handwriting BCI.)

Restorative Neurotechnology

This technology can restore an individual's confidence and sense of self. In regaining independence, these individuals can feel more in control of their disabilities and therefore their lives. Restoring their ability to communicate is a significant and positive change, especially for their mental health. This BCI can make it possible for people with disabilities to communicate exactly their needs, wants, thoughts, and feelings. Doctors can better understand their patients, and these individuals can get back those shared moments between themselves and their loved ones. Restorative

treatment does not just give back a learned skill, it centers the whole life of an individual and holistically aids them in being as “them” as they can be. This technology does just that.

³Shih, Jerry J, Dean J Krusienski, and Jonathan R Wolpaw. Brain-Computer Interfaces in Medicine. Mayo Clinic proceedings. Mayo Foundation, March 2012.

⁴Xue, Jiachi. Handwriting with Brain Computer Interface. Journal of Physics: Conference Series 1865 042026, 2021.

⁵Brain-Computer Interface Turns Mental Handwriting into Text. Tectales Tagging Medical Technology, May 21, 2021.

⁶Brain-Computer Interface Turns Mental Handwriting into Text.



Why Can't We Perform Brain Transplants?

In the past century, improvements in medical technology have allowed for us to accomplish procedures that save millions of lives each year. The concept of transplantation between humans, whether through organ, bone marrow, or blood, has healed millions and allowed for us to create life out of otherwise dead bodies. It's fascinating to think that we can simply take one's body and use it to heal another's. But, one part of us has been un-transplantable. While there are countless stories about heart transplants, liver transplants, and bone marrow transplants, the brain has never successfully been transplanted.

The history of brain transplants

While the concept of brain transplants remains mostly theoretical, a few notable attempts have been made in the past. In 1982, Dr. Dorothy T. Krieger, chief of endocrinology at Mount Sinai Medical Center in New York City, successfully completed a partial brain transplant among mice. In 1998, a team of surgeons from the University of Pittsburgh Medical Center at-

tempted to transplant a group of cells, originally from a cancer patient and chemically modified to turn into brain cells, to 62 year-old Alma Cerasini in an effort to reduce brain damage following a stroke she experienced. This was the first recorded time that lab-grown adult cells were implanted into a human brain, and, while there are limited updates, she suffered no initial ill effects from the procedure. While attempts such as the ones mentioned above have been performed, the full transplantation of the brain has proved not physically possible.

What's stopping us?

For one, plasticity. The brain is plastic, meaning that it's always changing, through experience, age, and exposure. Such exposure allows for new neural pathways to strengthen, while inactive neural pathways eventually become weak and die. This process is called synaptic pruning. As we grow older, plasticity decreases to stabilize what we have already learned, making it more difficult for older brains to perform tasks that require brain

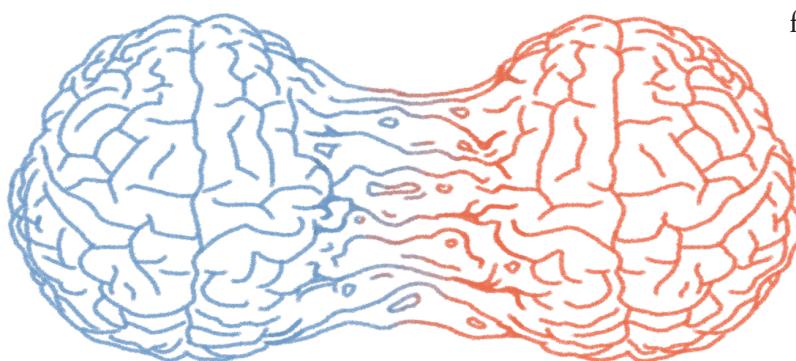
function, such as learning and executing functions like movement and memory. This could easily decrease the quality of life for a hypothetical recipient due to a lack of neuroplasticity, which could dampen the recipient's ability to perform such executive functions. Furthermore, all cells in organisms have antigens, foreign substances that induce immune responses, on their cell membrane. As noted by the Wu Tsai Neuroscience Institute at Stanford University, "Most antigens are specified to individuals and if they are in contact with immune cells of another person, the immune cells will see the antigens as foreign objects and want to kill the cells." The immune system of our hypo-

thetical recipient could easily reject the new antigens which come with the new brain, and this could lead to major, deadly problems. Another factor to consider is the placement of the brain in the new body. With current technology, while we are able to perform surgeries on both the skull and spinal cord, we are not yet capable of implanting the brain into a new skull, in a way which fuses with the spinal cord, which would be necessary to physically complete a brain transplant, as nerves are fragile, and can be easily injured by any kind of physical manipulation; this can be detrimental because damaged nerves can prevent signal transmission to the brain.

Let's say we became physically capable of brain transplants. Are we good to go?

Imagine waking up in a world where your brain is transplanted into someone else's body, with the skin and bones of someone new, who has perhaps grown of old age and once possessed a life's worth of experiences and memories—someone who has grown mentally through the course of their life and has now passed on with your soul in their body. How would you feel?

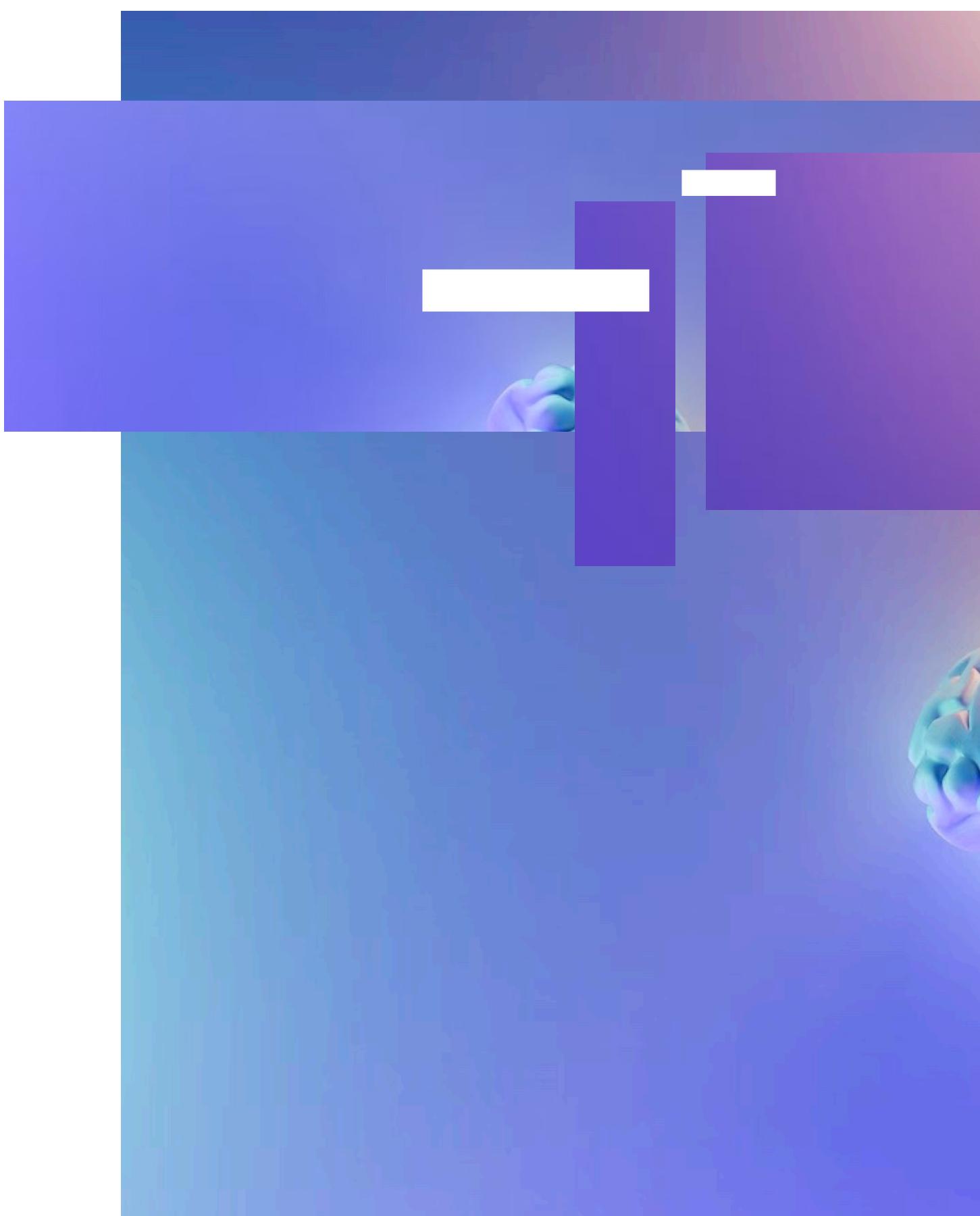
Would you even feel? And would you feel it as yourself, or as them? These are all part of the grand question we must consider when thinking about the ethics of brain transplants. The Journal of Medicine and Philosophy: A Forum for Bioethics and Philosophy of Medicine, for one, argues the idea that "one's personal identity depends on the continuity of one's mind (namely, one's consciousness, memory, emotion, feeling, and the like), not on the intactness of one's body."



While there is no ethical standard put in place just yet, the concept of going forward with such a procedure is one that will need to be discussed and approved by the patient, recipient, government, and the medical authorities involved. Considering the question of identity that follows brain transplantation, getting that consent may prove difficult or impossible. After all, whose conscience is consenting?

What we can-and are expected-to be able to do

While brain transplants are not yet possible, doctors are expecting major advancements within the next few decades. Dr. Bruce Matthew, a former clinical lead for neurosurgery at Hull University, has suggested moving the entire spinal column along with the brain, which would get rid of the need to attach the brain to the new spine, and thus could bring us one step closer to achieving the brain transplant. As we consider current physical and ethical barriers, along with the potential future of brain transplants, it is fascinating to reflect upon the power of medical developments to influence our lives. While brain transplants may or may not ever become a practiced procedure, considering the physical and philosophical components of its effects can bring us one step closer to understanding the intricacy and complexity of our brilliant brains.



An Imposter in Your Brain

By Tessanya Gunatilake

Most of us have heard of the game Among Us. Players must complete their tasks while trying to find the imposter, who is trying to kill everyone on board. While this game seems far from reality, what if I told you there was an imposter in your brain? An imposter who, like in Among Us, is destructive and can be the leading cause of your downfall.

Growing up in the Bronx with a single mother and brother, a girl felt like a fraud and that she did not deserve the accomplishments she made. As she said in a speech, “I have spent my years since Princeton, while at law school and in my various professional jobs, not feeling completely a part of the worlds I inhabit. I am always looking over my shoulder, wondering if I measure up.” “She” is Sonia Sotomayor, the first Hispanic American justice to serve on the Supreme Court. Countless successful people have experienced what Sotomayor describes, indicating that feelings of self-doubt do not discriminate. What all of these incredible people have in common is a feeling known as imposter syndrome, and more than 80% of the population experience it at some point in their life¹.

While you may think these geniuses and celebrities are miles above you, you may have more in common with them than you think.

Imposter syndrome is a psychological trick your brain plays to doubt your skills and accomplishments, and believe deep down that you are a fraud.

And what happens when you tell a person you suffer from this?

You need more confidence.

Like that will fix it.

Just focus on your accomplishments.

Don't you think I've already tried that?

While this advice may come from a good place at heart, you need to get to the root cause of why you

have imposter syndrome and remind yourself that that feeling is normal.

Why do people feel imposter syndrome?

Now that we have established- hopefully- that you are not the only one who has this lingering feeling, let us get to the root cause of why people have imposter syndrome. Imposter syndrome is a vicious cycle of gaining accomplishments but believing you don't deserve them. Various factors, including societal and familial pressures, prejudice, and the workplace environment. Imposter syndrome is especially prevalent in underrepresented students. This prevalence is due to biases, especially systemic biases, and microaggressions- brief actions or comments that target marginalized groups. These microaggressions build up and can cause one to doubt themselves. Furthermore, lack of representation causes an individual to feel like they need to fit in. When their identity is not represented, the feeling of not fitting in- which correlates to imposter syndrome- is intensified. This is another reason why imposter syndrome impacts underrepresented identities at a greater level².

Imposter Syndrome at Berkeley

In a 2019 study, it was found that up to 82% of people may face imposter syndrome at some point in their lives. Imposter syndrome is especially prevalent in college. Students are constantly surrounded by peers and assume their academic work does not align with their grades. Dr. Sahar Yousef, a Cognitive Neuroscientist at UC Berkeley, says “We’re seeing imposter syndrome and burnout start to speak to each other in ways that are of concern, but it also makes sense.”³ UC Berkeley is

Formatting/
Artwork:
Hannah Corr

¹8 in 10 experience Imposter Syndrome because they ignore a universal ... (n.d.). Retrieved October 23, 2022, from <https://www.inc.com/jeff-haden/8-in-10-experience-impostor-syndrome-because-they-ignore-a-universal-truth-about-success.html>

²BBC. (n.d.). Why Imposter Syndrome hits women and women of colour harder. BBC Worklife. Retrieved October 22, 2022, from <https://www.bbc.com/worklife/article/20200724-why-impostor-syndrome-hits-women-and-women-of-colour-harder>

³Lucchesi, N. (2022, July 21). How to stop imposter syndrome and burnout before they take hold of your team. The Asana Blog. Retrieved October 22, 2022, from <https://blog.asana.com/2022/06/impostor-syndrome-burnout-employees/>

⁴Swart, T. (2019, August 8). Why you need to understand the neuroscience of imposter syndrome. Forbes. Retrieved October 22, 2022, from <https://www.forbes.com/sites/taraswart/2019/08/08/why-you-need-to-understand-the-neuroscience-of-impostor-syndrome/?sh=67ed1c8c80e7>

⁵DARPA RSS. (n.d.). Retrieved October 20, 2022, from <https://www.darpa.mil/program/targeted-neuroplasticity-training>

among the most academically rigorous universities in the world. With over 45,000 students who were at the top of their class in high school, one can feel that they are not worthy of attending this school or will not be able to keep up academically.

Neuroplasticity and Imposter Syndrome

Despite this natural instinct to feel like you are not enough, there are many resources to combat this feeling. First, there are holistic treatments such as positive affirmations, building self-trust, celebrating your successes, and accepting that perfection is not possible.

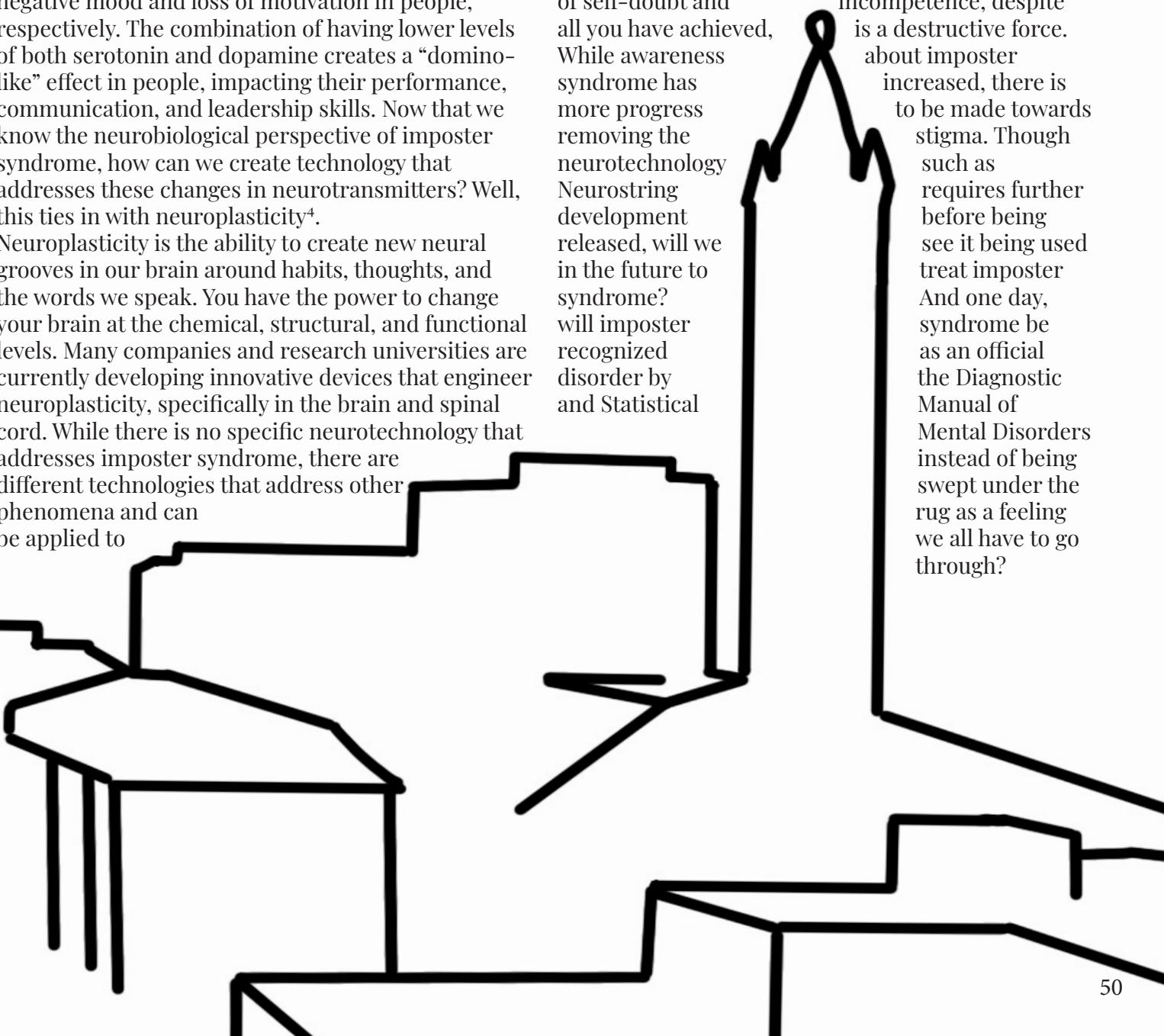
What is interesting, though, is how technology is trying to treat this feeling. To address this field, we need to know the biological mechanisms of imposter syndrome. Imposter syndrome is linked to lower levels of serotonin and dopamine which lead to a negative mood and loss of motivation in people, respectively. The combination of having lower levels of both serotonin and dopamine creates a “domino-like” effect in people, impacting their performance, communication, and leadership skills. Now that we know the neurobiological perspective of imposter syndrome, how can we create technology that addresses these changes in neurotransmitters? Well, this ties in with neuroplasticity⁴.

Neuroplasticity is the ability to create new neural grooves in our brain around habits, thoughts, and the words we speak. You have the power to change your brain at the chemical, structural, and functional levels. Many companies and research universities are currently developing innovative devices that engineer neuroplasticity, specifically in the brain and spinal cord. While there is no specific neurotechnology that addresses imposter syndrome, there are different technologies that address other phenomena and can be applied to

combatting imposter syndrome in the future. For example, the Targeted Neuroplasticity Training (TNT) program provides accelerated military training through non-invasive neurotechnology that elevates neurological signaling, thus improving cognitive skills⁵.

Along with this, NeuroString, a soft sensor for monamine neurotransmitters, has a high potential to manage imposter syndrome. NeuroString uses fast-scan cyclic voltammetry, a method that rapidly changes the voltage applied to a probe that reduces and oxidizes a target neurotransmitter. This technique generates a neurotransmitter-specific current. Transition-metal nanoparticles are created using laser carbonization enabling the sensor to analyze the neurochemistry of monamine neurotransmitters such as dopamine and serotonin. Imposter syndrome doesn't discriminate. The feeling of self-doubt and

incompetence, despite all you have achieved, is a destructive force. While awareness about imposter increased, there is to be made towards stigma. Though such as requires further before being see it being used treat imposter And one day, syndrome be as an official the Diagnostic Manual of Mental Disorders instead of being swept under the rug as a feeling we all have to go through?



Rapid EEG for EMTs? Fad or Possibility?

Re-assessing the approach of
brain-related injuries in the Standard
of Practice for Emergency Medicine

By Yoosung Jung

You are a paramedic and are called to a location where a mother reported that her daughter had a brief seizing episode. After arriving on the scene, the daughter seems alert and aware of her surroundings. You learn that she has a history of epileptic seizures and does not need to be taken to the hospital for a more detailed exam. After the mother signs the release forms and you have left the near vicinity, the girl experiences another tonic-clonic seizure with no

paramedic by her side and no benzodiazepines available. You could not have prepared for this, right?

It is difficult in the field of emergency medicine to take into account the internal physiology of the brain without neural technologies. In addition, the lack of time to perform life-saving interventions puts aside the importance of the brain. Though, in fairness, paramedics and EMTs alike have limited abilities regard-

ing their subjective methodology for identifying brain-related emergencies like oncoming seizures. It's difficult to tell neurological deficits through signs and symptoms that are often hard to observe--sometimes those symptoms don't even show until a problem like a seizure or a stroke exists.

¹The limitation of emergency responders is why using neural technologies in the field can save lives.



In 2017, Ceribell released a Rapid EEG headset in which physicians in hospitals could triage their patients through the collection of neuro-electrical data in minutes to identify whether seizures were expected. The headset uses a monitoring method called electroencephalography to interpret the brainwaves in a compact and portable system. This test involves small metal discs that attach to the scalp to interpret the brain's electrical signals on an EEG recording. The headset, taking only five minutes to set up changed the landscape in which emergency medicine physicians carried on with their approach to triage.

²Ceribell's Rapid EEG headset proved to be a groundbreaking aid for an area that requires intensive focus in a short amount of time. Therefore, an accurate rapid EEG could accompany an emergency field provider through their assessments, given their limitations of correctly identifying ambiguous symptoms.

³Those in charge of local budgeting could argue that neural technology would be too much of an expensive purchase in terms of training and inventory

for ambulance companies and fire departments but the same argument could be placed for the rise of the LUCAS, a device in the field of emergency medicine that saved countless of lives through its innovation of automatic CPR with a click of a button. This device eliminated the need for manual CPR saving both time and money while performing CPR better than any human could. In fact, the LUCAS had a 25% success rate of resuscitation for those who went into cardiac arrest versus the historical 10% from manual CPR. We are entering an era of emergency medicine where subjective intuition is not enough to meet the demands of the human body and especially the intricate enigma of the brain.

Ceribell's Rapid EEG in the field would allow for better triage, diagnosis, choice of transportation, and relaying of information to medical command. This would in return have a greater impact on the lives



of patients while efficiently saving

the time and resources of many ambulance and fire departments.

It can be seen as a concern that the use of neural technologies in emergency medicine is a violation of privacy for patients because of the possibility of data collection. In regards to this, it is important to consider that medical data is protected by HIPPA which, through the law, prevents a patient's health information from being disclosed. And in the situation of an emergency, unless a non-altered patient refuses care, implied consent gives the EMT and paramedic alike the right to care for the patient given the notion that they would rather be cared for than have concerns about privacy. Though in a scenario where the patient is awake and alert, they have the right to refuse care by signing an AMA, or Against Medical Advice form.

The convoluted politics of emergency medicine in regard to the division

of power among provider roles could limit such new technologies from entering the field, let alone allow EMTs to use such machinery with their standard of practice.

It is not a matter of if, but when these new technologies will accurately help patients on the field to help advance the importance of brain-related injuries. Dealing with the political and public push-back of introducing neural technologies into the world of emergency medicine can be a mind f***ck in itself. The understandable concern of privacy along with a natural fear of the fast-paced growth of neural technologies can sound confusing and worrisome to anybody who hears the word “EEG”. But through the careful navigation of these challenges through education on these new devices along with a leap of faith, we may be able to achieve just this:

You walk into the same scene where the mother previously called for her daughter’s seizing episode, but this time you place the EEG headset on her and in minutes are warned of an oncoming seizure. You load her up in the ambulance to take her to the hospital and possibly prevented a devastating situation.



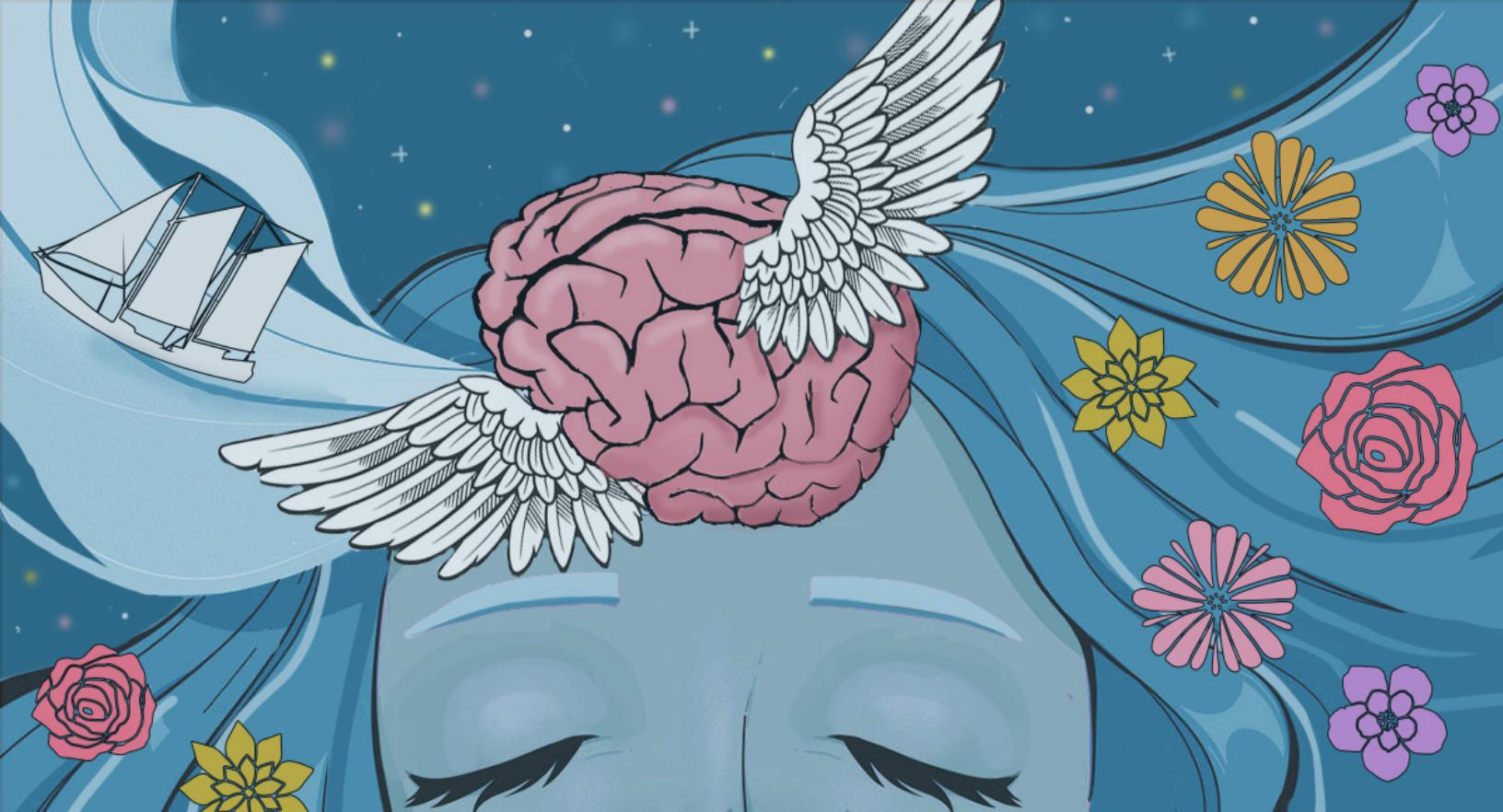
Artwork: Ceribell;
Whipsaw

And the reality is clear: You can prepare for this.

¹Ceribell EEG. (2022, October 8). Rapid Response EEG for seizure triage in minutes. Ceribell. <https://ceribell.com/rapid-response-eg/>

²Rapid Response EEG: The Future is Here and Techs Need to Get Onboard. (2020, September 3). Neurology Insights. <https://www.neurologyinsights.com/2020/09/04/rapid-response-eg-the-future-is-here-and-techs-need-to-get-onboard/>

³Clinical evidence - LUCAS - Chest Compression System. (n.d.). Retrieved October 29, 2022, from https://www.lucas-cpr.com/clinical_evidence/



Artwork: Hilary Lu

The Green Screen

By: Meltem Su

Your alarm starts raucously ringing and you suddenly wake up during the best part of your dream. You were imagining the plot for the next Oscar worthy movie, but now cannot remember any part of your dream. However, you have a weird feeling, putting weight on your heart. While dreaming, you had a heated argument with your best friend and now feel as though your friend is furious at you in “real life”. You quickly call your friend and tell them about the argument and ask if there is any problem, but your friend laughs and reassures that everything is fine.

The beauty of dreams can be a mind fuck. Being able to cherry pick the trajectory of your dreams (lucid dreaming), the false thought that you have already prepared yourself breakfast (false awakening), and the sense that you are falling down into a void (hypnagogic hallucinations) are all part of various dreaming phenomena. These peculiar sensations that you experience, while meant to relax you, can actually remove you from reality.

How does our mind even get to the point of imagining things that are not real or creating events you have never witnessed before? Where does imagination take place? Where in our brain does our consciousness live? Where do we gain inspiration for dreams? Is there a subconscious mind? Where do we store memories? Why do we see fantastical dreams?

Where Do Our Thoughts Live?

To answer the questions about why we have dream phenomena, we need to first meet the voice in our head. Therefore, it is essential to scientifically understand consciousness to get a good grasp on dreams.

Having self-awareness about your thoughts, psychology, memories, surroundings, and feelings is what consciousness boils down to. Our consciousness allows us to think about what we will eat while we play the cello and allows us to review math problems in our dreams. Our consciousness never goes to sleep. Even when we are sleeping and dreaming, it is active. Consciousness can be altered with various biological environments such as sleep deprivation and comas¹. In recent studies, scientists have started exploring the electromagnetic field of our brain as a source of consciousness. Scientists hypothesize that the electrical signal is sent to nearby tissues and through commonly used technology such as an electroencephalogram or magnetoencephalography we can clearly witness the presence of these magnetic fields ultimately creating our consciousness².

Our consciousness allows us to experience life and our brain allows us to store these memories. Inside the temporal lobe of our brain lies the hippocampus, which is responsible for memory formation and storage³. Memories are formed by activating certain synaptic pathways in our brain, in other words, forming biological connections. Some memories are more strongly consolidated such as knowing your name versus remembering the name of ten new people you just met. This is due to having more experience activating the synaptic connections relating to remembering your name rather than the new synaptic connections made to remember the name of newly met people. Additionally, in neurogenesis, the formation of new neurons in the hippocampus contributes to the formation of new memories⁴.

Dreaming

Dreaming remains one of the biggest curiosities and unanswered questions in science. Many neuroscientists claim that the purpose of dreaming is to recreate memories that occur in one's life in different scenarios. Though a rudimentary explanation, there is much fact to this idea.

When we sleep, we go through five distinct stages: "wake", N1, N2, N3, and rapid eye movement (REM). A typical night's sleep cycle progresses from "wake" to N1, then repeats N1, N2, N3, N2, and REM accordingly. We spend most of our sleep in non-REM cycles; therefore, many of us wake with the thought that we did not see a dream when we did. A typical night of sleeping includes 4 to 5 cycles (about 1.5 to 2 hours per cycle). We hypothetically experience 4 to 5 dreams a night however, when we wake up we may only remember the dream we saw last if we can even remember the dream at all⁵.

REM sleep is when dreaming is said to be occurring, and this is typically viewed as a less restful stage, since our brain waves are similar to those we have while awake. With each progressive cycle, our REM becomes longer, with a difference of 10 minutes in the first REM cycle to about an hour in the next REM cycle. As the name suggests, our eyes are indeed moving rapidly and our brain is very energetic⁵. By researching patients with unhealthy brainstems and forebrains, scientists have discovered that REM is attributed to the brainstem and dreaming is attributed to the forebrain⁶.

Dream Phenomena

Our brain's creative outlet is highly expressed while dreaming, so naturally it is expected to have irregularities. Among some of the most well known dream phenomena are lucid dreaming, false awakenings, and hypnagogic hallucinations.

Lucid Dreaming

Imagine a scenario where you are at a social gathering with your friends. Think about what you would say and how you would possibly interact with different people. Or, think about what you would be wearing. Just thinking about these surface level details, your mind probably drifted off into thinking about deeper details in that setting. As you read this paragraph, you were able to consciously pick and choose how the scenario of your dream would progress. Lucid dreaming is exactly this — just in dreaming! Having conscious awareness of your dream and not being awoken, is the ability to lucid dream.

Lucid dreaming is a state of dreaming where the dreamer has an awareness of their dreams and control over their dreams. Simply put, the dreamer has some degree of consciousness during their REM cycle sleep⁷. Lucid dreams arise from non-lucid dreams which are dreams in which dreamers are aware of the happenings in the dream, but are not aware that they are indeed dreaming.

Although much is known about sleeping, there is a lot more to discover about lucid dreaming. Due to the difficult nature of studying this phenomenon, scientists have created unique methods to induce lucid dreaming in order to study it further. The most basic example is doing reality testing where participants perform reality checks to distinguish sleeping and waking states. Another technique is to manifest lucid dreaming by stating sentences such as “I will be aware of my dream when I sleep”. This technique is also used in everyday life for people who want to try inducing lucid dreaming and is referred to as mnemonic induction of lucid dreams (MILD). Often partnered with MILD is the technique of wake back to bed (WBTB) which requires dreamers to wake at certain times in the night and then fall back asleep corresponding to specific times of the dream cycle. Lastly, external stimulations such as lights or temperature can also help activate lucid dreaming.

After the dreamer has fallen asleep, researchers are able to measure the brain waves through electroencephalogram (EEG) technology by placing electrodes onto the dreamer’s head. Additionally, an electrooculogram (EOG) is used to record eye movements which are crucial during the REM sleep and a strong indicator that the dreamer is in REM⁸.

False Awakening

A false awakening is a dream phenomenon that can occur during REM. The dreamer believes they are performing actions, but in reality, they are still sleeping. For example, a dreamer may believe that they have dressed up for the day and completed breakfast but then proceed to wake up with disappointment that they haven’t actually completed these actions.

False awakenings commonly occur just before waking up in the morning, though they occasionally occur in the middle of the night, but are less graphic.

People who experience false awakenings tend to have the ability to lucid dream and have out of body experiences, all of which often occur during the steps between REM and “wake”. Someone experiencing a false awakening may be led into lucid dreaming if not awoken.

The reason behind false awakening is attributed to either disturbances or hyperarousal, both of which can be linked to insomnia. People who often experience disturbances during REM are closer to an awakened state, giving false awakenings their realistic nature. Additionally, stressors can be the cause of a false awakening, such as remembering to send an important email. False awakenings can ultimately be used to free the mind of these worries⁹. Due to much variation in what someone may visualize during a false awakening, two families of false awakenings have been assumed. Type 1 false awakenings are composed of more mundane tasks, such as brushing teeth and preparing

breakfast. Type 2 false awakenings occur when the dreamer is anticipating something bad or stressful¹⁰.

Hypnagogic Hallucinations

Lastly, inherently different from the two aforementioned types of dream phenomena, as the name suggests, hypnagogic hallucinations are a form of hallucination. These hallucinations tend to occur as a person is falling asleep and tend to involve the hallucination of multiple senses including auditory, visual, sensory, and olfactory, however most tend to be visual. All of these senses combined can often produce a physical sensation, such as falling into darkness.

The cause of hypnagogic hallucinations are not well understood, however research suggests that these hallucinations are created similar to daydreaming¹¹.

So, is my friend truly mad at me?

When we sleep, our minds enter a new headspace as it goes through the cycles of sleep with REM being the most fascinating of them due to all the phenomena that can exist during this state. Our minds store our memories that we collect throughout the day and compress them into a movie that we call our dream. However sometimes our minds go astray during dreaming and cause lucid dreaming where the dreamer chooses the trajectory of the dream. Or sometimes our dreams turn into false awakenings where dreamers believe that they have already awoken. Or our minds experience hypnagogic hallucinations where we hallucinate sensations that are actually not present.

The mind is a marvel that isn't entirely understood. The relationship between our consciousness and imagination and how it leads to dreaming and the production of intense dream phenomena remains a mystery with many surprises yet to be uncovered.

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Contributors



Mary Shahinyan | Publications Lead and Editor

Mary Shahinyan is a senior studying Molecular & Cell Biology: Neurobiology and Theater at UC Berkeley. She is passionate about neurobiology, affective science, and cognitive development. From delving into human behavior on-stage via performance to studying the neurological and anatomical basis of behavior via research, she is constantly expanding her knowledge of the human brain and behavior. Outside of academics, she enjoys singing, volunteering, acting, and



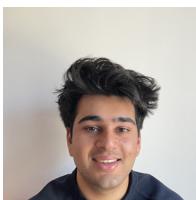
Jacob Marks | Publications Lead and Editor

Jacob Marks is a senior studying Cognitive Science and Data Science at UC Berkeley. He loves learning about the brain and interned at the National Institute of Neurological Disorders and Stroke over this past summer. While not in class, Jacob enjoys playing for the Cal Club Golf Team, rooting for his hometown Los Angeles Dodgers, and spending time with his dog. He is looking forward to his third and final semester as Publications Lead and hopes to pursue medical school, then neurology after undergrad.



Parvathy Nair | Author

Parvathy Nair is a first-year student planning to major in Cognitive Science and minor in Data Science. She is fascinated by the field of social psychology and hopes to learn more about brain structures and artificial intelligence in the future. In her free time, she enjoys practicing Bharatanatyam, painting, and journaling.



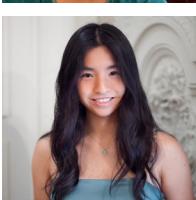
Shobhin Logani | Author

Shobhin Logani is a sophomore studying Molecular and Cell Biology. He is passionate about harnessing the power of biology to create technologies that directly improve the lives of the vulnerable. His research interests lie in the neurobiology and biochemistry of the brainstem and cerebellum, and a summer internship at an early stage biotech startup also sparked an interest in how lab research is translated into innovation through business. In his free time, he enjoys reading cool maps and baking (it's just edible chemistry, after all).



Sarita Celaya | Author

Sarita Celaya is a freshman planning to major in Molecular and Cell Biology. She plans to become a doctor to work as a researcher in neurology/neurosurgery. She is passionate about reaching underserved patient communities and aspires to aim her research at how to best reach and serve them. She is currently working as a physics research assistant in a laser lab and enjoys singing, dancing, running, and reading old thrillers (such as the Hercule Poirot mysteries).



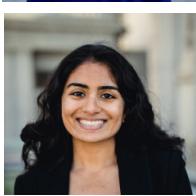
Catherine Duan | Author

Catherine Duan is a freshman studying Cognitive Science and Molecular & Cell Biology at UC Berkeley. She is passionate about cultivating a holistic understanding of the human consciousness, hoping to explore the biological and metaphysical backdrop of a future in medical school and neurology. Her interests include neurodegenerative disease progression, neuropsychopharmacology, and increasing accessibility at the intersection of healthcare and social justice. Outside of academia, she enjoys dancing, playing the piano, and listening to true crime podcasts.



Yoosung Jung | Author

Yoosung Jung is a freshman studying Public Health at UC Berkeley on the pre-medical track. He is a proactive advocate for re-innovating the diagnoses of clinical mental disorders as an empirical science through advanced neuro-technologies rather than with subjective DSM criteria. He loves to serve his community as a nationally and state licensed EMT provider since it allows him to connect with his community on a personal basis.



Oce Bohra | Author

Oce Bohra is a junior studying Molecular & Cellular Biology and Cognitive Science. She's passionate about exploring the intersection of genetics, neuroscience, and child development. In her free time, she loves to work with kids, fail at pottery, and investigate how to engineer viruses into tools for gene therapy.



Tessanya Gunatilake | Author

Tessanya Gunatilake is a freshman studying Molecular & Cellular Biology. She is fascinated by the field of neuroscience and plans to explore the progression of neurodegenerative disorders as well as public health and access to healthcare. In her free time, she enjoys playing tennis, reading, and baking.



Ananya Chawla | Author

Ananya Chawla is a Junior studying Molecular & Cellular Biology: Neurobiology and CS at the University of California, Berkeley. She is passionate about merging Neuroscience and technology to create effective medicinal instruments. After graduating, she hopes to go into the healthtech industry in order to help develop and increase access to procedures and devices that cure neurodegenerative diseases. Ananya loves the theatre, and in her personal time, you can find her singing, dancing, and performing as much as she possibly can.



Siri Tantry | Author

Siri Tantry is a Freshman studying Microbial Biology at the University of California, Berkeley. She is extremely passionate about neuroscience and wants to further her studies in neurodevelopmental diseases. In her free-time is typically volunteering with children on the autism spectrum, reading, singing, dancing, recording voice narrations for storytelling YouTube Channel, recording/writing episodes for her podcast, or emceeing for events around the Bay Area.



Meltem Su | Author

Meltem Su is an undergraduate student specializing in Molecular and Cell Biology. When she is not writing, you can find her ballroom dancing and learning new languages. Her personal experiences with lucid dreaming and false awakenings has always made her curious in understanding how dreams function and their significance. Her curiosity drove her towards writing about dreaming and presenting her findings to a larger audience who may also relate to her experiences.



Suhina Sharma | Author

Suhina Sharma is a sophomore studying Bioengineering. She is interested in the intersection of neuroscience and technology and further hopes to explore methods of early diagnosis for neurodegenerative diseases. She hopes to be involved in healthcare and aid in increasing access to medicinal devices to the public. In her free time, Suhina loves traveling, watching Bollywood movies, and listening to ASMR.



Zane Ashkar | Author

Zane Ashkar is a senior studying Molecular and Cellular Biology with an emphasis in Neurobiology. He plans on taking his interests into the medical field, and is currently interested in neurovascular surgery and inpatient vascular neurology. In his free time, you can expect to find Zane in his hometown of Oakland, CA, with family and friends, at a car show, or cooking.



Niki Parker | Author

Niki Parker is a first year student hoping to pursue cognitive science and computer science at Berkeley! Ever since childhood, she's always been interested in the brain and how its study and understanding can be applied in various directions, stemming from biological to psychological to computational applications and much more. As a member of NT@B, she deeply enjoys contributing to the ever growing fields associated with neuroscience and technology! Out-



Hilary Lu | Design

Hilary Lu is a sophomore at UC Berkeley studying Molecular and Cell Biology. While pursuing her interdisciplinary interests, she hopes to further her studies in understanding how underlying neurobiological mechanisms guide human behavior and perception. From the applications of neurotechnology in visual impairments to integrative medicine for neurodegenerative diseases, she is passionate about expanding her knowledge through research and service in her community. Outside of school, she loves to explore hiking trails, visit art galleries, and play tennis.



Megan Lui | Design

As a junior studying Molecular and Cell Biology (Neurobiology) and Cognitive Science at UC Berkeley, Megan Lui is passionate about neurodegenerative disease progression and neural mechanisms of cognitive reactivity. Through the exploration of neural pathways in mice with Alzheimer's, blood flow patterns in children with plagiocephaly, cognitive symmetry bias in children, and polysomnography studies for the elderly, she hopes to apply her passions of research through biotechnology. Outside of school, Megan enjoys playing volleyball and tennis, dancing, jamming on the guitar with her roommates, exploring new cafes, and testing the limits of friendships with unbearable puns.

Contributors



Hannah Corr | Design

Hannah Corr is a first year studying Molecular and Cell Biology and Psychology at UC Berkeley. She is interested in pursuing a career in the medical field and using her passion for neuroscience to study developmental disorders in children. In her free time, she enjoys teaching children at an Oakland elementary school, rock climbing, and making music.



Alex Buntin-Nakamura | Design

Alex Buntin-Nakamura is a Cognitive Science major completing her sophomore year at UC Berkeley. She is avidly interested in sleep and dream science, as well as neurobiology - Alex hopes to pursue research in a related field as a career, post-undergrad. Additionally, Alex is pursuing a certificate in Innovative Design from the Jacob's Institute at Cal, wherein she hopes to apply and develop her knowledge of design - whether it be logo, magazine, 3D, architectural, or UX/UI design. In her free time Alex does social and behavioral research at the Language and Cognitive Development Lab, plays competitive golf individually and with the Cal Club Golf Team, and enjoys music and film media.



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