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Complete a
Chemistry Lab'*
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LETTER FROM THE EDITOR

William Ellsworth

Readers,

I hope you enjoy the third issue of the YNCA Journal! We greatly appreciate your continued (or new) readership.

We spend approximately a third of our lives asleep, and a plethora of disorders result from misregulation of sleep; thus, it is crucial to unlock the secrets of what happens during this period. For these reasons, we chose sleep for this month's theme.

We all know that sleep is important--but what exactly does it do for us? In the neuroscience and society section, Alexander Skvortsov describes the myriad benefits of sleep.

Like any neurological process, though, sleep can be disrupted by neurological disorders. In the disease section, Christian Gonzalez profiles narcolepsy, Brendan Mitchell covers sleep apnea, Alexander Skvortsov discusses parasomnias, and Priya Vijayakumar explains REM sleep behavior disorder--all debilitating disorders of sleep.

The New Technology section details devices that have the potential to ameliorate many sleep-related disorders. Dhanya Mahesh describes the CPAP, a device that has proven effective for Sleep Apnea Patients. Neelu Paleti discusses SleepGuard, an invention used to reduce pain associated with Temporomandibular Joint Syndrome

Most medical technology could not be developed without basic research. The research section describes some of the new studies associated with sleep. Jacob Umans and Meenu Johnkutty discuss how the EEG is used in diagnostics and research, and provide new insight into the role of yawning. Jacob Umans and Shreyas Parab explain the link between sleep and memory, proving that pulling all-nighters before big exams isn't as beneficial as you may think.

As always, it is critical that we recognize all of our dedicated staff for helping us make this issue the success that it is. You can find all of their names and positions on our Contributors page. If you have any questions, comments, or suggestions for us, please feel free to contact

us at YNCA.info@gmail.com. We hope you enjoy our third issue as much as we enjoyed writing it!

Best Regards,

William Ellsworth
Editor-in-Chief, YNCA Journal

Update from the Chairmen

YNCA Journal Reader,

We are very glad to see that you are reading our journal! Whether you are a returning reader or first-time reader, we hope you enjoy this issue of the YNCA Journal. Behind the scenes, though, there is a significant amount of work going on. We would like to inform you of the changes occurring within our organization.

Leadership: During this month, the YNCA has made significant changes to the structure of its leadership. We have developed a two-tiered leadership, with the Board of Directors as an upper board and the Leadership Board as a lower board. The Board of Directors has six members, namely two presidents, two executive vice presidents, an Editor-In-Chief, and an Outreach Director. The Leadership Board currently has eight members, two of whom were recently promoted within the past month. There are plenty of opportunities open to all members, even you, so we encourage everyone interested in our organization or reading our journal to join us.

Outreach: Our Outreach committee has been very active over the past month. Our main goal has been to better spread awareness of our organization, through a variety of channels. We have established a temporary publicity committee, chaired by Kyle Ryan (Outreach Director), aiming to publicize the YNCA. Anyone who is interested in the joining the YNCA individually or starting a chapter club should contact ynca.outreach@gmail.com to begin the process of becoming a part of the YNCA.

Non-Profit Status: Over the course of the last month, the YNCA has begun to consider registration as a tax-exempt nonprofit organization under 501(c)3s. While this measure will take up a considerable amount of time, effort, and capital, we believe that registering as a tax-exempt nonprofit will allow us to be more effective in completing our mission, provide more opportunities for our chapter organizations, and to hold fundraisers for medical and scientific research.

YNCA Constitution: Just recently, we have ratified the first article of the YNCA club constitution, setting our organization on the path towards better organization and structure. We hope to ratify further articles, which would discuss topics such as committee organization, meeting policy, etc.

We here at the YNCA hope that you enjoy reading this month's journal, and that you return next month for our next edition. We also hope that all of you will consider joining the YNCA yourselves, and becoming part of our mission to share our passion for neuroscience with students all over the country.

Jacob Umans and Nicholas Chrapliwy
YNCA Presidents

Alexander Skvortsov and Janvie Naik
YNCA Executive Vice Presidents

An Overview of What Happens When We Sleep

Kyle Ryan

Sleep is vital to the human body, but can often seem like a pain or a nuisance to any six-year-old. Scientists have long thought of sleep as a mystery, not understanding why we spent 1/3 of our lives on our pillows. Nevertheless, ancient scientists understood that sleep was important: our bodies tell us this every night when we experience the strong urge to sleep. Our desire to sleep is driven by the neurotransmitter adenosine which builds up throughout the day and causes our sleepiness. Intriguingly, adenosine is closely related to adenosine triphosphate (ATP), a molecule that helps store energy to be used in various ways. When we drink coffee, the caffeine blocks adenosine receptors, which then makes it more difficult for the adenosine to signal to our body that we are tired.

At a molecular level, adenosine mediates sleepiness-- but what evolutionary purpose does sleep serve? There are many theories as to why we sleep, and many are still being formulated today. One such theory was the Inactivity Theory; this theorized that being inactive during sleep may be advantageous for prey in keeping predators from finding or noticing them. Many have disagreed with this theory, positing that while asleep we are in fact more vulnerable to attack. Other theories for sleep are the Energy Conservation Theory and restoration theories. These theories are more plausible and have scientific basis (resulting in large part from research in the last 10 years). The theories believe that during sleep, our bodies have increased periods of muscle growth, tissue repair and protein synthesis. Another important theory that has been studied is the Brain Plasticity Theory; this says that the brain develops greatly during times of sleep--especially during infancy--and helps us develop long-term memories. Many aspects of these theories have been studied, but there is still so much that we don't know about sleep. (Why Do We Sleep, Anyway 2007)

Sleep is more complex than most would imagine. Each night we go through a number of 90-110 minute sleep cycles that consist of 5 stages: Stage 1, 2, 3, 4 and REM. In order to measure these brain waves and see the varying stages, scientists use an EEG (electroencephalogram) which measure electrical activity in the brain. The first stage we enter, stage 1, is generally a very light sleep that one can be very easily woken from. This is the stage during which some people experience *hypnic myoclonia*, which is the feeling of falling. We continue then into stage 2 in which our brain waves slow down considerably and our eyes do not move. After stage 2, our body moves into deep sleep. In stage 3, our brain has decreased activity down to delta waves (which are the slowest type of waves) with spurts of fast waves. Stage 4 consists of only delta waves, and there is no muscle activity; those asleep during this stage are often hard to wake. Then we begin to switch into REM or rapid eye movement sleep. In REM, our eyes move rapidly, our heart rate increases, and we experience atonia (the paralysis of our muscles) and dreaming. As the cycles continue throughout the night we spend

much less time in deep sleep, and so when the sun begins to rise and morning comes we sleep generally only in stages 1, 2 and REM. (Brain Basics: Understanding Sleep 2014).

Sleeping is complex and is monitored by the circadian rhythm, which is our internal clock helping to monitor for one thing our sleeping cycle. Generally the day we live in – 24 hours – is what we base our clock on, with daytime and nighttime. However if the sun did not rise and fall, and there were no external time indicators, our body clock would default to 25 hour cycles. The circadian rhythm is monitored by the suprachiasmatic nucleus in the hypothalamus which regulates hormones like melatonin that can cause us to become sleepy.

Sleep is a fascinating part of our lives. It's easy to see, even with this brief introduction, that sleep is much more than many of us think.

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Basics of Neuroscience III: Neuronal Communication

Alexander Skvortsov, Jacob Umans, William Ellsworth

Hello YNCA readers, and welcome to our third Basics of Neuroscience lesson. Today, we will explain how neurons communicate with each other. Last month, we explained how each neuron acts as an independent relay stations. In this issue, we hope to discuss the molecular events underlying communication between neurons. These first three lessons will set the foundation for future lessons as we delve deeper into the fascinating world of neuroscience. Understanding how neurons are structured and how they communicate is, we believe, essential to a thorough understanding of the workings of the brain.

External Communication

You may already know that a dendrite from one cell and an axon from the other cell are joined in special areas called synapses. But what exactly goes on at the synapse? To communicate with a nearby dendrite at a synapse, axons release **neurotransmitters**, specialized signaling molecules used in the nervous system.

Neurotransmitters are stored in special axonal structures known as **synaptic vesicles**. When an action potential (see previous issue) reaches the edge of the axon (**axon terminal**), a series of molecular events cause the synaptic vesicles to release neurotransmitter into the gap between the axon and dendrite, known as the **synaptic cleft**.

The neurotransmitter then binds to specialized proteins on the receiving dendrite known as **receptors**. One broad class of receptor is the **ionotropic receptor**. When a neurotransmitter binds to an ionotropic receptor, the receptor acts as an ion channel. There are two main types of ion channels; one excitatory, the other inhibitory. The Na⁺, or sodium ion channel, when opened, causes sodium ions to flow into the receiving neuron, increasing the membrane voltage, exciting the neuron. The K⁺, or Potassium ion channel allows potassium ions to leave the neuron, lowering the membrane voltage. The reaction of the neurotransmitter with the ionotropic neuroreceptor causes

gates in the ion channels to open, thus allowing movement of ions along their concentration gradient. Depending on the neuroreceptor, this can create an EPSP or IPSP. The second broad class of neuroreceptor is the **metabotropic receptor**. This class of receptors acts indirectly; as opposed to acting as an ion channel, they activate a signaling cascade that can result in protein modification or altered transcription, among other things. Found in all types of cells, **G-Protein Coupled Receptors** are a major type of metabotropic receptor-- Upon contact with a neurotransmitter molecule, their alpha subunit dissociates from the protein, moving away from the receptor. This subunit then activates a signaling cascade of chemical reactions, which lead to the release of intracellular molecules known as second messengers. It is these second messengers that perform a function in the cell. This function can range from cellular contraction to protein synthesis to a mitotic split. This second messenger system allows cells to perform complicated functions by working together. In this manner, the nervous system performs a similar function to the intracellular genome; allowing the organization of responses to a stimulus.

Neurotransmitters and Neuroreceptors: An Overview

Probably at the very core of interneuronal communication are the chemicals most involved in the communication process: neurotransmitters. Neurotransmitters occur in two basic types: small-molecule neurotransmitters and neuropeptides. Small-molecule neurotransmitters are synthesized inside of the axon terminal, while neuropeptides are synthesized out of amino acids outside of the axon terminal, and then transported there. Both types are then held in synaptic vesicles until needed. When an action potential occurs, voltage-dependant Ca^{2+} ion channels on the presynaptic axon terminal membrane open. This allows Ca^{2+} ions to flood into the cell. These calcium ions then react with the proteins linked to the synaptic vesicles, causing the synaptic vesicle to release its contents into the synaptic cleft and react with the postsynaptic neuron.

As stated above, neurotransmitters can be either ionotropic or metabotropic. Ionotropic neurotransmitters are involved with passing on an electric signal while metabotropic neurotransmitters trigger a change in the receiving neuron. Here at the YNCA, we've compiled a list of many of the most common neurotransmitters and their functions.

One of the most prevalent amino acid neurotransmitters is glutamate. Glutamate is an excitatory neurotransmitter, and it acts on several different receptors. Perhaps the two most important of these are the AMPA and NMDA receptors, named for other molecules that activate

them. These two neurotransmitters are involved with the process of Long-Term Potentiation, the cellular mechanism by which memories are formed.

Two important inhibitory neurotransmitters in the Central Nervous System are GABA (Gamma-Aminobutyric Acid) and Glycine. GABA acts primarily within the brain, and is linked to various fear, anxiety, and anguish conditions. Some anti-anxiety drugs act as GABA agonists, helping to increase inhibitory signals in the brain. Much like anti-anxiety drugs, alcohol acts on GABA at first, which can explain how it reduces people's inhibition. At higher concentrations, though, alcohol interferes with NMDA receptor functioning, harming the formation of short term memories. Furthermore, abnormalities in GABA and Glycine signaling contribute to seizures. Glycine acts in the spinal cord, so it lacks the importance to consciousness that GABA has. Nonetheless, Glycine also works as a very important inhibitory neurotransmitter alongside GABA.

Much more complex are the neurotransmitters that react with metabotropic receptors. These neurotransmitters can serve various functions, and, in some cases (like acetylcholine), act as ionotropic receptors as well. The first neurotransmitter to be discovered, and perhaps one of the most famous, is acetylcholine, abbreviated as ACh. This neurotransmitter acts on two receptor subtypes: nicotinic (ionotropic) and muscarinic (metabotropic). Outside of the CNS, ACh acts on nicotinic cholinergic receptors at the neuromuscular junction to trigger movement. Within the central nervous system, it is responsible for many higher processes, and has been implicated in many neurological disorders including Alzheimer's disease (see our previous issue for more information on this).

Serotonin, another neurotransmitter, is highly implicated in mood. So much, in fact, that many commercial antidepressants target serotonin to have their effects. Fluoxetine, more commonly known by its trade name Prozac, acts as an SSRI (Selective Serotonin Reuptake Inhibitor). All SSRIs block reuptake channels in the synapse, allowing Serotonin to remain in the synapse longer than is biologically necessary and hence allowing it to create a stronger signal in the post-synaptic neuron. This helps to improve mood significantly.

Another neurotransmitter highly implicated in controlling mood is dopamine. Dopamine abnormalities have been implicated in many psychiatric disorders, including Schizophrenia, illustrating its significant effects on higher functions. Furthermore, dopamine is also linked to drug addiction, and through a pathway involving the ventral tegmental area, nucleus accumbens, and prefrontal cortex. In another pathway, dopamine is also involved in motor control. The substantia nigra is one of the most important regions producing dopamine in the brain, so it is no coincidence

that Parkinson's disease is caused by the loss of dopaminergic neurons in this region (more specifically the substantia nigra pars compacta).

Endorphins, the abbreviated name for endogenous opioids, are responsible for the so-called "runner's high" some people feel when exercising. They are powerful painkillers, which explains why drugs having the ability to mimic their action (i.e. morphine) have such a powerful analgesic effect. Interestingly enough, endorphins were discovered by researchers who hoped to discover how opiate drugs alter brain activity.

As some of you may have guessed by now, neurotransmitters are involved in many processes. Stress is one of these processes. The two main stress neurotransmitters, epinephrine (adrenaline), and norepinephrine (noradrenaline) are monoamine catecholamines. Both are synthesized in the adrenal glands, located on top of the kidneys. Epinephrine is released under conditions of stress, and works to energize the body by increasing blood flow to muscles, quickly using blood sugar, dilating the pupils. Epinephrine does have some involvement in the brain, but no clear role has been identified. Norepinephrine, on the other hand, functions primarily as a neurotransmitter and secondarily as a hormone. Norepinephrine is released in the brain and in the sympathetic nervous system of the peripheral nervous system. In the sympathetic nervous system, norepinephrine functions alongside epinephrine to help respond to stress. In the Central Nervous System, however, norepinephrine has wider implications involving arousal, mood, alertness, and memory.

Perhaps the most relevant neurotransmitter to this month's theme is orexin. Also known as hypocretin, orexin is an excitatory neurotransmitter, necessary for wakefulness. A neuropeptide, orexin regulates functions such as appetite and arousal. Low levels in orexin are linked to narcolepsy (as discussed in significant detail in the disease section). Also linked to sleep is the neurotransmitter adenosine (ATP without phosphate groups). Adenosine signaling indicates that the brain is using up its energy supply; thus, adenosine acts to induce sleep so the brain's supply of ATP is restored.

How (Not) To Complete a Chemistry Lab

YNCA Satire Team

Disclaimer: DO NOT FOLLOW THIS PROCEDURE. THIS IS ONLY MEANT TO BE A SATIRE ARTICLE, AND BY NO MEANS SHOULD ANYONE TRY THIS. IT IS DANGEROUS.

Today is the day you will conduct your first calorimetry lab! Calorimetry is the measurement of the heat of chemical reactions and heat capacity. How fun! This guide will explain exactly how you can be as successful in completing this lab!

I. First, gather your materials:

- 24 pack of soda (diet coke works best with mentos)
- Thin Mint Oreos
- Double Stuffed Oreos
- Regular Oreos (don't worry, you won't have to eat these)
- Birthday Cake Oreos
- Mint Oreos
- Any other oreos you can find
- Milk (to drink with oreos)
- Styrofoam Cups
- Acetone
- Aluminum Foil
- Hydrochloric Acid (HCl)

2. Alright next test the oreos. The goal is to minimize staleness. To ensure that this is controlled its best you try at least one of each of the oreo flavors. Or maybe ten if you really feel like it.

3. Crush the oreos and place them in some aluminium foil and mash them.

4. Leave the HCl in a beaker near the oreos.

5. Get a drink of water to clear your palette

→ Note: Don't drink the HCl

→ Note: If you accidentally drink the HCl:

1. Drink sodium hydroxide to neutralize the hydrochloric acid
 2. You will then become dehydrated because mixing HCl and sodium hydroxide creates salt
 3. Get a drink of water because you're probably thirsty from all that salt
- Note: You can, and most certainly should, put the HCl away after this step

6. Next place just a few drops of acetone into the aluminium foil and place it in the styrofoam cup.

→ Note: Acetone is HIGHLY HIGHLY flammable = fire = nociceptor neurons firing = ouch

7. Get a lighter and light a wooden stick and place it into the acetone-drenched oreos

8. Let the flame burn out, while measuring the change in water temperature.

➤ ***If you accidentally drop too much acetone, burn it anyway.***

Note: The styrofoam cup may become completely engulfed in flames

Note: Don't worry, it's just fire?

Note: Don't breathe in the burning plastic.

Note: Yes, styrofoam is plastic.

Note: I'm sure you could, but most likely won't die.

Note: But just keep to the procedure, ok?

And then if you do that, you will have wasted lots of time, and due to the lack of consideration for the energy of the acetone and lack of controlling acetone, you will have gotten incorrect data, so next just cry and eat the oreos?

Or burn them all with the extra acetone.

Keep a safe distance from the fire, kids.

Don't worry - I don't speak from experience.

The Necessity of Sleep

Alexander Skvortsov

Introduction

The average human adult sleeps for 8 hours a night. This amounts to one third of your lifetime. Now at first glance, one might wonder: why must we spend so much time doing nothing? While it seems as if sleep is just a waste of time, a worthless period that we must spend doing absolutely nothing, those 8 hours of rest set the bar for the next 16. Sleep is a period of rest and refreshment for your body, especially your brain. This period must be of a certain length in order to lead a healthy life. Sleep is involved in many critical processes, including memory consolidation, conservation of energy, and recovery of the body. Too much or too little can have drastic effects on your physical, mental, and emotional status.

Physical

Sleep is an essential part of your physical bodily functions, as it allows your body to heal, recover, and grow. It has been linked to various physical processes, such as muscle mitosis, cellular repair, and energy conservation through lowering of body temperature and blood pressure.

Sleep provides the body a time to recover and grow: it is extremely important for maintenance of healthy levels of the androgen Testosterone, as seen in a study by Dr. LeProult and Dr. Van Cauter regarding the effects of sleep deprivation on testosterone levels. The study found that subjects that were deprived of 3 hours of sleep each night for a week had testosterone levels 15% lower on average than subjects with normal sleep patterns (LeProult & Van Cauter, 2011). Testosterone has been implicated as one of the driving factors of muscle mass growth through muscle protein synthesis (Groggs et al., 1989). Maintaining healthy levels of sleep can therefore increase levels of muscular regeneration and growth, quickening recovery from injuries, increasing strength, and muscle mass gains.

Speaking of overall bodily health, sufficient sleep can also help prevent obesity and lose excess fat. Studies find that people across all genders and ages who are deprived of sleep can be much more likely to become obese (“Sleep and Obesity,” 2012). Not only does sleep deprivation directly increase energy consumption by giving more time to eat, as well as decrease energy expenditure, but it also changes hormone balances to make people hungrier. The two major hunger hormones, Ghrelin and Leptin work as counterbalances to maintain our hunger levels in a healthy males. However, when study participants were sleep deprived, levels of ghrelin, which increases appetite, were higher while levels of leptin, which decreases appetite, were lower. The study reports that participants who received less than 7.7 hours of sleep each night had, on average, an elevated BMI (body mass index) compared to participants who received more than 7.7 hours of sleep each night (Taheri, Lin, Austin, Young, & Mignot, 2004).

Mental

The mental and emotional risks of insufficient sleep are as serious as the physical risks. Obtaining sufficient sleep can vastly improve attention, increase motivation, and reduce stress. Sleep is also considered to be a critical part of the memory consolidation process, by which we store and remember the information that we acquire throughout the course of the day. Insufficient sleep can also lead to various emotional problems, such as depression.

Sleep deprivation has often been associated with lower levels of attention. In fact, you yourself may have felt yourself falling unable to focus and concentrate after a night of hard studying or binge-watching *your favorite shows on Netflix*. The reason behind this is the following: while we are awake, our brain releases a neurotransmitter called adenosine in increasing amounts. This neurotransmitter works together with the circadian cycle system, among others, to regulate when we are tired and when we are not. With normal sleeping patterns, adenosine buildups are dispelled while we sleep. However when we receive insufficient sleep, adenosine buildups remain through the work day, inhibiting cognitive function to a degree that, while not necessarily life-threatening, can severely inhibit your capability to focus and complete a given task quickly and efficiently. Under conditions of sleep deprivation, you may often find your mind wandering, or even find yourself struggling to stay awake. Studies have found that sleep deprivation can hinder our ability to respond to external stimuli, such as lectures, sudden road situations (Lim & Dinges, 2008).

Sleep deprivation can also hinder your motivational capabilities and willpower. Similar to its effect on concentration, sleep deprivation often causes severely reduced motivational capacities in

study participants (Alhola, P., & Polo-Kantola, P., 2007). Getting less than 6 hours of sleep a night can be very taxing on your willpower reserves. When sleep deprived, your prefrontal cortex, or the part of your brain that evaluates decisions, is hit particularly hard, making you much more vulnerable to cravings and other unhealthy temptations (Steakley, 2011).

One of the most important functions of sleep for us is the memory consolidation process. During the day, we are constantly taking in information through a process known as acquisition. During this process, outside stimuli are detected by our various sensory receptors, information is sent through the thalamus (The brain's information relay station), and to the various parts of the cerebral cortex which then process said information. This information is now encoded in the brain. During sleep, the brain shifts to a status optimal for consolidating, or solidifying said information into long term memory. Through this process, memories are strengthened and integrated into the information already located in the brain (Rasch, B., & Born, J., 2013) For more information on memory consolidation during sleep, see our article on sleep and memory. The theory that sleep provides the domain for memory consolidation is further solidified by the fact that sleep deprivation can negatively impact information retention ("Sleep, learning, and memory," n.d.).

Another very interesting function of sleep is the removal of waste from the brain. While we sleep, the brain removes toxins that build up during our waking hours with the help of the glymphatic system, named after the glial cells that perform these cleaning functions. During the day, proteins such as Beta Amyloid build up in the brain's interstitial space, or the space between neurons. If allowed to remain, these protein buildups can cause disastrous problems, such as Alzheimer's Disease, as mentioned in our article on Alzheimer's Disease in our previous edition. Here's where the glymphatic system comes in. At night, neurons shrink to a fraction of their normal size, increasing interstitial space by about 60%. This allows Cerebrospinal Fluid, or CSF, to pump through the brain, washing out the protein buildups (Jessen, Munk, Lundgaard, & Nedergaard, 2015). This works similarly to the lymphatic system, which performs a like function throughout the rest of the body parallel to the circulatory system by pumping a fluid called lymph fluid throughout the body, which then absorbs various proteins, molecules, and toxins too large to be absorbed by veins ("What is the Lymphatic system?," n.d.). However, as the brain has a tight blood-brain barrier that does not allow lymph fluid to pass through to the brain, the glymphatic system takes over this cleaning function. (Jessen, Munk, Lundgaard, & Nedergaard, 2015). By cleaning the brain of various harmful proteins such as the aforementioned Beta-Amyloid, sleep may help prevent neurological diseases such as Alzheimer's.

Insufficient sleep can also lead to various emotional problems. A heightened sense of stress and aggression is often prevalent among sleep deprived people. One can often find oneself somehow angrier, less patient, and more aggressive when receiving insufficient sleep. While the physiological causes of this correlation are unknown, sleeping more has been seen to improve a stressful situation, and to greatly lift the mood of someone suffering from sleep deprivation almost instantly.

Conclusion

Insufficient levels of sleep can lead to a variety of diseases. Insufficient sleep has been implicated in various problems ranging from the aforementioned Alzheimer's and obesity to diabetes, heart disease, immune problems, alcoholism, and even shorter life expectancy ("Sleep and disease risk," n.d.). However, too much sleep can be unhealthy as well. Although the dangers of sleeping too much have become urban myth, speculated to be linked to everything from strokes to Parkinson's, oversleeping is not as bad as made out to be. However, studies conducted on the topic find that the only health risks that long sleeping(defined as sleeping, on average, 10 or more hours per night) pose, besides being late to school or work, are obesity and depression, and even those risks are slight compared to the risks of insufficient sleep (Léger, Beck, Richard, Sauvet, & Faraut, 2014). Sleep is an extremely important part of human life. It provides us with the time period for rest and growth, and regulates hormone levels. Maintaining healthy levels of sleep can help prevent diseases such as obesity, depression, Alzheimer's, heart disease, Alcoholism, sleep problems, among many others. As a result, it is very important to get the proper amount of sleep in order to lead a healthy, happy life.

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Snorts and Slumber: An Overview of Continuous Positive Airway Pressure

Dhanya Mahesh

For many of us, the time before we fall asleep is filled with thoughts about the future, reflections upon the day before, or even wild fantasies. For those with Obstructive Sleep Apnea, the time before Morpheus's embrace may be consumed by worries about failing to breathe during the night.

Obstructive Sleep Apnea and its family of disorders constrain many from obtaining a good night's rest and cause persistent problems during the day. Those affected are unable to carry out the day's tasks with fervor as they are so ill-rested due to their condition.

More specifically, Sleep Apnea causes breaths to become shallow and accounts for pauses in breathing. These hitches cause a normally deep sleep to become a lighter one, preventing those affected from obtaining a high quality of sleep ("What is Sleep Apnea?", 2012). One of the most common conditions of this type is Obstructive Sleep Apnea (OSA) which is marked by the airway collapsing or becoming blocked during sleep (Mayo Clinic Staff, 2016).

OSA is most common among those who are overweight and have high blood pressure. It occurs when the throat muscles at the back of the neck relax and thus close the airway. The brain then senses that some sort of breathing impairment has occurred and rapidly reawakens the person which allows proper breathing to occur for a few seconds before the person goes back to sleep. This rapid awakening and breathing is marked by loud snorts, gasps, and choking sounds (Mayo Clinic Staff, 2016).

Because these awakenings may occur frequently throughout the night, OSA sufferers are unable to be alert during the day. This can affect mood and the ability to focus or concentrate. It can even be dangerous as a lack of sleep is a leading cause of car accidents ("What is Sleep Apnea", 2012).

A treatment for OSA comes in the form of Continuous Positive Airway Pressure (CPAP), a mechanism which exerts air pressure to keep one's airway open. CPAP was invented by Colin Sullivan, a physician at the Royal Prince Alfred Hospital. Sullivan devised the idea of CPAP in 1980 when a patient with a severe form of OSA refused a tracheotomy. Sullivan needed to develop a different treatment for the patient, leading to a series of experiments in which Sullivan applied increasing amounts of pressure into the patient's airway. He ultimately found that with a specific amount of continuous pressure, the airway was kept open and the patient was able to enter a deep sleep. After the success of the first experiment, Sullivan continued to experiment with CPAP until it was made commercially available in 1985 ("Past, Present, and Future of CPAP?").

CPAP machines consist of three parts: a motor, a tube, and a mask. The mask fits over the user's head with straps to keep it in place during sleep. The mask is attached to one side of the tube while the motor is attached to the other side of the tube ("What is CPAP?", 2011). Masks can come in all shapes and sizes--from nasal masks to full face masks to nasal pillow masks--with a certain

amount of adjustability for different facial structures. The motor retrieves the air from the surrounding area, filters it, and applies it with a certain amount of pressure to maintain the airway opening (Phillips, 2014). CPAP is also used to help preterm infants breathe without well developed lungs. It is safer and more effective than its alternative, ventilator therapy which includes a breathing tube being placed into the mouth and down the windpipe (“What is CPAP?”, 2011).

The main problems that are associated with CPAP machines are the side effects which include congestion, runny nose, sneezing, burping, headaches, dry throat, skin irritation, and a mild amount of noise. The mask also induces feelings of claustrophobia in many users (Phillips, 2014). Sensitive users may experience slight discomfort due to the sudden onset of air pressure. But as new and improved CPAP machines are developed, these discomforts are being addressed with additions to the machine such as humidifiers to relieve nasal problems, as well as nasal pillow masks to relieve mask discomfort (Drake, 2011).

CPAP machines are distributed and sold by many medical supply companies including Fisher and Paykel, Invacare, and Circadiance (“Shop by Manufacturer”). With the recent improvements in CPAP machine patient interface, CPAP machines are marketed and sold widely (Antonelli, 2003).

While CPAP is currently one of the best methods of treatment for Obstructive Sleep Apnea, it may be replaced by new types of technology such as pacemakers that stimulate the throat muscles and keep the throat from collapsing, as well as dental devices which push the lower jaw downward to prevent airway blockage (Rodriguez, 2015). While these new therapies seem promising, researchers suggest that CPAP machines will still be the best method of treatment for the foreseeable future (“Past, Present and Future of CPAP”).

With the availability of CPAP machines, no one should go to bed worrying about their ability to breathe throughout the night--and more importantly, everyone should get a good night's sleep.

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Biofeedback & SleepGuard: Training the Brain to Relieve Temporomandibular Joint Syndrome

Neelu Paleti

Waking up to a jolting headache, stiff jaw muscles, and excruciating face pain seems quite an unfortunate fate to endure each morning. This is the reality of the condition that currently affects nearly ten million Americans (NIDCR, 2013). Temporomandibular joint syndrome can be a painful condition for many people, yet new research to halt its progression is currently underway. Founded in 2001, SleepGuard is a form of therapy that uses biofeedback to train the brain, relieve this pain, and ultimately allow for a good night's sleep (SleepGuard™).

What is temporomandibular joint syndrome?

Temporomandibular joint syndrome, more commonly referred to as TMJ disorder, collectively refers to the conditions resulting from dysfunctional jaw joints and muscles. The temporomandibular joint, which connects the mandible to the side of the head, works with the condyles, the round ends of the lower jaw, to freely move the jaw up and down (NIDCR, 2013). However, through increased pressure from grinding teeth and clenched jaws, this efficient interaction can be inhibited. Bruxism, the habitual grinding of teeth during sleep, and other circumstances of tense jaws are often primary factors influencing TMJ pain (SleepGuard™). Following such behavior, the arisal of muscular irritation, shifted discs, and arthritis may provoke changes that leave the jaw joint vulnerable to shock and progressive damage (NIDCR, 2013).

TMJ disorder is capable of eliciting symptoms ranging from scattered pain to mild headaches. What starts as a light sensation of pain in the jaw or neck may proceed to cause jaw muscle stiffness and painful jaw movement (McAllister, 2015). As the condition progresses, simple tasks, such as chewing or yawning, may prove to be painful for the patient. Often, a dull ache across the jawline persists for many months to years, and this is finally coupled with headaches and muscle spasms (NIDCR, 2013). Soon enough, the original sleep habits that induced TMJ pain are enough to keep a patient awake into the late hours of the night.

SleepGuard and TMJ Pain Relief

Following significant research for new advancements in TMJ pain relief, SleepGuard has opened a new avenue for this pain treatment. Before the arrival of SleepGuard technology, much of TMJ pain treatment concerned symptomatic relief through the sole use of overnight mouth guards,

general anti-inflammatory drugs, and Botox (McAllister, 2015). Although surgery and jaw joint implantations still persist as options, their invasive nature may render them more useful in advanced rather than basic stages of this condition.

In contrast, SleepGuard technology focuses on treating the behavioral causes behind TMJ pain in order to relieve the condition. By preventing sleep habits, such as clenching jaws and grinding teeth, the root cause of the pain can be terminated efficiently. The main objective of SleepGuard is to improve sleep habits in order to fully combat the sleep disturbances caused by pain, making it a new portrayal for preventative treatment.

Underlying Mechanisms: Biofeedback

Unlike other traditional treatments, SleepGuard technology is built on the foundation of biofeedback to train the brain to stop a targeted behavior. Since TMJ pain and Bruxism are commonly seen as the results of clenched jaw muscles and grinding teeth, these actions can be the focus of treatment in order to relieve pain (Hudzinski et al., 1997).

Appearing as a white headband, SleepGuard includes a white strip with a controllable monitor in the middle (SleepGuard™). Conductive silicone rubber pads in the headband are placed near the temples in order to detect electrical signals (SleepGuard™). Every time a patient's jaws are clenched, the electrical signals that are coupled with the action are recognized by these rubber pads. These slight signals are then be sent to the electronic module at the middle of the headband for inspection as to the strength of the clenched jaws (SleepGuard™). Following this, different volumes of sounds will be transmitted for the patient to hear and relax the muscles.

Before beginning this therapy, this headband is to be worn in the daytime for practice. The patient is directed to respond to a repeated tone by relaxing clenched jaws each time the sound is heard (SleepGuard™). By training the brain to respond to these signals while awake, the brain is able to subconsciously respond during sleep stages, as well (SleepGuard™). As practice sessions get longer, the brain is able to adapt to this signal by responding with relaxation of clenched jaw muscles even when asleep (SleepGuard™). Ultimately, when this headband is worn over the forehead during sleep, it is able to transmit the practiced sounds to encourage the relaxation of clenched muscles. With different volumes and levels of feedback, SleepGuard is able to notify the brain of the varying degrees of clenched muscles (SleepGuard™). Continued use may discourage the clenching of jaw muscles and grinding of teeth due to prolonged training. With this change in behavior, a significant decrease in TMJ syndrome and its relevant symptoms is often charted.

While the field of biofeedback and SleepGuard technology may have its fair share of limitations in treating TMJ syndrome, it is simply another option to consider. With the historical shortcomings of mouthguards and general pain medication, biofeedback has been a popular method of relieving the pain from its original roots. Research is still necessary to ensure the safety of users and to measure the ultimate efficacy of this technology on a larger scale. In the meantime, this may be an alternative for some people waking up to the manifestations of temporomandibular joint pain each morning.

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A Compendium of Narcolepsy

Christian Gonzalez

History of the Disease

In the late 19th century, French physician Jean-Baptiste-Edouard Gelineau described the curious case of a wine merchant suffering from short yet pronounced sleep attacks. Observing the merchant's lifelong drowsiness, Gelineau proposed that the man had **neurosis** and described his findings in two scientific papers in Paris in 1880. Occurring up to 200 times per day but lasting just 1 to 5 minutes, the pattern of occurrence of the attacks resembled in many respects that of epilepsy. Gelineau believed, however, that the case was unique and named it narcolepsy. Before the coining of the term narcolepsy by Gelineau in the 1880s, German physicians had encountered similar cases in the 1870s and observed a correlation between sleep attacks and muscle weakness initiated by emotional stress. The history of narcolepsy stretches even further back, though, with earlier reports of similar situations that were described by English doctor Thomas Willis in the 17th century. Since Gelineau's findings, the scientific community has achieved a greater understanding of his observations, due to extensive sleep disorder research. Presently, neurologists have a comprehensive understanding of the pathology of narcolepsy and are working towards finding a cure (Todman, 2008).

Summary and Symptoms

Characterized by sudden and recurrent attacks of sleepiness, fatigue, and muscle weakness, narcolepsy is a chronic, debilitating neurological disease that affects approximately 1 out of every 2000 people in the US. With a total of nearly 3 million patients suffering from the condition worldwide, narcolepsy is one of the most commonly observed sleep disorders. The symptoms of narcolepsy include abnormal sleep cycles (similar to those experienced by patients with insomnia), **excessive daytime sleepiness**, loss of muscle tone, and **hypnagogic hallucinations**, accompanied by episodes of muscle weakness induced by emotional stimuli, also known as **cataplexy**, in 70% of patients ("Narcolepsy Fact Sheet," n.d.). The pathology of narcolepsy results in the brain losing the ability to maintain standard regulatory control of the sleep-wakefulness cycle, producing abnormal sleep patterns and persistent daytime sleepiness that can significantly interfere with daily activities such as driving vehicles and functioning at work. The cataplexy that most patients

experience in addition causes temporary muscle weakness while a patient maintains full consciousness. These ephemeral incidences typically torment patients for most of their lives, and can be quite devastating, especially in emotional situations that may trigger such reactions.

Etiology

While there is currently no known cause for the onset and pathology of narcolepsy, many researchers believe that there are several factors which may contribute to the development of the disease. The most common factor is low levels of the neuropeptide **hypocretin** in **cerebrospinal fluid** brought about by hypothalamic dysfunction. This molecule, also known as **orexin**, is involved in the regulation of the sleep-wakefulness cycle and proper REM sleep. When lower concentrations of hypocretin are present in a patient, the regulation of normal sleep patterns is offset, resulting in narcolepsy (“Narcolepsy: Causes,” n.d.). More specifically, researchers speculate that the immune system mistakenly attacks specific brain parts, such as the **lateral hypothalamus**, which produces hypocretin to yield the symptoms of narcolepsy. In the case of narcolepsy with cataplexy, over 90% of patients have deficient amounts of hypocretin-1 and hypocretin-2, supporting this conclusion (“Narcolepsy Research - FAQs,” n.d.). Other factors thought to trigger narcolepsy include genetic abnormalities, significant psychological stress, and infections or viruses (“Causes of narcolepsy,” n.d.).

Diagnosis

Diagnosis of narcolepsy is relatively straightforward, as the process is primarily based on symptoms and physical signs. Typically, neurologists can identify narcolepsy when a patient exhibits frequent sleep attacks and muscle weakness caused by cataplexy. Unfortunately, when these signs are less severe, diagnosis of narcolepsy can be more difficult and often requires further testing. Medical and family histories are helpful in diagnosis, and can provide information such as brain injuries and infections, as well as genetic predisposition for autoimmune diseases. The main symptoms of narcolepsy are daytime sleepiness, muscle weakness, fatigue, sleep attacks, and cataplexy. If a patient experiences sleepiness but not attacks of sleep, for example, narcolepsy may be eliminated as a possibility. Doctors usually utilize two medical tests to validate a diagnosis of narcolepsy. The first of these tests is called a **polysomnogram** (see the research methods section for more information). The other test is a **multiple sleep latency test**; this technique enables a physician to identify abnormalities in the amount of time it takes a patient to transition from being awake to falling asleep. Additional investigations may also be conducted with the use of a **hypocretin test**, in which a lumbar puncture is performed to quantify the

amount of hypocretin present in a patient's cerebrospinal fluid. If the concentration is lower than normal, the patient will likely receive a diagnosis of narcolepsy ("How Is Narcolepsy Diagnosed?," n.d.).

Molecular Underpinnings

Although narcolepsy with cataplexy is primarily the result of low hypocretin levels in cerebrospinal fluid, the genetics of an individual play a key role in their susceptibility to the disease. Having an immediate family member affected by narcolepsy increases one's chances of being afflicted by 20-40 times, compared to those without a narcoleptic relative. Still, even having a parent with narcolepsy will only place an individual at a 1-2% risk of developing the condition in their life. Genes that control the human leukocyte antigen (HLA) are speculated to be involved in the autoimmune etiology of narcolepsy, as differences in their alleles have been identified to be predictive markers for the probability of a patient being diagnosed with the disease. Particularly, the most accurate marker in determining hereditary bases of narcoleptic susceptibility is the allele HLA-DQB1*0602 in the **HLA-DQB1** gene. With over 90% of patients with narcolepsy-cataplexy carrying this allele, it is by far the most accurate genetic predictor of narcolepsy to date ("Narcolepsy Research – FAQs," n.d.). Still, while genetics is an accurate measurement of susceptibility, mutations in hypocretin-related genes associated with hypothalamic dysregulation are exceedingly rare in narcoleptic patients. Accordingly, etiology is less understood compared to other stages of narcoleptic pathology. (Taheri, Chabas, Renier, & Mignot, 2003).

Treatment

Although there is no cure for the disease, several treatments can be taken to lessen the severity of narcolepsy's effects on the life of individuals. Some of the most common types of medications include stimulants, SSRIs, tricyclic antidepressants, and sodium oxybate. Stimulants are the leading method used to abate sleep attacks in narcoleptic patients because they are very effective in keeping patients awake during the day. SSRIs are prescribed in order to ease episodes of cataplexy in narcoleptics. Through suppressing REM sleep, SSRIs such as fluoxetine (better known as Prozac) are also effective in treating narcolepsy. Other types of antidepressants besides SSRIs, such as tricyclic antidepressants, may also be used in certain cases, but have adverse side effects such as lightheadedness and dry mouth, and are therefore less attractive. Cataplexy can also be treated with **sodium oxybate**, sometimes referred to as xyrem. Unfortunately, xyrem has severe side effects including headache, nausea and sleepwalking. Other medication options include the drugs **methylphenidate** and **atomoxetine**, which both result in adverse side effects such as irregular heartbeat and psychosis. ("Narcolepsy: Treatments and drugs," n.d.).

Advocacy and Awareness

In order to truly improve the lives of patients suffering from narcolepsy, additional efforts beyond research must be taken. Becoming an advocate to help raise public awareness of the disease is an important key in making narcoleptic lives easier to live. If you would like to spread awareness of narcolepsy and get involved in volunteer programs on local, national, and international levels, please contact the following organizations listed below.

Narcolepsy Network

<http://narcolepsynetwork.org/get-involved/>

Youth Ambassador Program

<http://narcolepsynetwork.org/get-involved/youth-ambassador-program>

Wake Up Narcolepsy

<http://www.wakeupnarcolepsy.org/>

Key Terms

Neurosis- Class of mild functional mental disorders that cause distress but not psychosis

Excessive daytime sleepiness- Condition of persistent sleepiness throughout the day

Hypnagogic hallucinations- Hallucinations that occur during the transitional period between wakefulness and sleep

Cateptexy- Temporary weakness or loss of muscle function spurred on by emotional stress

Orexin/Hypocretin- Neuropeptide involved in regulation of sleep-wakefulness cycle and appetite

Cerebrospinal Fluid- Clear liquid that fills the ventricles of the brain and the vertebral column

Lateral Hypothalamus- Orexinergic area of the brain involved in regulation of body temperature, blood pressure, pain perception, and normal sleep functioning

Polysomnography- Method also known as sleep study that is conducted to diagnose disorders based on electronically measuring activities during sleep

Multiple sleep latency test- Test for measuring how long it takes patients to fall asleep

Hypocretin test- Lumbar punctures may be performed to quantify hypocretin levels in cerebrospinal fluid of patients to diagnose narcolepsy more accurately

HLA-DQB1- Gene whose DQB1*0602 allele can cause a predisposition to narcolepsy in over 90% of patients

Sodium oxybate- Anti-sleep medication; only treatment for narcolepsy approved by the World Anti-Doping Agency

Methylphenidate- Stimulant drug used to treat narcolepsy and ADHD

Atomoxetine- Cognition enhancing medication used to treat narcolepsy and ADHD

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REM Sleep Behavior Disorder: An Overview

Priya Vijayakumar

Introduction

Rapid eye movement (REM) sleep behavior disorder (RBD) was first acknowledged by the scientific community in 1985; doctors Mark Mahowald and Carlos Schenck of the University of Wisconsin published four cases of men over the age of 50 with RBD. These case studies detailed RBD's hallmark symptom of violent dream enactment during REM stages of sleep ("History of REM Sleep Behavior Disorder," n.d.). REM is a stage of sleep that alternates with slow wave sleep. Characterized by rapid eye movement, active dreaming, and wakeful-like brain activity, REM increases in frequency and duration until wakefulness. A landmark characteristic of REM sleep is atonia or full-body paralysis, except for the muscles used in respiration and eye movement. Patients with RBD fail to lapse into atonia during REM sleep and thus, experience abnormal and often dangerous mobility during dreaming states while sleeping ("Chapter 6: Sleep," n.d.).

Physiology

During normal REM sleep, atonia is achieved by inhibition of locomotor activity in the pons and neurons in the nucleus reticularis magnocellularis, a region of the medulla in the brain. In the case of RBD, these inhibitors of motor function during REM sleep are impaired, thus giving patients the ability to act out their dreams during this stage of sleep ("REM Behavior Disorder (RBD) as an Early Marker for Development of Neurodegenerative Diseases," n.d.).

Notable Features

There are two forms of RBD: idiopathic and symptomatic. Emerging with no apparent triggers, Idiopathic RBD is linked to a 12-year risk of 52.4% for developing a neurodegenerative disorder (Iranzo, Santamaria, & Tolosa, 2009). Idiopathic RBD is strongly linked to the onset of Parkinson's disease (PD) although not all PD patients experience RBD (Coeytaux, Wong, Grunstein, & Lewis, 2013). Symptomatic RBD, on the other hand, occurs alongside other neurological disorders, such as narcolepsy. Disorders that may occur prior to, during, or after the development of RBD include multiple system atrophy, prior encephalitis, progressive supranuclear palsy, Lewy body dementia, and brainstem infarction (Olson, Boeve, & Silber, 2000).

RBD is characterized by persistent movements and vocalizations during REM sleep. Dream-enacted behavior can injure both patients suffering from RBD, as well as bed partners. While reports show that it is rare for patients with RBD to leave their beds, as noted in sleepwalking, 20%

of patients are likely to face unconsciousness and head injuries as a result of RBD (Iranzo, Santamaria, & Tolosa, 2009).

Trends and Risk Factors

Affecting nearly 0.5% of the general population, RBD is nine times more likely to occur in men than in women (“REM Behavior Disorder (RBD) as an Early Marker for Development of Neurodegenerative Diseases,” n.d.). The predominance of RBD in men has been inconclusively theorized to involve a correlation between sex hormones and aggressive behavior. RBD may also occur in women; however, it is less likely to be diagnosed due to less violent dream enactment (Coeytaux, Wong, Grunstein, & Lewis, 2013; Irfan & Howell, 2016). RBD is predominant in men over the age of 50, most likely due to the predated onset of neurological disorders in such an age range (Coeytaux, Wong, Grunstein, & Lewis, 2013). Two proven risk factors of RBD include exposure to pesticides and smoking cigarettes (“Risk Factors Identified for REM Sleep Behavior Disorder,” n.d.).

Diagnosis

The first diagnostic tool for RBD is polysomnography, also known as a sleep study. Polysomnography monitors brain activity, blood oxygen levels, heart rate, respiration and muscle tone in a laboratory setting. Electromyography during sleep is also effective in RBD diagnosis; it is used during sleep studies as well (Mayo Clinic Staff, n.d.; Coeytaux, Wong, Grunstein, & Lewis, 2013). Reports of abnormal mobility during sleep also serve to indicate the presence of RBD when paired with clinical forms of diagnosis (“REM Behavior Disorder (RBD) as an Early Marker for Development of Neurodegenerative Diseases,” n.d.).

Treatment and Preventative Measures

Treatment of RBD reduces the likelihood of injuries due to dream enactment to the patient and his or her bed partner. One drug, Clonazepam, reduces RBD symptoms in 90% of patients (“History of REM Sleep Behavior Disorder,” n.d.; Iranzo, Santamaria, & Tolosa, 2009). Clonazepam acts on GABA receptors to suppress abnormal mobility during REM sleep. Melatonin is another potent drug used to treat RBD. The use of antidepressant medications are heavily discouraged to those with RBD. Other preventative measures include removing obstructive furniture or placing mattresses at lower elevations to reduce the risk of injury itself (Iranzo, Santamaria, & Tolosa, 2009).

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Parasomnias: Sleep Abnormalities

Alexander Skvortsov

Introduction

Sleep is an essential part of life. As described in our article on the necessity of sleep, sleep is extremely important to our physical, mental, and emotional wellbeing. However, sleep isn't always the tranquil resting place that it should be. We are sometimes disturbed in our sleep by conditions known as parasomnias. Parasomnias, a word derived from the greek "para", meaning as a part of, and "somnia", or sleep, are abnormal behaviors that occur during sleep. However, unlike Sleep Apnea or Narcolepsy, Parasomnias are not necessarily related to sleep disorders, are common among the general population, and are usually not a cause for medical concern. Parasomnias are divided into 2 distinct categories: REM Parasomnias, which occur during the REM stage of sleep, and NREM Parasomnias, which occur during the non REM stages of sleep. REM Parasomnias are most prevalent among adult males above 50 years of age, while NREM Parasomnias are generally more common among children. Most Parasomnias are harmless, although some may cause sleepiness during the day, or create a health hazard. Treatment of Parasomnias is often unnecessary, as most have only a minimal effect on a person's life. However, if episodes of the disorder are severe or abnormally frequent, consulting a sleep specialist is recommended (Markov, Jaffe, & Doghramji, 2006).

NREM Parasomnias

There are several types of NREM parasomnias, which may have underlying genetic causes. These mainly affect children and infants. NREM parasomnias primarily concern arousal or problems with transitions between sleep stages.

Confusional Arousals are episodes in which an individual abruptly awakens in a confused state—somewhat awake, yet with a foggy state of mind. Episodes of Confusional Arousal are often accompanied by rapid breathing, sweating, automatic behaviors, and a general sense of confusion (Markov, Jaffe, & Doghramji, 2006), and are generally much more common in infants and children. In these cases, it is recommended to let the episode run its course without interruption (Stores, 2001). Episodes of Confusional Arousal often follow being woken up.

One of the most well-known parasomnias, sleepwalking, or somnambulism, affects mostly children, and decreases in prevalence after puberty; despite this, about 25% of children with somnambulism continue to suffer from it in adulthood. Somnambulism is, in basis, an arousal disorder in which the individual unconsciously performs various motor tasks, such as walking, speaking, or engaging in some sort of activity. These episodes generally last from several minutes to an hour, and are followed by complete amnesia regarding the event. Somnambulistic episodes have been associated with insufficient sleep, an irregular sleep schedule, stress, sickness, medications, and other arousal-related sleep disorders. Somnambulism is thought to be linked with epilepsy, as 47%

of sleepwalkers experience abnormal EEG readings. Treatment is generally unnecessary unless the sleepwalking is frequent or results in harm to self or others (Kumar & Bharadwaj, 2007).

Night terrors are occurrences of waking up with a feeling of fear or terror. These occur mostly in children from 3 to 7 of age. During an episode of sleep terrors, the individual usually sits up, screams, and appears frightened. The individual often exhibits an elevated pulse, dilated pupils, and intense sweating (Markov, Jaffe, & Doghramji, 2006). The individual may run around, and is often unresponsive at attempts to approach. Afterwards, amnesia generally sets in, with the individual usually only remembering difficulty breathing or a primal sense of terror (Stores, 2001).

REM Parasomnias

These parasomnias occur during REM sleep, which is characterized by atonia (a type of paralysis). The brain is generally most active during the REM stage of sleep, so REM parasomnias are generally more visually intensive than NREM parasomnias. These include nightmares, hallucinations, and REM Sleep Behavior Disorder, which we explore in depth in our REM sleep behavior disorder article.

Perhaps the most famous and common parasomnia is the nightmare. Not to be confused with night terrors, or the state of arousal while in a terrified state, nightmares are vivid, disturbing dreams that occur during the REM stage of sleep. Throughout the course of the nightmare, the dream becomes increasingly disturbing, terrifying, and threatening. Nightmares generally concern subjects of danger such as pursuit or imminent death. To be classified as a nightmare, the dream in question has to wake the sleeper. The individual almost never experiences the unconscious sleep movements or confusion. The amnesia experienced in most arousal NREM parasomnias is also absent in nightmares. Nightmares are often triggered by a traumatic event, or PTSD, in which case the nightmare will be centered around the event (Hasler & Germain, 2009).

Alongside arousal-related NREM parasomnias and REM parasomnias, there are also several parasomnias not necessarily associated with a sleep stage. These include sleep paralysis, or episodes during which an individual is unable to move their limbs while waking up or falling asleep; sexsomnias, or sexual acts while sleeping; bruxism, or tooth grinding during sleep; and sleep eating, which is an occurrence during which an individual awakens and eats/drinks excessively. Most parasomnias are harmless, and can be safely ignored, however, if unsure, you should always consult a trained sleep specialist.

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Sleep Apnea: The Apneas that Plague the Night

Brendan Mitchell

Introduction

Sleep Apnea is a prevalent and potentially life-threatening disorder in which a person experiences intermittent cyclical cessations, or reductions, of airflow during sleep. As of 2016, 42 million Americans suffer from sleep-disordered breathing (ResMed, 2016). There are three main forms of the disorder. Obstructive sleep apnea, the most common form, occurs when throat muscles relax, causing throat tissue to collapse and consequently obstructing the flow of air to the lungs. Central sleep apnea is caused by abnormal signaling from the brain to the muscles that control breathing. Complex sleep apnea, also known as treatment-emergent central sleep apnea, is characterized by the presence of both Obstructive and Central Sleep Apnea (Mayo Clinic Staff, 2015). The prevalence of Sleep Apnea varies based on a multitude of risk factors such as excess weight, gender, hypertension, genetics, and chronic nasal congestion. Both Obstructive and Central sleep apnea can cause complications to arise such as daytime fatigue and sleepiness, however, Obstructive apnea can cause cardiovascular problems like arrhythmia, eye problems such as glaucoma, and sleep-deprived partners (Mayo Clinic Staff, 2016).

There are several treatment methods for ameliorating the symptoms of Sleep Apnea such as Continuous positive airway pressure (CPAP), in which the patient wears a mask to receive a continuous flow of compressed air, and oral appliances such as Jaw Advancing Devices (JAD) to bring the lower jaw forward to open the airway (Afzal, 2015). In some cases, Sleep Apnea can be cured by weight loss and surgery, however these are situational. Despite the various ways in which the symptoms of Sleep Apnea can be treated, they are not universally effective therefore more research is exhorted.

History

Observations of periodic breathing during sleep were first reported in the mid 1850s, and in the 1870s British Physicians described obstructive apneas as “fruitless contractions of the inspiratory and expiratory muscles against glottic obstruction with accompanying cyanosis during sleep” (Lavie, 2003). There were some reports in the 1950s of obese patients who experienced daytime sleepiness, but were labeled under “Pickwickian Syndrome”—a condition of hypoventilation caused by excessive weight (Bickelmann et al., 1956). It was not until the mid 1950s that a link between obesity and disordered breathing was fully acknowledged as the Pickwickian Syndrome was rediscovered. Surprisingly, no link to sleep disorders were considered. Indeed, physiologists studying the control of breathing never considered the extrathoracic upper airway as an integral factor in this control

system, and at that time, we knew little about its neuromuscular regulation. Furthermore, descriptions of the connection between sleep and ventilation were not reported until the comprehensive studies of Bulow in the 1960s (Bulow, 1963). Eventually, by the mid 1960s, Gastaut et al. recognized obstructive sleep apnea in obese patients as cyclical airway obstruction with frequent arousals, thereby providing the first comprehensive links between excessive weight, sleep-induced airway obstruction, sleep fragmentation, and daytime sleepiness (Dempsey et al., 2010). One early form of treatment for obstructive sleep apnea, following this key discovery, was chronic tracheostomy in the early 1970s (Lugaresi et al., 1971). Since then, research has progressed with case reports about patients with sleep-disordered breathing.

Distinctions

The most noticeable sign of Sleep Apnea is chronic snoring, which can be observed by a partner or family member. There are many shared symptoms between Obstructive and Central Sleep Apnea patients such as excessive daytime sleepiness (hypersomnia), difficulty concentrating during the day, and mood changes like depression and irritability. Because of these similarities, advanced tests such as esophageal pressure (Pes) and diaphragm electromyogram (EMGdi) are required to distinguish the two forms. Pes monitoring can be performed during polysomnography with a thin, water filled catheter connected to a transducer to determine what fraction of airway pressure is applied to overcome lung and chest wall elastance (Akoumianaki et al., 2014). EMGdi recorded from an electrode which can be used to assess neural respiratory drive, a physiological biomarker indicating exacerbated breathlessness, and diaphragm function (Luo et al., 2008). Despite the close similarities, the two forms of the disorder are fundamentally different. Uniquely, patients with Obstructive Sleep Apnea experience awakening to a sore/dry throat, high blood pressure, nighttime sweating, and decreased libido (Mayo Clinic Staff, 2016). Those with Central Sleep Apnea experience insomnia, chest pain, and shortness of breath during the night (Mayo Clinic Staff 2016).

The clearest distinction between both forms of Sleep Apnea is their causes. Obstructive Sleep Apnea occurs when muscles in the throat over relax, disrupting normal breathing. These muscles bolster structures like the soft palate, the uvula, the tonsils, and the tongue. When the airway becomes obstructed by the relaxed muscles, breathing is stopped for a short amount of time (usually 10-20 seconds) which may lower the amount of oxygen in the blood and increase the amount of carbon dioxide. The brain senses the impaired breathing and briefly awakens the person to open the airways (the awakening is too short to be remembered). The continuous disruption of breathing during the night impair the person's ability to reach the desired sleep, completion of sleep stages, and will leave the person sleepy during the day. Typically, those who suffer from Obstructive Sleep Apnea fail to notice their interrupted sleep, which leads them to believe they slept well (Mayo Clinic Staff, 2016).

As briefly mentioned above, Central Sleep Apnea is a result of the brain's failure to communicate with the motor neurons that control breathing by interacting with muscle tissue in the throat. This failure is caused by various factors that affect the brainstem--which links the CNS and controls involuntary actions such as heart rate and breathing--to control breathing. Central Sleep Apnea has a multitude of forms caused by specific factors. One notable example of a variation of Central Sleep Apnea is Cheyne-Stokes, this form is associated with congestive heart failure or stroke. This condition is characterized by an insidious progression and regression in breathing and

airflow. During the weakest breathing effort, a complete lack of airflow (Central Sleep Apnea) can occur (Mayo Clinic Staff, 2016).

There are various methods of treating and curing sleep-disordered breathing, however these solutions are, for the most part, unique to the individual. A constellation of factors contribute to the onset and the progression of Sleep Apnea, which is why there is not a universal remedy.

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A Thermal Window for Yawning in Humans: Yawning as a Brain Cooling Mechanism

Jacob Umans and Meenu Johnkutty

Yawning: whether after a long movie or a late night, it happens to all of us. When people think of yawning, the first thing that comes to mind is sleepiness. But why, from an evolutionary standpoint, would yawning when sleepy help us? One widely believed explanation for yawning is that it serves as a means to bring in more oxygen to the brain. However, at the present, no studies definitively confirm this hypothesis. A recent study sheds new light on a factor which may explain this phenomenon: yawning actually helps to cool the brain. Previous research, according to this study, identified that yawning follows an increase in temperature in the brain, so the researchers posited that the efficacy of yawning as a cooling mechanism is determined by the ambient air temperature.

To conduct their research, the researchers gathered pedestrians, aged 18 and above, to study contagious yawning, or yawning induced by another person yawning. As is standard protocol for human research, all participants had given informed consent to be participants in the study. While conducting this study, the researchers provided participants with eighteen photos of people yawning. After this, they had participants record how many times they yawned, if at all, and whether or not they felt an urge to yawn (if they did not yawn). The researchers also gathered information on the time they slept the night before, length of time they spent outside before participation in the study, and age. While they did this, the researchers recorded the temperature and humidity, and gathered all data within a two-hour window each day to ensure that people's circadian rhythm did not affect the data gathered.

After gathering all of the data, the researchers used statistical analysis to consider the effects of all of these variables. After doing so, their results strongly suggested that both the presence and frequency of yawns were related to air temperature. No other factor the researchers recorded (age, gender, or even amount of time spent asleep the previous night) had a significant effect on the probability of yawning during the study. The fact that the amount of time spent asleep is not correlated to the presence or absence of a yawn strongly

suggests that yawning is not caused by sleepiness; instead, it acts as a thermoregulatory mechanism to ensure the brain stays within its optimal temperature range.

Furthermore, the team identified factors influencing the number of yawns. The researchers identified both temperature and age as factors influencing the frequency of yawns. The effect of age on the frequency of yawns was also reported in previous research.

This study (which included measurements of lower temperatures from approximately 2-20°C) was paired with a study conducted in Arizona, which measured temperatures in a range from about 20°C to 37°C. The study conducted there concluded that the frequency of yawns increases with temperature up to 20°C, **but as the temperature rises past 20°C, the frequency of yawns begins to decrease. In more recent studies, though, researchers found that the** highest rates of yawning occurred between 28-31°C, and still decrease as the temperature approaches body temperature (37°C). These results suggest that the cooling function of yawning only works within a certain range of temperatures.

The conclusion that yawning serves as a thermoregulator satisfies a number of questions related to this often misinterpreted behavior. The benefits resulting from thermoregulation in the brain as a result of contagious yawning could serve as a coordinator of arousal in a group and would enhance overall group vigilance in a primitive setting. Though there may be numerous other facets of yawning which have yet to be understood, the basic understanding of yawning can be used in the treatment and diagnosis of patients with thermoregulatory problems. So, the next time you catch yourself yawning, don't think of it as a sign that you should take an 8 hour nap; rather, think of it as your brain staying fit and getting ready for the next big challenge.

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Research Methods: EEG

Jacob Umans and Meenu Johnkutty

History

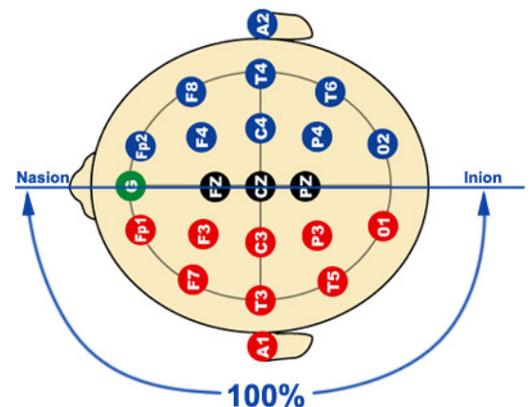
Electroencephalography, more commonly known by its abbreviation EEG, is a widely used technique to study the brain. Some of the earliest work on the electrical properties of neurons goes back to Galvani, who experimented on frog tissue to determine that electrical stimulation allowed communication within the nervous system (see article on the history of neuroscience in our first issue for more information). While researchers already had developed the EKG (electrocardiogram), newer advancements in measuring the weak electrical activity of the brain were necessary to develop the EEG.

With new developments in technology, including fine electrodes, Richard Caton was able to measure electrical activity in the brain of exposed rabbits. He observed significant differences between animals in different states, including being awake and asleep. Later, Hans Berger pioneered the use of EEG on humans through experimentation and technological research. After extensive study, research, and experimentation, Herbert H. Jasper became the first person in North America to confirm Berger's reports and conduct the first human EEG recording along with Leonard Carmichael, the chairman of the Department of Psychology at Brown University west of the Atlantic (Collura, 1993).

These developments illustrate a key theme in neuroscience: technology often precedes discovery. New technologies allow researchers to illuminate previously unknown details. And now, as technology is improving at a rapid pace, neuroscience is undergoing a massive transformation, with influential discoveries made at a rapid pace.

EEG Methods

Recording EEG--as complex as it looks in the right image--is actually quite simple! By locating landmarks on the skull and using something called the "10-20" rule, the electrodes are placed in a symmetrical fashion. After measuring the midline (the prime meridian of the brain), the brain is sectioned off into 10% and 20% sections sagittally and coronally, and lines are drawn. The intersection of these horizontal and vertical lines represents where electrodes will be placed. After sufficient skin preparation, the electrodes are



placed and the electrical activity of the brain is recorded through the use of computers, which have software specialized for the recording of EEG (Rahey, 2009). The ease at which EEG can be recorded allows it to be used in many situations where further information about the brain's electrical activity is needed. Such situations are often apparent in cases related to sleep disorders, epilepsy, and psychiatric disorders.

EEG in Sleep

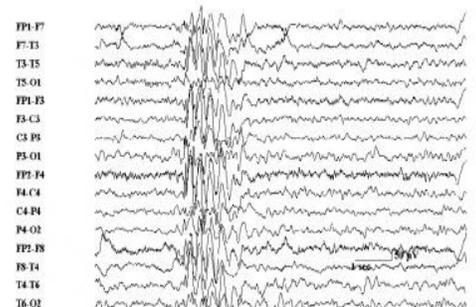
EEG studies during sleep have been essential to understanding the overall brainwave patterns occurring during sleep. As sleep is a phenomenon that induces changes throughout much of the brain, an EEG scan is a very useful tool.

EEG recordings vary during the four stages of sleep and REM (rapid eye movement) sleep. Theta waves are found during Stage 1 and Stage 2. These stages are relatively "light" because if someone were to be awoken during these stages, they wouldn't be able to recall that they were even sleeping in the first place. Though they are very similar, Stage 2 sleep is characterized by the presence of **sleep spindles** and **k complexes**. Sleep spindles are clusters of high-frequency waves, brought about by interactions between the thalamus and cortex, while k complexes are high-amplitude waves thought to be brought about by external stimuli. As the sleeper transitions into stage 3 sleep, the EEG displays delta waves. These delta waves increase in amplitude (height) but decrease in frequency in comparison to the waves recorded in the first two stages of sleep. These delta waves continue on in Stage 4. Stages 3 and 4 of sleep are collectively referred to as Slow Wave Sleep (SWS). REM sleep is categorized by the same waves found during Stage 1 and wakefulness (Purves et. al., 2001).

In sleep analysis, EEG can be conducted along with with EMG (Electromyography, which measures muscle movement) and EOG (Electrooculography, which measures eye movements) in a technique known as polysomnography. With this, researchers can gain a much more holistic view of a person's sleep. Polysomnography clearly identifies REM sleep, which is characterized by atonia (a lack of muscle movement) and, of course, rapid eye movement. Abnormalities in polysomnographs can indicate the presence of certain disorders. For example, a lack of atonia during REM sleep can indicate REM Behavioral Disorder.

EEG in Epilepsy

The use of EEG is crucial in the diagnosis and treatment of epilepsy, a neurological disorder defined by the presence of two or more unprovoked seizures. Epilepsy is a spectrum disorder and presents itself in various forms such as **absence seizures** and **tonic-clonic seizures**. The diversity found in epilepsy necessitates the use of EEG to track and pinpoint the nature of a patient's seizures and possible treatment options. Aside from diagnostics, EEGs allow



researchers to better understand the precise mechanisms behind epilepsy and how it occurs. This allows other researchers to develop more effective treatments.

The image to the right shows the brain activity in an epileptic patient. As stated before, epilepsy is a disorder which results from *unprovoked* seizures. The waves before and after the irregular pattern found towards the left end of the image are normal for any human. The disturbance found in the EEG shows abnormal interictal patterns known as **hypsarhythmia**. This electrical disturbance in the brain results in various effects in the brain and all throughout the body.

EEG in Research:

In research, the EEG scan has significant benefits. Researchers have found that many disorders, even psychiatric disorders, cause patients to have abnormal EEG signals. For example, one research team found a way to identify the differences between the EEG scans of depressed patients, insomniac patients, and healthy controls (Gillin, Duncan, Pettigrew, Frankel & Snyder). By better characterizing the EEG signals of patients suffering from certain diseases, researchers will be able to more efficiently diagnose and treat them.

Furthermore, EEG has provided new insights into movement. One study (Cochin Barthelemy, Roux, & Martineau, 1999) found that observing and executing movements activate the same brain networks. When studying function on a large scale, especially in the cerebral cortex, the EEG is incredibly useful because it can detect very rapid events.

In using an EEG scan to conduct any sort of research, researchers must take into account the strengths and weaknesses of this method. On one hand, the EEG has very high temporal resolution compared to other types of brain scans. This means that the EEG can detect changes in electrical activity far faster than other types of functional brain scans, such as fMRI or PET. On the other hand, though, the EEG has weak spatial resolution. By virtue of its setup, it is difficult to find out exactly what region in the brain causes a specific spike on an EEG readout. Furthermore, studying deep brain structures is next to impossible with the EEG. To work around these weaknesses, researchers have developed a new method: using EEG and fMRI together. This lets researchers identify more clearly when and where significant events are occurring. In research, this allows researchers to develop a clearer picture of how the brain functions. Clinically, this can be used in diagnostics and to better determine the origin of seizures of epileptic patients.

One new device, the MEG (Magnetoencephalogram) threatens to take the place of the EEG because of its incredibly high temporal resolution. The MEG uses very weak magnetic fields produced in the brain by neuronal activity. It may take time for the MEG to fully replace the EEG, though, since the MEG requires extremely low temperatures to function. At the present, this is impossible to produce without incredibly bulky equipment. In the future, though, new neuroimaging techniques may eventually replace the EEG.

Like all neuroimaging technologies, the EEG has applications both in basic research and clinical work. Whether it be paving the way for a better understanding of sleep, or creating new ways of tackling diseases such as epilepsy, EEG has and will continue to be a useful tool for neuroscientists and clinicians alike. As technology continues to advance at a rapid pace, there is hope for neuroimaging technologies which will surpass our expectations just like EEG did for its founders.

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Research Summary: Hippocampo-cortical coupling mediates memory consolidation during sleep

Jacob Umans and Shreyas Parab

Research Overview:

In this study, a team led by Dr. Maingret analyzed the linkage between sleep and memory. For decades, scientists have struggled to understand the complex relationship between sleep and memory storage. Dr. Maingret and his team's research effectively points to an exchange of electrical signals between the hippocampus and cortex (referred to as a hippocampo-cortical dialogue) which occurs during sleep that can assist in long-term memory storage and also determines its cause. This research, performed at the Center for Interdisciplinary Research in Biology at the Collège de France, could lead to several breakthroughs in the study of human memory disorders and the development of techniques to improve memory through the reproduction of such interactions during sleep.

Chasing Brain Waves

For many years, scientists have hypothesized certain types of brain oscillations, or patterns of electrical activity within the brain, are responsible for consolidating memory through a hippocampo-cortical dialogue. Specifically, researchers implicated the following types of oscillations: hippocampal sharp wave-ripples (SPW-Rs), cortical slow oscillations, delta waves, or thalamo-cortical spindles. Despite this, they lacked the evidence to prove the precise cause of the activation of the hippocampo-cortical dialogue during sleep. Before attempting to identify a causal relationship between this hippocampo-cortical dialogue and memory consolidation, the researchers first wanted to characterize the activity of both structures thought to be involved in this dialogue during sleep.

Honing in on specifically the medial prefrontal cortex (mPFC), known to receive signals directly from the hippocampus, Maingret's team recorded the brain oscillations of the mPFC and the hippocampus during slow-wave sleep. The oscillations the researchers hoped to study are known to occur during SWS; therefore, it would be optimal part of the sleep cycle to study these oscillations. The data indicates a causal relation between the SPW-Rs which occurred in the hippocampus and the cortical delta waves and spindles in the mPFC: when sharp wave-ripples were triggered in the hippocampus, delta waves followed at an average of 130ms afterwards. These delta waves, in turn, trigger hippocampal SPW-Rs. This call and response observed between the

hippocampal SPW-Rs and the cortical delta spindles showed that these oscillations are vital for dialogue between the hippocampus and the neocortex.

Maingret and his team designed an experiment to demonstrate that the dialogue between the hippocampus and the cortex causes long-term memory consolidation. The researchers split rats into two groups--one group would be left in a room to memorize the position of two objects for 20 minutes, while the other group stayed in the room for only 3 minutes. That night, the researchers measured the brain oscillations of the rats. The rats left in the room for 20 minutes showed greater signs of hippocampo-cortical coupling during slow-wave sleep, which, according to their hypothesis, indicated memory consolidation. The next day, the researchers tested the rats by placing them in the same room and determining whether or not they could identify which object the researchers moved. Unsurprisingly, the researchers identified a statistically significant difference in the performance of rats in the discrimination task discussed above in each of the two conditions. Consistent with their hypothesis, the researchers observed a direct correlation between hippocampo-cortical coupling and observed memory consolidation.

From Correlation to Causation

The researchers then repeated the experiment with a slight variation, to determine whether or not this observed correlation represented a causal relationship. In this iteration of the experiment, they only used rats that had spent three minutes inside the box. While the rats slept, the researchers artificially generated delta waves in the brains of sleeping rats whenever an SPW-R was detected. The researchers placed these rats into two conditions: coupled and delayed stimulation. Rats in the coupled stimulation condition had delta waves generated at times which mimicked the normal dialogue between the two, while in the delayed stimulation condition this did not occur. The next day, the researchers observed a statistically significant improvement in the performance of rats that received coupled stimulation, but not delayed stimulation. This difference indicates that the precisely-coordinated dialogue between the hippocampus and mPFC during sleep are linked to memory consolidation.

The Bigger Picture

Maingret and his team were able to evidence the clear relationship between long-term memory and the hippocampo-cortical dialogue caused by specific brain oscillations. This research could significantly improve understanding complex memory disorders, for which there is much to learn. The potential applications could include treatment to help individuals retain information better and assist those who suffer from learning disorders by stimulating these oscillations in the brain during sleep. This evidence is a stepping stone for a better understanding of the interconnectedness of sleep, learning, and memory.

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On Law and Individual Responsibility

Karina Bao

When discussing individual responsibility, it is impossible not mention group responsibility and group dynamics. Although current research is lending more information about the mechanisms and social impact of one individual or group upon another individual or group, human group dynamics are too complex for our current discussion. However, there is still something remaining to be discussed: one individual's mental status, something quantifiable, both clinically and chemically.

With increased awareness and discussion of mental illnesses, neuroethicists must discuss the relativity of an individual's nervous system on that individual's actions. Scientists understand the critical role the nervous system has as the director of many human behaviors and the origin of all human thoughts. It does this by feedbacking and altering signals within and through interactions with other organ systems, but nonetheless is the leading organ of the human body.

We marvel and study brains of great scientists, like Einstein's, to see the "secrets" to a brilliant brain, because we *know* that his brain is what lead to those incredible discoveries. The human brain is not only the root of discovery and thought, but also of personality, mood, and actions. So, when things go wrong, we know which organ system is responsible. But there are massive implications to this conclusion. Does this mean that no one has control over their own brains? Or is our control only the result of billions of neurons firing at trillions of synapses? If we can see changes representative of a disorder through imaging, what must we do? And what if an individual shows sign or is following a typical clinical path that can be presented to physicians, what is their responsibility to address this individual? Must this be reported, and sound like a certain dystopian novel? Or should we as a society wait until they have shown their state of mind or level of irresponsibility in order to implicate them?

The answers to these pressing questions must developed and discussed in order for our society to be more fair. It is critically important that these issues are discussed in context of the literature, so that its impact can be relayed effectively in an efficient justice system. The role of neuroethics in relation to law is including and discussing the new research and how laws can be changed to reflect our new understandings. In most criminal justice systems, there are strict liability systems in place where an individual may not have *mens rea* (guilty mind) but has committed an *actus reus* (guilty act). Right now it is acceptable to convict based only the guilty act because the act in it of itself is guilty. But can there exist an act without the mind? And if there are complications with the mind, what changes must we make in our justice system to make sure what is responsible is held responsible?

We must strive to understand the processes and progression of mental illnesses which may cause an individual to have difficulties making responsible decisions. Of course many people learn, adapt, and don't let it define who they are. And ideally, we would exert more resources in helping these individuals and find ways to prevent any of them from entering the criminal justice system in

the first place. But with the systems in place now, we have to pressure our lawmakers to know and understand the science that is the core of their jobs and ours, as members of collective society.

For further reading, visit:

<http://neuroethics.upenn.edu/wp-content/uploads/2015/06/farah-Neuroethics-The-Ethical-Legal-and-Societal-Impact-of-Neuroscience.pdf>

<http://plato.stanford.edu/entries/collective-responsibility/>

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We could not have made this issue of the YNCA possible without the following contributors

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Neuroethics: Karina Bao and Nicholas Chrapliwy

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