PHYSIOLOGICAL CHANGES AS A RESULT OF THE LSVT BIG AND LOUD PROGRAMS FOR THOSE AFFECTED BY PARKINSON'S DISEASE

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ABSTRACT
Parkinson’s disease (PD) is a progressive neurological disorder primarily affecting motor function through the degeneration of dopaminergic neurons in the substantia nigra. Additional brain changes include Lewy body accumulation, impaired thalamic and cortical activity related to tremor and rigidity, and degeneration of the limbic and prefrontal regions contributing to mood disorders. Environmental factors like pesticide and metal exposure, alongside genetic variants have been linked to increased PD risk. Neuroplastic changes induced by the Lee Silverman Voice Treatment (LSVT) programs, particularly LSVT LOUD, increase activity in the right motor cortex, auditory cortex, and dorsolateral prefrontal cortex, improving speech production. LSVT BIG promotes cortical and sensorimotor pathway adaptations through large movements leading to enhanced balance and gait. Still, the LSVT program lacks accessibility, long-term effectiveness and patient-friendliness and thus, continued efforts must be made to feasibly implement this therapy.

INTRODUCTION

Parkinson's disease (PD) is a complex, progressive neurologic disorder often characterized by symptoms including bradykinesia, asymmetric tremor, imbalance, stiffness and potentially mood disorders (American Association of Neurological Surgeons, 2024). PD is measured in five stages, which diagnose a patient's progression based on mobility and the degree to which PD interferes with their day-to-day tasks.

In the United States, about one million people live with Parkinson's disease and 90,000 cases are diagnosed per year. PD is most common in older adults, with about 96 percent of cases diagnosed after the age of 50 (Parkinson's Foundation, 2024). Although more prevalent in men, the primary risk factor for PD is still age. While a majority of PD cases are idiopathic, incidence has also been linked to environmental factors such as pesticide exposure, manganese metal and inorganic pollutants such as polychlorinated biphenyls (PCBs), which are known carcinogens. Thus, PD incidence may be higher in the Rust Belt regions of the United States due to certain industrial activities (Johns Hopkins Medicine, 2019).

A common non-medication treatment program is the Lee Silverman Voice Treatment (LSVT) Program, which includes the LSVT LOUD for vocal function and the LSVT BIG for balance and mobility. The five concepts of the program include:

i. Exclusive focus on voice (specifically vocal loudness)
ii. Stimulation of high-effort productions with multiple repetitions
iii. Intensive delivery of treatment
iv. Enhancing sensory awareness of increased vocal loudness and effort (calibration)
v. Quantification of behaviors (Fox et al, 2002)

It is also important to note that the LSVT programs are used for a variety of other neurologic disorders that fall under the category of parkinsonisms due to its effectiveness (Cleveland Clinic, 2022). While proven to be relatively successful, there are still criticisms of the LSVT programs, which include both methodological and treatment concerns.

UNDERSTANDING THE NEUROPHYSIOLOGY OF PARKINSON'S DISEASE

Parkinson's disease is rather complex, with neurophysiology varying across patients. While some cases present mild atrophy of the frontal lobe, a vast majority present loss of the substantia nigra pars compacta (SNpc) and locus coeruleus regions near the brainstem. The substantia nigra is primarily responsible for dopamine production, voluntary movement, and cognitive executive functions such as problem-solving. The locus coeruleus (LC) region is involved in attention, vigilance, and stress regulation, and its cAMP pathways are sensitive to chronic stress (Kouli, 2018).

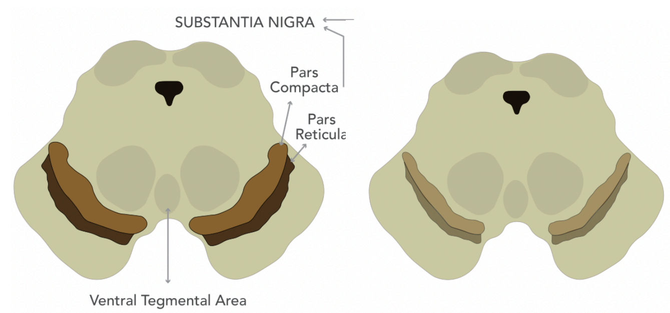

Figure 1: Two depictions of a brain cross section, the one on the left is a typical, healthy patient. while the one on the right is someone with PD. The substantia nigra in the patient with PD is noticeably thinner due to cell death and degeneration of neurons (MNC, 2019)

In many PD cases, the degeneration of dopaminergic neurons in the substantia nigra (Fig. 1), along with cell death in these regions is common. Motor symptoms typically worsen as neuron degeneration increases. This cell loss is responsible for the fundamental motor symptoms of PD and the degeneration of the nigrostriatal pathway, which is a circuit that connects the SNpc to the dorsal striatum and controls motor function (Sonne & Beato, 2022). Furthermore, the accumulation of Lewy bodies, which are abnormal protein clumps, in the SNpc can prevent the production and transmission of dopamine and may cause PD-related dementia.

Specific physical symptoms also present unique brain activity. For patients with an asymmetric tremor, EEG data in the thalamus, subthalamic nucleus, internal globus pallidus have shown cells firing at tremor frequency. Additionally, studies of cell activity reveal that different cell types are involved in tremors in different regions of the body, which explains why there is no consistent tremor across the body. While both motor and non-motor factors play into gait instability, it appears that abnormal cerebellar function and cognitive multitasking worsen cortical compensation. When looking at rigidity, it seems that the primary factor is excitability changes at the cortical and subcortical levels. More research is also unraveling the relationship between stiffness and long-latency reflexes (Chen et al., 2022).

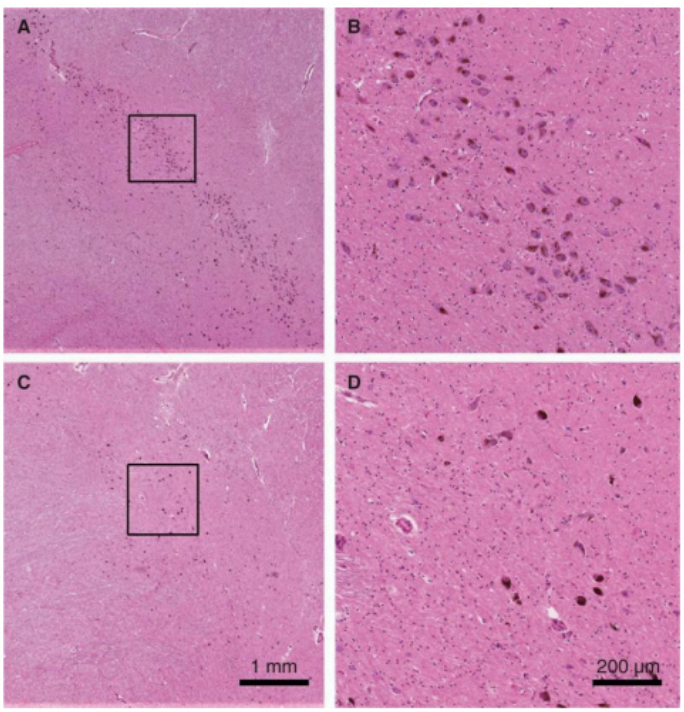

Figure 2: Imaging a coronal section of the substantia nigra pars compacta (SNpc) of a control (A-B) and PD (C-D) brain. Dark brown spots mark dopaminergic neurons. Dopaminergic cell loss is present in the PD brain (Stoker & Greenland)

Mood disorders are also affected by the neurophysiology of PD. In particular, 30-35% of PD patients report depression. This may be related to dysfunction in several regions including the prefrontal cortex, striatal-thalamic-prefrontal cortex circuits, and brainstem indoleamine systems (i.e. dopamine, serotonin and norepinephrine). For those with genetic PD, a few genes have been identified in increasing risk for PD-related depression such as variants in the SLC6A15, TPH2,84 and BDNF genes. In particular, the BDNF gene, or brain-derived neurotrophic factor gene, is primarily responsible for dopaminergic activity and its mRNA expression is significantly reduced in the substantia nigra in PD patients (Howells et al., 2000).

Anxiety disorders have been linked to the loss of function in the fear circuit and limbic cortico-striato-thalamocortical circuit. Notably, the amygdala is a part of the fear circuit and its degeneration is linked to low dopamine output. Some PD patients also experience hallucinations but the mechanism behind this is not understood (Weintraub et al., 2022).

Cell death also occurs in the LC, hypothalamus, olfactory bulb and several other regions that lead to the non-motor symptoms of PD. Because these regions are largely affected by non-dopaminergic neural networks, these symptoms are difficult to treat with typical dopamine replacement therapies (Kouli, 2018).

DIFFERENCES IN BRAIN PHYSIOLOGY WITH THE LSVT PROGRAMS

LOUD:

The LSVT LOUD program includes intensive, consistent treatment aimed to improve speech production and recalibrate the brain to recognize vocal volume more accurately (Fox et al., 2006). Exercises include speaking at varying volumes and pitches, reading aloud, and repetition of daily speaking tasks such as answering the phone, ordering food and more. According to a study hypothesizing right-shift theory post-LSVT, there were significant differences in several regions of the brain controlling speech production and understanding.

By treating PD patients with the LSVT program, researchers concluded that their hypothesis was mostly correct, as almost every region in the right hemisphere saw more activity with the program. In particular, the LSVT LOUD program directly modified the right side of the cortical motor, auditory and prefrontal areas as well as the M1-mouth region. The right auditory region is responsible for pitch discernment and timbre discrimination, which the LSVT LOUD program targets through its speech repetition.

Activating the right dorsolateral prefrontal cortex caused a type of “top-down” effect in which other connection regions such as the motor and subcortical connections (basal ganglia - thalamic inputs) improved during speech production. Additionally, the change to thalamic nuclei and the basal ganglia at large can also be attributed to modulation effects from the dorsolateral prefrontal cortex (Narayana et al., 2018).

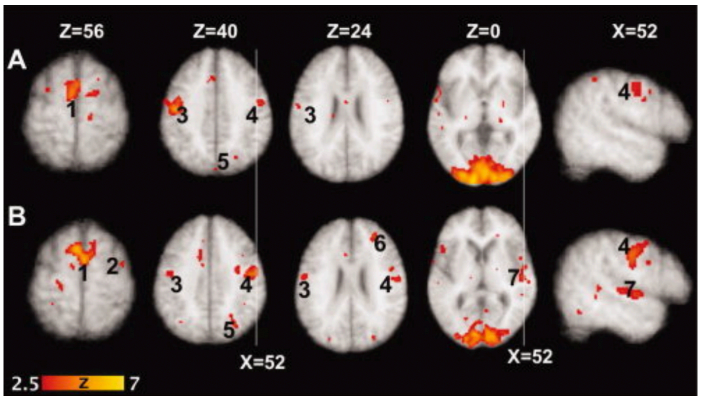

Figure 3: The top row shows brain activity before the LSVT program and the bottom shows brain activity after the program. There is significant increase in activity in the right motor cortex and right superior temporal gyrus, which contains the Wernicke''s area and the primary auditory cortex (Narayana, S. et al., 2018)

In another study, the LSVT program was utilized in children with cerebral palsy (CP). While not under the branch of parkinsonisms, this study provides valuable insights about the potential of the LSVT program to improve neurological conditions. The study discovered that the CP group treated with the LSVT LOUD program displayed increased brain activity (PSC) in the left anterior cingulate gyrus (which controls emotions, motivation and attention) as well as in the supramarginal gyrus, a region in the parietal lobe responsible for cortical speech and language. The study also found that all groups showed significant positive correlation with activity in the bilateral inferior frontal gyrus and anterior cingulate gyrus, further highlighting their role in speech and language. Interestingly, the same group also displayed decreased brain activity in the right and left precentral gyrus, which controls motor movement, and in the right cerebellum. This contrasts with previous studies regarding adult Parkinson’s patients. However, this is likely due to the different onset times of PD versus CP (Bakhtiari et al, 2017).

BIG:

The LSVT BIG program has the goal of moving “BIG.” Because PD patients will often lose their range and speed of mobility, the therapy that involves increasing the amplitude of movements will help to delay those physical symptoms. Exercises include large movements such as outstretched arms and fingers, exaggerated strides and reciting phrases while walking to engage more areas of the brain (Fig 4). These large movements have resulted in increased trunk rotation, improved balance and gait, faster limb movements and a generally better quality of life.

Figure 4: The exercises in the LSVT BIG’s program emphasize the idea of moving “BIG” (LSVT Global)

In a study conducted with multiple PD patients, researchers looked at proprioceptive function, which measures body awareness and sensation, and found that the LSVT program led to significant improvements in these individuals. While difficult to discern the exact mechanism, one proposition is that sensory information acquired throughout the program is utilized in the basal ganglia. This subsequently causes rearrangement of dopaminergic pathways, allowing for sensorimotor improvement. This change was further confirmed by PET data. Alternatively, the newly acquired sensory information may be used on a cortical level to actually overcome sensorimotor function in the basal ganglia, which is similar to the neural mechanism of those with amputation or stroke (Peterka, 2020).

There has been notably less research into the LSVT BIG program's physiological effects, primarily because of alternative treatments. Across the literature, it is well understood that exercise and repetitive movements are an effective way to manage PD symptoms. Exercise is known to increase the neuroplasticity of the brain. Neuroplasticity entails several processes, but it is simply the process by which the brain learns and solidifies new experiences and also modifies neural networks (Petzinger, G et al., 2013). Exercise acts on many aspects of neuroplasticity (i.e. releasing neurotrophic factors that improve cognitive function) which ultimately point to better outcomes. Additionally, in a recent study, there is also evidence to show that aerobic exercise also increases dopamine release in the anterior striatal region, possibly slowing the development of Parkinson's (Johansson, 2022).

CONCLUSION

It is clear that the LSVT programs have proven to be effective in rewiring the brain and improving outcomes for Parkinson's patients. Still, there remain several criticisms of the program. A major concern is the long-term use of such an intensive program. Typically the LSVT exercises are taught to patients by a certified physical or occupational therapist. The program also requires daily maintenance, so there is a need for strategies that will assist patients in implementing these exercises outside of therapy sessions. The scheduling of the program is also quite time intensive as many clinics recommend three to four sessions per week. For many working or older adults, this is simply not feasible and would require modifications by a therapist or other professional (Clark, 2013).

Additionally, the initial research done on the LSVT program was completed by its program developers. It will be beneficial to have independent studies that validate its promises. While it is beyond the scope of this specific program, it is important to acknowledge that there remains a large population that cannot access these services if they live in certain regions, cannot afford therapy or do not have consistent internet access.

With Parkinson's disease still the second leading neurodegenerative disease among adults, it is a continuing public health concern. Through more education and increased awareness of these programs, its benefits can be delivered to many more individuals. With the advent of artificial intelligence and virtual learning, the programs are potentially revolutionary for those who live alone, in rural areas or without access to therapeutic care. As Western medicine works to combine pharmaceutical treatment with non-invasive medicine, the LSVT poses great potential to be a part of treatment programs for various neurodegenerative disorders.

REFERENCES

1. American Association of Neurological Surgeons. (2024).
Parkinson's Disease. AANS.

2. Bakhtiari, R., Cummine, J., Reed, A., Fox, C. M., Chouinard, B., Cribben, I., & Boliek, C. A. (2017). Changes in brain activity following intensive voice treatment in children with cerebral palsy. Human brain mapping, 38(9), 4413–4429.

3. Burciu, R. G., & Vaillancourt, D. E. (2018). Imaging of Motor Cortex Physiology in Parkinson's Disease.
Movement Disorders, 33(11), 1688–1699.

4. Clark, H. M. (2013-2014).
Effectiveness of the Lee Silverman Voice Treatment (LSVT) in improving communication in individuals with Parkinson's disease: A critical review.
Western University.

5. Clark, J.fox, Cl, M., & Sc. (n.d.).

6. Cleveland Clinic. (2022, April 15). Parkinsonism: What It Is, Causes & Types. Cleveland Clinic.

7. Chen, R., Berardelli, A., Bhattacharya, A., Bologna, M., Chen, K. S., Fasano, A., Helmich, R. C., Hutchison, W. D., Kamble, N., Kühn, A. A., Macerollo, A., Neumann, W. J., Pal, P. K., Paparella, G., Suppa, A., & Udupa, K. (2022).
Clinical neurophysiology of Parkinson's disease and parkinsonism.
Clinical neurophysiology practice, 7, 201–227.

8. Dementia with Lewy Bodies and Parkinson Disease Dementia - Neurologic Disorders. (n.d.). Merck Manuals Professional Edition.

9. Fox, C. M., Morrison, C. E., Ramig, L. O., & Sapir, S. (2002). Current Perspectives on the Lee Silverman Voice Treatment (LSVT) for Individuals With Idiopathic Parkinson Disease. American Journal of Speech-Language Pathology, 11(2), 111–123.

10. Howells, D. W., Porritt, M. J., Wong, J. Y., Batchelor, P. E., Kalnins, R., Hughes, A. J., & Donnan, G. A. (2000). Reduced BDNF mRNA expression in the Parkinson's disease substantia nigra. Experimental neurology, 166(1), 127–135.

11. Johansson, M. E., Cameron, I. G. M., Van der Kolk, N. M., de Vries, N. M., Klimars, E., Toni, I., Bloem, B. R., & Helmich, R. C. (2022). Aerobic Exercise Alters Brain Function and Structure in Parkinson's Disease: A Randomized Controlled Trial. Annals of neurology, 91(2), 203–216.

12. Johns Hopkins Medicine. (2019). Can Environmental Toxins Cause Parkinson’s Disease? Johns Hopkins Medicine.

13. Kouli, A., Torsney, K. M., & Kuan, W.-L. (2018). Parkinson’s Disease: Etiology, Neuropathology, and Pathogenesis (T. B. Stoker & J. C. Greenland, Eds.). PubMed; Codon Publications.

14. Locus Coeruleus - an overview | ScienceDirect Topics. (n.d.).

15. Narayana, S., Fox, P. T., Zhang, W., Franklin, C., Robin, D. A., Vogel, D., & Ramig, L. O. (2010). Neural correlates of efficacy of voice therapy in Parkinson's disease identified by performance-correlation analysis. Human brain mapping, 31(2), 222–236.

16. NHS. (2022, November 3). Causes - Parkinson’s Disease. NHS; NHS.

17. Parkinson’s Disease Symptoms, Causes & Parkinsonism | MNC. (2019, November 27). Miamineurosciencecenter.com.

18. Parkinson's Foundation. (2024). Statistics. Parkinson’s Foundation.

19. Peterka, M., Odorfer, T., Schwab, M., Volkmann, J., & Zeller, D. (2020). LSVT-BIG therapy in Parkinson’s disease: physiological evidence for proprioceptive recalibration. BMC Neurology, 20(1).

20. Petzinger, G. M., Fisher, B. E., McEwen, S., Beeler, J. A., Walsh, J. P., & Jakowec, M. W. (2013). Exercise-enhanced neuroplasticity targeting motor and cognitive circuitry in Parkinson's disease. The Lancet. Neurology, 12(7), 716–726.

21. Right-Shift Theory - an overview | ScienceDirect Topics. (n.d.).

22. Saffarian, A., Amiri Shavaki, Y., Shahidi, G. A., Hadavi, S., & Jafari, Z. (2019). Lee Silverman voice treatment (LSVT) mitigates voice difficulties in mild Parkinson’s disease. Medical Journal of the Islamic Republic of Iran, 33(5), 5.

23. Sonne, J., & Beato, M. R. (2022, October 24). Neuroanatomy, Substantia Nigra. Nih.gov; StatPearls Publishing.

24. Stoker, T. B., & Greenland, J. C. (Eds.). (2018). Parkinson’s Disease: Pathogenesis and Clinical Aspects. Codon Publications.

25. Weintraub, D., Aarsland, D., Chaudhuri, K. R., Dobkin, R. D., Leentjens, A. F., Rodriguez-Violante, M., & Schrag, A. (2022). The neuropsychiatry of Parkinson's disease: advances and challenges. The Lancet. Neurology, 21(1), 89–102.

THE EMOTIONAL PROCESSING OF DREAMING AND DREAM ANALYSIS IN EXPLORING AND RESOLVING EMOTIONAL CONFLICTS

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ABSTRACT
This article explores how dreams and dream analysis contribute to emotional regulation and psychological development. Drawing from both neuroscience and psychoanalysis, the study considers how dreams may process traumatic and emotional material during REM sleep and how their interpretation in therapy can promote self-awareness and behavioral change. Two complementary frameworks are used: a connectionist model of emotional processing, which explains how dreams link emotionally similar memories to reduce psychological distress, and a cognitive-experiential model of dream analysis, which outlines a three-stage therapeutic approach—exploration, insight, and action. Together, these models highlight the interdisciplinary significance of dreaming as both a biological process and a psychotherapeutic tool.

INTRODUCTION

Often when people think about dreams, the immediate association is with a fantasy created inside the mind. But going beyond that, dreams can be defined as a kind of mental activity that takes place during sleep and mostly consists of visual content perceived as vivid and hallucinatory, often with bizarre elements or irrational narratives (Cheniaux, 2006). These experiences are also connected to how the unconscious mind continues to function during sleep. Dreams can be interpreted and analyzed in relation to what is happening in a person's life and, through an understanding of psychoanalysis, can help individuals better understand themselves and, in doing so, achieve psychical growth. Consideration can also be given to the meanings and functions of dreams and how those aspects are related to each other.

With this in mind, several questions may arise that can be better addressed with insights from psychoanalytical and neuroscientific perspectives. These questions include: What are the reasons dreams occur? Is there a physiological reason for dreaming? How can dreams provide meaningful insights into someone's psychological condition and assist in treating that condition? How can neuroscience and physiological perspectives help explain the purpose of dreams?

To address such questions, the theory that dreams can be evolutionary adaptations is considered—adaptations that involve processes tied to survival, memory, and trauma. A deeper focus is placed on the processing of traumatic or emotional material within this theory. By converging those ideas, it becomes possible to explore how dreams and their interpretation might mitigate disruptive emotions. This approach makes it possible to examine how the emotional processing function of dreaming and the analysis of dream content can help reduce distressing feelings. Two models are brought together in this context: a physiological model supported by emotion processing theory, and a cognitive-experiential model of dream analysis. The first model examines the emotional processing function of dreaming through a psychological framework of connection nets. This model proposes that dreams make it easier for memories to connect with one another, which allows traumatic memories to be dissolved and emotional discomfort to be alleviated. The second model centers on dream analysis using a cognitive-experiential approach that includes three main discussion stages: exploration, insight, and action. These are the steps a therapist and patient go through to connect dream content with the patient's emotional conflicts. Through this process, the dreamer becomes more aware of the underlying schemas contributing to those conflicts, helping ease emotional distress and support personal development through behavioral change.

INTERDISCIPLINARY FOUNDATIONS OF DREAM STUDY

To develop a more complex understanding of dreams, it is essential to consider the fields of psychoanalytical study, dream analysis, and neuroscience. Psychoanalysis explores how the unconscious and conscious mind contribute to diagnosing and treating mental conditions, often using dreams as a pathway to explore unconscious material. Dream analysis involves a collaborative process between patient and therapist in which dream content is explored to uncover issues connected to emotional struggles. The goal of this process is to generate insight, which may lead to psychological change.

A complete understanding of dreams and their functions must also include biological and neurological activity as examined through neuroscience. Neuroscience explores the chemical and biological functions of the brain at a molecular level, including the nervous system, brain, spinal cord, and peripheral system. Tools such as EEGs can reveal electrophysiological properties of dreaming, while neural net models may compare brain activity during wakefulness and dreaming. These tools support theories such as emotional processing during dreaming, where emotional experiences, particularly traumatic ones, are diffused through memory reconsolidation during sleep.

To address how dreams and their interpretation can alleviate emotional stress, two models are considered: the "connectionist nets" model and a cognitive-experiential model of dream analysis. The connectionist model demonstrates how neural links are distributed widely, facilitating easier connection among memories. This idea is grounded in the theory of emotional processing. Meanwhile, the cognitive-experiential model describes dream interpretation as a process involving exploration, insight, and action. This model views dreams as personally meaningful; by understanding the schemas within dream images through elaboration and association, psychological development may occur.

NEUROLOGICAL BASIS OF DREAMING

Before diving into the emotional processing aspect of dreams, their interpretations, and how they collaborate in solving emotional conflicts, it is important to understand the processes involved in the formation of dreams in the brain, when they occur, and other possible cognitive and emotional functions they might serve. It has also been scientifically observed that dreams are constructed through internally generated sensory, cognitive, and emotional experiences, primarily during REM sleep (Desseilles et al., 2011). REM sleep is the condition in which rapid eye movements occur and cortical blood flow—that is, the delivery of blood to the outermost layer of the brain—is very intense (Cheniaux, 2006). Cerebral blood flow is essential for eliminating neural waste and for delivering nutrients and oxygen to the brain (Tsai et al., 2021). It has also been demonstrated through positron-emission tomographic studies (PET scans) that during REM sleep, the associative visual cortex and the limbic and paralimbic regions are active, while the primary visual cortex and the prefrontal cortex are deactivated.

In the context of dream analysis by psychiatrists, these findings have been used to explain specific characteristics of dream content. Psychiatrists can connect the richness of visual imagery to the activated associative visual cortex and the deactivated primary visual cortex; the strong emotional response to the activation of the limbic and paralimbic regions; and, finally, the bizarreness, incoherence, loss of criticism, and forgetfulness to the deactivation of the prefrontal cortex. In addition to these neurophysiological aspects of dreaming, other possible purposes of dreaming beyond emotional conflict resolution have also been investigated. These include mental activity, threat simulation, wish fulfillment, and a variety of other proposed functions such as the discharge of psychical energy, problem solving (both intellectual and emotional), creativity, self-knowledge, integration of the mind, adaptation, learning, stress neutralization, and communication.

THE EMOTIONAL PROCESSING FUNCTION OF DREAMING

After understanding how dreams occur in the brain, when they happen, and why, attention can now shift to the first half of this study: the emotional processing function of dreaming in resolving emotional conflicts, viewed through the aforementioned psychological connectionist nets model. According to psychiatrist, psychoanalyst, and associate professor Elie Cheniaux, this function relates to an elaboration process described in a model of computational neuroscience, which recognizes dreams as relevant to processing trauma and psychological conflict.

First, let’s consider the dream’s function of processing emotions and how this corresponds to the idea that it can alleviate stress. Emotional aspects are encoded as implicit memories (Cheniaux, 2006). Implicit memory implies that remembering can be an unconscious process (Martin & Li, 2016). Those implicit memories are likely consolidated during REM sleep, which is also when dreams occur the most. Scholars have agreed about there being a relationship between emotions that are felt in waking moments and the content that appears in our dreams (Cheniaux, 2006). The same study claims that, since REM sleep has been shown to collaborate in the processing of emotional memories, it is safe to conclude that emotions have a big impact when dreams are formed. This also allows us to understand that those mental activities have a therapeutic role as they process traumatic experiences and conflicts, just as sessions of therapy would.

To further comprehend this elaboration process, a model of computational neuroscience can be used. This process relies on the frequent neural network connections that occur during dreaming, which associate traumatic memories with other memories that have the same affective connotation, making those traumas less distressing and powerful. In other words, researchers argue that these neural connections occur more easily during dreaming than during wakefulness (Cheniaux, 2006). This would, therefore, allow more connections and more elaborations to take place. Additionally, these neural networks are organized, as previously described, according to the emotions being experienced, which means that mental connections are grouped together based on affective similarity and tone. As a result, during sleep, more recent events are connected to past and more remote ones that share emotional relatedness before being stored as memories in the brain. This suggests that unpleasant situations—whether traumatic experiences or less disturbing, stressful memories—can be more easily associated and connected with other encoded memories while dreaming. These new connections would lead to fewer disturbing and unpleasant emotions being tied to those powerful and traumatic memories.

Another author uses the same model of computational neuroscience to argue that dreams serve the function of processing and mitigating negative emotions. Clinical psychologist Matthew Merced supports the idea that dreams have the ability to dilute emotional intensity, granting them a partial therapeutic role. He explains that dreams continuously add new memories to older, traumatic ones, resulting in the diffusion of the emotional intensity of those powerful memories. In comparison to the previously discussed scholar, Cheniaux, Merced explores in greater depth the ideas of one of the more specialized psychiatrists in the field of dream function. This allows for a clearer explanation of how the connection between remote and newer memories might occur. Merced examines Ernest Hartmann's studies on dreams, including his methodology, analyses, and conclusions.

Hartmann, a renowned psychiatrist, psychoanalyst, and sleep researcher, conducted a series of dream reports over several months with individuals who had experienced traumatic events. A recurring pattern was observed in which natural disasters appeared as common dream content. Over time, Hartmann recorded that the anguish linked to these nightmares diminished as they became associated with older, previously encoded material. Merced argues that Hartmann noticed a gradual decrease in the negative emotions connected to the traumatic experience and that the trauma began to have a smaller presence in the patient's waking life and dreams (Merced, 2012). Merced places more emphasis on the idea that these connections occurred between traumatic and older memories, rather than with newly encoded content. This difference could be due to Cheniaux referencing earlier interpretations of Hartmann's work. In addition, Merced includes the idea that the trauma's role becomes diminished in both dreams and waking life, offering further insight into how emotional resolution and integration may occur through the dreaming process.

Another author who supports the emotional processing function of dreams is Matthew Walker, a professor of psychology and neuroscience at the University of California, Berkeley, and director of the university's Center for Human Sleep Science. Walker states that dreaming can help reduce negative emotions and traumatic experiences because, during REM sleep, the brain is free from an anxiety-triggering molecule and the emotional and memory-related regions of the brain are activated. He also claims that while dreaming, the brain becomes more creatively active, mixing and combining memories in abstract and novel ways that help solve problems that may be unsolvable in waking life (Walker, 2017). This idea of connecting old and new memories to solve unresolved problems is closely related to the arguments presented by Cheniaux and Merced within the computational neuroscience framework. Creativity, in this context, would enable the brain to combine memories in ways that help resolve internal emotional "puzzles."

This aligns with the emotional processing theory, where creativity plays a role in mitigating strong and distressing emotions. These insights also introduce new elements to the theory of dream function—such as the brain's freedom from anxiety-inducing molecules during REM sleep and the active participation of brain regions involved in memory and emotion. In addition, the creative capacity of the brain to join and reconfigure memories and emotional material adds a significant perspective not emphasized by the previous two scholars who contributed to the emotional processing theory of dreams.

COGNITIVE-EXPERIMENTAL DREAM ANALYSIS AND ITS THERAPEUTIC POTENTIAL

The second half of this study deals with the argument that working with dreams within the field of psychotherapy—specifically, through a cognitive-experiential model of dream analysis—can provide positive psychological development for the patient. In support of this, Merced also argues that dreams are events full of meaning, and that dream work, which assumes dreams are part of a cognitive process and have personal relevance, is a well-founded and therapeutically relevant activity. He claims that for dream work to be done, the psychologist must clarify and comprehend the images in the dreams through the dreamer's elaborations, thoughts, feelings, and associations. By understanding the visual content of the dream, both the patient and the psychoanalyst are better able to visualize previously hidden schemas, and from that, the possibility of positive change can emerge.

Merced adds that the simple act of talking about dreams also has therapeutic effects (Merced, 2012). He defends a three-step model developed by Clara E. Hill, a professor of psychology at the University of Maryland. The first step is exploration, the second is insight, and the third is action. Merced notes that these three stages mirror the structure of therapy more broadly. First, the therapist and the patient explore possible issues that brought the patient into treatment. Then, the therapist tries to make sense of the patient's underlying personal schemas and how they relate to emotional conflicts. Finally, both attempt to initiate change in the client's life.

Merced states that during the exploration stage of dream analysis, there is often an initial sense of not knowing exactly what the images in the dream mean. However, with open-ended questions the therapist can ask about the dream's visual content, the client can more easily connect the images to personal life experiences through free associations. With the help of the therapist, it becomes possible to arrive at current conflicts or situations in the patient's life that may relate to the dream's content. In the second stage, both participants work to combine the descriptions and associations in order to gather possible interpretations of the dream. In this stage, as in the first, the therapist asks the patient for initial impressions, and they both try to form interpretations that fit the individual's past and current history. The therapist should avoid relying on fixed or stereotypical meanings; interpretations should be personal. Again, the bizarre and figurative aspects of dreams are connected to the specific areas of the brain that are either activated or deactivated during REM sleep.

The third and final stage allows the patient to decide whether to change certain behaviors or reconsider perspectives after gaining insight into the dream's meaning. This may lead to psychological development, considering that behaviors and thoughts can change over time. If the client decides to make a change, the therapist can offer strategies the patient may follow. However, it is important to recognize that behavioral change is not required. Some patients may be more resistant to change than others; nevertheless, all responses are valid in the context of dream work. Simply working with the dream presented by the individual can already increase the dreamer's awareness of psychological issues, schemas, and personality dynamics. The therapist can also help evaluate the benefits and drawbacks of specific choices and offer encouragement (Merced, 2012). In this way, the patient is given tools to achieve personal development and ease emotional conflict.

Other authors agree with the view that dream work can contribute to psychological development and hold therapeutic value. In one article, Hill's cognitive-experiential model of dream analysis, along with other dream work frameworks, is used to support the argument that dream interpretation can promote cognitive restructuring and emotional benefit. These authors emphasize that the model focuses on building a shared understanding between therapist and patient based on the patient's associations and descriptions of the dream, with the aim of identifying personally meaningful insights and increasing self-awareness (Scarpelli, 2022). These scholars also note that, while the effectiveness of dream work in psychotherapy has not yet been definitively established, many models—such as Hill's—show consistency with experimental findings in the study of dreams. This is partly because they avoid stereotypical interpretations and instead emphasize the individual's cognitive, emotional, and behavioral experience (Scarpelli, 2022).

CONCLUSION

In conclusion, dreams and dream analysis can have powerful consequences in cognitive, emotional, and behavioral functions. Dreams have an emotional processing ability that allows traumatic events or simply stressful emotions to have their intensity decreased so that the brain can work less disruptively. For this, the model of computational neuroscience helps demonstrate how, during dreaming, new connections are more easily made, allowing negative emotions to become less intense, less worrying, and less influential in the mind. In addition, the cognitive experimental model of dream analysis also shows potential benefits for psychological growth and therapeutic value by connecting the visual aspects of dreams with the dreamer's associations and descriptions in order to identify underlying conflicts. Through this process, psychological development can be achieved by fostering greater awareness of the self. In this way, both dreams and dream analysis can lead to personal growth, particularly through the cognitive development that results from these processes.

REFERENCES

1. Cheniaux, Elie. "Os Sonhos: Integrando as Visões Psicanalítica E Neurocientífica." *Revista De Psiquiatria Do Rio Grande Do Sul*, vol. 28, no. 2, 2006. DOI: 10.1590/s0101-81082006000200009.

2. Desseilles, Martin, et al. "Cognitive and Emotional Processes during Dreaming: A Neuroimaging View." *Consciousness and Cognition*, vol. 20, no. 4, 2011. DOI: 10.1016/j.concog.2010.10.005.

3. Martin, Jane, and Clara Li. "Normal Cognitive Aging." *Brocklehursts Textbook of Geriatric Medicine and Gerontology*, Section D, Elsevier, 2016. DOI: 10.4324/9780203882504-7.

4. Merced, Matthew. "Dreaming: Physiological Sources, Biological Functions, Psychological Implications." *The Journal of Mind and Behavior*, vol. 33, no. 3/4, 2012.

5. Scarpelli, Serena, et al. "What about Dreams? State of the Art and Open Questions." *Journal of Sleep Research*, vol. 31, no. 4, 2022. DOI: 10.1111/jsr.13609.

6. Tsai, Chia-Jung, et al. "Cerebral Capillary Blood Flow Upsurge during REM Sleep Is Mediated by A2A Receptors." *Cell Reports*, vol. 36, no. 7, 2021. DOI: 10.1016/j.celrep.2021.109558.

7. Walker, Matthew. "Why Your Brain Needs to Dream." *Greater Good*, 24 Oct. 2017.