

Mind-Brain Dynamics in the Pathophysiology of Psychiatric Disorders

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Abstract: Short of a comprehensive understanding of psychiatric disorders, two parallel but phenomenologically different schools of thinking continue to guide treatment: the psychological school and the biological school. Yet both of these schools of thinking have major shortcomings. The psychological school does not explain how psychopathology is related to neuropathology, and the biological school does not explain how neuropathology is related to psychopathology. However, a new hypothesis contends that the mind and the brain influence each other. “Mind” in this sense does not refer to a psychic manifestation of complex neurological activity but rather an independent entity that has the ability to think, emote, and access memory either in connection with neurological activity or independent of it. An important consequence of this is that mental stress could hyperactivate the brain, and hyperactivity in the brain could cause mental stress, thus creating a vicious cycle of mutual overstimulation between the mind and the brain. According to the multi-circuit neuronal hyperexcitability (MCNH) hypothesis of psychiatric disorders, psychiatric symptoms develop when normal thoughts and emotions become abnormally amplified, prolonged, or distorted by pathological hyperactivity in the related circuits in the brain. Although this pathological hyperactivity can sometimes be initiated by the brain alone, it is almost always initiated by a superimposition of mental and emotional stress upon an underlying hyperexcitability of the neurological system. This article will discuss how the interactions between the mind and the brain influence: 1) the development of psychiatric symptoms; 2) the nature of the psychiatric symptoms; and 3) the severity of the psychiatric symptoms. It will also discuss the possible means by which the cognitive-emotional system interacts with the neurological system and speculate about where, based on brain architecture and detailed clinical observations, that interaction occurs. Acquiring a better understanding of mind-brain dynamics could help solve the mystery of mental illness and allow clinicians to treat mental and neuropsychiatric disorders with greater precision and with greater success.

Keywords: Mind-Brain Dynamics, Pathophysiology of Psychiatric Disorders, Neuronal Hyperexcitability, Diathesis-Stress Model of Mental Illness

1. Introduction

After more than a century of rigorous scientific study and philosophical debate, the pathophysiology of psychiatric disorders remains elusive. Short of a comprehensive explanation for these highly common and perplexing disorders, two parallel but phenomenologically different schools of thinking continue to guide treatment: the psychological school and the biological school. The psychological school believes that psychopathology arises in the mind and, therefore, can be treated by correcting the psychological abnormalities that drive psychiatric symptoms. In contrast, the biological school believes that

psychopathology arises in the brain and, therefore, can be treated by correcting the neurological abnormalities that drive psychiatric symptoms. Yet both of these schools of thinking have major shortcomings. The psychological school does not explain how psychopathology is related to neuropathology, and the biological school does not explain how neuropathology is related to psychopathology.

However, a new hypothesis contends that the mind and the brain influence each other. “Mind” in this sense does not refer to a psychic manifestation of complex neurological activity but rather an independent entity that has the ability to think, emote, and access memory either in connection with neurological activity or independent of it. An important

consequence of this is that mental stress could hyperactivate the brain, and hyperactivity in the brain could cause mental stress, thus creating a vicious cycle of mutual overstimulation between the mind and the brain [1]. This conceptualization integrates, for the first time, the psychological and the biological schools of thinking and lays the foundation for a comprehensive understanding of the means by which psychiatric symptoms develop and perpetuate in the lives of those who are plagued by them.

According to the multi-circuit neuronal hyperexcitability (MCNH) hypothesis of psychiatric disorders, psychiatric symptoms develop when normal thoughts and emotions become abnormally amplified, prolonged, or distorted by pathological hyperactivity in the related circuits in the brain [1]. Although this pathological hyperactivity can sometimes be initiated by the brain alone, it is almost always initiated by a superimposition of mental and emotional stress upon an underlying hyperexcitability of the neurological system. The recognition of this is highly relevant to the treatment and prevention of mental illness because it explains, for the first time, how cognitive-emotional stress translates into neurological dysfunction and vice-versa.

This article will take a detailed look at the cognitive-emotional system in distinction to the neurological system and discuss how the interactions between the mind and the brain influence: 1) the development of psychiatric symptoms; 2) the nature of the psychiatric symptoms; and 3) the severity of the psychiatric symptoms. It will also discuss the possible means by which the cognitive-emotional system interacts with the neurological system and speculate about where, based on brain architecture and detailed clinical observations, that interaction occurs. Acquiring a better understanding of mind-brain dynamics could give mental healthcare practitioners a better understanding of how to treat and prevent mental, emotional, and behavioral disorders.

2. The Mind-Brain Dialogue

Unlike other functions of the body, mental and emotional functions are invisible and intangible. This creates a conundrum for the medical field, which, being scientifically-based, relies on the ability to see and measure the things that it attempts to treat. However, the recent development of more sophisticated neurostimulatory and neuromonitoring techniques has opened the door to a better understanding of the relationship between mental functions and neurological functions. The first of these advances came at the turn of the 20th century, when Sir Victor Horsley made the first use of intraoperative electrical brain stimulation as a means of brain mapping [2]. This was followed by the seminal work of Wilder Penfield, who found that stimulating the brain in specific places caused his patients to experience specific thoughts and emotions [3]. More recently, it was similarly demonstrated that the behavior of laboratory animals could be influenced by stimulating or inhibiting specific neurons [4, 5]. This helped clarify the means by which the touch of Penfield's electrical probe was stimulating related thoughts

and emotions. Yet these experiments only demonstrated that brain function affects mental function. However, the latest studies have found that the reverse is also true: that mental function affects brain function. In a set of elegant experiments, Cerf et al. [6] found that willful thoughts and emotions readily stimulated specific neurons when subjects were asked to perform specific mental tasks. This observation, taken together with the previously observed effects of brain stimulation on thoughts, emotions, and behavior, provides compelling evidence that the mind and the brain influence each other. This bidirectional influence could help explain why psychiatric symptoms tend to develop under the influence of mental and emotional stress. The mind, when under stress, could overstimulate specific neurons and circuits, thus causing them to become hyperactive. The hyperactive neurons and circuits, in turn, could reactivate the associated thoughts and emotions. This would result in a vicious cycle of mutual overstimulation between the mind and the brain that could cause the associated thoughts and emotions to become abnormally intense and abnormally persistent. The relevance of this to psychopathology is that an abnormal increase in the intensity and persistence of specific thoughts and emotions is precisely what distinguishes psychiatric symptoms from normal thoughts and emotions.

3. The Biological Vulnerability Trait

However, a vicious cycle of mutual overstimulation between the mind and the brain would not explain why some persons are more vulnerable than others to developing psychiatric symptoms. As suggested by the long-held diathesis-stress model, susceptible individuals must harbor some vulnerability trait that acts as an accelerant when cognitive-emotional stress begins to hyperactivate the neurological system. Strikingly, the top candidate genes for the major psychiatric disorders—disorders that together express all of the most common psychiatric symptoms—involve ionchannelopathies [7-10]. Specifically, the protein products of the candidate genes fail to adequately regulate the firing of neurons, thus increasing the excitability of the neurological system. What makes this unlikely connection so relevant is that a hyperexcitability of the neurological system would have a natural tendency to amplify the mind-brain dynamic, and this effect could, particularly under high levels of stress, cause an intolerable elevation in the intensity and persistence of the associated thoughts and emotions.

That is not to say that persons with normoexcitable neurons would be completely immune to developing psychiatric symptoms. However, their stress levels would have to be high enough for long enough to induce enough kindling to precipitate symptoms even in the absence of an inherent hyperexcitability of the neurological system. First observed by Graham Goddard in his experiments on rats [11], kindling describes the natural tendency for neurons to become increasingly responsive when stimulated repeatedly. This adaptive process, which under normal physiological conditions is more aptly described as “primed burst

potentiation” [12], is the MCNH explanation for why stress can drive the development of psychiatric symptoms even in persons with normoexcitable neurons. In essence, kindling itself increases the excitability of the symptom-related neurons and circuits. Kindling also helps explain why the onset of psychiatric symptoms tends to be delayed relative to the onset of a triggering stressor.

4. Distinguishing the Cognitive-Emotional System from the Neurological System

Although cognitive-emotional stress has long-been recognized to be the most ubiquitous precipitant of psychiatric symptomatology, the precise mechanism by which stress precipitates psychiatric symptoms remains unclear. Central to understanding the stress response is the need to better define “cognitive-emotional stress.” Based on the most widely accepted definition, cognitive-emotional stress is a real or perceived demand on the cognitive-emotional system to respond to a mental or emotional stressor. This is different than physiological stress, which is a demand on the physical body to respond to a physical or biological stressor, such as an injury, an infection, or a toxin. That raises the question: what exactly is the cognitive-emotional system? The most widely held conceptualization is that thoughts and emotions are products of complex brain function. But how can atoms and molecules—the basic building blocks of the brain—create the subjective experience of awareness and mediate psychological phenomena such as thoughts, emotions, and intrapsychic conflicts? Obviously, it is not the atoms and molecules that experience these phenomena but the mind. This implies that the cognitive-emotional system is something other than the neurological system.

Another observation that suggests that the cognitive-emotional system is something other than the neurological system is the preservation of consciousness even in the absence of large parts of the brain. Although the cerebral cortex has traditionally been thought to be the seat of cognitive function, it has been found that children who are born without a cerebral cortex exhibit conscious awareness [13]. Also, in their pioneering work, Penfield and others found that awareness of self and environment were fully preserved even as they surgically removed relatively large areas of the cortex to treat refractory seizures [13, 14].

Further evidence that the cognitive-emotional system is something other than the neurological system is the large separation between the anatomical location where emotions are experienced and where the emotional wiring is located. Unlike the experience of mentation, which being in the head is not surprising given that the head is where the mental wiring is located, emotions are experienced in the chest despite the fact that the emotional wiring is likewise in the head. Unsurprisingly, it was once believed that emotions emanated from the beating heart. However, that idea began to

change with the discovery that the heart was just a pump in the chest. Of course, the brain is connected to the heart, and so one could surmise that perhaps the brain relays emotional messages to the heart. However, the connections between the brain and the heart are severed during heart transplant surgery, and there is no evidence that those who undergo such operations experience their emotions any differently than those who undergo other operations [15]. Then again, one could still speculate that perhaps the brain communicates emotion to the transplanted heart by signaling the release of circulating hormones or that perhaps the heart itself carries the emotion. However, an increasing number of modern-day heart transplants involve artificial hearts, and there is no evidence that those who receive such hearts experience their emotions any differently than before the surgery. Then again, the fact that there are connections between the brain and other areas of the body where emotions are sometimes experienced, such as the stomach, the intestines, and the genitals, still fails to explain why the associated feelings are not experienced in the head where the emotional wiring is located.

Yet another example that illustrates the distinction between the cognitive-emotional system and the neurological system is the differential response to an identical stimulus in different contexts. For example, the sense of alarm that one would experience upon hearing an unexpected knock at the door in the middle of the night would naturally be greater than if an identical knock were unexpectedly heard in the middle of the day. Given that the sound would stimulate the same auditory receptors at either time, the dramatic difference in one’s psychological, emotional, and physical response at one time versus the other could not possibly be explained by neurophysiology alone. The more plausible explanation is that the mind would react differently in the two different contexts. Because the knock in the middle of the night would be less expected than the knock in the middle of day, the mind would react with greater alarm, which in turn would cause a greater increase in heart rate, respiratory rate, muscle tone, and other indicators of autonomic arousal.

Beyond explaining these observable phenomena, a duality of mind and brain could help explain why some persons with mental illness become more symptomatic as they age, whereas others become less symptomatic [16]. Based on the hypothesis that mental illness involves a pathological dialogue between mind and brain, the long-term course of the symptomatology would depend upon the differential changes that occur in the mind and the brain over time. Personal growth, the development of more effective coping skills, and the establishment of stronger support systems would tend to reduce the amount of stress that the mind would experience on a day-to-day basis. This, in turn, would take stress off the brain, thereby reducing the neurological kindling through which stress is hypothesized to precipitate psychiatric symptoms and symptom-recurrences. Hence, such persons would tend to “grow out” of mental illness. Conversely, persons who failed to progress in the aforementioned ways would tend to become increasingly vulnerable to symptom

recurrences due to the progressive kindling that would occur with each psychiatric episode [17].

Note that each of the foregoing examples presumes that the mind is an independent entity that has the capacity for reasoning, memory, will, and all the other attributes that are generally associated with brain function only. Although this may sound inconsistent with the observation that consciousness is lost when the brain stops working, it is only inconsistent if one equates a loss of corporeal consciousness (consciousness in connection with the body) with a loss of consciousness in general. The idea that a loss of corporeal consciousness equates to a loss of consciousness in general is only an assumption, and it is an assumption that, particularly after death, has yet to be verified. On the contrary, many persons claim to have been conscious during a time when they were medically deemed to be unconscious, and there is growing evidence that consciousness persists even after death [18, 19]. An increasing number of persons, most commonly those who have been involved in a life-threatening emergency or highly traumatic event, have reported experiencing a sense of detachment from their bodies and feelings of levitation while on the brink of death [18, 19]. Some of these persons claim to have watched themselves being resuscitated in the emergency room or operated upon despite being deeply anesthetized. The detailed reports of some of these persons have been corroborated by the discovery of factual information that they could not possibly have known unless they really had been able to see, hear, and think during a time when hospital staff, based on subjective and objective clinical criteria, had assessed them as being unconscious [18-20].

Though the reports of these so-called “near-death experiences” (NDEs) had initially been considered weak evidence of consciousness beyond corporeal consciousness, the growing number of them, which is now in the millions globally, and the consistency of them across diverse ethnic, cultural, and religious groups, is making them increasingly hard to ignore [21]. Further adding to the credibility of NDEs is the fact that they are primarily unsolicited, unpaid, and apt to draw social scrutiny. Moreover, the NDE literature has, in recent years, not only become science-based in that it has been studied systematically, but it is arguably more credible than most scientific analyses in that the data involve large numbers of independent eye-witnesses rather than just one researcher or group of researchers.

Taken together, the aforementioned observations suggest that the cognitive-emotional system is a fully rational, self-willed entity that has the ability to function independent of the neurological system though it interacts with, and is dependent upon, the neurological system while in the corporeal state.

5. The Psychophysiology of Stress

It is self-evident that the mind has both carnal instincts and moral instincts. Carnal instincts, which fundamentally involve the desire for pleasure and the fear of pain, guide our

conduct in relation to ourselves [22]. Moral instincts, which include honesty, integrity, charity, patience, faith, hope, and love, guide our conduct in relation to others. These two sets of instincts often come into conflict in that we cannot always satisfy our carnal instincts without violating our moral instincts. Such conflict creates cognitive-emotional tension. There are also some stressors that create cognitive-emotional tension simply because they trigger strong emotion or require intense concentration. As previously mentioned, cognitive-emotional tension accelerates kindling because a stressed mind involves more energy than a relaxed mind, and that energy stimulates the associated circuits in the brain [23, 24]. That would explain why any intervention that calms the mind, whether it be proper rest, moderate exercise, mindfulness meditation, or psychotherapy, tends to reduce psychiatric symptoms. Note, however, that this explanation would not be possible apart from a duality of mind and brain.

6. Many Disorders, One Cause

Continuing with the premise that the cognitive-emotional system is a fully rational, self-willed entity that stimulates cognitive and emotionally-specific neurons and circuits as it thinks, emotes, and behaves, one would expect that the constellation of symptoms that any given individual would experience when under stress would be as different as one person is from another and one stressor is from another. Weighed against this, however, would be the natural tendency for each individual to develop his or her own stereotypical ways of reacting to stress. If these stereotypical profiles were categorized based on shared characteristics, they could easily be misconstrued to reflect different pathological processes. Hypothetically, it is this grouping of profiles that has led some experts to believe that different constellations of symptoms reflect different disease processes [16].

Another phenomenon that has led to symptom-based diagnostic distinctions is that of bipolar switching. Traditionally, patients who exhibited bipolarity were singled out and treated differently than patients who did not exhibit bipolarity [25]. Although symptom-cycling is now recognized to be a more general phenomenon that can involve any combination of symptoms [26], it is still singled out as a diagnostically, if not pathophysiologically, distinct disorder. However, the MCNH hypothesis posits that pathologically hyperactive circuit loops have a propensity to aberrantly fuel hyperactivity in inappropriate circuit loops when the neurological system is hyperexcitable [27, 28]. Computerized simulations of brain development suggest that the path taken by axons and dendrites as they sprout is more random than previously thought, an observation that suggests that the formation of neuronal-to-neuron connections is determined more by accidental collisions than biological programming [29]. This raises the possibility that neural signaling during cognitive-emotional processing could sometimes deviate from its intended path. Of course, the likelihood of aberrant neural signaling would be small

because neuronal-to-neuron connections that are used more frequently tend to be reinforced, whereas those that are used less frequently tend to be pruned [30]. However, the risk of aberrant signaling would increase if the neurological system were hyperexcitable, and it would increase even more if the individual were under stress because stress further increases the excitability of the neurological system [23, 24]. Under such conditions, hyperactive circuit loops could, with increasing frequency, aberrantly fuel hyperactivity in inappropriate circuit loops while themselves quieting down due to synaptic fatigue [31] (Figure 1). Note that once initiated, the rise in aberrant circuit-specific activity would draw mental attention to it, thus reinforcing the activity until the same process repeated itself through other (and possibly reciprocal) aberrant neuron-to-neuron connections (Figure 1). This is the MCNH explanation for why stress tends to kick up the waves of symptom-cycling. Note also that a patient's characteristic cycling frequency (for example, hours-to-days vs. weeks-to-months) would likely be determined by the number of aberrant neuron-to-neuron connections, which in turn would be proportionate to the total number of neurons and the degree of connectedness of the individual's neurological system [28]. Consistent with this hypothesis, symptom-cycling, though traditionally associated with bipolar disorder, actually appears to be an epiphenomenon of neuronal hyperexcitability [27].

However, the reason that symptom-cycling tends to go unrecognized is that the waxing and waning of symptoms tends to be overshadowed by the patient's chief complaint. For example, common symptoms, such as anxiety and depression, in addition to being part of the cycling, may reflect the patient's emotional response to the unpredictability that symptom-cycling creates. Also, a patient's perception of his or her symptom history tends to be state-dependent, and any perceived fluctuation of symptoms can easily be misattributed to external factors because they too are fluctuating [32]. Of course, a true absence of symptom-cycling would suggest that the neurological system was *not* hyperexcitable; however, such patients would be relatively rare because persons with normoexcitable neurons tend to be resistant to developing psychiatric symptomatology [28, 33].

Thus, the differences in symptomatology that have traditionally been thought to reflect different pathological processes are more likely reflective of differences in the way that different individuals manage stress psychologically and process stress neurophysiologically. Additional support for this conceptualization comes from the wide range of symptomatology that most psychiatric patients experience and the succession of different disorders that they tend to be diagnosed with [16]. The idea that different psychiatric disorders are more reflective of psychological differences than neurological differences is also suggested by the broad utility of antidepressants, anticonvulsants, and other drugs that are used to treat them. Taken together, these observations again highlight the distinction between the cognitive-emotional system and the neurological system.

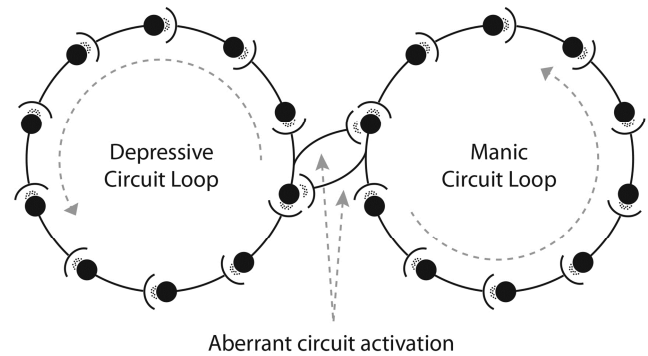


Figure 1. Schematic illustration of the means by which one hyperactive circuit loop could potentially fuel hyperactivity in an inappropriate circuit loop. In this example, the depressive circuit loop and the manic circuit loop inappropriately excite each other.

7. The Mind-Body Problem

The concept of mind as a distinctly different entity than the brain is certainly not new. Some of history's most renowned thinkers, including Socrates, Plato, Descartes, Popper, and Eccles, believed that the essence of the mind was different than that of the brain. The first to write extensively about a duality of mind and brain was the 16th century pioneer in mathematics, science, and metaphysics René Descartes. Descartes believed that the mind, though working closely with the brain, had a completely different nature than the brain [34]. Like many of those before him, he contended that the substance of the mind had to be different than that of the brain because the mind was rational, whereas the brain was physical. He also believed that the mind was able to function independent of the brain and the rest of the body. However, these ideas gave rise to the historic mind-body problem: how could the mind and the brain communicate with each other if their natures were different? Short of an answer to that historic question, the mind-body duality has largely been overshadowed by the idea that the mind is a product of complex brain function.

Yet the many advances that been made in the medical and physical sciences over the past two centuries have led researchers closer to solving the mind-body problem. The most logical way to explain how an invisible and intangible mind could interact with a physical and tangible brain is via the induction of magnetic fields. It is evident that mental activity induces magnetic fields because mentation is an energy-dependent process [35]. Conversely, it is well-known that neural signaling induces magnetic fields because it involves the movement of charged particles and the flow of current [36, 37]. What this implies is that the mind and the brain could communicate with each other in the same language; namely, electromagnetic energy. Also, quantum communication could explain how the behavior of laboratory animals, as referenced earlier, could be finely controlled with a beam of light (i.e., electromagnetic energy) [4, 5]. Similarly, it could explain how willful thoughts and emotions were readily able to stimulate specific neurons when subjects were asked to perform specific mental tasks [6]. What's more, a two-way dialogue between the mind and the brain seems

empirically correct. It is self-evident that the nervous system conveys sensory input to the mind and that the mind, using the same system, sends intention-specific signals to the body.

Note that this conceptualization also provides a psychophysiological basis for Freud's structural theory of the mind. A Century ago, Austrian psychiatrist Sigmund Freud divided the mind into functionally different compartments that he referred to as the "conscious mind" (what one is aware of), the "preconscious mind" (what one could become aware of through selective attention), and the "unconscious mind" (what one is unaware of though processing at a deeper level) [38]. Freud related these three levels of mental function to the three parts of the mind that he called the "id," the "ego," and the "superego" [22]. Here, the id was regarded as entirely unconscious whilst the ego and superego were regarded as having conscious, preconscious, and unconscious aspects. Though Freud had initially relied on neurological terms and concepts to formulate his theories, his desire to build a "scientific psychology" eventually caused him to part ways with neurological research [39]. Consequently, the psychophysiological distinction between the Freudian parts of the mind have largely remained unexplained. However, a duality of mind and brain offers an explanation for these distinctions. From the perspective of the mind-brain hypothesis, the id and the superego would represent carnal and moral instincts, respectively, whilst the ego would represent reality testing as mental impulses and neurological impulses became synchronized. Conscious thoughts would

be those that arose when neurological impulses fully synchronized with mental impulses; preconscious thoughts would be those that the brain could fully synchronize with if the mind were to turn its attention to them; and unconscious thoughts would be those that the brain, whether by the will of the mind or otherwise, was not fully synchronizing with (Figure 2).

Also note that as an energy body, the mind would process information at approximately 300,000,000 meters/sec (the speed of electromagnetic energy). In contrast, the brain can process information at a top speed of about 100 meters/sec (the speed of saltatory conduction), which is about 3,000,000 times slower than the mind would process information. This would imply that conscious thoughts are just the tip of the iceberg and that the vast majority of thought-life is unconscious, just as Freud proposed. Then again, the relatively slow speed of saltatory conduction and the further delays that occur at chemical synapses should not be viewed negatively. On the contrary, these delays, together with the reverberation of neurons and circuits that occurs as neurological signals synchronize with cognitive-emotional signals, would give the mind time to integrate, contemplate, and modify specific thoughts, beliefs, and attitudes in conjunction with sensory input from the environment. In other words, it would facilitate learning and personal growth through real life experiences while at the same time allowing the mind to regulate the more essential functions of the body with astounding swiftness, agility, and precision.

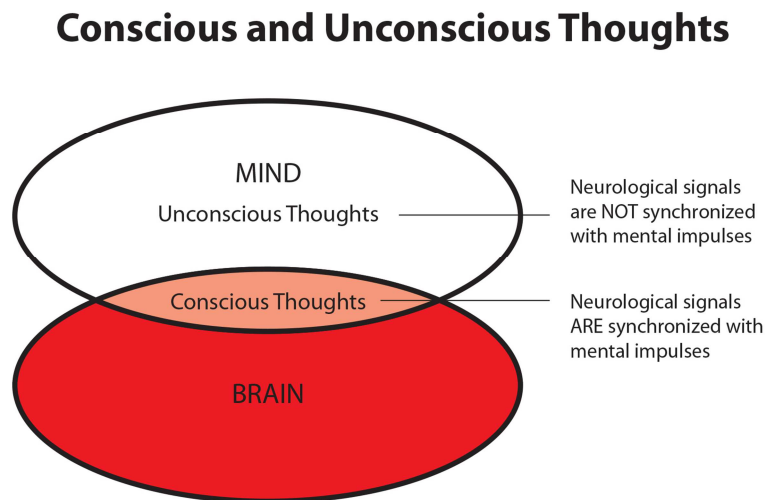


Figure 2. *Overlap between the mind and the brain illustrating the psychophysiological distinction between conscious and unconscious thoughts.*

Therefore, although the brain is often thought of as a complex, amazing, and somewhat mysterious organ, it appears more likely that most of these attributes actually belong to the mind, not the brain. The preponderance of evidence suggests that the brain is merely a computer in the head, just as the heart is a pump in the chest. Recall that because emotions are experienced in the chest, it was once thought that the heart was the seat of emotion. Likewise, because thoughts are experienced in the head, it is logical to think that the brain is the seat of cognition. However, it seems more likely that just as the heart was eventually

discovered to be little more than a pump in the chest, the brain will ultimately be discovered to be little more than a computer in the head.

Of course, that raises the question: where anatomically would the cognitive-emotional system be? The best answer to that question is likely the simplest one. The cognitive-emotional system, like the neurological and the circulatory systems, would likely be anatomically matched to the physical body (Figure 3). The mind, as the head of the cognitive-emotional system, would be in the head just as the brain, as the head of the neurological system, is in the head.

Likewise, the spiritual heart, as the heart of the cognitive-emotional system, would be in the chest just as the physical heart, as the heart of the circulatory system, is in the chest. This would also be consistent with the observation that thoughts are experienced in the head, whilst emotions are experienced in the chest.

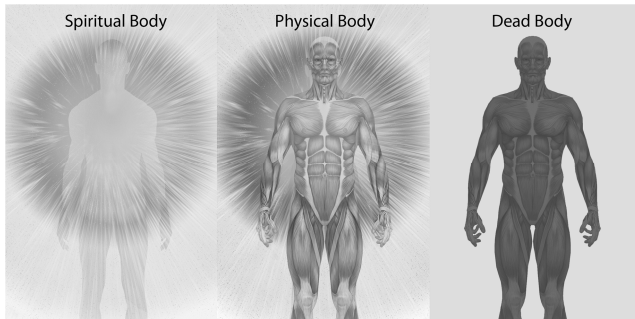


Figure 3. Proposed anatomy of the cognitive-emotional system (spiritual body) in comparison to the living body (physical body) and the dead body. Note the glove-like fit of the spiritual body to the physical body. Also note that because the magnetic field of the living body would include the quantum effects of physiological processes, it would be stronger than the magnetic field of the spiritual body. The dead body, being void of the life-giving spiritual body, would have neither of these magnetic fields.

Recognizing the energetic nature of the cognitive-emotional system, it would not be unreasonable to think that its activities would influence the physiological processes that occur in the physical body. Unfortunately, however, the wave interference that would be created by the superimposition of cognitive-emotionally-induced magnetic fields upon physiologically-induced magnetic fields would tend to preclude the ability to distinguish one from the other. Then again, the activity of the cognitive-emotional system is self-evident, as thoughts and emotions are the very fabric of our lives. Moreover, it may be that cognitive-emotionally-induced magnetic fields and physiologically-induced magnetic fields are rooted in the same energy source; namely, the spirit of life. Notably, this would be consistent with the NDE literature, which documents a close temporal relationship between: 1) the time that an NDE begins and the time that the heart stops beating; and 2) the time that an NDE ends and the time that the patient is successfully resuscitated [18, 19]. Note also that this would give death a clear definition: the separation of the spiritual body from the physical body. It would also be one that aligned with the common expression “passed away” in reference to death.

Of course, all of this raises another question about the mind-body connection. Where anatomically would the cognitive-emotional system interact with the physical systems of the body? The answer to that question can be deduced by studying what happens when various parts of the physical body are damaged. With the exception of injury to the brain, injury to any part of the body leaves corporeal consciousness intact. Therefore, the mind-body connection must occur in the head. Also, with the exception of damage to the neurological system, damage to any part of the body can be sensed by the mind. Therefore, the mind-body

connection must depend upon the neurological system. The only part of the neurological system that is in the head is the brain. Therefore, the mind-body connection must occur in the brain.

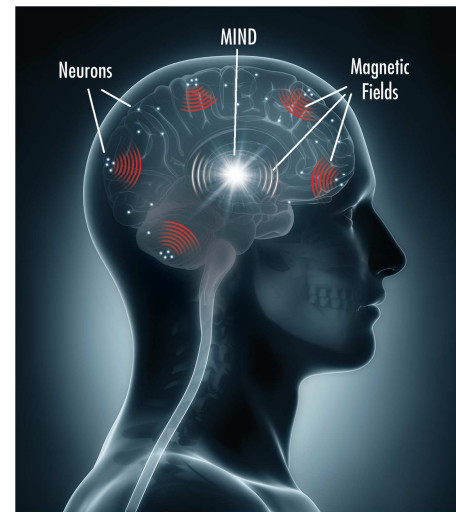


Figure 4. Schematic illustration of mentally-induced magnetic fields (large white burst and white radiations) interacting with neurologically-induced magnetic fields (pinpoint white bursts and red radiations) in the mind-brain dialogue.

That leads to another question: where in the brain is the mind? Because the mind is proposed to be made of energy rather than matter, it would be more appropriate to ask how the mind uses the brain to govern the body. Once again, the best answer is likely the simplest one. The mind appears to use the brain the same way that it uses a computer. Just as it uses a computer to interact with other computers connected to the internet, it uses the brain to interact with other parts of the body connected to the nervous system. More specifically, the mind sends messages to the brain by stimulating specific neurons [6], and the neurons relay the messages to other parts of the body via the peripheral nervous system (Figure 4). The computer analogy also helps explain how the mind knows which neurons to stimulate. The mind would learn this the same way it learns the spatial location of the keys it needs to punch on a computer keyboard. Conversely, the mind can keep abreast of what is happening in the body by being sensitive to the magnetic fields that are induced as the brain processes neural feedback from the body. Based on this conceptualization of the mind-body connection, learning and memory would be obligate functions of the mind, but they would stimulate neuroplastic changes in the brain that would increase the ease with which the related neurons and circuits could be reactivated by subsequent mental and emotional impulses. With each repetition, the responsivity of the associated neurons and circuits would increase, thus explaining why learning requires repetition.

Strikingly, the nature of the brain, its anatomical structure, and its functional characteristics are precisely what they would need to be were the brain actually serving the mind as a computer. To begin with, the functional units of the brain—the neurons—are electrical cells that, like the

transistors of a computer, relay electrical signals back and forth. Second, when viewed under a high-powered microscope, the brain looks just like the inside of a computer; that is, it's a clump of wires—not metallic wires—but biological wires called “axons” and “dendrites.” Third, just as a computer relays nearly all input to one specific location, namely, the computer monitor, the neurological system relays nearly all sensory input to one specific location, namely, the thalamus. The thalamus is a symmetrical, walnut-sized structure located at the core of the brain. From this strategic location, it is able to present to the mind (Figure 5A) all incoming neurological input and relay from the mind all willful output to various parts of the

brain for higher processing (Figure 5B). The convergence of sensory input upon the thalamus also solves the “binding problem,” which refers to the challenge of explaining time-dependent integration and segregation requirements of neurological information based on brain function alone. For example, in order to properly execute a tennis shot, the mind would have to know where the ball was, where the racket was relative to the ball, where the opponent was, where the fault line was, and many other pieces of information simultaneously. The convergence of sensory input upon the thalamus makes that possible, as it gives the mind simultaneous access to all the pieces of information that it needs to make fully informed responses.

Mind-Brain Dynamics

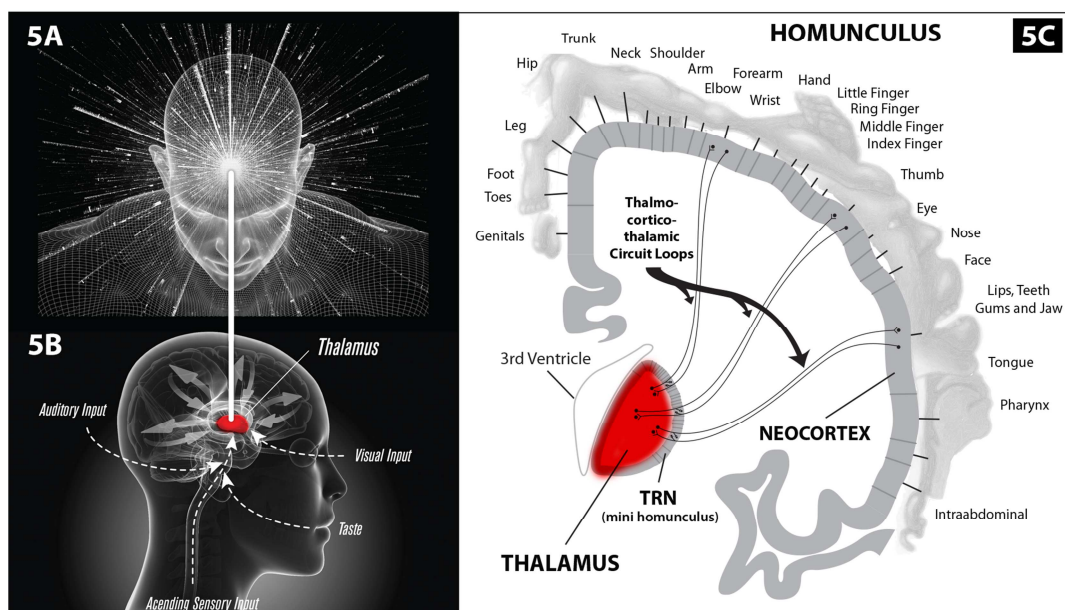


Figure 5. Upper left image depicts mental energy emanating from the core of the brain, where mental attention is hypothetically focused. Lower left image depicts the activity of thalamo-cortico-thalamic circuit loops as thoughts and emotions are processed by the brain. Right image is a schematic enlargement of the lower left image illustrating thalamo-cortico-thalamic circuit loops connecting the mini homunculus formed by the neurons of the TRN to that formed by the neurons of the neocortex.

Looking more closely at the thalamus (Figure 5C), one finds that a portion of the surface is comprised of a sheath of neurons called the *thalamic reticular nucleus* (TRN). All neuronal projections from the thalamus to the cerebral cortex pass through it, as do all reverse projections from the cortex to the thalamus (Figures 5B and 5C) [40]. Additionally, for all groups of neurons whose projections pass through a specific section of the TRN en-route to the cortex, there is a reverse projection from the cortex that passes through the same section of the TRN [40]. Thus, the cells of the TRN are topographically arranged to form a map or “homunculus” of the body that corresponds to the same homunculus in the cortex [41] (Figure 5C). This makes the TRN a spatially arranged hub that is capable of modulating nearly all of the informational traffic that is being processed by the brain.

Though once thought to be a passive relay center, more recent studies have found that the thalamus is heavily involved in sensory processing and, like a conductor in an

orchestra, coordinates the flow of information through various parts of the brain. For example, when light stimulates the retina of the eye, the information is relayed to the thalamus before being sent to the visual cortex for higher processing. But in addition to modulating the flow of information to the cortex, the thalamus continues to be part of the conversation as the information is being processed. In a set of elegant imaging experiments, Theyel et al. [42] found that severing the connections between two separate but communicating parts of the cortex in the mouse brain did not prevent the communication from occurring. Instead, the communication continued via circuit loops between the two different parts of the cortex and the thalamus, thus indicating that cortico-thalamo-cortico circuit loops are involved in the higher processing of information.

Another observation illustrates how important these circuit loops are. When visual input from one eye is continuously blocked early in development, there is a reduction in the

number of cortical neurons responding to the blinded eye together with a corresponding increase in the number of neurons responding to the seeing eye. Eventually, the imbalance of activity between the two eyes results in a decrease in the number of connections between the thalamus and the blinded eye together with a corresponding increase in the number of connections between the thalamus and the seeing eye [43]. This indicates that cortico-thalamo-cortico circuit loops are not just alternative pathways but are essential to normal cortical function. Other research has found that, in addition to visual processing, the thalamus coordinates cortical synchrony, executive function, sensory-motor activity, goal-directed behavior, levels of arousal, emotional states, behavioral flexibility, and the neural correlates of memories [44]. Clearly, the thalamus is the operational cockpit of the brain. But who could imagine a group of cells being able to think, emote, reflect on the past, and make all our decisions for us? Lung cells don't do that; kidney cells don't; liver cells don't. So how could brain cells, which are made of the same building blocks as these other cells, carry out all these human functions? Such a thing would make us mindless robots whose lives were dictated by spontaneous biological processes and neurological reflexes.

Clearly, it is the mind that carries out these functions, and it is the mind that has human experiences; but as the mind thinks, emotes, and makes decisions, the associated magnetic fields hypothetically influence the firing of neurons, including the cells of the TRN.

The pioneering work of Anne Treisman and her colleagues [45-47], supported more recently by a set of elegant experiments by Julesz [48, 49] and Bergen and Julesz [50], has suggested that there is an "attentional searchlight" that scans and selects information coming into the TRN [40]. The searchlight is not proposed to light up areas of a completely dark landscape but rather, like a searchlight at dusk, is thought to illuminate those parts of a dimly lit landscape that are of particular interest to it. According to the investigators, it does this by stimulating select assemblies of cells in the TRN (Figure 6). Although the collaterals of these cells are largely (if not entirely) inhibitory, specialized burst activity allows them to enhance the activation of select neural networks when stimulated [51]. The mechanism by which this occurs is based on the unique physiology of thalamic neurons. Elegant studies on thalamic slices from the guinea pig have confirmed that when hyperpolarized thalamic neurons are stimulated, they respond by producing a single spike (or short burst of rapid spikes) followed by a brief period during which they are unresponsive to continued stimulation [52-54]. This implies that when the attentional searchlight, which could be none other than the human mind, turns its attention to a point of interest, the excitatory phase initiates a wave of inhibition that turns down irrelevant information while the refractory phase allows activity in select circuits to be turned up. In this way, the TRN allows the mind to scan the information coming into the thalamus, highlight select inputs, and then shift attention to other areas of potential interest. This could explain how, on a

psychophysiological basis, the mind is able to contemplate or, conversely, repress various thoughts, emotions, and images.

Circuitry of the Mind-Brain Interface

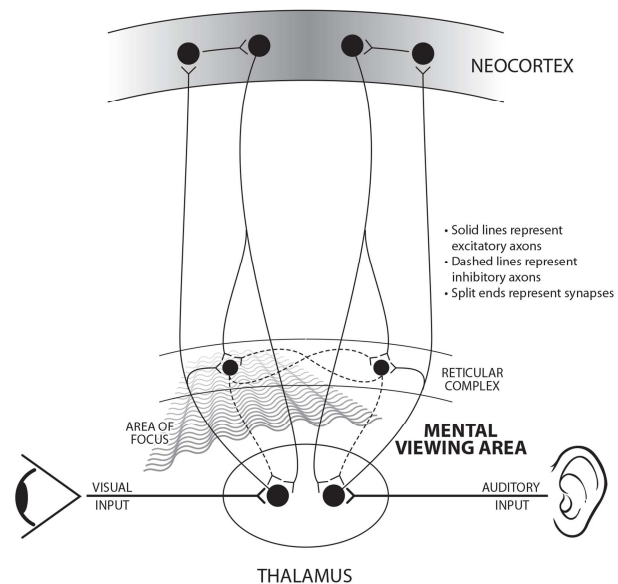


Figure 6. Schematic illustration of the human mind (wavy grid) scanning information coming into the TRN at the mind-brain interface. Input from the eyes, ears, and other sensory organs is relayed directly to the corresponding nuclei of the thalamus (bottom center). From there, the signals are sent to the corresponding areas of the cerebral cortex for higher processing before looping back to the corresponding nuclei of the thalamus. Both going and coming, collaterals from these informational tracts synapse with target cells of the reticular nucleus, thereby creating a mental touchscreen through which the mind can monitor and modulate nearly all of the information that is being processed by the brain. Note that the reticular neurons are inhibitory, thus allowing the TRN to act as a circuit-breaker that keeps the mind from becoming distracted by information that it does not intentionally select. Diagram is adapted from Crick, 1984: "Function of the thalamic reticular complex: The searchlight hypothesis" [40].

In addition to supporting the mind-brain hypothesis, the structure and function of the brain in conjunction with the MCNH hypothesis provides greater insight into the cause of specific mental illnesses. For example, it can help explain both the cause of symptoms in attention deficit hyperactivity disorder (ADHD) and the mechanism by which various medical and natural interventions exert their therapeutic effects in persons who have the disorder. Based on extensive clinical observations, nearly all patients with ADHD have hyperexcitable neurological systems [28]. It is hypothesized that the hyperexcitability of the system increases neuronal synchrony, thus flooding the TRN and overwhelming its modulatory capacity. This can cause the mind to experience intrusive thoughts, which are distracting, and it can cause behavior to be impulsive due to both an overabundance of neural signaling to the motor cortex and a failure of the slower functioning brain to fully think things through in conjunction with the mind before the mind decides to act upon its thoughts and feelings. This could explain the triad of symptoms that tend to occur in ADHD unless the neurological system is either quelled by drugs like clonidine

and guanfacine, or the activity of dopamine and norepinephrine, which enhance the modulatory capacity of the TRN [55], is bolstered by drugs like methylphenidate and dextroamphetamine [56]. Note that these catecholamines can likewise be increased by activities that involve pleasure and excitement, thus explaining why such activities can be as effective as medication in reducing ADHD symptoms. However, the potential liability of stimulating the brain is that it can exacerbate the underlying problem of neuronal hyperexcitability. This in turn can partially negate the therapeutic effects of psychostimulants, especially over time. It can also aggravate co-occurring psychiatric symptoms or even precipitate new ones, thus explaining the potential for psychostimulants to cause irritability, insomnia, and other activating effects. For this reason, ADHD is best managed with brain-calming interventions. Such intricate explanations and practical recommendations for treatment of a disorder that has heretofore remained unexplained would not be possible without a mind-brain duality of the cognitive-emotional system in conjunction with the MCNH hypothesis of psychiatric disorders.

The necessity of the mind-brain hypothesis becomes even more apparent when the role of stress is factored in. As previously discussed, there is general agreement that cognitive-emotional stress is the most ubiquitous precipitant of psychiatric symptomatology. This has been the foundation for the many forms of psychotherapy that have been practiced over the centuries, particularly after Freud cited the importance of intrapsychic conflict in the development of psychopathology. According to mind-brain hypothesis, intrapsychic stress causes cognitive-emotionally-induced magnetic fields, like stones being thrown at a beehive, to overstimulate the brain. As the associated neurons and circuits become pathologically hyperactive, the magnetic fields that they induce begin to overtake the more subtle magnetic fields that are induced by willful thoughts and emotions. In other words, they begin to make a person think thoughts and experience emotions that he or she may not intend to think or feel. For example, pathologically-elevated activity in cognitive circuits could cause obsessive *thoughts* of contamination, and pathologically-elevated activity in anxiety circuits could cause obsessive *fears* of contamination, thus explaining the classic symptoms of obsessive-compulsive disorder. The same could be said of other thoughts and emotions, such as those that characterize mood disorders, eating disorders, and other persistent cognitive-emotional states. Hypothetically, it would also be possible that cognitive functions that would normally activate the corresponding emotional circuitry would be unable to do so because hotspots of neuronal activity were competing for dominance [57]. As a result, the person's emotions, rather than being dictated by the thought content, would be dictated by inappropriate firing in limbic circuitry. It would also be possible that the thought content, rather than being dictated by the emotions, would be dictated by inappropriate firing in cognitive circuitry. Sensory systems could also be affected by pathologically-elevated neurological activity. For example,

pathologically-elevated activity in the auditory processing system could cause the person to think that the auditory nerve were being stimulated. This could explain auditory hallucinations (Figure 7). Likewise, pathologically-elevated activity in the visual processing system could cause the person to think that the optic nerve were being stimulated. This could explain visual hallucinations, etc... Other forms of psychosis, such as paranoia and delusional thinking, could occur when the intensity of internally-generated, circuit-specific signaling began to approach the intensity of signaling that would normally be driven by the higher processing of auditory, visual, and other sensory input. In other words, the hyperexcitable brain could amplify purely internal processes to the point that the mind, believing that the impetuses were coming from the environmental, began to weave the content into narratives to explain what it believed to reflect external reality. The risk of such aberrant signaling would be increased by intrapsychic stress, stimulant-type drugs, rapid hormonal changes, or any factor that increases excitation in the brain, thus explaining why psychotic symptoms are more likely to develop under such circumstances [1]. In extreme cases, the willful intentions of the individual could be completely usurped by this intensive, spontaneous, electrical activity. Such chaotic brain signaling would be more likely to occur in persons with exceedingly high levels of neuronal excitability, such as those with schizophrenia, bipolar disorder, borderline personality disorder, and other severe psychiatric disorders. That such persons have exceedingly high levels of neuronal excitability is corroborated by the elevated risk of seizures that they have in comparison to those with less debilitating psychiatric disorders [10, 58, 59].

Psychotic Symptoms

(Auditory Hallucinations)

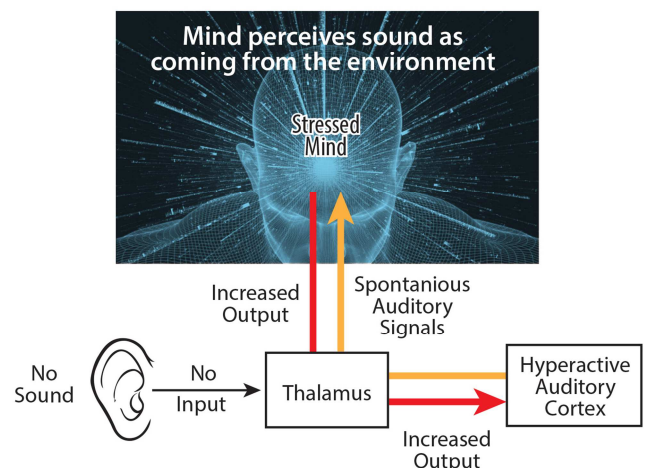


Figure 7. Psychophysiology of hallucinatory experiences and psychotic states. In the example above, a small network of neurons that would normally be activated by input from the auditory system begins to fire spontaneously or, more commonly, in response to stimulation from the stressed mind (red line). The rise in circuit-specific thalamo-cortical activity (red arrow) induces magnetic fields that the mind interprets as sounds from the environment. The likelihood of this phenomenon would increase as the level of electrical activity in the brain increased.

Of course, this reasoning would not be limited to cognitive and emotional systems. Pathologically-elevated activity in specific neurons and circuits could also affect motor systems of the body. For example, pathologically-elevated activity in the skeletal muscular system could cause such things as physical hyperactivity, chronic muscle tension, and motor tics, thus explaining why persons with mental illness tend to have corresponding behavioral and physical symptoms. Similar effects on smooth muscle, such as in the bowel, could cause either hypermotility (diarrhea) or spastic immobility (constipation), thus explaining the link between mental illness and irritable bowel syndrome. Note that when these symptoms are experienced by the mind, the cognitive-emotional stress would tend to further exacerbate them because the associated rise in the intensity of cognitive-emotionally-induced magnetic fields would tend to further increase the level of excitation in the associated neurons and circuits, thus creating a vicious cycle of stress, symptomatology, and more stress.

Other systems of the body that could be affected by this dynamic include the endocrine, the metabolic, and the immunological systems, thus helping to explain the link between mental illness and obesity, diabetes, heart disease, asthma, thyroiditis, rheumatoid arthritis, and other health conditions that are associated with a dysregulation of these systems [60]. Moreover, it is hypothesized that even in the absence of cognitive-emotional stress, an inherent hyperexcitability of the neurological system could drive a hyper-reactivity of the system, thus explaining the link between mental illness and postural orthostatic tachycardia syndrome (POTS) [61]. It could also drive a subtle increase in heart rate, respiratory rate, and blood pressure [60], thus explaining why upper-end-of-normal resting vital signs are, from an early age, predictive of the early development of any of a wide range of mental, emotional, and physical illnesses [60].

Note, however, that although a superimposition of stress upon a hyperexcitable neurological system could explain the aforementioned health conditions, it would not explain the symptoms themselves, nor would it explain the person's response to them. To explain these phenomena, an interpretive aspect is needed. In other words, there must be an observer who experiences the symptoms, interprets them, and reacts to them. That observer would logically be the person who lives inside the body; the person who has the ability to think, feel, and react to whatever it is that he or she experiences in association with the body. Also note that if, as previously suggested, the non-physical or "spiritual body" were anatomically aligned with the organs of the physical body, it would experience sight in the spiritual eyes, where the physical eyes are located; it would experience hearing in the spiritual ears, where the physical ears are located; it would experience thoughts in the mind, where the brain is located; and it would experience emotions in the spiritual heart, where the physical heart is located (Figure 3). Of course, all of these senses are known to be associated with the corresponding organs of the physical body, but it seems more likely that they and their connections to the brain are

merely conduits through which the sensory input is communicated to the person who senses it, interprets it, and reacts to it.

How then would this system function psychophysiologically? As is currently recognized, sensory input would be relayed from the physical body's sensory organs to the thalamus. During this process, the information would be communicated to the mind via the magnetic fields that are induced as the associated neurons depolarize and repolarize. Subsequently, the mind, working at the speed of light, would process the information through the spiritual body's cognitive-emotional system. Simultaneously, the magnetic fields thus induced would influence the activity of the related neurons and circuits. Through this highly efficient process, the mind (and by extension the rest of the spiritual body) could readily experience what the physical body experiences and reciprocally communicate its responses to the physical body via the brain.

In addition to providing a logical mechanism for the psychophysiological processing of cognitive and emotional information, this conceptualization is consistent with the long-held belief that the physical body is indwelt by a non-physical entity, traditionally referred to as the "psyche," "soul," or "spirit." That such an entity is invisible and intangible should not deter us from being open to its existence, as bacteria and other microorganisms, which are now known to outnumber the body's own cells [62], were once thought to be non-existent because they were invisible and intangible. The same could be said of electromagnetic radiation, most of which is invisible, yet has clearly been demonstrated to exist. In addition, the belief that a non-physical entity dwells within and animates the physical body has been held for far longer and with much more consistency than any modern scientific belief. For scientists to renounce such a belief without convincing proof of its falsity would be contrary to science and more fool-hardy than to acknowledge it without convincing proof of its accuracy.

8. Discussion

Despite the many efforts that have been made to elucidate the cause of mental illness, the psychophysiological mechanism by which psychiatric symptoms develop and perpetuate remains unclear. However, a new hypothesis—one that integrates the effects of both cognitive-emotional processes and neurological processes—may have solved the mystery. According to the MCNH hypothesis of psychiatric disorders, psychiatric symptoms are precipitated by an acute or chronic stressor superimposed upon an inherent hyperexcitability of the neurological system and perpetuated by a vicious cycle of mutual overstimulation between the mind and the brain. This integrative hypothesis offers: 1) a psychophysiological mechanism by which different constellations of symptoms can be driven by a shared neurophysiological abnormality; 2) a psychophysiological means by which different constellations of symptoms can be experienced by the same person at different points in time; 3) a

psychophysiological means by which symptoms can meld into one another and oscillate back and forth in a wide range of psychiatric disorders; 4) a psychophysiological means by which a split or “schism” between thoughts and emotions can occur in some persons with severe mental illness; 5) a psychophysiological means by which psychotic symptoms and other perceptual abnormalities can be experienced in persons who are vulnerable to developing psychiatric symptomatology; 6) a psychophysiological means by which various physical symptoms can occur in conjunction with psychiatric symptomatology; 7) a psychophysiological means by which symptoms can, in rare instances, develop in persons with normoexcitable neurological systems; 8) a psychophysiological explanation for the characteristic delay between the onset of a cognitive-emotional stressor and the onset of psychiatric symptoms; 9) a psychophysiological explanation for the observation that some persons with mental illness become more vulnerable to symptom recurrences over time, whereas others become less vulnerable; 10) a psychophysiological conceptualization of the distinction between conscious, preconscious, and unconscious thoughts; 11) a psychophysiological explanation for why persons who are more vulnerable to developing mental illness are also more vulnerable to developing any illness, whether mental or physical, that can be precipitated or exacerbated by stress; 12) a psychophysiological explanation for the bidirectional link between upper-end-of-normal resting vital signs, an inherent hyperexcitability of the neurological system, and an increased risk of developing any of a wide range of the psychiatric, functional, and physical illnesses.

It should be reiterated, however, that the concept of mind that is integral to the foregoing hypothesis is not merely mind as a manifestation of complex brain function but mind as an independent entity that has its own thoughts, emotions, hopes, fears, and dreams. It also refers to mind as having the faculties of selective attention, memory, and will independent of brain function. In other words, it is a mind that has the capacity for consciousness and all its attributes whether in or out of the corporeal state.

This concept of mind is very different than that which has been proposed by reductionist theories of consciousness, such as the electromagnetic field theory of consciousness and the conscious electromagnetic information (CEMI) field theory of consciousness. For example, the electromagnetic field theory of consciousness proposed by Susan Pockett posits that mental processes are a manifestation of neurologically-induced magnetic fields and that the distinction between conscious and unconscious neurological processes is determined by the 3-dimensional shape of a cortically-induced electromagnetic field [63]. Similarly, the CEMI field theory of consciousness proposed by Johnjoe McFadden posits that mental processes are a manifestation of neurologically-induced magnetic fields, but in CEMI field theory, the distinction between conscious and unconscious processes is determined by the interference patterns of electromagnetic waves: constructive interference increases the amplitude of some waves, thereby making them

conscious, whereas destructive interference reduces the amplitude of other waves, thereby preventing them from rising to the level of consciousness [64, 65]. CEMI field theory further asserts that ongoing mental processes, such as reasoning and recall, are driven by “quantum entanglement,” which can be defined as the aggregate of all the magnetic fields that are induced by neurological activity at any point in time. Because quantum entanglement is instantaneous, it proposes to solve the binding problem.

The weakness of these and other reductionist theories, however, is that they fail to account for the previously-described faculties of the mind, the most basic of which are self-awareness, emotion, and will. They also fail to explain the phenomenology of stress on a cognitive-emotional level and how, psychophysiological, stress tends to precipitate psychiatric symptomatology. However, the strength of these theories is that they highlight the importance of electromagnetism in mind-brain dynamics. As previously discussed, electromagnetism is, hypothetically, the shared language through which the mind and the brain communicate with each other.

Although some of the concepts that have been presented herein have yet to be proven scientifically, Occam’s razor states that the most likely explanation is the simplest one. The simplest way to explain how psychiatric and functional physical symptoms develop is through a duality of mind and brain. Particularly when the neurological system is hyperexcitable and the mind is under stress, tension in the cognitive-emotional system induces a pathological elevation in the activity of stress-related circuits in the brain. This in turn adds fuel to the tension in the cognitive-emotional system, thus creating a vicious cycle of mutual overstimulation that gradually increases the strength of the associated magnetic fields. As the mind-brain dialogue continues to ramp up, new hotspots of electrical activity tend to develop in the brain, and symptoms tend to cycle as pathologically-hyperactive circuit loops fuel activity in inappropriate circuit loops via aberrant neuron-to-neuron connections [27, 28]. Meanwhile, the intensity of the associated magnetic fields, though not rising high enough to cause a loss of consciousness (as in complex seizures), can eventually overpower the relatively low-intensity magnetic fields that the mind induces as it labors to control what it is thinking and feeling. In extreme cases, the willful intentions of the individual can be completely usurped by this rising electromagnetic activity.

Thus, persons with mental illness are not mindless automatons passively riding the waves of neurologically-induced magnetic fields but rather living beings who are desperately trying to maintain control of their lives amidst the racing thoughts and surging emotions that are caused by cognitive-emotional stress superimposed upon a hyperexcitability of the neurological system. Although pathologically-elevated circuit-specific neurological activity can potentially develop spontaneously if the inherent excitability of the neurological system is high enough, only a mind-brain duality of the cognitive-emotional system in

conjunction with the MCNH hypothesis can fully explain the psychophysiology of mental illness.

9. Suggestions for Future Research

Urgently needed are clinical studies aimed at determining: 1) the benefits of focusing treatment on reducing the excitability of the neurological system; 2) the benefits of anticonvulsant therapy alone in comparison to other pharmacological agents alone, psychotherapy alone, or a combination of the two for a wide variety of psychiatric disorders; and 3) the benefits of combining different anticonvulsants with one another for a wide range of psychiatric disorders.

10. Conclusion

Despite unprecedented strides in the ability to visualize and monitor neurological activity, the underlying cause of mental illness remains poorly understood. However, a fresh new look at the relationship between the cognitive-emotional system and the neurological system in conjunction with the MCNH hypothesis of psychiatric disorders may have lifted the veil on the most common yet most perplexing group of disorders known to humankind. According to the mind-brain hypothesis, the mind is not merely a manifestation of complex brain function but an independent entity that has the ability to think, reason, and access memories both in conjunction with and independent of the neurological system. It is thought that the mind, as the head of the cognitive-emotional system, interacts with the brain via the induction of magnetic fields and that these energy fields, when escalated to pathological levels of intensity by psychosocial stress superimposed upon an inherent hyperexcitability of the neurological system, drive the development of psychiatric symptomatology.

Although the mind-brain hypothesis has yet to be validated through rigorous scientific experimentation, the logic, simplicity, and explanatory power of the hypothesis bear witness to its validity. Many of the greatest scientists and thinkers throughout history have said that the beauty and simplicity of a theory is greater evidence of truth than scientific experimentation. "Beauty brings with itself evidence that enlightens without mediation," wrote Hans Von Balthasar, one of history's most renowned philosophers. The broad explanatory power of the mind-brain hypothesis should not be surprising given that it recognizes the independence and versatility of the cognitive-emotional system in relation to the neurological system and more clearly defines the functional anatomy of psychophysiological processes. My hope is that the conceptualization that has been presented herein will better orient future research in psychiatry and hasten the development of more precise and effective treatments for what has been called "the world's largest single health problem."

Conflicts of Interest

The author declares that he has no competing interests.

References

- [1] Binder MR. The multi-circuit neuronal hyperexcitability hypothesis of psychiatric disorders. *AJCEM* 2019; 7 (1): 12-30.
- [2] Ritaccio AL, Bruner P, Schalk G. Electrical stimulation mapping of the brain: Basic principles and emerging alternatives. *J Clin Neurophysiol* 2018; 35 (2): 86-97.
- [3] Penfield W. Epilepsy and surgical therapy. *Archives of Neurology and Psychiatry* 1936; 36 (3): 449-484.
- [4] Aravanis AM, Wang L-P, Zhang F, et al. An optical neural interface: in vivo control of rodent motor cortex with integrated fiberoptic and optogenetic technology. *Journal of Neural Engineering* 2007; 4 (3).
- [5] Boyden ES, Zang F, Bamberg E, Nagel G, Deisseroth K. Millisecond-timescale, genetically targeted optical control of neural activity. *Nature Neuroscience* 2005; 8: 1263-1268.
- [6] Cerf M, Thiruvengadam N, Mormann F, et al. On-line, voluntary control of human temporal lobe neurons. *Nature* 2010; 467: 1104-1108.
- [7] Ferreira MAR, O'Donovan MC, Sklar P. Collaborative genome-wide association analysis supports a role for ANK3 and CACNA1C in bipolar disorder. *Nat Genet* 2008; 40 (9): 1056-1058.
- [8] Yuan A, Yi Z, Wang Q, et al. ANK3 as a risk gene for schizophrenia: new data in Han Chinese and meta analysis. *Am J Med Genet B Neuropsychiatr Genet* 2012; 159B (8): 997-1005.
- [9] Green EK, Grozeva D, Jones I, et al., Wellcome Trust Case Control Consortium, Holmans, PA, Owen, MJ, O'Donovan, MC, Craddock N. The bipolar disorder risk allele at CACNA1C also confers risk of recurrent major depression and of schizophrenia. *Mol Psychiatry* 2010; 15 (10): 1016-1022.
- [10] Lopez AY, Wang X, Xu M, et al. Ankyrin-G isoform imbalance and interneuronopathy link epilepsy and bipolar disorder. *Mol Psychiatry* 2017; 22 (10): 1464-1472.
- [11] Goddard GV. Development of epileptic seizures through brain stimulation at low intensity. *Nature* 1967; 214: 1020-1021.
- [12] Rose GM, Diamond DM, Pang K, Dunwiddie TV. Primed burst potentiation: lasting synaptic plasticity invoked by physiologically patterned stimulation. In: Haas HL, Buzsáki G. (eds) *Synaptic plasticity in the hippocampus*. Springer, Berlin, Heidelberg, 1988.
- [13] Merker B. Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences* 2007; 30 (1): 63-134.
- [14] Kawkabani K. Preserved consciousness in the absence of a cerebral cortex, the legal and ethical implications of redefining consciousness and its neural correlates: A case for a subcortical system generating affective consciousness. *Neuroscience and Neurobiology Commons, Honors Research Projects* 2018; 734.
- [15] (<http://blogs.discovermagazine.com/seriouslyscience/2014/07/31/getting-heart-transplant-change-personality/#.W1YU0cu9KK0>). Accessed 5/16/18).

- [16] Caspi A, Houts RM, Ambler A, et al. Longitudinal assessment of mental health disorders and comorbidities across 4 decades among participants in the Dunedin Birth Cohort Study. *JAMA Netw Open* 2020; 3 (4): e203221.
- [17] Post RM. Mechanisms of illness progression in the recurrent affective disorders. *Neurotox Res* 2010; 18 (3-4): 256-271.
- [18] Moody RA. *Life after life*. Mockingbird Books, 1975.
- [19] Long J. *Evidence of the afterlife: The Science of near-death experiences*. HarperOne, 1975.
- [20] Parnia S, Spearpoint K, de Vos G. AWARE—AWAREness during resuscitation—A prospective study. *Resuscitation* 2014; 85: 1799-1805.
- [21] Van Lommel P. Near-death experiences: the experience of the self as real and not as an illusion. *Ann N Y Acad Sci* 2011; 1234: 19-28.
- [22] Freud S. (1915). The unconscious. *SE*, 14: 159-204.
- [23] Fuchs E, Flügge G. Chronic social stress: effects on limbic brain structures. *Physiology & Behavior* 2003; 79 (3): 417- 427.
- [24] Mehler B, Reimer B, Coughlin JF, Dusek JA. Impact of incremental increases in cognitive workload on physiological arousal and performance in young adult drivers. *Transportation Research Record: Journal of the Transportation Research Board* 2009; (2138): 6-12.
- [25] Kahn DA, Sachs GS, Printz DJ, Carpenter D. Medication treatment of bipolar disorder 2000: A summary of the expert consensus guidelines. *Journal of Psychiatric Practice* 2000; 6 (4): 197-211.
- [26] Akiskal HS. The bipolar spectrum: new concepts in classification and diagnosis. In: Grinspoon L, editor. *Psychiatry Update; The American Psychiatric Association Annual Review*. Vol. 2. Washington DC: American Psychiatric Press 1983, pp. 271–292.
- [27] Binder MR. Electrophysiology of seizure disorders may hold key to the pathophysiology of psychiatric disorders. *AJCEM* 2019; 7 (5): 103-110.
- [28] Binder MR. The Neuronal Excitability Spectrum: a new paradigm in the diagnosis, treatment, and prevention of mental illness and its relation to chronic disease. *AJCEM*; 2021; 9 (6): 187-203.
- [29] Perin R, Berger TK, Markram H. A synaptic organizing principle for cortical neuronal groups. *PNAS* 2011; 108 (13): 5419-5424.
- [30] Yuan P, Monie M. Activity shapes neural circuit form and function: A historical perspective. *Journal of Neuroscience* 2020; 40 (5): 944-954.
- [31] Henkel AW, Welzel O, Groemer T W, et al. Fluoxetine prevents stimulation-dependent fatigue of synaptic vesicle exocytosis in hippocampal neurons. *Journal of Neurochemistry* 2010; 114 (3): 697-705.
- [32] Lubloy A, Kereszturi JL, Nemeth A, Mihalicza P. Exploring factors of diagnostic delay for patients with bipolar disorder: a population-based cohort study. *BMC Psychiatry* 2020; 20 (75).
- [33] Binder MR. A pathophysiologically-based approach to the treatment and prevention of mental illness and its related disorders. *AJCEM* 2021; 9 (6): 223-232.
- [34] David Cunning. *The Cambridge Companion to Descartes' Meditations*. Cambridge University Press, 2014. p. 277. ISBN 978-1-107-72914-8.
- [35] Pandya SK. *Understanding brain, mind and soul: Contributions from neurology and neurosurgery*. Mens Sana Monogr 2011; 9 (1): 129-149.
- [36] Forbes N, Mahon B. *Faraday, Maxwell, and the electromagnetic field: How two men revolutionized physics*. Prometheus Books, New York, 2014.
- [37] Xu Y, Jia Y, Ma J, Hayat T, Alsaedi A. Collective responses in electrical activities of neurons under field coupling. *Sci Rep* 2018; 8: 1349.
- [38] Freud, S. (1924). *A general introduction to psychoanalysis*, trans. Joan Riviere.
- [39] Dimkov P. Large-scale Brain Networks and Freudian Ego. *ResearchGate* 2018 <https://www.researchgate.net/publication/326468259>.
- [40] Crick F. Function of the thalamic reticular complex: The searchlight hypothesis. *Proceedings of the National Academy of Sciences* 1984; 81: 4586-4590.
- [41] Thomson AM, Lamy C. Functional maps of neocortical local circuitry. *Front Neurosci* 2007 <https://doi.org/10.3389/neuro.01.1.1.002.2007>
- [42] Theyel BB, Llano AL, Sherman SM. The corticothalamocortical circuit drives higher-order cortex in the mouse. *Nature Neuroscience* 2010; 13: 84-88.
- [43] Baroncelli L, Braschi C, Spolidoro M, et al. *Brain Plasticity and Disease: A Matter of Inhibition*. Neural Plasticity; 2011.
- [44] Herrero MT, Insausti R, Estrada C. (2015) Reference Model in Neuroscience and Biobehavioral Psychology. *Brain Mapping: An Encyclopedic Reference*. Vol. 2: Anatomy and Physiology Systems: 219-242.
- [45] Treisman A. Focused attention in the perception and retrieval of multidimensional stimuli. *Perception & Psychophysics* 1977; 22 (1): 1-11.
- [46] Treisman A, Gelade GA. A feature integration theory of attention. *Cognitive Psychology* 1980; 12: 97-136.
- [47] Treisman A, Schmidt H. Illusory conjunctions in the perception of objects. *Cognitive Psychology* 1982; 14: 107-141.
- [48] Julesz B. Spacial nonlinearities in the instantaneous perception of textures with identical power spectra. *Philosophical Transactions of the Royal Society of London B* 1980; 290: 83-94.
- [49] Julesz B. Textons, the elements of texture perception, and their interactions. *Nature* 1981; 290: 91-97.
- [50] Bergen JR, Julesz B. Parallel versus serial processing in rapid pattern discrimination. *Nature* 1983; 303: 696-698.
- [51] Kwasniak J. Looking at the thalamic reticular nucleus. <http://charbonniers.org/2013/02/13/looking-at-the-thalamic-reticular-nucleus/>. (Accessed 5/17/18).
- [52] Llinas R, Jahnsen H. Electrophysiology of mammalian thalamic neurons in vitro. *Nature (London)* 1982; 297 (5865): 406-408.

- [53] Jahnsen H, Llinas R. Electrophysiological properties of guinea-pig thalamic neurones: an in vitro study. *J. Physiol* 1984; 349 (1): 205-226.
- [54] Jahnsen H, Llinas R. Ionic basis for the electro-responsiveness and oscillatory properties of guinea-pig thalamic neurones in vitro. *J Physiol* 1984; 349: 227-247.
- [55] Erlij D, Acosta-García J, Rojas-Márquez M, et al. Dopamine D4 receptor stimulation in GABAergic projections of the globus pallidus to the reticular thalamic nucleus and the substantia nigra reticulata of the rat decreases locomotor activity. *Neuropharmacology* 2012; 62 (2): 1111-1118.
- [56] Faraone SV. The pharmacology of amphetamine and methylphenidate: Relevance to the neurobiology of attention-deficit/hyperactivity disorder and other psychiatric comorbidities. *Neuroscience & Behavioral Reviews* 2018; 87: 255-270.
- [57] Hargreave E (2006). The neuroplasticity phenomenon of kindling. <http://hargreaves.swong.webfactional.com/kindle.htm>. (Accessed 5/19/18).
- [58] Begh M, Beghi E, and Cornaggia CM. Epilepsy in psychiatric disorders. In: Mula M. (eds) *Neuropsychiatric Symptoms of Epilepsy. Neuropsychiatric symptoms of neurological disease*. pp. 289-302. Springer, Cham Publishing Company, 2016. ISBN: 978-3-319-22158-8.
- [59] Josephson CB, Lowerison M, Vallerand I, et al. Association of depression and treated depression with epilepsy and seizure outcomes: a multicohort analysis. *JAMA Neurol* 2017; 74 (5): 533-539.
- [60] Binder MR. FLASH syndrome: tapping into the root of chronic illness. *AJCEM* 2020; 8 (6): 101-109.
- [61] Anderson JW, Lambert EA, Sari CI. Cognitive function, health-related quality of life, and symptoms of depression and anxiety sensitivity are impaired in patients with the postural orthostatic tachycardia syndrome (POTS). *Front Physiol* 2014; 5: 230.
- [62] Sender R, Fuchs S, Milo R. Revised estimates for the number of human and bacteria cells in the body. *PLoS Biology* 2016. <https://doi.org/10.1371/journal.pbio.1002533>.
- [63] Pockett S. The electromagnetic field theory of consciousness: A testable hypothesis about the characteristics of conscious as opposed to non-conscious fields. *Journal of Consciousness Studies* 2012; 19 (11-12): 191-223.
- [64] McFadden J. Synchronous firing and its influence on the brain's electromagnetic field: Evidence for an electromagnetic theory of consciousness. *JCS* 2002; 9 (4): 23-50.
- [65] McFadden J. Integrating information in the brain's EM field: The cemi field theory of consciousness. *Neuroscience of Consciousness* 2020; 2020 (1).