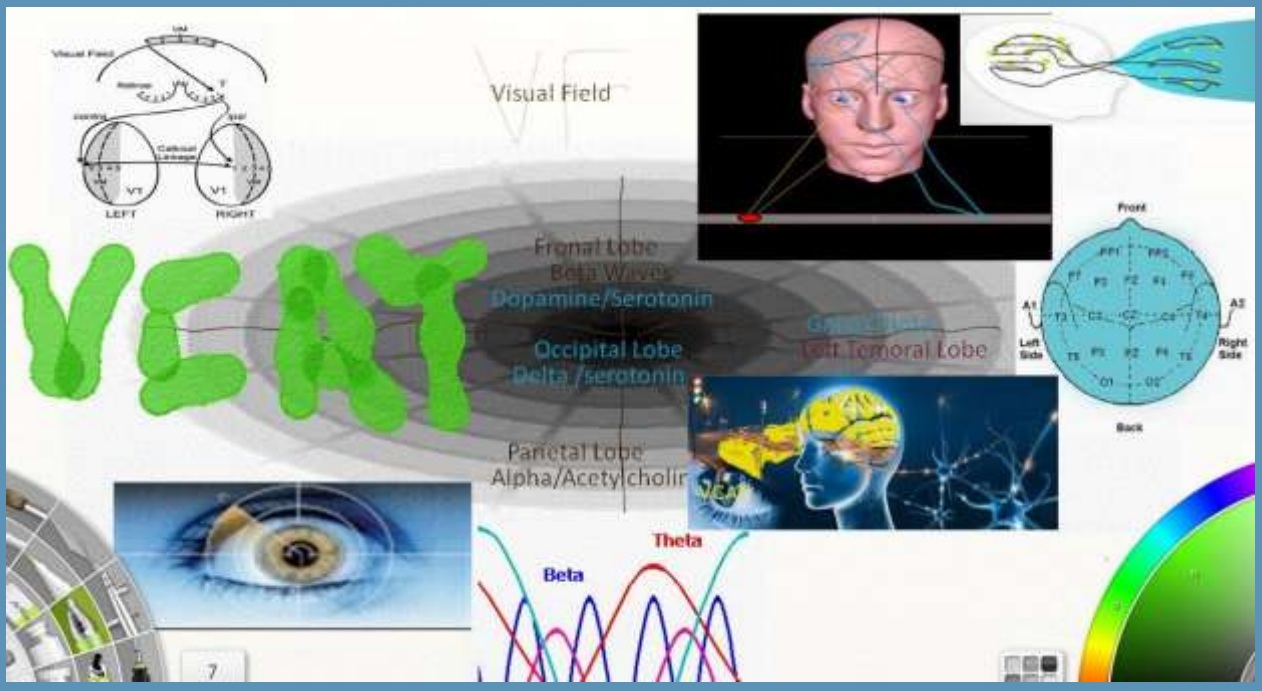
VCAT

Welcome to the World of Visual Stimulations and NueroPathway Therapy

Brain Functioning System Stimulated With Visual Concentration Attention Therapy (VCAT) a Neuropathway Therapy

By

Nader Siahdohoni, Ph.D.



The Science behind VCAT

Cognitive Neuroscience, with its concern about brain's chemical imbalance, perception, concentration, Attention, and memory will increasingly come to represent the central focus of psychological disorders and Neurosciences in the 21st Century

The world of psychology and mental health is constantly changing. The future of mental health now focuses more into an objective way of assessing and treating psychological disorders. It is more about how brain functioning system including neurons, neurotransmitters, and certain brain waves working together to enable the way we think, behave, move, feel, and maintain homeostasis. Recent research and studies have confirmed a strong relationship between brain's chemicals and psychological disorders. VCAT is the most effective neuropathway/neurocognitive treatment methodology in addressing and treating brain's neural network related dysfunction and its chemical imbalance. It is the most powerful solution for the millions of people with the life restricting symptoms. It is safe, fast, and has the major benefit of being drug free.

VCAT, Brain, and The Visual Field (VF)

Visual Concentration Attention Therapy (*VCAT*)

Idling, Balancing, Neuroplasticity, and Brain Performance

Visual Concentration Attention Therapy (VCAT) is developed and maintained by Nader Siahdohoni, PhD. VCAT is the absolute power in regulating brain's chemical imbalance. It is a sophisticated brain training and exerting technology with therapeutic approach designed to fit many different neural network related dysfunction in a very special way. It uses variety of patterns, strategies and techniques with sophisticated steps to empower its efficacy and validity (See VCAT-Treatment Plan). VCAT predicts that such steps will have an enormous impact in relieving neuropsychological as well as psychological related disorders. VCAT assumes that brain has the capability to self-train itself automatically after it is conditioned to the VCAT's treatment. Thus, the brain will automatically compute VCAT principles in real daily life practice. Visual Concentration Attention Therapy (VCAT) is the absolute power in regulating neural network dysfunction and brain's chemical imbalance caused by disease, substance abuse, or traumatic brain injuries. It is combined with Neurofeedback/ Brain Mapping Technology (wireless EEG/QEEG) to illustrate brain's activities on the computer screen while exercising VCAT (Real time Brain mapping and live monitoring of brain's performance). VCAT's treatment-plans consist of direct attentional stimulations to the brain within the visual field leading to improving symptoms of anxiety, depression, addictions, schizophrenia, ADHD, dementia, pre-Alzheimer's, seizures, and etc. According to the quantitative pre and post treatment research study conducted by Dr. Nader Siahdohoni on attention, concentration, and memory in individuals with ADHD as well as hundreds pilot studies, empirical and evidence-based-studies, programs such as VCAT could be an effective way in promoting efficient communication among the many nerve cells (neurons) and functional centers located throughout the brain and sensory motor system leading to improvement of attention, concentration, perception, cognition, and memory. VCAT improves brain function by producing higher activity and plasticity, increases blood circulation and the oxygen and glucose throughout the brain tissue, restoration, and the regeneration of neurons (Neurogenesis). It optimizes brainwave patterns that increase cognitive abilities and trains the brain to produce these activities on its own. Improved brain activities lead to memory improvement and sustained attention, which could also produce gains in focusing and concentration. These are the very mechanisms that

underlie psychological disorders such as ADD and ADHD, both of which could be improved by regular use of VCAT without the use of any medication. Exercising VCAT might delay the onset and progression of some neurodegenerative diseases such Alzheimer's disease. Unlike many neurocognitive and brain training instruments, VCAT -Family Tree represents a simple, yet comprehensive, synthesis of research by some of the
world's leading psychologists and neuroscientists into how the human brain works, and why people, who have similar backgrounds, intelligence, experience, skills, and knowledge, behave in very different ways. VCAT explains behavior in terms of the activities of the brain. How the brain organizes its billions of individual nerve cells to produce behavior, and how these cells are influenced by external visual stimulations through the visual field.

VCAT- Template

VCAT Template consist of scientific, research, and evidence based brain stimulations as follow:

1. Visual Field (VF)- Template (See "**The effect of VCAT's external Stimulations from the Visual Field to the Brain).**"

2. Build-in Eye/Object tracking, Attentional, Neural, and Memory tracking Stimulations within the VF on the VCAT-VF-Template (See VCAT Family Tree)

3. Build-in Neuropathways/Neurons stimulations to different parts of the brain based on the effected locations within the VCAT-VF-Template (See **Key Factors of the Effects of VCAT's Model).**

4. Build-in Adjustment Stimulation System within the VCAT-VF-Template to influence overall brain's Chemical imbalance by adjusting the Brain Waves and the Neurons' Firing Rate (See " **Brain Functioning System and Chemical Imbalance).**"

VCAT-Treatment Plan (TRP)

VCAT-TRP is based on therapeutic theories of attention, neural network, and memory track (see VCAT-Family Tree) within Visual Field (VF). It uses the science of Visual Field, Attentional Stimulation, and Neurocognitive Theory to create variety of therapeutic patterns that VCAT integrates with its sophisticated strategies to stimulate and enhance brain's functioning system accordingly depending on where and how the stimulations are penetrate/guided to the affected area of the brain from the visual field (See VF). VCAT-TRP starts with attentional track including sustained, divided, and selective attention, gaze cueing of attention, shifting attention, directions of attentional shifting (bottom-up and top-down), as well as feature integrated model of object

and space based attention including overt and covert attention and further continues with the neural and memory track including **s**ingle and multiple objet tracking and attention processing, crowding, single and multiple cognitive processing, and maximal cognitive tracking. VCAT-TRP consists of 4 progressive levels of treatment to precisely affecting brain's functioning system and its chemical imbalance by balancing related brain waves throughout the brain as well as rejuvenating and regulating brain's neural network functioning and connectivity.

How VCAT-TRP is developed

All of the following steps are conducted, compared, and collaborated in evaluation and creation of VCAT-Treatment plan.

1. A Clinical Interview
2. Mental Status Exam
3. Collateral Information
4. Psychological Testing
5. Brain Mapping Testing and Assessments
6. Brain mapping evaluation

VCAT Stages of Treatment Development and Progress

A. Plug-in / Diagnostic (Wireless EEG-Headset)

1. Brain Mapping Assessments
2. EEG Recording /Evaluation

B. Plug-in / VCAT- Stages of Treatment

Idling Phase

Coordination Stimulations (Eye, Head, Shift-Attentional Eye/Head Movements-L3.5)

NeuroPathway Visual Field Stimulations (VCAT- L1/L2)

Balancing Phase

Cellular Network Activation / Connection (VCAT- L2)

Cellular Network /Pathways Connection and Balance (VCAT- L2/L3)

Neuroplasticity/Performance Phase

Regulation Stage (VCAT-L4)

Rejuvenation Stage

C. Plug-in / Post Treatment Evaluation

D. Maintenance

A typical VCAT- session

There are three stages in each session

1. ***Resting Stage Brain Mapping,*** occurs when a subject is not performing an explicit task (VCAT) which can be used to evaluate regional interactions. Brain activity is present even in the absence of an externally prompted task; any brain region will have spontaneous fluctuations. The resting state approach is useful to explore the brain's functional organization as a pre-assessment and baseline for treatment.

2. ***VCAT Treatment Plan (TRP)*** is facilitated to address those mal functioning regions of the brain (Balancing the under active, over active, none active areas). Facilitating neural network action potential and enhancing functioning to the specific individual's stage of treatment (See- **B. Plug-in / VCAT- Stages of Treatment)**

3. ***Individual Feedback and Review*** of the recorded EEG, real time brain mapping, and the result of the VCAT-treatment leading to any possible changes in the performance of the brain in real time monitoring.

Key Factors of the Effects of VCAT's Model

Gaze Cueing of Attention

The eyes are the key to visual attention in cognitive search (Perrett, Hietanen, Oram, & Bensen, 1992). According to Perrett et al. (1992), gazing based on attentional cueing is important for interpretation and recognition of objects in the visual field. Such gazing influences the neural transaction including neural responses in the areas of the cortical region and superior temporal sulcus (STS; Perrett et al., 1992; Emery, 2000). For example, looking forward with eyes fixed on an object.

Attentional Gaze Cueing and Body Parts Movement

Gaze cueing combination of eyes and body parts such as head, and body position cues, or eyes and hands movement activate STS neural responses. These are assumed to be the central part of neural system in regard to collective perception (Pelphrey, Morris, Michelich, Allison, & McCarthy, 2005). For example, STS neural network will correspond to syndicate direction of head and gaze such as looking with the head forward to own moving hands and arms with eyes fixed on the fingers only. STS is also connected to the amygdala, a structure of the limbic system that is activated in emotional depressive circumstances (Thomas, Drevets, Whalen, Eccard, Dahl, Ryan et al., 2001). Damage to the amygdala lead to deficiency in judgment of gazing direction and the identification of facial manifestations of others (Young, Aggleton, Hellawell, Johnson, Broks, & Hanley, 1995).

Shifting Attention

Shifting attention from one object to another (object by object or object and object) relates strongly to parietal cortex, which is connected to STS and the reciprocal connections between STS and the intraparietal sulcus (IPS) (Rafal, 1996; Nobre, Sebestyen, Gitelman, Mesulam, Frackowiak, & Frith, 1997). According to Corbetta et al. (1991) and Nobre et al. (1997), through such connections data regarding the attentional covert/ overt shifting and the eye-gaze direction are being analyzed and processed for the proper response. (As shown in figure 11)

Directions of Attentional Shifting (Bottom-up and top-down)

Fixing the eyes on a target (Gaze Point) and voluntarily shifting the attention to any direction to a particular object within the visual field leads to controlled attention and cueing by top-down (endogenous) (Posner, 1980). Bottom-up (exogenous) happens, when attentional sifting from the gaze point is reflexive or stimulus driven.

Attentional controls such as bottom-up (exogenous) activated the neural network in posterior attention system involving subcortical structures such as the pulvinar and the superior colliculus (SC) (Rafal, Calabresi, Brennan, & Sciolto, 1989). Top-down (endogenous) is predicted to influence neural functioning in the cortical areas in anterior including cingulate gyrus and the supplementary motor area which are related to positive emotional feeling such as hope and expectancies (Carr, 1992; Corbetta et al., 1993), and posterior areas of the brain including intraparietal sulcus (IS) (Corbetta et al., 2000).

Crowding

Crowding is a well-documented spatial interference phenomenon (Levi, 2008; Pelli & Tillman, 2008) that is likely to affect object tracking, as it affects an observer's ability to select or individuate an object (Intriligator & Cavanagh, 2001). In the literature, crowding is typically used to refer to the spatial limits on a very particular task—identifying a single target in the presence of nearby stimuli. For such single-target identification tasks, the spatial range of crowding is rather well understood. Interference is absent or very small when objects are separated by more than half their eccentricity (Bouma, 1970). This is known as Bouma's Law, although it may not apply in the fovea, and systematic departures from it occur in the periphery (which are minor relative to the range of separations used here; Gurnsey, Roddy, & Chanab, 2011), so it should be considered more a heuristic than a law. As yet there have been no investigations of whether the range of spatial interference in tracking is the same as that of crowding. But some evidence suggests that attentional facilitation or interference may extend over a longer range than that documented in the

crowding literature.

Maximal cognitive tracking (deep stimulation)

It is widely accepted that visual attention can be shifted from one location to another independently of eye movements, and that the processing of stimuli appearing at attended locations is enhanced. The methodological paradigm that produced much of the evidence for this is the attentional cueing procedure (Posner, Snyder, & Davi dson, 1980). In a cueing experiment, visual attention is shifted to a predetermined location either endogenously, in which the shift is under the volition of the observer, or exogenously, in which the shift is involuntarily elicited by a highly salient cue.

Feature integration theory (Treisman)

This is a theory for explaining which patterns are easily discriminable and why. The idea is that the front-end of the visual system breaks the stimulus down into its constituent parts. It separately analyzes each local patch of the visual field to determine: pattern; motion; shape (depth and size); color, etc. Attention is the glue in feature integration theory. Example: seeing red is preattentive, and seeing rightward motion is preattentive, but seeing a red thing moving to the right requires attention to connect the red thing with the moving thing. The theory does not go so far as to say *how* attention accomplishes this gluing. Feature integration theory is based in part on the functional specialization hypothesis, e.g., that "red" and "moving rightward" are represented in separate visual brain centers. One needs attention to bring those separate neuronal representations together.

Overt attention

This is the most direct way to shift attention, called overt attention.

Covert attention

You can also shift attention without eye/head movements to "filter out" unattended locations.

Resting Potentials and Action Potentials

Resting Potentials (brain at rest) happens when neurons are firing in the resting phase without any cognitive challenge, concentration, and or brain stimulation. This phase is used for pre and post assessing brain function before and after VCAT treatment.

Action potential is a short-lasting event in which the electrical membrane potential of a cell rapidly rises and falls, following a direct stimulation to the brain (VCAT). Action potential's main function is to activate intracellular processes. Action potentials in neurons are also known as

"**nerve impulses**" or "spikes", and the temporal sequence of action potentials generated by a neuron is called its "**spike train**." A neuron that emits an action potential is often said to "fire." Action potential relates directly to the release of Brains' neurotransmitters and endorphins.

In other words, when a neuron sends a signal down it's axon to communicate with another neuron; this is called an action potential (firing). The electrical impulse carried by the action potential must trigger the release of neurotransmitter into the synaptic gap. Neurotransmitters are the chemical messengers that carry the message from one neuron to the next. If this does not happen and the neuron does not reach the firing phase (action potential) to complete the transfer of neurotransmitters, such neuron goes to misfiring phase.

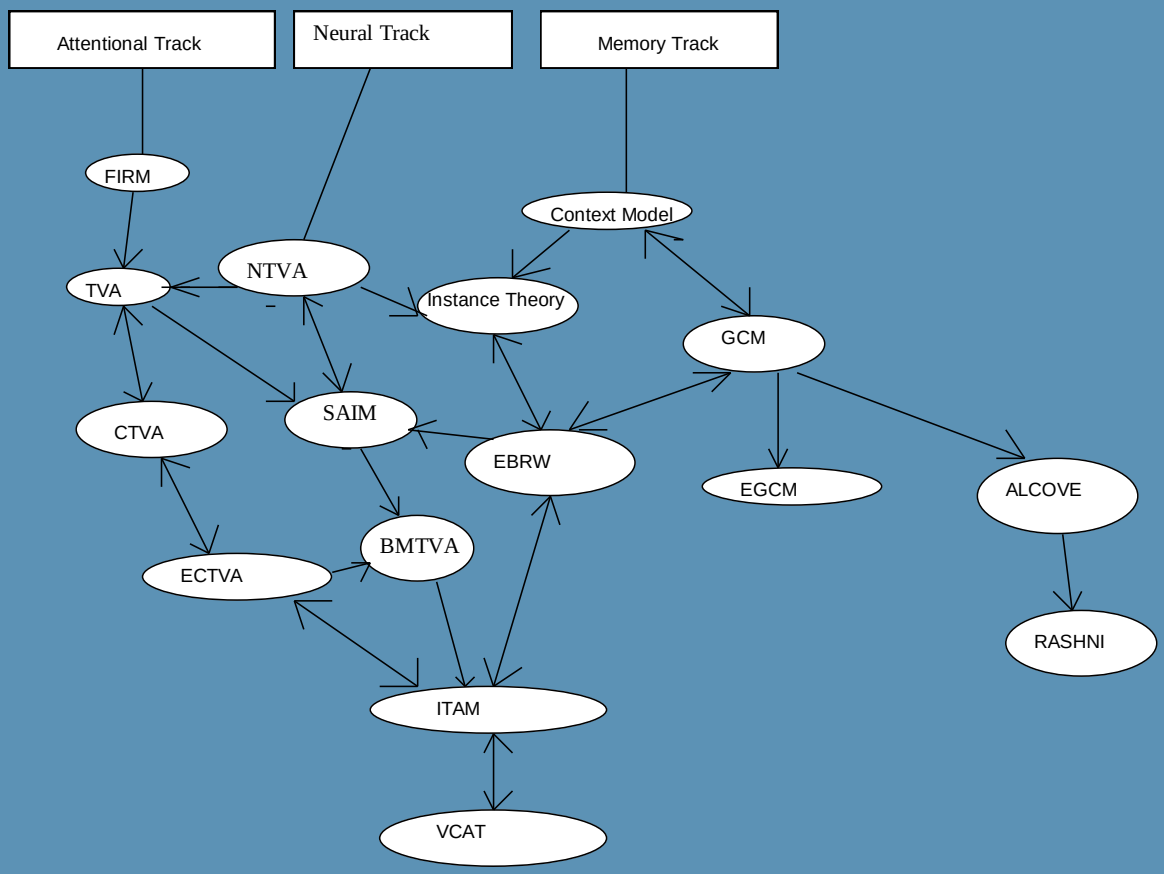
What is Brain Plasticity?

The brain is one of the largest and most complex organs in the human body. It is made up of more than 100 billion nerves (neurons) that communicate in trillions of connections called synapses. The brain is a highly organized organ that serves as the center for all of one's mental, behavioral, emotional, and cognitive functioning including the overall psychological wellbeing. It contains billions of nerve cells arranged in patterns that coordinate thought, emotion, behavior, movement and sensation. A complicated highway system of nerves connects the brain to the rest of your body, so communication can occur in split seconds. The neurons communicate with one another by means of long protoplasmic fibers called axons, which carry trains of signal pulses called action potentials (firing neurons when stimulate) to distant parts of the brain or body targeting specific recipient cells. The level of stimulation that a neuron must receive to reach action potential is known as the threshold of excitation, in which the brain discharges certain chemicals (neurotransmitters) to facilitate a proper communication between different networks of neurons. When a neuron is not able to reach such threshold it goes to misfiring stage. By discharging the right amount of neurotransmitters brain creates a balanced harmonized environment for itself to regulated, process, and function accordingly with an increased plasticity. Neuroplasticity is the brain's ability to reorganize, repair, and re-wire itself by forming new neural connections. Neuroplasticity allows the neurons (nerve cells) in the brain to compensate for injury and disease and to adjust their activities in response to new situations or to changes in their

environment. It also regulates and improves both the gray matter (facilitates nerve connections and processing) along with white matter, which acts as the information highway of the brain to speed the connections between distant parts of the brain and body.

Healthy people possess levels of brain chemicals that lie within a certain normal range. When internal or external factors either deplete the brain of these chemicals or stimulate it to produce excess amounts, a chemical imbalance occurs. Imbalances in the brain's chemistry can cause psychiatric conditions such mood disorders, learning disabilities, and substance abuse.

VCAT's Family Tree



Theories linked together by bidirectional arrows are identical to each other and to VCAT

The attentional track is started with fixed-capacity independent race model (FIRM) (Bundesen, 1987; Bundesen, Pedersen & Larsen, 1984; Shibuya & Bundesen, 1988). FIRM relates to

functioning and presentation of partial and whole report tasks, which is changed by Bundesen (1990, 1998a, 1998b; Bundesen & Harms, 1999) in order to develop the theory of visual attention (TVA). TVA consists of wide varieties of attentional tasks such as feature search, cueing, single-item identification, and priority learning. Distance and grouping effects in flanker tasks and feature-and conjunction-search tasks are related to each other and they should be connected; therefore, TVA and the theory of perceptual grouping by proximity [COntour DEtector (CODE) (Van Oeffelen & Vos, 1982; 1983)] are combined to the CODE theory of visual attention (CTVA) (Logan, 1996; Logan & Bundesen, 1996). The theory of executive control of TVA (ECTVA) (Logan & Gordon 2001) relates to crosstalk and set-switching effects in dual-task situations. The instance theory of attention and memory (ITAM) (Logan & Gordon, 2002) is the integration of the previously explored theories of attention and memory. VCAT is related to different outlooks in attentional concepts and appearance in the visual field.

The memory track is started with the context model of classification. There is the recognized pattern of arrangements (Medin & Schaffer, 1978), which was improved by Nosofsky (1984) in regard to similarity in objects to establish generalized context model (GCM). GCM provides precise collections of categorized data such as identification categorization and recognition categorization. Exemplar-based random-walk model (EBRW) (Nosofsky & Palmeri, 1997) is the combination of GCM and ITAM, which includes categorization, precision, and perception and learning configurations such as automatization.

VCAT is similar to ITAM in that both are models of attention and attentional selectivity functioning theory based on visual cognition. VCAT also resembles ITAM in utilizing the constancies of previous theories in performance, selection, categorization, and memory concepts. VCAT unlike ITAM covers attention in dimensions based of attentional weights (Logan & Gordon, 2002). Similarity between targeted objects and their distractors are sustained as the most important basis for the formal attentional theories, but not in VCAT's concept. VCAT supports all kind of different objects and images with various shapes, size, color, and space position. Memory track does not reveal attention to objects, but it concerns the theory of classification and dimension, theories of learning , theory of similarity, and similarities influence on categorization performance (Logan & Gordon, 2002). The most priority in VCAT is the attention and the shift of attention to and from the objects in a display by gazing, concentrating, and shifting on one or multiple objects in visual field as partial or whole.

The Neural track relates to both memory and the attentional track, since it stimulates the neural network through attentional stimulus to generate memory (Bundesen, 1990). VCAT shares many similarities to the Neural Theory of Visual Attention (NTVA) (Bundesen, 1990), which is an extended theory of TVA. It relates to attentional selectivity stimulus and its influence to neural

activity, the mind, and the behavior (Bundesen). According to Bundesen NTVA affects both psychological and neural interactions. NTVA includes two selections: filtering (selection of objects) and pigeonholing (selection of features). Filtering affects the individual's cortical neurons, (where objects are analyzed) and pigeonholing happens in encoding and interpreting of selected features in a high level of neural activities (Bundesen).

The Brain

The brain is one of the largest and most complex organs in the human body. It is made up of more than 100 billion nerves (neurons) that communicate in trillions of connections called synapses. The brain is a highly organized organ that serves as the center for all of one's mental, behavioral, emotional, and cognitive functioning including the overall psychological wellbeing. It contains billions of nerve cells arranged in patterns that coordinate thought, emotion, behavior, movement and sensation. A complicated highway system of nerves connects the brain to the rest of your body, so communication can occur in split seconds. The neurons communicate with one another by means of long protoplasmic fibers called axons, which carry trains of signal pulses called action potentials (firing neurons when stimulate) to distant parts of the brain or body targeting specific recipient cells. The level of stimulation that a neuron must receive to reach action potential is known as the threshold of excitation, in which the brain discharges certain chemicals (neurotransmitters) to facilitate a proper communication between different networks of neurons. When a neuron is not able to reach such threshold it goes to misfiring stage. By discharging the right amount of neurotransmitters brain creates a balanced harmonized environment for itself to regulated, process, and function accordingly with an increased plasticity. Neuroplasticity is the brain's ability to reorganize, repair, and re-wire itself by forming new neural connections. Neuroplasticity allows the neurons (nerve cells) in the brain to compensate for injury and disease and to adjust their activities in response to new situations or to changes in their environment. It also regulates and improves both the gray matter (facilitates nerve connections and processing) along with white matter, which acts as the information highway of the brain to speed

the connections between distant parts of the brain and body.

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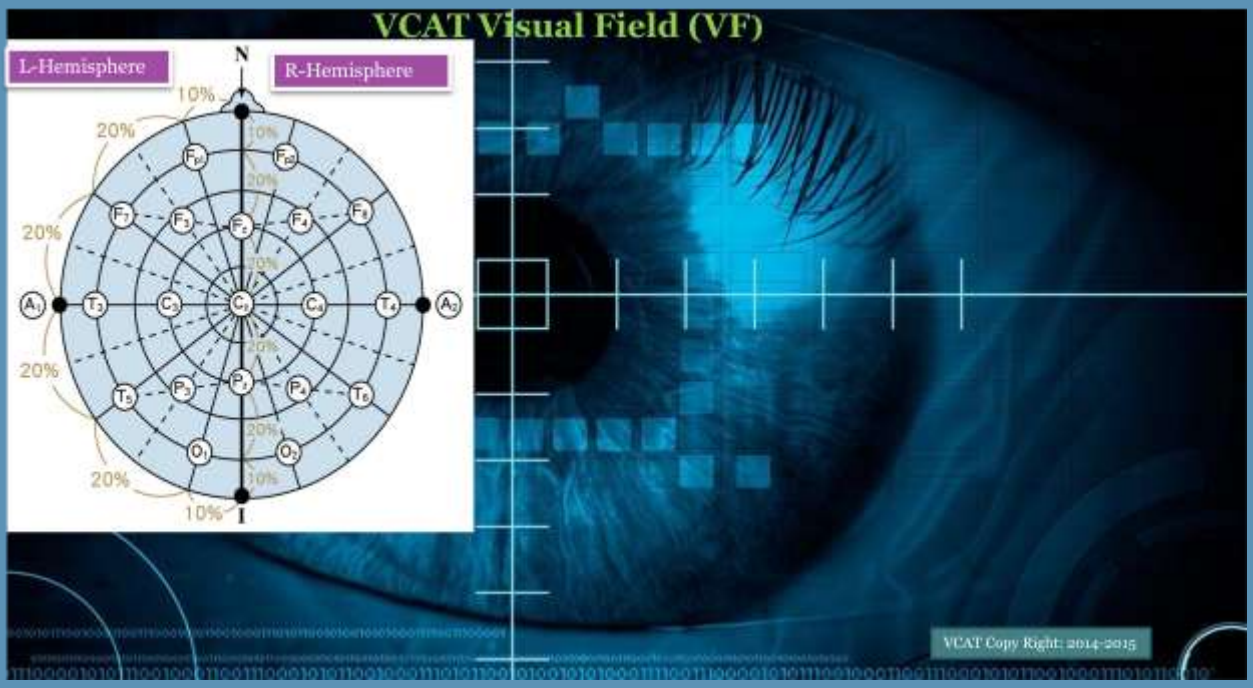
Visual Field (VF)

The eyes are an extension of the brain and the Visual Field (VF) is the reflection of brain. The visual system including the visual field are intimately connected with the nervous system and endocrine system by way of the eyes which through the light receptors of the retinas send electrical messages not only to the visual cortex but also to the hypothalamus and pineal glands. The light coming in through the eyes can augment the function of the nervous system and the endocrine system and put the brain and body into balance. Published scientific and clinical studies have reported visual stimulations to be effective in inducing relaxation, reducing stress, reducing blood pressure, relieving tension headache, managing chronic pain, relieving insomnia, and reducing the discomfort of premenstrual syndrome.

Visual stimulation means are currently being used by professional psychologists in their practices and by the general public for relaxation, stress management, Insomnia, mind expansion, accelerated learning and retention, breaking limiting beliefs, phobias, anxiety, sports training, promoting physical wellness.

Visual areas of the brain are capable of reacting to impulse going from the retina. To improve the eye-sight and mental health, it is necessary to activate corresponding areas of the brain. If some sectors of the brain have been damaged, or their work impaired, electric signals don’t go through such damaged ways, and therefore, some areas of the brain may not function. In some cases, brain energy massage may help restore normal functioning of the various sectors of the brain by means of the visual stimulation of the brain.

The effect of VCAT's external Stimulations from the Visual Field to the Brain



Eye exercises and brain trainings work on two factors to improve eyesight and brain. Firstly, it is training the muscles in the eye. Secondly, it is training the various areas of the brain.

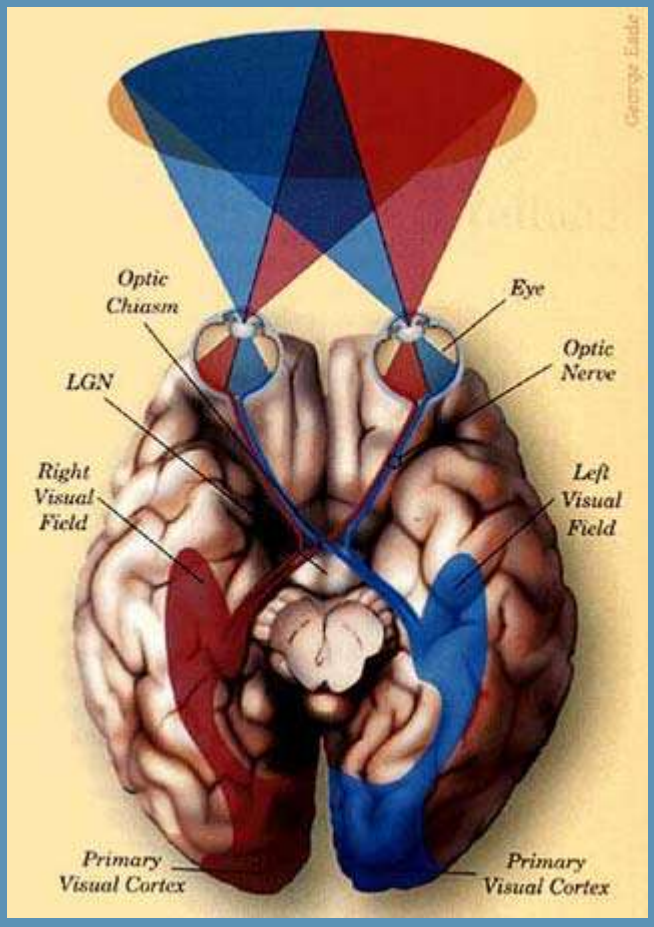
Visual areas of the brain are capable of reacting to impulse going from the retina. To improve the eyesight and mental health, it is necessary to activate corresponding areas of the brain. If some sectors of the brain have been damaged, or their work impaired, electric signals don't go through such damaged ways, and therefore, some areas of the brain may not function. Such kind of condition cannot disappear by itself. In some cases, training the visual areas of the brain may help restore normal functioning of the various sectors of the brain. Brain visual stimulation can be used for improve memory, attention, concentration, executive functions, decision-making, mental flexibility, and other core capabilities. Like physical exercises, brain training can be based by various challenging activities such as special training based on visual stimulation of the brain. Recently research shows that brain stimulation can help prevent age-related cognitive decline, reverse behavioral assessment declines in dementia and Alzheimer's, and can also improve normally functioning minds.

According to the study conducted by Gandhi, Heeger and Boynton (1998), external attentional stimulus coming from the right or the left half of an exhibit would **af**fect the brain activity in

visual cortex in the way that the stimulus coming from the left side would be processed by the neurons in the right hemisphere and those from the right side would be process by the neurons in

the left hemisphere.

In addition, the right side of the brain controls muscles on the left side of the body and the left side of the brain controls muscles on the right side of the body. Also, in general, sensory information from the left side of the body crosses over to the right side of the brain and information from the right side of the body crosses over to the left side of the brain. Therefore, damage to one side of the brain will affect the opposite side of the body. VCAT's diagnostic assessments are developed to determine the under active brain functioning in cases such as the Traumatic brain Injuries (TBI) and stroke.

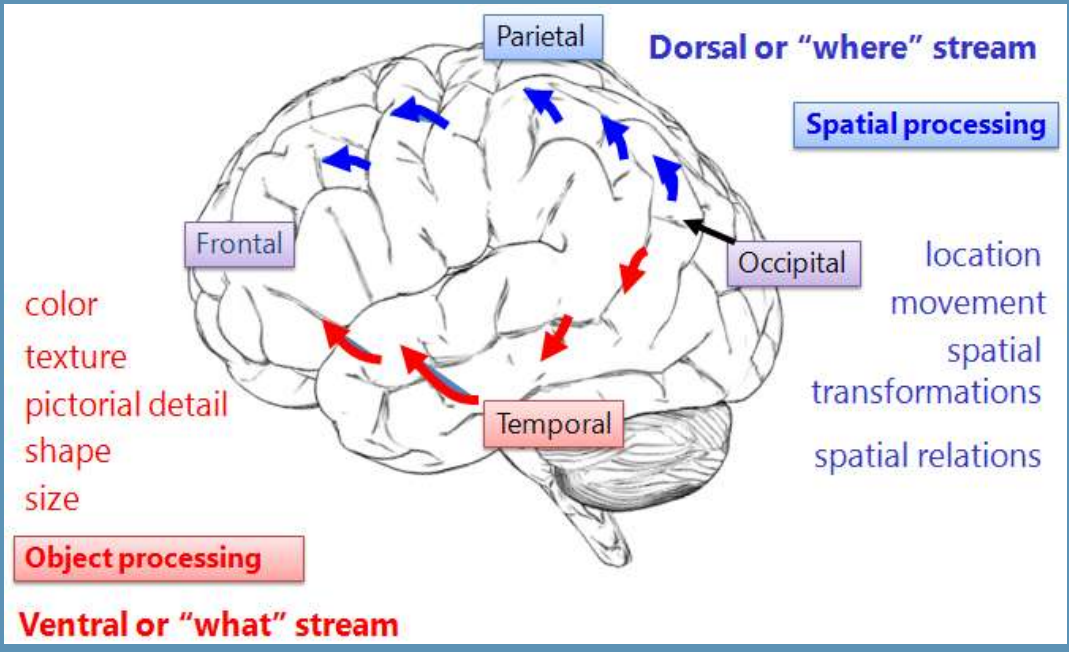


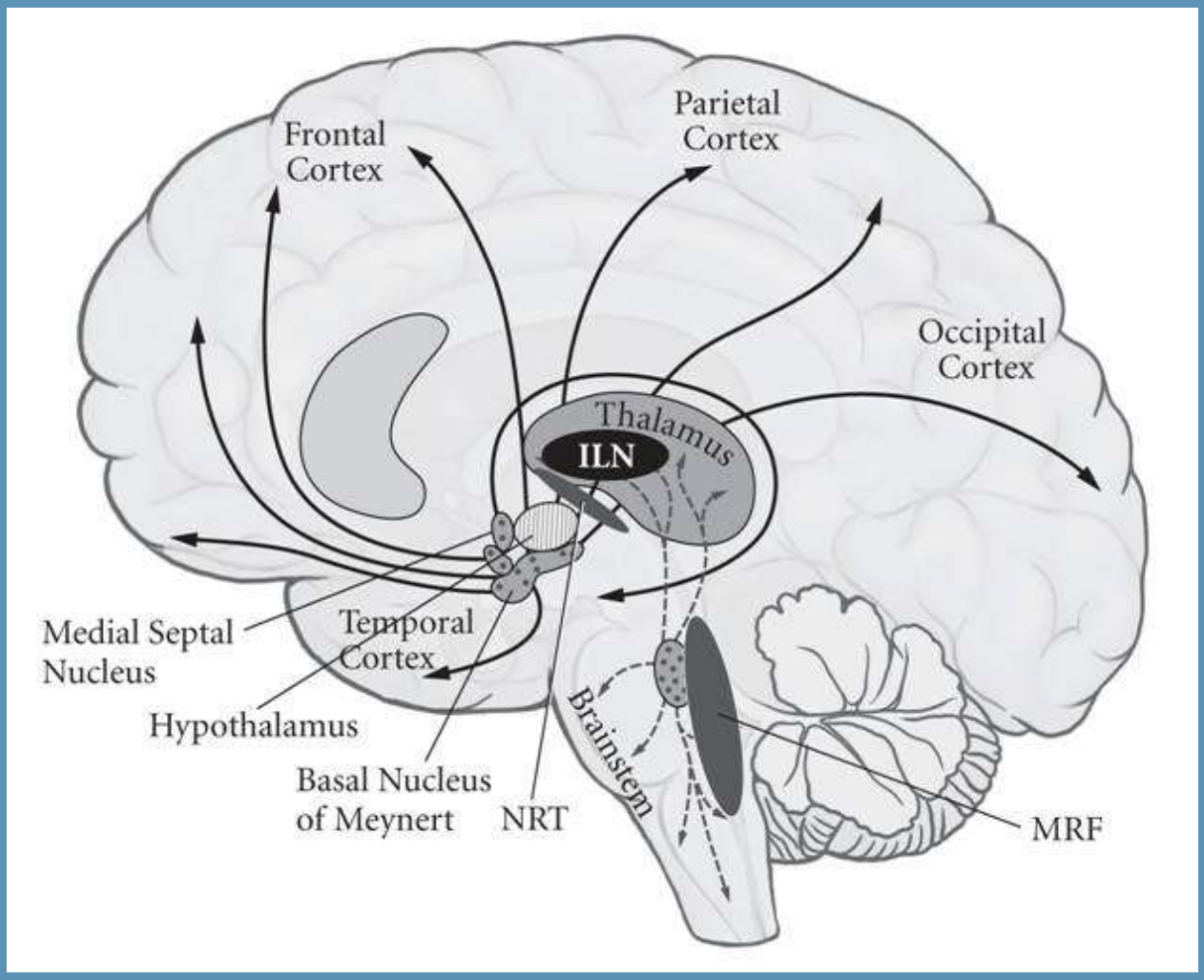
Visual Processing in the Brain

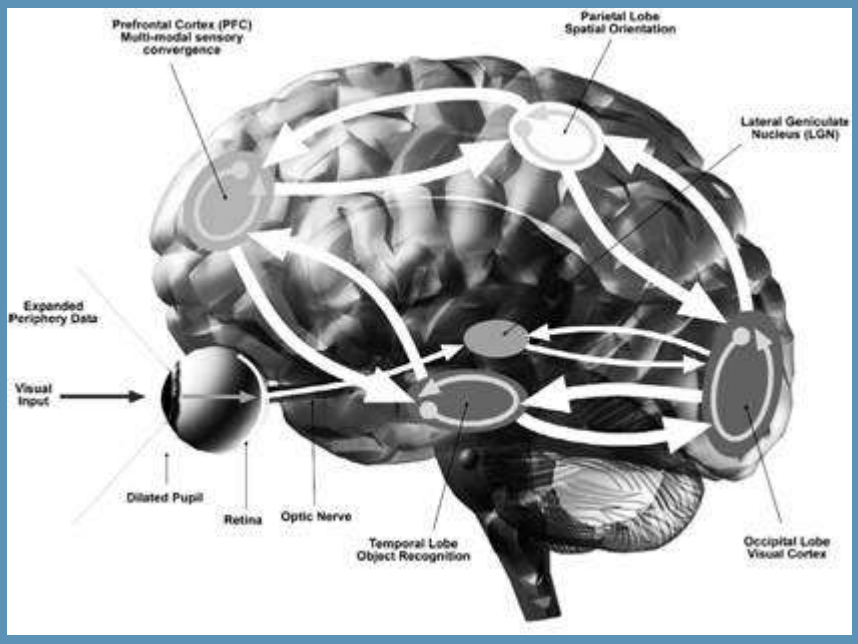
After being processed in the thalamus and different areas of the brain, visual signals eventually reach the primary visual cortex in the occipital lobe of the brain's cerebrum. In the 1960s, David Hubel and Torsten Wiesel demonstrated that highly specialized cells called **feature detectors** respond to these visual signals in the primary visual cortex. Feature detectors are neurons that respond to specific features of the environment, such as lines and edges.

From the visual cortex, visual signals often travel on to other parts of the brain, where more processing occurs. Cells deeper down the visual processing pathway are even more specialized than those in the visual cortex. Psychologists theorize that perception occurs when a large number of neurons in different parts of the brain activate. These neurons may respond to various features of

the perceived object such as edges, angles, shapes, movement, brightness, and texture.




Frontal Cortex
Parietal Cortex
Occipital Cortex
Thalamus
ILN
Medial Septal Nucleus
Temporal Cortex
Hypothalamus
Basal Nucleus of Meynert
NRT
Brainstem
MRF


Prefrontal Cortex (PFC)
Multi-modal sensory convergence
Parietal Lobe
Spatial Orientation
Lateral Geniculate Nucleus (LGN)
Expanded Periphery Data
Visual Input
Dilated Pupil
Retina
Optic Nerve
Temporal Lobe
Object Recognition
Occipital Lobe
Visual Cortex

As is quite well-known, the brain is split into two roughly similar **hemispheres**, separated by the deep longitudinal fissure. Also well-known is that the brain is **"cross-wired"**, with the **left hemisphere** controlling movement on the **right side of the body**, and the **right hemisphere** controlling the **left side of the body**. Most, but not all, of the different structures, lobes and organs of the brain have a left and right hemisphere element, and **communication between the hemispheres** is achieved by means of a thick bundle of nerve tissues known as the **corpus callosum**, which effectively makes a full brain out of two half-brains (Ref).

The two hemispheres of the cerebral cortex are linked by the corpus callosum, through which they communicate and coordinate actions and decisions. Communication and coordination between the two hemispheres is essential because each hemisphere has some separate functions.[4] The right hemisphere of the cortex excels at nonverbal and spatial tasks, whereas the left hemisphere is more dominant in verbal tasks, such as speaking and writing. The right hemisphere controls the primary sensory functions of the left side of the body. In a cognitive sense the right hemisphere is responsible for recognition of objects, timing and in an emotional sense it is responsible for empathy, humor and depression. On the other hand, the left hemisphere controls the primary sensory functions of the right side of the body and is responsible for scientific and math skills, and logic.[5] The extent of specialized brain function by an area remains under investigation. It is claimed that the difference between the two hemispheres is that the left hemisphere is "analytic" or "logical" while the right hemisphere is "holistic" or "intuitive." [6] Many simple tasks, especially comprehension of inputs, require functions that are specific to both the right and left hemispheres and together form a one direction systematized way of creating an output through the communication and coordination that occurs between hemispheres.[7]

Feedback circuits in visual perception pathways. Sensory signal in human consciousness projects up and around the brain like a wave as consciousness arises in distinct stages. All visual data and stimulations coming from Visual Field (VCAT) are fed into the thalamus (through the LGN for visual pathways), which filters and routes those incoming VF data into higher areas of the cortex for processing. Recognition happens quickly and instinctively in the thalamus and medial temporal lobe, focusing attention on salient information. Memory identifies incoming signal moving upward in the cortex, passing data through parallel layers of spatial and object recognition along dorsal and ventral pathways. Multisensory perception finally converges in the pre-frontal cortex as a parsed reconstruction of reality is presented for informing real-time behaviors.

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4. **Jump up^** Witelson, Sandra F; Wazir Pallie (1973). "Left Hemisphere Specialization for Language in the Newborn". *Neuroanatomical Evidence of Asymmetry* **96** (3): 641–646. doi:10.1093/brain/96.3.641. Retrieved 28 April 2014.
5. **Jump up^** http://bama.ua.edu/~st497/pdf/rightorleftbrain.pdf
6. **Jump up^** Borod, Joan; Fani Andelman; Loraine K Obler; James Tweedy; Joan Wilkowitz (1992). "Right Hemisphere Specialization for the Identification of Emotional Words and Sentences". *Neuropsychologia* **30** (9): 827–844.doi:10.1016/0028-3932(92)90086-2.
7. **Jump up^** O'Shea R. P. (2003). "Binocular rivalry in split-brain observers". *Journal of Vision* **3**: 610–615.
8. **Jump up^** Mooshagian, Eric (2008). "Anatomy of the Corpus Callosum reveals its Function". *Journal of Neuroscience* **28** (7): 1535–1536.doi:10.1523/JNEUROSCI.5426-07.2008

Brain Conditioning to Self-Training and Automatically Functioning

VCAT assumes that brain has the capability to self-train itself automatically after it is conditioned to the VCAT's steps. Thus, the brain will automatically compute VCAT principles in real daily life practice. According to the studies conducted in sustained automatic attention the basal forebrain cholinergic function, amygdala central nucleus (CEA), the magnocellular cholinergic neurons of the sublenticular substantia innominata/nucleus basalis (SI/nBM), and the posterior parietal cortex (PPC) were recognized as important factors for sustained automatic attention in the presentation of a precise selective attention method (Pearce & Hall, 1980; Holland, Han, & Gallagher, 2000).

Clinical Evidence for VCAT

Thomas et al. (2001) stated that attentional shifting and eye-gaze cueing affect the neural network associated with the limbic system and damages in this area especially to amygdala will cause deficiency in neural transaction. Damage in this region has been determined to be the cause of emotional related psychological disorders (Mulrow, Williams, & Trivedi et al., 1998).

According to Vuilleumier (2002), proper functioning of neural system plays a significant role in eye-gaze cueing. Vuilleumier presented evidence of patients with damages to tempor oparietal area of the right hemisphere suffering from attentional discrepancy for computing stimulations reflected to the left (contralesional) side of the visual field (unilateral visual neglect). Vuilleumier also provided evidence that ignorance to such effect will lead to a total blindness in the left side of visual field. Eye-gaze cueing and attentional shifting exercises have shown tremendous improvements in patients with visual neglect disorders.

Cognitive functioning such as thinking, computing, learning, ability in memorizing, and multi tasking are components that strongly depend on an efficient working memory (WM) (Keefe, 2000). According to Keefe, destruction to temporal and frontal lobes is the cause of cognitive decline by not being able to use the resources of the WM, which may lead to psychological disorders such as schizophrenia (SC). Patients with SC have shown deficiency in cognitive processing that has been related to impairment of WM and attentional functioning (Barch, 2005; Zubin, 1975). Attention is suggested to be vital in selection and data transfer of cognitive related tasks in to WM and the effective use of WM is positively correlated with efficient use of attention (Zubin). Selective attention is predicted to be guidance in WM encoding, which is investigated by Sperling (1960) and Averbach and Coriel (1961) in their experiments with iconic memory. These researchers established that attentional shifting cues affect the retained data from short visual exhibits. Also recent studies on top-down and bottom-up cues predicted that attentional selectivity affects the data representation and encoding in WM (Schmidt, Vogel, Woodman, & Luck, 2002; Woodman, Vecera & Luck, 2003). Further, the study conducted by Gold, Wilk, McMahon, Buchanan, and Luck (2003), showed congregate proof that attentional selectivity's stimulus can be used by patients with SC to guide WM encoding.

Brain Functioning System and Chemical Imbalance

Healthy people possess levels of brain chemicals that lie within a certain normal range. When internal or external factors either deplete the brain of these chemicals or stimulate it to produce excess amounts, a chemical imbalance occurs. Imbalances in the brain's chemistry can give rise to mood disorders, learning disabilities, substance abuse and muscle weakness. The most easily noticeable imbalances in the brain's chemistry are imbalances in neurotransmitters that function as stimulants or inhibitors in the neurological system.

For example:

Depression

Depression is a symptom of an imbalance in the brain's chemistry. In depression, the brain levels of the well-being hormone serotonin, the reward hormone dopamine and the neurological inhibitor chemical GABA is lower than normal.

Anxiety

Anxiety and panic disorder, too, can be symptoms of an imbalance in the brain's chemistry. Anxiety is attributed to low levels of serotonin and GABA and high levels of stress hormones such as cortisol.

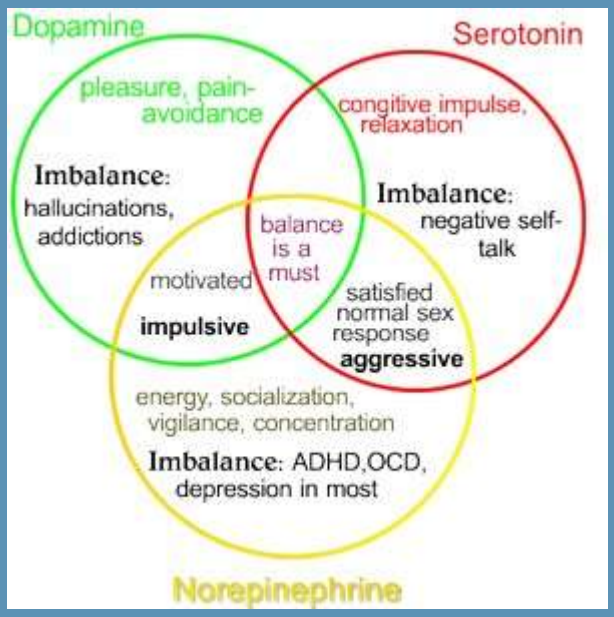
Alcoholism

Alcohol is a temporary depressant of the neurological system. At the time of drinking, alcohol stimulates the secretion of the inhibitory molecule GABA. However, excessive amounts of alcohol can deplete the brain's natural resources of GABA, serotonin, dopamine and opioid peptides. Alcohol furthermore triggers the release of stress chemicals that create depression, distress and tension. This asymmetry between the short-term calming effects and long-term distressing effects of alcohol may tempt alcoholics into relapse.

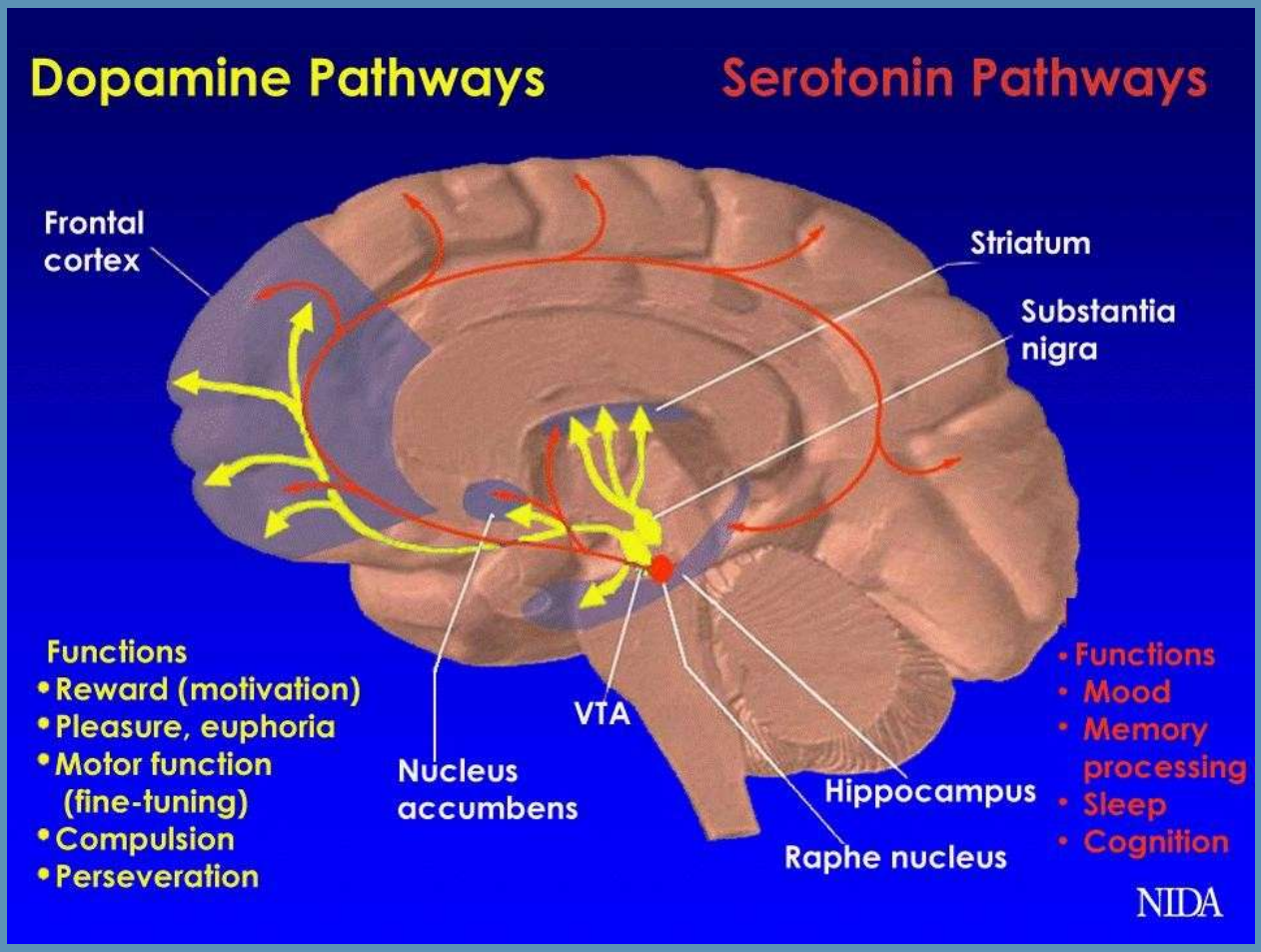
Attention Deficit Hyperactivity Disorder

People with attention deficit hyperactivity disorder may have low levels of dopamine markers, reports a research team in the September 9, 2009, issue of the "Journal of the American Medical Association." Dopamine is released in response to exciting activity and gives rise to a feeling of pleasure or reward, which can encourage a person to engage in the same kind of activity again. The researchers speculate that the low levels of dopamine in people with ADHD may explain their lack of motivation and inability to focus.

The Most prominent Chemicals in Brain



Dopamine and Serotonin Pathways within Brains' Processing System



Dopamine Pathways affecting Brains' Functioning System

In the brain, dopamine functions as a neurotransmitter—a chemical released by nerve cells to send signals to other nerve cells. The brain includes several distinct dopamine systems, one of which plays a major role in reward-motivated behavior. Most types of reward increase the level of dopamine in the brain, and a variety of addictive drugs increase dopamine neuronal activity. Other brain dopamine systems are involved in motor control and in controlling the release of several other important hormones.

Several important diseases of the nervous system are associated with dysfunctions of the dopamine system. Parkinson's disease, a degenerative condition causing tremor and motor impairment, has been related to the loss of dopamine-secreting neurons in the midbrain area called the substantia nigra. There is evidence that schizophrenia involves highly altered levels of dopamine activity, and the antipsychotic drugs that are frequently used to treat it have a primary effect of attenuating dopamine activity. Attention deficit hyperactivity disorder (ADHD) and restless legs syndrome (RLS) are also believed to be associated with decreased dopamine activity.

Serotonin affecting Brains' Functioning System

In the brain, serotonin is involved in regulating several important functions including sleep, appetite, feeding, and body weight. Abnormal serotonin levels are also associated with problems such as suicidal tendency, obsessive compulsive disorder, alcoholism and anxiety.
Serotonin (5-hydroxytryptamine, 5-HT) is a neurotransmitter that is involved in just about everything. It helps ensure proper cell growth, maturation and migration during development. Serotonin is also important in regulating emotions, cognitive functions, appetite, pain, circadian rhythms, and our endocrine system in adulthood.

The Term “NeuroMax”

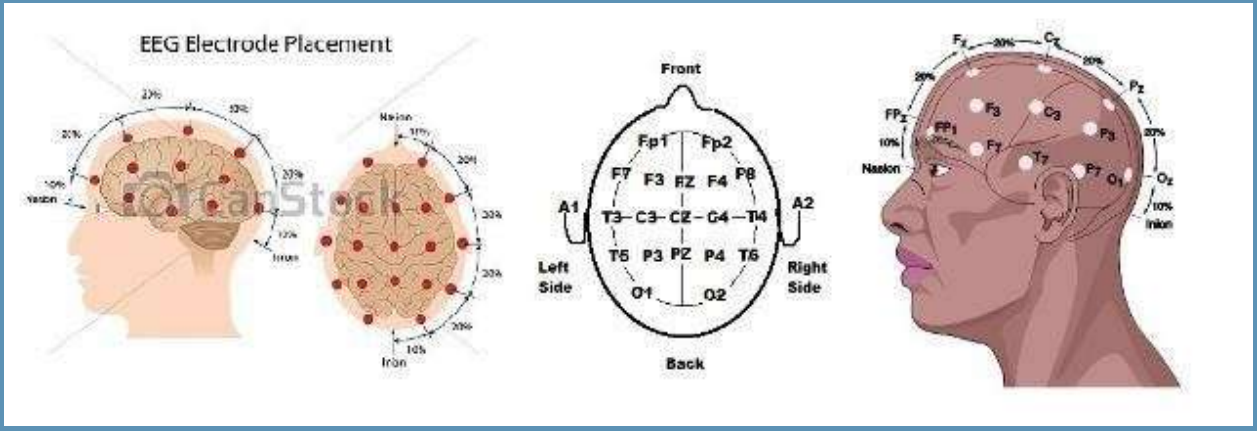
The term “NeuroMax” is used by Dr. Nader Siahdohoni for the Integrated Cognitive Neurfeedback therapy for VCAT in combination with Electroencephalography (EEG, neurofeedback technology and brain mapping). The Neurofeedback technology and brain mapping is used to screen the effectiveness of VCAT in enhancing brian’s functioning system by stimulating the brain’s electrical activities (Neurons firing rates and brain waves). Electroencephalography (EEG) in combination with VCAT (NeuroMax) is an optimal treatment approach for Neural Network Disarray. NeuroMax (EEG/VCAT) is a powerful method for training poorly regulated brainwave activity. While looking at VCAT displays on a computer monitor, you learn to control the activity of the brain for effective learning. The prefrontal cortex is involved with executive functions such as short-term attention and organizing; the temporal lobes are involved with memory functions, auditory processing, word finding and emotional responses and the parietal lobes are involved with direction. Visual Concentration Attention Technology (VCAT) is a unique system of brain exercises using sophisticated

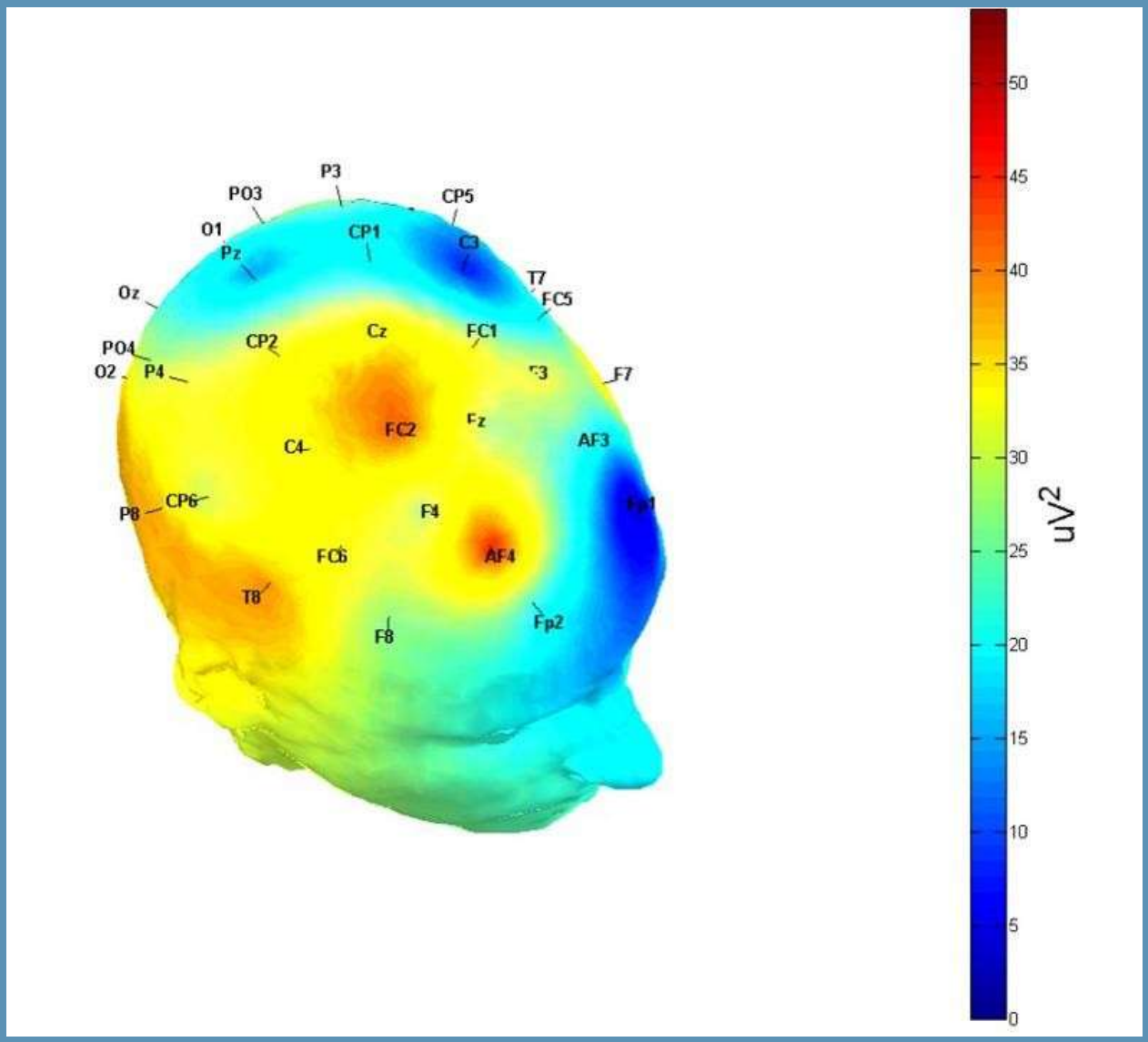
attentional visual stimulations within the visual field to affect the brain's functioning system by enhancing its neural activities, leading to improvement of perception, cognitive, memory, attention, focusing and concentration. The Neurofeedback technology is used to screen the effectiveness of VCAT in enhancing brain's functioning system by stimulating the brain's electrical activities (Neurons firing rates and brain waves).

Brain Mapping Using Wireless
EEG/QEEG

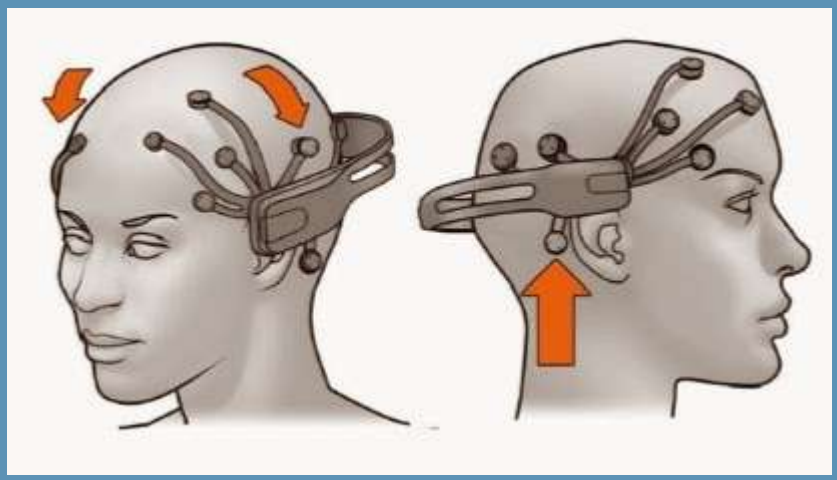
The brain is a highly complex organ made up of billions of cells called neurons. Neurons send and receive messages to and from all parts of your body. These messages are electrical impulses that create brain waves. The brain map (also called a neuro map) is an important tool that is being used to evaluate brainwaves and identify opportunities to improve communication between various regions of the brain using international 10-20 system. The brain map is able to capture a window of brain activity, analyze the data, and create a visual representation for each lobe of the brain and each specific brain wave (Delta, Theta, Alpha, Beta).

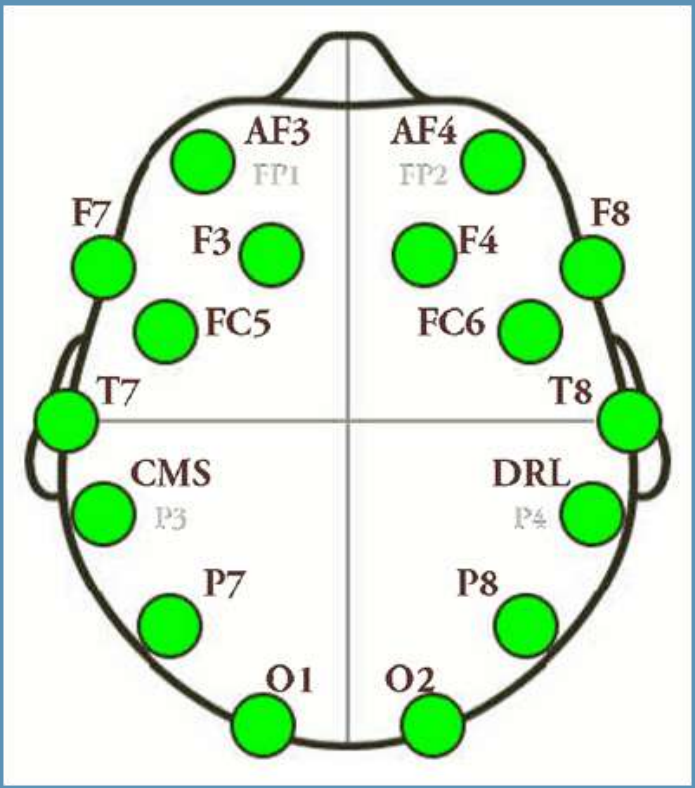
International 10-20 System





Emotiv (EEG-Headset)



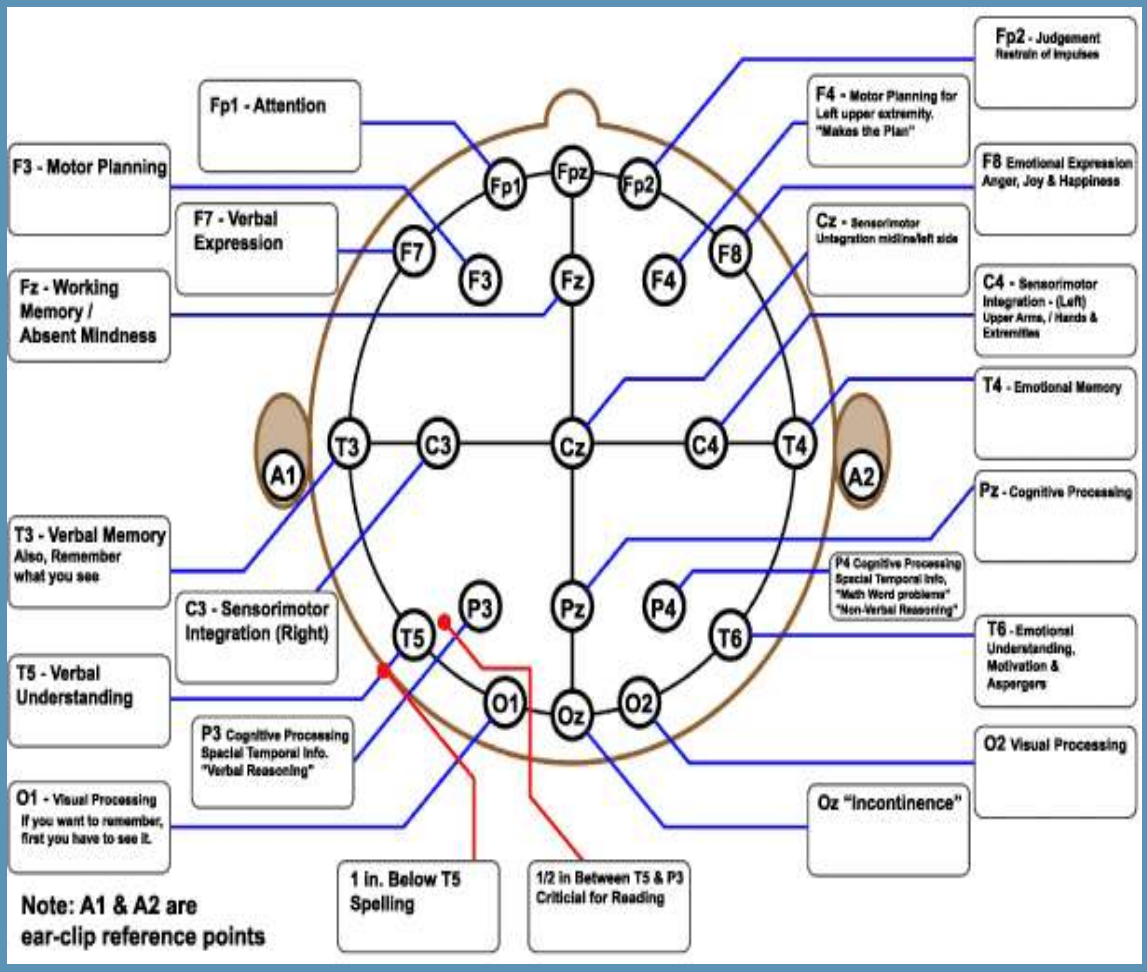


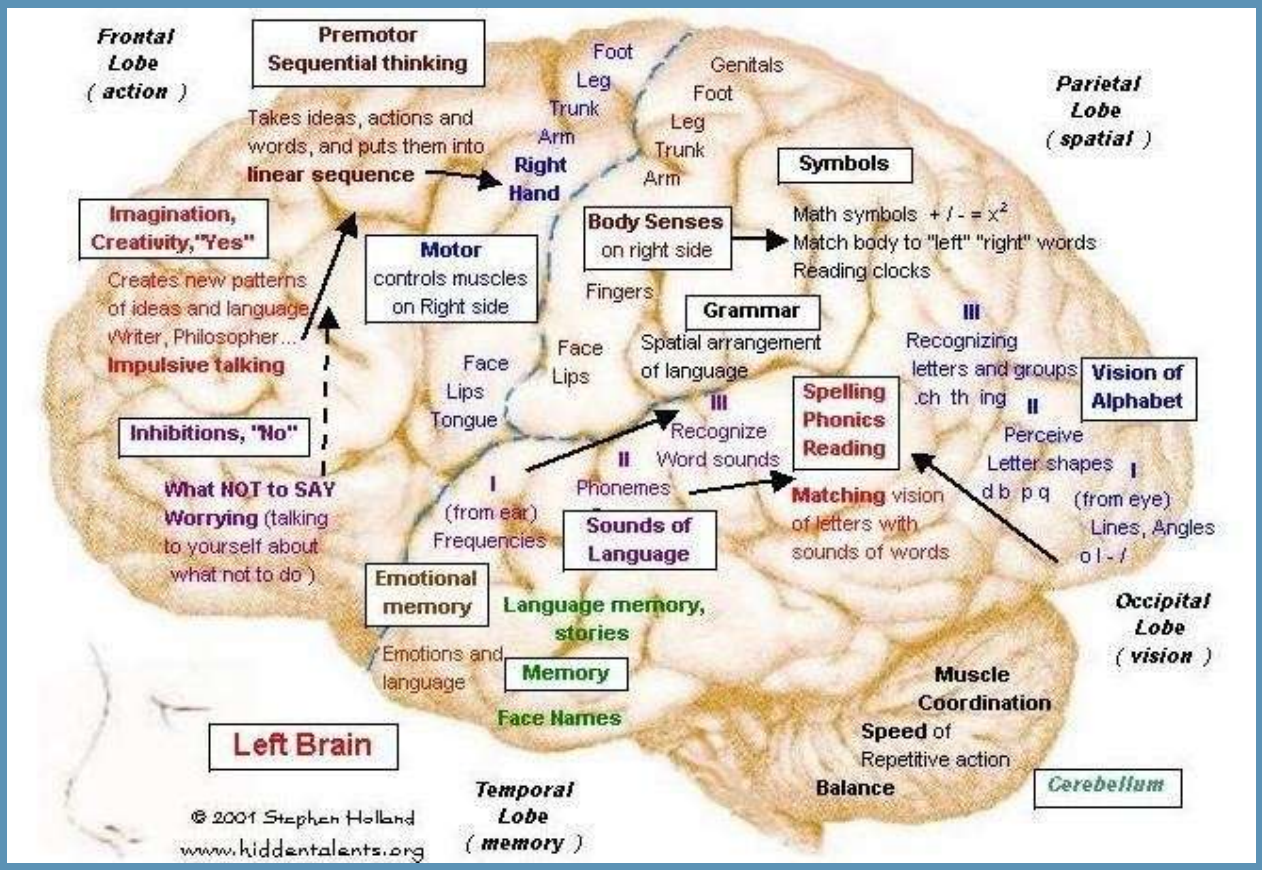
Most EEG (Emotiv) brain maps show the distribution of EEG activity in amplitude (i.e., microvolts /hertz) or power (i.e., microvolts squared/hertz) units over the cortex in terms of a set of pre-defined frequency bands— namely, Delta (1-4 Hz), Theta (4-8 Hz), Alpha (8-12 Hz), SMR or Beta1 (12-
16 Hz), Beta2 (16-20 Hz), Beta3 (20-24Hz), High Beta (24-32 Hz), and Gamma (38-42 Hz). Sometimes maps are produced for each single frequency from 1 to 40 Hz, called "single-bin" maps. Each map is color-coded to show the amount of activity from lowest to highest. Maps are also sometimes produced to show other variables related to brain functioning such as left versus right power asymmetry, phase and coherence. These latter two are measures of the amount and efficiency of communication between different cortical regions.

The normal eye can detect stimuli over a 120º range vertically and a nearly 160 degree range horizontally. From the point of fixation, stimuli can typically be detected 60º superiorly, 70º inferiorly, 60º nasally, and 100 degrees temporally, though the true extent of the visual field depends on several features of the stimulus (size, brightness, motion) as well as the background conditions. The field of vision is often depicted as a three dimensional hill, with the peak sensitivity to stimuli occurring at the point of fixation under photopic conditions, decreasing rapidly in the 10º around fixation, and then decreasing very gradually for locations further in the

periphery. Nerve fibers pass through the sclera at the optic nerve head, typically 10-15º nasal to fixation. At this location, no photoreceptors are present, creating a normal absolute scotoma.

Map of the Brain Based on International 10-20 System




Frontal Lobe (action)
Premotor Sequential thinking
Takes ideas, actions and words, and puts them into linear sequence
Right Hand
Foot Leg Trunk Arm
Genitals Foot Leg Trunk Arm
Parietal Lobe (spatial)
Symbols
Math symbols + / - = x²
Match body to "left" "right" words
Reading clocks
Imagination, Creativity,"Yes"
Creates new patterns of ideas and language
Writer, Philosopher...
Impulsive talking
Motor controls muscles on Right side
Body Senses on right side
Fingers
Grammar
Spatial arrangement of language
III Recognizing letters and groups .ch th ing
II Perceive Letter shapes d b p q
I (from eye) Lines, Angles o l - /
Vision of Alphabet
Face Lips Tongue
Face Lips
Inhibitions, "No"
What NOT to SAY
Worrying (talking to yourself about what not to do)
III Recognize Word sounds
II Phonemes
I (from ear) Frequencies
Sounds of Language
Spelling Phonics Reading
Matching vision of letters with sounds of words
Emotional memory
Language memory, stories
Emotions and language
Memory
Face Names
Occipital Lobe (vision)
Muscle Coordination
Speed of Repetitive action
Balance
Cerebellum
Left Brain
Temporal Lobe (memory)
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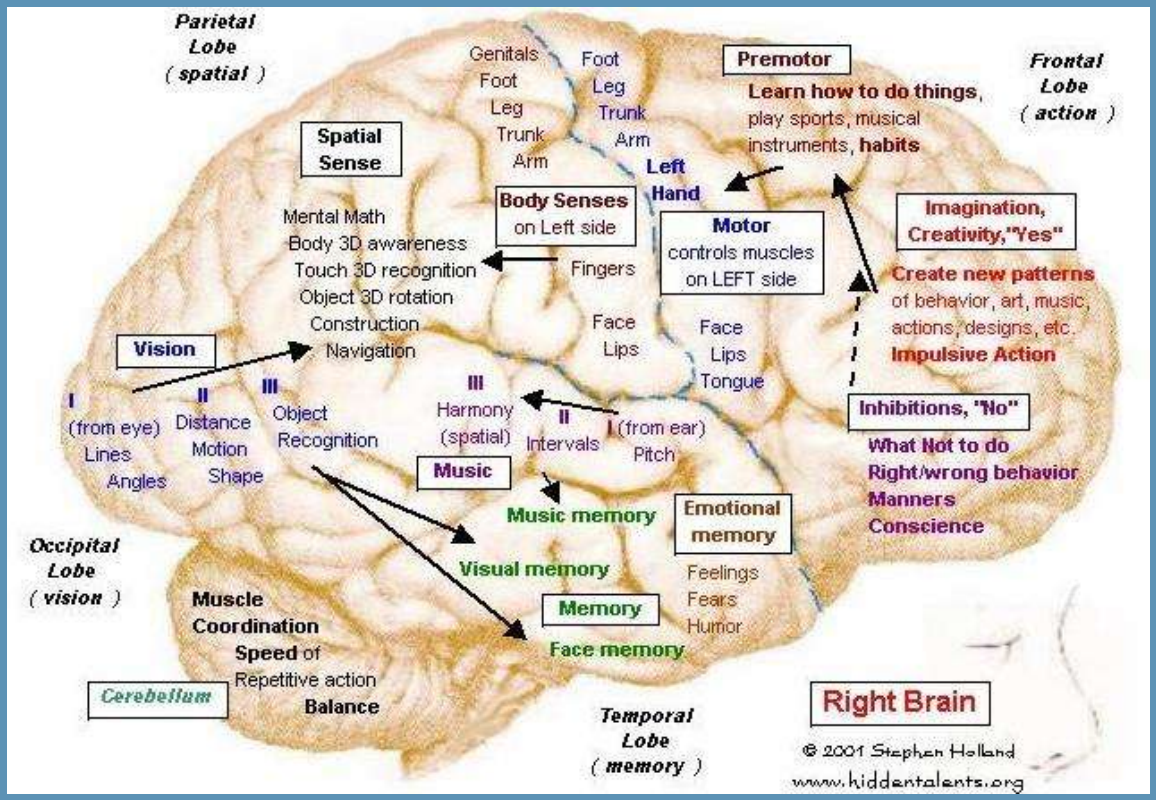
Parietal Lobe (spatial)
Spatial Sense
Genitals Foot Leg Trunk Arm
Foot Leg Trunk Arm
Premotor
Learn how to do things, play sports, musical instruments, habits
Frontal Lobe (action)
Left Hand
Body Senses on Left side
Fingers
Mental Math
Body 3D awareness
Touch 3D recognition
Object 3D rotation
Construction
Navigation
Motor controls muscles on LEFT side
Imagination, Creativity,"Yes"
Create new patterns of behavior, art, music, actions, designs, etc.
Impulsive Action
Face Lips
Face Lips Tongue
Vision
I (from eye) Lines Angles
II Distance Motion Shape
III Object Recognition
III Harmony (spatial)
II Intervals
I (from ear) Pitch
Music
Music memory
Emotional memory
Feelings Fears Humor
Inhibitions, "No"
What Not to do
Right/wrong behavior
Manners
Conscience
Occipital Lobe (vision)
Visual memory
Memory
Face memory
Muscle Coordination
Speed of Repetitive action
Balance
Cerebellum
Right Brain
Temporal Lobe (memory)
© 2001 Stephen Holland
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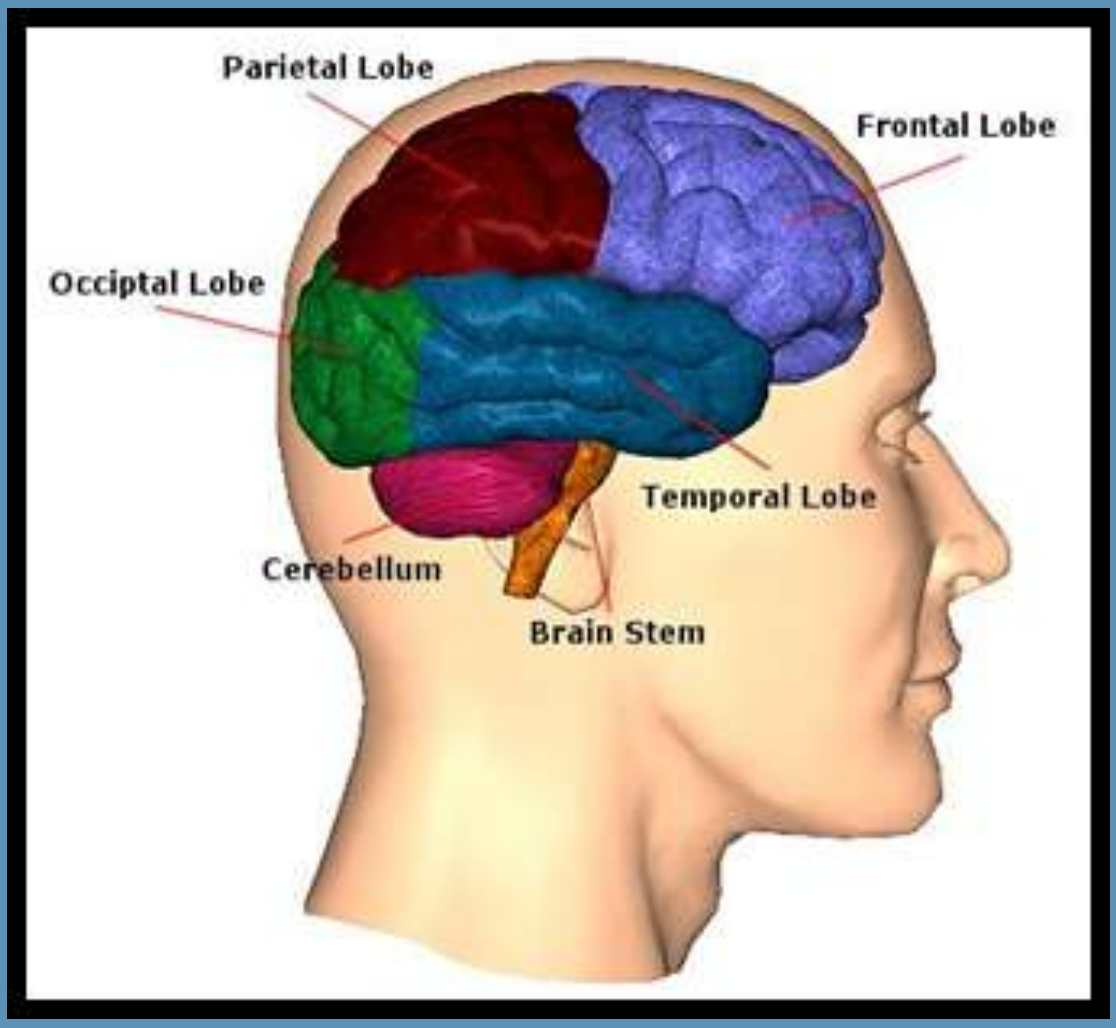
MAP of The Brain and Its Function



1. Frontal Lobes

Function	Observed dysfunction
Conscious thought Concentration Perseverance Judgement Attention span Impulse control – self monitoring and supervision Problem solving Organisation Critical thinking	Paralysis Difficulty in sequencing (Inability to plan a sequence of complex movements need to complete multi-stepped tasks) Loss of spontaneity in interacting with others Loss of flexibility in thinking Perseveration (persistence of a single thought) Difficulty attending (Inability to focus on task) Emotionally labile (mood changes)

Forward thinking Ability to feel and express emotion Empathy Memory for habits and motor activities	

For more information see: http://www.health.qld.gov.au/abios/asp/bfrontal.asp

2. Parietal Lobes

Function	Observed Dysfunction
Visual attention Touch perception Monitors sensation and body position Control reading Face recognition Understanding time Goal directed voluntary movements Manipulation of objects	Inability to attend to more than one object at a time Anomia (Inability to name an object) Agraphia (Inability to locate the words for writing) Alexia (Reading difficulties) Difficulty drawing Difficulty in distinguishing left from right Dyscalculia (difficulty with mathematics Apraxia (Lack of awareness of certain body parts and/or surrounding space) Inability to focus visual attention Difficulties with hand-eye coordination

For more information see: http://www.health.qld.gov.au/abios/asp/bparietal.asp

3. Occipital Lobes

Function	Observed Dysfunction
Receives visual information Interprets colour, shape, distance	Visual field deficits Difficulty locating objects Colour Agnosia (difficulty identifying colour) Production of hallucinations Visual illusions Inability to recognize words (word blindness) Difficulty recognizing drawn objects Movement Agnosia (inability to recognize movement of an object) Difficulty reading and writing

For more information see: http://www.health.qld.gov.au/abios/asp/boccipital.asp

4. Temporal Lobes

Function	Observed Dysfunction
Memory and new learning Receives auditory messages Understands spoken language and rhythm Controls how things are ordered and categorized Some visual perception	Prosopagnosia (difficulty in recognizing faces) Wernicke's Aphasia (difficulty in understanding spoken words) Disturbance with selective attention to what we see and hear Difficulty with identification of and verbalization about objects Short-term memory loss Interference with long term memory Increased or decreased interest in sexual behaviour Inability to categorize objects Persistent talking (right lobe damage) Increased aggressive behaviour

For more information see: http://www.health.qld.gov.au/abios/asp/btemporal.asp

5. Brain Stem

Function	Observed Dysfunction
Breathing Heart Rate Swallowing Startle Response (reflexes to seeing and hearing) Autonomic Nervous system (sweating, blood pressure, digestion, temperature) Affects level of alertness Ability to sleep Vestibular function (sense of balance)	Decreased vital capacity Dysphagia (swallowing) Difficulty with balance and movement Vertigo (dizziness and nausea) Insomnia, sleep apnoea (sleeping difficulties)

For more information see: http://www.health.qld.gov.au/abios/asp/bbrain_stem.asp

6. Cerebellum

Function	Observed Dysfunction
Coordination	Asynergia (Loss of coordination of motor

Balance and equilibrium	movements) Dysmetria (inability to judge distance and when to stop) Adiadochokinesia (inability to perform rapid alternating movements) Intention tremor Abnormal/ataxic gait (staggering wide based walking) Tendency to fall Hypotonia (weak muscles) Dysphonia (slurred speech) Nystagmus (abnormal eye movements) Loss of ability to coordinate fine movements
For more information see: http://www.health.qld.gov.au/abios/asp/bcerebellum.asp	

VCAT Training with real time Brain mapping and EEG Readings

VCAT optimizes brain function using leading-edge computer software technology in brain mapping and wireless EEG headset. The wireless EEG records the brainwave readings from around the brain matching with the recorded real time brain mapping results. The resulting brain map shows which parts of the brain are overactive, underactive, and non-active that leads to the brain mal function and chemical imbalance. The Emotional and Cognitive Analysis compares the results of the self-assessment against the results of the EEG readings and brain mapping results to identify problem areas within the brain. The brain map is a revolutionary new tool in accurately identifying the problem areas of the brain. It takes the guesswork out of the assessment process and provides an accurate road map for customizing a precise individual VCAT-treatment plan to suit that particular disorder. The individual will then be able to be treated with VCAT while watching his/her brain performance in real time.

Typical VCAT/NeuroMaxfeedback Treatment Process

It begins with an assessment which includes an intake questionnaire and checklists, a personal interview, and a computer-based assessment related to your particular issue/disorder. Additional testing and assessment for developmental disorders, neurological problems, or psychological

issues may be necessary on a later time. Each session includes 15 minutes of counseling, 35 minutes of NeuroMax (VCAT/EEG) including real time brain mapping. At the beginning depending on the issue the treatment sessions are recommended at least 2 times weekly. Improvement is frequently seen starting within 8 sessions. Generally 13 to 16 sessions are needed to create lasting change. Some conditions require more sessions. A progress evaluation is usually conducted after each session.

VCAT-Treatment Plan supports overcoming: anxiety, depression, racing thoughts, brain injury, poor memory, sleeping problems, addictions and many other issues such as following disorders:

Developmental Disorders:

ADD/ADHD
Learning Disability
Autism
Asperger's

Mood:
Brighten mood
Reduce or eliminate depression, anxiety and temper
Cognitive:
Enhancing Brain-Plasticity
Restore clarity & focus, problem solving, common sense,
Reduce or eliminate impulsivity
Improve memory
Improve ability to follow direction
Improve ability to follow conversation
Improve reasoning skills
Racing thoughts
Movement:
Improve sensory-motor integration issues
Restore movement and balance
Reduce spasticity
Improve coordination
Improve pain control
Energy:
Improve sleep quality:
getting to sleep, staying asleep, waking rested
Restore energy
Reduce fatigue
Reduce stress reactions
Others
OCD
Traumatic Brain Injury (TBI)
PTSD
Migraine

Research, studies, and publications done on VCAT

http://gradworks.umi.com/34/61/3461443.html

http://www.chapters.indigo.ca/books/Effect-External-Attentional-Stimulations-Such-Nader-Babai-siahdohoni/9781249033967-item.html?cookieCheck=1

http://www.ebay.com.au/itm/The-Effect-of-External-Attentional-Stimulations-NEW-/280966996643

http://www.buybooksindia.com/the-effect-of-external-attentional-stimulations-such-as-visual-concentration-attention-techniques-v-0-13146317.html

https://books.google.com/books/about/The_Effect_of_External_Attentional_Stimu.html?id=HBrAoAEACAAJ

http://www.betterworldbooks.com/the-effect-of-external-attentional-stimulations-such-as-visual-concentration-attention-techniques-id-1249033969.aspx

http://www.shimply.com/p/the-effect-of-external-attentional-stimulations-such-as-visual-concentration-attention-techniques-vcat-on-sustained-attention-in-adults-with-attention-deficit-hyperactivity-disorder-adhd-by-nader-babai-siahdohoni-9781249033967-p11917051

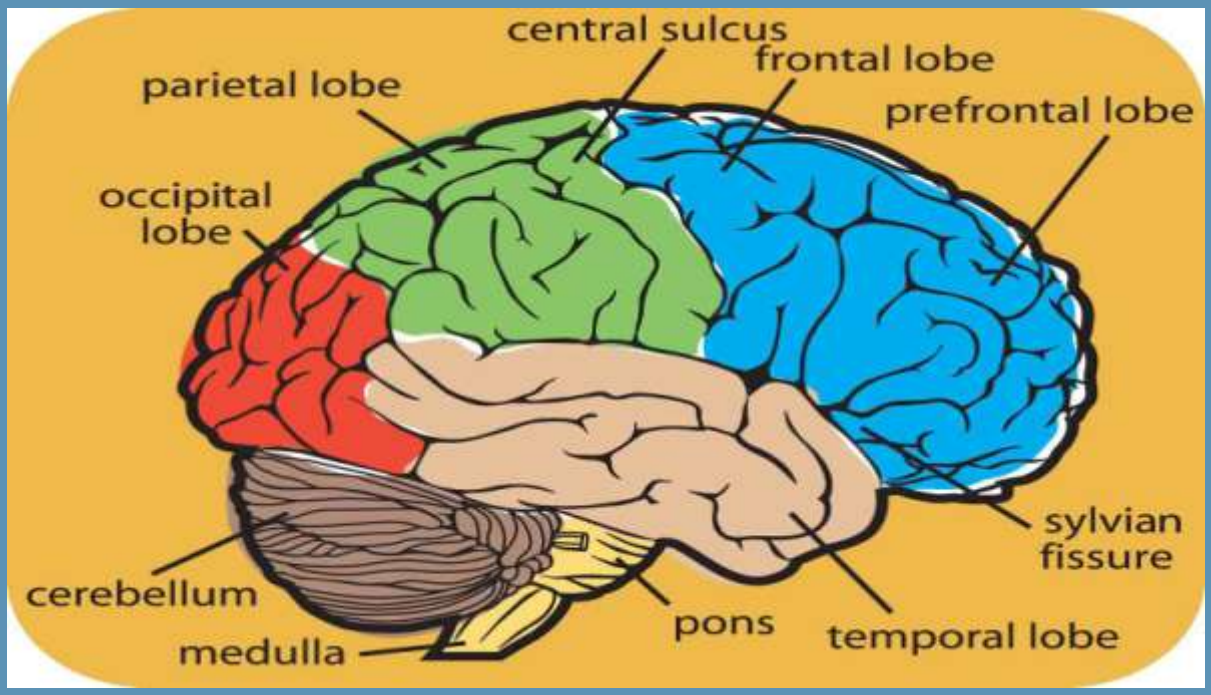
http://www.amazon.co.uk/s?_encoding=UTF8&field-author=Nader%20Babai-Siahdohoni&search-alias=books-uk

http://www.youtube.com/watch?v=txoxhYpGXwE

http://www.amazon.com/Attentional-Stimulations-Concentration-Techniques-Hyperactivity/dp/1249033969

Supplemental Information related to VCAT

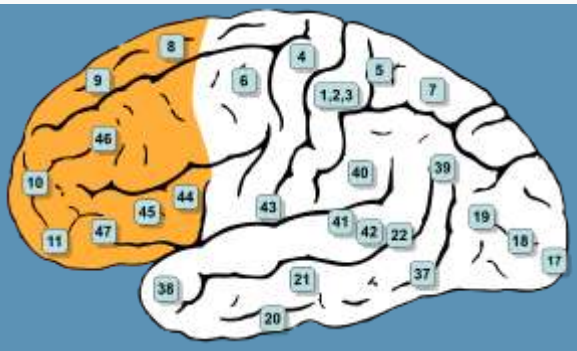
Frontal lobe



The **frontal lobe**, located at the front of the brain, is one of the four major lobes of the cerebral cortex in themammalian brain. The frontal lobe is located at the front of each cerebral hemisphere and positioned in front of the parietal lobe and above and in front of the temporal lobe. It is separated from the parietal lobe by a space between tissues called the central sulcus, and from the temporal lobe by a deep fold called the lateral sulcusalso called the Sylvian fissure. The precentral gyrus, forming the posterior border of the frontal lobe, contains the primary motor cortex, which controls voluntary movements of specific body parts.

The frontal lobe contains most of the dopamine-sensitive neurons in the cerebral cortex. The dopamine system is associated with reward, attention, short-term memory tasks, planning, and motivation. Dopamine tends to limit and select sensory information arriving from the thalamus to the forebrain. A report from the National Institute of Mental Health says a gene variant that reduces dopamine activity in the prefrontal cortex is related to poorer performance and inefficient functioning of that brain region during working memory tasks, and to a slightly increased risk for schizophrenia.[1]

1. "Gene Slows Frontal Lobes, Boosts Schizophrenia Risk". National Institute of Mental
2.
3. Health. May 29, 2001. Retrieved 2013-06-20.
4. **Jump up^** Giedd JN, Blumenthal J, Jeffries NO et al. (October 1999). "Brain development during childhood and adolescence: a longitudinal MRI study". *Nature Neuroscience* **2**(10): 861–3. doi:10.1038/13158. PMID 10491603.

Prefrontal cortex	
 Brodmann areas #9, #10, #11, #12, #46, and #47 are all in the prefrontal cortex.	
Details	
Latin	*Cortex praefrontalis*
Part of	Frontal lobe
Components	Superior frontal gyrus Middle frontal gyrus Inferior frontal gyrus
Artery	Anterior cerebral Middle cerebral
Vein	Superior sagittal sinus

The PFC contains Brodmann areas 9, 10, 11, 12, 46, and 47.

Many authors have indicated an integral link between a person's personality and the functions of the prefrontal cortex.[1] This brain region has been implicated in planning complex cognitive behavior, personality expression, decision making, and moderating social behavior.[2] The basic activity of this brain region is considered to be orchestration of thoughts and actions in accordance with internal goals.[3] Destruction of the anterior two-thirds results in deficits in concentration, orientation, abstracting ability, judgment, and problem solving ability; destruction of the orbital (frontal) lobe results ininappropriate social behavior.

The most typical psychological term for functions carried out by the prefrontal cortex area is executive function. Executive function relates to abilities to differentiate among conflicting thoughts, determine good and bad, better and best, same and different, future consequences of current activities, working toward a defined goal, prediction of outcomes, expectation based on actions, and social "control" (the ability to suppress urges that, if not suppressed, could lead to socially unacceptable outcomes).

Frontal cortex supports concrete rule learning. More anterior regions along the rostro-caudal axis of frontal cortex support rule learning at higher levels of abstraction.[4]

7. Interconnections

The prefrontal cortex is highly interconnected with much of the brain, including extensive connections with other cortical, subcortical and brain stem sites.[13]The dorsal prefrontal cortex is especially interconnected with brain regions involved with attention, cognition and action,[14] while the ventral prefrontal cortex interconnects with brain regions involved with emotion.[15] The prefrontal cortex also receives inputs from the brainstem arousal systems, and its function is particularly dependent on its neurochemical environment.[16] Thus, there is coordination between our state of arousal and our mental state.[17]

The medial prefrontal cortex has been implicated in the generation of slow-wave sleep (SWS), and prefrontal atrophy has been linked to decreases in SWS.[18] Prefrontal atrophy occurs naturally as individuals age, and it has been demonstrated that older adults experience impairments in memory consolidation as their medial prefrontal cortices degrade.[18] Significant atrophy can also occur as a result of neuroleptic or antipsychotic psychiatric medication.[19] In older adults, instead of being transferred and stored in the neocortex during SWS, memories start to remain in the hippocampus where they were encoded, as evidenced by increased hippocampal activation compared to younger adults during recall tasks when subjects learned word associations, slept, and then were asked to recall the learned words.[18]

There is much current research devoted to understanding the role of the prefrontal cortex in neurological disorders. Many disorders, such as schizophrenia,bipolar disorder, and ADHD, have been related to dysfunction of the prefrontal cortex, and thus this area of the brain offers the potential for new treatments of these conditions.

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2. DISORDERS ASSOCIATED WITH DAMAGE TO prefrontal cortex PFC

As it has been known that frontal lobe not only mediates cognitive aspects of the personality, but also its affective and emotional aspects as well.

1. Apathy

It results from widespread lesions of the PFC. In the affective sphere, the hallmark of the disorder is the generalized blunting of affect and emotional responses. The patient's underlying mood is frequently one of profound indifference, and so is his or her attitude toward others.[16]

2. Depression

Experience of depressed mood, can be a result of left PFC lesions, especially those involving anterior (polar) aspects of the frontal lobes.[83]

3. Social behavior

PFC lesions are likely to have great impact on social behavior particularly in case of OFC lesion. It commonly results in euphoria. Instinctual urges may be released or exacerbated. Some patients with OFC lesions show a tendency to have voracious appetite, driven to satiate an apparently insatiable hunger. The sexual drive also appears frequently disinhibited by prefrontal, especially orbital lesions.[84]

4. Prefrontal syndromes

To have a holistic and comprehensive understanding of these syndromes, a more specific terminology based on structural, functional, and clinical features, which recognize *dorsolateral*, *medial*, and *orbital*prefrontal regions, was developed.

5. Dorsal convexity Dysexecutive syndrome

It is characterized by deficits in cognitive flexibility, temporal ordering of recent events, planning, regulating ones actions based upon internal, and external stimuli.[85] This results in a reduced state of mental control, perseveration, and impairment of sustained attention. The capability to retrieve information is altered despite evidence of intact recognition. Patients present with diminished judgment, impaired working memory, insight, self-care, and there is often a general reduction in verbal and nonverbal fluency. There is impaired priming of stereotypes if the lesion is of ventromedial PFC.[86]

6. Medial frontal apathetic syndrome

The hallmark feature of medial apathetic syndrome is a severe reduction in spontaneity, motivation, and lack of interest in the environment. Memory of recent events is relatively intact. It is thought that the overall alteration in motivation and motor activity is a result of the lesion involving the medial motor cortices.[3]

Orbitofrontal disinhibition syndrome

Patients with OFC damage are characterized generally by an acquired disturbance of personal and social behaviors.[39] There are marked abnormalities in the realms of reasoning, decision-making, and emotional control. This often results in explosive aggressive outbursts characterized by socially unacceptable, tactless, and vulgar presentation.[3]

7. Association with expression of psychiatric disorders

The specification of the component “executive” processes and their localization to particular regions have been implicated in a wide variety of psychiatric disorders ranging from depression to anxiety disorders to schizophrenia as well as in a number of other disorders like attention deficit hyperactivity disorder (ADHD), autism, conduct disorder, etc.

1. Schizophrenia:

Findings from WM studies in schizophrenia indicate that schizophrenia patients are consistently impaired on WM tasks irrespective of WM domain or processing requirements. This pattern of WM performance may further implicate DLPFC dysfunction in the liability for schizophrenia and has implications for future cognitive, genetic, and developmental research.

2. Mania:

Study conducted by Lebowitz *et al*. indicated that impairment in verbal fluency was found to be greater with the increase in number of episodes of mania.[87] Both number of episodes and total number of hospitalizations have been found to be related to poorer performance on several aspects of WCST.[88]

3. Depression:

Based on a meta-analysis of 13 studies, Veiel concluded that cognitive deficiencies associated with major depression are similar to those seen in moderately traumatic head injury.[89] Merriam *et al*.reported that unipolar major depression patients demonstrated significant deficits on the WCST.[90]

4. Dementia:

Patients with Alzheimer's disease (AD) and Parkinson's disease can be distinguished on the basis of certain cognitive and behavioral features. Performances on different tests have given rise to the similarities and differences in the cognitive profile of these two groups.[91] Similarities between the groups were seen in visuo-motor speed and attention, but differences were found in executive functioning, memory, sequencing abilities, set shifting, and word fluency. Thus, cortical patients (AD) perform significantly worse than the subcortical group (Parkinson's disease) in the memory abilities while the latter group showed greater deficits on the executive functions. Studies have found that patients with a high-cognitive reserve, i.e., higher education, occupation, etc. attain a higher neuropsychologic performance than those with a low cognitive reserve and this plays an important protective role in the incidence of cognitive deterioration and dementia.[92]

3. CONCLUSION

Large-scale distributed networks coordinate all complex behavior domains. The performance of a relevant task engages all components of the pertinent network, and damage to any network component can impair behavior in the relevant domain. Experimental data and lesion based-behavioral analyses and functional imaging observations demonstrate that the appropriate and skilled execution of higher-order tasks depend not only on PFC, but also on the integrity of other cortical and subcortical structures that are interconnected with the PFC.

4. Footnotes

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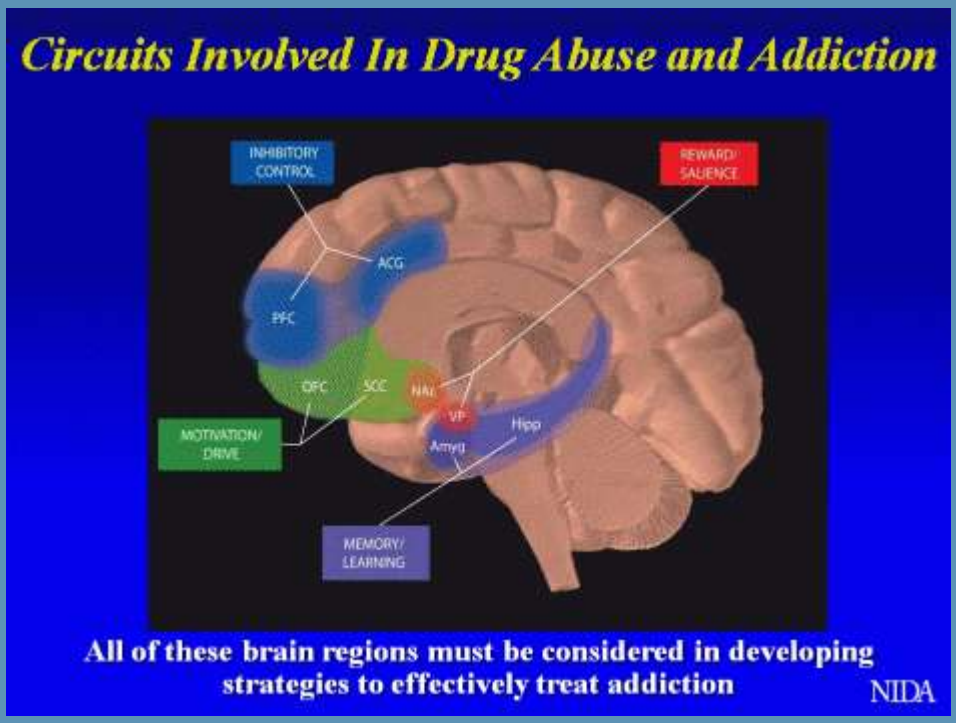
Substance abuse and Addiction

The areas depicted contain the circuits that underlie feelings of reward, learning and memory, motivation and drive, and inhibitory control. Each of these brain areas and the behaviors they control must be considered when developing strategies to treat drug addiction.



Key:
PFC – prefrontal cortex;
ACG – anterior cingulate gyrus;
OFC – orbitofrontal cortex;
SCC – subcallosal cortex;
NAc – nucleus accumbens;
VP – ventral pallidum;
Hipp – hippocampus;
Amyg – amygdala.

Low dopamine levels and lack of pleasure
How Does the Brain Become Addicted? *Typically it happens like this:*

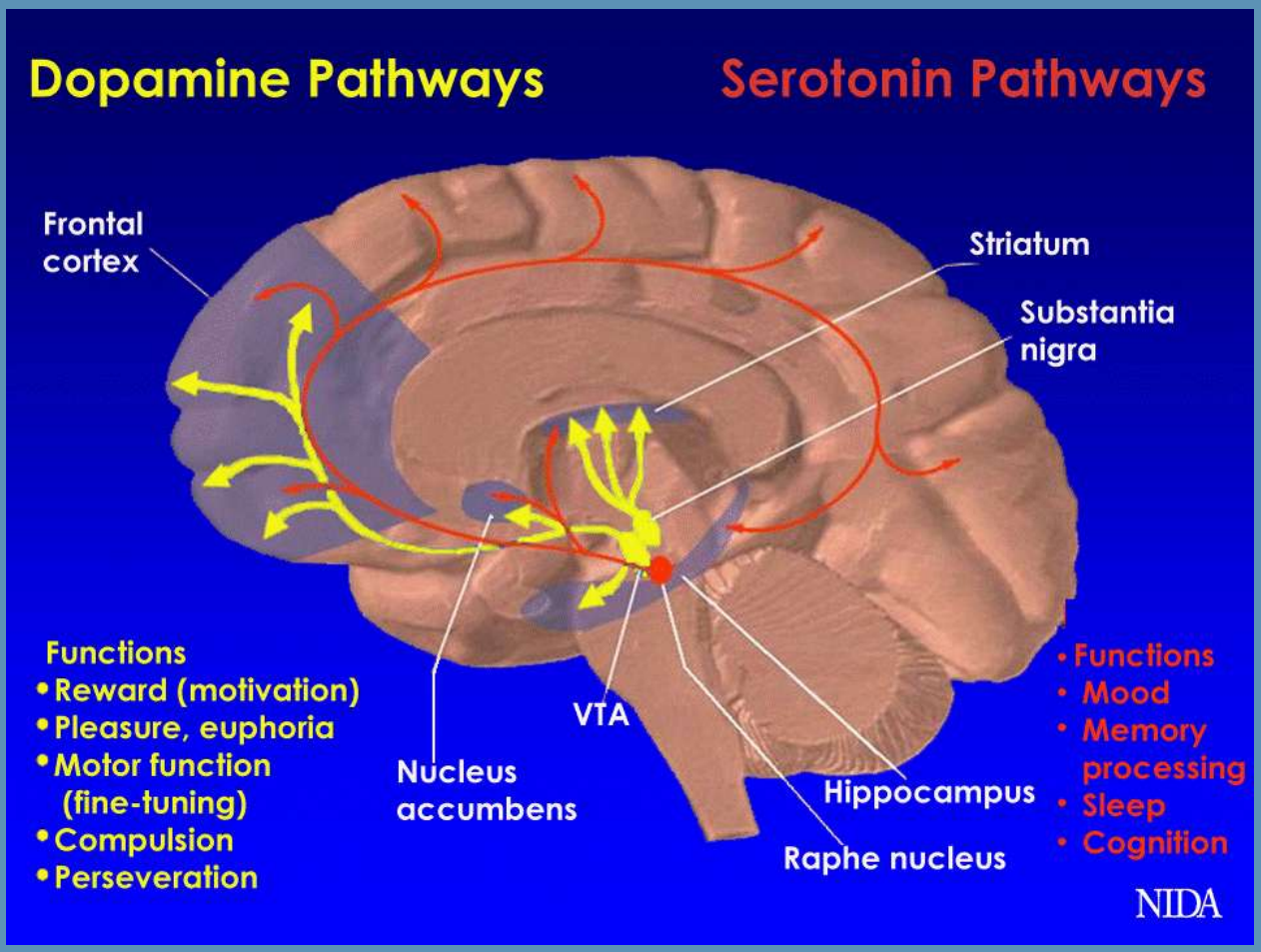
* A person takes a drug of abuse, be it marijuana or cocaine or even alcohol, activating the same brain circuits as do behaviors linked to survival, such as eating, bonding and sex. The drug

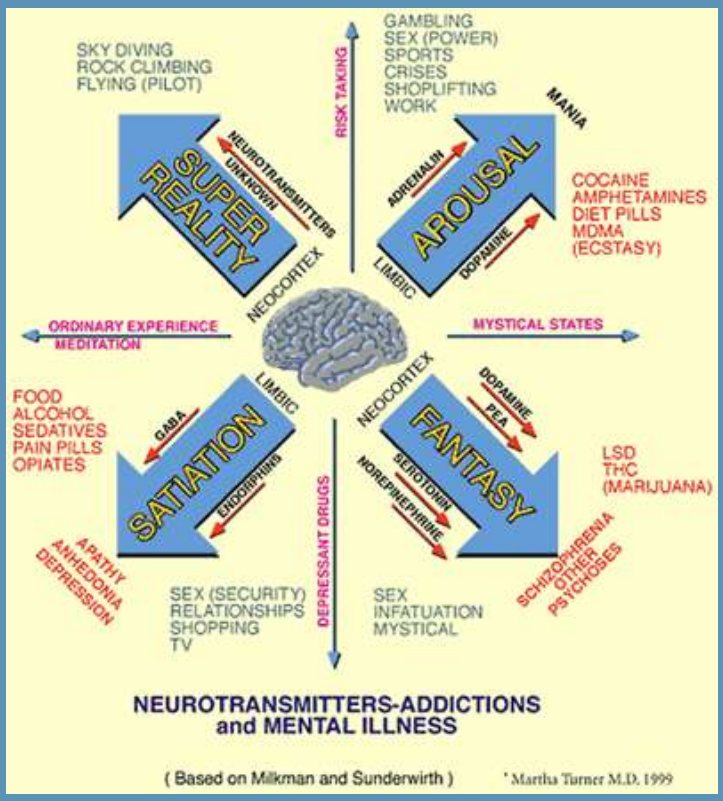
causes a surge in levels of a brain chemical called dopamine, which results in feelings of pleasure. The brain remembers this pleasure and wants it repeated.

* Just as food is linked to survival in day-to-day living, drugs begin to take on the same significance for the addict. The need to obtain and take drugs becomes more important than any other need, including truly vital behaviors like eating. The addict no longer seeks the drug for pleasure, but for relieving distress.

* Eventually, the drive to seek and use the drug is all that matters, despite devastating consequences.

* Finally, control and choice and everything that once held value in a person's life, such as family, job and community, are lost to the disease of addiction.





1.*The neural circuits of the ventral tegmental area (VTA), the nucleus accumbens, and the prefrontal cortex are critical in learning both natural and drug reward behavior.*

Upon exposure to either a naturally rewarding stimuli or a drug, there is a increase in dopamine signaling from the ventral tegmental area (VTA) to the nucleus accumbens. The nucleus accumbens "perceives" this dopamine signal and measures the "goodness" of the agent or the natural reward based on the size of the dopamine release.

2.*Glutamate projections from the nucleus accumbens instruct the prefrontal cortex to remember the environment and behaviors which lead up to the occurrence of the "goodness".*

3. *In addiction, excess signaling of glutamate neurons in the prefrontal cortex stimulate the nucleus accumbens, triggering drug seeking behaviors at the expense of naturally rewarding or good behaviors.*

5.

6.

7. Anxiety and Brain

8.

9. Neurotransmitters and Anxiety

Your brain responds directly to neurotransmitters – little chemicals inside your body that send messages to your brain about how you should feel, think, act, and more. Many neurotransmitters have been linked to anxiety, including:

- Serotonin
- GABA
- Norepinephrine

Even dopamine may play a role in anxiety, or at least have a calming effect on those already living with anxiety symptoms. Interestingly, too much or too little of any hormone may also effect anxiety in different ways. The problem is with balance. If your brain doesn't have enough serotonin, for example, it may cause you to experience anxiety symptoms.

When it comes to neurotransmitter production, the truth is that cause and effect are rarely known. It's often impossible to distinguish between poor neurotransmitter balance as a result of life experience, or poor neurotransmitter balance as a result of genetics. Both can occur in anyone living with anxiety, and in some cases a combination of both may be responsible for anxiety symptoms.

10. Anxiety and Brain Activation

There are two different parts to an anxiety disorder, and someone with anxiety may suffer from one or both. The first part is mental – verbal worries, nervous thoughts, etc. The second part of anxiety is physical. For example, a racing heartbeat, panic attacks, lightheadedness, and other physical symptoms.

It's possible to experience physical symptoms with less worry, and it's possible to worry often without many physical symptoms. Researchers also found that both of these excited different parts of the brain. Those with worried thoughts showed more left brain activity when nervous. Those with physical symptoms experienced more right brain activity.

Another study looked at the way that those with a spider phobia reacted to the belief that they were going to encounter a spider. They found that those with the phobia had their dorsal anterior cingulate cortex (ACC), insula, and thalamus become more active than those without a phobia.

Yet another study at the University of Wisconsin – Madison found that those with generalized anxiety disorder appeared to have a weaker connection between the white matter area of the brain and the pre-frontal and anterior cortex. This was compared to those without generalized anxiety disorder and the results appeared to be significant.

These are just some of the ways that anxiety can activate the brain.

11. Hormones and Anxiety

Hormone balances may affect anxiety as well. Many different hormones have an effect on brain chemistry and neurotransmitter production and balance, so if these hormones appear to be out of balance, anxiety may be the result.

Some examples of hormones affecting the brain include:

- **Adrenaline/Epinephrine** – Adrenaline is one of the most common causes of anxiety symptoms. Your body releases it when your fight or flight system is active, and it causes the increase in heart rate, muscle tension, and more. In some cases, long term stress and anxiety may damage your ability to control adrenaline, leading to further anxiety symptoms.
- **Thyroid Hormone** – Thyroid hormone appears to regulate the amount of serotonin, norepinephrine, and Gamma-aminobutyric acid (GABA) produced and distributed to the brain, so problems with your thyroid may also increase your risk for developing anxiety.

Several hormones may cause anxiety, and a change in brain chemistry may increase the production of hormones that lead to further anxiety symptoms.

12. Panic Attacks and the Brain

Panic attacks are a particularly distressing form of anxiety, and these may be due to the health of the brain too. Researchers have found that those with panic attacks often have an overactive amygdala. While it's not clear what creates this over activity, the fact that that area of the brain appears to contribute to panic attacks indicates that some aspect of the brain is in control of the panic attack experience.

13. Other Links Between Anxiety and the Brain

Another interesting relationship between anxiety and the brain is that long term anxiety may damage the brain in a way that could cause further anxiety. Researchers have found that when you leave your anxiety disorder untreated, the dorsomedial prefrontal cortex, anterior cingulate, hippocampus, dorsolateral prefrontal cortex, and orbitofrontal cortex all appear to decrease in size. The longer the anxiety goes untreated, the smaller and weaker they appear to be.

What's interesting is that not only do these changes affect anxiety symptoms – they also create anxious thoughts. Those with anxiety may feel their thoughts are completely natural, when in reality the brain contributes to that type of negative thinking.

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Natalya Chechko, Renate Wehrle, Angelika Erhardt, Florian Holsboer, Michael Czisch, Philipp G. Sämann. Unstable prefrontal response to emotional conflict and activation of lower limbic structures and brainstem in remitted panic disorder. PLoS ONE (2009), 1–15, Online-Vorabpublikation 20. May 2009

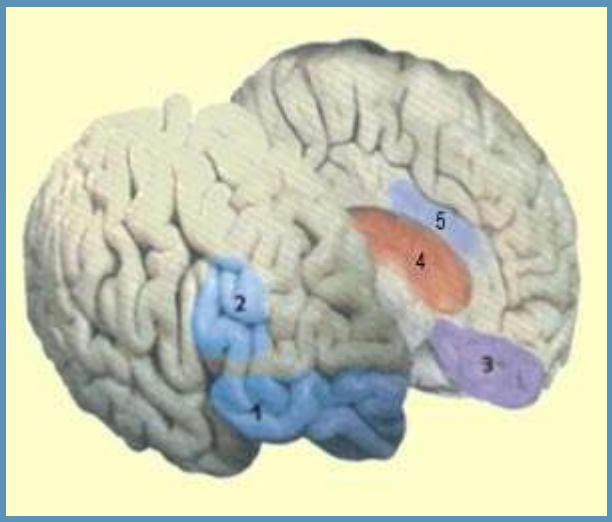
NA, Reduced Brain Connections Seen in People With Generalized Anxiety Disorder. ScienceDaily. ScienceDaily, 04 Sept. 2012. Web. 16 Dec. 2012.

Depression and Brain

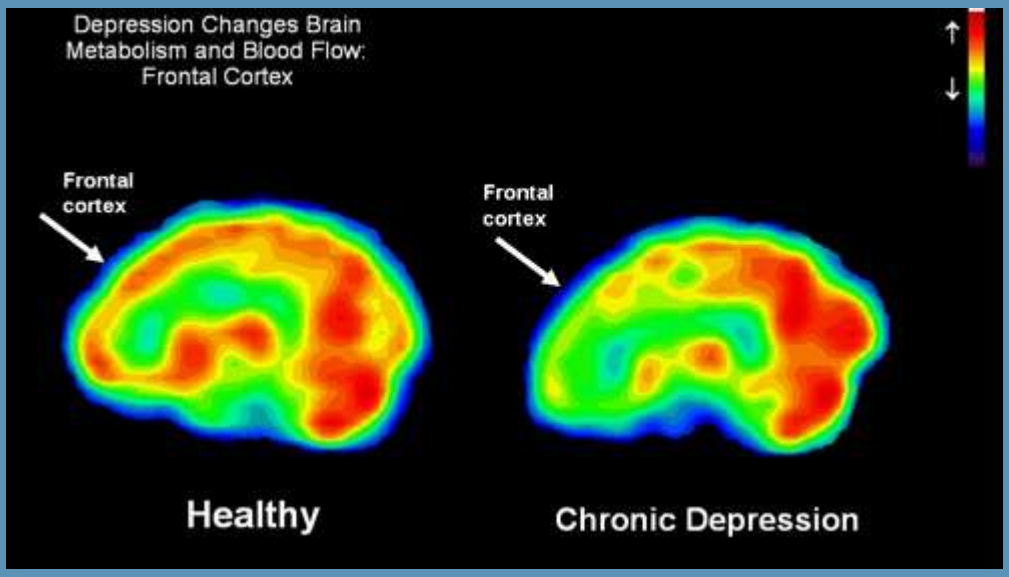
One of the regions of the prefrontal cortex that is most affected both by depression and by the manic phase of manic depression is the **ventromedial cortex** (also known as the subgenual cortex, because it sits beneath the genua, or knee, of the corpus callosum). This area deep inside the frontal lobes, on either side of the centre line separating the two hemispheres, lets us switch from one kind of affect to another. It is also heavily involved in feelings of pleasure and positive reinforcement.

The ventromedial cortex has very dense connections with the limbic system. These connections make the ventromedial cortex an ideal structure for linking the conscious to the unconscious and for ascribing meaning to perceptions by associating them with a meaningful whole. Also, this region is strongly modulated by the neurotransmitters involved in depression.
We already know that all of these structures are interconnected by neural pathways. Thus someone's **amygdala**, which can generate a number of emotions related to fear, might produce a negative emotion, the **prefrontal lobes** would revive some bad memories associated with this emotion, the **anterior cingulate cortex** would prevent the person from thinking of anything else, and the **thalamus** would promote the activity of the circuits that form this "depression loop".
A substantial reduction in the number of glial cells in certain parts of the brain, such as the ventromedial prefrontal cortex, has been observed in people who are clinically depressed.

This discovery is interesting, because it might explain some other observations associated with depression, such as atrophy or a decline in neural activity in certain parts of the brain. The function of certain glial cells, such as astrocytes, is to supply the neurons with energy. Hence, if the number of astrocytes decreased, a decline in the activity of the associated neurons could be expected.

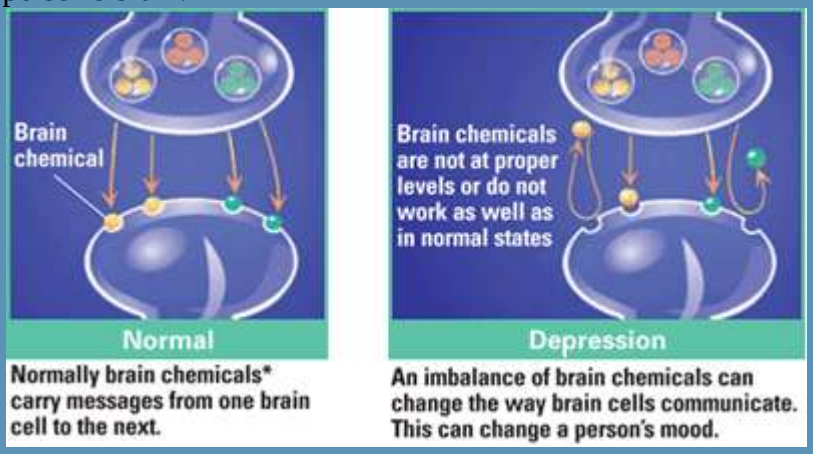


1) orbitofrontal cortex
2) lateral prefrontal cortex
3) ventromedial cortex
4) limbic system
5) anterior cingulate cortex



Chemical Imbalance

Depression has been linked to problems or imbalances in the brain with regard to the neurotransmitters serotonin, norepinephrine, and dopamine. The evidence is somewhat indirect on these points because it is very difficult to actually measure the level of neurotransmitter in a person's brain.



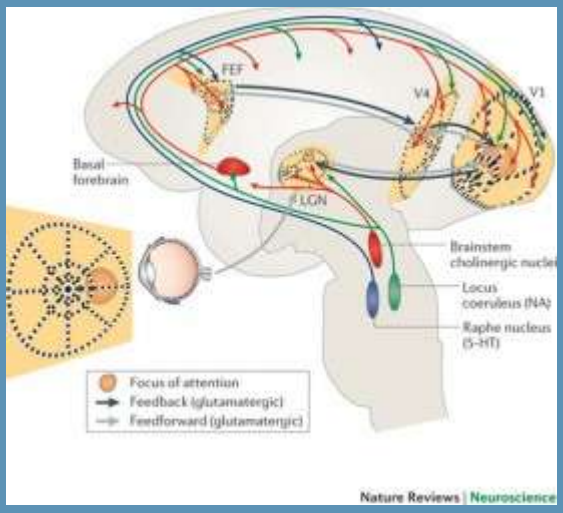
Attention Processing System Related to VCAT

Neuroscience of Attention

Ed. George R. Mangun. Oxford University Press (2012)

Summary and review of the above book

INTRODUCTION: Voluntary attention is shown to be involved with slower and more deliberative processing, as opposed to the quick reaction of involuntary attention. Voluntary attention involves the frontal brain regions dealing with both emotional/evaluative and planning/working memory processing; these are some of the areas most closely correlated to conscious experience. They are thought to influence the frontal eye field (FEF) and other parts of the fronto-parietal network that controls attention.



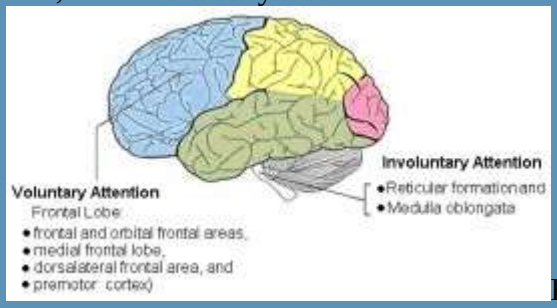
Mechanisms of attention are seen as crucial for the brain to select behavior-relevant information from the environment, with only some incoming signals beinge selected for enhanced processing. Top-down influences are argued to be the drivers in giving preference to inputs that are more likely to be relevant to goal-directed behavior. Attention is seen as based on top-down signals feeding back through the cortex, with fronto-parietal areas modulating activity in both the thalamus and the sensory cortex. Perceptual processing is demonstrated to be more facilitated at attended locations than at unattended locations; inputs from unattended locations tend to be repressed. Moreover, neural activity is increased just by directing attention to a location, even in the absence of incoming stimuli.

Spatial attention allows organisms to concentrate information processing on a limited spatial region. Top-down control is related to working memory, and to fronto-parietal areas tuning sensory neurons to attend to a particular location. With top-down or voluntary attention,

working memory and related brain areas control the responsiveness of sensory neurons to particular stimuli and inhibit neurons that might attend to other stimuli.

INVOLUNTARY and VOLUNTARY ATTENTION

With exogenous or involuntary attention, the trigger for attention comes from outside the brain, for instance loud bangs or flashes. One distinction between involuntary and voluntary attention is that involuntary attention takes only about 50 ms after the initial input of a stimulus to register data, while voluntary attention takes 300 ms or longer.



However, involuntary attention last for only a few hundred milliseconds, after which, either voluntary attention is attracted to the stimulus, or there is an involuntary inhibition of attention to the intruding stimulus. By contrast, voluntary attention can be focused for long periods. In general, voluntary attention and top-down expectations have more influence on the later stages of processing, and the length of time for which attention is held may be important in determining subsequent action and behavior.

SENSORY INPUT & INTERNAL MODULATION

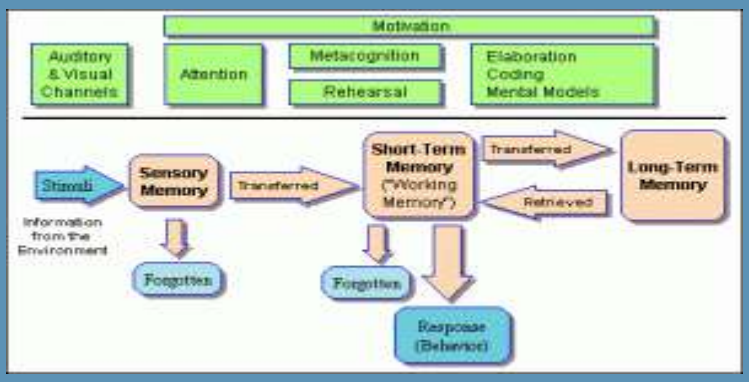
Internal neural signals are seen to modulate the perceptions that result from sensory inputs. The research of the last 20 years demonstrates a clear divide between sensory input, and the internally driven modulation of sensory processing. Top-down signals from the prefrontal cortex bias the sensory system towards stimuli that are relevant for behavior. The dorsal fronto-parietal attention network (DAN) is involved in the selection of task-relevant stimuli. Regions in the DAN respond to behaviorally relevant stimuli, in preference to equally strong signals that are not behaviorally relevant.

FRONTAL EYE FIELD

In the forebrain, the frontal eye field (FEF) and the parietal both encode the relative importance of stimuli. However studies suggest that the frontal eye field is still downstream of the actual direction of attention coming from the dorsolateral prefrontal which controls the working memory. The forebrain system relates to slower more deliberative activity involving the working memory, while midbrain areas deal with responses to sudden threats. The direction of attention is seen as being coded by the activity of particular neurons, notably in the FEF. The FEF is thought to have connections to both the ventral and the dorsal stream, and attention is directed to where targets are likely to appear. Enhanced neural activity can be observed prior to the arrival of attended objects in the locations at which they are expected. Both visual and movement responsive neurons in the FEF were found to attend the location of spatial attention, and this neural activity is predictive of what the subject will detect. A third type of neuron in the FEF is capable of remaining highly active, even after the stimuli that originally gave rise to the activity has disappeared. The frontal eye field (FEF) receives converging inputs from many cortical areas, and may use visual representations in working memory to select targets for spatial attention. The operation of the frontal eye field (FEF), as distinct from other brain regions, is suggested to be independent of current sensory inputs, thus functioning in a purely top-down fashion, and having a unique role in the voluntary control of attention.

WORKING MEMORY

Working memory is viewed as holding representations while a visual search is underway. Top-down influences direct visual attention, while shared neural mechanisms decide the items that are held in short-term memory, and these in their turn act as drivers of behavior. Cognitive functions are seen as depending on information being retained in the brain, after the original signal input is no longer present.



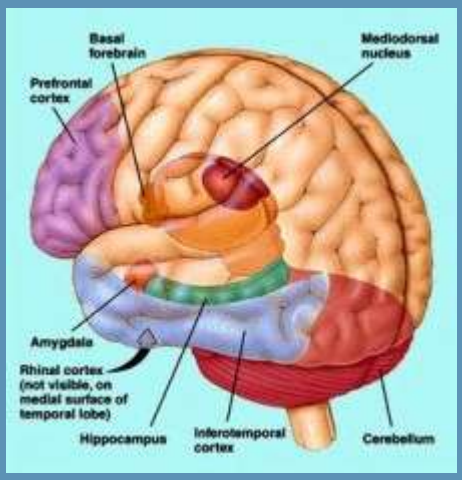
Visual short-term memory allows retained visual information to guide behavior. The process of retaining a visual memory is suggested to involve a subset of the neurons that encode the original signal. The mechanism of attention that influences the original perception is also suggested to direct the visual short-term memory. Existing biases also help to determine which items survive longest in short-term memory.

There is evidence of capacity limitations in the short-term memory. Thus change-detection for color or shape declines when there are more than four items in the visual memory. This forces access to working memory to be selective and driven by existing biases. Preparatory attention involves top-down signals from the frontal and parietal areas, so that areas of the primary and secondary visual cortex can become activated even where no relevant signal subsequently appears. Biases also help to determine which items survive longest in short-term memory. The stability of frontal brain processing such as the short-term memory is seen to require protection from the sensory bombardment from the lower sensory cortex.

ATTENTION and EMOTION

The dorsal fronto-parietal network influences attention sensory neurons, but it is not seen as the real origin of voluntary biases, which are suggested to come ultimately from the brain's reward systems. A distinction is made here between internally driven voluntary attention on the one hand, and on the other, involuntary attention given to unanticipated inputs from the environment.

Attention and emotion are argued to be parallel processes interacting at many stages in the brain. Limbic regions act so as to bias sensory processing, and several emotional-attentional hubs are identified in the frontal areas of the brain. Emotional and motivational aspects are considered to drive attentional control, sensory representation and goal-directed behavior. The emotional areas are seen to interact with the executive/planning/working memory areas. Attention and emotion are argued to be intertwined. Processes in the amygdala and other limbic areas are viewed as interacting with attentional controls in the fronto-parietal area and the ventral visual stream. Thus emotional arousal is viewed as altering the focus of attention.

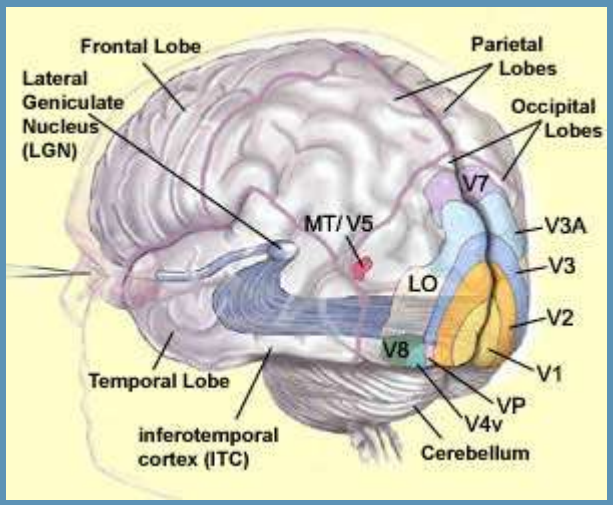


The amygdala is the most connected area of the forebrain and interfaces with the anterior cingulate, the insula and the orbitofrontal. These limbic regions interact with the dorsolateral fronto-parietal network to bias sensory processing and eventually behavior. The emotional areas also have feedback influences on sensory processing, such as the ventral visual stream where the representation of emotionally significant events can be enhanced. More sustained attention can be supported by the emotional content of images.

1. Switching channels: Attention in the brain!

Selective visual attention in the brain is connected through the pulvinar area in the thalamus.

For most of us, a primary experience of consciousness is vision, and more specifically selective control of our visual experience. Our eyes take in far more than we can "process" at any time, so it is essential to limit this stream, at the same time that it empowers our sense of sovereignty, even if our attention is pulled by external events and easily manipulated by conjurers, film directors, and advertisers.

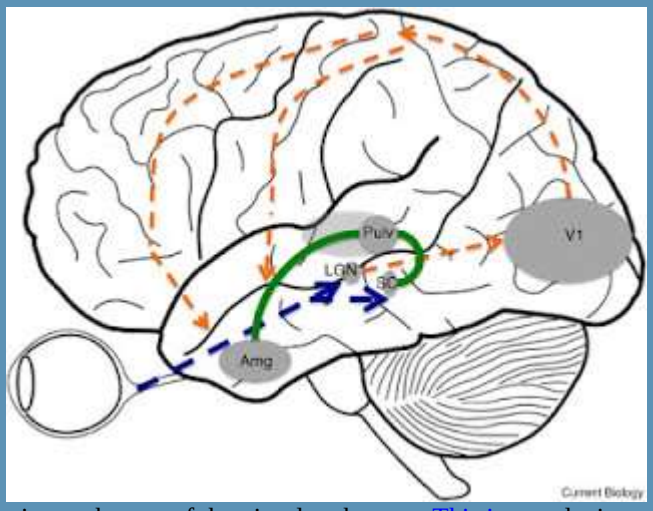


Visual pathways, early stages. Signals coming from the retina are channeled through the LGN to the visual cortex at the back of the brain. They percolate up through the V1 to V8 areas and

branch into upper (dorsal) and lower (ventral) streams to other areas of the brain. Vision flows in complicated pathways in the brain, from the eyes though a central waystation (geniculate nuclei in the thalamus) to the visual cortex at the very back of the brain for processing into ever more abstract and meaningful representations, during or after which it forks into two streams. The upper (dorsal stream) coming into the parietal cortex forms most of our unconscious visual awareness of motion and location, directing motor control, while the lower (ventral stream) heads back to the thalamus and into the temporal cortex, and seems to provide identification (what) information, (interconnected with memory), and emotional salience information. It seems to be the more consciously available pathway.

Of course we want to focus on the most emotionally salient information. And we want to notice aspects of both streams at the same time, for unified perception. A recent paper describes pathbreaking techniques in identifying and recording key routes of the visual pathway in macaques, and finds that an area in the thalamus may serve as an orchestrator of visual attention.

What is attention? In neuroscience, it is increasingly recognized as synchronized neural firing among distributed brain regions, binding together various processed aspects of a particular scene. While consciousness per se remains unresolved, attention has been the focus of great, er, attention. Most perceptual pathways appear to have feedback pathways going back from higher abstract thinking levels into their primary processing systems which can either shut off or call forth activity that is synchronized with the calling regions. In the visual system, there is the added problem of synchronization between the two streams.

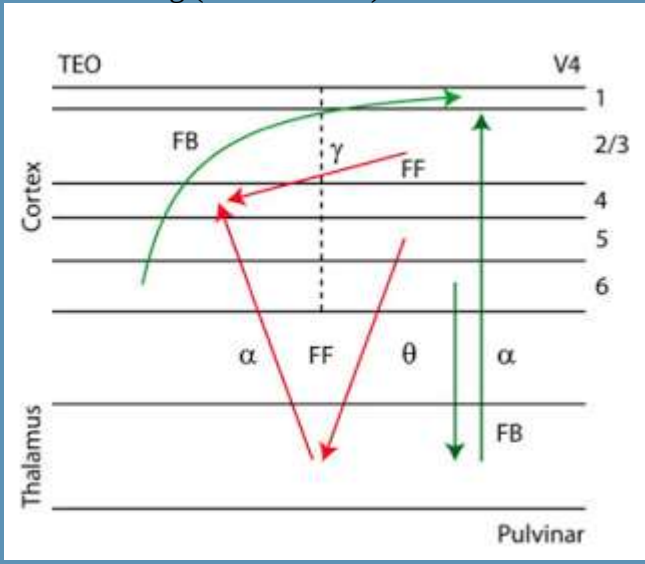


Location of the pulvinar relative to the rest of the visual pathways. This image depicts some of the visual pathway, but not the key connections studied in this paper.

That is where the current work comes in. The pulvinar area of the thalamus is sort of a transgressive part of the brain, sending connections to many higher cortical levels rather than residing within linear tracts of visual or other forms of processing. Indeed, most of the cortex maps to various and overlapping regions of the small and central pulvinar nuclei. And lesions in the pulvinar cause attention deficits. A decade ago, the pulvinar nuclei of the thalamus were proposed to play a role in this synchronization and binding for visual attention specifically:
"The scheme requires that multiple groups of neurons, distributed within and across separate areas, be capable of attaining synchronous firing by means of re-entrant circuitry (Tononi et al. 1992). It is by facilitating this process that the pulvinar could play a coordinating role in cortico-cortical communication."
"Ultimately, the synchronized neural assembly is proposed to mediate the perceptual binding of different object features (von der Malsburg & Schneider 1986; Tononi et al. 1992; Eckhorn 1994; Singer & Gray 1995). If the pulvinar is a key element of the assembly, damage to the

pulvinar should have a noticeable effect on feature binding. There is already some preliminary evidence in favour of this prediction, documenting one patient's report of illusory conjunctions of colour and letter form (i.e. 'misbinding') in the visual hemifield contralateral to a pulvinar lesion (Ward et al. 2002)." -From Shipp, 2003

So the current researchers carefully mapped the connections they were interested in, between the ventral stream of the visual pathway (occipital V4 to the temporal-occipital border area, called TEO) and the pulvinar area of the thalamus. Using amazing MRI/DTI imaging, they could do this individually for each monkey they experimented on, finding precisely where the nerve projections from the two cortical areas overlap in the pulvinar (a method called tractography). That is where they stuck their electrodes, taking electrophysiology to a new level of long-range circuit specificity. They had electrodes both in the overlap area in the pulvinar and in the originating locations in the V4 and TEO cortical areas, tracking precisely connected signals over long distances in a living (more or less!) brain.



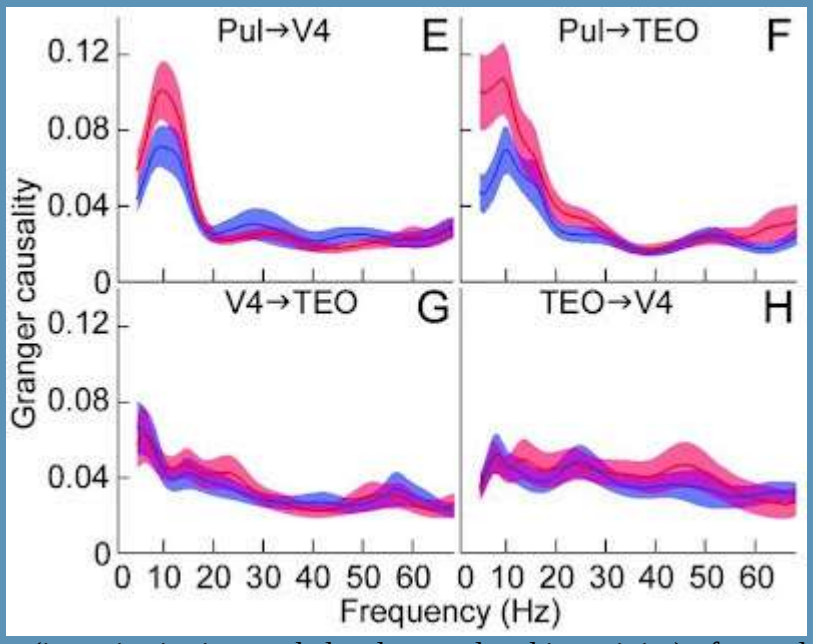
Mapped connections between relevant areas, in schematic terms. Red arrows are forward processing (feed forward, FF), going with the flow of visual information, while green arrows are feedback signals (FB). The numbers refer to the cortical layer being targeted, and the greek symbols refer to the frequency band of the nerve firing. TEO refers to the temporal/occipital boundary region which is part of late-stage visual processing. The experiments were oriented to detecting the alpha-band signals going from the pulvinar back into V4 and the TEO, and telling whether they have coordinating effects.

The point was then to test whether the pulvinar leads the synchronization of visual signals going between the cortical areas, as though it generates attention based either on rapid perceptions in lower areas of the visual pathway (say, suddenly seeing a snake/stick), or on signals from executive areas that exert voluntary attention control.

The task the macaques were set was to follow a spot on a video screen and rapidly report whether the subsequent shape at that spot was one of two possibilities, for a juice reward. In the experimental cases, the spot was engineered to happen in what the researchers knew was the receptive field (RF) for the neurons they had previously mapped and stuck with electrodes. So the question was whether the monkey's rapt attention (verified with infrared gaze tracking) raised the level of synchrony among the areas being recorded, and whether the pulvinar played a leading role in setting that synchrony.

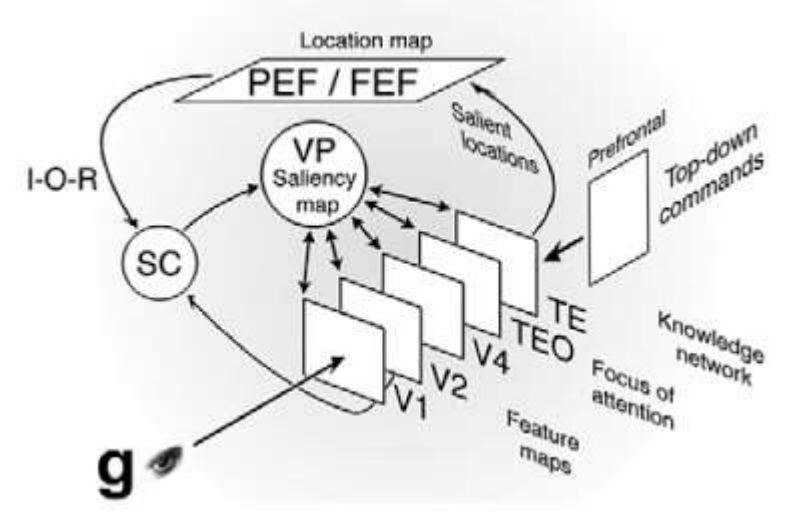
The authors present data showing that these visually connected pulvinar neurons fire more when the monkey is paying attention to their receptive fields. They also find that the frequency of this

firing is maximal in the alpha band around 10 Hz. Firing during attention was also closely correlated between the points they connected anatomically- the pulvinar and the V4 and temporal-occipital cortices. And finally, they use a relative timing method (Granger causality) to argue that, when the monkey was attending to the receptive field of the recorded neurons, and was in the attending period between presentation of the cue-spot and presentation of the puzzle shapes, neuron firing in the pulvinar significantly led the correlated firing in each cortical area it was tied to, not the reverse.



Inferred causality (i.e. prior in time and closely correlated in activity) of neural firing, between the tested areas of the visual system in macaque. Significant signals and causality in the alpha frequency band (10 Hz) are only seen going from the pulvinar (PUL) to the cortical visual processing areas V4 and TEO which are sequential in the usual sequence of visual feature processing. This supports the theory that the pulvinar drives attention-related synchronicity across the the visual processing system.

So one can ask where the regulatory decision of the pulvinar arises from. One hypothesis is that we have an immaterial soul that directs these things. Just kidding! As mentioned above, our attention may be involuntarily drawn by features of the scene (some visual pathways connect to the amygdala), or by a higher voluntary decision. In either case, the model would be that once a receptive field was decided on, the pulvinar helps pull the perceptual scene together by coordinating the firing of much or all of the visual pathway for that receptive field, and perhaps coordinating it as well with higher levels that receive that information.



Model (from Shipp, 2004) for attention in the visual system. The pulvinar (VP) receives signals and returns feedback from all areas of the linear/parallel visual feature processing pathway, governing which features or receptive fields are synchronized and thus attended to. The pulvinar receives input to drive its selection of what to attend to from both high level (prefrontal and parietal and frontal eye fields; PEF, FEF) and from low-level areas (the superior colliculus, SC and amygdala, not shown.

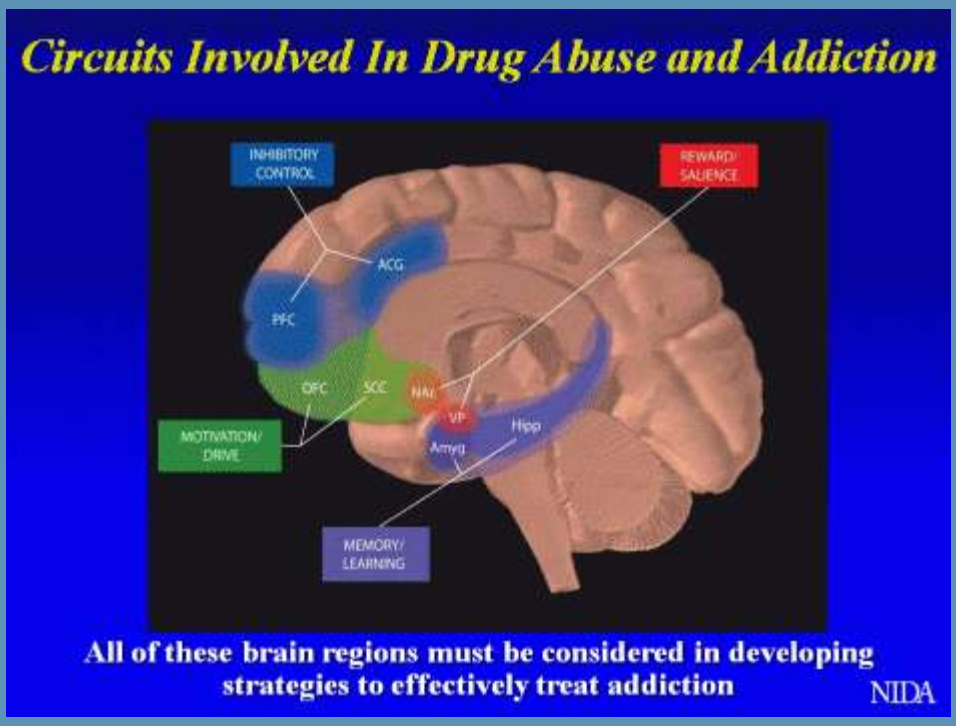
The alpha frequency band seems central to vision: *"Evidence suggests that the rhythmic excitability of alpha oscillations gates visual events, with the phase of the alpha oscillations being critical for the transmission of visual information."*

And, since consciousness seems more closely tied with the gamma band of higher frequency oscillations, the current authors add: *"Because low-frequency oscillations modulate higher-frequency oscillations, we tested whether attention increased cross-frequency coupling between alpha and gamma oscillations within V4 and TEO. To measure cross-frequency coupling, we calculated the synchronization index between cortical alpha oscillations and the gamma power envelope. Across the population, there was a significantly greater synchronization index for V4 and TEO during the delay period, when attention was directed to the RF location rather than outside the RF (sign tests, $P < 0.05$; fig. S3, A and B), suggesting that alpha oscillations contributed to the attention effect on gamma frequencies."*

So there you have it.. a few steps towards a physical theory to flesh out the "spotlight of attention" theory that has been an important subject of recent cognitive psychology and neurobiology, not to mention armchair philosophy.

Substance abuse and Addiction and the Chemical Imbalance

The areas depicted contain the circuits that underlie feelings of reward, learning and memory, motivation and drive, and inhibitory control. Each of these brain areas and the behaviors they control must be considered when developing strategies to treat drug addiction.



Key:
PFC – prefrontal cortex;
ACG – anterior cingulate gyrus;
OFC – orbitofrontal cortex;
SCC – subcallosal cortex;
NAc – nucleus accumbens;
VP – ventral pallidum;
Hipp – hippocampus;
Amyg – amygdala.

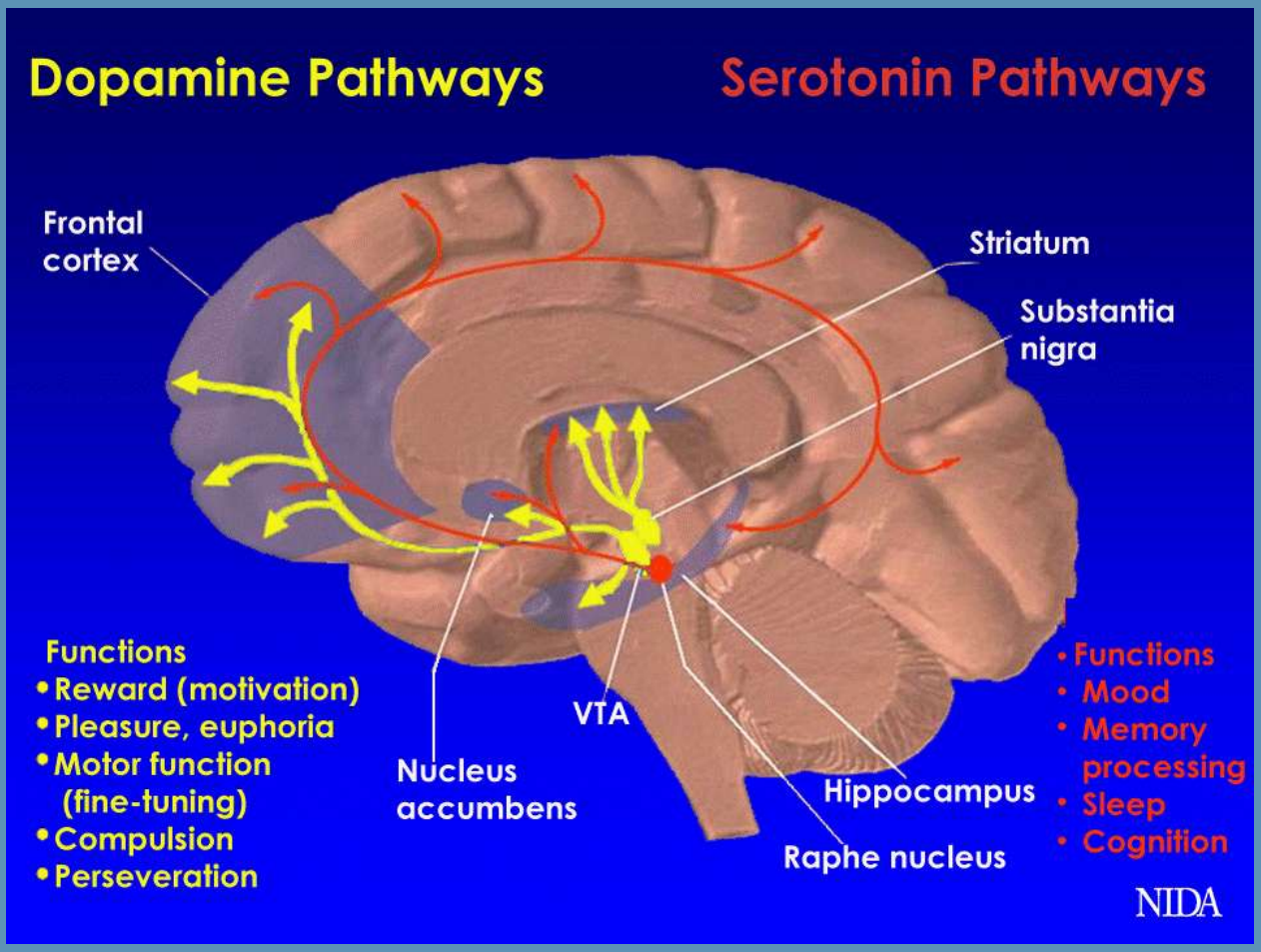
Low dopamine levels and lack of pleasure
How Does the Brain Become Addicted? *Typically it happens like this:*

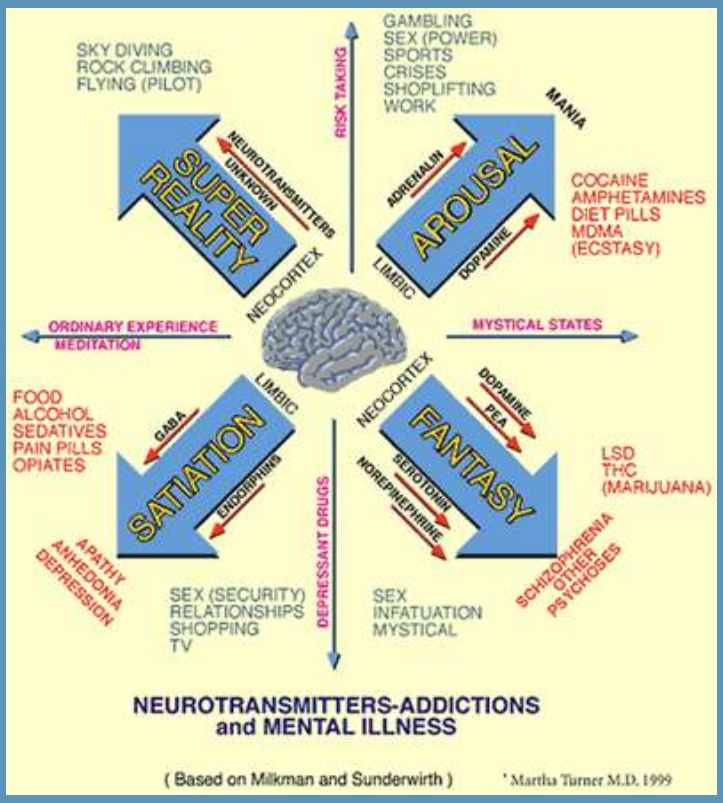
* A person takes a drug of abuse, be it marijuana or cocaine or even alcohol, activating the same brain circuits as do behaviors linked to survival, such as eating, bonding and sex. The drug causes a surge in levels of a brain chemical called dopamine, which results in feelings of pleasure. The brain remembers this pleasure and wants it repeated.

* Just as food is linked to survival in day-to-day living, drugs begin to take on the same significance for the addict. The need to obtain and take drugs becomes more important than any other need, including truly vital behaviors like eating. The addict no longer seeks the drug for pleasure, but for relieving distress.

* Eventually, the drive to seek and use the drug is all that matters, despite devastating consequences.

* Finally, control and choice and everything that once held value in a person's life, such as family, job and community, are lost to the disease of addiction.





1.*The neural circuits of the ventral tegmental area (VTA), the nucleus accumbens, and the prefrontal cortex are critical in learning both natural and drug reward behavior.*

Upon exposure to either a naturally rewarding stimuli or a drug, there is a increase in dopamine signaling from the ventral tegmental area (VTA) to the nucleus accumbens. The nucleus accumbens "perceives" this dopamine signal and measures the "goodness" of the agent or the natural reward based on the size of the dopamine release.

2.*Glutamate projections from the nucleus accumbens instruct the prefrontal cortex to remember the environment and behaviors which lead up to the occurrence of the "goodness".*

3. *In addiction, excess signaling of glutamate neurons in the prefrontal cortex stimulate the nucleus accumbens, triggering drug seeking behaviors at the expense of naturally rewarding or good behaviors.*

14. Neurotransmitters and Anxiety

Your brain responds directly to neurotransmitters – little chemicals inside your body that send messages to your brain about how you should feel, think, act, and more. Many neurotransmitters have been linked to anxiety, including:

- Serotonin
- GABA
- Norepinephrine

Even dopamine may play a role in anxiety, or at least have a calming effect on those already living with anxiety symptoms. Interestingly, too much or too little of any hormone may also effect anxiety in different ways. The problem is with balance. If your brain doesn't have enough serotonin, for example, it may cause you to experience anxiety symptoms.

When it comes to neurotransmitter production, the truth is that cause and effect are rarely known. It's often impossible to distinguish between poor neurotransmitter balance as a result of life experience, or poor neurotransmitter balance as a result of genetics. Both can occur in anyone living with anxiety, and in some cases a combination of both may be responsible for anxiety symptoms.

15. Anxiety and Brain Activation

There are two different parts to an anxiety disorder, and someone with anxiety may suffer from one or both. The first part is mental – verbal worries, nervous thoughts, etc. The second part of anxiety is physical. For example, a racing heartbeat, panic attacks, lightheadedness, and other physical symptoms.

It's possible to experience physical symptoms with less worry, and it's possible to worry often without many physical symptoms. Researchers also found that both of these excited different parts of the brain. Those with worried thoughts showed more left brain activity when nervous. Those with physical symptoms experienced more right brain activity.

Another study looked at the way that those with a spider phobia reacted to the belief that they were going to encounter a spider. They found that those with the phobia had their dorsal anterior cingulate cortex (ACC), insula, and thalamus become more active than those without a phobia.

Yet another study at the University of Wisconsin – Madison found that those with generalized anxiety disorder appeared to have a weaker connection between the white matter area of the brain and the pre-frontal and anterior cortex. This was compared to those without generalized anxiety disorder and the results appeared to be significant.

These are just some of the ways that anxiety can activate the brain.

16. Hormones and Anxiety

Hormone balances may affect anxiety as well. Many different hormones have an effect on brain chemistry and neurotransmitter production and balance, so if these hormones appear to be out of balance, anxiety may be the result.

Some examples of hormones affecting the brain include:

- **Adrenaline/Epinephrine** – Adrenaline is one of the most common causes of anxiety symptoms. Your body releases it when your fight or flight system is active, and it causes the increase in heart rate, muscle tension, and more. In some cases, long term stress and anxiety may damage your ability to control adrenaline, leading to further anxiety symptoms.
- **Thyroid Hormone** – Thyroid hormone appears to regulate the amount of serotonin, norepinephrine, and Gamma-aminobutyric acid (GABA) produced and distributed to the brain, so problems with your thyroid may also increase your risk for developing anxiety.

Several hormones may cause anxiety, and a change in brain chemistry may increase the production of hormones that lead to further anxiety symptoms.

17. Panic Attacks and the Brain

Panic attacks are a particularly distressing form of anxiety, and these may be due to the health of the brain too. Researchers have found that those with panic attacks often have an overactive amygdala. While it's not clear what creates this over activity, the fact that that area of the brain

appears to contribute to panic attacks indicates that some aspect of the brain is in control of the panic attack experience.

18. Other Links Between Anxiety and the Brain

Another interesting relationship between anxiety and the brain is that long term anxiety may damage the brain in a way that could cause further anxiety. Researchers have found that when you leave your anxiety disorder untreated, the dorsomedial prefrontal cortex, anterior cingulate, hippocampus, dorsolateral prefrontal cortex, and orbitofrontal cortex all appear to decrease in size. The longer the anxiety goes untreated, the smaller and weaker they appear to be.

What's interesting is that not only do these changes affect anxiety symptoms – they also create anxious thoughts. Those with anxiety may feel their thoughts are completely natural, when in reality the brain contributes to that type of negative thinking.

Schizophrenia

Schizophrenia is a complex brain disorder, it likely results from the interplay of genetic, behavioral, developmental and other factors. The exact cause of this group of illnesses is not known but stress, trauma and viral infection at an early age are factors thought to be involved. An imbalance in the concentrations of dopaminergic and glutamatergic systems in the brain is also thought to play a role in the development of schizophrenia. The dopamine hypothesis states that the behavioral patterns typical of schizophrenia are a result of overactivity of dopamine in certain regions of the brain. Serotonin is also important in schizophrenia and it may be that the serotonin system interacts with the dopamine system, modulating the way in which it operates. The serotonin receptors which are important in the treatment of schizophrenia are 5-HT1, 5-HT2 and 5-HT3.

The areas of the brain implicated in schizophrenia are the forebrain, hindbrain and limbic system.

It is thought that schizophrenia may be caused by a disruption in some of the functional circuits in the brain, rather than a single abnormality in one part of the brain. Although the brain areas involved in this circuit have not been defined, the frontal lobe, temporal lobe, limbic system,(specifically the cingulate gyrus, the amygdala and the hippocampus) and the thalamus are thought to be involved. The cerebellum, which forms part of the hindbrain, also appears to be affected in people with schizophrenia.

Several structural changes are found in the brains of people with schizophrenia, most of which occur in the forebrain. Reductions in the volume of grey matter in the frontal lobe, and decreased brain volume and activity, have been repeatedly noted among people with a schizophrenic disorder. The ventricles are commonly found to be larger than normal, as are the basal nuclei, while the hippocampus and amygdala are often smaller. The disease is also associated with alterations in blood flow to certain areas of the brain.

Neuroscientists have discovered abnormal neural activity in the brain that may cause people with schizophrenia to experience unorganized thought processes, according to a study published in the journal *Neuron*.

Neurochemical Abnormalities in Schizophrenia

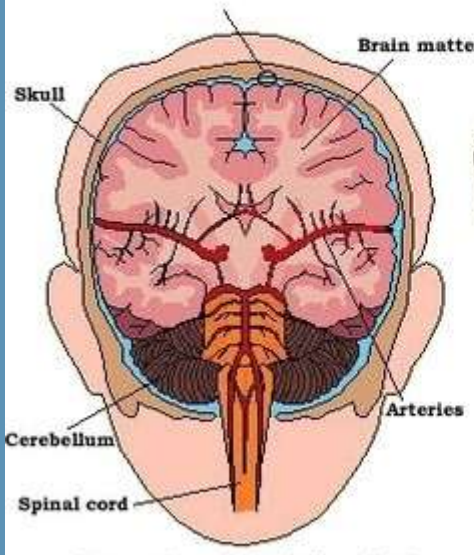
- Multiple neurotransmitters likely involved
- Dopaminergic abnormalities demonstrated, but abnormalities in other neurotransmitter systems likely as well
- Classic "dopamine model" of **too much dopamine somewhere or everywhere in the brain** is not fully explanatory

Carlsson et al. (1997), Life Sci 61:75-94

Area of the brain involved with schizophrenia

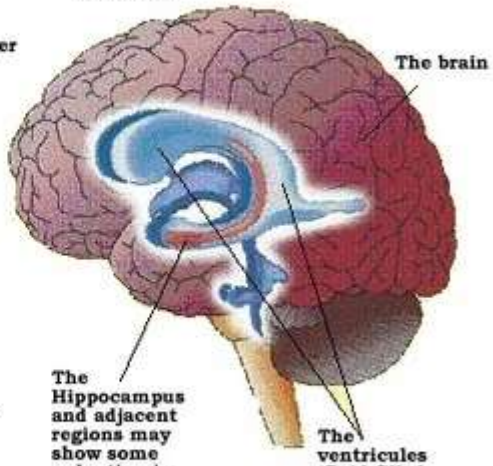
The cerebros pinal fluid (fluid surrounding the brain) may contain different relative amounts of chemicals associated with the transmitting of nerve impulses.

There may be changes in the frontal lobes, the part of the brain concerned with emotional and some higher mental functions.



Cross section of the skull and brain

(view from the front)



The Hippocampus and adjacent regions may show some reduction in size. This can affect the sensory filtering that takes place in this region of the brain.

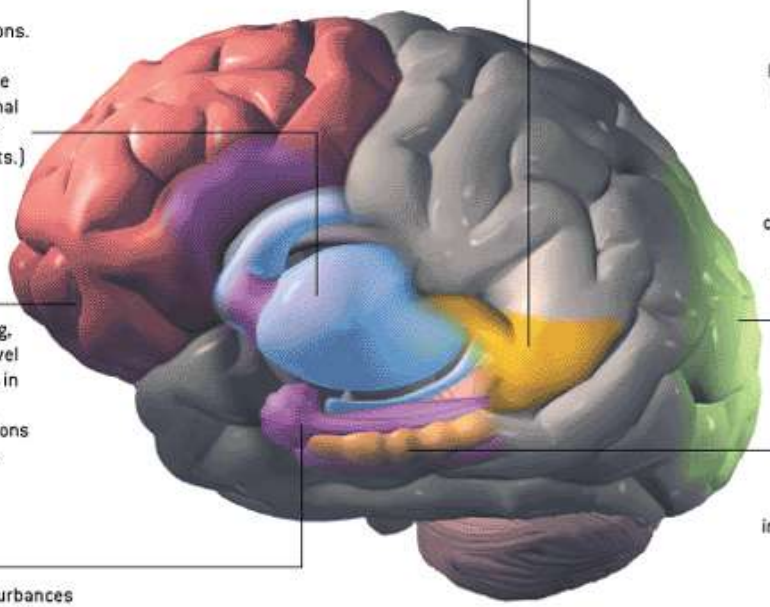
The ventricules (fluid-filled spaces) may be larger than normal. This may put pressure on surrounding brain tissue.

THE BRAIN IN SCHIZOPHRENIA

MANY BRAIN REGIONS and systems operate abnormally in schizophrenia, including those highlighted below. Imbalances in the neurotransmitter dopamine were once thought to be the prime cause of schizophrenia. But new findings suggest that impoverished signaling by the more pervasive neurotransmitter glutamate—or, more specifically, by one of glutamate's key targets on neurons (the NMDA receptor)—better explains the wide range of symptoms in this disorder.

BASAL GANGLIA
Involved in movement and emotions and in integrating sensory information. Abnormal functioning in schizophrenia is thought to contribute to paranoia and hallucinations. (Excessive blockade of dopamine receptors in the basal ganglia by traditional antipsychotic medicines leads to motor side effects.)

AUDITORY SYSTEM
Enables humans to hear and understand speech. In schizophrenia, overactivity of the speech area (called Wernicke's area) can create auditory hallucinations—the illusion that internally generated thoughts are real voices coming from the outside.



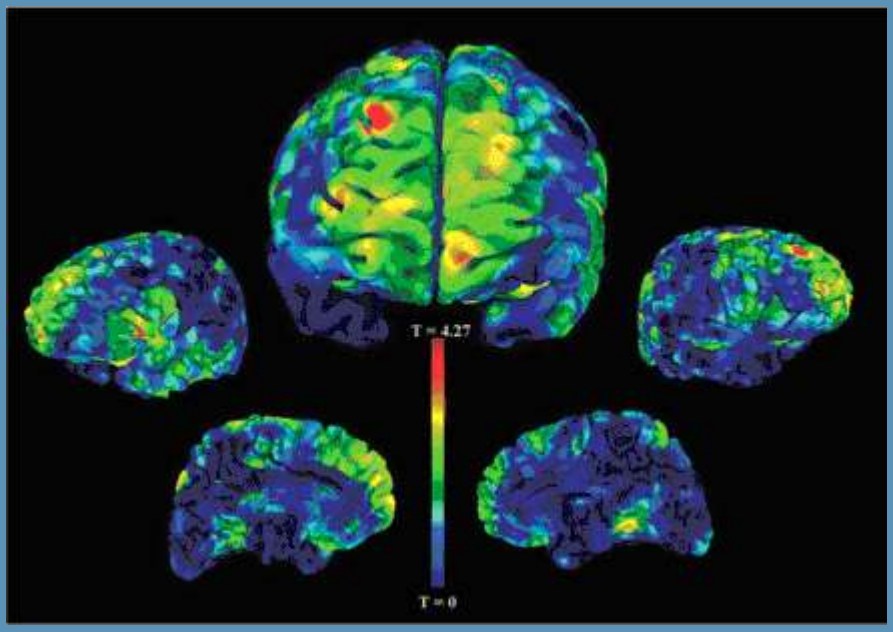
OCCIPITAL LOBE
Processes information about the visual world. People with schizophrenia rarely have full-blown visual hallucinations, but disturbances in this area contribute to such difficulties as interpreting complex images, recognizing motion, and reading emotions on others' faces.

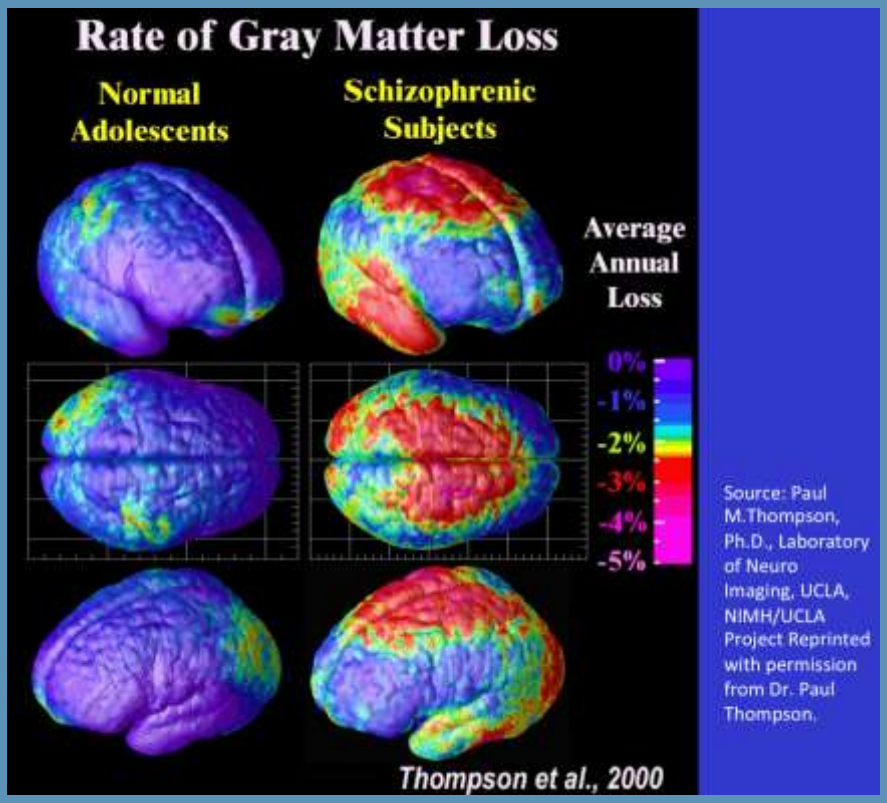
FRONTAL LOBE
Critical to problem solving, insight and other high-level reasoning. Perturbations in schizophrenia lead to difficulty in planning actions and organizing thoughts.

HIPPOCAMPUS
Mediates learning and memory formation, intertwined functions that are impaired in schizophrenia.

LIMBIC SYSTEM
Involved in emotion. Disturbances are thought to contribute to the agitation frequently seen in schizophrenia.

ALFRED T. KAMAJIAN





2. Brain Wave Activity Seems Slower in Schizophrenics

But some suggest the finding may be due to medication

Please note: This article was published more than one year ago. The facts and conclusions presented may have since changed and may no longer be accurate. And "More information" links may no longer work. Questions about personal health should always be referred to a physician or other health care professional.

By Randy Dotinga
HealthDay Reporter

THURSDAY, Nov. 11, 2004 (HealthDayNews) -- New research suggests that schizophrenics process information at a lower frequency than healthy people, almost as if their minds were filled with bad connections.

"It is like a glitch," said Dr. Robert McCarley, deputy chief of staff for mental health services at VA Boston Healthcare System. An "out-of-sync" brain leads to symptoms such as hallucinations and disorganized thinking, some of the characteristics of schizophrenia, he explained.

The findings could lead to better understanding of the mysteries of schizophrenia, a complex brain disorder that affects an estimated one in every 100 Americans.

However, one neurologist suggested the apparent dysfunctional brain activity detected in the study may simply be a product of medication, not the disease itself.

The study appears in this week's issue of the *Proceedings of the National Academy of Sciences*.

An estimated 2.2 million people in the United States struggle with schizophrenic. They lose touch with reality -- they see and hear things that aren't real, have trouble focusing and may become paranoid. In some cases, schizophrenics become unresponsive.

Using electroencephalogram (EEG) technology, McCarley and his colleagues scanned the brains of 20 healthy people and 20 schizophrenic patients. They studied the 40 people's responses when they looked at an optical illusion that forced them to create an imaginary square shape in their mind.

The researchers found that the neurons of the schizophrenic patients communicated less quickly than those of the healthy subjects. It took the schizophrenics about 200 milliseconds longer to process the optical illusions.

McCarley and colleagues also found that neurons in those with schizophrenia communicated at a lower frequency than in the healthy people. Neurons send different types of information to each other and help the brain piece together a variety of facts into one coherent whole.

"For example, if you see a fire truck, you hear the bell, you smell the motor, you see the red, you see the movement," McCarley said. "All of these sensations are registered in different parts of the brain. You put them together to get your picture of a fire truck."

The neurons of the brain communicate most effectively at a frequency of 40 cycles per second, McCarley said. But in the schizophrenic patients, they communicated at lower frequencies; they were lowest in the patients with the worst symptoms.

The difference in frequencies doesn't mean it takes longer for information to travel between neurons, McCarley said. Instead, it means the information transmission isn't very efficient. "It's as if your brain is not putting things together quite right," he said.

The next step is to figure out ways to use drugs to improve the brain's inner communication system in schizophrenic patients, McCarley said. "You look for possible reasons why these cells aren't communicating at the best frequency," he added. "It may be that we need a new generation of drugs."

However, not everyone agrees with McCarley's interpretation of the importance of brain waves in individuals with schizophrenia.

Dr. James Grisolia, a neurologist at Scripps Mercy Hospital in San Diego, said the brain waves of schizophrenics slow down because of the medications they take. Indeed, the schizophrenic patients

in the study were all on medication. But McCarley said the frequencies of the brain waves remained low, regardless of how much medication the patients were taking.

Grisolia added that the focus on brain waves misses the point. "The schizophrenic brain is different from the normal brain in ways that we still don't understand, but brain wave patterns will always be a sign of the problem, rather than the cause," he said.

More information

To learn more about schizophrenia, visit the National Institute of Mental Health.

SOURCES: Robert McCarley, M.D., deputy chief of staff, mental health services, VA Boston Healthcare System, and professor, psychiatry, Harvard Medical School, Boston; James Grisolia, M.D., neurologist, Scripps Mercy Hospital, San Diego; Nov. 8-12, 2004, *Proceedings of the National Academy of Sciences*