The Brain and Botanicals
Introduction

Generally speaking, there are two physiological systems that share responsibility for maintaining homeostasis within the body: the **endocrine system** and the **nervous system**. The **endocrine system** maintains control by releasing substances called hormones that travel through the blood to target specific receptors. The **nervous system** exerts its influence by the transmission of electrical impulses through nervous tissue. The **brain** is an interesting blend of these two systems, where both function and communicate with each other.

The nervous system has three basic functions:

1. **Sensory**: sensing certain changes (stimuli) within the body (e.g. a change in the pH of the blood or stretch receptors in the gut) and without (such as the sensation of a raindrop or the sun shining on one’s arm)
2. **Integrative**: analyzing sensory information, stores information and makes decisions regarding behavior
3. **Response**: responding to stimuli by initiating muscular contraction or glandular secretions

**Consciousness**

When discussing consciousness, there are many models that one can look at. The prominent bio-medical model explains consciousness (both mental and emotional) as being a subset of brain function. Although some herbalists prescribe to this model, many more have adopted a vibrational medicine model.

**The vibrational medicine model** shows that consciousness itself is not just a by-product of physical and biochemical mechanisms, but it also plays a primary role in both physical health and physical disease. While biomechanical medicine has been looking for the part of the brain that controls consciousness and thought, vibrational medicine accepts that consciousness takes place in an entirely different energy field. To illustrate this point, think of a radio. When you hear Beethoven’s fifth symphony on the radio you wouldn’t take the radio apart to try and find
Beethoven inside. Well, vibrational medicine looks at the brain like a radio receiver that receives information from the mental body. We are not simply sophisticated meat machines, with a bio-computer brain and nervous system; we are conscious beings with a physical body that is but one dimension of our existence. Our emotional ‘body’, our mental ‘body’ and our spiritual ‘bodies or fields’ all influence us.

The vibrational medicine model has a separate emotional (astral) body and up to 3 separate mental bodies. These bodies communicate into the endocrine and nervous system of the physical body via an etherical body that includes chakras, acupuncture points. Many of the health issues that we will discuss in this lesson arise from the level of consciousness, outside the physical body. Understanding the communication systems between the bodies is just as important as the biomechanical aspect of it.

Gaia Hypothesis

Many Herbalists prescribe to the Gaia hypothesis that consciousness is an electromagnetic field effect between the planet and us; including all the other organisms that live in this field. We can easily say that Herbalists have a direct interaction between humans, planet and Earth. Since all of these, including the Earth are living fields, it is the interaction between these that stimulates consciousness. There is no doubt that some botanicals, fungi and animals can have profound effect on consciousness. Does this mean that these reactions are all biochemical or electromagnetic? This is a question to take into consideration when looking at various traditional medicines from around the world.

Many indigenous people have a variation of this hypothesis, projecting super natural, spiritual or esoteric meanings on these interactions.

Some people proscribe that consciousness and the sleeping cycles and/or dreaming have to do with the rotation of the planet, as it journeys around the sun and the solar system through the galaxy. Yet we know that these rotations create an electromagnetic field similar to the way electricity is created from a generator.
Scope

Botanical medicine, while providing non-specific support for recuperation and repair, can have specific action as part of a strategy to treat several ailments pertaining to the functioning of the brain, including:

- Insomnia
- Depression
- SAD (Seasonal Affective Disorder)
- ADD/ADHD (Attention Deficit Disorder/Attention Deficit Hyper-activity Disorder)
- Autism
- Parkinson’s disease
- Alzheimer’s disease
- Epilepsy

Herbalists should be cautious when treating cases that involve:

- Severe psychosis
- Patients on strong antipsychotic, antiepileptic, and anesthetic prescriptions
- Addictive personalities

Many of the botanicals that are used in the area of the brain have chemicals that work as, or mimic, neurotransmitters. These chemicals are often quite strong, functioning on both the nervous systems and the endocrine systems.

Psychoneuroimmunology (PNI) is the scientific discipline that involves health issues having psychological, neurological and immunological aspects. This is of course an important area to understand when treating some of these conditions. We will be looking at the immune system in another lesson (9. Herbal Immunity: Nonspecific resistance, Immunity and Botanical medicine.) But suffice to say that neuro-transmitters and attitude, in the realm of this lesson, do play a huge role in immunity and general health. A positive attitude is one of the important foundations for good health.

Depression and the harboring of negative emotions have been shown to increase risk of cancer in several ways. We will discuss this in more detail in the lesson on cancer (11. Meeting Life: Cancer, Immunodeficiency, Death and
Botanical Medicine). Depression has a negative effect on natural killer cells, hindering the cell’s ability to repair DNA. A positive attitude is one of the most important strategies for good health.

In addition to the brain, the immune system and immunity, the cardiovascular system is closely linked to emotion and attitude.

### Neuroanatomy and physiology

Material from this section can be found in your mandatory text: *Principles of Anatomy & Physiology* by Tortora and Grabowski.

**Nervous tissue**
Organization of the nervous system

The nervous system consists of two principal divisions, the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS is comprised of the brain and spinal cord and its function is to integrate and correlate all incoming sensory information. Medical science considers the CNS to be the repository of the mind, emotion and memory. Holistic medicine presents other models to interpret how mind, emotions and memory are stored and transmitted. The CNS is connected to sensory receptors, muscles and glands in the peripheral nervous system. The PNS consists of cranial nerves that arise from the brain and spinal nerves that emerge from the spinal cord. The input component of PNS consists of nerve cells called sensory or afferent neurons. These conduct nerve impulses from sensory receptors in various parts of the body to the CNS. The output component of the CNS consists of nerve cells (called motor or efferent neurons), originating in the CNS. They conduct nerve impulse from the CNS to muscles and glands.

The PNS can be further subdivided into the somatic (SNS) and the autonomic (ANS) nervous systems. The SNS consists of sensory neurons that convey information from cutaneous and special sense receptors (smell, taste, vision, touch, equilibrium and hearing) primarily from the head, body wall and extremities to the CNS. These impulses are then integrated by the CNS and conducted by motor neurons from the CNS to skeletal muscles only. The SNS is (mostly) consciously controlled and thus voluntary. A single nerve that travels from the CNS to the skeletal muscle, without interruption, directs skeletal muscles. Therefore, stimulation in the SNS is an “all or none” phenomenon. The ANS consists of sensory neurons that convey information from receptors found primarily in the viscera to the CNS, which are then integrated and conducted to motor neurons in smooth muscle, cardiac muscle and glands. The ANS is (mostly) unconsciously controlled and thus involuntary. Effectors are directed by the ANS through a network of nerves that arise from the CNS. The activation, therefore, of autonomic effectors is modified by the connection between these networks (i.e. by the synapses).
The ANS consists of two further subdivisions, the sympathetic and parasympathetic divisions. The viscera receive instructions from both the sympathetic and parasympathetic divisions, which have opposing actions. The parasympathetic division is the energy conservation-restorative system, regulating those activities that conserve and restore body energy during times of rest and recovery. Parasympathetic responses include salivation, lacrimation, urination, defecation and a decrease in heart rate. The sympathetic division, on the other hand, prepares the body for emergency situations, concerned primarily with processes involving the expenditure of energy. Activation of the sympathetic aspect of the nervous system generally results in setting in motion a set of physiological responses called the fight or flight response.

**Histology of nervous tissue**

Within nervous tissue there are two principle kinds of cells, neuroglia and neurons. **Neuroglia:** support, nurture and protect the neurons; generally smaller than neurons, outnumber them by 5 to 50 times; able to multiply and divide in mature nervous systems; some produce the **myelin sheath** (a multi-layered lipid and protein covering
that electrically insulates the axon of the neuron, increasing the speed of nervous conduction). There are four types of neuroglia found in the CNS, including astrocytes, oligodendrocytes, microglia and ependymal cells; specific function: protecting neurons and forming the blood-brain barrier (BBB), to digesting foreign pathogens and cellular debris. There are two types of neuroglia within the PNS, neurolemmocytes (Schwann cells), which produce the myelin sheath around PNS neurons, and satellite cells, which support neurons in ganglia of the PNS.

Neurons: three parts, a cell body, a dendrite and an axon. Cell body: nucleus surrounded by cytoplasm, responsible for the metabolism of the neuron. Dendrites: extends outward from the cell body, often tree-shaped and unmyelinated, conducting impulses toward the cell body. Axons: extends outward from the cell body, may or may not be myelinated, conducting impulses away from the cell body; junction between the cell body is a cone-shaped elevation called the axon hillock, nerve impulses can arise at this junction, called the trigger zone, conducted along the axon to another neuron, muscle fiber or gland cell. At the terminal end of the axon the tip swells into a bulb-shaped structure, the synaptic end-bulb, and contains synaptic vesicles that contain neurotransmitters.
Synapse: functional contact between two neurons, between a motor neuron and a muscle fiber is a **neuromuscular junction**, between a neuron and glandular cells is called a **neuroglandular junction**.

**Classification of neurons**

Neurons: **unipolar**, having just one process that is a fusion of a dendrite and an axon, specialized to monitor environmental changes, sometimes aided by receptors and impulses that arise at the first neurofibral node; **bipolar**, having one dendrite and one axon, typically found in the retina of the eye, the inner ear and within the olfactory region of the brain; or **multipolar**, having several dendrites and a single axon, found in the brain and spinal cord.

Functional classification: based upon the direction in which neurons transmit impulses. **Afferent neurons** transmit sensory impulses from receptors in the skin, sense organs, muscles, joints and viscera to the spinal cord and brain. **Efferent neurons** convey motor nerve impulses from the brain and spinal cord to effectors, which may be either muscles or glands. A third type of neuron are **association neurons**, which carry nerve impulses from one neuron to another, and make up 90% of all neurons in the body.

Within the spinal cord and brain there is a differentiation of neuronal tissue based on colour. **White matter**: aggregations of myelinated processes from several neurons, most nerve cells in the PNS and all tracts in the CNS. **Gray matter**: unmyelinated axons and neuroglia. Within the spinal cord, the white matter surrounds the gray matter, whereas in the brain, the gray matter is a thin outer shell around the brain that forms the two cerebral hemispheres.

**Neurophysiology**

Communication between neurons is dependent upon two basic properties of their plasma membranes, the **resting membrane potential (RMP)** and **ion channels**. A cell that exhibits a membrane potential is said to be **polarized**. Ion channels open and close, allowing the transmembrane flow of ions to regulate the membrane potential. There are two types of ion channels, **leakage channels**, which are always open, and **gated channels**, which only open and close in response to some sort of stimuli. Stimuli could be changes to the RMP, the binding of ligands such as...
neurotransmitters and hormones, mechanical pressure, vibration and even light.

An action potential or nervous impulse is the depolarization of adjacent areas of the plasma membrane along an axon, much like a domino effect. In unmyelinated axons there is a step-by-step depolarization of adjacent areas of the plasma membrane, called continuous conduction. In myelinated axons the plasma membrane is insulated against ionic currents and membrane depolarization occurs at intervals along the axon that interrupt the myelin sheath, called nodes of Ranvier. At these locations the nervous impulse is propagated along a myelinated axon by the ionic current that flows through the axoplasm from one node to the next. This apparent jumping of the nervous impulse from one node to the next is called saltatory conduction (saltare (F) = to leap).

Transmission at synapses

A synapse is the area between two neurons, or between a neuron and an effector, filled with extracellular fluid. The neuron that sends the signal is the presynaptic neuron and
the neuron receiving the message is the **postsynaptic neuron**. There are two kinds of synapses, electrical and chemical. Electrical impulses are sent through gap junctions, minute fluid-filled tunnels between the plasma membranes of each neuron. Electrical synaptic transmissions are faster than chemical synapses, and can provide two-way communication between cells, synchronizing the activity of a group of neurons or effectors. Chemical synaptic transmission requires the presynaptic neuron to convert the electrical signal into a chemical one, called a **neurotransmitter** resulting in a one-way communication.

The advantage that chemical transmission offers is that these neurotransmitters may be **excitatory**, causing the depolarization of postsynaptic neuron, or **inhibitory**, causing the hyperpolarization of the postsynaptic neuron.

The removal of the neurotransmitter from the postsynaptic neuron, and therefore the inhibition of further neural activity, is a process of enzymatic degradation (e.g. acetylcholinesterase). This removal of the neurotransmitter can also take place through the reuptake of the transmitter by the presynaptic neuron. Many drugs can cause the neurotransmitter to remain in the synaptic cleft for a longer period of time, by either interfering with enzymatic degradation or with presynaptic uptake. An example is the activity of cocaine that inhibits the presynaptic reuptake of dopamine, causing dopamine to linger longer in the synaptic cleft, stimulating brain centers.

Exogenous agents that facilitate the activity of a neurotransmitter are agonists, whereas exogenous agents that inhibit the activity of a neurotransmitter are antagonists.

**Neural circuits**

The CNS contains billions of neurons organized into complicated patterns, called neuronal pools. A neuronal pool may contain thousands or even millions of neurons. Neuronal pools are arranged in **circuits**, and are of four basic types: divergence circuits (single presynaptic neuron that synapses with many postsynaptic neurons), convergence circuits (several presynaptic neurons that synapse with a single post synaptic neuron), reverberating circuits (when a presynaptic cell is stimulated it will cause
the postsynaptic cell to transmit a series of impulses) and parallel-after-discharge circuits (single presynaptic cell that stimulates a group of neurons, each of which synapses with a common postsynaptic cell).

The Brain

The brain is made up of about 100 billion neurons and weighs about 1.3 kg. It is vaguely mushroom-shaped, and can be divided into four principle parts: the brain stem (the stalk of the mushroom), the diencephalon (above the brain stem), the cerebrum (the cap of the mushroom), and the cerebellum (behind the brain stem). The brain stem is continuous with the spinal cord, consisting of the medulla oblongata, pons, reticular formation and midbrain. The diencephalon consists of the thalamus and hypothalamus. The cerebrum spreads over the diencephalon, occupying most of the cranium and contains the cerebral cortex, limbic system and basal ganglia. The cerebellum consists of the anterior, posterior and flocculonodular lobes.

The brain is protected externally from injury by the cranial bones (the skull) and the cranial meninges. Both the brain and the spinal cord are bathed and protected against chemical and physical injury by a nourishing fluid called the cerebrospinal fluid (CSF); (80-150 ml) essentially, the brain and spinal column float within this fluid, delicate
balance of nutrients that provide an optimal environment for neuronal functioning, and even slight alterations in its composition can affect nervous transmission; a medium of exchange between the nutrients needed, and the wastes produced, by the brain.

**Blood supply to the brain**
The brain is well supplied with oxygen and nutrients, and consumes about 20% of the total serum O₄ at rest, and up to 25% of the total serum glucose. The brain is protected by a barrier called the **blood brain barrier (BBB)**, which prevents the entry of most substances into the brain. The BBB however, is completely permeable to glucose, O₂, CO₂, water, most lipid soluble substances such as alcohol, caffeine, nicotine, heroin and anesthetics, and partially soluble to substances such as creatinine, urea and most ions (including Na⁺, K⁺ and Cl⁻). Other substances such as proteins and antibiotics are inhibited from passing through the BBB, and thus the direct treatment of the brain is difficult.

**Brain stem**
The **brain stem** is comprised of the medulla, pons, reticular formation and the midbrain. **Medulla:** continuous with the spinal cord, forming the inferior part of the brain stem; contains all of the ascending and descending tracts to and from the brain; most of the tracts cross over from one side to the other within the medulla; relay motor and sensory impulses between other parts of the brain and spinal cord; vital reflex centers regulate the heart beat, breathing (with the pons) and blood vessel diameter; contains reflex centers that coordinate swallowing, vomiting, coughing, sneezing and hiccupping; contains the nuclei of origin for cranial nerves VIII (hearing and equilibrium), IX (swallowing salivation and taste), X (the vagus nerve, relaying impulses to and from the thoracic region and viscera), XI (head and shoulder movements) and XII (tongue movements).

**Pons:** directly above the medulla and serves as a bridge, relaying impulses between the spinal cord and the higher brain. Along with the medulla, the pons maintains the pneumotaxic and apneustic area to control respiration; the nuclei of origin for cranial nerves V (chewing and facial sensation), VI (eyeball movements), VII (taste, salivation and facial expression) and VIII (equilibrium).
Reticular formation: small areas of gray matter interspersed among fibers of white matter, extending into the diencephalon and the spinal cord; both sensory and motor functions, receiving input from higher parts of the brain that control skeletal muscles, regulates muscle tone and alerts the cortex of incoming sensory signals; contains the reticular activating system (RAS), maintaining consciousness and awakening from sleep, activated by sensory stimuli (eyes, ears and skin) and body position.

Midbrain: (mesencephalon) lies directly above the pons and relays motor impulses from the cerebral cortex to the pons and spinal cord and relays sensory impulses from the spinal cord to the thalamus; ventral portion of midbrain is composed of the cerebral peduncles, containing nerve tracts that connect the upper parts of the brain with the lower parts; posterior to the cerebral peduncles is the substantia nigra, which controls unconscious muscle movement; dorsal portion of the midbrain is called the tectum, and is comprised of four parts; superior colliculi is located in the dorsal regions of the midbrain, and serves as a reflex center for movements of the eyes, head and neck in response to sensory stimuli; inferior colliculi is located below the superior colliculi, serving as a reflex center for movements of the head and trunk in response to auditory stimuli; left and right red nuclei function with the basal ganglia and cerebellum to co-ordinate muscular movement. The midbrain also contains nuclei of origin for cranial nerves III (eyeball movement and changes in pupil and lens shape) and IV (eyeball movement).

Diencephalon
Comprised of the thalamus and the hypothalamus.

Thalamus: oval structure above the midbrain; 80% of the total mass of the diencephalon; relay all sensory input to the cerebral cortex, including hearing (medial geniculate), vision (lateral geniculate), taste and somatic sensations such as touch, pressure, vibration, heat, cold and pain (ventral posterior nuclei). Other nuclei within the thalamus are synapses in the somatic motor system, the ventral lateral and ventral anterior nuclei mediating voluntary motor actions and arousal, and the anterior nucleus functioning in some aspects of emotion and memory.
Hypothalamus: is located below thalamus, and can be divided into the mammillary, tuber al, supraoptic and preoptic regions. The mammillary region is the posterior portion of hypothalamus, consisting of the mammillary bodies and the posterior hypothalamus. The mammillary bodies serve as relay stations in reflexes related to smell and controls feeding reflexes, and the posterior hypothalamus controls blood pressure, pupillary dilation and shivering. The tuberal region is located in the middle portion of hypothalamus, and consists of the dorsomedial, perifornical, ventromedial and arcuate nuclei. On the ventral surface of the tuberal region is the tuber cinereum, a mass of gray matter that connects to the infundibulum, a stalk-like structure that connects the hypothalamus to the pituitary gland. The medial eminence of the tuber cinereum encircles the site where the infundibulum becomes the stalk of the pituitary gland and contain neurons which synthesize the hypothalamic regulating hormones, regulating hormonal secretions of the anterior pituitary gland (i.e. hGH, prolactin, ACTH, MSH, TSH, FSH and LH). The supraoptic region lies above the optic chasm (point of crossing of the optic nerves), anterior to the tuberal region, and contains nerve fibers that extend from the paraventricular and supraoptic nuclei to form the supraopticohypophyseal tract, extending into the
infundibulum to the posterior pituitary, transporting ADH and oxytocin. The preoptic region, anterior to the supraoptic region, regulates autonomic activities in association with the rest of the hypothalamus.

Sensory input into the hypothalamus comes from afferent pathways in the somatic and visceral sense organs, and it is the task of the hypothalamus to coordinate responses to these stimuli.

The major functions of the hypothalamus include:

1. The control and integration of activities of the ANS (e.g. smooth and cardiac muscle contraction, glandular secretion, heart rate, movement of food through the GIT, contraction of the urinary bladder)
2. Rage and aggression responses
3. Regulation of body temperature
4. Regulation of food intake through the inhibitory activity of the satiety center
5. Regulation of thirst, stimulated by the rising osmotic pressure in the extracellular fluid
6. Assisting in the coordination of arousal and sleep patterns.
The cerebrum is located superior to the diencephalon, and although it is the largest portion of the brain, relatively little is known about its function. The surface is composed of gray matter called the cerebral cortex, and lying under the cerebral cortex is the cerebral white matter. During embryonic development the gray matter enlarges much faster than the underlying white matter, and as a result the cortical region rolls and folds upon itself. These folds are called gyri or convolutions, the deep grooves between the gyri are called fissures, and the shallower grooves are called sulci.

The most prominent fissure is called the longitudinal fissure, and divides the cerebrum into the left and right hemispheres. These hemispheres are connected internally by white matter that forms a large bundle of transverse fibers called the corpus callosum. The cerebral hemispheres are further divided into four lobes, named after the bones that cover them, and are the frontal, parietal, temporal and occipital lobes. The central sulcus separates the frontal lobe from the parietal lobe. A major gyrus, called the precentral gyrus, is located immediately anterior to the central sulcus and is the primary motor area of the cerebral cortex. The postcentral gyrus is located immediately posterior to the central sulcus and contains the primary somatosensory area of the cerebrum. The lateral cerebral sulci divide the frontal lobe from the temporal lobes. The parietooccipital sulcus separates the parietal lobe from the occipital lobe. A fifth region is located inside the cerebrum and cannot be seen externally, called the insula or Isle of Reil.

The white matter of the cerebrum consists of myelinated axons that extend in three principle directions. They are called the association (fibers connect and transmit nerve impulses between gyri in the same hemisphere); commissural (fibers transmit impulses from gyri in one hemisphere to the corresponding opposite gyri in the other hemisphere); and projection (fibers form ascending and descending tracts that transmit impulses to and from the cerebrum to other parts of the brain and spinal cord).

The cerebral cortex contains three types of functional areas: sensory, motor and association. Sensory areas are
located in the posterior half of the hemispheres, and can be divided into two types of areas, primary and secondary. Primary sensory areas have a direct connection with peripheral sensory receptors, whereas secondary sensory and sensory association areas lie adjacent to the primary sensory areas, receiving input from the primary sensory areas and from other parts of the brain. The secondary areas participate in the interpretation of sensory experience into meaningful patterns of recognition and awareness. Motor areas control muscular movement, and flow primarily from the anterior portions of each hemisphere.

Association areas of the cerebral cortex consist of association tracts that receive and analyze signals from multiple regions of the cortex, as well as from other parts of the brain, and are comprised of the parieto-occipitotemporal, prefrontal and limbic association areas. The parieto-occipitotemporal association area is responsible for the continuous analysis of the spatial coordinates of all parts of the body, as well as the surroundings of the body. It assists the brain in controlling body movements and analyzing incoming sensory signals.

Perhaps the most important area within the parieto-occipitotemporal association area is Wernicke’s area, a confluence of somatic, visual and auditory secondary and association areas, located in the posterior portion of the superior temporal lobe. Wernicke’s area is highly developed in the dominant hemisphere of the brain, and plays the single greatest role of any part of the cerebral cortex in the higher levels of brain function called intelligence. Areas adjacent to Wernicke’s area assist in reading comprehension and the capacity to name objects. The prefrontal association area functions in close association with the motor cortex in planning complex patterns and sequences of motor movement. It contains Broca’s area in the frontal cortex, which provides the neural circuitry for word formation, where the plans and motor patterns for the expression of individual words and short phrases are initiated. The limbic association area is connected to the limbic system, and is concerned with behaviour, motivation and emotion.

Lateralization of the cerebrum
Brain lateralization refers to subtle anatomical and obvious physiological differences between the left and right
The left hemisphere controls the right side of the body, written and spoken language, numerical skills, scientific skills and reasoning. The right hemisphere controls the left side of the body, as well as musical and artistic awareness, space and pattern perception, insight, imagination and the generation of mental images to compare spatial relationships. About 95% of the population display a left brain dominance, in which the Wernicke’s area is as much as 50% larger than in the right hemisphere at birth. Thus, the left hemisphere gets a head start in its development because of the tendency to direct one’s attention to the better developed hemisphere. Broca’s area is often larger in the dominant hemisphere as well. The remaining 5% of the population either develops both sides simultaneously, or more rarely, the right side alone is more highly developed.

The basal ganglia are several groups of nuclei in each cerebral hemisphere that coordinate gross automatic muscle movement and regulate muscle tone. The largest nucleus of the basal ganglia is the corpus striatum, consisting of the caudate and lenticular nuclei. The lenticular nucleus is further subdivided into the putamen (laterally) and the
The basal ganglia is a group of interconnected nuclei in the brain that play a critical role in the regulation of voluntary movement. It consists of the globus pallidus, subthalamic nucleus, substantia nigra, putamen, and caudate nucleus. The basal ganglia are involved in the control of both voluntary and involuntary movements, as well as in the regulation of pathologic movements such as tremors and chorea.

The putamen circuit is responsible for executing learned, complex patterns of motor activity, such as writing letters of the alphabet and other skilled movements. It receives input mainly from those parts of the brain adjacent to the motor cortex, but not from the primary motor cortex itself. The putamen circuit then outputs to the primary motor cortex. When the putamen circuit is damaged, the cortical system of motor control can no longer provide these patterns and the writing is crude, as if one were learning for the first time. Lesions of the globus pallidus lead to spontaneous writhing movements of the limbs, neck or face, called athetosis. Lesions in the subthalamus lead to flailing movements of an entire limb, called hemiballismus. Multiple lesions in the putamen lead to flicking movements of the limbs, neck and head, called chorea. Lesions of the substantia nigra lead to paralysis agitans (e.g. Parkinson’s disease).

The caudate circuit of the basal ganglia plays a major role in the cognitive control of motor activity. It receives input from all lobes of the cortex, including information from the associative areas, and outputs to the areas adjacent to the motor cortex, to accessory motor areas concerned with patterns of movement instead of individual muscle movements.

The interplay of neurotransmitters (discussed later) within the basal ganglia include a dopamine pathway from the substantia nigra to the caudate nucleus and putamen, a GABA pathway from the caudate nucleus and putamen to the globus pallidus and substantia nigra, and an acetylcholine pathway from the cortex to the putamen and caudate nucleus. Both dopamine and GABA function as inhibitory agents, lending stability to motor control systems, while acetylcholine is excitatory to motor control. The basal ganglia also receives multiple, general pathways from the brain stem that secrete serotonin, enkephalin and norepinepherine.
The **limbic system** is a ring of structures below the basal regions of the cerebrum that surround the brain stem, and is not so much an individual structure as aspects of several brain structures *that appears to* control emotional behavior and motivational drives. The major structure of the limbic system is the **hypothalamus**, which serves as the main communication link between the other structures of the limbic system and the rest of the brain. Other major structures include the limbic lobe (parahippocampal and cingulate gyri, the hippocampus), the dentate gyrus, the amygdala, the septal nuclei, the anterior nuclei of the thalamus and the olfactory bulb. Aside from the vegetative and endocrine function of the hypothalamus, stimulation or lesions of the hypothalamus can have profound effects on behaviour. The stimulation of the lateral hypothalamus increases general activity levels, sometimes leading to rage and aggression, as well as hunger and thirst. Lesions of the lateral hypothalamus can lead to extreme passivity, a lack of desire for food or drink, with a generalized loss of motivation. Stimulation of the ventromedial nucleus can cause the opposite reaction to stimulation of the lateral hypothalamus, including tranquility and satiety. Lesions of the ventromedial nucleus are opposite to those of the lateral...
hypothalamus, increasing the desire to eat and drink, increasing activity levels and feelings of extreme rage and aggression. Stimulation of the thin zone of the periventricular nucleus can lead to fear and punishment reactions. Stimulation of areas within the anterior and posterior regions of the hypothalamus can stimulate sexual activity.

The limbic system is concerned with the affective nature of sensory experience, the determination of whether these sensations are pleasant or unpleasant. Researchers have identified certain areas in the limbic system that they associate with sensations of reward, punishment, learning and rage. The areas that are associated with pleasure and reward are the lateral and ventromedial nuclei areas of the hypothalamus. However, excessive stimulation of the lateral nuclei seems to produce feelings of rage and aggression. Other areas of secondary importance in pleasure and reward include the amygdala, and certain areas of the thalamus and basal ganglia. Prolonged stimulation of these centers for 24 hours or more can cause severe sickness and even death. The stimulation of these regions also seems to inhibit the reward centers. The most potent areas associated with punishment are the central gray areas surrounding the aqueduct of Sylvius in the midbrain, and the periventricular areas of the hypothalamus and thalamus. Other, less potent areas include regions in the amygdala and the hippocampus. Strong stimulation of punishment centers in the periventricular nucleus and reward centers in the lateral nuclei of the hypothalamus can lead to a rage pattern, such as defensive posturing, hissing, spitting and growling, piloerection, wide-open eyes and pupil dilation. Stimulation of the ventromedial nucleus of the hypothalamus, as well as the amygdala, hippocampus and anterior portions of the limbic cortex help to suppress the rage pattern. Damage to these structures can make the animal or human more susceptible to bouts of rage.

**Amygdala**

The amygdala is located immediately beneath the cortex of the medial anterior pole of each temporal lobe, receiving information from all portions of the limbic cortex as well as other parts of the brain, but especially from the auditory and visual association centers. In lower animals the amygdala is “hardwired” with the olfactory bulb, and while this is still true for humans to a certain extent, the amygdala
has many other functions not associated with olfactory stimuli. Electrostimulation of certain areas within the amygdala can cause many of the same reactions as stimulation of the hypothalamus, such as changes in arterial pressure and heart rate, gastrointestinal motility, defecation and urination, pupillary dilation, piloerection, the secretion of anterior pituitary hormones, as well as rage, reward and punishment reactions. Stimulation of other areas in the amygdala can cause sexual activities including erection, ejaculation, ovulation, uterine activity and premature labour. The amygdala seems to function on a semiconscious level, integrating thought and emotion with specifics of the environment, allowing for appropriate behaviours on specific occasions.

Hippocampus

The hippocampus is located posterior to the amygdala, below the temporal lobe and stretches along upward and inward to form the ventral surface of the inferior horn of the lateral ventricle. Like the amygdala, the hippocampus is a channel through which incoming sensory information can lead to the appropriate behavioral reactions. Weak stimulation of the hippocampi causes psychomotor epileptic seizures that extend in duration beyond the period of stimulation. Specifically, the hippocampus seems to play a role in learning. In lower animals the hippocampus is part of the olfactory cortex. Olfactory stimuli is important for the animal to make decisions regarding the edibility of a given food, whether a certain smell indicates danger, whether a certain smell suggests sexual activity and many other functions. Thus, in the evolution of mammals, the hippocampus took on the role of decision making, determining the relative importance of certain stimuli, and the brain began to rely upon it for this function. If the hippocampus decides that a given stimuli is important, then the brain will store it as memory. Removal of the hippocampi in epileptic patients has resulted in a unique condition called anterograde amnesia, in which the person has full memory prior to the surgery, but after, cannot commit newly learned activities to long-term memory. These patients can only remember what happens throughout their day for a few minutes, after which the memory is completely lost. This dysfunction of memory however, only refers to symbolic or verbal forms of information, and persons afflicted with lesions to the hippocampi can continue with reflexive learning, such as
physical skills learned through repetition, as in sports. Therefore, the hippocampus is responsible for the consolidation of long-term memory.

**Thought, consciousness and memory**

According to modern biomedicine, thoughts, consciousness and memory seem to be the preserve of the higher functions of the brain. A thought is said to result from a pattern of stimulation generated by many parts of the nervous system, determined and colored by the limbic system, thalamus and reticular activating system as being pleasurable or painful, and given discrete characteristics by the cerebral cortex. Consciousness is the continuous stream of sequential thoughts. Other models suggest that these centers are biomechanical receivers of impulses that are generated in the mental and emotional (astral) bodies. These bodies appear to take on a holographic nature, thus can communicate directly with these centers.

Memory is classified into three types, immediate, short term and long term. Immediate memory lasts for seconds, and is the capacity to remember highly specific but limited amounts of information (such as a telephone number), only as long as the person actively thinks about it. There are several theories as to how immediate memory functions, and one theory is that it is the activation of vibrating neural circuits that continue to vibrate as long as the person exerts a conscious effort to remember. Another model suggests that active connection to other bodies is necessary. Various traumas, age, or lack of vitality can influence this connection. Sometimes background consciousness seems to be focused on other matters, thus making the ‘mundane’ connection hard to keep. This function of being ‘spaced-out’ seems to arise from that lack of connection.

Short-term memory is the capacity to remember information for days and even weeks, but unless the information becomes consolidated, the memory is lost. Long-term memories last for years. The bio-medical model believes that immediate or short-term memories initiate structural and chemical changes at synapses that enhance or suppress impulse conduction. This consolidation of information into long term memory is believed to require at least 5 – 10 minutes to be minimally functional, and up to
an hour to be completely retained. The brain engages in a rehearsal process, continually going over the information until consolidation has taken place. Other models believe that long term memories are stored in a holographic archive, that take stimulus to ‘unravel’.

An **electroencephalogram (EEG)** can be used to determine the nature of the electric potentials generated by the brain, called **brain waves**, measured in cycles per second (hertz, Hz). To record an electroencephalogram 16 to 30 electrodes are attached to the scalp, connected to wires which are routed to an amplifier and an electroencephalograph. The electrical discharge of a single neuron is insufficient to be recorded through the skull, so brain waves are indicative of several thousands and even millions of neurons firing simultaneously. Thus, the strength of the brain wave is dependent upon the synchronous firing of neurons, not on the total level of electrical activity in the brain. There are four types of brain waves, alpha, beta, theta and delta. **Alpha waves** (8 – 13 Hz) occur most intensely in the occipital region of the brain, and are present in the resting wakened state, (meditative state), when the eyes are closed, but disappear during sleep. **Beta waves** (14 – 30 Hz) are recorded primarily in the parietal and frontal regions of the brain, and appear when the nervous system is active, during periods of sensory input and mental activity. These waves may occur in a waking state or during REM sleep. **Theta waves** (4 – 7 Hz) are found in the parietal and temporal
regions and occur normally in children and in adults experiencing stress, frustration or disappointment. Theta waves occur in many brain disorders. Delta waves (1 – 5 Hz) arise from the cerebral cortex and occur in infants and in adults during deep sleep. In an awake adult, delta waves indicate a serious organic brain disease. An EEG reading can show considerable variability in the nature and number of spikes on the graph paper. An EEG of petit mal epilepsy for example, is typified by a spike and dome pattern.

As seen, the brain waves can determine mood and are theorized by some to also determine states more connected to other bodies. When the brain waves are in an alpha state (found in regular meditators) a clearer connection with the mental body is made. This means the person can be clearer about memory or get levels of inspiration from other bodies. If the brain waves are in an alpha state and in harmony with other wave frequencies such as heart waves, a feeling of bliss is obtained. This would mean both the mental and the emotional bodies are communicating with the physical body and are in harmony. Many types of meditation, exercise (yoga, Tai Chi etc.), hobbies, and creative endeavours can put a person into an alpha state.

When a person is in a state of anxiety, they are often in a state of non-connectedness with their other bodies. This is characterized by more erratic or delta brain waves.

**Cerebellum**

The cerebellum is located inferior to the occipital lobes of the cerebrum and posterior to the medulla and pons. It is separated from the cerebrum by the transverse fissure and by an extension of the cranial dura mater. It is shaped like a butterfly and the wings are representative of the left and right hemispheres. In turn, each hemisphere can be divided into an anterior and posterior lobe. The surface of the cerebellum is called the cerebellar cortex and is comprised of gray matter, arranged in a series of slender, parallel ridges called folia. Beneath the gray matter are white matter tracts called arbor vitae because they resemble the branches of a tree. Within the heart of this tree are the cerebellar nuclei, comprised of gray matter that give rise to the nerve fibers that convey information to the brain and spinal cord.

**Theanine:** an amino acid found in green tea and some mushrooms, will help put a person into an alpha state, 30 60 min after ingesting.
The cerebellum controls the skeletal muscle contractions required for skilled movements, coordination, posture and balance. It has no direct ability to cause muscle contraction however, but functions by sequencing motor activities and monitoring and adjusting motor activities elicited by other parts of the brain. It compares actual movements recorded by the peripheral sensory feedback system with movements intended by the motor system, and if the two signals do not compare favorably, then corrective impulses are sent back to the motor system to increase or decrease activation of the specific muscles.

Cranial nerves

There are 12 pairs of cranial nerves that arise from the brain and pass through the various foramina of the cranial bones. Each pair of cranial nerves are designated by a roman numeral, indicating the order, anterior to posterior, in which the nerves arise from the brain. A name is further appended with the numeral, giving indication of that nerve’s distribution or function. Some of these nerves have motor functions and others are sensory, but most have both sensory and motor functions. The cranial nerves are listed as followed, with a brief mention of whether they are sensory or motor (or mixed), what areas of the body they innervate, and their basic functions.
<table>
<thead>
<tr>
<th>Cranial Nerve</th>
<th>Type</th>
<th>Area</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Olfactory</td>
<td>sensory</td>
<td>olfactory bulb</td>
<td>smell</td>
</tr>
<tr>
<td>II. Optic</td>
<td>sensory</td>
<td>retina</td>
<td>vision</td>
</tr>
<tr>
<td>III. Oculomotor</td>
<td>mixed</td>
<td>upper eyelid, eyeball</td>
<td>Sensory: proprioception</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: eye movement</td>
</tr>
<tr>
<td>IV. Trochlear</td>
<td>mixed</td>
<td>superior muscle of eye</td>
<td>Sensory: proprioception,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: eye movement</td>
</tr>
<tr>
<td>V. Trigeminal</td>
<td>mixed</td>
<td>eye, face, mouth, tongue</td>
<td>Sensory: touch, pain,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>temperature, proprioception</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: chewing</td>
</tr>
<tr>
<td>VI. Abducens</td>
<td>mixed</td>
<td>lateral muscle of eye</td>
<td>Sensory: proprioception</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: movement</td>
</tr>
<tr>
<td>VII. Facial</td>
<td>mixed</td>
<td>tongue, face, glands</td>
<td>Sensory: proprioception,</td>
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<td></td>
<td></td>
<td></td>
<td>taste</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: facial expression,</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>saliva, tears</td>
</tr>
<tr>
<td>VIII. Vestibulocochlear</td>
<td>mixed</td>
<td>ear</td>
<td>Sensory: hearing</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: adjust function of</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>hair cells in ear</td>
</tr>
<tr>
<td>IX. Glossopharyngeal</td>
<td>mixed</td>
<td>tongue</td>
<td>Sensory: touch, pain,</td>
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<td></td>
<td></td>
<td></td>
<td>temperature</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: swallowing,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>salivation</td>
</tr>
<tr>
<td>X. Vagus</td>
<td>mixed</td>
<td>throat, viscera</td>
<td>Sensory: touch, pain,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>temperature, breathing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: swallowing,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>coughing, heart rate,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>digestion</td>
</tr>
<tr>
<td>XI. Accessory</td>
<td>mixed</td>
<td>neck, shoulders</td>
<td>Sensory: proprioception</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: swallowing,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>movement</td>
</tr>
<tr>
<td>XII. Hypoglossal</td>
<td>mixed</td>
<td>tongue</td>
<td>Sensory: proprioception</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor: swallowing,</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>speech</td>
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</tbody>
</table>

**The autonomic nervous system**

The autonomic nervous system (ANS) is activated by centres within the spinal cord, brain stem and hypothalamus, and also by portions of the cerebral cortex, especially the limbic system, via the hypothalamus. Efferent autonomic signals are transmitted to the body by either the sympathetic or parasympathetic division. In the
ANS, both the sympathetic and parasympathetic ganglia are supplied by preganglionic fibers that exit the CNS. A **ganglion** (pl. ganglia) is a collection of neuronal cell bodies outside the CNS.

Within the sympathetic nervous system, ganglia are of two types: **paravertebral** ganglia and **prevertebral** ganglia. Paravertebral ganglia lie next to the vertebrae, concentrated in two long chains that extend on either side of the spinal column, from the neck to the coccyx, and are all interconnected. Prevertebral ganglia are located in front of the vertebrae, supplying the viscera with sympathetic nerves.

Preganglionic nerves in the sympathetic nervous system are short, synapsing with a postganglionic nerve that is much longer. Parasympathetic ganglia, on the other hand, are located close to the effector they innervate. Thus, the preganglionic fibers in the parasympathetic division are long and the postganglionic fibers are short, opposite of the sympathetic nervous system. The functional difference between this arrangement is that parasympathetic responses are rapid and precise, and more likely to affect a single effector, such as a specific organ. Sympathetic responses, on the other hand, tend to be slower and more diffuse, affecting the entire body (e.g. the “fight or flight” response). Not all sympathetic responses are diffuse however: pupil dilation and sexual orgasm are both sympathetic responses that need not occur simultaneously, otherwise going from a brightly lit room into a darkened one could be a potentially embarrassing situation. The primary difference, therefore, between the parasympathetic and sympathetic nervous systems is anatomical placement, and secondarily, their function.

There are two principle types of synapses found in the ANS, called **cholinergic** and **adrenergic**. Cholinergic synapses are nerve synapses that release **acetylcholine** (ACh), whereas adrenergic synapses release **epinephrine** (E) and **norepinephrine** (NE).

All preganglionic nerves are cholinergic in both the sympathetic and parasympathetic divisions. Postganglionic nerves of the parasympathetic system are also cholinergic.
After binding with the receptor site and initiating a nervous impulse in the postsynaptic neuron, ACh quickly degrades into acetic acid and choline under the influence of an enzyme called acetylcholinesterase (AChE). Choline is then pumped back into presynaptic neuron to be recycled into ACh. **Cholinergics** are exogenous substances (e.g. drugs, botanical agents and bacterial toxins) that promote the release, prevent the degradation or otherwise enhance the actions of ACh. **Anticholinergics** are exogenous substances (e.g. drugs, botanical agents and bacterial toxins) that inhibit the synthesis, release or receptor binding of ACh.

**Cholinergic receptors** are of two basic types: **nicotinic** receptors, located in the brain, smooth muscle, heart and glands such as the adrenal medullae; and **muscarinic** receptors, located in the brain, sweat glands and blood vessels. The binding and stimulation of nicotinic receptors leads to the up regulation of autonomic functions such as increased muscle tone, blood pressure and heart rate. Nicotinic receptors are called such because the alkaloid nicotine (derived from tobacco) was found to stimulate both sympathetic and parasympathetic postganglionic nerves. Examples of exogenous agonists to nicotinic receptors include nicotine from tobacco and the piperidine alkaloids of *Lobelia inflata*. An example of a nicotinic antagonist is curare, a plant poison coated on arrows used by some of the First Nations people in South America to bring down prey.

The binding and stimulation of muscarinic receptors promotes parasympathetic responses such as sweating, salivation, pupil constriction, peristalsis, hypotension and a decrease of heart rate. Muscarinic receptors are also associated with learning, memory, posture and temperature regulation. Muscarinic receptors are called such because the alkaloid muscarine, derived from the mushroom *Amanita muscaria*, was found to stimulate parasympathetic responses. An example of a muscarinic agonist is pilocarpine, an alkaloid of *Pilocarpus jaborandi* used in the treatment of glaucoma. Examples of muscarinic antagonists are the tropane alkaloids (atropine, hyoscine and hyoscyamine) found in *Atropa belladonna, Datura stramonium* and *Hyocyamus niger*, parasympathylytics used in the treatment of spasmodic afflictions, including asthma, cerebral palsy and Crohn’s disease.
Adrenergic synapses are nerve synapses that release epinephrine and nor-epinephrine. Most postganglionic sympathetic neurons are adrenergic. Adrenergics are agents that accumulate in the synaptic cleft and lead to an increase in norepinephrine release, such as amphetamines, the amino acid tyrosine, and the alkaloid ephedrine from Ephedra sinica.

Adrenergic receptors are of two major types, alpha (α) and beta (β), and have somewhat opposing effects. Alpha-receptors are stimulated primarily by norepinephrine and to a lesser extent by epinephrine, and promote vasoconstriction, iris dilation, intestinal relaxation, intestinal sphincter contraction, pilomotor contraction and bladder sphincter contraction. An example of an α-receptor antagonist is the alkaloid yohimbine from Pausinystalia yohimbe, which induces a down-regulation of α-adrenergic function. There are two primary types of β-receptors, stimulated mostly by epinephrine, and to a lesser extent by norepinephrine. Stimulation of β₁-receptors cause cardioacceleration, increased myocardial strength and lipolysis. Stimulation of β₂-receptors cause vasodilation, intestinal relaxation, uterine relaxation, bronchodilation, glycogenolysis and bladder wall relaxation. Many botanicals such as Cimicifuga racemosa and Viburnum opulus can act as β₂-agonists, and are thus uterine antispasmodics. Pharmaceuticals used to inhibit β-adrenergic activity are called “beta-blockers,” and are often used in the treatment of hypertension and angina pectoris. Some of these drugs, such as propranolol hydrochloride, are non-specific beta-blockers, inhibiting both β₁ and β₂-receptors. While these drugs may be effective for reducing blood pressure, they can have unwanted side-effects such as bronchoconstriction, mediated by β₂-receptors. If these side effects pose any risk, a β₂-receptor blocker such as metoprolol hydrochloride may be used instead.

The types of receptors in the organs determine the relative effects of norepinephrine and epinephrine on different effector organs. If they are all β-receptors, for example, then epinephrine will act as a stimulant. If the effector contains both α and β-receptors then the activity of epinephrine will be considerably less.
Neurotransmitters

**Acetylcholine**

It’s difficult to say with any surety what the effect of *acetylcholine* (ACh) is on the brain, because depending upon the nature of the postsynaptic neurons, ACh may be excitatory or inhibitory. Within the brain, cholinergic pathways can be organized into those that function locally, and those that connect two or more different regions. Of the cholinergic pathways that are of the latter type, two major constellations have been found: in the basal forebrain and in the pontomesencephalotegmental cholinergic complex. Together these pathways extend to almost all areas of the brain.

Within cholinergic neurons, acetylcholine is synthesized in a reaction catalyzed by choline acetyltransferase (CAT), combing *acetyl coenzyme A* with *choline* to form ACh and *coenzyme A* (CoA). Choline appears to be the rate-limiting factor in the synthesis of ACh.

The release of ACh is stimulated by the invasion of an action potential into the synaptic bulb, causing the intracellular flow of Ca$^{2+}$, which in turn promotes exocytosis of the synaptic vesicles and the release of ACh into the synaptic cleft. ACh is rapidly degraded by acetylcholinesterase to choline and acetic acid. Choline is then taken up by the presynaptic neuron to resynthesize ACh.

There is some evidence to suggest that ACh may participate in pain reception. This is evidenced in the painful ‘stinging’ rash elicited by the tiny hairs of the *Urtica dioica*, which contain ACh as well as histamine. ACh may also act as a sensory transmitter in thermal receptors, taste fiber endings and chemoreceptors.

Acetylcholine plays a role in a few nervous disorders, such as myasthenia gravis, an autoimmune disorder that directs antibodies against ACh. Alzheimer’s disease and Huntington’s chorea involve the destruction of cholinergic pathways in the brain.
Amino acid neurotransmitters

Glutamic acid
Glutamate is found in uniquely high concentrations in the CNS and it has been known for some time that it can exert a powerful stimulatory effect on neuronal activity. Within the brain L-glutamate is synthesized in presynaptic neurons by two sources: from glucose, via the Krebs cycle and the transamination of β-oxoglutarate, and from glutamine that is synthesized in glial cells, transported into nerve cells and converted into glutamate by glutaminase. Synaptic vesicles containing L-glutamate are induced to exocytosis by the depolarization of the terminal end of the presynaptic axon and the movement of Ca$^{2+}$ into the intracellular fluid. Glutamate binds with receptors on the postsynaptic cell and promotes the opening of chemically gated ion channels permeable to Na$^+$ and Ca$^{2+}$. After causing the depolarization of the postsynaptic cell, glutamic acid is reabsorbed by the presynaptic neuron, or taken up by the glial cells and converted into glutamine by glutamine synthetase. Although glutamate is ubiquitous in the CNS, it seems to be predominant in the spinal cord, released by primary afferent nerve endings. Other high-density regions for glutamate include the hippocampus and cerebral cortex.

Glutamate, as well as aspartate, are called excitatory amino acids (EAAs) and bind to at least five different receptor subtypes that have slightly different activities, some initiate fast EPSPs and some initiate slow EPSPs. EAA receptors appear to play an important role in memory and learning. A relative deficiency of EAAs has been implicated in long-term depression. Excessive levels of EAAs, however, have been implicated in neurotoxicity and brain cell damage, and play a role in the neurodegenerative aspects of such diseases as Huntington’s disease, cerebral ischemia, epilepsy, hypoglycemia and AIDS.

Neuralathyrism is a spastic disorder common to East Africa and Southern Asia that is associated with the consumption of chick peas (*Lathyrus sativus*), which contains the amino acid β-N-oxalylamino-L-alanine (BOAA) and is an agonist to a specific EAA receptor subtype. The consumption of domoic acid, synthesized by seaweeds and consumed by mussels that feed on the seaweed, is a potent excitatory neurotoxin that can damage the hippocampus and produce

**Gamma aminobutyric acid (GABA)**

GABA is also an important neurotransmitter in the human body, found in no other location except the CNS and the retina. GABA is synthesized from glutamic acid by glutamic acid decarboxylase (GAD) and has a discrete distribution in the brain, primarily in the superior and inferior colliculi, thalamus, hypothalamus and occipital lobes of the cerebrum. The enzyme GAD requires pyridoxal phosphate (vitamin B6) as a coenzyme, and the use of this vitamin is an important adjunct in the treatment of seizure disorders. GAD is also known to coexist in the β-cells of the pancreas, perhaps playing a role in the endocrine pancreas. In almost all type I diabetes patients, antibodies to GAD can be observed, and it is thought that it is these antibodies that are responsible for the destruction of the pancreatic β-cells. GABA is metabolized by GABA-transaminase, which also requires vitamin B6 as a cofactor, into succinic semialdehyde.

There are two major types of GABA receptors in the human brain that have been determined largely through pharmacological evidence, GABA_A and GABA_B. The binding of GABA to GABA_A receptors causes a shift in the membrane permeability to inorganic ions, in particular to chloride, inhibiting the firing of the neurons by inducing a state of hyperpolarization. Thus GABA_A receptors have an inhibitory action in the CNS. Benzodiazepines and barbiturates are commonly used drugs that function as GABA_A agonists by enhancing the electrophysiological effects of GABA. Other drugs, including muscimole, a metabolite of Amanita muscaria that appears upon drying, and the volatile oil isovaleric acid and valepotriates of Valeriana officinalis, appear to be GABA mimetics. Other botanicals that either improve receptor sensitivity to GABA or are GABA mimetics include Passiflora incarnata, Withania somniferum and Tilia cordata. Certain hormones are also known to bind with GABA_A receptors, such as deoxycorticosterone and progesterone, and thus hormonal changes experienced during puberty, pregnancy and during the luteal phase of the menstrual cycle could account for certain behavioral adaptations to stress. GABA_B receptors are present in lesser volume than GABA_A receptors, and the binding of GABA to GABA_B receptors initiates inhibitory
postsynaptic potentials by increasing the transmembrane permeability of $K^+$. $GABA_B$ receptors are unaffected by $GABA_A$ agonists, and $GABA$ antagonists appear to have none of the activity on $GABA_B$ receptors as they do on $GABA_A$ receptors (i.e. seizure). This has lead to the hypothesis that $GABA_B$ receptors may only be activated under certain psychological states.

GABA appears to have an inhibitory (hyperpolarizing) activity in the brain, but an excitatory (depolarizing) role in the spinal cord. Within the brain GABA is released in amounts up to three times higher in delta wave EEG patterns than in alpha wave patterns. It is interesting to note that there is a 30-40% increase in GABA levels postmortem, in part resulting from a transient activation of GAD.

**Glycine**

Glycine is a simple amino acid that is ubiquitous in the human body, essential in the metabolism of protein, peptides, nucleic acids, porphyrins and bile salts, as well as neurotransmitters in the CNS. Little is known about glycine synthesis and metabolism, but much evidence has been accumulated to suggest that it is an inhibitory neurotransmitter, primarily in the gray matter of spinal tissue. Glycine also seems to play an important role in increasing the responsiveness of EAA receptors.

**Biogenic amine neurotransmitters**

The biogenic amines are monoamine neurotransmitters synthesized from dietary sources of amino acids. The monoamines include the catecholamines and the indolamines. The catecholamines (dopamine, epinephrine, norepinephrine) are derived from tyrosine, a non-essential amino acid found in foods such as almonds, avocados, and bananas, and can be synthesized from phenylalanine by phenylalanine hydroxylase in the liver. The catecholamines are called such due to a common catechol nucleus (a six carbon benzene ring and two adjacent hydroxyl groups) and an amine group. Inactivation of these catecholamines occurs after they are pumped back into the presynaptic neuron and destroyed by either catechol-O-methyltransferase (COMT), monoamine oxidase (MAO), or recycled back into synaptic vesicles. The indolamines (serotonin and melatonin) are derived from the essential
amino acid **tryptophan**, found in animal products including meat, eggs and dairy products. Tryptophan is also the biological precursor to **tryptamine** through the activities of amino acid carboxylase, which provides the indole unit of monoterpenoid-indole and derived alkaloids that have psychoactive properties. Unlike the catecholamines, the indolamines are degraded only by MAO.

MAO inhibitors such as phenelzin are drugs that prolong the activity of monoamine neurotransmitters and thus can act as stimulants, and are used in some circles as antidepressants. Examples of botanicals that have MAO inhibitory activity include *Peganum harmala* and *Banisteriopsis caapi*, the latter of which is often found in the indigenous entheogenic ‘Ayahuasca’ brews used by shamans in South America. Such natural MAO inhibitors prevent the gastrointestinal enzymatic degradation of hallucinogenic compounds such as DMT (N,N-dimethyltryptamine) found in plants such as *Psychotria viridis* and *Diplopterys cabrerana*.

**Dopamine**

Neurons that contain **dopamine (DA)** are clustered primarily in the midbrain within the substantia nigra. Some of the dopaminergic axons project from the substantia nigra into the cerebral cortex, where DA is thought to be involved in emotional responses. Other axons project into areas of the basal ganglia, where DA is known to have an inhibitory activity upon the autonomic movements of the skeletal muscles, lending stability to motor control. It is the destruction of these dopaminergic neurons in the basal ganglia that plays a role in the dopamine deficiency of Parkinson’s disease. Dopamine is
also known as prolactin-inhibiting hormone, inhibiting the activity of prolactin which promotes milk production. Peripherally, DA has a variety of functions, including the inhibition of gastric emptying.

Dopamine synthesis, like all of the catecholamines, is derived from the non-essential amino acid tyrosine, synthesized from phenylalanine in the liver by phenylalanine hydroxylase, and is transported across the BBB into dopaminergic neurons. The absorption of tyrosine, as well as tryptophan (which is used in serotonin production) is regulated by the presence of other amino acids that compete for absorption. In conditions such as phenylketonuria\(^1\) in which serum levels of phenylalanine are abnormally high, both tyrosine and tryptophan uptake by the brain may be diminished. Once tyrosine enters the neuron it is converted into L-dihydroxyphenylalanine (L-DOPA) by tyrosine hydroxylase (TH). The activity of TH is dependent upon molecular \(O_2\), \(Fe^{2+}\) and a tetrahydropteridine cofactor. L-DOPA is then rapidly converted into DA by L-aromatic amino acid decarboxylase, which is dependent upon pyridoxal phosphate (vitamin B6) as a cofactor. A deficiency of vitamin B6 can interfere with the rate of repletion of adrenal catecholamines.

Calcium dependent release of dopamine is thought to occur as the result of an action impulse reaching the terminal end of the axon. The release of dopamine appears to be dependent upon the rate and pattern of neuronal firing. Dopamine release is also inhibited by the presence of presynaptic release-modulating autoreceptors. Upon release and stimulation of the postsynaptic cell, DA is rapidly metabolized into dihydroxyphenylacetic acid (DOPAC) by intracellular MAO after reuptake by the presynaptic neuron. The majority of the released DA in the human brain however, is converted into homovanillic acid (HVA) by MAO, and concentrations of HVA in the brain and CSF can be used as an index of dopaminergic activity. Reduction of HVA from normal can be detected in the CSF of patients with Parkinson’s disease.

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\(^1\) Phenylketonuria is a defect of metabolism that is characterized by an elevation of phenylalanine in the blood, due to a genetic deficiency of the enzyme phenylalanine hydroxylase which converts phenylalanine into tyrosine. Excessive levels of phenylalanine in early development leads to brain damage and mental retardation. It is prevented by performing a PKU test on newborns, and in positive infants, special steps are taken to limit dietary phenylalanine.
At present there are five known postsynaptic receptors of DA, and D₂ in particular is thought to be associated with behavior and emotion. In post mortem studies of dopamine receptors in schizophrenia, the number of D₂ receptors appears to be abnormally elevated.

Some herbs such as *Vitex agnus castus* seed appear to have a dopaminergic activity. This is why *Vitex* is useful in hyperprolactinemia, since dopamine is a prolactin antagonist. In contrast, botanicals such as *Asparagus racemosus* act as a dopaminergic antagonists, enhancing gastric emptying and allowing prolactin to function unopposed, thereby increasing milk production.

**Norepinepherine and epinepherine**

Like dopamine, norepineph(e)rine (NE) and epineph(e)rine (E) belong to a class of compounds called catecholamines. Epinepherine, and to a lesser extent, norepinepherine, are concentrated in the tissues of the adrenal medullae and are secreted in response sympathetic stimulation. The adrenal medullae are actually modified sympathetic ganglionic fibers, as opposed to an effector organ, and secrete E and NE directly into the blood stream. The highest concentration of these compounds in the brain is usually found in the hypothalamus and other areas of central sympathetic representation, more often in gray rather than white matter. As previously mentioned, all of the catecholamines require tyrosine as the base nutrient for their creation. Tyrosine is converted into L-DOPA by TH, and L-DOPA is converted into NE by the enzymatic activity of dopamine-β-hydroxylase. This enzyme is dependent upon molecular O₂ and ascorbic acid (vitamin C) as cofactors. NE can be further synthesized into E by phenylethanolamine-N-methyl transferase. The enzymes that facilitate the metabolic degradation of the catecholamines are MAO and catechol-O-methyl-transferase.

The presynaptic release of catecholamines is generally dependent upon Ca²⁺, and a deficiency of serum Ca²⁺ can inhibit their release. Drugs such as cocaine, amitryptaline and other related tricyclic antidepressants inhibit the reuptake of norepinepherine, allowing it to linger longer in
the synaptic cleft. These drugs, along with MOA inhibitors, potentiate adrenergic transmission.

“Adrenal stress” is a term often used by herbalists in a rather unspecific way, to describe, in part, a depletion of the cofactors needed to secrete NE and E. The theory is that there is some disruption to homeostasis, typically sympathetic in origin, but diffuse and mediated by external factors such as social customs and life experiences. These stressors of ‘everyday life’ create physical reactions that: 1) cause an increase in the utilization of O$_2$ and vitamin C, and; 2) cause physical reactions such as visceral vasoconstriction that inhibit nutrient supply to key organs, creating an even greater need for these and other nutrients. It is a subtle group of mechanisms at best, addressed by herbalists with comparatively subtle remedies and nutritional supplementation. The adrenal stress dynamic also refers to the exhaustion of the adrenal cortex in its role as a mediator of sympathetic stress, and a relative deficiency of adrenal corticoids used to down-regulated the sympathetic response.

There are two major clusterings of norepinepherine cell bodies in the brain, one localized in an area called the **locus ceruleus**, contained within the caudal pontine central gray anterior to the cerebellum, and the other, scattered loosely through the ventral tegmental fields. Noradrenergic pathways that have been studied physiologically are efferent fibers from the locus ceruleus to the cerebellum, the hippocampus, and the cerebral cortex. Noradrenergic neurons of the lateral tegmental fields send fibers downward into the mesencephalon and the spinal cord, and anteriorly to the forebrain and diencephalon, including the amygdala. Norepinepherine has been implicated in maintaining arousal, dreaming and the regulation of mood.

There are considerably fewer epinepherine-containing neurons than noradrenergic neurons, but their discrete anatomical distribution in the brain is believed to play a unique role in assisting in neuro-endocrine control and blood pressure regulation.

**Serotonin**

**Serotonin (5-hydroxytryptamine, or 5-HT)** is found in many cells that are not neurons, including platelets, mast cells and the enterochromaffin cells of the intestinal
mucosa. Only 1-2% of the serotonin that is produced in the body is actually made in the brain, but since this neurotransmitter cannot pass the BBB, the brain must manufacture its requirements. Serotonin synthesis in the brain begins with the amino acid tryptophan derived from the diet. The botanical *Avena sativa* is exceptionally high in this amino acid, but there are many other sources of dietary tryptophan, including turkey, eggs and aged cheeses. The rate-limiting enzyme tryptophan hydroxylase (TH) then acts upon tryptophan to complete its hydroxylation into 5-hydroxytryptophan (5-HTP), which undergoes almost immediate decarboxylation by amino acid decarboxylase (AADC) to form 5-hydroxytryptamine (5-HT).

Drugs such as Lithium increase tryptophan uptake across the BBB, increasing the amount of 5-HT produced. Over a 14-21 day period however, tryptophan uptake is still increased, but the activity of TH is gradually decreased so that normal levels of 5-HT are eventually produced. Thus in manic depressive-psychosis there is a minimum of 7-10 days before Lithium can foster this state of equilibrium. The activity of TH also appears to be dependent upon the level of molecular oxygen in the brain, and in rats permitted to breathe 100% O₂ the level of 5-HT is greatly increased. The administration of the precursor 5-HTP avoids the rate-limiting activities of TH and results in the non-specific formation of serotonin in any sites containing AADC.

Serotonin production is particularly concentrated in an area of the brain stem called the raphe nuclei, which enerates areas of the hypothalamus involved with pituitary releasing hormones such as prolactin, growth hormone and adrenocorticotropin. All the enzymes needed to produce 5-HT are also found in the pineal gland however, and 5-HT concentrations within the pineal are 50 times greater (per gram) than in the rest of the brain. Within the pineal, 5-HT undergoes two additional enzymatic steps to form melatonin. Melatonin production is increased by darkness and decreased by light, and its secretion has a suppressive
effect upon sexuality. Perhaps the increasing conversion of 5-HT to melatonin during the winter season is a possible cause of seasonal affective disorder (SAD). At present, there are at least eight subtypes of serotonin receptors known to exist in the brain, and fifteen total in the whole body.

Upon the release of free serotonin in the synaptic cleft, it is metabolized by MAO into 5-hydroxyindolacetaldehyde, which is further oxidized into 5-hydroxyindoleacetic acid (5-HIAA). Drugs such as isocarboxazid and phenelzine inhibit the activity of MAO, as does the Middle Eastern herb *Peganum harmala*, which contains the β-carboline alkaloids such as harmine and harmaline. Another mode of serotonin metabolism is its uptake by the presynaptic neuron into synaptic vesicles. Some antidepressant drugs such as fluoxetine and sertraline selectively inhibit the reuptake of 5-HT by the presynaptic neuron, as does cocaine.

Serotonergic activity cannot be characterized simply: it seems to be responsible in coordinating a wide range of complex sensory and motor patterns during varied behavioral states. Serotonergic activity has been found to be highest during periods of arousal, reduced in quiet waking, reduced even more in slow-wave sleep and absent during REM sleep. An increase in the activity of serotonergic neurons during waking states serves to enhance motor neuron excitability. A suppression of sensory input during REM sleep impedes motor activity, even though there is increased internal arousal.

Altered serotonergic function has been reported in several psychopathological conditions, including schizophrenia, hyper-aggressive states, major depression, anxiety, eating disorders, migraine, obsessive compulsive disorder and suicidal behavior. The diversity of receptors and receptor subtypes in different areas of the brain, as well as other factors in serotonin metabolism, help to explain why it’s possible for a single neurotransmitter to be associated with such a large array of behaviors, clinical conditions and drug activities.
There is a wide range of psychotropic agents that are known to affect 5-HT neurotransmission, including antidepressants (e.g. fluoxetine), antipsychotics (e.g. clozapine), antiemetics (e.g. ondansetron), appetite suppressants (e.g. fenfluramine) and antimigrane drugs (e.g. sumatriptan), all working in a unique fashion.

Hallucinogens such as lysergic acid diethylamide (LSD) and psilocin are said to affect 5-HT neurotransmission as well, and can be either serotinergic agonists or antagonists dependent upon their location within the brain (i.e. the mode by which LSD functions in the brain is still poorly understood).

**Neuroactive peptides**

The **neuropeptides** represent the brave new world of neuropharmacology. New peptides are constantly being discovered and the race is on to understand their biological properties so pharmacologists can tweak them and pharmaceutical manufacturers can amass even greater fortunes. So far they are the largest family of neurotransmitters, and can have both inhibitory and excitatory activities. Neuropeptides consist of chains of 3 to about 40 amino acids. In 1974 scientists discovered that certain neurons in the brain have receptors for opiate drugs such as heroin and morphine. The quest to find the endogenous complement to these drugs provoked the discovery of the first neuropeptides. These two molecules, comprised of a chain of 5 amino acids, were **enkephalin** and **endorphin**. Some of the more important neuropeptides, beyond the two already mentioned, include substance P, dynorphin, hypothalamic regulating hormones, angiotensin II, cholecystokinin, oxytocin and vasopressin.

Neurons that secrete peptides are different from other neurons in the manner of how each synthesizes its respective neurotransmitters. Unlike amino acids or monoamine-releasing neurons that utilize dietary sources of amino acids for synthesis, synthesis of neuroactive peptides is directed by mRNA on ribosomes, located only in the dendrites or cell bodies of peptide-secreting neurons. This synthesis creates large protohormones, which are then cleaved by proteolytic enzymes, packaged into vesicles in the smooth endoplasmic reticulum and transported to the axon terminals for eventual release.
The release of neuroactive peptides seems to be mediated, however, by the same factors that are responsible for neurotransmitter release in the monoamine and amino acid secreting neurons. An action potential travels down the axon and invades the synaptic bulb, initiating the influx of extracellular Ca$^{2+}$, thereby promoting exocytosis of the neuroactive peptides. The postsynaptic effects of neuropeptides, as well, appear to be similar to its cousins, regulating ion channels through secondary messengers. Neuropeptides also seem to exhibit many properties of hormones, such as the capacity to target sites that are distant from the site of release. Additionally, the immune system seems to be much more closely linked to the nervous system than was previously realized.

Lymphocytes have been found to manufacture and secrete neuropeptides, such as endorphins. Researcher Candice Pert describes in her book *Molecules of Emotion* how she and her colleagues found that every known neuroactive peptide receptor present in the brain could also be found on the plasma membrane of a monocyte, a finding among others that has led to the development of psychoneuroimmunology (PNI), a branch of science which examines the correlation between the nervous, immune and endocrine systems.

Neuropeptides challenge orthodox ideas about how the nervous system works, and the belief that the nervous system is in any way independent from other bodily systems. At one time all pharmacologists believed that there were only two kinds of autonomic neurons, cholinergic and adrenergic. But soon neuroactive peptides began to show up in autonomic neurons where they shouldn’t be found. So far, neuropeptides have been found in every neuron that has been looked at, and in many non-nervous tissues. This has lead to the theory that neuropeptides may be used to modulate the activity of neurotransmitters to refine the postsynaptic effect. It also shows, as Candace Pert postulates, that neuropeptides may be a link between the diverse functions of different body systems, possibly regulated by emotional control.

**Enkephalins**

Enkephalins are concentrated in the thalamus, the hypothalamus, parts of the limbic system and in spinal pathways that relay impulses for pain. They are believed to be the body’s way of mediating the negative effects of pain.
by suppressing substance P release, and are on average, over 200 times stronger than morphine. The effects, however, are short lived.

**Dynorphins**

Dynorphins are found in the posterior pituitary gland, hypothalamus and small intestine. Like enkephalins, dynorphins are important in mediating the effects of pain by inhibiting substance P release, and seem to have some activity in controlling emotion.

**Endorphins**

Endorphin is a generalized term for any endogenous agent that resembles morphine. They are concentrated in the pituitary gland and function similarly to enkephalins in inhibiting substance P release. Endorphins have also been linked to improved memory and learning, feelings of pleasure and euphoria, control of body temperature, the initiation of puberty and sexual activity.

**Substance P**

Substance P is found in sensory nerves, spinal cord pathways and parts of the brain associated with pain transmission, such as the substantia nigra, basal ganglia, amygdala, hypothalamus and cerebral cortex. When it is released from neurons, substance P transmits pain-related input from the peripheral pain receptors into the CNS. Substance P has also been found to counter the activities of neurotoxins, prompting speculation that it might be useful in nerve regeneration. *Capsicum spp.* is a counter-irritant to sensory nerve endings and is known to promote the release of substance P.

**Nitric oxide**

Nitric oxide (NO) is a gas that is found throughout the body, as a neurotransmitter and a regulatory molecule, but also as a toxin, used by components of the immune system to kill microbes and tumor cells. It has been implicated in the production of a free radical called peroxynitrite, when it reacts with super oxide. Nitric oxide is formed by the combination of a single atom of oxygen and a single atom of nitrogen, catalyzed by the enzyme nitric oxide synthetase from the amino acid arginine. Unlike other neurotransmitters, NO is not synthesized in advance, but is formed on demand and acts immediately. The activities of NO were first elucidated when it was discovered that
endothelial cells release NO to promote vasodilation (and was previously called endothelium-derived relaxing factor, EDRF), which is the activity that is harnessed by the drug sildenafil (Viagra) that promote erections in men. NO is thought to play a role in memory and learning.

**Arachidonic acid**

Arachidonic acid is synthesized from dietary linoleic acid and is responsible for a large number of metabolites called eicosanoids. There are three major groups of eicosanoids, the prostaglandins, leukotrienes and thromboxanes. The activity of these compounds has been well studied and although they seem to play an important modulatory role in nervous tissue, how and where they act is still a question. Unlike neurotransmitters they are not stored in tissues, but are synthesized on demand, acting for short periods of time in very low concentrations. The eicosanoids perhaps act as secondary messengers: a neuroactive substance binds with its receptor and either inhibits or stimulates the release of an enzyme called phospholipase A2. This enzyme then promotes the release of arachidonic acid from phospholipids, which undergo further enzymatic change by either lipoxygenase, cyclooxygenase or epoxygenase to form leukotrienes (LTs), hydroxyeicosatetraenoic acids (HETEs), prostaglandins (PGs), thromboxanes (TXs) and epoxides. Researchers have had difficulties in developing assay techniques for these chemicals, but it is clear that they have a profound activity in neurophysiology.

In the late 1980’s, researchers found a specific protein receptor for tetra-hydrocannabinol (THC), (the active ingredient of marijuana), in mouse nerve cells. THC is well known for its activity to promote changes in mood, memory, appetite, movement and perception, and pain. The wide-ranging activities of THC are probably due to an abundance of THC receptors found in many parts of the brain including the hippocampus, basal ganglia and cerebral cortex. It is unlikely however that THC receptors evolved for the singular purpose of ‘getting high,’ so researchers put themselves to the task of finding an endogenous compound that binds with these receptors. In the early 1990’s a fat soluble hair-pinned shaped chemical was teased out from bovine brain tissue which bound to
these receptors to cause a parallel activity to THC, and was termed **anandamide (arachidonoylethanolamine)**, from the Sanskrit word ‘ananda,’ meaning ‘bliss.’ It appears that the brain is able to enzymatically synthesize anandamide and the existence of cannabinoid receptors for this eicosanoid suggests the presence of anandamide-containing (anandaergic) neurons. Researchers so far have been unable to create a compound that has all of the benefits of anandamide, such as its antispasmodic properties, without also creating the characteristic mood altering effects. The medical use of marijuana has become a prominent issue in our society, and even though the jury is still out as to the negative, cumulative effects of using this drug, its usage, both medically and recreationally, is unlikely to go away any time soon.

**Part II: Etiology, Pathology and Treatment of Nervous system disorders**

As Herbalist, we acknowledge the interconnectedness of physiological and psychological factors, as they interact with the whole Being and the environment. This of course means that a vital part of treatment must take into consideration diet, lifestyle as well as emotional, mental and spiritual life.

**Insomnia**

**Insomnia** is a general term used to describe the patient’s perception that the amount or quality of sleep he or she experiences is inadequate, despite the fact that the opportunity for adequate sleep exists. The problems can range from difficulty falling asleep, easily disrupted with multiple awakenings, or early morning awakenings with an inability to fall back asleep. It is important to note which particular symptom the patient expresses, as this may not only indicate the cause but also the possible treatment. As such insomnia isn’t really a disease at all, but a symptom that can be related to numerous disorders including anxiety, depression, and chronic pain, or may be the result of a variety of medications or drugs such as alcohol (which can cause night-time wakening from rebound hypoglycemia) and psychostimulants such as cannabis. Insomnia is however associated with a variety of complaints.
experienced during the daytime, including an impaired ability to concentrate, poor memory, difficulty coping with minor stressors, and a decreased ability to engage in social relationships.

Sleep and rest help the body replenish depleted energy reserves and allows the maintenance of normal physical and mental function. Adequate sleep is essential for good physical and mental health.

There is a natural rhythm (circadian cycles) that is slightly over 24 hours and resets according to light and temperature cues. There is no set amount of sleep that a person should have. It can vary from person to person.

Insomnia is extremely common; with up to 30% of the population suffer from it sometime during the year and roughly 10% having a chronic problem. Approximately 12.5% of the adult population use some form of prescribed medication for insomnia. In any given day 2% of the population take prescriptions for insomnia. Over 100 million prescriptions are written for this area yearly.

There are three basic types of insomnia that can be found, classified on the basis of the duration of the complaint:

**Transient insomnia**: lasting no more than one week, usually caused by acute stress such as a new job, a project deadline, or an upcoming exam. In many cases it is a pattern that repeats itself with new or similar stressors.

**Short-term insomnia**: lasting for a period of one to six months, usually associated with a persistent stressful situation, such as the chronic illness or death of a loved one, or environmental factors such as an uncomfortable bed, room temperature, and extraneous noise.

**Chronic insomnia**: lasting more than six months, associated with a wide variety of disorders.

As previously mentioned, the specific symptoms of insomnia can indicate possible causes. For example, depression is most commonly associated with early morning awakening and an inability to fall back asleep. Schizophrenia and bipolar mania are often are associated with difficulty falling asleep, whereas anxiety disorders are
associated with difficulty falling asleep and maintaining sleep. The drugs taken to treat these conditions can also affect the quality and nature of sleep.

Another possible cause of chronic insomnia is obstructive sleep apnea, in which the patient awakens because of difficulty breathing. In some cases the causes of insomnia may relate to restless leg syndrome (RLS) or periodic limb movement disorder (PLMD), a sleep disorder characterized by spasmodic movements of the limbs that awakens the patient. Shift workers frequently complain of insomnia, due to a fundamental disruption of normal circadian cycles that are guided by the interplay of light and melatonin secretion in the brain. If no cause can be ascertained however, the condition is referred to as primary insomnia, a fairly rare idiopathic condition that often manifests early in childhood.

Sleep apnea is a major cause of sleep disorders, with approximately 20 million people in North America suffering from it. In this disorder the person stops breathing for brief periods of time. It is most often caused by narrowing of the air passages – which in turn is often caused by accumulation of fat tissue. Snoring is frequently associated with sleep apnea. Ingestion of alcohol or sleeping pills can increase sleep apnea. The most common therapy for sleep apnea is the use of a nasal continuous positive airway pressure (CPAP) device. The patient wears a mask over their nose during sleep and pressure is blown through the nasal passage. Sometimes surgery is performed, but it is not that effective and complications can develop within a year. Often by simply sleeping on one’s side, with a pillow between their knees, the sleep apnea can be resolved without the use of the uncomfortable CPAP.

Again it is important to note that insomnia is a subjective experience. Although most people require a minimum of eight hours sleep to feel refreshed upon awakening, some people appear to have a decreased requirement for sleep, and appear to be able to function on less sleep than others. Elderly people often complain of difficulties sleeping, and while it is more difficult for them to experience sustained sleep, their sleep requirements are about the same as for an adolescent.
Some practitioners theorize a system related to units of sleep instead of hours of sleep. For example, if a person does not get a deep sleep, they might get .5 units per hour, only having a restless surface sleep. They can sleep for 10 hours and get 5 units of sleep. Another person can get into a deeper sleep, with 4 units per hour, sleeping for 5 hour, receiving 20 units of sleep. Even though the second person slept only half the time as the first person, they accumulated four times the level of sleep.

Some people get the deepest sleep at certain hours, always waking at specific times. This can often be a diagnostic tool for other problems that may be related to sugar, liver or some other health issue. Often these sleep patterns can be loosely correlated to Chinese meridian clock.

“Cat napping” is quite useful for some people. Some of the most productive people through history, such as Leonardo Da Vinci, Churchill, Florence Nightingale to name a few, only slept four hours a night, but were active cat nappers.

**The nature of sleep**

Sleep is a term used to describe an altered state of consciousness or partial unconsciousness from which an individual can be aroused. Normal sleep consists of two components, **non-rapid eye movement (NREM)** and **rapid eye movement (REM)**. NREM consists of four distinct stages that can measured by an EEG:

1. **Stage one**: the transition between wakefulness and sleep, usually lasting between 1-7 minutes. The subject’s eyes are closed and they may have fleeting thoughts. The α-waves that mark normal consciousness gradually diminish. Subjects that are roused from this state usually feel that they have not been sleeping.

2. **Stage two**: the first stage of true sleep, in which the subject is more difficult to arouse. Fragments of dreams may be experienced, and the eyes can be seen to roll from side to side. EEG readings demonstrate the characteristic **sleep spindle**, a burst of sharply pointed waves that occur in the 12-14 Hz range, lasting 1-2 seconds.

3. **Stage three**: a period of moderately deep sleep, occurring about 20 minutes after falling asleep, in
which the subject is difficult to arouse. There is a reduction in body temperature and blood pressure, and the EEG shows a mixture of sleep spindles and larger, lower frequency waves.

4. **Stage four:** the period of deepest sleep, in which large, slow delta waves dominate the EEG reading, with a reduction in brain metabolism, body temperature, and muscle tone. The subject is most difficult to arouse in this state. Sleep-walking or bedwetting will only occur during this stage of NREM sleep.

The movement from stages one to four typically occurs in less than an hour. These different phases of NREM sleep are interspersed with progressively longer periods of REM sleep during a normal 8 hour sleeping cycle, characterized by the movement of the eyes rolling back and forth under closed eyelids. The brain waves observed in REM sleep are small and irregular and resemble the waking state. The blood pressure can increase drastically and the pulse rate and breathing may become irregular, which is why REM sleep is associated with an increased risk of a myocardial infarction. Although the subject’s large muscle groups are literally paralyzed, the small muscles in the face, toes and fingers may twitch, and both men and women can experience vascular engorgement of the penis and clitoris even when the content of the dream isn’t sexual. The presence of penile erection in men suffering from erectile dysfunction indicates that the problem is more likely related to psychogenic factors.

The first episode of REM sleep usually occurs before the second hour of sleep and lasts only 10-20 minutes, followed by an interval of NREM sleep. REM and NREM sleep then alternate throughout the sleep period about every 90 minutes. The alternating period of REM sleep however gradually lengthens, such that the last period of REM sleep can last upwards of 50 minutes. In total REM sleep accounts for about 90-120 minutes of a normal sleep cycle. In an infant however REM sleep can account for up to 50% of the total sleep period, in contrast to about 35% for toddlers, and 25% for adults, with the percentage declining with age. Sleep spindles seen in stages two and three of NREM sleep usually begin by about the 3rd month of life. Although the purpose of sleep and REM sleep specifically isn’t clear, it is thought that the high percentage of REM
sleep in the young is related to proper neuronal development.

There are different parts of the brain that appear to regulate NREM and REM sleep, with neurons in the preoptic region of the hypothalamus, the basal forebrain and medulla oblongata regulating NREM sleep, and the neurons in the pons and midbrain activating and inhibiting REM sleep. The sleep spindles specifically are thought to arise in the thalamus. One of the more important chemicals thought to play a role in sleep is adenosine which accumulates when high levels of ATP are catabolized. Adenosine binds to specific (adenosine) $A_1$ receptors in the brain, inhibiting cholinergic neurons in the reticular activating system (RAS) that promote arousal. This is the neuropharmacological basis of the stimulatory effects of methylxanthines such as caffeine, which bind to $A_1$ receptors and prevent adenosine from binding, thereby inhibiting sleep.

**The purpose of sleep**

Despite observing and defining the sleep cycle, researchers are still hard pressed to come up with a reason for sleep. A multitude of theories have been put forward, many of which contradict each other. One recent Bio-medical hypothesis is that sleep is a homeostatic mechanism used to replenish glycogen stores within the brain (Kong et al 2002). During normal wakefulness, the brain catabolizes stored glycogen and glucose to manufacture ATP, which is used to power neuronal activities in the brain. The result of this is the accumulation of adenosine, which as previously described, inhibits arousal of the RAS. According to this theory, sleep is initiated when adenosine levels are high enough to promote sleep (Benington and Heller 1995). As this process is theorized to occur only during NREM sleep, a question arises as to what the purpose of REM sleep is. Benington and Heller suggest that REM is a homeostatic mechanism to induce another cycle of NREM restorative sleep (1994a, 1994b).

The concept that sleep is essentially a restorative mechanism is supported by Ayurvedic medicine. During normal wakening, ingested dietary nutrients and oxygen from breathing are catabolized in a step-wise manner to nourish the different tissues (*dhatus*) of the body. When these tissues are properly nourished, specifically those associated with reproduction (i.e. *shukla*, semen; *andanu*,...
ovum), the result is the production of *ojas*, the vital essence of the body. The function of *ojas* is to nourish and protect that body, to provide energy and endurance, and to counter-balance the catabolic effects of *agni*, the fire of digestion and metabolism. During the day *ojas* circulates through the body, supplying it with energy, ‘sacrificing’ itself so that *agni* has the intrinsic energy needed to break down the nutrients consumed in the diet. During sexual activity *ojas* concentrates in the reproductive tissues, and with orgasm, is released to create life. The depletion of *ojas* is noted by tiredness and fatigue, by an inability to continue work, and thus from an Ayurvedic perspective, it is a deficiency of *ojas* that promotes tiredness and the need to sleep. During sleep the tissues of the body can concentrate on replenishing *ojas* instead of performing work, allowing it to circulate through the body, nourishing and revitalizing the tissues. Thus in Ayurvedic medicine sleep is one of the three pillars of life, along with food and sex.

Other esoteric models suggest that sleep is time for the physical body to replenish itself, and is also a time for the other bodies (emotional, mental, spiritual) to ‘metabolize’ energy and to release built up tension. It is also thought to be a time when one can be almost free of the restraints of the physical body. This means that sleep is a way to release various emotional (or astral) conflicts in the form of dreams (especially in traveling dreams). If an emotional dream (a nightmare or simply a vivid dream sequence) is too strong it will awaken a person.

### Causes of insomnia

<table>
<thead>
<tr>
<th>Sleep-onset insomnia</th>
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<tr>
<td>Anxiety or tension</td>
<td>Depression</td>
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<td>Environmental change</td>
<td>Environmental change</td>
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<td>Emotional arousal</td>
<td>Sleep apnea</td>
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<td>Fear of insomnia</td>
<td>Restless Legs</td>
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<td>Phobia of sleep</td>
<td>Hypoglycemia</td>
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<td>Disruptive environment</td>
<td>Parasomnias</td>
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<td>Pain or discomfort</td>
<td>Pain or discomfort</td>
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<tr>
<td>Caffeine</td>
<td>Drugs</td>
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Medical management of insomnia

The medical management of insomnia depends upon the cause, and may include analgesics for patient’s suffering from chronic pain, or the use of specific psychiatric drugs if the insomnia is thought to be secondary to a psychiatric disorder such as depression. Drug therapy for insomnia usually consists of hypnotics such as benzodiazepines (e.g. clonazepam, triazolam, estazolam, flurazepam) and pyrazolopyrimidines (e.g. zaleplon, zolpidem), and tricyclic antidepressants (e.g. amitriptyline, trazodone) if depression is a part of the symptom picture. These drugs are typically advised to be used for only transient and short-term insomnia, although in many cases end up being used on a long-term basis. Many of these drugs interfere with the normal sleep cycle, inhibiting both REM and stage 4 NREM sleep, and thus can interfere with the restorative benefits of sleep. Hypnotics with a rapid onset of action, such as zolpidem, zaleplon, and triazolam are used when the patient has difficulty in falling asleep. If the patient has difficulty maintaining sleep however, a hypnotic with a slower rate of elimination may be prescribed instead, such as temazepam, estazolam, and flurazepam. Rebound insomnia is a fairly common phenomenon when the drugs are withdrawn abruptly.

Beyond specific drug therapies, behavioral therapies are increasingly recognized as a more appropriate treatment for patients with primary insomnia. These consist of:

Relaxation therapy. In relaxation therapy the patient is taught to recognize and control somatic muscular tension through a series of exercises that consist of first tensing and then relaxing each major muscle group in a systematic way, such as beginning with the feet and working upwards. Guided imagery and meditation can help the patient focus on neutral or pleasant thoughts and feelings instead of the myriad of thoughts, concerns and worries that may typically race through their head when trying to get to sleep.

Biofeedback. Biofeedback is a form of relaxation therapy that can provide the patient with direct information about physiological processes that he or she isn’t typically aware
of, such as muscle tension, body temperature, heart rate, and peripheral vasoconstriction. With the usage of a biofeedback device and special sensors attached to the skin, the technique essentially creates an external loop by which the patient can monitor these physiological states and eventually learn to control them. These devices measure physiological changes associated with emotional states such as stress and anxiety, and when physiological patterns associated with these states are sensed, the biofeedback device provides the patient with a signal, such as a colored light or sound. Patients will then try to control the lights or sound by consciously modifying their behavior. Among the different devices, there are three principle types that are commonly available to consumers: the electromyogram, temperature biofeedback, and galvanic skin response. The electromyogram (EMG) measures muscle tension by placing two electrodes on the skin over the muscle to be monitored, such as the frontalis, masseter and trapezius, which are often contracted in states of emotional stress. Temperature biofeedback monitors skin temperature by attaching a sensor to the foot or middle or small finger of the dominant hand. A decrease in body temperature is correlated with peripheral vasoconstriction that occurs with the ‘fight or flight’ response. Galvanic skin response (GSH) measures electrical conductance in the skin by discharging a small electrical current through the skin, measuring changes in salt and water content. In heightened states of arousal most people will typically sweat, increasing the electrical conductivity of the skin.

**Stimulus control therapy:** stimulus control therapy is a technique that attempts to re-associate the bed with sleepiness instead of arousal. This includes: not using the bed for activities such as reading, watching television, eating, or working. It is also important to ensure that the patient lies down only when sleepy, and if they are unable to fall asleep within a 15-20 minutes period, to get out of bed and engage in another activity until he or she feels sleepy.

**Environmental Stimulus:** There are several parameters that aid in sleep. Since a large percentage of our genetic past was literally living in caves or as hunter gathers, approaching this state is useful for sleep. This means keeping the **sleeping chamber dark, low or no noise and**
cool. Temperature is often one of the most import items that a person misses.

**Holistic treatment of insomnia**

While insomnia is similarly recognized as a symptom of an underlying disorder, herbalists have traditionally classified insomnia into two basic types: sthenic (‘hot’) and asthenic (‘cold’). **Sthenic insomnia** manifests as irritability, anger, impatience and frustration, whereas **asthenic insomnia** will manifest as anxiety, fear, worry and grief. Thus on the basis of these differences in symptomology, different approaches may be taken to promote a healthy, restful sleep. Where depression or pain is a factor in the condition, these are addressed simultaneously. Quite often the underlying problem of insomnia, especially when it occurs several hours after retiring, is related to hypoglycemia, and thus measures may also be taken to ensure proper control over blood sugar.

Another factor to consider is the disruption of normal circadian rhythms through exposure to artificial light sources, including reading lamps and television or computer monitors late at night. Like many mammals, humans traditionally awakened with the rising of the sun and retired when it set. This dynamic is still clearly observed in many traditional, ‘primitive’ societies where electricity is not available. Artificial light is a relatively recent artifact of modern civilization, and being exposed to light sources at night could down-regulate melatonin release and thereby promote nighttime waking.

From an Ayurvedic perspective the best time to sleep is when kapha is most active, between the hours after sunset and late evening. As the night progresses, pitta becomes active, and if the patient stays awake during this period the fires of the body become very active, stimulating the mind and even digestive processes (i.e. a craving for the ‘midnight snack’). Thus the best time to retire is during the kapha period of time, between the hours of 8 to 11 pm, to take advantage of the natural lethargy and somnolence that it produces in the body.

**Sleep Direction:** Many people sleep best when their head is pointing **North**, while others sleep best when their head is pointing **South**. Strong geographical features such as rivers, hills and magnetic field can change this.
**Botanicals**

- **Sthenic insomnia**, with heat, palpitations, flushing, and irritability: *Leonorus, Scutellaria, Passiflora, Lycopus, Verbena, Tilia*
- **Asthenic insomnia** with coldness, anxiety and fear: *Ganoderma, Valeriana, Anenome, Piper methysticum, Nardostachys*
- **Cerebral insomnia**, for overthinking, racing thoughts, best taken throughout the day in divided doses e.g. *Ganoderma, Withania, Avena*
- **Non-specific hypnotics**: *Lactuca, Eschscholzia, Meconopsis, Papaver*
- **Delayed onset sedatives**: *Myristica*
- **Antispasmodics**, to ease somatic muscular tension: *Cimicifuga, Piper methysticum, Piscidia, Myristica*
- **Flushing and night sweats associated with menopause**: *Salvia, Vetivera, Mesua, Santalum, Schizandra, Astragalus, Phellodendron, Ligustrum, Cornus*
- **Stomachics and carminatives**, to relieve indigestion and bloating: *Foeniculum, Zingiber, Carum, Zanthoxylum, Elettaria, Coriandrum, Acorus*

**Suggested Program**

**Breakfast**  Reishi premium (15:1 extract; 2 capsules), Nerve Formula (2 capsules).

**Lunch**  Same as breakfast.

**Supper**  Same as breakfast.

**30 min before bed**  Valerian tincture (30 drops), Melatonin (3-6 mg).

**Nerve Formula**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Dosage</th>
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<tbody>
<tr>
<td>Valeriana officinalis</td>
<td>100 mg</td>
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<tr>
<td>Oats Avena sp.</td>
<td>100 mg</td>
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<tr>
<td>Hops</td>
<td>100 mg</td>
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<tr>
<td>Humulus lupulus (2:1)</td>
<td>100 mg</td>
</tr>
<tr>
<td>Scutellaria lateriflora</td>
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</tr>
<tr>
<td>Black Cohosh</td>
<td>75 mg</td>
</tr>
<tr>
<td>Passiflora spp.</td>
<td>50 mg</td>
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</tbody>
</table>

**Nutritional supplements**

- Vitamins: B complex, 50-100 mg, taken before 12 pm
- Minerals: calcium/magnesium for RLS/PLMD, 1:1 ratio, 800-1200 mg each before bed; chromium, to control blood sugars, 200 mcg t.i.d. with meals
- melatonin: 3 mg before bed. This will only work if melatonin levels are low.
- tryptophan: 3 – 5 g; or 5-HTP, 100 – 300 mg

**Diet**

- calorie-restricted diets may interfere with sleeping patterns
- eliminate coffee, methylxanthine containing foods (tea, chocolate) and caffeine-containing medications
- alcohol used as a sedative may cause night-time awakening due to rebound hypoglycemia
- rule out food sensitivities (e.g. cow’s milk, wheat, sugar, food additives and preservatives)
- high carbohydrate diets may promote rebound hypoglycemia
Aromatherapy
- Lavender, Ylang, Bergamot, Melissa, Lemon verbena, Vetivert, Jasmine, Rose, Sandalwood

Flower Essences
- Chamomile, Trembling Poplar
- Night, Night

Night, Night Flower Essence
Night, Night is beneficial for the person who has a hard time falling asleep, or who wakes up after only a few hours and is not able to fall back to sleep. It is especially useful for the person who is awakened by vivid or disturbing dreams. Night, Night has been specifically designed to aid in a deeper, more restful sleep.

Trembling Poplar – aids in allowing a calm sleep, reducing anxiety and bad dreams
Chamomile – reduces the hyperactivity and anxiety that prevents a person from getting into a high-quality deep sleep; releases emotional tension stored in the stomach and solar plexus
Dill – helps a person have a restful sleep when they would normally experience sensory and nervous overload; helps integrate daily experiences
Mugwort – reduces sleep interruption caused by overactive dreaming
Saint John’s Wort – reduces dream disturbances associated with fear or psychic stress

Lifestyle
- encourage the patient to write their thoughts, worries and concerns onto paper or in a journal before bed-time
- implement a meditation regimen before bed and in the morning
- biofeedback
- Reversal: To help lower the conflict in dream states, an ancient “Blue” mystery school technique from the Middle East, is beneficial. The technique is performed as follows. Think about the last thing you did before going to bed, then the thing before etc. This is continued until you recall back to the first thing that happened in the day, or until you fall asleep. If a person comes across any incident that is highly ‘charged’ (eg being cut off by a car, causing you anxiety), tell yourself to release this emotion. Check to see if it is
released, if the answer is no, repeat the clearing. If after
the second time it seems to still contain a ‘charge’,
recall a short time before the incident and tell yourself
that it will not effect you, (eg. I know a car is going to
cut me off, but I will not react negatively to it). This
releases some of the mundane energies contained in the
emotional body, so that dreams don’t have to do it later.

- Heal-Toe Clapping: this ancient Korean Qi Gong
exercise is quite simple. While laying down on one’s
back, with bare feet, heels touching, a person ‘claps’
their toes together as fast as they can, for as long as
they can, up to 2 minutes. This seems to release
electrostatic electricity in the head (brain) area,
releasing it through the bottom of the feet.
- suggest that the patient turn the lights down low by
about 9 pm, and discourage reading, watching
television, or working on the computer afterwards
- encourage regular exercise during the day to discharge
somatic tension
- warm oil massage with unrefined sesame oil, over the
ears, large joints and feet, particularly in the ‘cold’
forms of insomnia, characterized by over-thinking,
anxiety and fear
- adjust the room temperature to a comfortable sleeping
temperature, typically a few degrees cooler than room
temperature. For ‘hot’ insomnia the room may need to
be considerably cooler. Fresh air while sleeping is
important. For patient’s complaining of coldness
however, ensure proper bed clothing and even socks to
keep warm
- remove extraneous noises as best as possible;
encourage treatment for partners that snore.
Affective disorder: Depression

Affective disorder (Depression) is a family of diseases characterized by changes in mood. Major depression occurs in 10 – 20% of the world’s population in the course of a lifetime. There are more than 30 million people in North America taking antidepressants on a regular basis. There is a 2:1 ratio of women to men who suffer from depression. Female hormones, childbirth and menopause all seem to be contributing factors for some women. There are two basic types: the melancholia of unipolar depression, and the manic elevation and desperate lows of a bipolar affective disorder. The prevalence of unipolar depression is fairly equally distributed, but bipolar disorders seem to affect more women and young adults.

The fourth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV) provides the diagnostic criteria for a major depressive episode. At least five of the following symptoms must be present in the same two week period, and also must include either (a) or (b):

(a) Depressed mood  
(b) Diminished interest or pleasure  
(c) Significant weight loss or weight gain  
(d) Insomnia or excessive sleep and lethargy  
(e) Psychomotor agitation or retardation  
(f) Fatigue or loss of energy  
(g) Feelings of worthlessness  
(h) Diminished ability to think or concentrate; indecisiveness  
(i) Recurrent thoughts of death, suicidal ideation, suicide attempt, or specific plan for suicide

There are a number of predisposing factors in depression, including:

• grief due to the illness or loss of a loved one  
• the recent diagnosis of a severe medical condition, e.g. cancer, multiple sclerosis  
• chronic pain and disability  
• nutrient deficiencies, e.g. vitamin B12  
• mood changes in the elderly, and in particular, depression associated with aging and diseases
associated with aging that affects the quality of life, including arthritis, Alzheimer’s disease, Parkinson’s disease, and stroke

- endocrinial disorders, e.g. hypothyroidism, Addison’s disease, Cushing’s disease, hyperthyroidism, prolactinomas, hyperparathyroidism

- side-effects from drug therapy, e.g. antihypertensive medications (especially β-blockers, reserpine, methylldopa, and calcium channel blockers); steroids; medications that affect sex hormones (e.g. estrogen, progesterone, testosterone, gonadotropin-releasing hormone [GnRH] antagonists); H2 blockers (e.g. ranitidine, cimetidine); sedatives; muscle relaxants; appetite suppressants; chemotherapy agents (e.g. vincristine, procarbazine, L-asparaginase, interferon, amphotericin B, vinblastine).

- substance use, abuse, or dependence, e.g. alcohol, cocaine, amphetamines, marijuana, sedatives/hypnotics, and narcotics

- post-partum depression

- seasonal affective disorder (SAD)

- anxiety disorders, including panic disorder, obsessive-compulsive disorder, generalized anxiety disorder, posttraumatic stress disorder, and phobia

- eating disorders, e.g. anorexia nervosa, bulimia

- borderline personality disorders

- psychosis (Berkow 1992, 1594-95)

The physical signs and symptoms of depression can be mild to acute. Most patients present with a relatively normal appearance, but with severe symptoms may show an inattention to personal appearance and hygiene. There may be a general decline in spontaneous movements, often commensurate with a flattening of emotional expression, evidenced by speaking patterns that are slow, monotonic, or lacking in spontaneity and content. The patient may have gained weight recently, or lost a significant amount of weight, and may suffer from vertigo and poor coordination, fatigue and lethargy. Psychological symptoms may include:

- sadness

- a feeling of heaviness

- emotional numbness

- irritability

- mood swings

- a loss of interest or pleasure in their usual activities
• difficulty concentrating
• decreased motivation
• feelings of worthlessness, hopelessness, or helplessness
• suicidal or homicidal ideation

While there are no objective criteria to examine for depression a number of laboratory tests may be undertaken to rule out underlying factors, including thyroid-stimulating hormone, vitamin B₁₂, liver function tests, toxicology and EEG.

Organic and Physiologic Causes of Depression

• Preexisting physical condition
  ◦ Diabetes
  ◦ Heart disease
  ◦ Lung disease
  ◦ Rheumatoid arthritis
  ◦ Chronic inflammation
  ◦ Chronic pain
  ◦ Cancer
  ◦ Liver disease
  ◦ Multiple sclerosis
• Prescription drugs
  ◦ Antihypertensives
  ◦ Antiinflammatory agents
  ◦ Birth control pills
  ◦ Antihistamines
  ◦ Corticosteroids
  ◦ Tranquilizers and sedatives
• Premenstrual syndrome
• Stress/low adrenal function
• Heavy metals
• Food allergies
• Hypothyroidism
• Hypoglycemia
• Nutritional deficiencies
• Sleep disturbances
Medical treatment of depression

From a neuropharmacological perspective, depression is primarily correlated with a disturbance in CNS serotonin, and to a lesser extent problems related to norepinephrine (NE) and dopamine (DA) function. The role of serotonin in depression is supported by clinical trials that have suggested the efficacy of serotonin reuptake inhibitors (SSRIs), as well as other studies that demonstrate an acute, transient relapse of depressive symptoms in tryptophan deficiency. The idea that it is a deficiency of serotonin that causes depression is a perspective that has become strikingly popular among medical professionals such that these drugs account for a significant percentage of profits enjoyed by the drug companies that produce them. It is a recipe that is convenient and ideally suited to a clinical practice where a doctor may spend only a few minutes with a patient before arriving at a diagnosis of depression and prescribing a serotonergic therapy. Unfortunately SSRIs have multiple negative side effects including weight gain, loss of libido and headache, and are notorious for interacting with other drugs. The drug companies appear to be very aggressive in protecting their market share however, and have published a few studies indicating that often used alternatives to SSRIs such as St. John’s Wort (Hypericum perforatum) are no better than placebos. In one double-blind placebo-controlled clinical study of 340 patients suffering from mild to moderate depression, a positive response to St. John’s Wort (SJW) was only found in 23.9% of the patients, compared to 31.9% for placebo (Hypericum Depression Trial Study Group 2002). What the press release failed to mention however is that the study also examined the efficacy of sertraline (Zoloft®), which accounts for upwards of two billion dollars in sales, and found that this drug was no better than St. John’s Wort in treating mild to moderate depression (with a 24.8% response). Despite the fact that SJW was never traditionally used for depression per se, other clinical trials have consistently shown the benefits of SJW over placebo and that it is at least equivalent, or more effective than drugs such as fluoxetine (Prozac®), (Behnke et al 2002; Schulz 2002; Schrader 2000). More than 27 double-blind, randomized trials, involving more than 2200 patients with mild to moderately severe depression, have shown St. John's Wort yields excellent results in the treatment of depression with far fewer side effects than standard antidepressant medications.¹
Pharmacotherapy
Pharmacotherapy for depression usually consists of a two to six week introductory period, during which time the benefits of the drug will begin to be seen. Among the different drugs are:

- selective serotonin reuptake inhibitors (SSRIs): fluoxetine (Prozac®), paroxetine (Paxil®), sertraline (Zoloft®), fluvoxamine (Luvox®), citalopram (Celexa®), and escitalopram (Lexapro®). SSRIs block the reuptake of serotonin with little effect upon norepinepherine. Common side effects include anorexia, nausea, headache, fatigue, insomnia, sexual dysfunction, drowsiness, dizziness and agitation.

- serotonin noradrenergic reuptake inhibitor (SNRI): venlafaxine (Effexor®). Venlafaxine inhibits the reuptake of both serotonin and noradrenaline. Side effects include sedation, nausea, headache, and sexual dysfunction.

- serotonin 2 antagonist/reuptake inhibitor (SARI): trazodone (Desyrel®). Trazodone affects serotonin and noradrenaline secretion, increases REM sleep and promotes sedation. Prominent side effects include a dry mouth, sedation, dizziness, and nervousness.

- norepinepherine, dopamine reuptake inhibitor (NDRI): bupropion (Wellbutrin®). Bupropion is a weak inhibitor of the neuronal uptake of serotonin, norepinephrine and dopamine, but the specific mode of action is not known. Side effects include headache, insomnia, agitation and seizure.

- noradrenergic/specifc serotoninergic antidepressants (NaSSA): mirtazapine (Remeron®). Mirtazapine increases noradrenergic, dopaminergic and serotoninergic activities. Side effects include sedation, increased appetite, weight gain, dizziness and dry mouth.

- tricyclic antidepressants (TCAs): amitriptyline (Elavil®), nortriptyline (Pamelor®), desipramine (Norpramin®), clomipramine (Anafranil®), doxepin (Sinequan®), protriptyline (Vivactil®), trimipramine (Surmontil®), and imipramine (Tofranil®). Tricyclic antidepressants are so-called because of their 3-ring chemical structure. Generally speaking, TCAs increase the activity of norepinepherine and serotonin by blocking neuronal uptake, with increased stimulation of postsynaptic receptors. Their relative lack of specificity however leads to anticholinergic effects and
cardiotoxicity. The most common side effects include sedation (antihistamine H1 blockade), dry mouth, constipation, urinary retention, blurred vision, orthostatic hypotension, dizziness, and weight gain.

- monoamine oxidase inhibitors (MAOIs): phenelzine (Nardil®) and tranylcypromine (Parnate®). MAOIs inhibit the enzymatic degradation of monoamines neurotransmitters including norepinephrine, dopamine and serotonin, increases post-synaptic stimulation. Side effects include dizziness, anxiety, tremors, insomnia, weight gain, and sexual dysfunction, and have many drug interactions. With the concomitant consumption of foods rich in tyramine (e.g. fermented foods such as aged cheeses, red wine, prepared meats, coffee etc.), TCAs, and SSRIs, MAOIs can promote a life-threatening hypertensive crisis.

**Non-pharmacotherapeutic medical options**

In addition to drug therapy, a variety of non-drug options may be used instead of or in conjunction with standard drug treatments.

- Electroconvulsive therapy (ECT): ECT or “electroshock” involves the application of electrodes placed above each temple or above the temple of one side of the brain and in the middle of the forehead (unilateral ECT). An electrical current is then passed through the brain via the electrodes, inducing a grand mal epileptic seizure. Prior to the application of the electrical current the patient is anesthetized with an intravenous injection of a barbiturate or an anaesthetic, and the muscles are temporarily paralyzed with a drug such as succinylcholine, preventing the violent jerking motions that can cause bones to break. Risks include those associated with anesthesia, as well as post-treatment confusion, short-term memory difficulties, and death. ECT is typically used when a rapid antidepressant response is prescribed, when drug therapies have failed.

- Light therapy: Broad-spectrum light exposure has long been shown to be effective for SAD, with some recent evidence indicating that it may be helpful in non-seasonal depression.

- Psychotherapy: Psychotherapies employ a belief that unconscious conflicts and distortions are at the heart of depression. Through a dialogue with a skilled practitioner, the aim of psychotherapy is to help the
patient to create a new perception and relationship with the world. In some cases psychotherapy may involve many years of treatment.

**Holistic treatment of depression**

At the heart of affective disorders is the perception of the human condition. Life, for all that it offers, has long been recognized as a source of pain and suffering for some. The reality of suffering is reflected in many of the religious and philosophical teachings of the world, from suffering of the Jews recounted in the Bible, to the painful crucifixion of Jesus. These teachings show us however that suffering is not only an inevitable reality, but that it is a vast storehouse of wisdom and knowledge. Affective disorders can be ‘hardware problems’, where there is an organic cause to a problem. Or they can be ‘software problems’ where one’s own thought processes cause the issues. Everyone has good days and bad days. Depression happens when the perception that there is more bad than good. Often what we ‘learn’ from the bad times helps us see the good times as all that much brighter.

The Yogic and Buddhist teachings of India suggest that life is an infinite wheel of birth, death and rebirth called *samsara*. When each of us is born, we are taught that we have an identity and a place within society. We are encouraged to develop this identity, into a career, into social and personal obligations, and soon we invest a great deal in supporting and protecting this sense of individuality (ego). Somewhere down the line however, the existential realities of life cause a disruption of this ego-complex, and he who was once a powerful CEO soon becomes an old man that has to be tended to as an invalid: we lose it all. The heart of the issue is this identification with the ego-complex, that we attach such an enormous significance to that which is transient, impermanent and subject to decay. We think we know who we are, and we have our many preferences and aversions to support it. But, in the end, we lose it all.

Buddhist teachings tell us that it is this conditioned ignorance, perhaps necessary for survival that binds and confines our unconditioned inner natures to a narrow and specific circumstance. Yogic teachings posit that we are holograms of God, a microcosm of the macrocosm, and it is the persistent belief that the self exists independently of
God that binds us to the wheel of *samsara*. Viewed in this context depression is something quite natural: coming face to face with the face that isn’t there. Who are you with no face, with no identity? Depression is the precursor to the next stage of spiritual development, where we escape the limitations of identity and learn to realize our unconditioned existence and merge with the bliss of the Infinite.

Holistic therapies treat a broad range of possible causes of depression, including emotional disturbances, chronic stress, fear, low self-esteem, loneliness, nutritional deficiencies, food allergies, hypothyroidism, obesity, candidiasis, heavy metal toxicity and hypoglycemia. In many cases of depression there is a concurrent issue of immune dysfunction. In women who are obese, hirsute (eg. facial hair) and depressed, there is a strong possibility of polycyclic ovarian disease, or at least, a relative androgenization and deficient progesterone. In such cases it is helpful to assess hormone levels, either through laboratory testing or by asking the woman to keep a symptothermal chart (see *The Human Flower: Reproductive Health and Botanical Medicine*).

In Ayurvedic medicine depression is a symptom of an underlying imbalance of the doshas. Thus treatment is centered around balancing or pacifying the affected doshas. Vataja depression is noted by concomitant symptoms of fear, anxiety, being “spaced-out” and confusion, with physical symptoms of coldness, dryness, numbness and fatigue. Pittaja depression is manifest as emotional irritability, rage, intense anger, and violence, with physical symptoms of heat and flushing. Kaphaja symptoms are recognized by feelings of worry, grief, and sentimentality, with physical symptoms of catarrh, heaviness, lethargy and coldness. Combined symptoms pictures are recognized as a combination of the doshas.

**Caution is warranted when withdrawing antidepressant drugs in patients that have been on these drugs for more than eight weeks. Withdrawal symptoms include headaches, dizziness, restlessness, tremors, insomnia and diarrhea.** To avoid these symptoms wean the patient off the drugs over a period of two weeks – to two months, gradually increasing the dosage of alternative remedies. Avoid the use of botanicals with an MAOI activity (e.g.
Peganum harmala, Banisteriopsis caapi) with any antidepressant or tyramine-rich foods.

Botanicals
- Thymoleptics, to allay sadness and promote happiness: Hypericum, Melissa, Anenome, Verbena, Passiflora, Piper methysticum, Bupleurum chinense, Ganoderma, Cordyceps, Rhodiola, Withania, Peganum
- Antimanics, for rage and aggression: Rauwolfia, Gelsemium, Scutellaria, Passiflora,
- Nervine trophorestoratives, for neurasthenia: Avena, Bacopa, Nardostachys, Centella, Acorus Turnera, Withania, Panax, Schizandra, Eleuthrococcus, Rhodiola, Glycyrrhiza, Zizyphus, Ganoderma
- Cholagogues, for detoxification: Gentiana, Taraxacum radix, Berberis
- Stimulants, for lethargy and mental confusion: Capsicum Piper longum, Zingiber, Zanthoxylum, Rosmarinus officinalis, Ocimum
- Cerebrovascular stimulants, for the aged: Ginkgo, Vaccinium
- Entheogens: MAOIs Banisteriopsis caapi and Peganum harmala, with DMT-containing plants such as Psychotria viridis, Diplopterys cabrerana, or Desmanthus illinoensis. **USE ONLY UNDER EXPERIENCED SUPERVISION**

Supplements
- vitamin B complex, 200 mg daily, with B12 and folic acid, 800 mcg each
- vitamin C, to bowel tolerance
- chelated multiminerals with trace minerals
- chromium, to control blood sugar, 200 mcg t.i.d.
- EPA/DHA, 3000 mg each daily
- L-tryptophan (taken with B vitamins)
- 5-hydroxytryptophan
- Vitamin D3, 4,000 IU daily
- Omega 3 fatty acids (2 – 3,000 mg; twice daily)

Topical
- cold water showers in manic states
- paste of Sandalwood powder mixed with cool milk applied to forehead (pitta)
- warm oil massage in asthenic conditions (vata)
• Ayurvedic *udvartana* (herbal powder massage) and *garshana* (massage with raw silk gloves) (kapha)

**Diet**
• remove food allergens with an elimination-challenge diet
• eliminate methylxanthines, in coffee, tea, chocolate and certain medications
• avoid alcohol: regular alcohol consumption can reduce serotonin levels
• rule out hypoglycemia
• rule out aluminum, lead, mercury and heavy metal toxicity

**Aromatherapy**
• lavender, rose, vetivert, sweet marjoram, bergamot, lemon, clary sage, myrrh, frankincense, sandalwood, cinnamon (vata)
• chamomile, lavender, rose, gardenia, honeysuckle, ylang, vetivert, jasmine and sandalwood (Pitta)
• cedar, pine, rosemary, basil, frankincense, myrrh, eucalyptus, cajeput, camphor, ginger and clove (kapha)

**Flower Essences**
• Buttercup (self-esteem), Cerato (self-esteem), Mimulus (fear), Gorse (hopelessness), Mustard (melancholy), Red Chestnut (worry), Rock Rose (panic), Sweet Chestnut (despair)

**Depression and Apathy Up Lifter (DAU) Flower Essence**
This combination of flower essence and homeopathic gold is used to open the heart and help one give and receive joy, love, and compassion. Used for simple to deep-seated depression, it brings a ray of sunshine into an otherwise gloomy life. It will help one to embrace higher spiritual principles in ordinary living. We have had great success with this formula over the last few decades. Depression and apathy come in many forms. This flower essence formula works on the deeper aspects of self-worth and self-love.

**Borage** – helps release heavy-heartedness, lack of confidence in facing difficult circumstances, depressive behavior.

**Wild Rose, Alberta** – gives us motivation to live with joy and release apathy.
Chamomile – produces a peaceful, sunny disposition, emotional balance full of self worth.

Saint John’s Wort -- creates an illuminated consciousness, filled with spiritual contact and solar strength.

Aurum – homeopathic gold has been used for various levels of depression for well over 100 years. It aids in rebuilding one’s self worth.

Gorse – equanimity and light-filled optimism and a builder of self-worth, used when a dark cloud comes over one’s life.

Other
- regular exercise
- meditation, tai chi, hatha yoga

Attention deficit disorder and attention deficit hyperactive disorder

Attention deficit disorder (ADD) and attention deficit hyperactive disorder (ADHD) are chronic disorders of attention span and impulse control, which can begin in infancy and continue through adulthood. This health issue, or more correctly, syndrome is quite common with 5 – 15% of school age children being diagnosed. It is responsible over 50% of referrals for childhood diagnosis. ADD is characterized by short attention span and poor impulse control, whereas ADHD includes these features in association with hyperactivity. Many children are not diagnosed with ADD/ADHD until specific behaviors are noticed in the classroom environment. However, many children who appear to have ADD/ADHD characteristics such as inattentiveness, impulsivity, underachievement and hyperactivity do not have this condition. Unfortunately ADD/ADHD is all too often over diagnosed, and as a result, over treated. A confirmed diagnosis of ADD/ADHD requires an ongoing joint assessment by the physician, a psychologist and (if it’s a child) the teacher. In the case of children it’s important to realize every child has unique needs in learning and play that cannot be addressed by one system of education. A new term, called “spirited,” is a more descriptive term for some children who are otherwise intelligent and perceptive, but have a difficult time reconciling their unique attributes within a homogenous
standard. Recently the term ‘indigo children’ has been used to describe these children, and advocates of this title appreciate these children for their vivacious natures that are often confronting for older generations. Like ADD/ADHD people, spirited people may have a difficult time screening out external stimuli in order to complete a given task, but unlike ADD/ADHD, can usually perform to an acceptable level if effort is made to accommodate their unique needs in the learning environment. ADD/ADHD has also been linked to periods of high stress in the home during early childhood.

There is no definitive diagnosis for ADD/ADHD, but rather a spectrum of behaviors that depending upon the context, may or may not be criteria for an accurate diagnosis. When EEG tests have been conducted on ADD/ADHD children there appears to be diminished electrical activity of the right frontal, central and temporal regions of the brain when engaged in such activities as listening to a story or figuring out math problems. This literally means they have diminished Executive control centers. These parts of the brain are thought to attend to the individual’s ability to plan, organize, and control one’s social, motor and emotional behavior, as well as to form one’s ability to concentrate. These same brain centers are indicated in addiction issue. Often people with ADD/ADHD have a higher than normal tendency to becoming addict, especially if drugs are used to suppress the symptoms.

Individuals with ADD may express the following symptoms:

• Easily distractible
• Difficulty listening and following directions
• Difficulty focusing and sustaining attention
• Inconsistent performance in school work
• Difficulty remaining seated
• Disorganized, lose things easily
• Poor study skills, difficulty working independently
• Talks excessively
• Doesn’t listen to what is said
ADHD is used to describe the student, who, in addition to the above conditions, displays the following behaviors:

- High activity level
- Trouble with transitions and making changes
- Aggressive behavior
- Socially immature
- Impulsivity and lack of control
- Low self esteem and high frustration level

It appears that the goal of modern education is to reduce human behavior to an acceptable norm, and that any behavior outside this norm prompts a quick “evaluation” of the offending child, leading to an equally quick “diagnosis” and “treatment.” The problem with such an approach is obvious: we are all unique individuals with specific learning styles. Large classroom sizes, overworked teachers and under-funded schools all contribute to the difficulty in accommodating a wide range of behaviors.

Two hundred years ago children never spent most of their waking lives in the classroom: they were outside shoveling out the hen-house or feeding the cows, helping in the kitchen or with the family business, learning skills as they could use them. Formal education was often based on the apprenticeship model, one or two students working under a teacher’s direct supervision. But, society changed, and as the industrial revolution fell upon us the whole dynamic of the social fabric was altered. Cottage industries based in the home were replaced by jobs at the factory, and people were uprooted from an agricultural, pastoral lifestyle to an urban environment.

Unfortunately, school can only teach a very limited number of skills, and most of these skills are out of context with the reality of children’s lives. Make no mistake: school is work, and for some children, the worst kind of work. Is it any surprise that many children look for ways to turn off
their mind when they get home from school, by playing video games or watching hours of television? Could it be that the high number of ADD/ADHD children is simply an artifact of our industrialized society, where children are no longer children and are expected to behave like adults, but with none of the privileges?

The cause of ADD/ADHD is multifactorial. Apart from inappropriate learning environments, there are very often allergies or sensitivities to a wide range of dietary articles, such as food preservatives, artificial colours and flavorings, herbicides and pesticides. In many cases there is reactive hypoglycemia, and considering the level of sugar that some of these children are consuming this should be no surprise. Additional factors may include air-borne environmental toxins, mercury amalgams, a lack of fresh air, a lack of exercise, overexposure to the rapid-fire images of multimedia, and chronic infections such as Candida. Above all, these children are extremely sensitive to their environment and need much love and patience.

Medical treatment of ADD/ADHD

Medical treatment for ADD/ADHD consists of the use of psychostimulants such as methylphenidate (Ritalin) or antidepressants such as desipramine (Norpramin). Possible side effects to methylphenidate include: appetite suppression, sleep disturbances, irritability, motor and facial tics, depression and lethargy. Possible side effects to desipramine include: nervousness, sleep problems, fatigue, stomach upset, dry mouth, tachycardia and arrhythmia. It is a sad statement that parents are so often coerced into drugging their children to treat what is truly a complex phenomena. No information on diet. No information on possible allergens. No information: just a prescription waiting to be filled. The conclusion one could make that in our increasingly disposable society, we can dispense with a child’s unique personality like it is a Styrofoam container.

Pharmaceutical restraints on these children can be highly problematic, especially in the long run. Many authorities feel that the lack of frontal lobe activity (often considered the executive center), shares the same EEG as with addiction. The dopaminergic system, particularly dopamine D2 receptors have been closely linked. These receptors are often considered reward centers, needing to be stimulated to gain a sense of self-worth. Many forms of
substance abuse (e.g. drugs, alcohol, food, sexual addiction, tobacco, gambling) will also stimulate these centers. This means several things. Often pharmaceutically restrained ADHD children start changing to stronger drugs in adolescents, selling their prescription drugs to pay for new drugs; with enhanced addiction tendencies. When a person with these tendencies is pharmaceutically suppressed, it also suppresses development of executive centers in late adolescent, thus resulting in semi-sociopathic tendencies. On the other hand, usually these children are some of our brightest, most creative and inventive children. Left to develop mostly on their own, with coaching on self-esteem, they can become some of the most productive individuals in our society.

A plea to teachers: please remain flexible, committed and willing. Don’t buy into pathologizing something that is a reflection of a wider problem. Research into the way children learn indicates that most children generally retain only 10% of what they read, 26% of what they hear, 30% of what they see, 50% of what they say and hear, 70% of what they say, and 90% of what they say and do. Some children learn better visually, some by listening, and others by doing: include all three aspects in the classroom. An extremely useful tool to use in class is Brain Gym, a series of simple exercises based on Bio-Kinesiology that can be performed every morning, after breaks, after lunch hour and anytime things are getting out of hand in the classroom. Ensure that fresh, clean water is available to all your students in the classroom.

A plea to parents: if both of you work, if you are a single parent, or if you are having problems of your own, don’t be surprised that the conflict that these situations engender may be reflected in your child. Part of this issue rests with you, and if it means changing your lifestyle, cutting back the number of hours you work or seeking the help of a counselor, your child is worth it. The opportunity to build something profound with your child is such a short period of time.

While ADD/ADHD is often misdiagnosed, there are legitimate cases, especially in fetal alcohol syndrome, prenatal drug use, birth complications and in children who were raised in high stress environments as infants. The important thing to realize about ADD/ADHD is that there
is a spectrum of behaviors, not just one type, from “spiritedness” up to severe forms of ADD/ADHD that resemble autism. The following protocol applies to all children with behavioral issues, whether spirited, misdiagnosed or truly ADD/ADHD. The length and scope of treatment may be considerably different in each case, however. Implement dietary changes first, and then over a period of time add treatment strategies as needed.

**Holistic treatment of ADD/ADHD**

**Botanicals**

- Nervine relaxants, to promote relaxations and equanimity: *Matricaria, Lavandula, Passiflora, Verbena, Valeriana, Scutellaria, Stachys, Nepeta, Melissa*
- Nervine trophorestoratives: *Avena, Ganoderma, Rhodiola, Panax quenifolium, Ginkgo, Centella, Bacopa, Acorus, Withania, Eleuthrooccus, Turnera*
- Alteratives, to promote detoxification: *Trifolium, Rumex, Curcuma, Guaiacum, Fucus, Berberis*

**Supplements**

- vitamin B complex, 100 mg t.i.d.
- vitamin C, to bowel tolerance
- iron, 20 mg b.i.d.
- calcium/magnesium, 1:1, 800 mg each b.i.d.
- zinc, 50 mg daily
- chromium, 200 mcg t.i.d.
- EPA/DHA, ensures proper neural development, 2000 - 4000 mg each daily, or Krill oil 500 – 1500 mg daily
- Theanine, 100 – 200mg, b.i.d.

**Diet**

- high protein, low carbohydrate and sugar-free diet
- eliminate common allergens, e.g. dairy, wheat, citrus
- avoid packaged foods
- organic produce and free range meat only
- rule out reactive hypoglycemia

For allergens associated with gut problems we often follow the gastrointestinal rehabilitation program outlined in Pizzorno, *Textbook of Natural Medicine* (3rd edition). This

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2 NOTE: All doses are approximated for a 65 kg adult. To determine an appropriate dose for a child divide the child’s weight in kilograms by 65 kg to determine the percentage of the adult dose. Thus an adult dose of 5 mL for a 30 kg child would be (30/65=) 46% of 5 mL, (5 mL x 0.46 =) or 2.3 mL.
program is included as an appendix to this lesson. This approach to gut rehabilitation follows a rational order that provides for optimal physiologic responses to treatment. The result of following this program to its completion in children with ADHD can be quite dramatic.

**Focus Flower Essence**
Excellent for the student that needs to focus more on the task at hand, whether that task is intellectual or physical. It helps a person to see the bigger picture so they can focus on the whole, not just the parts. Focus will aid in bringing one’s center of attention to a place where they can perform the task for their highest good.

**Shasta Daisy** – helps bring all the details into a more holistic focus

**Cosmos** – helps integrate and focus thought and speech

**Dill** – used when a person is overwhelmed due to over-stimulation, hypersensitivity to their environment or to other activity, sensory congestion

**Yarrow, Pink** – maintains self-aware focus so one doesn’t feel the need to merge with others

**Nasturtium** – helps balance the focus of intellectual life with inner strength of heart energy

**Lady Slipper** – brings higher spiritual values into one’s daily work

**Aromatherapy**
- citrus, melissa, chamomile, hops, lavender, rosemary, sage

**Topical**
- oil massage, medicated with Acorus, Bacopa, Centella and Withania

**Counseling**
- Biofeedback
- Biokinesiolgy (Brain Gym)
- NLP

**Additional factors**
- heavy metal toxicity (aluminum, cadmium, lead)
- rule out environmental toxins (at home, school or workplace)
- rule out candidiasis and immunodeficiency
Autism

Autism is an abnormal condition of early childhood in which the child has difficulty establishing relationships with other people, exhibiting extreme withdrawal and a refusal to communicate. Similarly to ADD and ADHD, EEG readings in autistic children indicate a dysfunction in the electrical activity of the frontal and temporal lobes. It is one of three recognized disorders in the autism spectrum (ASDs), the other two being Asperger syndrome, which lacks delays in cognitive development and language, and Pervasive Developmental Disorder-Not Otherwise Specified (commonly abbreviated as PDD-NOS), which is diagnosed when the full set of criteria for autism or Asperger syndrome are not met. The number of people with autism is about 1–2 per 1,000 people worldwide; however, the Centers for Disease Control and Prevention (CDC) reports approximately 9 per 1,000 children in the United States are diagnosed with ASD.\(^2\) The number of people diagnosed with autism has increased dramatically since the 1980s, partly due to changes in diagnostic practice; the question of whether actual prevalence has increased is unresolved.\(^3\) There has been links to environmental factors like vaccinations and bowel microorganisms (from antibiotic use), but none of this evidence is conclusive.

Autism is a complex issue, most likely made up of many factors and described by many theories. One theory for the more severe forms of autism are linked to Fragile X syndrome, a genetic disorder in which a small portion of the tip of the X chromosome is susceptible to breakage. Fragile X is the leading cause of mental retardation among newborns, and is more common in males. Among the features of Fragile X children are learning difficulties and emotional impairment, oversized ears, elongated forehead, enlarged testes and double jointedness. Individuals that display features in the autistic spectrum but without any evidence of mental retardation may be classified as suffering from Asperger’s syndrome.

An autistic child may exhibit obsessional motions such as erratic movements of the fingers or limbs, facial grimacing or twitching. Seizure disorders are common in individuals with autism. If the autistic child’s normal environment is disturbed he may fly into a rage or retire into an anxious
brooding. Efforts by the parents to correct this behavior will often result in hostility and even hysteria. Autistic children grow up to be very sensitive, temperamental adults, with some even displaying exceptional talents.

The cause of autism is for the most part idiopathic, although several hypotheses exist, including genetic abnormalities, obstetric complications, exposure to toxic agents, and prenatal, perinatal, and postnatal infections. Maternal rubella in particular is associated with significantly higher rates of autism. There is also strong anecdotal evidence and some clinical evidence that autism is linked to early vaccination, and specifically, the usage of the mercurial preservative thimerosal that was and still is found in some childhood vaccines such as DPT.

Just as ADD/ADHD is often associated with ‘Indigos’, many associate the increase numbers of autism with the next group of ‘crystal’ and ‘rainbow’ children. Could there be an esoteric/spiritual component to these health issues? That answer seems to only be answered on an individual basis, reminding us not to group all people with a disease or syndrome into one category. Each patient is unique unto himself or herself.

**Holistic treatment of autism**

**Botanicals**

- Nervine relaxants, to promote relaxation and equanimity: *Matricaria, Lavandula, Passiflora, Verbenae, Valeriana, Scutellaria, Stachys, Nepeta, Melissa*
- Nervine trophorestoratives: *Avena, Ganoderma, Centella, Bacopa, Acorus, Withania, Eleuthrococcus, Rhodiola, Turnera*
- Alteratives, to promote detoxification: *Trifolium, Rumex, Curcuma, Guaiacum, Fucus, Berberis*
- Antispasmodics, to control spasm and seizure: *Acorus, Withania, Valeriana, Lobelia, Cimicifuga*

**Supplements**

- vitamin B complex, 50 mg t.i.d.
- vitamin C, to bowel tolerance
- iron, 20 mg b.i.d.
- calcium/magnesium, 1:1, 800 mg each b.i.d.
- zinc, 50 mg daily
- chromium, 200 mcg t.i.d.
- EPA/DHA, ensures proper neural development, 1000 – 4,000 mg each daily
- dimethylglycine (DMG), 125 mg, 1-8 times daily; if hyperactivity is noted add folic acid, 1600 mcg per 125 mg dimethylglycine

**Diet**
- high protein, low carbohydrate and sugar-free diet
- eliminate common allergens, e.g. dairy, wheat, citrus
- avoid packaged foods
- organic produce and free range meat only
- rule out reactive hypoglycemia

**Children’s Super Hug Flower Essence**
This formula is specially designed for children ages 0 – 16 years old. Children’s Super Hug will allow the child to feel that the world loves them and wants to give them a big hug. This formula of course can be used by any age, but we have found it beneficial to help during the cranky and irritable stages that children go through. It is specially designed to help the user feel more empowered. We most often use this formula in a spray form, as it can be given orally and/or sprayed into a room where children are ‘acting up’ in order to get attention.

**Buttercup** – strengthens feelings of self-worth and ability to experience one’s own inner-light and uniqueness

**Chamomile** – reduces moodiness and irritability; releases emotional tension, especially in the stomach or solar plexus

**Chicory** – freely accepting love, strength and freedom without a need to be demanding, manipulative, or use attention getting behaviour

**Yarrow, Yellow** – having inner protection while remaining open to others

**Self Heal** – a healthy, vital sense of Self; feeling a deep sense of wellness and wholeness

**Sunflower** – a balanced sense of individuality, and a sun-radiant personality

**Aromatherapy**
- citrus, melissa, chamomile, hops, lavender, rosemary, sage

**Topical**
- oil massage, medicated with Acorus, Bacopa, Centella and Withania
Counseling

- Biofeedback
- Biokinesiology (Brain Gym)
- NLP

Additional factors

- heavy metal toxicity (aluminum, cadmium, lead)
- rule out environmental toxins (at home, school or workplace)
- rule out candidiasis and immunodeficiency

Parkinson’s Disease

Parkinson's disease (PD), also known as paralysis agitans and Parkinsonism, is a progressive disorder of the CNS that typically affects older adults around the age of 60, and is prevalent in 1% of the population over the age of 55. The disease results in the widespread destruction of the areas of the basal ganglia that send dopamine secreting nerve fibers to the caudate nucleus and putamen. With the destruction of dopaminergic neurons, the caudate nucleus and putamen become overly agitated, sending excitatory signals to the corticospinal motor system, leading to excitation and muscular rigidity. The high feedback gains without the inhibitory control of dopamine leads to tremors at a fixed rate of 3 to 6 cycles per second. In addition to the loss of dopaminergic neurons in the basal ganglia, other dopamine, monoamine and non-monoamine neurons may be destroyed in other parts of the brain. Diminished levels of norepinephrine, GABA (gamma amino butyric acid), GAD (glutamic acid decarboxylase, which acts on glutamic acid to produce GABA), serotonin, substance P, enkephalin, cholecystokinin and somatostatin have been observed in patients with PD. Reduced levels of homovanillic acid, a metabolite of dopamine, has been observed in the cerebrospinal fluid of PD patients, and is diagnostic marker. (Rubin 2001, 739-740; Berkow 1992, 1496-97)

PD is characterized by rigidity of the musculature, involuntary tremors and an inability to initiate movement (akinesia). Motor performance is impaired and the patient will have great difficulty in performing everyday activities such as shaving, brushing the teeth, eating with utensils,
buttoning shirts and opening door handles. As the disease progresses, handwriting becomes illegible, walking difficult and speaking unintelligible. Rigidity of the facial muscles can give the face a mask-like appearance, characterized by a wide, unblinking stare, an open mouth and uncontrolled drooling. (Rubin 2001, 739-740; Berkow 1992, 1496-97)

The cause of PD is unknown, although many have speculated that it may be the result of environmental toxins. Epidemiological and experimental data suggests the potential involvement of specific agents that act as neurotoxicants (e.g. pesticides) in the pathogenesis of nigrostriatal degeneration (Di Monte et al 2002). In one epidemiological study an increased risk for PD appeared to be associated with occupational exposure to Mn, Fe and Al, especially when the duration of exposure is longer than 30 years (Zayed et al 1990). These studies in support of PD as a post-industrial disease however is in contrast with a disease described in ancient Indian medical texts called kampavata, that very much resembles PD (Manyam 1990). There is an increasing amount of evidence to suggest that the pathogenesis of PD relates to oxidative stress and a reduced ability to deal with it, primarily in the mitochondria of the dopaminergic neurons of the substantia nigra. Often PD is preceded by a severe stress.

Secondary PD can be drug induced by antipsychotic drugs (e.g. haloperidol) that are dopamine antagonists. The chronic use of resperine, an alkaloid from Rauwolfia serpentina, leads to dopamine depletion and can precipitate secondary PD as well. Researchers have found an increased prevalence of PD in patients born during an influenza pandemic. Only 5% of patients with PD have a family history of the condition. (Rubin 2001, 739-740; Berkow 1992, 1496-97)
Hoehn and Yahr grading for motor dysfunction

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No signs of disease</td>
</tr>
<tr>
<td>1</td>
<td>Unilateral disease</td>
</tr>
<tr>
<td>1.5</td>
<td>Unilateral plus axial involvement</td>
</tr>
<tr>
<td>2</td>
<td>Bilateral disease, without impairment of balance</td>
</tr>
<tr>
<td>2.5</td>
<td>Mild bilateral disease, with recovery on pull test</td>
</tr>
<tr>
<td>3</td>
<td>Mild to moderate bilateral disease; some postural instability; physically independent</td>
</tr>
<tr>
<td>4</td>
<td>Severe disability; still able to walk or stand unassisted</td>
</tr>
<tr>
<td>5</td>
<td>Wheelchair bound or bedridden unless aided</td>
</tr>
</tbody>
</table>

Medical management of PD

The focus of medical management of PD is to control the signs and symptoms for as long as possible while minimizing adverse effects. The various medications used in PD usually provide good symptomatic control for about four to six years, after which the condition usually progresses to postural instability and dementia. One focus in the medical management of PD is the development of neuroprotective drugs to prevent a loss of dopaminergic neurons. Of the drugs currently being investigated, selegiline holds out the most promise, demonstrating neuroprotective effects in laboratory animals, but has provided conflicting data in clinical trials. Currently selegiline is employed as monotherapy in early PD to delay the usage of levodopa.

The symptomatic management of PD consists of levodopa (L-dopa), usually given with a peripheral decarboxylase inhibitor (PDI) such as carbidopa to inhibit dopamine formation outside of the brain, which can lead to heart and liver dysfunction. Unfortunately, L-dopa has a finite usage as its effectiveness declines over years of use, and thus it is reserved for the latter stages of the illness. Synthetic L-dopa also causes CNS side effects such as dyskinesia and psychosis; supplementing with L-tryptophan may reduce
these side effects. There is also evidence that the L-
dopa/carbidopa combination may accelerate disease
progression or contribute to the development of motor
fluctuations and dyskinesia. Specifically, levodopa
administration increases dopamine turnover and may
increase free radical production. As PD progresses fewer
and fewer dopaminergic neurons are available to store and
release the L-dopa-derived dopamine, resulting in clinical
fluctuations.

In contrast to L-dopa, the long-acting dopamine agonists
such as bromocriptine and pergolide can provide relatively
smooth and sustained receptor stimulation. Other drugs
include the use of anticholinergics such as benzotropine
mesylate to reduce ACh secretion, and MAOIs to inhibit
dopamine metabolism. Surgical methods involve the
destruction of portions of the basal ganglia, the thalamus
and even the motor cortex to block basal ganglial feedback
to the cortex, and are of varying success. The use of
transplanted dopamine-rich fetal nerve tissue has been used
as well, with some success, but this has raised some
significant ethical issues. More recently, the use of an
electrical oscillating unit that has been surgically implanted
in the brain to inhibit ACh secretion has been used with
success in a few patients, but is invasive and costly.

Other treatments include the use of the amino acid
glutamine and/or GABA to have a relaxing effect upon the
rigidity and tremor in PD. Tryptophan supplementation has
also been shown to be helpful, improving serotonin levels,
and in reducing the side effects of L-dopa therapy. Vitamin
B6 is an important cofactor in the synthesis of dopamine, as
is iron and molecular oxygen, and thus these are important
considerations (e.g. B6 and iron supplementation,
hyperbaric oxygen).

**Holistic management of PD**

The focus of holistic management of PD is concentrated
around keeping the patient off medical treatment for as
long as possible, and using a variety of therapies to limit
free-radical damage and destruction to dopaminergic
neurons. As PD is primarily a disease of aging, general
measures are taken to promote good vascular health as
well. One theory of the free-radical damage seen in PD is
an underlying atherosclerotic condition, causing ‘mini’
strokes and progressive damage to the brain.
**Food Source Levodopa:** Anecdotal reports have demonstrated that patients with PD show improved symptom control when consuming meals of broad beans. In some cases, the response to *Vicia faba* (fava beans) may be even greater than to conventional levodopa medication. Fava beans are a good source of levodopa: a 100-g serving of *V. faba* pods contains about 250 mg of levodopa, equivalent to the levodopa content of one of the standard pharmaceutical formulations. Until more is known about how to use fava beans as an l-dopa source, unsupervised replacement or coadministration of l-dopa with fava beans is not recommended.

**Green tea:** In animal models of Parkinson's disease, both green tea and the oral administration of (−)-epigallocatechin-3-gallate prevented the loss of tyrosine hydroxylase positive cells in the substantia nigra and of tyrosine hydroxylase activity in the striatum. They also prevented neurotoxin-induced elevations in the antioxidant enzymes superoxide dismutase and catalase. These treatments also retained striatal levels of dopamine and its metabolite homovanillic acid and inhibited nitric oxide synthetase in the substantia nigra. There are recent indications that Black Tea can also be useful.

**Ginkgo biloba (Ginkgo):** *Ginkgo biloba* extract (GBE) exerts profound, widespread tissue effects including membrane-stabilizing, antioxidant, and free radical-scavenging effects. GBE also enhances the utilization of oxygen and glucose. GBE is an extremely effective inhibitor of lipid peroxidation of cellular membranes. Although there are no clinical studies in Parkinson's patients, ginkgo is well researched for its beneficial effects in Alzheimer's disease and has been shown useful in animal models of Parkinson's disease. GBE possesses a protective effect on the Parkinson's disease models both in vivo and in vitro. The antioxidation and antiapoptosis may be one of the mechanisms underlying the neuroprotective effect of GBE.

Ginkgo may also help decrease levodopa toxicity. In order to observe the toxic neuronal effect of levodopa and investigate if using levodopa together with GBE would be a feasible method to treat Parkinson's disease, rat models with Parkinson's disease were administered either levodopa
(50 mg/kg every day for 3 days, 5 days, 7 days) or levodopa combined with GBE (100 mg/kg daily). The results showed that in the L-dopa group, the numbers of apoptosis of substantia nigra, which are rings of rotational behavior, were more than those in the group that took levodopa with the ginkgo. These results suggest that levodopa had a neurotoxic effect and that GBE may decrease the toxicity of levodopa. The authors concluded that a combined use of GBE with levodopa may be a practical method to treat Parkinson's patients and may be better than using levodopa alone.

**Phosphatidylserine:** Deficient phospholipid metabolism found in the Parkinson's brain may be due to toxic insult or oxidative stress. Normally the brain can manufacture sufficient levels of phosphatidylserine, but if there is a deficiency of methyl donors like S-adenosylmethionine (SAMe), folic acid, and vitamin B12 or essential fatty acids, the brain may not be able to make sufficient phosphatidylserine. This may make adequate intake of these nutrients important as well.

**Mucuna puriens:** In Ayurvedic medicine, Parkinson's disease, or "kampavata," is described as an imbalance of the vata dosha. *Mucuna puriens* is a legume and is a rich source of the antioxidant vitamin E. Rat studies have demonstrated a clear anti-parkinson effect, and although not well understood, it may be due to components other than levodopa or that it has a levodopa-enhancing effect. One 12-week open clinical trial of 60 patients with Parkinson's disease were treated with a powdered preparation of this legume. Of these patients, 26 patients were taking synthetic levodopa/carbidopa formulations before treatment, and the remaining 34 had not used the medications. The preparation is called HP-200 and is a powder supplied as a 7.5-g sachet that is mixed with water and given orally three to six times a day. Statistically significant reductions in Hoehn and Yahr stage and UPDRS scores were seen from baseline to the end of the 12-week treatment. Adverse effects were mild and mainly gastrointestinal in nature.

**Vitamin D:** There has been some indication that patients with PD have a severe insufficiency of Vitamin D. Thus it is recommended to have at least 4-10,000 IU of this vitamin in early stages to prevent its progression.
Botanicals

- Cerebrovascular stimulants: *Gingko, Green Tea (Camellia), Vinca, Rosmarinus, Vaccinium, Capsicum, Zingiber, Zanthoxylum, Crataegus*
- Nervine trophorestoratives, to protect neurons: *Centella, Bacopa, Acorus, Withania, Rosmarinus, Avena Hypericum, Ganoderma, Rhodiola, Eleuthrococcus, Turnera, Sida cordifolia, Phyllanthus emblica, Panax spp., Polygonum, Angelica sinensis, Cordyceps, Grifolia, Coriolus*
- Antioxidant botanicals: *Gingko, Green Tea (Camellia), Curcuma, Boswellia, Commiphora, Crataegus, Phyllanthus emblica, Bacopa, Tinospora, Shilajitu, Rosmarinus, Centella, Silybum, Bupleurum, Astragalus, Spirulina, Ganoderma*
- Antispasmodics, to relieve dyskinesia: *Acorus, Withania, Valeriana, Lobelia, Cimicifuga*
- *Mucuna* (up to 7% natural source L-dopa), to supplement declining L-dopa levels, *Astragalus* (also containing natural L-dopa)
- Anticholinergics, to relieve dyskinesia when other measures fail: *Datura, Hyocyamus, Atropa*

Supplements

- vitamin A, 20,000 IU daily
- vitamin B complex, 50 mg b.i.d.
- vitamin C, to bowel tolerance
- vitamin D₃, 4 – 10,000 daily
- vitamin E, 800-1200 IU daily
- EPA/DHA, 1000 mg each daily
- iron, 20 mg b.i.d. especially, Krill due to phospholipids
- phosphatidylserine 100mg, tid
- calcium/magnesium, 1:1, 800 mg each b.i.d.
- chromium, 200 mcg t.i.d.
- selenium, 200 mcg b.i.d.
- zinc, 50 mg daily
- CoQ10, 400 - 800 mg t.i.d.
- grapeseed extract, 50 mg t.i.d.
- superoxide dismutase, 100 mg b.i.d.
- bioflavonoids, 3-5 g daily
- melatonin, 1 – 5 mg daily
- 5-HTP (only if taking L-dopa), 75 – 125 mg daily

Homeopathy: No clinical studies are available to support the use of homeopathy for Parkinson's disease, although
anecdotal success stories are known. Some of the Parkinson's remedies and their symptom picture may include the following:

- **Agaricus muscarius**: crawling sensations, vertigo with impulse to fall backwards, symptoms worse in cold weather
- **Antimonium crudum**: Parkinsonism movements associated with gastric symptoms; desire for sour foods that do not sit well in the digestive tract; a thickly white coated tongue; stubbornness; anxiousness; a general disgust of life with worse symptomology caused by heat, wine, or moonlight
- **Argentum nitricum**: tremulousness

**Diet**
- Low protein, low fat, low simple carbohydrates, high vegetable fiber
- Emphasize antioxidant foods, e.g. garlic, onions, cruciferous vegetables; foods rich in anthocyanidins, e.g. blueberries, huckleberries, elderberries, red and black grapes
- Emphasize foods rich in tryptophan, e.g. oats, turkey, hard cheeses
- Consider including fava beans in diet, natural source of L-dopa
- During L-dopa therapy limit protein to 1/7 of total caloric intake, as amino acids can interfere with L-dopa’s transport across the BBB

**Topical**
- *Balashvagandha taila abhyanga, shirodharara, shirovasti*
- Acupuncture

**Other**
- Regular exercise
- Meditation, tai chi, *hatha yoga*
- *Acupuncture* has had great success in some patients
Alzheimer’s disease

Alzheimer's disease (AD) is a degenerative condition of the brain that destroys memory, disrupts personality and accelerates death. Dr. Alois Alzheimer, who had a 51-year-old female patient suffering from a severe form of dementia, first described the disease in 1907. Upon autopsy, he noticed the deposition "of a peculiar substance in the cerebral cortex," which has since been determined to be a 40 amino acid long protein fragment called amyloid beta-protein. Alzheimer's disease affects 5% of the population over 65 years old, more than 5 million people in North America, with an estimated 250,000 new cases every year. (Rubin 2001, 742-43; Berkow 1992, 1403; Roberts 1996)

The tendency to develop AD is probably multifactorial rather than a case of simple genetics or infection; in the case of familial prevalence, this may or may not be genetic, but rather evidence of similar predisposing factors. Above all, AD appears to be a 20th century disease, resulting from the combined effect of dietary and environmental changes with genetic and immunological factors.

The clinical features of AD include dementia, disturbances in comprehension and language, as well as several other behavioral clues. Dementia refers to an impaired ability to pursue one's occupation and social activities. This may include confusion, difficulty in memory recall, difficult speech, altered judgment and impaired abstract thinking. There may also be a feeling of "not being oneself," a loss of interest in favourite pastimes, diminished creativity and a diminished ability to express affection. Language disturbances include the inability to find the correct word or the use of "filler" words in conversation, circumlocution (talking around the subject), an inability to express one's thoughts in a written form or comprehend the written word, and difficulties in performing everyday physical tasks such as dialing the telephone or unlocking the door. Concomitant behaviours may include a deterioration of personal hygiene, inappropriate dress, a loss of social graces, losing and misplacing items, repeated traffic accidents, irritability, stubbornness, suspiciousness, a short attention span, an inability to perform simple instructions.
and an obsessive attention to trivial matters. The diagnosis of **probable Alzheimer's disease (PAD)** occurs when other possible causes of the above clinical features have been ruled out. This includes multi-infarct disease, Parkinson’s, depression, alcoholic dementia, hypothyroidism, adverse reactions to pharmaceuticals, vitamin B₁₂ deficiency, hydrocephalus (increased CSF in the brain's ventricles) and infections such as syphilis. (Rubin 2001, 742-43; Berkow 1992, 1403; Roberts 1996)

There are characteristic changes to the morphology of selective brain structures in AD, specifically of the hippocampus and cerebral cortex, which can be detected by MRI (multiple resonance imaging). The characteristic findings in AD are amyloid plaques, neurofibrillary tangles and the loss of nerve cells and synapses. Amyloid plaques are spherical structures that have a central core of beta-amyloid and varying degrees of inorganic aluminum. The number of these plaques directly corresponds with the severity of the disease. Beta-amyloid is cleaved from a much larger protein called amyloid precursor protein (APP), which is encoded on the 21st human chromosome, which in Down's syndrome is triplicated (trisomy 21). The production of beta-amyloid is not limited to the brain however, but also occurs in the walls of peripheral blood vessels. Thus, the gradual production and accumulation of amyloid plaques may occur well before neuronal degeneration in the brain: thus, AD may begin in early adulthood. The enzyme which cleaves APP has a genetic determinant and current research is focused on finding substances that block the activity of this enzyme. Neurofibrillary tangles are the twisted ends of dead nerve cells, and although not specific to AD, large quantities of them have been correlated with severe dementia. These tangles slow down nerve transmission and impair cellular function. There is a significant loss of brain cells and nerve synapses in AD, within the cerebral cortex and subcortical structures, the major suppliers of acetylcholine, norepinephrine and serotonin that serve the higher cortical centres. (Rubin 2001, 742-43; Berkow 1992, 1403; Roberts 1996)

In cases of AD, a marked depletion of acetylcholine has been noted in an area of the brain called the nucleus basalis of Maynard, in contrast to other neurotransmitters such as dopamine and GABA which remain normal. This has lead
to the theory that AD is a degenerative nerve cell disorder that targets cholinergic neurons. Acetylcholine is a neurotransmitter that binds with M₁ muscarinic receptors to evoke changes in that tissue, and is quickly broken down by acetyl cholinesterase. It has been suggested that there may be a deficiency of acetyl-L-carnitine, which provides acetyl groups for the production of acetylcholine. (Rubin 2001, 742-43; Berkow 1992, 1403; Roberts 1996; Mitchell 1996)

The average adult brain requires 112 grams of glucose to maintain proper brain function. Impaired glucose levels can alter brain cells, initiate the induction of a neurotoxin called glutamate and cause dramatic alterations in the synthesis and metabolism of acetylcholine. The areas of the brain that seem to be highly vulnerable to glucose deprivation are the same regions of the brain that are affected by AD. Reactive hypoglycemia, caused by the over consumption of refined carbohydrates, the usage of exogenous insulin in the absence of dietary precautions, calorie restriction, as well as the chronic usage of caffeine, alcohol and tobacco, are all possible causes of impaired glucose metabolism in the brain. (Roberts 1996; Mitchell 1996)

Reduction in the levels of brain oxygen, necessary for the production of ATP in oxidative phosphorylation, may affect various neurotransmitters, acetylcholine and nerve growth factor and is another possible contributing factor in AD. Factors that limit brain O₂ include smoking, lung and heart disease, anesthesia, air travel, excessive sleep, poor breathing habits, migraine related brain blood vessel spasm and cerebral atherosclerosis. The latter of these factors may be caused by chronic states of hyperinsulinemia. (Roberts 1996; Mitchell 1996)

Certain food additives have been implicated in the development of AD, such as MSG and aspartame (NutraSweet®). Increased concentrations of glutamate and aspartate have been found in the CSF of AD patients. When MSG was tested on young experimental animals it lead to the rapid destruction of brain cells, leading it to be banned in baby foods. Aspartame consists of 50% phenylalanine, 40% aspartic acid and 10% methyl ester. Upon entering the stomach the methyl ester is transformed in free methyl alcohol, which in small amounts can cause blindness, permanent neurological damage and even death.
There is increasing evidence that mutation involving a single amino acid may be the cause of the production of amyloid precursor protein (APP). In a family with three generations of early onset autosomal dominant AD, DNA sequencing revealed the substitution of phenylalanine for valine in the transmembrane domain of APP. Excessive amounts of D-aspartate and other stereoisomers have been found in the neurofibrillar tangles of AD patients, as well as in the amyloid plaques. (Roberts 1996)

A substantial amount of evidence has indicated that aluminum plays a role in Alzheimer's disease. As much as four times the amount of aluminum as normal has been found in the brain of AD patients. Aluminum is known to interfere with essential enzymes needed to metabolize glucose for ATP production. It is also known to cause the destruction of the blood brain barrier and transform L-aspartic acid into the neurotoxin D-aspartic acid. (Roberts 1996)

Demographic studies have found a greater preponderance of AD in women, possibly due to a higher incidence of excessive sugar consumption, fad dieting (causing reactive hypoglycemia), and greater longevity. On average, men have a higher metabolic activity in the temporal and limbic regions of the brain than women, which may confer a preventative benefit. (Roberts 1996)

**Fingerprint Patterns:** Abnormal fingerprint patterns are associated with both AD and Down syndrome. Compared with the normal population, Alzheimer and Down patients show an increased number of ulnar loops on the fingertips, with a concomitant decrease in whorls, radial loops, and arches. Ulnar loops (pointing toward the ulnar bone, away from the thumb) are frequently found on all 10 fingertips. Radial loops (pointing toward the thumb), when they do appear, tend to be shifted away from the index and middle fingers - where they most commonly occur - to the ring and little fingers. In patients with this fingerprint pattern characteristic of AD, it is recommended that an aggressive, preventive approach be instituted immediately.

You can use any ink pad or watercolor paint to make fingerprints on the Fingerprint Chart (see below). Study
the examples of fingerprint patterns in Chart #1, then compare your fingerprints with these.

**Potential Alzheimer's markers to look for:**

a) 8 or more fingertips with ulnar loops, with the base pointing AWAY from the thumb

b) 3 or less fingertips displaying whorls and / or arch patterns

c) Radial loops that point TOWARD the thumb, on the ring and little fingers.

**Holistic treatment of AD**

The treatment of AD disease is similar in scope to other neurodegenerative conditions such as PD. As AD is likely a condition that progresses slowly from repeated insult and damage to the brain, preventative measures based on eating a healthy diet and following a healthy lifestyle are highly recommended for anyone with a family history of AD.

**Huperzine A:** Huperzine A is an alkaloid isolated from the moss *Huperzia serrata*, long used in China to treat primarily fever and inflammation. Although it has been shown to have no antipyretic or antiinflammatory properties in experimental models, Huperzine A has been shown to be a potent inhibitor of acetyl-cholinesterase. In fact, it is significantly more selective and substantially less toxic than the acetylcholine esterase (ACE) inhibitors currently used in conventional medicine (physostigmine, tacrine, and donepezil). Toxicity with synthetic ACE inhibitors has been a major drawback in their clinical use. In contrast, Huperzine A, purified from *H. serrata*, has been used as a prescription drug in China since the early 1990s and has reportedly been used by more than 100,000 people with no serious adverse effect. First studied for use in myasthenia gravis, clinical studies conducted in China have shown considerable benefit in the treatment of dementia. One double-blind clinical study found that Huperzine A at a dose of 200 µg twice daily produced measurable improvements in memory, cognitive function, and behavioral factors in 58% of Alzheimer patients.
**Botanicals**

- Cerebrovascular stimulants: *Rosmarinus, Vaccinium, Ginkgo, Vinca, Crataegus, Capsicum, Zingiber, Zanthoxyllum*

- Nervine trophorestoratives, to protect neurons: *Rhodiola, Centella, Bacopa, Acorus, Withania, Rosmarinus, Avena Hypericum, Ganoderma, Eleuthrococcus, Turnera, Sida cordifolia, Phyllanthus emblica, Panax spp., Polygonum, Angelica sinensis, Cordyceps, Grifolia, Coriolus*

- Antioxidant botanicals: *Curcuma, Boswellia, Commiphora, Crataegus, Phyllanthus emblica, Bacopa, Tinospora, Shilajitu, Ginkgo, Rosmarinus, Centella, Silybum, Buplerum, Astragalus, Spirulina, Ganoderma*

- M1-mimetics, to provide cholinergic stimulus in the nucleus basalis of Maynard: *Pilocarpus*, 10-30 gtt b.i.d.

- Acetyl cholinesterase inhibitors, to inhibit the enzymatic degradation of acetylcholine: *Physostigma venenosa*, 5 gtt b.i.d.

**Supplements**

- vitamin A, 20,000 IU daily
- vitamin B complex, 50 mg b.i.d.
- vitamin C, to bowel tolerance
- vitamin E, 800-1200 IU daily
- EPA/DHA, 3000 mg each daily
- phosphotidylserine, to support biosynthesis of acetylcholine, 100 mg t.i.d.
- L-acetylcarnatine, to support biosynthesis of acetylcholine, 500 mg t.i.d.
- iron, 20 mg b.i.d.
- calcium/magnesium, 1:1, 800 mg each b.i.d.
- chromium, 200 mcg t.i.d.
- selenium, 200 mcg b.i.d.
- zinc, 50 mg daily
- CoQ10, 50 mg t.i.d.
- grapeseed extract, 50 mg t.i.d.
- superoxide dismutase, 100 mg b.i.d.
- bioflavonoids, 3-5 g daily

**Diet**

- Paleolithic diet, low carbohydrate diet to prevent CVD
- emphasize antioxidant foods, e.g. garlic, onions, cruciferous vegetables; foods rich in anthocyanidins,

**Suggested Program:**

- GBX (24%) 80 – 240 tid
- Huperzine A: 200 mcg, bid
- Krill: 1500 mg daily
- Rhodiola 1000 mg bid

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e.g. blueberries, huckleberries, elderberries, red and black grapes

- Eating large amount of fish or fish oils has dramatically reduced speed of dementia.
- Low carbohydrate diet reduces dementia
- Emphasize foods rich in the biochemical building blocks of acetylcholine, e.g. free-range eggs, lecithin
- Avoid transfatty and hydrogenated fats
- Avoid all aluminum containing foods or foods packaged in aluminum (e.g. various antacids, dolomite; aluminum cans, foil and cookware),
- Emphasize artichokes to enhance liver metabolism

Topical
- Brahmi taila abhyanga, Brahmi or Vacha nasya

Epilepsy

Epilepsy is a brain disorder characterized by short, recurrent, periodic attacks of motor, sensory or psychological malfunction. These attacks, called epileptic seizures, are caused by the abnormal, synchronous electrical discharge of millions of neurons in the brain, perhaps resulting from abnormal reverberating circuits. The incidence of seizure disorders is about 2%, with about 0.5% of those suffering from epilepsy on a regular basis. At least four types of epileptic seizures have been identified: grand mal, temporal lobe, focal and petit mal. Grand mal (tonic-clonic seizure) is characterized by generalized involuntary muscular contraction and cessation of respiration followed by tonic and clonic spasms of the muscles. EEG recordings indicate a high voltage synchronous discharge that occurs over the entire cortex on both sides of the brain, originating in the basal regions of the brain that drive the cortex. The reticular activating system may be temporarily depressed so that the person loses consciousness. The teeth may be clenched, the tongue may be bitten and control of bowel and bladder may be lost. After the seizure consciousness shortly returns and breathing begins with noisy respirations. The person may feel sleepy, fall asleep or experience confusion and has no memory of the episode upon awakening. The prodromal symptoms of grand mal may
include auras, as well as unusual odours and sounds. (Berkow 1992, 1439)

Temporal lobe (psychomotor seizure) is characterized by psychic symptoms (visual or auditory hallucination, déjà vu), loss of judgment and autonomic behavior, and abnormal activity. Typically, there are no apparent convulsions, but there may be a loss of consciousness or amnesia for the episode. During the seizure the patient may appear drowsy, intoxicated, and violent or commit asocial behaviors, but other activities such as driving a car or eating remain unaffected. Psychic symptoms may be accompanied by chest pain, transient respiratory arrest, tachycardia, abnormal taste and/or smell sensations, and GI disturbances. EEG readings during a psychomotor attack show a low frequency rectangular wave with a frequency of between 2 and 4 Hz, superimposed with 14 Hz waves. (Berkow 1992, 1439-40)

Focal epilepsy can involve almost any region of the brain, resulting from localized lesions such as a tumor or damaged neural tissue, or from congenitally deranged circuitry. Such lesions can promote the rapid discharge of local neurons, and when this electrical activity passes a threshold of about 1000 Hz, it begins to spread to adjacent cortical regions, as slow as a few millimeters per second to as fast as a few centimeters per second. When such a wave progresses over the motor cortex, it often causes a progressive series of muscular contractions throughout the
body, while the person remains conscious. It may begin in the fingers and toes and progress upwards, or it may begin in the mouth region and progress downward to the legs. This particular manifestation of progressive muscular contraction is called Jacksonian epilepsy. (Berkow 1992, 1437-40)

Petit mal (absence seizure) is characterized by a sudden momentary loss of consciousness (10-15 seconds), occasionally accompanied by myoclonus of the neck or upper extremities, slight symmetric twitching of the face or a loss of muscle tone. EEG recordings in petit mal show a characteristic spike and dome pattern. (Berkow 1992, 1438-40)

Brain samples from epileptic foci of experimental animals have been shown to have abnormally low concentrations of GABA and abnormally high levels of glutamate. Physiologically, the role of glutamate is excitatory, promoting neuronal firing in the cerebellum and depolarization in the cerebral cortex. GABA, on the other hand, inhibits cerebral firing. Administration of glutamate in experimental animals has been shown to induce epileptic seizures. Thus, excessive amounts of glutamate may be a mechanism of seizure. Seizures are believed to stop because of neuronal fatigue, and the active initiation of inhibitory neurons. (Cooper 1996)

Epileptic seizures are known to be initiated by strong emotional stimuli, alkalosis caused by hyperventilation, drugs (e.g. metrazol, insulin), fever, loud noises and flashing lights. Other possible causes of epilepsy include severe head injuries (even in gestation), atherosclerosis, brain tumors or abscesses, intracranial infection, drug abuse, cerebral ischemia, exposure to rapid fire images common to some kinds of television programming and video games, food allergies, and Leaky-Gut syndrome.

Many epileptics report an unusual odor prior to seizure, which may or may not be present in the environment. Neural pathways from the basal ganglia and many other brain regions extend into the olfactory bulb, and thus odor may be a trigger or a symptom for seizure. Some clinicians have speculated that it may be possible to prevent seizures by anticonvulsant essential oils such as Aniseed, Celery seed and Lavender. Some kinds of essential oils are
reported to initiate seizures, such as *Artemisia* spp., and should be avoided.

**Medical treatment of epilepsy**
Medical treatment rests on the usage of a variety of anticonvulsant drugs that act in a variety of ways. Barbiturates such as phenobarbital and primidone depress excitatory postsynaptic discharges, thereby raising the convulsive threshold for electrochemical stimulation. Benzodiazepines such as clonazepam enhance the activity of GABA. Hydantoins such as phenytoin inhibit seizure activity by blocking the propagation of electrical impulses, through decreasing sodium transport or by blocking calcium channels. Other anticonvulsants such as valproic acid act as GABA agonists by inhibiting GABA metabolism and presynaptic uptake, and enhancing post synaptic uptake. Surgical options in the treatment of epilepsy may consist of the removal of the corpus callosum, the hippocampi and other brain regions. Electroconvulsive therapy is another technique used to treat epilepsy.

**Holistic treatment of epilepsy**
When attempting to wean a patient off of anticonvulsants it is imperative that it is performed slowly, and only in mild to moderate cases. Treatment can be given with the anticonvulsant, as long as it doesn’t have a similar activity, such as using a GABA agonists like valproic acid with *Valeriana* spp. The use of botanical GABA agonists can be gradually increased as the medication is being weaned. This initial phase of botanical support that doesn’t directly interfere with the medication can occur over a one to three month period. Weaning is performed over a six-month period, gradually reducing the dosage of the anticonvulsant, but depending upon the symptoms of the patient, the entire weaning process may actually occur a little faster. Regardless, at all time during the weaning process and for the length of the holistic treatment, the patient should keep his or her anticonvulsant medication on hand, and be directed to pay strict attention to prodromal symptoms such as headaches, visual disturbances, olfactory disturbances, odd sensations, or dizziness. When the weaning process is completed, the patient should be symptom free for at least two years before discontinuing the supporting therapy. Even if weaning is not attempted however, many of the recommendations below are useful.
**Chinese Herbal Medicine:** a Chinese herbal medicine combination, Saiko-Keishi-To (SK). SK demonstrated dramatic therapeutic effects on some difficult cases of epilepsy that had long been unsuccessfully treated with standard allopathic anticonvulsive drugs. SK is a combination of nine botanical drugs:

- Bupleuri radix (5 g)
- Scutellaria radix (3 g)
- Pinelliae tuber (5 g)
- Paeoniae radix (6 g)
- Cinnamon cortex (2 g)
- Zizyphi fructus (4 g)
- Ginseng radix (30 g)
- Glycyrrhizae radix (1.5 g)
- Zingiber rhizoma (2 g)

In one experiment, seizure like activity was induced in snail neurons by the drug pentylenetetrazol (PTZ). SK was found to do the following:

- Inhibit the intracellular shift of Ca\(^{2+}\) toward the cell membrane
- Inhibit the binding of Ca\(^{2+}\) to Ca\(^{2+}\)-receptive membrane proteins and the Ca\(^{2+}\)-calmodulin complex
- Inhibit the conformational changes of the Ca\(^{2+}\)-receptive membrane proteins
- Inhibit the pathologic transmembrane current of Na\(^+\), K\(^+\), and Ca\(^{2+}\)

The researchers presumed that SK's experimental effects are similar to its mode of action in humans. Interestingly, when an attempt was made to isolate purified chemicals from the component herbs, the crude drug's efficacy was lost. This suggests a synergistic effect between the component botanical agents. More recent clinical research has shown some benefit in epileptic patients. One study reported that 8 of 28 epileptics experienced a 25% decrease in number of seizures after 8 weeks of treatment.

**Botanicals**

- Anticonvulsants: Valeriana, Ganoderma, Withania, Cimicifuga, Viscum, Acorus, Lobelia, Piper methysticum, Ruta, Tilia
- GABA agonists: Valeriana, Piper methysticum, Tilia, Withania, Passiflora

**Suggested Program**

<table>
<thead>
<tr>
<th>SK 300 ml before bed</th>
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<tbody>
<tr>
<td>B6 – 50 mg tid</td>
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<tr>
<td>Melatonin – depending on age</td>
</tr>
<tr>
<td>Zinc 25 mg</td>
</tr>
<tr>
<td>Krill 500 mg, bid</td>
</tr>
<tr>
<td>Reishi Premium (2 capsule, bid)</td>
</tr>
<tr>
<td>Multi vitamin mineral 1 bid</td>
</tr>
<tr>
<td>Hypo glycemic or Ketogenic Diet</td>
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</tbody>
</table>
• Nervine trophorestoratives, to protect neurons:
  Centella, Bacopa, Acorus, Withania, Rosmarinus, Avena Hypericum, Ganoderma, Eleuthrococcus, Turnera, Sida cordifolia, Phyllanthus emblica, Panax spp., Polygonum, Angelica sinensis, Cordyceps, Grifolia, Coriolus
• Antioxidant botanicals: Curcuma, Boswellia, Commiphora, Crataegus, Phyllanthus emblica, Bacopa, Tinospora, Shilajitu, Ginkgo, Rosmarinus, Centella, Silybum, Buplerum, Astragalus, Spirulina, Ganoderma
• Hepatics and aperients, to ensure the proper elimination of wastes: Berberis, Rumex, Taraxacum, Rhamnus purshiana, Rheum, Operculina, Embelia

Supplements
• vitamin A, 20,000 IU daily
• vitamin B complex, 50 mg b.i.d.
• vitamin B 6 50 mg tid
• Folic acid 0.4 – 4 mg daily
• vitamin C, to bowel tolerance
• vitamin E, 800-1200 IU daily
• EPA/DHA, 1000 mg each daily
• calcium/magnesium, 1:1, 800 mg each b.i.d.
• chromium, 200 mcg t.i.d.
• selenium, 200 mcg b.i.d.
• zinc, 50 mg daily
• melatonin 3 mg in children up to 20 mg in adults

Diet
• implement elimination-challenge diet
• consider a Hypoglycemic diet
• consider a Ketogenic Diet (see appendix)
• emphasize antioxidant foods, e.g. garlic, onions, cruciferous vegetables; foods rich in anthocyanidins, e.g. blueberries, huckleberries, elderberries, red and black grapes
• avoid stimulating foods and beverages such as coffee, tea, aspartame and chocolate
• avoid refined foods such as sugar

Aromatherapy
• lavender, celery seed, aniseed

Topical
• Balashvagandha taila in shirodhara
• cranial sacral therapy

**Other**
- ensure proper bowel elimination
- de-stress environment, biofeedback, massage
- lower exposure to high stimulatory media (e.g. video games) and emotionally disturbing stimuli (e.g. horror movies)

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**References and Bibliography**


Appendix\(^3\)

Gastrointestinal rehabilitation

Gastrointestinal rehabilitation is a concept familiar to practitioners of natural medicine. This involves therapeutic steps to reduce intestinal permeability, improve nutrient absorption, and increase immune response to gut pathogens while diminishing hypersensitivities. A useful approach follows the mnemonic "ANT PIE" (Abstain, Nourish, Toxins/detoxification, Probiotics, Identify, Eliminate).

- **Abstain** from junk foods, deep-fried foods, sugary drinks, and other foods that harm or irritate the gut, as well as unnecessary drugs or excessive alcohol.
- **Nourish** the digestive tract with nutrients that support gut healing. Functional foods that combine low allergy potential protein and a number of nutrients helpful for gastrointestinal healing are readily available.
- **Toxins/detoxification** refers to the avoidance of pesticides by eating organic foods and consuming nutrients that improve the efficiency of detoxification processes such as L-glutamine, N-acetylcysteine, dietary fiber, and cruciferous vegetables. Regular exercise and stress reduction are also important parts of this step.
- **Probiotics** are therapeutic bacteria (and some yeast) that may improve the gastrointestinal micro-ecology, improve immune response toward gut pathogens, reduce immune hypersensitivities, and stimulate gut repair.
- **Identify** allergenic or intolerant foods and gut pathogens. Food allergies (Type I or immediate hypersensitivity food reactions) can be identified by skin prick testing, radioallergosorbent testing, or enzyme-linked immunosorbent assay testing for IgE antifood antibodies. Patients with evidence of Type I hypersensitivity food allergies (e.g., postprandial swelling of the lips, urticaria, wheezing) should be referred to an allergist for definitive testing, as serious anaphylaxis could occur without proper avoidance. Delayed hypersensitivity reactions (usually Type III mediated) are best identified with a properly conducted elimination test diet followed by carefully observed reintroductions of individual food items eliminated in the test diet. IgG antifood antibodies may also be tested but only before any dietary manipulation has occurred.
- **Intestinal parasites, yeast, and pathogenic bacteria** may also be identified by stool testing, *Candida* and *H. pylori* serology, breath testing, and urinary organic acids.
- **Eliminate** foods found to be allergic or intolerant with elimination test diet or laboratory testing. Gut pathogens, parasites, and *Candida* overgrowth may be treated.

\(^3\) From Pizzorno Murray et al.
Ketogenic Diet

Originally introduced by Wilder in 1921, the ketogenic diet has a long history of use for the reduction of seizure activity. The diet consists of large amounts of fat and minimal amounts of protein and carbohydrates. The low carbohydrate intake inhibits fat metabolism, resulting in the production of excessive levels of ketone bodies (acetone, aceto-acetic acid, and beta-hydroxybutyric acid), the intermediary oxidation products. Presumably the beneficial effects of such a diet are due to its induction of metabolic acidosis, which corrects an underlying tendency of epileptics towards the spontaneous development of alkalosis. This acidification is thought to normalize nerve conductivity, irritability, and membrane permeability.

The two main types of ketogenic diets are (1) the classic ketogenic diet and (2) the medium-chain triglyceride ketogenic diet. The classic ketogenic diet produces ketosis by limiting intake of carbohydrates and protein to less than 10% of energy combined. The medium-chain triglyceride ketogenic diet uses medium-chain triglyceride fat to produce ketosis. This allows for a larger intake of carbohydrates and protein.

Research continues to document the efficacy of these types of dietary therapy. For example, one study of 27 children from 1 to 16 years old using the classic diet found that 40% experienced a reduction of seizures of more than 50%, with 25% becoming seizure free. However, 35% discontinued the diet due to difficulty in following the rigorous guidelines. A review from 1996 concluded that the ketogenic diet had efficacy in one third to one half of epilepsy cases in children and was partially effective in another one third of cases. A review article from 1997 stated that the ketogenic diet's success rate "greatly exceeds that of the medications" and that its side effects were fewer and the therapy cheaper.

One prospective, nonrandomized study measured nutrient intakes, growth, and biochemical indexes of 30 children from 1 to 16 years old who had intractable epilepsy before and after a 4-month protocol using a ketogenic diet. Fourteen children on the classic diet and 11 children on the medium-chain triglyceride diet completed the study for an 83% completion. The results indicated that linear growth was maintained in patients from baseline to 4 months on both therapies. However, body weight decreased for children on both diets, which could be a result of inadequate energy intake. Protein intake met recommended intakes for both diets. In the medium-chain triglyceride group, there was a 0.7% decrease in the ratio of total cholesterol to high-density lipoprotein ratios at 4 months. All biochemical indexes including albumin levels remained within normal limits. However, this was only a 4-month study. Longer-term evaluations may show eventual unwanted changes in these parameters. The authors concluded that the medium-chain triglyceride diet therapy may be more nutritionally adequate and thus confer an advantage over the classic ketogenic diet.
Although the previously mentioned study demonstrated a relatively short period of 4 months, the ketogenic diet is not without side effects. The long-term risks of a high-fat diet are well known, and a ketogenic diet may prove unhealthy for a growing child. One retrospective investigation found that the linear growth of some children might be retarded. When treating children on a ketogenic diet, clinicians should recommend adequate intake of energy and protein, a higher proportion of unsaturated to saturated dietary fats, and also consider vitamin and mineral supplements. Also, children should not be allowed to eat large meals, as these may predispose them to seizures. Small, frequent meals may be appropriate and may decrease hypoglycemic episodes.

The Atkins diet theoretically may be useful to treat epilepsy, for like the ketogenic diet, it too produces a ketotic state but creates this effect with less restriction on protein intake. In one pilot study six patients ranging from 7 to 52 years old were prescribed the Atkins diet for intractable focal and multifocal epilepsy. Five of the patients maintained moderate to large ketosis for periods of 6 weeks to 24 months. Three patients experienced seizure reduction and, as a result, were able to reduce antiepileptic medications.