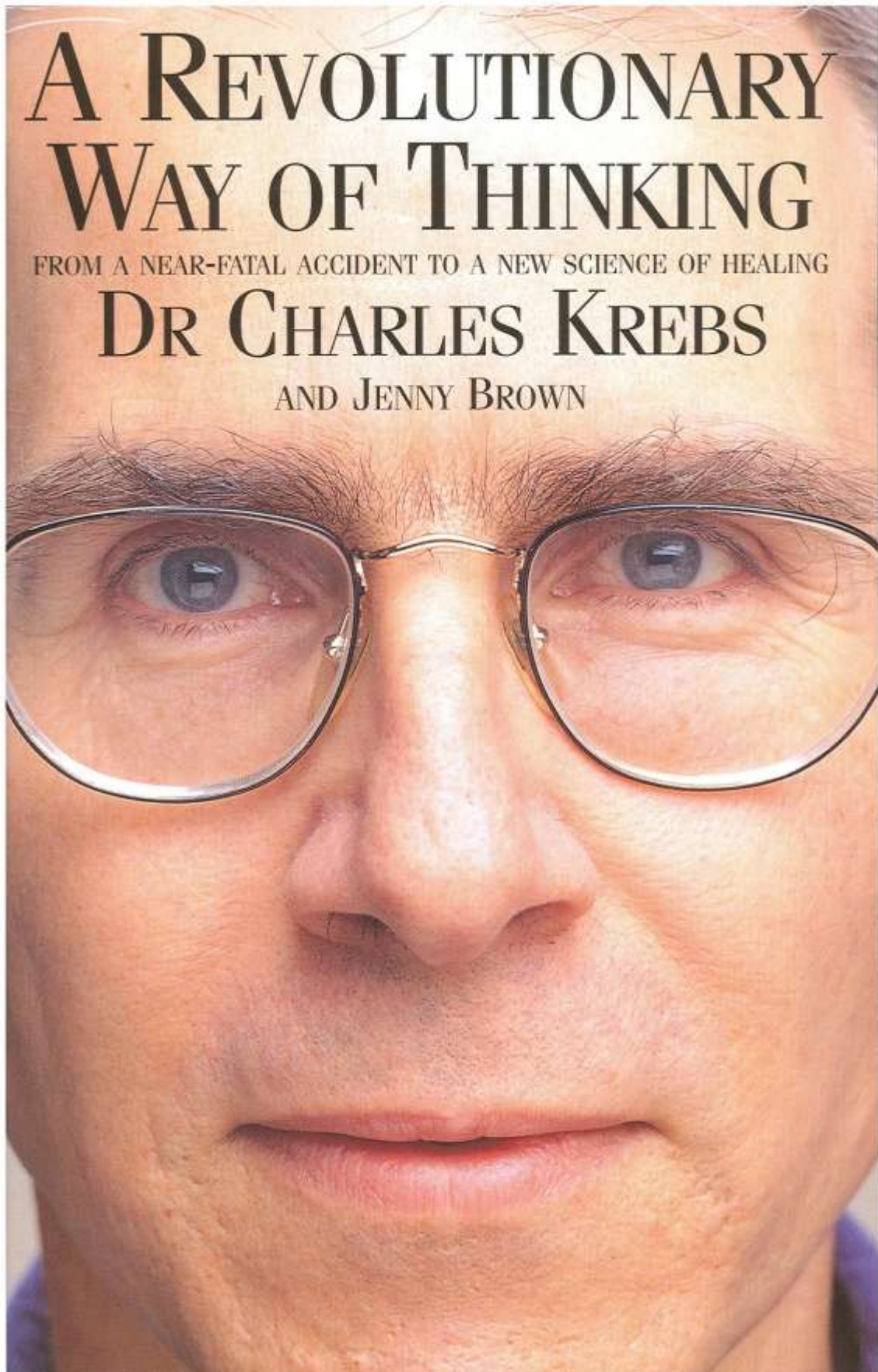


A REVOLUTIONARY WAY OF THINKING

FROM A NEAR-FATAL ACCIDENT TO A NEW SCIENCE OF HEALING

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LEAP[®]: The Neurological Basis of Assessing and Correcting Learning and Memory Problems with Acupressure and Kinesiology.

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ABSTRACT:

The Learning Enhancement Advanced Program, or LEAP[®], has been developed since 1985 in conjunction with clinical psychologists, speech pathologists, neurologists and other health professionals, as a very effective program for the correction of most learning difficulties. LEAP[®] is based on a new model of learning integrating recent concepts in neurophysiology of the brain and uses highly specific acupressure formatting to address stress within specific brain structures. The application of specific non-invasive acupressure and other energetic techniques can then resolve these stresses resulting in a return to normal function.

In the LEAP[®] model of learning Gestalt and Logic functions are not simply localised in the right or left cerebral hemisphere as in the popular Right Brain/Left Brain model of learning. Rather, each type of conscious brain function or process appears to have a cerebral "lead" function that is either predominantly Gestalt or Logic in nature. These cortical "lead" functions provide a "point of entry" into a widely distributed system comprising many subconscious cortical submodules in both hemispheres and many subconscious subcortical modules throughout the limbic system and brainstem.

While the Gestalt and Logic "lead" functions are conscious, these functions are dependent upon many levels of subconscious processing at many levels within the nervous system. While this processing through multiplexing and parallel processing at many different levels is highly efficient, it means that brain processing is "time bound". Since many components of any mental function are performed in many different parts of the brain, and often at different speeds, coherent output in the form of "thinking" requires integration and synchronisation of all of these separate processes.

Loss of integrated brain function, termed loss of Brain Integration in LEAP[®], thus results in the loss of a specific mental capacity, the ability to perform a specific type of mental task. When these specific mental capacities are required for academic performance, their loss can result in Specific Learning Disabilities.

Specific Learning Disabilities (SLDs) arise in this model by either lack of access to specific subconscious processing modules, either cortical or subcortical, or the de-synchronisation of neural flows in the integrative pathways between processing modules. Thus to resolve these SLDs, you need only "open up" access to the "blocked" processing modules or re-synchronise the timing of information flow between them to re-instate integrated brain function.

The LEAP[®], program provides an integrated acupressure protocol using kinesiology as a tool to identify "stress" within specific brain nuclei and areas that have "blocked" integrated function. The application of the LEAP[®] acupressure protocol using acupressure and other energetic based techniques to re-synchronise brain function resolves learning and memory problems in a high percent of cases.

HISTORY OF SPECIFIC LEARNING DIFFICULTIES.

Difficulties with learning academic tasks such as reading, spelling and mathematics have been recognised for over a century, with Kussmaul in 1877 ascribed as the first person to specifically describe an inability to read, that persisted in the presence of intact sight and speech, as word blindness.¹ The word *dyslexia* was coined by Berlin in 1887.² Within a decade a Glasgow eye surgeon James Henschelwood (1895) and a Seaford General Practitioner Pringle Morgan (1896) observed students who were incapable of learning to read and hypothesised that this was based on a failure of development of the relevant brain areas which were believed to be absent or abnormal. This model was based on the assumption that developmental dyslexia (congenital dyslexia) was similar in form to acquired dyslexia, which is dyslexia due to brain damage after a person has already learned to read. Deficits in other types of learning, such as mathematics, would also result from some other underlying brain damage or abnormality.³

Work in the early part of the twentieth century, particularly by Samuel T. Orton in the 1920s and 1930s suggested that learning difficulties such as dyslexia were not based on anatomical absence or abnormality, but rather it was delay in the development of various areas that caused these dysfunctions. This belief was largely ignored until the 1960s when it was revived by a growing interest in neuropsychology. However, more recent developments in neuropsychology and neurophysiology support the hypothesis that dysfunctions within the brain, both anatomical and developmental, may be causal in many learning problems.⁴

It was not until 1963, in an address given by Samuel Kirk, who argued for better descriptions of children's school problems that the term "learning disabilities" originated. Since that time there's been a proliferation of labels that attempt to dissociate the learning disabled from the retarded and brain damaged.

Definitions

In the context of this paper, Specific Learning Disorders or Disabilities relates to problems with physical co-ordination and acquiring the academic skills of reading, writing, spelling and mathematics including both dyslexia and Attention Deficit Disorder (ADD) with or without hyperactivity. ADD with hyperactivity is now commonly called Attention Deficit Hyperactivity Disorder (ADHD) or hyperkinetic disorder in Europe. Historically, dyslexia has been widely defined in terms of deficits in the areas of reading, spelling and language. However, more recent conceptualisations have included a definition that also encompasses a wide range of problems, including clumsiness and difficulty with rote learning.⁵ Fawcett and Nicolson have also challenged the prevailing hypothesis that dyslexia is merely a language based problem, suggesting that it might be a more generalised deficit in the acquisition of skills.⁶

The term dyslexia is not defined in the DSM IV (1994) although it is still commonly used in literature discussing various learning difficulties. The term Learning Disorders (DSM IV) currently encompasses various types of learning difficulties including dyslexia and Attention Deficit Disorder (ADD). Learning Disorders are defined in the DSM IV as being essentially a persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development. The performance of these individuals on standardised tests for reading, mathematics, or written expression is substantially below, more than 2 standard deviations (SDs), same age peers even though their IQ scores are average or above average.⁷

Incidence

Frequently, children diagnosed as learning disabled are also inattentive and deficient in linguistic skills, most often in reading.⁸ Rutter and Yule examined a large population of children from a number of different studies and found 3.5% of Isle of Wight 10-year-olds, 4.5% of 14-year-olds and over 6% of London 10-year-olds showed reading difficulties.⁹ Gaddes looked at the proportion of children with learning disorders in various studies in both North America and Europe and found that the need for special training for learning disorders ranged between 10-15% of the school age population.¹⁰ However, estimates of the prevalence of learning disorders for broad age ranges is problematic because a learning disability is an emergent problem that is often not evident until later years in schooling. Using the criteria of defining learning disorders as being two years behind on standardised tests, less than 1% of 6-year-olds are disabled, 2% of 7-year-olds and so on until at age 19, 25% would be classified as learning disabled. So these children fall progressively behind as they mature and the complexity of work increases.¹¹

In an address given by the Australian Federal Schools Minister, Dr David Kemp, in October 1996, Kemp stated that a study of 28,000 students in four surveys in Australia found 30% of year 9 students lacked basic literacy skills. This high incidence of learning disorders in school children indicates a need for effective treatment. Studies in other countries, both English, French and German support these figures, so specific learning difficulties, which cover all types of learning disabilities from dyslexia, reading problems, ADD to ADHD, probably represent greater than 15% of school-aged children, and may be as high as one third of all school-aged children.

Causes

Currently hypotheses concerning learning disorders suggest that they are primarily the result of one or more of five major factors; 1) structural damage, 2) brain dysfunction, 3) abnormal cerebral lateralisation, 4) maturational lag and 5) environment deprivation. While none of these theories is unequivocally supported by current data, all of these factors may contribute in varying degrees to learning disabilities.¹²

Brain damage and overt brain dysfunction would appear to account for a relatively small percentage of children with learning disorders. The great majority of other children with learning disorders do not typically show many of the neurological symptoms associated with brain damage in adults. For instance, EEG and CT studies have not shown structural damage and abnormal EEGs correlated with known brain damage are not consistently observed in children with learning disorders.¹³ Rather than direct brain damage, there is evidence that abnormal physiological or biochemical processes may be responsible for malfunction in some part of the cerebral cortex. Electrophysiological recording studies have associated specific high frequency EEG and AEP (averaged evoked potentials) abnormalities with various types of learning disorders.¹⁴ Recent studies with SSVEP (Steady state visual evoked potential) have shown that children diagnosed with Attention Deficit Disorder demonstrate similar abnormal SSVEP patterns when compared to normal subjects while performing the same cognitive task.¹⁵ The brain dysfunction hypothesis suggests that the dysfunction may be a consequence of defective arousal mechanisms resulting in some form of inadequate cerebral activation.¹⁶

This is supported by studies of children with learning disorders that show they have difficulty on continuous performance tests requiring attention and low distractibility; had slower reaction times to stimuli, and increased errors due to impulsivity on tests of visual searching.¹⁷ Douglas proposed that the deficits on these tasks resulted from inadequate cerebral activation. Learning disorders of some types at least, do improve with drugs like amphetamines that cause cerebral

activation via increasing subcortical arousal. In fact this is the basis of treating hyperactive children with Ritalin.¹⁸

An alternative model of learning disorders is based on recent neurophysiological findings that suggest it is the timing and synchronisation of neural activity in separate brain areas that creates high order cognitive functions. Any loss or malfunction of the timing mechanism may cause disintegration of neural activity and hence dysfunction in cognitive tasks.¹⁹ Clearly, brain dysfunction due to inadequate cerebral activation may indeed lead to disruption of the timing and synchronisation of neural flows, and thus these two hypotheses may just be different aspects of the same process.

This model supports the approach in the Learning Enhancement Advanced Program (LEAP®) that Krebs and McCrossin developed in the late 1980s early 1990s.²⁰ In the LEAP® Model, Specific Learning Disorders are based on the disruption or loss of timing and synchronisation between the neural activity in the diverse brain regions, both cortical and subcortical, that must be synchronised in order for successful integration to produce normal cognitive activity. *Learning disorders would arise in this model from a lack of integration of functions that occur simultaneously in separate brain regions.*

If the brain does integrate separate processes into meaningful combinations we call ‘thought’ or cognitive ability, then the main risk is mis-timing or loss of synchronisation between these processes. To quote Damasio “any malfunction of the timing mechanism would be likely to create spurious integration or *disintegration*”.²¹ For synchronous firing of neurons in many separate brain areas to create cognitive functions would require maintenance of focused activity at these different sites long enough for meaningful integration of disparate information and decisions to be made.

THE LEAP® MODEL OF LEARNING:

From a review of the major brain structures and the workings of learning and memory in the neurological literature, it is clear that both memory and learning do not involve a single, global hierarchical system in the brain. But rather, learning involves interplay between many inter-linked sub-systems or modules.²² Also, the timing and synchronisation of information flow between these sub-systems and modules appears to be critical to the success of learning and coherent cognitive function.

However, the sub-systems or modules underlying both learning and memory are both conscious and subconscious with most of the early leveling processing being totally subconscious, and only the highest levels of neural processing reaching consciousness. Yet, it is indeed these conscious modules that initiate and direct the processing to be done by the subconscious modules, as both learning and memory require “conscious” effort to occur. This means that the memory and learning processes can be disrupted at both the conscious and subconscious levels, depending upon which neural substrates or integrative pathways are disrupted.

Sensory processing of all types is initially a relatively linear chain of neural impulses originating from a generator potential of the sensory receptor, and following a chain of neurons into the Central Nervous System (CNS) and brain. However, this initially linear stream of nerve impulses, the data of the CNS, rapidly becomes divergent and multiplexed at higher levels of cortical processing. Conscious perception only arises at the highest levels of these multiplexed data flows as they are re-integrated back into unified conscious perception by the cortical columns directing all conscious brain activity.

Thinking and other cognitive abilities rely upon all of the proceeding levels of subconscious sensory processing, which are predominately bilateral initially, but which become progressively asymmetrical and lateralised with increasing levels of conscious awareness. Sensory information is processed initially as neural flows of increasing complexity that generate preverbal images and symbols, but becomes increasingly defined by language in higher level cognitive processes. And language by its very nature is based upon abstract representations of external reality (called words), that follow linear rules (grammar), and word order linked to meaning (syntax). Hence it is predominately sequential and linear in form, which permits analytical evaluation of the thoughts generated following rational rules of Logic. From the perspective of Logic, the world is interpreted as parts that can be constructed into a whole via deductive reasoning.

Sensory and other mental data not suitable for language-based rational processing is processed via visuospatial image and symbols that permit global, holistic comprehension of the whole and is inherently non-rational.²³ This global, simultaneous, non-rational visuospatial processing has been termed Gestalt (German for pattern or form), with the meaning of the whole extracted via inductive reasoning. From the Gestalt perspective, the world is seen as a “whole” with intuitive understanding of the properties of the whole. There is no rational analysis of “Why?”, it just “Is”.

In the LEAP[®] Model of Learning, it is recognized that most of the lower level linear sensory processing occurs below conscious perception, and is either subcortical, being processed in the brainstem or other brain nuclei like the hypothalamus, thalamus, basal ganglia, etc., or is palaeocortical and limbic. Even the basal levels of cortical processing are largely bilateral and subconscious, and thus occur outside of conscious perception. All higher level cortical processing, which may become conscious, is thus reliant upon maintenance of integrated function and neural flows at these subconscious levels.

However, the more overtly cognitive components of learning rapidly become lateralised with processing dominated by activation of cortical columns, the functional units of the neocortex, in one hemisphere of the brain or the other. In right-handed people, Logic processing typically activates cortical columns in the left hemisphere, that then process the data in a linear analytical way, while activation of cortical columns in the right hemisphere process data in a Gestalt, visuospatial way. Thus, at the highest levels of conscious neural processing underlying cognition and thought, whether that “thought” be verbally based language of Logic, or global intuitively based “knowing” of Gestalt, the neural processing is highly lateralised and is predominately processed in the right or left hemisphere.

The neural substrates for all “conscious” functions therefore are cortical columns of the neocortex (Fig. 1). Conscious activation of a cortical column acts to initiate a cascade of neural flows that rapidly spread to other cortical areas both conscious and subconscious in both hemispheres, and also into many subcortical structures as well. These consciously activated cortical columns initiate either Gestalt or Logic functions depending in which hemisphere they are located. In LEAP[®] we term cortical columns activating Logic functions, Logic “lead” functions, and those activating Gestalt functions, Gestalt “lead” functions. These “lead” functions provide points of entry into an inter-linked set of cortical and subcortical modules performing our mental functions.

Indeed, it was a misunderstanding about the nature of these “lead” functions from which the popular “Right Brain – Left Brain” model of learning and brain function arose. Because damage to specific cortical columns caused loss of specific conscious functions, e.g. the ability to form an image, or figure out certain types of problems or solve certain types of puzzles, it was

assumed that the damaged area actually did that specific function. In reality, all that cortical column did was provide a point of entry into these inter-linked sets of cortical and subcortical modules that actually performed the function lost because of the damage to the cortical “lead” function.

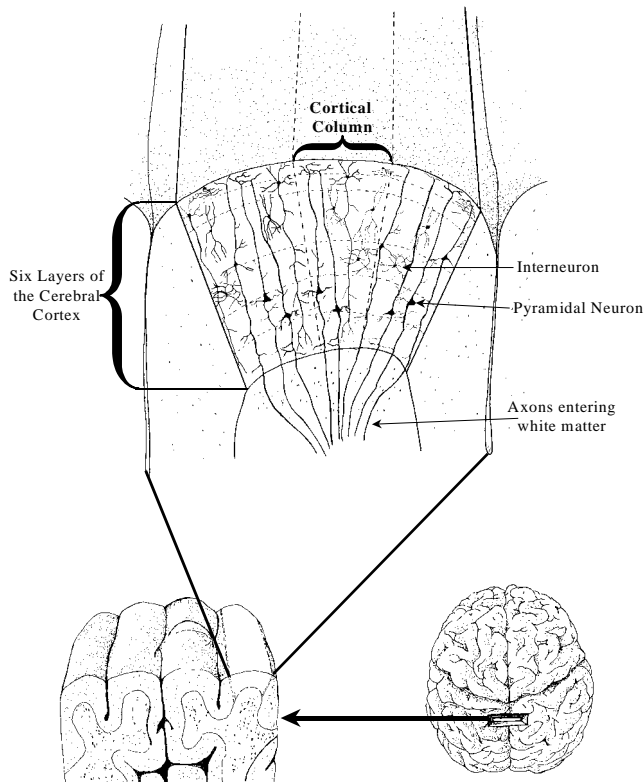


Figure 1. Cortical Columns. Vertical slabs of cortex consisting of all six distinct cell layers, called cortical columns, are the functional units of the cerebral cortex. Some of the cells like the large pyramidal cells have dendrites that extend through almost all layers and axons that exit the gray matter to become part of the white matter tracts carrying information to other parts of the brain and body. There are also innumerable interneurons connecting the cells within each cell layer and between the layers.

An analogy would be damage to the “K” key on your keyboard. Your consciousness is still intact and able to initiate “K” questions, and your computer system is still able to process and answer “K” questions, but the interface to initiate “K” processing in the computer has been damaged. Like wise, if a Gestalt “lead” function is damaged, the process initiated by this “lead” function no longer activates the inter-linked cortical and subcortical functions that are required for this process to occur. Thus, while damage to the area initiating a function, “blocks” the rest of the processing needed to perform the function, the initiating area never actually ever “did” the function in the first place.

Synopsis of the LEAP® Model of Learning:

In summary, the LEAP® Model of Learning is based on the following suppositions about the nature and location of neural processing underlying learning and memory:

- Sensory processing initiated by sensory receptors generates initially linear neural flows that rapidly diverge at each successive processing centre (spinal and cranial nerve ganglia, brainstem nuclei, subcortical nuclei, limbic cortices, and finally neocortical columns) into a number of different complex data streams. All processing below the neocortex is subconscious.
- Each processing centre, at each successive level within the spinal cord, brainstem, diencephalon, basal forebrain and cortex elaborates the sensory data, defining some aspect more than another, or adds additional types of information needed to define the sensory data further at the next level of processing. All processing below the neocortex is subconscious.
- At the higher cortical levels, input from many lower levels both cortical and subcortical is integrated to form a conscious perception of the initial sensory experience.
- These higher cortical levels not only integrate processing of the “raw” sensory data, but also include integration of input from memory areas about past experiences with similar sensory stimuli.
- At the highest cortical levels the conscious perceptions formed at lower cortical levels are further processed asymmetrically in either Gestalt or Logic cortical columns, and hence perceived as a visuospatial pattern or a Gestalt, or abstractly as a verbal word based language or an abstract symbol based mathematical language.
- The very highest levels of conscious processing that underlie our thinking about conscious perceptions, while dependent upon input from all areas of the brain, are generally frontal lobe and particularly involve working memory areas in the Dorsolateral Frontal Cortex.
- A whole set of basal brainstem mechanisms maintain the organism in a state of homeostasis, such that higher level conscious sensory processing can proceed effectively: These include the Reticular Activating System, the Periventricular Survival System, the Vestibular System and the Sensory-Motor System. Imbalances within or between these systems may disrupt on-going sensory processing and integration at this and higher levels. Processing at this level is totally subconscious.
- The initial “raw” data stream is “sampled” by the Amygdala and other survival centres in the brainstem, and coloured by the survival emotions paired or associated with the sensory stimuli being analyzed, including the physiological responses to these emotions, and is the basis of Conditioned Learning. These primary survival emotions may disrupt on-going sensory processing and integration at this and higher levels. Processing at this level is subconscious.
- When survival emotions of the Fight or Flight response are activated above some “threshold” value, the amygdala and other brainstem structures such as the Periaqueductal Grey Matter of the midbrain inhibit frontal cortical processing, interfering with reasoning and problem-solving. The cause of this loss of higher level conscious cortical processing is a direct consequence of activation of the subconscious primary survival emotions of the Limbic System and Brainstem.
- Secondary processing of the sensory stimuli in the Brainstem, Limbic System and lower cortical levels generates a series of control functions defining the nature of the

sensory data stream (e.g. control of pupils in vision) and second-order integration of this sensory data (e.g. movement, shape and location of object in space). Processing at this level is subconscious.

- Further processing in the palaeocortical components of the Limbic System (e.g. cingulate, subcallosal and orbitofrontal cortices) generates secondary emotions relative to the sensory data stream and primary emotions already supplied by the amygdala and other brainstem areas via sampling memory of related events. These secondary limbic emotions may disrupt on-going sensory processing and integration at this and higher levels. Processing at this level is largely subconscious.
- Initial cortical processing is predominately bilateral and subconscious, and is dependent upon earlier processing at brainstem and subcortical levels. Emotions, either primary or secondary, may disrupt on-going sensory processing and integration at this and higher levels.
- At some level of cortical processing the sensory data stream emerges into a conscious perception, and is dependent upon earlier processing at brainstem, subcortical, and earlier cortical levels. Emotions, either primary or secondary, may disrupt on-going integration at this and higher levels
- At the highest levels of cortical processing, the processing is largely done in one hemisphere or the other and perceived consciously as a logical, rational thought or a visuospatial Gestalt, and is dependent upon earlier processing at brainstem, subcortical and cortical levels. Emotions, either primary or secondary, may disrupt on-going integration at this level, and any “thinking” dependent upon this level of processing.
- Thinking about the fully processed and integrated sensory experience in the frontal lobes, based upon remembered sensory experiences relevant to the current experience may lead to decisions, which will be represented neurologically by activation of either Logic or Gestalt “lead” functions or both.
- These “lead” functions will then initiate a cascade of neurological flow, which is initially frontal cortical, but rapidly flows into other cortical areas and subcortical structures like the basal ganglia, thalamus, and cerebellum, which in turn feedback to the cortex and each other. Emotions, either primary or secondary, may disrupt on-going processing and integration at any level of this process, and thus overtly affect the final outcome of the cognitive functions taking place.
- Coherent neurological processing at any stage of the above process is dependent upon both uninterrupted flows along integrative pathways and within integrative processing centres. Disruption or de-synchronisation of the timing of these integrative neural flows or disruption or de-synchronisation of processing in any of the integrative centres may result in loss of cognitive function.
- Maintaining integration along all integrative pathways and within all integrative centres produces optimum function, a state called Brain Integration in LEAP.
- Loss of integrated brain function is the principal cause of dysfunction in both mental and physical performance, called Loss of Brain Integration in LEAP.
- The primary mechanism causing Loss of Brain Integration is de-synchronisation and loss of timing of neural flows along integrative pathways and within integrative

centres by inhibition or excitation of these pathways and centres by neural flows originating from brainstem and limbic survival related emotions.

- On-going Loss of Brain Integration is often generated by early childhood trauma that creates long-term disruption of Brain Integration as a mechanism of coping.
- Other factors affecting Brain Integration are genetic, structural, organic brain damage, and environmental stressors:
 - Structural defects or abnormalities can be of developmental origin, e.g. neuronal migration problems, or result from toxin exposure at specific critical periods of development, e.g. fetal alcohol syndrome. Many cognitive defects have been shown to correlate with abnormalities in brain structure.²⁴
 - Organic Brain Damage may result from a head injury, and this damage often results in sclerosis that disrupts neural flows underlying Brain Integration (e.g. hippocampal sclerosis and subsequent epilepsy are often associated with learning disorders).
 - Genetic Factors affecting Brain Integration are often genes that code for specific alleles for specific enzymes involved in maintaining normal levels of neurotransmitters or receptors in brain circuits.²⁵ Deficiencies in either neurotransmitters or receptors will compromise Brain Integration, and have behavioural consequences. This is both the basis of much ADHD behaviour and the justification for drug use to ameliorate these behaviours.²⁶

Other genes may code for alleles that affect fatty acid metabolism and utilisation, especially in maintaining neuronal membrane stability and function. This affects predominately physical co-ordination and reading.²⁷

- Diet and nutritional deficiencies may also compromise brain function and result in loss of Brain Integration. Diets rich in fast or junk foods often create marginal nutritional deficiencies that may disrupt brain function, and often contain various preservatives and additives, like the azo-food dye tartrazine, that may cause a total loss of brain integration in sensitive individuals²⁸.

Indeed, the misbehaviour and academic performance of children and young adults have been shown to improve significantly with diet change or nutritional supplementation²⁹, and several recent books have discussed this aspect of behaviour and learning problems³⁰.

- Environmental factors such as electromagnetic fields emitted from man-made electronic equipment and Geopathic stress from distortions in the earth's electromagnetic fields may affect the brain integration of sensitive individuals and result in learning problems.³¹

Loss of Brain Integration and Compensation

When Brain Integration is lost via disruption of the most efficient neural pathways and/or centres, either by organic damage or by functional inhibition of cortical or subcortical functions due to outputs from survival centres, specific conscious functions dependent upon this integration is also disrupted. The loss of overt conscious function is, however, often far less than the degree of interference with underlying functions might suggest because the brain will

automatically compensate for these disrupted flows by using other areas of the brain, both conscious and subconscious to produce the most efficient processing possible.

Thus, even children with considerable organic brain damage will often establish compensatory neurological patterns of activity to produce varying levels of function in spite of massive disruption of neural pathways underlying normal function, e.g. children with cerebral palsy may learn to walk and talk. It is indeed this tremendous compensatory capacity of the brain that allows even highly disintegrated brain function to produce some degree of function, however, the level of dysfunction controls the degree of compensation. Thus, the greater the degree of dysfunction present, the less compensation that is possible.

If the disruption of integrated function is at the more basal levels of integration, the ability to compensate for the resulting dysfunction is much more limited than if the loss of integration is at a higher level of processing because all higher levels of processing are dependent upon the quality of the data integrated at earlier levels of processing. For instance, damage to an early component of vision, say the retina or optic nerve totally disrupts sight, while damage and hence loss of integration in the V₃ area of the occipital cortex may only affect colour vision.

When the highest levels of cortical integration are disrupted directly or lower level cortical or subcortical functions underlying these higher cortical functions are disrupted, we may lose the capacity to “think” in certain ways. For instance, we may maintain Gestalt creative abilities (e.g. be good at art and design), but lose the ability to perform even simple mathematics because of the loss of the ability to abstract (e.g. are hopeless at maths). Specific Learning Disorders result from the loss of integration in or supporting higher-level cortical functions activated by consciousness.

Children and adults suffering Specific Learning Disorders usually know what they need to do, often even how to do it, e.g. I want to spell this word, so I need to sequence the letters and remember this sequence. But they just cannot activate the necessary subcortical and cortical processing to do what they want to do consciously because of loss of integration at some level of neural processing required to do this function, whether this be to read, spell, write or do mathematics. However, they will still attempt to perform these functions, but in some compensated way. For instance, a child that cannot spell words correctly still attempts to spell words, but using phonetics to compensate for the “mind’s eye” image he/she cannot create.

Because the level at which the integration is disrupted is unknown to the consciousness and compensation is largely subconscious and automatic, a person with Specific Learning Disorders is only aware that some function is difficult or not possible to perform, but not why this is so. Also, most of the time Brain Integration is lost in subconscious functions that are always inaccessible to our consciousness. So how are we to detect and correct the loss of Brain Integration blocking our function?

Kinesiology: Its Role in Assessing and Correcting Specific Learning Difficulties.

Since the relevant functions and processes that control our ability to perform most academic tasks are subconscious, how can we evaluate them, or know the type of “block” preventing access to them. Or how can we know at what level this block in processing occurs, particularly for more complex tasks that require several levels of neural processing? The answer is using Kinesiology because it provides direct access to subconscious functions via the interface of muscle proprioception. Muscle proprioception is totally subconscious, yet linked with other subconscious processing, including neural processing underlying all cognitive functions.³²

Kinesiology is a western muscle technology developed by traditional Structural Kinesiologists as a diagnostic tool to assess muscle dysfunction,³³ and then adapted and modified by Chiropractors as a diagnostic and therapeutic tool.³⁴ In past three decades it has been further developed as both a diagnostic and therapeutic tool in Energy Medicine for the assessment and treatment of energetic imbalances and their related physical, physiological, and psychological symptoms.

There are really three separate modalities currently covered by the single term Kinesiology, each with a different description and use of the same muscle response. Broadly speaking, Kinesiology can be divided into three major categories:

- 1) **Structural or Academic Kinesiology:** An academic discipline taught in universities that involves the study of biomechanics of motion. When the muscle response is performed manually, it is termed a Muscle Test and the muscle response is called either Strong or Weak. Individual muscles are placed into their position of greatest mechanical advantage, isolating them as the Prime Mover in a specific action and then force is applied against the muscle to evaluate its relative strength compared to normal function.
- 2) **Applied Kinesiology:** An academic discipline taught in Chiropractic Colleges in which the muscle response is still called a Muscle Test, and the muscle response is either Strong or Weak. The Muscle Test is used both diagnostically to assess the strength of the muscle, and as an indicator of the type of therapy required to be employed to correct the imbalance detected by the muscle response, as well as confirmation of the efficacy of the treatment.
- 3) **Energetic Kinesiology:** A group of Kinesiologic systems that use the muscle response as a biofeedback tool, and that use techniques based in eastern energetic models to correct imbalances located via muscle monitoring. Currently Energetic Kinesiology is taught predominately in an informal workshop system. One or more muscles may be used as an Indicator Muscle to provide biofeedback to monitor the muscle response to physical, physiologic, emotional and mental stresses. It is not a measure of strength, but rather information flow between the muscle sensors and the Central Nervous System, so when the muscle gives passively to the pressure applied, it is said to Unlock, whereas if it holds against moderate pressure it is said to Lock. The Indicator Muscle response is first used to identify the specific imbalance in the physiological or energetic systems, and then used to assess and identify the technique required for correction of imbalances within the energetic systems of the human body.

In Energetic Kinesiology you are not “testing” a muscle for its relative strength as in Structural Kinesiology, but rather “monitoring” its ability to maintain un-interrupted, coherent communication with the Central Nervous System. When a muscle “unlocks” during muscle monitoring, it is not “weak”, but rather it has been “inhibited” via another muscle or neurological circuit. This inhibition, however, may originate at the physical-structural level, the emotional-mental level, or at the etheric-energetic level due to disturbances in the etheric energy systems such as disrupted Ch'i flows of Acupuncture System or Pranic energy flows of the Chakra system. (For a more detailed discussion of Kinesiology and Acupressure, see Chapter 2; and for the Chakra-Nadi System see Chapter 12 of *A Revolutionary Way of Thinking*³⁵).

LEAP® uses Energetic Kinesiology as a diagnostic and therapeutic tool to assess and correct the energetic imbalances underlying Specific Learning Disabilities. The subconscious nature of

the muscle response provides an ideal interface with several levels of response from the body.³⁶ A muscle will respond in the same way, that is by locking or unlocking, to physical stress (e.g. tendon strain); an emotional/mental stress (e.g. anger or negative thoughts); or energetic stress (an acupuncture meridian or chakra imbalance). When a previously locked muscle suddenly unlocks, it indicates an active stressor has been located. A reciprocal change in muscle response (the muscle now going from unlocked back to locked) may be used to identify specific correction techniques via frequency matching.

Kinesiology is therefore an excellent tool to investigate stresses affecting subconscious brain function as it can provide a directly observable response to these stresses – an unlocking or locking muscle. Since subconscious mental functions form the basis of all higher cognitive functions like thinking, detecting stresses that may compromise these functions is vital to understanding the nature of learning problems. Throughout LEAP, the link between stress in subconscious processing and muscle response is used extensively to evaluate the extent of access to specific mental functions and the nature of the "block" that prevents full access to these functions. Kinesiology, therefore, provides an effective means of assessing the nature and degree of subconscious dysfunction resulting in the loss of Brain Integration underlying all Specific Learning Disabilities.³⁷

Kinesiology not only provides a means of identifying where these "blocks" in function occur, as noted above, but more importantly, provide a means of identifying the "nature" of the disturbance de-synchronising neural flows resulting in the "block" in function. Muscle monitoring provides an interface between neurological function and the more subtle energies of the energetic, emotional and mental bodies.³⁸ Disturbances at any of these levels can cause a change in muscle response during monitoring. The vibrational frequency of the underlying cause of the dysfunction resulting in the "indicator change" or change in muscle response can then be "matched" against various "frequency domains" of acupoints and finger modes enabling the source of the disturbance to be specifically identified.³⁹

Once the stress causing a "block" in function has been located, then by simply touching specific acupoints or holding specific finger modes and remonitoring the muscle, the Specific Indicator Point or Finger Mode causing a reciprocal "indicator change" identifies the exact nature of the factor causing the "block". For instance, if holding "emotion mode" changes the indicator muscle response, then the underlying cause of the "block" is an emotional disturbance that alters the underlying physiological function.

What is critical for successful long-term correction, however, is locating the exact subconscious function that is blocked. For some subconscious functions, simply touching specific acupoints or holding finger modes will detect these "blocks". However, many other functions, particularly those "deep" within the subcortical areas of the brainstem, limbic system and other brain nuclei, cannot be accessed by these simple methods. To access these very specific subconscious functions requires activating specific patterns and combinations of Specific Indicator Points and Finger Modes, termed "formatting" in Applied Physiology.

The Role of Acupressure Formatting in Accessing Specific Brain Structures and Functions:

Richard Utt, the founder and developer of Applied Physiology, developed a system called *acupressure formatting* to provide the specificity required to address specific physiological functions directly.⁴⁰ Formatting uses the frequency resonance "match" between specific combinations of acupoints of the Acupuncture Meridian System, called Specific Indicator Points, and/or Finger Modes, based on Mudras of the Yogic system, and specific physiological functions or anatomical structures. If there is a frequency match denoted by a change in muscle response

when these multi-acupoint combinations are circuit-located (touched) and/or specific finger modes held simultaneously, this indicates stress in specific physiological functions or anatomical structures activated by these acupoint-finger mode combinations.

Although there has been no external validation of activation of specific brain structures via acupressure formatting, two types of information provide practical and theoretical support for this technique. First of all, there is ample anecdotal evidence to demonstrate that specific brain functions demonstrating “stress” by acupressure formatting changed significantly following treatment. Secondly, recent scientific studies have shown specific activation of brain areas by acupuncture stimulation.

For instance, when people have hypersensitivity to normal levels of light, often needing to wear sunglasses whenever outside, acupressure formatting specifically for the pupillary control mechanisms of the amygdala and posterior hypothalamic nuclei show “stress” via kinesiology. When these formats are activated, and followed by acupressure corrections or corrections using light/sound on acupoints, the hypersensitivity to light disappears. These people now respond normally to light levels, indicating that the function of this basal visual mechanism of pupillary control has indeed been normalised.

Likewise, when people demonstrate deficit Digit Span, a measure of auditory short-term memory that is highly dependent upon integrated hippocampal function, “stress” is found in the hippocampus and other memory areas of the brain via acupressure formatting and kinesiology. Once these “stresses” are eliminated by various acupressure techniques, people’s auditory short-term memories reproducibly improve to normal or in some cases to better than normal as measured on standard psychological testing.⁴¹ This is in spite of the fact that millions of Digit Span tests performed by psychologists have shown that Digit Span does not improve spontaneously, and basically is stable over your lifetime.⁴²

In a similar way, even highly complex functions like reading comprehension show measurable improvements after LEAP Brain Formatting to access “stress” caused by attempting this activity, followed by acupressure therapy.⁴³ Thus, even though the exact nature of the activation of cortical and subcortical neural substrates via acupressure formatting is not understood, reproducible observable and measurable normalisation of the functions directly reliant upon these neural substrates strongly suggest that these specific brain structures are indeed “targeted” by acupressure formatting.

While the mechanism of how these multi-acupoint-mode combinations activate or access specific subcortical and cortical structures and functions remains unknown, recent scientific evidence of highly specific activation of cortical and subcortical structures by specific acupoint stimulation is now available, providing at least a plausible mechanism for acupressure formatting.

Acupressure Effects On Brain Function.

Acupuncture or acupressure therapy consists of either stimulating or dispersing the flow of energy, called Ch’i by the Chinese, by activation of specific acupoints on the surface of the body.⁴⁴ The acupoints have been shown to have a unique histological microstructure and have been accurately mapped using electrical detection because they have been found to be ‘null’ points or points of least electrical resistance on the surface of the body.⁴⁵ The electrical mapping is very highly correlated with Chinese maps of these same points.⁴⁶

The Chinese propose that Ch’i energy is a dynamic force in constant flux that circulates throughout the body but that follows specific pathways and specific rules, and can be controlled

or modulated by stimulation of acupoints.⁴⁷ Further, one of the premises of both Energy Medicine and Traditional Chinese Medicine is that for every energetic imbalance, there are corresponding or related symptoms, or disturbance in neurological and physiological function. Thus, activation of acupoints will alter the energetic flows associated with these points causing a reciprocal activation or modulation of the neurology and physiology associated with these altered energetic flows.

In animal studies, Zhongfang et al., showed that the stimulation of specific acupoints using electro-acupuncture could activate or inhibit the electrical activity of specific neurons within the amygdaloid nucleus, the subconscious emotional control center of the brain, resulting in increasing and decreasing rates of neuronal discharge.⁴⁸ They also found that electro-acupuncture stimulation of different acupoints caused either consistent but different responses in the discharge rate of a single group of neurons in the amygdala or no effect on these neurons. Thus stimulation of specific individual acupoints caused highly specific patterns of discharge in this deep brain structure. They also found that stimulation of 'sham' points produced no detectable change in neuronal firing rates in the amygdala. Another study found that electro-acupuncture increased levels of various neuropeptides in the rat brain, with significant increases in the hippocampus, a limbic structure directly involved in short-term memory.⁴⁹

Traditional acupuncture techniques are stated to be helpful for strengthening cerebral function and improving intelligence.⁵⁰ In human subjects, Abad-Alegria et al. demonstrated that acupuncture stimulation of the acupoint 'Heart 7' produced long-lasting increases in the P300 wave, a late evoked EEG potential that has been associated with cognitive activities.⁵¹ Stimulation of another acupoint, 'Large Intestine 4', did not change the P300 wave, suggesting this cortical response to acupoint stimulation is highly specific.

Using f-MRI brain scanning researchers have shown that specific acupoint stimulation caused specific patterns of neuronal activity in the cerebral cortex that correlated with traditional Chinese Medicine's treatment for visual disorders.⁵² Even though the acupoints were on the side of the little toe and side of the foot, stimulation of individual acupoints activated specific and unique areas of the occipital cortex. Since each of the acupoints stimulated is associated in traditional Chinese Medicine with a specific type of eye or vision disorder, activation of specific occipital regions associated with different aspects of visual processing provides strong correlation between acupoint stimulation and relevant cortical activation.

Another f-MRI study showed needle acupuncture stimulation of Large Intestine 4, caused activation of the limbic system and subcortical structures associated with reward and punishment and emotional and behavioural regulation.⁵³ Again specific acupoints activated highly specific limbic and subcortical areas. In these studies as in the study of Zhongfang et al., stimulation of non-acupuncture 'sham' points had no effect on cortical or subcortical activation demonstrating the specificity of acupoint stimulation.

While none of the studies above provide a direct test of acupressure formatting, they do at least provide a theoretical mechanism by which it might work.

LEAP® Acupressure Formatting:

The LEAP® treatment is based upon activating specific brain structures using acupressure formatting,⁵⁴ and employs specific acupressure protocols to improve brain function.⁵⁵ The initial acupressure formats used in LEAP® were developed by Richard Utt in Applied Physiology, but over the years myself and others have greatly expanded the number of brain structures and functions accessed by acupressure formatting. Most recently, Hugo Tobar has contributed a large

number of acupressure formats to access brainstem and other brain structures and functions not accessible before by this powerful system of formatting.⁵⁶

LEAP[®] acupressure protocols follow the neurological flow of sensory information into the brain and between brain areas, with specific acupressure formats for most major integrative centres and pathways. While the frequency match of scanning neurological diagrams does give valuable information about the location of stress in specific structures or pathways, acupressure formatting for the same structure or pathway enters far more stress into the energetic circuit, and hence permits a more robust correction.

LEAP[®] acupressure treatment for the Correction of Specific Learning Difficulties:

Acupoint stimulation in the acupuncture studies discussed above was either electro-acupuncture where a small electrical current is applied to a needle inserted into an acupoint, or traditional needle acupuncture where a needle is inserted to an acupoint and then manually activated. However, using f-MRI to monitor the effects on cortical activation, Jones et al. have recently shown the effects elicited by conventional needle acupoint stimulation were indistinguishable from those produced by highly focused pulses of ultrasound directed to acupoints over a wide range of ultrasound parameters.⁵⁷

Thus, what appears to be important is that the acupoint is sufficiently stimulated, not the specific type of acupoint stimulation. In LEAP[®] acupressure treatments use either finger pressure or a Tei Shin, a blunt spring-loaded probe, that is rapidly tapped on an acupoint to apply specific acupoint stimulation. Both finger acupressure and stimulation with the Tei Shin, called needleless acupuncture by the Chinese, are standard techniques to stimulate acupoints with a long anecdotal history of effectiveness.⁵⁸

Acupressure has a number of advantages over traditional acupuncture in this application, as it is non-invasive, not painful, and well tolerated by children. Also the problem of sterile needles and bleeding are eliminated. Most importantly, the LEAP[®] acupressure corrections rely on multi-point sequential stimulation, something very difficult to do with needles, and not part of traditional acupuncture theory. However, this multi-point acupressure stimulation is capable of powerfully stimulating specific brain structures and re-synchronising brain function, as evidenced by the profound changes in people's performance on standardised psychological tests, and in the classroom following LEAP[®] treatment.

Results of Application of the LEAP[®] Acupressure Protocol:

The Learning Enhancement Acupressure Program (LEAP[®]) has been developed empirically since 1989 and has been applied to the improvement of specific learning problems on several thousand subjects with generally excellent results. The LEAP[®] acupressure protocols have been empirically demonstrated to reproducibly improve various learning dysfunctions including deficit Digit Span ability (Fig. 2) and poor reading comprehension (Fig. 3).⁵⁹

Pre and post-testing of subjects undergoing the LEAP[®] protocol with WISC-R has shown marked improvement on all of the subtests.⁶⁰ Improvement was consistently seen even on visuo-spatial subtests like Block Design that had not previously been observed to change over time, regardless of considerable periods of remedial treatment. Block design is often considered to be a measure of innate intelligence as it tests spatial reasoning not affected by acquired verbal knowledge.⁶¹

After LEAP[®] treatment some subjects have shown an increase in the Block Design task from a previous ranking in the 25th percentile (low average) of same aged children to the 75th

percentile (high average) and changes to as high as the 99.6 percentile (superior) have been observed. Changes of equal magnitudes have been observed on all subtests, for instance from the 1st to the 50th percentile ranking on the Digit Span subtest, a measure of auditory short-term memory.⁶²

Figure 2a. Digit Span Scores for the Non Treatment Group at the Pre-test (B) and Post-test (A) of the Study. * Zero backwards as subject could not understand concept of reversing digits.

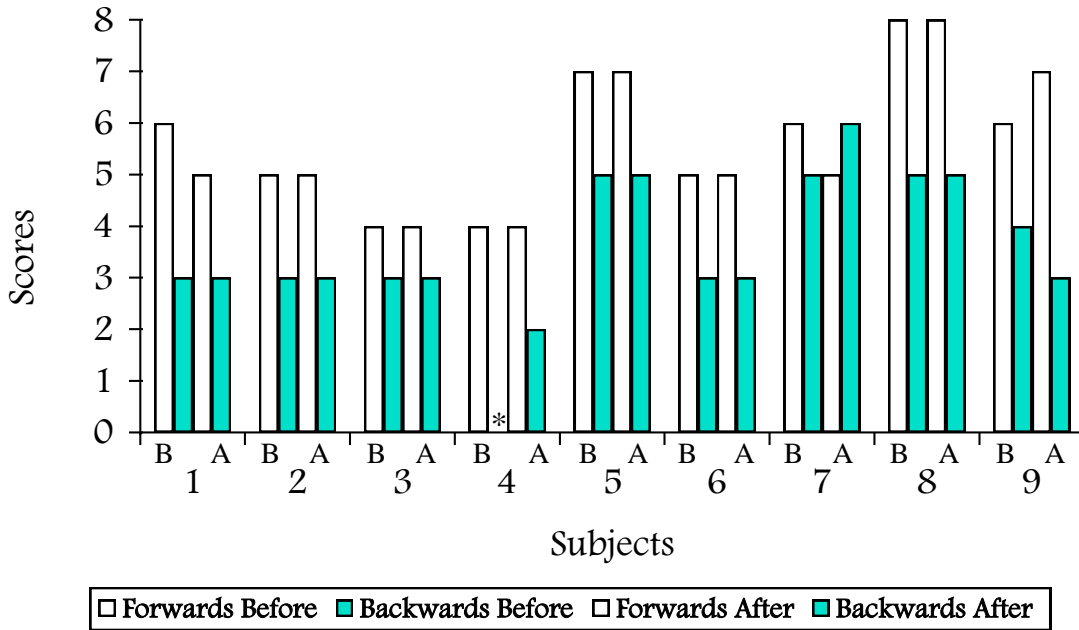


Figure 2b. Digit Span Scores for the LEAP® Treatment Group at the Pre-test (B) and Post-test (A) of the Study.

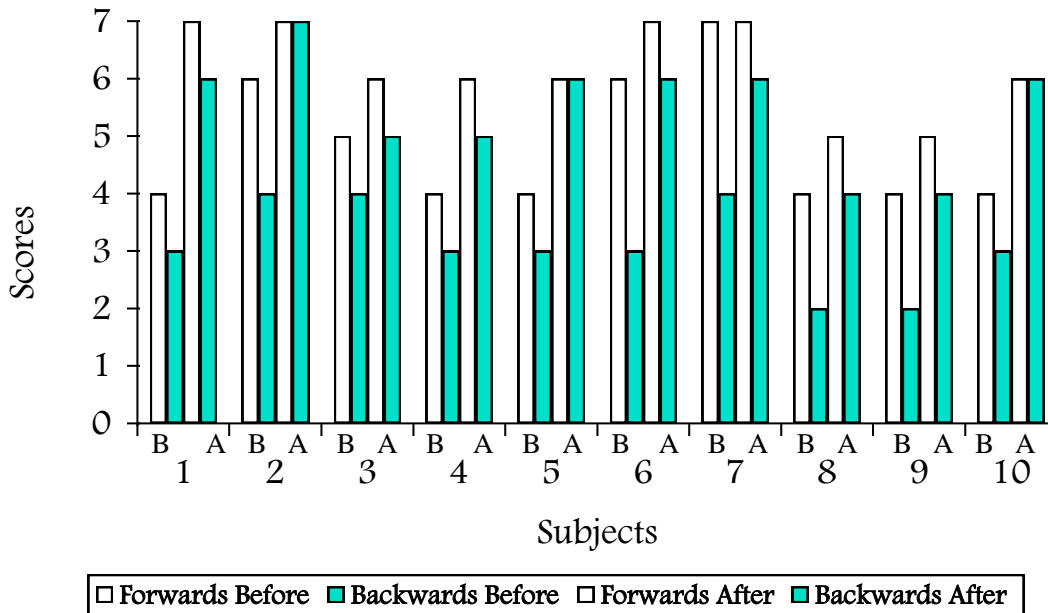


Figure 3a. Reading Comprehension Scores for the Non Treatment Group at the Pre-test and Post-test of the Study. * Six-year-old subject unable to read.

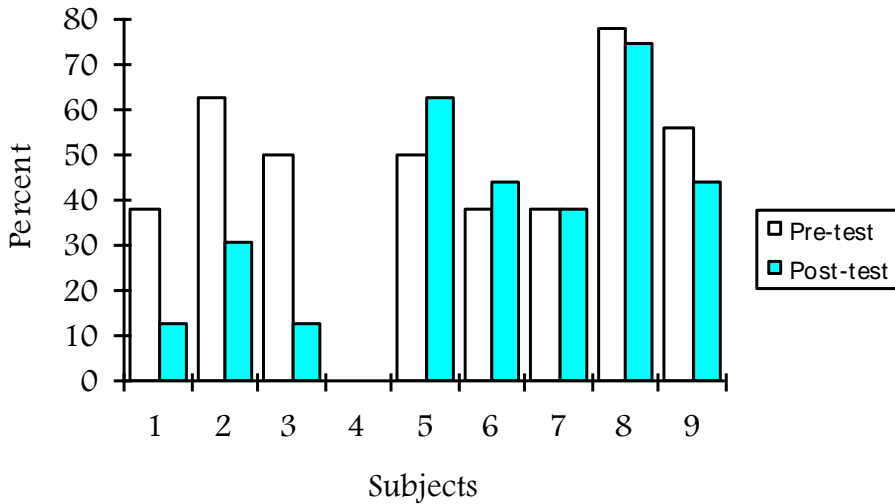
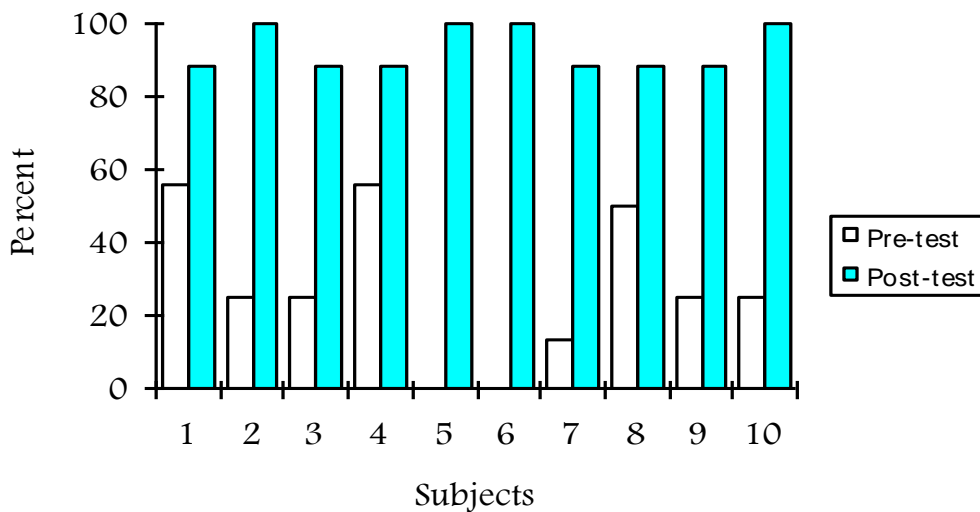
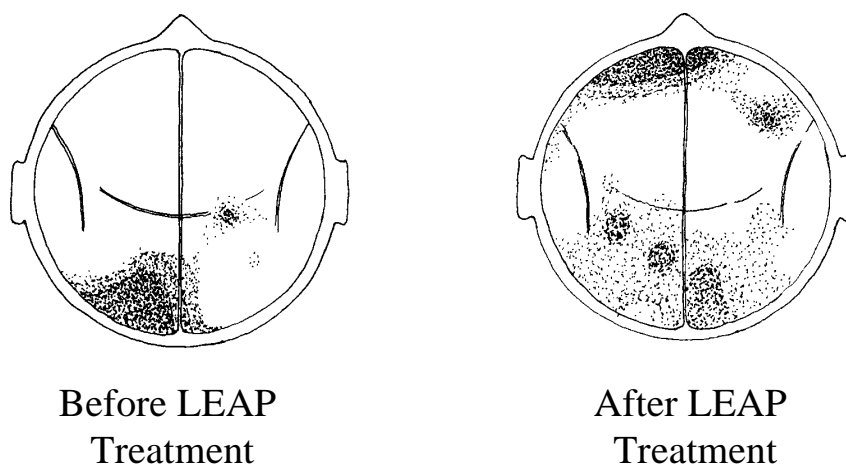


Figure 3b. Reading Comprehension Scores for the LEAP[®] Treatment Group at the Pre-test and Post-test of the Study. * 16-year-old subject unable to read.
** 11-year-old subject able to read a few small words.



LEAP® acupressure treatment has also been shown to normalize cortical activity during decision-making using Steady-State Visually Evoked Potential (SSVEP) cortical activity mapping before and after treatment.⁶³ While normal people activate their frontal cortex during attentional and decision-making, ADD children and adults do not. Rather they maintain activity primarily in their occipital lobes, even when they should be paying attention and during decision-making. After the LEAP® acupressure treatment, ADD adults switched cortical activity to their frontal lobes during both attentional and decision-making tasks, demonstrating normalisation of cortical activity following treatment (Fig. 5).

Figure 5. SSVEP Maps of typical subjects Before and After LEAP treatment. Degree of stippling indicates degree of activity. Before treatment subjects with learning difficulties showed the most activity in the occipital lobes when performing attentional and decision-making tasks. After treatment the cortical activity now switched to the frontal lobes on the same attentional and decision-making tasks, the same areas active when normal subjects perform these tasks.



In those cases where improvement was not observed or was marginal following the LEAP® treatment, either on several or on only a specific WISC-R subtest, neurological assessment demonstrated varying degrees of organic brain damage in almost all cases.⁶⁴ The damage observed varied from developmental problems such as neuronal migration problems, temporal lobe epilepsy, hypoxic damage resulting from birth difficulties to traumatic injury such as blows to the head.

In Summary:

The LEAP® treatment protocol permits the identification of the causal factors underlying the de-synchronization of neural flows within the brain, including the early childhood traumas, using kinesiology. Then the application of acupressure and other energetic techniques to re-synchronises these neural flows produces integrated brain function.

The person is then challenged to perform the function that was poorly integrated before treatment, e.g. reading, spelling or maths. If integration is lost again, as evidenced by muscle monitoring, the treatment protocol is repeated, re-synchronising and integrating the brain in the context of this specific function. Once the brain functions are fully integrated, and integrated function can be maintained under the stress of performing a previously stressful function, e.g. reading, there is a corresponding cessation or reduction of the original learning or memory problems with a concomitant normalisation of these functions.

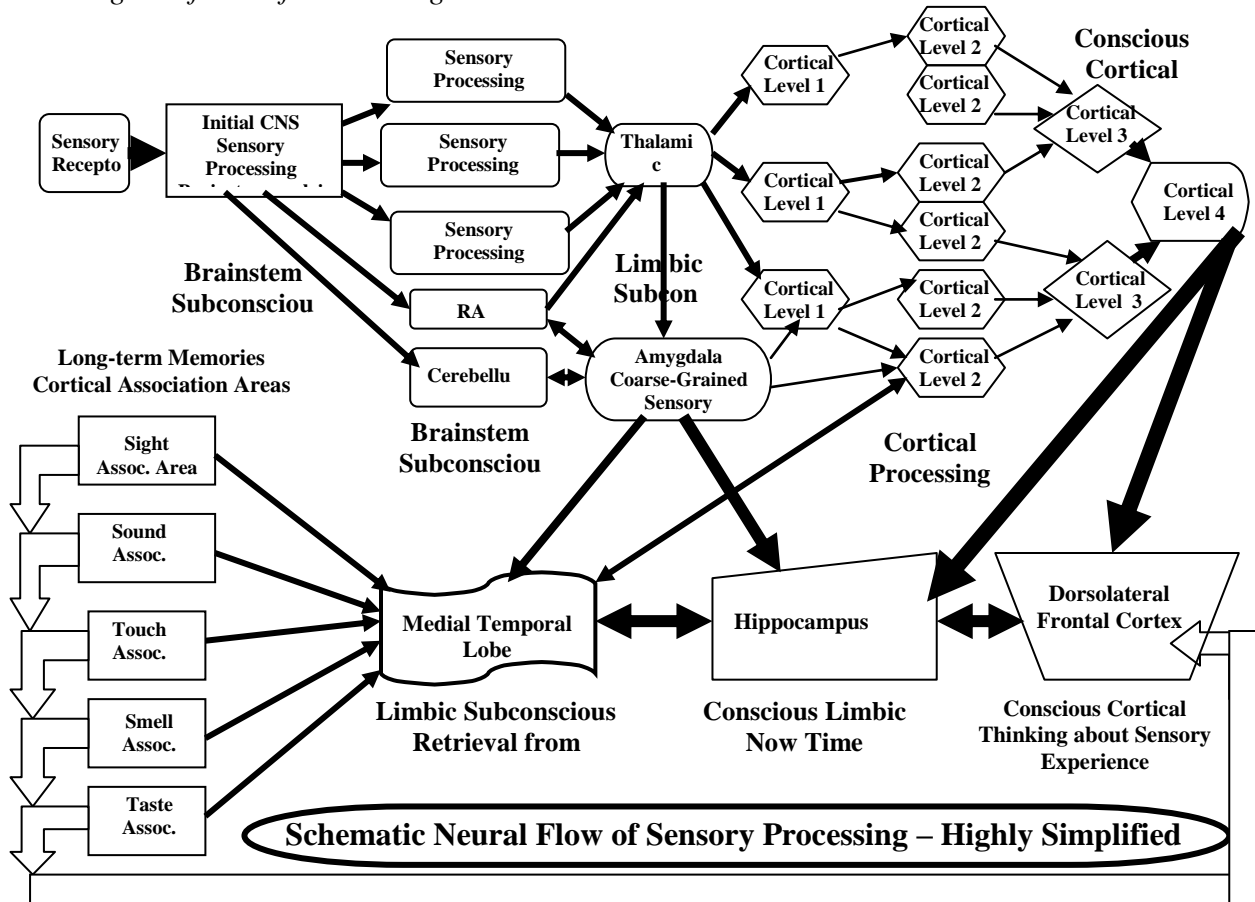
To summarise, the LEAP® model of learning is based upon the order and hierarchy of neural processing of sensory information in the brain that follows a roughly similar pattern for most senses: (See Figure 6 below and Appendix I)

1. Most neural processing starts initially as a single data stream from a receptor to the central nervous system and brainstem where it is initially processed as subconscious neural impulses in a brainstem or cranial nerve nuclei, and then partitioned and sent to several other subconscious brainstem nuclei for specific types of processing.
2. Output from most of these brainstem nuclei converge into one thalamic nucleus, which integrates this input and again generates neural flows to a number of subconscious limbic and cortical areas for further processing.
3. The subconscious limbic and cortical areas receiving this thalamic output further develop various aspects of the original sensory input at several levels, often with reference to similar information held in memory, before output of these separate integrative centres is integrated to create a conscious perception at higher cortical levels.
4. A conscious perception represents the integrated output from these subconscious limbic and cortical processing centres and once again becomes a single data stream.
5. This single integrated data stream usually is rapidly partitioned again into yet higher levels of cortical processing where further integration of conscious perceptions of all senses creates the unified multi-sensory experience of our world.
6. New neural flows are also created in the cortex via activation of information stored in memory, which then feed into the processing of the original sensory input at both subcortical and cortical levels.
7. Sensory based neural flows sampled by subconscious emotional and survival centres like the amygdalae generate new neural impulses that can combine with or disrupt neural flows and processing in higher brainstem, limbic and cortical areas.
8. At the highest levels of neural processing thinking becomes asymmetrical and is dominated by cortical columns in predominately one hemisphere. Neural processing becomes either linear, sequential and rational based upon the rules and principles of deductive reasoning - Logic, or global, holistic and non-rational based upon the simultaneous intuitive “knowing” of inductive reasoning – Gestalt.
9. The Logic and Gestalt functions are located in the cortical columns of the left and right cerebral hemispheres, and are termed Logic or Gestalt “lead” functions in LEAP®. For right-handed people, the Logic “lead” functions are located in the left cerebral hemisphere, and the Gestalt “lead” functions are located in the right cerebral hemisphere. In non right-handed people, left-handed and ambidextrous people, the Logic “lead” functions are located in the right cerebral hemisphere, and the Gestalt “lead” functions are located in the left cerebral hemisphere.
10. The Logic and Gestalt “lead” functions initiate a specific cognitive function, the type of task the consciousness wants the brain to perform. This consciously initiated neural processing rapidly becomes both bilateral and subconscious. The integration of these subconscious largely bilateral processes generates the “answer” or action requested by the consciousness, becoming conscious once more.
11. The consciously perceived “answer”, may then initiate another round of conscious activation of Gestalt and/or Logic cortical “lead” functions while we “think” about the

“answer” to our last question, and then via other Gestalt or Logic “lead” functions create a conscious “question”, which in turn initiates another stream of subconscious processing – and so on.

12. Logic and Gestalt “lead” functions only initiate cognitive functions, requesting certain types of processing to be carried out, they do not actually perform this processing. Rather, this processing is largely performed within subconscious cortical and subcortical integrative centres linked by integrative pathways such as the Corpus Callosum.

Figure 6. Schematic of the neural processing of sensory data. This diagram is highly simplified with many pathways omitted for the sake of clarity. Note that conscious perception only emerges at the highest level of cortical processing, and is dependent upon many levels of subconscious integration in brainstem, limbic and cortical centres linked by extensive integrative pathways. Loss of timing and synchrony in anyone of these integrative centres or pathways will result in some degree of loss of Brain Integration.



From this simple schematic of sensory processing, one of the most striking features is the relatively late emergence of consciousness, and only at the very highest level of neural processing does Logic and Gestalt “thinking” arise. Of all the neural processing involved in processing the sensation and what I feel about it, consciousness only emerges at the very last stages of integration in cortical processing. Furthermore, while I may have conscious awareness of “feelings” about this sensory experience, emotional processing outside of my consciousness largely generates these “feelings”.

The conscious act of learning or thinking therefore requires integration of sensory experience at many levels of neural processing from the basal integration of raw sensory input to the highest levels of conscious experience. Integration at each level is dependent upon precisely timed neural flows along integrative pathways that are synchronised with other neural flows from other integrative centres. The higher in the neural processing of sensory input, the greater the number of integrative pathways and integrative areas involved, with these integrative centres generating further output to yet higher integrative centres until consciousness overtly directs the highest levels of our integration, our cognitive thinking, upon which all learning relies.

Thus, for any type of learning to occur, all the lower levels of sensory and memory integration must be coherent and fully synchronised. Higher levels of integration in the frontal cortices involve the current sensory perception and integration of inputs from other sensory processing happening simultaneously as well as sensory experience imported directly from long-term memory to create our “thinking” about what was perceived.

Clearly, a breakdown of integration at any level in this processing will disrupt to varying degrees integration at all higher levels. Likewise, loss of precisely timed and synchronised neural flows in all integrative pathways linking these integrative centres will also cause loss of integrated function. Loss of integrated brain function either directly due to disrupted processing within integrative centres or indirectly via de-synchronisation of neural flows between these integrative centres will result at the highest levels of processing in the loss of a specific conscious mental capacity or ability.

This disruption of integration and loss of neural timing is termed “Loss of Brain Integration” in LEAP®, and is clearly a major cause of Learning Disorders. The schematic above also provides a plausible explanation of why using Kinesiology to detect stress in these subconscious processes and pathways, and the application of acupressure to re-synchronise these neural flows within and between these subconscious processing centres has been so successful in the LEAP® program.

From the perspective of the LEAP® model of learning, Specific Learning Disorders (SLDs) would result from the following factors:

1. Either “blocked” or disrupted neural flows linking gray matter integrative centres, such as brain and brainstem nuclei or cortical columns of the cerebral cortex. These are termed Integrative Pathways in LEAP®.

The Corpus callosum is the single biggest integrative pathway in the brain, and functionally as measured by muscle response, always “shutdown” to varying degrees when SLDs are present. In fact, a shutdown Corpus Callosum is a direct marker for the loss of Brain Integration.

2. Loss of Brain Integration is primarily the result of loss of synchronised neural flows through the various integrative pathways due to an excess of pre- or post-synaptic inhibition or excitation of the neurons comprising these pathways. This results most often from the output of the amygdala and other limbic and brainstem survival and emotional centres.
3. Or, loss of Brain Integration results from disruption of coherent function of gray matter integrative centres, such as cortical columns or brain nuclei, via loss of synchronised inputs to these centres or direct inhibition or excitation of neurons within these centres due to the output of the amygdalae and other limbic and brainstem survival and emotional centres.

4. The loss of coherent integrated processing within and between integrative centres disrupts either the subconscious or conscious processing underlying cognitive functions – our thinking. In either case, all we are ever “conscious of” is our inability to perform certain types of cognitive processes or thinking, but never “why” we cannot perform these functions.
5. Disruption of integrative centres and pathways, and hence loss of brain integration with concomitant learning difficulties can also be caused by the presence of toxins or allergens in sensitive individuals. In some people, even more subtle environmental factors such as electromagnetic fields or Geopathic stress may cause loss of Brain Integration.

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APPENDIX I

An Example of sensory processing from the Receptor to Consciousness.

For a person to “think” about something requires many levels of neural processing, only the last levels reaching consciousness. For example, for me to have a “thought” about something I “see”, for instance a chair, requires the following levels of neural processing:

- Light is reflected off the object into my eyes, which is absorbed by the photoreceptors in the retina generating a nerve impulse in the bipolar or horizontal neurons, which then transmit this nerve impulse either directly to the ganglion cells, or indirectly via the amacrine cells to the ganglion cells where these different inputs to the ganglion cells are integrated. Processing is subconscious.
- Once the ganglion cells “fire”, the nerve impulse travels via the optic tract (nerve) to the Lateral Geniculate Nucleus (LGN) as a single data stream. Processing is subconscious.
- The initial single data stream arriving at the LGN is partitioned into a faster Magnocellular pathway going to the Extrastriate cortex and a slower Parvocellular pathway to the Striate cortex. Processing is subconscious.
- The Magnocellular pathways are further partitioned to a variety of areas in the extrastriate cortex, generating information about the object’s movement (MT/V5), 3-D location in space (V5), general shape or form (V4) and colour or colour pattern on the object (V3, V3/VP). All of this information will form part of the conscious perception of the object, but at this level is still subconscious, but on the borderline of consciousness, e.g. the phenomenon of blind-sight.
- At the same time, the Parvocellular pathways direct the data stream largely from the central fovea of the retina to the striate cortex where image formation begins in area V1, and is further elaborated in area V2. Processing in V1 and V2 is largely subconscious with conscious perception only emerging near the end of processing in V2 when the highly developed image forms the basis of higher level visual processing in the Posterior Parietal Cortex and Inferior Temporal Cortex.
- Part of this elaboration is to correct for the reversal and inversion of the object in external reality caused by the lens system of the eye in forming the retinal image. You are conscious only of seeing the image, say the letter “b”. However, the “b” in external reality is first represented as a “p” in the retinal image, which is then first inverted to a “d”, and finally reversed to appear as the “b” of conscious perception in the striate cortex. These inversion and reversal processes are totally subconscious. Failure of the inversion and reversal processes leave a “p” in conscious perception, while failure only of the reversal process leaves a “d” in conscious perception. Indeed, a common problem for dyslexics is looking at a “b”, but perceiving a “d” due to the failure of this subconscious reversal mechanism.
- The Striate processing creates a high resolution static, black and white image, which is then synchronised and integrated with the Extrastriate processing to produce a coloured object of a specific shape, located in a specific place in 3-D space, that is either moving or not moving – in my case a stationary blue plastic chair at the desk. Thus, while this unified perception is conscious, all of the underlying processes that made up this conscious perception were subconscious.

- Part of this integrated striate and extrastriate processing now flows up into the Posterior Parietal Cortex where the object's (chair) location relative to other objects in three-dimensional space will be elaborated into the conscious perception of "where" the chair is –the "Where pathway".
- At the same time, the combined striate and extrastriate processing will also flow down along the Inferior Temporal Cortex to define via accessing long-term memory of objects of this shape, exactly "what" the object is – the "What pathway".
- Now that I have "seen" the blue plastic chair next to the desk in my room, this unified conscious image is then forwarded to my Hippocampus that creates a short-term memory trace of the perceived object. Thus I am aware of the blue plastic chair in y room even when I am no longer looking at it.
- At the same time, the Amygdala samples this hippocampal image and accesses emotional memories I have associated with blue plastic chairs, e.g. "Blue plastic chairs are ugly!" These emotional memories are then fed back into the hippocampus, and "colour" future thinking about the chair. Most of this processing is subconscious except the "feeling" that surfaces into consciousness.
- This conscious hippocampal perception may then be sent forward to my Working Memory in my Dorsolateral Frontal Cortex (DLFC) to think about and analyse the blue plastic chair in its current environment. I may then import information about chairs in general from long-term memory association areas of my occipital cortex directly into Working Memory to provide further information to evaluate both this chair in relation to other chairs, and/or my feelings about blue plastic chairs in particular.
- To evaluate this chair, I may choose to deductively analyse the details of each piece of the chair – the legs, the seat, the back, etc., and how these pieces go together to create a chair (Logic), or simply induce my overall impression of the chair in this context and get a "gut" feeling about the chair in this context – "It simply doesn't "go" with the desk!" (Gestalt).

General Schema of Sensory Processing and Integration:

All sensory processing follows roughly the same pattern. A single data stream of neural impulses from a sensory receptor (e.g. for hearing - cochlea), arrives via a cranial nerve (8th for hearing) or spinal tract at an initial brainstem integrative centre(s) the cranial nerve nuclei or other brainstem nuclei (e.g. for hearing - ventral and dorsal cochlear nuclei), where the data stream is partitioned and sent to several other brainstem integrative centres for further processing (e.g. for hearing - superior olivary nuclei, reticular nuclei of the medulla, nuclei of the medial longitudinal lemniscus and inferior colliculi), which then all send their integrative flows to thalamic nuclei of the diencephalon (e.g. for hearing - medial geniculate nuclei), which in turn relays this largely single neural data stream to the relevant area of the neocortex (e.g. for hearing - the primary auditory cortices). (See Figure 6)

It is only at the level of the neocortex that this highly integrated neural flow creates a conscious perception. This perception is then immediately sent to several areas for further processing and to be integrated into our overall awareness of what is happening around us. Thus, as soon as the conscious perception is formed, it is immediately sent into the hippocampus to become part of our now-time awareness, which imports referents from long-term memory to define the perception held in the hippocampal circuits, and is then usually imported into our working memory of the dorsolateral frontal cortices where we can "think" about it.