Symptom Check List and Functional Specialization in the Brain

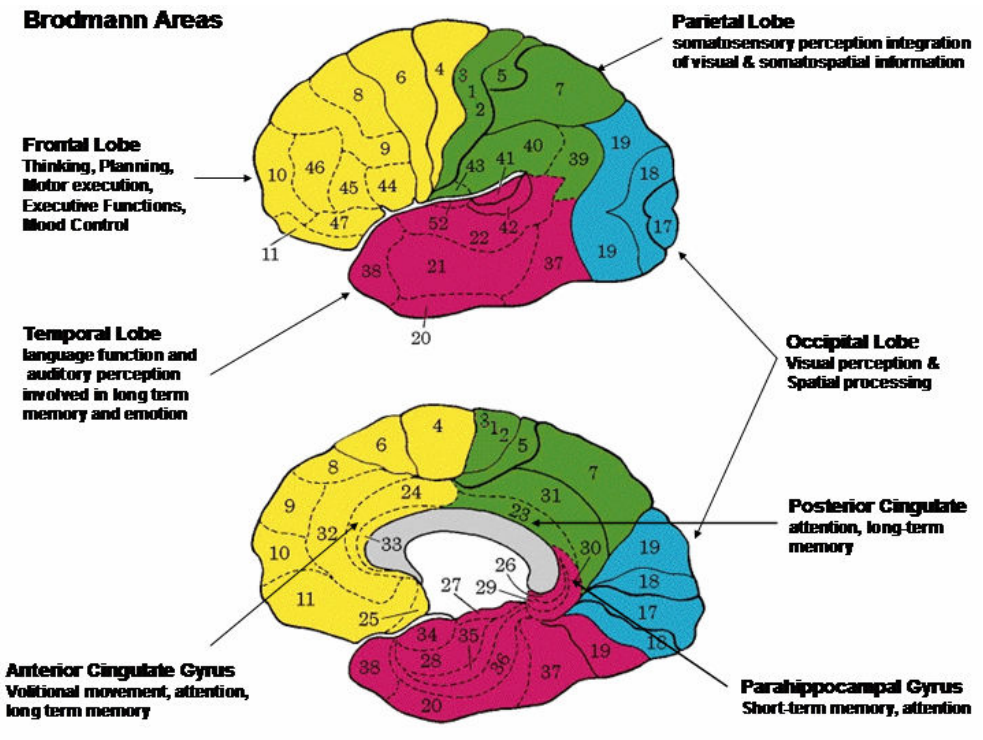
Link Between Structure and Function

Brodmann Areas

Korbinian Brodmann received a medical degree in Berlin in 1895 and then pursued studies in the histology and cytology of the human cerebral cortex. This was an exciting time in the history of neuroscience due to the fresh biological linkages between structure and function advanced by the invention of the microscope and the new mathematics of physics. The surge of powerful scientific tools and precise measurement gave rise to the first Brain Research Institutes in Germany with the goal of localization of function to particular regions of the brain. There was no separation between psychiatry and neurology and the linkage between structure and function was a driving force of all biological science. This linkage included understanding how the 'soul' of the human brain operates to give rise to consciousness, feelings, thoughts and action. In the late 1800s Nissl and Golgi stains, for the first time, were illuminating the cellular structure of the cerebral cortex demonstrating layering of pyramidal neurons and a differentiated cytoarchitecture of the cortex including clusters of different types of cortical neurons in different regions.

Brodmann integrated ideas on phylogenetic and ontogenetic influences with his theories of cortical structure, function and pathology. Brodmann argued that if a cortical region exhibits similar cytoarchitecture and layering then, based on structure and function, there must be a shared function of the neurons in that region. Further, he argued that regions with different cytoarchitecture exhibit different functions (Brodmann, 1909). He compared localization in the human cortex with a number of other mammals, including primates, rodents and marsupials. In man, he distinguished 47 areas, each carrying an individual number, and some being further subdivided. The subdivisions ranged in surface area from about 1 square cm to 6 square centimeters and are the same Brodmann areas used today and the original Brodmann text can be downloaded at: http://www.appliedneuroscience.com/Brodmann-pdf.pdf

A remarkable historical fact is that Brodmann's areas or subdivisions are a mainstay of modern neuroscience and neuropsychology in the 21st century. For example, the Human Brain Project uses Brodmann areas as one of the basic nomenclatures of cortical structure and function with co-registration between different imaging modalities such as MRI, PET, fMRI and EEG/MEG that also rely upon Brodmann areas (Thatcher et al, 1994; 1995). The use of Brodmann areas with coordinates, defined in the Talairach and MNI atlas, provides a common coordinate system for all modern neuroimaging and provides an immediate co-registration of structure-function measures in EEG/MEG based on the science of the MRI, fMRI, DSI, PET and SPECT.



The Human Electroencephalogram: What is the spatial extent of Function?

What is important is not that 100% of 10 - 100 million neurons must be simultaneously active to be detected, but rather that approximately 50% of the electrical potential at the scalp surface comes from generators directly underneath the recording electrode (1 cm) and 95% comes from sources 5 – 6 cm distant no matter what the amplitude. It is not necessary for 10 million neurons to be simultaneously active to generate an electrical potential at the scalp surface, rather the amplitude (A) of the EEG varies as a function of the number of synchronous synaptic generators (M) times the square root of the number of asynchronous generators (N) (Nunez, 1981; 1994),

$$A = M\sqrt{N}$$

thus waxing and waning of the EEG is due to the ratio of M to N from moment to moment and this proportion is oscillatory in time with abrupt phase shifts and couplings between local and distant neurons.

The highest amplitude of the EEG is produced by synapses located on radially oriented dendrites and soma in the crest of cortical gyri underneath each scalp electrode. Sources in the walls of gyri (tangential) and in the sulcus (radial) also contribute but less so and for MEG only tangential sources on the walls of the gyri contribute to the MEG signal. This is one of the major differences between EEG and MEG. Steriade (2006) reviews 40 years of science and shows the importance of cortico-thalamic, thalamo-cortical and cortico-cortical loops involved in the coordination of EEG oscillations. In local domains, Dwyer et al (2010) and Hutcheon (1999) show that EEG is produced by

circulation or oscillations in excitatory loops between the upper and lower layers of the cortex with electrical and chemical synapses regulating the local and horizontal synchrony of pyramidal neurons across centimeters of distance.

Studies by many clinical qEEG scientists, for example, Walter Freeman, Valeria, John, Buzsaki and many others in the 1980s and 1990s discovered a fundamental relationship between the probability of action potential firing and the Local Field Potentials (LFPs) in the general vicinity of neurons. Neurons fire only 50% of the time because they are most excitable on the depolarizing phase of the LFP where activation of neurons occurs when they are phase shifted "In-Phase" with respect to the LFP and are suppressed when phase shifted to "Anti-Phase" with respect to the LFP. Thus, at any instant of time the amplitude and frequency of the EEG constitutes the summation of LFPs and the EEG is a moment-to-moment measure of the excitability of action potential firing like gates opening and closing on the half cycle. The amplitude of the EEG is directly proportional to the probability distribution of synchronous action potentials. This does not mean that action potentials themselves contribute to the scalp EEG but rather that the probability of action potential firing is related to the depolarizing phase of the EEG recorded at any scalp location.

Phase differences between electrical sources located in different Brodmann areas are easily measured in the human electroencephalogram. Cross-frequency phase synchrony, for example, theta (4-7 Hz) rhythms phase locked to beta (13 – 20 Hz) or delta and alpha or alpha and gamma, etc. is an important information processing method in the human brain. Coherence is a measure of the temporal stability of phase differences between different scalp locations and different functional systems are phase locked across brain regions for varying periods of time (e.g, 100 msec to 500 msec) and then released by a subsequent phase shift that recruits a different set of neurons to then become phase locked. Phase shifts are correlated with reduced EEG amplitude and 'asynchrony' while phase lock is correlated with increased rhythm amplitude and 'synchrony'.

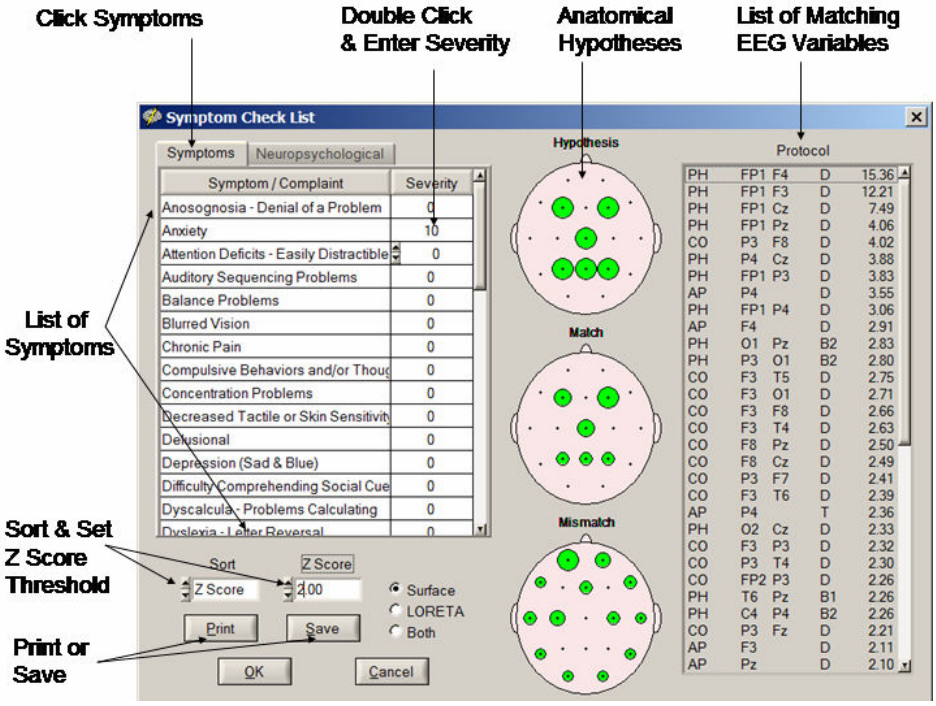
As previously mentioned, Brodmann (1909) argued from a "structure-function" point of view that if the cytoarchitecture is the same then function must also be the same and on this basis he created his sub-divisions. Thus, based on Brodmann areas then the EEG is measuring function that is macro in scale and spatially limited to Brodmann areas (1 to 6 cm). Importantly, neurons rapidly synchronize and the spatial extent of global or macro function is about 1 cm to 6 cm if fMRI or PET or any other imaging modality is used. This indicates that synchronization of large groups of pyramidal neurons is itself a fundamental property of information processing in the human brain. Another important fact is that the axonal connections of the human cortex are arranged in six basic clusters referred to as 'Modules' as measured by Diffusion Imaging Spectroscopy (Hagmann et al, 2008). The synaptic density of connections is spatially heterogeneous and clustered with phase shift and phase lock between clusters or Modules providing the 'vitality' or temporal dynamics of the EEG as determined in highly stable loops in thalmo-cortical, cortico-thalamic and cortico-cortical connections. Pacemakers and natural resonance of pyramidal neurons and loops give rise to stable rhythms that operate like a "Carrier Wave" in which phase shift of neurons to "In-Phase" with respect to the local field potential (LFP) are orchestrated by phase shift and phase lock mechanisms that are easily measurable in real-time by standard quantitative EEG methods (Wang, 2010).

Symptom Check List Linkage Between Structure and Function: Surface EEG

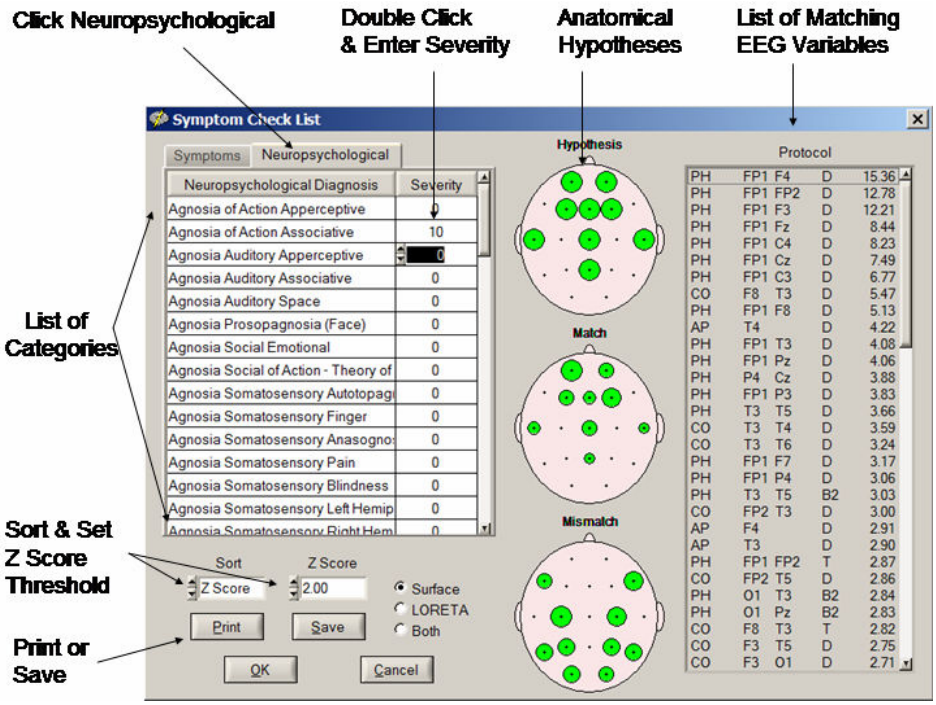
Based on these facts and other supporting science a "Symptom Check List" (SCL) was developed with the goal of linking structure to function based on the spatial overlap of functional and clinical studies using fMRI, PET and EEG/MEG as well as the clinical neurological science of strokes, tumors and lesions. The specific literature citations and sources for the SCL are provided in Appendix A.

To activate the symptom check list one must download, install and launch the free Neuroguide from:
http://www.appliedneuroscience.com/ContactDownloadDemo.htm then click File > Open > Lexicor > Lexicor NRS24 and then type in age 55, select the eyes closed condition and click OK. Then click Edit > Select All, then click Report > Symptom Check List Protocol Creation to activate the Symptom Check list window and scroll down to "Spatial Perception Problems" and double click on the Severity and type 10 because the EEG is from a patient with right parietal lobe damage and has symptoms of spatial neglect and left sided paresis. The SCL generates a scalp model of likely brain regions expected to be related to the patient's symptoms in the "Hypotheses" head. The "Match" head shows the EEG Z scores in the patient's record that match the hypothesized locations and the "Mismatch" head shows the significant EEG measures that failed to match the hypothesized locations. It is possible that the mismatches represent "compensatory" processes and in this way one can focus on the most likely "weak" brain systems linked to the patient's symptoms as discussed by Alexander Luria (Luria, 1973). The "Protocol" on the right is a listing of measures that matched the hypothesized locations and can be saved in a file and are automatically available to perform EEG biofeedback. For further details, click Help > NeuroGuide Help and then open the Chapter on Neurofeedback and Symptom Check list inside of the Neuroguide Manual. Below is an example of the SCL panel with the "Symptoms" selected. The second screen is the SCL panel with "Neuropsychological Diagnoses" selected. A listing of these two different symptom check lists is available in Appendix – A and B.

Behavioral Symptom Check List



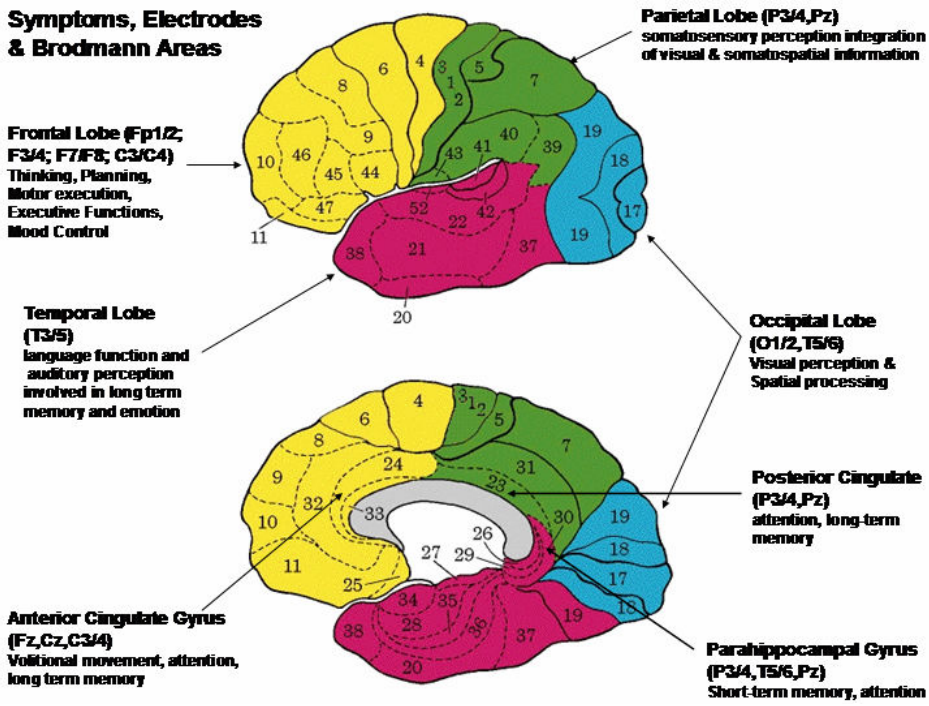
Neuropsychology Category Check List



Symptom Check List Linkage Between Structure and Function: LORETA

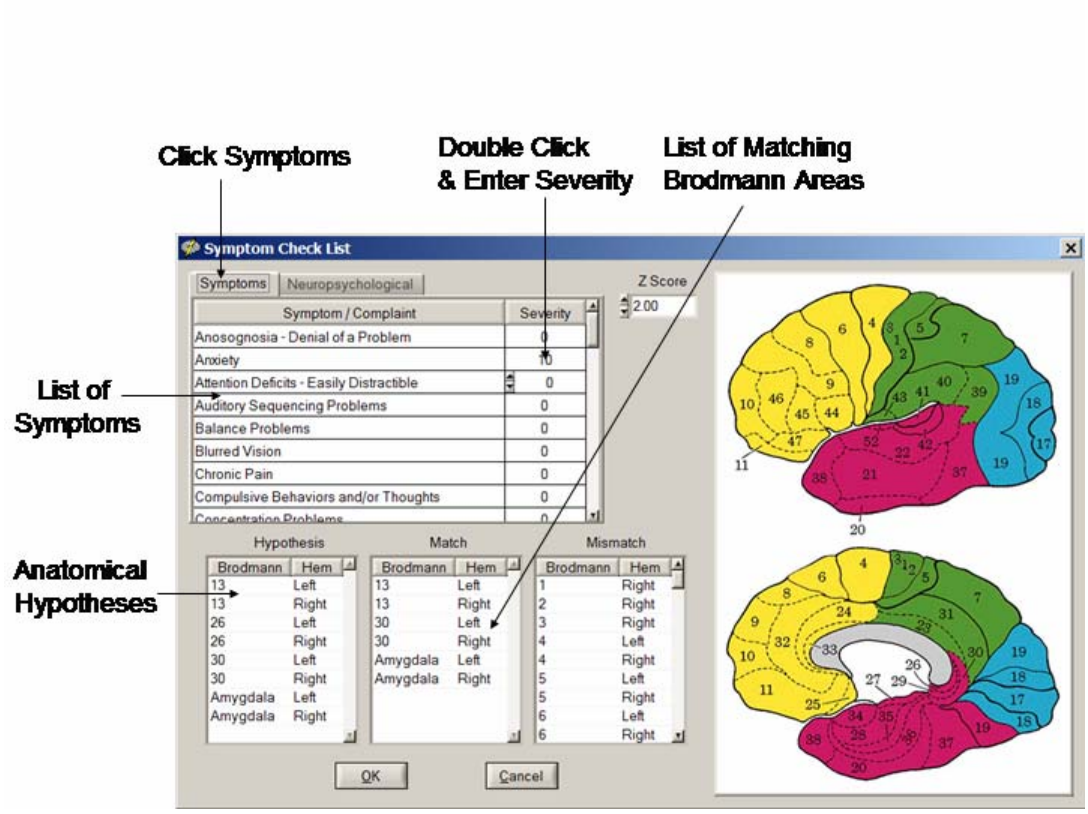
The same scientific literature review of the fMRI, PET and EEG/MEG literature that links symptoms and neuropsychological diagnoses to functional specialization in the brain was used to provide a linkage to the 3-dimensional sources of the scalp EEG. We use Low Resolution Electromagnetic Tomographic Analysis or "LOERTA" that has a 7 mm cubic spatial resolution and adequately measures all of the Brodmann areas as evidenced by 795 peer reviewed journal articles that can be read at:

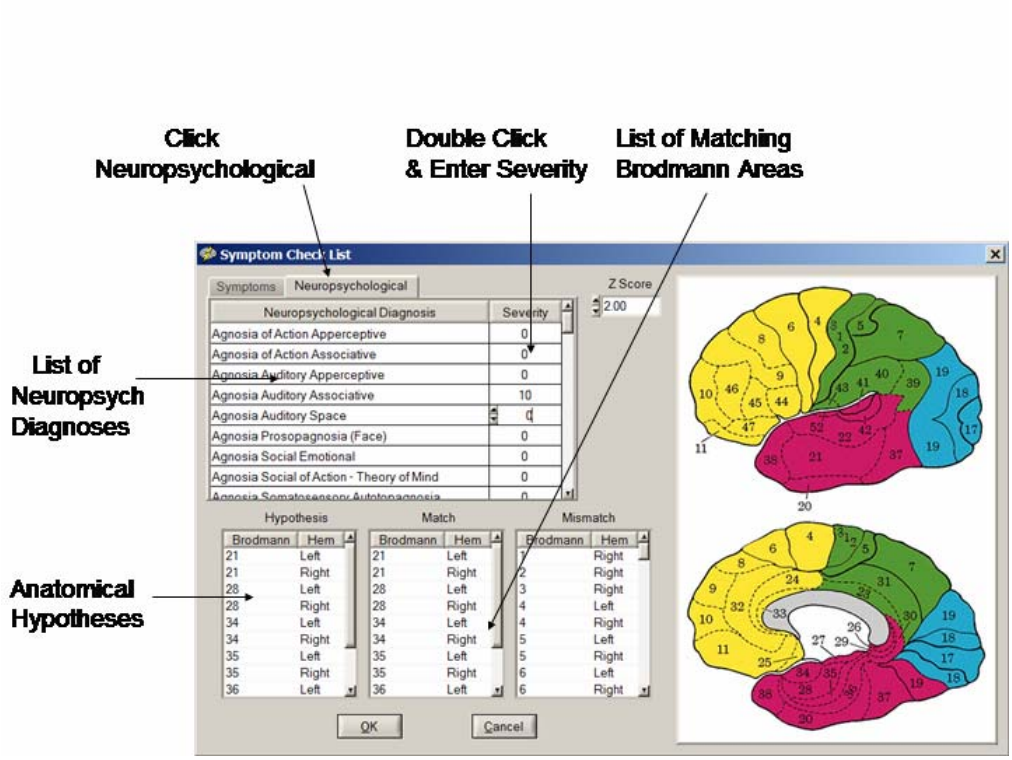
http://www.appliedneuroscience.com/LORETA%20publications.pdf



To activate the LORETA symptom check list one must download, install and launch the free Neuroguide from: http://www.appliedneuroscience.com/ContactDownloadDemo.htm then click File > Open > Lexicor > Lexicor NRS24 and then type in age 55, select the eyes closed condition and click OK. Then click Edit > Select All, then click Report > Create Symptom Check List Match and then save the .scl file in a folder. Then click Collection > Setup & Monitor and then click OK. Click Collection > Neurofeedback > LORETA Neurofeeback. Click Symptom Check List in the settings panel and open the .scl file that was previously saved. Scroll down to "Spatial Perception Problems" and double click on the Severity and type 10 because the EEG is from a patient with right parietal lobe damage and has symptoms of spatial neglect and left sided paresis. The SCL generates a list of likely Brodmann areas expected to be related to the patient's symptoms in the "Hypotheses" in the lower right list. The "Match" list shows the statistically significant Brodmann areas in the patient's record that match the hypothesized locations and the "Mismatch" shows the Brodmann areas that failed to match the hypothesized locations. It is possible that the mismatches represent "compensatory" processes and in this way

one can focus on the most likely "weak" brain systems linked to the patient's symptoms as discussed by Alexander Luria (Luria, 1973). Click OK to return to the settings panel and view the Brodmann areas and regions of interest that were automatically generated by the symptom check list match. For further details, click Help > NeuroGuide Help and then open the Chapter on Neurofeedback and Symptom Check list inside of the Neuroguide Manual. Below is an example of the SCL panel with the "Symptoms" selected and the Brodmann areas selected based on the match of symptoms and regions of the brain. The second screen is the SCL panel with "Neuropsychological Diagnoses" selected. A listing of these two different symptom check lists is available in Appendix – D.





Appendix A- Scientific Literature Used to Create the Symptom Check List

The literature that was relied upon is available in the National Library of Medicine's database at:
https://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed and at:
http://www.appliedneuroscience.com/Brain%20&%20Behavior.htm

The following textbooks were relied upon:

1- The Brain and Behavior: An Introduction to Behavioral Neuroanatomy, D.L. Clark, N.N. Boutros and M.F. Mendez, Cambridge University Press, 2010

2- Localization of Clinical Syndromes in Neuropsychology and Neuroscience. J.M. Tonkonogy and A.E. Puente, Springer Pub. Co, N.Y., 2009

3- Localization in Clinical Neurology. P.W. Brazis et al, Lippincott Williams & Wilkins, Philiadelphia, PA 2007

4- Brodmann's Localization in the Cerebral Cortex, Translated by L. J. Garey. Springer, London, 1994

5- Principles of Behavioral and Cognitive Neurology, 2nd ed., M.-Marsel Mesulam. Oxford University Press, 2000

6- The Working Brain: An Introduction to Neuropsychology, A. R. Luria, Penguin Press, 1973

References in Text:

Nunez, P. (1981). Electrical Fields of the Brain. Oxford University Press, New York.

Nunez, P. (1994). Neocortical Dynamics and Human EEG Rhythms, Oxford University Press, New York.

Appendix B- Surface EEG List of Symptoms

Symptoms

Anosognosia - Denial of a Problem
Anxiety
Attention Deficits - Easily Distractible
Auditory Sequencing Problems
Balance Problems
Blurred Vision
Chronic Pain
Compulsive Behaviors and/or Thoughts
Concentration Problems
Decreased Tactile or Skin Sensitivity
Delusional
Depression (Sad & Blue)
Difficulty Comprehending Social Cues
Dyscalcula - Problems Calculating
Dyslexia - Letter Reversal
Executive Function Problems
Face Recognition Problems
Failure to Initiate Actions
Hyperactive and/or Agitation
Impulsive Behaviors
Insensitive to Others Emotional Expressions
Insensitive to Other's Feelings
Low Motivation
Low Threshold for Anger & Loss of Control
Migrane Headaches
Mood Swings
Multi-Tasking Problems
Obsessive Thoughts about Self
Obsessive Thoughts and/or Hyper Focused
Oppositional Defiant Conduct
Orientation in Space Problems
Perception of Letters Problems

Poor Judgement
Poor Skilled Motor Movements
Poor Social Skills
Receptive Language Problems
Recognizing Objects by Touch Problems
Self-Esteem Problems
Sequential Planning Problems
Short-Term Memory Problems
Slow Reader
Slowness of Thought - Easily Confused
Spatial Perception Problems
Speech Articulation Problems
Symptoms of Fibromyalgia
Word Finding Problems

Appendix C – Surface EEG List of Links to the Neuropsychological Literature

Neuropsychological Diagnoses
Agnosia of Action Apperceptive
Agnosia of Action Associative
Agnosia Auditory Apperceptive
Agnosia Auditory Associative
Agnosia Auditory Space
Agnosia Prosopagnosia (Face)
Agnosia Social Emotional
Agnosia Social of Action - Theory of Mind
Agnosia Somatosensory Autotopagnosia
Agnosia Somatosensory Finger
Agnosia Somatosensory Anasognosia of Aphasia
Agnosia Somatosensory Pain
Agnosia Somatosensory Blindness
Agnosia Somatosensory Left Hemiplegia
Agnosia Somatosensory Right Hemiplegia
Agnosia Somatosensory Mental Illness
Agnosia Tactile Apperceptive
Agnosia Tactile Associative
Agnosia Visual Topographic
Agnosia Visual Acronomatopsia
Agnosia Visual Anomia Color
Amnestic Disorder Anterotrograde
Amnestic Disorder Working Memory
Amnestic Disorder Transient Global amnesia (TGA)
Amnestic Disorder Reduplicative Paramnesia
Aphasia Anterior Broca's
Aphasia Anterior Transcortical Motor
Aphasia Anterior Articulation
Aphasia Posterior Wernicke's

Aphasia Posterior Transcortical Sensory
Aphasia Posterior Conduction
Aphasia Posterior Word Deafness
Apraxia Motor Constructional
Apraxia Motor Dressing
Apraxia Motor Gate, Trunk
Apraxia Motor Ideational
Apraxia Motor Ideomotor
Apraxia Motor Limbkinetic
Apraxia Motor Oral
Apraxia Social Disorganization of social actions
Apraxia Visual Topographic
Aspontaneity Abulia and/or Apathy
Aspontaneity Akinetic Mutism
Aspontaneity Catonia
Attenional Disturbances Balint's Syndrome
Attenional Disturbances Neglect
Attenional Disturbances Excessive Shifting of Attention
Delusions Capgras Syndrome
Delusions Grandiose
Delusions Guilt
Delusions Persceutory
Disturbances of Self-Image Depersonalization
Disturbances of Self-Image Derealization & Self-Awareness
Disturbances Visual-Spatial Disorientation
Disturbances Visual-Spatial Topograhpic Agnosia
Disturbances Visual-Spatial Topograhpic Apraxia
Hallucinations Auditory Elementary
Hallucinations Auditory Complex
Hallucinations Visual Elementary
Hallucinations Visual Complex
Mood Disturbances Agression, Rage
Mood Disturbances Mania
Mood Disturbances Panic
Mood Disturbances Obsessive Compulsive
Mood Disturbances Secondary Depression
Loss of Visual Imagery Objects
Loss of Visual Imagery Space

Appendix D –Brodmann Areas and Structure-Function

Under Construction based on Links to the National Library of Medicine Database of EEG/MEG and/or fMRI and/or PET/SPECT studies.

Links are not compete and are adapted from David Kaiser
http://www.skiltopo.com/B1/functions.htm#B1123L

Brodmann Area 1 - Post Central Gyrus (Somatosensory Unimodal Cortex - Mesulam, 1999)

- Homunculus (1)
- Localize pain (1, 2, 3, 4)
- Localize touch and vibration (1, 2, 3, 4, 5, 6, 7)
- Localize temperature (1, 2, 3, 4)
- Sense of fingers (1, 2, 3)
- Sense of body (1)
- Move hands (1, 2)
- Move mouth and tongue (1, 2)
- Swallowing (1, 2, 3)
- Anticipate pain (1)
- Anticipate tickling (1)
- Mirror neurons (1, 2, 3)

Brodmann Area 2 - Post Central Gyrus (Somatosensory Unimodal Cortex - Mesulam, 1999)

- Homunculus (1)
- Agraphesthesia

Brodmann Area 3 - Post Central Gyrus (Somatosensory Unimodal Cortex - Mesulam, 1999)

- Homunculus (1)

Brodmann Area 4 - Pre Central Gyrus (Somatosensory Unimodal Cortex - Mesulam, 1999)

- Muscle sequences (1, 2, 3)
- Move hands(1, 2, 3, 4, 5)
- Limbs (1, 2, 3)
- Sense of our fingers (1, 2, 3)
- Softness and vibration (1, 2, 3)
- Move tongue and mouth (1, 2, 3, 4, 5)
- Actions, Touches (1, 2)
- Swallow (1, 2, 3, 4)
- Voluntary breathing (1, 2, 3, 4)
- Muscle imagery (1, 2, 3, 4)
- Blinking (1, 2)
- Eye movements (1, 2, 3)
- Bicycling (1)

- Tactile (1)

Brodmann Area 5 - Parietal Lobe Economo-Koskinas area PE **(Somatosensory Unimodal Cortex - Mesulam, 1999)**

- Ordered & chaotic pattern perception (1)
- Spatial imaging in deductive reasoning (1)
- Imagery of movement (1, 2, 3)
- Tool-use and gestures (1, 2)
- Imitation learning (1)
- Mirror neurons for movement (1)
- Bimanual coordination (1)
- Saccades (1, 2, 3)
- Working memory (1, 2, 3, 4)
- What & where of touch (1)
- Localizing pain (1)
- Motor attention (1, 2)
- Imitating finger movements (1)
- Spatial mnemonic processing (1)

Brodmann Area 6 - Frontal Lobe Economo-Koskinas area FB **(Motor Association Cortex Mesulam, 1999)**

- Sequential movements (1, 2, 3)
- Object recognition (1)
- Sensorimotor willed actions (1)
- Word repetition (1)
- Attention to visual motion (1)
- Action Knowledge (1)
- Feigned memory impairment (1)
- Action learning (1, 2, 3, 4, 5)
- Start moves (1, 2)
- Muscle imagery (1, 2, 3)
- Imagined moves (1, 2, 3, 4)
- Voluntary breathing (1, 2, 3)
- Saccades (1, 2, 3)
- Laughter, smiling (1)
- Words (a, b, c)
- Language (a, b)
- Language switching (a)
- Understand speech (a, b)
- Lipreading (a)
- Human voices (a)

- Acoustic rhythms (a, b)
- Generate melodies (a)
- Working memory (1, 2, 3, 4)
- Mnemonics (1, 2)
- Episodes (1)
- Topographic memory (1)
- Locations (1, 2, 3)
- Self-reflection (1)
- Update locations (1)
- Eye guidance (1)
- Observe Actions (Mirror neurons) (1, 2, 3, 4)
- Solve novelty (1, 2)
- Orchestrate (1)
- Calculation (1, 2)

- Speech muscles (1, 2) - Left Hemisphere
- Novel words (1) - Left Hemisphere
- Phonemes (1) - Left Hemisphere
- Object names (1, 2) - Left Hemisphere
- Deduction (1, 2) - Left Hemisphere
- Inhibit response (1) - Left Hemisphere

- Sequential moves (1) - Right Hemisphere
- Monitor errors (1) - Right Hemisphere
- Strong odors (1) - Right Hemisphere
- Same or different (1) - Right Hemisphere

Brodmann Area 7 - Supramarginal Gyrus Somatosensory Association Cortex

- Economic decisions (1)
- Conscious awareness of visual-spatial events (1) - Bilateral
- Goals (1) - Bilateral
- Activation at rest - Default Brain (1) - Bilateral
- Pain (1)
- Recall episodes (1) - Left Hemisphere
- Tool use (1) - Left Hemisphere
- Handwriting and spelling (1) - Left Hemisphere
- Shift attention (1) - Left Hemisphere
- Language (1) - Left Hemisphere
- Literalness (1) - Left Hemisphere
- Imageability (1) - Left Hemisphere

- Personal space (1, 2) - Right Hemisphere

- Estimate (1) - Right Hemisphere
- Meta-emotions (1) - Right Hemisphere
- Visuospatial attention (1) - Right Hemisphere
- Mental rotation (1, 2) - Right Hemisphere
- Stereopsis (1, 2) - Right Hemisphere
- Line bisection judgments (1) - Right Hemisphere

Brodmann Area 8 - Pre-Frontal Cortex **(Association Cortex - Mesulam, 1999)**

- Muscle learning (1, 2, 3, 4)
- Muscle imagery (1, 2)
- Muscle control (1, 2)
- Saccades (1, 2)
- Laughter, smiling (1)
- Executive control (1, 2, 3, 4)
- Planning (1, 2)
- Speech programs (1)
- Language processing (1)
- Language translation (1)
- Generating sentences (1)
- Lipreading (1)
- Working memory (1, 2, 3)
- Perceptual priming (1)
- Topographic memory (1)
- Visuospatial and visuomotor attention (1)
- Retrieval of words (1) - Right Hemisphere
- Sequence learning (1, 2)
- Proprioceptive stimulation (1, 2, 3)
- Pain anticipation (1)
- Processing uncertainty (1)
- Inductive reasoning (1)
- Calculation (1, 2)
- Auditory imagery (1)
- Memory retrieval (1, 2)
- Nonconscious saccades (1)

Brodmann Area 9 - Pre-Frontal Cortex Economo-Koskinas area FD **(Frontal Association Cortex)**

- Male and female sexual arousal (1)
- Word identification (1)
- Economic decisions (1)

- Lie detection (1)
- Viewing of unpleasant pictures (1)
- Short-term memory (1)
- Recency (1)
- Orchestrate behavior (1)
- Verbal fluency (1)
- Word-stem completion (1)
- Error detection (1)
- Human voices (1) Infer (1, 2,)
- Intentions (1)
- Action authorship (1)
- Energize (1)

- Generate sentences (1)
- Inference (1, 2)
- Idioms (1, 2, 3)
- Categorization (1)

- Suppress sadness (1)
- Working memory (1, 2, 3)
- Spatial memory (1, 2)
- Recognition (1, 2, 3)
- Recall (1, 2, 3)
- Recognize emotions (1)
- Planning (1)
- Calculation (1, 2)
- Familiar odors (1)

Brodmann Area 10 - Pre-Frontal Cortex Economo-Koskinas area FD **(Frontal Association Cortex)**

- Working memory (1, 2, 3) - Bilateral
- Spatial memory (1, 2) - Bilateral
- Recognition (1, 2, 3) - Bilateral
- Recall (1, 2, 3) - Bilateral
- Intentional forgetting (1) - Bilateral
- Nonspeech sounds (1) - Bilateral
- Recognize emotions (1, 2) - Bilateral
- Calculation (1, 2) - Bilateral
- Pain (1) - Bilateral
- Joint attention (1) - Bilateral

- Syntax(1) - Left Hemisphere
- Metaphor (1) - Left Hemisphere

- Lexical (1) - Left Hemisphere
- Verbs (1) - Left Hemisphere

- Self-reflection (1) - Right
- Self-appraise (1) - Right
- Economic decisions (1) - Right
- Inferences during reading (1) - Right Hemisphere
- Recall episode (1) - Right Hemisphere
- Nonspeech sounds (1) - Right Hemisphere
- Familiar odors (1) - Right Hemisphere
- Reward vs conflict (1) - Right Hemisphere
- Risk vs benefit (1) - Right Hemisphere

Brodmann Area 11 - Higher Order Assocation and Orbital Frontal Association Cortex (Paralimbic Posterior - Mesulam, 1999)

- Olfaction (1, 2) - Bilateral
- Nonspeech sounds (1) - Bilateral
- Reward vs conflict (1, 2) - Bilateral

- Unexpected outcomes (1) - Left Hemisphere
- Idioms (1) - Left Hemisphere
- Face-name (1) - Left Hemisphere

- Self-calm (1) - Right Hemisphere
- Odors (1) - Right Hemisphere
- Encoding of words (1) - Right Hemisphere

Brodmann Area 12 - Frontal Lobe (12/47 Economo-Koskinas area FD (Paralimbic & Association Cortex - Mesulam, 1999)

- Hypothesis generation (1)
- Same different discrimination (1)
- Decision making (1)
- Affective value of reinforcers (1)
- Sensitivity to reward and punishment (1)
- Expectation (1)

Brodmann Area 13 - Insular Cortex Economo-Koskinas area FF (Paralimbic Cortex - Mesulam, 1999)

- Dissociated PTSD (1)
  - Maternal and romantic love (1)
  - Seeing or making a smile (1)

- Interoceptive Body Status pain localization (1, 2, 3, 4)
- Attend to happy voices (1)
- Hearing pleasant music (1)
- Reward fluctuation (1)
- Pain processing (1, 2, 3, 4, 5)
- Feeling joyful (1)
- Female self reported orgasm ratings (1)
- Mothers viewing photos of their own child (1)
- Mirror movement neurons (1, 2)
- Female sexual arousal to erotica (1)
- Retrieval of words (1)
- Heat (1, 2)
- Touch (1, 2)
- Vibration (1)
- Vestibulation (1)
- Olfaction and taste (1, 2, 3)
- Verbal memory (1, 2, 3)
- Action planning (1)
- Swallowing (1)
- Categorization (1, 2)
- Error awareness (1)
- Motivated reasoning (1)
- Risk-taking (1)
- Emotional inhibition (1)
- Express fear or disgust (1, 2, 3)
- Vocal pitch (1)
- Humor appreciation (1)
- Phonemes (1)

Brodmann Area 14 - only present in monkeys

Brodmann Area 15 - only present in monkeys

Brodmann Area 16 - only present in monkeys

Brodmann Area 17 - Occipital Cortex Economo-Kosinas area OB **(Primary Visual Unimodal Cortex - Mesulam, 1999)**

- Light intensity (1, 2, 3, 4)
- Orderly visual patterns (1, 2)
- Contours (1, 2, 3)

- Colors (1, 2, 3)
- Spatial orientation (1, 2, 3)
- Motion (1)
- Visual attention (1, 2, 3, 4)
- Visual priming (1, 2, 3)
- Help saccade eyes (1)

Brodmann Area 18 - Occipital Cortex Economo-Kosinas area OB (Primary Visual Unimodal Cortex - Mesulam, 1999)

- Light intensity (1, 2)
- Motion (1)
- Orderly visual patterns (1, 2)
- Understand finger gestures (1)
- Monitor color and shape (1, 2)
- Visual priming (1, 2)
- Face-name (1)
- Help saccade eyes (1)

Brodmann Area 19 - Occipital Cortex Economo-Kosinas area OA (Primary Visual Unimodal Cortex - Mesulam, 1999)

- Conscious awareness of visual-spatial events (1) - Bilateral
- Dissociated PTSD (1)
- Light intensity (1, 2) - Bilateral

- Reflective self-awareness (1, 2)
  - Visual patterns (1, 2) - Bilateral
  - Motion (1, 2, 3, 4) - Bilateral
  - Understand finger gestures (1) - Bilateral
  - Activation at rest - Default Brain (1)
  - Sign language (1)
  - Monitor color and shape (1)
  - Feature-based attention (1)
  - Orientation-selective attention (1)
  - Visual priming (1) - Bilateral
  - Visual memory recognition (1)
  - Spatial working memory (1)
  - Phonological demands (1)
  - Saccades (1)
  - Visual imagery (1, 2)
  - Inferential reasoning (1)

  - Animacy (1)
  - Visuospatial challenge (1)

- Motion (1)
- Visuo-spatial (1, 2)
- Visual priming (1)
- Visual recognition (1)
- Spatial working memory (1)
- Face-name (1)
- Saccades (1)
- Visual imagery (1)

Brodmann Area 20 - Temporal Lobe (Auditory Association Cortex - Mesulam, 1999)

- Visual fixation (1) - Bilateral
- Identify intention (1) - Bilateral

- Suspicion (1) - Left Hemisphere
- Truth telling (1) - Left Hemisphere
- Process latter parts of sentences (1) - Left Hemisphere
- Visual categorization (1) - Left Hemisphere
- Lexical categories (1, 2) - Left Hemisphere
- Metaphor comprehension (1) - Left Hemisphere
- Language comprehension and production (1) - Left Hemisphere
- Monitor speech (1) - Left Hemisphere

- Category ambiguity (1) - Right Hemisphere
- Perceptual closure (1) - Right Hemisphere
- Working memory (1) - Right Hemisphere

Brodmann Area 21 - Temporal Lobe Auditory Economo-Kosinas area TE (Unimodal Cortex - Mesulam, 1999)

- Categorization (1, 2, 3) - Bilateral
- Observe motion (1) - Bilateral
- Complex sounds (1) - Bilateral
- Attribute intention (1) - Bilateral

- Verbal associated novelty (1) - Left Hemisphere
- Monitor text and speech (1, 2) - Left Hemisphere
- Sentence generation (1) - Left Hemisphere
- Word generation (1) - Left Hemisphere
- Deductive reasoning (1, 2) - Left Hemisphere

- Prosody (1, 2) - Right Hemisphere

Brodmann Area 22 - Temporal Lobe Auditory Economo-Kosinas area TA **(Auditory Association Cortex - Mesulam, 1999)**

- Sentence generation (1)
- Attend to happy and angry voices (1)
- Word generation (1)
- Monitor speech (1)
- Repeating words (1)
- Complex sounds (1)
- Deductive reasoning (1)
-
- Auditory language (1, 2)
- Categorization (1, 2)
- Learning tone-based 2nd language (1)

- Affective prosody (1)
- Nonverbal sounds (2)
- Previous eye movements (1)

Brodmann Area 23 - Posterior Cingulate Gyrus Economo-Koskinas area LC **(Paralimbic Cortex - Mesulam, 1999)**

- Activation at rest - Default Brain (1)
- Emotional words (1)
- Action memory (1, 2, 3)
- Episodic retrieval (1)
- Recall (1)
- Resolve proactive interference (1)
- Learn complex procedure (1)
- Finger movements (1)
- Temperature processing (1)
- Visual discrimination (1)
- Evaluative judgment (1)
- Self/other distinction (1)
- Classical conditioning (1)

- Lexical categories (1, 2)
- Fear conditioning (1)
- Threatening words (1)
- Mood swings (1)
-
- Cortical hub in infancy (1)
- Disconnects with trauma (1)

- Word and face encoding (1)

Brodmann Area 24 - Anterior Cingulate Gyrus (Paralimbic Cortex - Mesulam, 1999)

- Inhibition (1, 2, 3)
- Hearing happy music (1)
- Emotional cues (1, 2, 3)
- Muscle strategy (1, 2, 3)
- Muscle imagery (1)
- Vestibulation (1, 2, 3)
- Pain endurance (1, 2, 3)
- Working memory (1, 2, 3)
- Episodes (1)
- Prospective memory (1)
- Time-based memory (1)
- Visuospatial attention (1, 2, 3)
- Divided attention (1, 2)
- Auditory attention (1, 2)
- Mental timekeeping (1, 2, 3)
- Self-other overlap (1, 2)
- Familiar odors (1, 2, 3)
- Deductive reasoning (1, 2)
- Taste (1, 2)
- Sexual arousal to erotica (1, 2)

- Object-naming (1, 2)
- Verbal fluency (1)
- Induction (1)
- Remembering after delay (1)
- Multitasking (1)
- Worry (1)
- Mood swings (1)
-
- Sustain response set (1)
- Verbal initiation and suppression (1, 2)
- Memory retrieval (1, 2)
- Conflict monitor (1)
- Monitors consequences of actions (1)
- Suppress aggression (1)
- Mood swings (1)

Brodmann Area 25 - Anterior Cingulate Gyrus & Medial Prefrontal Cortex (Paralimbic Cortex; Mesulam, 1999)

- Moral reasoning (1)
- Evaluate emotional words (1, 2)

Brodmann Area 26 - Paralimbic Cortex (Mesulam, 1999)

- Verbal associated novelty (1)

Brodmann Area 27 - Parahippocampal Gyrus and Presubiculum - (Paralimbic Cortex - Mesulam, 1999)

- Pattern separation (overlapping representations are made less similar) (1, 2)
- Face memory (1, 2, 3)
- Picture memory (1, 2)
- Auditory memory (1)
- Emotions (1)
- Categories (1, 2, 3)
- Visual memory (1)
- Autobiographical memory (1, 2, 3)
- Olfactory and gustatory memory (1, 2, 3)
- Recognition, recall and retrieval (1, 2, 3)
- Procedural memory consolidation (1, 2, 3)
- Memory for novelty (1)
- Memory for negative stimuli (1, 2, 3)
- Face-based emotional perception (1, 2, 3)
- Erotica (1, 2)
- Experience stress (1)
- Congruent facial movements (mirror neurons) (1)
- Craving (1)
- Hunger (1)
- Embarassment (1)
- Spatial (1, 2, 3)
- Landmarks (1, 2, 3)
- Novelty (1)
- Past and future events (1, 2)
- Anticipating regret (1)
- Insight (1)
- Decision-making (1)

- Categorization (1, 2, 3,)

Brodmann Area 28 - Parahippocampal Gyrus, Uncus and Medial Temporal Lobe (Paralimbic Cortex - Mesulam, 1999)

- Deductive and probabilistic reasoning (1)
- Viewing of unpleasant pictures (1)
- Finer grain discriminantion (1)

Brodmann Area 29 - Posterior Cingulate & Superior Transverse Temporal Gyrus (Cortex Paralimbic Cortex -Mesulam, 1999)

- Novelty activation (1)
- Precautions awareness (1)
- Nonconscious formation of semantic associations (1)

Brodmann Area 30 - Posterior Cingulate & Cuneus (Paralimbic Cortex - Mesulam, 1999)

- Attending to speech (1)
- Listening to sentences (1)
- Activation to emotional words (1)
- Observation of actions by others (1)
- Activation at rest - Default Brain (1)
- Encoding of words (1)

Brodmann Area 31 - Posterior Cingulate Gyrus (Paralimbic Cortex - Mesulam, 1999)

- Self-evaluation (1)

Brodmann Area 32 - Anterior Cingulate Gyrus (Paralimbic Cortex - Mesulam, 1999)

- Visual uncertainty (1)
- Mood regulation (1)

- Sustain response set (1)
- Identify pain (1)
- Self-insight (1)
- Reward (1)
- Reappraise negative emotions (1)

Brodmann Area 33 - Anterior Cingulate Gyrus (Paralimbic Cortex - Mesulam, 1999)

- Hearing pleasant music (1)
- Female sexual arousal to erotica (1)

Brodmann Area 34 - Superior Temporal Gyrus & Subcallosal Gyrus-Entorhinal Area (Association Cortex)

- Processing words at different levels of specificity (1)
- Dissociated PTSD (1)

Brodmann Area 35 - Medial Temporal Lobe & Parahippocampal Gyrus (Association Cortex)

- Retrieval of words (1) - Right Hemisphere
- Acquisition of new memories
- Spatial mapping
- Novelty detection
- Short-term memory

Brodmann Area 36 - Medial Temporal Lobe & Parahippocampal Gyrus (Association Cortex)

- Retrieval of words (1) - Right Hemisphere
- Acquisition of new memories
- Spatial mapping
- Novelty detection
- Short-term memory

Brodmann Area 37 - Fusiform Gyrus & Middle Temporal Gyrus (Auditory-Visual Association Cortex - Mesulam, 1999)

- Episodes (1)
- Faces (1, 2, 3)
- Motion (1, 2, 3, 4)
- Visual fixation (1)
- Monitor color and shape (1, 2)
- Intentions (1)
- Drawing (1)
- Acquisition of new memories
- Spatial mapping
- Novelty detection

- Short-term memory
  - Categorization (1, 2, 3)
  - Word retrieval (1, 2, 3)
  - Sign language (1)
  - Metaphor (1)
  - Reading (1)
  - Face-name (1)
  - Deduction (1)
  - Numbers (1)

  - Familiarity judgments (1)
  - Face identity (1)

Brodmann Area 38 - Superior Temporal Gyrus and Middle Temporal Gyrus **(Paralimbic Cortex - Mesulam, 1999)**

- Dissociated PTSD (1)
  - Intentions (1, 2, 3)
  - Moral judgments (1, 2)
  - Experience emotions (1, 2, 3)
  - Identify emotions (1, 2, 3, 4, 5)
  - Attachment (1, 2)
  - Categorization (1, 2, 3)
  - Speech comprehension (1, 2)
  - Verbal disambiguation (1)
  - Stories (1, 2)
  - Aversive sounds (1)
  - Familiarity (1)
  - Humor (1, 2)

  - Self/other (1, 2, 3)
  - Attend to speech (1, 2)
  - Inferences (1)
  - Identify social emotions (1)
  - Aversiveness (1)
  - People's names (1)
  - Music appreciation (1)

  - Familiar voices (1)
  - Irony (1)
  - Faux pas (1)
  - Social behavior (1)

Brodmann Area 39 - Angular Gyrus & Inferior Parietal Lobe Association Cortex

- Reflective self-awareness (1, 2)
- Reading (1, 2)
- Spatial focusing (1)
- Theory of mind (1)
- Executive control (1)
- Action sequences (1)

- Sentence generation (1)
- Verbal creativity (1)
- Numerical facts (1, 2)
- Calculation (1, 2, 3, 4)

- See differently from others (1)
- Visuospatial (1)
- Sight reading of music (1)
- Self-relevance (1)
- Resolve surprise (1)
- Action authorship (1)
- Situational context (1)

Brodmann Area 40 - Inferior Parietal Lobe Angular Gyrus **(Somatosensory Association Cortex - Mesulam, 1999)**

- Attend to phonemes (1)
- Categorization (1)
- Economic decisions (1)
- Verbal creativity (1)
- Aversiveness (1, 2)
- Emotional working memory (1)
- Conscious recollection (1)
- Visual grasping (1)
- Gesture imitation (1, 2)
- Visuomotor planning (1)
- Repetitive passive movements (1)
- Intention/conflict detection (1)
- Somatosensory (1, 2)
- Motion (1, 2)
- Deduction (1, 2)
- Empathy (1)
- Music performance (1)
- Goals (1)

- Imitate novel actions (1)

- Calculation (integer computation) (1)
- Verbal creativity (1)

- Sentences (1)
- Self-reflection (1)
- Same or different (1)
- Sustain attention (1)

Brodmann Area 41 - Superior Transverse Temporal Gyrus - Auditory Primary (Unimodal Cortex - Mesulam, 1999)

- Auditory (1, 2, 3, 4, 5)
- Harmony (1, 2)
- Sound intensity (1, 2 ,3)
- Pitch (1, 2)
- Vowel segregation (1, 2)
- Auditory priming (1, 2)
- Auditory working memory (1)
- Visual speech perception (1, 2)

Brodmann Area 42 - Inferior Transverse Temporal Gyrus - Auditory Primary (Unimodal Cortex - Mesulam, 1999)

- Auditory (1, 2, 3, 4, 5)
- Harmony (1, 2)
- Sound intensity (1, 2 ,3)
- Pitch (1, 2)
- Vowel segregation (1, 2)
- Auditory priming (1, 2)
- Auditory working memory (1)
- Visual speech perception (1, 2)

Brodmann Area 43 - Gustatory Primary Cortex - Postcentral and Paracentral Lobule

- Tactile digit stimulation (1)
- Spoken language (1)

Brodmann Area 44 - Inferior Frontal and Extra Nuclear Gyrus of the Pre-Frontal Lobes

- Word generation (1)
- Unintelligible speech (1 ,2)
- Attend to speech (1)
- Syntactic working memory (1, 2)
- Working memory (1, 2, 3)
- Episodic memory (1)
- Declarative memory (1)
- Mirror other's movements (1, 2, 3)
- Speech programs (1)
- Tactile imagery (1)
- Goals (1)
- Word and face encoding (1)
- Arithmetics (1)
- Object manipulation (1)
- Familiar odors (1, 2)
- Music appreciation (1)

- Linguistic fluency (1, 2, 3)
- Grapheme-to-phoneme conversion (1, 2)
- Grammar (1, 2, 3, 4)
- Sequential sounds (1)
- Lexical inflection (1)
- Sentence comprehension (1)
- Imitate finger movements (1)
- Phonemes (1)

- Express emotions (1)
- Speech intonation (1, 2)
- Sentence comprehension (1)
- Inhibition actions (1)
- Generate melodies (1)
- Monitor actions (1)

Brodmann Area 45 - Inferior Frontal and Extra Nuclear Gyrus of the Pre-Frontal Lobes

- Semantic & phonological processing (1, 2, 3, 4, 5)
- Generate words (1, 2, 3, 4)
- Verbal fluency (1, 2, 3)
- Lexical search (1, 2)
- Phonemes (1, 2)

- Grammar (1)
- Categories (1, 2, 3)
- Sign language (1)
- Reason (1, 2)
- Metaphors (1, 2)
- Working memory (1, 2, 3)
- Episodic memory (1)
- Declarative memory (1)
- Digits (1)
- Mirror movement neurons (1, 2)
- Response inhibition (1, 2, 3)
- Mental rotation (1)
- Word and face encoding (1)
- Aesthetic and music appreciation (1, 2)
- Modulate emotions (1)

- Attend to speech (1)
- Lexical inflection (1)
- Generate melodic phrases (1)
- Familiar odors (1, 2)
- Retrieving categories (1)
- Phonemes (1)

- Affective prosody (1)
- Monitor actions (1)
- Timed behavior (1)

Brodmann Area 46 - Middle Frontal Gyrus

- Memory recognition (1, 2, 3)
- Working memory (1, 2, 3)
- Categorization (1, 2, 3)
- Monitor (1)
- Chewing (1)
- Drawing (1)
- Mirror neurons (1)
- Saccades (1)
- Intention/conflict detection (1)
- Music appreciation (1)
- Willfulness (1)
- Strategy (1)

- Verbal fluency (1, 2)
- Phonemes (1)
- Self-reflection (1)

Brodmann Area 47 - Inferior Frontal

- Dissociated PTSD (1)
  - Categorization (1, 2, 3, 4, 5)
  - Phoneme (1, 2)
  - Lexical inflection (1)
  - Attend to speech (1)
  - Working memory (1, 2)
  - Episodic memory (1)
  - Adverse emotional inhibition (1)
  - Temporal coherence (1)
  - Intention (1)
  - Deduction (1, 2)

  - Idioms (1)
  - Make inferences during reading (1)
  - Categorization (1, 2, 3)
  - Familiar odors (1, 2, 3)
  - Retrieve categories (1)
  - Music (1)

  - Affective prosody (1)
  - Behavioral inhibition (1, 2)
  - Manage conflict and reward (1)
  - Monitor actions (1)
  - Monitor unattended information (1)

Appendix E – Neuropsychological Linkages and Structure and Function for. This summary is based on the Clark et al, 2010 and Tonkonogy and A.E. Puente, 2009 and adapted from: http://www.dendrites.com/essays/cortical.htm

This summary description was chosen for its succinctness and the reader can verify that correctness of this section by reading the books:

The Brain and Behavior: An Introduction to Behavioral Neuroanatomy, D.L. Clark, N.N. Boutros and M.F. Mendez, Cambridge University Press, 2010

Localization of Clinical Syndromes in Neuropsychology and Neuroscience. J.M. Tonkonogy and A.E. Puente, Springer Pub. Co, N.Y., 2009

Principles of Behavioral and Cognitive Neurology, 2nd ed., M.-Marsel Mesulam. Oxford University Press, 2000

Cortical Layers

Plexiform or Molecular -- most superficial

- Mainly plexus of axons & dendrites (synapses)
- Axons to underlying cells
- Dendrites receive impulses from most peripheral areas Small Pyramidal Cells -- association neurons
- Not clearly separated from medium pyramidal cell layer
- Damage to this cell layer does not disrupt EEG

Medium Pyramidal Cells -- association neurons

Granule layer -- (sharply demarcated)

- Composed of multipolar granule cells Large Pyramidal Cells
- Betz cells in precentral gyrus Fusiform cell layer (efferent)

The part constituting the outer layers of the cerebral hemispheres is called the cerebral cortex. (cortex means "bark").

Layers I through VI vary in thickness in different cortical regions. Layer IV is most pronounced in the sensory projection cortex and layer V is most pronounced in the primary motor cortex (pre-central gyrus). The majority ot cortical neurons travel vertically through the layers as you will note in the diagram thus the concept of the (vertical unit organization). Layers II and III are composed on small and medium sized pyramidal cells and function primarily as association neurons. Layers I and 6b or 7 are derived from the same tissue as found in the mesencephalon.

General structure of the cortex has a thickness of 4 mm at the precentral gyrus to 1.5 mm at the visual center Layers: six layers -- two main zones

Outer--receptive--where incoming fibers synapse

Inner--efferent--cell bodies of fibers projecting to other areas

Structure -- vertical units (independent systems which communicate with each other via association of fibers). The functional unit is a representation of the manner in which information passes through the nervous system.

General Motor Association Cortex	<---	General Sensory Association Cortex
V		^
Motor Association Cortex		Sensory Association Cortex
V		^
Motor Strip		Sensory Projection Cortex
V		^
Motor Neuron Pool		Thalamus
V		^
Effectors (muscles & glands)		Receptors

Layer I: apical dendrites of the pyramidal cells
axons of the stellate cells, tangential to the surface, local intracortical communication
afferent from nonspecific thalamus

Layers II & III:
small pyramidal cells, intercortical information transfer
afferents from nonspecific thalamus

Layer IV:
specific thalamic afferents (terminating on stellate, pyramidal cells
distribution to other layers

Layer V:
large pyramidal cell (giant cells of Betz in the motor ctx)
transfer of information to the subthalamic parts of the brain

Layer VI:
back to the thalamus, corticothalamic projection

The above figure is numbered according to Broadman's areas.

Broadman's #	**NAME**	**FUNCTION**
17	Occipital Lobe	Visual Projection Cortex
18		Visual Association Cortex
19	Posterior Parietal Lobe	Visual Association Cortex
37	Tempero-pariteal-occipital area	General Sensory Association Cortex
39	Angular Gyrus	Word Recognition
40	Supramarginal Lobe	Somatosensory Association Cortex
1,2,3	Postcentral Gyrus	Somatosensory Projection Cortex
5, 7	Superior Parietal Lobule	General Sensory Association Cortex
41, 42	Middle 1/3 of Superior Temporal Cortex	Auditory Projection Cortex
22	Superior Temporal Gyrus	Auditory Association Cortex
21, 20, 38	Inferior Temporal Cortex	General Sensory Association Cortex
4	Precentral Gyrus	Primary Motor Cortex
1,2,3	Postcentral Gyrus	Somatosensory Projection Cortex
6,8,9	Premotor Cortex	Motor Association Cortex
41, 42	Middle 1/3 of Superior Temporal Cortex	Auditory Projection Cortex
44,45,46	Broca's Area	Motor Association Cortex - Specific to speech
10	Preftontal Cortex	General Motor Association Cortex
11	Orbital Gyri	General Motor Association Cortex

The frontal lobes of the human brain comprise all the tissue in front of the central sulcus. It lies anterior to the central (rolandic) sulcus and superior to the sylvian fissure. This represents 20% of the neocortex, having three distinct areas; motor, premotor, and prefrontal. These functionally different areas have been conventially assigned numerical numbers to represent specific regions (scheme devised by Brodmann). The motor cortex is area 4, projects to the spinal motor neurons to control limb, hand, foot, and digit movements and to the appropriate cranial nerve motor neurons to control face movements. It also projects to other motor neurons such as the basal ganglia and the red nucleus. The premotor cortex includes areas 6 and 8, which are divided into four areas: lateral area 6, or premotor cortex; medial area 6, or supplementary motor cortex (this area is where the integrity of voluntary movement is controlled); area 8 or the frontal eye field.

The frontal lobes are considered our emotional control center and home to our personality. There is no other part of the brain where lesions can cause such a wide variety of symptoms (Kolb & Wishaw, 1990). The frontal lobes are involved in motor function, problem solving, spontaneity, memory, language, initiation, judgment, impulse control, and social and sexual behavior. The frontal lobes are extremely vulnerable to injury due to their location at the front of the cranium, proximity to the sphenoid wing and their large size. MRI studies have shown that the frontal area is the most common region of injury following mild to moderate traumatic brain injury (Levin et al., 1987). There are important asymmetrical differences in the frontal lobes. The left frontal lobe is involved in controlling language related movement, whereas the right frontal lobe plays a role in non-verbal abilities. Some researchers emphasize that this rule is not absolute and that with many people, both lobes are involved in nearly all behavior.

Disturbance of motor function is typically characterized by loss of fine movements and strength of the arms, hands and fingers (Kuypers, 1981). Complex chains of motor movement also seem to be controlled by the frontal lobes (Leonard et al., 1988). Patients with frontal lobe damage exhibit little spontaneous facial expression, which points to the role of the frontal lobes in facial expression (Kolb & Milner, 1981). Broca's Aphasia, or difficulty in speaking, has been associated with frontal damage by Brown (1972).

An interesting phenomenon of frontal lobe damage is the insignificant effect it can have on traditional IQ testing. Researchers believe that this may have to do with IQ tests typically assessing convergent rather than divergent thinking. Frontal lobe damage seems to have an impact on divergent thinking, or flexibility and problem solving ability. There is also evidence showing lingering interference with attention and memory even after good recovery from a TBI (Stuss et al., 1985).

Another area often associated with frontal damage is that of "behavioral sponteneity." Kolb & Milner (1981) found that individuals with frontal damage displayed fewer spontaneous facial movements, spoke fewer words (left frontal lesions) or excessively (right frontal lesions). This is the same as the examples below of a mesolimbic based EPILEPTIC activity in the right versus left hemisphere.

behavioral sponteneity	**More Word**	**Fewer words**
Dec FOF Ablative	**RB**	**LB**
Inc FOF Epileptic	**LB**	**RB**

One of the most common characteristics of frontal lobe damage is difficulty in interpreting feedback from the environment. Perseverating on a response (Milner, 1964), risk taking and non-compliance with rules (Miller, 1985), and impaired associated learning (using external cues to help guide behavior) (Drewe, 1975) are a few examples of this type of deficit. This could be included in the Right Frontal Hemispheres association with ATTENTION, NOVELTY, NEW LEARNING.

The frontal lobes are also thought to play a part in our spatial orientation, including our bodies orientation in space (Semmes et al., 1963). There is a cross-modality association between the three functional zones: motor cortex, premotor cortex, and prefrontal cortex. The motor cortex is responsible for making movements, and the premotor cortex selects the movements which will be initialized. The prefrontal cortex controls the cognitive processes so that the appropriate movements are selected in accordance to time and place In the frontal lobes we see the home of our humanism, as these lobes are not seen in lower animals. The LEFT frontal hemishere can be characterized as a home of POSITIVE THOUGHT. The RIGHT frontal hemisphere can be characterized as the home of NEGATIVE THOUGHT.

LESION physiological or functional	**Depressive (more negative)**	**Manic (more positive)**
ABLATIVE (dec FOF)	**LB**	**RB**
SOMATIC effect	**Rt sided paresis of Speed, Intiation, Control**	**Lt sided paresis of Speed, Intiation, Control**
EPILEPTIFORM (inc FOF)	**RB (mesolimbic)**	**LB (mesolimbic)**
SOMATIC effect	**Left sided increase tone, dystonia, spont. movement**	**Right sided increase tone, dystonia, spont. Movement**

The major types of mood episodes are either depressive or manic. Below is a comparison of the direction, reactivity and duration of mood change in characterizing mood episodes:

Mood	**Depressive**	**Manic**
Direction	**Sad, blue, dejected**	**Elated, high, expansive, euphoric**
Reactivity	**constricted, blunted, flattened**	**labile, exaggerated**
	Allied symptoms	
Sleep	usually insomnia with early morning awakenings and midnight awakenings (can be hypersomnic in atypical cases)	reduced need for sleep
Appetite	reduced, often with weight loss	variable (can be increased but can't sit long enough to eat)
Libido	reduced interest	increased interest
Recreation	anhedonic	increased hedonism

As the affective psychopathology intensifies, the rest of the mental state begins to be involved. Below is a comparison of the extension of depressive and manic pathology into the rest of the mental state.

Component of the mental state	Depressive	Manic
Cognition	Feelings of guilt, worthlessness, hopelessness, reduced self-esteem, impaired concentration, nihilism, negation, suicidal ideation	expansive, exaggerated sense of self, impulsivity, overly optimistic, increased speed of thought (flight of ideas), poor judgment, grandiosity, hyper-religiosity
Activity	reduced goal directed activity, inertia, psychomotor retardation, catatonia	increased activity, pressured speech, spending sprees, increased risk taking, manic catatonia
Perception	Depressive content, "body rotting", somatization, can believe everyone is against them (paranoia). Can have delusions of guilt.	may develop frank psychosis with paranoid thought disorder. Can be virtually identical to schizophrenic psychosis

Frontal Lobes

Motor & higher cognitive functions; Initiative as well as behavioral inhibition;Sensitivity to social cues

1. Anatomy
    1. The frontal lobe is heavily interconnected with:
        1. -basal ganglia & other components of the motor system
        2. -all other lobes of cortex
        3. -limbic system
2. Beyond Motor Planning
    1. Frontal lobe has evolved from being the main motor planner/organizer to a higher level behavioral/strategic planner/organizer.
    2. Mental model, considering options, selecting behaviors based on context, feedback, stored knowledge
    3. Making predictions about what will work.
3. Impaired Divergent Thinking
    1. Decreased consideration of alternative strategies/behaviors; reduced flexibility
    2. Decreased spontaneity, initiative, may appear lazy, unmotivated
    3. Knowledge/intelligence may seem intact (e.g. IQ) but its not used to generate strategies or solve problems efficiently
4. Decreased Inhibition
    1. Problems inhibiting incorrect/ineffective responses & switching to a new strategy
    2. Perseverates; not responsive to feedback or changes in environment
    3. Violates rules, expectancies; takes risks
    4. Not adaptable
    5. Decreased social inhibitons as well
5. Impaired Associative Learning
    1. Reduced response to consequences
    2. Impaired on delayed response tasks
    3. Impaired responsiveness to social & contextual cues
6. Decreased Temporal Memory
    1. Impaired memory for order, recency
    2. Could affect problem-solving, planning and impair systematic, organized behaviors
7. Possible Personality and Emotional Changes
    1. Apathetic, indifferent, loss of initiative, lack of emotion or somewhat depressed, little verbal output. Most common after left frontal damage; called "pseudodepression"

8. Possible Personality and Emotional Changes
    1. Lack of tact & restraint, immature, coarse,lack of social graces, inappropriate sexual behavior, increased motor activity. More common after right frontal damage; called "pseudopsychopathic"
9. Some Neuropsych Tests Used
    1. Wisconsin Card Sorting Test
    2. Stoop Test
    3. Chicago/Thurstone Word Fluency Test
    4. Gotman-Milner Design Fluency Test
    5. Visual Search Test

Motor stength, speed and sequencing

FRONTAL LOBE SYNDROMES -
Human prefrontal cortex syndromes can be subdivided into three subtypes.

A. **Orbitofrontal Syndrome** - Damage in **Brodman areas 11, 12** results in prominent affect disturbances. Emotional lability and decreased impulse control contribute to poor social integration. Problems such as loss of control of anger and inappropriate laughing, crying or sexuality are often observed. Attention capacity is usually preserved, frontal release signs (i.e. snout, suck**,** palmomental reflexes) are absent and the patient is typically aware of the problem but unable to control their reflexive inappropriate behavior. The most common cause of the orbital syndrome is head trauma with contra coup damage. Olfactory groove meningiomas can also present with similar complaints.

B. **Mesial Syndrome** - Bilateral mesial prefrontal damage involving supplementary motor and cingulate cortex **(Brodmann areas 24, 25, 32, 33 and mesila 6, 8, 9**) produces an amotivational, akinetic state with motor programming deficits manifesting clinically as apractic disturbances. Unilateral mesial or mild bilateral disease yields lesser degrees of difficulty in the initiation and sustaining of motor and mental activity. A common cause is anterior cerebral artery infarction due to spasm from subarachnoid hemorrhage.

C. **Dorsolateral** - Damage in dorsolateral prefrontal cortex **(Brodmann areas 6, 68, 9, 10, 44, 45, 46)** leads to a complex range of behavioral disturbances since this is the most advanced phylogentic area in man. In early disease due either to tumors (i.e. glioma) or degenerative disease (i.e. Picks) sparing language cortex, subtle deficits in creativity and mental flexibility are often noted by the patient or family. As unilateral disease progresses or becomes bilateral pronounced behavioral problems become apparent. Abnormalities emerge in planning, goal directed behavior, temporal coding of external and internal events, metamemory (i.e. confidence about memory judgments), judgment and insight. Attention capacity is invariably impaired in advanced disease.

A common cause of acute unilateral dorsolateral prefrontal damage is infarction of the precentral branch of the middle cerebral artery. This occlusion results in variable amounts of damage to areas 6, 8, 9, 10, 44, 45, 46. Damage typically centers in area 46 which is

likely equivalent to the sulcus principalis region in monkeys critical for delayed response performance.

Acute unilateral infarcts involving area 46 and the frontal eye field (area 8) are often associated with a transient syndrome of mild global confusion and attention dysfunction. Right sided prefrontal infarcts are more likely to result in contralateral hemispatial neglect than comparable volume left sided infarcts. When motor and language cortex is spared, patients may not seek acute medical treatment. However, when seen at a later date they may complain of continued problems with attention, memory and mental quickness. In advanced bilateral dorsolateral disease due to tumor, degenerative or vascular disease, preservation and frontal release signs (snout, grasp, palmomental) are often present.

Premotor Cortex Area 6,8,9

Responsible for kinetic organization of movement once started, transfer, smooth sequencing. According to Luria, 1973, it is responsible for the conversion of individual motor impulses into consecutive kinetic melodies.

A. Oral (buccofacial) Praxis

1. Puff cheeks.
2. Click teeth together three times.
3. Whistle,
4. Protrude tongue
5. Lick lips.
6. Pucker lips.
7. Cough.
8. Blow out a match.
9. Retract lips to show teeth.
10. Sip on a straw.

B. Speech/Language

1. Spontaneous speech sample - Note agrammatism and dysprosody.
2. Diadochokinetic rates - Repeat the syllable "puh" as fast as you can: then "tuh": then "kuh"; then put thon together "puh-tuh-kuh".
3. Imitation of monosyllabic and polysyllabic words.
4. Repeat words of increasing length, i.e. thick, thicker, thickening; zip, zipper, zippering; and hope, hopeful, hopefully.
5. Imitation of sentences.

C. Intransitive Body movements (Motor Sequencing)

1. Touch fingers with thumb AFAP - reveals paresis, lack of precision, pathological dystonia, ataxia.

2. Separate fingers & bring together AFAP.
3. Alternately clinch and relax fingers of both hands for a long period of time.
4. Bilateral clinching & relaxing fingers AFAP.
5. Finger tapping.
6. Tapping as fast as possible (Speed)
7. Tapping two times with one hand and one time with the other hand and then reversing the pattern (Rhythm). With premotor lesions, movement loses smoothness and each tap is produced as isolated phenomenon; patient begins to make superfluous taps or performs identically with both hands.
8. Snap your fingers.
9. Salute;
10. Fist-Ring Test - can't do them in series; more problems when sequence is reversed.
11. Fist-Edge-Palm Test .
12. Circle Hands In Air .

D. Transitive Body Movements

1. Demonstrate playing the piano or typing; piano playing test is forward & then reversed; frontal patients can't reverse; premotor may improve with spoken commands or feedback; frontals may repeat correctly but can't make proper movements.
2. Thread a needle.
3. Tie shoelaces.
4. Cut paper with scissors.
5. Unlock and open a door.
6. Flip a coin.

E. Graphics

1. Write words from dictation - can form letters but often disarranges or transposes them.
2. Write sentences from dictation - words may be written correctly but may be in the wrong order.
3. Spontaneous writing.
4. Drawing various shapes,(circle, square, cross)
5. Draw zig-zag line alternating pointed and rectangular elements.

Prefrontal Cortex Area 10

Responsible for planning, structuring, and evaluating voluntary (goal directed behavior, i.e., activities requiring the constant comparison of planned acts with the effects achieved.

A. Deficits associated with lesions of the prefrontal cortex

1. With minor lesions
    - Inability to prevent rapid extinction of orienting response following verbal instruction.
    - Disturbances in regulatory role of speech.
    - Disturbances in complicated gnostic functions (e.g. understanding complicated pictures, thematic pictures, comprehension of written text).
    - Problem solving difficulties associated with disturbances in selective organization of mental activity (i.e. serial sevens).
    - Deficits in complex tasks requiring inhibition of habitual behavior patterns.
    - Difficulties with actions requiring a series of movements, less severe when accompanied by external verbalization (Fist, edge, palm).
    - Difficulties in task executions when instructions or prompts conflict with what would be expected, although ability to carry out simple instructions is unimpaired (visual or verbal).
    - Difficulties tapping at successive groups of rhythms or drawing a series of figures that alternate in pattern (tendency to perseverate).
    - Decreased spontaneity, decreased rate of behavior, decreased range of interests, loss of initiative, and impulsiveness without self-corrective action may be early signs.
    - Deficits in visual tracking and scanning, especially on complex tasks.
    - Difficulty in constructing mirror-image relationships, especially if complex.
2. With more extensive lesions.
    - Perseveration: difficulties in making behavioral shifts in attention, movement, and attitude.
    - Concreteness & decreased creativity.
    - Inflexibility in cognitive, perceptual and motor modalities.
    - Poor recall of thematic verbal material (paragraphs).
    - Poor recall of verbal & nonverbal series, with contamination of first & subsequent series.
    - Deficits in comprehension of logical-grammatical (prepositional) constructions (e.g. "place a cross beneath the circle.") .
    - Difficulties in writing associated with fatigue, progressively smaller characters, perseveration, loss of overall plan (i.e. letter transpositions, etc.).
    - Deficits in abstract/categorical intellect.
    - Take longer to learn go-no go tasks and make more false positive responses particularly with medial frontal lesions.
    - Diminished visual scanning and tracking, resulting in impulsive judgments which are based on the perception of a single aspect of a stimulus.
    - Performance of relatively simple task is impaired by perseveration; however, overlearned tasks are conducted without difficulty.
    - Increased level of distractibility, especially to small noises or events.
    - Memory curve plateaus early (approximately 5 items), even with rehearsal.

- Disturbances in selective memory with confabulation.
- Defects in time sense with respect to recency and time span Judgments and disturbances of temporal orientation occurring with bilateral frontal lobe lesions.
- More superior lesions produce motor disturbances, while inferior lesions produce speech disturbances
- Increased trend toward confabulation .
- Magnitude of deficit is associated with the cause of the lesion (i.e. surgery or degenerative) and the presence of generalized physical disorders (i.e. hypertension). Also, deficits are more severe with bilateral involvement.
- Constructive intellectual activity may be distributed when preliminary analysis and formation of a plan is required (not constructional apraxia per se) .
- Diminished critical self-evaluation of behaviors; no distress or attempts at correction.
- Able to perform firmly-established verbal analogies (father: son: Mother, but difficulty in forming unfamiliar analogies
- Poor capacity for arithmetic tasks involving a series of analytic steps, although often able to solve simple problems. Tend to make impulsive judgments.
- Emotional disturbances seen as two principle reactions: inhibition (apathy, narrowing of interests, flattered affect, withdrawal) and disinhibition (euphoria, impulsivity, irritability, anxiety, obscene language.
- Symptoms of frontal lobe lesions with increased intracranial pressure can include headache and somnolence.

3. **With medial orbital lesions**
    - More often associated with emotional changes such as apathy or hyperactivity.
    - Defects in sorting or abstraction tasks usually not observed; gross intellectual changes also not apparent.
    - Olfactory & visual disturbances may occur.
    - Emotional alterations can range from apathy to short-term lability.
4. **Lateral dorsal lesions**
    - Associated more with intellectual deficits.
    - Perseveration is more common: associated with impaired shifting in attention and thinking.
    - With extensive lesions, gross perseveration may occur even though patient recognizes as inappropriate.
    - Impaired categorical thinking apparent.
5. **Left hemisphere prefrontal lesions**
    - Deficits in tests of categorization and flexibility.
    - Problems with body schema (autopagnosia) due to problems of scanning, perceptual shifting and postural mechanisms.
    - Marked inactivity affects general intellectual processes and behavior.

- Cannot change verbal instructions into acts, especially when the instructions are complex or symbolic.
- Decreased spontaneity of speech; may result in complete loss of voluntary speech.
- Memory deficits for verbal material; however, deficits may be due to defective registration.

6. **Right hemis. prefrontal lesions**
   - Constructional apraxia, associated with motor rather than perceptual difficulties; deficits may occur as a function of impaired complex (3-D) spatial analysis.
   - Large lesions may exist without obvious symptoms; serious speech disorders usually not seen in right hemisphere lesions.
   - Difficulty with drawing tasks, though this is associated more with right hemisphere lesions in general.
   - Impaired visual-spatial integration, maze learning, non-verbal visual memory

With very severe lesions there may be a complete disintegration of behavior as observed in many of the following: nonreactive to environmental cues or instructions from self to others, reactive to irrelevant stimuli, echolalia, mutism (loss of voluntary speech), agraphia, confusion, and generalized slowing . * Even though, following the Luria approach , we have chosen to distinguish lesser from more extensive lesions, this distinction is somewhat arbitrary. Research suggests that the distinction is based on quantitative rather than qualitative differences. That is, with lesser lesions, one could expect to find deficits similar to those seen in cases of more extensive lesions; however, such deficits would obviously he of less magnitude. Likewise, the types of deficits associated with less extensive lesions would undoubtedly be observed in more extensive cases.

B. Tests - Prefrontal Lobe Function

1. Wisconsin Card Sort: left > right
2. Word fluency: L > R
3. Halstead Categories Test: L > R
4. Trail Making, Part B: L > R
5. Color and word page of Stroop Color and Word Test: L > R
6. Temporal orientation test: bilateral
7. Verbal associative learning: bilateral
8. Personal identification test(body schema): L>R
9. Picture Arrangement: R > L
10. Memory for Designs: R > L
11. 3-D Constructional praxis test: R > L
12. Object classification test: Right frontal and left parietal
13. Link's cubes (building a large cube from differently colored small cubes): R > L

14. Reaction of choice tests (two choices), such as go-no go: L > R on more complex tasks.
15. Impairment on tests requiring complex picture and Raven's Progressive Matrices)
16. Depressed digit span: L > R.

Supplementary Motor Area

The supplementary motor area (SMA) occupies an expanse of frontal agranular cortex rostral to the primary motor cortex (MI), largely in the medial surface of the hemisphere. It is basically organized topographically, although the topography is not as apparent as in the MI. The traditionally defined SMA is now regarded as including two separate areas.

The caudal part (SMA proper or F3) projects directly to the MI and to the spinal cord.

The rostral part (pre-SMA or F6) is more remote from MI and receive projections from the prefrontal cortex and the cingulate motor areas.

The supplementary eye field (SEF) is a small area separate from either the SMA or pre-SMA. The SEF is connected to cortical and subcortical areas related to oculomotor control. The SMA is active when subjects perform distal as well as proximal limb movement. The SMA activity is subject to functional plasticity. The SMA is more active than the primary motor cortex if motor tasks are demanding in certain respects. Similarities of lesion effects of the SMA and basal ganglia suggests their intimate relation linked anatomically by the cortico-basal ganglia loops.

Studies in both human subjects and in subhuman primates indicate the importance of the SMA in motor tasks that demand retrieval of motor memory. The SMA appears also crucial in temporal organization of movements, especially in sequential performance of multiple movements.

Transcortical motor aphasia - The lesion is anterior to Broca's area or in the supplementary motor area. The language syndrome is similar to Broca's except for preserved repetition and occasionally preserved writing.

Language is a motoric component, therefore, we should become a bit more adpet at noticing subtle changes in cadence, prosadic variation, mistakes. Etc.

Broca's Aphasia (= nonfluent = expressive = motor = anterior aphasia) - Broca's aphasia is seen in its pure form in lesions involving the inferior frontal gyrus. Due to the proximity of the precentral gyrus, there is typically an associated right central facial paresis and right hemiparesis (arm>leg). Speech is slow, effortful, and has articulatory errors. Phrase length varies from complete mutism to decreased number of words per sentence (2-3 with normal being 5-9). There is a parallel deficit in writing. Repetition, particularly short phrases ("no ifs, ands or buts")is severely disrupted. Comprehension is largely intact although detailed linguistic analysis reveals difficulty with prepositions such as into, on, etc. There is often associated anomia, production of paraphasic errors ("grish" for "dish") and oral facial apraxia. The apraxia is manifested by difficulty in sequencing oral movements. Patients may be able to hum a tune or sing words.

Anterior Temporal Cortex

Performs basic Auditory processing of auditory stimuli that are shorter in duration than similar stimuli processed more posteriorly in the temporal lobe. Also processes some visual material.

A. Dysfunctions Associated with Lesions of Anterior Temporal Lobes

1. Auditory memory disturbance
2. Left anterior temporal lobe damage impairs learning and retention of verbal material regardless of whether material is auditorily or visually presented and regardless of whether patient is tested using recall or recognition techniques.
3. Left anterior temporal lobe does not affect memory for places, faces, melodies- etc.
4. Right anterior temporal lobe damage impairs recognition and recall of visual and auditory patterns that do not lend themselves to verbal coding.
5. Difficulty remembering short auditory stimulation.
6. Deja vu.
7. Hallucinations (auditory and/or visual).
8. Disinhibited social behavior.

B. Assessment Strategies for Anterior Temporal Lobe Lesions

1. Paired associates
   Seashore Rhythm Test
2. Left vs. right
   - -Paired Associates Verbal Story for Immediate Recall
   - Visual Memory
     Seashore Rhythm Test

1. Visual Memory
   Seashore Rhythm Test
2. Repeat short and long sentences
3. Discern presence of deja vu. Presence of deja vu does not help to lateralize lesion .
4. Discern presence and nature of hallucinations
5. Obtain recent history concerning quality of social judgment

Middle Temporal Lobe

General association cortex integrating input from lower level auditory and visual areas.

A. Dysfunctions Associated with Lesions of Middle Temporal Lobe

1. Acoustico-mnestic disorders, can't retain series of sounds, syllables or words in memory.
   - Comprehension of individual words intact but cannot retain more than 2 or 3 at once.
   - In mild cases, patient can retain essential elements of series but cannot remember correct order.
   - Problem is due to increased mutual inhibition of auditory traces.

1. Do not have difficulty with phonemic learning.
2. Difficulty reproducing words or word series under complicated conditions.
3. Impaired ability to name series of objects.
4. Stimulation gives hallucinations, memory images, changes in state of consciousness.

B. Assessment Strategies for Middle Temporal Lobe

1. Paired associates, long vs. short series of words, verbal story for immediate recall.
    - Check for comprehension of individual words in above tests.
    - If items are remembered, check for proper ordering.
    - Increase intervals between presentation of individual items in series and see if performance improves.
1. Auditory Discrimination Test
2. Introduce a distraction interval before patient repeats series.
3. Ask to name several objects individually and then all at once.

Right Temporal Cortex

A. Effects of lesions-temporal cortex

1. Compared with similar lesions of the left temporal lobe, right temporal lesion effects tend to be notable statistically but of less clinical significance.
2. Visual analysis (nonverbal primarily)
    - Impairment of simple and complex visual analysis, but some negative findings.
    - Impairment of short-term nonverbal memory.
    - Impaired perception of tachistoscopically-presented letters.
    - Prosopoagnosia (especially with anterior lesions).
    - Impaired recognition of objects seen from unusual angles.

3. Auditory analysis (nonverbal)
    - Impairment of short-term auditory memory.
    - Perception of short sounds impaired.
    - Impaired recognition of familiar sounds.
    - Impaired tonal discriminations, timbre discriminations, and amplitude discriminations.
    - Amusia.
    - Impairment of contralateral ear input in dichotic listening.

.4. Constructional tasks
    - Visual construction impairment proportional to tissue loss.
    - Impairment in maze learning (visual and proprioceptive feedback).
    - Enlarged left-hand margin in dictation.

1. Psychiatric -- personality phenomena with right temporal epilepsy
    - Personality changes.

- Psychiatric symptoms.
- Deja vue.
- Metamorphopasias.

1. Psychometric findings
   - Temporary decline in Performance IQ following lobectomy.
   - Impairment on WAIS Picture Arrangement.
   - Impairment on Binet Memory for Designs
   - Possible impairment of WAIS Block Design?.
2. Persistence in maintaining a hypothesis even after being informed it was not correct.

B. Assessing possible right temporal lesions

1. Impairment on WAIS Picture Arrangement.
2. Impairment on Seashore Test of Musical Talent (especially Tonal memory, Timbre, Loudness, and Time).
3. Lezak and others
   - Have the patient identify a tune the examiner hums. If unsuccessful, try several other familiar melodies to check for amusia.
   - Test pitch discrimination with pitch pipe.
   - Have patient try to discriminate (or imitate) different rhythmic tapping patterns. A memory component may be added.
   - Test recognition of familiar sounds.
   - The examiner may pair verbal and nonverbal material to clarify the interpretation of a patient's failures.

1. Dichotic thresholds.
2. Impairment of short-term memory for other nonverbal acoustic tasks.
3. Visual recognition deficits (prosopoagnosia included)
   - Impairment of recognition of photographs of faces.
   - Impairment on Closure Faces Test (Mooney).
   - Impairment on McGill Picture Anomalies Test.
   - Difficulty in identifying pictures of objects viewed from unusual angles.

1. Impairment of other nonverbal visual tests of short-term memory and visual discrimination or Binet Memory for Designs Test.
2. Corsi's block-tapping test.
3. Impairment on stylus maze tasks (visual or proprioceptive feedback).
4. Impairment in enumeration of tachistiscopically-presented dots.
5. Enlarged left margins in dictation.
6. In epileptics a history of strong deja vue experiences or metamorphopsia.

Left Temporal

Primary functions of this lobe include: decoding of speech sounds, comprehension of speech and mediating verbal memory processes.

A. Dysfunctions associated with lesions of the left temporal lobe.

1. Auditory deficits (right ear)
    - Intracranial localization of sound is impaired.
    - Increased threshold for perception of short bursts of sound.
    - Increased threshold for some frequencies.
    - Failure to perceive brief simultaneous auditory stimulation.
2. Visual deficit (both eyes)
    - Upper right quadrantanopsia.
3. Other complex sensory deficits
    - Right hand tactile performance difficulty.
    - Right hand finger agnosia.
4. Language deficits
    - Decoding of speech sounds (phonemes) is impaired.
    - Problems with verbal repetition.
    - Problems with auditory comprehension of speech.
    - Receptive aphasia (deficits in all language qualities).
    - Impairment of dichotic listening to verbal material.
    - Intellectual impairment on verbally mediated intellectual processes.
1. Memory impaired for verbal material.
2. Impairment on measures of higher cortical functions (Trails A and Trails B).
3. Emotional disturbances
    - Perceptual distortions, alterations of mood, obsessional thinking, psychosis, temper outbursts, hypo and hypersexuality.

B. Assessment devises for left temporal lobe lesions

1. Auditory tasks
    - Binaural localization of clicks.
    - Simultaneous auditory messages.
    - Short duration tone bursts-threshold testing.
    - Frequency threshold testing.
    - Dichotic listening.
2. Visual testing
    - Test visual perimetry.
3. Complex sensory tests
    - Tactual performance test.
    - Finger agnosia test.
4. Linguistic abilities tests
    - Speech sound perception test.
    - K-V auditory discrimination test.
    - Token test (for comprehension).
    - Aphasia exams
    - Wepman-Reitan Aphasia Screening Test.

- Porch Index of Communicative Ability.
- Boston Diagnostic Aphasia Examination.
- Dichotic listening.

5. Measures of Intelligence
    - Wechsler Adult Intelligence Scale; sensitive subtests include similarities, arithmetic, and digit symbol.
6. Assessment of verbal memory
    - Hebb's recurring digits (digit span with every third list is repeated).
    - Consonant trigrams (recall of a spoken set of three consonants following distraction).
    - Discrimination of recency (subject required to indicate which of two verbal stimuli they have seen most recently).
    - Recall of Logical Memory from Wechsler Memory Scale after a 1 hour delay.
7. Assessment of higher cortical functions
    - Trails A and Trails C.
8. Emotional disturbances
    - MMPI.

Wernicke's Area

A region of the brain located in the posterior, superior temporal gyrus, adjacent to the cortical region for hearing. This area seems to be of focal importance for language and is involved in the recognition of the auditory patterns of language.
Wernicke's Aphasia (= fluent = receptive = sensory = posterior) The lesion is in the posterior region of the planum temporale of the superior temporal gyrus (Wernicke's area) and adjacent inferior parietal cortex. Motor weakness is typically minimal or absent. There may be a partial hemianopsia due to involvement of the geniculocalcarine fibers deep to this area. Word output is normal or increased, with many paraphasic errors, and speech is often incomprehensible (jargon aphasia). Paraphasic errors include elogisms ("Slep", "gort"), phonetic distortions ("good" for "wood") and semantic mistakes (Chevy for Ford). Auditory (speech) and visual (reading) comprehension are severely disturbed. The patient cannot follow 2 or 3 part commands (i.e. pick up the pen and give the keys to the doctor). One part commands can often be followed (i.e. stand up). Repetition is impossible. The severe comprehension disorder often makes testing of other cognitive functions difficult.

A. Dysfunctions

1. Speech may be very rapid, with rhythm, grammar and articulation preserved, but devoid of content.
2. The patient may fail to use the correct word and substitute circumlocutory phrases and "empty words" (e.g.,"thing").
3. Verbal paraphasia -- substitution of one word or phrase for another, sometimes related in meaning

4. Literal (phonemic) aphasia -- substitution of incorrect sounds in otherwise correct words.
5. A lesion in Wernicke's Area can produce a severe loss of understanding, even though hearing of nonverbal sounds and music may be fully normal.
6. Damage to Wernicke's Area causes difficulties in the comprehension of both spoken and written language since auditory pattern can't be aroused
7. Disturbance of auditory analysis and syntheses which leads to the loss of phonemic hearing.
8. Impaired ability to match an auditorily presented word to its equivalent picture when presented in an array of phonemically similar test items.
9. Normal articulation, prosody and average phrase-length; rate of speaking normal or increased; frequent verbal neologistic or unclassifiable paraphasias; periods of jargon and severe disturbance in verbal comprehension.
10. Word-comprehension disorders.
11. A case in which a right-handed male with a completely destroyed Wernicke's Area in the left hemisphere, did not result in aphasia. This case is first validated case of language dominance in the right hemisphere of a right-handed individual.
12. Impairment in the comprehension of language and associated mental decrement.
13. Disruption of the ability to obtain meaning from a stimulus and to use it as a basis for orderly symbol formation.
14. Impaired naming repetition, and comprehension in the presence of fluent speech. The speech pattern is markedly circumlocutory and void of specific semantic content. Production of neologistic jargon is also observed.
15. Disturbances in reading, writing, naming, repetition, and comprehension of the spoken language
16. A patient, though his hearing is normal, is unable to understand spoken speech.
17. Perseveration may occur, in which the same word is used repeatedly.
18. Accelerated speech may be one of the factors which leads to perseveration and lack of inhibition may be another.
19. Disturbance of the understanding of speech, defects in the repetition of words and the naming of objects, impairment in writing.
20. Patients displaying breakdown in the discrimination of speech sounds and consequent difficulties in the comprehension of speech and of word meanings, but without impairment of hearing per se, are most likely to have lesions in Wernicke's Area.
21. Comprehension deficit attributed to a selective impairment in phonological perception.
22. Proper auditory feedback is absent.
23. A Lesion in "the centre of auditory images" gives the classical Wernicke's aphasia (fluent speech, poor comprehension, and poor reception)

24. A lesion which interrupts the pathway from the primary auditory area to Wernicke's Area produces "pure word deafness." The patient has fluent and normal spontaneous speech but he cannot comprehend or repeat spoken language. Although he can comprehend written language and read aloud.

B. Tests for Aphasia

1. Porch Index of Communicative Ability (PICA)
2. Token Test
3. Token Test (3E-item version)
    - Appears to be a useful and convenient device to diagnose aphasia impairment of language comprehension.
1. Boston Diagnostic Aphasia Battery
2. Western Aphasia Battery
3. Minnesota Test for Differential Diagnosis of Aphasia
4. Halstead-Wepman Aphasia Screening Test
5. Head's Serial Tests
6. Language Modalities Test
7. Illinois Test of Psycholinguistic Abilities
8. Michigan Picture Language Inventory
9. Functional Communication Profile
10. Examining for Aphasia
11. Sklar Aphasia Scale
12. Neurosensory Center Comprehensive Examination for Aphasia

MEMORY - Memory the ability to store and retrieve information both on a short and long term basis. It is a critical factor in all forms of learning. Memory dysfunction is an early feature of numerous neurological disorders. In addition, it is a frequent complaint in psychotic patients. When valuating a patient's memory function, it is important to remember that inattention, decreased motivation and poor cooperation, all features of non-organic depression, can appear to decrease memory ability. In depression, however, the memory defect can be overcome by eliciting the patient's cooperation and concentration, while organic deficits in memory are not removed by increased patient effort.

Temporal Stages of Memory:

1. Sensory Store - From stimulus onset to about 250 msec. This is the stage during which a sensory input is converted into a perceived sensation.

2. Short Term Memory (STM) - This refers to a system which holds new sensory traces from a few seconds up to 1-2 min. If the information is of interest or requires more processing it is rehearsed (recycled) in STM or transferred to long term memory (LTM); if not, it is forgotten. STM is a limited capacity, modality -specific system; (i.e. may not

be able to hold any more auditory input in STM, but could add additional visual input). Clinical disorders of memory are not defects in sensory store or STM. Patients can usually store items for 15-45 seconds even with the most severe degree of anterograde memory loss. If the sensory store or STM is impaired it is usually due to an attentional disturbance. This can be assessed by digit span test. Memory disorders involve transfer of information from STM to LTM.

3. Long Term Memory - Formation of LTM requires both intact sensory store, short term memory and the consolidation mechanism which takes new data from short term store and converts it into LTM. Most clinical memory deficits involve breakdown in conversion from STM to LTM. This deficit is referred to as anterograde amnesia or the inability to form new LTM. Once information has been stored in LTM it can still decay if not rehearsed (recycled through STM?). Not all new facts that you learn and remember at the end of a day are present weeks or months later. LTM is the last memory to be lost in organic disease, with the most remote events (i.e. childhood events) remembered the longest. The loss of remote memory is referred to as retrograde amnesia. It is always accompanied by severe anterograde amnesia.

4. Neuroanatomy of Memory - The hippocampus plays an essential role in the transfer of memories from STM to LTM. Recent data suggest that critical structures in STM-LTM conversion include the hippocampus and the nearby temporal stem which contains fibers connecting the middle and inferior temporal gyri with the dorsomedial nucleus of the thalamus.

Lesions of the dorsomedial nucleus of the thalamus (which projects to orbito-frontal and temporal cortex), mammillary bodies (which project to anterior nucleus of thalamus, then to cingulate gyrus), fornix, and entorhinal cortex also cause memory disturbance. Of interest is the finding that unilateral lesions of any of these structures cause relatively minimal clinical deficit; bilateral lesions are needed for severe deficits in memory function.

PARIETAL LOBE SYNDROMES - The left and right parietal lobes have equal processing capabilities for light touch, tactile localization, 2-point discrimination, joint position sense, passive movement sense, and stereo gnosis. Lesions in the post-central gyrus or adjacent parietal cortex impair these functions. The remaining areas of parietal lobe are involved in complex trimodal associations which are lateralized. Language and sequential analysis ability are strongly lateralized to the left inferior parietal lobe and adjacent superior temporal plane (see aphasia). Spatial abilities are 1Q86 strongly lateralized than language. Both parietal lobes have substantial spatial abilities, with the right being superior. Disorders of spatial-perceptual abilities are seen with the highest frequency in right parietal association cortex lesions.

A. Right Parietal Lobe Syndrome:

1. Constructional apraxia is manifested by deficits in block design, stick design, drawing and geometric design. Patients tend to focus on individual elements (local properties) and lose the overall picture (global properties). This deficit extends to geographical space

disorientation.

2. The hemi-inattention or hemi-neglect syndrome is the most dramatic non- dominant hemispheric syndrome and is seen in cortical lesions involving the temporal-parietal junction or the frontal eye field. Neglect can also be seen in unilateral thalamic lesions. The patient shows neglect of the contralateral half of visual space, the most severe example being anosognosia or denial of illness. Patients may deny having hemiplegia and may even deny the hemiparetic arm is theirs. Mild forms include extinction of the contralateral (left) stimulus during simultaneous presentation of bilateral visual, tactile or auditory stimuli.

3. Dressing apraxia - The patient has difficulty dressing the left side of body.

B. Left Parietal Lobe Syndrome:
1. Gerstmann Syndrome: acalculia, finger agnosia, left/right disorientation, agraphia. The lesion is in the left angular gyrus. Since there is often some degree of receptive aphasia and anomia, some features may be due to language disturbance.

2. Fluent aphasia, due to lesions of the temporal/parietal junction (i.e. Wernicke's, conduction, see Aphasic Disorders).

3. Alexia with agraphia can occur with angular gyrus lesions; some cases produce profuse semantic errors (i.e. deep dyslexia).

4. There are less severe deficits in constructional ability and milder forms of hemi-inattention to contralateral space.

C. Bilateral Parietal Syndrome:
1. Simultagnosia - The patient sees only one section of the visual field and fails to perceive the rest, although formal visual fields are normal on tangent screen perimetry. The patient may not be able to recognize large objects and will have to grope around the room.

2. Visual Agnosia - This involves a bilateral occipital/parietal lesion. The patient cannot name an object and cannot tell what it is or describe what it is used for. However, the patient can visually describe the object. There is a disconnection of visual information from memory stores. The patient knows and can name the object as soon as it is touched.

Post Central Gyrus
This is the primary projection area for somatosensory and kinesth-etic/proprioceptive inputs. It is topographically organized, and important body areas for tactile analysis (hands, face, mouth, tongue) receive disproportionate representation. The sensory feedback loops that project into this region are intimately related to precise motor control as well. Thus, both sensory and motor impairment are possible sequelae of injury to the post central gyrus.

A. Kinesthetic Basis of Movement

1. Eyes closed - patient is to position hand to match position of other.
2. Passive finger detection.
3. Two point threshold.
4. Von Frey Hair threshold.
5. Vibration sense.
6. With lesion most severe changes are distal, coarse sensations return first.
7. Height discrimination.
8. Pinpoint vs. head.
9. Touch area on skin, have patient point to area on contralateral side.
10. Fasten a button.
11. Tie a shoelace.
12. Localized lesion by deficit interactions.

B. Post Central Gyrus Test Items:

1. Eyes closed - match one hand to position set by examiner. Used in "Luria Battery".
2. Passive finger detection, two point threshold, Von Frey Hairs Finger Agnosia - used in Halstead Reitan Battery In-Between test, two point finger test, matchbox test, finger-tip number writing, Rey's skin writing.
3. Vibration sense - tuning fork.
4. Weight discrimination.
5. Pinpoint vs. head - use a pin. Also see Talland, 1965 - cited in. Tactile Completion Test.
6. Touch area and have patient point to contralateral area.
7. Fine motor tasks - see also Grooved pegboard, motor steadiness, finger or stylus mazes.
8. Not on outline, but also look for unusual speech. Consonant substitutions (especially of similar sounds), without broken or jerky speech typical of Broca's Aphasia. May see writing errors due to role of articulatory movements in analysis of words.

Most tasks for assessing the integrity of this brain area are more neurological than psychometric in nature.

Inferior Parietal Lobule

A. Dysfunctions caused by lesions

1. Apraxia for dressing.
2. Constructional apraxia (spatial apraxagnosia) - problems in motor integration in constructional tasks.
3. Spatial orientation deficit (more severe for right hemisphere lesions than left:).
4. Right-left disorientation.
5. Planto-pokinesia (disorganization of discriminations in spatial Judgment).

6. Visuospatial agnosia.
7. Difficulty in performing reversible operations in extrapersonal space (difficulty in taking different perspectives) (more severe for right hemisphere lesions than left).
8. Inability to maintain visual image of patterned and verbal material.
9. Visuographic defects.
10. Unilateral neglect.
11. General intellectual impairment (lesions in left hemisphere).
12. Problems with writing and defective comprehension in reading.

B. Assessment

1. Inability to analyze positions of hands on a clock.
2. Confuses symmetrically arranged symbols (e.g., d & b).
3. Difficulty making rotations on a 2-D stick test.
4. Difficulty changing perspectives on a village scene test.
5. Difficulty with transformations on pool reflections test.
6. Problems on both visual and tactile route finding tests.
7. Difficulty in maze learning.
8. Inability to follow habitual routes.
9. Difficulty designating body parts on examiner.
10. Difficulty drawing common objects to demand.
11. Problems in visual memory for patterns and verbal matter.
12. Errors on the Bender.
13. Poor performance on Unknown Faces Test
14. Difficulty with simple addition, subtraction, multiplication, and division, both presented orally and written.
15. WAIS arithmetic subtest scores lowered.
16. Low test scores on Army General Classification Test.

Supramarginal Gyrus

A. This sensory association area integrates kinesthetic memories with auditory commands. Lesions may produce:

1. Ideomotor apraxia: disruption of organization of complex acts
    - Results from left hemisphere lesion
    - Usually affects both sides, may be worse on right side
    - Can affect the face (buccofacial) and/or the limbs
1. Conduction aphasia: results from left hemisphere lesion if the underlying arcuate fasciculus is cut
    - Severely defective repetition
    - Paraphasia in repetition and in spontaneous speech
    - Normal comprehension
    - Impaired writing, spontaneous and to dictation, errors in spelling, word choice, syntax
1. Astereognosis: impairment of somatosensory discrimination
    - Left hemisphere lesion: both hands affected
    - Right hemisphere lesion: deficit - left hand

1. Finger agnosia: inability to recognize, name, and point to individual fingers on self and others (left hemisphere lesion).
2. Right-left disorientation
    - Can't distinguish right from left on self or env.
    - More common with left hemisphere lesion

1. Acalculia
    - Loss of ability to understand & order numbers
    - More severe with left hemisphere lesion

1. Tactile perceptual disability: results from contralateral lesion
2. Gerstmann's syndrome: Researchers disagree as to the lesion site for this syndrome, but the supramarginal and/or angular gyrus is usually involved. This left hemisphere, inferior parietal disorder includes:
    - Right-left disorientation
    - Finger agnosia
    - Agraphia
    - Acalculia

1. Lesion of right hemisphere supramarginal gyrus can cause:
    - Constructional apraxia
    - Mild left side neglect and/or denial
    - Inability to interpret maps

B. Tests to measure the Above Deficits

1. Ideomotor apraxia
    - Carrying out motor acts to command: buccofacial (blow out a match, protrude tongue, drink through a straw)
    - Carrying out motor acts to command: limb (salute, use a toothbrush, flip a coin, hammer a nail, comb hair, snap fingers, kick a ball, crush out a cigarette)
2. Conduction aphasia
    - Repetition of words, phrases, & sentences
    - Write to dictation (letters, words, sentences)
    - Ask patient to write sentences describing a Job, the weather, or a picture
    - Confrontation naming of objects, clothing, body parts, parts of objects
3. Astereognosis (with eyes closed)
    - Patient identifies by touch such common objects as a coin, paperclip, pencil, or key (each hand tested separately)
    - Patient judges the relative size of a series of coins
    - Patient judges the texture of a series of objects, such as cloth, wire, sandpaper
4. Finger agnosia
    - In-between test, Two-Point Finger Test, and Match Box Test
    - Identifying named fingers on examiner's hands and naming fingers on self
5. Right-left disorientation
    - Identification of right and left limbs on self and examiner

- Crossed commands on self and examiner

6. Acalculia
    - Written addition, subtraction, multiplication, and division problems
    - Verbal complex problems
7. Fingertip number writing
8. Gerstmann's syndrome
    - Right-left disorientation
    - Finger agnosia
    - Agraphia: writing to dictation and writing sentences describing scenes in pictures
    - Acalculia
9. Lesions of right hemisphere supramarginal gyrus
    - Constructional apraxia
    - copying designs
    - match stick tests
    - block construction test
    - Left-side neglect
    - glove test: ask the patient to put on a pair of gloves
    - drawing to command: clock, bicycle, flower in pot
    - behavioral observations
    - Have patient locate cities on a map

Parieto-occipital Cortex

A. Right hemisphere - intact functioning of this area of the cortex contributes to the individual's ability to orient himself in space, reproduce constructions, and recognize objects through visual or tactile cues. Facial recognition appears to be a function of right parietal cortex as well.
Dysfunction may produce:

1. Right-left homonymous hemianopsia - severe visual field cuts. Test: visual exam, copy drawings, reading ability.
2. Contralateral hemianaesthesia - global anesthesia to all modalities affecting the left side of the body, but with grossly preserved motor and postural control of the involved limbs.
3. Paragnosia - difficulty with visual recognition of objects - also called visual agnosia. Test: Naming tasks, visual recognition tasks.
4. Prosopagnosia - recognition of faces. Test: Tachistoscopic recognition of faces, behavioral observation.
5. Spatial loss greater than with lesion to left hemisphere. Test: constructional ability tests.
6. Unilateral spatial agnosia - neglect or inattention to one side in spatial orientation. Test: Drawings of designs, block design, Incomplete Pictures.

7. Visuospatial Dysgnosia - loss of "whereness", relation of self to environment and relations of objects to each other. Test: Drawings of designs, loss of way in familiar surroundings, tests of right-left orientation.
8. Anosognosia - lack of awareness of defect.
   Test: Self-report by patient-interview.

OCCIPITAL LOBE SYNDROMES

A. Isolated area 17 lesion - contralateral hemianopsia, often with macular sparing. Patients with isolated, unilateral area 17 lesions have been shown to differentiate X from 0, detect grating patterns, and reach accurately for objects in their blind field. These visual capacities in "blind" fields may reflect extrageniculocalcarine connections between pulvinar, superior colliculus or accessory optic tract with visual association cortex areas 18 and 19. There may also be some involvement of uncrossed non-classical visual projection system.

B. Bilateral area 17 lesions - This results in cortical blindness with pupillary reflexes spared and no optic atrophy. The patient may be able to tell if a light is turned on or off and may perceive some form and movement. Form is often reported as shades of gray.

Occipital Lobes Area 17

9. Deficits associated with lesions
   - Homonymous hemaniopsia of contralateral visual field follows unilateral lesion; scotoma, or "blind spot" follows subtotal lesion.
   - Amblyopias, areas of intermediate degree of change in visual function, are often found near scotomas
   - A field defect may be reported as blindness in the eye on that side.
   - Some loss of visual function may occur in intact half fields in homonymous hemianopsia.
   - Central scotomas may lead to subjectively normal visual fields with reduced acuity.
   - Macular sparing may occur if the lesion is close to the occipital pole.
   - Bilateral ablation may result in "cortical blindness" (inability to see, with denial and confabulation, sometimes called Anton's syndrome), or to subtotal blindness with residual ability to discriminate luminous flux and a speckled from a grey field.
   - Interruption of optic pathways may produce similar, but not always identical symptoms to lesions of area 17.
   - Acute trauma may cause warping of visual coordinates, distortions, and polyopias. These symptoms may disappear within days or last for months. They may reappear ictally
   - Toxic states may produce transitions from visual distortion to blindness and back again.

- Eye movement may be affected differentially by right or left occipital lesions; left lesions will shift to compensate for a scotoma while right lesions may not follow in the area of the scotoma.
- Left occipital lesions with involvement of the splenium of the corpus callosum may result in alexia without agraphia.
- Focal seizures consisting of visual sensations have been associated with lesions found in area 17

1. Tests for dysfunction
    - Perimetry allows definition of limits of scotoma. Harms perimeter allows definition in terms of contour, spectral values, intensity, as well as critical flicker frequency.
    - Amblyopias may also be tested with Harms perimeter; deficits are found in critical flicker frequency, dark adaptation, and two point resolution
    - Homonymous hemianopsia indicates cortical lesion or interruption of optic radiations, while bitemporal hemianopsia indicates interruption of the optic pathway at the optic chiasm. Monocular blindness results from unilateral destruction peripheral to the chiasm, and quadrantonopsia from selective interruption of the radiation fibers in one hemisphere, either temporal or nasal but not both.
    - Macular sparing indicates that the lesion involves the occipital cortex, rather than the optic radiations. Occlusion of the posterior cerebral artery should be suspected.
    - "Filling in" of a scotoma may be tested by presentation of a horizontal line crossing the visual field with a gap in the scotomatous area. Patients report seeing a completed line if filling in occurs
    - Eye movement can be tested by having the patient follow a moving target. Occipital lobe and posterior occulomotor lesion patients can do this; frontal lobe or anterior ocularmotor lesion patients cannot follow the verbal command with voluntary following. Right occipital lesions will not follow into the left half field.
    - Forced choice (guessing) discrimination of a stimulus may result in identification of objects within a scotoma, although if asked to identify a stimulus the patient reports he does not see it.

Areas 18 and 19

Deficits associated with lesions

- Visual recognition (agnosia) may result from bilateral lesions since visual input is disconnected from other sensory association areas.
- Reading is affected if visual input does not have access to left inferior parietal areas
- In man selective lesions of 18 and 19 do not take place. Some temporal or parietal involvement always occurs.
- Limited unilateral ablation may product deficits in visual following.

- A unilateral (usually right) lesion (especially with parietal involvement) results in unilateral spatial agnosia since eye movements fail to compensate for the lost left visual field.
- The degree of deficit is related to the extent of the lesion, i.e., how much parietal or temporal involvement.

Tests for dysfunction

- Sensory deficits such as rapid visual fatigue, impaired visual adaptation, and raised visual thresholds indicate involvement of 18 or 19.
- Visual following is tested by looking for opticokinetic nystagmus generated by following moving vertical black lines on a white background.
- Unilateral spatial agnosia is tested by picture copying (details on the left may be omitted) or card sorting (cards on the left are ignored).
- Tests for degree of bilateral deficit may be done in order from simplest to most complex: identification of clearly drawn pictures, identification of complex or indistinct pictures, identification of scribbled-on pictures, identification of hidden structures or Raven's Progressive Matrices Test.

C. Types of Visual Agnosias

Visual agnosia: an associative visual deficit in which perception and acuity are relatively normal, while the recognition and meaning of the percept are absent. This deficit is generally associated with lesions in the occipitoparietal and occipitotemporal region of the right hemisphere. Recently it has been postulated that bilateral lesions of the inferior longitudinal fasciculi interrupt the transfer of visual information from the occipital lobe to the temporal lobe and the limbic system, permitting processing for perception, but not for meaning, and producing the necessary and sufficient conditions for this syndrome. However, since the different types of visual agnosia sometimes occur dissociatively, others hypothesize discrete, although contiguous, pathways for interpreting visual stimuli, reading, identifying familiar faces, and color naming.

Visual object agnosia-a deficit in which visual perception is intact, but recognition and ascription of meaning to objects seen is impaired.

- This defect is associated with a right occipitoparietal lesion although similar left hemisphere lesions are frequently present.
- Test-naming of simple objects and pictures, naming of complex or ambiguous pictures, locating visual stimuli with interference or with pictures superimposed on each other, fragmented letters, the Benton Visual Retention Test, and WAIS Object Assembly.

Prosopagnosia-the inability to recognize familiar faces. This is a special type of visual object agnosia and often occurs with other visual agnosias.

- It is most frequently seen with right occipitoparietal and occipitotemporal lesions.
- Test-matching of facial pictures and identification of famous faces

Color agnosia-a defect of color recognition which has a variety of forms, primarily an inability to name or discriminate between colors. The color name may be given in a

qualified or concrete form or a confabulation may occur. It sometimes accompanies alexia and aphasia.

- Usually results from a left occipital or occipitotemporal lesion.
- Test-matching names to colors and colors to names, color sorting, arranging shades of a color by intensity, coloring pictures appropriately, identification of inappropriately colored objects.

Simultanagnosia-the inability to absorb more than one object or aspect of a visual stimulus at a time.

- This condition occurs with bilateral lesions of the occipitoparietal region.
- When accompanied by incoordination of ocular movements, this defect is known as Balint's syndrome. Test-have patient attempt to draw a line around shapes (circle, square, triangle), or write with eyes open, then closed (with Balint's syndrome, closing eyes improves handwriting).

Metamorphopsia-objects are correctly recognized but are subjectively distorted. Variations-include alterations in object size or in the obliquity of vertical and horizontal components, inversions of objects, waviness or fragmentation of lines, and apparent movement of stationary objects or their contours. In other variations, objects may appear alien or unusually familiar, very sinister or very beautiful, or endowed with special personal meaning.

- Lesions of the occipital lobe produce simpler deficits, while parietal lesions tend to produce intermediate effects, and temporal lesions are associated with more complex disorders.
- Teleopsia objects appear small and at a distance
- Pelopsia objects appear to loom up close
- Loss of stereoscopic vision
- Palinopsia (paliopsia) -- perseveratory illusions which are superimposed upon objects currently in the visual field. This defect usually occurs in the presence of a right occipitotemporal lesion.
- Test-assess qualitative visual changes via patient report and tasks tapping visual disorientation, inattention, fluctuation, delayed recognition of forms, imperfect synthesis of moving objects, and altered rate of flicker-fusion.

D. Integrative Function

1. Optic-Kinesthetic motor organization. Since movement entails a number of systems, it is necessary to check all components of the movement function in assessing a deficit.
    - Integrity of the visual-spatial basis of movement.
        - Impairment of this component is due to a lesion in the inferior parietal and/or parietoccipital region, especially on the right.
        - Test-have patient reproduce the examiner's hand and pencil movements with examiner sitting beside, then opposite, the patient .
    - Integrity of the kinesthetic basis of movement.

- Impairment of this component, or afferent apraxia, is usually due to a lesion in the postcentral gyrus contralateral to the impaired side of the body. Movements tend to be awkward or diffuse in character.
- Test-check basic sensory adequacy; then with eyes closed place patient's hand in one position and ask him to reproduce the position with the other hand; ask patient to reproduce position of examiner's hands or follow verbal commands for hand position without looking at his own.

- Integrity of motor or dynamic basis of movement.
  - Impairment of this component, or efferent apraxia is due to lesions of the premotor area, especially in the dominant hemisphere. It results in perseverative movements and the loss of continuity of movement.
  - Test-tasks requiring reciprocal coordination of both hands, alternating tapping patterns, ring-fist test, fist-edge-palm test, and drawing of alternating designs.

2. Word fluency has come to be associated with the left frontal area due to its disturbance with lesions in this region. It is hypothesized that the left hemisphere is necessary for verbal facility and the frontal area for the ability to switch sets.
   - Test-have patient write or say as many words as possible that begin with a given letter in a given time period.

3. Severe memory deficits have been reported with bilateral temporal lesions and these have been thought to be due to hippocampal damage. However, recently damage to temporal stem has been postulated to produce severe learning and retention deficits in the presence of intact hippocampi.
   - Test-memory for verbal story, hidden objects, design reproduction from memory, paired associates, and memory for four unrelated words after interference.

Angular Gyrus

A. Functions

1. Tertiary in function: lies at the boundary between the occipital, temporal, and postcentral regions of the hemisphere, where the cortical areas for visual, auditory, vestibular, cutaneous, and proprioceptive sensations overlap.
2. Supramodal in function: plays a special role in inter-analyzer syntheses. The angular gyrus, as part of the inferior parietal lobule, is the association area of association areas and allows cross modal transfer and associations between either vision or touch and hearing . As the angular gyrus is important in the processing of associating a heard name to a seen or felt object, it is probably also important for associations in the reverse direction. A "name" passes through Wernicke's area, then via the angular gyrus arouses associations in the other parts of the brain. Thus, the angular gyrus acts as a way station between the primary sensory modalities and the speech area.

3. The development of language is probably heavily dependent on this area. Object naming, one of the simplest aspects of language, depends on associations between other modalities and audition.
4. Association cortex that combines visual and auditory information necessary for reading and writing. Designed for storing the memory of the "rules of translation" from written to spoken language.

B. Behavioral deficits

1. Alexia without agraphia: results when the inferior parietal lobule is disconnected from all visual input. Pure word blindness results due to a disconnexion from the "memory centre".
    - Reading aloud and comprehension of written words is lost.
    - Ability to name and recognize objects is preserved. Objects have rich, multiple associations in other areas, e.g. one can recognize an apple by vision, touch, taste, smell, even by texture. The arousal of such associations permits the finding of an alternative pathway across an uninvolved more anterior portion of the corpus callosum.
    - Persistent difficulty in color naming but can match colors by hue without error.
    - Loss of ability to read music.
    - Spelling and spelling comprehension way he quite normal
    - Writing should be normal or nearly so; however, subtle defects can usually present (e.g. letters are too large or too widely spaced, there may be an absence or misuse of punctuation, capitals may be disregarded, letters dropped or reduplicated).
    - This syndrome is referred to as agnostic alexia by Brown. He states that a right hemanopia is an almost constant.

2. Alexia with agraphia: results from damage to the angular gyrus itself and renders the patient unable to read and write. May be referred to as aqraphic alexia or angular gyrus alexia.
    - A loss of visual word memory returns the patient to the state of being illiterate; lack of reading, writing, and spelling, and an incomprehension of spelled words are all components of this more primitive state.
    - Reading has a global character, without facilitation by literal analysis or letter tracing. Paralexia is present in reading aloud, especially for letters.
    - Letters are misnamed and patients cannot Indicate or sort letters accurately to command, unless first given a visual model of the letter tested, nor can they select the correct letter name from a spoken group. Patients are unable to match spoken letter sounds to written letters.
    - There is an inability to spell all but the simplest words, either to command or to a presented object.
    - Printing is variable, but always impaired. The agraphia reflects the spelling deficiency, as well as, in severe cases, the loss of conceptualization of words as whole units.

Although specific assessment devices have not been mentioned, it would appear that qualitative analysis of reading, writing, and spelling abilities is warranted in assessing the above syndromes.

Parieto-temporal-occipital cortex

This area is a tertiary, general sensory association area that integrates visual, tactile, and auditory information.

A. Lesions of this area will produce complex disorders that may include:

1. Constructional Apraxia: defects in copying designs and in drawing to command.
    - Left hemisphere lesions: ordering of movements is disrupted, simplification of drawings, difficulty making angles.
    - Right hemisphere lesions: more severe deficits such as visuo-spatial defects, neglect of left side of drawing, disproportions.
1. Difficulties in serial ordering: comprehension of order and sequence.
    - Left hemisphere lesions: disruption of sequential organization of speech.
    - Right hemisphere lesions: cannot understand temporal relationships and is unable to make future plans.
1. Visual memory disturbance: defective revisualization
    - Left hemisphere lesions: inability to evoke visual image in response to a given word.
    - Right hemisphere lesions: inability to retain visual image of nonverbal, spatial figures.
1. Impaired recognition and comprehension of complex, symbolic stimuli.
    - Left hemisphere lesions only
    - Symptoms of aphasia may also be seen.

B. Tests to measure the above deficits

1. Constructional apraxia
    - Copying designs: diamond, cross, cube, pipe.
    - Drawing to command: clock, daisy in flowerpot, house in perspective.
    - Match stick pattern test.
    - Block construction test.
1. Difficulties in serial ordering
    - Observations of spontaneous speech
    - Ability to order events in time: both life history events and objective events such as the Presidential terms .
1. Visual memory disturbance
    - Left hemisphere: Ask patient to describe objects that are not present
    - Right hemisphere: Short-term visual memory for geometric patterns
1. Impaired comprehension of complex symbolic stimuli
    - Ask patient to explain complex logico-gram-matical constructions such as "brother's father"
    - Give commands such as "draw a circle under a square"